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Cerebral Asymmetry and Emotion: Conceptual and Methodological Conundrums

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A diversity of methods have been used to study cerebral asymmetries associated with emotion. Many different conceptual schemes have also been invoked to guide research on this topic. The purpose of this article is to survey the critical methodological and conceptual issues in this area of research. Research in this area must acknowledge the multi-componential nature of emotion. Asymmetries associated with the perception of emotional information and the posing of emotional expressions are not necessarily the same as those that accompany the actual production of emotion. Asymmetries vary along the rostral/caudal plane both in their magnitude and direction, as well as in their functional significance. Research in this area must explicitly take this variable into account. Different measures of asymmetry do not reflect the same underlying process and so cannot be used interchangeably. In particular, behavioural measures which lack extensive localising validation, must be used with caution. Finally, the nature of the causal connection between alterations in asymmetric activation and emotion is not a simple one and extant data indicate that an asymmetric shift is not sufficient for the production of emotion. This fact has serious implications for the types of experimental designs that must be used to adequately test for relations between cerebral asymmetry and emotion. The article concludes with a discussion of some of the major outstanding questions that will occupy a central position in the future research agenda in this area.

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INTRODUCTION

Research on emotion is burgeoning after many dormant years in the behavioural sciences. One of the most exciting and promising new areas in research on emotion is work on the underlying neural substrates of emotion (see LeDoux, 1987, for a general review). Within this general area, the study of cerebral laterilisation and emotion has assumed a position of major importance. Bearing witness to this claim is a recent volume dedicated entirely to this topic (Gainotti & Galtagirone, 1989), a large increase in the number of papers published on this topic within the past five years (for reviews see Davidson, in press; Davidson & Tomarken, 1989; Leventhal & Tomarken, 1986; Silberman & Weingartner, 1986), and the inclusion of chapters on cerebral asymmetry and emotion in several major volumes dedicated to modern emotion research (see e.g. Davidson, 1984; Heller, 1990). In addition, the literature on cerebral asymmetry and emotion has direct implications for research on the hemispheric substrates of affective disorders and other forms of psychopathology. Several recent volumes have appeared on this topic (e.g. Cutting, 1990; Flor-Henry & Gruzelier, 1983; Kinsbourne, 1988a; Takahasi, Flor-Henry, Gruzelier, & Niwa, 1987).

The growth of interest in the topic of cerebral asymmetry and emotion has been accompanied by a corresponding increase in the diversity of methods used to study their interrelations. Moreover, many different conceptual schemes have been used to guide research either implicitly or explicitly in this area. Often, scientists who survey the extant literature on this topic are struck by the inconsistencies in the conclusions that have been drawn and in the models invoked to organise the field (e.g. Gainotti, 1989; Gainotti, Caltagirone, & Zoccolotti, this issue). However, some of the inconsistency is more apparent than real because of failure to take account of important methodological and conceptual issues.

The purposes of this article are to survey briefly these issues and to propose a set of desiderata for research on this topic. I will first discuss the different components of emotion and urge that they be disentangled and not be treated in a uniform way. When the production of emotion (i.e. emotional experience) is the focus of study, I will underscore the need for procedures to verify the presence of the intended emotion. The heterogeneity of cerebral specialisation along the rostral-caudal plane will next be emphasised. This functional specialisation within a hemisphere will be shown to be critical in understanding the underlying hemispheric substrates of emotion. I will then turn to the various measures and methods that scientists have used to make inferences about the differential contributions of the two hemispheres to emotion and show that they are not equivalent and must not be assumed to reflect the same underlying process. The final major topic will be a consideration of the logical relation between patterns

of asymmetric brain activity and emotion. The question of whether particular patterns of asymmetric brain activity are necessary, sufficient or contributory causes of emotional phenomena will be addressed. The review concludes by suggesting that when these methodological and conceptual issues are taken into account, many of the inconsistencies in the literature disappear. However, many questions still remain and some of the most outstanding ones are presented at the conclusion of this article.

The Multi-componential Nature of Emotion

Although it may appear to be belabouring the obvious, it is important to underscore the differentiated nature of emotion subcomponents and avoid assuming that they share a common neural substrate. At grossest level, we can differentiate between the perception of emotional information and the production of emotion. The perception of emotional information might involve decoding facial or vocal expressive cues, whereas the production of emotion involves the actual generation of emotion in the subject.[1] Even though each of these broad classes of emotion-related behaviour can be further differentiated, I wish now only to emphasise the fact that even this distinction is not always made. Conclusions about the "hemispheric substrates of emotion" have occasionally been drawn on the basis of data primarily from the perceptual domain. A number of investigators have proposed that the anterior regions of the two cerebral hemispheres are differentially specialised for the *experience* of positive and negative emotions, with the left anterior region more activated during positive, approach-related emotion and the right more activated during negative, withdrawal-related emotion[2] (e.g. Davidson, 1984; Davidson, in press;

[1]It is true that the perception of emotional information might give rise to the production of emotion. However, one cannot assume that the perception of emotional information will necessarily give rise to the experience of emotion. In fact, it is likely that in most laboratory tasks of emotion perception, little actual emotion is generated in the subjects.

[2]The terms approach-related positive emotion and withdrawal-related negative emotion are specifically chosen to delineate the types of positive and negative emotion that are theoretically predicted to be associated with differential anterior lateralisation. Positive emotion that is approach-related is accompanied by action tendencies designed to reduce the distance between the organism and the source of positive stimulation. Negative emotion that is withdrawal-related is accompanied by action tendencies designed to increase the distance between the organism and the source of aversive stimulation. Disgust and fear are prototypic withdrawal-related negative emotions. On the positive side, we often fail to differentiate between approach and non-approach forms of positive affect. However, there are certain forms of positive affect that are unambiguously accompanied by approach-related action tendencies, such as when infants toward the end of the first year of life are approached by their mothers. In such situations, infants usually smile, reach toward their mothers with their arms and show increased left-sided frontal activation (e.g. Fox & Davidson, 1987, 1988).

Heller, 1990; Kinsbourne & Bemporad, 1984). These same investigators also agree that a different pattern of laterilisation is present for the perception of emotional information. Most studies support the conclusion that right posterior association regions are differentially specialised for the perception of emotional information, irrespective of the valence of the perceived emotion (see Davidson, 1984; Heller, 1990; Silberman & Weingartner, 1986, for reviews). Thus, data from the perceptual domain are not directly relevant to the hypothesis that select regions of the two cerebral hemispheres are differentially engaged during the experience of certain positive and negative emotions. Gainotti and colleagues (this issue), in marshalling evidence for their critique of the hypothesis of differential lateralisation for the experience of positive and negative emotion, invoke a large number of studies that did not involve actual emotion, either experimentally produced or naturally occurring. For example, many of the studies they (Gainotti et al., this issue) cited involve the perception of emotional information (e.g. Bowers, Bauer, Coslett, & Heilman, 1985; Duda & Brown, 1984; Etcoff, 1984; Hirshman & Safer, 1982; Suberi & McKeever, 1977), whereas other studies involve the posing of emotional expressions (e.g. Campbell, 1978). In neither case do the experimental subjects actually report experiencing any emotion whatsoever! The major point I wish to underscore is simply that data on the hemispheric substrates of emotional perception cannot be used to make inferences about the production of emotion. These two major components of emotional processing are likely to engage different neural substrates in a manner that is similar to Wernicke's and Broca's areas being differentially involved in speech perception and speech production, respectively.

There are two related issues that deserve emphasis. The first is that posed emotional expressions do not necessarily involve the same neural substrates as spontaneous expressions (see Hager & Ekman, 1983, for a review). Thus, data on posed expressions cannot be used to make inferences about the hemispheric substrates of either spontaneous expression or the production of emotion *per se*.

The second point is that if manipulations are performed to induce emotion in the laboratory, adequate procedures must be utilised to verify the presence of the intended emotion. It is not sufficient to assume that the intended emotion is produced simply because a specific stimulus has been presented. Nor is it sufficient to simply present group data to establish that on the average, a particular stimulus is reported to produce more emotion of a certain valence than a comparison stimulus (e.g. Borod, Koff, Lorch, & Nicholas, 1986). What is required is the use of idiographic verification methods. For each individual subject in each emotion condition, the investigator must verify that the intended emotion was actually produced. If the study is a complete within-subjects design, only those subjects who

experienced the intended emotion in each of the conditions would be retained for analysis. As an example, consider the methods used in one of our recent studies (Davidson, Ekman, Saron, Senulis, & Friesen, 1990) on the effects of film-elicited emotion on brain electrical asymmetries. In this experiment, subjects were presented with two short positive and two short negative film clips designed to elicit happiness and disgust, respectively. These clips had been used extensively in earlier emotion research (e.g. Ekman, Friesen, & Ancoli, 1980) and had been validated as effective elicitors of these emotions. In our study, we videotaped subjects unobtrusively while they viewed these clips. We also obtained ratings of their emotional experience immediately following each clip. To evaluate our major predictions, brain electrical activity was extracted for analysis based upon the presence of target facial expressions. During the positive clips, we extracted brain activity during the spontaneous display of facial signs of happiness, while during the negative clips we extracted brain activity during the spontaneous display of facial signs of disgust. In addition, we only included for analysis those trials (e.g. film clip periods) during which the subject reported the target emotion at a relatively high intensity and did not report the presence of any opposite valence emotion during the film clip (e.g. anger during the positive clips or amusement during the negative clips). The imposition of these strict verification criteria resulted in the elimination of more than half the original sample of subjects tested in the experiment. However, were the excluded subjects to be included in the analysis, we would no longer be comparing between relatively "pure" instances of happiness and disgust. And, when we examine the data in the absence of these verification criteria (i.e. we include all data from the positive and negative film periods, irrespective of facial expression) in the same restricted sample of subjects, no systematic difference in frontal asymmetry emerged between the positive and negative film clip conditions.

Rostral/Caudal Differences in Hemispheric Specialisation

As more precise methods have been used to make inferences about patterns of hemispheric activation, assumptions about global hemispheric activation are gradually being replaced by more differentiated claims about selective regional hemispheric activation. Modern research suggests that the whole hemisphere typically does not behave in a unitary fashion, but rather exhibits considerable regional specificity (see e.g. Wood, Flowers, & Naylor, 1991). In fact, activation asymmetries in rostral and caudal hemispheric regions have been reported to show *inverse* correlations in some contexts (Davidson, Schaffer, & Saron, 1985; Wood et al., 1991). These inverse correlations are based upon measurements of brain activity

in anterior and posterior cortical regions at the same points in time. Thus, an individual might show left frontal and right parietal activation simultaneously.

One source of evidence concerning the importance of rostral/caudal differences in hemispheric specialisation for emotion are the studies of unilateral stroke patients by Robinson and his colleagues (e.g. Robinson, Kubos, Starr, Rao, & Price, 1984). These investigators studied the relation between the location of a unilateral lesion based upon computerised tomography (CT) and magnetic resonance (MR) scan evidence and the severity of depressive symptomatology. They consistently found that among patients with unilateral left hemisphere lesions, those with more anterior lesions exhibit more severe depressive symptomatology (see Starkstein & Robinson, 1988, for a review).[3] Such specificity within a hemisphere is not unique to studies of individuals with brain damage. Studies of regional brain activation (assessed with quantitative electrophysiology and positron emission tomography: PET) in patients with psychiatric depression (e.g. Baxter et al., 1989; Henriques & Davidson, 1990, 1991; Martinot et al., 1990) clearly underscore the importance of the frontal region. These studies have reported that depressives show less activation than controls specifically in the left frontal region. Moreover, Baxter et al. (1989) found an inverse relation between glucose metabolism in the left dorsolateral frontal cortex and severity of depressive symptomatology, a finding recently replicated by Drevets et al. (1992b) using PET-O^{15} measures of regional cerebral blood flow. The more depressed the patient, the less left frontal glucose metabolic activity or blood flow was observed.

Consistent evidence is also available from studies of normal subjects. For example, individual differences in baseline measures of frontal activation asymmetry are related to the intensity of positive and negative affective responses to emotional challenges in both infants (e.g. Davidson & Fox, 1989) and adults (e.g. Tomarken, Davidson, & Henriques, 1990). In both of these studies, we demonstrated that individual differences in parietal asymmetry recorded from the same points in time were unrelated to measures of affective responding to the emotional challenges. In addition, we (Tomarken, Davidson, Wheeler, & Doss, 1992a) have found that

[3]Not all studies have found the same pattern of affective changes following unilateral lesions as the Robinson group has reported (see Gainotti, 1989, for a review). Failure to replicate this effect may arise from a multitude of causes. The most significant source of variability in findings related to the affective consequences of unilateral lesions is probably a function of failure to account for differences in premorbid history and environmental context. As I discuss in a later section, a lesion acts as a diathesis, which in interaction with the requisite environmental events and life stresses may culminate in affective psychopathology.

individual differences in frontal asymmetry in normal subjects are related to self-reports of dispositional positive and negative mood. Specifically, those subjects who exhibit extreme and stable left frontal activation report more positive and less negative dispositional mood compared with subjects who show the opposite pattern of frontal asymmetry. Measures of asymmetry from posterior scalp regions obtained at the same points in time are unrelated to subjects' reports of their mood. Finally, in several studies (Davidson et al., 1990; Sobotka, Davidson, & Senulis, 1992) we have demonstrated that when affect is experimentally manipulated among unselected normal subjects, withdrawal-related negative emotion is associated with a reliable shift toward greater right frontal activation compared with both a positive emotion elicitor and with activity at baseline. Simultaneous measures of asymmetry in posterior cortical regions do not reliably differentiate between such positive and negative emotion-arousing conditions.

There are several important consequences that follow from these observations. First, it is inappropriate to refer to hemispheric activation as if it were unitary and homogeneous. Secondly, behavioural measures which do not provide direct information on where within a hemisphere they might be mediated should be used with caution and with appropriate sensitivity to the fact that differential patterns of asymmetry that are opposite in direction may be present simultaneously.

These issues are particularly salient in the study of hemispheric substrates of emotion, because available evidence clearly suggests that rostral and caudal cortical regions contribute differentially to different subcomponents of emotion. The evidence briefly reviewed earlier suggests that activity in the frontal region is related to the experience/expression of emotion.[4] Simultaneous measures of activation asymmetries from posterior cortical regions are not reliably associated with either dispositional mood or phasically aroused emotion. However, asymmetries in certain posterior cortical regions, particularly the parietal region, appear related to the perception of emotional information, particularly facial expressions of emotion (Etcoff, 1986). The studies of asymmetrical parietal lobe involvement in the perception of emotional information have, for the most

[4]Other data implicate the anterior temporal region, in addition to the frontal region, in affective responding. For example, we have found that in response to experimentally aroused positive and negative affect, reliable shifts occur in the same direction in electrophysiological measures of frontal and anterior temporal activation (Davidson et al., 1990). In addition, research using PET measures of regional cerebral blood in response to experimentally aroused emotion also have uncovered reliable changes in the anterior temporal region (e.g. Reiman, Fusselman, Fox, & Raichle, 1989; but see a recent critique of this study by Drevets et al., 1992a).

part, suggested right hemisphere superiority for the perception of both positive and negative emotion (see reviews by Leventhal & Tomarken, 1986; Silberman & Weingartner, 1986). Thus, whereas the left and right frontal regions appear specialised for the processing of certain positive and negative emotions respectively, the right parietal region has been implicated in the perception of emotional cues irrespective of valence. This pattern of results underscores the importance of differentiating among subcomponents of emotion and suggests that the perception and experience of emotion may be mediated by different brain circuits, with the latter more rostrally located than the former.

Measures of Asymmetry: Are they Reflecting the Same Underlying Process?

Investigators have used a myriad of methods to make inferences about the differential involvement or activation of the two cerebral hemispheres. The methods range from rather gross behavioural measures of perceptual or motor biases to those that provide spatially detailed information on regional cerebral metabolism or blood flow. Although each of these methods has advantages and disadvantages, the most important issue when comparing the various methods is that different methods may reflect asymmetrical activity from different brain regions. This fact, in conjunction with the data reviewed earlier, which indicate that asymmetries in different cortical regions are often relatively independent and can even be inversely related, suggests that data derived from different methods are often not comparable and variability in patterns of asymmetry across methods should be expected. Thus, the fact that many investigators have reported left visual field advantages for the perception of emotional information irrespective of valence (e.g. Landis, Assal, & Perret, 1979; Ley & Bryden, 1979; see Etcoff, 1989, for a review) should not be taken to indicate a failure to confirm the hypothesis of differential lateralisation for the experience of positive and negative emotion. Most formulations of the hypothesis of differential lateralisation for positive and negative affect have focused on the production of emotion and have restricted their claims to anterior cortical regions (see Leventhal & Tomarken, 1986, for a review).

A large number of studies have appeared over the past decade on asymmetries in the facial expression of emotion. By literally measuring differences in visible (e.g. Sackeim & Gur, 1983) or covert (using electromyographic procedures) (e.g. Cacioppo & Petty, 1981; Schwartz, Ahern, & Brown, 1979) expression on each side of the face, investigators have made inferences about asymmetrical hemispheric contributions to emotion. The large increase in the number of studies measuring differences in visible facial asymmetry is probably a function of the simplicity with

which these measures can be obtained. Facial asymmetry can be easily assessed by photographing faces and constructing composites made of two left sides and two right sides. Judgements of differences in the intensity of emotion conveyed by each hemiface can then be obtained. Thus, this method offers the promise of providing a low-technology procedure for assessing putative differences in the contributions of two cerebral hemispheres to emotion.

A number of reviews of this literature have appeared over the past several years (e.g. Borod & Koff, 1984; Sackeim & Gur, 1983; Skinner & Mullen, 1991). I will concentrate here on only those methodological and conceptual issues which bear directly on the central concerns of this article. First is the problem of voluntary or posed vs. spontaneous emotional expressions. There is some suggestion that facial asymmetries are more pronounced in voluntary expressions than they are in spontaneous expressions (e.g. Hager & Ekman, 1983). In their recent meta-analysis of this literature, Skinner and Mullen (1991) found that when they directly compared spontaneous to posed emotional expressions, posed expressions were significantly more asymmetrical. However, the use of these labels implies that this distinction is categorical and can be clearly made. It is likely that there are gradations between the extremes of voluntary and spontaneous production and it is difficult to be certain what the relative contributions of each might be for any given expression. What is clear is that when a subject is aware of that her/his face is being observed, it is likely that some voluntary control of facial action will be present. A number of studies in the facial asymmetry area either did not use unobtrusive videotaping (e.g. Moscovitch & Olds, 1982; Wylie & Goodale, 1988) or do not specify whether subjects were aware of being videotaped or photographed (e.g. Borod et al., 1986; Buck & Duffy, 1980; Heller & Levy, 1981). A related problem occurs when attention is called to a subject's face, even if the subject is not explicitly aware that s/he is being videotaped or photographed. In one often-cited study of facial asymmetry during spontaneous emotion (Dopson, Beckwith, Tucker, & Bullard-Bates, 1984), subjects were required to fixate on a point located directly on a one-way mirror through which photographs were made. Exposing subjects to a mirror has been demonstrated to have profound effects on their behaviour which likely includes an increase in the extent to which facial expressions are voluntary regulated (e.g. Carver & Scheier, 1981).

Second is the issue of the intensity of the facial expression. If comparisons are being made between positive and negative facial expressions, it is desirable to have the intensities of the expressions matched. The *Facial Action Coding System* (Ekman & Friesen, 1978) contains codes for intensity of particular facial actions that comprise a number of frequently observed emotional expressions. These intensity codes can be used to

match facial expressions of different emotions on intensity. Alternatively, self-report measures can be used to match epochs of different emotion types on intensity (see Davidson et al., 1990, for examples). If systematic differences in intensity are present, any difference in facial asymmetry between the positive and negative expressions may be a product of the intensity difference between them rather than the qualitative emotional states that are expressed. In the Dopson et al. (1984) study, a massive difference in intensity between the positive and negative expressions was present.

Third is the problem of the neural control for facial action. Based upon clinical and experimental evidence, it appears that the neural circuits which mediate voluntary expression are different from those mediating more spontaneous expression (see reviews by Pizzamiglio, Caltagirone, & Zoccolotti, 1989; Rinn, 1984). It is also clear that not all facial regions are contralaterally controlled (Rinn, 1984). Contralateral control of the facial muscles is most apparent for the lower two-thirds of the face, yet the upper facial muscles play a crucial role in the definition of certain common facial expressions of emotion (e.g. happiness; see Ekman, Davidson, & Friesen, 1990). For those facial muscles that are clearly under contralateral hemispheric control, it is likely that cortical regions that lie within the motor strip are responsible. Accordingly, even in situations where facial behaviour is spontaneous and asymmetrical action occurs, it is likely reflecting asymmetrical cortical contributions from the motor region. Thus, it is possible for direct measures of regional cortical activation to show one pattern of frontal asymmetry and a different pattern of motor asymmetry simultaneously. In the best of circumstances, the presence of facial asymmetries during the spontaneous expression of emotion may only be reflecting asymmetries in the cortical control of the facial expression, localised in the motor strip. A different pattern of asymmetry may be present elsewhere within the hemispheres.[5]

In one of our recent studies referred to earlier (Davidson et al., 1990), we extracted brain electrical activity during the presence of objectively defined facial expressions of happiness and disgust. Videotaping of sub-

[5]It is also possible that asymmetrical facial action may be a function of peripheral asymmetries. Sackeim and Gur (1983) have reviewed the evidence on possible asymmetries in the physical characteristics of the face, such as muscle mass and fatty tissue deposits, and have concluded that asymmetries in these characteristics were minor and unlikely to contribute to visible asymmetries in facial action. However, one possibility not considered by these scholars is differential sensitivity of motorneurons on each side of the face to the same neural input. Thus, it is conceivable that facial asymmetries do not necessarily reflect asymmetrical cortical efferent control. However, much more research is required before we can accept or reject any of these hypotheses.

jects' facial behaviour was unobtrusive and subjects were completely unaware that their facial behaviour was being observed. As indicated in an earlier section, we found differences in measures of anterior activation asymmetry between happy and disgust expressions. However, when we examined the expressions themselves for asymmetry, we did not find that they differed. Although some individual expressions were slightly asymmetrical, there were no systematic differences in the asymmetry of happiness compared with disgust. These data directly indicate that brain electrical asymmetries may reliably differentiate between certain positive and negative expression periods, in the absence of any difference in the asymmetry of the facial expression itself.[6] In this study, we also examined asymmetries derived from scalp electrodes over the motor region. There were no significant differences in asymmetry in the motor region between the happy and disgust periods. *Both* happy and disgust expressions were associated with relative right-sided activation in the motor region. These data suggest that the neural control in the motor region for the expressive behaviour itself may be lateralised irrespective of valence. It is important to note that this pattern of motor asymmetry was accompanied by the *simultaneous* presence of differential lateralisation in frontal region for the happy and disgust periods. Equally relevant is the fact that asymmetries in physiological measures of regional hemispheric activation may be present in the complete absence of any facial expressions of emotion whatsoever. As noted in an earlier section, our research on individual differences in baseline measures of frontal asymmetry indicates that individual differences in these measures predict an individual's reactivity to affective challenges and dispositional mood. At the time that such baseline measures are obtained, virtually no facial behaviour is present (see e.g. Davidson & Fox, 1989; Tomarken et al., 1990). These data strongly argue against an assumption of a direct isomorphic relation between asymmetrical hemispheric activation and the presence of facial asymmetry.

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[6]Of course, it can always be claimed that the measures of facial asymmetry were insufficiently sensitive. If an electromyogram (EMG) were recorded from the left and right sides of the face, it is possible that much more subtle differences could be detected. This is indeed true and underscores the need for this type of multi-measure research to be performed. However, it should also be noted that by placing electrodes on the face, it is difficult *not* to call attention to the fact that the face is an object of study. This problem plagues all studies that use facial EMG as a measure. Although some investigators have concocted various rogues to mask the true purpose of the electrodes (see Cacioppo, Tassinary, & Fridlund, 1990, for a thoughtful discussion of this problem), it is difficult to evaluate their success independently.

Cerebral Asymmetry and Emotion: The Nature of the Causal Connection

What is the nature of the causal relation between activation asymmetries and emotion? There are two relevant domains of evidence that have been referred to earlier. One domain concerns the effects of the experimental arousal of emotion on measures of asymmetric hemispheric activation (e.g. Davidson et al., 1990). The other concerns the relation between individual differences in asymmetric hemispheric activation (either naturally occurring or lesion-produced) and emotional state, mood or psychopathology. From our own research, it appears that shifts in activation asymmetries as a result of the phasic arousal of emotion are superimposed upon relatively stable trait-like individual differences in asymmetric anterior activation (Davidson et al., 1990; Tomarken, Davidson, Wheeler, & Kinney, 1992b). It is in this latter domain of research where the causal connection between individual differences in asymmetric hemispheric activation and emotion requires clarification. Here, the independent variable is the pattern of asymmetric activation while the dependent variable is some measure of emotional state.

Explicating the nature of causal connection between individual differences in anterior asymmetry and emotion is especially relevant to interpreting the evidence on the effects of unilateral lesions on affective behaviour. Is a lesion-produced alteration in asymmetrical activation a sufficient cause for a change in affective behaviour? Is it a necessary cause? Just how this causal relation is conceptualised is critical in evaluating the extant literature, and in directing future research. I wish to argue here that asymmetrical anterior activation is not sufficient to produce a change in affective behaviour or experience. As I have mentioned elsewhere (e.g. Davidson & Tomarken, 1989; Davidson, 1992), basal measures of asymmetrical anterior activation are associated with an individual's vulnerability or propensity to experience certain positive or negative emotions, *given the requisite environmental elicitors*. This is an explicit diathesis/stress formulation where the diathesis is a particular pattern of anterior activation asymmetry. The "stress" in the model is simply meant to refer to any type of affective challenge.

According to the model we have formulated, an individual with hypoactivation in the left frontal region would be specifically vulnerable to depression in the presence of negative life events.[7] An already-depressed

[7]As I note in the next section, it is likely that early environmental stress can affect anterior activation asymmetry. Thus, the model allows for the possibility that negative life events accentuate right frontal activation and the latter, in turn, renders an individual more vulnerable to subsequent negative life events.

individual is one who was likely exposed to such negative events in the past and who possesses the requisite biological diathesis, left frontal hypoactivation (Henriques & Davidson, 1991).[8] On the other hand, an individual who is selected on the basis of showing left frontal hypoactivation would not necessarily be depressed because the frontal asymmetry difference is not sufficient to produce depression. In fact, when subjects are selected from a normal sample on the basis of left frontal hypoactivation, they do not report themselves to be significantly more depressed than their left frontally activated counterparts, although the former group does report more dispositional negative affect than the latter group (Tomarken et al., 1992a). Subjects with left frontal hypoactivation who are currently nondepressed would be expected to be more vulnerable to depression and to display more intense negative affect in response to negative affect elicitors than subjects with the opposite pattern of frontal asymmetry. Although we have not yet performed the requisite longitudinal studies to examine the long-term predictive validity of these measures for the development of affective disorders, we have established that individual differences in frontal asymmetry do predict affective responses to emotional film clips in three independent studies (Tomarken, Davidson, & Henriques, 1990; Wheeler, Davidson, & Tomarken, in press). Importantly, measures of emotion taken at the time baseline measures of brain activity were obtained do not differentiate between subjects with relative left vs. right frontal activation. These findings further support the idea that basal levels of frontal activation asymmetry reflect an individual's vulnerability or susceptibility to certain positive and negative emotions in response to specific environmental elicitors.

What is the import of this diathesis/stress model for studies of the affective sequelae of unilateral brain damage? The major implication is that considerable variability would be expected among patients with unilateral damage. Even if we restrict the sample to patients with unilateral frontal damage, not all patients with left-sided lesions would be expected to show depressive symptomatology and correspondingly, not all patients with right-sided lesions would be expected to show signs of inappropriate positive affect. Only those who have been exposed to particular negative or

[8]It is clear from the available evidence that a pattern of left frontal hypoactivation is not a necessary cause of depression. That is, some depressed individuals do not possess this biological diathesis (e.g. Henriques & Davidson, 1991). However, whether this pattern is a necessary cause of a specific subtype of depression is not known. There may be a subtype of depression, similar to melancholic depression, that is characterised primarily by a deficit in approach behaviour and an inability to experience pleasure. We have hypothesised that left frontal hypoactivation may be a necessary cause of this specific subtype of depression, but clearly not a sufficient cause.

positive events would be expected to show a difference in dispositional affective state. Our model explicitly recognises the possibility that individuals with left frontal lesions might be totally nondepressed. In such cases, we would expect that few, if any, negative life events have occurred. Furthermore, such individuals may be buffered from possible negative consequences by having unusually good networks of social support (e.g. Cohen & Wills, 1985). Thus, without adequate information on negative life events (including past history of depression) and social support, it is difficult to evaluate putative failures to replicate the observation that patients with left frontal damage are more prone to depressive symptomatology (see Gainotti, 1989, for a review).[9]

A useful strategy adopted by some investigators has been to probe affective reactivity in unilateral brain-damaged patients by exposing them to emotionally evocative stimuli and assessing their behavioural response (e.g. Borod et al., 1986; Mammucari et al., 1988). Unfortunately, several methodological limitations prevent any firm conclusions from being drawn from the Borod et al. study. These experimenters attempted to elicit emotion by presenting slides for 6 seconds with the experimenter sitting alongside the subject. Brief presentations of static slide images are not likely to elicit very strong emotion reactions. The manner in which expressive behaviour was measured was highly subjective and not based upon coding discrete facial actions. Moreover, the presence of the experimenter next to the subject maximises the degree to which subjects' knew they were being observed. Mammucari et al. (1988) used mostly well-validated film clips to elicit positive and negative emotion and measured facial behaviour unobtrusively with an established objective procedure. Interestingly, these investigators found the major difference between right and left brain-damaged subjects to be in response to the negative film clip, with the right brain-damaged subjects showing significantly less gaze aversion compared with both a normal control group and a left brain-damaged group. Precisely why the largest group difference appeared on the gaze aversion measures and not in facial expression itself is difficult to know from this one study. Perhaps with other types of negative emotional film clips group

[9]It must also be noted that damage to other brain regions may contribute directly or indirectly to the development of depression. A number of investigators have observed that damage to right posterior association cortex is often associated with depression (e.g. Finset, 1988; Robinson et al., 1984). Damage in this region may render an individual more vulnerable to a subtype of depression different from that produced by left frontal damage. Kinsbourne (1988b) has speculated that in severely depressed individuals, both of these regions (i.e. left frontal and right posterior) may be underactivated. Damage to yet other brain regions may also contribute indirectly to depression by simply adding additional stress to an individual's life.

differences in facial expressions of emotion might be present. It is interesting to note, however, that the difference between groups appeared in the one behavioural manifestation of withdrawal (gaze aversion) that was possible for subjects to produce in this constrained situation.

Although this finding is consistent with our general formulation, it is surprising that differences in positive expressions were not found between the left and right lesion groups. However, the overall incidence of happy facial expressions[10] was very low in this study. Across all three groups, the percentage of subjects who displayed at least one happy expression in response to either of the two positive film clips presented ranged between 10% and 22%. This figure is considerably lower than we have found in previous research using one of the same positive film clips (Davidson et al., 1990; Ekman et al., 1990). Thus, it was difficult to perform meaningful statistical comparisons. But, inspection of Mammucari et al.'s (1988) data on responses to the one positive film clip that has been extensively used in prior emotion research shows that the percentage of left brain-damaged subjects showing a positive emotional expression in response to the clip was less than half of the percentage of right brain-damaged subjects showing a positive expression to the same clip. Normal controls showed a pattern of response to this clip that was very similar to the right brain-damaged subjects. Although not significant, this is the pattern we would expect to obtain in response to a clip that elicited relatively pure positive affect. The failure to confirm this difference statistically between the two brain-damaged groups is likely a function of a lack of power.

The issue addressed in this section has important implications for the design of research strategies to investigate emotional differences between patients with different patterns of unilateral lesions, or in normal individuals who differ in asymmetric anterior cortical activation. The conceptual model of causal relations between anterior asymmetry and emotion that is proposed holds that individual differences in anterior asymmetry are not sufficient causes of emotional differences among individuals. Thus, a simple comparison of dispositional mood among patients who differ in the

[10]Mammucari et al. (1988) were careful to differentiate between smiles that included the upper face action (contraction of the muscles surrounding the eye which produce wrinkles at the external canthi) and those that consisted solely of lower face, i.e. contraction of the zygomatic muscles. It is only the former that have been consistently found to be associated with the felt experience of positive affect. In previous research with Ekman (Ekman, Davidson, & Friesen, 1990), we have labelled this type of smile the "Duchenne smile" to give appropriate historical credit to the anatomist Duchenne (1862/1990) who first commented on the difference between these smile types and speculated that only those that included the eye muscle component were frank signs of joy. My analysis of differences among the groups in the Mammucari et al. (1988) study is based upon Duchenne smiles only.

lateralisation of a lesion is not an adequate test of differential lateralisation for certain positive and negative emotions. The required strategy is exemplified by Mammucari et al. (1988) and by the studies conducted in our laboratory (e.g. Davidson & Fox, 1989; Tomarken et al., 1990; Wheeler et al., in press) where relations between individual differences in anterior asymmetry (either naturally occurring or lesion-produced) and affective reactivity in response to well-constructed probes are assessed. In addition, longitudinal studies of subjects who differ in anterior asymmetry will also be very informative. Such studies can assess differences among such subjects as they confront real life events. To date, such studies have yet to be performed.

Some Major Unanswered Questions: The Search for Proximal and Distal Causes

Among the most important questions concerning the differential anterior lateralisation for emotion is the reason for this asymmetry. It is useful to differentiate between proximal and distal causes. The distal causes of this asymmetry are apt to be a function of both genetic influences and early environmental effects. Although the necessary studies have not yet been performed, it is probable that there will be some heritable contribution to individual differences in anterior asymmetry. We (Rickman & Davidson, 1991) have reported moderate correlations (in the 0.4 range) between anterior activation asymmetry in parents and their offspring, consistent with the presence of heritable influences. However, this study did not permit the separation of heritable and environmental influences, so the findings are merely suggestive. Equally significant is the likelihood of robust environmental influences on affective lateralisation. Although the requisite data in humans are not available, animal studies do indicate that prenatal stress produces profound changes in the lateralisation of catecholamine asymmetry (Fride & Weinstock, 1988). In addition, early handling in rats has been found to induce massive changes in cerebral lateralisation inferred on the basis of behavioural deficits produced by unilateral lesions (e.g. Denenberg & Yutzey, 1985).

Although the question of distal cause will ultimately involve the complex interaction of genetic and early environmental factors, the study of the proximal causes of anterior lateralisation for emotion will necessarily provide important clues to mechanism. Such studies should help us understand why particular regions of the two hemispheres may be differentially sensitive to certain positive and negative incentive conditions as well as why certain individuals have greater activation in one or the other hemisphere.

Over the past several years, we have begun to embark upon a series of studies designed to address the issue of proximal cause. Our experiments to date have raised more questions than they have answered, but a brief description of what we have studied and found will, we hope, illustrate some promising directions for future research.

One of our first efforts in the analysis of proximal causes was to examine the possibility that individual differences in frontal asymmetry were associated with anatomical differences in the brain. We (Davidson, Wheeler, & Doss, in prep.) performed magnetic resonance imaging (MRI) scans on subjects who had been selected on the basis of their extreme and stable frontal asymmetry. These subjects were selected from an original sample of 90 right-handed individuals and consisted of those subjects who fell in the upper or lower 25% of the distribution of frontal asymmetry on two occasions separated by approximately three weeks (see Tomarken et al., 1992b, for details of the electrophysiological assessment). We quantified measures of frontal and occipital width, a measure of the asymmetrical shape of the head (plagiocephaly; see Myslobodsky et al., 1989) and measures of skull thickness in the region of the frontal electrodes. On none of these measures did any of the differences between the left and right frontally activated subjects approach statistical significance. The results of this study therefore implied that the differences between individuals who show extreme asymmetrical frontal activation are functional and not structural.

Another strategy we are currently pursuing in our quest to understand the proximal causes of the frontal asymmetry is to develop an animal model of this phenomenon. We (Davidson, Kalin, & Shelton, in press a) are studying brain electrical asymmetries in rhesus monkeys in response to pharmacological probes. In this way, we can begin to develop insights, albeit indirectly, into possible neurochemical mediators of the frontal asymmetries. In stressed monkeys, we found that diazepam changes frontal asymmetry from a right to a left activated pattern (Davidson, et al., in press a). This finding is consistent with a study performed with humans that found increased relative left-sided frontal cerebral blood flow in response to diazepam (Mathew et al., 1985). These data imply that benzodiazepines have a greater effect in the left compared with right frontal region. This asymmetrical effect may arise as a function of fewer benzodiazepine receptors in the right frontal region compared with the homologous left hemisphere region. The decreased number of receptors on the right side may be in part responsible for the greater susceptibility of the right frontal region to negative incentives. This speculation is a specific variant of the more general claim that a decreased density of benzodiazepine receptors may be associated with increased dispositional negative affect. Although such relations have not yet been examined at the human level, they have

been investigated with animal models of fearfulness. Maudsley reactive rats who have been bred for high fearfulness have been found to show a decreased number of benzodiazepine receptors in a number of different brain regions (Robertson, Martin, & Candy, 1978). Unfortunately, these investigators did not examine the two hemispheres separately.

We (Davidson, Kalin, & Shelton, in press b) have also found that the magnitude of the left-sided shift in frontal activation produced by diazepam in these monkeys is strongly associated with the animal's propensity to freeze in response to a novel challenge. The greater the amount of freezing behaviour, the greater was the increase in left frontal activation in response to diazepam. The precise mechanism underlying this effect is not currently known. It may be that the most fearful animals (i.e. those that show the most freezing) have the largest receptor asymmetry, with relatively few benzodiazepine receptors in the right frontal cortex compared with the left. Such a receptor pattern would be associated with a greater benzodiazepine effect in the left compared with the right hemisphere.

Although these are merely preliminary findings from our first study of this kind, they nevertheless point toward new avenues of research in the study of the neurochemical mechanisms that underlie the frontal asymmetry for emotion. It will be of interest to examine possible asymmetries in benzodiazepine receptor densities in intact humans using a positron-labelled benzodiazepine receptor antagonist (Persson et al., 1985). It will also be important to examine the contributions of other neurochemical systems, as several have been found to be distributed asymmetrically (see Tucker & Williamson, 1984, for a review).

Many complex questions remain. Even though the evidence reviewed in this article and elsewhere (Davidson & Tomarken, 1989; Starkstein & Robinson, 1991) clearly suggests that the dorsolateral frontal cortex is involved in the regulation of emotion, there is also abundant evidence of the involvement of this region in higher cognitive function (see Fuster, 1989, for a review). A major unanswered (and mostly unasked) question is how these two domains are related. Are the functions traditionally assigned to the dorsolateral frontal cortex (e.g. short-term memory and temporal organisation of behaviour; Fuster, 1991) also involved in emotional processing? Does the persistance of emotion following the offset of a brief emotionally provocative stimulus depend upon the dorsolateral frontal cortex? This region may also play an important role in response preparation for action tendencies associated with emotion.

Another major question concerns the precise nature of the difference in affective responding between left and right frontally activated subjects. Although we have established that such subjects differ in their dispositional positive and negative mood and in intensity of positive and negative affect in response to emotional stimuli, we do not know more specifically in

which stage of affective processing the individual differences are most apparent. For example, such subjects may differ primarily in their threshold for the elicitation of positive and negative emotion. On this view, left frontally activated subjects may require a less intense positive emotional stimulus to induce positive affect. Alternatively, left and right frontally activated subjects may not differ in the threshold for eliciting positive and negative affect, but they may differ in the duration of emotion once it is elicited. Right frontally activated subjects may have a more persisting negative emotional response compared with left frontally activated subjects. Such a difference in the ability to turn off an emotion once it is initiated may critically depend upon the dorsolateral frontal cortex.

It is clear that many questions remain regarding the causes of anterior lateralisation for emotion. As research on the proximal causes of this asymmetry continue, more mechanistic accounts of the underlying biological substrates of this anterior asymmetry will be forthcoming. These developments will facilitate increased contact between research on the neuropsychology of emotion, and other neurobiological studies of emotion and affective disorders.

SUMMARY AND CONCLUSIONS

In this article, I have reviewed theory and evidence bearing on cerebral asymmetry and emotion. Theories concerning differential lateralisation for certain positive and negative emotions or approach and withdrawal behaviour specifically refer to the production of these behavioural states (e.g. Davidson, 1992; Kinsbourne, 1978). It is therefore critical to differentiate among the various subcomponents of emotion. Research on cerebral asymmetries related to the perception of emotion, or to the posing of facial expressions in the absence of produced emotion, although interesting in its own right, does not directly bear on the issue of the neural circuits involved in the production of emotion. A related point concerns the importance of rostral/caudal differences in hemispheric specialisation. Different regions within a hemisphere are not specialised for the same functions, nor do they tend to become activated simultaneously. Many different measures have been used to make inferences about differential hemispheric involvement in particular emotional states ranging from measures of visibly observed facial asymmetry to regional glucose metabolism assessed with positron emission tomography. These measures reflect different underlying processes and should not be treated as if they were reflecting the same basic underlying brain state. It is especially important when using behavioural measures to understand that such measures may be reflecting activity more in certain hemispheric regions than others. Apparent inconsistencies between findings with different measures may reflect the fact that they are

sensitive to activity in different brain regions. The nature of the causal connection between alterations in asymmetric activation and emotion was also considered. The proposal was made that alterations in asymmetry act as diatheses which alter the probability for the production of particular emotion states, given the requisite environmental elicitors. According to this model, lesion-induced alterations in asymmetric activation are not *sufficient* for the production of particular emotional states or moods. Rather, they alter the probability that a particular emotional state would occur in response to environmental challenge. Without the challenge, we would not expect a change in emotional state to arise. This perspective helps to make sense of the many apparent inconsistencies in the literature on the emotional consequences of unilateral brain damage. Finally, some significant questions for future research were discussed. The types of studies that are needed to identify the proximal and distal causes of this anterior emotional asymmetry were described.

When the methodological and conceptual issues identified in this article are taken into account, the literature on cerebral asymmetry and emotion is remarkably consistent (for reviews see Davidson, 1992; Davidson & Tomarken, 1989; Leventhal & Tomarken, 1986; Silberman & Weingartner, 1986) in showing left-sided anterior activation during certain positive emotional states and traits and right-sided anterior activation (or left-sided hypoactivation) during certain negative emotional states and traits. Having said this, I hasten to add that many significant questions and problems remain. Among the most important for future research to address is the underlying neurobiological mechanisms which give rise to asymmetric anterior activation.

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