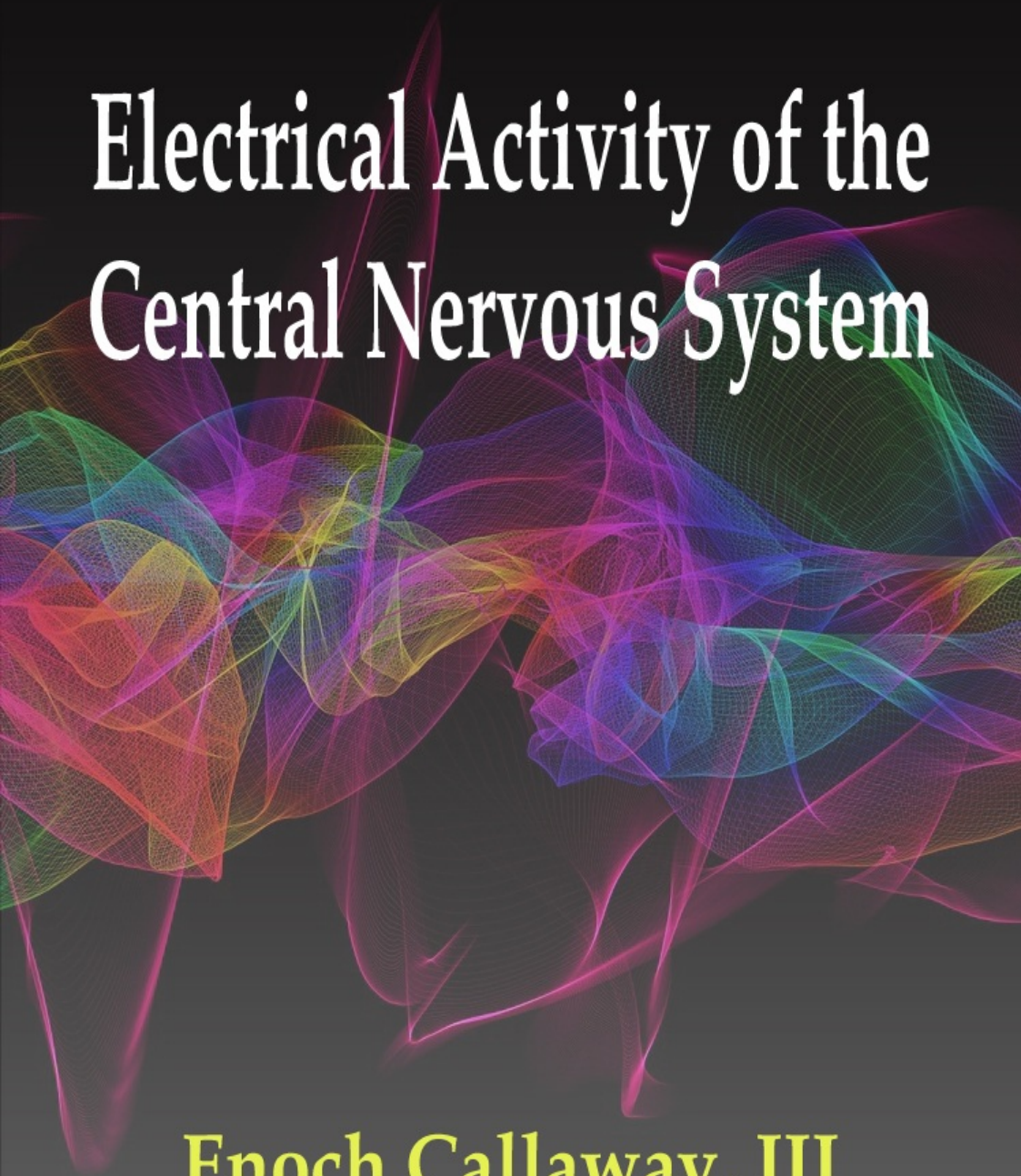
American Handbook of Psychiatry

Electrical Activity of the Central Nervous System



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e-Book 2015 International Psychotherapy Institute

From *American Handbook of Psychiatry: Volume 6* edited by Silvano Arieti

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ELECTRICAL ACTIVITY OF THE CENTRAL NERVOUS SYSTEM[1]

Sherrington, with his poet's eye, saw the brain as magically weaving a fabric of flashing and moving points of light. The passings of these fanciful shuttles in the enchanted loom of the cerebral cortex are in reality marked by shifting electrical potentials. In the brain itself the shifting potentials are exquisitely detailed and specific. Some are sharp, brief, and all-or-none; others are slow, persistent, and graded. At the surface of the head these potentials are attenuated and mixed. Much of the fine detail is blurred, but even so there is more than enough detail to go around since investigators are continuing to find new clues about what the brain is doing by studying this cerebral electrical activity.

These shifting potentials on the surface of the head are referred to as the electroencephalographic activity and a recording of this activity is, of course, an electroencephalogram. The initials "EEG" serve to indicate both the activity and its record. The EEG was probably first described by R. Caton, in 1875, but the human EEG was the discovery of Hans Berger. Berger was a psychiatrist and hoped the EEG would provide clues about the function of the mind. The logic is simple: if the EEG reflects operations of the brain, and if the brain determines functions of the mind, then the EEG should tell us something about the mind. That syllogism has sustained an amazing number of people in the faith that the EEG somehow contains clues as to mental

processes. It sustained them during a long period when empirical support was scanty at best. Now, however, there is increasing evidence that the EEG does have psychological significance and there is even reason to hope that it may have practical applications in areas more psychological than neurological. But such was not always the case.

The Physiology of the EEG, or Does It Come from the Brain?

People have seriously questioned whether the EEG really has anything to do with brain function. Thus, Kennedy insisted that a lot of brain electrical activity could be the result of shock waves produced by the arterial pulse passing through the polarized jell of the brain. More recently Lippold contended that oscillation of the eyeball produced a major part of the EEG. Without going into all the details, it seems now that these theories can by and large be rejected; not that some of the EEG may not be due to things unrelated to neural activity. Muscles, eye movement, even mechanical events inside the skull may make their contribution to the EEG. Nonetheless, it seems fairly clear that most of the EEG originates in neural activity of the brain. The dendrites and cell bodies of neurons in the cortex are continually responding to incoming volleys by graded potential changes. These postsynaptic changes may be inhibitory or excitatory. The sum of these changes at the surface of the head probably account for much of the EEG. The orientations of the neuronal population, as well as the changes in dendritic polarization, influence what

happens at the surface, and the possibilities in the cortex are large enough to account for almost anything that could occur. In special cases (the evoked responses, to be considered later) we know that certain arriving volleys depolarize apical dendrites that are oriented toward the surface of the head, and the result is a surface negative wave. In other cases, such as the far-field evoked potentials, volleys coursing along deep tracks of white matter may produce a positive wave at the surface if the action potentials are not moving at right angles to the electrode. Usually these far-field waves are much smaller than the slow potentials. Thus we have a rough idea of how the EEG could be generated, if not a detailed picture of how it is actually produced. The actual EEG is much like what one might expect from the first of the two sources just described. In general, most of the EEG is made up of relatively high voltage—50 microvolts (abbreviated /xv) slow waves—Hertz—up to 20 Hertz (abbreviated, Hz) or cycles per second—and these probably arise principally from the cortex.

Form of the EEG

If you want to put an active electrode on someone's head to measure an electrical potential, you will of course have to decide where to put another electrode, for a potential is a difference between two points. Sometimes we try to find an inactive reference (monopolar recording) but this is always relative since there is no true electrically inactive site on the human body.

Alternatively we can record between two active sites, but that has obvious problems, too, for it is very hard to tell whether a particular potential change is due to a drop in potential in one of the active electrodes or an increase in potential at the other.

Suppose, however, we choose the ear lobe as a relatively inactive site and put our so-called active electrode at the occiput. There we will usually be able to record a prominent sine-wavelike electrical activity at about 10 Hz in frequency and about 50 μv in amplitude. These are the alpha waves and they tend to appear when one is alert, relaxed, and ready to look but not yet visualizing anything. Close inspection of an EEG record will reveal other waves, including faster, lower voltage, so-called beta waves; slower theta waves; and occasionally even slower delta waves.

These Greek letter designations have now been formally defined by international agreement. Delta is below 4 Hz. Theta includes activity from 4 Hz up to but not including 8 Hz. Alpha goes from 8 Hz up to but not including 13 Hz, and beta goes from 13 Hz to above 30 Hz.

The form of the EEG can be described in terms of the visual impressions obtained by a trained encephalographer. One can also obtain an automatic frequency analysis which gives a power spectrum. This spectrum specifies the power at each frequency. The third common automatic approach is the period

or line cross analysis which gives a measure of the abundance of waves with specific time periods between the instants that they cross the base line. “Period” is, of course, the reciprocal of “frequency,” but conventional period analysis substitutes “abundance” for “power”—so although the two automatic analyses are related, they are not identical. Finally, if the power spectrum is obtained mathematically by Fourier analysis, one can also obtain coherence measures to specify relations between various areas of the brain.

In general, children have high voltage and lower frequencies in the EEGs. Voltages fall and frequencies increase both with maturity and with arousal. The highly alert subject usually shows a low voltage beta record. When he is moderately alert but not specifically attending, higher voltage alpha waves appear. With falling alertness, alpha may disappear and slow theta waves may appear. When sleep commences, however, a complex sequence of EEG changes may be observed, and these will be considered in Chapter 8 on sleep.

The EEG from different sites on the head may also differ. As noted, alpha is most prominent at the occiput, and beta is more prominent frontally. The two hemispheres may also differ, and this also may be a function of the state of the subject.

For example, when the subject is engaged in a verbal task such as

composing a letter, the EEG on the left, the "language" or "prepositional" hemisphere, is suppressed—much as alpha at the occipital "visual area" is suppressed by involvement in visual activity. By contrast, a nonverbal task such as mirror tracing will suppress the EEG over the right or "appositional" hemisphere.

The Neurologist's EEG—Epilepsy and Gross Intercranial Pathology

This basic EEG with its dominant occipital alpha was described by Berger. The next developments, however, were of more interest to neurologists than psychiatrists. If there is a space-occupying lesion in the cranium, the EEG may reflect this. The EEG may be suppressed over the lesion itself and at the edges of the lesion irritation may cause sharp spikes and abnormal slow waves to appear. Thus the EEG can help in the localization of a brain tumor. Gross pathological alterations of consciousness are frequently reflected in the EEG just as are those gross normal alterations of consciousness referred to as sleep. Figure 5-1 shows a normal EEG and EEGs from patients in two common pathological states: the EEG of a patient in hepatic coma and that of a post-alcoholic psychiatric patient receiving large doses of several tranquilizing drugs.

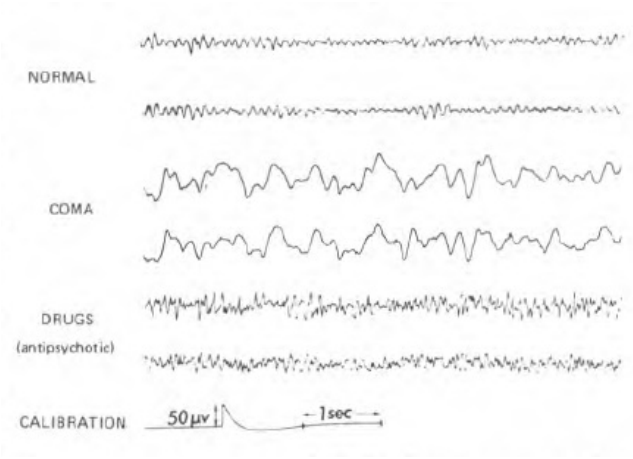

Figure 5—1.
Samples of EEGs showing a normal record, a record from a patient in hepatic coma, and a record from a patient with a history of alcoholism and psychosis who was receiving both phenothiazines and chlordiazepoxide.

Usually when patients have epileptic fits the EEG will also show signs of this. Petit mal attacks are characteristically accompanied by so-called spike and wave discharges, with typical alternating slow and fast waves. Grand mal seizures are marked by a buildup of activity that continues until the brain seems to be in the throes of an electrical storm. This is followed by deathlike stillness during the early parts of the postictal coma. Psychomotor fits may be accompanied by spiking discharges in the temporal area. These typical patterns are illustrated in Figure 5-2. Unfortunately, these pretty pictures are not as consistent as one would like. It may be very hard to catch an epileptic having a major fit and some people who have EEGs that look simply awful still manage to function surprisingly well. This imperfect correspondence

between the EEG and clinical epilepsy is puzzling. The normal EEG in the clinical epileptic is not so bad; we can always rationalize that we've missed the fit or that the electrical signs of the fit are buried too deep in the brain to be picked up at the scalp. But the ability of some people to function when a large part of the brain is obviously having an electrical fit is unsettling for those of us who would like one-to-one correlations between behavior and the EEG.

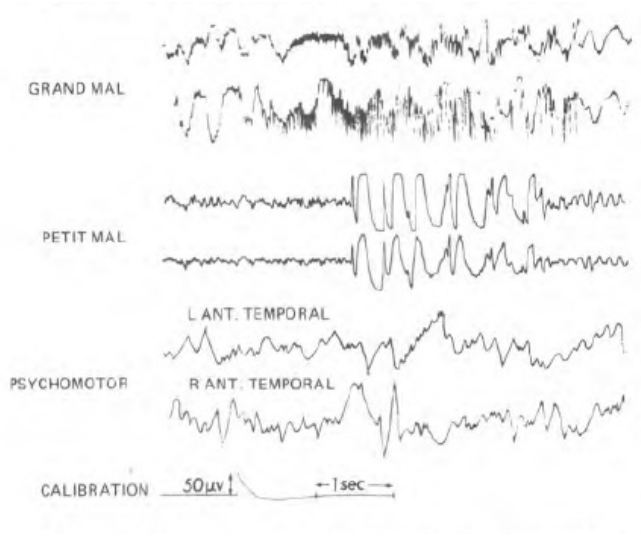

Figure 5-2.
Samples of EEG illustrating a brief major seizure (grand mal), spikes and waves accompanying an "absence" (petit mal), and a spiking discharge from the right temporal focus of a patient with psychomotor seizures.

If the above suggests the EEG may be a rubber crutch, this is not accidental. The surface EEG may give support to a clinical impression and as

such is justifiable. But the danger is that it may be used to justify poor clinical practice. For example, a geriatrics expert presented data on the EEG changes found in aging. Eighty percent of his cases who subsequently had demonstrable central nervous system (CNS) damage had an abnormal EEG. However, thirty percent who never developed evidence of CNS damage also had similarly abnormal EEGs. He concluded that the absence of EEG abnormalities should make one sensitive to the possibility of psychological problems. Should one also conclude that the presence of abnormal EEGs in a patient should allow one to ignore the possibility of psychological problems? I'm sure our geriatrics expert would be horrified at such a conclusion. No doubt he'd agree that optimum attention to psychological problems should be accorded to each person. Nonetheless, the EEG can be and, sad to say, often is an excuse for suboptimum clinical practice.

To summarize, we can say that the routine EEG arises mostly from graded potentials in the cortical feltwork. It reflects gross changes, such as sleep, space-occupying lesions, fits and death. An abnormal interictal EEG is common in seizure patients but not invariable, and abnormal EEGs can be encountered in people who never have had epilepsy. In short, outside of an overt major seizure, the correlation between epilepsy and EEG is statistical and not absolute. Most important, a normal EEG does not rule out gross cerebral pathology, nor does an abnormal EEG rule out psychologically mediated disability.

The EEG and Psychiatry

While the EEG was being applied to neurological problems (with more or less success), psychiatrists and psychologists worked, too, but with somewhat less to show for their efforts. Over the years, simple visual analysis of the EEG record has been supplanted by sophisticated and elegant computer-based systems, but psychologically relevant findings still remain controversial and meager on the whole.

Ellingson and Vogel and Broverman carried on an informative controversy about the relationship of the EEG to intelligence. Ellingson concluded that the EEG only correlated with intelligence by virtue of the fact that people with gross brain damage were more likely to have abnormal EEGs and to be less intelligent than people who had not suffered in this way. Vogel et al. marshaled considerable evidence to indicate that Ellingson was perhaps unduly pessimistic. But Ellingson's point is one to be taken seriously.

There has been some talk that slow EEGs (EEGs with lower frequency alpha) are associated with slower minds. Work on that continues, but the best bet is that no such simple correlation will be found. Correlations between EEG variables and such psychological traits as rigidity, automatizing, etc., continue to be reported from time to time. The EEG reflects the level of arousal, and since the level of a subject's arousal during specific situations will be related to such things as past experience, personality, and intelligence, it's not

surprising to find that EEG-personality correlations occur.

The relations between the EEG and the level of arousal are also a factor in studies on the effects of psychotropic drugs. Using elegant computer techniques, Fink and ltd have been able to show some more or less drug-specific EEG changes, but again, by and large, the EEG is more a function of the arousal of the subject than is the specific pharmacological agent employed. Social experience with alcohol should be sufficient for the reader to supply his own illustrations of how alcohol in equivalent doses can result in heightened interest and activity on one occasion (alcohol with friends at a cocktail party) and somnolence on another (alcohol alone in a strange hotel room). The EEG will in general reflect the state of the subject more than the dose of the drug.

EEG correlates in psychopathology also exist, and again the arousal factor may be important. There is some controversy about the correlation between the so-called six and fourteen EEG pattern and the psychopathic personality. These six and fourteen patterns are made up of mixed slow and fast waves and occur rather commonly in adolescents and youths, particularly when the subject is drowsy. The specific relationship of this to psychopathology is questionable, but one might wonder if psychopathic teenagers get drowsy in a testing situation faster than non-psychopathic teenagers.

The schizophrenic syndrome has also been associated with specific EEG patterns. In chronic, process, organic-like schizophrenics who show poor responses to phenothiazine drugs, one is likely to encounter abundant hyperstable alpha activity. In addition, schizophrenics in general are also likely to have an excessive amount of fast activity, which may give the record a choppy appearance. Finally, integrated voltage of the EEG is likely to be less variable in schizophrenia than in normal people. Specifically, the integrated voltage is computed for twenty-second samples of the EEG. After a series of these voltage intervals have been computed, the coefficient of variability is determined. This is obtained by dividing the standard deviation of these voltage integrals by the average voltage. Schizophrenics tend to have a lower coefficient of variability than do normal people. Average voltage itself does not distinguish schizophrenics from normal people so well, but the hyperstability does.

This again may be a function of arousal. The schizophrenic appears by many criteria to be in a chronic, poorly modulated state of hyperarousal. The normal person will show wide fluctuations in arousal during a testing session, and this may be reflected in a high coefficient of variability. The schizophrenic, on the other hand, is fixed in his anxious, defensive state and is notably lacking in adaptive plasticity, both by behavioral and by physiological criteria. This view, which attributes low coefficient of variability in schizophrenia to hyperarousal, finds support in the observation that

amphetamine decreases and sedation increases the EEG coefficient of variability.

Alpha Feedback

Behavioral modification by operant techniques is receiving more and more interest. Autonomic and other nonskeletal muscle (involuntary) responses were once supposed to be modifiable only by classical or Pavlovian conditioning. Some of the early attempts to test this dictum involved experiments intended to bring EEG alpha activity under operant control, since EEG alpha activity was considered a good example of involuntary behavior that was readily accessible to measurement. Although recently . called into question, the work of DiCara and Neal Miller has apparently proved that involuntary behavior can be operantly conditioned. In the meanwhile, the pioneering work of Kamiya indicated that the EEG alpha rhythm can also be brought under operant control. The method is fundamentally simple. The state of the EEG is made known to the subject. This can be done by providing a tone when the alpha waves are above a certain voltage. The subject is then asked to turn the tones on or off. Subjects learn to control the tones when they have no idea about the mediating physiological event.

The interpretation of these findings is something else. Eye position can affect alpha abundance, and just concentrating on tone bursts will also tend to

increase alpha activity. Of equal interest, however, is the fact that subjects tend to enjoy turning on alpha, and they report subjective experiences similar to those described by subjects involved in religious meditation. Finally, certain religious men who have become expert at meditation (Zen Buddhist priests, yoga teachers, etc.) appear to have unusual abilities to control their alpha activity.

This work on alpha control has important implications both for research on states of consciousness and for studies of comparative religions. Because of its exotic nature, it has captured the popular fancy. Varieties of do-it-yourself alpha-control programs are available, and some promise to help the purchaser obtain Nirvana quickly and painlessly. Some of these popularizations are naive to say the least. Others are frankly fraudulent. The fundamental importance of this work, however, should not be obscured by the fringe of carnival fast-buck operators it has attracted.

How to Average Evoked Potentials

The conventional EEG reflects global states. Death, fits, sleep, and general level of arousal can be determined with fair reliability. One suspects that correlations between more subtle psychological variables and the gross EEG also depend on indirect relations between the psychological variable, experimental conditions, and the resulting level of arousal in the subject.

The EEG, after all, looks at what the brain is doing moment by moment and since the waking brain is always busy at a variety of tasks, the potentials at the surface of the scalp are like a potpourri. Individual ingredients are hard to separate out and we notice principally the overall quality. How can we see potentials reflecting specific mental processes when they are buried in so much incidental activity? There are a variety of tricks, but most depend on getting the brain to repeat a specific act several times. If the incidental electrical babbling is random with respect to the specific activity in question, we can hope to distinguish between the repeating specific electrical events, and the other random or "noise" events.

The easiest trick is to average. As in all averaging procedures, the hope is that interfering variables will cancel out, leaving us with an accurate representation of the more or less consistent variable of interest. Specific brain responses can be seen in recordings from the cortex, and in some subjects, responses to single sensory stimuli are large enough to be apparent in the raw EEG in spite of all the background noise. Thus, evoked responses were known before averaging techniques were developed. The evoked response era, however, really began with averaging.

At first a variety of methods were tried. These included photographic techniques, magnetic tape methods, and so on. Condenser charging averagers are still used for special purposes, but today digital computers have pretty

much won the day. The usual method demands that we know more or less precisely when an event is occurring in the brain. We present a series of events and after each event, we take a set of samples of brain electrical activity timed as precisely as possible with the event. These voltages are converted to numbers in a computer, and voltage-values for corresponding times-after-stimulation are averaged. The simplest illustration is a sensory-evoked response. If the stimulus is repeated over and over, we can hope that the subject will respond in more or less the same way to each repeated representation. Figure 5-3 illustrates how average brain wave samples taken during the half second following a click can be averaged to disclose the averaged evoked potential, or AEP. The AEP is almost the electrical by-product of the specific CNS event. We can only say "almost," however, because of the requirement of averaging. The whole averaging process is based on the idea that the same response is evoked repeatedly, so that it can be made to stand out against the background noise. In fact, the brain rarely does anything exactly the same way twice, much less repeatedly, and the more important and complex the mental event is, the less likely it is to be repeated precisely.

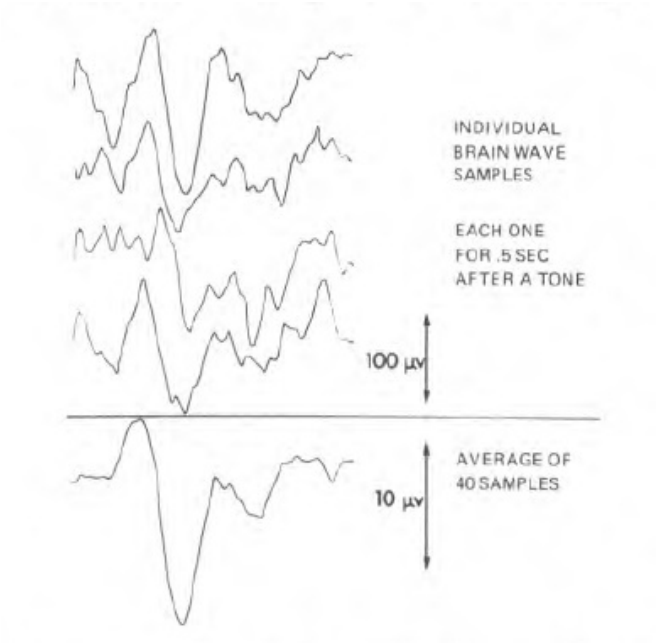

Figure 5-3.
The first five EEG samples of a set of forty samples are shown above the line, and the average of all forty are shown below the line. This subject had unusually large evoked responses in the first few samples.

Sensory-Evoked Responses and Neonatal Audiometry

Since the closest approach to repetitive brain response is its response to a periodic, meaningless sensory stimulus, evoked responses to such stimuli were the first to engage the attention of investigators, and it is not surprising that the most generally accepted clinical application of the AEP uses sensory-evoked responses to test sensory function.

By and large, if you want to find out if someone has received a stimulus, the best way is to ask him. Evoked responses become clinically useful when the person cannot or will not talk, or when we need some extralinguistic point of reference. Neonates can't talk, and it's important to determine promptly if they have hearing loss so that steps can be taken to supply sensory stimulation. Otherwise, there may be a permanent loss of sensory function. As one might expect, the ability to obtain an auditory-evoked response from an infant is suggestive evidence that the child is able to hear. It isn't proof since very large sounds can apparently produce an evoked response by their effect on the cochlear system, while, on the other hand, some children, and even adults, may fail to show demonstrable evoked responses and yet have intact hearing. In spite of these problems, evoked-response audiometry in neonates is in fairly wide clinical use today.[2]

Auditory-evoked responses have another practical application in the study of neonates. Gestational age can be measured in a number of ways. Best, of course, would be to know exactly when the child was conceived, but sometimes that isn't feasible. Weight at birth is also related to the gestational age, but some children are born unusually small or unusually large, even at term. Gestational age can be estimated from the raw EEG. This is best done by using EEG to determine stages of sleep and estimating gestational age on the basis of the nature of the sleep cycle. The auditory-evoked response can also be used to measure gestational age, since the latencies of the evoked response

tend to be long at first and become progressively shorter, particularly over the first six months after birth. Gestational age as estimated by the evoked response at birth predicts the time at which the child will walk. Gestational age, however, has very little predictive validity when it comes to any of the later talents of a child.

Evoked responses have been obtained using all of the other sensory modalities. Visual-, olfactory-, gustatory-, tactile-, and vestibular-evoked responses have been studied and, in principle, the AEP could be used to test sensory function in any of these modalities. In practice, however, the practical applications have not been impressive, although the use of visual-evoked responses for perimetry and refraction in mentally retarded subjects may prove to be of practical importance. Finally, there is promise that function of various sensory pathways and of primary sensory-receiving areas may be tested by methodically changing the nature of the stimulus and the site of recording. Such a neurophysiologically sophisticated approach may offer a useful alternative to angiography and brain scan for localizing intercerebral lesions.

Evoked-Potential Recovery Cycles

It is fortunate for us that Shagass, one of the first psychiatrists on the evoked-potential scene, has published a book that combines a review of

evoked-potential work relevant to psychiatry with a thorough presentation of his own work in the field.

Much of what Shagass and his followers have done involves recovery cycles. The basic notion is that one presents a conditioning stimulus and then, after a delay, a test stimulus. There tend to be periods of increased and decreased responsiveness in the interval following the conditioning stimulus, and these recovery cycles are probed by the test stimulus.

It is somehow intuitively appealing to consider that the impaired CNS of the psychotic patient might appear less efficient on neuro-physiological measures. Thus, one would predict that psychosis would be associated with depressed recovery. The test stimulus should generally evoke a smaller response in the psychotic patient. That is to say, the post conditioning-stimulus depression should persist longer.

In general, "'patienthood' appears to be associated with diminished amplitude recovery and earlier latency recovery. . . ." However, the problem is complicated by the relationship of the test response to the general level of responsiveness (as shown by a response to a test-type stimulus with no preceding conditioning stimulus) and by the dependence of the whole recovery-cycle phenomenon upon relative intensities of test and conditioning stimuli.

More recent work has shown that schizophrenics and drug abusers with a history of psychotic drug reactions both showed a reduced range of responsiveness when the intensity of the conditioning stimulus was varied. In general, the more intense the conditioning stimulus, the smaller the response to the test stimulus.

The complexity of these recovery functions may make them seem less appealing than some other simpler appearing measures. However, studies of recovery functions have provided a variety of significant correlations with psychopathology. Also, recovery functions may seem more complex simply because we know more about them (and hence about their complexity). Thus, they lay serious claim to the interest of the psychiatric investigator.

Cognitive-Evoked Potentials

The specific activity of sensory pathways is reflected in the early components and the AEP. These early components are of great interest to the neurologist and neurosurgeon, but are relatively insensitive to psychological factors. In fact, it is a general practice to sedate children for AEP audiometry, and to record the AEPs while the child is asleep.

The AEP does, however, have components that reflect phenomena of greater subtlety than the mere patency of sensory pathways, and these components might well be referred to as cognitive-evoked potentials.

To oversimplify a little, one can consider that there are two cognitive components to the AEP: (1) the contingent negative variation or CNV: and (2) the third positive wave or P_3 (at about 300 msec.).

If one records from the vertex and uses recording equipment with a very long time constant (i.e., with a DC or very low-frequency response), one can detect a negative-going change between a warning signal and subsequent stimulus that demands some response (imperative stimulus). This negative variation is most striking when the subject knows the imperative stimulus is contingent on the warning stimulus. Thus, it is referred to as the contingent negative variation or CNV.

Specifically, suppose we use a single click as a warning, and two seconds after each click we start a train of clicks that the subject must terminate by pressing a switch. The CNV will develop in the two seconds ready period. The magnitude of the variation may be as much as 15 to 20 μv and will depend on a variety of factors.

For example, it is increased by: (1) certainty that the imperative stimulus will occur. Thus it builds up with experience. (2) Interest in the imperative stimulus; and (3) physical force required to respond to the imperative stimulus.

It is reduced by (1) distraction; (2) boredom; and (3) sleep deprivation.

The CNV is correlated with behavior. In the usual paradigm, the faster the response to the imperative stimulus, the larger the CNV. But this is not always the case. For example, after sleep deprivation a subject can give normal reaction times, although his CNV is completely suppressed.

Physiologically the CNV probably represents an excitation or "priming" that is relatively specific. Thus, if a subject develops a CNV in readiness for a signal of 100 Hz, an interposed probe stimulus of 1000 Hz will evoke a response that is larger during larger CNVs—as though the excited state represented by the CNV augments the "probe" AEP. Such an augmentation is not found in the probe stimulus of a flash, or even a 600 Hz time.

The CNV holds some promise of practical clinical utility. It is reduced or absent in persons with psychopathic character disorders; it is variable in schizophrenics; it may be absent in retarded individuals; and there may be a second negative "equivocation" wave after the imperative stimulus in schizophrenics and obsessives—as though they tended to have afterthoughts following their responses.

At the moment, however, practical application is a mere possibility. The real value of the CNV lies in its use as a "convergent datum" in psychological constructs. For example, does the CNV represent awareness of contingency, interest, or arousal? How do we operationally define and separate these

concepts? In short, the CNV and P_3 bid fair to make honest men of heretofore loosely verbigerating psychologists.

This need for operational definitions of psychological constructs becomes even more obvious when P_3 is added to the general picture. Generally, the more physically intense a stimulus is, the larger the AEP components that occur before 250 msec, and the shorter are the latencies of these components. The more psychologically intense the stimulus is, the larger are the AEP components occurring after 250 msec, and the longer their latencies. Perhaps this means that physically strong stimuli get into the CNS quicker and involve more neural circuits but that psychologically more intense stimuli require more processing time—although they also call into play more neural circuits. Whether or not that neuro-physiological fantasy is true, it is a useful mnemonic.

This psychological augmentation in the AEP can be seen from about one hundred msec, on, but it is not seen in the negative wave which usually occurs around two hundred msec. That wave indeed may increase with light sleep! Cognition should take more time than simple sensory registration, and it is reassuring that psychological augmentation is most striking in the late positive component.

There is some confounding between the CNV and P_3. Since the

expectancy of a predictable but intrinsically interesting stimulus will produce both a CNV before the stimulus and a P3 on stimulus delivery, the question arises whether the P_3 is the "resetting" of the CNV. The best answer is to say that one can vary P_3 independently of the CNV, for the P_3 reflects the information delivered by the stimulus and the CNV reflects the subject's expectancy. On the other hand, expectancy and its accompanying arousal can influence both the information delivered by the stimulus and —perhaps independently—amplitudes of some AEP components.

In studies of attention, words like contingency, arousal, interest, uncertainty, information, etc., have a tendency to float about without anchor. CNV and P_3 seem destined to provide some means of tying these concepts down.

AEP and Intelligence

Having complained about the paucity of good correlations between the EEG and complex psychological processes, it would be worse than ungrateful to complain about the embarrassment of riches provided by the AEP. For example, while the EEG provided few convincing correlations with intelligence, this is not the case for AEPs. There are at least three (and perhaps four) different aspects of the AEP that correlate with the I.Q. The four AEP aspects are: (1) latency (short latency = high I.Q.); (2) plasticity (plastic

or adaptive response = high I.Q.); (3) asymmetry (asymmetrical visual AEP = high I.Q.); and (4) variability (variable response under stable conditions = low I.Q.). We will consider the first two in some detail to convey the flavor of the problems that still exist in this area.

The first work on the AEP and the I.Q. was done by Ertl, who sought a measure of neural efficiency in measures of AEP latency. The logic is impelling. A fast brain should go with a fast mind. The speed with which AEP waves appear seems to reflect the speed of the brain in processing data at least in some cases, so the latency of AEP waves in general might reflect neural speed (or efficiency) and hence provide a measure of intelligence.

There is no doubt that correlations (on the order of r = —0.30) can be found between visual AEP latency and I.Q. One needs to present from one hundred to eight hundred flashes that carry no special meaning to record the AEP from a central-parietal, bipolar-electrical pair, to measure latencies to the third or fourth wave—after defining "third wave" by counting peaks from stimulus onset.

But does AEP latency reflect neural efficiency? Perhaps. However, an alternate explanation can be found in AEP plasticity, which also correlates with the I.Q. Plasticity refers to the changes in the AEP that occur with changes in the demands of the occasion. Consider an experiment reported by

Dinand and Defayolle. While the subject faced a projection screen, flashes were presented at the periphery of his visual field. Most of the flashes were white and AEPs to the white flashes were recorded. Occasional flashes were red and the subject had to press a key when he saw a red flash. At one sitting the subject counted the white flashes and the AEP was large. At another sitting, complex logical problems were presented on the screen and, as would be expected, AEPs to the peripheral flashes were suppressed. The change in AEP amplitude (or AEP plasticity) correlated strongly with I.Q. ($r = 0.79$!).

Now it seems that "plasticity" explains, or at least plays a role, in the correlations between AEP latency and I.Q. Shucard replicated Ertl's work, adding two conditions. First, subjects pressed a key to each flash, then they counted each flash, and finally, as in Ertl's procedure, they just watched. AEP amplitudes were highest and latencies longest, when the flashes required an immediate response. Latency-I.Q. correlations were smallest when subjects were alert and responding, and largest when the subject just watched the flashes. Finally, the drop in AEP amplitude from the first (attending) to the last (non-attending) condition also correlated significantly with I.Q. After the entire session, bright subjects rated themselves as more bored than did the dull subjects. If bright subjects become bored more quickly, then in a moderately boring situation, they would develop smaller AEPs with shorter latencies than would dull subjects. In other words, bright subjects, by being more "plastic," develop shorter latencies, and thus "plasticity" rather than

"neural efficiency" may account for Ertl's finding.

The stories for the other AEP-I.Q. correlations are equally complex at this time but perhaps the above gives enough of the flavor of things to warn the reader against the uncritical ingestion of the brief survey that follows.

Rhodes et al. found that differences in visual AEP amplitudes recorded over the left and right hemispheres are greater in bright subjects. Lairy et al. also found alpha averages (averages triggered on alpha peaks rather than on stimuli) more asymmetrical in bright subjects. Conners, on the other hand, found striking AEP asymmetry in severe dyslexia; so asymmetry cannot be taken uncritically as an indication of a good intellect.

Variability refers to the trial-to-trial changes in the individual evoked potential (EP), and it cannot be measured in a single AEP. Schizophrenic patients have more variable EPs than do non-schizophrenic patients. Among schizophrenics, non-paranoids are more variable than paranoids. Over a period of time, a given patient will show increased EP variability when his thought-process disorder worsens, and decreased EP variability when he improves.

In children, EP variability is high and measures of EP variability are inversely correlated with age from six years to sixteen years.

Since with more maturity and less schizophrenia, EPs tend to be more stable, it is not surprising that correlations between EP variability and I.Q. can be found. Here too, there are problems. Measurements of EP variability are complex and sometimes difficult to interpret. For example, EP variability and background EEG are inextricably confounded. Then, too, maturity and cognitive stability are not perfect correlates of intelligence. We all know very bright colleagues who are quite immature and who may even be a trifle schizophrenic.

Cognitive Style

Perhaps one of the hazards in the way of applying AEPs to psychiatric problems lies in our tendency to try to force the information we get from AEPs into psychological constructs like I.Q., which are not all that good in the first place.

We mentioned above how EP variability is high in schizophrenia. This tells us more about how a schizophrenic thinks than about any innate disease process. Everybody knows that schizophrenics tend to show more variability in almost every sphere of behavior than is normal. However, certain sorts of simple tasks will allow the schizophrenic to perform with as little variability as a non-schizophrenic. With such a task, the schizophrenic's EPs are also no more variable than normal.

Thus, the late components of the AEP may best be looked on as another way of examining cognitive process rather than as a way of looking at fundamental neuropsychological substrates.

There are many new studies using AEPs to examine relatively enduring patterns of cognitive functioning, which (to borrow a term popular among psychologists a decade ago) we may call cognitive style. There are fascinating AEP studies of attention deployment and field dependence, but some consideration of repression and augmenting-reducing will serve again to give the flavor of the field. The field is moving so fast that by the time this is published the fine details will be in need of revision anyway.

The concept of augmenting-reducing goes back to the work of Petrie, but has been considerably elaborated by Buchsbaum and Silverman. With increasing stimulus intensity, there is a tendency for all responses to increase, and this is true also for AEP amplitude. In many subjects, however, intense stimuli result in responses that may be smaller than those evoked with moderately strong stimuli. This seems to reflect the operation of a kind of automatic gain control whereby subjects who are particularly sensitive to low level stimuli can avoid being damaged by high level stimuli. Subjects with particularly active automatic vain controls will “reduce” their AEP amplitudes at high stimulus intensities and they are referred to as “reducers.” Those with a more monotonic stimulus-intensity, AEP amplitude function are called

"augmenters." One's position on the augmenter-reducer continuum does seem to be a more or less enduring trait. It also seems to correlate with certain pathology. For example, lithium-responsive manics are augmenters while manic and lithium converts them into reducers.

The concept of repression is much more familiar. Shevrin et al. defined repression on the basis of Rorschach responses. For the evoked-response portion of the study, they used either meaningful or non-meaningful stimuli and presented the stimuli tachistoscopically so briefly that the subject could not consciously recognize them. Repressed subjects tended to show smaller evoked responses to the meaningful stimuli and larger responses to the non-meaningful stimuli than did the non-repressed subjects. The repressed subjects also gave fewer responses in their free associations that could be related to the meaningful stimulus. By contrast, when the stimuli were presented supraliminally (i.e., when they were presented for thirty msec, so that the subjects could clearly recognize the content of the meaningful stimuli) repressed subjects then gave larger responses to the meaningful stimuli and smaller responses to the non-meaningful stimuli. Although this work has no immediate clinical utility, it does provide valuable convergent data on the concept of repression.

Conclusion

As this is written, there are no established clinical procedures using the EEG that are relevant to the diagnosis and treatment of psychological problems, but I suspect that this is a temporary state of affairs.

The EEG will continue to supply clues when one is tracking down epilepsy or considering a gross intercranial lesion. Soon evoked potentials and other computer-based techniques (i.e., cross spectra) may become routine tools to help select the proper medication for psychotic patients. We may use such techniques to distinguish between various forms of minimal brain dysfunction in children. In special situations, they may supplement more conventional intelligence tests.

Before all the details are agreed on, there will be a time when extravagant claims and unrealistic expectations will make optimum use of these new tools most difficult. To keep a proper perspective, it is useful to consider that the EEG and its computer-derived products (such as the AEP) are examples of brain-controlled behavior. Some such behavior is simple and stereotyped, like pupillary reflexes —or early components of sensory AEPs. Other such behavior may be complex and rich in psychological significance as, for example, language and cognitive-evoked responses. Although arcane electronic devices may be used to observe and record this electrical behavior, it is neither more nor less "biological" than any other sort of behavior. When a man says he is frightened, we look for other convergent data such as tremor,

sweating palms, facial pallor, etc. to determine what inferences to make. We generally use several sorts of behavior to make inferences both about the structure of a person's brain and about its functions. EEGs and AEPs have more degrees of freedom than do the other types of behavior that don't depend upon skeletal muscles. So far there are only a handful of people who have voluntary control of their brain-electrical behavior. Thus, such behavior is resistant but not immune to mendacity.

In brief, newer techniques for observing and recording brain-electrical potentials give us access to a rich new set of behavior that can be used as other sorts of behavior are used, in the study of the human mind.

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Notes

1 The author is indebted to Mrs. Keiko LeVasseur for her work on both the illustrations and the manuscript, to Dr. Charles Yeager for the EEGs in the figures, and to Mrs. Hilary Naylar for the AEP Figure.

2 There now exists the ERA-Club (Evoked Response Audiometry) and the informative proceedings of their first international symposium have been published in English in Band 198, Heft 1, 1971 of Archiv für Klinische und Experimentelle Ohren-Nasen- und Kehlkopfheilkunde.