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CHAPTER 20

Laterality and emotion: an electrophysiological approach

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Introduction

Although functional differences between the two cerebral hemispheres in the control of emotional behavior have been noted for over fifty years (e.g., Alford, 1933; Goldstein, 1939), relatively little systematic study of this problem occurred prior to the last decade. During this latter period there has been a dramatic increase in research on this topic. Furthermore, this accumulating body of work encompasses a wide variety of methodological approaches. For example, neurological, psychiatric and normal populations have all been studied extensively, and a range of assessment techniques have been used. These latter include self-report and behavioral indices of emotion, and behavioral, electrophysiological and other indices of regional brain activity (for reviews, see Davidson, 1984; Leventhal and Tomarken, 1986; Silberman and Weingartner, 1986; Tucker, 1981; Tucker and Frederick, in press). In addition to this large corpus of new data in humans, animal findings consistent with the human data have been obtained, and promising animal models of the lateralization of emotion have been developed (for reviews, see Denenberg and Yutzey, 1985; Glick and Shapiro, 1985).

In this chapter, we will first present a brief review of representative findings in the area of lateralization and emotion. In the process, we will highlight several key conclusions from these data and discuss some significant gaps in our current understanding of the hemispheric substrates of emotion. After presenting this brief overview, we will consider some specific methodological requirements for research on the psychobiology of emotion. We will then present recent electrophysiological research from our laboratory on both task-dependent emotion-related EEG asymmetries and on individual differences in resting patterns of asymmetry which predict emotional reactivity.

Asymmetry and emotion: previous empirical observations

Although there has been a notable convergence of findings and conclusions across studies using different methodological approaches, there have also been inconsistent findings and differing interpretations of those results that have proven replicable (e.g., Davidson, 1984; Levy, 1983; Tucker and Williamson, 1984). Previously, we (Davidson, 1984; Leventhal and Tomarken, 1986) have argued that one major reason for disagreements and inconsistencies in this area is the failure to distinguish between the *perception* or *decoding* of emotional information, and the *experience* or *expression* of emotion. Additionally, investigators have often failed to distinguish regional variations in specialization or activation within a hemisphere, along the rostral-caudal axis. Taking these two sets of factors into account, a number of converging lines of evidence indicate that the posterior regions of the right hemisphere are relatively more special-

ized for the perception, or decoding, of affective information.

Posterior right hemisphere superiority for the perception of emotional information is supported by evidence from both brain-damaged and normal populations. Concerning the former, several studies have shown that posterior right hemisphere lesions impair the ability to detect the emotional tone of speech. No such impairments were found in patients with left hemisphere lesions (e.g., Heilman et al., 1975; Tucker et al., 1976). Right hemisphere parietal lesions have been found to impair the ability to recognize emotional faces, while comparable lesions in the homologous left hemisphere region have little effect (Etcoff, 1986). Consistent with these clinical findings, a plethora of studies in normal adults and children have shown right hemisphere superiority for the perception of emotional information. In the auditory modality, investigators have examined hemispheric differences in the ability to judge the emotional tone of speech or to identify non-verbal emotional sounds. For example, Carmon and Nachson (1973), in one of the first studies to evaluate hemispheric differences in the perception of emotional information, found that accuracy of emotional sound identification (e.g., baby crying) was better in the left ear (whose fibers project predominantly, although not exclusively, to the right hemisphere) than the right ear. In the visual modality, many similar studies have been performed. The essential method used in these studies has been to compare either reaction time or accuracy of recognition in response to hemi-retinal presentations of visual stimuli. When such stimuli are exposed to the left visual field, they project directly to the right hemisphere and vice versa. These studies have repeatedly found enhanced performance when emotional stimuli are presented directly to the left versus right visual field (e.g., Ley and Bryden, 1979; Saxby and Bryden, 1985; Suberi and McKeever, 1979).

Most of the studies which were designed to examine hemispheric differences in the processing of emotional versus non-emotional information have not explicitly considered the valence of the emotional stimuli. For example, some studies have not used an equal number of positive and negative emotional stimuli (e.g., Carmon and Nachson, 1973; Schwartz et al., 1975). Most studies which have carefully compared differences in performance as a function of the valence of the emotional cues have not found systematic differences in the processing of positive and negative stimuli. Right hemisphere superiority is typically found for both types of cue (e.g., Ley and Bryden, 1979; for review, see Etcoff, 1986; Silberman and Weingartner, 1986). Some studies, however, have reported hemispheric differences in the perception of positive and negative emotional information, with left hemisphere superiority in the perception of positive cues and right hemisphere superiority in the perception of negative cues (e.g., Reuter-Lorenz and Davidson, 1981; Reuter-Lorenz et al., 1983; Natale et al., 1983). We have suggested that the differences between studies may in part reflect the degree to which affect is actually recruited by experimental stimuli (Davidson, 1984).

In contrast to the preponderance of evidence relating to hemispheric substrates of emotional perception, evidence concerning the experience or spontaneous expression of emotion underscores the importance of valence. Specifically, certain left hemisphere regions are activated during the experience or spontaneous expression of particular positive emotions, while corresponding regions of the right hemisphere are relatively more activited during the experience or expression of particular negative emotions. Furthermore, this pattern of asymmetry is most strongly linked to activity in the *anterior* regions of the cerebral hemispheres.

Evidence from several sources supports this conclusion. Studies on the affective correlates of unilateral brain damage indicate that lesions in the left anterior region are more likely to result in depressive symptomatology compared with comparable right hemisphere lesions. In one of the first systematic studies to compare the emotional consequences of left versus right-sided brain damage, Gainotti (1972) reported that left-lesioned patients

had significantly more negative affect and depressive symptomatology compared with right-lesioned patients. Sackeim et al. (1982), in a review of the literature on pathological laughter and crying subsequent to unilateral brain damage, found a similar pattern indicating relative left hemisphere specialization for laughter and relative right hemisphere specialization for crying. More recently, using rigorous quantitative assessment of lesion location inferred from CT-scan evidence, Robinson and his colleagues (Robinson et al., 1984) reported a very strong correlation between the proximity of a lesion to the frontal pole within the left hemisphere and severity of depressive symptomatology based on a composite index (see also Kolb and Milner, 1981; Robinson and Benson, 1981; Robinson and Szetela, 1981).

Electrophysiological studies on non-lesioned populations point toward a similar conclusion. Such studies have typically used spectral analysis of the EEG to make inferences about patterns of regional activation (see Davidson, 1988, for an overview of these methods). In the next part of this chapter we will review an extensive series of studies from our laboratory which use such methods. A number of other workers, using similar measures, have reported effects similar to ours. This evidence indicates that the anterior region of the right hemisphere is relatively more activated during negative compared with positive emotion (e.g., Ahern and Schwartz, 1985; Tucker et al., 1981). In addition, depressives have been reported to show more relative right-sided activation in anterior electrode sites compared with non-depressed subjects (see Henriques and Davidson, 1989, for review).

A strong linkage between anterior regions of the cerebral hemispheres and affective experience is additionally consistent with evidence of the extensive neuroanatomical reciprocity between prefrontal and anterior temporal regions of the cerebral cortex and limbic circuits known to be directly involved in the control of motivation and emotion (e.g., Kelly and Stinus, 1984; Nauta, 1971). As a caveat, we should note, however, that recent evidence from our laboratory (to be reviewed below) also indicates an association between at least one affective state (clinical depression) and asymmetries in posterior regions. That the pattern of posterior asymmetry in this case is opposite to that found in anterior regions once again underscores the importance of regional specificity. It should be noted that this posterior asymmetry is most related to cognitive dysfunction which accompanies depression and may not be specifically associated with the primary affective disturbance.

Prior to reviewing our own recent studies in this area, we should note several significant unexplored or unresolved issues pertaining to evidence for hemispheric differences in emotion. One of the most significant among the unexplored issues concerns the temporal coordination among different response systems which specify emotion. For example, do the changes in asymmetrical anterior activation precede, accompany or follow changes in the subjective experience or facial expression of emotion? Another significant unexplored issue is the relationship between electrophysiological measures of emotion-related asymmetries and other biological indices (e.g., regional cerebral metabolism as assessed by emission-computed tomography). A final unexplored issue is the relationship between patterns of cortical and sub-cortical activation in specific brain circuits implicated in emotion and motivation. We know from extensive animal evidence that emotion and motivation are represented at various levels of the neuraxis from the brainstem to the cortex (e.g., Gallistel, 1980a). Some have suggested that heightened activation in a particular cortical region is associated with decreased activation in underlying subcortical tissue (e.g., Tucker and Frederick, in press). According to one variant of this view, relative *left-sided* anterior activation in *cortical* regions is associated with relative *right-sided* activation in *subcortical* regions (e.g., Levy, 1983). One source of evidence which we believe militates against such an interpretation is data from patients with subcortical lesions who have been found to show emotional symptoms which are highly similar to those

found in patients with cortical damage to the same hemisphere. Specifically, Starkstein et al., (1987) have found that left anterior lesions, *both* cortical *and* subcortical, are associated with the presence of depressive symptomatology. Other investigators have observed similar effects (e.g., Cummings and Mendez, 1984).

In this chapter, we will review recent findings from our laboratory that indicate differential contributions of the cerebral hemispheres to the experience or spontaneous expression of emotion. We will concentrate on the results of our most recent studies using electrophysiological methods, specifically spectral-analysed EEG. Two general types of studies will be reviewed. The first type focuses on changes in EEG measures of activation asymmetry brought about by the experimental induction of emotional *states* in normal subjects. In such studies, emotional states have typically been induced by visual stimuli (e.g., affective film clips or slides) or by the self-generation of positive and negative emotional imagery. The second type of study focuses on *individual differences* in patterns of resting activation, assessed while subjects are sitting quietly, in the absence of exposure to an emotion elicitor. Studies in this latter category assess whether patterns of resting asymmetry can discriminate between diagnostic groups (depressives and normals), or whether such patterns can predict the affective responses of normals when subjects are exposed to emotion elicitors at a later point in time. We will first review our recent work on EEG asymmetries associated with the elicitation of emotional states. Before doing so, however, it is useful to note a number of unique methodological requirements for research on the psychophysiological manifestations of emotional states (from Davidson et al., in press).

Methodological desiderata in psychophysiological research on emotional states

Research on the psychophysiology of emotion has traditionally been hindered by the failure to adhere to a number of important methodological desiderata particular to studies in this area. Fulfillment of these requirements helps ensure that valid exemplars of the intended emotions are compared on measures which are appropriately chosen to reflect changes brought about by the induction of emotion. Below, we note eight such desiderata for research on the biological substrates of human emotion. These apply to studies of both autonomic and central nervous system components of emotion. In this section, and in the subsequent section reviewing our own research on EEG correlates of emotional states, we note the degree to which these desiderata are satisfied in our own studies.

1. Emotion must actually be elicited. While this requirement may seem trivial, many experiments which purport to study emotion may not actually elicit significant emotion in subjects. Some studies focus more on the perception of affective cues. Other studies, particularly those that focus on subject's perceptions of how they *would* think and feel in hypothetical situations, may focus more on the structure of subjects' beliefs about emotions, or the language that they use to describe emotion. If the goal of the research is to characterize the psychophysiology of emotion, then there must be evidence that emotion was actually produced.

We acknowledge that researchers studying the psychophysiological correlates of negative emotions may face valid ethical constraints that may limit their ability to fulfill this requirement. For example, the elicitation of intense, phobic-like fear in the absence of any benefit to subjects is not acceptable. There are, however, options which permit the transcendence of such constraints. For example, in some of our current research, we are inducing emotion in therapeutic contexts, for example during the course of exposure treatment for phobias.

2. Adequate procedures must be used to verify the presence of the intended emotion. One of the noteworthy characteristics of emotion is the lack of an isomorphic relationship between an elicitor and a particular emotion (see Ekman, 1984). In other words, the same elicitor will often produce an array of different emotions across subjects.

Even in response to elicitors that are specifically chosen to target a particular discrete emotion, subjects typically will report a range of different emotions if given the opportunity (cf. Ekman et al., 1980). In our recent work, we have attempted to produce emotion using film stimuli that induce relatively 'pure emotion' (e.g., high levels of sadness, low levels of other emotions). Our experiences selecting film clips for use in these experiments have underscored for us the relatively non-specific effects of the great majority of affective stimuli. It is particularly difficult to dissociate specific combinations of emotions. For example, we have found that the overwhelming majority of film clips that elicit significant disgust in subjects also elicit significant fear. Clearly, self-report instruments used to assess affective responses must assess both the targeted emotion and other emotions that may also be elicited. The failure to assess multiple emotions is particularly grievous in work designed to assess emotion-specific psychophysiological profiles.

3. Epochs of different discrete emotions must be separable. Not only is it likely that any given elicitor will produce more than one emotion, the relative intensities of different emotions may also change sequentially over time. Unfortunately, studies on the psychophysiology of emotion typically ignore this possibility by aggregating data over the entire duration of the eliciting stimulus. Simply put, it is imperative to identify separate periods of time during which different discrete emotions are present. Only in this way can the physiology that accompanies different emotions be directly compared.

Note that this requirement necessitates a method which provides a continuous or near-continuous measure of emotional state. One possibility here would be to have subjects continuously rate their emotional experiences using a joy-stick or similar device (e.g., Davidson et al., 1979a). However, one obvious limitation of this method is that the requirement of continuous rating may distract subjects from the experimental stimulus and weaken its emotional impact. As a better alternative, in our recent studies, we have surreptitiously videotaped subjects' affective facial expressions during exposure to stimuli. Using this unobtrusive method, we have been able to extract those periods in time during which particular discrete emotions were present.

4. The physiological measures chosen for study must have a sufficiently fast time constant to reflect brief periods of emotion. Quite often, emotional states are relatively fleeting, lasting perhaps less than four seconds (Ekman, 1984). High levels of emotional intensity are especially likely to be brief. It is clear that only those physiological events which have a relatively fast time constant are capable of being the core substrate for such affective phenomena. Correspondingly, only those measures that have sensitive temporal resolution are capable of tracking the physiological correlates of emotion in these circumstances. In our work, a major reason that we have relied on EEG measures of emotion is the excellent time resolution of EEG relative to competing techniques (e.g., cerebral blood flow, PET) for assessing the neural correlates of emotional states (see Davidson, 1988, for a review of the relative strengths and weaknesses of EEG). Certainly, when affective phenomena are more long-lasting (e.g., clinical depression), other physiological systems and measures whose response properties are slower may also be appropriate foci of study.

5. At least two emotions must be compared. An experimental design which allows for assessment of the effects of only one emotion makes it impossible to conclude whether any physiological changes observed are unique to that specific emotion or are non-specific changes associated with *any* emotion. Thus, at least two emotion conditions are required. In addition, it is preferable to have a baseline condition against which to compare the emotion-arousing conditions. It is possible that two emotion conditions will differ from one another, but only one condition will differ significantly from baseline. Such a finding could have an important bearing on the interpretation of a particular result.

6. Each emotion under consideration should be induced by at least two distinct stimuli or methods. This requirement is an extension of the preceding one. Simply put, in the absence of multiple stimuli or methods for inducing each emotion, it is unclear whether experimental effects are produced by emotion per se, or by the unique, potentially non-emotion-related, features of the stimulus. In our recent work, where feasible, we have endeavored to use at least two exemplars of each emotion category (e.g., two distinct film clips designed to elicit happiness) and to replicate results across multiple methods.

7. The intensity of the elicited emotion must be matched between conditions. When two or more emotions are compared, it is imperative to match the intensity of emotions so that differences in intensity do not confound the emotion-specific comparisons. Unfortunately, many investigators who have compared two or more emotions have not utilized any procedure to match the intensity of the elicited emotions (e.g., Schwartz et al., 1981). If two emotions that differ in intensity also differ in some physiological parameter, it is impossible to say whether emotion-specific factors or intensity account for the physiological differences.

It is important to note that stimuli matched under certain contextual conditions may not remain matched under others. For example, in our experience, ratings of the happiness elicited by film clips tend to be higher when subjects view films, and make ratings, in a group setting, rather than alone. Conversely, fear and disgust ratings tend to be higher when subjects are run individually rather than in a group. Thus, two sets of emotional stimuli matched on intensity in a group context may not be matched in an individualized setting. This point highlights the need to make the stimulus selection context as similar as possible to the actual experimental context.

8. For each emotion under study, a sufficient duration of data must be obtained from each subject. In order to obtain stable estimates of physiological activity, a certain minimum amount of artifact-free data is required. Precisely what constitutes a sufficient duration will vary as a function of the physiological measures of interest. If the dependent measure is EEG, a minimum of approximately 10 seconds would be required in order to obtain a stable estimate of spectral power (Davidson, 1988). In order to have sufficient durations of data, we have frequently used a within-subject aggregation strategy. For example, if physiological activity during smiling was of interest, all instances of the artifact-free physiology during target smiles would be extracted, analysed and then aggregated for each subject.

Patterns of brain electrical asymmetry during emotional states

Before reviewing our recent studies on patterns of EEG asymmetry during facial signs of emotion in adults and infants, we will briefly describe the EEG recording and analysis methodology which is common to all of the studies from our laboratory reviewed in this chapter. For additional details, the reader is referred to Davidson (1988).

In certain respects, brain electrical activity measures are ideally suited for the study of neural activity underlying emotional behavior. For example, such measures can be recorded non-invasively from the scalp surface. In addition, as noted above, brain electrical activity measures have a very fast time resolution. Indeed, events in the millisecond range can be resolved with certain measures. This makes them ideally suited for the study of emotion, where the critical periods for analysis are often very brief due to the fleeting nature of affect. In studies of emotional state, we often examine brain electrical activity which is coincident with brief facial expressions of emotion. We use the face as a flag to provide us with information on periods during which peak emotional activity is present. The brain activity is then extracted for these periods and subjected to quantitative analysis.

There are two salient disadvantages of brain electrical activity measures in the study of emotion which should also be noted. The first is a problem

endemic to all applications of these measures. Topographic differences in scalp-recorded brain electrical activity do not necessarily reflect activity from underlying neural tissue. The source generators of scalp-recorded brain electrical activity are difficult to unambiguously describe, particularly for neural activity which may be generated by large, multiple regions of the brain. Thus, the spatial resolution of this method is less than ideal. The second problem is that most features of brain electrical activity which are recorded from the scalp surface and analysed using the methods described below are generated in the cortex. Since we know that significant contributions to emotional behavior are made by structures which are subcortical, the analysis of cortical activity may seem tangential to the study of the neural substrates of emotion.

As we will illustrate below, certain cortical regions do participate in emotional behavior. It is those cortical areas which have extensive anatomical connections with limbic structures implicated in the control of emotion. The crude spatial resolution of EEG remains a significant methodological obstacle. However, confidence in the localization findings is increased as convergence across methods is obtained. As we note below, there are now striking parallels in the findings on regional electrophysiological asymmetries in 'non-neurological' depressed subjects and the effects of localized unilateral lesions on depressive symptomatology.

The principal method of quantitative analysis that will be presented in this chapter is the Fast Fourier Transform (FFT). This procedure decomposes the complex waveforms of the EEG into their underlying sine-wave constituents. From this analysis, estimates of power in different frequency bands can be obtained. To obtain stable estimates of spectral power for brain activity accompanying a particular emotional expression, we require a minimum of about 10 seconds of artifact-free EEG. Once power values are obtained, they are log-transformed to normalize their distribution. Before analysis, all data are artifact-scored by hand to eliminate data confounded by eye movements, muscle activity and other sources of artifact. The FFT is then performed on artifact-free epochs of EEG. When analysing EEG contemporaneous with facial expressions, these epochs are approximately 1 second in duration. They are extracted via a Hamming window, which minimizes spurious frequency components (see Davidson, 1988, for a detailed discussion of these technical issues), and are overlapped by 75%. The output of the FFT is then averaged over all epochs extracted from the same condition so that a minimum of 10 seconds is obtained.

In the studies presented in the second part of this chapter on individual differences in asymmetry and their relation to affective reactivity, brain electrical activity is measured during a short resting period when subjects are sitting quietly. Due to the greater proportion of artifact-free data during resting baselines, the epoch length for the baseline-period FFTs is approximately two seconds in duration. The output of the FFT is aggregated over all epochs within the baseline period.

Although we typically extract, and perform statistical analyses upon, measures of power density in several frequency bands, in studies using adult subjects our experimental hypotheses and major analyses concern the alpha frequency band (8 – 13 Hz). This is because there is a wealth of evidence indicating that increased alpha activity is associated with decreased cerebral activation (e.g., Shagass, 1972) and because asymmetrical activation in this frequency band has been most consistently linked both to emotional states and to individual differences (for reviews, see Davidson, 1988; Davidson et al., 1989). The specific measure of asymmetry derived is the difference between the log of alpha power in the right hemisphere and the log of alpha power in the homologous left hemisphere region (i.e., log right alpha – log left alpha). Since increased alpha indicates decreased cortical activity, higher scores on this index indicate greater relative left hemisphere activation. In our studies with infants in the first year of life, we began by examining whole band power (1 – 12 Hz), since the specific frequency equivalent of

alpha activity in the infants is not definitively known. In adults, measures of whole band power (1 – 30 Hz) have been found to change in a fashion quite similar to alpha activity (see Davidson, 1988). In more recent studies with infants, we have found that most of the variance in both task-dependent changes in EEG asymmetry as well as individual differences in resting activation are in the 6 – 8 Hz frequency band (see review in Davidson and Fox, 1988).

Below we will briefly review two recent studies performed in our laboratory which examine EEG asymmetry during facial signs of emotion. The first experiment was a collaborative study with Ekman and Friesen (Davidson et al., in press) in which right-handed adult subjects were presented with brief film clips designed to elicit happiness and disgust. We chose happiness and disgust since these were emotions that we expected to be associated with approach and withdrawal respectively. As will be discussed more extensively below, we have hypothesized that approach vs. withdrawal may be the underlying dimension indexed by asymmetries in anterior regions. Subjects were presented with two positive and two negative film clips in a darkened room while we unobtrusively videotaped them and recorded brain electrical activity from the left and right mid-frontal, central, anterior temporal and parietal regions. All EEG leads were referenced to vertex.

In accord with the methodological desiderata described above, the films were carefully selected to elicit positive and negative affect at comparable intensities. The predominant emotion elicited by the positive clips was amusement and by the negative films the predominant emotion was disgust. The mean intensity of amusement reported in response to the two positive film clips (on a 0 – 8 point scale) was 5.88. The mean intensity of disgust reported in response to the two negative film clips was 5.44. These self-report data suggest that the two classes of films were well matched on the intensity of the predominant emotion elicited by each.

The video record of each subject was coded with Ekman and Friesen's (1978) Facial Action Coding System FACS). FACS distinguishes 44 action units. These are the minimal units that are anatomically separate and visually distinctive. Any facial movement can be described in terms of the particular action unit, or units, that produced it. The scorer identifies the action units, such as the one which pulls the lip corners up, or which lowers the brow, rather than making inferences about underlying emotional states such as happiness or anger. FACS scoring of the facial data from this experiment revealed that the two types of expression which occurred with the most frequency were happy expressions in response to the positive film clips and disgust expressions in response to the negative film clips. For each subject, the onset and offset of each happy and disgust expression was identified with FACS. These times were then entered into the computer so that the EEG coincident with these expressions could be extracted.

Artifact-free EEG during facial expressions of happiness and disgust was Fourier transformed to yield measures of power density. As noted above, power density in the alpha frequency band was of prime interest, and it is these data that will be reported below. Based on previous findings from our laboratory and other laboratories reviewed above, we hypothesized greater relative right hemisphere activation in anterior regions during

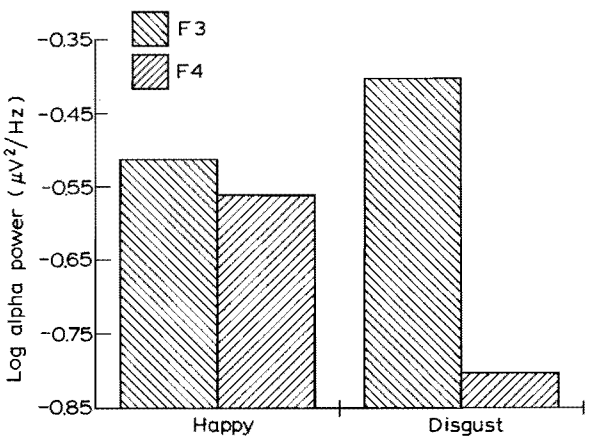

Fig. 1. Mean log-transformed alpha-power (in μV^2/Hz) from the left and right mid-frontal regions (F3 and F4) during facial signs of happiness and disgust. From Davidson et al. (in press).

facial expressions of disgust compared to facial expressions of happiness. As shown in Fig. 1, the data supported our hypothesis. In the mid-frontal region, assessed by the F3 and F4 electrode sites in the international 10–20 system, disgust periods were associated with greater right-sided activation (i.e., less alpha power) than happy periods. The pattern of greater right-sided activation during disgust compared with happiness was also found in the anterior temporal region. That no significant between-condition differences were found for the central and parietal regions underscores the regional specificity of the effects observed.

Fig. 2 displays frontal asymmetry scores (log right minus log left alpha power) from the happy and disgust periods for individual subjects. Two aspects of this figure are noteworthy. First, every subject shows a lower score during disgust expressions than happy expressions. Thus, *100%* of the subjects showed more relative right-sided frontal activation during disgust compared with happy periods. The second important feature of these data is the pronounced individual variability in the absolute magnitude of the asymmetry scores. Some subjects show highly positive asymmetry scores overall, denoting tonic left-sided frontal activation, while other subjects show highly negative asymmetry scores denoting overall right-sided

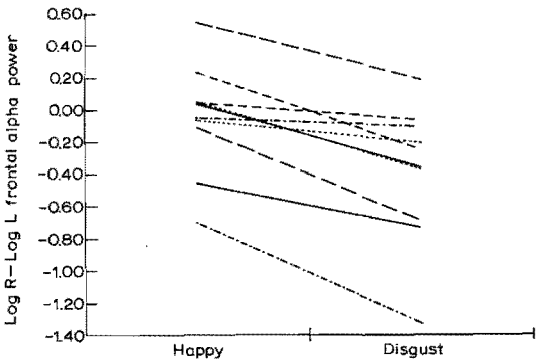

Fig. 2. Frontal asymmetry laterality scores (log right – log left alpha power) for individual subjects during facial signs of happiness and disgust. Higher numbers on this index denote greater relative left-sided activation. Note that every subject shows a higher score during happiness compared with disgust. Based upon data from Davidson et al. (in press).

frontal activation. In other words, the between-condition differences in asymmetry (i.e., between happy and disgust) are superimposed upon widely varying individual differences in the overall magnitude and direction of asymmetry. As will be considered in the next section of this paper, these individual differences in tonic asymmetry may be related to important dimensions of affective style.

In order to examine whether the procedures we used to verify the presence of an emotion (i.e., facial expression) actually made an important difference in uncovering patterns of asymmetry, we performed the type of analysis which is more typical in research on the psychophysiology of emotion. We simply compared all artifact-free EEG epochs extracted from the positive film clips with the comparable epochs extracted from the negative film clips. The epochs used for this analysis were selected irrespective of the subject's facial behavior. While the means were in the expected direction for the frontal leads, with the negative film clips producing more right-sided activation compared with the positive clips, we found no significant differences between these conditions on any of the measures of asymmetry. The lack of significant effects when the analyses were performed independent of facial behavior suggests that our method of using the facial expressivity to flag epochs of peak emotional state was effective. In this study, significant between-condition differences in asymmetry were obtained only when facial expression was used to verify the presence of emotional states. We should note, however, that previous studies in our laboratory and other laboratories (e.g., Ahern and Schwartz, 1985; Tucker et al., 1981) have found EEG differences between positive and negative emotional states without using facial expressive measures as an index of peak emotional intensity. Thus, the presence of discrete facial expressions of emotion does not appear to be a *necessary* condition for the emergence of hemispheric asymmetries relevant to emotion.

One line of research from our laboratory that has used both a facial-expressive strategy and the more traditional whole-epoch strategy is a series of

collaborative studies with Nathan Fox. Focusing on EEG asymmetries in infants, this research effort has been designed to document the early manifestations of cerebral asymmetries which underlie emotion. In our first study with this age group (Davidson and Fox, 1982), we presented 10-month-old infants with a videotape of an actress displaying laughter and distress while EEG was recorded from the left and right frontal and parietal regions. We found that the positive condition elicited more left-sided frontal activation than the negative condition. This finding was obtained in two separate samples of infants. In a more recent study, we (Fox and Davidson, 1986) tested newborn infants to determine whether this asymmetry was present at birth. Neonates were presented with tastes which differed in hedonic valence while brain electrical activity was recorded. We found that tastes which produced facial signs of disgust were associated with more right-sided activation compared with tastes which produced a more positive facial expression (sucrose). In this study, we found that the asymmetry difference between conditions was present in both the frontal and parietal regions. Whether this reflects the lack of functional differentiation in these cortical regions at this age is a question that must await additional research.

In more recent work with Fox (Fox and Davidson, 1988), we have studied brain electrical activity during the expression of different facial signs of emotion in 10-month-old infants. Emotion was elicited via the approach of a mother and a stranger. Of prime interest to us was a comparison between two types of smile. One of these involves activity in both the zygomatic muscle (cheek) *and* orbicularis oculi (around the eye) while the other involves activity only in the zygomatic region. The difference between these two smile types was first described by the French anatomist Duchenne (1862). Duchenne's work figured heavily in Darwin's 1872 book *The Expression of the Emotions in Man and Animals*. According to Darwin's discussion, Duchenne suggested that the emotion of 'frank joy' is accompanied by activity in both the zygomatic and orbicularis oculi muscles, while smiles not associated with felt happiness are accompanied only by activity in the zygomatic region. Ekman and Friesen (1982) provided the first modern empirical support for this proposal by showing that smiles involving activity in both of these facial regions were much more highly correlated with self-reports of happiness than smiles produced by activity in only the zygomatic region. In the light of this evidence, we were intrigued by the possibility that these two types of smile could be discriminated electrophysiologically.

Artifact-free EEG data were obtained on 19 infants, all born to two right-handed parents. EEG was recorded from the left and right frontal and parietal regions and quantified in the same manner as described above for the study with adults. Infants were exposed to episodes of both mother approach and stranger approach. Facial behavior and EEG were recorded in response to each episode. Facial behavior was coded with Ekman and Friesen's (1984) EM-FACS system.

We first computed the incidence of each of the two smile types in response to both stranger and mother approach. Seventy-five percent of the infants displayed smiles without orbicularis oculi activity to the stranger, while in response to mother approach 78% of the infants displayed smiles with orbicularis oculi activity. The difference in the frequency of occurrence of these two smile types is highly significant ($p < 0.005$). In other words, smiles indicative of felt happiness were more likely to occur in the situation in which genuine positive affect would be expected (mother approach), while a potentially threatening situation (stranger approach) was more likely to elicit 'unfelt happiness'. We also coded the duration of the two smile types, since Darwin (1872) suggested that the more 'genuine' smiles (i.e., those with orbicularis activity) were longer in duration compared with the other type of smile. Our results confirmed Darwin's (1872) suggestion: the mean duration of smiles with orbicularis activity was 2.39 seconds, while the mean duration of smiles without orbicularis activity was 1.49 seconds ($p < 0.01$). The central

question which we posed in this study was whether the two smile types could be discriminated on the basis of frontal brain electrical asymmetry. We specifically predicted that smiles with orbicularis activity would be accompanied by more relative left-sided frontal activation compared with the other smiles. As shown in Fig. 3, the data strongly supported this hypothesis. Consistent with the majority of our previous findings, regional specificity was once again indicated by the absence of significant differences in the parietal region (see Fig. 3).

We have recently examined EEG asymmetry during these two types of smile in adults (Ekman et al., in press). In this study, we were also able to examine the relationship between the duration of these smile types and self-reports of emotion. We found that higher intensities of self-reported amusement were associated with increased duration of smiles with orbicularis oculi ($r = 0.70$), while the duration of smiles without orbicularis activity were not associated with self-reports of amusement ($r = 0.14$). Most importantly, smiles with orbicularis activity were associated with significantly more left anterior activation than smiles lacking orbicularis activity. This pattern of anterior asymmetry is precisely the same found to discriminate between these two smile types in infants.

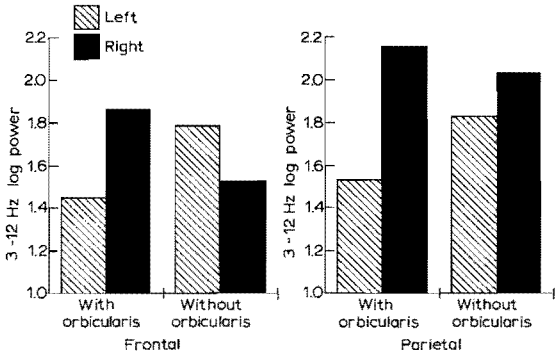

Fig. 3. Mean log-transformed band power (3 – 12 Hz in $\mu V^2/Hz$) for the left and right frontal and parietal regions during two types of smile in 10-month-old infants. One smile type includes activity in orbicularis oculi and the other does not. Adapted from Fox and Davidson (1988).

Anterior asymmetry, affective style and psychopathology

In recent years, we have begun exploring individual differences in resting anterior EEG asymmetry and its relation to emotional reactivity and psychopathology. We have adopted two converging strategies in our studies in this area. The first strategy involves comparing groups of subjects who differ on a known psychological or behavioral variable which theoretically should be related to anterior asymmetry. One of the most important variables we have examined is depression. The second strategy is to examine the relationship between individual differences in resting anterior activation asymmetry and affective reactivity at a later point in time in an unselected, normal cohort. Studies which represent each of these two approaches will be presented below.

Our first study in this area (Schaffer et al., 1983) was designed to determine whether patterns of resting frontal asymmetry would discriminate between depressed subjects and non-depressed subjects. Subjects for this study were selected on the basis of extreme scores on the Beck Depression Inventory. They were required to score at the extremes on two separate occasions separately by approximately 6 weeks. We then examined resting frontal and parietal EEG asymmetry using methods described above. As hypothesized, we found that the depressed subjects showed more relative right-sided frontal activation at rest compared with the non-depressed subjects. The difference between groups was primarily in the left frontal region, with the depressed subjects showing less activation.

We have recently replicated this study on a much larger sample size ($n = 23$ per group) and found the same pattern of results (Davidson et al., 1987). Depressed subjects were found to exhibit less relative left-sided frontal activation compared with non-depressed subjects. We also found that the depressed subjects showed less relative right-sided parietal activation at the same points in time. This pattern of parietal asymmetry was the same as we

found in the original study (Schaffer et al., 1983), but, given the small sample size in that first study, it was not significant. In the replication study, both the frontal and parietal asymmetry differences between groups were significant.

These findings indicate the pronounced specificity which typifies regional brain asymmetry. Simultaneously, the frontal and parietal regions showed opposite patterns of asymmetry. Clearly, hemispheres as a whole are not activated in a uniform fashion. Rather, different regions within a hemisphere may become very selectively activated. Our findings indicating EEG differences between depressives and non-depressives are also noteworthy because of their convergence with recent neurological data on the emotional sequelae of unilateral brain damage. Our finding of decreased left anterior EEG activation in depressives is paralleled by a recent finding by Robinson and his colleagues (Robinson et al., 1984) which indicates a very high correlation ($r = 0.92$) between the proximity of a lesion to the frontal pole within the left hemisphere (based upon CT scan evidence) and the severity of depressive symptomatology. Consistent with our data, in Robinson et al.'s study the closer the left hemisphere lesion to the frontal pole, the more severe was the depressive symptomatology. Our finding that depressives show less relative right-sided activation in posterior regions is also consistent with Robinson et al.'s data. In addition to reporting an association between left anterior lesions and depression, these authors observed that right posterior lesions were associated with depression.

Both our findings and those of Robinson et al. (1984) suggest that there may be two neuropsychological paths to depression: (1) deficits in left anterior activation, and (2) deficits in right posterior activation. The latter type of deficit would be expected to result in impairments in certain forms of visuo-spatial processing. We (Davidson et al., 1987) recently compared subclinically depressed subjects and non-depressed controls on psychometrically matched verbal and spatial tasks. The verbal task consisted of word-finding problems and the spatial task consisted of dot-localization problems. The tasks were matched on overall difficulty, the standard deviation of item difficulty, and internal consistency reliability. As predicted, the depressed subjects showed a selective deficit on the spatial task. No differences between groups were found for the verbal task. These findings confirm the suggestion in the literature that depression is associated with a selective impairment on right hemisphere cognitive tasks (for a review, see Silberman and Weingartner, 1986). Some investigators (e.g., Willner, 1985) have speculated that the right hemisphere cognitive performance deficit in depressives may underlie certain symptoms of depression, such as deficits in interpersonal skills and in the perception of non-verbal emotional information. For example, individuals with such a deficit may show impairments in the recognition of facial expressions and intonation patterns which specify emotion.

One important question to which we have recently turned is the degree to which the differences between depressed and non-depressed subjects in resting asymmetry are state-independent. That is, does the pattern of asymmetry characteristic of depression vary as a function of the clinical status of the individual, or is it a trait marker that pre-dates the onset of depression and/or remains stable despite changes in clinical status? In a recent study, we (Henriques and Davidson, in press) have addressed this question by comparing two groups of subjects who were currently non-depressed. One group ($n = 9$) had a past history of major or minor depression according to Research Diagnostic Criteria (Spitzer et al., 1978), as assessed by the Schedule for Affective Disorders and Schizophrenia (SADS). All subjects in this group had been free from depressive symptoms for at least one year and free of all psychoactive medication for at least one month. We compared this group to a group of non-depressed subjects who had never been depressed ($n = 11$). Subjects in this latter group were additionally required to have an absence of psychiatric illness in their first-degree relatives. The groups did not differ in age, sex distribution or socioeconomic

status. Most importantly, the groups did not differ in the current depression, as assessed both by the Beck Depression Inventory (M for past depressed group = 1.67 (SD = 2.25); for never depressed group = 1.43 (SD = 2.15)) and the Hamilton Depression Rating Scale (M for past depressed group = 1.16 (SD = 1.60); for never depressed group = 1.75 (SD = 1.49)).

EEG was recorded during an eyes-open and an eyes-closed baseline period from 14 scalp locations. In this study, we derived the EEG with three different reference montages. Since the major effects were significant with each reference montage, only the data from the averaged ears reference will be presented here*. No differences in asymmetry were found between the two baseline types and the data were combined across them. As we predicted, the differences in asymmetry that we found in previous studies to differentiate between acutely depressed and non-depressed subjects were present in this sample when we compared previously depressed and never depressed subjects. Specifically, there was a significant ($p < 0.05$) Group × Hemisphere interaction for the mid-frontal region (F3/F4 electrode sites)**. The previously depressed group showed less activation in the left frontal region compared with the never depressed group. The trend in the parietal region was opposite to that observed in the frontal region, although this difference did not reach significance. This pattern of decreased left frontal and decreased right parietal activation can be easily observed in a topographic difference map. Fig. 4 presents the difference between the mean alpha power data for the never depressed subjects and the previously depressed subjects (i.e., never depressed – previously depressed). In this figure, lighter shading indicates greater activation (i.e., less alpha power). As can be seen from this topographic map, the never depressed subjects show more activation in both the left frontal and right posterior regions compared with the previously depressed subjects.

These results indicate that patterns of asymmetry characteristic of depression (decreased relative left frontal activation and decreased relative right parietal activation) remain even when depres-

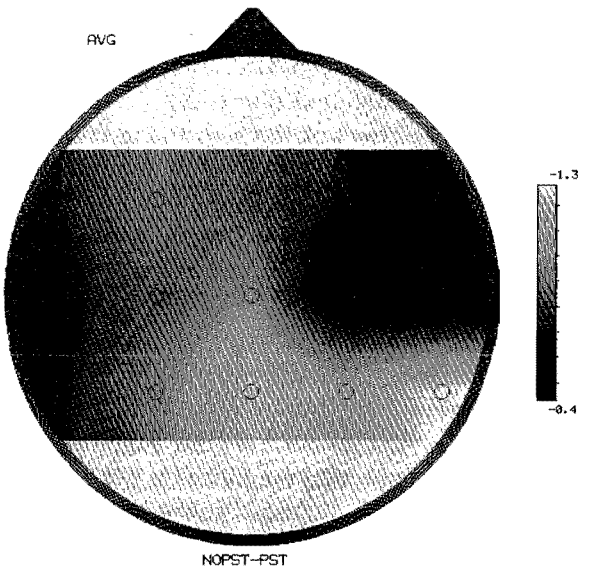

Fig. 4. Topographic map displaying the difference in mean log-transformed alpha power between never depressed and past depressed subjects. Lighter shading denotes areas where the never depressed subjects have more activation (i.e., less alpha power) than the past depressed subjects. Actual electrode locations used in producing the map are denoted by the circles. From Henriques and Davidson (in press).

* We developed the averaged ears reference to eliminate a potential problem with the more traditional linked ears reference. A number of electrophysiologists (e.g., Nunez, 1981) have suggested that physically linking the ears provides a low-resistive shunt across the head which attenuates the magnitude of observed asymmetry since it forces the two sides of the head to be isoelectric. Some researchers have empirically found that the magnitude of observed asymmetry is attenuated with a linked ears reference compared with other reference montages (e.g., Van Petten and Kutas, 1988). We developed a procedure which eliminates this possibility yet also permits us to reference to the two ears. We record each electrode referred to vertex (Cz). In addition, we record two channels of Cz, one referred to A1 (left ear) and the other referred to the right ear (A2). We then average these two channels and use this average of the two ears to re-compute each channel with respect to this new reference.

** The mid-frontal region is measured by activity in the F3 and F4 electrode sites. These are sites which we have previously found to differentiate between acutely depressed and non-depressed subjects. In addition, they were the sites used in the studies described in the first part of this chapter on state-dependent changes in frontal asymmetry in response to affect elicitors.

sion is remitted. In turn, these findings suggest that 'depressogenic' asymmetry patterns may be a *state-independent* marker that indexes risk for depression. Clearly, to more comprehensively test this hypothesis, a prospective design is required in which subjects are classified on the basis of asymmetry patterns and followed-up over time. We would predict that, relative to comparison groups, a higher proportion of subjects who demonstrate decreased left anterior activation and right posterior activation would develop subsequent psychopathology. We will be conducting such a study in the future.

Although we have not yet conducted a prospective, longitudinal study on the development of psychopathology, we have recently conducted several studies which indicate that patterns of resting asymmetry can indeed *predict* affective reactions at a later point in time. These studies have been conducted with both infant and adult samples. We have completed two studies to date in adults (Tomarken et al., 1989). These examined the relationship between resting baseline asymmetry measures obtained at the beginning of an experimental session and subjects' subsequent reactivity to short emotional film clips.

The design of each of the two studies was similar, although the subject samples were entirely independent and the film clips used in each study were different. In the first study, 24 right-handed females were assessed. Resting measures of EEG asymmetry from a 30-second eyes-open period were recorded at the beginning of the session. Subjects were then presented with two positive film clips, followed by two negative film clips. The positive clips were designed to elicit happiness and amusement and the negative film clips were designed to elicit fear and disgust. The duration of each film clip was approximately 2 minutes. Following each baseline and film trial, subjects were asked to rate their emotional experience during the clip on a series of 0–8 rating scales.

The major issue addressed in this study was the relationship between the resting measures of asymmetry (log right minus log left alpha power) taken at the start of the session and subjects' subsequent reports of their emotional responses to the film clips. We found that relative right frontal activation at rest significantly predicted fear responses to each of the two film clips ($r = -0.45$ and -0.43, $p < 0.05$) and composite (i.e., mean) fear responses across the two clips ($r = -0.48$, $p < 0.025$). Correlations between frontal asymmetry and disgust responses were not significant, although trends in the predicted direction were evident. The results of several additional sets of analyses were also revealing. First, the correlations between resting anterior asymmetry and fear responses were not simply due to the confounding effects of pre-existing mood at the time of the baselines. Resting EEG was uncorrelated with all individual mood scales assessed at the time of the baseline. In addition, it was uncorrelated with composite mood indices (e.g., composite negative affect). Moreover, when baseline mood was partialed out, correlations between frontal asymmetry and fear responses remained significant and almost identical in magnitude to their zero-order values (e.g., partial r between frontal asymmetry and composite fear $= -0.47$, $p < 0.05$). Thus, consistent with the notion that anterior asymmetry is a state-independent marker indexing affective predispositions, it predicted fear responses in this sample independent of concurrent mood state.

A second, revealing set of analyses focused on the relationship between anterior asymmetry and both positive and negative affective responses. In this sample, although the correlation was in the predicted direction, frontal asymmetry did not significantly predict happiness responses to positive films ($r = 0.18$). However, for both fear and disgust, frontal asymmetry was significantly correlated with the *difference* between negative affective responses to negative films and positive affective responses to positive films. The correlation between resting asymmetry and the disgust – happiness difference score was -0.41 ($p < 0.05$) and the correlation between resting asymmetry and the fear – happiness difference score was -0.59 ($p < 0.01$). This important finding suggests that resting

anterior asymmetry may primarily index the individual's *relative balance* of positive and negative affective response tendencies.

That anterior asymmetry significantly predicted the *difference* between positive and negative affective responsivity also suggests that it does *not* index generalized emotional reactivity. This is an important observation given recent findings by Diener and his colleagues. This research group has identified stable individual differences in affective reactivity that are independent of the valence of emotion (e.g., Diener et al., 1985; Larsen et al., 1986). To conduct a more direct test of a generalized affective reactivity interpretation of frontal asymmetry, we computed what might be considered a 'generalized reactivity' index consisting of the *sum* of subjects' negative affective responses to negative films and positive affective responses to positive films. For both fear and disgust, frontal asymmetry was uncorrelated with this index (fear + happiness $r = 0.10$; disgust + happiness $r = 0.09$). These data indicate that it is not affective reactivity per se that is indexed by anterior asymmetry but a valence-specific bias in reactivity.

One additional set of results in this study is also noteworthy. Specifically, for all analyses conducted, resting asymmetry in parietal sites was unrelated to any measure of affective reactivity (e.g., zero-order r between parietal asymmetry and composite fear = 0.13). This observation once again underscores the regional specificity of the relationship between resting asymmetry and emotional responsivity.

In the second study in this series (Tomarken et al., 1989; Study II), subjects were 15 right-handed females. As in the first experiment, EEG was recorded during a 30-second eyes-open resting baseline, after which subjects completed a set of 0–8 scales assessing current mood. Subjects then watched two positive and six negative film clips selected from contemporary movies (e.g., The Godfather) and rated their emotional responses immediately after each clip. Film clips were different from those used in Experiment 1. EEG was recorded from three sets of anterior sites: mid-frontal (F3/F4), lateral frontal (F7/F8) and anterior temporal (T3/T4). In addition, EEG was recorded from two posterior sites: posterior temporal (T5/T6) and parietal (P3/P4).

We computed correlations between resting EEG asymmetry and composite fear and disgust responses across the four films which elicited at least moderate fear and disgust and we computed correlations between resting asymmetry and happiness ratings across the two films which elicited at least moderate happiness. As in the first experiment, greater relative right hemisphere activation in the mid-frontal site was associated with greater fear ($r = -0.35$), although this correlation was not significant due to the small sample size ($n = 14$). Furthermore, resting asymmetry in both the mid-frontal and lateral frontal sites was highly correlated with disgust responses to negative films ($r = -0.62$ and -0.68, respectively; $p < 0.025$). In addition, although resting frontal asymmetry did not predict happiness responses, anterior temporal asymmetry was significantly correlated with composite happiness ratings ($r = 0.52$), in the direction hypothesized (greater relative left anterior temporal activation associated with increased happiness).

As in Experiment 1, resting anterior asymmetry predicted affective responses independent of concurrent mood at the time of the baselines. Resting asymmetry was either uncorrelated with baseline mood, or correlated in a direction opposite to that which would be expected. Furthermore, when baseline mood was partialed out, correlations remained significant, and either identical or actually somewhat greater in magnitude (e.g., $r = 0.71$ between anterior temporal asymmetry and happiness). Finally, as in the previous experiment, EEG asymmetry in posterior sites failed to significantly predict any affective responses to films.

Although there were some inconsistencies across the two studies (e.g., the significant prediction of fear but not disgust in Experiment 1, and the reversal of this pattern in Experiment 2), the findings from these two studies indicate that in normal, unselected cohorts of adults, resting frontal asym-

metry can predict positive and negative responses to affect elicitors. We have recently extended these findings and conclusions in a study examining the relationship between individual differences in resting asymmetry and affective reactivity in infants (Davidson and Fox, 1989). In this study, resting EEG asymmetry was measured from 14 female 10-month-old infants prior to their exposure to a brief episode of maternal separation. The episode was 60 seconds in duration unless the infant was judged by the experimenter to be extremely upset, at which point the episode was terminated. We focused on individual differences in response to this stressor, since several researchers have noted the pronounced differences among infants of this age in response to maternal separation (e.g., Shiller et al., 1986; Weinraub and Lewis, 1977). Moreover, infants' response to maternal separation at this age period is one component of a constellation of behaviors that are associated with individual differences in vulnerability to distress. This is a dimension of temperament for which impressive longitudinal stability has been demonstrated (for a review, see Kagan, 1984).

From the videotaped record of the session, we coded infants' responses to the maternal separation challenge. Examination of these responses revealed that 7 infants cried and 7 did not cry. An infant was classified as a non-crier only if she showed no evidence of crying for the entire duration of the maternal separation episode. The classification of an infant as a crier or non-crier was done prior to any EEG analysis and was therefore completely blind.

In accord with our previous findings, we predicted that infants who responded with distress to maternal separation would show more relative right frontal activation during a baseline assessment of EEG than infants who were not distressed by the situation. As indicated by Fig. 5, infants who cried in response to maternal separation did in fact show greater right-frontal and less left-frontal activation at rest than the non-criers. Consistent with our previous findings in adult subjects, parietal asymmetry recorded at the same time as the frontal asymmetry measures failed to discriminate between groups.

In order to examine the consistency of the group difference in frontal asymmetry on an individual subject basis, we computed a laterality difference score for each subject (log right minus log left power). As before, higher numbers on this metric indicate more relative left-sided activation. As shown in Fig. 6, every one of the criers fell below the mean score for the non-criers and every one of the non-criers fell above the mean score for the criers. Moreover, all but one of the criers had absolute right-sided frontal activation.

One concern which might be raised about these findings is that the difference in asymmetry between the two groups might simply reflect differences in pre-existent mood, with the criers perhaps in a more irritable mood at the time of the resting baselines. In order to obtain data relevant to this issue, we coded infants' facial behavior during the resting baseline according to Izard's MAX

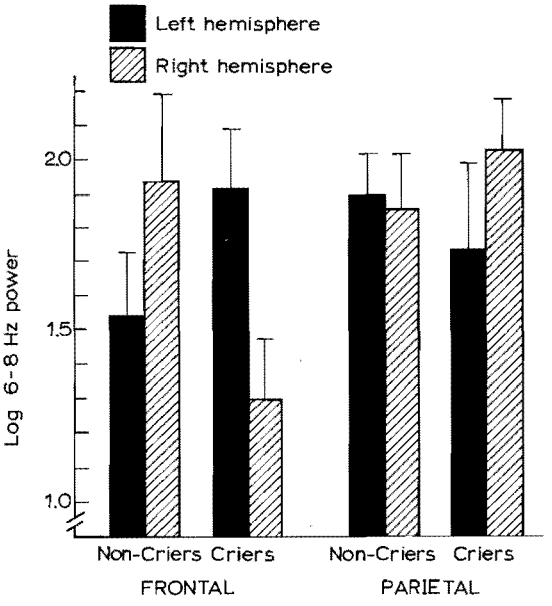

Fig. 5. Mean log-transformed band power (6–8 Hz) for the resting baseline period in the left and right frontal and parietal regions for criers and non-criers. Decreases in 6–8 Hz power are indicative of increases in activation. From Davidson and Fox (1989).

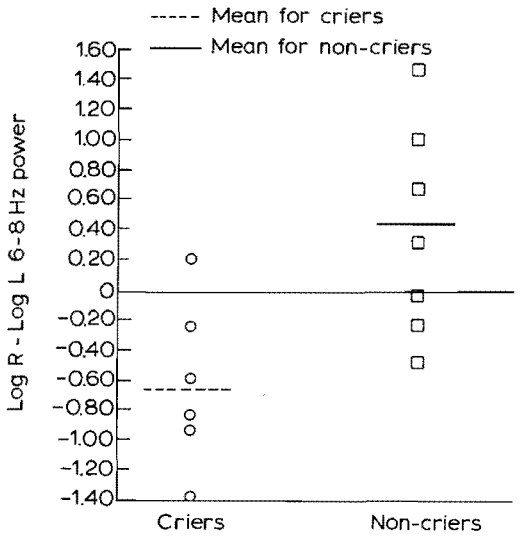

Fig. 6. Frontal asymmetry laterality scores (log right – log left power) for individual infants, split by which infants subsequently cried or did not cry in response to a brief episode of maternal separation. The means for each group are denoted by the lines. Note that all the criers fell below the mean of non-criers and all the non-criers fell above the mean of the criers. Based upon data from Davidson and Fox (1989).

TABLE 1

Facial signs of emotion during the baseline period

	Criers		Non-criers
Interest	M	9.5	11.3
	SD	8.6	7.8
No expression	M	17.0	15.1
	SD	8.2	8.9
Joy/surprise	M	2.4	3.2
	SD	2.9	3.2
Negative affect	M	0.5	1.4
	SD	0.9	1.3

Mean duration in seconds of facial affect for criers (n = 6) and non-criers (n = 7) during the baseline period. The 'no expression' category represents the mean number of seconds during which no facial signs of emotion were present. The 'negative affect' category represents the mean number of seconds during which facial signs of any of the negative emotions (anger, fear, distress, sadness and disgust) were expressed. From Davidson and Fox (1989).

system. Table 1 presents the duration in seconds of different facial signs of emotion during the baseline period. As indicated by this table, on all facial signs of emotion, those infants who subsequently cried in response to maternal separation failed to differ from those who did not cry. While certainly not definitive, these findings do suggest that the two groups of infants were not in a different emotional state at the time that the EEG measures were recorded.

In sum, we have conducted two adult studies and one infant study, each of which assessed the relationship between resting EEG asymmetry and subsequent affective responses to an emotion elicitor. In each study, we found evidence that resting asymmetry recorded from anterior sites can predict affective responses to emotion elicitors. Furthermore, in each study, anterior asymmetry predicted affective responses independent of subjects' emotional state at the time of the baselines. Taken together, these two sets of findings suggest the following conclusions: (1) anterior asymmetry is a state-independent marker indexing the individual's readiness or predisposition for affective responsivity, and (2) this readiness is only released when the individual is exposed to a sufficiently potent affect elicitor. Finally, in each of these three studies, resting EEG asymmetry in posterior sites did not significantly predict subsequent affective responses. Thus, regional specificity is indicated.

We should note that our findings on the relationship between resting *anterior* asymmetry and affective responsivity parallel our previous results (Davidson et al., 1979), and those of other investigators (e.g., Furst, 1976; Glass and Butler, 1977), which indicate a relationship between resting asymmetry in *posterior* sites and performance on cognitive tasks. This evidence indicates that resting asymmetries recorded from parietal or posterior temporal sites predict performance on verbal and spatial cognitive tasks in a manner consistent with neuro-anatomical specialization for these functions (e.g., greater relative left hemisphere activation associated with better performance on verbal tasks). Similar findings have been

obtained in studies assessing the functional significance of individual differences in cerebral blood flow (e.g., Dabbs and Chou, 1980; Gur and Reivich, 1980). In addition, Levy and her colleagues have found strong relationships between behavioral indicators of hemispheric arousal and performance on verbal and non-verbal cognitive tasks (Levy et al., 1983). As Levy (1983) has argued, these findings support a distinction between hemispheric *specialization* for particular cognitive functions and hemispheric *activation*. In this view, diversity among individuals in activation may be '. . . superimposed on a relatively invariant pattern (Levy, 1983, p. 476) . . .,' in specialization, and account for individual differences in cognitive performance.

By the same token, variation among subjects in resting *anterior* asymmetry may be superimposed on invariant patterns of hemispheric specialization for emotion. One pattern of specialization has been revealed by the studies that we reviewed in the earlier section on EEG responses during experimentally induced affective states. These studies revealed that negative affective states were associated with relative right anterior activation and that positive affective states are associated with relative left anterior activation. Our findings on individual differences in resting anterior asymmetry suggest that diversity among individuals in resting activation may account for differences in affective reactivity that occur despite invariant patterns of affective specialization.

One possible conceptualization of the effects of such individual differences is that they represent differences in *thresholds* for affective responsivity. According to such a view, individuals with relative right anterior activation have a relatively low threshold for negative affective responses to affect elicitors, while individuals with relative left anterior activation have a relatively high threshold. Thus, a more potent affective stimulus may be required to trigger negative affect in the latter group, relative to the former. The converse would be expected in the case of reactivity to positive affective elicitors.

This conceptualization is consistent with recent evidence that individual differences in emotion or temperament are linked to altered thresholds for perception of, or responses to, affective stimuli (for a review, see Derrybery and Rothbart, 1984). Our interpretation is also consistent with infrahuman research indicating that activation of neural structures known to regulate important aspects of emotion does in fact produce altered thresholds for significant stimuli. An example of such threshold adjustment is the finding that an increase in electrical current administered to the lateral hypothalamus of a rat produces a *downward* shift in the concentration gradient of sugar water that rats are willing to consume (Stellar et al., 1979). Gallistel (1980a, 1980b) has reviewed infrahuman evidence of this sort, and has eloquently described the process of threshold adjustment as one of *selective potentiation and depotentiation* of lower-order effectors by higher-order centers (e.g., the hypothalamus) that regulate behavior in motivational contexts.

In future studies, we hope to test the threshold adjustment notion more directly by assessing the relationship between resting anterior asymmetry and responses to affective stimuli that vary parametrically in intensity. In addition, consistent with the argument that a process of threshold adjustment may underlie mood-congruent biases and other effects of emotions on cognitive processes (e.g., Derrybery and Rothbart, 1984; Esposito, 1984), we plan to assess whether resting anterior asymmetry predicts selective processing of positive and negative affective stimuli in cognitive tasks.

An additional important question addressed by current, on-going research is the stability of anterior asymmetry over time. Simply put, if resting anterior asymmetry does in fact index individual differences in affective predispositions and/or risk for psychopathology, it should be a stable attribute of the individual. Previous evidence has indicated that individual differences in resting EEG asymmetry recorded from posterior sites are highly stable over a 1–3 week period (Ehrlichman and Wiener, 1979; see also Amochaev

and Salamy, 1979). The stability of posterior EEG asymmetry is probably a specific feature of the more general stability of the spectral composition of the resting EEG (e.g., Gasser et al., 1985; Mocks and Gasser, 1984). Unfortunately, to our knowledge, no studies have as yet assessed the stability of EEG asymmetry recorded from anterior sites. As a result, we are currently conducting a study designed to assess the test-retest stability of resting EEG asymmetry in adults over the course of a 3 – 4-week period. In addition, we will assess whether it is specifically those subjects with both *stable* and *extreme* patterns of asymmetry who are most likely to demonstrate notable affective responses to stimuli. For instance, are those subjects with stable, extreme right anterior activation most likely to respond with negative affect to emotion elicitors?

Future research directions

An additional goal of our current and anticipated future work is relevant to research both on EEG patterns during experimentally elicited affective states and on the affective correlates of individual differences in resting asymmetry. This goal is to clarify the underlying dimensional basis of affective asymmetry. Previously, Davidson (1984) and Kinsbourne (1978) have speculated that anterior asymmetries reflect a fundamental neuro-anatomical asymmetry in the control of approach and withdrawal behaviors. This speculation is consistent with the evidence that approach and withdrawal are fundamental components of motivation (e.g., Schneirla, 1959), and that this dichotomy is a useful framework for a functional analysis of neural structures implicated in important aspects of motivation and emotion (e.g., Depue and Iacono, 1989; Glickman and Schiff, 1967; Stellar et al., 1979; Stellar and Stellar, 1985; Wise and Bozarth, 1987).

Our findings to date are at least broadly consistent with the notion that anterior asymmetries for emotion assess the relative balance of approach and withdrawal tendencies. For example, studies reviewed above have shown that experimentally induced disgust (Davidson et al., in press; Fox and Davidson, 1986) is associated with relative right frontal activation and that resting right anterior activation predicts subsequent fear and disgust responses to affect elicitors (Tomarken et al., 1989). These results are notable given that both fear and disgust are commonly associated with *withdrawal* from eliciting stimuli (cf. Davidson et al., in press; Marks, 1987). Indeed this may be the major commonality between the two emotions.

Our findings indicating a linkage between frontal asymmetry and depression (e.g., Schaffer et al., 1983; Henriques and Davidson, in press) may also be consistent with the notion that anterior asymmetry indexes the relative balance of approach and withdrawal motivation. From this perspective, in accord with several recent conceptualizations of affective disorders (e.g., Depue and Iacono, 1989; Higgins, 1987; Tellegen, 1985), depression may primarily index a deficit in approach motivation. In this regard, it is notable that difficulty initiating voluntary actions, psychomotor retardation, lowered energy and an apparent absence of reward-oriented motivation are all cardinal features of depression. It may well be that this set of symptoms co-occur because they all reflect disrupted functioning in neural structures that regulate approach-oriented motivation toward positive hedonic stimuli (cf. Depue and Iacono, 1989). Notably, in our data, the greatest differences between depressives and non-depressives have consistently been observed in the left frontal region, with depressives showing left frontal *hypo-activation*. It is also significant that lesions in the left frontal region most consistently result in depression in patients with unilateral cortical lesions (Robinson et al., 1984). Perhaps, then, the specific activation levels of the left anterior region reflect the strength of the individual's approach motivation.

In future research, we hope to test these speculations by assessing whether a more fine-grained classification of patterns of anterior asymmetry can yield better prediction of discrete affective

responses. For example, is a pattern of left frontal hypo-activation selectively associated with sadness or depression, while right frontal hyper-activation is selectively associated with fear and disgust? This possibility would be consistent with the model of the dimensional structure of emotion recently advanced by Tellegen and his associates (e.g., Tellegen, 1985; Watson and Tellegen, 1985). They have characterized fear and disgust as heightened negative affect and sadness and depression as lowered positive affect.

We also intend to test the approach-withdrawal model through experiments designed to forge a linkage between our research focusing on cortical asymmetries and that of other investigators studying the neurochemistry of approach and withdrawal systems. Dopamine is a neurotransmitter which has been directly implicated in reward motivation. Dopamine agonists have been found to increase self-stimulation behavior elicited from dopamine-rich sites in mesolimbic and mesocortical regions (see review by Kelly and Stinus, 1984). A variety of recent evidence has uncovered asymmetries in dopamine concentration and receptor density in both human and non-human species (see Tucker and Williamson, 1984, for review). For example, Glick and his associates (e.g., Glick and Shapiro, 1984) have reported higher concentrations of dopamine on the side contralateral to the direction in which rats habitually turn. Even more relevant to the research described in this chapter, Schneider et al. (1982) have found that the density of D2 dopamine receptors is 23% higher in the left compared with the right nigrostriatal region in rats. Alterations in the normal pattern of dopamine asymmetry may also be found in certain forms of psychopathology. For example, Reynolds (1983; Reynolds and Czudek, 1987) has reported significant differences in the concentration of dopamine in the left and right amygdala of schizophrenic patients based upon post-mortem examination of brains. In the light of the documented importance of dopamine to positive affect, reward and approach motivation and animal findings suggestive of a left-sided lateralization of dopamine in at least some brain regions, we believe that the examination of the relationships between dopamine levels, emotion and electrophysiological measures of asymmetry is warranted. We are currently addressing this issue by studying Parkinson's patients who exhibit cyclic changes in motor function *and* mood over the course of a medication cycle. Such patients have been labeled as exhibiting 'on-off' phenomena (e.g., Cantello et al., 1986; Girotti et al., 1986). We are measuring changes in EEG asymmetry in patients of this type over the course of several medication cycles. In addition to the electrophysiological measures, we are also carefully assessing changes in affect using both self-report and behavioral measures. Finally, both the electrophysiological and the behavioral changes will be related to cyclic changes in levels of dopamine and its metabolites that we assay from repeated plasma samples.

Because they are completely non-invasive, relatively inexpensive and possess excellent time resolution, quantitative electrophysiological methods are ideally suited to the study of emotion. We have illustrated how such methods can be used to make inferences about regional patterns of brain asymmetry which are associated with different emotional states and, when they occur in the resting state, predict trait-like characteristics of emotional reactivity. In the future, the combined use of brain electrical activity measures and behavioral measures of emotional expression in patients with well-defined neurological disorders offers great promise in furthering our understanding of the brain systems which underlie human emotional behavior.

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References

Ahern GL, Schwartz GE: Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. *Neuropsychologia: 23,* 745–756, 1985.

Alford LB: Localization of consciousness and emotion. *Am. J. Psychiatry: 12,* 789–799, 1933.

Amochaev A, Salamy A: Stability of EEG laterality effects. *Psychophysiology: 16,* 242–246, 1979.

Carmon A, Nachson I: Ear asymmetry in the perception of emotional non-verbal stimuli. *Acta Psychol.: 37,* 351–357, 1973.

Cantello R, Gilli M, Ricco A, Bergamasco B: Mood changes associated with 'end-of-dose deterioration' in Parkinson's disease: a controlled study. *J. Neurol. Neurosurg. Psychiatry: 49,* 1182–1190, 1986.

Cummings JL, Mendez MF: Secondary mania with focal cerebrovascular lesions. *Am. J. Psychiatry: 141,* 1084–1087, 1984.

Dabbs JM, Chou G: Left-right carotid blood flow predicts specialized mental ability. *Neuropsychologia: 18,* 711–713, 1980.

Darwin C: *The Expression of the Emotions in Man and Animals.* Chicago: University of Chicago Press, 1872/1965.

Davidson RJ: Affect, cognition and hemispheric specialization. In Izard CE, Kagan J, Zajonc R (Editors), *Emotion, Cognition and Behavior.* New York: Cambridge University Press, 1984.

Davidson RJ: EEG measures of cerebral asymmetry: Conceptual and methodological issues. *Int. J. Neurosci.: 39,* 71–89, 1988.

Davidson RJ, Fox NA: Asymmetrical brain activity discriminates between positive versus negative affective stimuli in human infants. *Science: 218,* 1235–1237, 1982.

Davidson RJ, Fox NA: Cerebral asymmetry and emotion: developmental and individual differences. In Molfese DL, Segalowitz SJ (Editors), *Brain Lateralization in Children: Developmental Implications.* New York: Guilford Press, pp. 191–206, 1988.

Davidson RJ, Fox NA: Frontal brain asymmetry predicts infants' response to maternal separation. *J. Abnorm. Psychol.: 98,* 127–131, 1989.

Davidson RJ, Schwartz GE, Saron C, Bennett J, Goleman DJ: Frontal versus parietal EEG asymmetry during positive and negative affect. *Psychophysiology: 16,* 202–203, 1979a.

Davidson RJ, Taylor N, Saron C: Hemisphericity and styles of information processing: individual differences in EEG asymmetry and their relationship to cognitive performance. *Psychophysiology: 16,* 197, 1979b.

Davidson RJ, Schaffer CE, Saron C: Effects of lateralized stimulus presentations on the self-report of emotion and EEG asymmetry in depressed and non-depressed subjects. *Psychophysiology: 22,* 353–364, 1985.

Davidson RJ, Chapman JP, Chapman LJ: Task-dependent EEG asymmetry discriminates between depressed and non-depressed subjects. *Psychophysiology: 24,* 585, 1987.

Davidson RJ, Ekman P, Saron C, Senulis J, Friesen W: Approach/withdrawal and cerebral asymmetry. Emotional expression and brain physiology, I. *J. Pers. Soc. Psychol.:* in press.

Davidson RJ, Chapman JP, Chapman LP, Henriques JB: Asymmetrical brain electrical activity discriminates between psychometrically-matched verbal and spatial cognitive tasks. Submitted for publication, 1989.

Denenberg VH, Yutzey DA: Hemispheric laterality, behavioral asymmetry and the effects of early experience in rats. In Glick SD (Editor), *Cerebral Lateralization in Nonhuman Species.* New York: Academic Press, pp. 109–133, 1985.

Derryberry D, Rothbart MK: Emotion, attention and temperament. In Izard CE, Kagan J, Zajonc RB (Editors), *Emotion, Cognition and Behavior.* New York: Cambridge University Press, pp. 132–166, 1984.

Depue RA, Iacono WG: Neurobehavioral aspects of affective disorders. In Rosenzweig MR, Porter LY (Editors), *Annual Review of Psychology.* Palo Alto: Annual Reviews, Inc., pp. 457–492, 1989.

Diener E, Larsen RJ, Levine S, Emmons RA: Intensity and frequency: dimensions underlying positive and negative affect. *J. Pers. Soc. Psychol.: 48,* 1253–1265, 1985.

Duchenne B: *Mechanisme de la physionomie humaine ou analyse electrophysiologique de l'expression des passions.* Paris: Bailliere, 1862.

Ehrlichman H, Wiener MS: Consistency of task-related EEG asymmetries. *Psychophysiology: 16,* 247–252, 1979.

Ekman P: Expression and nature of emotion. In Scherer KR, Ekman P (Editors), *Approaches to Emotion.* Hillsdale, NJ: Lawrence Erlbaum Associates, pp. 319–343, 1984.

Ekman P, Friesen WV: Felt, false and miserable smiles. *J. Nonverbal Behav.: 6,* 238–252, 1982.

Ekman P, Friesen WV: *Emotion Facial Action Coding System (EM-FACS).* San Fransisco: University of California, San Fransisco, 1984.

Ekman P, Friesen WV, Ancoli S: Facial signs of emotional experience. *J. Pers. Soc. Psychol.: 39,* 1125–1134, 1980.

Ekman P, Davidson RJ, Friesen WV: The Duchenne smile. Emotional expression and brain physiology, II. *J. Pers. Soc. Psychol.:* in press.

Esposito RU: Cognitive-affective integration: some recent trends from a neurobiological perspective. In Weingartner H, Parker ES (Editors), *Memory Consolidation: Psychobiology of Cognition.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1984.

Etcoff NL: The neuropsychology of emotional expression. In Goldstein G, Tarter RE (Editors), *Advances in Clinical Neuropsychology, Vol. 3.* New York: Plenum Press, pp. 127–179, 1986.

Fox NA, Davidson RJ: Taste-elicited changes in facial signs of emotion and the asymmetry of brain electrical activity in human newborns. *Neuropsychologia: 24,* 417–422, 1986.

Fox NA, Davidson RJ: Patterns of brain electrical activity during facial signs of emotion in ten month old infants. *Dev. Psychol.: 24,* 230–236, 1988.

Furst CJ: EEG asymmetry and visuospatial performance. *Nature: 260,* 254–255, 1976.

Gainotti G: Emotional behavior and hemispheric side of lesion. *Cortex: 8,* 41–55, 1972.

Gallistel CR: *The Organization of Action: A New Synthesis.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1980a.

Gallistel CR: From muscles to motivation. *Am. Sci.: 68,*

398 – 409, 1980b.
Gasser T, Bacher P, Steinberg H: Test-retest reliability of spectral parameters of the EEG. *Electroencephalogr. Clin. Neurophysiol.: 60,* 312 – 319, 1985.
Girotti F, Carella F, Grassi MP, Soliveri P, Marano R, Caraceni T: Motor and cognitive performances of Parkinsonian patients in the on and off phases of the disease. *J. Neurol. Neurosurg. Psychiatry: 49,* 657 – 660, 1986.
Glass A, Butler SR: Alpha EEG asymmetry and speed of left hemisphere thinking. *Neurosci. Lett.: 4,* 231 – 235, 1977.
Glick SD, Shapiro RM: Functional and neurochemical asymmetries. In Geschwind N, Galaburda AM (Editors), *Cerebral Dominance: The Biological Foundations.* Cambridge, MA: Harvard University Press, pp. 147 – 166, 1984.
Glick SD, Shapiro RM: Functional and neurochemical mechanisms of cerebral lateralization in rats. In Glick SD (Editor), *Cerebral Lateralization in Nonhuman Species.* New York: Academic Press, pp. 157 – 183, 1985.
Glickman SE, Schiff BB: A biological theory of reinforcement. *Psychol. Rev.: 74,* 81 – 109, 1967.
Goldstein K: *The organism.* New York: Academic Press, 1939.
Gur RC, Reivich M: Cognitive task effects on hemispheric blood flow in humans: evidence for individual differences in hemispheric activation. *Brain Lang.: 9,* 78 – 92, 1980.
Heilman KW, Scholes R, Watson TR: Auditory affective agnosia: disturbed comprehension of affective speech. *J. Neurol. Neurosurg. Psychiatry: 38,* 69 – 72, 1975.
Henriques JB, Davidson RJ: Asymmetrical brain electrical activity discriminates between previously depressed subjects and healthy controls. *J. Abnorm. Psychol.:* in press.
Higgins ET: Self-discrepancy: a theory relating self and affect. *Psychol. Rev.: 94,* 319 – 340, 1987.
Kagan J: *The Nature of the Child.* New York: Basic Books, 1984.
Kelly A, Stinus L: Neuroanatomical and neurochemical substrates of affective behavior. In Fox NA, Davidson RJ (Editors), *The Psychobiology of Affective Development.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1984.
Kinsbourne M: The biological determinants of functional bisymmetry and asymmetry. In Kinsbourne M (Editor), *Asymmetrical Functions of the Brain.* New York: Cambridge University Press, 1978.
Kolb B, Milner B: Observations on spontaneous facial expression after focal cerebral excisions and after intracarotid injection of sodium amytal. *Neuropsychologia: 19,* 505 – 514, 1981.
Larsen RJ, Diener E, Emmons RA: Affect intensity and reactions to daily life events. *J. Pers. Soc. Psychol.: 51,* 803 – 814, 1986.
Leventhal H, Tomarken AJ: Emotion: today's problems. In Rosenzweig MR and Porter LY (Editors), *Annual Review of Psychology,* Vol. 37. Palo Alto: Annual Reviews, Inc., pp. 565 – 610, 1986.
Levy J: Individual differences in cerebral hemisphere asymmetry: Theoretical issues and experimental considerations. In Hellige JB (Editor), *Cerebral Hemisphere Asymmetry: Method, Theory and Application.* New York: Praeger, 1983.
Levy J, Heller W, Banich MT, Burton LA: Are variations among right-handed individuals in perceptual asymmetries caused by characteristic arousal differences between the hemispheres? *J. Exp. Psychol. Hum. Percept. Performance: 9,* 329 – 359, 1983.
Ley R, Bryden M: Hemispheric differences in processing emotions and faces. *Brain Lang.: 7,* 127 – 138, 1979.
Marks IM: *Fears, Phobias and Rituals.* New York: Oxford University Press, 1987.
Mocks J, Gasser T: How to select epochs of the EEG at rest for quantitative analysis. *Electroencephalogr. Clin. Neurophysol.: 58,* 89 – 92, 1984.
Natale M, Gur RE, Gur RC: Hemispheric asymmetries in processing emotional expressions. *Neuropsychologia: 21,* 555 – 565, 1983.
Nauta WJH: The problem of the frontal lobe: a reinterpretation. *J. Psychiatric Res.: 8,* 167 – 187, 1971.
Nunez PL: *Electric Fields of the Brain: The Neurophysics of EEG.* New York: Oxford University Press, 1981.
Perris C, Monakhov K: Depressive symptomatology and systemic structural analysis of the EEG. In Gruzelier J, Flor-Henry P. (Editors), *Hemisphere Asymmetries of Function in Psychopathology.* New York: Elsevier/North Holland, 1979.
Reuter-Lorenz P, Davidson RJ: Differential contributions of the two cerebral hemispheres to the perception of happy and sad faces. *Neuropsychologia: 19,* 609 – 613, 1981.
Reuter-Lorenz P, Givis RP, Moscovitch M: Hemispheric specialization and the perception of emotion: evidence from right-handers and from inverted and non-inverted left-handers. *Neuropsychologia: 21,* 687 – 692, 1983.
Reynolds GP: Increased concentrations and lateral asymmetry of amygdala dopamine in schizophrenia. *Nature: 305,* 527 – 529, 1983.
Reynolds GP, Czudek C: Neurochemical laterality of the limbic system in schizophrenia. In Takahashi R, Flor-Henry P, Gruzelier J, Niwa S (Editors), *Cerebral Dynamics, Laterality and Psychopathology.* Amsterdam: Elsevier, pp. 451 – 456, 1987.
Robinson RG, Benson DF: Depression in aphasic patients: frequency, severity and clinical-pathological correlations. *Brain Lang.: 14,* 282 – 291, 1981.
Robinson RG, Kubos KL, Starr LB, Rao K, Price TR: Mood disorders in stroke patients: importance of location of lesion. *Brain: 107,* 81 – 93, 1984.
Robinson RG, Szetela B: Mood change following left hemispheric brain injury. *Ann. Neurol.: 9,* 447 – 453, 1981.
Saxby L, Bryden MP: Left visual field advantage in children for processing visual emotional stimuli. *Dev. Psychol.: 21,* 253 – 261, 1985.
Sackeim HA, Weinman AL, Gur RC, Greenberg M, Hungerbuhler JP, Geschwind N: Pathological laughter and crying: functional brain asymmetry in the expression of positive and negative emotions. *Arch. Neurol.: 39,* 210 – 218, 1982.
Schaffer CE, Davidson RJ, Saron C: Frontal and parietal EEG asymmetries in depressed and non-depressed subjects. *Biol. Psychiatry: 18,* 753 – 762, 1983.
Schneider LH, Murphy RB, Coons EE: Lateralization of striatal dopamine (D2) receptors in normal rats. *Neurosci Lett.: 33,* 281 – 284, 1982.
Schneirla TC: An evolutionary and developmental theory of biphasic processes underlying approach and withdrawal. In Jones MR (Editor), *Nebraska Symposium on Motivation.* Lincoln: University of Nebraska Press, pp. 1 – 42, 1959.

Schwartz GE, Davidson RJ, Maer F: Right hemisphere lateralization for emotion in the human brain: interaction with cognition. *Science: 190,* 286–288, 1975.

Schwartz GE, Weinberger DA, Singer JA: Cardiovascular differentiation of happiness, sadness, anger and fear following imagery and exercise. *Psychosom. Med.: 43,* 343–364, 1981.

Shagass C: Electrical activity of the brain. In N.S. Greenfield NS, Sternbach RA (Editors), *Handbook of Psychophysiology.* New York: Holt, Rinehart and Winston, pp. 263–328, 1972.

Shiller VM, Izard CE, Hembree EA: Patterns of emotion expression during separation in the strange situation. *Dev. Psychol.: 22,* 378–383, 1986.

Silberman EK, Weingartner H: Hemispheric lateralization of functions related to emotion. *Brain Cognition: 5,* 322–353, 1986.

Spitzer RL, Endicott J, Robins E: Research diagnostic criteria: rationale and reliability. *Arch. Gen. Psychiatry: 35,* 773–782, 1978.

Starkstein SE, Robinson RG, Price TR: Comparison of cortical and subcortical lesions in the production of poststroke mood disorders. *Brain: 110,* 1045–1059, 1987.

Stellar JR, Stellar E: *The Neurobiology of Motivation and Reward.* New York: Springer-Verlag, 1985.

Stellar JR, Brooks FH, Mills LE: Approach and withdrawal analysis of the effects of hypothalamic stimulation and lesions in rats. *J. Comp. Physiol. Psychol.: 93* 446–466, 1979.

Suberi M, McKeever WF: Differential right hemispheric memory storage of emotional and non-emotional faces. *Neuropsychologia: 15,* 757–768, 1977.

Tellegen A: Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In Tuma AH, Maser J (Editors), *Anxiety and the Anxiety Disorders.* Hillsdale, NJ: Erlbaum, pp. 681–706, 1985.

Tomarken AJ, Davidson RJ, Henriques JB, Saron CD, Straus A, Senulis J: Anterior activation asymmetries predict emotional reactivity to film clips. In preparation, 1989.

Tucker DM: Lateral brain function, emotion and conceptualization. *Psychol. Bull.: 89,* 19–46, 1981.

Tucker DM, Frederick SL: Emotion and brain lateralization. In Wagner H, Manstead T (Editors), *Handbook of Psychophysiology: Emotion and Social Behaviour.* London: Wiley, in press.

Tucker DM, Williamson PA: Asymmetric neural control systems in human self-regulation. *Psychol. Rev.: 91,* 185–215, 1984.

Tucker DM, Watson RT, Heilman KM: Discrimination and evocation of affectively intoned speech in patients with right parietal disease. *Neurology: 27,* 947–950, 1977.

Tucker DM, Stenslie CE, Roth RS, Shearer SL: Right frontal lobe activation and right hemisphere performance decrement during a depressed mood. *Arch. Gen. Psychiatry: 38,* 169–174, 1981.

Van Petten C, Kutas M: The use of event-related potentials in the study of brain asymmetries. *Int. J. Neurosci.: 39,* 91–99, 1988.

Watson D, Tellegen A: Toward a consenual structure of mood. *Psychol. Bull.: 98,* 219–235, 1985.

Weinraub M, Lewis M: The determinants of children's responses to separation. *Monogr. Soc. Res. Child Dev.: 42,* (Serial No. 172).

Willner P: *Depression: A Psychobiological Synthesis.* New York: Wiley, 1985.

Wise RA, Bozarth MA: A psychomotor stimulant theory of addiction. *Psychol. Rev.: 94,* 469–492, 1987.