Medical Education Systems, Inc.

EEG Principles

Electroencephalography

Learning Objectives

Upon successful completion of this course, you will be able to:

1. Define the term Electroencephalography (EEG) and explain its role in diagnostic medicine today.
2. List the clinical uses for EEG.
3. Identify the advantages and limitations of EEG in clinical use.
4. Explain what is meant by "artifacts" and provide some examples of the different types of artifacts.
5. Describe the use of artificial neural networks for automatic description and interpretation of the electroencephalogram.

Electroencephalography is the neurophysiologic measurement of the electrical activity of the brain by recording from electrodes placed on the scalp or, in special cases, subdurally or in the cerebral cortex. The resulting traces are known as an **electroencephalogram (EEG)** and represent an electrical signal (postsynaptic potentials) from a large number of neurons. These are sometimes called **brainwaves**, though this use is discouraged [1]. The EEG is a brain function test, but in clinical use it is a "gross correlate of brain activity" [2]. Electrical currents are not measured, but rather voltage differences between different parts of the brain.

EEGs are frequently used in experimentation because the process is non-invasive to the research subject. The subject does not need to make a decision or behavioral action in order to log data, and it can detect covert responses to stimuli, such as reading. The EEG is capable of detecting changes in electrical activity in the brain on a millisecond-level. It is one of the few techniques available that has such high temporal resolution. The other common technique is MEG.

(***Magnetoencephalography (MEG)*** *is an imaging technique used to measure the magnetic fields produced by electrical activity in the brain via extremely sensitive devices such as SQUIDs. These measurements are commonly used in both research and clinical settings. There are many uses for the MEG, including assisting surgeons in localizing a pathology, assisting researchers in determining the function of various parts of the brain, neurofeedback, and others*.)

Clinical use

EEG in various forms is most useful as a tool for monitoring and diagnosis in certain clinical situations:

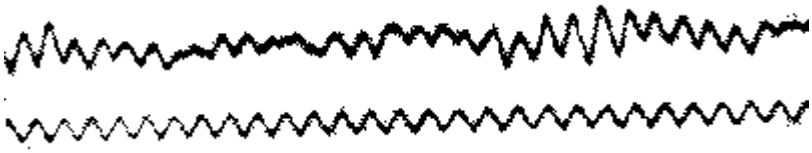
- **epilepsy and syncope (fainting)**
- **sleep disorders**
- **coma and brain death**

It is sometimes useful in assessing dementia, when other examinations are equivocal. In some jurisdictions it has a legal significance and formal criteria are used to assess brain death. Current

research is being done to determine if EEG may also be used to help monitor clinical depression treatment, but such studies are still in the clinical stages.

Research use

Neuroscientists and biological psychiatrists use EEGs to study the function of the brain by recording cerebral activity during controlled behavior of human volunteers and animals in lab experiments. Theories to explain sleep often rely on EEG patterns recorded during sleep sessions.



The first EEG recording, obtained by Hans Berger in 1924.

Methods

In conventional scalp EEG, the recording is obtained by placing electrodes on the scalp, usually after preparing the scalp area by light abrasion and application of a conductive gel to reduce impedance. Each electrode is connected to an input of a differential amplifier (one amplifier per pair of electrodes), which amplifies the voltage between them (typically 1,000–100,000 times, or 60–100 dB of voltage gain). The resulting voltage signal is filtered by a high-pass filter and a low-pass filter, typically set at 0.5 hertz and 35-70 Hz, respectively. The high-pass filter typically filters out slow electrogalvanic signals, whereas the low-pass filter filters out electromyographic signals.

The filtered signal is then output on paper (in older systems), or displayed on a computer screen. The amplitude of the EEG is about 100 µV when measured on the scalp, and about 1-2 mV when measured on the surface of the brain.

The electrode-amplifier relationships are typically arranged in one of three ways:

Common reference derivation
: One terminal of each amplifier is connected to the same electrode, and all other electrodes are measured relative to this single point. It is typical to use a reference electrode placed somewhere along the scalp midline, or a reference that links both earlobe electrodes.

Average reference derivation
: The outputs of all of the amplifiers are summed and averaged, and this averaged signal is used as the common reference for each amplifier.

Bipolar derivation
: The electrodes are connected in series to an equal number of amplifiers. For example, amplifier 1 measures the difference between electrodes A and B, amplifier 2 measures the difference between B and C, and so on.

This distinction has become void with the advent of digital or *paperless* EEGs, which record all electrodes against an arbitrary reference and will calculate the above relationships (called *montages*) post hoc.

Perspectives:

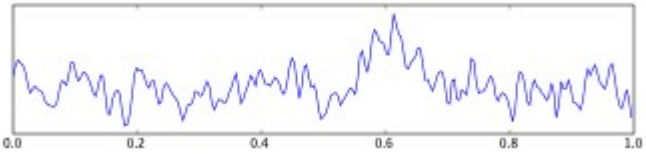
Limitations

EEG has several limitations. Scalp electrodes are not sensitive enough to pick out individual action potentials, the electric unit of signaling in the brain, or whether the resulting electrical activity is releasing inhibitory, excitatory or modulatory neurotransmitters. Instead, the EEG picks up the activity of large groups of neurons, which produces a greater voltage than the firing of an individual neuron. Secondly, EEG has limited anatomical specificity when compared with other functional brain imaging techniques such as functional magnetic resonance imaging (fMRI). Some anatomical specificity can be gained with the use of EEG topography, which uses a large number of electrodes to triangulate the source of the electrical activity.

Advantages

EEG has several strong sides as a tool of exploring the brain activity. The time resolution is very high. As other methods for researching brain activity have time resolution between seconds and minutes, the EEG has a resolution down to sub-millisecond. As the brain is thought to work through its electric activity, EEG is the only method to measure it directly. Other methods for exploring functions in the brain rely on blood flow or metabolism which may be decoupled from the brain electric activity. Newer research typically combines EEG or MEG with MRI or PET to get high temporal and spatial resolution.

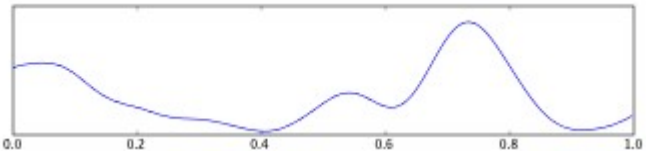
Activity types



One second of EEG signal

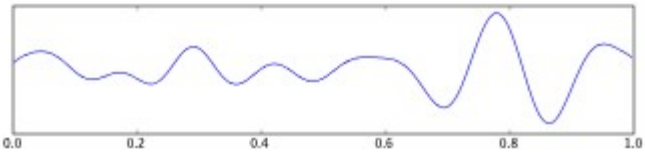
Historically four major types of continuous rhythmic sinusoidal EEG activity are recognized (alpha, beta, delta and theta). There is no precise agreement on the frequency ranges for each type.

- **Delta is the frequency range up to 4 Hz and is often associated with the very young and certain encephalopathies and underlying lesions. It is seen in stage 3 and 4 sleep.**



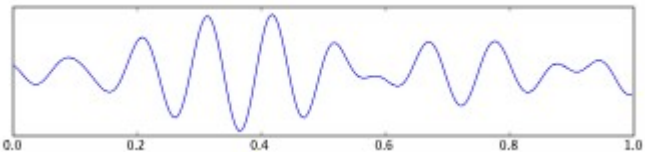
Delta waves.

- Theta is the frequency range from 4 Hz to 8 Hz and is associated with drowsiness, childhood, adolescence and young adulthood. This EEG frequency can sometimes be produced by hyperventilation. Theta waves can be seen during hypnagogic states such as trances, hypnosis, deep day dreams, lucid dreaming and light sleep and the preconscious state just upon waking, and just before falling asleep.



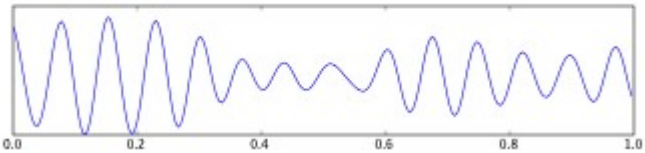
Theta waves.

- Alpha (Berger's wave) is the frequency range from 8 Hz to 12 Hz. It is characteristic of a relaxed, alert state of consciousness. Alpha rhythms are best detected with the eyes closed. Alpha attenuates with drowsiness and open eyes, and is best seen over the occipital (visual) cortex. An alpha-like normal variant called mu is sometimes seen over the motor cortex (central scalp) and attenuates with movement, or rather with the intention to move.



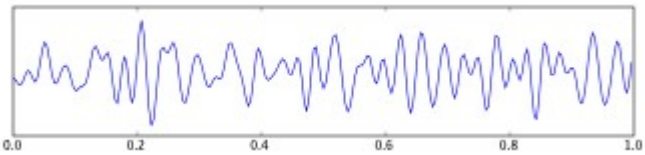
Alpha waves.

- **sensorimotor rhythm** (SMR) is a middle frequency (about 12–16 Hz) associated with physical stillness and body presence.



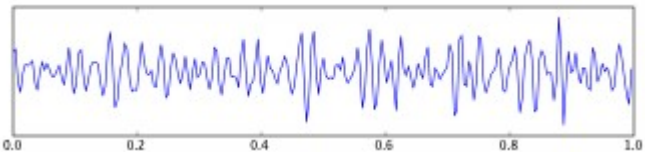
SMR waves.

- **Beta** is the frequency range above 12 Hz. Low amplitude beta with multiple and varying frequencies is often associated with active, busy or anxious thinking and active **concentration.** Rhythmic beta with a dominant set of frequencies is associated with various pathologies and drug effects, especially benzodiazepines.



Beta waves.

- Gamma is the frequency range approximately 26–100 Hz. Gamma rhythms appear to be involved in higher mental activity, including perception, problem solving, fear, and consciousness.



Gamma waves.

Rhythmic slow activity in wakefulness is common in young children, but is abnormal in adults. In addition to the above types of rhythmic activity, individual transient waveforms such as sharp waves, spikes, spike-and-wave complexes occur in epilepsy, and other types of transients occur during sleep.

In the transition from wakefulness, through Stage I sleep (drowsiness), Stage II (light) sleep, to Stage III and IV (deep) sleep, first the alpha becomes intermittent and attenuated, then disappears. Stage II sleep is marked by brief bursts of highly rhythmic beta activity (sleep spindles) and K complexes (transient slow waves associated with spindles, often triggered by an auditory stimulus). Stage III and IV are characterized by slow wave activity. After a period of deep sleep, the sleeper cycles back to stage II sleep and/or rapid eye movement (REM) sleep, associated with dreaming. These cycles may occur many times during the night.

EEG under general anesthesia depends on the type of anesthetic employed. With halogenated anesthetics and intravenous agents such as propofol, a rapid (alpha or low beta), nonreactive EEG pattern is seen over most of the scalp, especially anteriorly; in some older terminology this was known as a WAR (widespread anterior rapid) pattern, contrasted with a WAIS (widespread slow) pattern associated with high doses of opiates.

Artifacts

Biological Artifacts

Signals in the EEG that are of non-cerebral origin are called artifacts. The EEG is nearly always contaminated by such signals. This is one of the reasons why it takes considerable experience to interpret EEGs clinically. The most common types of artifacts are:

- Eye artifacts (including eyeball, ocular muscles and eyelid)
- EKG artifacts
- EMG artifacts
- Glossokinetic artifacts

Eyeball artifacts are caused by the potential difference between the cornea and retina, which is quite large compared to cerebral potentials. When the eye is completely still, this is not a problem. But there are nearly always small or large reflexive eye movements, which generates a potential which is picked up in the frontopolar and frontal leads. Eye movements - whether vertical or horizontal [saccades] - are caused by ocular muscles, which also generate electromyographic potentials. Purposeful or reflexive eye blinking also generates electromyographic potentials, but more importantly there is reflexive movement of the eyeball during blinking which gives a characteristic artefactual appearance of the EEG (see Bell's phenomenon).

Some of these artifacts are useful. Eye movements are very important in polysomnography, and is also useful in conventional EEG for assessing possible changes in alertness, drowsiness or sleep.

EKG artifacts are quite common and can be mistaken for spike activity. Because of this, modern EEG acquisition commonly includes a one-channel EKG from the extremeties. This also allows the EEG to identify cardiac arrythmias that are an important differential diagnosis to syncope or other episodic/attack disorders. Glossokinetic artifacts are caused by the potential difference between the

base and the tip of the tongue. Minor tongue movements can contaminate the EEG, especially in parkinsonian and tremor disorders.

External Artifacts

In addition to internal artifacts, there are many artifacts which originate from outside the patient. Movement by the patient, or even just settling of the electrodes, may cause *electrode pops*, spikes originating from a momentary change in the impedence of a given electrode. From a completely different source, within the United States, poor grounding of the EEG electrodes can cause a significant 60 Hz artifact (50 Hz in many other countries). A third source of possible interference can be the presence of an IV drip; such devices can cause rhythmic, fast, low-voltage bursts, which may be confused for spikes.

Artifact Correction

Recently, source decomposition techniques have been used to "correct" or "remove" EEG artifacts. These source decomposition models, in one way or another, assume the ability to "unmix" EEG signal into some number of independent sources. If one happens to agree with the principle behind a particular decomposition approach, then there is no argument against "remixing" only those sources that do not resemble artifact. There is no proof yet as to the validity or preciseness of these methods since simulated EEG recordings are the only way to know beforehand, the exact properties of the uncontaminated signal. In reality, EEG is to some extent stochastic.

History

A brief timeline is given here [3]. Richard Caton (1842–1926), a physician practicing in Liverpool, presented his findings about electrical phenomena of the exposed cerebral hemispheres of rabbits and monkeys in the British Medical Journal in 1875. In 1890, Beck publishes an investigation of spontaneous electrical activity of the brain of rabbits and dogs which included rhythmic oscillations altered by light.

In 1912, Russian physiologist, Vladimir Vladimirovich Pravdich-Neminsky published the first EEG and the evoked potential of the mammalian (dog)[4]. In 1914, Cybulsky and Jelenska-Macieszyna photograph EEG-recordings of experimentally induced seizures.

German physiologist Hans Berger (1873–1941) began his studies of the human EEG in 1920. He gave the device its name and is sometimes credited with inventing the EEG, though others had performed similar experiments. His work was later expanded by Edgar Douglas Adrian.

In 1934, Fisher and Lowenback first demonstrate epileptiform spikes. In 1935 Gibbs, Davis and Lennox describe interictal spike waves and the 3 cycles/s pattern of clinical absence seizures, beginning the field of clinical electroencephalography. In 1936 Gibbs and Jasper report the interictal spike as the focal signature of epilepsy. The same year, the first EEG laboratory opened at Massachusetts General Hospital.

Franklin Offner (1911-1999), professor of biophysics at Northwestern University developed a prototype of the EEG which incorporated a piezoelectronic inkwriter called a Crystograph (the whole device was typically known as the Offner Dynograph).

In 1947, The American EEG Society is founded and the first International EEG congress is held. In 1953 Aserinsky and Kleitmean describe REM sleep.

In the 1950s, English physician William Grey Walter developed an adjunct to EEG called EEG topography which allowed for the mapping of electrical activity across the surface of the brain. This enjoyed a brief period of popularity in the 1980s and seemed especially promising for psychiatry. It was never accepted by neurologists and remains primarily a research tool.

In 2004, Antoine Lutz et al., collaborating with Richard J. Davidson, reported that long-term meditators could "self-induce high-amplitude gamma synchrony during mental practice" in the Proceedings of the National Academy of Sciences[5].

References

1. Cobb, WA (1983). *Recommendations for the practice of clinical neurophysiology*. Amsterdam: Elsevier.
2. (2002) John S. Ebersole: *Current Practice of Clinical Electroencephalography*. Lippincott Williams & Wilkins.
3. Swartz, B.E (1998). "Timeline of the history of EEG and associated fields". *Electroencephalography and clinical Neurophysiology* 106: 173-176.
4. Pravdich-Neminsky VV. Ein Versuch der Registrierung der elektrischen Gehirnerscheinungen (In German). Zbl Physiol 27: 951–960, 1913.
5. Antoine Lutz et al. "Long-term meditators self-induce high-amplitude gamma synchrony during mental practice". *Proceedings of the National Academy of Sciences* 101:46, 16369-16373, 2004. (full text)

Electroencephalography Overview

INTRODUCTION

The first recording of the electric field of the human brain was made by the German psychiatrist Hans Berger in 1924 in Jena. He gave this recording the name *electroencephalogram (EEG)*. (Berger, 1929). (From 1929 to 1938 he published 20 scientific papers on the EEG under the same title "Über das Elektroenkephalogram des Menschen".)

1. spontaneous activity,
2. evoked potentials, and
3. bioelectric events produced by single neurons.

Spontaneous activity is measured on the scalp or on the brain and is called the electroencephalogram. The amplitude of the EEG is about 100 µV when measured on the scalp, and about 1-2 mV when measured on the surface of the brain. The bandwidth of this signal is from

under 1 Hz to about 50 Hz, as demonstrated in Figure 13.1. As the phrase "spontaneous activity" implies, this activity goes on continuously in the living individual.

Evoked potentials are those components of the EEG that arise in response to a stimulus (which may be electric, auditory, visual, etc.) Such signals are usually below the noise level and thus not readily distinguished, and one must use a train of stimuli and signal averaging to improve the signal-to-noise ratio.

Single-neuron behavior can be examined through the use of microelectrodes which impale the cells of interest. Through studies of the single cell, one hopes to build models of cell networks that will reflect actual tissue properties.

THE BRAIN AS A BIOELECTRIC GENERATOR

PRECONDITIONS:
SOURCE: *Distribution of impressed current source elements* $\bar{J}^{i}$ *(volume source)*
CONDUCTOR: *Finite, inhomogeneous*

The number of nerve cells in the brain has been estimated to be on the order of 10^{11}. Cortical neurons are strongly interconnected. Here the surface of a single neuron may be covered with 1,000-100,000 synapses (Nunez, 1981). The electric behavior of the neuron corresponds to the description of excitable cells introduced in the earlier chapters. The resting voltage is around -70 mV, and the peak of the action potential is positive. The amplitude of the nerve impulse is about 100 mV; it lasts about 1 ms.

The bioelectric impressed current density $\bar{J}^{i}$ associated with neuronal activation produces an electric field, which can be measured on the surface of the head or directly on the brain tissue. The electric field was described by Equation 7.10 for a finite inhomogeneous model. This equation is repeated here:

$$4\pi\sigma\Phi(r) = \int_{v} \bar{J}^{i} \bullet \nabla\left(\frac{1}{r}\right) dv + \sum_{j} \int_{sj} \left(\sigma_j'' - \sigma_j'\right) \Phi \nabla\left(\frac{1}{r}\right) \bullet d\bar{S}_j \qquad \textbf{(13.01)}$$

While for most excitable tissue the basis for the impressed current density $\bar{J}^{i}$ is the propagating action potential, for the EEG it appears to arise from the action of a chemical transmitter on postsynaptic cortical neurons. The action causes localized depolarization - that is, an excitatory postsynaptic potential (EPSP) - or hyperpolarization - that is, an inhibitory postsynaptic potential (IPSP). The result in either case is a spatially distributed discontinuity in the function $\sigma\Phi$ (i.e., $\sigma_o\Phi_o$ - $\sigma_i\Phi_i$) which, as pointed out in Equation 8.28, evaluates a double layer source in the membranes of all cells. This will be zero for resting cells; however, when a cell is active by any of the aforementioned processes (in which case Φ_o - $\Phi_i = V_m$ varies over a cell surface), a nonzero primary source will result.

For distant field points the double layer can be summed up vectorially, yielding a net dipole for each active cell. Since neural tissue is generally composed of a very large number of small, densely packed

cells, the discussion in Section 8.5 applies, leading to the identification of a continuous volume source distribution $\bar{J}^i$ which appears in Equations 7.6 and 7.10.

Although in principle the EEG can be found from the evaluation of Equation 7.10, the complexity of brain structure and its electrophysiological behavior have thus far precluded the evaluation of the source function $\bar{J}^i$. Consequently, the quantitative study of the EEG differs from that of the ECG or EMG, in which it is possible to evaluate the source function. Under these conditions the quantitative EEG is based on a statistical treatment, whereas the clinical EEG is largely empirical..

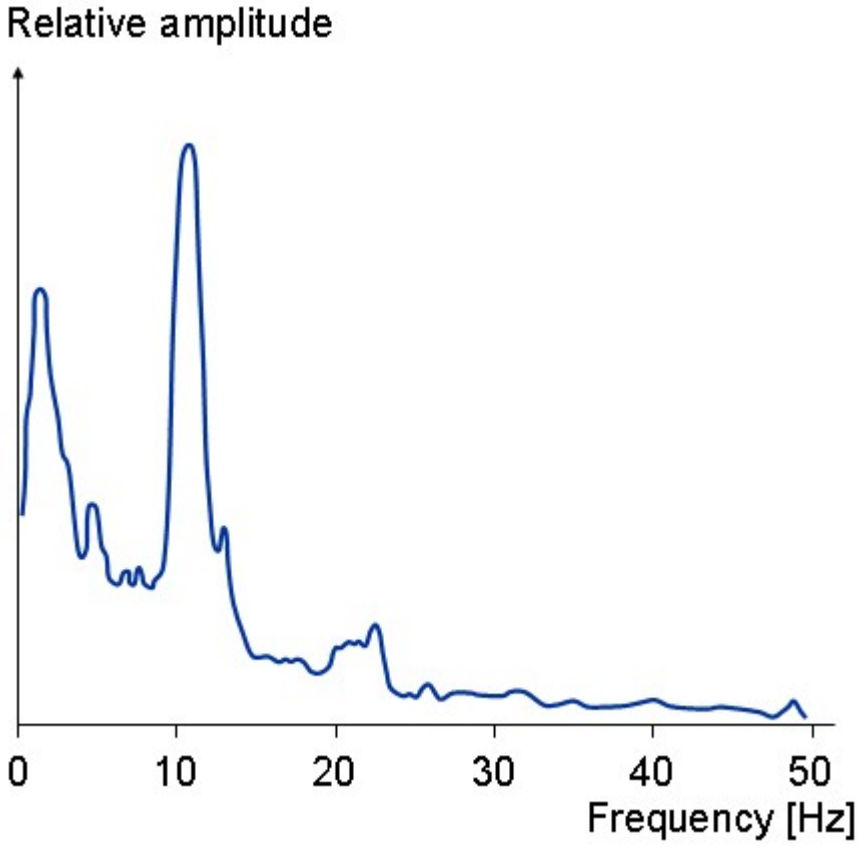


Fig. 13.1. Frequency spectrum of normal EEG.

EEG LEAD SYSTEMS

The internationally standardized *10-20 system* is usually employed to record the spontaneous EEG. In this system 21 electrodes are located on the surface of the scalp, as shown in Figure 13.2A and B. The positions are determined as follows: Reference points are *nasion*, which is the delve at the top of the nose, level with the eyes; and *inion*, which is the bony lump at the base of the skull on the midline at the back of the head. From these points, the skull perimeters are measured in the transverse and median planes. Electrode locations are determined by dividing these perimeters into 10% and 20% intervals. Three other electrodes are placed on each side equidistant from the neighboring points, as shown in Figure 13.2B (Jasper, 1958; Cooper, Osselton, and Shaw, 1969).

In addition to the 21 electrodes of the international 10-20 system, intermediate 10% electrode

positions are also used. The locations and nomenclature of these electrodes are standardized by the American Electroencephalographic Society (Sharbrough et al., 1991; see Figure 13.2C). In this recommendation, four electrodes have different names compared to the 10-20 system; these are T_7, T_8, P_7, and P_8. These electrodes are drawn black with white text in the figure.

Besides the international 10-20 system, many other electrode systems exist for recording electric potentials on the scalp. The *Queen Square system* of electrode placement has been proposed as a standard in recording the pattern of evoked potentials in clinical testings (Blumhardt et al., 1977).

Bipolar or unipolar electrodes can be used in the EEG measurement. In the first method the potential difference between a pair of electrodes is measured. In the latter method the potential of each electrode is compared either to a neutral electrode or to the average of all electrodes (see Figure 13.3).

The most recent guidelines for EEG-recording are published in (Gilmore, 1994).

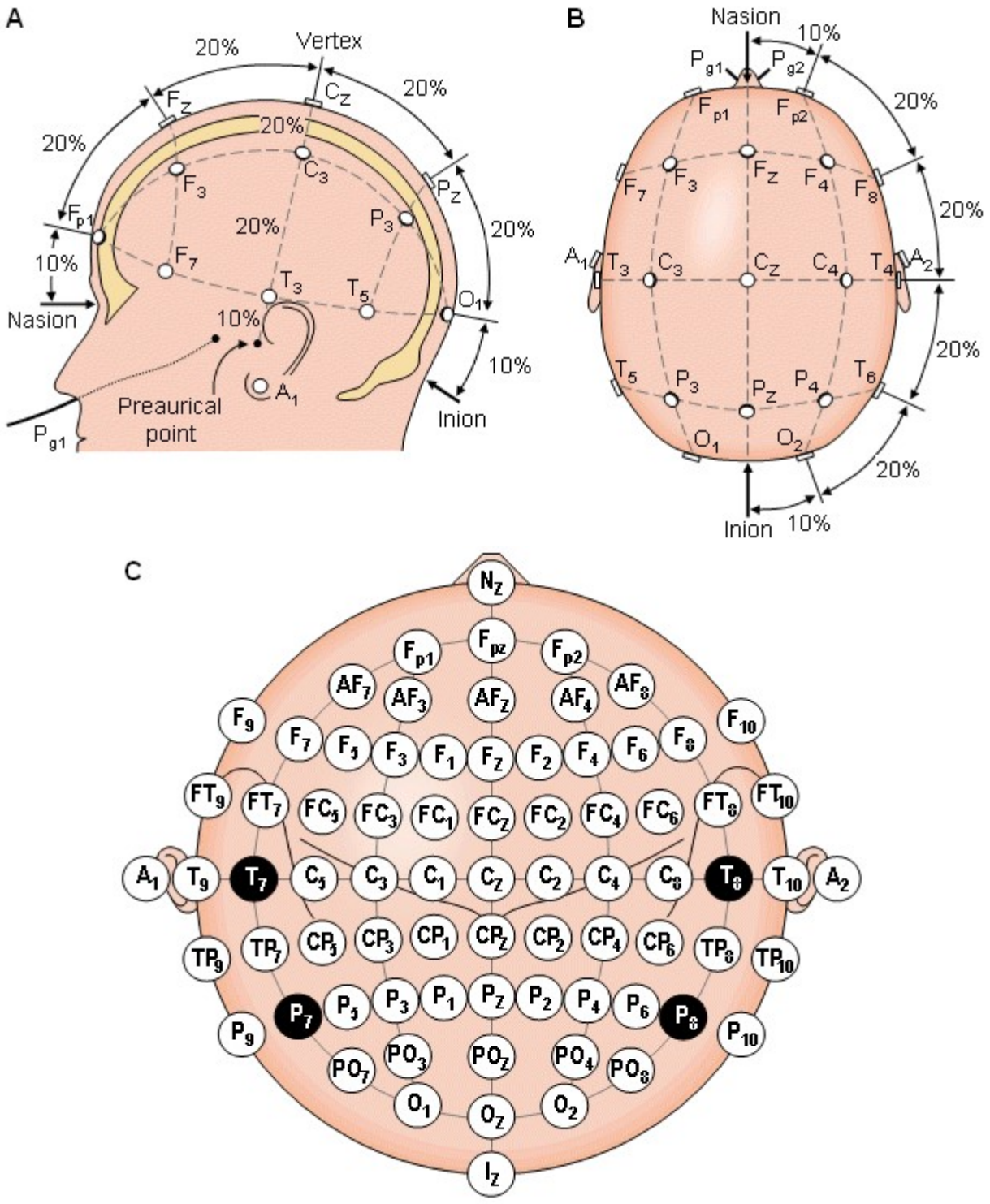


Fig. 13.2. The international 10-20 system seen from (A) left and (B) above the head. A = Ear lobe, C = central, Pg = nasopharyngeal, P = parietal, F = frontal, Fp = frontal polar, O = occipital.
(C) Location and nomenclature of the intermediate 10% electrodes, as standardized by the American Electroencephalographic Society. (Redrawn from Sharbrough, 1991.).

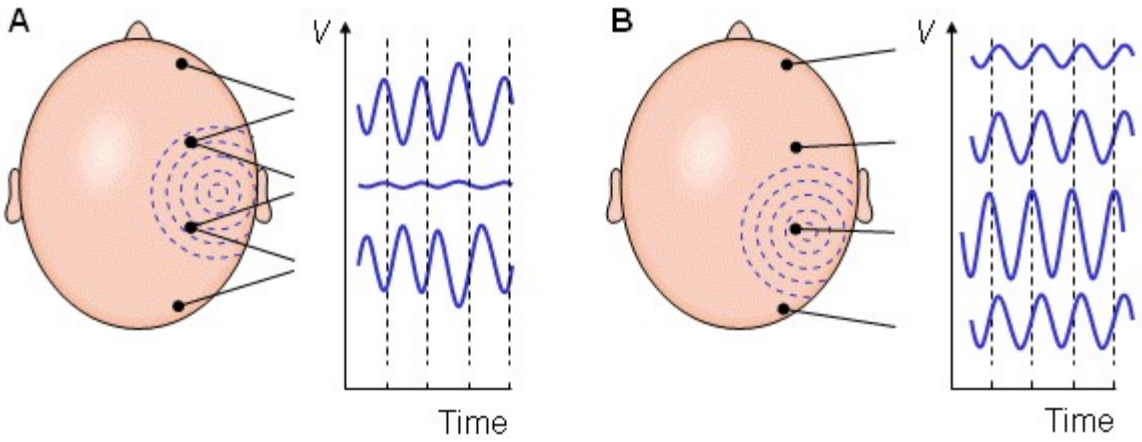


Fig. 13.3. (A) Bipolar and (B) unipolar measurements. Note that the waveform of the EEG depends on the measurement location.

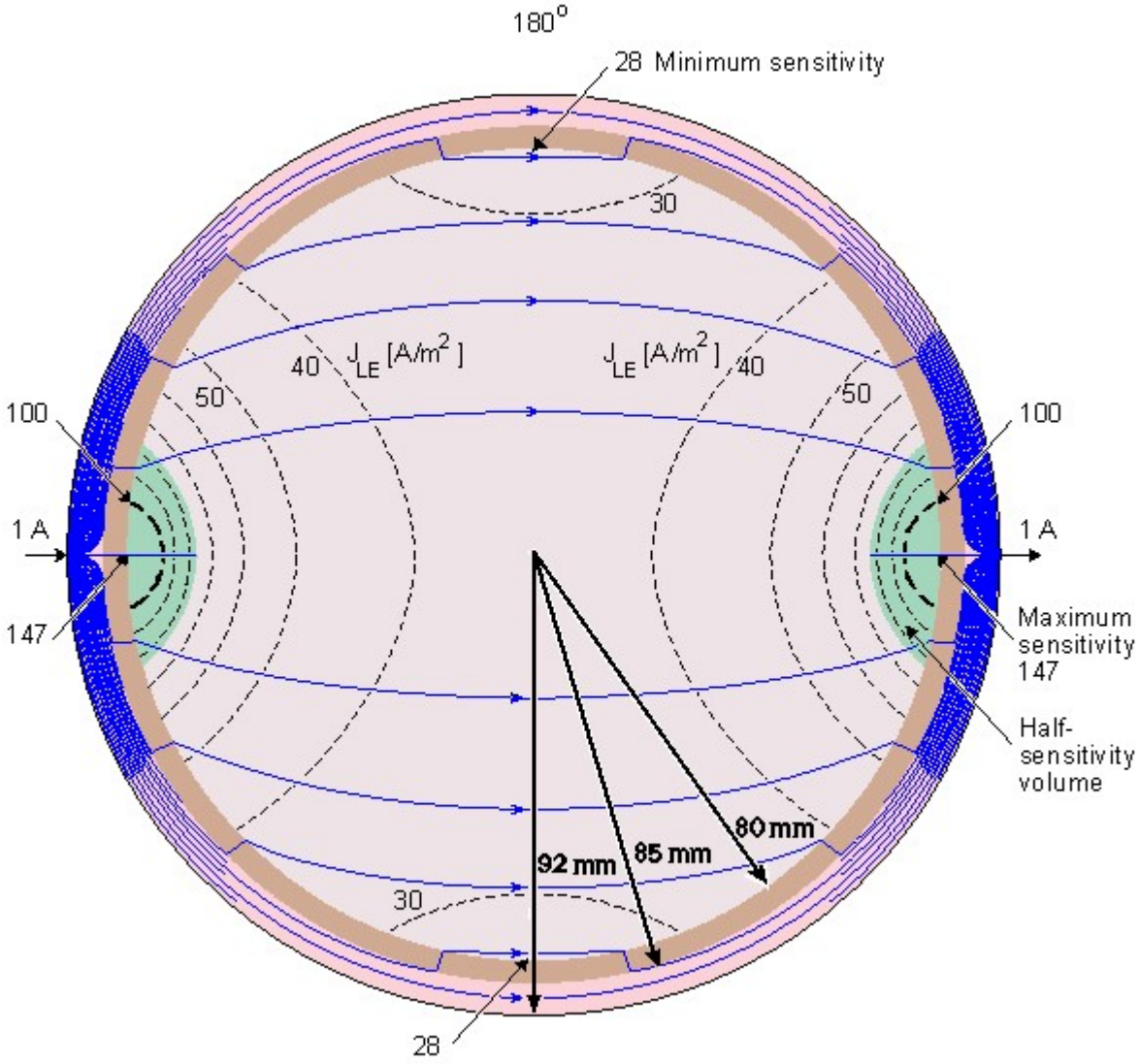
SENSITIVITY DISTRIBUTION OF EEG ELECTRODES

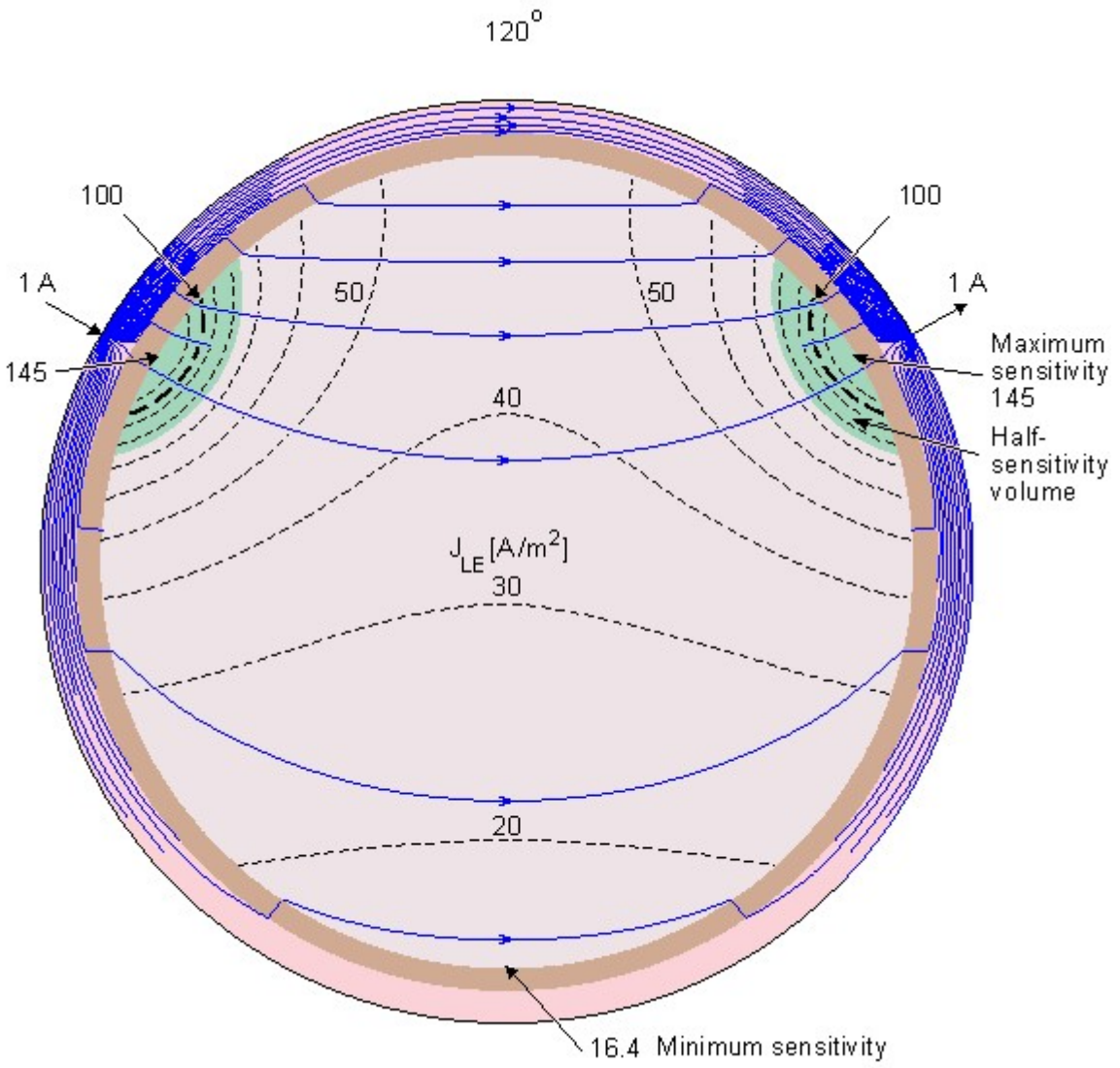
Rush and Driscoll (1969) calculated the sensitivity distribution of bipolar surface electrodes on the scalp based on a concentric spherical head model. They published the results in the form of lead field isopotential lines. The direction of the lead field current density - that is, the direction of the sensitivity - is a negative gradient of the potential field. This is not immediately evident from such a display.

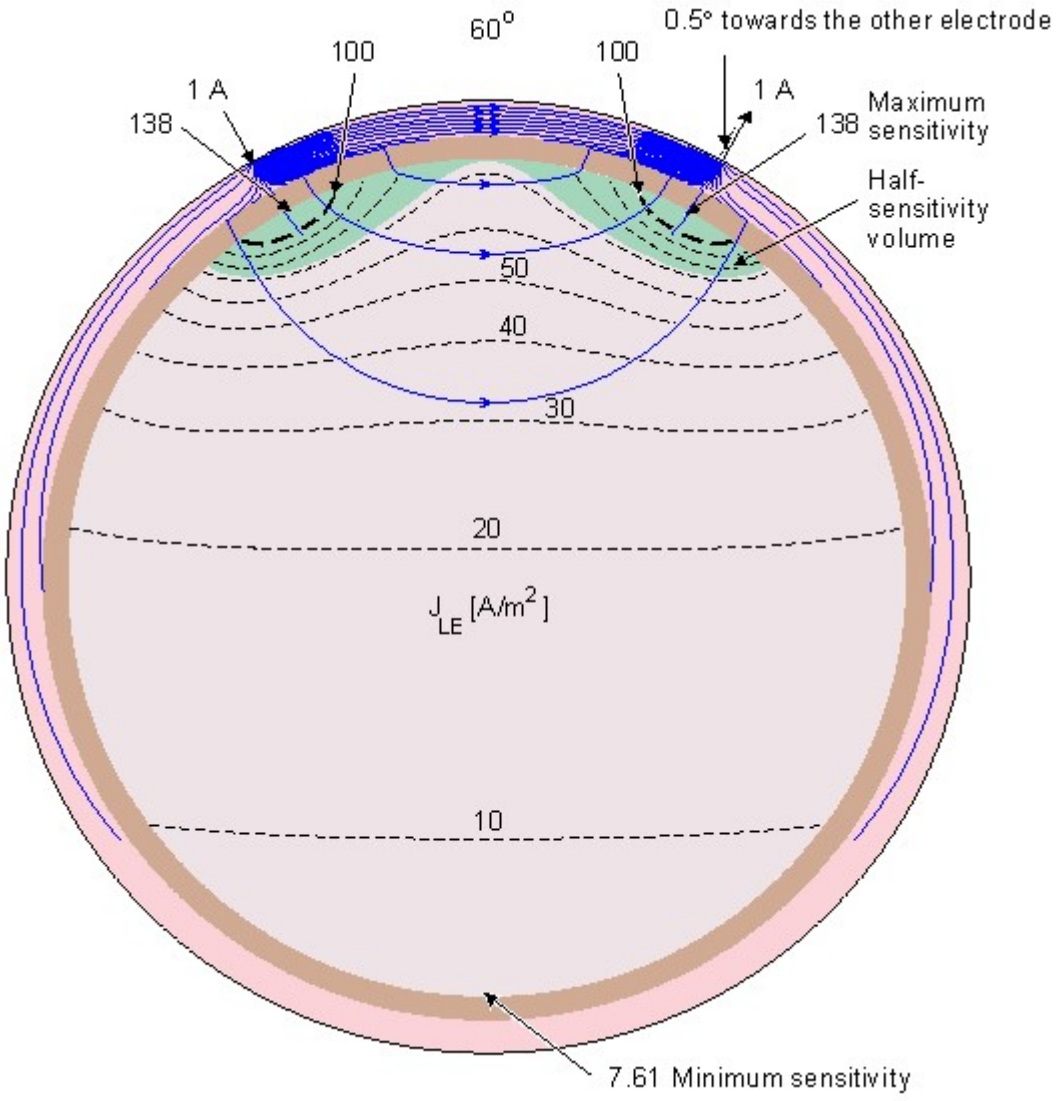
Puikkonen and Malmivuo (1987) recalculated the sensitivity distribution of EEG electrodes with the same model as Rush and Driscoll, but they presented the results with the lead field current flow lines instead of the isopotential lines of the lead field. This display is illustrative since it is easy to find the direction of the sensitivity from the lead field current flow lines. Also the magnitude of the sensitivity can be seen from the density of the flow lines. A minor problem in this display is that because the lead field current distributes both in the plane of the illustration as well as in the plane normal to it, part of the flow lines must break in order to illustrate correctly the current density with the flow line density in a three-dimensional problem. Suihko, Malmivuo and Eskola (1993) calculated further the isosensitivity lines and the *half-sensitivity volume* for the electric leads. As discussed in Section 11.6.1, the concept half-sensitivity volume denotes the area where the lead field current density is at least one half from its maximum value. Thus this concept is a figure of merit to describe how concentrated the sensitivity distribution of the lead is. As discussed in Section 11.6.6, when the conductivity is isotropic, as it is in this head model, the isosensitivity lines equal to the isofield lines of the (reciprocal) electric field. If the lead would exhibit such a symmetry that adjacent isopotential surfaces would be a constant distance apart, the isosensitivity lines would coincide with the isopotential lines. That is not the case in the leads of Figure 13.4.

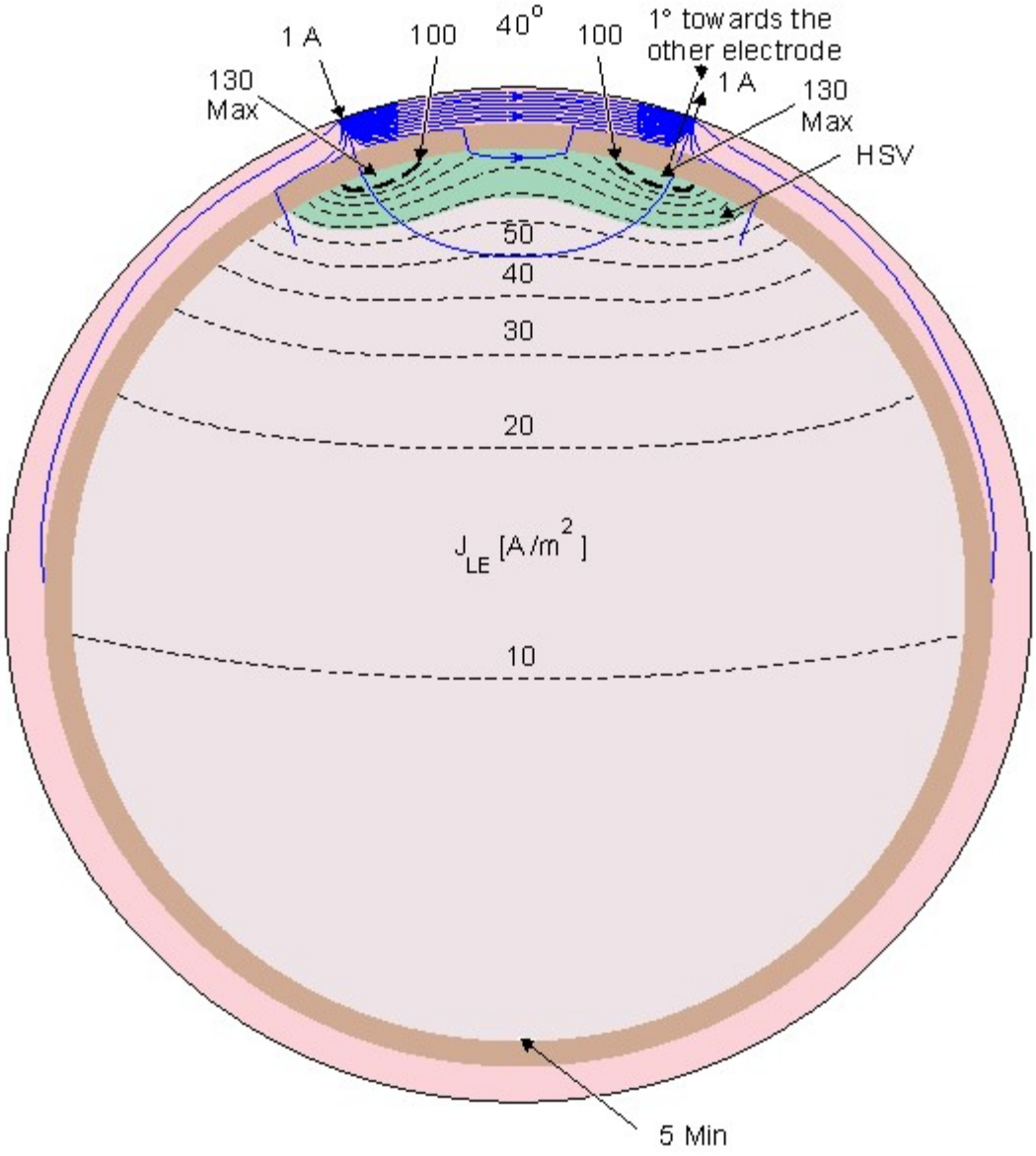
Figure 13.4 displays the lead field current flow lines, isosensitivity lines and half-sensitivity volumes for the spherical head model with the electrodes located within 180°, 120°, 60°, 40°, and 20° angles. Note that in each case the two electrodes are connected with 10 continuous lead field flow lines. Between them are three flow lines which are broken from the center, indicating that the lead field current distributes also in the plane normal to the paper. The figure shows clearly the strong effect of the poorly conducting skull to the lead field. Though in a homogeneous model the sensitivity would be

highly concentrated at the electrodes, in the 180° case the skull allows the sensitivity to be very homogeneously distributed throughout the brain region. The closer the electrodes are to each other, the smaller the part of the sensitivity that locates within the brain region. Locating the electrodes closer and closer to each other causes the lead field current to flow more and more within the skin region, decreasing the sensitivity to the brain region and increasing the noise.




120°
100
100
1 A
1 A
50
50
145
40
Maximum sensitivity 145
Half-sensitivity volume
J_{LE} [A/m^2]
30
20
16.4 Minimum sensitivity


60°
0.5° towards the other electrode
100
100
1 A
1 A
138
138
Maximum sensitivity
Half-sensitivity volume
50
40
30
20
J_{LE} [A/m^2]
10
7.61 Minimum sensitivity


1 A
100
40°
100
1° towards the other electrode
1 A
130 Max
130 Max
HSV
50
40
30
20
J_{LE} [A/m^2]
10
5 Min

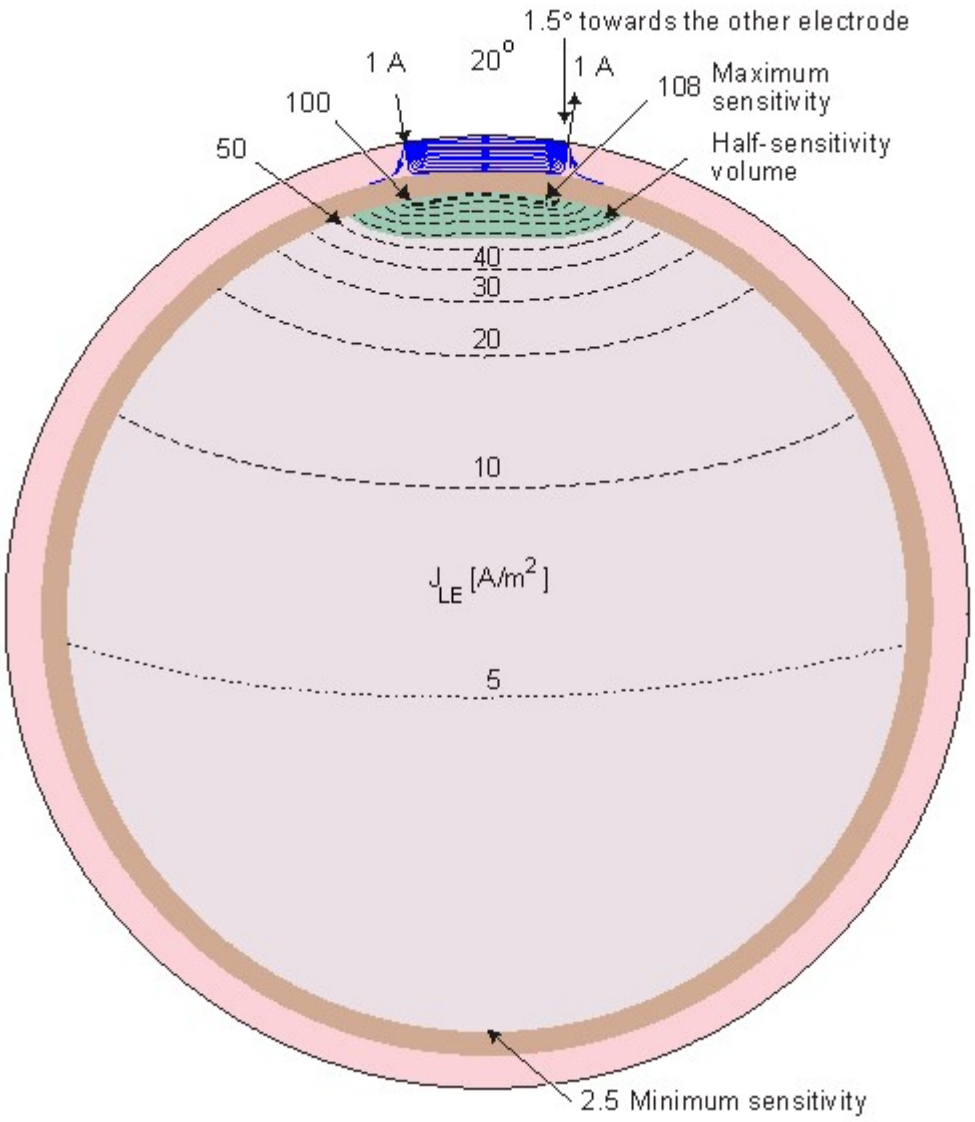


Fig. 13.4. Sensitivity distribution of EEG electrodes in the spherical head model. The figure illustrates the lead field current flow lines (thin solid lines), isosensitivity lines (dotted lines) and the half-sensitivity volumes (shaded region). The sensitivity distribution is in the direction of the flow lines, and its magnitude is proportional to the density of the flow lines. The lead pair are designated by small arrows at the surface of the scalp and are separated by an angle of 180°, 120°, 60°, 40°, and 20° shown at the top of each figure.

THE BEHAVIOR OF THE EEG SIGNAL

From the EEG signal it is possible to differentiate alpha (α), beta (β), delta (δ), and theta (Θ) waves as well as spikes associated with epilepsy. An example of each waveform is given in Figure 13.5.

The alpha waves have the frequency spectrum of 8-13 Hz and can be measured from the occipital region in an awake person when the eyes are closed. The frequency band of the beta waves is 13-30 Hz; these are detectable over the parietal and frontal lobes. The delta waves have the frequency range

of 0.5-4 Hz and are detectable in infants and sleeping adults. The theta waves have the frequency range of 4-8 Hz and are obtained from children and sleeping adults..

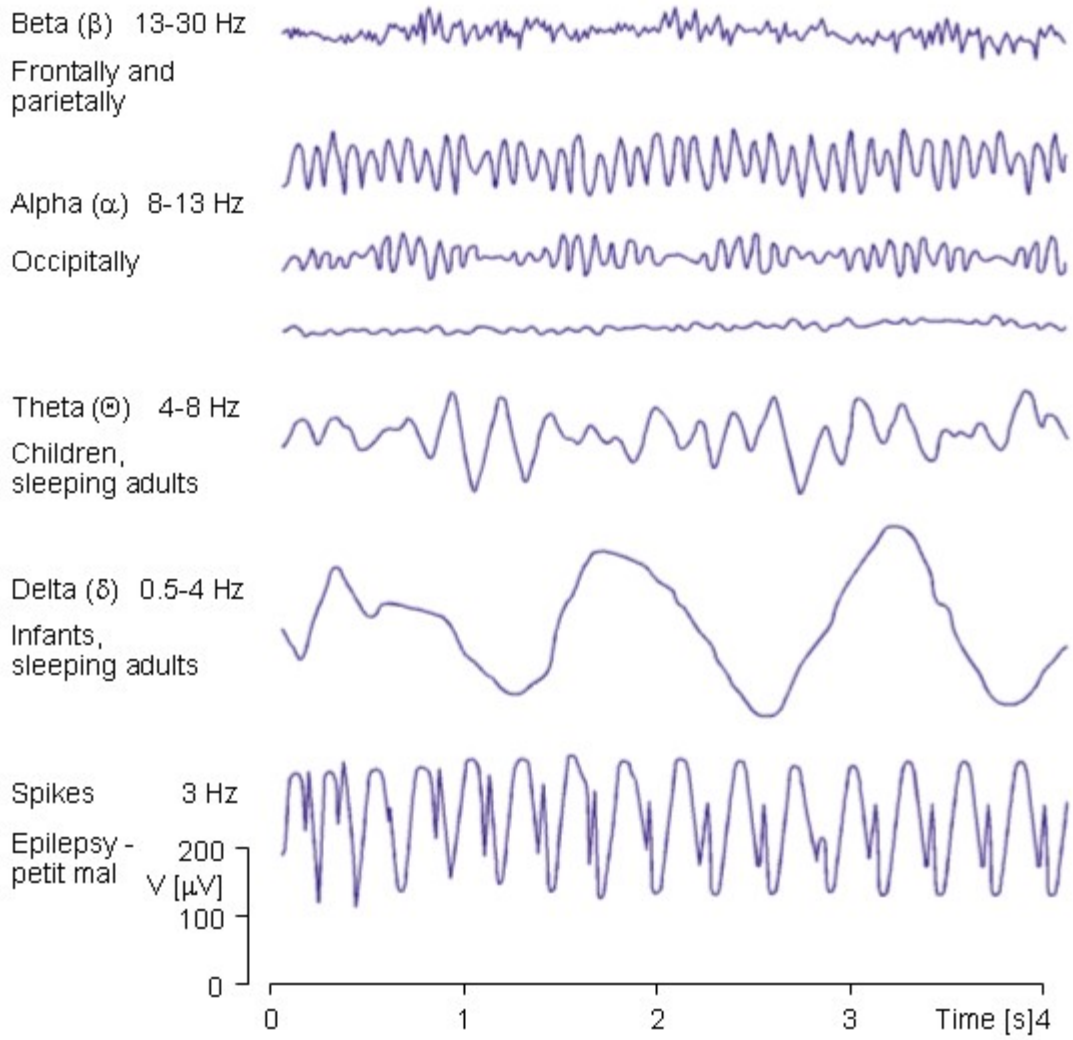


Fig. 13.5. **Some examples of EEG waves.**

THE BASIC PRINCIPLES OF EEG DIAGNOSIS

The EEG signal is closely related to the level of consciousness of the person. As the activity increases, the EEG shifts to higher dominating frequency and lower amplitude. When the eyes are closed, the alpha waves begin to dominate the EEG. When the person falls asleep, the dominant EEG frequency decreases. In a certain phase of sleep, rapid eye movement called (REM) sleep, the person dreams and has active movements of the eyes, which can be seen as a characteristic EEG signal. In deep sleep, the EEG has large and slow deflections called delta waves. No cerebral activity can be detected from a patient with complete cerebral death. Examples of the above-mentioned waveforms are given in Figure 13.6.

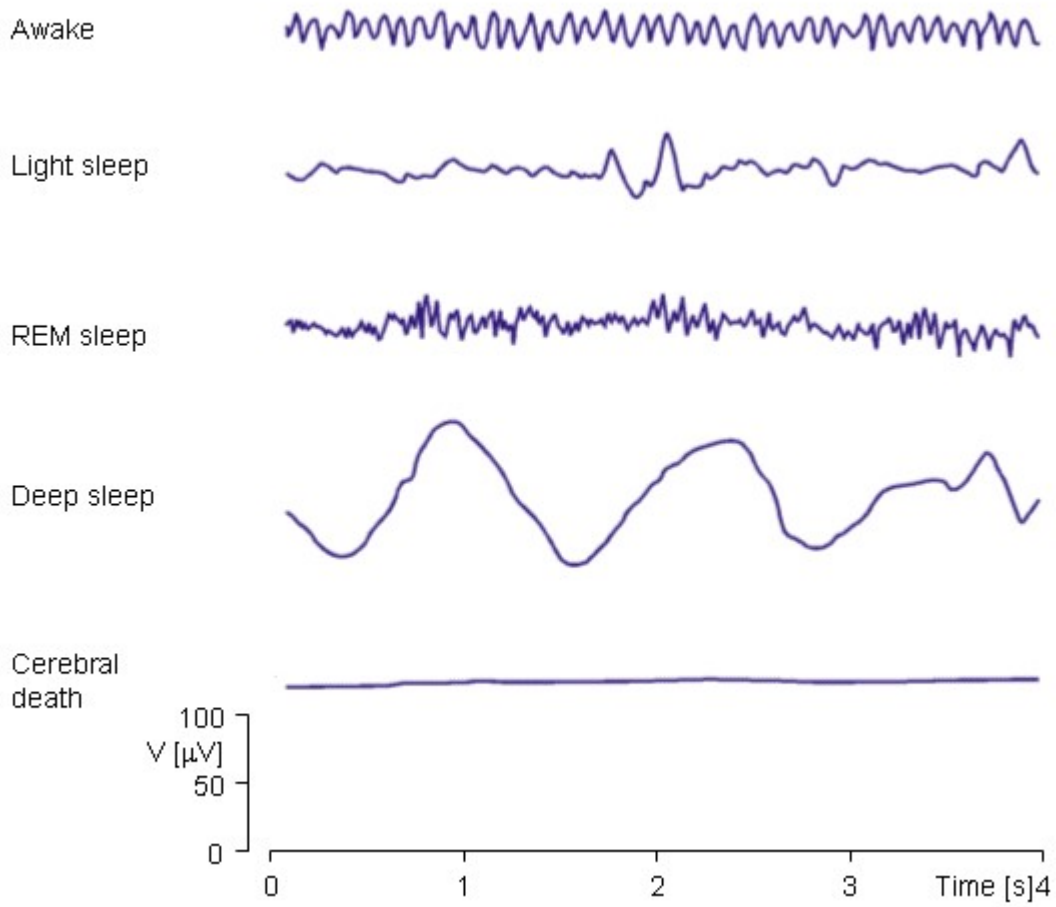


Fig. 13.6. **EEG activity is dependent on the level of consciousness.**

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Use of artificial neural networks for automatic description and interpretation of the Electroencephalogram

Introduction

The objective is to enhance the diagnosis of neurological and neuropsychiatric diseases or disorders based on electroencephalogram (EEG) responses, by exploiting artificial neural network (ANNs) methods to automatically classify and interpret EEG data considering subject's healthy conditions and the abnormalities hereof due to the progress to a possible neurological disorder.

The expected results will lead to advanced devices for neurological diagnosis, for instance through providing different ways of classification of a set *(epilepsie)*, into subsets (e.g. *absence epilepsy, hypsarithmia and benign focal epilepsy of childhood*). Since better diagnosis means earlier treatment, the idea contributes to public health in the Netherlands. The practicability and feasibility of the application of such a methodology will be evaluated together with the neurologist.

An optimum accessibility of the monitoring and diagnosing system is obtained by designing a *Clinical Engineering Environment (CEE)* platform. Such a platform, enabling the user to set up a clinical neurophysiological experiment by graphically composing a block diagram out of a selection of software building blocks, not only foresees in a dramatic reduction in development time but also offers a user friendly experimenting environment. The *CEE* platform accesses centralized EEG data.

The electroencephalogram (EEG) response

An EEG, recorded by positioning 21 or more electrodes on the intact scalp, represents the changes of the electrical field within the brain. Generally even up to 128 and more EEG channels, each corresponding with a standard electrode position on the scalp, can be displayed simultaneously. The results of the EEG signals, after being registered as voltage differences between pairs of electrodes *(bipolar derivations)* or between an active electrode and a suitably constructed reference electrode *(referential derivations)*), are measured, amplified and next displayed on paper or on a monitor. The EEG itself is recorded during different behavioral conditions such as *eyes closed, eyes open, hyperventilation* and *photic stimulation* to provoke abnormalities. However EEGs can also be recorded during *sleep* or during *operative procedures*.

An *intelligent* system will be developed, that first defines the fingerprint of a cardio-vascular response and next identifies and assesses the nature of the abnormalities hereof, the characteristic properties of which are specified by multiple cardiologists.

The exact origin of the EEG is still not completely understood, but it is generally assumed that the measured responses are generated by neurons in the cortex. To be more specific, changes in the depolarisation of the membrane of these neurons *(excitatory and inhibitory post synaptic potentials)* cause extra-cellular currents perpendicular to the surface of the cortex. These currents measured at the crown of the scull, the amplitudes and frequency components of which vary from 10 to 100 μV and from 0.5 to 30 Hz respectively, give rise to the EEG signal through volume conduction. Recordings made directly from the cerebral cortex contain higher frequencies but these are filtered in the scalp recordings. Generally speaking, the EEG signals recorded as a function of time are considered to be stochastic and not deterministic. Consequently, phase information is considered unimportant, and the power spectrum is assumed to fully describe the EEG characteristics. However, this view has recently been challenged by the results of non-linear EEG analysis.

The information consists of different types of periodicity's, characterized by their corresponding frequency, their amplitude, the topography and the conditions under which they occur. The alpha rhythm, for example, registered as a periodic activity with a frequency range varying from 8 to 13 Hz and amplitudes ranging from 20 and 100 μV, occurs across the visual areas of the brain during attentive wakefulness with eyes closed. What is still unclear is how these various EEG rhythms are generated in populations of coupled neurons.

Clinical application of the EEG

Although the origin of EEG responses is not completely brought to light, the signal itself proved to be a valuable tool for diagnosis in the environment of clinical medicine, in particular in neurology, in neurosurgery and in psychiatry. In addition to that, EEG recordings still require additional investigations in studying epilepsy. In indicating epilepsy, it is able to detect abnormalities in waveforms, such as spikes, sharp waves and spikewave discharges. Not only that specific forms of epilepsy *(absence epilepsy, hypsarithmia and benign focal epilepsy of childhood)* can be found, but also *non-epileptic focal brain dysfunctions* possibly caused by cerebrovascular disorders, tumors, infections or traumas and *generalized brain dysfunction* in case of metabolic encephalopathy, intoxication, encephalitis or degenerative dementia are reflected by the EEG signal. Such defects can be classified as either occurring periodically or befalling in a more continuous fashion.

In most cases the EEG is considered to be a sensitive rather than a specific diagnostic instrument, making it a suitable instrument to monitoring the course of a disorder on the one hand and to determining a prognosis of the abnormality on the other. That is, the EEG can pick up very mild degrees of brain dysfunction, but it seldom gives much information about the exact cause of the abnormalities. In general, one should not try to derive *etiologic diagnoses* from the EEG.

Current methods for EEG analysis and interpretation

First analyzing and next interpreting EEG records is hampered by an incomplete knowledge of the origin of the various rhythms and the lack of specificity of the abnormalities. Visually analyzing the raw data and quantitatively examining the time series are the two methods that are available today.

In the first alternative the complete EEG record, involving 16 to 32 channels of information and 20 to 30 minutes of recording, is examined visually by the clinical neurophysiologist. The analysis is performed systematically.

The record is first described for the present EEG periodicity's, like the waveforms itself, the amplitudes of it, the topographical distribution and the changes in the regularity due to *opening of the eyes, hyperventilation and photic stimulation.* The main characteristics of the EEG are than described in a short text of about 200 to 400 words. The next phase is to decide whether the data is normal or that it indicates potential anomalies. If this is the case than the type of abnormality is determined. The last step is to consider the relevancy of the EEG findings with respect to the clinical problem that actually initiated the reason for the investigation. An answer must be given to a well-formulated clinical question. This requires neurological expertise.

Two actualities should be emphasized here. Visually analyzing the EEG data is very much an empirical science. Secondly, interpreting the EEG findings in terms of their clinical relevance requires a considerable amount of clinical, in particular neurological, knowledge. Therefore trained physicians have to be involved for the indispensable interpretations, while trained EEG technicians perform the description of the recordings.

In applying time series analyzing techniques, it is expected that it could facilitate an objective description of the defects. This is the field of a quantitative analysis.

As stated before, EEG responses are considered to be stochastic. This specifically explains the necessity to use power spectra and coherence estimations as the main tools for their analysis. Applications of advanced time series analyzing techniques have so far proved to be of limited use, because quantitative techniques are rather sensitive to artifacts, whereas an experienced electroencephalographer can recognize these more easily. A second argument is that power spectra are not very suitable to detect transients, such as spikes, whereas the human eye is very sensitive for deviations of the background information. On the other hand, a computerized analysis of the EEG data is preferable for long term monitoring.

To summarize: the analysis of EEG records still depends almost completely upon the visual analysis of the raw registrations by a trained technician or physician. Quantitative analysis can assist in this analysis, but so far has proved unable to replace visual assessment. Even more difficult than describing the phenomenon is interpreting it. Consequently, clinical knowledge must be merged with the description of the EEG record to decide whether the found abnormalities have any relevance. Visual analysis is an empirical science, it not only requires a relatively long training (at least 6 to 12 months) to obtain even moderate levels of performance, but also appears to be a time consuming process, roughly 10 minutes per EEG. Developing a new technology, that could improve on analyzing and interpreting the EEG information definitely has a considerable cost saving effect.

Performing through processing

A major task in data acquisition is defining the conditions to process the fingerprint data and to adapt to a suitable indicator for diagnostics purposes. An input space, representing the responses from twenty-one electrodes, positioned on predetermined positions of the scalp, is composed of information obtained from an electroencephalogram, monitoring the brain activities.

Signal preprocessing, leading to data reduction, offers more tractability for other purposes. It can also amount to selective emphasis, including procedures for trend detection and pattern recognition. The

requirements for some of these tasks are quite arduous. It is therefore necessary to develop methods that permit recognition of a consistency of adequate strength in the derived signal that could reasonably be considered as a developing or one to be developed pattern. Determining and recognizing the characteristics of a pattern would be the most important step.

Another stringent demand in practicing signal preprocessing is that various purposes require the availability of techniques to discover interrelations between the signal of interest and other responses. The latter is essential because specific features may only become evident when compared to other signals.

The way input-space data is preprocessed strongly determines the overall efficiency of the performance of the classificator. Smoothing, spectral analysis, statistical analysis, time-frequency and time-scale analysis, correlation and convolution operations, and matched filtering all come in the category of relevant linear signal operations with continuous or sampled signals. But also trend detection and certain aspects of curve fitting and regression analysis belong to the to be investigated processing methods. The various procedures selectively accent features of interest, some of them are emphasized explicitly, others implicitly.

Special attention will be paid to investigating the advantages and disadvantages of time-frequency and time-scale analysis methods. The time-frequency analysis -also known as the short-time Fourier Transform or Gabor Transform gives preference to investigating quasi-stationary responses. The time-scale analysis or Wavelet Transform mode on the other hand offers a preprocessing scheme, in which a high frequency resolution is offered at low frequencies and a high time resolution is offered at high frequencies. Rendering signals, containing all the information, in a two-dimensional domain (the scalogram) is a feature that is only offered by Wavelet Transform operations. The inverse Wavelet Transform procedure ends in a precise time localization, making this operation suitable to compose characteristic properties of a healthy person.

The role of the feature generator is to extract characteristic properties out of the preprocessed signal first and next putting them together in a feature space for further classification.

The role of the classificator is to categorize the elements of the feature space in defining certain classes in reference to the actual state of monitoring and diagnosing.

Diagnosing through neural networking

Neural networks are able to recognize differences in patterns based on automatic learning procedures. The application is attractive, not only that it provides faster responses, but especially because of its capability to automatically discover irregularities in patterns not seen or detected before. Another important feature is that it enables the discovery of regularities in the training signal itself as a consequence of the actual learning process. A training procedure can be performed by three different categories.

Supervised training, requiring the presence of one or more supervisors (in the case considered the neurologists), labels the data to be used in teaching the network. The system, knowing the correct answers, inputs an error signal the moment the network produces an incorrect response. It continues to do so by feeding the difference in assessments back into the network until the error has been decreased to a predetermined minimum value. The error signal as it were teaches the network the correct response. Unsupervised or self-organized training, using unlabelled training sets, do not need a supervisor. Internal clusters, compressing offered data into classification categories, are formed the

moment data is presented to the network. The supervisor is also absent in self-supervised training. The error signals, generated by the network, are fed back into the network itself until a correct response is produced.

Neural networks for analyzing and interpreting EEG recordings

Many processes in medicine depend on pattern recognition. Analyzing and interpreting EEG records are in many respects an example of it. During the years it became clear that conventional (artificial intelligence oriented) approaches, based on obeying a set of rules, are not very suitable to perform such a task. Artificial neural networks (ANN), on the other hand, are effective in detecting and classifying patterns in all kinds of data sets. Recognizing these features has led to a search for possible applications of ANNs in medicine, but so far, only a few attempts have been made in applying these techniques to detecting one specific type of activity in the EEG record. ANNs showed excellent performance in detecting *epileptic* activities in EEG recordings (Gabor and Seyal, 1992; Jando et al., 1993; Gabor Leach and Dowla, 1996).

A logical next step is expanding the scope of ANN applications to EEG analysis. The goal would be to develop a system, which takes the EEG record and clinical data as its inputs, and produces a judgment, whether the record is abnormal, and a clinical interpretation as its outputs.

Using raw EEG time series as an input for an ANN is technically unfeasible. So, some form of processing and data reduction is required. A logical choice would be to use the power contents of a few frequency bands as an input to the ANN. However, power spectra may not be very suitable to detect *transient epileptic* activity. Alternatively, some form of Gabor transformation must be used. The whole record of 20 to 30 minutes will have to be split up into epochs of a few second's lengths to be further processed.

Another difficult problem to be addressed is the choice of the overall architecture. More specifically: which tasks should be solved by which networks? One option might be to use a collection of networks, each trained for a specific well-defined task. This would make the system flexible because new tasks could be added later on, and individual networks could be changed without affecting the architecture of the whole system. As an example, networks can be trained to detect the alpha rhythm; to anticipate on possible artifacts; to demonstrate the appearance of delta waves; the presence of spikes, etc. An advantage of such an approach is that different types of pre-processing can be applied to networks tailored to their dedicated task.

Each EEG epoch is than simultaneously scanned by the networks, resulting in a situation that each network is able to assign a code to that epoch. After having processed all EEG epochs, the codes generated by all the networks are than used as inputs for a secondary level of networks that decide whether the EEG recordings were normal or abnormal. A similar type of strategy might be used for the clinical interpretation of the EEG recordings.

Reliability analysis based on risk analysis

It cannot be expected from a diagnostic system to be fully reliable. There is always an intrinsic uncertainty, because there will never be an exact one-to-one mapping from measured input space to diagnostic output space. This may be caused by uncertainties fundamental to the physics of the problem, or by measurement errors, or by the fact that only a subset of the relevant variables is measured. But there is also an unreliability originating from the errors in a model when the mapping function, used to identify the faults from the input variables, does not correspond to the correct

mapping. A third uncertainty is due to statistical errors, because a mapping function is estimated from a finite set of learning samples and the coefficients can therefore not be estimated exactly.

For classifying applications, it therefore does not suffice to make a certain diagnosis, but it is also important to quantify its reliability (the probability that alternative diagnoses are correct).

The diagnosis system should therefore include a risk analysis option. Putting together a reliability module, in which a relative contribution of the different types of errors is investigated, determines the accuracy of the diagnosis system in question. The results of this could provide important clues on how to improve the performance of the ANN system. Defining a decision module, that assists in estimating the consequences of an inaccurate classification of the rules applied to information supplied by the reliability module and by the users, is a second option.

Defining a measure for the reliability of a diagnosis system and finding a method to separate the different errors are problems that need investigation.

Sleep Studies and Analysis are not the only uses for EEG data. Another fairly recent and important use can be seen in the following study:

Use of Visual Evoked-Potential Studies and EEG Data to Classify Aggressive, Explosive Behavior of Youths

Donald R. Bars, Ph.D., F. La Marr Heyrend, M.D., C. Dene Simpson, Ph.D. and James C. Munger, Ph.D.

Abstract

OBJECTIVE: Data from electroencephalograms (EEGs), including data from visual evoked-potential studies, were analyzed to assess their association with a specific type of explosive behavior in children and adolescents. **METHODS:** Data for 326 children and adolescents treated in a psychiatric clinic were examined. Eighty-two percent exhibited behavior consistent with intermittent explosive disorder, although diagnosis was not an inclusion criterion for the study. The presence of explosive behaviors was indicated by reports from the legal system, schools, parents, health care workers, and psychiatric intake interviews. A quantitative EEG and a series of pattern-reversal evoked-potential studies were administered to each patient. In these studies, children are shown a rapidly reversing checkerboard pattern or rapid flashes of light, and their brain waves (evoked potentials) are measured. **RESULTS:** Logistic regression indicated that patients who exhibited explosive behaviors were significantly more likely to produce high-amplitude P100 wave forms in the evoked-potential studies than patients who did not exhibit explosive behaviors. Forty-six percent of those with explosive behaviors met the clinically defined electrophysiological criteria for the high-amplitude P100 wave forms. **CONCLUSIONS:** The use of visual evoked-potential studies helped us classify a large subset of youths who exhibited out-of-control explosive behaviors. The findings suggest that a subgroup of individuals exhibiting explosive behaviors may have a predisposition for violent or explosive behavior

that is an innate characteristic of their central nervous system. An understanding of the etiology of explosive behaviors permits the use of more appropriate intervention and treatment strategies.

Introduction

With each violent outburst by an adolescent in our society, early identification of children who exhibit explosive, aggressive, and uncontrollable outbursts becomes more critical. This difficult population was identified more than 30 years ago by Stevens and colleagues (1).

The deterioration of family structure and the noticeable moral decline in society (2) have only increased the need to accurately identify individuals with explosive and aggressive tendencies and to develop methods to help them achieve success in the world around them. In a study by Cadoret and associates (3), a significant amount of the variability in the aggressivity of adopted children, as well as in conduct disorder and adult antisocial behavior, was accounted for by environmental effects and genetic-environmental interactions. Empirical evidence of these interactions is important to both basic science and applied social sciences for intervention and prevention. As Elliott (4) noted, "As with all behavior, aggression is the result of interactions between the brain (including psychological experience) and environmental challenges—a biopsychosocial phenomenon."

Although environmental factors are important, a review of the literature reveals that many aggressive behaviors have a biological basis, and when such behaviors are not identified and addressed appropriately, positive treatment outcomes are extremely difficult to obtain (5). Matthews and his colleagues (5) have labeled some patients "neurobehavioral" because they believe that underlying abnormalities in brain function are the primary cause of their explosive behavioral outbursts.

Studies evaluating the electrophysiological correlates of aggressive, explosive, and assaultive behaviors have relied primarily on standard electroencephalogram (EEG) data (6,7,8,9,10,11). In two previous studies, we evaluated the relationship between pattern reversal, visual evoked potentials, and explosive behaviors. An evoked potential is a regular pattern of electrical activity recorded from neural tissue that is evoked by a controlled stimulus. In pattern-reversal visual evoked-potential studies, a subject is shown a rapidly alternating pattern, and the subject's EEG data are examined for amplitude and latency of the wave forms, the symmetry of the responses, and any abnormal activity. Such abnormalities often indicate difficulties with behavior and learning. The etiology of explosive behaviors may be different in children who exhibit such abnormalities.

In our first study of 278 children and adolescents, 71 (26 percent) exhibited explosive behaviors, and 237 (85 percent) exhibited at least one type of brain electrical abnormality (12). The other study compared six explosive individuals (four males and two females) who were matched by age, sex, and handedness with six children who did not have a psychiatric diagnosis (13). The mean±SD age of the children was 13.5±2.06 years. In the pattern-reversal evoked-potential studies, the children with explosive behaviors were found to have significantly higher N75 and P100 wave forms than the controls.

This study investigated whether a relationship exists between quantitative EEG data, visual evoked potentials, and explosive behavior of children and adolescents referred to our clinic. During the exploratory studies (12,13), one variable in the visual evoked-potential series—the P100 wave form—consistently showed a significant relationship with explosive behavior. Therefore, in the study reported

here we hypothesized that the amplitude of the P100 wave form in the pattern-reversal visual evoked-potential studies would be significantly related to explosive, aggressive, and out-of-control behaviors.

The data we used are a subset of an extensive database gathered over two years involving electrophysiological parameters of various psychiatric behaviors. It should be emphasized that our research deals with behaviors and not diagnostic categories, because explosive behaviors cut across a variety of diagnoses. The results of this study provide possible evidence of a specific class of explosive disorders and may lead to more effective treatment based on neurophysiology and scientific principles.

Methods

An evaluation of electrophysiological, behavioral, and medication data was conducted for all children through 18 years of age who successfully completed a standard protocol of computerized electroencephalographic and evoked-potential evaluations during 1995 and 1996. Before coming for evaluation, many of these patients had been placed in one or more residential treatment facilities and had been seen by several therapists and doctors, yet their uncontrollable behaviors continued.

Of the 454 children and adolescents evaluated, complete data were available for 326—105 females (32 percent) and 221 males (68 percent)—who were included in this study. Children excluded from the study were those who had only a sleep-deprived EEG—that is, those for whom evaluation of evoked potentials was not completed or those who did not correctly count the number of target stimuli given during the auditory-event-related potential studies. We did not consider the amplitude of wave forms or any specific behavioral characteristics for the children not included in the study.

The mean±SD age of the 326 children was 13.27±2.92 years; the mean age of those with explosive behaviors was 13.03±2.95 years, and the age of those without explosive behaviors was 14.32±2.52. Explosive behavior was defined as that with apparent limbic system involvement. Typically it occurs illogically, in situations that would not be expected to prompt an outburst. Such children control themselves in many situations in which a strong emotional response would be understandable; however, an inconsequential situation causes an "explosion" of emotions. The outburst lasts at least 20 minutes and sometimes goes on for a day or more. When the children are approached, the behavior is escalated, and the length of the outburst is extended. Usually they are apologetic after the outburst, and they sometimes deny remembering what occurred.

In our study, an individual was considered to be explosive if any mention of explosive rage, out-of-control anger, out-of-control aggression, verbal or physical attacks on another individual, intermittent explosive disorder, or episodic dyscontrol syndrome was recorded in the clinical file. A total of 267 of the 326 children (82 percent) met these criteria. In our 1991 exploratory study, only 26 percent of the sample exhibited explosive behaviors. The difference reflects an increase in the proportion of explosive individuals among our clinical population because of our clinic's growing reputation for working with explosive, out-of-control individuals.

To account for any effect medication might have had on the electrophysiological data, a dichotomous variable, medications, was created, which indicated whether or not an individual was taking medication at the time of testing.

Informed consent was given by the participants or their parents or guardians after we reviewed the procedure and answered their questions.

Electrophysiological procedure

Each patient underwent a series of evoked-potential studies and a quantitative EEG in accordance with guidelines of the American Electroencephalographic Society (14,15). We used the Brain Atlas III, a product of the Bio-Logic Corporation in Chicago. Electrode placements were in accordance with the international 10-20 system, using an Electro-Cap, with 16 active electrodes. A monopolar montage with forehead ground with linked-ear reference was used. Electrode impedance was maintained at less than 2 kohms, and the impedance between homologous sites was maintained within 1 kohm. The gain was set at 30,000, the low-pass filter at 100 Hz, the high-pass filter at 1 Hz, and the 60-Hz notch filter was set to the "in" position.

Patients were comfortably seated in a padded reclining chair in a small, sound-attenuated room. A channel-by-channel calibration was performed before and after each recording session.

The electrophysiological test series consisted of four visual evoked-potential studies, including pattern reversal (both eyes, left eye, and right eye) and flash (both eyes); three auditory-event-related potential studies (the odd-ball paradigm at three different speeds); two brainstem auditory evoked potentials; and 20 minutes (after two minutes of hyperventilation) of computerized electroencephalography. Digital EEG data were evaluated, and artifact-free data were used to create eyes-open and eyes-closed (resting) fast Fourier transformed files. We analyzed data on visual evoked potentials and fast Fourier transformed files.

Visual evoked potentials

Data from the pattern-reversal visual evoked-potential evaluation were recorded for each individual in accordance with guidelines of the American Electroencephalographic Society (14,15). The checkerboard pattern-reversal paradigm used 19 millimeter, black-and-white alternating squares displayed on a model TC1115 RCA monitor positioned at eye level, 76 centimeters in front of the patient, subtending a visual angle of 23 degrees. The pattern reversed every .59 seconds for a total of 1.7 stimuli per second. A 256 ms epoch was used with a 5 ms prestimulus time. The flash paradigm used a 512 ms epoch with 10 ms of prestimulus time. The intensity of stimulus from the checkerboard pattern-reversal was 12.69 candelas per square meter (cd/m^2) and that of the flash 19.26 cd/m^2.

The patient was instructed to visually fixate on a red dot centered on the RCA monitor, not to speak, and to remain relaxed with as little movement as possible throughout the recording time. Artifacts were detected and removed using the Bio-Logic online artifact rejection program. For each patient, 200 artifact-free trials were averaged together to produce the final wave form.

Procedure

Patients' clinical files were reviewed for information about sex, age, medications, head injuries, loss of consciousness, explosive behaviors, narcissism, rumination, symptoms of bipolar disorder, or impulsivity. These clinical variables were evaluated for association with several electrophysiological variables. They included the maximum amplitudes of the pattern-reversal evoked-potential N75 and P100 wave forms at O1 and O2; the flash evoked potential P100 wave form at O1 and O2 and P200 at F3 and F4; the auditory-even-related evoked potential P50 wave form at F3 and F4, the N200 wave form at F3, F4, and CZ; the P300 wave form at CZ and PZ; and the eyes-open FFT data recorded from occipital lobe electrodes, O1 and O2, the frontal lobe electrodes, F3 and F4, two central locations, CZ and PZ, and the temporal lobes, T5 and T6 (16,17,18).

All clinical data except explosivity, sex, age, and medications were excluded from the final analysis because although the concordance decreased slightly when clinical data were removed, we decided that the small decrease in concordance was less important than the inclusion of variables that are subjectively based and that may vary dramatically between professionals.

Logistic regression analysis was used to determine which electrophysiological variables were significantly associated with the presence of explosive behaviors. Stepwise analyses were completed on various combinations of EEG data, data from the evoked-potential evaluations, and clinical information, using a cutoff probability value of .25 to produce the preliminary model.

Interactions between sex, age, medications, and the electrophysiological variables identified in the preliminary model were assessed individually. All interaction effects with p values of .15 or less were added to the preliminary model. We then sequentially assessed variables that had p values in excess of .05, starting with the least significant variables. A confounding variable was defined as one that changed the parameter estimate of a significant variable by 20 percent or more.

To gain a basic understanding of the relationship of evoked potentials to underlying brain electrical activity, correlations were determined between amplitudes of the P100 wave forms in the pattern-reversal evoked-potential studies and the EEG data from the occipital electrodes (O1 and O2).

Results

Stepwise logistic regression analysis of the EEG data alone and the EEG data plus the evoked-potential variables indicated that the concordance percentage increased with the addition of evoked-potential data and that the addition of these data significantly improved the analysis, as shown by the -2LOG L chi square statistic in Table 1.

Model and variable	p	Odds ratio
Model 1[1]		
Delta F4	.001	3.354
Sex	.183	.664
Age	.823	.948
Medications	.597	1.177
Model 2[2]		
OP100	<.001	1.256
Delta F4	.007	2.931
Sex	.022	.469
Age	.149	1.127
Medications	.585	1.190

[1] Model 1 included only the EEG data. –2LOG L=22.745, df=4, p<.001; concordant pairs=68.6 percent; discordant pairs=31 percent

[2] Model 2 included the EEG data and the evoked-potentials variables. –2LOG L=44.518, df=5, p<.001; concordant pairs=75.8 percent; discordant pairs=23.9 percent

Table 1. Two logistic regression models predicting behavior among 326 children and adolescents

The final model consisted of five variables, which are shown in Table 1. They were the OP100 wave form—that is, the maximum P100 amplitude at the O1 and O2 electrodes—from the pattern-reversal evoked-potential studies; delta F4—that is, the delta absolute power in the right frontal lobe from the EEG data; sex; age; and medications.

Concordance in the models was determined as follows: All possible pairings of individuals who had explosive behavior and who did not have explosive behavior were created. A pair was defined as concordant if the individual who had explosive behavior was also the individual predicted by the logistic regression model to be the one more likely to have explosive behavior on the basis of physiological predictor variables. A pair was defined as discordant if the model incorrectly predicted that the individual who did not have explosive behavior was more likely to be the individual who had explosive behavior.

Higher amplitudes of the OP100 wave form were significantly associated with explosive behaviors, and the amplitude was somewhat higher for the female explosive individuals. As Table 2 shows, males were more likely than females to exhibit explosive behaviors, and the explosive group was slightly younger. Male explosive individuals were the youngest overall. However, these differences were not statistically significant.

Variable	N	Age		OP100[1]		Delta F4[2]	
		Mean	SD	Mean	SD	Mean	SD
Females	105	13.96	2.67	9.91	4.23	65.44	32.57
Males	221	12.93	2.98	9.58	5.07	82.70	46.40
Explosive	267	13.03	2.95	10.31	4.96	81.67	44.52
Nonexplosive	59	14.32	2.52	6.80	2.60	56.64	28.68
Explosive females	80	13.75	2.86	10.68	4.33	68.60	34.60
Nonexplosive females	25	14.64	1.85	7.46	2.73	55.33	22.72
Explosive males	187	12.72	2.95	10.15	5.20	87.27	47.13
Nonexplosive males	34	14.89	2.93	6.48	2.46	57.60	32.69

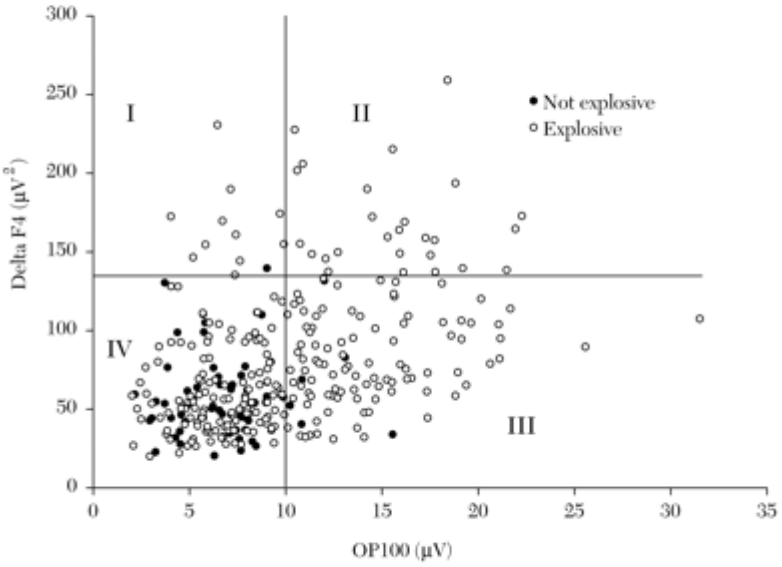
[1] Maximum amplitude, in microvolts, between the O1 and O2 electrodes. The normal range is 5 to 8 μV.
[2] Absolute power in microvolts squared. The normal range is 30 to 45 μV^2.

Table 2. Mean±SD ages and EEG values for 326 children and adolescents, by sex and by whether or not they exhibited explosive behavior

Explosive individuals also had higher delta absolute power at the F4 electrode; the most intense delta F4 activity was found in males. Using a 95 percent reliability level, our clinical criteria require a P100 of 10 microvolts or greater to indicate a predisposition to explosive behavior (Figure 1).

Figure 1

Absolute power of delta at the F4 electrode and OP100 values (maximum P100 amplitude at the O1 and O2 electrodes) for 326 children and adolescents, by whether or not they exhibited explosive behaviors[1]



[1] The four quadrants are based on breakpoints of 135 μV² of delta F4 and OP100 values of 10 μV. The breakpoints are set such that 95 percent of the individuals in quadrants I and II exhibited explosive behaviors.

Figure 1. Absolute power of delta at the F4 electrode and OP100 values (maximum P100 amplitude at the O1 and O2 electrodes) for 326 children and adolescents, by whether or not they exhibited explosive behaviors

In both regression models shown in Table 1, the absolute power of delta F4 was significantly higher in the explosive group than in the nonexplosive group. In the model that included the EEG data plus the evoked-potential variables, the OP100 was significantly correlated with explosive behavior. The medication variable was not significantly associated with explosive behavior in either model. A further set of analyses was performed in which the subjects taking medication at the time of testing were analyzed separately from those who were not.

A correlation analysis was used to evaluate whether the underlying brain electrophysiology might be the driving force of the amplitude of the P100 wave form. An increase in the EEG absolute power was significantly related ($p<.001$) to an increase in P100 amplitude in the occipital lobe at the O1 and O2 electrodes (O1; delta, beta, and at O2; delta, theta, alpha, and beta). However, the two were not highly correlated; the highest correlation occurred between the delta activity and the P100 amplitude, recorded at the O2 electrode; the correlation accounted for only about 20 percent of the variance in the amplitude of the P100 wave form.

Discussion

The purpose of this study was to evaluate whether EEG data predicted behaviors exhibited by a group of children and adolescents referred to a psychiatric clinic for evaluation of a wide variety of psychiatric disorders, including explosive, aggressive, and out-of-control behaviors. These individuals were referred by psychiatrists, psychologists, social workers, educators, and parents. They had been treated with little success, given various medications, and given a profusion of diagnoses.

Our study showed that individuals with a high-amplitude P100 wave form in evoked-potential evaluations and high delta band absolute power in the right frontal lobe were more likely to exhibit explosive behaviors.

It was not surprising to find that most explosive individuals had high frontal delta band absolute power (quadrants I and II in Figure 1); most experts in neurophysiology would expect such a result (Nuwer M, personal communication, 1998). Convit and associates (19) found a positive relationship between the level of violence and the amount of delta activity in psychiatric patients with violent behaviors. Fishbein and colleagues (20) reported that aggressive subjects in their study had more delta and less alpha activity in a spontaneous EEG, which they note has been observed in psychopaths and criminals.

What is new in this study is the finding that individuals with higher P100 wave form amplitudes in pattern-reversal evoked-potential studies were more likely to be explosive. Indeed, we believe that the P100, which occurs within the obligatory portion of the brain's electrophysiological response to sensory stimulation (approximately within the first 200 milliseconds after stimulus), is a biological signature and represents an individual's unique biological receptive process underlying a predisposition to process incoming stimuli in a given manner.

Although not all explosive individuals in our study had a high P100 wave form (Figure 1), the results suggest that the higher the amplitude, the more probable the explosive behaviors. Overall, these patients constitute a distinct psychiatric population in need of the unique medical treatment provided by the psychiatric community—not identification and incarceration as sociopaths by the justice system. Further studies are needed to confirm our results with children and adolescents, to investigate adult populations, to evaluate the predictability of the behavior before it is actually present, and to evaluate the relationship between the wave form and various medication regimens.

We wish to emphasize that we do not hypothesize that the high P100 amplitudes are the cause of explosive behavior. Instead, as described above, it is best to conceive of high P100 amplitudes as a marker for explosive behavior. Descending inhibitory pathways parallel the ascending sensory systems and modulate responsiveness at a very early level of processing, either at the receptors themselves or soon after, and in any event before the cortical events responsible for the generation of scalp evoked potentials (21).

As a visual stimulus moves past the retina, it proceeds down two distinct paths. The first allows it to be transported to the primary receptive area in the occipital lobes (Brodmann's area [17]). The hypothalamus also receives one-way afferent connections directly from the retina (22). This is the pathway of interest because the hypothalamus participates in autonomic or behavioral expressions of emotions as part of the survival mechanism controlled by the limbic system (23). If the intensity of the stimuli is excessive, they may cause a kindling effect as seen in seizure disorders. Andy and Velamati (24) concluded that "repeatedly recurring limbic system seizures through superkindling mechanisms can eventually render the limbic-basal ganglia-preoptico-hypothalamic aggressive system hyper-responsive to both recurring seizures and to exteroceptive stimuli with resulting aggressive behavior with or without an accompanying seizure."

These patients appear to respond positively to various combinations of anticonvulsant, antidepressant, antipsychotic, and stimulant medications. Of these, the anticonvulsants appear to deliver the most consistent benefits (5,18,25-27). Because explosive behavior appears to be a problem with emotional expression or control, defects in limbic system function have been proposed by Tancredi and Volkow (26) and by Matthews and associates (5). The positive effect of anticonvulsants may be related to the large number of gamma-aminobutyric acid (GABA) receptors located in the amygdala, a critical component of the limbic system; several of the anticonvulsants are thought to exert their effects via the GABAergic systems (17).

This study found that results for patients who were taking medication at the time of the evaluation were not significantly different from the results of those who were not. The effect of medication on visual evoked potentials, both checkerboard and flash, has been evaluated in several studies involving normal subjects and individuals with seizure disorders. The majority of studies found no effect on the amplitude of the wave forms. Although the effect of valproic acid may be nonspecific, a wide variety of anticonvulsant and sedative agents have been reported to lower the amplitude of flash evoked potentials (28). Faught and Lee (29) reported a trend toward higher amplitudes among photosensitive patients when pattern reversal was used as the visual stimulus, but the differences were not statistically significant.

No significant treatment effect was found for simple reaction time, nor was there any significant effect on either the latency or the amplitude of the visual evoked potential. Another study concluded that sodium valproate has no effect on simple reaction time or the visual evoked potential in normal subjects, although it may cause a slight increase in slow-wave sleep (30). Among patients with seizure disorders, no pronounced influence of the disorder itself on the parameters and wave form of the normal visual evoked potential using pattern reversal was demonstrated if the patients were not taking anticonvulsant drugs; the findings for patients under complete seizure control with anticonvulsants were similar (31).

The question arises as to whether the increased amplitude of the P100 wave form can be explained by the augmenting-reducing (A-R) phenomenon, also known as stimulus intensity modulation. The A-R refers to the central nervous system's modulation of responses to sensory stimuli of different intensities. In 1976 Buchsbaum (32) suggested that A-R is related to central control factors rather than peripheral mechanisms. Several authors have suggested that this central mechanism is designed to protect the cortex from overstimulation (32,33,34,35,36).

Blenner and Yingling (21) reported that their visual evoked-potential data were in agreement with those of Stenberg and colleagues (37), who found a weak augmenting pattern at both FZ and CZ but a reducing pattern at OZ. Overall they found in the visual system a slight augmentation at the vertex, but a reduction at the occiput. In our study the effect of the A-R response in our patients could not be evaluated because the stimulus intensity remained constant.

This study found a group of patients exhibiting explosive behaviors who had low-amplitude OP100 wave forms in pattern-reversal evoked-potential evaluations and low absolute-power delta (Figure 1, quadrant IV). These patients represent other subsets of individuals with explosive behaviors of different etiologies, perhaps those with temporal lobe syndrome or frontal inhibition problems. This finding is consistent with the literature indicating that several different roads can lead to explosive, aggressive behaviors. Research in our laboratory is focusing on electrophysiologically delineating these other groups. However, the importance of the amplitude of the P100 wave form in classifying and predicting explosive behaviors should not be overlooked.

The study had several limitations. First, the results are generalizable only to individuals with psychiatric problems severe enough for them to be identified by their parents, school system personnel, or health care professionals. Second, evoked potentials were recorded from scalp electrodes, and therefore the subcortical involvement identified can only be hypothesized. Third, we did not evaluate all psychophysiological response systems (for example, the electrodermal or cardiovascular) and their relationships to explosive behaviors.

Conclusions

Our findings suggest that individuals who exhibit explosive behaviors may have a predisposition for violent or explosive behavior that is an innate characteristic of their central nervous systems. The use of noninvasive visual evoked potentials and quantitative EEG data appears to allow accurate identification of this substantial subgroup of patients who have explosive disorders that appear to be biologically based. Once we identify such patients, we can direct them into the most appropriate treatment regimens.

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Principles of EEG Examination

Select the *best* answer to each of the following items. Mark your responses on the Answer Form.

1. EEG in various forms is most useful as a tool for monitoring and diagnosis in certain clinical situations, including _____.

a. to distinguish epileptic seizures from other types of spells
b. monitoring depth of anesthesia

c. as an indirect indicator of cerebral perfusion in carotid endarterectomy
d. All of the above

2. EEGs are frequently used in experimentation because the process is non-invasive to the research subject.

a. True
b. False

3. There are many uses for the MEG, including _____.

a. assisting surgeons in localizing a pathology
b. assisting researchers in determining the function of various parts of the brain
c. providing neurofeedback
d. All of the above

4. In conventional scalp EEG, the recording is obtained by placing electrodes on the scalp, usually after preparing the scalp area by light abrasion and application of a conductive gel to reduce impedance.

a. True
b. False

5. Scalp electrodes are sensitive enough to pick out individual action potentials.

a. True
b. False

6. Gamma is the frequency range approximately 26–100 Hz. Gamma rhythms appear to be involved in higher mental activity, including perception, problem solving, fear, and consciousness.

a. True
b. False

7. Signals in the EEG that are of non-cerebral origin are called _____.

a. noise
b. waves
c. artifacts

d. None of the above

8. In 19__, Russian physiologist, Vladimir Vladimirovich Pravdich-Neminsky published the first EEG and the evoked potential of the mammalian (dog).

a. 04
b. 12
c. 20
d. 26

9. An EEG, recorded by positioning _____ or more electrodes on the intact scalp, represents the changes of the electrical field within the brain.

a. 12
b. 17
c. 21
d. 26

10. The EEG itself is recorded during different behavioral conditions such as *eyes closed, eyes open, hyperventilation* and *photic stimulation* to provoke abnormalities. However EEGs can also be recorded during *sleep* or during *operative procedures*.

a. True
b. False

11. The exact origin of the EEG is still not completely understood, but it is generally assumed that the measured responses are generated by neurons in the cortex.

a. True
b. False

12. EKG artifacts are quite common and can be mistaken for spike activity.

a. True
b. False

13. First analyzing and next interpreting EEG records is hampered by an incomplete knowledge of the origin of the various rhythms and the lack of specificity of the abnormalities.

a. True
b. False

14. Visually analyzing the EEG data is very much an empirical science. Secondly, interpreting the EEG findings in terms of their clinical relevance requires a considerable amount of clinical, in particular neurological, knowledge.

a. True
b. False

15. _____ are able to recognize differences in patterns based on automatic learning procedures. The application is attractive, not only that it provides faster responses, but especially because of its capability to automatically discover irregularities in patterns not seen or detected before.

a. PET studies
b. Neural networks
c. MRI studies
d. None of the above

16. Although the origin of EEG responses is not completely brought to light, the signal itself proved to be a valuable tool for diagnosis in the environment of clinical medicine, in particular in neurology, in neurosurgery and in psychiatry.

a. True
b. False

17. Recent advances in EEG instrumentation and analysis have improved the spatial resolution of the technique and rendered the inverse problem tractable in many cases.

a. True
b. False

18. In most cases the EEG is considered to be a sensitive rather than a specific diagnostic instrument, making it a suitable instrument to monitoring the course of a disorder on the one hand and to determining a prognosis of the abnormality on the other.

a. True
b. False

19. *Home Ambulatory Electroencephalograms* (EEG) with the patient not modifying medication is a valuable test as the patient's symptomatology can be monitored day and night in a natural environment of home using computerized filtering of artifact.

a. True
b. False

20. *Computerized EEG* monitoring allows breakdown of wave forms and allows correlation with evoked potentials including cognitive evoked potentials.

a. True
b. False

21. The EEG signal is the summation of brain cell synaptic firing originating from the cerebral cortex. General anesthesia (GA), sedation and hypnosis can be considered to be different states on the same continuum, ranging from death, coma, to full cognitive awareness and beyond to hyper-arousal.

a. True
b. False

22. Interpreting EEG is difficult but made easier by spectral analysis. Fast Fourier transforms have until fairly recently been the standard method of analyzing raw EEG signals.

a. True
b. False

23. The main disadvantage of the Burg algorithm in EEG is its handling of signals in noisy environments where spectral line-splitting occurs.

a. True
b. False

24. It has been found that when a person is sedated, but not yet anaesthetized, their brain waves contain a frequency component which occurs between 8 Hz and 12 Hz, and is known as the alpha rhythm.

a. True
b. False

25. There are a large number of potential applications for low cost, reliable, easy-to-interpret EEG monitors throughout medicine and “soft” medical applications such as relaxation, study, personal and arts performance/concentration improvement, self-administered psychological training (bio/neurofeedback), and sports performance.

a. True
b. False