A trauma-related dissociation model may explain psychopathology of the difficult-to-treat BPD patients

Dana Maria Bichescu-Burian

Abstract

There is a difficult-to-treat core group with severe psychopathology among borderline personality disorder (BPD) patients. Treatment advance for this group calls for extended etiological knowledge. I propose an evolutionarily-based model of trauma-related dissociation that will help explain psychopathology of severe BPD cases and has not yet been investigated in this area. Clinical and neurobiological evidence shows that trauma-related dissociation is a significant factor in the BPD etiology and indicates the model validity. Further evidence and model elaboration should be obtained from clinical and psychophysiological assessments of dissociative phenomena in BPD.

Keywords: Dissociation; Traumatic Stress; Borderline Personality Disorder; Dissociative Disorders; Complex PTSD;

1. Introduction

Clinical research recognized a core group of difficult-to-treat BPD patients with dissociative disorders (DDs), functional impairment, severe comorbidities, history of childhood abuse, and high use of health services (Korzekwa et al., 2009; Fig. 1). The “complex PTSD” notion was discussed as a proper diagnosis to classify the effects of severe neglect, emotional, physical, and sexual abuse also seen in BPD (Herman, 1992). Since BPD and DDs are adaptations to severe early trauma seen as “trauma spectrum disorders”, a dissociation model for PTSD can be proposed to explain severe dissociative pathology in BPD.

Dissociation is an altered state of consciousness characterized by a disintegration of sensation, emotion, and cognition. Evolutionary theories assume that dissociation belongs to the biological defense mechanisms developed along with the classic Freeze-Flight-Fight reactions as a coherent system of...
survival responses that progresses along several stages. Although involving risks for injuries, dissociation is the remaining strategy that reduces extreme affect and facilitates adaptive behavior to near, inescapable threat by a superior aggressor (Bracha et al., 2004). Hence, anxiety causes the medial prefrontal cortex to inhibit emotional processing by the limbic system, sympathetic output is reduced and dissociation arises. Defense responses are modulated by specific threat characteristics and by previous experiences, i.e. peritraumatic responses that have become classically conditioned (Adenauer et al. 2009). The view of dissociation as a classically conditioned defense mechanism recurring during confrontation with traumatic cues complies with the documentation of peritraumatic dissociative reactions (e.g. automatic derealization and depersonalization; Birmes et al., 2003). The persistence of such reactions is highly impairing resulting in disorders of the trauma spectrum, e.g. PTSD and DDs. There is substantial evidence that peritraumatic dissociation robustly predicts psychological problems including PTSD (Birmes et al., 2003), as well as DDs and BPD (Sar et al., 2004). In what follows, I shortly describe the model and argue for its relevance in the area of BPD providing clinical and neurobiological evidence.

Fig. 1. Schematic illustration of the relationship between dissociation, BPD, and psychological trauma according to the research findings on prevalence of traumatic antecedents and dissociative pathology in BPD. The middle of the overlap (the darkest area) indicates the group of the severely impaired, difficult-to-treat BPD patients.

2. An evolutionarily-based etiological model for trauma-related dissociation

Despite great clinical significance, current BPD models fail to offer thorough account of the onset of dissociation. I therefore propose a PTSD model of dissociation (Schauer & Elbert, 2010) also in order to explain dissociative pathology of severe BPD cases. I show how the model postulations apply to BPD:

- A sixth defense reaction ("Flag"/"shutdown dissociation"; Schauer & Elbert, 2010) has been included along with other reactions (Freeze-Flight-Fight-Fright-Flag-Faint; Table 1), all of which are distributed on 2 dimensions of the defense cascade – succeeding in increasing (sympathetic arousal) and decreasing activation levels (parasympathetic dissociative reactions; Fig. 2). By incorporating the "shutdown dissociation" in the conventional defense models, using this model in BPD, I argue, has the potential to overcome an important shortcoming of previous models, i.e. their inability to specify where and how exactly dissociation arises among defense responses.

- The shutdown peritraumatic dissociation is dominated by parasympathetic activity. This is in line with findings on neurophysiological activation patterns seen during dissociative states in BPD, as I indicate later. The specification that shutdown dissociation corresponds to severe trauma (overwhelming, inescapable threat by a superior aggressor, e.g. child abuse) corroborates findings on high occurrence of pathological dissociation in BPD patients with a history of early severe physical and sexual abuse.

- Specific peritraumatic responses are connected in the memory system with features of the threatening situation enabling prompt danger-focused reaction, but they can become pathologically disconnected from the spatio-temporal context. The actual peritraumatic reaction will be reenacted when the trauma-related memory is reactivated, generating at least 2 main symptom profile subtypes (hyperarousal/dissociation). Accordingly, the shutdown dissociation will produce a dissociative group of patients...
with a dominant parasympathetic activation and a dissociative responding pattern to trauma-related stimuli. My approach suggests that this dissociative pattern in BPD will become generalized, so that it may be even caused by minor stressors and these patients also exhibit severe DDs and other problems. This is consistent with the subsequently presented findings on BPD types, dissociative reaction patterns in severely traumatized BPD patients, and great treatment difficulties. Although the model is biologically well-documented, it made no descriptions of psychological and behavioral reactions. I propose that the psychological component of shutdown dissociation consists of submissive, clinging thinking and behavior and these patients also show disorganized attachment and traumatic reenactment (self-destructive/masochist behavior, re-victimization), and avoidance of trauma memories as expressions of dissociative fragmentation intrinsic to the specific neurobiological modifications.

2.1. Evidence coming from the clinical research

![Graphic illustration of the defense cascade as it evolves along 6 stages](image)

**Fig. 2.** Graphic illustration of the defense cascade as it evolves along 6 stages (see Schauer & Elbert, 2010). The sympathetic arousal reaches a maximum at the fright stage, and is eventually replaced by the onset of dissociative "shut-down" (the gray area).

**Table 1.** Short description of the 6 responses within the defense cascade according to Schauer & Elbert (2010).

<table>
<thead>
<tr>
<th>Stages</th>
<th>Short description</th>
<th>ANS activation pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freeze</td>
<td>“stop, look, and listen”/orienting response: focused attention, information processing, preparedness</td>
<td>moderate sympathetic</td>
</tr>
<tr>
<td>Flight &amp; Faint</td>
<td>“alarm response”: increased arousal &amp; heart rate, cardiac &amp; muscular vasodilatation, faster &amp; deeper breath</td>
<td>marked sympathetic</td>
</tr>
<tr>
<td>Fright</td>
<td>“tonic immobility”: high alertness, unresponsiveness, and preparedness for a possible escape in cases of direct physical contact (e.g. sharp objects, injury)</td>
<td>sympathetic &amp; parasympathetic</td>
</tr>
<tr>
<td>Flag &amp; Faint</td>
<td>“flaccid immobility” with unresponsiveness, derealization, depersonalization, emotional numbness, memory and central information processing decline</td>
<td>marked parasympathetic</td>
</tr>
</tbody>
</table>

2.2. Evidence coming from the clinical research

Dissociation is central in BPD (Zanarini et al., 2000) and may become a chronic reaction elicited by daily hassles (Birmes et al., 2003). Epidemiological BPD studies described highly prevalent dissociative
pathology up to 76.2 % (Korzekwa et al., 2009). BPD is diagnosed in up to 70% of the DDs patients (Sar et al., 2004). In BPD, a strong link between dissociation and psychological trauma is empirically supported, with the dissociative level as predicted by the severity of early trauma (Dutra et al., 2009). Numerous early adversities (Zanarini et al., 2002), substantially more than other personality disorders (Yen et al., 2002) and high PTSD rates up to 70% (Golier et al., 2003) are being reported in BPD.

2.3. Evidence coming from the field of treatment research and practice

Severe dissociation in BPD constrains affective processing and learning and impedes treatment (Ebner-Priemer et al., 2009). Clinical trials identified dissociative symptoms in the wake of childhood sexual abuse as a high-ranking predictor for negative treatment outcome in BPD (Paris, 2009). Moreover, dissociation is difficult to treat (Foa et al., 1998). Treatment failures due to dissociative reactions in severely traumatized BPD patients are frequent, followed by self-harm, suicide attempts and readmissions. A metaanalysis of RCTs (Brazier et al., 2006) indicates treatment efficacy to a certain degree (e.g. reducing impulsive and parasuicidal behavior, suicidal ideation). Anyway, there are not sufficient remedies for dissociative and posttraumatic symptoms in BPD patients (Dyer et al., 2009). This could be due to the scarce consideration of trauma-related dissociation during treatment.

2.4. Neurobiological evidence

A BPD investigation of neural processing in dissociative states (Ludäscher et al., 2010) shows a frontolimbic activation resembling that seen in dissociative PTSD patients. Moreover, BPD patients with high state dissociation exhibit reduced acquisition of differential aversive delay conditioning with respect to emotional learning aspects compared with healthy and non-dissociative BPD participants (Ebner-Priemer et al., 2009). A neuropsychological review (Irle et al., 2010) indicates that, similarly to PTSD, early traumatized BPD patients reveal small hippocampal and amygdala volumes and impaired cognition.

3. Conclusion

Different sources of empirical evidence on the prevalence of dissociative phenomena and their pathogenic development mechanisms in BPD indicate that the proposed evolutionary-based model of trauma-related dissociation offers a helpful framework of understanding dissociative phenomena in severely traumatized BPD patients. Despite this, none of the evaluated treatments up to date is directly related to an explicit model of trauma-related dissociation. So far, the psychophysiological and neurobiological correlates of dissociation in BPD favor the notion of trauma-related dissociation by indicating a parasympathetic activation pattern and emotional modulation mediated by the inhibitory effect of the medial prefrontal cortex on the limbic system. Clinical BPD investigations with a focus on the dissociative phenomena and psychophysiological assessments of reactions to the reactivation of traumatic memory within a script-driven imagery paradigm may provide substantial evidence for the relevance of the model I propose to apply in the area of BPD. This offers a useful paradigm for understanding this complex disorder with important treatment implications. In particular, it helps enrich trauma-focused treatment approaches for successfully addressing the specific needs of traumatized BPD patients with dissociative pathology.
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References


