We are IntechOpen, the world’s leading publisher of Open Access books
Built by scientists, for scientists

4,200 Open access books available
116,000 International authors and editors
125M Downloads

154 Countries delivered to
TOP 1% Our authors are among the most cited scientists
12.2% Contributors from top 500 universities

WEB OF SCIENCE™
Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com
Chapter 2

Brain Lateralization of Emotional Processing in Depression

Danielle M. Pereira and Azizuddin Khan

Abstract

There are three major hypotheses regarding the lateralization of emotion in the brain—the right-hemisphere hypothesis (RHH), the valence hypothesis, and the approach-withdrawal hypothesis. The approach-withdrawal hypothesis, which is the most widely accepted, states that emotions that elicit approach behaviors are lateralized to the left hemisphere, while emotions that elicit withdrawal behaviors are lateralized to the right hemisphere. In line with this hypothesis, it has been found that persons with depression show left frontal hypoactivity and right frontal hyperactivity. This hemispheric asymmetry appears not to influence mood but rather emotional reactions to affective stimuli. That is, a person with such an asymmetry does not show a predominant negative mood, but rather heightened negative reactions to occurrences in the environment. The asymmetry may also be a biological marker of depression, with research evidence that it is found in remitted depressives and in infants of depressed mothers. Currently, research in this area focuses on identifying the mechanism underlying the link between the asymmetry and depression.

Keywords: biological marker, depression, emotion regulation, frontal hyperactivity, hemispheric asymmetry

1. Introduction

The cortex is the neuron-rich outer layer of our brains that is believed to be responsible for all higher-order mental processes. This cortex is divided anatomically into two hemispheres—left and right. While the two hemispheres are similar in appearance, they have, however, been found to show differences in function. The idea that the two hemispheres of the brain were specialized for different areas can be said to have gained popularity after Broca’s discovery in the 1860s that language is lateralized to the left hemisphere. Since then, researchers
have continued to explore the possibility of lateralization of other human functions. One such area of interest is human emotion. An emotion is a complex psychological state that involves three distinct components: a subjective experience, a physiological response, and a behavioral response [1]. Emotional responses, as distinguished from other responses emitted by the organism, are brief, often quick, organized, involve complex patterning across a number of different systems, and are difficult to control [2].

2. Brain lateralization of emotion: hypotheses over time

Research on brain lateralization of emotion has a long history, originating with lesion studies and more recently getting a new breath of life with the emergence of more sophisticated neuroimaging methods. In spite of research spanning over a century, strong trends in results are seen but nothing is conclusive, and debates in this area abound—this is evidence of the complicated relationship between emotions and brain and of how much scientists still have to discover about this complex organ. Views about the hemispheric specialization of emotion can now be categorized into three main hypotheses.

The oldest of these is the Right-Hemisphere Hypothesis (RHH) that, in its original version, states the right hemisphere of the brain is specialized for the perception, expression, and experience of emotion. Most work on the right-hemisphere hypothesis was done in the area of perception. For example, in lesion studies, subjects with right-hemisphere lesions were found to be more deficient than left-lesioned subjects in the perception and discrimination of emotional tones in both speech [3, 4] and emotional facial expressions [5]. With respect to the expression of emotion, several studies demonstrated that registration of emotional expression is stronger in the left than the right half of the face. Since there is contralateral control of the hemifaces, these results point to the greater role of the right hemisphere in the expression of emotion. A prolific researcher in this area was Borod, who first showed the right-hemisphere hypothesis to be superior to the handedness hypothesis that states that facial expression of emotion is dominant on the right hemiface for left-handed persons and dominant on the left hemiface for right-handed persons [6]. He later showed the right-hemisphere hypothesis to be superior to the valence hypothesis, which proposed that the dominant hemiface for emotion expression shifted as a function of valence [7]. In another study, he showed that the right-hemisphere hypothesis was upheld for facial expression irrespective of whether the expression was spontaneous or posed [8]. Later, in the history of right-hemisphere research, it has been identified that specifically posterior regions of the right-hemisphere activate during emotional perception. A study by Sato et al. [9] used Functional magnetic resonance imaging (fMRI) technique and found that the occipital and temporal cortices of the right hemisphere were activated in response to presentation of emotional faces (both happy and fearful), but not to control faces that were emotionally neutral.

However, when testing the right-hemisphere hypothesis in the area of experience, evidence has tended to be inconclusive. Most prominently, it was noticed that though studies supported right-hemisphere advantage when a negative emotion versus a control was compared, when positive emotion was studied, clear right-hemisphere dominance was not seen. Currently,
therefore, when the RHH is cited, it is framed as pertaining specifically to the perception of emotion [10, 11].

An alternate hypothesis was then derived called the valence hypothesis [12]. This hypothesis holds that certain regions of the left hemisphere are specialized for the processing of positive affect, while certain regions of the right hemisphere are specialized for the processing of negative affect [2]. The bulk of evidence for the valence hypothesis came from observations in early lesion studies. It was found that persons with unilateral left hemisphere damage showed a high incidence of “catastrophic reactions,” which included negative affect, tears, guilt, and pessimism about the future. At the same time, persons with unilateral right-hemisphere damage showed either indifference or euphoric reactions, characterized by inappropriate positive affect, joking and laughing, mimicry, relaxation, and a sense of well-being [13–16]. This was interpreted as evidence for contralateral inhibition, the theory that each hemisphere plays a role in inhibition of the emotional expression of the other, and when one is damaged by a lesion, the undamaged hemisphere is able to produce its emotion unchecked. The results of the lesion studies were then confirmed by studies using more precise techniques for localizing cortical lesions, such as the Wada test. In the Wada test, localized incapacitation of either hemisphere is carried out through injections of sodium-amobarbital into the left or right carotid artery. Amytal injections in the left artery produced the same catastrophic reactions, while those in the right artery produced euphoric reactions [17–21]. Moreover, Robinson et al. [22] and Starkstein et al. [23], among others, found that the closer the location of the lesion to the left frontal pole, the more severe the negative reaction observed. Thus while the posterior regions of the brain were the major focus in the right-hemisphere hypothesis studies of emotion perception, especially the temporal and occipital lobes, anterior asymmetry became the focus of studies on emotion expression and the valence hypothesis.

Attempts were made to formulate alternate explanations for the results of these studies. Gainotti [15] proposed that patients’ psychological response to their own illness may account for some of the emotional asymmetries observed. Since right-hemisphere damaged patients are often unaware of their deficits, a condition called anosognosia, perhaps they do not show negative reactions to their own illness like left-hemisphere-lesion patients do. However, this hypothesis does not account for the WADA test results. Since the right- versus left-hemisphere incapacitation in those studies is merely temporary, the negative emotions observed in left-hemisphere injection patients is not a reaction to an illness. Other researchers theorized that the emotional asymmetry observed in these studies should be interpreted as symptoms of emotional communication disorder rather than differences in emotional experience. Since emotional prosody or speech and emotions in facial expressions are right-hemisphere lateralized, right-hemisphere damaged patients cannot show depressive reactions and may be seen as indifferent. However, a study by Gasparini et al. [24] refuted this hypothesis. The Minnesota Multiphasic Inventory (MMPI) was administered to both left- and right-hemisphere damaged patients. It was found that left-hemisphere-damaged patients scored significantly higher on the depression scale than right-hemisphere-damaged patients. Since the MMI does not require emotionally intoned speech or facial expressions, the observed difference in scale scores can be assumed to reflect difference in emotional experience.
With the emergence of electroencephalography (EEG) as a research technique, a large amount of supportive evidence was obtained for the valence hypothesis. In this method, brain waves are recorded by amplifying voltage differences between electrodes placed on the scalp. There are four major types of brain waves that are identified—alpha, beta, gamma, and theta. Alpha waves are associated with relaxation, cortical idling, and cognitive inactivity. Thus power in the alpha-band frequency is considered a reverse measure of tonal brain activation, that is, if greater frequency of alpha waves is recorded in a particular brain area, activity of that area is assumed to be low. Davidson et al. [25] published the first paper linking positive and negative affect to frontal EEG asymmetry, where relative left frontal activity was associated with positive affect, while relative right frontal activity was associated with negative affect. This was followed by a large number of EEG studies yielding results that supported the association between the frontal left hemisphere and positive emotion and frontal right hemisphere and negative emotion. For example, Tomarken et al. [26] recorded EEG data from normal participants and administered the Positive and Negative Affect Schedule (PANAS) to them. Results showed that subjects who were characterized by relatively greater left versus right frontal activation reported increased positive and decreased negative affect. The reverse was found in subjects with the opposite pattern of asymmetry. Versions of the valence hypothesis now differ as to whether the right-left distinction is seen for all emotional processing or whether perception of emotion irrespective of valence is right-hemisphere dominant.

One limitation of the valence hypothesis, however, is its inability to account for the lateralization of anger, which though popularly considered a negative emotion has found to be left-hemisphere dominant [27]. This limitation is dealt with in the third and most recent hypothesis regarding the lateralization of emotion, namely the approach-withdrawal hypothesis. The approach-withdrawal hypothesis is phylogenetically relevant because it looks at emotion from the point of view of the evolutionary purpose it serves. In terms of motivation, emotions serve one of two purposes—to elicit approach behaviors or withdrawal behaviors in response to stimuli or situations. For example, happiness and anger elicit approach behaviors, to celebrate or to attack, respectively. Both fear and disgust elicit withdrawal behaviors—fear elicits the flight response, while disgust elicits a termination response, the shutting down of input from a sense organ. This hypothesis states that the left and right anterior regions of the left and right hemispheres are dominant for approach and withdrawal behaviors, respectively. The left frontal cortex is important for intention, planning, and regulation, which are important components of approach behaviors. The right prefrontal cortex has links to behavioral inhibition, which is important for withdrawal. According to Davidson [28], there are individual differences in approach versus withdrawal temperament, which are state-like and stable over time, and these can be linked to stable differences in baseline measures of activation asymmetry in the anterior regions of the brain. Approach versus withdrawal behavior to specific stimuli is then superimposed on this state. Since positive emotion motivates approach behavior and negative emotion is associated with withdrawal, this hypothesis overlaps significantly with the valence hypothesis. However, the approach-withdrawal hypothesis overcomes one of the limitations of the valence hypothesis in that it accounts for left-hemisphere dominance of the emotion anger, which elicits approach behavior even though it is considered to have negative valence. However, one limitation of the approach-withdrawal hypothesis is...
the contradiction of associating the right hemisphere with withdrawal behavior when it has been established that the right hemisphere is responsible for creativity, which is definitely an approach behavior [29].

3. Depression and hemispheric asymmetry

If the approach-withdrawal theory of emotion lateralization is accepted, with its view that the frontal right hemisphere is specialized for withdrawal emotions and the frontal left hemisphere for approach emotions, one could then reasonably hypothesize that clinical depression would show a stronger relationship with the frontal right hemisphere than the frontal left hemisphere. This hypothesis has been supported by a variety of studies using the different techniques for exploring brain lateralization.

Early studies of patients with unilateral lesions who developed clinical depression analyzed the location of lesion in relationship to the severity of the depression experienced. Two features of the lesions were repeatedly associated with greater likelihood for depression—first, that it was located in the left rather than the right hemisphere and second, that it was located in anterior rather than posterior regions [30–33]. Interpreting this in the light of contralateral inhibition it appears that when the inhibitory capacity of the frontal left hemisphere is affected due to damage, the right hemisphere is free to produce negative emotion unchecked. The results of these and other similar studies led Ross and Rush [34] to theorize that depression may be initiated by structures in the right hemisphere.

While lesion studies were informative, it was necessary to determine whether the hypothesized relationship between depression and the right hemisphere could be demonstrated in patients with intact brain function. With the development of more advanced methods, studies using the technique of positron emission tomography (PET) found relatively greater right frontal than left frontal activation in depressed subjects [35, 36]. But the largest body of evidence in favor of this relationship comes from studies using the electroencephalography method to study the brains of persons with depression. Early studies by Tucker et al. [37] and Schaffer et al. [38] found a relationship between relatively greater right frontal activation and higher scores on self-report measures of depression among students. Henriques and Davidson [39] found that clinically depressed patients had relatively greater right-hemisphere activation than left-hemisphere activation, which was not seen in controls. The same asymmetry has been observed in multiple studies of persons with depression [40–44].

Moreover, it appears that this demonstrable anterior hemispheric asymmetry could function as a trait marker for depression. There are two main lines of evidence supporting this claim. First, studies that have compared EEG activation in currently depressed patients to remitted patients have found no significant difference in the asymmetry seen in the two groups [39, 43, 45, 46]. Thus, right-hemisphere hyperactivation is not only limited to depressed patients with currently active symptoms, suggesting a trait-like rather than state-like nature. Allen et al. [46] examined resting EEG alpha in 30 women diagnosed in major depression at four-week intervals over a course of 8–16 weeks. They found lesser left than right-hemisphere activity
(characterized by greater left than right alpha-band activity) in these women, and this asymmetry showed over the course of the study, evidence that this is a fairly stable characteristic of depression.

Second, developmental research studies have found that the anterior asymmetry can be seen in infants as well. In a study by Davidson and Fox [47], EEG recordings from 10-month-old infants showed that there is relatively greater right frontal activation in infants who cry in response to maternal separation as compared to those who do not cry. Studies have found that 1-month-old infants [48] and 3-month-old infants [49] of depressed mothers with anterior asymmetry also have relatively greater right-hemisphere activation than left-hemisphere activation. Moreover, this asymmetry is stable across time. A second study by Jones et al. [50] found that right-hemisphere hyperactivation in 3-month-old infants of depressed mothers persisted when the infants were tested at age 3 years. Thus evidence appears to support the idea that right-hemisphere activation is a biological vulnerability marker for depression that is inherited. One should note though, that twin studies [51, 52] have found that only 11–28% of the variance of frontal alpha power asymmetry in children and 27% of variance in adults is accounted for by genetics.

However, there are some inconsistencies in results across studies, suggesting that the lateralization of emotion suggested by the approach-withdrawal hypothesis and the relationship between anterior hemispheric asymmetry and depression are not as simple as they seem. Gainotti [53] reviewed a number of studies on left- versus right-brain-damaged patients that did not find a clear difference in lateralization of emotion. Similarly, several groups have failed to demonstrate lateralization of emotion and anterior asymmetry in depression in EEG studies [54, 55]. A meta-analysis by Wager et al. [56] found limited support for a simple valence-based lateralization of emotion in the brain and concluded that lateralization of emotion in the brain is more complex and region-specific than early theories proposed.

Davidson [28] argued that the reason for differing results in some studies is that anterior cerebral asymmetry predisposes positive versus negative responses only in the presence of a specific emotion elicitor. Thus studies using the same procedure may yield different results because the participants’ emotional behavior depends not merely on asymmetry in frontal cortical activation but more importantly on the emotion elicitors present during the experiment, which differ from study to study. Davidson and Fox [47] demonstrated that in healthy controls, baseline anterior asymmetry in the hemispheres predicts emotional reactions to a specific emotional challenge but is unrelated to general emotional state. In a study by Tomarken et al. [57], baseline EEG was recorded, following which participants were made to view film clips designed to elicit either positive or negative emotion. They found that baseline frontal right-hemisphere activation was associated with a heightened negative affective reaction to the negative film clips. Similarly, Wheeler et al. [58] measured baseline EEG recordings of participants at two separate occasions three weeks apart. Following the second recording, participants were shown brief positive and negative emotional film clips. Researchers
found that if a recorded frontal asymmetry was stable across the three-week interval, then those participants with greater left frontal activation reported more intense positive affect in response to the positive film clips, while those participants with greater right frontal activation reported more intense negative affect in response to the negative film clips. In other words, these studies demonstrate that anterior hemispheric asymmetry predisposes affective reactions in the individual and not mood. Thus, the right-hemisphere hyperactivity seen in depression would not precipitate a negative mood, but would be associated with heightened negative affective reactions to stimuli.

Recent research in the area has focused on determining what underlies the link between this particular hemispheric asymmetry and depression. The above distinction between mood and emotional reaction makes it tenable that the frontal right and left hemispheres are involved in emotion regulation. That is, the anterior asymmetry observed in depression may not be associated with differences in production of negative emotions, but may be associated with a difficulty regulating negative affective reactions to stimuli. Pereira and Khan [59] tested this hypothesis using a tachistoscopic task that measured the ability to disengage attention from affective stimuli. In this experiment, emotion regulation was not defined as a higher-order strategy like cognitive restructuring. Rather, the focus was on the basic cognitive process of attention, specifically the ability to disengage or inhibit attention to a stimulus. This ability can be considered highly relevant in the context of emotion regulation since an important first step in regulating emotion is shifting attention away from the emotion-producing stimulus. Furthermore, impaired disengagement of attention is a repeatedly found cognitive deficit in depression. In the experimental task used in this study, affective stimuli (pictures in one version of the task and words in another version) were primed to either the left or right hemisphere by manipulating position of presentation on the screen. This was followed by immediate presentation of the affective primed stimulus with a neutral stimulus both in the center of the screen, one below the other. In half the trials, the neutral stimulus was placed over the affective stimulus, in the other half, the affective stimulus was placed over the neutral one. Participants had to indicate the position of the neutral stimulus by pressing either the “Up” or “Down” arrow keys, thus requiring them to shift attention away from the primed affective one. Apart from the visual field presentation, valence of the affective stimulus was manipulated at two levels—positive and negative. The dependent variable measured was the reaction time of the participants in pressing the key on each trial. The assumption was that the longer the reaction time, the longer the time taken by the participant to disengage from the affective stimulus and note the location of the neutral stimulus. The same task was given to both clinically depressed participants and healthy controls to perform. In controls, a distinct right-hemisphere advantage was seen for disengaging attention, that is, shorter reaction times for affective stimuli primed to the left visual field irrespective of valence. This was found to be in line with research that has identified right-hemisphere structures as the seat of behavioral inhibition. In the depressed group, however, this right-hemisphere advantage was not observed. These results support the inference that the right-hemisphere hyperactivity seen in depression is due to some dysfunction of the inhibition system.
Author details

Danielle M. Pereira and Azizuddin Khan*

*Address all correspondence to: khanaziz@iitb.ac.in

Psychophysiology Laboratory, Department of Humanities and Social Sciences, Indian Institute of Technology Bombay, Mumbai, Maharashtra, India

References


