Dissociative Disorders

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Hoyle Leigh

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H. Leigh, MD, DLFAPA, FACP, FAPM (✉)
Professor of Psychiatry, Department of Psychiatry, University of California, San Francisco, CA, USA

Director, Psychosomatic Medicine Program & Psychiatric Consultation-Liaison Service, UCSF-Fresno, 155N. Fresno St., Fresno, CA 93701, USA
e-mail: hoyle.leigh@ucsf.edu

18.1 Vignettes

1. A patient complained that she was feeling numb, and felt as it her surroundings were unreal, and that she was in a dream. These feelings occurred since 2 days ago. On careful history, the physician found that the patient had discontinued paroxetine 40 mg per day 3 days prior as she ran out of the medication. Depersonalization/derealization associated with SSRI withdrawal was diagnosed, and the drug was resumed. The symptoms disappeared within a day. Then paroxetine was gradually tapered over 2 weeks to successfully avoid any discontinuation syndrome
2. Agatha Christie, the British mystery writer who invented Hercule Poirot and Miss Marple, disappeared on 3 December 1926 only to reappear 11 days later in a hotel in Harrogate, apparently with no memory of the events which happened during that time span (http://www.straightdope.com/columns/read/361/why-did-mystery-writer-agatha-christie-mysteriously-disappear)
3. Jeff Ingram, appeared in Denver in 2006 with no memory of his name or where he was from. After appearing on national television to appeal for help identifying himself, his fiancée Penny called Denver police identifying him. The episode was diagnosed as dissociative fugue. Jeff has experienced three incidents of amnesia: in 1994, 2006, and 2007.

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(http:/www.npr.org/2012/12/14/167187734/for-man-with-amnesia-love-repeats-itself) (from fugue state cases, Wikipedia)

18.2 Introduction

Dissociation is a phenomenon in which there is a lack of connection in a person's thoughts, memories, feelings, actions, or sense of identity. During the period of dissociation, certain information is split off from other information with which it is normally connected. Dissociative experience is probably a continuum, from complete absorption in a task with total unawareness of surroundings, to fugue states, to total amnesia.

Dissociation can be interpreted as an "emergency defense," or a "shut off mechanism."

It may be an evolutionarily adaptive mechanism designed to prevent overwhelming flooding of consciousness at the time of trauma. Once the individual has learned to dissociate in the context of trauma, he or she may subsequently transfer this response to other situations and it may be repeated thereafter arbitrarily in a wide variety of circumstances. The dissociation therefore may destabilize adaptation and becomes pathological (Allen and Smith 1993).

Patients who receive treatment interventions that address their trauma-based dissociative symptoms show improved functioning and reduced symptoms (Gentile et al. 2013).

Dissociation is closely related to conversion syndrome (hysteria, hysterical dissociation), and some consider the latter to be a subset of dissociation syndrome. Hypnosis is a widely used technique to induce dissociation. There is evidence that identical functional brain changes occur in conversion paralysis and hypnotically induced paralysis of the lower limb (Halligan et al. 2000). Dissociation is an important symptom in posttraumatic stress disorder (PTSD), as well as in the borderline personality (see Chap. 25). Conversion, PTSD, and borderline personality disorder, however, are not classified under the rubric of dissociative disorders in DSM-5. Syndromes included in the DSM-5 as dissociative disorders are dissociative identity disorder (multiple personality), dissociative amnesia, dissociative amnesia with dissociative fugue, depersonalization/depersonalization disorder, and other specified or unspecified dissociative disorder.

18.3 Depersonalization and Derealization

Depersonalization refers to a psychological state in which the perception or experience of the self feels detached or unreal. One feels as if one is an outside observer of one's mental processes or body, as if in a dream. Depersonalization is accompanied by feelings of disembodiment and subjective emotional numbing. It has been proposed that depersonalization is caused by a fronto-limbic (particularly anterior insula) suppressive mechanism—presumably mediated via attention—which manifests subjectively as emotional numbing, and disables the process by which perception and cognition normally become emotionally colored, giving rise to a subjective feeling of 'unreality'. Depersonalization syndrome patients show increased prefrontal activation as well as reduced activation in insula/limbic-related areas to aversive, arousing emotional stimuli. Parietal mechanisms may underlie feelings of disembodiment (Reutens et al. 2010; Sierra and David 2011)

Derealization is an alteration in the perception or experience of the external world so that it seems strange or unreal. In depersonalization, there is increased alertness that may be associated with an activation of prefrontal attentional systems (right dorsolateral prefrontal cortex) and reciprocal inhibition of the anterior cingulate, leading to the experiences of "mind emptiness" and indifference to pain that are often seen in depersonalization. In derealization, there may be a left-sided prefrontal inhibition of the amygdala resulting in dampened autonomic output, hypoemotionality, and lack of emotional coloring, resulting in feelings of unreality or detachment. Derealization and depersonalization may be

conceptualized as a syndrome of corticolimbic disconnection (Sierra and Berrios 1998). Depersonalization and derealization may serve an evolutionarily adaptive function of intensifying alertness and dampening potentially disorganizing emotion (Stein and Simeon 2009).

Depersonalization and derealization experiences often occur in normal people in situations of severe anxiety, as in medical settings where a serious diagnosis or medical procedures may be discussed. Furthermore, many drugs, particularly analgesics and sedatives, as well as mild delirium that may be associated with a medical condition, may predispose patients to depersonalization/derealization. Specific neurological conditions such as partial complex seizures as well as encephalopathies and strokes may be associated with these phenomena. In the CL setting, psychological support and reassurance may alleviate the frightening aspect of these experiences. Reduction or change in a medication that might be associated with the condition, as well as treatment of delirium and the underlying medical condition may be therapeutic.

When there is functional impairment due to depersonalization or derealization, depersonalization or derealization *disorder* may be diagnosed. According to DSM-5, the lifetime prevalence of depersonalization/derealization disorder is 3 %, equally in males and females.

Depersonalization and derealization are common features of other psychiatric conditions, particularly borderline personality and posttraumatic stress disorder (PTSD).

18.3.1 Treatment of Depersonalization/ Derealization Disorder

Cognitive-behavioral therapy, mindfulness training, and repeated exposure are the psychotherapeutic techniques that have been reported useful in depersonalization/derealization disorder (Hunter et al. 2005; Michal et al. 2007; Stein and Simeon 2009; Weiner and McKay 2013).

SSRIs, clonazepam, naltrexone, methylphenidate, and lamotrigine, in monotherapy or in combination, have been used effectively in treatment of depersonalization/derealization syndrome (Aliyev and Aliyev 2011; Foguet et al. 2011; Rosagro-Escamez et al. 2011; Sierra 2008).

18.4 Dissociative (Psychogenic) Amnesia and Fugue

18.4.1 Vignette

A 25-year-old man was hospitalized with no memory of who he was, where he was from and with no identification. When tested however he could do serial 7's and remember new things he was told. Under hypnosis he revealed that he lived in another state. He came home late one night intoxicated, tried to make popcorn and accidentally set the house on fire. His parents died in the fire. After the funeral, he disappeared, apparently traveling to a distant state. After the hypnosis session, his memory gradually returned and he was helped to grieve.

18.4.2 Definition and Subtypes

Dissociative amnesia is characterized by a pervasive loss of memory of significant personal information, such as name, occupation, and residence. Aspects of dissociative amnesia may be present in dissociative identity disorder (multiple personality), factitious syndromes, psychosis, and the borderline syndrome. Dissociative amnesia is diagnosed when the amnesia cannot be directly attributed to a neurological cause such as trauma or to another major psychiatric condition, and is extensive enough to impair function. In head trauma, there may be localized amnesia that may be retrograde or anterograde. According to DSM-5, the 12 month prevalence for dissociative amnesia is 1.8 % (1 % for males, 2.6 % for females). Dissociative amnesia has been linked to overwhelming stress, such as abuse, accidents, disasters, or war that the patient has experienced or witnessed. It is more common in women, and tends to be more prevalent in stressful periods

such as wars and natural disasters. Dissociative amnesia following general anesthesia has been reported (Chang et al. 2002).

Dissociative amnesia has a variable course, with some resolving rapidly while others may persist for decades or longer. Dissociative amnesia often recurs. Dissociative capacity may decline with age.

The subtypes of dissociative amnesia include the following:

Selective amnesia: The patient can recall only small parts of events that happened during a defined period of time. For example, a victim of abuse may have only fragmentary memory of her abuse.

Generalized amnesia: The amnesia encompasses the person's entire life.

Continuous amnesia: The patient has no memory for events beginning from a certain point in the past continuing up to the present.

Systematized amnesia: A loss of memory for a specific category of information.

For example, a person may have no memories about one particular family member.

With Dissociative Fugue

In a dissociative fugue, the person leaves home suddenly and unexpectedly and goes off on a journey, often to distant places (see Vignettes 2–3). The journey may last hours, days, months, or even years. A person in a fugue state is unaware of or confused about his/her identity, and in some cases will assume a new identity.

18.4.3 Differential Diagnosis

18.4.3.1 Vignette

A young man was hospitalized unable to identify himself or answer questions other than by responding "OK." He was given food that included a hard-boiled egg. He was observed to attempt to bite ito it shell and all. The consultant was suspicious and ordered an EEG. It showed A continuous epileptiform pattern. His mental status normalized with anticonvulsant medication. He then was able to reveal that he had been on vacation and ran out of his epilepsy medication.

18.4.3.2 General Considerations

In the CL setting, patients who manifest a global amnesia are likely to be referred for a psychiatric consultation. Major differential diagnostic considerations in such cases include memory disturbance associated with neurocognitive disorders including delirium and dementia, ictal and postictal states, head trauma, as well as transient global amnesia discussed below. Amnestic syndromes associated with alcohol abuse (e.g., Korsakoff's psychosis) should also be considered. Comorbidity with other psychiatric disorders is common with dissociative amnesia, and depression and other major psychiatric syndromes may emerge as the amnesia clears.

18.4.3.3 Transient global amnesia (TGA)

Transient global amnesia (TGA) is a neurologic condition that usually occurs in persons over the age of 50, and is characterized by abrupt anterograde memory loss with repeated questioning ("Where am I?" "What's my name?"). The duration is usually 1–8 h with full recovery, though durations of 15 min and of 24 h have been reported.

Emotional and physical stress may precipitate these attacks, and in younger patients, migraine headaches appear to be a risk factor. In females, anxiety, depression, and emotional instability may be risk factors (Quinette et al. 2006a, b).

MRI data suggest that a transient perturbation of hippocampal function may underlie transient global amnesia. Various factors such as migraine, focal ischemia, venous flow abnormalities, and epileptic phenomena may contribute to the risk. The vulnerability of hippocampal neurons to metabolic stress may play a pivotal part in the pathogenesis of TGA (Bartsch and Deuschl 2010).

18.4.3.4 Treatment of Dissociative Amnesia

Various psychotherapeutic modalities may be used to treat dissociative amnesia including cognitive behavioral therapy (CBT), exploratory psychotherapy, creative therapies (art therapy, music therapy), and hypnotherapy. Intravenous sedative interview has been reported to be effective

(Lee et al. 2011). There are no specific medications for dissociative amnesia, but antidepressants and anxiolytics may be used for symptomatic indications.

18.5 Dissociative Identity Disorder (Multiple Personality)

In this condition, two or more identities or personalities alternatively take over the person's behavior. One or more of the personalities may be aware of the other identities, while others may be totally unaware of the existence of other personalities. Patients with this condition often have amnestic periods during which another identity had taken over.

Many patients, in addition, have symptoms of anxiety, depression, derealization, and depersonalization. Substance abuse is common, as well as suicide attempts.

This condition is relatively common in acute psychiatric settings (3–4 %), or very rare depending on the observer's orientation, and may cause serious functional impairment. The risk of suicide is high in patients suffering from dissociative identity disorder.

More than 90 % of patients with dissociative identity disorder report experiencing childhood physical or sexual abuse. Dissociative identity disorder has been conceptualized as a neurodevelopmental disorder caused by traumatic childhood that prevented an integration of the child's experiences and interactions (Forrest 2001).

Reinders et al. (2003) demonstrated specific changes in localized brain activity on PET scan, consistent with their ability to generate at least two distinct mental states of self-awareness, each with its own access to autobiographical trauma-related memory. The findings revealed the existence of different regional cerebral blood flow patterns for different senses of self.

Thus, specific brain functional differences in the medial prefrontal cortex and posterior association areas may be associated with different personalities in dissociative identity disorder (Reinders et al. 2003). Smaller hippocampal and amygdala volume has also been associated with dissociative identity disorder, as well as in PTSD and borderline syndrome with a history of childhood abuse and depression (Vermetten et al. 2006).

18.5.1 Diagnosis and Treatment

In the CL setting, psychiatric consultation may be requested on patients who have a known diagnosis of dissociative identity disorder (multiple personality), or in the course of an evaluation concerning amnesia, depersonalization, anxiety, depression, or unexplained physical symptoms.

Interviewing patients after placing them under hypnosis or after administering an intravenous sedative (see Chap. 34) may facilitate the diagnosis. The diagnosis is established when an alternate personality is demonstrated, either spontaneously or in an altered state. Great care must be taken not to subtly reinforce the development of altered states by expressing great interest in them.

The treatment of choice is psychotherapy (individual, couples, group) on an outpatient basis. Antidepressants (SSRIs, SNRIs, tricyclics, etc.), antipsychotic mood stabilizers (aripiprazole, quetiapine, etc.), antianxiety agents, beta-blockers, anticonvulsant mood stabilizers (carbamazepine, valproate, etc.), and naltrexone (for self-injurious behaviour), and other drugs are sometimes helpful in conjunction with psychotherapy (Burton and Lane 2001; Fine 1999; Kluft 1996).

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