

Are attachment disorders best seen as social impairment syndromes?

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ATTACHMENT ON AXES 1 AND V

It has often been noted that one of the origins of the paradoxical position of attachment concepts in clinical research lies in different research traditions using the theory. On the one hand, the development of the early attachment research tradition in developmental psychology producing a taxonomy of normative attachment relationships and a wealth of robust research on early socialization. On the other hand attachment disorder concepts within clinical psychopathology, which have had a more intermittent research history although receiving a tremendous boost from recent studies of cross-cultural adoption of severely institutionalized children. Another way of representing this contrast is within the conceptual framework of the multi-axial classification. Here 'attachment disorders' fall within 'Axis 1' – clinical syndromes – and attachment processes fall within 'Axis V' – as a psychosocial influence on development. As O'Connor and Zeanah note, much confusion has resulted from trying to integrate these two approaches: and yet the core rationale of developmental psychopathology contains just this integration of developmental process and clinical symptomatology (Cicchetti, 2001). The integration should thus be beneficial. Part of the difficulty may be that the very term 'attachment disorder' itself conflates these two different logical concepts. 'Attachment...' refers to a relationship concept that is a key experience out of which primary sociability grows (in the sense that early relations with others come to feel vital and useful in their power to assuage personal distress). '...disorder' refers to a clinical syndrome defined in terms of current child functioning.

I want to suggest here that there may be benefit in re-conceptualizing these Axis 1 attachment disorders as disorders (or possibility even just one disorder) of *current social impairment*. This recasting might help logical clarity and also generate testable clinical hypotheses.

ATTACHMENT DISORDERS AS SYNDROMES OF SOCIAL IMPAIRMENT?

O'Connor and Zeanah note that the empirical base of *disinhibited attachment disorder* has been much strengthened recently. The face validity, stability and predictive validity of a syndrome of social difficulties associated with lack of early attachment relationships is now quite well characterized at the behavioural level.

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Evidence of the cognitive and neuropsychological concomitants of this syndrome is increasing. Additional aspects of the presentation are also described; such as attentional difficulties, mood disorder and oppositional disorder. I would argue that these latter may most usefully be characterized as patterns of *co-morbidity* rather than part of a core social syndrome. What could be the argument for privileging the social impairment in this way? Simply that problems in socialization and sociability are not only the salient aspect of the clinical presentation but at the core of the developmental psychology of attachment relationships. Here then the two attachment traditions inform each other in a classic way.

Perhaps the most difficult area in which to make this 'comorbidity' case is for the associated attentional difficulties. An association of core ADD disorders with social impairment is increasingly recognized with clinical studies suggesting that up to 30% of children presenting with complex ADHD show social impairments above threshold on autism screener questionnaires (Taylor and Santosh personal communication). If the social impairment within inhibited attachment disorder was merely that associated with attentional problems in a similar way then there would be no need of a separate attachment disorder. Experience and evidence however indicates that the social impairment of disinhibited attachment disorder is much more than this.

The authors point to the logical argument against including a presumed aetiological component (*viz.* lack of early attachment relationships) within the diagnosis of a current state. There are of course a number of other syndromes that include in their definition a presumed aetiology; for instance, post traumatic stress disorder, acute stress reaction, organic brain syndromes. Nevertheless I agree that there is a good argument *a priori* at this stage not to include the putative antecedent cause in the definition, on the pragmatic grounds that this pre-supposes closure on what the precise aetiology is and confuses Axis 1 and Axis V concepts. (This argument is even more salient in the case of inhibited attachment disorders.) But if the presumed aetiology is not to be included in the definition of the syndrome – why include it in the name? It may be argued that the disorder is marked by *concurrent* disturbance of attachment behaviour or attachment representation. But while this is no doubt the case, the pervasive social impairment of the syndrome is much wider than even a 'broad' (Fox, 1995) concept of attachment could encompass – affecting all aspects of social functioning. Thus it is more logically in my view thought of as a (undoubtedly attachment-related) 'social impairment' disorder rather than an 'attachment' disorder.

As O'Connor and Zeanah state, the situation with regard to *inhibited attachment* disorder (Reactive Attachment disorder, RAD, in ICD 10) is rather more complex. There are several points to be made.

Firstly, it is arguable that what demands our clinical attention and sets RAD apart from other reactive behavioural difficulties is also a social impairment syndrome. The literature on RAD normally contains references to a widespread complex impairment of this sort. Parent groups, who have taken to RAD with intensity, will usually put this forward as the key component of their concern. They say it is impossible to make a 'relationship' with these children; to 'get through to them'; to help them adjust to family life etc. The clinical and research literature on follow-up is full of reports of their difficulties in social adjustment (Hanson & Spratt, 2000).

Secondly, I suggest that the concept of co-morbidity can be applied even more fruitfully in this disorder. As has been often recognized, the notion of RAD is confounded in complex ways with maltreatment syndromes, oppositional disorder, post-traumatic stress etc. Thus if one applies to the problem the existing parallel

literature on type 2 post-traumatic stress disorder in the context of maltreatment (Terr, 1991) or characterizes the behavioural syndrome of oppositional defiant disorder or conduct disorder or mood disorders seen in the syndrome; then all these might usefully be considered as co-morbid with a core social impairment syndrome linked to the 'attachment' part of the problem.

Thirdly, there is the relationship between attachment disorder and disorganized attachment. O'Connor and Zeanah argue that there is evidence of distinction between these concepts, in that their base rate prevalences are very different and the inhibited attachment disorder represents a state of 'non-attachment' rather than distorted attachment. However against this view, there is I think some face validity for much symptomatic commonality between the two entities (see Green & Goldwyn, 2002). It is possible that at least some of the phenomena encompassed by the concept of 'inhibited attachment disorder' can be seen as on the extreme end of a spectrum of attachment disorganization. The symptomatic continuity and what is known about the antecedents of the two conditions would support this. Furthermore, I am not sure that, clinically, inhibited attachment disorder always really represents a 'non-attachment' state: in my experience it commonly presents as a complex, distorted attachment state.

We know a good deal about the consequences of disorganized attachment, both in terms of psychopathology and social adjustment. Although studies have not progressed to the point of being able to define a specific social impairment syndrome associated with disorganized attachment, there is now good early evidence as to the powerful associations between pre-school disorganization and specific patterns of middle childhood peer rejection and social adaptive failure (e.g. Verschuere & Marcoen, 1999; Verschuere, 2001). There is evidence that Attachment disorganization leads to social cognition deficits as well as a strong association with associated psychopathology (Green & Goldwyn, 2002).

The arguments above against including a putative cause in the definition of a current state syndrome are particularly strong in relation to RAD. As Ritchers and Volkmar (1994) argued, the syndrome may include aspects of other child developmental difficulties. While attachment disorganization is highly associated with atypical parenting, there is evidence that in some contexts it may also be associated with child neurological vulnerability (Pipp-Siegal, Siegal & Dean, 1999), language delay (Green, Goldwyn, Peters & Stanley, 2001) and possibly a genetic polymorphism at the DRD4 gene locus (Lakatos *et al.*, 2000, 2002).

Thus, although speculative, I do not think it is unreasonable to postulate a 'core social impairment syndrome' associated with RAD as well. Some of the key additional characteristics of the ICD10 definition of RAD (hypervigilance, mood fluctuation, oppositionality) could be then understood as the influenced by 'co-morbidities' related to associated PTSD, oppositional defiant disorder, depression and so on.

Whether such a putative social impairment syndrome in RAD would be similar to the social impairment syndrome associated with disinhibited attachment disorder is an interesting question that could be tested empirically. Clinical experience of social dysfunction in the two groups certainly sometimes seem similar. From a social cognition viewpoint one might postulate that, in the RAD group, there would be more *distortion or misattribution* of social cognition (related to the maltreatment), compared to a more mentalising type *impairment* of social cognition (related to absence of attachment experience) in the disinhibited attachment disorder group – this at least leads to hypotheses that could be tested empirically.

These two attachment related social impairment disorders might then usefully be considered to lie on a continuum of such disorders within the taxonomy, for instance with the PDD spectrum. (In a way they already do so within ICD10 – this proposal merely makes this more explicit.) Evidence supporting this notion comes from the much-discussed finding of phenocopies of autistic symptomatology in a proportion of the disinhibited attachment disorder group. It also usefully accords with clinical experience, where there is often a difficulty in distinguishing between PDD spectrum and attachment disorder presentations (or their interaction). My own clinical practice mirrors this in running a ‘social development clinic’ that is equipped to deal with a full range of social impairments from PDD to Attachment difficulties – and allowing for their distinction and combination to be assessed and differentially treated. The need in future work would be to characterise the syndrome phenotype more precisely and undertake controlled studies of predictive and concurrent validity and treatment interventions.

Underneath these admittedly rather technical arguments is the reality of the profound impact that disturbance of early attachment relationships can have on later social understanding and functioning in the child. The shift of conceptual emphasis suggested here is aimed at allowing us to understand this important reality more directly in the highly complex and vulnerable groups of children we see.

TREATMENT

The authors present a balanced and helpful account of different forms of treatment advocated for attachment disorders, and in particular usefully tackle the issue of holding therapies. Is there a possible rationale to this treatment? As they point out, the therapist’s described behaviour during holding therapy bears no relationship to any quasi care giving behaviour found to promote attachment. Thus the rationale cannot be equivalent to the ‘corrective emotional experience’ of dynamic psychotherapy (where the therapist’s deep attention is felt to be homologous to sensitive parental type care).

Other possible rationales for the treatment depend on assumptions regarding the psychopathology of the syndrome. One might be to see the disorder as essentially an anxiety related behavioural avoidance of social contact and relatedness analogous to other anxiety related social avoidance. Holding therapy could then be seen as a variant of flooding technique. Of course, this rationale depends on anxiety related avoidant behaviour actually being the key component of the RAD social syndrome. Similar arguments were used to justify holding therapy in autism until the notion that it was primarily a hyperarousal syndrome received no sustained empirical support. The rationale could similarly be tested here. A weaker rationale might perhaps be based on distorted social learning – with the behavioural syndrome the result of avoidance of threat and confusion from toxic care giving. However this is close to the notion (likely to be often the case) that RAD is partly a post traumatic syndrome – in which case, as the authors state, the worry is that holding therapy is likely to be experienced as a frightening re-enactment of abuse.

A third possibility relates to the discussion above: that RAD is a social impairment syndrome containing not uncommonly a core developmental impairment of social cognition. Should this be the case then holding therapy might do no more than further mystify an already confused child.

Intensive and dramatic therapies of this kind have an intuitive appeal for serious disorders; they seem a fitting response somehow, like intensive care units for life threatening illness or intrusive behavioural treatments for autism. It is the better conceptualization and understanding of the characteristics and aetiology of the disorder itself that will clarify whether holding therapy can ever be justified or useful – or whether serious empirical trials of the approach should be mounted. If it is to be used it would, I suggest, need to be within the most carefully defined, contracted and consenting relationship of trust between therapist and patient, and with the greatest sensitivity to feedback following holding sessions. The practice is obviously highly vulnerable to abuse in misguided or unscrupulous hands and for that reason alone may not be justified. Intrusive holding on a first visit as a ‘diagnostic exercise’ seems particularly unpleasant and inappropriate. As O’Connor and Zeanah point out, many of the children included in treatment programmes of this kind probably don’t have ‘attachment disorder’ in any strict sense at all.

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