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## THE OVERLAP BETWEEN ALEXITHYMIA AND ASPERGER'S SYNDROME

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Dear Editor

Alexithymia is characterised by an inability to express emotions verbally and by impoverished fantasy and imaginal life. Individuals so affected may also experience difficulty in distinguishing emotional states from bodily sensations. Alexithymia is thought of as a psychosomatic disorder since the inability to identify and verbalise feelings may predispose to somatization. The term was coined by Sifneos (1972) but descriptions of conditions with alexithymic features predate this work. Aetiological theories of the disorder have variously emphasised genetic, neuropsychological, developmental and psychodynamic factors (Krystal, 1998; Parker & Taylor, 1997). Like Alexithymia, Asperger's disorder is also characterised by core disturbances in speech and language and social relationships. Here we aim to demonstrate that there is considerable overlap in the clinical presentation of persons with a diagnosis of Alexithymia and Asperger's syndrome.

As John Nemiah (1996) points out there is now a large literature devoted to the construction of standardised Alexithymia rating scales and their applications to clinical research. Alexithymia may now be measured as a valid and reliable clinical phenomenon (Nemiah, 1996, 1977). It is interesting that the description of Alexithymia focusing on aetiology and treatment all are similar to the literature on Asperger's syndrome. Taylor, Bagby and Parker (1997) describes persons with Alexithymia as having difficulty in describing feelings and having difficulty in distinguishing between feelings and bodily sensations. He also points out their difficulties with affective self-regulation. Thus sufferers may have difficulties in the appraisal and expression of emotion and in the ability to use feelings to guide behaviour. Indeed Taylor et al. (1997) state that persons with Alexithymia “know very little about their own feelings and, in most instances, are unable to link them with memories, fantasies, and higher level effects, or specific situations” (page 29). It is argued that the inability of the patient with Alexithymia to express and modulate feelings, may lead to a discharge of tension through, for example, impulsive acts or compulsive behaviour such as binge eating (Bagby & Taylor, 1997).

### COGNITIVE PROBLEMS IN ALEXITHYMIA/ASPERGER'S SYNDROME

Taylor et al. (1997) notes that individuals with Alexithymia have problems with introspection, poor capacity for fantasy, and that they show a stimulus bound, externally orientated cognitive style. Indeed James Grotstein (in Disorders of Affect Regulation by Taylor et al., 1997) describes Alexithymia as “an affect processing disorder that interrupts or seriously interferes with the organisms self-organising and reorganising processes” (page 12). This means that they have a diffuse sense of self. Indeed there is an earlier disorder called Pensee Operatoire (operative thinking) described by Marty and de M'Uzan (1963) where there is a similar utilitarian thinking style to that seen in Alexithymia and indeed

Asperger's disorder. Krystal (1998) has described patients with La Pensee Operatoire as showing a “dull, mundane, unimaginative, utilitarian, and sequential recitation of concrete facts” (page 246). He also notes that these patients show a cognitive style where there is an “absence of the human quality (which) contributes to making these patients thoughts “operative” or thing orientated” (page 247). Such patients may often be described as dull, colourless and boring even when they are intellectual and clever (Taylor, 1984). Of course many persons with Alexithymia or Asperger's syndrome or La Pensee Operatoire can operate very well or indeed at superior levels in their work as mathematicians, engineers, etc. (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001).

## **PROBLEMS WITH SOCIAL RELATIONSHIPS IN ALEXITHYMIA AND ASPERGER'S SYNDROME**

Taylor et al. (1997) noted that Alexithymic individuals are “unable to identify accurately their own subjective feelings, verbally communicate emotional distress to other people very poorly, thereby failing to enlist others as sources of aid and comfort” (page 30). In addition they note that persons with Alexithymia have a preference to be alone or avoid people altogether. Krystal (1998) notes their tendency “to be like a rock or an island” and that “the degree of detachment in relation to others and their attitudes towards themselves is sometimes so flat that they feel psychotic to the interviewer and give the impression that the patient has deadened his object and self-representations or at least sapped it of all humanity” (page 246). Wing's (1981) criteria for Asperger's disorder (Asperger, 1944) emphasise a lack of normal interest and pleasure in people around them and a significant reduction in shared interests. Thus difficulties with social relationships are common to both Alexithymia and Asperger's disorder. Krystal notes the tendency of Alexithymic individuals to treat people as if they were machines. McDougall (1984) notes their great fear in intimacy situations and the problems experienced by these individuals in understanding social groups. Persons with Asperger's syndrome have similar problems. They have problems with social “know-how,” have empathy deficit and are often very over controlling, and have difficulty reading the non-verbal cues other people demonstrate in social interaction.

## **SPEECH AND LANGUAGE PROBLEMS IN ALEXITHYMIA AND ASPERGER'S SYNDROME**

Taylor et al. (1997) describes persons with Alexithymia as showing speech which is deficient in nuance, meagre in the use of metaphor and lacking affect, which of course is typical of the kind of speech that persons with Asperger's syndrome produce. He also notes that their language is characterised by flatness, banality and is fact based. Krystal (1998) notes that “the associations of these patients are characterised by an almost complete absence of thoughts relating to inner attitudes, feelings, wishes, or drives; and a recounting in great and often boring detail, of events in their external environment and their own actions in this setting” (page 248) and they also show a decreased use of the pronoun I. They show clear impairment in symbolic function. Indeed McDougall (1978) observed that Alexithymic patients use speech as “an act rather than a symbolic means of communication of ideas or affect” (page 45). Krystal noted that these patients with Alexithymia showed a “poorness and flatness of the contents of their communication” (page 246) as shown by their prosody and their monotone tone of voice. They can learn approximately socially appropriate responses but these are often excessively formal but these expressions lack nuance, lack proper prosody, are often very formal, and pedantic. They strike the listener as being somewhat unusual or overly formal. Speech and prosodic abnormalities are widely reported in autism spectrum disorders, including Asperger's disorder (Shriberg et al., 2001). Such abnormalities are perhaps indicative of dysfunction to the right hemisphere (see below)

in these disorders. Krystal also observes that patients with Alexithymia “give a boring recital of information with a mentality approaching that of mental retardation” (page 272). This description is again very similar to Asperger's syndrome. In our own clinical experience (M.F.) we have noted that it is not uncommon to under estimate the IQ of some persons with Asperger's syndrome. Indeed, the opposite can also happen because of the great knowledge apparent in a single narrow area.

## **NON-VERBAL BEHAVIOUR IN ALEXITHYMIA AND ASPERGER'S SYNDROME**

Bagby and Taylor (1997) note “a somewhat stiff wooden posture, and a prosody of facial emotional expression” (page 29) in patients with Alexithymia. They also note their problems in identifying non-verbal facial emotion. Difficulties in the perception of facial emotions expressed by others is a cardinal feature of autism spectrum disorders. Damasio and Maurer (1978) suggested that autism is accompanied by dysfunction within mesolimbic brain circuits, including the ventromedial prefrontal cortex, medial temporal lobe, striatum and limbic thalamus. Damage to these areas causes deficits in social and emotional functioning, stereotype and obsessionality that also characterise autism spectrum disorders. Indeed, functional imaging studies have reported aberrant activation patterns within the above defined circuitry when autistic individuals perform tasks requiring the recognition of facial expressions (e.g., happy, sad, disgust) (Ogai et al., 2003).

It appears from this discussion that there is considerable overlap between Asperger's syndrome and Alexithymia with social interactional difficulties, unusual language, problems in non-verbal behaviour, problems with affective interaction, and a rather factual based thinking style. In addition Alexithymia like Asperger's syndrome, presents as a dimensional, rather than categorical, construct, suggesting multiple aetiological routes.

## **AETIOLOGY OF ALEXITHYMIA**

At an aetiological level there also appears to be overlap between Alexithymia and Asperger's disorder, in terms of the brain circuitry that is thought dysfunctional in these conditions. One source of information regarding the cerebral localisation of affective function has come from patients who have undergone commissurectomies. Hoppe and Bogen (1977) reported on 12 commissurectomized patients who experienced, among other difficulties, Alexithymia. Cerebral commissurectomies also result in impoverishment of dreams and fantasies, and difficulty in describing feelings, as well as a pronounced operative style of thinking. A similar clinical presentation has also been reported in a case of agenesis of the corpus callosum. Hoppe and Bogen (1977) has postulated that Alexithymia may involve interruption of the normal flow of information between the two hemispheres. An alternative hypothesis to a deficit in inter-hemispheric communication is that the right-hemisphere itself may be dysfunctional in Alexithymia (Krystal, 1998). Supporting evidence for this proposition comes from lesion studies that demonstrate abnormal prosody in right-hemisphere patients. Prosodic abnormalities are a feature of both Alexithymia and Asperger's disorder. The right-hemisphere may also be specialised for processing the whole, rather than the detail. Patients with right-hemisphere lesions show deficits on visual-perceptual tasks where they are required to process the whole at the expense of the detail (Robertson, Lamb, & Knight, 1988). Influential cognitive theories of autism, such as weak central coherence, propose an alternative information processing style such that affected individuals show detailed-focussed processing that occurs at the expense of the global form (Happe, 1999). This processing style may extend to the semantic domain where for example, people with autism spectrum disorder do not show the usual advantage for recalling sentences better than unconnected word strings (Hermelin & O'Connor, 1967). It would be

interesting to investigate whether individuals with Alexithymia demonstrate similar behavioural profiles in verbal and perceptual tasks. Persons with Alexithymia have greater tendencies to alcohol abuse, “psychosomatic complaints,” and have a tendency to social conformity. Alexithymia has been seen as a response to chronic illness. Clearly however differences exist between aetiological accounts of the disorders. Whereas Asperger's syndrome is a neurodevelopmental disorder, Alexithymia may be more of a personality trait.

Despite the apparent overlap between the disorders, aetiological differences may exist. One point of difference may lie in the genetic and neurochemical underpinnings of the disorders. Although not conclusive, a number of studies have indicated an association between polymorphisms of the Serotonin transporter gene and Autism Spectrum Disorder (Cook *et al.*, 1997; Kim *et al.*, 2002). By contrast associations with polymorphisms of the dopamine system have not been reliably identified in Autism Spectrum Disorders. In a recent study Ham *et al.* (2005) reported an association between the catechol-*O*-methyltransferase Val 108/158Met polymorphism and Alexithymia. Interestingly, an association was reported with the Valine allele which is associated with lowered prefrontal dopamine tone and is also thought to confer a small amount of risk to schizophrenia (Egan *et al.*, 2001). In contrast to Autism Spectrum Disorders, Ham *et al.* did not report any association between Alexithymia and polymorphisms of the Serotonin transporter gene. These very preliminary findings suggest a greater involvement of the dopamine system in Alexithymia and the Serotonin system in Autism Spectrum Disorders. Other points of difference between the disorder lie in the greater tendency on the part of persons with Alexithymia, relative to those with Asperger's disorder, to alcohol abuse, psychosomatic complaints and social conformity. Alexithymia has also been seen as a response to chronic illness.

## TREATMENT OF ALEXITHYMIA

Not surprisingly, persons with Alexithymia, like persons with Asperger's syndrome, show a very poor response to psychoanalytic psychotherapy (Freyberger, 1977; McDougall, 1982). Alexithymic patients need a more cognitive behavioural therapy like patients with Asperger's syndrome. Krystal (1998) described patients with Alexithymia as being “antianalytic.” The same could be said of persons with Asperger's syndrome. Formal psychoanalytic psychotherapy is an impediment with patients with Alexithymia (Task Force Report of American Psychiatric Association, 1989) and Asperger's syndrome (Klin & Volkmar, 2000a, b).

Ego psychological approaches which is similar to cognitive behavioural therapy are helpful. These work better with patients who have problems working with feelings and phantasies.

It is important that psychiatrists making a diagnosis of Alexithymia consider Asperger's syndrome in the differential diagnosis.

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## REFERENCES

- Asperger H. Die ‘autistischen Psychopathen’ im Kindesalter. Archives für Psychiatrie und Nervenkrankheiten. 1944; 117:76–136.
- Baron-Cohen S, Wheelwright S, Skinner K, Martin J, Clubley E. The autism spectrum quotient: Evidence from Asperger's syndrome on high functioning autism, males and females, scientists, and

- mathematicians. *Journal of Autism and Developmental Disorders*. 2001; 31:5–17. [PubMed: 11439754]
- Cook EH, Courchesne R, Lord C, Cox NJ, Yan S, Lincoln A, Haas R, Courchesne E, Leventhal BL. Evidence of linkage between the Serotonin transporter and autistic disorder. *Molecular Psychiatry*. 1997; 2(3):247–250. [PubMed: 9152989]
- Damasio AR, Maurer RG. A neurological model for childhood autism. *Archives of Neurology*. 1978;35. [PubMed: 718482]
- Egan MF, Goldberg TE, Kolachana BS, Callicott JH, Mazzanti CM, Straub RE, Goldman D, Weinberger DR, et al. Effect of COMT Val 108/158 Met Genotype on Frontal Lobe Function and Risk for Schizophrenia. *Proceedings of the National Academy of Science*. 2001; 98(12):6917–6922. [PubMed: 11381111]
- Freyberger, H. Supportive psychotherapeutic techniques in primary and secondary Alexithymia. In: Brautigam, W.; von Rad, M., editors. *Theory of Psychosomatic Disorders*. Basil: Karger; 1977.
- Freyberger, H. Howels modern perspective in the psychiatric aspects of surgery. New York: Brunner-Mazel; *Psychosomatic aspects of an intensive care unit*.
- Ham BJ, Lee MS, Lee YM, Kim MK, Choi MJ, Oh KS, Jung HY, Lyoo IK, Choi IG. Association between the Catechol O-Methyltransferase Val 108/158Met Polymorphism and Alexithymia. *Neuropsychobiology*. 2005; 52(3):151–154. [PubMed: 16127282]
- Happe F. Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences*. 1999; 3:216–222. [PubMed: 10354574]
- Hermelin B, O'Connor N. Remembering of words by psychotic and subnormal children. *British Journal of Psychology*. 1967:58. [PubMed: 5588649]
- Hoppe KD, Bogen. Split brains and psychoanalysis. *Psychoanalytic Quarterly*. 1977; 46:220–244. [PubMed: 870923]
- Kim SJ, Cox N, Courchesne R, Lord C, Corsello C, Akshoomoff N, Guter S, Leventhal BL, Courchesne E, Cook EH. Transmission disequilibrium mapping at the Serotonin transporter gene (SLC6A4) region in autistic disorder. *Molecular Psychiatry*. 2002; 7(3):278–288. [PubMed: 11920155]
- Klin A, Volkmar F. Treatment and intervention guidelines for individuals with asperger's syndrome. 2000a
- Klin, A.; Volkmar, F.; Sparrows, S., editors. *Asperger's syndrome*. New York: Guildford Press; 2000b.
- Krystal, H. *Integration and self-healing: Affect-trauma – alexithymia*. New Jersey: Published by Lawrence Erlbaum Associates; 1998.
- Marty P, de M'Uzan M. La Pensee Operatoire. *Revue Francaise De Psychanalyse*. 1963; 27(Suppl.)
- McDougall J. Primitive communication and the use of countertransference. *Contemporary Psychoanalysis*. 1978; 14:173–209.
- McDougall J. Alexithymia, psychotomatois and psychosis. *International Journal of Psychoanalytic Psychotherapy*. 1982; 9:377–388. [PubMed: 7152820]
- McDougall J. The 'disaffected' patient: Reflections on affect pathology. *Psychanalytic Quarterly*. 1984; 53:386–409. [PubMed: 6473578]
- Nemiah J. Alexithymia Psychother. *Psychosomatics*. 1977; 28:199–206.
- Nemiah J. Alexithymia, present, and future. *Psychosomatic Medicine*. 1996; 58:217–218. [PubMed: 8771620]
- Ogai M, Matsumoto H, Suzuki K, Ozawa F, Fukuda R, Uchiyama I, Suckling J, Isoda H, Mori N, Takei N. MRI study of recognition of facial expressions in high-functioning autistic patients. *Neuroreport*. 2003; 14:559–563. [PubMed: 12657885]
- Parker, J.; Tylor, G. The neurobiology of emotion, affect relation, and Alexithymia. In: Taylor, G.; Bagby, RM.; Parker, JD., editors. *Disorders of affect regulation*. Cambridge: Cambridge University Press; 1997.
- Robertson LC, Lamb MR, Knight RT. Effects of lesions of temporal-parietal junction on perceptual and attentional processing in humans. *Journal of Neuroscience*. 1988; 8:3757–3769. [PubMed: 3193178]

- Shriberg LD, Paul R, McSweeney JL, Klin AM, Cohen DJ, Volkmar FR. Speech and prosody characteristics of adolescents and adults with high functioning autism and asperger's syndrome. *Journal of Speech Language and Hearing Research*. 2001; 44(5):1097–1115. [PubMed: 11708530]
- Task Force Report of American Psychiatric Association. *Treatment of psychiatric disorders*. Washington DC: American Psychiatric Association; 1989.
- Taylor, G.; Bagby, R.; Parker, J. *Disorders of affect regulation*. Cambridge: Cambridge University Press; 1997.
- Taylor J. The boring patient. *Canadian Journal of Psychiatry*. 1984; 29:217–222.
- Wing L. Asperger's syndrome. *Psychological Medicine*. 1981; 11:115–130. [PubMed: 7208735]