DEPERSONALIZATION DISORDER
Etiologic theories & biological correlates

By: David Gorman
Lecture Outline

- What is dissociation?
- DSM-IV-TR criteria for depersonalization disorder (DPD): prevalence and associated features
- Comorbidity with Axis I & II disorders
- Cross-cultural symptom presentations (culture-bound syndromes)
Lecture Outline Continued

- Self-induced (trance), substance-induced, and organic antecedents. Differentiation from depersonalization disorder.

- Main focus: Etiologic theories and hypotheses

- Brief discussion on treatment: both psychopharmacological and therapeutic interventions

- Case Vignette and recommended readings
What is dissociation?

- A loss of the integration of faculties or functions that are normally integrated into consciousness evidenced by: memory (amnesia), sensory (hallucinations), cognitive (detachment from mental processes), or identity impairments (loss of sense of self). Dissociative features may be transient or chronic (Castillo, 1997).
Persistent or recurrent experiences of feeling detached from, and as if one is an outside observer of, one’s mental processes or body (Criterion A).

Depersonalization is marked by intact reality testing (i.e. awareness that it is only a “feeling as if” one is an automaton) (Criterion B).

The depersonalization causes clinically significant distress or impairment in critical areas of functioning (Criterion C).

The experience does not occur exclusively during the course of another mental disorder; or direct physiological effects of a substance, or general medical condition (Criterion D) (APA, 2000).
DSM-V Changes

- Criterion A: the presence of either/or depersonalization or derealization, instead of just depersonalization Sxs. Thus, a clinician must specify if DPD is: depersonalization only, or derealization only (APA, 2010).
- Criterion D: differentiated into two parts for more efficient Dx of comorbid disorders (APA, 2010).
Associated Features

- A feeling or sense of being an automaton, or as if in a dream or movie (derealization).
- A sense of being an observer to one’s mental processes or body (locus of control)
- Sensory anesthesia, lack of affective response (blunting), and general lack of control over actions or speech (APA, 2000).
Associated Lab findings

- Found that individuals with DPD may display high hypnotizability and high dissociative capacity (APA, 2000).

- Implication: suggests an acquired or inherited dysfunction in the ability to cope in a normative fashion when faced with conflict (intra or interpersonal).

- Individuals who typically use disavowal level defense mechanisms (denial, projection, and rationalization) may evidence lower dissociative capacities (Bluml, Stastka, & Szerencsics, 2009).
Transient Depersonalization is common among the general and clinical population. Figures range from 19-23% (one year). 3rd most reported symptom behind depression and anxiety (Sadock & Sadock, 2007)

2.4% prevalence of DPD in the general public (Simeon, 2004). Age of onset: 16

DSM-IV-TR states that the disorder is diagnosed at least twice as often in women than in men (APA, 2000).

However, Meta-analyses find a 1:1 gender ratio of DPD diagnoses (Simeon, 2004).
Comorbidity with Axis I & II disorders

- There is often high comorbidity rates of mood and anxiety Axis I disorders; however, onset of DPD is often found to precipitate these disorders (Simeon, 2004).

- Extensive comorbidity with Axis II personality disorders (PD) (60%). Most common PD associated with DPD are: borderline, avoidant, and OCD (Simeon, 2004).
Culture-Bound Syndromes

- In pre-modern societies, depersonalization is experienced as spirit attack or possession (Castillo, 1997).

- Depersonalization experiences can be self-induced via state training in various Eastern religious traditions. These meditative injunctions are not to be confused with DPD.
Culture related schemas

- South Asian Cultural schemas: phenomenological structures such as ghosts, demons, or gods (Castillo, 1997).

- Eastern contemplative traditions: cognitive schemas of an inherent duality in the world (illusionary). Meditative practices therefore seek release through dividing consciousness (Castillo, 1997).
Organic correlates

- Individuals that experience migraines and those that have a history of epilepsy may exhibit depersonalization symptoms (Sadock & Sadock, 2007).

- Substance intoxication and/or abuse may temporarily alter brain chemistry or induce depersonalization after prolonged use (Simeon, 2004).
Chronicity of Depersonalization

- The course of DPD is typically chronic, thus, hypotheses that indicate transience of depersonalization may not be an indication of psychopathology (Castillo, 1997).

- It is important to differentiate dissociative symptoms that may be the result of acute stress disorder or PTSD.
Etiologic Hypotheses

- Integrative stress diathesis
- Psychodynamic processes in defense of the ego (i.e. repression, splitting).
- Child-Parent maladaptive attachment: abuse and neglect.
- A spontaneous stress response inducing a trance state (Castillo, 1997).
- Inherent or acquired Biological abnormalities.
Integrative Stress Diathesis

- Personality, cognitive abilities
- Psychodynamic mechanisms (i.e. immature)
- Biological Dysfunction
- Cumulative Stress (i.e. maladaptive attachments)
Psychodynamic processes

- The universality of the psychological process of dissociation without much cultural variation (phenomenology) over time may indicate inherent or culturally acquired defense mechanisms.

- In addition, dissociative symptoms are common in correlation to known stress-related disorders, such as: Acute stress disorder, PTSD, panic attacks, and phobias.
Attachment theories

- John Bowlby, among many other attachment theorists, found through their observations that humans are hardwired for attachment in service of survival.

- John Bowlby described psychological defenses against disorganization: one of which is deactivation. Deactivation is akin to emotional detachment, which may correlate to DPD Sx’s like emotional blunting (subjective expression of feelings) (Bowlby, 1980).

Source: as cited in Howell, 2008
Child-parent maladaptive attachment: abuse and neglect

- Simeon et. al (2001): found that emotional abuse was a significant predictor of DPD and symptom severity; emotional abuse plus sexual abuse was more predictive of more severe general dissociative scores (i.e. dissociative amnesia, dissociative identity disorder).

- Relationships between childhood trauma and depersonalization are well documented via meta-analyses (Simeon, 2004).
Personality and Cognitive factors

- Personality factors of DPD (Simeon, 2002): harm-avoidant temperament, immature defenses, over-connection (dependency, vulnerability), and Disconnection (defectiveness, emotional inhibition).

- Simeon et. al (2000): Focus on attention and memory using the WAIS-R.
- Results: No diffuse cognitive deterioration, lowered processing speed, or LTM impairment. However, had clear impairments on visual-spatial tasks. Also had impaired functioning on continued performance tasks.
- Implications: DPD patients seem to have impaired attentional control, which may lead to STM deficits. Poor block design scores may indicate diminished concentration as well as perceptual deficits.
Biological dysfunctions

- NMDA subtype of the glutamate receptor has been hypothesized as the central genesis of depersonalization symptoms (Sadock & Sadock, 2007).
- The NMDA antagonist ketamine (known for its dissociative capabilities) increases glutamate transmission at non-NMDA sites (Simeon, 2004).
- Simeon (2001) found a pattern of Hypothalamic-pituitary-adrenal axis dysregulation evidenced by significant hypo-suppression to low-dose dexamethasome and elevated cortisol in plasma. (See example).
Hypothalamus: control of the body’s hormonal system. Releases CRF to pituitary gland.

Pituitary gland secretes: ACTH to the adrenal glands.

Adrenal glands secrete cortisol in an attempt to alleviate the effects of stress through negative feedback to the hypothalamus and pituitary gland.
Biological dysfunctions

- Dysfunctional brain metabolic activity and sensory cortex abnormalities in areas responsible for an integrated body schema (Simeon, 2000).
- In an fMRI study by Phillips et. al (2001), they found reduced neural responsiveness in emotion-sensitive regions in correlation to blunted affect (a symptom of depersonalization).
Biological dysfunctions

- Study that tested skin conduction responses of DPD sufferers found: a significant decrease in response to unpleasant stimuli; response latency was significant; however, DPD’s had faster responses to non-specific elicitors (clap, cough) (Sierra et. al, 2002).
- Conclusion: Autonomic responses to unpleasant stimuli are depressed in DPD sufferers. Faster responses to non-specific elicitors indicates heightened state of alertness for DPD sufferers.
Etiological Conclusion

Depersonalization disorder may be influenced by multi-developmental domains, such as: Constitutional factors (i.e. personality), immature psychodynamic defense mechanisms, cumulative stress (dysfunctional attachment models), and inherent or acquired biological abnormalities.
Treatment interventions

- Psychopharmacological: SSRI’s (Fluoxetine) found to be ineffective despite possible serotonergic involvement (Kutcher, 2004).
- Use of Lamotrigine, an NMDA antagonist: conflicting results.
- Opioid antagonists have seen some success: Naloxone and Nalmefene (reduces emotional numbing).

Source: Simeon, 2004
Treatment interventions

- Psychotherapy: no evidence-based practices are particularly supported.
- Psychodynamic, trauma-based, CBT, hypnotherapy, and cognitive interventions may be used to “re-integrate” personality features, self-object relations, negative automatic thoughts (reinforcement), and controlled regressive techniques in order to resolve trauma-related material (Simeon, 2004).
“Sam was 17 years old when he was first seen by a psychiatrist. He grew up at home with a bipolar mother, a relatively intact father and a younger brother. From as far back as he could remember his mother would have decompensations at home, during which she would get violent, rage at the family, feared that neighbours were out to hurt her because she had special powers, and would lock herself up in her room. From around the age of eight, Sam could recall feeling fleetingly unreal, like he was living a dream, when his mother lost it. These bouts of unreality would typically last for a few hours. Sam was otherwise a well-adjusted kid, had many friends and hobbies and was a good student. When he was 16, his mother had a severe decompensation at home, during which she grabbed a kitchen knife in front of her children and attacked her husband believing that he was having an affair. He was able to subdue her and called 911, and she was taken to the hospital. Sam felt that his family was irreparably damaged, and that he may have lost his mother for good. As the Emergency Medical Service personnel forced her out of the home, Sam felt as if a veil fell in front of his eyes; existence finally felt like a dream, his body felt deadened, and he went numb. Despite his father’s efforts to comfort him and his mother’s return home two weeks later, Sam continued to feel unreal and deadened over the next year. He did not want to burden his already burdened father with his terrifying state of which he could not make sense. He knew that what he felt was strange and inexplicable, and did not want to share his state with any of his friends in case they thought he had lost his mind. About a year later he finally confided in his father, who arranged for him to see a psychiatrist. The psychiatrist diagnosed him with depersonalization disorder.”

Reference: Moskowski, Schafer, and Dorahy, 2009.
Test Questions

1. What is the typical age of onset for Depersonalization disorder?
   Answer: 16

2. True or false: Depersonalization sufferers have intact reality testing.
   Answer: True, depersonalization is an “as if” feeling with no psychotic features.
Recommended Readings


- These books are good resources regarding etiology of dissociative disorders and include case vignettes. They are current, and free to look at on ebrary reader.
References

References II