Cognitive and emotional characteristics of alexithymia
A review of neurobiological studies

Junilla K. Larsena,*, Nico Branda, Bob Bermondb, Ron Hijmanc

a Department of Health Psychology, Utrecht University, Postbus 80140, Utrecht 3508 TC, Netherlands
b Department of Neuropsychology, University of Amsterdam, Amsterdam, Netherlands
c Department of Psychiatry, University Medical Center of Utrecht, Utrecht, Netherlands

Received 7 November 2001; accepted 29 April 2002

Abstract

Objective: To review neurobiological studies of alexithymia in order to achieve a better understanding of the relationship between alexithymia and psychosomatic diseases and psychiatric illnesses. Methods: Neurobiological studies of alexithymia were reviewed with a special focus on how emotional and cognitive elements of alexithymia are reflected in earlier research. Results: Studies that have correlated alexithymia to corpus callosum dysfunctioning have mainly found impairments in cognitive characteristics of alexithymia, whereas from studies of right hemisphere and frontal lobe deficits, it may be concluded that both cognitive and emotional characteristics of alexithymia are impaired. Conclusion: The fact that there is no general agreement on how to define alexithymia seems to have hampered theoretical and empirical progress on the neurobiology of alexithymia and related psychosomatic diseases and psychiatric illnesses. Alexithymia should no longer be approached as one distinct categorical phenomenon and follow-up studies should monitor subjects according to both the cognitive and emotional characteristics of alexithymia.

Keywords: Alexithymia; Neurobiology; Right hemisphere; Commissurotomy; Interhemispheric transfer; Frontal lobe

Introduction

Definition of alexithymia

Over the past two decades, there has been an expanding scientific interest in the regulation of emotion and in the impact of dysregulated emotion on mental and physical health [1]. Alexithymia refers to difficulties in emotional self-regulation and is thought to be one of several possible risk factors in a variety of medical [2–7] and psychiatric disorders [8–17]. The alexithymia concept stems from the field of the psychology of emotions and psychosomatics. A large proportion of patients with psychosomatic complaints showed difficulties in emotional self-regulation. Sifneos [18] introduced the term “alexithymia” for these emotional difficulties, which was derived from “the Greek alexis (no words), thymos (emotion).” Five salient characteristics of alexithymia have been described: (1) a reduction or incapacity to experience emotions; (2) a reduction or incapacity to verbalize emotions; (3) a reduction or incapacity to fantasize; (4) an absence of tendencies to think about one’s emotions; and (5) difficulty in identifying emotions [15,19]. From the first introduction of the term alexithymia, it has been evaluated, criticized and refined. Whereas some researchers regard the cognitive, evaluative aspects of alexithymia as most important [17], others suggest that the fundamental deficit in alexithymia is a limited — and in extreme cases nonexistent — ability to consciously experience emotion [20]. Drawing on recent knowledge from neurobiology, Bermond [21,22] distinguishes two main forms of alexithymia (Types I and II). Type I alexithymia is characterized by the absence of the emotional experience and, consequently, by the absence of the cognition accompanying the emotion. Type II alexithymia is characterized by a selective deficit of emotional cognition with sparing of emotional experience. Given the important psychological differences between the two types of alexithymia, it is suggested here that the classification into two types of
alexithymia is useful towards examining the existing literature on the neurobiology of alexithymia, since it may offer an explanation for the distinct neurobiological and related physiological findings in the literature.

**Measurement of alexithymia**

Many of the early studies on alexithymia are of questionable validity, as they were conducted with measures that were shown to lack reliability and validity, such as the Schalling Sifneos Personality Scale (SSPS) and the MMPI alexithymia scale [1]. For several years, however, the Toronto Alexithymia Scale (TAS) has proven to be the most reliable and valid method for measuring several characteristics of alexithymia [23–25]. The TAS demonstrated a replicable four-factor structure: difficulty identifying feelings (Factor 1); difficulty describing feelings (Factor 2); reduced daydreaming (Factor 3); and a tendency to think in externally oriented ways (Factor 4). Since the items assessing daydreaming showed little coherence with the other factors, two successive revisions of the scale have led to the 20-item Toronto Alexithymia Scale (TAS-20), eliminating all items assessing imaginal activity [26–28].

Recently, the validity and reliability of a new instrument to assess alexithymia, the Bermond–Vorst Alexithymia Questionnaire (BVAQ), have been established [29,30]. The BVAQ contains five subscales: (1) emotionalizing, (2) fantasizing, (3) identifying, (4) analyzing and (5) verbalizing emotions. Analogous to the distinction in cognitive and emotional aspects of alexithymia within the hypothesized subtypes of alexithymia [21,22], the subscales of the BVAQ exhibit a second-order (two-factor) structure, with the subscales emotionalizing and fantasizing representing an emotional component, and the subscales identifying, analyzing and verbalizing emotions representing a cognitive component of alexithymia, respectively. The total TAS-20 score shows high correlations with the cognitive, but not the emotional, component of the BVAQ [29,30]. Both difficulty fantasizing and difficulty emotionalizing within the BVAQ remained statistically uncorrelated with the total TAS-20 and weakly correlated or uncorrelated with the TAS-20 subscales [29]. Therefore, as a diagnostic instrument, the TAS-20 may emphasize the cognitive and underestimate the emotional component of alexithymia. It is suggested here that both the original and revised versions of the TAS emphasize the cognitive component of alexithymia, since most, if not all, factors of the TAS represent cognitive characteristics of alexithymia.

**Research aim**

Alexithymia has often been attributed to neurobiological dysfunctioning. Some studies found evidence for dysfunction of the corpus callosum in alexithymia, whereas other studies found a relationship between alexithymic characteristics and dysfunctioning of the right cerebral hemisphere, anterior cingulate cortex (ACC) or the orbitofrontal cortex. The aim of this article is to review neurobiological studies of alexithymia in order to achieve a better understanding of the relationship between alexithymia and psychosomatic diseases and psychiatric illnesses. To that aim, neurobiological studies of alexithymia were reviewed with a special focus on how emotional and cognitive elements of alexithymia are reflected in earlier research, seeking evidence for the two main forms of alexithymia as originally defined by Bermond [21].

**Alexithymia and neurobiological dysfunctioning**

Several models have been proposed to explain how the brain mediates emotion. A parsimonious and convincing model states that the right hemisphere is more involved than the left in all aspects of emotional behavior [31,32]. This model is largely based on the fact that in most people, the verbal, conscious and serial information processing takes place in the left hemisphere [33,34], while the unconscious, nonverbal and emotional information processing mainly takes place in the right hemisphere [35–38]. From this model, two neurobiological views on alexithymia have been derived: (A) alexithymia is the result of a deficit in interhemispheric communication, involving the corpus callosum [39–42]; and (B) alexithymia is the result of a dysfunction of the right cerebral hemisphere [43,44]. Another model states that the brain organizes emotion differently as a function of valence, with positive emotions mediated by the left hemisphere and negative emotions by the right hemisphere [30]. This model has been further expanded by Davidson et al. [45], who postulate that the existence of separate approach (correlating with positive emotion) and withdrawal systems (correlating with negative emotion) is lateralized to the left and right frontal lobes, respectively [45]. A third neurobiological hypothesis has been derived from this model: (C) alexithymia is the result of dysfunctional mechanisms in the frontal cortex. The above-mentioned models should be considered more as attempts at systematization, aiming to classify neurobiological studies on alexithymia, than as firmly established theories.

**Corpus callosum dysfunctioning**

According to Gazzaniga and LeDoux [46], cognitive components of stimuli presented to the right hemisphere reach the conscious left hemisphere directly by means of the corpus callosum, while the emotional value is first projected downward to the limbic system and from there reaches the left hemisphere by the anterior commissure. Consequently, blockage of the function of the corpus callosum can result into a specific type of alexithymia (Type II), in which a person still experiences basic emotional feelings, but has no conscious cognition concerning these feelings [21]. The hypothesized relationship between blockage of corpus cal-
loss of functioning and Type II alexithymia has been strengthened through the descriptions of persons with corpus callosum transections [47,48]. When emotional stimuli were presented to the right hemisphere of these persons, they were unable to verbalize their emotional feelings, whereas from nonverbal reactions, it was clear that emotional feelings had been induced. On the basis of the reasoning of Gazzaniga and LeDoux, it should be expected that in case of complete cerebral commissurotomies, i.e., sectioning both the corpus callosum and the anterior commissure, there should be deficits in both emotional and cognitive aspects of alexithymia. In a preliminary investigation of 12 split-brain individuals, Hoppe and Bogen [39] observed that patients following complete cerebral commissurotomies for intractable epilepsy developed a decreased capacity for fantasy, a pronounced operative style of thinking and difficulties in describing feelings. While the pronounced operative style of thinking and difficulties in describing feelings are cognitive characteristics of alexithymia, the decreased capacity for fantasy is an emotional aspect of alexithymia [30]. Thus, these results are in line with the hypothesis that complete cerebral commissurotomies would lead to deficits in both cognitive and emotional aspects of alexithymia.

TenHouten et al. [40,49,50] compared the spoken and written responses of eight patients who underwent cerebral commissurotomies with those of eight neurologically intact control subjects to a film that was aimed at evoking emotions. In comparison with the control subjects, commissurotomized patients showed a decreased capacity for fantasy and use of symbols (emotional aspects of alexithymia) and difficulties in describing feelings (cognitive aspect of alexithymia).

As a result of the studies with split-brain patients, researchers have become interested in the relationship between alexithymia and interhemispheric transfer (corpus callosum functioning). Studies have been performed in which alexithymic and nonalexithymic patients have been compared on interhemispheric transfer through the corpus callosum. Support for the interhemispheric communication deficit hypothesis of the cognitive component of alexithymia was provided by studies using a tactile finger localization task for intractable epilepsy, a decreased capacity for fantasy, a pronounced operative style of thinking and difficulties in describing feelings. While the pronounced operative style of thinking and difficulties in describing feelings are cognitive characteristics of alexithymia, the decreased capacity for fantasy is an emotional aspect of alexithymia [30]. Thus, these results are in line with the hypothesis that complete cerebral commissurotomies would lead to deficits in both cognitive and emotional aspects of alexithymia.

A study by Zeitlin et al. [51] compared the efficiency of interhemispheric transfer in 15 alexithymic and 7 nonalexithymic patients suffering from posttraumatic stress disorder (PTSD) and 10 controls in using the tactile finger localization task. Alexithymia was assessed by the total TAS score, emphasizing the cognitive aspects of alexithymia. A strong association was found between the TAS global alexithymia score and the lack of bidirectional interhemi-

spheric transfer. Parker et al. [42] also found a strong association between the TAS global alexithymia score and a bidirectional deficit in interhemispheric transfer. From both studies, it appeared that the deficit in interhemispheric communication in the cognitive alexithymic patients was not as great as in commissurotomized patients. A possible explanation for this is that the tactile finger localization task is not the most appropriate way to assess a deficit in interhemispheric communication associated with emotional processing [51]. Another explanation is that in the noncommissurotomized alexithymic patients, the disconnection is less absolute and more functional in nature. In an extension of the study of Zeitlin et al. [51], Dewaraja and Sasaki [54] examined the relationship between interhemispheric transfer and alexithymia using both linguistic and nonlinguistic Japanese symbolic stimuli. Alexithymia was assessed using the MMPI alexithymia scale and the Schalling Sifneos Personality Scale—Revised (SSPS-R). While both instruments have proven unsatisfactory psychometric qualities [55], their factors point mainly to a cognitive deficit in alexithymia (i.e., capacity to describe feelings, preference for describing events in detail and the ability to communicate with others). Whereas Zeitlin et al. [51] found a bidirectional relationship between cognitive aspects of alexithymia and the callosal transfer process, Dewaraja and Sasaki [54] found a unidirectional (right-to-left) relationship. The processing of linguistic information is known to be mainly associated with the left hemisphere, while sensorimotor processing is not known to be associated with a particular hemisphere. Therefore, the finding of a unidirectional relationship by Dewaraja and Sasaki [54] may be more significant regarding the concept of alexithymia.

In a recent study, the relationship between the cognitive Type II alexithymia and interhemispheric transfer deficit was examined [41] using the BVAQ. The EEG was continuously recorded from homologous occipital, parietal, temporal and frontal recording sites during the presentation of films excerpts, one of neutral and two of emotional content. An association was found between the cognitive, but not the emotional, component of alexithymia and interhemispheric transfer.

Type II alexithymics, as compared to nonalexithymics, had reduced coherence between the right frontal lobe and the left hemisphere, independent of film. This result is also in line with the reduction in interhemispheric alpha band coherence found by TenHouten et al. [56,57] for commissurotomized patients. Because the corpus callosum appears to be necessary for the transfer of more complex information between the hemispheres, there is some indication for reduced callosal function between the right frontal region and the left hemisphere in Type II alexithymic individuals.

Dysfunctioning of the right cerebral hemisphere

In normals, right hemisphere advantage has frequently been observed in the perception of emotion. Similarly,
deficits in the perception of emotion have been found more frequently in patients with right than left hemisphere damage [58–62]. Several studies have shown that individuals with high levels of alexithymia are poorer than those with low levels of alexithymia in the recognition of facial expressions of emotions [20,43,44,63]. Since the ability to recognize facial expressions of emotions is predominantly linked to the right cerebral hemisphere, dysfunctioning of the right cerebral hemisphere was suggested in alexithymia [43,44]. Ross [64] and Fricchione and Howanitz [65] have described studies of patients with large right unilateral cortical lesions. These patients showed the symptoms of a major depression, but denied feelings of depression. After treatment with antidepressants, all expressive symptoms of a major depression disappeared, however, with the exception of the mental emotional numbness. Thus, right cortical lesions may result into the absence of affect, representing the emotional component of alexithymia. Following Bermond [21,22], the absence of the emotional experience — and, consequently, absence of the cognition accompanying the emotion — has been defined as Type I alexithymia. Thus, right cortical lesions may result into Type I alexithymia.

Although studies have been performed in which alexithymic and nonalexithymic patients have been deliberately compared on interhemispheric transfer, as far as we know, no valid studies have been performed in which alexithymic and nonalexithymic individuals have been directly compared on functioning of the right cerebral hemisphere. Conjugated lateral eye movements (CLEMs) have sometimes been used as an index of hemispheric activation, since a predominance of eye movements in one direction is thought to reflect activation of the contralateral hemisphere.

In a study of Parker et al. [66], the relationship between CLEMs and alexithymic characteristics, measured by the TAS, was examined. A positive relationship was found between left hemisphere activation, indicated by a predominance of right CLEMs, and the cognitive component of alexithymia, as measured by a high global score on the TAS. While the direction of CLEMs may be a function of an individual’s consistent tendency to rely on a particular cerebral hemisphere, there is evidence that the direction can be influenced by the experimental situation and by the type of questions asked. Moreover, much more research is required to determine whether a possible tendency to rely on the left hemisphere indeed represents a dysfunction of the right hemisphere [66].

**Frontal lobe dysfunctioning**

Many studies have attributed the expressive components of emotional processing to frontal lobe structures of the brain. In a model that has been proposed to explain how the brain mediates emotion, an interaction between emotional valence and frontal lateralization has been suggested [45]. However, experimental studies of right-brain-damaged and left-brain-damaged patients show that when the intrahemispheric site of lesion was examined, anterior regions (i.e., frontal lobe) were more critical for emotional expression than posterior regions, independent of valence [67]. Stuss et al. [68] describe many studies finding a decrease in emotional expression after frontal lobe damage. Since a reduction in emotional expression has also been described for alexithymic patients [69], characteristics of alexithymia may be the result of deficits in frontal lobe functioning. However, the question remains as to which parts of the frontal lobe will be specifically involved in cognitive and/or emotional characteristics of alexithymia.

Higher cognitive and affective functions are mainly localized in the prefrontal cortex, which occupies approximately 30% of the total amount of cortical space. The prefrontal cortex can grossly be divided into dorsolateral and ventromedial regions. The ventromedial frontal area (Brodmann’s areas 24, 25, 32, 11, 13 and 14) is connected with limbic structures in the medial temporal lobe, and hence is well situated to integrate motivational and emotional processes. Both the orbitofrontal cortex and the ACC are located in the ventromedial frontal area and are especially important for emotional functioning [70,71]. Analysis of the orbitofrontal cortex and ACC suggest that these regions are involved in emotion-related learning [72]. In modern approaches to emotion, emotions are often considered to be states elicited by rewarding and punishing stimuli, so that any failure to alter behavior when the reinforcement value of environmental stimuli changes will lead to impairments in inappropriate emotional experience and related behavior [73]. Electrical stimulation of the orbitofrontal cortex produces many autonomic responses [74] and, moreover, orbitofrontal lesions are associated either with a blunting of emotional responses, including the attenuation of autonomic accompaniments of emotion, or intense expression of affect with loss of inhibitions [70]. Emotionally disturbed alexithymic individuals resemble the blunted rather than the disinhibited type. Lesions of the ACC have also been associated with emotional aspects of alexithymia, such as blunting of emotional experiences [75,76]. In addition, it was found that the reduction in emotional experience in a group of patients with ventral frontal lobe damage was correlated with impairments in the identification of facial and vocal emotional expression [73], merely reflecting cognitive characteristics of alexithymia.

Since normal subjects’ emotional awareness was associated with greater activation in the ACC during elicitation of film- and recall-induced emotion [77], it was hypothesized that deficits in ACC activity are associated with alexithymia. This hypothesis was tested in a comparative study of the cerebral regions activated during viewing of emotional stimuli in a group of high and low alexithymic males [78]. Intergroup comparisons revealed that alexithymics had higher activation associated with positive pictures, but showed no effect of arousal with negative
pictures. Main differences were concerned with medial prefrontal (Brodmann's area 9) and ACC regions. Although these findings should be considered preliminary until they are replicated, they might suggest that positive and negative emotions should be treated in a different manner in Type I alexithymic patients. Hornak et al. [73] studied 10 patients with ventral prefrontal lesions and observed that all reported changes in their ability to feel emotions compared to their premorbid state. However, the extent to which there were changes (increases or decreases) in the capacity to feel negative or positive emotions was quite variable across patients. It is possible that different subregions within the ventromedial prefrontal cortex could be associated with the elaboration of different types and intensity of emotion, consistent with its role in integrating information about rewards and punishments. In conclusion, there appear to be distinct regions in the ventromedial prefrontal cortex, such as the ACC and the orbitofrontal cortex, that participate in emotion and that may be associated with impairments in both cognitive and emotional aspects of alexithymia, reflecting Type I alexithymia.

Local depletion of dopamine in parts of the prefrontal cortex by application of 6-OHDA produces effects on emotions similar to those of lesions [70,79]. Dopamine neurons are mainly organized in the substantia nigra, which contains over 90% of the dopamine cells and mainly projects to the striatum. It is now generally accepted that the basal ganglia, in addition to movement control, fulfill important functions in emotions [80]. Two basal ganglia neural circuits have been described: the dorsolateral prefrontal circuit and the lateral orbitofrontal circuit (for detailed descriptions, see Ref. [80]). The basal ganglia are therefore, well connected with prefrontal structures that fulfill important functions for the emotional experience. Furthermore, it may be assumed that these circuits are modulated by dopamine, since the substantia nigra participates in both circuits [22]. In line with this, Parkinson patients, in whom the functions of nigrostriatal dopamine circuits have been reduced, show numbness in affect and motivation. This numbness in affect and motivation is related to the reduced dopaminergic activity in the prefrontal cortex [81]. Overall, depletion of dopamine in the ACC or the orbitofrontal cortex may produce both cognitive and emotional characteristics of alexithymia, defined by Bermond [21,22] as Type I alexithymia.

Alexithymia, sympathetic activity, somatic diseases and psychiatric illnesses

Sympathetic activity

Based on preliminary findings in the mid-1980s, it was suggested that alexithymia is related to illness because it produces hyperarousal to situational stressors [82,83]. Whereas in some studies alexithymia was associated with higher tonic or baseline levels of sympathetic activity [84–86], other studies found either hypoarousal or normal arousal during exposure to a stressor [87–89]. A possible explanation for these different results may be that different types of alexithymia formed by distinct neural malfunction produce different levels of sympathetic activity.

Lesions of the corpus callosum may result into higher sympathetic activity, since patients with corpus callosum deficits can be affected by several situations without having any explanation for their feelings [1]. Therefore, they will experience prolonged stress and related physiological hyperarousal, which may be a risk factor for a variety of psychosomatic diseases and psychiatric illnesses. No studies on the physiology of corpus callosum deficits have been performed so far. However, data from brain-damaged and normal subjects strongly suggest that the right hemisphere is more closely related to the autonomic nervous system, and thus, more intimately linked with the physiological and automatic components of the emotions than the left hemisphere [90,91]. Heilman and Gilmore [92] have suggested that the right hemisphere is more in touch with subcortical systems that are important for arousal and intention. Moreover, it appears that cerebral arousal is mediated by noradrenaline (NE), which, although widespread, is mainly supposed to be innervated by right hemispheric pathways [93]. On the basis of these findings, it may be expected that alexithymia produced by right hemisphere deficits is associated with reduced physiological responses. With respect to the orbitofrontal cortex, it has been demonstrated that electrolystimation of this area results in a great number of autonomic and endocrine responses [74] and that lesions of this area result in reduced physiological responses [70,94,95]. It is, therefore, suggested that alexithymia due to orbitofrontal lesions or a reduced neural or dopaminergic innervation of this area also results in reduced emotional physiological responses [22].

Overall, it can be stated that it is impossible to predict the level of the emotional physiological responses without knowledge on which neural malfunction alexithymia is based, since facilitation as well as inhibition may be expected. This is clearly illustrated in a study of Henry et al. [96] demonstrating that, although the mean level of sympathetic activation (as measured by 3-methoxy-4-hydroxyphenylethylene glycol levels, MHPG) in alexithymic patients is in the normal range, the MHPG levels of 16 of 17 patients studied were either clearly above or below that normal range.

(Psycho)somatic diseases and psychiatric illnesses

Neafsey [74] refers to literature demonstrating that the risk for psychosomatic diseases is reduced after prefrontal lesions. Moreover, it is suggested here that the reduced physiological responses due to deficits in the right hemisphere will also result into a reduction of psychosomatic
influence the immune system indirectly via (SNS), elevated levels of adrenaline and noradrenaline may result of stimulation of the sympathetic nervous system to sustained arousal and chronic dysregulation of the physiological component of emotion response systems. As a result of stimulation of the sympathetic nervous system (SNS), elevated levels of adrenaline and noradrenaline may influence the immune system indirectly via α- and β-adrenergic receptors that have been found on immune cells and organs [97]. In agreement with this, some research has found an association between alexithymia and reduced levels of immunity [98,99]. Dewaraja et al. [99] found that highly alexithymic men had a significantly lower number of cells of the cytotoxic natural killer (NK) subset. These results suggest that the negative modulation of cellular immunity, combined with other factors, results in the association between alexithymia and somatic diseases.

Alexithymia has been associated with a variety of (psychosomatic) diseases, including functional gastrointestinal disorders and chronic pain [2–7]. Moreover, several researchers have explored the relationships between alexithymia and psychiatric illnesses. Empirical studies have reported elevated levels of alexithymia in individuals with eating disorders, such as anorexia nervosa and bulimia nervosa [8–11]. Moreover, several studies have found a strong positive correlation between alexithymia and depression in normal [12,13] as well as clinical samples [14,15] and, furthermore, empirical studies have found elevated levels of alexithymia in patients with panic disorders [16,17] and PTSD [51]. Although many studies found varying correlations between alexithymia and psychiatric illnesses, no attention has been paid to the fact that different characteristics of alexithymia could be differentiated. With reference to possible psychiatric complaints, such a differentiation is important. Although Type I alexithymics do experience a host of problems, they are — due to the seriously reduced level of emotionalizing — still free from emotional problems [100], whereas Type II alexithymics do mention such problems. This is understandable since they do get emotionally aroused, but cannot — due to the lack of emotion accompanying cognitions — understand the felt emotional arousal, resulting in further emotional problems [11]. Moreover, because of the cross-sectional design of most of the studies, it is not possible to make any causal connection between alexithymia and the various psychiatric illnesses. Prospective studies are needed for that. From longitudinal studies, it appears that alexithymia is a stable trait, in contrast to functional somatic symptoms and psychological distress that change over time [101,102].

Discussion

The focus of this study has been on current insights into the different biological mechanisms of alexithymia. Findings from research exploring the neurobiology of alexithymia suggest that different features of alexithymia are associated with a variation in brain organization. However, the fact that there is no general agreement on how to define alexithymia seems to have hampered theoretical and empirical progress, as it has been a source of confusion and misunderstanding. Some neurobiological studies on alexithymia and emotion emphasize cognitive aspects, and other studies emotional aspects, producing different outcomes. In this article, neurobiological studies on alexithymia were reviewed with a special focus on how emotional and cognitive elements of alexithymia are reflected in earlier research, seeking evidence for the two main forms of alexithymia as defined by Bermond [21,22].

From studies of split-brain patients, it can be concluded that complete cerebral commissurotomy, i.e., sectioning the corpus callosum and the anterior commissure, results into distinct deficits in both cognitive and emotional aspects of alexithymia. Studies in which neurological intact subjects have been compared on interhemispheric transfer through the corpus callosum [41,42,51,54] show that specifically cognitive aspects, but not emotional aspects, of alexithymia are associated with deficits in interhemispheric transfer. Thereby, the findings of a unidirectional relationship in the callosal transfer process [41,54] may be more significant regarding the cognitive characteristics of alexithymia. Following Bermond, it may be concluded that Type II alexithymia, in which the cognitive but not the affective component of alexithymia is disturbed, is associated with a right-to-left unidirectional deficit in interhemispheric transfer.

Studies of patients with right unilateral cortical lesions show that these lesions often result into deficits in the perception of emotion [58–62], which may be comparable to deficits in cognitive characteristics of alexithymia. Moreover, right unilateral cortical lesions often result into emotional numbness and mental emotional indifference [32,64,65], which may be interpreted as impairments in affective aspects of alexithymia. Thus, right unilateral cortical lesions may be associated with Type I alexithymia, characterized by the absence of the emotional experience and, consequently, by the absence of the cognitions accompanying the emotion.

Many studies of patients with frontal lobe damage find a reduction in emotional expression [68], which has also been found in alexithymic patients [69]. More specifically, patients with orbitofrontal cortex and ACC lesions show impairments in affective [70,73–76] and cognitive [73,78] aspects of alexithymia. Thus, orbitofrontal cortex and ACC lesions may result into Type I alexithymia, as originally defined by Bermond [21,22], characterized (in its extreme
form) by the absence of the emotional experience and, consequently, by the absence of the cognition accompanying emotion. Since depletion of dopamine produces effects on emotions similar to lesions, it is assumed that depletion of dopamine in the lateral orbitofrontal circuit may also result into Type I alexithymia. Since little direct research has been done on the relationship among the right hemisphere, the ACC, the orbitofrontal cortex and alexithymia so far, the suggested link between these brain areas and Type I alexithymia is rather premature.

We believe that the most important implication of the reformulation of the alexithymia construct is that it provides a better explanation for the physiological basis of the association between alexithymia and physical disease. Without knowledge on which neural malfunction a specific type of alexithymia is based, it is impossible to predict anything meaningful about the level of emotional physiological responses. So far, the research on the neurobiology of alexithymia has mainly relied on indirect methods for studying brain functioning. Additional research should focus on the use of modern brain imaging techniques, including magnetic resonance spectroscopy (MRS), positron emission tomography (PET) and single photon emission computed tomography (SPECT) to compare changes in regional brain chemistry and brain activity between the different types of alexithymic individuals and nonalexithymic individuals during the elicitation and processing of emotional states. Finally, alexithymia should no longer be approached as one distinct homogenous phenomenon and, subsequently, follow-up studies should measure the various components of the alexithymia concept separately.

References


[63] Berman KE, Weinberger DR. The prefrontal cortex in schizophrenia


[95] Damasio AR, Tranel D, Damasio H. Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. Behav Brain Res 1990;41:81–94.


