ARTICLE

Post-traumatic stress disorder and attachment: possible links with borderline personality disorder

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SUMMARY
This article discusses the aetiology of both simple and complex post-traumatic stress disorders (PTSDs) in terms of attachment theory, and points out the similarities between the diagnosis of complex PTSD and of borderline personality disorder. Case vignettes illustrate an outline of the assessment and treatment of the psychobiological symptoms of PTSD informed by attachment research.

DECLARATION OF INTEREST
None.

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In both ICD–10 (World Health Organization 1994) and DSM–IV (American Psychiatric Association 1994), the diagnostic criteria for post-traumatic stress disorder (PTSD) require that the individual has been exposed to ‘a stressful event or situation of exceptionally threatening or catastrophic nature likely to cause pervasive distress in almost everyone’ (according to ICD–10) and eliciting a response involving ‘intense fear, helplessness or horror’ (according to DSM–IV). However, more recent research shows that the events resulting in most diagnoses of PTSD are actually quite common and that none of these traumas is so powerful that exposure typically leads to the disorder (Kessler 1999: p. 55). The disorder is more frequent and severe in victims of natural than of man-made disasters (Lifton 1976: pp. 10–14), and is less likely to occur in well-integrated communities than in fragmented ones (Quarantelli 1985: p. 192). These findings tie in with the main conclusion of treatment guidelines published by the National Institute for Health and Clinical Excellence (NICE), which singles out the lack of social support as the most important risk factor for PTSD (National Collaborating Centre for Mental Health 2005: p. 94).

Social support is a vague term, but there is little doubt that it includes the attachments that individuals develop with each other, in both family and community life. It is through the study of attachment research that we can begin to make sense of findings that show strong links between the pathophysiology of PTSD and that of attachment disorders (Van der Kolk 1996; Henry 1997; Wang 1997; Schore 2000).

An understanding of the development of attachment and its disorders is therefore helpful to our understanding of PTSD, and particularly of complex PTSD (Herman 1992a,b; Roth 2006). Attachment research can also guide the development of therapeutic approaches for complex PTSD (de Zulueta 2006a,b), an important area that is not addressed by the NICE guidelines (National Collaborating Centre for Mental Health 2005).

In addition to the key diagnostic symptoms of PTSD, DSM–IV also refers to ‘an associated constellation of symptoms’ that ‘may occur and are more commonly seen in association with an interpersonal stressor’ such as childhood sexual or physical abuse, domestic violence, torture, or being a hostage or a prisoner of war (American Psychiatric Association 1994: p. 425). This combination (Box 1) has been variously referred to as complex PTSD (Herman 1992a) and ‘disorders of extreme stress not otherwise specified’ (Pelcovitz 1997). The list resembles very much the symptoms of DSM–IV borderline personality disorder except for the last item, referred to as ‘a change from previous personality characteristics’ which is probably best covered by the ICD–10 diagnosis of ‘enduring personality change’. 

BOX 1 Associated symptoms of PTSD

- Impaired affect modulation, self-destructive and impulsive behaviour
- Dissociative symptoms
- Somatic complaints
- Feelings of ineffectiveness, shame, despair, hopelessness, guilt; feeling permanently damaged; loss of previously sustained beliefs
- Hostility, social withdrawal, feeling constantly threatened, impaired relationships with others
- Change from previous personality characteristics

(American Psychiatric Association 1994; Van der Kolk 2005)
Complex PTSD and borderline personality disorder

The distinction between the diagnosis of complex PTSD and that of borderline personality disorder (Table 1) remains controversial. Both can be seen to result from damage to the attachment system (Fonagy 1997; de Zulueta 1999, 2006b). However, the latter is often thought of as a stigmatising diagnosis that elicits a negative response from healthcare workers (Nehls 1998). In addition, feminists point out that the diagnosis of complex PTSD acknowledges the sexual abuse that many women with a borderline personality disorder have suffered (Shaw 2005).

There is some debate about renaming borderline personality disorder. One suggestion is that it should be called an ‘emotional regulation disorder’, a term favoured by Linehan, the creator of dialectical behavioural therapy, a very useful approach to the treatment of these patients’ symptoms (Koerner 2000). Quadrio (2005) favours the concept of a ‘post-traumatic personality disorganisation’, in the belief that it is a common outcome of both developmental and attachment trauma.

The development of attachment behaviour† and PTSD

Like all mammals, human infants are genetically predisposed to seek contact with another living creature – an ‘attachment figure’ – and it is believed that this behaviour is essentially triggered by fear, the same fear and sense of helplessness that are inherent to the experience of psychological trauma. In explaining this fear of separation, Bowlby wrote:

Man, like other animals, responds with fear to certain situations, not because they carry a high of pain and danger, but because they signal an increase of risk. Thus, just as animals of many species, including man, are disposed to respond with fear to sudden movement or a marked change in level of sound or light because to do so has survival value, so are many species, including man, disposed to respond to separation from a potentially caregiving figure and for the same reasons. (Bowlby 1988: p. 30. Italics as in original)

Attachment behaviour involves the limbic and paralimbic areas of the right hemisphere and the supra-orbital area of the brain. The latter area is necessary to acquire specific forms of knowledge to regulate interpersonal behaviour. With deep connections to the autonomic system, the supra-orbital area is critical to the modulation of emotional and social behaviour, the affect-regulating functions involved in attachment processes (Schore 2000: pp 30–32).

It is through the process of attunement that takes place between infant and caregiver that the infant becomes able to modulate his or her emotions. The different types of attachment behaviour that develop between infants and their various caregivers were originally identified by Ainsworth and her team (Ainsworth 1978). Using the ‘strange situation’, a structured separation test carried out on 1-year-old infants, they described three types of attachment: secure; insecure avoidant; and insecure anxious – ambivalent or resistant.

Secure attachment

As the infant interacts with their caregiver, a secure child is thought to develop a mental representation of the caregiver as responsive in times of trouble. This type of attachment becomes a primary defence against trauma-induced psychopathology (Schore 1996). In addition, if the caregiver or another important attachment figure in the child’s life is able to give meaning to the child’s experiences and share and predict their behaviour, the child can internalise this capacity. Such a developmental acquisition, described by Fonagy & Target as ‘reflective functioning’ or ‘mentalisation’, enables people to understand the mental states of others and thereby foster successful social interactions (Fonagy 1997). Its development in a child provides them with further protection against future re-traumatisation.

The development of the child’s representation of their self is closely intertwined with the internal representation of their attachment figure. Hence, securely attached children will tend to feel loved and valuable.

Insecure attachment

Insecure attachments are thought to develop when infants do not have a mental representation of a

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**TABLE 1**

<table>
<thead>
<tr>
<th>Complex PTSD</th>
<th>Borderline personality disorder*</th>
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<tbody>
<tr>
<td>Impaired affect modulation</td>
<td>Impulsivity in at least two potentially self-damaging areas; recurrent suicidal or self-damaging behaviour; affective instability; inappropriate, intense anger or difficulty in controlling anger</td>
</tr>
<tr>
<td>Dissociative symptoms</td>
<td>Transient, stress-related paranoid ideation or severe dissociation; identity disturbance such as a markedly and persistently unstable self-image of sense of self; chronic feeling of emptiness</td>
</tr>
<tr>
<td>Impaired, insecure relationships with others</td>
<td>Frantic efforts to avoid real or imagined abandonment; a pattern of unstable and intense interpersonal relationships</td>
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responsive caregiver in times of need: they develop various strategies to gain access to their caregiver in order to survive (Ainsworth 1978). The most insecurely attached are infants with a disorganised/disoriented attachment (Main 1992), who show an unpredictable response to their caregiver in the strange situation and are seen to freeze in a trance-like state, very much like adults with PTSD. Often these infants have either been abused or neglected by a frightening parent or their parent has appeared frightened; such a parent is likely to suffer from PTSD triggered by the infant (Main 1992).

**Case vignette 1**

A Kurdish woman from Turkey with a diagnosis of complex PTSD presented to us with her small son, who had been referred to social services for failure to thrive. The mother had been severely beaten by the child’s father and, unfortunately for this little boy, when he became distressed, his eyes resembled those of his father. This triggered in his mother a reliving of her past experience of domestic violence, with all the anger and fear that this involved, rather than the comforting maternal behaviour the child needed.

In such cases, the caregiver not only induces traumatic states in their children, but also cannot interactively repair the infant’s negative affective states because they are unable to put themselves in the mind of their child. The result is very damaging, both to the child’s future capacity to regulate emotions and because it can lead to the development of early dissociation and what US authors commonly refer to as complex PTSD and dissociative disorders or what we refer to more often in the UK as a borderline personality disorder (Fonagy 1997).

The infant’s psychobiological response to feeling threatened by their caregiver can involve three stages (Perry 1995).

The first is the fight-or-flight response, mediated by the sympathetic nervous system. This bypasses the cortical centres and their capacity for symbolic processing, with the result that traumatic experiences are stored in somatic, behavioural and affective systems.

If the fight-or-flight response is not possible, as will usually be the case with a very small child, the parasympathetic state takes over and the child ‘freezes’, which in nature may be linked to feigning death, thereby fostering survival. Vocalisation may also be inhibited and children, like young animals, may lose the capacity to speak and become mute. This phenomenon is related to the release of endogenous opiates and the shutting down of Broca’s speech area as observed in positron emission tomography scans of adults with PTSD (Rauch 1996).

If the caregiver’s threat or rejection continues, the infant enters a state of ‘fear without solution’, in which both responses are activated, which can lead to a dissociative response (Main 1992). Although in fear of their caregiver, the child must maintain their vital attachment to their caregiver. This can be achieved by resorting to dissociation, i.e. creating different representations of themselves in relation to their caregiver. This results in a lack of self-continuity in relation to the ‘other’ as can be seen in people with borderline personality disorder (Fonagy 1997; Ogawa 1997; Ryle 1997; de Zulueta 1999), complex PTSD (Herman 1992a,b) and dissociative disorders.

In other words, to ensure their survival these infants develop an idealised attachment to their caregiver, on whom they continue to rely emotionally, and their terrifying ‘self–other interactions’ with the same caregiver become dissociated – or unavailable to the conscious self – with the result that the self becomes fragmented.

At a cognitive level, this means that these children, and later adults, will tend to feel guilty and blame themselves rather than their caregiver for what happens to them. By taking the blame, being ‘bad’ and keeping the caregiver as an idealised figure in their mind, they retain a sense of control in the face of otherwise unbearable helplessness. They can also preserve the hope that in the future, if they behave well, they will finally attain the love and care they never had. This cognitive defence, aptly called the ‘moral defence’ by Fairbairn (1952), is ferociously maintained to avoid the unbearable realisation, and its accompanying grief and anger, that there is no such idealised caregiver.

The cost of maintaining a ‘traumatic attachment’ to an abusing or neglectful caregiver can be a heavy one. In seeking the parental care they never had in childhood, as adults these individuals tend to destroy intimate relationships. In therapy, they tend to sabotage their achievements and progress in order to continue their search for the idealised parent they still yearn for, albeit unconsciously (de Zulueta 2006c). In extreme cases, this inner conflict and resulting sense of intense vulnerability can lead, paradoxically, to homicide (de Zulueta 2006a: pp.137–151; de Zulueta 2006c). Addressing the ‘traumatic attachment’ and its cognitive distortions may be central to the treatment of patients with a history of childhood abuse or severe neglect.

Although a similar sense of blame may also arise in adults with simple PTSD, the sense of responsibility and associated fear of helplessness are more readily amenable to change (Ehlers 2000).

Henry (1997) noted that many people with complex PTSD arising from childhood abuse and neglect also have alexithymia, finding it difficult to speak about their emotions and thereby share and cope with disturbing feelings. Consequently, they tend
to re-enact their traumatic experience rather than think about it, thus experiencing re-traumatisation (Van der Kolk 1989). Alexithymia also appears to be associated with an interhemispheric transfer deficit (Zeitlin 1989) and is more likely to occur if the trauma is repeated, as in sexual abuse (Zeitlin 1993).

The psychobiology of neglect and abuse

Evidence suggests that traumatisation in early life can result in damage to the cortical and subcortical limbic systems of the right hemisphere, leaving the child with a reduced capacity to play, to empathise and to form sustaining relationships (Schore 2001; Siegel 2001). The inability to modulate emotions is central to an understanding of patients with complex PTSD: they cannot deal with sympathetic-dominant affects such as terror, rage and elation, nor can they deal with parasympathetic-dominant affects such as disgust and, in particular, shame.

Shame is an emotion that is often ignored to our peril. People who have been neglected or abused in childhood can grow up as adults whose sense of self is that of an individual who has been made to feel totally invalidated. When exposed to shame or humiliation, they often resort to attack in order to ward off an unbearable sense of total annihilation. As one patient with a history of homicide said to his therapist ‘Better be bad than not be at all’ (Gilligan 1986). Our prisons are full of such individuals.

The inability to modulate emotions often causes survivors of trauma to self-medicate using alcohol or drugs or to resort to immediate violence when feeling out of control or frightened of the ‘other’.

The reliving of traumatic experiences can result in release of natural opiates and accompanying analgesia: this effect can fuel acts of re-traumatisation, self-harm or violence as a form of relief from unbearable emotional states. The following vignette gives a vivid illustration of this phenomenon (Van der Kolk 1989: p. 391).

Case vignette 2

One night in 1968, a Vietnam Vet lit a cigarette which led to his ‘buddy’ next to him being killed by a Vietcong bullet. From 1969 to 1986 (when he ended up in this psychiatrist’s clinic), on the anniversary of his friend’s death, this man would commit ‘armed robbery’ by putting his finger in his pocket and carry out ‘an armed hold-up’ in order to provoke gunfire from the police. His compulsive and unconscious re-enactment came to an end when he understood its meaning through the process of psychotherapy.

Females are more likely to dissociate when exposed to a traumatic event: they show a mild tachycardia and ‘internalise’ their symptoms. Males are more likely to display fight-or-flight responses and tachycardia when exposed to a fearful experience, but in some adolescent boys the heart rate normalises as they grow older and some violent young men reported a ‘soothing’ feeling when they began ‘stalking’ a potential victim (Perry 1995).

Understanding PTSD as a ‘sensitisation disorder’ of the attachment system

Post-traumatic stress disorder is classified in ICD–10 as a stress-related disorder and in DSM–IV as an anxiety disorder. However, in 1997 Yehuda reported that ‘contrary to all initial expectations and hypotheses, the neuroendocrinology of PTSD does not resemble the neuroendocrine alterations observed in stress’ High cortisol levels have traditionally been associated with stress but, among her sample of victims of road traffic accidents, only those who showed a lower than normal release of cortisol developed PTSD. She commented, ‘It may be that PTSD reflects a biologic sensitisation following stress due to preexisting risk factors. If so, perhaps it might be more appropriate to consider the symptoms and neurobiologic changes following trauma as reflecting a posttraumatic sensitisation disorder rather than a posttraumatic stress disorder’ (Yehuda 1997: p. 69. Italics as in original).

This hypothesis is supported by evidence showing correlations between suppressed cortisol levels and insecure avoidant behaviour in 1-year-old infants (Tenenes 1977) and in maltreated children (Hart 1995), Yehuda’s view that PTSD is a ‘sensitisation disorder’ that may be attributed to a priming of the hypothalamic–pituitary axis seems very likely. This could be the result of either trauma-induced damage to the attachment system during early development or to repeated or chronic traumatisation in later life (Wang 1997; Cichetti 2001; Gunnar 2002).

In adults, low urinary cortisol levels have been found in Holocaust survivors with PTSD and in Vietnam veterans, where the urinary cortisol levels were strongly negatively correlated with degrees of emotional numbing (Yehuda 1997: pp. 58–62).

There also appears to be evidence of transmission of this sensitisation to PTSD down the generations: Yehuda notes low cortisol levels both in Holocaust survivors and in their adult offspring (Yehuda 2002) as well as in women suffering from PTSD as a result of the destruction of the World Trade Centre and in their infant offspring (Yehuda 2005).

Although a recent review of research on cortisol and PTSD concluded that low cortisol levels do not relate to PTSD in general but seem to reflect particular types of trauma or subgroups of individuals (Meewise 2007), this literature search only went up to March 2005 whereas Yehuda’s paper was published in July 2005. In addition, when carrying out their subgroup analyses Meewise...
et al found that testing plasma or serum showed significantly lower levels of cortisol in people with PTSD than in controls not exposed to trauma. Lower levels were also found in afternoon samples of people with PTSD and, of particular relevance here, when females were included, they found low cortisol levels in studies on physical or sexual abuse.

The apparent transmission of low cortisol levels and its significance in terms of predisposing individuals to PTSD is an important finding which, if confirmed, highlights the role of the transgenerational transmission of vulnerability to PTSD through the attachment system. It further confirms Schore’s view that a secure attachment is a primary defence against trauma-induced psychopathology (Schore 1996). This possibility is even more likely if we take into account Van Ijzendoorn & Bakermans–Kranenberg’s (1997) review of the research in the field of attachment, which shows a 75% correspondence between a mother’s attachment and that of her offspring. These findings are important when analysing possible genetic transmission.

The conceptual basis to the treatment of complex PTSD

The NICE guidelines on the treatment of simple PTSD recommend trauma-focused cognitive–behavioural therapy (CBT) or eye movement desensitisation and reprocessing (EMDR) as the first line of treatment (National Collaborating Centre for Mental Health 2005). It is suggested that medication be tried only if a trauma-focused psychological therapy fails.

The success of trauma-focused EMDR and CBT is confirmed in a relatively recent review of randomised controlled trials of psychological treatments for chronic PTSD (present for more than 3 months) (Bisson 2007). Stress management sessions and group cognitive–behavioural therapy over 8–12 weeks also improved PTSD symptoms compared with waiting-list or ‘usual care’ control conditions. However, the review does not state whether the patients involved had simple or complex PTSD, nor whether they included refugees and victims of torture. It does mention that Vietnam veterans deteriorated with trauma-focused CBT and EMDR: as these individuals must have been suffering from PTSD for a very long time, it is likely that most of them had complex rather than simple PTSD. The authors conclude by suggesting that extending the number of sessions and making the treatment more acceptable to patients (withdrawal from some studies was as high as 30%) may be necessary, as well as adding a course of pharmacological treatment.

The sad fact remains that there are currently no established guidelines regarding the treatment of complex PTSD. This is in large part due to the confusion surrounding its diagnosis and the lack of randomised controlled therapeutic trials in this field. There are, however, an increasing number of outcome studies reporting on different therapeutic modalities for the treatment of borderline personality disorder (Bateman 2001, 2003; Beecham 2006), which, as discussed above, is very similar to complex PTSD. Both Bateman and Fonagy use an attachment-based model for their therapeutic work.

It therefore makes sense to extend the use of attachment theory and research in the treatment of people with developmental or complex PTSD (de Zulueta 2006b; Lab 2008). It also allows for the inclusion of psychoanalytically derived treatments such as cognitive analytic therapy (CAT) (Ryle 1997) and affect-focused therapies such as dialectical behaviour therapy (Koerner 2000) and EMDR.

**Eye movement desensitisation and reprocessing**

In EMDR, the processing of traumatic memories is achieved by evoking the memory and applying bilateral stimulation in accordance with a clearly outlined protocol (Shapiro 1995). The stimulation can involve eye movements, sounds or tapping the patient above the knees, depending on the client’s preference and with their full consent. Henry (1997) pointed out that since PTSD can produce a functional dissociation of emotional processing across the two hemispheres of the brain, the therapeutic role of EMDR may be that of enabling this processing to be restored across the corpus callosum.

It is not necessary for the patient to give a detailed account of their traumatic experiences during EMDR: they only have to give the therapist a rough idea of what they are processing.

It is the bilateral stimulation in EMDR that essentially distinguishes it from CBT and may also be why it is so effective in the treatment of somatic symptoms. Such symptoms often predominate in the presentation of patients from the Middle East, Africa and the Far East who have complex PTSD (Al Krenawi 2005). The additional fact that these individuals often do not speak much English and that they feel so much shame in relation to their experiences of rape and torture makes the use of EMDR a very useful tool in their therapy.

Mollon (2005) gives a vivid clinical description of work in this field, as well as describing the ‘energy therapies’ that are being used to improve affect modulation in PTSD.

All these developments are very helpful in building up a positive attachment between a distrustful patient and their therapist while addressing the patient’s overriding fear of facing the memories of their past traumatic experiences.
Assessment of patients with complex PTSD

The assessment of patients with complex PTSD can extend to two or even three sessions (Lab 2008). In the Traumatic Stress Service at the Maudsley Hospital, we include an evaluation of the individual’s social support system and try to determine how securely attached they are by focusing on their early attachment patterns and their current levels of dissociation (measured using the Dissociation Evaluation Scale; Bernstein 1986). The primary purpose of the assessment is threefold:

- to establish whether the patient has the resources, both internal and external, to be treated for their symptoms of PTSD
- to establish whether it is safe to proceed with therapy without risk of re-traumatising the patient
- to determine how complex the treatment is likely to be bearing in mind the attachment history and current levels of support.

Substance and alcohol addiction are contra-indicated and the patients are referred to addiction services before any PTSD treatment can be contemplated.

Self-destructive behaviour and suicide risk tend to increase in the early phase of treatment. It is therefore important to assess these and put in place support systems if necessary, involving community mental health teams or general practitioners. Risk assessment should take into account the patient’s potential for violence to self and others and their levels of dissociation in relation to their behaviour with other members of the family. Usually, such information can be obtained only by meeting with the partner or another close relation or friend, who can also provide essential information about changes in the patient’s behaviour and personality over time. A family meeting is often useful to further establish levels of risk and need in the family.

Another important function of a family meeting is to provide the assessor with an opportunity to explain to family members the nature of PTSD, treatment must be carried out from what Bowlby (1988) called a ‘secure base’ (further discussed below). This is particularly obvious when dealing with domestic violence: therapy should not begin before the (usually) female victim has established a safety net of individuals, a potential refuge and contact with the police. This is because as a woman gains confidence through treatment, she becomes more of a threat to her controlling partner (who, in serious cases, often has a borderline personality disorder himself). This can lead to increasing violence or even homicide. In cases of child abuse, victims who are still living with their family, be it with either the abuser or the caregiver who allowed the abuse to happen, may need to move out of the family home before engaging in therapy addressing their traumatic experiences.

Case vignette 4

A West Indian woman was taken on in therapy and asked to keep a diary of the work she did. She had moved back into her mother’s house while on the waiting list for therapy but the therapist had not considered the possible implications of this move.
When her mother found this diary, the young woman was subjected to the same anger and abuse she had incurred when, as a child, she had tried to inform social services of abuse perpetrated by her father. On that occasion, she withdrew her allegations. On this occasion, the experience of re-traumatisation led to her admission to hospital because of the risk of suicide.

**The need for phase-oriented treatment**

The conceptual infrastructure underpinning the therapeutic work in the Maudsley’s Traumatic Stress Service follows the guidelines set up in the USA by Herman (1992b) and Bloom (1997). It is based on an attachment model within a systemic framework in which PTSD symptoms are understood as the psychobiological manifestations of a disrupted attachment system requiring an approach that addresses both the internal world of the individual and their social attachment network (de Zulueta 2006b).

Once the patient has been assessed by a team member, the findings are discussed in a team meeting and the treatment best suited to the patient’s individual and social needs is decided. Therapists provide a range of therapeutic orientations, including psychodynamic, systemic and attachment-based psychotherapy, CBT and EMDR. Within these different individual approaches, however, narrative exposure therapy (Schauer 2005), CBT techniques (Ehlers 2000) and particularly EMDR (Shapiro 1995) are used to process the traumatic memories at the appropriate stage in treatment.

**Phase 1: Stabilisation**

The focus here is providing safety – a secure base. This can take a relatively long time with severely traumatised or unstable patients. It is achieved through psychoeducation, techniques focusing on affect regulation and the establishment of a cohesive support network, all of which also results in the development of a good therapeutic alliance.

With asylum seekers, it is often important to attend to housing and immigration problems to give them the sense of safety they need before they can work through their traumatic experiences. At this stage, generic rather than specialised skills are required. Medication can play an important role in treating concomitant severe depression, and overwhelmingly high levels of arousal and flashbacks can be reduced by prescribing very low levels of an antipsychotic medication such as olanzapine, as outlined in the NICE guidelines (National Collaborating Centre for Mental Health 2005).

**Phase 2: Remembering, processing and grieving**

Avoidance and fear of the traumatic memory are central to post-traumatic psychopathology. Therefore a paced and modulated approach to this material is essential to the reactivation of the traumatic memory.

At the Maudsley we use CBT and EMDR techniques to process traumatic memories, but in combination with another therapeutic approach such as narrative exposure therapy (Schauer 2005).

In many patients suffering from complex PTSD, often with borderline traits if not a diagnosis of borderline personality disorder, resolution of intense insecure attachments to abusive or neglectful caregivers or other abusers often needs to be addressed during this phase (de Zulueta 2006c). Some patients do not feel ready to go through this stage and they should not be forced to do so because of the risk of re-traumatisation.

**Phase 3: Personality integration and rehabilitation**

Personality integration and rehabilitation can be ongoing throughout the therapy. However, it becomes increasingly important towards the end of treatment, when patients begin to reconnect with life in the ‘here and now’ and are encouraged to engage in gratifying activities such as sports, dance, art or other right-hemisphere based activities. They examine with their therapist the changes they have made and, by reconnecting with others, facilitate the process of ending the therapy. Termination of therapy is often painful as it can bring back the feelings of separation and loss related to their past, but for this reason it is extremely important.

**Group therapy**

By its very nature group therapy is, as Yalom (1995) has outlined in his 11 curative factors (Box 2), an excellent treatment mode for people with PTSD: the possibility of sharing and helping others to make sense of what has happened to them while working together towards recovery and rehabilitation gives patients hope and dispels their overriding sense of loneliness. In the early phase of stabilisation, the group model can be used to teach and practise general psychoeducation techniques. Subsequently, therapy can continue with specific groups of individuals who have suffered a common trauma.
Conclusions

When Lindemann in 1944 defined psychological trauma as ‘the sudden cessation of human interaction’, he was addressing its essential feature, the sudden disruption of an individual’s attachment system and all its manifestations. Attachment research provides a comprehensive understanding of the psychobiological symptoms of both simple and complex or ‘developmental’ PTSD (Van der Kolk 2005) and supports Yehuda’s view that PTSD is in fact a sensitisation disorder of the hypothalamic–pituitary axis. It also accounts for the fact that many people with complex PTSD, usually those with an earlier experience of attachment failure, do not respond to CBT or EMDR alone, requiring the other interventions outlined above, focusing particularly on the different manifestations of emotional dysregulation and dissociation.

If we understand PTSD as resulting from the disruption of the attachment system, we can also understand why the lack of social support is the most important risk factor for this disorder. This finding makes sense of the fact, for instance, that an asylum seeker with a history of severe political abuse can remain free of symptoms of PTSD while living within his London community, only to present with severe symptoms when removed from those to whom he is attached. It also explains why many people with a history of childhood abuse can remain free of symptoms of complex PTSD until they experience the loss of an important attachment figure in adulthood.

Finally, an attachment perspective may contribute to the resolution of the diagnostic impasse between complex PTSD and borderline personality disorder.

References


MCQs

1. The following are characteristics of PTSD:
   a. PTSD is more frequent and severe in victims of natural disasters than of man-made disasters
   b. Good affect regulation
   c. Reduced autonomic response
   d. The most important risk factor is the lack of social support
   e. An increased activation of Broca’s area (the speech area on the left hemisphere).

2. As regards PTSD:
   a. The NICE guidelines cover the treatment of both simple and complex PTSD
   b. A stressful event of a threatening or catastrophic nature always leads to PTSD
   c. Securely attached individuals are more likely to develop PTSD
   d. Attachment behaviour is activated by fear
   e. The NICE guidelines do not recommend the use of EMDR in the treatment of PTSD.

3. Attachment research shows that:
   a. Children with a disorganised attachment show good affect regulation
   b. Dissociation due to abuse or neglect in early infant development has no impact on the individual’s sense of self in later life
   c. There is a 75% correspondence between a mother’s attachment and that of her offspring
   d. Patients with a borderline personality disorder or complex PTSD are untreated
   e. Attachment behaviour involves mainly the left hemisphere.

4. Complex PTSD:
   a. Exists as a specific diagnostic entity in DSM–IV
   b. May follow persistent and prolonged traumatic events often in association with an interpersonal stressor
   c. Is not associated with dissociation
   d. Can be treated without prior stabilisation of the patient
   e. Is not associated with re-traumatisation.

5. Cortisol levels in people who develop PTSD:
   a. Are often higher than normal
   b. Are often lower than normal
   c. Have not been found to be low in the children of adults with PTSD
   d. Do not indicate any sensitisation of the hypothalamic–pituitary axis
   e. Are normal in victims of child sexual abuse who suffer from PTSD.
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