Advances in Psychiatric Treatment (2006), vol. 12, 297–305

Personality disorders as disorganisation of attachment and affect regulation

Jaydip Sarkar & Gwen Adshead

Abstract If personality disorder is no longer to be a diagnosis of exclusion it needs a conceptual framework that fits both the symptoms of the illness and the behavioural problems that constitute its current diagnostic criteria. In this article, we suggest that personality disorder is best understood as disorganisation of the capacity for affect regulation, mediated by early attachments. We present evidence for this argument based on both developmental and neurobiological research.

Personality disorders are common psychiatric disorders, especially in primary care (Moran *et al*, 2000; Singleton *et al*, 2001), and they carry significant costs for healthcare services (Moran *et al*, 2000). People with personality disorders present a problem in psychiatry because they demonstrate both symptoms of psychological distress and social rule-breaking behaviour. They therefore invite punitive as well as therapeutic responses, which can lead to confusion and negativity in service providers (Watts & Morgan, 1994).

In the past, people with personality disorders were frequently excluded from mental health services, on the grounds that they could not be treated or that they were not ill. However, exclusion is no longer an option in the UK. Department of Health policy emphasises that service providers should address the needs of people with personality disorders, especially those who harm themselves (Department of Health, 2003; National Collaborating Centre for Mental Health, 2004) or others (Home Office & Department of Health, 1999).

Current models of personality disorder used by psychiatrists tend to focus largely on the problem behaviours. However, behaviours cannot be symptoms: they involve assumed intention, especially where social rules and conventions are breached or broken. It is also not clear how to treat a behaviour, in the absence of an understanding of its underlying cognitive schema and neurobiological basis.

What might be helpful, then, is a model that would allow personality disorders to be understood much as we understand other illnesses that cause harmful dysfunctional states (Wakefield, 1992). This might offer both a better understanding of why symptoms occur and why and how certain treatments work, and further options for treatment. In this article, we suggest that the major feature of personality disorders is a failure of affect regulation. We present evidence on the neurobiology of affect regulation and on its development within attachment relationships in a heuristic model that explains both the symptoms of and effective treatment strategies for personality disorders. Being heuristic in nature, this model will, we hope, form the basis of further empirical research.

Affects

What are they and where are they formed?

E(x)motion indicates a departure from a basic state of calm (Freeman, 1999: p. 124) (emotions and affects are essentially similar terms and we use them interchangeably in this article). Damasio (1994) posits that emotions are bodily experiences (somatosensory states) in response to external and internal influences. Several areas of the somatosensory cortex are associated with the recall of emotional experiences, especially the insula, cingulate cortex, hypothalamus and several nuclei of the brain-stem tegmentum (Damasio, 2003). The bodily states created include autonomic, neuroendocrine and somatomotor responses that are subjectively experienced as feelings and are expressed through a range of somatomotor responses, including facial, gestural, vocal and behavioural reactions. Thus, behaviour is merely one expression of an affective state; individuals also use words (written and spoken) and facial expression to communicate affective states.

Jaydip Sarkar is a consultant forensic psychiatrist, currently working at the Shaftesbury Clinic (South West London and St George's Mental Health NHS Trust, Springfield University Hospital, 61 Glenburnie Road, London SW17 7DJ, UK. Email: amdeep176@aol.com). Gwen Adshead is consultant forensic psychotherapist at Broadmoor Hospital, Crowthorne, UK.

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

What are their functions?

Affects act as a driving force or catalyst to assist humans in pursuit of goal-directed behaviours that help us to find sources of energy, fend off external obnoxious agents, and make and maintain social relationships to support a life-sustaining homoeostasis (Panksepp, 1998; Rose, 1998; Damasio, 2003). This is achieved by a complex interplay of multiple systems and events within the body that lead to an automated regulation of life. The 'machinery' involved includes a number of systems (Box 1), nested within each other, that are ultimately driven by emotions. No one system acts in isolation: simple systems are regulated by more complex ones (Damasio, 2003). Affect regulation is one aspect of the more complex systems required for optimal homoeostasis. This nested principle, with the emotions governing the motivational machinery of the body, includes, but goes beyond, the reductionist view of affects simply as states elicited by rewards and punishments (Rolls, 2000).

Humans are unique among animals in their long period of total dependence on others for survival after birth. Like other non-human primates that live in social groups, people instinctively make and maintain different types of social relationship for survival. These relationships are a function of time, complexity and interpersonal attachments. Interpersonal affective responses need to be both regulated and organised to be effective. In particular, the most favourable management of relationships requires the capacity to regulate negative affects such as anger and anxiety. This is especially true of relationships characterised by discrepancies of power and those that involve dependency and neediness, for example relationships with partners, children, family members and professional carers.

We suggest that the need and the ability to coexist with others in order to survive optimally are fundamental to the development and maintenance of affect regulation. In other words, the affects fine-tune the organism's struggle for survival, but affect regulation can improve the quality of that survival.

Box 1 Key components of the body's emotion-driven multisystem machinery

- The endocrine system
- Simple reflexes (e.g. the startle reflex, which is protective in nature and has survival value)
- The immune system
- Drives and motivations (e.g. hunger, thirst and curiosity)
- Approach and avoidance behaviours that lead to appetites and desires

Affect production and regulation

Regulation in any homoeostatic system (including that of affect) means not only initiating a response to a stimulus, but also modulating it appropriately and turning it off when no longer required. Regulation also implies that the response itself is organised and effective. Phillips *et al* (2003) suggest that affective experience involves:

1 identification of the emotional significance of a stimulus;
2 production of an affective state in response;
3 regulation of the affective state.

Identification of emotional significance

Two areas of the brain – the amygdala and the insula – are involved in the identification of the emotional significance of a stimulus. The amygdala is responsible for modulation of vigilance and attention to emotionally salient information. The insula conveys aversive sensory information to the amygdala, and the two areas act in concert to detect and respond to threatening and aversive stimuli. They can be conceptualised as a defence radar alerting the organism to the presence of threat in its environment and stimulating a fight or flight self-preservative response (see Phillips *et al*, 2003).

Production of a responding affective state

Sites implicated in triggering the production of affective states in response to a stimulus include the amygdala, insula, parts of the anterior cingulate gyrus, striatum, and orbitofrontal and ventromedial prefrontal cortices.

The amygdala subserves fear-conditioning (Bechara *et al*, 1995) and autonomic reactions associated with feelings of fear (Gloor, 1992). The insula is implicated in induced sadness, and anticipatory, phobic and traumatic anxiety (Charney & Drevets, 2002). It is also activated during internally generated self-directed disgust, i.e. social emotions such as guilt and shame (Shin *et al*, 2000). Stimulation of the ventral (affective) division of the anterior cingulate gyrus evokes autonomic and visceromotor changes and spontaneous emotional vocalisations (Bancaud & Talaraich, 1992). The ventral striatum appears to be involved in craving (Breiter *et al*, 1997), anticipation of reward (Pagnoni *et al*, 2002) and romantic love (Bartels & Zeki, 2000). The orbitofrontal cortex is

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

associated with autonomic changes accompanying affective states such as anger (Dougherty *et al*, 1999) and physical aggression (Pietrini *et al*, 2000). The ventromedial prefrontal cortex is involved in induction of sad mood (Pardo *et al*, 1993) and guilt and in responding to facial expressions of negative emotions (Sprengelmeyer *et al*, 1996).

Regulation of the affective state

Affect regulation is largely dependent on the functioning of two neural systems: a ventral and a dorsal system (Phillips *et al*, 2003).

The ventral system includes the amygdala, insula, ventral striatum and ventral (affective) regions of the anterior cingulate gyrus and prefrontal cortex. It is important for rapid appraisal of emotional material, and automatic affective regulation in response to social interactions, including the capacity for interpersonal empathy.

The dorsal system includes the hippocampus and dorsal (cognitive) regions of the anterior cingulate gyrus and prefrontal cortex. It supports selective and sustained attention, planning and effortful (rather than automatic) regulation of affective states, and autonomic responses to those states. Here affect regulation involves cognitive appraisals: using logic and rational evaluations, based on past experience and anticipated future outcomes.

These contributions of the two systems might be summarised as insight and foresight respectively (Freeman, 1999: p. 124) (Box 2).

The role of attachment relationships in affect regulation

Schore (2002, 2003) has set out an explanatory framework for affect dysregulation, based on research into the neural development of the infant brain. He reviews the evidence that the rearing environment (in the form of the infant's relationship with the mother or other primary carer) has a direct effect on the development of brain structures and pathways involved in affect regulation. Animal research by Suomi (1999, 2003) has also demonstrated the importance of the interaction between the genetic basis for neural and synaptic development (temperament) and the developing infant's socio-emotional environment (nurture) in the development of neurotransmitter systems and cytoarchitecture .

Box 2 The neural systems that govern affect regulation

Insight is mediated by the ventral system:

- amygdala
- insula
- ventral striatum
- ventral (affective) regions of the anterior cingulate gyrus and prefrontal cortex

Foresight is mediated by the dorsal system:

- hippocampus
- dorsal (cognitive) regions of the anterior cingulate gyrus and prefrontal cortex

Secure attachment

In humans, attachment operates through the interaction of two behavioural systems: caregiving and care-eliciting (George & Solomon, 1996). These foster identification of affects, the response to them and the regulation of the affective system. It is useful to conceptualise the interaction between a caregiver and care-elicitor as one that regulates the experience of emotions through a crescendo–decrescendo process (Schore, 2002). A distressed infant responds to threats in his (or her) environment by experiencing a high degree of arousal, mediated by the sympathetic division of the autonomic nervous system. This is a catabolic system, making available large amounts of energy to prepare the infant for a self-preservative action repertoire of a fight/flight mode. The infant experiences the peripheral and central effects of noradrenaline (norepinephrine) (e.g. more rapid heart and pulse rate, increased blood pressure, dilated pupils), which are uncomfortable. By soothing the infant, the mother helps in recruiting the infant's parasympathetic system, which has opposite effects and restores homoeostasis. There is a return to normal rate and rhythm of the autonomic system. The sympathetic system supports an action-consuming state, whereas the parasympathetic system supports a withdrawal–conservation state.

The earliest attachment figure conceivably acts as a primary affect regulator, one that ameliorates and terminates the infant's distress, augments within reasonable limits its experience of happiness and pleasure, and offers predictable and replicable affect regulation. The basic language of attachment relationships thus consists of episodes of interactive signals produced by the autonomic nervous system in both infant and caregiver. These episodes emerge at about 2 months of age, and they are highly arousing, affect-laden and short interpersonal events that expose the infant to high levels of cognitive and social information (Feldman *et al*, 1999). As the infant grows, it is the relationship, rather than a particular caregiver, that becomes the (accessory) affect regulator.

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

A right prefrontal cortex regulation of the autonomic nervous system lies at the heart of the development of affect regulation in an infant. The right hemisphere is also centrally involved in corporeal self-identity and its relation to the environment, distinguishing the self from non-self (Devinsky, 2000). Infant–maternal attachment behaviour is almost exclusively body to body, and it is now accepted that the right hemisphere is involved in the social and biological functions of the attachment system in the infant (Wang, 1997). Furthermore, this hemisphere is crucial in the receptive and expressive empathic processes (Adolphs *et al*, 2000), which are processed unconsciously using extensive reciprocal connections with both the limbic systems.

A good-quality affect regulatory system, based on secure attachment, leads to optimal right hemispheric maturation at a critical period during the first 2–3 years of life (Schore, 2002). Any experience that disturbs the development of secure attachment at a time of heightened dependence (e.g. abuse, neglect or inconsistent caring) will lead to impaired development of neural pathways that subserve emotional behaviours, such that impaired emotional regulation is likely to persist throughout the individual's lifetime.

The final task in terms of emotion processing involves the internalisation of affect-regulating capacity. Up to about 5 years of age, children locate both affects and their stimuli outside of the self. Any adverse emotional experience is therefore ascribed to the object (including humans) causing it: an externalisation of affects. Later, children locate emotions internally and still later they can identify mixed and conflicting emotions (Levine *et al*, 1997). Thus, emotion is initially perceived as being caused, and is in reality regulated, by others, but over the course of early development it becomes increasingly self-regulated as a result of neurophysiological development (Thompson, 1990: p. 371).

Insecure attachment

The successful outcome of secure attachment is the development of the basic machinery to self-regulate affects later in life (Fonagy *et al*, 2002). Insecure attachment prevents the development of a proper affect regulatory capacity. The individual is left with either an inability to balance sympathetic hyperarousal in response to threat, or the production of an untimely or inadequate parasympathetic response. Dysregulation of this nature leads to prolonged persistence of a catabolic state of fight/flight hyperarousal, or a sudden and inappropriate shift into an anabolic withdrawal–conservation state of 'freezing'. The latter occurs when a situation is perceived to be hopeless and one's own agency inadequate, leading to inhibition and avoidance in order to become 'unseen' (a state of dissociation) as a defensive strategy of last resort (see Schore, 2002). Alternatively, there could be rapid cycling between states of hyperarousal and withdrawal, resulting in gross disorganisation of both affects and associated behaviours.

Affect dysregulation and symptoms of personality disorder

Individuals who have experienced insecure attachment are at risk of developing dysregulated and disorganised affective systems. Both small-scale (Patrick *et al*, 1994; Fonagy *et al*, 1997) and larger studies (Johnson *et al*, 1999) have found that early childhood adversity, especially neglect, is a risk factor for the development of personality disorders. Childhood sexual abuse is also a risk factor for the development of self-harming and suicidal behaviour in adulthood (Andrews *et al*, 2003) – behaviour commonly seen in people with personality disorders.

One major outcome of this is that people with personality disorders have significant difficulty in establishing and maintaining interpersonal relationships that require good affect regulation. They seem to withdraw from and alienate others and/or engage in confusing and disorganised relationships. This characteristic is observed particularly within dependency relationships during adulthood (e.g. relationships with peers, partners, children and professional carers), which may be experienced as disparities of power and vulnerability, giving rise to a sense of threat and fear. Inability to regulate negative affects within dependency relationships increases the chance of responding with unregulated hostility or anger. This puts these individuals at a double disadvantage: not only do they tend to alienate caregivers, but they are likely to do it at times of greatest need.

In exhibiting this behaviour, individuals with personality disorders are moving away from the recently (in evolutionary terms) evolved adaptive species-preservative behaviour seen in mammals towards a more ancient self-preservative behaviour. As the name suggests, species-preservative behaviour has evolved to improve the chances of survival of a species, and it is based on parental care, nursing, social interaction, pair-bonding and mutual defence (Henry & Wang, 1998). If trauma results in a stressful loss of control, the self-preservative fight/flight catecholamine coping-response takes priority. Problems arise when this style becomes the default coping response to a wide range of events, people and circumstances. It is then maladaptive and inappropriately accessed.

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

Regulation of negative affect

The problem is not that people with personality disorders are 'affectless' but that they have too much or too little affect, depending on the perceived social stimulation, i.e. the affective system is dysregulated and the responses disorganised. Affective dysregulation also implies an unpredictability that goes beyond either an excessive or diminished response.

Heightened perception of threat seems to be a major problem for people with personality disorders, one that emphasises a lack of safety with and an essential untrustworthiness of others. This is compounded by an inability to repair the emotional states stimulated by threat or fear. They seem to lack the capacity to soothe themselves after fearful experiences (van der Kolk & Fisler, 1994), becoming and remaining hyperaroused in an uncontrollable, dysregulated manner. Their difficulty in providing an internal discourse for themselves to manage negative affects leads to the expectation or requirement of an external solution when they feel bad – preferably from another person whom they identify as having a caring role.

In people with personality disorders there appears to be a deficit, if not an absence, of the shift in locus from external to internal affect regulation. They appear to continue to believe that emotions are almost always an outcome of external developments caused by other people. This is a problem of excessive externalisation of experience of negative affects, a task that should have been resolved around 5 years of age. Such responses are therefore age inappropriate and immature.

Box 3 Affect dysregulation with personality disorder

Cluster A
Prototype: paranoid personality disorder
- Consistent under-regulation of the affects of fear and terror
- Over-regulation of positive affects – narrow range of affective expression
- Over-regulation (muting) of all affects in schizoid personality disorder

Cluster B
Prototype: borderline personality disorder
- Dysregulation (under- or over-) of both positive and negative affects, but predominantly demonstrated with fear, anger, sadness and anxiety
- Under-regulation of fear, arousal and anger, and over-regulation of feelings of empathy, remorse and guilt in antisocial and narcissistic personality disorders
- Under-regulation of most affects in histrionic personality disorder

Cluster C
Prototype: anxious/avoidant personality disorder
- Under-regulation of social emotions, e.g. shame and guilt
- Under-regulation of anxiety and sadness
- Positive affects usually experienced only when with others

Regulation and specific personality disorders

Both ICD–10 (World Health Organization, 1992) and DSM–IV (American Psychiatric Association, 1994) implicitly endorse, without prioritisation, the characteristic disturbances in affect regulation found in most personality disorders and all personality clusters (Geiger & Crick, 2001) (Box 3).

Cluster A

Cluster A personality disorders (paranoid, schizoid, schizotypal) are characterised by increased paranoia and suspiciousness of others. People with paranoid personality disorders have increased suspiciousness and arousal based on the excessive fear that arises from their heightened perception of threat, under-regulation of fear and a fight/flight response pattern. Those with schizoid and schizotypal personality disorders also have a predominantly constricted affect. Individuals with schizoid personality disorder also experience lack of pleasure and an affective indifference towards others, suggesting perhaps a muting of all affective responses on account of over-regulation of affects. Schizotypal personality disorder is characterised by inappropriate affect and heightened social anxiety, secondary to paranoia, and lacking in habituation.

Cluster B

Cluster B personality disorders (borderline, antisocial, histrionic and narcissistic) are the classic example of dysregulation, and borderline personality disorder is the prototype. In this cluster there is clinical evidence of dysregulation of all negative affects, primarily involving fear and anger, but including depression and anxiety.

People with borderline personality disorder alternate between having either no trust in others or a highly risky tendency to fail to see threat when it is present. They also experience predominantly depressive mood disorders and poorly controlled anger, and form under-regulated, intense attachments to others that are often a source of further affective distress and arousal.

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

Histrionic personality disorder is characterised by shallow or labile affect, excitement seeking and an exaggerated emotional expression.

Individuals with antisocial personality disorder show an excessive capacity for blaming others (externalisation of affect) and have little or no regard for the feeling of others, as exemplified by impairment in empathy, remorse and guilt. They share certain characteristics with those with narcissistic personality disorder, who also show little empathy but also excessive envy and jealousy. People with either disorder seem to see others as highly risky and unstable sources of aggression or threat, and their own aggression, paranoia and cruelty to others is likely to be due to under-regulation of arousal in response to threat. They seem to have difficulty in regulating emotions that have a social valence, suggesting a dysfunction based in the prefrontal cortex. Not surprisingly, people with these particular disorders have the greatest difficulty in adapting to social norms and customs.

Cluster C

The avoidance behaviour so characteristic of Cluster C personality disorders (avoidant, dependent and anakastic) may be seen as avoidance of situations, people and thoughts that provoke unmodulated affect, usually severe anxiety and panic, in a classic behavioural style.

Whereas people with anakastic personality disorders show excessive doubt and caution, avoiding risks altogether, those with avoidant personality disorders have a heightened fear of criticism and disapproval, with a possible heightened sense of shame and ridicule.

People with dependent personality disorders have exaggerated fears of their own ability to care for themselves and therefore avoid being alone, depending on others to validate their existence.

Substance misuse

Substance misuse is a common feature of personality disorders. It is likely that the pathways that mediate the hedonic properties of psychostimulants evolved as neural systems for social attachment. There is evidence that brain activation patterns in adults responding to attachment figures (partners or children) are similar to neural responses to cocaine-induced euphoria (Bartels & Zeki, 2000). The brain structures involved include bilateral activation in the anterior cingulate gyrus, medial insula and ventral striatum. These findings suggest that the high rates of substance misuse by people with personality disorders may therefore lie, in part at least, in their dysfunctional or absent social attachments. Substance use (and misuse) acts as a social 'integrator', both externally with peers and internally through the induction of a pleasurable state. This state replaces the very basic human quality of gregariousness. Furthermore, substances are used as external regulators of negative affects because the individual perceives these affects to be externally, not internally, caused.

Violence

Some personality disorders are associated with high rates of violence and rule-breaking. Blair (2001) suggests that violence can take one of two forms: reactive violence, which is elicited in response to frustration or threat, and instrumental violence, which is goal-directed, purposeful and apparently unprovoked.

Reactive violence has been conceptualised as a response to perceived threat, mediated by the hypothalamus–periacqueductal grey matter system. The amygdala feeds information into the periacqueductal grey matter system on the current state of threat, thus determining whether the response is fight or flight. The orbitofrontal cortex has extensive projections to autonomic control centres in the medial hypothalamus and periacqueductal grey matter, and it is damage specifically to this part of the frontal lobe that leads to greatest risk of reactive violence (Grafman *et al*, 1996). Thus, reactive violence is a consequence of inadequate regulation of threat-based affects, largely by the dorsal prefrontal cortex.

Instrumental violence is a function of cruelty and lack of empathy (Hare *et al*, 1991), which in turn has been linked to muted autonomic responses to sad and fearful facial expressions. In this type of violence, it has been speculated that the affect regulating system of the prefrontal cortex remains intact, but there is a fundamental problem within the amygdala, the area concerned with properly identifying fearful and sad emotions (Blair, 2001). Other studies have implicated bilateral frontotemporal-based cognitive strategies for processing affective material (Blair *et al*, 1997), and disturbed functional connectivity of brain areas related to emotion processing (Müller *et al*, 2003). Clearly, the final word has not been said on this issue.

Implications for treatment

Essentially, all therapeutic interventions in psychiatry seek to regulate affects by various means (Bradley, 2000: p. 146), and it is no different for personality disorders. Cognitive psychotherapies are likely to engage the dorsal prefrontal system, which is involved

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

in the use of reason, logic and foresight, to influence affect regulation. Relationship-based therapies (including individual and group psychodynamic therapies), which are based on emotional experiences, are likely to be processed in the ventral prefrontal cortices. This is consistent with evidence that mild to moderate degrees of personality disorder can be treated using a combination of psychotherapies (Bateman & Tyrer, 2004).

An affect-regulation model of personality disorder also helps to explain the use of polypharmacy to treat it. This includes all classes of psychotropic drugs, which are often used on a trial and error basis (Tyrer & Bateman, 2004). It has been proposed that, although most psychotropics have some specificity for psychiatric disorders, most have a generic affect- (anxiety-) regulating function. Antipsychotic medications are most effective for the most intense and disorganising anxiety (psychotic reactions), whereas antidepressants and sedatives have an anxiolytic effect in the less disorganising types (LeDoux, 1996). This may explain the efficacy of mood-stabilising agents in the management of personality disorders. Given the prevalence of substance misuse in personality disorder, it is hardly surprising that any prescribed drugs that reduce arousal or regulate affect will be as effective (or ineffective) as illicit drugs; nor is it surprising that people with personality disorders may misuse prescribed drugs.

Affect regulation is also relevant in group processes such as therapeutic communities, which are clearly effective for mild to moderate personality disorder (Lees & Manning, 1999). Community members report feeling more confident in dealing with their own (insight) and others' negative feelings (empathy), especially hostility and rage. The therapeutic benefit of such communities for people with personality disorders may arise from the secure attachment to the community that they can make, which allows them to develop a greater capacity to manage negative affect internally.

Conclusions

Our heuristic model is based on an integrative synthesis of recent empirical evidence from the fields of attachment and neurobiology, relating it to current strategies for treating personality disorders. It proposes a biologically grounded system that is nevertheless sufficiently based on clinical findings to be clinically relevant.

Affect regulation is only one, but arguably the most critical, aspect of personality disorder. Given its developmental origins, it is a key foundation on which other aspects of personality – thoughts, perceptions and behaviour – are built.

Our model is purposely limited in scope and does not incorporate neuroendocrine regulation or the involvement and interaction of various neurotransmitters and neuromodulators. Neither does it deal with the problem of comorbid mental illness and the fact that personality disorders rarely occur singly. Finally, the model does not explain all types of personality disorder, especially psychopathy and schizotypy. We suggest that there are fundamental differences in the brain mechanisms underlying the latter disorders, which may be the result of altered patterns of neural connectivity and responses that are largely genetically based rather than a product of gene–environment interaction.

Our key conclusion is that a personality disorder is like many other complex medical conditions. It has degrees of severity and can manifest with varying levels of behavioural dysfunction and symptomatic distress. Mild degrees of personality disorder are probably compatible with reasonable mental health and functioning; more severe disorder or comorbid psychiatric conditions will cause more dysfunction and result in referral to mental health services. If there is to be a national strategy for personality disorder services, clinical teams will need explanatory models to help them understand their patients' problems and plan treatment accordingly.

Declaration of interest

None.

References

Adolphs, R., Damasio, H., Tranel, D., *et al* (2000) A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. *Journal of Neuroscience*, **20**, 2683–2690.

American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders* (4th edn) (DSM–IV). Washington, DC: APA.

Andrews, G., Corry, J., Slade, T., *et al* (2003) Child sexual abuse. In *Comparative Quantification of Health Risks, Global and Regional Burden of Disease Attributable to Selected Major Risk Factors* (eds M. Ezzati, A. D. Lopez, A. Rodgers, *et al*), pp. 1851–1940. Geneva: World Health Organization.

Bancaud, J. & Talaraich, J. (1992) Clinical semiology of frontal lobe seizures. *Advances in Neurology*, **57**, 3–58.

Bartels, A. & Zeki, S. (2000) The neural basis of romantic love. *NeuroReport*, **11**, 3829–3834.

Bateman, A. W. & Tyrer, P. (2004) Psychological treatment for personality disorders. *Advances in Psychiatric Treatment*, **10**, 378–388.

Bechara, A., Tranel, D., Damasio, H., *et al* (1995) Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science*, **269**, 1115–1118.

Blair, R. J. R. (2001) Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery and Psychiatry*, **71**, 727–731.

Blair, R. J. R., Jones, L., Clark, F., *et al* (1997) The psychopathic individual: a lack of responsiveness to distress cues? *Psychophysiology*, **34**, 192–198.

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

Bradley, S. J. (2000) *Affect Regulation and the Development of Psychopathology*. New York: Guilford Press.
Breiter, H. C., Gollub, R. L., Weiskoff, R. M., *et al* (1997) Acute effects of cocaine on human brain activity and emotion. *Neuron*, **19**, 591–611.
Charney, D. S. & Drevets, W. C. (2002) The neurobiological basis of anxiety disorders. In *Psychopharmacology: The Fifth Generation of Progress* (eds K. Davis, D. S. Charney, J. Coyle, *et al*). Philadelphia, PA: Lippincott.
Damasio, A. R. (1994) *Descartes' Error. Emotion, Reason and the Human Brain*. New York: Penguin Putnam.
Damasio, A. R. (2003) *Looking for Spinoza. Joy, Sorrow and the Feeling Brain*. London: William Heinemann.
Devinsky, O. (2000) Right cerebral hemisphere dominance for a sense of corporeal and emotional self. *Epilepsy and Behaviour*, **1**, 60–73.
Department of Health (2003) *Personality Disorder: No Longer a Diagnosis of Exclusion*. London: Department of Health.
Dougherty, D., Shin, L. M., Alpert, N. M., *et al* (1999) Anger in healthy men. A PET study using script-driven imagery. *Biological Psychiatry*, **46**, 466–472.
Feldman, R., Greenbaum, C. W. & Yirmiya, N. (1999) Mother–infant affect synchrony as an antecedent of the emergence of self-control. *Developmental Psychology*, **35**, 223–231.
Fonagy, P., Target, M., Steele, M., *et al* (1997) Morality, disruptive behavior, borderline personality disorder, crime, and their relationship to security of attachment. In *Attachment and Psychopathology* (eds L. Atkinson & K. J. Zucker), pp. 233– 274. New York: Guilford Press.
Fonagy, P., Gergely, G., Jurist, E. L., *et al* (2002) *Affect Regulation, Mentalisation and the Development of the Self*. New York: Other Press.
Freeman, W. J. (1999) *How Brains Make up Their Minds*. London: Orion Books.
Geiger, T. & Crick, N. (2001) A developmental psychopathology perspective on vulnerability to personality disorder. In *Vulnerability to Psychopathology: Risk Across the Lifespan* (eds R. E. Ingram & J. M. Price), pp. 57–106. New York: Guilford Press.
George, C. & Solomon, J. (1996) Representational models of attachment: links between caregiving and attachment. *Infant Mental Health Journal*, **17**, 198–216.
Gloor, P. (1992) Role of the amygdala in temporal lobe epilepsy. In *The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction* (ed. J. P. Aggleto), pp. 505–538. New York: Wiley-Liss.
Grafman, J., Schwab, K., Warden, D., *et al* (1996) Frontal lobe injuries, violence, and aggression. A report of the Vietnam head injury study. *Neurology*, **46**, 1231–1238.
Hare, R. D., Hart, S. D. & Harpur, T. J. (1991) Psychopathy and the DSM–IV criteria for antisocial personality disorder. *Journal of Abnormal Psychology*, **100**, 391–398.
Henry, J. P. & Wang, S. (1998) Effects of early stress on adult affiliative behaviour. *Psychoneuroendocrinology*, **23**, 863–875.
Home Office & Department of Health (1999) *Managing Dangerous People with Severe Personality Disorder. Proposals for Policy Development*. London: Home Office & Department of Health.
Johnson, J. G., Cohen, P., Brown, J., *et al* (1999) Childhood maltreatment increases risk of personality disorder during early adulthood. *Archives of General Psychiatry*, **56**, 600–606.
LeDoux, J. (1996) *The Emotional Brain: The Mysterious Underpinnings of Emotional Life* . New York: Simon & Shuster.
Lees, J. & Manning, N. (1999) *Therapeutic Community Effectiveness: A Systematic International Review of Therapeutic Communities for People with Personality Disorder and Mentally Disordered Offenders* (CRD Report 17). York: NHS Centre for Research and Dissemination, University of York.
Levine, D., Marziali, E. & Hood, J. (1997) Emotion processing in borderline personality disorders. *Journal of Nervous and Mental Disease*, **185**, 240–246.
Moran, P., Jenkins, R., Tylee, A ., *et al* (2000) The prevalence of personality disorder among UK primary care attenders. *Acta Psychiatrica Scandinavia*, **102**, 55–57.
Müller, J. L., Sommer, M., Wagner, V., *et al* (2003) Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths: evidence from a functional magnetic resonance imaging study using pictures with emotional content. *Biological Psychiatry*, **54**, 152–162.
National Collaborating Centre for Mental Health (2004) *Self-harm: The Short-term Physical and Psychological Management and Secondary Prevention of Self-harm in Primary and Secondary Care* (Clinical Guideline 16). London: National Institute for Clinical Excellence.
Pagnoni, G., Zink, C. F., Montague, P. R., *et al* (2002) Activity in human ventral striatum locked to errors of reward prediction. *Nature Neuroscience*, **5**, 97–98.
Panksepp, J. (1998) *Affective Neuroscience*. Oxford: Oxford University Press.
Pardo, J. V., Pardo, P. J. & Raichle, M. E. (1993) Neural correlates of self-induced dysphoria. *American Journal of Psychiatry*, **150**, 713–719.
Patrick, M., Hobson, R. P., Castle, P., *et al* (1994) Personality disorder and the mental representation of early social experience. *Developmental Psychopathology*, **94**, 375–388.
Phillips, M. L., Drevets, W. C., Rauch, S. L., *et al* (2003) Neurobiology of emotion perception. I: The neural basis of normal emotion perception. *Biological Psychiatry*, **54**, 504–514.
Pietrini, P., Guazzelli, M., Basso, G., *et al* (2000) Neural correlates of imagined aggressive behaviour assessed by positron emission tomography in healthy subjects. *American Journal of Psychiatry*, **157**, 1772–1781.
Rolls, E.T. (2000) Précis of brain and emotion. *Behavioral and Brain Sciences*, **23**, 177–234.
Rose, S. (1998) *Lifelines: Biology beyond Determinism*. New York: Oxford University Press.
Schore, A. N. (2002) Dysregulation of the right brain: a fundamental mechanism of traumatic attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian and New Zealand Journal of Psychiatry*, **36**, 9–30.
Schore, A. N. (2003) The human unconscious: the development of the right brain and its role in early emotional life. In *Emotional Development in Psychoanalysis, Attachment Theory and Neuroscience. Creating Connections* (ed. V. Green), pp. 23–54. Hove: Brunner–Routledge.
Shin, L. M., Dougherty, D. D., Orr, S. P., *et al* (2000) Activation of anterior paralimbic structures during guilt-related script-driven imagery. *Biological Psychiatry*, **48**, 43–50.
Singleton, N., Bumpstead, R., O'Brien, M., *et al* (2001) *Psychiatric Morbidity among Adults Living in Private Households 2000*. London: TSO (The Stationery Office).
Sprengelmeyer, R., Young, A. W., Calder, A. J., *et al* (1996) Loss of disgust: perception of faces and emotions in Huntington's disease. *Brain*, **119**, 1647–1665.
Suomi, S. (1999) Attachment in rhesus monkeys. In *Handbook of Attachment* (eds J. Cassidy & P. Shaver), pp. 181–197. New York: Guilford Press.
Suomi, S. J. (2003) Gene-environment interactions and the neurobiology of social conflict. *Annals of the New York Academy of Science*, **1008**, 132–139.
Thompson, R. A. (1990) Emotion and self-regulation. In *Nebraska Symposium on Motivation, 1990. Vol. 38: Perspectives on Motivation* (ed. R. A. Dienstbier), p. 371. Lincoln: University of Nebraska Press.
Tyrer, P. & Bateman, A. W. (2004) Drug treatment for personality disorder. *Advances in Psychiatric Treatment*, **10**, 389–398.
van der Kolk, B. A. & Fisler, R. (1994) Child abuse and neglect and loss of self-regulation. *Bulletin of the Menninger Clinic*, **58**, 145–168.
Wakefield, J. C. (1992) Disorder as harmful dysfunction: a conceptual critique of DSM–II–R's definition of mental disorder. *Psychological Review*, **99**, 232–247.
Wang, S. (1997) Traumatic stress and attachment. *Acta Physiologica Scandinavica*, **161** (suppl. 640), 164–169.
Watts, D. & Morgan, G. (1994) Malignant alienation. Dangers for patients who are hard to like. *British Journal of Psychiatry*, **164**, 11–15.
World Health Organization (1992) *The ICD–10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. Geneva: WHO.

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press

MCQs

1 Personality disorders:
a are common psychiatric disorders
b are associated with behavioural problems
c do not co-exist with other disorders
d invite punitive or rejecting responses from others
e are associated with subjective distress.

2 Affect regulation:
a is the same as emotional regulation
b involves both mental and physical experience
c is an essential part of social relationships
d is a function of several complex neurological systems
e does not influence behaviour.

3 Affects are produced in the following brain areas:
a amygdala
b insula
c orbitofrontal cortex
d occipital cortex
e parietal lobes.

4 Secure attachment in mammals:
a regulates affects through modulation of the noradrenergic system
b affects the development of right orbitofrontal cortex
c promotes exploratory behaviour and learning
d is associated with successful social relationships with peers
e is associated with successful parenting in adulthood.

5 People with personality disorders:
a are affectless
b do not feel distress or anxiety
c have increased capacity for down-regulation of affect
d have decreased capacity for recognition of affective signals in others
e are unresponsive to all therapies.

MCQ answers

1	2	3	4	5
a T	a T	a T	a T	a F
b T	b T	b T	b T	b F
c F	c T	c T	c T	c T
d T	d T	d F	d T	d T
e T	e F	e F	e T	e F

https://doi.org/10.1192/apt.12.4.297 Published online by Cambridge University Press