Alexithymia, Gender, and Hemispheric Functioning

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It has been hypothesized that alexithymia is related to an impairment of the right hemisphere or a deficiency in interhemispheric transfer. We used the Toronto Alexithymia Scale-20 (TAS-20) and the tactile finger localization task of Zeitlin et al. to test these relationships on nonclinical samples of college men and women, and also considered the role of short-term memory. Among 47 men, the TAS-20 facets of difficulty identifying feelings or difficulty describing feelings were correlated with poorer performance by the right compared with the left hemisphere in uncrossed trials and poorer interhemispheric transfer of information on crossed trials; short-term memory was not related. Thus, both hemispheric hypotheses were supported for men. However, among 58 women, alexithymia was completely unrelated to either index of hemispheric functioning; instead, poorer short-term memory (specifically digits backwards) strongly predicted poorer interhemispheric transfer. We conclude that deficiencies in right hemisphere function and interhemispheric transfer may contribute to alexithymia in men, but not in women.

RECENT DECADES have witnessed substantial theoretic and empirical attention given to the construct of alexithymia, which is defined as a deficit in one's ability to identify and describe emotions, along with an externally oriented cognitive style. Alexithymia is hypothesized to predispose to or exacerbate psychiatric and medical disorders in which affect regulation problems play key roles.[1] Theoretically, the inability to identify one's emotions, differentiate emotions from other physiologic states, and regulate negative affect via adaptive cognitive and interpersonal processes leads to excessive physiologic arousal, as well as maladaptive behavioral attempts to regulate affect. These physiologic and behavioral consequences of alexithymia can contribute to somatoform, anxiety, substance abuse, and eating disorders, as well as interpersonal difficulties.

However, the etiology of alexithymia remains unknown, although several neurobiological models have been proposed. Over five decades ago, MacLean[2] theorized that impaired communication between limbic and neocortical systems interferes with the integration of affective and verbal processes. In support of this, Lane et al.[3] have provided empirical evidence that patients with limited emotional awareness have reduced activity in the anterior cingulate cortex, which suggests a disconnection between basic neural emotion centers and higher cortical consciousness and regulation functions. In contrast to these models which focus on the integration of cortical and subcortical processes, others have proposed that alexithymia is dependent on the functioning of the two cerebral hemispheres. Taylor et al.[1] describe two hemispheric models of alexithymia, one based on a relative dysfunction of the right hemisphere and the other based on a deficit in interhemispheric transfer.

The right hemisphere dysfunction model of alexithymia derives from observations that among right-handed people the right hemisphere shows an advantage for nonverbal and holistic processing and emotionality, whereas the left hemisphere shows an advantage for verbal process and analytic thinking. Given these functions, one might hypothesize that alexithymia, where emotional deficits are found in conjunction with an overly analytic, sometimes verbose cognitive style, is due to a relative deficit or dysfunction in the right hemisphere relative to the left. This supposition has some empirical support. For example, right hemisphere learning disabilities share features with alexithymia,[4] and alexithymic-like deficits in fantasy and verbalization of feelings have been found among people with right hemisphere lesions.[5,6] Experimental studies have shown that alexithymia, or low emotional awareness, is related to deficits in emotion identification and interpretation,[7,8] as well as preferences for right-directed conjugate lateral eye movements[9] and right visual field search of chimeric faces.[10-12] These findings suggest a left hemisphere dominance or right hemisphere weakness.

The second hemispheric model suggests that alexithymia stems from a deficit in communication

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between the cerebral hemispheres, which are connected by the corpus collosum and other commissures. The alexithymic person's inability to apply linguistic descriptions (presumably a left hemisphere function) to emotional experience (presumably a right hemisphere function) suggests a disconnection between hemispheres,[13] and there is both clinical and experimental support for the interhemispheric transfer deficit hypothesis. Patients who have undergone commissurotomies for intractable epilepsy manifest the key features of alexithymia—deficits in the ability to talk about feelings, limited dream recall, and an externally oriented cognitive style,[14,15] which differ significantly from the findings for neurologically intact controls.[16]

In an important test of the interhemispheric transfer deficit hypothesis with neurologically intact subjects, Zeitlin et al.[17] assessed alexithymia (Toronto Alexithymia Scale [TAS]) and hemispheric functioning among 25 male combat veterans with posttraumatic stress disorder (PTSD). They used the tactile finger localization task, in which the fingers of blindfolded subjects are touched by the experimenter in a random order and subjects are required to repeat the sequence by touching their fingers with their thumbs in the same order as done by the experimenter. Either hand can be stimulated, and responses can be made on the same hand, in which case sensorimotor activity involves only one hemisphere (ipsilateral), or responses can be made by the other hand, necessitating transfer of information across the hemispheres (contralateral). Zeitlin et al.[17] found that the error rate for contralateral trials compared with ipsilateral trials was strongly positively correlated with alexithymia (accounting for 35.58% of the variance in TAS scores) even after controlling for intelligence and PTSD severity. They found no evidence that the deficit in interhemispheric transfer was limited to one direction or the other, and their data did not suggest alexithymia-related dysfunction solely of the right hemisphere.

The methods and findings of Zeitlin et al.[17] are impressive and have been widely cited, but concerns about the generalizability of the study necessitate its replication, especially on a nonclinical sample. First, the sample was relatively small and quite atypical; all of the subjects were male combat veterans with PTSD and 80% had comorbid diagnoses of alcohol and/or substance abuse. Also, the patients had exceptionally high levels of alexithymia. Sixty percent of the patients were classified as alexithymic, and their TAS scores were highly elevated (mean $\pm$ SD, 89.3 $\pm$ 11.3), averaging over 5 SD above the mean of a group of healthy control subjects in the study. However, recently, Parker et al.[18] replicated the findings of Zeitlin et al.[17] These authors tested tactile finger localization on 29 college men who scored as either alexithymic ($n = 14$) or nonalexithymic ($n = 15$) on the 20-item TAS, and found that alexithymic subjects were significantly less efficient at transferring tactile information between cerebral hemispheres than nonalexithymic subjects.

In addition to further replication, other methodologic advances are needed. First, it is not known whether women demonstrate an association between alexithymia and hemispheric functioning; the studies by neither Zeitlin et al.[17] nor Parker et al.[18] included women. Women appear to demonstrate less hemisphericity during dichotic listening, tachistoscopic, and brain lesion studies,[19,20] and women appear to have larger corpus collosa than men,[21] raising the possibility that the neurologic underpinnings of alexithymia differ in women and men. Second, successful tactile finger localization requires not only intact neurologic functioning but also adequate short-term memory, because the subject needs to recall a sequence of touches. Thus, it is important to test whether short-term memory deficits may account for the observed alexithymia effect. Third, there is evidence that the different facets of alexithymia as measured by the 20-item TAS—difficulty identifying feelings, difficulty describing feelings, and externally oriented thinking—have different external correlates.[22-24] Although the full scale measures the broad construct of alexithymia, it is important to test the correlates of the individual facets as well, which was not done in the two prior studies of alexithymia and tactile finger localization.[17,18]

We studied separate samples of men and women and assessed global alexithymia as well as its facets, short-term memory, and tactile finger localization based on the protocol of Zeitlin et al.[17] This task was used to test both the right hemisphere dysfunction hypothesis, because finger localization is sensitive to damage or impairment in one hemisphere,[25] and the interhemispheric transfer deficit hypothesis. The first hypothesis was tested by relating alexithymia scores to the performance of the right hemisphere relative to the left hemisphere