Depersonalization Disorder

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Depersonalization is an intriguing psychiatric symptom that was first described and named in the European literature over a century ago. Its essence is a subjective experience of unreality and detachment from various aspects of the self, manifested as a sense of disconnection from one's own body, mentation, feelings, or action. Individuals who have experienced depersonalization may report these sensations:
- feeling as if they are watching themselves from a distance; going through the motions of life as if they are watching a movie of themselves;
- having one part that acts while another part observes; feeling disconnected from one's physical being;
- emotional numbness and an inability to experience feelings;
- being in a dream, fog, or trance;
- looking in the mirror and not recognizing one's self; or
- feeling that one's voice, movements, or behavior are not under full control.

Depersonalization is not infrequently accompanied by derealization, a sense of detachment, unreality, and altered relationship to the outside world.

As a symptom, depersonalization is fascinating for the rather vast variety of physical and emotional states, psychiatric symptoms, organic factors, and life experiences with which it can be associated. There is evidence to suggest that depersonalization occurs on a continuum in the general population, ranging from the "normal" (mild and transient experiences without clinical significance) to the "abnormal" (recurrent or persistent episodes associated with morbidity).

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Transient depersonalization is known to occur in "normal" individuals under conditions such as sleep deprivation, sensory deprivation, travel to unknown places, or acute intoxication with hallucinogens, marijuana, or alcohol. In college population samples, from one third to one half of all students report having had depersonalization experiences. A similar high incidence of brief depersonalization has been reported in individuals without psychiatric histories after exposure to life-threatening danger such as serious accidents.

Various organic conditions can be associated with depersonalization, the more common ones being temporal lobe epilepsy, tumors, and migraines. Indeed, the variety of precipitants linked to depersonalization led Mayer-Gross to speculate in 1935 on "the limited value of psychological theories in explaining the syndrome," suggesting instead that "we have to regard it as an unspecific preformed functional response of the brain."

Depersonalization has also been associated with a variety of functional psychiatric disorders, mainly anxiety and panic, depressive disorders, schizophrenia, personality disorders, and dissociative disorders. Depersonalization and derealization comprise one of the DSM-III-R diagnostic criteria for panic attacks, and about one third of patients with panic disorder experience these symptoms. Sir Martin Roth described in 1959 the "phobic anxiety-depersonalization syndrome," having found in his experience that most patients with depersonalization exhibited phobic, anxious, and panic symptoms precipitated by severe emotional stress.

Other investigators have emphasized the occurrence of depersonalization in association with depression rather than anxiety. Although the older literature stressed the link between depersonalization and psychosis, the degree and nature of the association are somewhat unclear, and the loss of reality testing in schizophrenia may render this a qualitatively different experience. Severe personality disorders, such as borderline, can manifest depersonalization experiences, and a frantic effort to relieve these sometimes leads to repetitive self-mutilation.

Finally, extensive depersonalization and derealization occur in over half of patients with severe dissociative disorders such as multiple personality. In one study of general psychiatric inpatients with various diagnoses, 12% reported severe and lasting depeersonalization experiences.

The widespread occurrence of depersonalization has led some investigators to view it as a nonspecific symptom of various psychiatric disorders. However, clinical experience, backed by a number of case reports and series, attests to the fact that depersonalization can occur as a primary symptom in its own right. In such cases, comorbid symptoms, if present, appear secondary in intensity or severity of distress, and may not fulfill criteria for major Axis I disorders. Alternatively, if another Axis I disorder is present, there may be a clear history of depersonalization predating that disorder, or occurring persistently and extensively beyond the manifestations of the particular Axis I disorder. In such patients, depersonalization appears to comprise a discrete syndrome with its own characteristic onset, phenomenology, course, and treatment response, and the diagnosis of depersonalization disorder can be made.

**CLINICAL CHARACTERISTICS**

Depersonalization disorder is currently classified as a dissociative disorder because its key feature involves an altered sense of self. According to the DSM-III-R, the diagnosis of depersonalization disorder is made when an individual suf-
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...fears from “persistent or recurrent experiences of depersonalization” during which “reality testing remains intact” and which are “sufficiently severe and persistent to cause marked distress.” The diagnosis is made when depersonalization constitutes the primary disturbance, and is not purely secondary to other disorders such as “schizophrenia, panic disorder, agoraphobia without panic disorder but with limited symptom attacks, or temporal lobe epilepsy.”

The onset of depersonalization most often occurs in the teens or 20s; onset in middle or late life is infrequent. Many patients recall their first episode vividly, whose beginning is usually abrupt or almost instantaneous, and frequently no precipitating event can be elicited. At other times, patients may be conscious of some precipitating event, such as the ingestion of marijuana or LSD, a severely anxiety-provoking emotional experience, or a life-threatening experience. The duration of episodes can be highly variable from individual to individual, and can last for hours, weeks, or months, usually resolving gradually in contrast to their sudden onset. The course of the disorder is usually chronic. Some patients experience recurrent episodes, while others have a more unremitting course with continuous depersonalization that can last for years.

Although not backed by systematic investigation, primary depersonalization disorder is thought to be rare. Some studies have reported a higher incidence in females, while others have found no gender difference, and thus the sex ratio remains unknown.

Depersonalization disorder is, by definition, accompanied by marked distress. Patients report various distressing feelings such as prominent secondary anxiety related to the sense of alienation from the self and from others; fears of not being in control of themselves, of going crazy, or of losing their minds; impaired interpersonal relationships hampered by the sense of disconnectedness; and decreased productivity and creativity because of the sense of detachment. Symptoms reported in association with depersonalization include other dissociative symptoms such as derealization or identity confusion; anxiety; depression; hopelessness; heaviness, dizziness, imbalance, and other somatic symptoms; obsessive preoccupations with the presence of depersonalization and compulsive rituals to check if the detachment is still present; impaired attention, concentration, and recall; and altered subjective sense of time.

THEORIES OF DEPERSONALIZATION
Psychodynamic Theories

A wide range of psychodynamic formulations have been proposed to explain depersonalization, which have been reviewed and critiqued by a number of authors. One psychodynamic approach to understanding depersonalization centers on its defensive functions. Depersonalization can be viewed as an affective response used by the ego to defend against painful and conflictual impulses or affects. This dynamic may be applicable to depersonalization phenomena in patients with neurotic character structure.

An ego or self psychology perspective may be useful in understanding depersonalization phenomena in many patients. Depersonalization has been linked extensively to a poorly integrated ego or sense of self, resulting from the presence and activation of conflictual and inadequately integrated partial identifications or self-representations. This might explain the higher frequency of depersonalization experiences in adolescent populations, where the developmental task of identity formation has not been fully completed. Early experiences of lack of validation of the self may lead to narcissistic pathology accompanied by a sense of falseness and unreality, as parentally unrecognized or
distorted aspects of the true self are scrutinized and dissociated. The relationship between the ego, the self, and depersonalization experiences has been lucidly discussed by Francis et al, correlating vulnerability to depersonalization in psychotic, borderline, or narcissistic patients with deficits in self-object differentiation, self and object constancy, or incompletely internalized self-constancy, respectively.9

**Biological Theories**

As with most “functional” psychiatric disorders, a biological basis has been postulated for depersonalization. Nonspecific evidence arises from its wide occurrence in a number of central nervous system anatomic and physiologic disturbances, such as temporal lobe epilepsy, tumor, encephalitis, head injury, migraines, Meniere’s disease, and intoxication with marijuana and hallucinogens. More specifically, preliminary evidence has implicated serotonergic dysfunction in depersonalization.

Attention has been drawn by several authors12-14 to the phenomenological similarities between obsessive-compulsive symptomatology and depersonalization. Patients with depersonalization are often reported to have obsessive personality traits; the split between the observing and participating self that is often characteristic of depersonalization is in some ways reminiscent of the isolation and intellectualization of obsessive personalities. Patients may be obsessively preoccupied by their depersonalization symptoms and may repetitively scrutinize themselves in a compulsive fashion to check out feelings of unreality. For example, one patient with depersonalization reported that the thought that he must be depersonalized often preceded the experience, and he would compulsively pace a distance to check out whether his legs felt connected.

The association of depersonalization with migraines and marijuana intoxication may implicate serotonergic mechanisms in its genesis. The recently reported favorable pharmacologic response to serotonin reuptake inhibitors15 has also implicated serotonergic dysfunction, especially in light of the refractoriness of depersonalization to a great variety of pharmacological treatments. A case report on the occurrence of depersonalization with acute fluoxetine administration is suggestive of serotonergic hypersensitivity, similar to OCD where transient exacerbation can occur with the initial administration of serotonin agonists before serotonin receptors gradually become downregulated.16

Depersonalization has also been reported to occur with abrupt clomipramine discontinuation.17 A case report of a patient with primary depersonalization disorder without other Axis I comorbidity found left hemispheric frontotemporal activation and decreased left caudate perfusion, findings reminiscent of obsessive-compulsive disorder.18 In a sample of depressed patients, a relative decrease in plasma L-tryptophan, a serotonin precursor, correlated with depersonalization, psychic anxiety, and obsessions, but not with other depressive symptoms.19

**Trauma and Stress Theories**

Although presented here separately, trauma-generated psychopathology may involve both psychological and biological mechanisms and may provide a framework for integrating the two. Systematic trauma histories in patients with DSM-III-R depersonalization disorder have not been reported to date, but extensive literature exists linking dissociative symptoms in general to childhood trauma. Both depersonalization and derealization experiences have been found to predict childhood sexual abuse, derealization being the single strongest predictor of all the variables examined in one study.20 In nonclinical samples, dissociative scores were correlated with retrospective reports of childhood stress, threats of violence, psychological abuse, and physical abuse.21

Severe stress in later life has also been associated with depersonalization. Sir Martin Roth reported that most patients with “phobic anxiety-depersonalization syndrome” had experienced overwhelming stress such as a threat to life or bereavement,5 and over one third of vic-
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Tims of near-deadly accidents experience transient depersonalization. Thus, it has been suggested that depersonalization may be an adaptive response to overwhelming stress, permitting continuous function by protecting against potentially disorganizing anxiety, as if the danger is not happening to the self but to a stranger.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The DSM-III-R criteria, phenomenology, and clinical characteristics of depersonalization disorder have already been described in the previous sections. In taking a psychiatric history, it is important to use patients' own words in describing the symptoms and to probe further with open-ended or more specific questions, as the experience is often elusive and difficult for patients to verbalize. The clinician should ask about age of onset, course, precipitating factors, and treatment history of the depersonalization. Other dissociative symptoms, such as derealization experiences, memory lapses, and altered identities should be inquired about in order to establish an accurate differential diagnosis of the different dissociative disorders.

A history of other psychiatric symptoms and disorders must be elicited, in particular anxiety, panic, depression, obsessions and compulsions, and phobias, and their relationship to the depersonalization. One should determine whether depersonalization has occurred historically only in the context of other illnesses, such as major depression or panic attacks, or whether the depersonalization predated, persisted after the resolution, or occurred at least in part independently of these.

Organic etiologies must be ruled out. Histories of head injury, seizures or other CNS pathology, or the presence of neurol ogic or behavioral symptoms suggestive of such pathology would be indications for brain imaging and/or EEG with nasopharyngeal leads. The clinician should obtain a medication history, since a number of psychotropic and nonpsychotropic medications may induce depersonalization, and obtain a history of drug use, especially marijuana and hallucinogens.

Once the presence of depersonalization independent of other Axis I disorders is documented, organic factors have been ruled out, and the experiences are recurrent or persistent enough to lead to significant distress or dysfunction, the diagnosis of primary depersonalization disorder can be made.

TREATMENT

Although a variety of treatments for depersonalization have been reported in the literature, efficacy has not been demonstrated convincingly for any treatment, and depersonalization is generally regarded as rather poorly responsive and refractory to treatment. There are no controlled treatment studies of depersonalization, either pharmacologic or psychotherapeutic.

Psychodynamic Psychotherapy

In the older literature, a rather pessimistic view prevailed of the psychoanalytic treatment of patients with depersonalization. It was cautioned that the analysis could be very lengthy, and that the use of the couch and the lack of visual contact could exacerbate the sense of unreality and detachment in some patients. Reports of successful psychodynamic treatment for depersonalization emphasize certain approaches and goals: acceptance of the symptom while this is explored and better understood, and psychoeducation; identifying its defensive functions; treatment of underlying narcissistic pathology by recognizing those parts of the self that are scrutinized, distorted, or repudiated; and recovering, validating, and integrating traumatic memories.
Behavioral Therapy

Although only a few case reports have been published on the behavioral treatment of depersonalization, these have noted some success in individual patients. Such behavioral techniques include in vivo exposure (to situations that exacerbate depersonalization); imaginal flooding (taped descriptions of severe episodes); paradoxical suggestion (to induce an episode and then gain a sense of control); negative reinforcement (performing a chore after each episode); and reward contingency (rewarding oneself for decreasing episodes). As no systematic studies exist comparing the different techniques, their relative efficacy is unknown, and they should probably be customized to the particular symptoms. It also remains to be seen whether behavioral therapy can be effective in treating continuous rather than episodic depersonalization.

Pharmacotherapy

Although numerous pharmacological agents have been tried in the treatment of depersonalization, the overall outcome has been rather grim. In the older literature, the use of intravenous amphetamines and of barbiturates was highlighted, either separately or combined, and frequently reported efficacious in relieving, at least transiently, episodes of depersonalization. Similarly, neuroleptics had previously been advocated, but outcome is unpredictable; some patients were reported to benefit while other patients experienced stronger feelings of unreality.

The frequent association of depersonalization with anxiety and depression has led to the widespread use of antidepressants and anxiolytics in an attempt to treat depersonalization. Certainly, if the primary diagnosis is one of major depression or an anxiety disorder, and depersonalization appears to be a manifestation of these, standard pharmacologic treatment for these disorders is indicated. However, even when disorders such as major depression and panic are comorbid with depersonalization, the latter may not respond during treatment for the former, supporting its independent status in at least a proportion of patients. Although there are reports on the success of antidepressants such as desipramine or benzodiazepines such as clonazepam in treating depersonalization, their success has not been documented in any patient series, and reports of their failure abound. ECT has been at times employed to treat refractory depersonalization, and its outcome has been unsuccessful in more cases than not, sometimes even in the presence of major depression.

Of more promise, in the last several years, has been the suggestion that depersonalization treatment may respond to serotonin reuptake inhibitors. In a series of eight patients with depersonalization, Hollander et al reported that six had a good response to fluoxetine or fluvoxamine. Although all these patients also had panic or obsessive-compulsive symptoms, six were considered to have independent depersonalization disorder, and most patients had previously been refractory to many somatic treatments. Since then, another case report of a patient with primary depersonalization disorder who responded to fluoxetine has been published.

It appears then that serotonin reuptake blockers may hold some promise for the treatment of this rather refractory condition, and controlled double-blind pharmacologic trials in patients with systematically diagnosed depersonalization disorder should be undertaken. The possibility that depersonalization disorder has a selective efficacy in response to serotonin reuptake inhibitors is of therapeutic interest in discussing the relationship between depersonalization and other obsessive-compulsive spectrum disorders.

REFERENCES