Dissociative disorders unclear? Think ‘rainbows from pain blows’

Visual metaphor answers the question, “What’s ‘dissociated’ in dissociative disorders?”

Mr. D, age 45, presents to his primary care physician with panic attacks, nausea, shortness of breath, nightmares, and dizziness 6 months after being assaulted and robbed at an ATM. Following a routine medical workup, the physician diagnoses posttraumatic stress disorder (PTSD) and refers Mr. D for exposure and response prevention therapy.

During graded exposure sessions, Mr. D’s eyes sometimes glaze over and he seems to “float away” from the discussion. When the therapist asks about these symptoms, Mr. D reports having had them as long as he can remember. In school, he says, teachers thought he was a slow learner, a daydreamer, or had attention-deficit/hyperactivity disorder. From what he can recall of his childhood, he describes a history of trauma and neglect with a violent, drug-abusing father and absent mother.

Patients with a history of early abuse or neglect are at risk for dissociative phenomena and other trauma-related psychiatric disorders. The heterogeneous dissociative disorders are often hidden and unrecognized—as in Mr. D’s case—or present with unfamiliar or atypical symptoms. Understanding and identifying dissociative symptoms is important because:

- Dissociative symptoms worsen prognosis, whether patients have conversion disorders or psychogenic seizures or are in psychotherapy.
- Dissociative states may impair memory encoding and decrease patients’ ability to remember therapeutic information.

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Dissociative disorders

Clinical Point
Like colors in a beam of white light, dissociable components of experience constitute normal consciousness.

5 components of consciousness. Just as separable wavelengths compose a beam of white light, dissociable "colors" or components of subjective experience constitute a normal state of consciousness. Five implicit components of normal consciousness—present in various degrees, at different times—are seamlessly integrated and associated in real time.

One paired component is a detached "observer" and a more embodied, feeling "experience." The observer is a perspective that begets metacognition (thinking about one's inner world) and self-observation; it resides in the same body as soma-based "feelings" that unconsciously contribute to the sense of "being present" with oneself and the world in the moment.

A second component is voluntary access to one's autobiographical memories (memories about the self in time), which are constantly "updated" and integrated with current experiences. This component allows one to distinguish between remembered (past) experiences and "firsthand" (present) experience.

Three other components of normal consciousness are:

- a sense of agency and voluntary control over one's mental contents, mental activity, and bodily movements
- an ongoing connection with one's body and mind and an understanding of where sensations and images come from
- a sense of sequential experience, with relatively smooth transitions (from self at work to self at home, self a week ago to self today, etc) that have a singular referent (an identity).

Pathologic dissociation occurs when a prism of distress disperses one of these component "wavelengths" from the main "beam" of consciousness. For example:

### Table 1

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dissociative amnesia</td>
<td>A reversible loss of memory, typically preceded by a stressor</td>
</tr>
<tr>
<td>Dissociative fugue</td>
<td>Loss of memory and identity, along with travel away from home</td>
</tr>
<tr>
<td>Dissociative identity disorder (formerly multiple personality disorder)</td>
<td>Presence of different identity states, often with lack of connection between them; current models highlight the presence of recurrent dissociative intrusions into many aspects of executive function and self</td>
</tr>
<tr>
<td>Depersonalization disorder</td>
<td>Detachment from oneself as a present, feeling person (depersonalization) and the world (derealization)</td>
</tr>
<tr>
<td>Dissociative identity disorder NOS</td>
<td>Functionally disturbing dissociative symptoms that do not fit into any of the above</td>
</tr>
</tbody>
</table>

NOS: not otherwise specified

Dissociation’s neurobiology: Evidence of brain ‘disconnections’

From a neurophysiologic perspective, mental states may be viewed as arising from synchronized integration of the activity of functionally specialized brain regions. Functional neuroimaging of dissociation supports an understanding of these symptoms as ‘disconnection syndromes.’

Functional neuroimaging. Different ‘identities’—sometimes called traumatic personality state and neutral personality state—demonstrate different patterns of cerebral blood flow, subjective reports, and peripheral physiologic parameters (blood pressure, heart rate). Functional imaging of traumatic dissociation shows active suppression of limbic regions (amygdala) and increased activity in dorsolateral prefrontal areas. Similarly, neuroimaging of depersonalization disorder show increased neural activity in prefrontal regions associated with affect regulation and decreased activity in emotion-related areas.

Speed. Dissociative responses occur extremely rapidly. Using EEG, which allows finer temporal resolution than functional imaging studies, Kirino et al. showed reversible attenuation of a specific EEG signal within 300 msec during dissociative episodes. This ultra-rapid neural reflex was correlated with allocation of attentional and working memory resources, perhaps with the goal of minimizing memory activation and resurgence of affect-laden memories.

Hormonal. Stress-related disorders cause perturbations in neurohormonal function. Simeon et al. found a distinct pattern of stress-induced HPA axis dysregulation in dissociative patients compared with PTSD patients and healthy controls. Similar results were seen in patients with borderline personality disorder and dissociative symptoms.

Structural imaging. Stress-related neurohormonal perturbations are known to affect critical neural structures, including the hippocampus. Using MRI, Vermetten et al. found significantly decreased amygdala and hippocampal volumes in patients with dissociative identity disorder.

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Box

Clinical Point

Personality traits that may predispose to dissociation include mental absorption, suggestibility, and a tendency to fantasize.

Separation of the “observer” and “experiencer” occurs in depersonalization disorder.

Reversible loss of ability to access memories characterizes dissociative amnesia.

Disconnection between sequential experiences is a part of dissociative identity disorder.

This modular perspective of dissociative disorders parallels a neurophysiologic perspective of mental states as arising from the synchronized integration of the activity of separate, functionally specialized brain regions. Functional neuroimaging of dissociation supports an understanding of these symptoms as “disconnection syndromes” (Box).

Causes of dissociative disorders

As with many psychiatric disorders, the etiology of dissociative phenomena is thought to include the individual patient’s temperamental or constitutional predispositions as well as a strong contribution of environmental trauma (early abuse, neglect).

Constitutional predisposition for developing a dissociative disorder may include personality traits such as being easily hypnotized, mental absorption, suggestibility, and a tendency to fantasize. These characteristics fueled concerns in the 1990s that therapists may contribute to dissociative identity disorder by “digging” for repressed memories in susceptible patients and creating “pseudomemories” of events that did not happen.

The issue of repressed traumatic memory and its role in therapy is extremely controversial and contributes to the complexity of psychotherapeutic treatment of dissociation.
Identifying ‘hidden’ phenomena

Dissociative disorders have been called “diseases of hiddenness” because:

• Many of their clinical characteristics—sense of identity, memory, connectedness, somatosensory phenomena—are alterations in subjective phenomena that lack clearly observable symptoms.

• Patients are often reluctant to seek help or divulge their symptoms to clinicians.

• When dissociative symptoms are obvious—such as multiple personalities or sudden loss of memories—they may be dismissed or evoke skepticism because of their dramatic presentation.

Screening tools. To identify at-risk patients, consider screening with a validated questionnaire such as the Childhood Trauma Questionnaire (CTQ), particularly for patients with psychiatric comorbidity (Table 2). Using the CTQ—which assesses physical, emotional, and sexual abuse and neglect—is a high-yield procedure, given the role of early trauma in brain development and future mental health.

For more targeted screening, the self-report Dissociative Experiences Scale (DES) is useful for clinical assessment in conjunction with the clinician-administered diagnostic Structured Clinical Interview for DSM-IV Dissociative Disorder (SCID-D).

Differential diagnosis. Diagnosing dissociative disorders includes ruling out psychopathologies that can present with “look-alike” symptoms (Table 3, page 81).

As in Mr. D’s case, dissociative phenomena may attenuate the benefit of post-trauma therapeutic interventions, especially those involving exposure. Therefore:

• assess post-trauma patients for dissociation before you start treatment
• make specific alterations in psychotherapy for such patients, as described below.

Educating trauma patients that detachment is a normal response to threat can reduce shame about not fighting back.

Medical causes. Because complex partial seizures can cause dissociative symptoms, consider evaluating patients for seizures, continued on page 81

<table>
<thead>
<tr>
<th>Table 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>With these findings, consider screening for dissociation</strong></td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>Posttraumatic stress disorder</td>
</tr>
<tr>
<td>Certain personality disorders (especially borderline personality disorder)</td>
</tr>
<tr>
<td>Somatoform disorders (conversion disorders and nonepileptic seizures)</td>
</tr>
<tr>
<td>Eating disorders</td>
</tr>
<tr>
<td>Substance use disorders</td>
</tr>
<tr>
<td>Extensive history of trauma or neglect</td>
</tr>
<tr>
<td>Self-harm behavior</td>
</tr>
</tbody>
</table>

**Clinical Point**

To reduce shame about not fighting back, educate abused patients that detachment is a normal response to threat.

**Early trauma.** Factors that make it difficult to define the specific role of early trauma in dissociative disorders include:

• shame and secrecy of early sexual or physical abuse and potential for victims to repress traumatic memories
• lability of memory, potential for suggestibility, and difficulty with verification

Some experts—influenced by attachment theory—view dissociative phenomena as manifestations of an innate, reflexive relational pattern called disorganized attachment. Attachment theory notes that:

• early relationships are one of the primary ways that humans learn to regulate distress
• early trauma frequently includes pathology in caregiving relationships, including overt role reversal, abuse, and neglect.

Empathic treatment of dissociation, therefore, is based on appreciating the difficulties that arose from an individual’s experience of being alone with overwhelming distress. The relation of dissociation to attachment theory has specific therapeutic implications, including a focus on constructing a safe therapeutic relationship for patients.

Finally, remember that transient dissociative symptoms can be considered normal in high-stress situations. Intensive military training has been found to be associated with a very high incidence (96%) of dissociative symptoms in army recruits.
head trauma, and structural lesions. Psychogenic nonepileptic seizures (PNES) often occur in conjunction with early trauma, dissociative symptoms, and PTSD. Recreational drugs such as ketamine, methylenedioxymethylamphetamine (“Ecstasy”), hallucinogens, marijuana, and dextromethorphan also can induce dissociative states. Consider evaluating for use of these substances, some of which may not be detected on a routine drug screen.

CASE CONTINUED

A tactical shift

Internal distress—such as when remembering painful events—clearly is linked with the appearance of Mr. D’s symptoms. The therapist—recognizing unacknowledged dissociative phenomena—changes Mr. D’s therapeutic strategy from exposure therapy to affect and anxiety regulation, with an explicit focus on attachment security (safety).

The therapist explains to Mr. D that dissociation symptoms are a response to distress, and he can learn more adaptive distress regulation in therapy. The in-session focus shifts to include more direct attention to components of the therapy relationship, including overt disclosure of the therapist’s positive regard and commitment to help the patient and frequent pauses to “check in” that the patient feels present, safe, and understood. With this new focus, Mr. D’s dissociative symptoms resolve and he feels more ready to face and overcome his fear and avoided memories.

**Psychotherapy: Putting pieces together**

Psychotherapy is the primary treatment, based on understanding dissociative disorders as manifestations of distress-related, traumatic fragmentation of the sense of self, interpersonal relatedness, and capacity for adaptive affect regulation (Table 4, page 82).

**Depersonalization disorder.** Cognitive-behavioral integration has been proposed, based on the idea that detachment from one’s self creates anxiety and reinforces efforts to avoid this internal state and events that trigger it. In an open study of 21 patients with depersonalization disorder, individual cognitive-behavioral therapy (CBT) reduced avoidance, safety behaviors, and symptom monitoring. Measures of dissociation, depression, anxiety, and general functioning also improved.

**Dissociative identity disorder (DID)—**the quintessential dissociative disorder—is usually treated by specialists. Treatment is complex, but some components are appropriate

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**Table 3**

Differential diagnosis: Dissociation ‘look-alikes’

<table>
<thead>
<tr>
<th>Dissociation symptom</th>
<th>Can be confused with:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual or auditory hallucinations, other ‘first-rank’ psychotic symptoms in dissociative identity disorder</td>
<td>Psychotic disorder</td>
</tr>
<tr>
<td>‘Blanking out’ (cognitive disruption)</td>
<td>ADHD, seizures</td>
</tr>
<tr>
<td>Somatoform (conversion) symptoms</td>
<td>A variety of nonpsychiatric medical problems, including pelvic or abdominal pathology and headaches</td>
</tr>
<tr>
<td>Dissociative memory lapses</td>
<td>Learning disability, not paying attention</td>
</tr>
<tr>
<td>‘Switching’ between states</td>
<td>Bipolar disorder, rapid cycling</td>
</tr>
<tr>
<td>Lack of emotional reaction to traumatic stimuli (numbing response)</td>
<td>Healthy coping</td>
</tr>
<tr>
<td>ADHD: attention-deficit/hyperactivity disorder</td>
<td></td>
</tr>
</tbody>
</table>

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**For more on PNES**

See this article at CurrentPsychiatry.com

**Psychogenic nonepileptic seizures: Ways to win over skeptical patients**

JANUARY 2008
for less severe forms of dissociation, including dissociation as part of PTSD.28

Safety, stabilization, and symptom reduction. Providing a safe therapeutic relationship is a primary and necessary part of DID treatment. On that platform, a first step in reintegrating distressing material into the self involves building the patient’s capacity for conscious, flexible affect regulation. This keeps anxiety and distress within a therapeutic “window.”

Graded exposure. Exposure to feared mental contents—typically traumatic memories—is central to trauma-focused therapy. Dissociation is conceptualized as driven by distress greater than the system can bear, loss of adaptive integration, and subsequent fear-based, reflexive avoidance.29 Re-experiencing trauma-related memories in a safe relationship with a new regulatory capacity may work by anchoring patients in an autobiographical memory base.28

Integration of identity and person. Treatment ends when formerly unintegrated or dissociated experiences or parts of the self are integrated into a coherent whole, and the patient can deal adaptively with interpersonal relationships and distress without fragmentation.

Adjunctive medications

Few studies have addressed using psychopharmacologic interventions in the heterogeneous dissociative disorders. GABA_A antagonism and 5-HT_2A/2C agonism have induced psychotic and dissociative-like symptoms in healthy men,29 and alterations in enzymes such as catechol-O-methyltransferase (COMT) may explain individual vulnerability to trauma.30 Reports of dissociation related to ketamine31 and marijuana32 implicate other neurotransmitter systems in their etiology.

DID. Similar to guidelines for borderline personality disorder,33 guidelines for DID suggest using medications to treat the most prominent symptom clusters such as insomnia, affective instability, and posttraumatic intrusions.

Depersonalization disorder. Trials of fluoxetine and lamotrigine showed no benefit in depersonalization disorder.34,35 In an open trial of 14 patients, naloxone (mean 120 mg/d) reduced depersonalization symptoms by 30%, as measured by 3 validated scales.36

PTSD-related dissociation. If dissociative symptoms are associated with PTSD, selective serotonin reuptake inhibitors are considered first-line pharmacologic treatment.37 In a 10-week trial of 70 mostly minority adult outpatients with PTSD, paroxetine, ≤60 mg/d, was more effective than placebo in reducing dissociative symptoms, as shown by changes in DES scores.38

References


**Related Resources**


**Drug Brand Names**

- Fluoxetine • Prozac
- Naloxone • Narcan
- Lamotrigine • Lamictal
- Paroxetine • Paxil

**Disclosure**

Dr. MacDonald is a speaker for Eli Lilly and Company, Janssen, L.P., and Pfizer Inc.

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**Bottom Line**

Think of dissociative disorders as distress-related breakdowns in the functional connection and integration of components of normal consciousness. Neurobiologic changes underlie these disorders’ often-unique symptoms. Screen at-risk patients with the Dissociative Experiences Scale, and consider dissociation when assessing patients with significant early trauma, somatiform disorders, or posttraumatic stress disorders, in particular.