LATERAL BRAIN FUNCTION IN NORMAL AND DISORDERED EMOTION: INTERPRETING ELECTROENCEPHALOGRAPHIC EVIDENCE

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Given the developing awareness of the lateral specialization of the human brain for both cognitive and emotional processes, the recent findings of characteristic hemispheric asymmetries in psychopathological groups suggest a neuropsychological model may be explanatory for abnormal psychology. Since the activity of arousal systems of the brain is a primary issue in interpreting both biochemical abnormalities and thought disorders in psychopathology, electroencephalographic (EEG) measures of cortical arousal are relevant. A better understanding of the relation of EEG measures to normal emotional arousal and cognitive effort may facilitate interpretation of the functional significance of EEG asymmetries in psychopathology.

1. Introduction

The question for this symposium is how basic research on hemispheric asymmetries in EEG data may contribute to clinical research on electrophysiology and psychopathology. My associates and I have considered a particular perspective on this question: The study of lateral asymmetries in normal emotional arousal. An initial question in EEG studies of emotional disorders is whether certain groups, depressives or schizophrenics for example, will be reliably characterized by a specific pattern of EEG asymmetry. There are a number of methodological issues in dealing with just this elementary question, but an associated question is also important: What is the functional significance of these asymmetries of cortical activity? Are the asymmetries due to some insidious organic lesion, perhaps coupled with the hypothesized biochemical impairment? Or are the asymmetries due to a transient emotional state associated with the patient’s current psychological distress? A careful treatment of these questions is important not just to understanding the results of EEG research in psychopathology, but to designing meaningful experiments.

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In our research on asymmetries in normal emotion, my colleagues and I have attempted to gather EEG data to supplement observations with measures of attentional biases and lateralized cognitive tasks. We have been particularly interested in EEG asymmetries in a depressed mood that seem to implicate the involvement of the right hemisphere. In attempting to interpret our finding, and to relate these to research with clinical populations, we have faced a number of difficult theoretical issues, questions of interpreting data on emotion and brain arousal in general, and interpreting EEG data in particular. In this paper, I will first discuss some general issues in current EEG research in psychopathology. Then in describing our research on normal depression, I will attempt to illustrate how basic questions of emotional arousal may be important to EEG research. Finally, I will attempt to review some interpretative issues in reasoning from EEG data to the emotional arousal and cognitive capacities relevant to psychological adjustment.

2. Electroencephalographic studies of psychopathology

Clinical study of brain electrical activity was first conducted within a psychiatric hospital. Berger's hope was that the study of brain waves could shed light on the alterations in brain function in disordered emotion (Perris, 1980). Although no clear explanations of psychopathology were immediately forthcoming in EEG research, this method has been applied to research with psychiatric patients in a number of systematic research programs over the last several decades (see Perris, von Knorring and Kemali, 1980).

The problems that plague much of the research on psychopathology, such as nosological vagaries and the difficulty of evaluating drug-free subjects, have hampered EEG studies as well. In addition, the ambiguity of the EEG measure itself has proved confusing. Before computer quantification of the EEG signal, research with EEG measures could occur at a grossly descriptive level at best. Unless frank neural pathology were evident in the raw record, clinical descriptions of the characteristics of the EEG were highly variable, subject to demand characteristics of the experiment, and quite difficult to compare across laboratories.

With the advent of digital computers, quantitative analysis promised not only consistency and reliability of measurement, but the capacity to extract new and revealing information from the EEG signal. Unfortunately, the richness of the information in the EEG was paralleled by multiplicity in the quantitative analysis methods developed in laboratories around the world. Each investigator or laboratory team experimented with a particular quantification scheme. These varied approaches often produced interesting results, but the research field as a whole lacked a paradigm (Kuhn, 1962), a standard-mode of operation.
The development of a workable computation method for Fourier analysis of time-series data by Cooley and Tukey allowed the precise delineation of the power or energy in the EEG waveform. Power spectral analysis has been the most consistently applied method in modern EEG research, and has had important applications to psychopathological disorders (Flor-Henry and Koles; Etevenon, Pidoux, cottereau, Peron-Magnan, Zarifian, Verdeaux and Denker, 1980). In addition, covariance measures among pairs of electrodes have been used to study normal cognitive processes (Beaumont, Mayes and Rugg, 1978), and have been applied to research on psychiatric groups (Flor-Henry and Koles, 1981). Although the application of methods of time-series analysis adapted from communications engineering (Bendat and Piersol, 1971) to EEG research has provided standardization as well as quantitative sophistication, comparison of results across laboratories remains a problem. These methods yield a voluminous array of data, and researchers who reduce their data in one form will not be able to compare findings to those selecting another data reduction method. The problem of concisely characterizing the most relevant information from time series analysis of a multichannel EEG promises to remain a challenge for some time to come (Tucker and Roth, 1984).

The selection of measures of the EEG in quantitative analysis should be determined at least in part by a priori consideration of the features of cortical electrophysiology relevant to the research questions. In much of the early work on psychopathology, the focus was on pathognomonic signs, indications of neural pathology. In some cases these indications are positive, and the question of psychiatric symptoms and inappropriate psychiatric diagnoses in cases of clear organic lesions remains important in neuropsychological research (Heaton and Crowley, 1981). But it seems fairly clear now that the majority of psychiatric patients will not be found to have organic lesions or epileptiform pathology that are conventionally indicated by a clinical EEG. For this research area, the question has become whether a more subtle analysis of the EEG will reveal a more latent neuropsychological disorder.

The construct of hemispheric specialization has provided an important hypothetical framework for EEG studies of psychopathology, as it has done for other areas of neurology, psychology and psychiatry. With the affective disorders, for example, astute researchers noticed early on that patients manifesting clinical depression showed asymmetrical EEG patterns (Perris, 1975). Other findings included asymmetries in event-related potentials in depressive patients (Perris, 1974). Even at this early stage, methodological issues clouded the interpretation of results. One of the most interesting findings with the depressives concerned not the power measures but a measure of the variability of the EEG amplitude (Perris, 1975). Although this measure has shown quite interesting relations to psychological processes in Goldstein's laboratory (Goldstein, 1981), it has not been adopted widely by other researchers, and its definition in neuropsychological terms is difficult to clarify.
Another methodological issue illustrated by these initial studies of psychiatric depression is the use of asymmetry scores. With differences among patient groups characterized by a left–right difference, the statistics do not reveal whether the relevant phenomenon occurs in the right or the left hemisphere. While this problem is often difficult to avoid in research on lateralization with performance measures such as tachistoscopic presentation or dichotic listening (Bruder, 1982), individual measures of each hemisphere's function are provided by EEG data. Some further studies of brain electrophysiology in the affective disorders (Perris and Monakhov, 1979; Flor-Henry, 1976) have suggested that it is abnormality of the right hemisphere particularly that contributes to the left–right asymmetry of function.

The complex issues in quantifying and interpreting EEG data can lead to a focus on the measurement aspect of the research, and failure to attend to the characteristics of the phenomenon being measured. The implicit assumption in most studies of psychiatric patients is that the asymmetric brain function is a stable, characteristic feature of the pathological condition. This assumption has not proved tenable in neuropsychological testing studies of depressives. While depressed patients show poor right hemisphere function (Goldstein, Filskov, Weaver and Ives, 1977), those that show clinical improvement also improve in right hemisphere task performance (Kronfol, Hamsher, Digre and Waziri, 1978).

Throughout EEG research, the inferential question that is too often unexamined is what the EEG measure has to do with the phenomenon in question, whether the phenomenon is cognitive processing or a psychopathological disorder. One explanation for EEG changes that are not frankly pathological is that they reflect changes in the arousal status of the cortex. Given that the neuropsychological test results of psychiatric patients change as their affective state changes, and given that this effect is not diffuse but is laterally-specific, the possibility arises that the EEG asymmetries also observed in these patients reflect not some insidious neurophysiologic lesion, but the normal hemispheric asymmetry associated with a strong emotional state. This is the possibility that my associates and I have examined in our studies of EEG measures recorded during a depressed emotional state in normal subjects.

3. Alpha asymmetries in normal emotion

In some initial questionnaire data collected on normal university students, we observed that students who reported greater depression described themselves as having poor visual imagery (Tucker, Stenslie, Roth and Shearer, 1981). Although self-report biases (Swenson and Tucker, 1983) and other factors could account for this relationship, we were interested in whether this effect in normals might parallel the poor right hemisphere neuropsychological
task performance observed in clinical depression. To manipulate depressive emotion experimentally, we asked students to engage in mood-induction procedures. The first question was whether depressive mood would alter right hemisphere performance ability. The next question was how this might occur. Most likely would be a change in the arousal level of the right hemisphere during the transient depression. As an index of cortical arousal level, we examined the power in the alpha frequency band of the EEG.

3.1. Mood induction studies

Students in psychology courses were recruited to participate in research on emotion and brain function. Careful screening of the applicants and openness in discussing the procedures of the research insured the experiment would be an interesting and educational experience for the subjects. In the first study, 4 male and 6 female right-handed students were given relaxation instructions, then listened to hypnotic suggestion of either depressive or euphoric mood states. During each mood state, mental arithmetic, visual imagery and lateral attentional bias tasks were performed. In the attentional bias task, the subject listened to two tones presented simultaneously to the two channels of stereo headphones; a bias in reporting the perceived loudness of tones in left and right ears was used to indicate relative hemispheric activation (Kinsbourne, 1970).

The results of this first study showed imagery to be poorer during the depressed mood while arithmetic performance was unchanged. Although such performance differences are interesting, the auditory attentional bias measure was a more convincing indication that some change in hemispheric activity had occurred. Compared to relative symmetry of the loudness judgments during euphoria, a right-ear louder bias was observed during depression, which we interpreted as reflecting either higher left or lower right hemisphere activation in depression (Tucker et al., 1981).

To more directly index each hemisphere’s activation level, a second mood-induction study was conducted. Eight male and sixteen female students engaged in similar mood-induction procedures as the first study. During each mood condition the subject performed simple arithmetic and imagery tasks; these were not designed to yield performance measures but to involve generally hemisphere-specific ideation as the EEG data were collected during the mood condition.

Electrodes were attached to the left and right frontal (F3 and F4), central (C3 and C4), parietal (P3 and P4) and occipital (O1 and O2) locations, referenced to linked ears with a vertex ground. The signals were amplified with a 0.1 sec time constant and digitized at 500 Hz. For each task in each mood condition 1024 samples were digitally filtered at 32 Hz each of 15 epochs. After tapering the epoch with a Hanning window, the data were spectrally
analyzed to produce power spectra of 0.5 Hz resolution. Peak power in the alpha band (7.5 to 12.5 Hz) was extracted from each spectrum and averaged to yield one alpha power value per task per mood condition per subject. Alpha asymmetry (left minus right power) was computed for each lobe (ie. frontal, central, parietal and occipital) and subjected to one-way analyses of variance.

The results of alpha asymmetry analysis showed a significant main effect for the mood condition on the asymmetry in the frontal electrodes. Examination of means showed relative symmetry between the frontal regions during the euphoria condition, compared to greater right frontal activation (desynchrony of alpha) during the depressed mood.

These findings suggest that both lateralized cognitive and electrophysiological measures will vary with normal depressive affect, but the EEG data were more complex than we had expected. The decrement in imagery in the first study seemed to replicate the association between depressive mood and poor imagery we observed in the questionnaire data, paralleling the findings with psychiatric depressives. The shift in lateral auditory attentional bias offers support for the interpretation that a change in relative hemispheric activation was responsible for the imagery decrement. Recent observations of parallels between cycling mood levels of affective patients and lateral biases in localizing sound in space (Sackei and Decina, 1983) are congruent with our observations with normals. With greater depression, the localization shifts rightward. A number of our students actually reported the tone loudness task as a spatial localization phenomenon.

Our hypothesis was that with a shift in attentional bias we would also find a change in hemispheric activation measurable in the EEG data. The results showed that, instead of the expected decrease in right hemisphere arousal across all locations, there was actually an increase in activation (indexed by a decrement in alpha activity) over the right frontal region. Given the apparent impairment of right hemisphere cognitive and attentional performance, and given the substantial evidence of the inhibitory roles of the frontal lobes (Flor-Henry, 1977; Hecaen, 1964), we speculated that the activity shown by the right frontal region may have been inhibitory in nature, representing a mechanism through which brain processes in the depressive state inhibit right hemisphere cognitive function.

Independently of our research, Davidson, Schwartz, Saron, Bennett and Goleman (1979) also observed a frontal lobe asymmetry in alpha activity during positive and negative mood states, with relatively greater right frontal activation also indicated for the more negative emotion in their data. However, Davidson et al. (1979) did not interpret the frontal alpha asymmetry as inhibitory, but as suggesting general left hemisphere involvement in positive emotion, compared to a general increase in right hemisphere activity in negative emotion.
3.2. Method actor study

More recently, Dawson and I (Tucker and Dawson, in press) have conducted further research on EEG data in normal depression, working with actors as they created emotional states in the laboratory. Previous research with autonomic measures has found that, unlike non-method actors, method actors show psychophysiological responses as they portray the experience of emotion (Stern and Lewis, 1968). The acting technique used by these persons draws on personal emotional memories to recreate a subjectively-felt emotional state. Four male and three female faculty and graduate students from the Theatre Arts department of the University of North Dakota participated in this research.

Depression was one target emotion in this study, to maintain continuity with our previous findings. We selected erotic arousal as the contrasting emotion for several reasons. The findings on frontal asymmetry, and our interpretation of them, raise the issue of the function of the hypothesized frontal inhibitory effects. Perhaps right frontal activation in a depressive mood serves to inhibit right posterior processing of imagery. If so, the opposite frontal lobe effect, left frontal activation in positive emotion, should be relevant to the inhibition of left hemisphere cognition in positive emotion. Perris and Monakhov (1979) have found that anxiety in psychiatric patients is related to left frontal EEG activity, whereas mood level covaries with right frontal EEG measures. Flor-Henry, Yeudall, Koles and Howarth (1979) found poor left frontal lobe cognitive function in severely obsessive-compulsive patients, and speculated that the left frontal region was not inhibiting the ruminative cognition of the left hemisphere. We have observed highly anxious normals to show high left hemisphere activation (Tucker, Antes, Stenslie and Barmhardt, 1978) and detail-oriented perception (Tyler and Tucker, 1982), further suggesting a link between anxiety and left hemisphere cognitive operations.

The clinical observations of Masters and Johnson (1970) suggest that anxiety is antithetical to sexual arousal. We hypothesized that as they created an erotic emotional state, the method actors would show left frontal lobe activation which would serve to inhibit left hemisphere cognitive operations. Thus in hypothesizing the regional cortical function contrasting the emotions of depression and erotic arousal, we predicted opposite effects for anterior and posterior locations. We expected right hemisphere perceptual and cognitive function, and thus right posterior brain activation, to be strong during sexual arousal and minimal during depression. For the frontal lobes, we expected right frontal activation during depression, contrasted with left frontal activity during sexual arousal.

The EEG data were appropriately filtered, windowed and spectrally analyzed (Tucker and Dawson, in press), and alpha power analyzed with analysis of
variance across mood conditions and electrode locations. The results confirmed some of our hypotheses and not others. A significant mood by hemisphere interaction showed low right hemisphere activation (high alpha) in depression and higher right hemisphere activation in sexual arousal. The right/left asymmetry of alpha reversed between the mood conditions. Inspection of the data and tests for individual comparisons showed the effect to be strongest for the more posterior (central, parietal and occipital) locations, while no significant alpha asymmetries were found for the frontal lobes.

4. Interpreting EEG data

These studies of normal persons suggest that asymmetries in the EEG may occur during normal emotional processes. Such results emphasize the importance of considering transient, emotional state effects when interpreting EEG studies of psychopathology. To consider more specifically the interpretation of asymmetries of alpha in normal depression requires facing some difficult issues of the meaning of EEG data, how it relates to cortical arousal, and what brain arousal systems have to do with the disordered emotion and thought of psychopathology.

4.1. Electrophysiologic and neural arousal

The most straightforward implication of our observations on normal depression is that changes in the emotional arousal of the brain may be intrinsically asymmetric, involving one hemisphere more than the other. The traditional interpretation of the clinical EEG is with reference to a neurologic lesion or neurophysiological pathology, as in epilepsy. This sort of approach may not be relevant to much of the research on psychopathology, particularly if studies of normal emotion suggest that asymmetries of brain arousal systems may underlie the EEG asymmetries observed in psychiatric disorders.

It is readily shown that gross changes in an individual's arousal level, from sleep to an alert resting state to concerted cognitive effort, will alter the EEG. It is thus reasonable to interpret asymmetries of an EEG parameter such as alpha power as indicating asymmetries in cortical arousal. Yet this is an inference and the relation of alpha activity to the arousal status of the cortex is too often assumed rather than considered explicitly in EEG research on brain lateralization. From a measurement perspective, alpha is only one of several distinct frequency characteristics of the on-going EEG. So little is known about changes in the EEG with cognition that a focus on just one band of the signal seems premature. Furthermore, power measures are one form of a number of measures generated by signal analysis methods. Covariance indices, such as coherence between electrode pairs, have shown interesting changes as a function of cognitive effort (Beaumont et al., 1978) and may eventually prove more informative than straight power measures. However, it is not easy to establish
the meaning of this measure in neurological terms.

Even with the frequently used measure of alpha power, it remains an
inference that cognitive function involving a certain brain region will influence
alpha activity. Assuming that increasing cortical arousal will reduce the level of
alpha in a consistent, linear fashion, it may not be the case that a given
cognitive process will necessitate an observable change in cortical arousal.
Perhaps only cognitive effort that draws on brain arousal systems to increment
processing capacity (Kahneman, 1973) will influence alpha. Insufficient care in
delineating the neurophysiological inferences relating the EEG measure to the
psychological process could lead to poorly designed experiments and failure to
replicate results across laboratories.

In autonomic psychophysiological research, the 'law of initial values'
(Wilder, 1957; Sternbach, 1966) states that a response to a stimulus may be
preconditioned by the activity level in the response system. For example, heart
rate acceleration to a loud tone can be expected to be less in a person with a
prestimulus level of 200 beats per minute than in someone starting at 60 beats
per minute. This sort of consideration may be relevant to EEG research as
well. Alpha desynchrony, asymmetrical or not, may be much greater in an
individual with abundant resting alpha, or in a psychological state that allows
the alpha rhythm to appear. We have observed substantial individual differ-
ences in baseline alpha that influence the effects of cognitive tasks on alpha
asymmetry. These individual differences in baseline EEG relate to personality
and cognitive style measures (Dawson, 1982). Previous research has shown
baseline asymmetries in EEG alpha (Furst, 1976) and regional cerebral blood
flow (Gur and Revich, 1980) to be predictive of visuospatial task performance.
Baseline alpha asymmetries also have been found to predict symbolic verbal
performance (Glass and Butler, 1977). To meaningfully examine alpha asym-
metries in relation to emotion or cognition, it is important to consider carefully
subject characteristics, the pre-existing state of the subject, and how the
psychological phenomenon of interest will alter the subject's neural arousal.

A closely allied question is whether changes in neural arousal level will
produce focal changes in cortical function and thus the EEG. The current
model of neuropsychology is localization of function to discrete areas. This
model has been influenced by clinical studies of the psychological effects of
discrete lesions, and may be of limited utility when describing the dynamic
function of the intact brain. Kinsbourne (1982) has called for network
cornerstone of neural organization, since populations of neurons are richly
interconnected across wide regions of the cortex, rather than narrowly segre-
gated among specific loci. It is quite possible that EEG changes during
emotional arousal or cognitive effort may be more diffuse than focal; such
network effects may be important to consider in efforts to localize cognitive
processes with EEG data.

Given the assumption that regional differences in EEG activity will reflect
psychological variables in an intact brain, it is still an open question whether a lateral distinction is more explanatory of a psychological process than a regional differentiation, for example a front–back one. The construct of hemispheric specialization has been a remarkably heuristic one in neuropsychological research, yet if relied on exclusively it may blind researchers to other meaningful regional patterning of brain function. When multiple electrode studies are conducted and analyzed appropriately, of course, the data itself may point to other distinctions than lateral asymmetry as being relevant to the psychological phenomenon of interest. This is what has occurred in our research on depression. When we began we were interested only in differential hemisphere contributions, but soon we were forced to consider the differential involvement of anterior and posterior regions.

In both the mood induction and the method actor studies of depression, the right hemisphere showed the clearest contrasts between mood conditions. However, under the assumption that alpha desynchrony indicates cortical activation, depression increased right frontal activation in the first study and decreased right posterior activation in the second study. An obvious difference between the studies was the contrast emotions; inherent differences between the euphoria and erotic arousal conditions would influence the contrasts with depression that emerged. Another important methodological point is that the subjects in the first study were performing arithmetic and imagery cognitive tasks as the frontal activation was observed in the depressed mood, whereas in the method actor study the actors used naturalistic cognition to generate the emotional states. The relation between the on-going cognition and the emotional state thus differed in important respects between the two studies.

Along with these questions as to the actual nature of the psychological processes contributing to the alpha asymmetry are basic questions on the differences in the functions of anterior and posterior brain regions in emotional processes. The front–back difference has become an unavoidable issue in EEG research on normal emotion (Tucker et al., 1981; Davidson et al., 1979). It seems likely to be important to moving beyond dichotomous notions of laterality in psychopathology and brain damage as well. Controversy in interpreting the effects of unilateral lesions on positive and negative emotions has appeared in the literature. Sackeim, Weiman, Gur, Greenberg and Hungerbuhler (1982) suggested a unilateral lesion disinhibits the emotional tone of the contralateral hemisphere, while I have suggested that disinhibition of the ipsilateral hemisphere’s affective orientation may occur (Tucker, 1981).

Robinson, Kubos, Starr, Rao and Price (in press) have shown recently that the caudality of the lesion is a major variable in determining the emotional valence following a unilateral lesion. The degree of depression following a lateralized lesion was found to correlate positively with distance from the frontal pole in the right hemisphere but to correlate negatively with distance from the frontal pole in the left hemisphere. Congruent findings on the relation
of lesion caudality to depression in the right hemisphere have been reported recently by Finset (1982). Assuming the role of the frontal lobe to be inhibitory (Flor-Henry, 1977; Hecaen, 1964), the more negative response with anterior left hemisphere lesions could be interpreted as consistent with a disinhibition of left-lateralized catastrophic anxiety, whereas less depression with more anterior right hemisphere lesions may be consistent with a disinhibition of that hemisphere's more positive characteristic affective valence (Tucker, 1981). Although firm conclusions still remain elusive, the data of Robinson et al. (in press) show that for emotional effects of lateralized lesions, as with alpha asymmetries in positive and negative emotion, anterior–posterior differences can no longer be ignored and may be crucial to understanding hemispheric specialization itself (Tucker and Williamson, 1982).

Because there are intrinsic differences in EEG data between anterior and posterior cortical regions, with much greater alpha power, for example, recorded from occipital than from frontal cortex, comparisons between front and back electrodes may be more difficult than those between left and right. Homologous left and right cortical areas are assumed to have anatomically and neurophysiologically parallel structure and organization; thus any asymmetries in the EEG are assumed to be related to neuropsychological function. However, given the increasing evidence of differing anatomical organization of the left and right hemispheres (Galaburda, LeMay, Kemper and Geschwind, 1978; Gur Packer, Hungerbuhler, Reivich, Obrist, Amarnick and Sackheim, 1980), this sort of reasoning may no longer be appropriate. We have observed lateral asymmetries of the topographic distribution of EEG coherence that seem to parallel the anatomical asymmetries that differ between anterior and posterior regions (Tucker, Roth and Bair, 1984). Coherence appears higher on the right side, especially in anterior regions; this may be relevant to the larger frontal lobe on the right (Galaburda et al., 1978) or the greater proportion of white matter in the right hemisphere (Gur et al., 1980).

With the necessity of considering concepts of neural arousal in designing and interpreting electrophysiological experiments on psychological processes, and with the need to formulate concepts of differential anterior–posterior brain function as interacting with left–right differences, research on EEG asymmetry can become substantially more complicated than the early studies of alpha asymmetry that had one electrode over each hemisphere. However, if it is possible to more accurately characterize the electrophysiology of the primary systems regulating neural arousal, EEG research may prove fundamental to integrating biological and psychological approaches to emotional disorders.

4.2. Biochemical and emotional arousal

To proceed beyond simplistic interpretations of abnormalities in brain arousal processes in psychopathology will require the development of a better
understanding of how emotional arousal is accomplished through neural regulatory circuitry, and how neural control systems are integral to psychological self-regulation. The primary arousal systems of the human brain are neurotransmitter-specific; concepts relevant to these systems may prove explanatory for the effects of pharmacologic treatment of psychiatric disorders.

From the electrophysiologic researcher's perspective, the question is how activity of a particular neurotransmitter system will manifest itself in cortical electrical activity. From the perspective of the biochemical researcher, the question is how the function of a neurotransmitter pathway can be observed in brain activity. Although behavioral and psychological models of neurotransmitter action are the desired end goal, conventional notions of neurotransmitter action hold that changes in the EEG are a major form of evidence that neurotransmitters regulate brain function through somehow altering neural arousal (Cooper, Bloom and Roth, 1974).

EEG measures may thus be important in mediating between concepts of neurotransmitter action and the psychological processes relevant to mental disorder. To achieve some explicitness in the characterization of this mediation, however, a theoretical formulation is required of the emotional significance of a change in neural arousal resulting from neurotransmitter activity. Important initial concepts of emotional arousal emerging from the operation of the reticular activating system were suggested by Lindsley (1957). More specific notions of emotional characteristics of brain arousal processes have been suggested by evidence of lateral brain asymmetries in normal and abnormal emotion, and by initial evidence that the major monoamine neurotransmitter systems of the human brain are asymmetric in both anatomy and function.

Given the suggestions of left hemisphere over-activation and dysfunction in schizophrenia (Flor-Henry, 1976; Gur, 1978), and given the current biochemical model of this disorder as stemming from an excess of dopamine system function (Metzler, 1979), it seems quite relevant that dopamine metabolites correlate with left but not right hemisphere event-related potentials (Gottfries, Perris and Roos, 1974). Since anxious normals seem to exhibit high left hemisphere activation and a detail-oriented perceptual style (Tucker et al., 1978; Tyler and Tucker, 1982) and highly anxious subjects may show left hemisphere dysfunction (Tucker et al., 1978), it may be that anxiety is the psychological characteristic of the operation of dopaminergic brain systems in humans, and these systems may augment left hemisphere function specifically (Tucker, 1981).

Given our observations of changes in EEG data from the right hemisphere in depression, my associates and I have been particularly interested in evidence that the neurotransmitter systems controlling mood level are more important to the right hemisphere than to the left. The catecholamine hypothesis of the affective disorders (Schildkraut, Orsulak, Schatzberg, Gudeman, Cole, Rohde
and LaBrie, 1978) holds that high norepinephrine activity characterizes mania, while low norepinephrine characterizes depression. Norepinephrine may be more highly concentrated on the right side in the thalamus (Oke, Keller, Mefford and Adams, 1978). The changes in right hemisphere functional capacity observed in normal and abnormal mood changes (Sackeim and Decina, 1984; Kushnir, Gordon and Heifetz, 1980) may thus be influenced by altered norepinephrine activity. Current models of mood suggest serotonin modulates norepinephrine activity level (Treiser, Cascio, O'Donohue, Thoa, Jacobowitz and Kellar, 1981). In research with psychiatric patients, serotonin metabolites were found to correlate with right but not left hemisphere event-related potentials (Gottfries et al., 1974).

Thus if the effect of disordered brain biochemistry on psychological disorganization is mediated through alterations in neural arousal, EEG measures should provide relevant research information.

If hemispheric asymmetry in emotional states results from the intrinsic asymmetry of brain arousal systems, then effective pharmacologic treatment should reverse or normalize the asymmetric brain activation. Serafetinides (1973) found chlorpromazine to normalize the high left hemisphere activation observed in schizophrenics' EEGs. Mandell and Knapp (1981) observed that cocaine increases the asymmetry of serotonin in mice brains, while lithium decreases it. Flor-Henry and Koles (1981) have shown that administration of lithium to normals produces EEG changes that are specific to the right parietal region.

4.3. Arousal and function

If the neurotransmitter-specific arousal systems of the human brain are asymmetrically organized, and if their operation produces subjectively meaningful emotional states, it would be reasonable that the hemisphere asymmetries of function observed in psychopathology would have parallels in normal emotional processes. In the attempt to provide a foundation in basic research for the clinical work with brain function in psychopathology, there should be an important place for studies of EEG asymmetries in normal emotion. This research, coupled with an increasing appreciation of the brain's neurotransmitter control systems, could suggest new concepts of emotional processes.

But in many respects the major issue in psychopathology is not abnormal brain arousal, nor even an abnormal subjective emotional state, but disorders of cognitive function that have insidious and cumulative effects on the person's ability to cope. They key theoretical issue in understanding brain arousal systems is how the brain self-regulates its cognitive capacity through altering its own arousal as a function of internal states and environmental demands. There is growing interest within cognitive psychology in the influence of emotional arousal on cognition (Bower, 1981). Yet with the exception of Kahneman's (1973) important work, there has been little effort in cognitive
psychology to develop concepts of adaptive self-regulation of arousal that are congruent with the neurophysiological evidence.

An important model of the neural circuitry regulating attention and brain activity has been suggested by Pribram and McGuinness (1975). They differentiate between an activation system which handles motor readiness and an arousal system that regulates the brain’s phasic response to perceptual input. Within this framework, qualitatively-specific forms of attentional control emerge from the systems controlling neural arousal. Williamson and I (Tucker and Williamson, 1982) have reviewed the cognitive and behavioral effects of the dopamine and norepinephrine systems that are integral to the motoric activation and perceptual arousal systems, respectively. The neurotransmitter literature indicates qualitative attentional control features of these systems that are substantially in agreement with the Pribram and McGuinness (1975; McGuinness and Pribram, 1980) model. Through considering the affective characteristics of these neurotransmitter systems that are shown in the research on psychopathology, it may be possible to develop a theory of neural control systems that describes adaptive, emotional regulation of on-going brain activity and cognitive capacity.

From this perspective, a major interpretive issue in research on psychopathology parallels closely a major question in EEG research on lateralized cognitive processes: How does the brain self-regulate its arousal status to adaptively control cognitive capacity? In EEG research on localization of cognitive function, the question presents itself in the context of interpreting EEG data, which seems to have something to do with cortical arousal. It may not occur to researchers studying cognitive function that the neural systems regulating cognitive effort and electrophysiology will have affective characteristics, such as anxiety or mood. But this is what seems to be suggested by the confluence of evidence on brain arousal systems: The operation of arousal systems are often experienced subjectively as affective states, and the effect of their operation is a change in attentional capacity (Tucker and Williamson, 1984).

The regulation of cortical arousal, attention and cognitive capacity by systems that are inherently emotional suggests an important new framework for understanding psychopathology and emphasizes the importance of a solid research base in the electrophysiology of normal emotion and cognition. The explanatory power of the new theoretical models will come from the descriptions of how cognition is altered in qualitatively specific ways, rather than diffusely, by neural arousal systems.

References


