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Case study

Organic alexithymia: a study of acquired emotional blindness

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The phrase 'organic alexithymia' is introduced as a clinically and theoretically useful construct for furthering understanding of alexithymia and the occurrence of alexithymic symptoms in patients with acquired brain injury (ABI). The construct is illustrated by the case study of HR, a 21-year-old man seen at the State Head Injury Unit, Perth, for neuropsychological and clinical assessment 2 years following a motor vehicle accident. HR's case supports the hypotheses that a syndrome substantially similar to alexithymia can occur in patients with acquired brain injury, and that the acquired nature of the organic disorder may cause it to be systematically different from the established syndrome. The case demonstrates the clinical relevance of a construct like organic alexithymia when treating patients with ABI. It also highlights the need to develop instruments capable of identifying the condition and differentiating it from symptoms of depression.

Introduction

Alexithymia is a psychosomatic condition characterized by difficulty identifying and verbalizing emotions, constricted imaginal processes (including a paucity of fantasies), and a stimulus-bound, externally oriented cognitive style [1]. Although research in the field largely ignores the population of patients with acquired brain injury (ABI) [1], clinical experiences with this population can suggest the existence of a sub-category, which might be referred to as 'organic alexithymia'.

The symptoms of people with organic alexithymia subsequent to ABI closely resemble those displayed by persons with non-organic alexithymia, including all those mentioned above. However, they offer an interesting contrast for two reasons. First, the symptoms of the majority of patients with organic alexithymia become manifest at the time of their primary brain injury. This is, therefore, an acquired, rather than a developed, disorder. Secondly, its acquired nature means that organic alexithymia often occurs in the absence of other significant personality or emotional disorders. As a corollary, the individual symptoms of alexithymia more often occur in isolation in the organic variant of the disorder. For example, many people with ABI display difficulty verbalizing emotions, or stimulus-bound cognitive styles, without displaying other symptoms. Therefore, organic alexithymia might demonstrate more pure examples of each symptom than non-organic alexithymia.

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This would allow a more precise description of the characteristics and mechanisms of alexithymia by providing cleaner contrasts before and after the acquisition of symptoms.

It is an open question as to whether the symptoms displayed in organic alexithymia have the same character and mechanisms as other forms of alexithymia. Given the similarity of symptoms, there should be an acknowledged possibility of similar aetiology, which can guide without limiting research.

The literature suggests the need for work on issues of co-diagnosis and/or differential diagnosis [1, 2]. Links between alexithymia and certain psychosomatic disorders have been established. For example, Porcelli *et al.* [3] reported a positive correlation between alexithymia and patients suffering from functional gastrointestinal disorders (FGID). The construct was originally associated exclusively with psychosomatic disorders, but it is now observed in a range of other presentations, diagnosed using tools such as the DSM. Taylor *et al.* [4] hypothesized that alexithymia is related (perhaps aetiologically) to psychological traits found in patients who have been diagnosed with an eating disorder. This has received partial support from other studies [5]. Some studies have reported high rates of alexithymia in patients with panic disorders [6, 7]. There is also evidence of an association between alexithymia and patients diagnosed with Post-Traumatic Stress Disorder (PTSD) [8, 9]. The specific contribution and/or causal direction of these relationships is yet to be clarified. Taylor *et al.* [1] stated that, although the alexithymia construct shares variance with some psychopatholgies, current results led them to believe that alexithymia is not just a secondary response to other disorders.

The aetiology of traditional alexithymia has been investigated from different angles. Psychoanalysts have emphasized the influence of developmental factors and early deficiencies [1], particularly the effects of traumatic experiences [8]. A higher incidence of alexithymia has been reported in PTSD patients [9]. Some authors have reported poor processing of emotions in college students scoring high on an alexithymia scale [10]. Others suggest that alexithymia may be primarily related to poorly developed verbal ability [11]. One study showed that a set of family-environment variables predicted alexithymia in a multiple regression analysis study utilising university students [12].

In the context of ABI, perhaps the most relevant theories regarding the aetiology of alexithymia are those related to putative neurophysiological factors. One prominent theory suggests that alexithymia is associated with functional commissurotomy [13]. Functional commissurotomy is a state of inhibition of neuronal transmission across the corpus callosum, which resembles the effects of cerebral commissurotomy, the operation that surgically divides the corpus callosum [14]. The theory associating non-organic alexithymia with functional commissurotomy was based on the demonstration of alexithymic symptoms by 12 patients after cerebral commissurotomies [13]. Some of the features displayed by these patients resembled alexithymia. The authors postulated that a deficit in interhemispheric communication is the basis of this disorder. Another important study examined combat veterans diagnosed with PTSD. It found that the efficiency of interhemispheric communication, assessed by a tactile finger localization task, predicted alexithymia [15]. Results in the control condition on the speed of information processing of each hemisphere allowed the investigators to conclude that the problem was poor communication between hemispheres, not a unilateral hemispheric deficit. In direct and indirect ways, this hypothesis has received support from the literature [16–18]. The proponents of this view suggest that there is an inter-

ruption of the normal flow of communication between hemispheres in the brains of patients with alexithymia. This is thought to impair the transfer of emotions from the right hemisphere to the 'language centre' of the left hemisphere, where labelling of the emotion would take place.

Another view is that alexithymic patients suffer a deficit in emotional processing in the right hemisphere [1, 19]. One group investigating this claim assessed university students' alexithymia scores and hemispheric activation through their Conjugate Lateral Eye Movements. They found a relationship between right hemisphere activation and alexithymic features [20]. Similar results were reported following a study of the relationship between alexithymia and hemispatial bias in university students [21]. The authors reported a correlation between the levels of alexithymia and levels of right hemisphere activation.

Another theory suggests that alexithymia may be related to a deficit in anterior cingulate cortex activity during emotional arousal [22]. Lane *et al.* [22] used 12 right-handed females whose emotional awareness was assessed before PET imaging. The authors reported a strong correlation between emotional arousal and blood flow in the cingulate cortex.

Of several scales developed to assess alexithymia, that developed by Taylor *et al.* [23] has been the most well validated. A new version of this scale, the Toronto Alexithymia Scale (TAS-20) [24] is a 20-item self-report questionnaire. The authors postulate the existence of a three-factor structure, which is congruent with the alexithymia construct. The three factors are: (1) Difficulties identifying feelings; (2) Difficulties describing feelings; and (3) Externally oriented thinking. This test has demonstrated strong factorial validity [25]. In essence, the TAS-20 includes seven questions covering factor 1, five questions covering factor 2 and eight questions covering factor 3.

With this conceptual and technical background, many interesting research questions present themselves, with various theoretical and clinical implications. Possible differences between organic and non-organic alexithymia remain to be described. Significant numbers of papers in the traditional literature report findings using university students. However, the proportion of cases of organic alexithymia in the population of people with ABI has not been investigated. There appears to be a strong potential for underreporting of the phenomenon, for three reasons. First, seemingly flat affect might elicit immediate suspicions of depression, a common sequela of ABI. This may lead the therapist to tackle depression in a traditional fashion, neglecting in some cases the primary condition. Secondly, difficulties in identifying and expressing feelings might mask this condition. In this case, therapists might focus on other issues endemic to the population of people with ABI, such as family, legal and financial problems.

Thirdly, it is possible that diagnosticians might be tempted to use the category of Dysexecutive Syndrome (DES), also known as 'Frontal lobe syndrome'. The latter label is currently out of favour because it is thought to be too specific in locus. Unfortunately, the existence and assessment of DES is still the subject of debate. A practical clinical approach to this area identifies DES as a cluster of deficits in self-monitoring, self-direction, self-care, planning, problem solving skills, and processing environmental feedback [26–28]. DES has also been associated with emotional disturbance [29]. Investigators have postulated that emotional disturbances in DES may be related to lesions in circuits between the frontal cortex and limbic system. These lesions would lead to an inability to withhold

responses to negative stimuli, increased aversion reactions, and lack of regard for consequences of their behaviours. DES is diagnosed with the aid of formal tests of planning, use of feedback and problem solving, such as the Wisconsin Card Sorting Test, Towers of Hanoi and some Maze Learning Tests [28]. However, a clinical interview with the family of the client is generally also considered necessary, as the symptoms of DES may be more obvious in everyday life activities than formal testing [28, 30, 31]. As an ambiguous affective condition amongst a cluster of executive deficits, organic alexithymia might be readily subsumed under the broader label. As this case study will demonstrate, however, it is possible to observe symptoms of organic alexithymia in the absence of the broader syndrome. This suggests that it might be useful treating DES and organic alexithymia as different constructs.

There are also interesting research topics to explore in the neuropsychological domain. For instance, there may be a correlation between poor visualization or constricted imaginal processes, and tests of visuo-spatial and visuo-constructional skills. On the other hand, there may be links between alexithymia and interhemispheric communication, or other neuropsychological indices such as information processing speed or IQ.

The clinical implications of research into organic alexithymia might also be very useful in practice. For example, if depression, organic alexithymia and DES could be clearly disambiguated, then therapy and rehabilitation could be adjusted appropriately. It has been suggested that therapy for alexithymia should focus on enhancing patients' emotional awareness, including attempts to re-direct patients attention to non-verbal manifestations of their emotions [32]. These suggestions appear to be applicable to the general population. However, within the population of people with ABI, therapy would need to be adjusted to accommodate potential cognitive deficits and general psychosocial difficulties specific to the ABI domain. Even if symptoms were identical across organic and non-organic alexithymia, an organic aetiology might eliminate the effects of some techniques, as the absence of some psychological function is less malleable than the unusual development of that function. Appropriate recognition and treatment of organic alexithymia may be particularly important, as the intake criteria of some psychiatric institutions exclude disorders with organic aetiologies. This might lead to inadequate care for patients with organic alexithymia. Recognition of the distinct condition of organic alexithymia could help alleviate such a failure.

Therefore, the purpose of this case study is to illustrate the construct of organic alexithymia and characterize it by comparing it to the established construct of alexithymia. The structure of the paper is guided by the hypotheses that a syndrome substantially similar to alexithymia can occur in people with ABI, and that the acquired nature of the organic disorder may cause it to be systematically different from the established syndrome.

Method

Case history

HR is a 21-year-old right-handed male who was referred to the State Head Injury Unit, Perth, for therapy due to anger-related issues. He was involved in a motor-

cycle accident without a helmet in mid 1998 and sustained a closed head injury. HR was admitted with a Glasgow Coma Scale of 3, and spent 28 days in the Intensive Care Unit. Subsequently, he spent 10 weeks in a rehabilitation hospital. HR's clinical presentation at the emergency room included: fracture of the left frontal bone extending to the roof of the orbit; traumatic fluid levels in the sinuses; intracranial blood within a few areas of the brain; traumatic blood in the subarachnoid space; contusions of the right frontal lobe and right temporal lobe; diffused cerebral swelling with effacement of the sulci; wedge fracture of T9 (acute) and wedge fracture of T10 (acute); and minor body wounds. In the Intensive Care Unit, an elevation of intracranial pressure was treated with intraventricular drain and drugs. On admission to the Rehabilitation Hospital, neurological examination indicated: a full third nerve palsy on the right; a left partial third nerve palsy; fixed and dilated right pupil; left pupil dilated, but slowly reacting to light; limited movements of left eye; inconsistent hand squeeze to command; confusion; obvious upper motor neuron involvement of all limbs. Outpatient programmes for occupational therapy, speech therapy and physiotherapy were organized before HR was discharged.

Upon beginning clinical counselling at the State Head Injury Unit, HR reported that his anger problems were mainly due to frustration and perceived lack of understanding. Most of the problematic incidents took place at home. During the first interview, he reported that he could not easily identify the feelings he was experiencing, which led to frustration and depression. HR stated that, while he was socially active before his accident, he had since become withdrawn and shy. He also reported having fewer dreams than before. His mother corroborated HR's account and added that she thought that his 'emotional confusion' was currently his main difficulty. Irritability and aggression were treated following a standard format used at HIU. The following is a simplified version of this intervention:

(a) *Education about anger.* This included identification of cognitive and physiological changes when angry, triggering factors, benefits of and problems with anger, etc.;
(b) *Controlling anger.* This included calming techniques, relaxation, time out, etc.; and
(c) *Planning for future risks.* This included benefits from planning, building plans, anticipating situations and building alternatives, readiness, etc.

HR engaged in therapy for a few sessions and showed considerable positive gains in relation to the anger outbursts at home. However, his difficulties with the identification of his feelings persisted. Further assessment was considered appropriate.

Neuropsychological testing

Except where noted, the subject was administered a standard battery of neuropsychological tests including the Wechsler Adult Intelligence Scale-Revised Version (WAIS-R) [33]; the Wechsler Memory Scale (WMS) [34]; the Rey Auditory Verbal Learning Test [35]; the Rey-Osterrieth Complex Figure test [36]; the Controlled Oral Word Association test [37]; the Category Naming test (animals) [38]; the Symbol Digit Modality Test [39]; the Trail-Making Test [40], and the Austin Maze test [41].

Psychological measures

HR's psychological state was measured with the Toronto Alexithymia Scale (TAS-20 form) and the Beck Depression Inventory (BDI) [42]. The BDI administration followed the standard procedure. As the TAS-20 gives an indication of the 'current' presence of alexithymia, the subject was additionally asked to estimate each item 'before' the brain injury. In that way, a contrast between the subject's estimate of the current symptoms and pre-morbid symptoms could be conducted. The difference in the general score is the discrepancy score. Given that people with ABI often suffer from memory difficulties and experience confusion about their pre-morbid state, HR's mother also completed a modified version of the TAS-20 with questions about HR's present and pre-morbid emotional state. An example of the modifications is given for item (1): 'I am often confused about what emotion I am feeling' (original); this item was modified to: 'He is often confused about what emotions he is feeling'. In summary, both HR and his mother were asked to rate his emotional capacities on the questions of the TAS-20 at present and before the accident.

Procedure

All tests listed above were administered as described in the relevant reference. The only non-standard test, the Austin Maze, is described here in greater detail. The subject is presented with a maze consisting of a 10×10 grid of square buttons with blank white faces. Patients are told that they must work out a path from the bottom left square to the top right square by trial and error. When they stray from the correct path, they are warned by a red light and a beeping sound. Several rules are also described: they must only move one square left, right, up or down; skipping, moving diagonally or repeatedly pressing the same button are treated as errors. Subjects are allowed 10 trials to determine the correct path, which does not change from trial to trial.

Results

Neuropsychological profile

Table 1 reports HR's neuropsychological profile. His overall profile indicated selective areas of deficit. His Full Scale IQ of 83, in the *low–average* range, is consistent with his educational and work history. Inspection of WAIS-R sub-tests suggests poor pre-morbid schooling (Information) and some residual cognitive slowing (Digit Symbol). HR's Performance and Verbal IQ scores were not statistically different, although there was a slight advantage to Performance, as expected.

HR's visuospatial abilities appear to be within the normal range, as assessed by the Block Design sub-test of the WAIS-R and the Rey Complex Figure Test (RCFT). However, HR's reproduction of the RCFT figure from memory following a 3-minute delay was at the 30th percentile. While still within the normal range, this demonstrated poor incidental recall of non-verbal material given his copy performance. HR's performance on the Austin Maze was mixed. His overall performance placed him within the 50th percentile, but his error reduction was erratic. A qualitative analysis of his performance suggests some difficulties with his non-verbal learning, and problem solving. HR's score on the Hooper Visual

Table 1. Results of HR on neuropsychological measures

	HR	Comparison
WAIS-R (scale scores)		
Information	5	
Digit span	8	
Arithmetic	7	
Similarities	7	
Picture completion	8	
Object assembly	10	
Block design	10	
Digit symbol	5	
Verbal IQ	83	
Performance IQ	87	
Full Scale IQ	83	
WMS (raw scores)		
Information	4	
Orientation	4	
Mental control	8	
Logical memory	5	$M = 11.5$; SD = 6.66
Delayed logical memory	2	$M = 9.92$; SD = 6.67
Digit span	10 (forward = 5; back = 5)	
Visual reproduction	11	$M = 10.5$; SD = 1.9
Delayed visual reproduction	12	
Paired associate learning	11	
MQ	82	
Hoopers	24.5	Mild organic deficit
RAVLT: Total words	32	$M = 53.4$; SD = 5.4
List b	3	$M = 6.9$; SD = 1.9
6th trial	2	$M = 11.2$; SD = 1.6
Recognition	7	$M = 14$; SD = 0.9
RCFT: Copy	35	90%ile
Memory	19	30%ile
COWA	24	<10%ile
TMT: A	46 s	10%ile
B	95 s	20%ile
Austin Maze—10th trial	60	$M = 61.1$; SD = 49.9

Organization Test was suggestive of a mild organic deficit in the ability to mentally manipulate fragmented images. This may be related to difficulties in maintaining mental image problems with conceptual recognition of abstract shapes or both.

HR also demonstrated some language difficulties, with remarkably poor word production on the COWA, and markedly impaired memory for the RAVLT word lists, both for list B, and for list A following interference. This is suggestive of both proactive and retroactive interference.

HR's overall MQ (82) was not different from his Full Scale IQ. His scores on the non-verbal sub-tests were in the normal range, while his weakness on the verbal sub-tests was a little worse than might be expected given his background.

Assessment of the potential presence of Dysexecutive Syndrome (DES) was conducted via formal testing and a clinical interview with the client and his mother. On interview, the salient feature of HR's presentation was his inability to identify and communicate his emotions and his irritability, which was reported by both his mother and himself. His irritability appeared to be accentuated at home, and he seemed to exercise control over his reactions in other environments. Thus, there did not appear to be a cluster of symptoms indicating DES. On formal testing, his score on the Austin Maze Learning Test placed him within the 50th percentile.

Psychological measures

HR's results on the Beck Depression Inventory placed him within the 'mild mood disturbance' range. See table 2 for items endorsed by HR on the BDI. At the HIU, scores that reflect objective changes after an ABI are discounted from the total score as they do not reflect mood or 'subjective views'. In his case, for example, his choice in item 14 reflects his accurate perception of physical changes on his face (scars on forehead and side of the face). On item 15, his choice reflects objective weight loss due to changes as a consequence of physiotherapist's recommendations on dietary issues but not loss of appetite. Subtracting the pertinent scores, HR falls within the normal range.

Table 3 reports the TAS-20 results. The first thing to note is the enormous difference in HR's self-rating before and after the accident. This supports the clinical impression, and HR's report, of symptoms consistent with alexithymia at the time of testing. It also supports the hypothesis that HR's alexithymic symptoms have developed since the accident. The pattern of scale scores suggests that HR believes that he has become less able to identify and describe his feelings, although he has not noticed the pattern of external thinking.

HR's mother's answers endorse a similar pattern, although these results should be interpreted with caution pending the development of a tool specifically designed to measure alexithymic symptoms through the observations of others. Nevertheless, her scores indicate that HR's behaviour before the accident was fairly normal, while since the accident he has become much less able to identify his feelings. It is interesting that HR's scores suggest that he has noticed a difference in his ability to describe and identify his feelings, while his mother has only noticed a difference in his ability to identify his feelings. This probably reflects the nature of those capacities, one of which, describing feelings, is probably only accessible to the

Table 2. Items endorsed by HR on the Beck Depression Inventory [a]

4.	I don't enjoy things the way I used to.
5.	I feel guilty a good part of the time.
8.	I am critical of myself for my weaknesses or mistakes.
10.	I used to be able to cry, but now I can't cry, even though I want to.
13.	I have greater difficulty in making decisions than before.
14.	I feel that there are permanent changes in my appearance that make me look unattractive.
15.	It takes an extra effort to get started at doing something.
16.	I wake up several hours earlier than I used to and cannot get back to sleep.
19.	I have lost more than five pounds.

[a] Items missing are those in which HR chose option '0'.

Table 3. HR's and his mother's scores on the Toronto alexithymia scale (TAS) and the three factors, both before and after the ABI

	Before ABI	After ABI	Discrepancy score[a]
Total TAS: Self	41	71	30
Identifying feelings	7	24	17
Describing feelings	8	25	17
External thinking	20	16	– 4
Total TAS: Mother	52	68	16
Identifying feelings	14	26	12
Describing feelings	12	16	4
External thinking	20	20	0

[a] The discrepancy scores were obtained by subtracting the scores on the estimates of each item 'after the ABI' from the estimates on each item 'before the ABI'.

subject, as only the subject can identify whether descriptions are accurate or not. Simple production of emotional behaviour is evidence of the identification of feelings, which anyone can observe.

It is significant that the discrepancy before and after the accident is caused by only two of the three factors, identifying and describing feelings. Neither HR nor his mother noticed a difference in external thinking. This supports the hypothesis, based purely on clinical judgement, that patients with organic alexithymia are less likely to demonstrate this facet of non-organic alexithymia, as their symptoms are associated with organic damage rather than long-established personality characteristics.

It is also possible that the wording of the external thinking factor may present difficulties for people with ABI. Both HR and his mother reported problems with these questions. This probably reflects the difficulty of using an instrument developed for use in the general population (usually with university students, an atypical sub-group) with a group as unusual and heterogeneous as people with ABI. HR tended to answer these questions with the neutral answer, and his comments often indicated a lack of understanding of or interest in these questions, in contrast to his engagement with the other questions.

Discussion

HR's case suggests that there exist in the population of patients with ABI people who have acquired significant and debilitating symptoms originally identified with the personality disorder alexithymia. HR and his mother both reported that, despite a normal history, following brain injury HR's ability to identify and describe his feelings was impaired. However, neither HR nor his mother noticed changes in the tendency to an external thinking pattern.

This case demonstrates that a condition that one has chosen to call organic alexithymia can exist in a population with an organic aetiology, and that it may have characteristics in common with alexithymia. However, it is not unlikely that it will be significantly different in some ways, due to the lack of a cluster of personality factors developed over time, as well as the presence of characteristics of brain injury, such as cognitive slowing, anger problems, impulsivity, and so on. These similarities and differences have implications for treatment, not least the fact that personality

disturbances with an organic aetiology may escape treatment, particularly if they are not recognized as a diagnostic group.

The term 'organic' was favoured to describe patients whose symptoms have developed as a consequence of a brain injury, rather than an early classification suggested by Sifneos [43]. 'Secondary' alexithymia refers to alexithymic symptoms resulting from developmental arrests, psychological traumas and the like, whereas 'primary' alexithymia refers to symptoms developed as a consequence of neurobiological deficits (including genetic defects). This classification does not help differentiate the aetiological contributions of environment and genetic disposition. Particular patients may have developed alexithymia as a result of a combination of 'primary' and 'secondary' contributing factors. Taylor *et al.* [1] rightly suggest that the aetiology of alexithymia involves multiple factors, including inherited variations and family and social environment. The term 'organic' would help to disambiguate the presentation of individuals in whom alexithymic symptoms have developed over time, and those in whom symptoms have appeared *de novo* after an acquired brain injury.

HR appeared to be suffering significant distress as the result of an inability to identify and describe his feelings. This distress was shared by his mother, and is likely to have played a part in HR's increasing social isolation. The lack of change in characteristics of external thinking is consistent with clinical intuition, but may also reflect the inadequacies of a general instrument used with a special population. The acquired nature of organic alexithymia also points to the need for a specialist instrument that can measure the changes in alexithymia over time, which is less important in personality disordered patients who, it may be assumed, have developed alexithymia as part of a personality cluster. One has initiated an effort to develop such an instrument using the population of patients with ABI passing through the State Head Injury Unit. This is designed to identify: (a) the presence of alexithymia in the population of people with ABI before and after ABI; (b) possible competing explanations for alexithymic symptoms, such as depression; (c) cognitive patterns in patients with and without organic alexithymic symptoms; and (d) brain lesion sites associated with organic alexithymia. This effort will shed light on the organic hypotheses of the aetiology of alexithymia (lesion site) and potential correlations with other medical information (length of PTA, type of injury, etc.).

To the authors' knowledge there is little research into these issues. One recent study does investigate the presence of alexithymic symptoms in a population of people with ABI [44]. However, in this study subjects' head injuries were self-reported, and they were recruited from a family practice service, so objective measures such as PTA and GCS were not used. Furthermore, control for competing explanations (e.g. depression levels) were not considered. Most importantly, this study does not shed light on the existence of alexithymia before the reported head injury and, therefore, cannot rule out a pre-morbid aetiology. Therefore, while it does establish the presence of alexithymic symptoms in a sample of people with ABI, it cannot effectively illuminate the role of ABI in the acquisition of those symptoms.

HR's case may be compared with the different hypotheses of the aetiology of alexithymia. Clearly, organic alexithymia shares some characteristics with non-organic alexithymia. However, it remains to be determined whether the phenomenological similarity is due to neurological or clinical similarities. Depression cannot account for HR's emotional profile, as he did not appear to be depressed and his

difficulties were not related to 'sad' emotions but to 'difficulties in identifying and describing them'. This was supported by his answers on the BDI.

HR's case does not strongly support any of the neurophysiological theories about the genesis of alexithymia. His injuries were for the most part confined to the surface areas of the cortex, particularly in the frontal and parietal lobes. This is not consistent with a functional commissurotomy. The damage was fairly evenly divided between the left and right hemispheres, thus making it difficult to assess the right hemisphere activation hypothesis. Finally, the areas of the anterior cingulate cortex were not visibly damaged, suggesting that HR's alexithymic symptoms cannot be explained by the ACC theory of alexithymia.

HR's injuries, and the neuropsychological results (principally impaired verbal and non-verbal learning and memory) suggest that, while he can remember and analyse information when it is simple and repeated, his faculties are impaired by either complexity or interference. He seemed to have difficulty in organizing efficient learning of new situations. This pattern is unlike any of the extant theories of alexithymia, and not greatly similar to a Disexecutive Syndrome. It suggests that the aetiology of organic alexithymia involves different factors to that of non-organic alexithymia.

Therefore, it appears that HR, while demonstrating the salient features of alexithymia, cannot be explained within the current theoretical environment. In order to examine the construct of organic alexithymia thoroughly, it is necessary to develop instruments capable of measuring alexithymic symptoms, and disambiguating them from other conditions such as depression, within a population of people with ABI. A study has been initiated that will determine the empirical characteristics of alexithymia, brain damage and psychological distress among ABI patients, which should begin to answer some of the questions raised by this case study.

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