FEATURE ARTICLE

Borderline pathology in children and adolescents

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ABSTRACT: Mental health nurses have historically been pessimistic about and often unsympathetic towards clients diagnosed with borderline personality disorder. By the time these clients reach adult mental health services their behaviours are often difficult to manage and they often suffer significant re-victimization by health services. Questions need to be raised about how best to avert the consolidation of the problems associated with the disorder. This paper explores the concept of ‘borderline pathology’ in children and adolescents and examines the best available evidence for utilizing an early identification and intervention model for children and adolescents who exhibit this constellation of symptoms.

KEY WORDS: borderline pathology, borderline personality disorder, child and adolescent mental health, self-harm.

INTRODUCTION

Borderline personality disorder (BPD) in adults represents 15–20% of reported psychiatric illnesses and 70–77% of these are women (Nehls 1999; Swartz et al. 1990). Gallop et al. (1989) report that, historically, mental health nurses have demonstrated a lack of understanding and pessimism for this client group which has been reflected in nursing practices that often lack empathy for their pain and distress. These clients are stigmatized as incurable and repeated admissions creates frustration in clinicians (Bender et al. 2001; Horsfall 1999; Meares & Stevenson 2000; Nehls 2000). This paper challenges mental health nurses, particularly those working with children and adolescents, to take a new perspective. Evidence presented in this article suggests that mental health nurses can make observations of children and families to predict which children are potentially at risk for the later development of borderline personality disorder. If such children or adolescents could be identified before the onset of serious psycho-pathology, early intervention approaches become a distinct possibility. This article explores the concept of ‘borderline pathology’ in children and adolescents as a possible precursor to BPD in adults.

DIAGNOSTIC UNCERTAINTY OF BORDERLINE PERSONALITY IN CHILDREN AND ADOLESCENTS

Debate has raged for decades concerning the merits of diagnosing personality disorders in children and adolescents. According to the 4th edition of the Diagnostic and Statistical Manual (DSM-IV)-TR (American Psychiatric Association 2000) the diagnosis of BPD is an Axis II disorder (i.e. personality disorders or mental retardation) that can only be made before the age of 18 years if maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular developmental stage or an episode of an Axis I disorder. This criterion accurately reflects that dysfunctional personality traits in children and adolescents do not necessarily persist through to adulthood (Guzder et al. 1996). Not surprisingly, many child and adolescent psychiatrists are loath to make the diagnosis of BPD in childhood (Rey 1996). Famularo et al. (1991) argue that the label ‘post-traumatic stress disorder’ (PTSD) might be more appropriate for children and adolescents given that 60–80% of BPD clients presenting with this constellation of symptoms...
have been the victims of child abuse (Meares et al. 1999). Arguments against the use of the BPD diagnosis in children and adolescents are based on the dynamic and evolving nature of the adolescent personality.

The majority of children who are exposed to the psychosocial risk factors generally thought to be associated with borderline states, do not develop the anticipated psychopathology. An array of protective factors in childhood (such as gender, intelligence, temperament, positive expectations, and positive relationships) provides a sense of resilience and the capacity to effectively adapt to the 'circumstances normally associated with psychological dysfunction' (Stein et al. 2000: p. 282).

The adolescent personality is dynamic in nature as adolescents continuously learn and develop as a result of interpersonal exposure, reflection and ongoing neurocognitive changes. As a result, static diagnostic labels given today could effectively become obsolete as adolescents incorporate new experiences into their inner schema, changing the way they perceive and interact with the world around them. Garnet et al. (1994) found that even when the borderline diagnosis was correctly made in adolescents, the diagnosis was fairly unstable; their research indicating that of a group of 21 adolescents diagnosed with BPD only 33% continued to meet the diagnostic criteria after 2 years. Similarly, Mattanah et al. (1995) found that of a group of 30 adolescents with BPD only 23% were still diagnosable after 2 years. In a later study of 14 adolescents diagnosed with BPD, only two were still diagnosable after 3 years (Meijer et al. 1998).

However, despite the results of all three studies, a large number of borderline symptoms continued to persist in subjects who no longer met the diagnostic BPD criteria. Persistent symptoms include: conflicting emotions about the experience of giving and receiving care, dependency, masochism, and depression. This would suggest that the juvenile form of BPD clearly does not necessarily lead to BPD in adult years. However, as Kernberg (1990) argued, children with moderate to severe borderline pathology rarely outgrow such psychopathology.

**BORDERLINE PATHOLOGY IN CHILDREN AND ADOLESCENTS**

'Borderline pathology' describes a condition inherently similar to BPD as seen in adults with all the subsequent internalizing, externalizing and cognitive symptoms (Bemporad et al. 1982; Greenman et al. 1986). Internalizing symptoms appear to relate to the child’s internal sense of self – the 'neurotic' symptoms of anxiety, depression, worry, and somatic complaints. Internalizing symptoms arise from the child’s efforts to deal with their problems internally at a mental or cognitive level. Conversely, externalizing symptoms are the exact opposite with symptoms of distress directed outward resulting in overt symptoms of aggression, problems with impulse control, and para-suicidal behaviours. Cognitive symptoms of borderline states reflect the actual thought processes that are often intrinsically linked to the internalizing and externalizing behaviours. These disturbed thought processes habitually involve the lack of a stable self-image, transient psychotic symptoms with paranoid ideation, chronic feelings of emptiness, and perceived fears of abandonment. Contemporary evidence indicates that internalizing and externalizing disorders in early childhood are predictive of psychopathology in later childhood years (Campbell & Ewing 1990; Lavigne et al. 1998; Mesman & Koot 2001). Borderline pathology was first described by Ekstein and Wallerstein (1954 as cited in Bleiberg 1994) while studying the prognosis of children initially thought to manifest the early prodromes of schizophrenia. Ekstein and Wallerstein described these children, who failed to develop schizophrenia, as presenting with a 'characteristic pattern of unpredictability which is paradoxically one of their most predictable aspects' (as cited in Bleiberg 1994: p. 170). They described such children as frequently vacillating between 'bordering' upon the neurotic and 'bordering' on the psychotic in terms of their object relations, reality contact, and ego defences. These children tended to be highly impulsive, experienced transient psychotic symptoms, and were often depressed to the point of suicide (Paris 2000a).

Psychoanalytic researchers Marcus (1963) and Frijling-Schreuder (1969) described presentations of children with borderline pathology including impulsivity, poor frustration tolerance, uneven psychosexual development, withdrawal into fantasy or regression into autism, poor separation from primary caretakers, intense and pervasive anxiety features, and multiple neurotic features including phobias and ritualistic behaviours, somatic complaints, and sleep problems. These observations were later reflected in the work of Freud (1969) who proposed that children with borderline pathology suffered enormous developmental impediments; they were seemingly unable to be comforted by others and had poorly developed psychological defence mechanisms. These same traits have also been used to describe observations made of adult BPD patients in clinical practice (Adler & Buie 1979).

In a prospective follow-up study of children meeting the description of Ekstein and Wallerstein’s (1954 as cited in Bleiberg 1994) ‘borderline pathology’, Lofgren et al. (1991) found that whilst many of these children grew to develop an array of Axis II personality disorders in adulthood it did not necessarily follow that they would...
develop BPD as adults. It would seem then that either there is no corresponding relationship between borderline pathology in children and the adult BPD or that early descriptions of borderline pathology are too broad and not sufficiently articulated to capture the specifics of the phenomena equating to the adult BPD. Linear relationships between other forms of juvenile psychopathology and adult psychopathology might serve to strengthen arguments towards the possible existence of links between child borderline pathology and adult BPD. Certainly there has been a wealth of well-defined studies evidencing a clear relationship between conduct disorder in children and antisocial personality disorder in adulthood (Caspi et al. 1996; Kagan 1994; Robins & Regier 1991; Robins 1996; Tremblay et al. 1994; Zanarini et al. 1989).

What is clear is that adult personality is shaped by experiences during childhood and adolescence (Rey 1996). Mental health problems during these early years are likely to impinge upon later personality development, thus, increasing one’s risk of developing later psychopathology. Ultimately, validation of the borderline pathology construct in children and adolescents and its subsequent application to the clinical setting requires the identification of the aetiological pathway through which the disorder manifests. By exploring the risk factors underlying the development of adult BPD and determining if those same risk factors exist in children with borderline pathology it is hoped to establish whether a linear relationship exists between the two disorders (assuming diagnostic differences exist). As Rey (1996) suggests, borderline pathology in childhood and adolescence may be a precursor to adult BPD. This being the case, early intervention for borderline pathology in children and adolescents might offset the later development of adult BPD that is, by nature, distressing and costly in terms of resource utilization.

PSYCHOLOGICAL RISK FACTORS FOR BORDERLINE PATHOLOGY AND BORDERLINE PERSONALITY DISORDER

Traumatic events

Empirical observations arising out of clinical practice and spanning a broad spectrum of the literature on borderline pathology suggests that borderline children come from chaotic families and have experienced substantial stressors such as physical, emotional or sexual abuse, neglect, and traumatic separations (Ad-dab’bagh & Greenfield 2001; Bemporad et al. 1982; Goldman et al. 1992; Kestenbaum 1983; Paris 2000a). Furthermore, Goldman et al. (1993) found that the parents of these children often demonstrated serious psychopathology such as depression, substance abuse, and antisocial personality disorder interfering with their capacity to carry out normal parental functions.

Interestingly, retrospective studies of adults with BPD consistently demonstrate striking similarities with respect to early traumatic experiences such as sexual abuse (Briere & Zadli 1989; Guzder et al. 1996; Herman et al. 1989; Links et al. 1988; Paris et al. 1994; Shearer et al. 1990; Zanarini et al. 1989), physical abuse (Herman et al. 1989; Links et al. 1988; Paris et al. 1994), emotional neglect (Zweig-Frank & Paris 1991), as well as traumatic separations (Paris et al. 1988; Zanarini et al. 1989). In fact, there are an abundance of studies suggesting that sexual abuse occurs more frequently in the childhood histories of adult BPD patients than in any other disorder (Herman et al. 1989; Ogata et al. 1990; Paris et al. 1994). Given that female children are more likely to be sexually abused than male children (Finkelhor et al. 1990) it is not surprising that 70–77% of clients who receive the BPD diagnosis are also female (Swartz et al. 1990; Widiger & Weissman 1991). Of course, not all adult women or men with BPD report having been sexually abused as children so the BPD diagnosis cannot be considered conditional on the basis of sexual abuse, however, sexual abuse during childhood, according to Paris (1994) could be considered a substantial risk factor for the development of BPD.

Emotional abuse and neglect take many forms and the prevalence of these phenomena is high when reviewing the childhood histories of adult borderline patients. According to Walsh (1977) up to 87% of adult BPD sufferers reported conflictual relationships with their parents marked by emotional abuse and neglect. These findings are similar to those of Zweig-Frank and Paris (1991) that adult BPD patients reported both their mothers and fathers as less caring and more controlling than non-borderline patients. As these were the only two reasonably well controlled studies to examine neglect and emotional abuse in the childhood experiences of BPD patients, few assumptions can be made linking this phenomena to the development of either borderline pathology in children or adult BPD. However, these studies lend support for psychodynamic models postulating the role of parental emotional withdrawal in the pathogenesis of borderline states.

Other studies highlight the importance of early separations in the pathogenesis of borderline pathology. Walsh (1977) observed that a large number of adults with BPD had experienced significant separations via divorce or death. Similarly, Bradley (1979) found that 64% of children with borderline pathology had experienced extended separations from their parents early in life and
that these separations were more likely to have occurred to borderline children than children with other forms of psychopathology. These results were comparable to those of Soloff and Millward (1983) who compared a group of 45 adult BPD patients to groups of depressed and schizophrenic patients. The BPD patients were significantly more likely to have come from families in which divorce, separation, or parental death had occurred. Seemingly then, separation of the pre-borderline child from one or both parents would appear to be a contributory factor towards the development of borderline pathology; although, Zeanah et al. (1989) argue that insecure attachments were more influential on later child development.

As mentioned above, adults with BPD often report the experience of traumatic events. Terr (1991) organized the abuse histories of children diagnosed with PTSD into two distinct categories: Type A, pathology secondary to one off traumatic experiences; and Type B, pathology resultant to chronic or repetitive exposure to trauma. Inherent to this model is the conceptualization that whilst singular traumatic events do not necessarily lead to the development of severe psychopathology, exposure to multiple or frequent trauma renders the child susceptible to a plethora of stress-related psychopathology (including the development of borderline states). Adolescent psychiatric inpatients who had been abused as children manifested higher levels of dependency, suicidality, aggression, impulsivity, and substance use disorders than nonabused inpatients (Grilo et al. 1999) consistent with adult BPD. Comparing a number of children with and without borderline pathology, Guzder et al. (1996) and later Paris (2000a) found higher rates of cumulative trauma in the borderline children suggesting that the experience of multiple traumatic events is more predictive of borderline pathology than any singular event. Children, adolescents and adults, whose victimization was a product of protracted trauma, have far more complex symptomatology impacting upon their long-term personality development, their sense of identity, and their capacity to relate to others (Herman 1992).

Whilst these studies would seem to point towards the experience of multiple traumas in the aetiology of borderline pathology and BPD, Rutter (1987) emphasizes the resiliency of childhood. While many children experience sexual abuse, neglect, and various other traumatic experiences, only a few actually go on to develop serious psychopathology. Hunter and Chandler (1999) defined resiliency as the array of internal and external protective factors which allow one to overcome adversity. Internal protective factors involve a complex interplay of gender, temperament, intelligence, sense of humour, empathic abilities, and an internal locus of control (Hunter & Chandler 1999; Stein et al. 2000). External factors, such as having a warm and cohesive family, support the development of internal resiliency (Garmezy 1991; Hunter & Chandler 1999; Stein et al. 2000). Paris (1998) explains the discrepancy between abused children who develop psychopathology and those who do not by suggesting that traumatic events are filtered by neurocognitive processes. Children predisposed to borderline pathology lack the biological capacity to adequately process these events, thus, producing the constellation of symptoms seen in borderline states.

**PSYCHODYNAMIC UNDERSTANDINGS OF BORDERLINE PATHOLOGY**

Psychoanalytic schools of thought have investigated the genesis of borderline pathology. As early as the 1940s Mahler et al. (1949) identified a group of children presenting with ego and object relational pathology far more severe than neurotic children, whilst less severe than those children diagnosed with psychotic disorders. This led to speculation by Mahler and colleagues that they were witnessing the early prodromal phases of later schizophrenic disorders with the borderline psycho-pathology the earliest overt symptoms that could be identified. It was not until the work of Ekstein and Wallerstein (1954 as cited in Bleiberg 1994) that it was realized that a large number of these children were not developing schizophrenia as first thought.

Adopting a developmental perspective, Masterson (1972) considered the significance of what Mahler (1971) termed the ‘separation-individuation’ (p. 419) stage of child development. Masterson suggested that when children begin to act upon their natural autonomous urges and start to act independently of their parents during the separation-individuation phase, the child is highly susceptible to perceived abandonment. According to Sable (1997) this insecure attachment between parent and child results in the child vacillating between a strong desire for intimacy and a dreaded avoidance of engagement. Masterson and Risley (1975) hypothesized that the mothers of these children take great pride in their child’s reliance on them and that fostering this dependency, thus, serves the interests of the mother. Bowlby (1988) hypothesized that the relationship between parent and child would likely become conflicted if the parent (especially the mother) experienced a significant loss or trauma that they find themselves unable to resolve. Unresolved issues of stress, loss, and trauma can potentially reverse the normal attachment pattern with the child becoming the primary emotional carer for the parent, thus, the parents emotional needs become the priority (Liotti et al. 2000). Liotti et al. (2000) observed that the
most significant losses impinging upon attachments include losses incurred by the mother primarily around the period of pregnancy and childbirth.

Contemporary psychoanalytic thought on the aetiology of borderline pathology began with Kernberg’s (1975) treatize on borderline conditions and pathological narcissism. Kernberg proposed that early trait aggression in children led to a division between positive and negative images of themselves and their mothers. Consequent to this split in self-image the child finds they are unable to reassess these conflicting images and as such continue to view both themselves and the world around them as extremes of positivity or negativity. This extreme sense of duality impacts adversely on the borderline individuals’ capacity for intimacy and can cause altered states of consciousness including dissociation, amnesia, depersonalization, and derealization (Streeck-Fischer & van der Kolk 2000).

Concurrent with Masterson’s (1972) and Kernberg’s (1975) theories of the aetiology of BPD, is that of Adler and Buie (1979) who suggested that, as children, people with BPD failed to develop a stable sense of object consistency due to early failures by the maternal figure. Their premise, that the mothers of pre-borderline patients inconsistently lacked sensitivity and empathy, suggests that as children BPD patients failed to learn the necessary self-soothing resource of a consistent self-image and perception of others. In response to this failure, infants experience extreme levels of anger, anxiety, and desire, which, given their incapacity for self-soothing, can precipitate dissociation or self-harming behaviours (Streeck-Fischer & van der Kolk 2000). Alternatively, the child may develop an ‘inner sense of emptiness’ (Bleiberg 1994: p. 181) leading to a dependency on transitional experiences such as food and drugs which supplant their own incapacity for self-soothing and a reliance on dramatic efforts to procure attention and involvement from others (Winnicott 1953). In light of the work of Links et al. (1988) and Zanarini (1993), that the parents of BPD patients oftentimes experienced serious psychopathology themselves, the Adler and Buie model may have some merits.

Exploring these psychoanalytic theories of the pathogenesis of BPD, Bezirganian et al. (1993) examined a group of 776 normal adolescents and their families at baseline and again 2.5 years later. Screening the families for evidence of maternal over-involvement and/or inconsistent parenting, the authors showed that maternal inconsistency was predictive for the emergence of BPD, but not of any other Axis II disorders. Interestingly, this effect occurred only in the context of inconsistent maternal over-involvement such that neither factor alone (i.e. maternal over-involvement or maternal inconsistency) was sufficient to predict the emergence of BPD (Bezirganian et al. 1993).

Clearly, psychoanalytic theories of the pathogenesis of borderline states rely very much on the presence of inherent temperamental traits that make such children vulnerable to the stressors of abnormal upbringings. As Paris (2000a) observed, whilst many children might have unusual temperaments only a few actually go on to develop some form of major psychopathology. Elucidation of the nature of these trait temperaments might lie in the domain of biological and genetic theories explaining the trait vulnerabilities in some children that give rise to the symptomatology reflected in borderline states. Such models might better serve to assist in an understanding of why some children display such marked pathology having the experience of abnormal upbringings whilst others do not and provide opportunities for early intervention (Zeanah et al. 1989).

In addition to the psychoanalytic models, behavioural and learning models of stress responses can also be used to explain many of the overt symptoms associated with borderline states. According to van der Kolk and Greenberg (1987) the aversive experience of abuse, neglect, or separation from significant carers (unconditioned stimuli) produces an unconditioned defensive response as per the autonomic ‘fight or flight’ response. Repeated exposure to such forms of adversive experiences eventually come to be associated with ‘intrinsically non-threatening cues’ (p. 64) or other benign events that have a relationship to the initial adversive experience. Subsequently, in borderline individuals, primal or regressed defensive behaviours are seen in the context of seemingly non-threatening events, the responses being conditioned according to the principles of classical conditioning.

**BIOLOGICAL RISK FACTORS**

**Neurological**

In an overview of borderline disorders in children, Petti and Vela (1990) discussed the frequent presence of organic deficits including learning disabilities, attention deficit disorder, and abnormal electroencephalogram (EEG) patterns. Exploring these ideas further Paris et al. (1999) administered a battery of neuropsychological tests (including the Continuous Performance Test and the Wisconsin Card-Sorting Test) to children manifesting borderline pathology. Comparing borderline versus non-borderline children, Paris et al. found that the borderline children experienced severe problems with attention, impulse control, concept formation, and serious executive functioning deficits.

These findings led Paris et al. (1999) to suggest a ‘stress-diathesis’ (p. 770) model for the formulation of
borderline pathology in children and adolescents. According to this model a series of biological vulnerabilities, not fully identified, are impacted upon by environmental stressors (e.g. multiple traumatic events or dysfunctional parenting) to produce the clinical symptoms associated with borderline pathology and later BPD.

Whilst there does not seem to be any specific biological markers for BPD in adults (Guzder et al. 1996) several studies are highly suggestive of neurological impairment by virtue of findings of ‘soft’ signs such as impulsivity, cognitive inflexibility, poor self-monitoring, and preservation of thought (Judd & Rugg 1993; O’Leary et al. 1991; O’Leary & Cowdry 1994; van Reekum et al. 1993; van Reekum et al. 1996) suggestive of frontal lobe deficits. Using neuropsychological testing methods van Reekum et al. (1993) reported findings that BPD patients show indications of frontal lobe dysfunction. Interestingly, Goyer et al. (1994) reported similar results in a study in which they found a significant relationship between abnormal frontal lobe activity and a history of impulsivity and aggression using positron emission tomography to analyse glucose metabolism in a group of 17 adult BPD patients. However, there exists some evidence that such abnormalities in brain glucose metabolism could in fact be the result of genetic influences (Plomin et al. 1994). In a later magnetic resonance imaging (MRI) study Lyoo et al. (1998) revealed that adult BPD patients have smaller frontal lobe volumes compared to healthy controls. These findings are of particular interest when one considers the current prevailing thought on the notion of ‘self’. Damasio (1996) and Meares et al. (1999) supposed that frontal lobe activity, whilst associated with impulse control, is also the centre from which one’s sense of ‘self’ emerges. Dysfunction of the frontal lobes, therefore, may be the key to understanding the poor sense of self that many children, adolescents and adults with BPD experience. However, the cause of this anomaly is not fully understood (Guzder et al. 1996) and could be the result of an acquired process (e.g. head injury or infection), inheritance (i.e. genetic), or experiential failures (e.g. under-stimulation or dysfunctional parenting).

One possible explanation for these anomalous frontal lobe findings put forth by Meares et al. (1999) proposes that borderline states are the product of failures in the socialization process or ‘sociogenesis’ (p. 837) which impairs the experience-dependent growth of neural connections in the frontal regions of the brain, thereby, impeding the normal development of a healthy self-concept. Linkage of the frontal regions to other areas of the brain, including the amygdala and hippocampus, result in a cascade of aberrant responses including somatization, problems in attending, affect regulation, memory, and dissociation. Interestingly, these two particular brain regions, the amygdala and the hippocampus, were the focus of another BPD study by Driessen et al. (2000). In this study the authors reported a 16% reduction in hippocampus volume and an 8% reduction in the volume of the amygdala in borderline patients as opposed to healthy controls. Schore (1997; 2000) also suggests that emotional development, stress-regulation, and coping strategies rely on the experience-dependent maturation of the orbito-frontal cortex. Therefore, the aetiological model for BPD proposed by Meares and colleagues, supported by the work of Schore, grounded in biological science, and emphasizing environmental and sociological factors, represents an evolution on the psychodynamic theories of the genesis of borderline states.

As has previously been highlighted, many children and adolescents with borderline pathology have experienced recurrent and chronic patterns of abuse, yet there are few neurobiological studies to determine the impact of this abuse on neurological development. Therefore, studies of neurobiological changes found in adults, who have suffered physical abuse as children, might serve a predictive analogous function hinting at some of the neurological changes at work in children and adolescents with borderline pathology who have been the victim of abuse. Teicher et al. (1993) showed that adults who had been physically abused as children had decreased corpus callosum volumes resulting in differential activation between brain hemispheres under aversive and peaceful conditions. In addition, they found that limbic system activity (thought to play a roll in agitation and emotional regulation) was increased by 38% in adults who had been physically abused. 49% in adults who had been the victim of sexual abuse, and that limbic system activity was increased by 113% in adults who had been both physically and sexually abused as children. Importantly, the structural changes found in the brains of these adults, who had been abused as children, were not the result of head trauma caused in the assault.

Exploring these concepts in children, Guzder et al. (1996) examined a group of 89 patients aged between 7 and 12 years with an array of mental health problems (e.g. conduct disorder, oppositional defiant disorder, attention-deficit hyperactivity disorder, major depressive disorder, over-anxious disorder, and borderline pathology). This group was again divided into a borderline group (n = 38) and a non-borderline control group (n = 51). Whilst both groups displayed marked abnormalities in a range of neuropsychological measures, this is not unexpected given that both groups were highly dysfunctional. However, using the Wisconsin Card Sorting Test, which is commonly used to assess cognitive deficits in psychiatric patients, the borderline group proved to
have far more severe deficits in frontal lobe, executive functioning, and working memory. In effect, this result translated into the borderline children having more difficulty completing tasks, making more mistakes, being incapable of learning from those mistakes, and possessing an overall lower level of conceptualization of the tasks involved in the testing. Of note, these results are indicative of the poor planning and inflexibility seen in borderline patients clinically.

Other authors have focused on neuro-chemical changes rather than structural anomalies in people with borderline states. Coccaro et al. (1989), in a study of 18 patients with diagnosed personality disorders, found that impulsive self-destructive behaviours and aggression aimed at others were associated with reduced central serotonergic 5HT functioning. Importantly, the confounding variables of substance abuse and major affective disorders were factored out in this study. Rinné et al. (2000) reported similar findings in a study of 12 adult BPD patients who reported a high incidence of childhood physical and sexual abuse. Consequently, in a later study, Coccaro and Kavoussi (1991) proposed that the cluster of symptoms seen in adult BPD patients might be the result of errors in certain neurotransmitter pathways. Transient psychotic symptoms are accordingly described as the result of over activity in the functioning of dopaminergic systems, whilst the high degree of impulsivity seen in many BPD patients is considered reflective of central nervous system serotonergic dysfunction. However, it is unknown whether these neurotransmitter anomalies are in fact the fundamental cause of BPD symptoms or the result of abnormal life experiences.

One possible explanation for this broad array of physiological anomalies found in borderline individuals might, hypothetically, lie in the concept of 'kindling' (Streeck-Fischer & van der Kolk 2000; van der Kolk & Greenberg 1987). This concept was first described by Goddard (1967) who found that a small electrical stimulus, just large enough to trigger a brief burst of epileptiform activity, if repeatedly applied, will eventually generate seizures that can lead to fully generalized behavioural convulsions. The concept has been broadened somewhat over the years. The traumas, suffered by children and adolescents with borderline pathology, can result in permanent neurobiological changes (most likely involving reduced frontal lobe functioning and limbic system hypersensitivity) and subsequent cognitive changes (Meares et al. 1999; Streeck-Fischer & van der Kolk 2000; van der Kolk & Greenberg 1987). As a product of this emotional and neurobiological ‘kindling’ process children and adolescents with borderline pathology continue to react to subsequent minor stressors with the same degree of emotional intensity as they did in the early trauma (Streeck-Fischer & van der Kolk 2000).

Genetic risk factors

Environmental perspectives on psychological development dominated developmental literature until the 1960s when research interest into the relationship between environmental and genetic influences of human behaviour began to emerge (Plomin et al. 1994). It was not until recent times when Bouchard (1994) published confirmatory evidence for the genetic basis of personality that the genetic origins of BPD became a viable source of scientific enquiry. Bouchard et al. (1990) argued that the ‘personality similarity between biological relatives is almost entirely genetic in origin’ (p. 1701) which suggests that environmental influences play only a minor role in the development of personality. Livesley et al. (1993) found that of a group of 90 monozygotic twins (mostly sisters) and 85 dizygotic twins (mostly sisters), who were all healthy, there were continuities between heritable normal personality traits and abnormal or disordered personality dimensions. In a larger replication of this study Jang et al. (1996) found that of a group of 236 monozygotic twins (mostly sisters) and 234 dizygotic twins (mostly sisters) personality disorders generally have a substantial genetic component. The authors concluded that whilst the personality disorders themselves might have a strong genetic component, individual differences in presentation (e.g. self-harm, labile emotions, cognitive rigidity) were susceptible to either genetic or environmental influences. More recently, Coolidge et al. (2001) found that BPD has a high component of heritability and hypothesize that the lack of emotional regulation and unstable self-concept, seen in these borderline children, is suggestive of a possible inherited neurobiological dysfunction.

Whilst many of the traits inherent to the borderline construct show evidence of being influenced by genetics, there is currently insufficient evidence to say to what extent borderline pathology or BPD is genetically determined (Torgersen 2000). Borderline studies of monozygote and dizygote twins, whilst seemingly appealing, must take into account the potent environmental factor of the family and culture. Hypothetically, the results of twin studies have the potential to overshadow the effects of traumatic exposure and familial dysfunction. Given the obvious difficulties faced with the prospect of studying twins raised apart, the likelihood of twin studies making valuable contributions to our understanding of borderline pathology is negligible. In addition Fonagy (2001) argues convincingly that the expression of individual genotypes is moderated by mental mechanisms that are linked to the early attachment relationships between the
infant and the caregiver. Until the outcomes of the now completed human genome project are applied to the study of BPD, studies of generational psychopathology might yield more valuable results.

Comparing borderline versus non-borderline children, Guzder et al. (1996) found that children with borderline pathology more often had a first-degree relative with either a substance abuse problem or antisocial traits. Interestingly, Zanarini (1993) suggested that BPD, substance abuse, and antisocial personality disorder might share a common biological origin. Added to this is the work of Weiss et al. (1996) who compared 21 children of mothers with BPD to a similar aged group with mothers diagnosed with other Axis II personality disorders. Strikingly, the 21 children with BPD mothers had more psychiatric diagnoses in the impulse spectrum range including childhood borderline pathology (Weiss et al. 1996). These results, whilst suggestive of BPD mothers being a substantial risk factor for second generational BPD, neglect the fact that in clinical practice only a minority of adults with BPD have mothers diagnosed with the disorder (Paris 2000a).

Links et al. (1988) and Zanarini (1993) both found high rates of serious psychopathology amongst the parents of adult BPD sufferers. Both of these studies counted high rates of substance abuse, depression, antisocial personality disorder, and BPD amongst the parents of adult borderlines. Whilst these results could lead to speculation about a possible genetic basis for borderline pathology in children, it might be of equal, if not greater, importance to consider the cost in terms of parenting these disorders impart. It is conceivable that the expression of these disorders bears a heavy environmental burden on the development of many children, the stress of which could be the pinnacle factor underlying the development of borderline pathology. It has further been suggested that the aetiology of borderline pathology in children and adolescents could be reflective of a combination of parental psychopathology and traumatic life experiences (Feldman et al. 1995).

DIAGNOSIS OF CHILDHOOD BORDERLINE PATHOLOGY

Presently, there is no internationally agreed systematized diagnostic classification for childhood borderline pathology. Diagnosing of adult BPD is fraught with methodological and stigmatic problems. The strict diagnostic systems of DSM-IV-TR and ICD-10, whilst capturing the component characteristics of adult BPD, fail to acknowledge the early developmental phases in childhood or adolescence that give rise to the disorder. Clearly, as has been shown, the borderline construct in childhood and adolescence has some categorical and diagnostic validity, even in the absence of the capacity to evidence a linear relationship between the juvenile disorder and the adult BPD diagnosis. However, concerns about the negative stigma of the ‘borderline’ label must be given due consideration. Ad-Dab’Bagh and Greenfield (2001) have suggested that the same fatalistic beliefs that many clinicians hold of adult BPD patients might be imbued upon children labelled with borderline pathology. Whilst this concern may have some validity, the lack of an accurate form of nomenclature could conceivably deny these children appropriate interventions.

There are few diagnostic tools developed for use in children or adolescents. Perhaps the most useful tool is the Child Diagnostic Interview for Borderlines (C-DIB-R) by Greenman et al. (1986) which is an adaptation of the Diagnostic Interview for Borderlines used in adults developed by Gunderson and Kolb (1978). Whilst this semistructured interview is extremely useful in diagnosing borderline pathology in children, it is important to note that the final resultant diagnosis does not necessarily comply with the strict DSM criteria (Petti & Vela 1990).

Without a clear classification system for borderline pathology, clinicians might care to look upon the borderline construct in children and adolescents as a syndrome; a group of signs and symptoms that characteristically occur together. This paper has discussed a number of signs and symptoms that could conceivably be grouped together for the formulation of the borderline syndrome. Thus, it has been suggested that the method of diagnosing of adult BPD could conceivably change with future research into the biological basis of the disorder and with advances in medical imaging techniques (Paris 2000b). Similarly, MRI, EEG, and neurocognitive findings of frontal lobe dysfunction in children and adolescents might also be used in the formulation of the borderline syndrome construct.

In isolation however, frontal lobe dysfunction could not be considered indicative of borderline pathology. Such findings would need to be put into context with accurate and extensive history taking. Reports of chronic and enduring childhood abuse, neglect, traumatic separations from attachment figures, and turbulent family functioning must be superimposed over biological data before making categorical assumptions. Additional support for the diagnosis of borderline pathology might come from family interviews in which the existence of generational psychopathology is elucidated.

CONCLUSION

This paper has highlighted the results of several decades of research and theory into the borderline construct as it
applies to children and adolescents. Children with borderline pathology presently escape inclusion into present systematized diagnostic systems such as ICD-10 and DSM-IV-TR. However, the literature indicates that children who fit the profile for borderline pathology tend to share a number of common characteristics, namely, the experience of repeated chronic trauma (e.g. sexual abuse, neglect, physical abuse, emotional abuse), early separations from attachment figures, irregular parenting styles, familial psychopathology, and cognitive deficits with evidence of neurological irregularities (especially frontal lobe reduction). Additional evidence presented suggests there may be a genetic component to borderline pathology with many of the personality traits found in parents being reflected in their children.

Clearly some form of pathological process is taking place in children and adolescents who manifest extremely labile and impulsive behaviours. Without appropriate intervention children with borderline pathology may continue to experience intrapsychic distress through to adulthood. Developmentally appropriate interventions, which encourage self-mastery, distress tolerance and cognitive remediation, have the potential to reduce the negative impact of borderline pathology on future development. Possibilities for treatment interventions and the implications for mental health nursing will be discussed in a further paper.

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REFERENCES


Masterson, J. F. & Rinsley, D. B. (1975). The borderline syndrome: The role of the mother in the genesis and psychiatric...


