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Clinical application of electroencephalography in the study of epilepsy : present status

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THE CLINICAL APPLICATION OF

ELECTROENCEPHALOGRAPHY

IN THE STUDY OF

EPILEPSY: PRESENT STATUS.

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THE CLINICAL APPLICATION OF ELECTROENCEPHALOGRAPHY IN STUDY OF EPILEPSY: PRESENT STATUS.

HISTORICAL INTRODUCTION

The history of electroencephalography had its beginning in 1874 when Caton, an Englishman, discovered evidences of electrical activity in the brains of living animals. He noted(11) electrical fluctuations in rabbit cortex and thought these were related to functional activity of the brain cortex. Caton measured currents directly from the cortex by means of a galvonometer. He found that the external surface of the cortex was electrically positive in relation to the cut surface of a section through it and that "...when any part of the gray matter is in a state of functional activity, its electric current exhibits negative variation."

Beck, in 1890, noted large fluctuations in potential in the visual cortex of the dog when the eyes were illuminated, and that smaller fluctuations occur without stimulation or relation to respiratory or cardiac rates(5). His experiments also included a study of the brain, cord and sciatic nerve of the frog which had been removed and placed on a glass plate. He found that without stimulation, continuous potential changes were observed from two electrodes on the cortex which

he regarded as "action currents." These potential
changes were also shown not to be related to organic
rhythms, such as respiration or pulse. They continued
independent of controlled afferent stimulation al-
though they were altered by such stimuli and by certain
drugs(5).

After Beck's paper was reported, the Vienna Aca-
demy opened a sealed paper by Fleischl von Marxow.
He said(69) that he had recorded, from the visual area,
large potentials when an animal's eyes were illuminated;
that these potentials could be obtained through the
dura and skull and were abolished by chloroform and
cooling(69). Apparently von Marxow was not sure of
his work for the Academy directors were not to open
this paper until other reports of the electrical acti-
vity of nervous tissue were published.

Gotch and Horsely(32) believed that special
cortical areas gave an electrical response to certain
types of peripheral stimulation. Their work was con-
ducted in 1892 using the "galvanometric method" to
obtain resting potentials between the cut end of the
cord and different parts of the cord and brain. They
were chiefly concerned with the electrical changes in
the cord and sciatic nerve in response to direct cor-
tical excitation. Excitation was found to produce,

after the usual negative variation, a marked positive
after-potential which remained several seconds after
the stimulus period. In addition, they also discovered
a "complete fusion of impulses" of no detectable inter-
mittency, as observed from the electrometer records of
cord potentials, during the tonic phase of cortical
excitation. During the after discharge following the
stimulus, smooth cord potentials at about ten-per-second
were associated with muscle contraction rhythms of the
same frequency. This frequency was considered a func-
tion of certain rhythmic characteristics in the dis-
charge of the higher centers. The exactitude of these
workers' measurements and their report of ten-per-second
rhythm will become apparent in the succeeding pages.

Beck, in 1892, working with Cylbuski, showed that
a local injury to the cortex modified its electrical
activity and that the stimulation of a dog's leg pro-
duces a response in the contralateral cruciate area(6).

Danilewsky, in 1891, reported studies of cortical
localization by galvanometric responses of the cortex
to tactual, auditory, olfactory and direct sciatic
nerve stimulation(12).

In 1904, Tchiriev(68) concluded that potential
fluctuations of the cortex were not related to nervous
activity but are the result of pulsations of blood in

the blood vessels. In 1912, however, Kaufmann(44) re-affirmed the work of earlier investigators that elec-trical potentials from the cortex are modified by stimulation, and are indeed related to nervous function.

Prawdicz-Neminski first used the Einthoven string galvanometer in 1913 after it had become available in 1906. He reported(56) that cortical potentials can be observed when a dog's sciatic nerve is stimulated and that spontaneous waves could be observed from the motor and occipital cortex. In 1925, Prawdicz-Neminski gave a complete account of his extensive experiments on the cortical potentials of the dog with a classification of the types of spontaneous potentials in terms still used today. Neminski stated(57) that the electrocerebrogram consisted of spontaneous potential fluctuations with a frequency of ten- to fifteen-per-second, which he des-ignated as waves of the first order, and other faster fluctuations ranging from twenty- to thirty-two-per-sec-ond, called waves of the second order.

The studies from 1874 onward were, with but few exceptions, in general accord, but overlooked the possi-bilities of directly studying an electrical component of brain activity for 40 years (1874-1933).

Hans Berger, however, recognized the importance of these observations and in 1902 and 1907 he recorded spon-

taneous fluctuations in potential from animal brains,
but was skeptical of their origin since on neither
occasion was he able to show that these fluctuations
were modified by sensory stimulation. He confirmed
Prawdicz-Neminski's observations on dogs in 1928.
In 1924, he attempted successfully to record the elec-
trical activity of the human brain. The first report(8)
of these results in 1929 were greeted with skepticism
and incredulity. Adrian, of England, the distinguished
Nobel Prize Physiologist, was very doubtful, repeated
Berger's work and verified(2) the phenomenon.

By 1934, Berger had shown that the brain of man
has an electrical beat; that this beat comes from ner-
vous activity of neurons, not from blood vessels or
connective tissue; that it changes with age, sensory
stimulation, and with various physico-chemical state
changes in the body. These beats were found to occur
in sinusoidal fluctuations with a frequency of from
1-60-per-second, and that the most prominent rhythm has
a frequency of ten-per-second in normal adults. He des-
ignated these as alpha waves and showed that they tend
to be abolished by attention. Waves of 15 to 60-per-
second were called beta waves. Berger also renamed
the graphic record of electrical brain pulsations be-
cause he objected to the use of combined Latin and Greek

study of epilepsy as heretofore believed(42).

Much work has been done by other investigators but a paper of this type precludes even mentioning them. Excellent summaries and bibliographies can be obtained, however, by those interested. Jasper has provided a thorough summary of the subject up to 1937(38). Gibbs and Gibbs, in their monumental Atlas of Electroencephalography, give a comprehensive bibliography up to 1940(26). Barnes has given an interesting bibliography in a recent publication(4).

BASIC PRINCIPLES UNDERLYING ELECTROENCEPHALOGRAPHY

Nature of the Phenomenon:

The activity of all cells show mechanical, thermal, chemical and electrical components. A study of these quantities have shown them to be directly related to the activity of the tissue in question. These factors are closely integrated and cannot be considered separately. Poor instrumentation or lack of comprehension, however, necessitate the consideration of only one of the components of cellular activity. Thus, when we speak of cortical activity in terms of electrical activity, the accompanying chemical, thermal, and mechanical changes are implied and should not be forgotten or divorced from the electrical changes even though the

former are too infinitesimal to be studied by available methods at the present time(26).

The electrical activity of the cortex shows a marked evidence of rhythmicity which is also manifested by other tissues, particularly smooth muscle, nerve, and heart muscle. Normally, the recorded beat in the EEG responds to stimuli, such as illumination of the retina, with a decrease in amplitude(8) and an increase in frequency(4). This inverse relationship between frequency and amplitude is also usual in smooth muscle, nerve, and heart muscle.

The Gibbs(26) have pointed out that the closest relationships exist between the cortical electrical activity and the respiratory center. The latter is a rhythmically-acting mass of nervous tissue innervating a recording apparatus, the muscles of respiration. Many agents affecting the frequency of cortical potentials also affect the frequency of respiratory potentials in the same way. Abnormalities of cortical rhythms have their homologues in abnormalities in respiratory rhythm. Agents tending to correct abnormalities in one, tend to correct abnormalities in the other. The likenesses of these two masses of nervous tissue are pointed out in Fig. 1.

ELECTROENCEPHALOGRAM

GRAND MAL EPILEPSY   PETIT MAL EPILEPSY   PSYCHOMOTOR EPILEPSY

RESPIRATORY MOVEMENTS

TACHYPNEA   PERIODIC RESPIRATION   BRADYPNEA
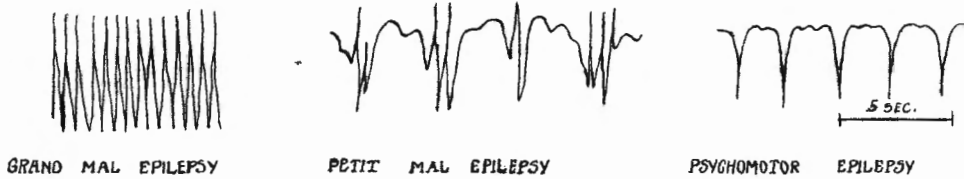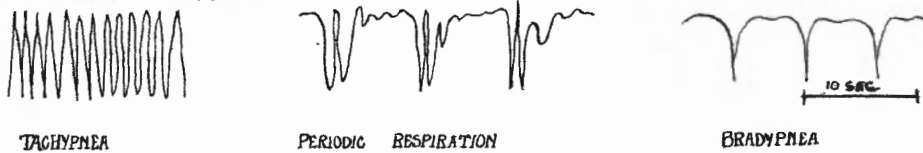
Fig. 1 . Similarities Between Cortex and
Other Tissues.

The commonest disorders of cortical rhythm
have their counterpart in certain disorders
of respiratory rhythm. The new brain and the
old brain resemble each other in their path-
ological as well as in their normal activity.
It should be noted that the time scales for
the electroencephalogram and for the record
of respiratory movements are not the same.

From Gibbs (26)

The Electrical Activity of the Cortex:

A. Origin.

By 1934, Berger(9) had proved that the electrical
activity of the brain came from the activity of neurons
and not blood vessels or connective tissue. These
cortical potentials have been attributed to summations
of potentials of individual neurons and groups of
neurons(2) and cell bodies or synapses(29). It is now
generally believed that the spontaneous potentials
of the cortex originate principally in the nerve cell
bodies(17)(51) rather than in the axons. Jasper and
Kershman(43) state the views of the Russian physiol-
ogist, Beritoff, who does not believe cortical waves
are due to integrated synchronized nerve cell potentials
but attributes them to the "neuropile" which he defines
as "dendritic and synaptic masses of gray matter" and
to self-exciting "closed neuron circles" which are
continuously active under the influence of internal
physico-chemical conditions.

B. Properties of cortical potentials.

The more important properties of cortical poten-
tials can be determined by studying the waves seen in
the EEG. Those properties which are generally agreed
upon include the following:

    1. Omniprescence and continuous action are the

most striking things about cortical potentials.
Cortical waves are abolished only in great stupor,
extremely deep anaesthesia, and death(38)(26).

2. Spontaneous automatism is an important
property which has been repeatedly emphasized(18)(40)
(39). Davis(18) has further pointed out that spontaneous
cortical activity is not maximal.

3. The organization (synchronization) of cortical
discharges into patterns is one of their recognized
properties. All workers are not in agreement, however,
as to how this is accomplished. Adrian(2) noted that
cortical effects are mainly built up out of repeated
brief potentials and are not due to gradually develop-
ing potential gradients. He felt that the cortical
neurons beat in phase in small groups over three to
four millimeter-areas of the cortex. Davis(18) further
emphasized that cortical potentials probably represent
summated potentials of individual neurons and went on
to state that they represent organized patterns of
activity(15). Jasper(38) believes that some records
give an indication of the form, frequency or amplitude
of cortical cell discharges. However, as he brings out,
some men are doubtful of these results so that one cannot
say exactly how cell discharges synchronize to produce
the measured potentials. The electrical activity from

a given brain area does represent the resultant poten-
tial from thousands of individual cells. These rhythm-
ic waves of cortical activity provide a sensitive in-
dicator of the frequency and intensity, but principally
of the nature of the organization, of cortical dis-
charge.

4. There is some degree of correlation between
the form of cortical discharge and cyte-architectonic
structure over different areas of the brain. Davis(16)
quotes from Kornmüller in the belief that the general
pattern of cortical activity differs from place to place
depending upon the histologic structure. Others,
in studying this problem, have disagreed and no definite
answer can be given at this time(38). There is con-
clusive proof, however, that laminar cell structure
determines certain elements of the complexes of po-
tentials obtained(38).

5. There is a remarkable constancy of frequency
in an individuals record when "basal" conditions for
recording are maintained(38). This has prompted the
belief by some that this might be due to a period of
refractoriness in the cells or circuits involved.

6. Wide gradient of frequency is manifested by
cortical potentials. Gibbs(45) speculated on a "spec-
trum" of cortical frequencies as an aid to interpre-

tation of the EEG. The long wave-lengths or slow waves, are correlated with surgical anaesthesia, stupor and sleep. The shorter wave-lengths are correlated with attention, fright, confusion, or ether and alcohol excitement. Such a spectrum has been questioned by some but the concept has found some acceptance. Gibbs(18) reiterates this belief by stating that it is more accurate to regard cortical frequencies as a continuum. Analysis by the Fourier Transform shows that energy is released at all frequencies from 1 to 60-per-second.

7. The individuality of cortical discharges seen in the records of different persons is a rather less important property. Roughly, an individual's electre-encephalogram is as distinctive as his own style of handwriting(53). As Davis(14) has so aptly stated, "...inevitably we compare the electroencephalegram with handwriting, each word different from what comes before and after, yet made up of the letters of a limited alphabet." Some writers have felt that an individual's EEG is as distinctive as his fingerprints but this is generally not conceded(51). Second records on indi-viduals closely resemble the first after weeks or months(16), and the individuality of an individual's record is supported by studying the records of identical

twins. These records show very similar patterns(18).
Although EEGS are characteristic of the individual,
many persons have EEGS which are indistinguishable from
the EEGS of other individuals.

8. The alteration of electrocortical discharges
by various stimuli is exceedingly important. This
property of brain waves will be considered in detail
at another point in this paper.
Theoretical Interpretations of Electrocortical Dis-
    charges:

What produces electrical discharges and what
factors regulate periodicity, amplitude, and other
properties of these discharges? The answers to these
and other questions are largely unknown and form the
basis for controversy among different workers in this
field although a fairly large amount of experimental
work has been done in an attempt to elucidate them.

It seems clear that the ability for spontaneous,
periodic discharge is intrinsic in the cortical cells.
This is somewhat analogous to the inherent rhythmic
contractions of cardiac muscle fibers, but unlike the
latter, it has never been demonstrated directly that
a single cortical neuron is capable of sustained rhythm-
ic discharges without periodic activation by nerve
impulses arriving at its synapses(38).

The theory has been proposed by some that chains
of central neurons form closed circuits around which
nerve impulses may circulate indefinitely, so long as
the refractery period in each neuron or circuit remains
short enough or the rate of travel of the impulses is
not too rapid(38). Such intra-cortical circuits have
been demonstrated histologically. The importance of
these "loop" or "reverberating" circuits is pointed
out by Lindsley(51) who believes that they may be
necessary to the maintenance of certain types of
spontaneous activity.

Jasper and Kershman(43), in summarizing recent
progress in the field of electroencephalography, take
a positive stand in this matter. They state that the
alpha rhythm of the cortex is dependent upon intact
projection fibers to certain subcortical (thalamic?)
nuclei as evidenced by cortical potential facilita-
tion after hypothalamic stimulation, and a generalized
inhibition of cortical activity following stimulation
of the striatum.

The importance of certain electro-tonic or chemical
conditions has been pointed out by some workers(38)(51)
(43) as being necessary for the maintenance of cortical
potentials. Lennox, Gibbs, Gibbs and their co-workers
have been particularly interested in the development

of this conception(25)(26)(27)(28)(47). In particular
they emphasize the importance of the proper blood sugar,
oxygen and carbon dioxide levels in the maintenance
of a "normal" type of potential discharge. Hoagland(35)
has referred to these as "chemical pacemakers."
Level of Cerebral Function as Correlated with Electro-

encephalographic Findings:

It has been repeatedly demonstrated that the pre-
dominant ten-per-second waves obtained from most indi-
viduals undergo certain physiological changes. This
rhythm is accelerated by heat, abolished by retinal
stimulation, mental effort or psychic changes. The
so-called beta rhythm of 25-per-second is also modified
by certain stimuli although the results are not so
clear or constant. The best records of relatively
large amplitude alpha ten-per-second activity are ob-
tained with a relatively low level of waking cerebral
function.

Because cortical potentials represent the summated
potentials from thousands of individual nerve cells, and
are an indicator of their functional activity, it is
paradoxical that relatively large amplitude alpha of
ten-per-second should be obtained with a relatively
low level of cerebral function(13)(39). A possible
explanation for this might be that the large amplitude

ten-per-second waves are indicative of an ontogene-
tically low level of activity whereas the rapid and
low voltage type of record is in accord with a higher
level of cortical activity. Thus the slower, high-
amplitude rhythm is dispersed or desynchronized by the
arrival of a volley of afferent impulses(34).

Darrow(13) has advanced an interesting theoret-
ical explanation for this phenomenon. He attributes
the paradox to inhibitory effects of subcortical ten-
per-second pacing of activity in the cortex and to
associated autonomic effects causing cerebral vaso-
constriction. On the other hand, fast activity is
associated with cortical excitation, metabolite (car-
bon dioxide) production, and fast activity in choli-
nergic nerves accounting for vasodilatation. Subcor-
tical pacing produces ten-per-second rhythm through
"loop" circuits and associated vasoconstriction whereas
the metabolite production, cholinergic nerve activity
and vasodilatation inhibit the ten-per-second pacing
when the cortex is functioning in a state of higher
activity. In this way, by two opposed effects of fast
versus ten-per-second slower activity, cortical and
subcortical mechanisms regulate one another. Thus,
the excitation in the cortex is normally prevented
from producing excessive discharges or from self-

perpetuation.

The level of cortical activity as measured by the
EEG and its relation to muscle function levels, studied
by the EMG, has indicated that cortical potential
rhythms may be definitely related to nerve impulses
reaching the final common path to the muscles. Under
normal conditions, secondary rhythms in muscle also
appear to be synchronized to a certain degree with
similar characteristics of subcortical centers(38).
Not only efferent stimuli can be related to cortical
potential discharges. Specific stimulation effects,
including direct cortical stimulation by drugs and
electrical stimuli, may abolish existing rhythms or
invoke new discharge patterns in the cortex. Afferent
stimulation causes depression of spontaneous activity
as previously mentioned and the stimulus after-effects
outlast the time through which the stimulus is applied
and is followed by a "rebound" effect in activity.
Adaptation also occurs after stimulation and there is
a gradual return of rhythmic activity with continuous
or repeated stimuli. In addition to these effects, "ev-
oked" potentials are obtained with afferent stimulation.
Auditory, visual, proprioceptive, cutaneous, olfactory,
and gustatory and labyrinthine stimuli are found to
evoke potentials with "on" and "off" effects, at the

same time abolishing (desynchronizing) the spontaneous
autonomous activity in the cortical projection area
specific for these sensibilities.

Methodology:

The wide variation of technique employed in
different laboratories makes the evaluation and com-
parison of results quite difficult. Placement and
types of electrodes, types of recording instruments
and other inconstancies in technique and methodology
obscure comparative studies.

The value of some criteria set up for statistical
study is questionable for general clinical work so
they will not be considered in this paper unless re-
ferred to specifically at which time a brief descrip-
tion of the method will be given. These purely des-
criptive or statistical studies are of limited value
anyway until more is known regarding the brain mech-
anisms they represent.

For this paper, a brief description of the gen-
erally used electrode placements, leads, instrumentation
and conventions will be given.

A. Basal Conditions.

Evoked potentials from a restricted cortical area,
due to impulses from specific sensory stimuli, often
pass undetected, being masked by the more prominent
autonomous rhythms. Clinical electroencephalography

is concerned almost entirely with autonomous rhythms, potentials not evoked by specific sensory stimulation. One of the factors affecting the excitatory state is the amount and kind of afferent influx, so that autonomous rhythms are conditioned by sensory stimulation. Evoked potentials are transient waves in response to each stimulus. Therefore, in recording EEGS for clinical work, it is desirable to eliminate any evoked potentials that might be picked up so the patient is allowed to sit or recline (with his eyes closed) in a comfortable position and in a darkened and adequately screened cage-room. It is paramount that these basal conditions are maintained for a "normal" record. Drowsiness or sleep must be carefully avoided.

B. Electrodes.

The spontaneous, apparently autonomous rhythms of electrical activity normally develop voltages in the order of 100 to 1000 microvolts at the surface of the cortex. The resistance offered by the dura, skull and scalp reduce them to 1/10 or 1/20 of this amount at the scalp surface or to the order of 5 to 100 microvolts(39). The electrodes which pick up these minute currents should therefore be easy to apply and maintain firmly in position. They should be easy to remove and artifacts should not be produced by them. Many devices

have been used in the past including small pellets
of conductive metal held in place with collodion, rubber
bands, tapes, and bandages. Needle electrodes have
been placed in the scalp but give trouble with oozing
tissue juices and pain is involved in their application.

The most commonly used type of electrode is des-
cribed by Gibbs and Gibbs(26). A small plate of solder
metal is covered with a salt paste on one side and
applied to the scalp with collodion. Fine flexible
insulated wires connect the electrodes to the record-
ing apparatus.

C. Electrode Placement and Leads.

In actuality, only the activity immediately under
an electrode is recorded. Gibbs and Gibbs(26) state
that the "short-circuiting action of the tissue is so
great that the voltage recorded from a given cortical
area decreases as approximately the square of its dis-
tance from the electrode." They advocate the use of
monopolar leads. In this placement one electrode is
placed on the lobe of each ear and serves as an indif-
ferent electrode. The other electrode is placed over
the cortical area where one wishes to measure the
electrical activity. Here the inactive area provides
a zero reference point or base line. If a record is
made by leading between two electrodes, both over active

cortex (bipolar lead), the record obtained consists essentially of the algebraic sum of the activity under each electrode.

For monopolar leads the standard placement of leads is described by Gibbs(26). One electrode on the lobe of each ear serves as an indifferent electrode. Two electrodes high on the forehead serve as the frontal electrodes. Two more electrodes are placed over the parietal area two or three centimeters from the midline and directly above the external auditory meatus; these are the parietal electrodes. Two occipital electrodes are placed two centimeters above the external occipital protuberance and approximately two centimeters from the midline. Monopolar leads are more often used by workers in this country and bipolar leads by men in England and Canada.

There is no standard placement of bipolar leads and each worker gives his own designation to this type of recording. A commonly used method is to use the electrode placements as described by Gibbs and to record between these placements. Thus recordings can be taken between two leads on the same side or between different leads on the two sides of the skull.

C. Localization Methods.

The most accurate localization of surface cortical

potentials may be obtained with bipolar leads and has
been described by Jasper(39). Their disadvantage lies
in the fact that electroencephalograms obtained from
them suffer much distortion due to the interference of
simultaneous activity from each electrode. Monopolar
leads are often used and it is necessary that the
active electrode be placed close to the source of
activity with the diffuse lead at a relatively long
electrical distance from active cerebral tissue. When
both electrodes are equidistant from the area in ques-
tion, bipolar leads are obtained. When properly carried
out the monopolar recording obtains a more accurate
picture of the true form of electrical activity with-
out the distortion from simultaneous activity at each
electrode. However, it is often difficult to do this.
For example, if there is a very active focus of abnormal
activity on the inferior surface of the temporal lobe
with very little activity at the "active" electrode,
the so-called "indifferent" electrode on the ear lobe
will become the "active" electrode when judged in
terms of its proximity to the source of disturbance.

The method of phase reversals is often used(34)
(39). The fundamental principle involved is simply
that a potential gradient on the head surface is up-
ward until its peak is passed and it reverses direction

to go down on the other side. If a number of elec-
trodes are placed in a line across the peak and sim-
ultaneous records taken (in successive pairs) of the
potential appearing, up one side and down the other,
the peak may be located by the point at which a rever-
sal in direction of the deflection of the recording
pens indicates a change in sign. Abnormal electrical
activity arises in gray matter adjacent to an area of
pathology in the border zone of pathological nerve
tissue between the lesion proper and normal brain.
It is abnormal function, rather than demonstrated
structural alteration (except as related to function),
which is localized by the EEG.

The principles of triangulation are the same as
those used in phase reversals along a line of elec-
trodes, except that only three electrodes are used
and these are placed in the form of a triangle. Three
simultaneous electroencephalograms are then recorded
from the three sides of the triangle and the method of
phase reversals is applied to locate an area which
has a different type of discharge than the tissue
surrounding it.

Other methods of localization include the use of
the basal lead, which is in contact with the perios-
teum of the sphenoid bone and is a type of triangulation

(34), and the dural lead and the pial lead.

D. Instrumentation.

The method of amplification of minute cortical potentials is accomplished by using vacuum tube amplification. The order of amplification is about 10 X 10^6 and the greatly amplified current records its fluctuation either by ink-writing pens or electrically heated wires writing on the recording paper. In either event, the paper is fed through the machine at a constant rate which is usually three or four centimeters-per-second thereby making frequency determination possible. Calibrating circuits make it possible to adjust the order of amplification in each case and to interpret it later by a calibrating device.

E. Conventions.

The polarity of the system is arranged so that a negative charge on the ungrounded side of the input registers as an upward deflection on the recorder. Voltage is measured from peak to trough of a wave. Frequency is the number of times a wave recurs in one second. Duration of a wave is the actual time it takes to complete a cycle between trough and trough or crest and crest.

THE NORMAL ELECTROENCEPHALOGRAM

The Rhythms Present:

A. Alpha Waves.

The most prominent normal rhythm from the adult human cortex is a regular series of rather smooth waves of about ten-per-second (8 to 12-per-second)(39). Various ranges of frequency have been cited: 6 to 13-per-second(26); 8 to 13-per-second(40); 8 to 13-per-second with a mean of ten-per-second(51).

The amplitude of the normal alpha rhythm is usually between 20 and 60 microvolts (5 to 100 micro-volts)(39). Ranges given include: 5-100 microvolts with a mean of 25 by bipolar methods to 40-50 micro-volts with a mean of 25 by bipolar methods for mono-polar leads(39). Parieto-occipital areas give rise to higher amplitude and more continuous alpha rhythms than the other regions.

B. Beta Waves.

A lower amplitude rhythm at frequencies ranging between 18 to 32-per-second with an average of 25(39). are designated as the beta rhythm. Ranges of frequency and amplitude are fairly generally accepted but there is more confusion than in the case of the alpha rhythm. The ranges given include: 14 to 50-per-

second(26); 17 to 30-per-second with an amplitude of
10-12 microvolts(40).

C. Gamma Waves.

A more rapid frequency of 35 to 55-per-second(39)
has been recorded from the anterior head regions.
These waves are of very low amplitude, are often not
seen at all, and their functional significance is
unknown.

D. Delta Waves.

All waves slower than alpha waves are called
delta waves. Gibbs and Gibbs(26) define the delta
"band" to be from 1 to 5-per-second. There is some
evidence of activity slower than the alpha rhythm in
some normal waking subjects but it is not present in
over 10-15% of the record and is of very low amplitude,
usually being masked by alpha and beta waves(39).
General Consideration of Amplitude, Frequency and
Wave Form:

It has been emphasized that it would be better
just to state the frequency of a wave, its amplitude
and wave form(26) rather than designating it as alpha,
beta, etc. By stressing the study of cortical fre-
quency as a continuum(24) Gibbs reiterates the concept
that the expenditure of energy in the normal cerebral
cortex is indicated by the reciprocal relationship of

frequency and amplitude. The general level of over-all cerebral activity is stated to be 20-75 microvolts(16) and a study of wave patterns in EEGS shows a wide variation to be consistent with normality.

A useful criterion of study has been advanced by Davis and Davis(16) where they consider the percentage of time (inna 100 centimeter strip of record) occupied by alpha waves. Records are classified as being rare alpha when they contain less than 25% alpha, mixed alpha when they consist of 25-50% alpha, subdominant alpha when they are 50-75% alpha, and dominant alpha when they contain over 75% alpha. The amount of alpha rhythm in a given period of time (index of alpha) varies in normal individuals. Gibbs and Gibbs state(26) that of their records, 20% are dominant alpha, 35% subdominant, 20% mixed alpha, and 20% rare alpha. Jasper(39) cites an alpha index of between 10 and 95% for the occipital region in the normal individual.

Variations in Records Taken from Different Cortical Areas:

Berger held that the alpha waves originated from all cortical areas(9) whereas Adrian and Yamagiwa(3) concluded that they arose only from area 19 of the occipital lobes. Most evidence substantiates Berger's view. Gibbs and Gibbs(26) state that the alpha may be

equal in all areas or more prominent in parietal areas but only rarely seen only in the frontal areas and then perhaps of a pathological nature. It is now felt that the alpha rhythm arises from all cortical areas but predominantly in the parieto-occipital regions(39) (41)(51). Jasper and Andrews(40) showed the occipital and precentral potentials to be of different origins as evidenced by their different reactions to stimuli. The occipital alpha potentials may be depressed by light or even show localized "on" effects after visual stimulation whereas the precentral alpha is more readily affected in the same manner by auditory stimuli.

The beta potentials appear with greatest regularity and amplitude over the regions of the central fissure(41) or precentral and frontal areas(51)(40)(39)(26). They are independent of occipital alpha potentials in frequency, regularity, and response to afferent stimuli. They are most depressed by tactual stimulation and are usually not affected by light as are the occipital alpha rhythms(40). They may show localized "on" and "off" effects after reflex tactual stimulation(40).
Ontogenesis:

It is generally accepted that there is an exponential rise in the frequency(39) of the normal electroencephalogram from birth until the characteristics

of the normal adult record are obtained. That this is
a negatively accelerated curve of increasing frequency
has been amply confirmed by Henry(36).

The electroencephalogram of the normal newborn
infant shows irregular slow waves with a frequency of
1/2 to 2-per-second and a voltage of 20-50 microvolts(26).
There are superimposed fast waves of 20-50 per-second
with an amplitude of 5 microvolts. Smith(65) has re-
ported finding slow waves emanating for the first few
days of life and believes them to be rhythmic and not
alpha waves. At birth, the parietal areas show more
activity but the occipital lead develops more rapidly
and the others appear to lag behind it(26). Rhythmic
activity is present only from the central areas at
birth(39). During the first 2-1/2 months there are no
steady frequencies present and the record shows only
random waves of the indefinite character of "base-line"
sway(65). No rhythmic activity is seen over the
occipital cortex before the third or fourth month(65)
(39). Although short bursts of 3 to 4-per-second waves
may begin to appear from the occipital areas during
the 10th to 12th weeks(65), they are not well estab-
lished until the fourth month(50)(39) when their
voltage is approximately 37 microvolts.

At eight months, waves of 4 to 5-per-second are

prominent(26) and at one year bursts of 4 to 8-per-second are seen in the occipital areas but not elsewhere(26). Lindsley reports the average frequency at one year to be 6.3-per-second and the voltage to be 52 microvolts(50). Jasper(39) gives the values of 6 to 7-per-second for the occipital alpha at one year. The predominant activity elsewhere is still slower than 4-per-second(26). The voltage remains at the stated level for two years and then falls sharply to 30 microvolts at four years. This is probably due to a cause extrinsic to the brain. Closing of the anterior fontanelles and thickening of the skull and scalp may account for this(50). The frequency at four years is 7 to 8-per-second for the occipital areas(26).

The frequency gradually reaches 9-per-second by eight years(65) and ten-per-second from the occipital area by the ninth year(26). The activity of the parietal areas is still much slower, however(26). Jasper(39) does not hold all of these views and gives a somewhat different developmental pattern of central and precentral alpha. He states that at birth, frequencies between 1 and 20-per-second are seen under different conditions. Seven to 8-per-second waves are seen and persist until the age of one year when they increase to reach the adult frequency of 8 to 10-per-second

between 2 and 4 years of age. Three to 6 and 1 to 3-
per-second rhythms are seen early from these regions
but seem to decline in prominence as development
proceeds.

From 12-15 years there are common 4 to 7-per-second
waves in the parietal areas and ages 14-17 often show
5 to 7-per-second waves in the frontal areas. The
low voltage, fast-activity of the occipital areas
(rare alpha of Davis) is seldom seen before the 14th
year.

The adult level of 10.2-per-second is reached at
8-12 years(39). There is a slow decline of voltage
to reach an adult level of 13 microvolts at 15 years(50).
Lindsley has also reported a short period of accel-
eration beyond the adult level during the 10th to 12th
years but Smith(65) failed to confirm these results.

Henry(36) reports that in his large series of
cases, the records "stabilize" by age 13 at a frequency
of 9 to 11 waves-per-second and the 10-per-second
adult norm is reached first by the 13 year-olds,
68% of 293 cases of this age or older being distrib-
uted within plus or minus 0.75 cycles of the 10-per-
second point. His data pertaining to alpha frequency
showed that females tend to have faster alpha fre-
quencies than males and that the low-voltage, fast-

activity type of record is more common in females.

No consistent changes accompany the presumed onset of puberty(36) and at 19 years of age, all normal persons show adult types of records in all areas(26).

The faster waves superimposed on the slow waves of infants' recordings persist and increase in amplitude and frequency from infancy until the adult level is established(26)'in much the same manner that the alpha waves change. These waves become those of beta frequency.

The influence of age on the resting EEG beyond the 20 year level is to cause acceleration with increasing age up to the age of 60 after which there is some slowing(23).

That changes in frequency and amplitude of the alpha do not vary as a direct function of age and are not directly related has been pointed out by Lindsley(50) and Henry(36). The latter has made extensive attempts to correlate alpha frequency and I.Q. and alpha frequency and skeletal maturity but did not support his expectation that there might be significant relationships between these variables.

In general, waves of the delta frequency band tend to become less prominent with aging. In studies between

percent-time-delta and delta frequency, Henry(36) has
shown that all children exhibit slow activity in their
resting records but that the central areas show a con-
sistently higher mean percent-time-delta value than
the occipital areas and show an equally consistent
slower mean delta frequency. With increasing age,
there is a tendency for the percent-time-delta to
decrease in both areas and for the delta frequency to
increase in both areas. Thus there is an inverse
relationship between percent-time-delta and delta
frequency and an inverse relationship between alpha
frequency and percent-time-delta.

That there may be considerable variation in the
form of individual growth curves is apparent from the
work of most men. Many of these curves are charac-
terized by irregular shifts in alpha frequency from
year to year. The energy peaks have been shown to be
highest in those frequency bands of waves with the
largest percent-time as shown by Fourier transform
studies(45).

Heredity and the Electroencephalogram:

The individuality of the electroencephalogram has
been discussed and it was noted that one factor in
support of a genetic influence upon an individual's
record was the close similarity between the records

of identical twins. The Davis's noted(16) this similarity in the records of identical twins and concluded that this probably reveals some inborn feature of cortical activity which is hereditarily transmitted. Suffice to say for the present that this view has been quite generally accepted and frequently confirmed(26)(53)(51)(46)(48).

Psycho-Physiological Variations in the Normal Human
    Electroencephalogram:

    A. Personality and the Electroencephalogram.

    Saul, Davis and Davis(64) report a positive correlation between the amount of alpha activity and the "passivity" of the individual. They believe that there is some correlation between a low alpha index and activity and a correlation between a high alpha index and passivity or inhibition. These trends were measured from the behavior as revealed by actions and attitudes toward others, food, work, sleep, and sex. Davis and Davis(16) had previously mentioned that psychic changes seem to abolish the occipital alpha rhythms in some tracings.

    That there is a rough correlation is generally accepted but it is also generally believed that the correlation is so coarse that it does not easily make the relationships between the personality and the EEG

apparent(26) and little has been accomplished in any such attempts at correlation(51).

B. Emotion.

Many workers have found the alpha waves to be depressed(51)(16) and delta waves to be increased in frequency with emotional stimulation(41). Here again, however, such correlations are so coarse as to be of little or no value.

C. Intelligence.

Many attempts have been made to correlate EEG findings with intelligence. The general attitude toward this view is that there are no relationships(48) (59)between these variables. The relationships that have been found are of limited value. In Mongolian and hereditary types of idiocy there is a tendency for frequency, amplitude and amount of alpha to increase with increasing mental age. This is most marked at the MA's of 6 to 8 years. These changes are not striking and marked differences at different MAs have been noted.

D. Attention.

Attention tends to abolish ten-per-second activity and flattens the record in all areas. This is striking-ly true when the eyes are opened to light after having been shut. However, when the attention fails (e.g./ when the eyes have been open a few minutes) the waves tend

to reappear unless the subject is looking at something
which holds his interest. Any type of visual, auditory
or tactile sensation, if it attracts the subjects'
attention, will tend to flatten the amplitude and make
the alpha waves disappear and often increase the fre-
quency(26). Retinal stimulation is most efficient in
suppressing or abolishing occipital alpha(26)(39)
whereas tactile, cutaneous, pain, gustatory, auditory,
and other stimuli block the alpha rhythm at least in
their respective cortical areas if not in the occipital
area also(51)(40).

E. Sleep.

The changes occuring in the EEG during sleep are
essentially opposite of those occuring with attention.

In the first stage when the individual feels as
though he were drifting, the ten-per-second activity
diminishes in amplitude provoding he had ten-per-second
activity in his waking record. If the waking record is
of the rare alpha(15) or low-voltage, fast-activity
type, more ten-per-second activity appears as the
subject "drifts". In all types of records, the am-
plitude diminishes.

With deeper sleep many "spindles" or spontaneous
bursts of 14 to 16-per-second waves appear in runs of
one to one and one-half seconds(52). The amplitude of

waves with a frequency of over 15-per-second decreases.
Three to 4-per-second waves begin to appear and always
before the alpha waves leave the record(10).

As sleep deepens, the 15-per-second waves (beta)
drop out and the alpha waves also are not seen. The
record then shows only high voltage slow waves with
frequencies of 1/2 to 3-per-second (delta waves)(26).

In general, the deeper the sleep, the slower the
waves. However, sleep is not a steady state and is
characterized by sudden fluctuations from moment to
moment(26). As the gradual hour to hour trends occur,
the minute to minute changes are superimposed.

Stimuli during sleep cause a shift from slower
type records to faster types but this shift may occur
without obvious stimulation(26). Loomis, Harvery and
Hobart have described(52) a brief arousal reaction or
"K" complex following an abrupt stimulus. These consist
of a train of slow waves with 9 to 10-per-second waves
superimposed on them. The complex may last from 5 to
8 seconds and then dies out.

Blake, Gerard and Kleitman(10) have described a
type of record for the period of diminished sleep during
the late part of the night. It follows the state of
deep sleep where the record is composed of delta waves
and occasional "spindles". It is characterized by

low voltage, slow activity resembling an earlier sleep
record where the delta waves appear before alpha waves
leave. In this record of diminishing sleep or "null"
stage, however, the delta waves leave before the alpha
waves return.

The sequence of events in the two commonly used
terminologies is given in Table 1.
Changes in the Blood Level of Certain Constituents and
    The Effects of Such Changes on the Electroencephalogram:
    A. Oxygen Lack.

With a diminution of oxygen intake, the electrical
activity suddenly slows and then ceases. This is true
even if the supply be cut off gradually(26). The nor-
mal blood oxygen level is 60% and the changes mentioned
occur at 30%. The causes of such a condition could
include carbon monoxide poisoning, sudden heart failure,
drowning, suffocation, and hemorrhage. The initial
lack may be heralded in the EEG by a few fast waves
or seizure-like discharges(26). When the oxygen supply
is replenished, the record assumes a normal tracing.
The changes observed, however, are not of measurable
significance until unconciousness sets in(51).

    B. Low Carbon Dioxide.

With hyperventilation, the carbon dioxide level
of the blood becomes lowered when that gas is "blown-

Table 1.   POTENTIALS IN STAGES OF SLEEP

	Amt. Alpha	Amt. Delta	Amt. 14 per sec.	Depth of sleep	Present Nomen-clature	Nomenclature of Blake et al.
1	+ +	—	—	Awake	Alpha	A. Interrupted α
2	+	+ +	+	Light sleep	Alpha + delta (+ 14 per sec.)	B. Low voltage C. Spindles
3	—	+ + +	+	Deep sleep	Delta (+ 14 per sec.)	D. Spindles-random E. Random
4	—	—	+	Light sleep	Null (or low voltage)	B. Low voltage
5	(+)	—	(+)	Sleep to wake	Intermittent alpha	B. to A. Low voltage to interrupted alpha
6	+	—	—	Awake	Alpha (low intensity)	

From Blake, Gerard, and Kleit-
man (10)

SCHEMA OF POTENTIALS DURING A NIGHT'S SLEEP
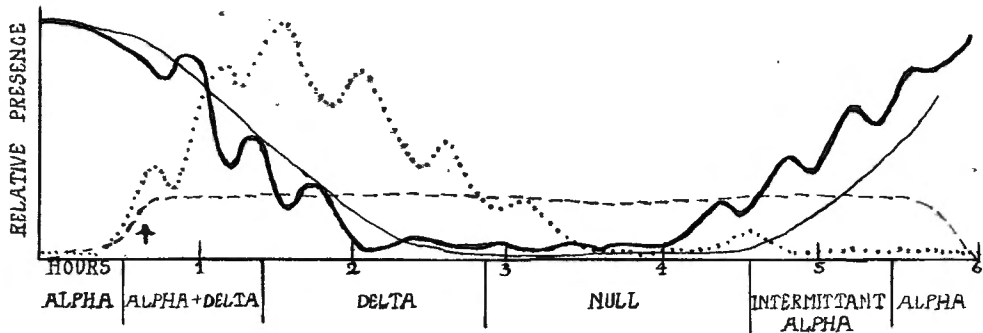
Fig. 2. Predominance of brain potentials through the night. Alpha waves (black heavy line) in per cent presence; 14 per sec. waves (dashes) in per cent presence and delta waves (dots) in extent of predominance. Oral temperature (black thin line). Below the stages of sleep are indicated. Record begun at time of retiring arrow indicates beginning of sleep. Wavy lines represent changes in waves due to shift of state of sleep. Depth of sleep by auditory response method roughly parallels delta curve.

From Blake, Gerard and Kleitman (10)

off". When the tension of carbon dioxide in the
cerebral blood does fall, slow waves occur more fre-
quently and the amplitude of the record increases(47)
(26). The ten-per-second waves tend to disappear and
are replaced by 2 to 4-per-second waves(26). This
change does not usually take place in less than a
three minute period of overbreathing in a normal
subject(51). It has been shown that the brain is more
sensitive to changes in the carbon dioxide tension in
the cerebral blood than in the tension in arterial
blood(26) and this is believed to be due to constric-
tion of cerebral vessels on hyperventilation which
tends to preserve the carbon dioxide level.

Low atmospheric pressures are subjected on aviators
and mountaineers and such records might normally be
encountered in them under the proper circumstances.
That the appearance of delta waves with hyperventilation
is related to age in normal subjects has been pointed
out by Gilman(31). The amount of delta decreases
rapidly up to 15 to 18 years of age, more slowly up to
35 years, and then shows few changes beyond this age.

C. High Carbon Dioxide.

Apnea or the breathing of air-carbon dioxide
mixtures high in carbon dioxide, causes an increase in
the frequencies of potentials. This has been said(26)

to be due to dilatation of the blood vessels with increased carbon dioxide tension. Such findings might be expected in respiratory disorders and in cases where the cortical blood flow is impeded.

D. Blood Sugar.

It is quite generally known that glucose is extremely important to the metabolism of nervous tissue. Lowering of the blood sugar levels causes cerebral activity to be greatly slowed(26)(51). No changes are apparent, however, until the critical level of 35 milligrams per-cent(26) is reached. The waves occuring are then of a frequency of 2 to 4-per-second and animal experiments have shown that all activity ceases if this level is held for very long(26). It has therefore been recommended(31) that for comparative or diagnostic studies, the blood sugar level be kept above 120 milligrams per-cent.

Hyperglycemia causes the opposite effect upon the EEG and tends to cause an upward shift in the cortical frequency spectrum(51).

E. Acid-Base Balance.

Alkalotic tendencies cause a slowing of cortical frequencies and acidotic tendencies (associated with dehydration) cause an acceleration in cortical frequencies(51).

F. Increase of Temperature or of Basal-Metabolic-Rate.

An increase in temperature or in the BMR has been shown to cause an increase in the frequency of the alpha waves in normal adults(26)(51).

THE ELECTROENCEPHALOGRAM IN EPILEPSY

I. Electroencephalographic Seizure Patterns in Epilepsy:

A. Criteria of Abnormality.

There have been several attempts made in setting up tentative criteria of abnormality in the EEG for adult subjects under standard waking conditions. A summary of the more important points that have been advanced should include the following:

1. Mean frequency and mean duration of regular sequences.

(a) Slow dysrhythmias or bradyrhythmias are composed of waves which regular, occur at frequencies of less than 7 to 8-per-second. These waves are considered to be abnormal(39) and are related to a number of conditions which tend to depress normal cortical function(39)(26)(7).

(b) Rapid dysrhythmias (tachyrhythmias) are usually related to states of abnormal cortical

excitation. When regular, the waves of this group occur at frequencies of 12 to 40 or 50-per-second.

(c) Combined forms of bradyrhythmia and tachyrhythmia are considered to be abnormal(26).

(d) Dysrhythmias may be either slow or rapid as they vary below or above the normal. They may be either regular or random depending upon the regularity of rhythmic repitition. Lastly, they may be continuous or paroxysmal in their mode of appearance dpending upon whether they represent a chronic state of the cortex or a recurring transient alteration in its condition(39).

(e) Regularity of period is a criterion of normality. Potentials of the alpha wave frequency or below which vary in period more than 30 milliseconds are abnormal(38).

(f) Too great a variability of frequency in regular sequences is abnormal. Frequencies of major potential rhythms varying from the mean by plus or minus one cycle-per-second are abnormal(38).

2. Waves having a duration of over 125 milliseconds are considered to be abnormal(38).

3. An amplitude exceeding 125 microvolts is abnormal(39)(42). Jasper(38) has termed these high

voltage waves as hypersynchrony regardless of form or frequency. He has also pointed out that hypersynchrony may occur without dysrhythmia (i.e./ in the normal range of frequencies).

4. Gibbs, Gibbs and Lennox have pointed out repeatedly that the configuration of waves in the EEGS of epileptics is of great importance. Jasper and Kershman(42), on the other hand, believe that wave forms and patterns are of less importance and stress localization of abnormal discharges as being a more valuable method of studying the EEGS of epileptics. These views will be considered in detail at another point in this paper.

5. Disorganization of bilaterally homologous rhythms is evidence of abnormality. Jasper(38) pointed out the importance of this point. It has been emphasized more recently(7).

6. Disorganization of simultaneous alpha frequencies in unilateral regions suggests malfunction of cortical neuronal discharges. Differences of more than 10-20% in the average alpha frequency of a single cortical region is stated to be abnormal(38).

II. Discussion of Theoretical Causes of Epilepsy as Advanced by Electroencephalographic Study:

The electroencephalogram in epilepsy has received

varying, though not wholly opposed, theoretical inter-
pretations. Gibbs, Gibbs and Lennox have related
epileptic discharges to general metabolic disorders
(involving oxygen, carbon dioxide, sugar, acid-base
balance and others) affecting the rate regulators of
neuronal discharge, a "dysrhythmia"(27)(29). They
have shown that epileptic patients show a lack of
competent control of cerebral rhythms antecedent to
gross abnormalities of rhythm(29). Increased carbon
dioxide tension and increased blood sugar were found to
suppress or prevent seizures. Lowering of the carbon
dioxide or sugar level of the blood, however, often
caused a post-inhibitory rebound or "over-shooting"
in the number of abnormal discharges and seizures
occuring(27). Hoagland supports(35) this work and
refers to the regulatory activity of these bodily moieties
as chemical pacemakers. In this theory, it is this
biochemical disorder and abnormal frequencies of dis-
charge of the brain as a whole which determines the
seizure patterns in epilepsy. They have held that this
underlying "paroxysmal cerebral dysrhythmia" is the
essential feature of epilepsy and that the disease,
clinically, is a condition characterized by defective
rate at which energy is released by the central nervous
system(26). This defective rate leads to the occasional

accumulation of an excessive quantity of energy which
is discharged in seizures (which serve as a discharging
mechanism) when the energy accumulating exceeds a
certain threshold value(60).

In hyperventilation tests for petit mal, Gibbs
and his associates maintain that it is the inability
of the body to adjust rapidly to shifts in the acid-
base balance which causes excessive electrocortical
discharge(25).

Jasper(37) has approached the problem of epilepsy
in a somewhat different manner. He maintains that
the rate of neuronal discharge is of little importance
but that the fundamental disorder is related to factors
which produce excessive facilitation with synchronous
discharge of large masses of neurons into unified mass
discharge or "hypersynchreny." Adrian and Morruzzi(1)
have supported this theory as does Echlin(19). The
different forms of clinical seizures are explained
upon the basis of the physiology of the local brain
area primarily involved and upon its functional relation-
ships. That these changes need not be associated with
dysrhythmia has been mentioned previously. In the
formation of the slow waves (random or rhythmic in the
EEGS of epileptic subjects) it is suggested(19) that,
although the periods of activity are more or less syn-

chronous (are simultaneous--show hypersynchrony), the
resulting composite impulse discharges which presumably
contribute to the slow pattern changes are asynchronous
and often of high frequency. In the case of fast waves
(at least when rhythmic), on the other hand, the periods
of activity of the neurons, as well as the impulse
discharges, may be synchronous.

III. Classification of Seizure Discharge Patterns and
    the Value of Such Classification in the Study of
    Epilepsy:

A. The Boston Classification. These views largely
reflect the work done by Gibbs, Gibbs and Lennox(26)
(29).

Gibbs, Gibbs and Lennox have attempted to classify
their observations during convulsive seizures into
four types. This was done because of the importance
of clinically distinguishing the various types of
epilepsy and because there are obvious correlations
between the special types of seizure discharges and
the clinical classification(26). For this reason,
the clinical terms, such as grand mal, petit mal and
the term psychomotor have been employed to describe
seizure patterns.

1. Petit mal pattern. A three-per-second slow
wave and fast spike characterize petit mal according to

these workers. The spike is negative with regard to the ear and occurs during the positive phase of the slow sinusoidal-type wave(26). This wave form has been said to be the only pathognomonic wave form of epilepsy(7)(29) but other workers do not agree upon this point(39).

The discharge may come from all leads, from certain areas only or may be confined to one circumscribed spot(26). Often the focus of discharge shifts from one cortical area to another. These discharges are often accompanied by the clinical seizure of petit mal epilepsy (transient aprosexia with the only muscular movement being a slight rhythmic three-per-second twitching of the facial muscles.)

In the three-per-second type discharges, over-ventilation can precipitate the disorder and carbon dioxide (4 to 10%) prevents, diminishes or shortens them. Over-breathing will not produce the pattern in normal individuals nor will it cause the appearance of delta waves within a three minute period whereas it does in those predisposed to the abnormality and usually in a one minute period(51). Also, decreased blood sugar precipitates and raised blood sugar prevents, discharges of the three-per-second type(27)(47). It has also been pointed out(26)(66) that mental activity

has an inhibiting influence upon epileptic attacks of
the petit mal type and also upon the abnormal electro-
cortical discharges. These finding have caused the
therapy of this type of epilepsy to change quite rad-
ically and patients are now urged to pursue an occu-
pation which they really like and which captures their
attention.

The frontal lobe is said to be more intimately
connected with the source of pathological electrical
activity associated with petit mal than any other
cortical area accessible from the scalp(30). The type
of pathological activity also tends to be somewhat
characteristic for the particular cortical area which
produces it.

2. Petit mal variant pattern. This type of
discharge has been described by Gibbs and associates(26)
but has not received general support or recognition.
The discharges are described as being two-per-second
waves and spikes, the spikes having a duration of
longer than one-fifteenth of a second. It is stated
that this type of discharge is seen in chronic insti-
tutionalized cases and in cases where there was evi-
dence of gross injury to the brain before the onset
of seizures. This type tends to show constant foci
of activity and is rarely accompanied by visible clin-

ical manifestations. The short tonic seizures many
patients have are not associated with this pattern
even though it may be present. They are usually
associated with very short grand mal discharges.
Petit mal discharges of this variant type are not
affected by changes in the blood sugar or carbon
dioxide content of the blood(47).

As with the petit mal discharges, petit mal variant
discharges may be interrupted or suppressed by such
factors as visual or auditory stimulation, attention,
activity or embarrassment(26).

3. Grand mal pattern. The discharges proper
are waves of 15 to 40-per-second. The frequency is
high at the onset and the amplitude may decrease causing
the record to resemble the decrease of amplitude occur-
ing with attention. After this initial decrease in
amplitude, the fast waves increase in amplitude.
During the tonic phase of the attack there is a cresc-
endo burst of fast waves. In the clonic phase these
are mixed with slow waves and the clonic jerks are
synchronous with the slow waves. As the clonic phase
ends, the slow waves decrease in amplitude and occur
1/2 to 1-per-second. At the end of the seizure, the
stuporous phase sets in. The records may be almost

flat but there may be slow 1 to 3-per-second waves present. As the stupor begins to wear off, the cortex gradually begins to recover. One to 3-per-second waves of high amplitude occur and are succeeded by waves of increasing frequency until supplanted by the type of waves that the patient shows in his inter-seizure record.

There may be an increase in fast waves long before the seizure sets in, sometimes days before(25) but sometimes this is not true. Often, when there is an early increase in fast activity, the different cortical areas don't show the same increase simul-taneously.

Many variations are possible and petit mal or psychomotor discharges may be admixed in the record.

4. Tonic stupor records. Tonic stupor is a partial grand mal characterized by aprosexia with slight generalized tonus. The patient is often cata-leptic and can be "moulded" like a catatonic schizo-phrenic. The electroencephalogram shows accompanying fast high-amplitude activity.

5. Psychomotor seizures. Gibbs, Gibbs and Lennox(29) have described what they believe to be a rather characteristic wave form for psychomotor seiz-ures. Psychomotor seizures are manifested clinically

as amnesic states, somnambulisms, fugues, temper-
tantrums or psychic equivalents. The full-blown seizure
is characterized by irrational and apparently purpose-
ful movements, impaired conciousness and some evidence
of emotional disturbance.

The waves are flat-topped or saw-toothed waves
formed by a series of widely-spaced, spike-like com-
ponents, usually positive to the ear lead with mono-
polar leads. The frequency is 2 to 4-per-second and
often 14-per-second waves are superimposed. The dis-
charge may last 5 to 6 seconds although it is often
shorter or even absent. It is followed by a discharge
of high-voltage, 6-per-second waves of a sinusoidal
form which are interrupted with the rectangular slower
waves.

6. Mixed seizures. Most seizure discharge
records show elements of two or three or all types of
abnormal discharges. Indeed, the petit mal record is
thought to show the components of stupor and grand mal
in that it represents a combination of the extremes of
the "cortical frequency spectrum" as described by Gibbs.
The slow waves represent the stupor type discharge,
the spikes represent the grand mal component(26).

7. Concept of subclinical and larval discharges.
Because many epileptic patients show the prescence of

abnormal discharges in their EEGS without accompanying clinical seizures, the belief has arisen that they represent asymptomatic seizures. When the discharge is of extremely short duration it is called a larval seizure. Longer periods of discharge are referred to as subclinical seizures. They have been said(26) to be quite common in persons with petit mal, less so in those with psychomotor and rare in those with grand mal seizures.

B. Discussion of the Boston Classification.

1. Relation of seizure patterns to cortical areas. The proponents of this classification believe that any formation of waves may be seen as a focal discharge in any cortical area. The petit mal type and grand mal type are seen more often in the frontal and pre-central areas. Single and sometimes multiple diphasic spikes of 15 to 25-per-second with an amplitude of 75-300 microvolts may appear in interseizure records of one type of grand mal discharge. These are often focal and are rare. Focal seizure discharges of the petit mal variant and psychomotor types are seen most often in the occipital and post-central areas.

2. Factors affecting the spread of seizure discharges. There are many factors which affect the spread of discharges. These include the afferent connections

of neurons, resistance to transmission of impulses (including fibers and synapses), and amplitude and duration of the discharges.

3. Present degree of acceptance of this classification. The evaluation of such a classification must resolve itself into an attempt to answer two questions: (1) are the wave forms described specific enough to allow one to make an electroencephalographic diagnosis between actual clinical convulsive seizures of epilepsy and other non-epileptic conditions, and (2) are they specific enough to allow differentiation between the several types of convulsive seizures said to be epileptic and idiopathic?

The first topic in this evaluation will be considered at a later point in this paper.

The specificity of the patterns in the different types of clinical convulsive seizures has been seriously questioned. It is well to reiterate that the Gibbs-Lennox theory of epileptic disorders entails the concept of a (biochemical) disorder of the brain as a whole. Their classification is based upon records obtained during clinical seizures whereas the routine diagnosis is attempted on records taken between the clinical seizures. It is consequently believed(39) that the types they describe, which occur during clin-

ical seizures, may not always be identified with
the form of onset. The second contention(39) is that
the clinical seizure cannot accurately be predicted in
most cases from the EEG observation of the three
main patterns of electrical activity bearing these same
names(39)(70)(55)(30)(16)(19).

That these relationships hold frequently, but not
always, has been pointed out(55), however, trends as
groups are said to be evident(29). Jasper and Kershman(42)
showed that the wave and spike pattern was found in 77
patients of whom 66 had major convulsive seizures from
clinical observation. There were only 14 per cent
whose attacks were confined to the petit mal form.
Petit mal clinical attacks, either alone or combined
with major seizures, were observed in only 44% of the
cases, so that about half of these patients with the
wave and spike electroencephalogram did not have petit
mal attacks, as judged from clinical examination and
history. Similar results were obtained in the McGill
analysis of the so-called "psychomotor" EEG pattern.
Analysis of the clinical validity of the Gibbs-Lennox
grand mal category gave somewhat different results.
Patients with this type of attack did not show a charac-
teristic form of EEG. All forms of EEG abnormalities

were represented including localized, bilaterally synchronous, and diffuse. This is due to the fact that all forms of epilepsy may appear quite similar at the height of a major attack after the primary discharge has spread to beome generalized. It was concluded that only confusion arises from the attempt to use clinical terms to describe types of electroencephalographic tracings. The one refers to clinical symptomatology, while the other refers to phenomena of a different order, the electrical activity of the brain.

Gibbs and Lennox(22) have defended their classification by stating that they have observed the EEG during many fits of all kinds and that the EEG changes witnessed during a fit are constant for that type of fit. Hoefer(58) has found the Gibbs-Lennox classification to be a good working hypothesis and states that during clinical seizures he obtains the specific EEG patterns, except in grand mal where petit mal electrical discharges may sometimes be found between grand mal seizure discharges. He finds the specific pattern in 55-60% of cases and in the others he obtains mixed patterns. He advances the explanation that these cases might actually be subclinical attacks of both forms of the disease, though only the more dramatic of the two

is detected clinically. However, he does think that
the brain wave patterns might not be as specific as
was once thought.

Jasper and Kershman(42) did not find the use of
this classification, in routine examination, to be
completely successful in that many patients show mixed
types of discharges. This type of paroxysmal outburst
as described(26) simply indicates the prescence of
epileptic activity and presumably warrants the diagnosis
of epilepsy in a patient in whom there occur unexplained
periods of unconciousness(70).

Although these observations do not necessarily
affect the validity of Gibbs' conclusions, it raises
the whole question of the status of the EEG in the
diagnosis of epilepsy(70).

C. The McGill Classification of Electroencephale-
    graphic Seizure Patterns in Epilepsy as Based
    on Localization Studies Made by Jasper and
    Kershman(42).

The uncertainty of many workers concerning the
Gibbs-Lennox classification has necessitated a somewhat
different approach for clinical study. As has been
pointed out, Jasper(37) believes the underlying dis-
order of epilepsy to be a summation of synchronous
discharges of large numbers of neurons producing hyper-

synchrony. According to him, this facilitation arises in some type of constant focus and the discharge spreads throughout the cortex before a seizure occurs. Echlin(19) reports observations in keeping with the hypothesis that the disturbance in most patients with epilepsy (65%) may occur and originate as a relatively local or focal disturbance and, under certain circumstances, spread to other regions of the brain. This belief led to the classification about to be discussed in which the epilepsies are described entirely upon the electro-encephalographic observations according to localization and form of paroxysmal discharge between seizures. Reference to other forms of clinical data and wave forms and patterns play a secondary role in the detailed analysis of each type of discharge.

1. The division of the dysrhythmias as to the nature of their discharge. The division of dysrhythmias according to the McGill workers(39) can be made as follows:

    (a) Regular (rhythmic) discharges which may be either

        (1) Continuous, or

        (2) Paroxysmal, and

    (b) Random (arrhythmic) discharges which may be either

(1) Continuous, or

(2) Paroxysmal.

2. Wave forms and Patterns. Wave forms and patterns were important for the detailed analysis of each case. But wave patterns are not emphasized so greatly in this classification. Patterns were divided into random waves and paroxysmal rhythms. Three types of frequently seen random waves are spikes, sharp waves and delta waves. The six forms of paroxysmal rhythms include 3-per-second waves and spikes, 3-per-second waves, 10-per-second waves, 14-per-second waves and 25-per-second waves.

(a) Random Waves.

(1) Spikes. These appear suddenly out of the background activity at irregular intervals. They are approximately 0.02 seconds in duration.

(2) Sharp waves. The rising phase of the sharp wave is similar in its magnitude to that of the spike wave, but the descending phase is prolonged. The duration of sharp waves is longer, and they appear to be a summation of spikes not perfectly synchronized.

(3) Slow waves. Waves having a duration of more than 0.15 seconds (usually nearly 0.3 seconds) are delta waves, if they have a smooth

rather than a sharp wave front. Delta waves of this type are paroxysmal in epileptic records.

(b) Paroxysmal rhythms. It is the rhythm at a definite frequency that is fundamental in paroxysmal rhythms. There is only a minor tendency for isolated single waves to appear. It is their paroxysmal hypersynchrony that is important. There are also certain rhythmic patterns which do not appear normally.

(1) Three-per-second wave and spike. The fundamental aspect of this rhythm is a series of regularly repeated slow waves of about three-per-second. The spikes are subordinated to the three-per-second slow wave. The amplitude of this form of paroxysmal discharge varies between 100 and 1000 microvolts, as recorded with monopolar leads. There is usually some deceleration of the rhythm toward the end of a long series of waves.

(2) Three-per-second (2 to 4-per-second) waves. Three-per-second waves might be considered a minor form of wave and spike. They are, in fact, transitional forms in which the spike component is barely detectable. Some patients with the wave and spike pattern may show only a

lower amplitude slow wave form, with medication. However, some patients with the simple slow wave rhythm, fail to show the wave and spike sequence under any circumstances.

(3) Six-per-second (5 to 7-per-second) waves. Similar in pattern to the three-per-second waves and of comparable or slightly less amplitude, this rhythm may be associated in the same patient with a three-cycle rhythm, or in other cases with random sharp wave forms.

(4) Ten-per-second (8 to 12-per-second) waves. It is difficult to consider this waveform a dysrhythmia. However, some clinical seizures begin with a paroxysmal outburst of large-amplitude, regular, ten-per-second waves. Amplitudes are of the same order of magnitude as the six-cycle waves.

(5) Fourteen-per-second (13 to 17-per-second) waves. Short bursts of rhythmic waves, with frequencies falling between 13 and 17-per-second in each burst, characterize the EEG from certain epileptic patients. Although seen in the normal sleep record ("spindles"), in certain epileptic patients this rhythm is observed while they are wide awake. Between bursts, the EEG

is usually composed of low-amplitude, disorgan-
ized, rapid waves, with occasional sharp wave
forms, sometimes a few delta waves, or even an
occasional spike.

(6) Twenty-five-per-second (18-to 32-per-
second) waves. This disorder is not a dysrhythm-
ia because the frequency is the same as the normal
beta rhythm. The amplitude is somewhat smaller
as a rule than that of the 14-per-second waves.
They may be associated with random spike dis-
charges.

In general, the decreasing pattern of waves found
with areas of epileptic discharge are as follows: spikes:
sharp waves; wave and spike pattern; delta waves.

3. Localization of discharges in epilepsy.
Jasper and Kershman found that localization in epilepsy
is perhaps more important for description or classifi-
cation of disorders of the EEG than any other method
they had tried. The three principal types of loci were:
(1) localized unilateral cortical discharges; (2) bila-
terally synchronous discharges from homologous areas,
and: (3) diffuse discharges.

Abnormality localized to a discrete area of one
hemisphere was found in about one-half the cases studied.
The principal localized forms were random spikes, random

sharp waves, random delta waves and paroxysmal rhythms
of the ten-per-second or more rapid type. The random
spike was the best indication of superficial local
cortical epileptic discharge. Seizures with focal
cortical onset and gradual march were most common in
patients with this pattern, the specific type of onset
being related to the function of the cortical area
primarily involved.

Bilaterally synchronous abnormality, observed in
35% of the cases, appeared chiefly bifrontal or bi-
temporal and occasionally bioccipital. The principal
forms were the three-per-second wave and spike pattern,
sharp waves, three-per-second waves and six-per-second
waves. The three-per-second wave and spike rhythm and
the three-per-second waves were usually bifrontal and
were often present in the same patient. Minor attacks,
usually of the petit mal form and major attacks, which
became generalized, were seen clinically. Sharp waves
usually appeared bitemporally and were present in patients
with onset of major seizures and minor attacks referable
to structures lying within or deep to the temporal
lobes. Many had viscerel auras with complex disturb-
ances of thinking or behavior (i.e./automatisms and
psychomotor attacks). Six-per-second waves appeared
either bifrontally (often associated with three-per-sec-

ond waves) or bitemporally, when associated with the sharp wave forms. Visceral auras and epileptic autom- atisms were often prominent.

Diffuse abnormality, without bilateral synchrony, was found in about 15% of the cases. Random paroxysmal waves were more common although rhythmic sequences could be developed occasionally. Three principal forms were diffuse multiple spikes, diffuse multiple sharp waves and diffuse multiple delta activity. Patients with diffuse multiple spikes and diffuse multiple sharp waves most commonly had major generalized seizures with no consistent focal cortical onset.

Jasper(39) has also attempted to show the relation- ships between the electroencephalographic and clinical classifications stressing that the clinical type of seizure depends upon localization of the discharging focus or foci.

D. Discussion of the McGill Classification.

In order to evaluate the validity of this method of classification it is necessary that three questions be answered. First, do all (or most) epileptic seizure discharges arise from epileptogenic foci and spread diffusely over the cortex with the onset of a clinical convulsive seizure, and (2) may the localization of abnormal waves be correlated precisely enough with

clinical seizure patterns to be of aid in the study
(and particularly in the diagnosis) of epilepsy, and
(3) are the wave form patterns as specific, as has
been outlined, for the proposed classification by
localization?

In the main, there have not been enough reports
concerning this method of study and an adequate eval-
uation cannot be made at this time. However, Gibbs and
Gibbs(26) believe that any formation of electrocortical
discharge patterns may be seen as a focal discharge in
any cortical area. One worker(61) has not duplicated
the results of the Montreal group as to the focal origin
of generalized seizures but has found them to shift.
Rheinberger(61) has favored the McGill point of view.

That the clinical seizure patterns are correlated
with the localization of abnormal electrical discharges
has not yet received enough attention to conclusively
state any decisions as yet. Echlin(19) has presented
a paper in which he classified 100 cases in an attempt
to correlate these findings. He concludes that guarded
predictions of the type of clinical seizure to be ex-
pected could be made from the form of the EEG (when
classified as to localization of the disturbance).
Whereas Jasper and Kershman found abnormalities localized
to a discrete are in one-half of their cases, Echlin

found that 30% of his cases had unilateral localized foci but stated that this figure was high due to the method used in selecting the cases under study. Sharp waves were the most common feature (80%), spikes being present in the records of 6.6% and slower waves in 13.3%. Bilaterally synchronous activity was found in 35% of the cases with paroxysmal rhythmic activity of three-per-second wave and spike patterns, three-per-second waves and six-per-second waves being seen. The only random waves were sharp waves obtained from the temporal lobes in some cases. He found diffuse non-localized activity in only 3% of his records whereas Jasper found a percentage of 15 for this type of abnormality. These records were characterized by a "moderate variation in the voltage and frequency of the alpha rhythm and the prescence of scattered random delta waves of low voltage. Discharges of rapid, low-amplitude sharp waves at 14 to 25-per-second were occasionally present." In 20% of his cases normal records were obtained even though they had a history of convulsive seizures.

In only a little over 65% of the patients with convulsive seizures were paroxysmal high voltage waves present in interseizure records. In the prescence of a history of petit mal, the abnormality almost always

was of the bilaterally synchronous type. In the case
of psychomotor or grand mal clinical seizures, no
predictions could be made concerning the type of EEG
from the standpoint of localization. In the majority
of patients with clinical focal seizures, a localized
cortical abnormality was found in the EEG. When
studying the electroencephalographic data, Echlin
found that when a local cortical disturbance was pres-
ent, one could expect focal clinical seizures in 70%
of the patients. However, 40% of these same patients
had psychomotor attacks, and 80% had grand mal in
addition. When bilaterally synchronous waves were the
outstanding feature, grand mal seizures, without
localizing features, were the rule (80%), and petit
mal attacks were common (34.2%). Psychomotor seizures
were also frequent (42.8%) in this group. Patients
with normal EEGS or having a slight diffuse abnormality
had seizures essentially similar to those of the pre-
vious group except that clinical attacks of the focal
cortical type were a little more frequent (14.2%).

Echlin thus concluded that the clinical mani-
festation resulting from such abnormal or excessive
electrical activity appeared to depend largely on the
function of the area of the brain involved or influenced
and also on the intensity of the discharge. He also

corroborates, in part, the findings of Jasper and
Kershman that the EEGS, taken from epileptic patients
between seizures, may be classified on the basis of
localization studies.

E. Comparative Analysis of the Gibbs-Lennex and
the Jasper-Kershman Classifications.

The relative value of these two purely descriptive
classifications and the varying theories underlying them
cannot, at this time, be definitely stated. It is
important, however, to realize that the views of
Jasper and Kershman do not conflict with the findings
of Gibbs and co-workers. The latter workers took their
records during clinical attacks whereas Jasper and his
co-workers took records between clinical attacks.
Their primary differences of opinion come in the prac-
tice of the Boston workers in describing subclinical
discharges by clinical terms since the McGill studies
show that abnormalities may occur in the EEGS of patients
who do not have attacks of the type implied. Finley
and Dynes(20) reflect the view of choice (in my opinion)
when they state that probably neither the Jasper-
Kershman nor the Gibbs-Lennex classifications are
wholly satisfactory and that "the time has not yet
arrived to make any formal classification of brain wave
patterns." This time will probably not arrive until

more is understood concerning the underlying electro-
physiology of the brain and until standardized tech-
niques are adopted(31).

IV. Psycho-Physiological Variations in the Electro-
    Encephalograms of Epileptics:

    A. Interseizure Records.

    Interseizure records have been discussed largely
in speaking of the views of Jasper and Kershman. The
findings of Gibbs, Gibbs and Lennox and their assoc-
iates have been stated to some extent in the consider-
ation of the concept of subclinical seizure discharges.
Williams(70) has studied the transient outbursts in
the records of clinical epileptic patients and states
that even minute and evanescent disturbances might
represent epileptic activity. Gibbs(24) has found that
abnormalities occur thirty times more commonly in the
interseizure records of epileptics than in normals
and that such activity should therefore suggest epilepsy
or a related disorder.

    B. Records of Deteriorated Epileptics.

    These patients commonly show slow activity composed
of 4 to 8-per-second sinusoidal waves and there is no
relationship between the slowness of the waves and the
degree of deterioration(26).

    C. Sleep Records of Epileptics.

    Most epileptics show more abnormalities during sleep

and some show abnormalities only at this time. This
is particularly true of the three-per-second wave and
spike pattern(26). Psychomotor activity is best studied
with the patient awake because it resembles normal
sleep too much. Aside from seizure discharges, the
sleep patterns of epileptic patients is like that
of normal subjects(26).

D. Correlation Between Degree of Severity of Epilepsy
   Electroencephalographic Findings.

Generally accepted, is the belief that there is
no accurate correlation between the severity of clinical
epileptic seizures and the number or type of abnormal
electrocortical discharges.

V. Specificity of Abnormal Electrocortical Discharges
   (Dysrhythmias and/or Hypersynchrony) for Epilepsy:
   A. Epileptic and Non-Epileptic Conditions.

In order to evaluate electroencephalographic find-
ings, it must be determined whether or not the type of
abnormal waves found in the epilepsies are specific
enough to differentiate these conditions from non-
epileptic conditions.

The appearance of 3 to 4-per-second waves at
random for 1-2 seconds throughout the various leads,
followed by a regular rhythm which is again broken up
by these slow waves is the so-called paroxysmal cerebral

dysrhythmia which is considered to be diagnostic of an epileptiform state(7). Gibbs(25) states that the grand mal or petit mal types of cerebral disorder rarely occur in those not suffering from clinically obvious epilepsy. The psychomotor type of discharge, on the other hand, is common in problem children, in patients with psychopathic personalities, in schizophrenics and among the near relatives of epileptics. Lindsley(51) has reaffirmed this view stating that some of the variations associated with epilepsy may also be found to some extent in certain non-epileptic conditions. The wave forms found in migraine(58) and cataplexy are not similar to those of epilepsy, however, the abnormalities associated with narcolepsy and pyknolepsy are similar to those of petit mal and psychomotor epilepsy respectively.

In a study devoted to the transient outbursts in the records of selected groups of idiopathic epileptics, patients with head injuries, neurotics, and normals(70), it has been found that even minute disturbances might represent epileptic disturbances but that fully developed larval disturbances were not found in normal non-epileptics. Other paroxysmal outbursts occured in 0.5% of non-epileptics compared with 29% of epileptics. This indicates that paroxysmal

disturbances of all kinds are over 100 times as common
in epileptics as in others and a certain degree of
specificity of these paroxysmal outbursts for epileptic
conditions are characterized by the occasional occurence
of slow waves and seizure discharges. Gibbs(26) has
pointed this out when speaking of cerebral abscess,
cortical tumors, intracranial hemorrhage, subdural
and epidural hemorrhage, increased intracranial pres-
sure and cerebral arteriosclerosis. Other conditons
showing delta wave discharges include meningitis,
brain trauma, encephalomyelitis, Schilder's disease
and multiple sclerosis. Cases of athetoid chorea
usually have normal tracings but some cases have high-
voltage fast waves. Schizophrenics may show deficien-
cies in alpha waves and a greater incidence of seizure
discharges than normal control groups(26) and some have
increased delta indices. Paresis may show abnormally
fast and abnormally slow waves. That other conditions
may show abnormal waves similar to those seen in the
epilepsies has been intimated in discussing various
types of mental deficiency, behavior disorders and
thyroid disease.

The rather commonly accepted viewpoint concerning
specificity of waves is well stated by Echlin(19)
when he says that "all abnormal forms can occur in non-

epileptics with the possible exception of the three-per-second wave and spike," remembering that abnormal paroxysmal cerebral discharges are over 30(24) to 100(70) times as common in epileptics than in non-epileptics. Also to be remembered is that 5 to 15% of clinical epileptics show no EEG changes as is to be pointed out at a later time.

B. Normal-Abnormal Records.

It has been frequently pointed out that 5% of clincal epileptics show no abnormalities electroencephalo-graphically(39). A frequent point in the differential diagnosis of epileptic seizures and hysterical seizures is that the latter show no electroencephalographic findings thus it is important to bear in mind that epilepsy need not be associated with abnormal electro-cortical discharges.

Jasper and Kershman(42) report the occurence of hypersynchrony as being 80% of their series of cases whereas Echlin(19) finds that 65% of his patients had high voltage waves in interseizure records. Gibbs(24) has reported the incidence of cerebral dysrhythmias among epileptics as being present in 90% of all cases.

The explanation of why clinically epileptic patients should show no electroencephalographic findings has not as yet been stated satisfactorily. All clinical

cases show abnormal electrocortical findings during an actual clinical seizure.

C. Abnormal-Normal Records.

Minor, or even some moderately severe abnormalities in the EEG of 5 to 15% of clinically "normal" control subjects has been repeatedly confirmed(58)(53)(48)(21) (24). Palmer(58) reports that only 5% of persons with dysrhythmias have actual convulsive epilepsy. Some of these cases are truly non-epileptic but a large percentage may be conditons related to epilepsy as suggested on the basis of electroencephalographic findings. Indeed, Gibbs(16) believes that these cases of asymptomatic dysrhythmia are cases of asymptomatic epilepsy. Jasper and Kershman(43) also believe that this may be due to (a) inherited predisposition to dysrhythmia, or (b) brain damage from which there was apparently complete clinical recovery.

VI. The Hereditary Aspect of Epilepsy as Indicated

by the Electroencephalograph:

Lennox, Gibbs and Gibbs(49) studied 74 "normal" pairs of twins and found that in the monozygotic twins 85% showed "identical" EEGS while only 5% were alike in the dizygotic twins. These findings indicate a good deal of similarity in the records of persons of the same parentage. However, it has been shown, that the record

of a normal person has certain distinctive qualities
for that individual. It is thus difficult to show any
great degree of similitude between normal records of
members of the same family. One of the main ways to
demonstrate such similarities, however, is to study
the records of families in whom one or more members
are known to have abnormal cerebral rhythms. Many
such studies have been undertaken and all show that
abnormal discharges are more common in parents and near
relatives of epileptics than in the normal population
as a whole. These findings have prompted most persons
to believe that the general liability or predisposition
to abnormal waves is an inherited factor.

The incidence of abnormalities in the records of
the families of epileptic patients varies quite widely,
but all findings are very statistically significant.
Lennox, Gibbs and Gibbs(48) found abnormal electro-
encephalograms in 60% of the relatives (parents, sib-
lings, and children) totaling 183 of 94 epileptic
patients. They found abnormalities in 10% of a control
group of 100 persons who had no near relatives with
epilepsy and this value falls within the range of the
abnormal-normal records previously discussed. Dys-
rhythmias were found to occur as often among the

relatives of patients with symptomatic epilepsy as
among the relatives of patients with "essential epilep-
sy." Lowenbach(54) obtained abnormal EEGS for 17(40%)
of 37 non-epileptic relatives of two epileptic patients.
Strauss, Rahm and Barrera(67) found abnormal EEGS in
26.9% of 93 relatives of 31 epileptic patients.
Robinson(62) found abnormal tracings in 36% of 36
relatives of 13 epileptic patients and 27% questionably
normal records in the same groupd. In an earlier
report(25), Gibbs found that when both the parents of
an epileptic patient were studied, abnormalities of the
same type as are seen in epilepsy were observed in one
or both parents in 94% of the pairs. Such abnormali-
ties occured in only 6% of the control series and in one-
half the near relatives of an epileptic. Palmer(58)
believes that the frequency of dysrhythmia among sib-
lings and parents of epileptics probably averages over
90%.

That the brain wave pattern is a hereditary trait
has further been pointed out by Gibbs(22). He con-
cluded that "although the EEG is not 100% conclusive,
it does furnish presumptive evidence that epilepsy
per se is not inherited but that cerebral dysrhythmias
are inherited and these genetic factors combined with
acquired factors can produce seizures." This inheri-
table factor is believed to be a dominant trait(48).

Lowenbach(54) does not believe that the dysrhythmias in the near relatives of epileptics are symptoms of epilepsy not yet severe enough to produce convulsions but are an expression of a non-specific functional instability of the central nervous system. The general concensus of opinion of most workers, however, favors the former view. They have found it reasonable to assume that these persons, even though they have no clinical symptoms, are the "carriers" of a hereditary dysrhythmia. Persons with dysrhythmias outnumber actual epileptic subjects 25 to 1. The incidence of epilepsy is about 0.5% hence persons with a predisposition to epilepsy form about 12% of the population according to this reasoning.

VII. The Borderlands of Epilepsy:

A. The Concept of Genetic and Acquired Factors in the Causation of Epilepsy.

The prescence of electrical disturgances of an epileptic kind in disorders which might or might not otherwise have been recognized as epileptic has made the field of epilepsy even wider than was suggested by Gowers in his book, The Borderland of Epilepsy. There can be little credulity expressed at the difficulties one might expect to encounter when trying to differentiate between the abnormal waves of true idio-
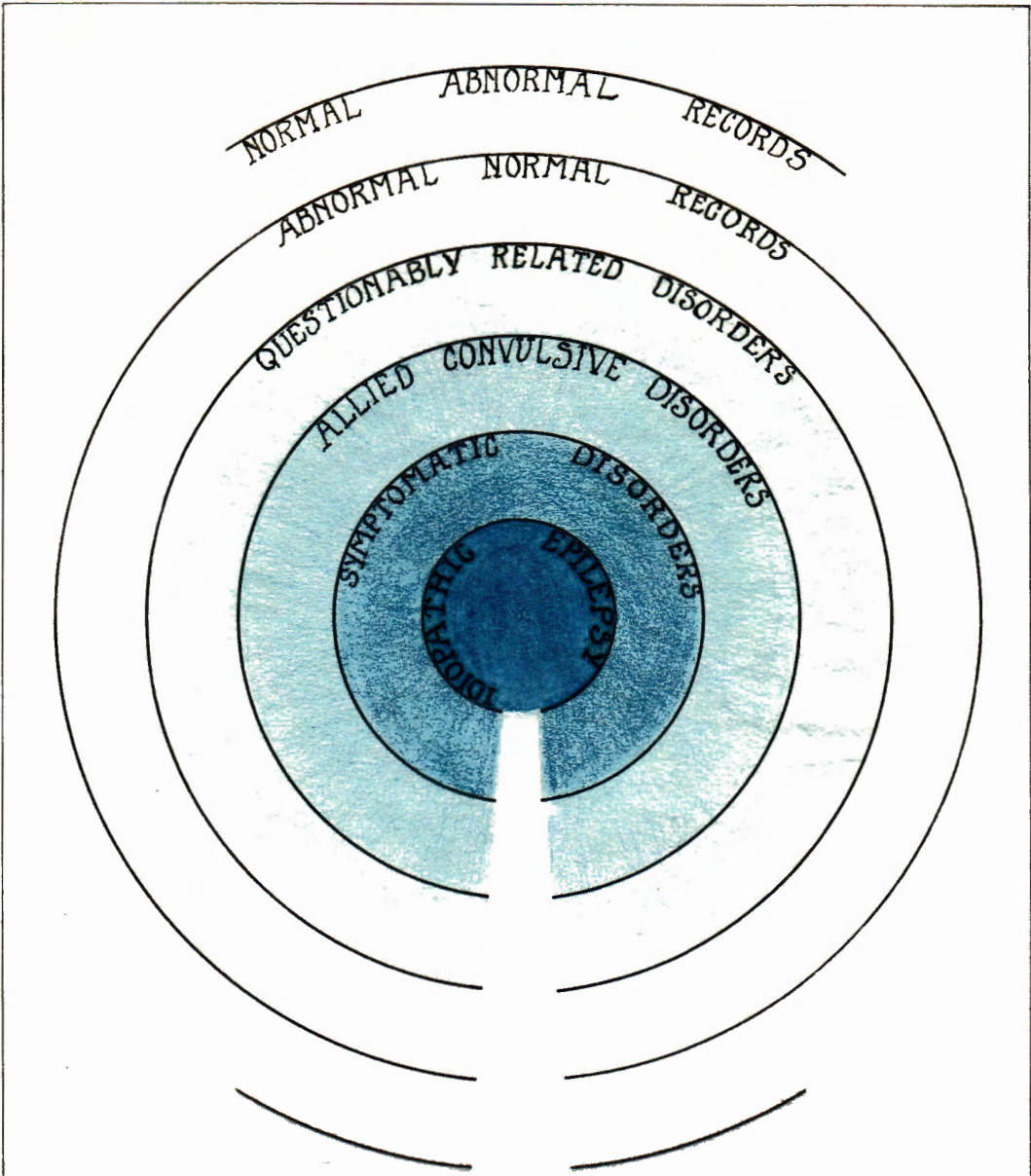
The "Borderlands of Epilepsy"
and
The Occurence of Cerebral Dys-
rhythmias and/or Hypersynchronies

pathic epilepsy and other related (closely or other-wise) conditions. This point is brought home particularly by the realization that it is exceedingly difficult to differentiate between the interseizure records of epileptics (with the possible exception of those patients showing the three-per-second wave and spike pattern) and the records of non-epileptics.

That there are many non-epileptic conditions in which the electrocortical discharges may resemble those of "true idiopathic" epilepsy has previously been discussed. The abnormal tracings found in the 10% of the normal population who have no history of convulsive seizures has also been mentioned as have the normal records found in 5% of cases with a history of clinical convulsive seizures. There are, in addition to the clinically diagnoses cases of epileptics (with or without cerebral dysrhythmia and/or hypersynchrony) three other groups of patients who must be considered in the attempt to relate electroencephalographic findings with the clinical picture. These groups are: (1) those cases with symptomatic epilepsy; (2) those suffering with convulsive seizures closely allied to idiopathic or symptomatic epilepsy but outside of the usual classification; and (3) those cases suffering from disorders which are questionably related to epilepsy.

The primary concern of this paper has been with the consideration and evaluation of electroencephale- graphic findings as correlated with the clinical find- ings in cases of so-called "true," "idiopathic," or "functional" epilepsy. "idiopathic", "functional", "essential", "true", or "cryptogenic" are all terms by which we dissemble in an attempt to express our ignor- ance. The simple term of "epilepsy, cause unknown" would probably be a more descriptive term. The clinical term "epilepsy" in this use has been employed to denote the occurences of attacks of transient aprosexia accompanied (and in some instances, unaccompanied) by convulsive seizures, sensory symptoms, visceral complaints, or psychic alterations in varying degree. The electroencephalographic term of "epilepsy" has been used in the discussion of one method of classification (Gibbs-Lennox) but has been avoided in the other (Jasper-Kershman. The characteristics of these wave types have been described considering their various aspects. It is now desirable to attempt to relate the EEG findings of the three more or less related groups previously mentioned and to attempt to derive some con- clusions that might be helpful in a better understanding of the symptom complex of epilepsy.

For this purpose, the accompanying diagram on page 78 has been constructed. The central core depicts

the group of idiopathic epileptics who show abnormal
EEG findings. Closely related is the symptomatic
epileptic group included in the first concentric ring.
Less closely related is the group of allied convulsive
disorders and, in the next outward ring, are a group
of questionably allied conditions. The outermost ring
includes those normal subjects who show abnormal
findings but have no clinical history of convulsive
seizures. It will be noted that as one travels from
the inner-most ring to the outer-most, one finds
conditions which are, at the outset, closely related
to the group of idiopathic epileptics, but as one con-
tinues outward, the conditions are less and less
related to the central core. The outer uncircled
margin representsthe group of clinical idiopathic ep-
ileptics who show no abnormal electroencephalographic
findings in their interseizure records. That this
group is, none the less, closely related to the cen-
tral group is indicated by the free passageway between
them. Conditions which can, in no way, be considered to
be epileptic or epileptiform, are excluded from the
diagram.

Symptomatic epilepsy includes those cases in which
convulsive seizures can be attributed to easily demon-
strable causes. Cerebral lesions found at autopsy

(those of post-traumatic epilepsy); fever; heart
block or hypoglycemia, all of which impair the metab-
olism of the brain; renal disease and vascular diseases;
are a few of the conditions which are implied when one
speaks of symptomatic epilepsy. Jacksonian seizures,
a subspecies of grand mal, are an often seen form of
symptomatic epilepsy.

A description of the EEG findings of these various
types of symptomatic epilepsy does not fall within
the scope of this paper. However, as has been pointed
out, many persons have cerebral dysrhythmias with an
abscence of clinical seizures. This has prompted many
workers to speak of a "latent" inherited liability or
predisposition to convulsive states as indicated by
the EEG. In some cases with such dysrhythmia, some
unknown factor causes the disorder to be so marked
that clinical attacks occur without any known causes
and an "active" epileptic state is said to exist.
This is designated clinically by the use of such terms
as "idiopathic". In others, the underlying regulatory
mechanisms of (a) rate of cerebral discharge, or
(b) facilitation of neuronal discharges is so poor that
added insult by any of the factors said to cause sympte-
matic epilepsy, causes the appearance of seizures.
Thus inherited and latent genetic factors are combined

with acquired factors to produce seizures. The conception of "latent" and "active" epilepsy, which has for long had clinical support, is advanced on the evidence which has been obtained from correlating clinical and electroencephalographic data.

The application of this concept has not only been made to both idiopathic and symptomatic forms of epilepsy, but also to certain clinically allied conditions. Although the work done along this line is still inadequate, there is strong evidence according to some(39), that myoclonia, masticatory seizures, simple adversive seizures and tonic postural seizures are other manifestations of motor epilepsy. Somatic sensory seizures believed to be epileptic include pure sensory seizures, and seizures of the auditory, vertiginous and olfactory types. It is said that visceral seizures of an epileptic nature exist. Psychical seizures of epilepsy include dreamy states, automatisms, psychotic states secondary to epileptic convulsions and the much discussed petit mal seizures. Pyknolepsy is now considered (31) to be a form of petit mal seizure. None of these conditions (with the exception of the latter) has as yet been studied adequately by the EEG. The existence of underlying cerebral dysrhythmia in most of them indicates that they may actually be forms of idiopathic

epilepsy or are closely related to it.

A questionable relationship is claimed between epilepsy and a large number of truly borderland conditions. Behavior disorders in children and adults have been said by some workers to be the manifestations of epilepsy and attempts have been made to correlate the EEG patterns in the two symptom complexes. Other such studies have been conducted in the study of anaesthetic convulsions(71), syncopal fits or hyperactive carotid sinus reflex(70), puerperal toxemia associated with convulsions(63), transient spells of mental dullness in children and other conditions. Some of these other conditions in which a faint or fit occurs only in response to a specific "trigger" mechanism include those patients who habitually faint after inoculation, on postural change, or at the sight of blood. Many of the patients suffering from the above conditions have been shown to have EEG changes which are indistinguishable from those in constitutional epileptics(70).

The group of normal persons having abnormal records has previously been discussed.

B. Comment and Discussion.

The concept has been presented wherein epilepsy is looked upon as being a much-varied symptom complex. It has been shown that cerebral dysrhythmia or predis-

position or liability to epilepsy may be inherited.
In addition, a large number of conditions more or less
related to epilepsy have been shown to have quite a
similar cerebral rhythm as a part of their measurable
manifestations. From these observable facts, many have
proposed that this genetic factor, in itself, is enough
in some persons, to cause convulsive seizures. In
others, it requires the additional acquired factors
such as emotional disturbances, bodily disorders or
brain damage in order to precipitate convulsions. In
still others, even more specific trigger mechanisms are
needed to induce seizures. In the final and largest
group, an exceedingly slight genetic factor or abscence
of sufficient acquired factors causes an abnormality of
brain waves to exist in the abscence of any clinical
symptoms.

This hypothesis has led to a rather unfortunate
widening of the term "epilepsy", and such interpretation
will widen the limits of the word so far as to make
it meaningless. It has been cautioned(70) that great
care is consequently required in translating EEG
findings into clinical terminology. Lennox, aware of
this, has suggested his phrase of "cerebral dysrhythmia"
when speaking of the undesirability of broadening the
meaning of epilepsy to include these subjects with a

latent tendency as well as those with active epilepsy.
In any final analysis, it should be remembered that
abnormal cerebral rhythms are only one of the few
measurable moieties of abnormal brain function. The
electrocortical discharges have been used only as an
indication of the activity of the cells because we are
as yet, unable to purify our methods of analysis to the
point of studying the minute chemical, thermal and
mechanical factors which are undoubtedly present. It
is therefore held(70) that the inclusion of all these
subjects under the heading of "epileptic" is undesirable,
and that with further knowledge gained by the EEG,
and by other methods of study, new insight into the
study of epilepsy will be gained which will probably
necessitate a new nomenclature.

VIII. Use of Electroencephalography in the Evaluation

of Therapy in Epilepsy.

A. Surgical Therapy.

It is believed by some that the EEG is of use in
prognosis and in following the progress of cases follow-
ing surgical therapy(51)(39). It has some value in the
selection of cases for surgery in that operable cases
are those having unilater], localized foci of discharge.
There is no assurance, however, that even these cases

will be seizure-free or show no abnormal electrocortical discharges following surgery(39).

B. Medical Therapy.

General correlations are numberous between electrical activity of the cortex and clinical manifestations but are not always present. Thus a severe clinical case may show little EEG evidence or a subject with an extremely abnormal EEG may have no clinical manifestations(26). Even so, the EEG may be used in evaluating the effectiveness of anticonvulsant therapy. Anticonvulsant drugs modify rather than abolish seizure discharges in epileptics(26)(47). Amphetamine sulphate has been found(66) to be effective in preventing attacks of petit mal both clinically and electrocortically. Phenobarbital and sodium bromide prevent or alter the pathologic activity associated with a seizure of several types of epilepsy(47)(27). Other newer therapeutic agents have also been shown to modify pathological electrocortical activity(31)(51). The EEG is particularly useful in that the technique may be used to evaluate therapy on a short-time basis without having to wait and see if a particular dosage of some drug is going to prevent seizures.

IX. An Evaluation of the Clinical use of the Electroencephalograph: Summary and Conclusions.

The principle ways in which the EEG may be used in

the clinical study of the epilepsies is in diagnosis
and in the evaluation of treatment. Other uses of
this laboratory method include the classification of
the epilepsies, use in observing subclinical seizures,
aid obtained in discovering cerebral disorders allied
to epilepsy and use in estimating the role played by
genetic factors in the causation of epilepsy.

In diagnosis, the EEG serves a most useful role.
It is most reliable when the three-per-second wave and
spike pattern is found and less valuable in the diagnosis
of grand mal and psychomotor types of disorder. As
yet, it has not been adequately applied to the diag-
nosis of related types of epileptic disorders. The
clinical diagnosis of petit mal is difficult so the
EEG performs a noteworth service when it establishes
a diagnosis in these cases. The EEG findings of grand
mal are difficult to read in interseizure records,
but here, clinical findings most often make the diag-
nosis. Because of the unspecificity of slow waves in
the EEG, its use in the diagnosis of the psychomotor
form of the disease is of questionable value. Although
slow waves and paroxysmal discharges of cerebral dys-
rhythmia and/or hypersynchrony are somewhat non-specific,
their appearance in the records of a patient suspect of
having epilepsy is considered to be significant. A

20 minute study on six leads with the eyes closed is
considered to be sufficient in obtaining a diagnostic
record. A two to five minute period of overventilation
is usually included. In any event, it is important to
remember that this method of study is a laboratory
method which has its particular indications and limi-
tations. It should be needed as a diagnostic aid in
only a minority of cases suspected of having epilepsy
and carefultclinical case-taking, including a history
and physical examination should not be omitted. When
EEG findings help confirm clinical findings it lends
credence to a diagnosis of epilepsy. Failure to con-
firm a clinical diagnosis, or the establishment of an
EEG diagnosis in the abscence of clinical findings should
probably not be made on the basis of EEG findings
alone. If such is done, extreme caution is required.

The EEG is also of limited value in therapy. In
selection of patients with local as opposed to general-
ized disorders and as a guide to the area of localiza-
tion, it is of some aid when surgery is contemplated.
The success of the EEG in preoperative diagnosis is not
always borne out in postoperative prognosis and after
operation there is no assurance that attacks and abnormal
records may not recur. In some cases, the EEG may be
used as a rough guide in determining the kind and
amount of anticonvulsant medication most effective.

The most certain guide, however, is still the number of clinical seizures and the general clinical status of the patient.

Although many attempts are made to classify the epilepsies on an electroencephalographic basis, it is felt that such attempts are premature and ill-advised until more is known regarding the normal and abnormal electro-physiology of the brain.

The use of the EEG in studying the interrelationships of the epilepsies and their possible correlations with other conditions has, unfortunately, tended to widen the meaning of the term epilepsy. It is felt, however, that this may be of great significance in setting the pattern of clinical research.

During its few years of rapid growth, this ingenious method of study has emerged from a state of pure experiment and has become a somewhat reliable clinical tool. As is often the case with new developments in any field, there is an over-enthusiastic reception accorded the newcomer. This acceptance of a new modus operandi as a panacea unfortunately too often leads to a later neglect when the method does not live up to its expectations. It is to be hoped that such excessive optimism will not lead to undue pessimism in the case of the EEG.

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