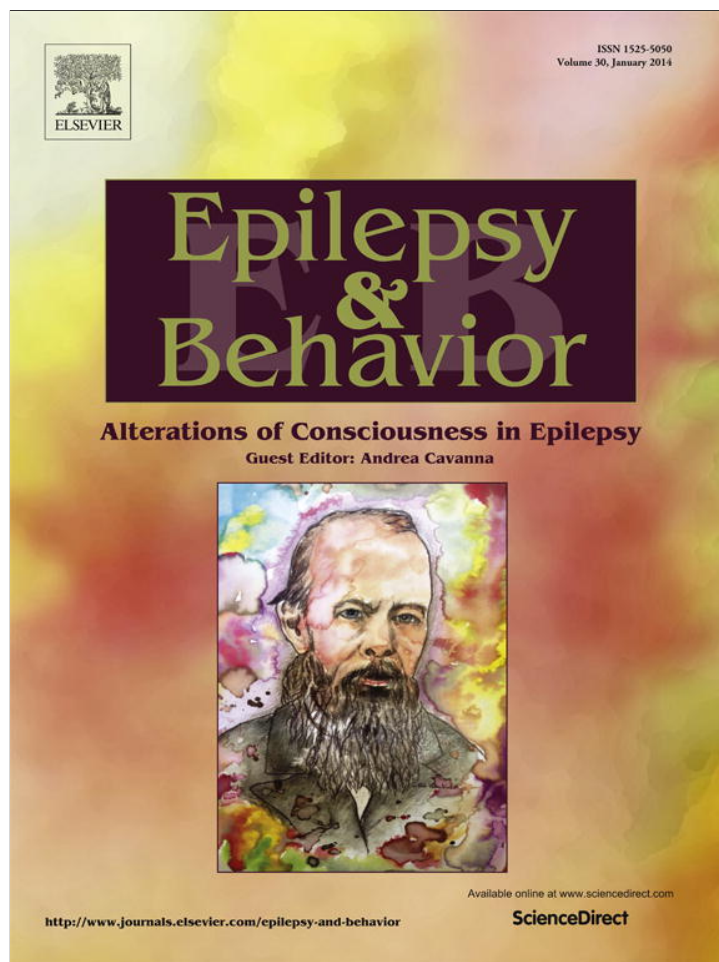


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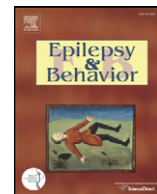
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Review

Alterations of consciousness in psychogenic nonepileptic seizures: Emotion, emotion regulation and dissociation

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ABSTRACT

Impairment of consciousness and reduced self-control are key features of most psychogenic nonepileptic seizures (PNESs), although, compared with patients with epilepsy, those with PNESs demonstrate greater conscious awareness during their seizures. The neurobiological underpinnings of PNESs and of alterations of awareness associated with PNESs remain relatively unknown. We suggest that an understanding of conscious experiences and discrepancies between subjective impairment of consciousness and the lack of objectifiable neurobiological changes in PNESs may benefit from an examination of emotion processing, including understanding sensory, situational, and emotional triggers of PNESs; emotional and physiological changes during the attacks; and styles of emotional reactivity and regulatory capacity. We also suggest that in addition to the typical comparisons between patients with PNESs and those with epilepsy, studies of PNESs would benefit from the inclusion of comparison groups such as those with PTSD, dissociation, and other forms of psychopathology where dissociative and emotion regulatory mechanisms have been explored more fully. We conclude that current evidence and theory suggest that impairment of consciousness in PNESs is only “dissociative” in one subgroup of these seizures, when consciousness is suppressed as a collateral effect of the excessive inhibition of emotion processing. We propose that PNES behaviors and experiences of reduced control or awareness may also represent direct behavioral manifestation of overwhelming emotions, or that minor emotional fluctuations or relatively neutral stimuli may trigger PNESs through conditioning or other preconscious processes. Future studies exploring the neurobiological mechanisms underpinning PNESs are likely to be more fruitful if researchers bear in mind that it is unlikely that all PNESs result from the same processes in the brain.

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1. Introduction

Disruptions in consciousness characterize many types of epileptic seizures, and the nature of these disruptions is a complex topic of investigation [1,2]. Arguably, the nature of disruptions of consciousness in psychogenic nonepileptic seizures (PNESs) is even more complex. In PNESs, patients have experiences and show behaviors which superficially resemble those associated with epileptic seizures but without any identifiable concomitant electrophysiological abnormalities [3,4]. Nearly two-thirds of observers report that patients lose awareness or the ability to react during their PNESs, and over half of observers endorse that patients' attacks “always” involve a “complete loss of consciousness or blackout” [5]. Compared with the observers of PNESs, patients themselves are less likely to state that they lose consciousness,

but 30% still endorse that they “always” “have no idea what is happening around them during their attacks” [5]. On the other hand, compared with patients with epilepsy, those with PNESs demonstrate greater general awareness/responsiveness (level of consciousness) and more subjective experiences (content of consciousness) during their seizures [6].

If most patients are not in fact losing consciousness, and their subjective level of awareness is greater than it may appear to observers, this raises the question of how consciousness is in fact altered in PNESs. Here we discuss how an understanding of conscious experience and alterations in consciousness in PNESs may benefit from considering models of dissociative and affective and emotion regulatory processes more broadly.

1.1. PNESs and consciousness

In their review of this topic, Reuber and Kurthen define consciousness broadly as “the interaction and temporal coordination of a wide range of neural subsystems of the human brain (including but not limited to those underpinning sensation, attention, voluntary

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movement, and memory)” [7] (p. 96). As noted above, evaluations of consciousness in PNEs and epilepsy have examined both level and content [8], which are thought to be subserved by different neural systems [2,9]. The study of consciousness is complicated by the fact that there can be different levels of conscious awareness. For instance, Frith and Lau [10] distinguish between (1) *the subject is fully aware of the stimulus*; (2) *the subject claims not to be aware of the stimulus, but can make guesses about the stimulus better than chance*; and (3) *the subject claims not to be aware of the stimulus, guesses at chance levels, but nevertheless responds to the stimulus in terms of brain activity and/or behavior* (p. 761). These levels are, to a large extent, amenable to testing through experimental manipulations. Although they were not developed with patients with seizures in mind, the consideration of these levels may be of particular value in PNEs because they offer ways of testing some of the types of discrepancies mentioned above (e.g., subjective awareness versus behavior or observer perceptions).

Reuber and Kurthen also present the distinction of *phenomenal* consciousness, or what it feels like to have a particular experience, and *access* consciousness, or having access to higher-order cognitive processes [7] (see also [11,12]). Understanding phenomenal consciousness can offer important information (see [10] for a general discussion of consciousness and the value of introspection). For example, phenomenal consciousness in seizures has been examined by analyzing the metaphors patients use for their paroxysmal experiences: whereas patients with epilepsy tended to conceptualize their seizures as an agent/force or event/situation (i.e., as an independently acting entity treating the patient as the target or observer of the seizure), those with PNEs were more likely to conceptualize their attacks as spaces/places that the patient “goes into” or “cannot come out of” [13] (also discussed in [7]). As discussed later, this may be an indication that the seizure-related subjective experience is more dissociative for patients with PNEs than for those with epilepsy. This, in turn, could have treatment implications: examining a patient’s seizure metaphors and sense of agency may offer the patient and therapist a linguistic starting point from which to create an acceptable and credible treatment formulation and increase the amount of control the patient has in and over the seizures [14,15].

1.2. PNEs as a dissociative disorder?

Psychogenic nonepileptic seizures are classified in ICD-10 as a dissociative disorder [16] and anticipated to become a dissociative/functional disorder in ICD-11 [17], and PNEs are often referred to as dissociative seizures [18–21]. On this basis, it could be argued that, by definition, the apparent alterations in consciousness associated with PNEs must be “dissociative” in nature. However, dissociation is a multifaceted and contested construct, and its relationship to altered states of consciousness has been debated. For example, Nijenhuis and van der Hart [22] propose that dissociation should be defined more precisely along the lines of its early conception as a personality construct [23]. They suggest that “normal” altered states of consciousness (e.g., absorption, feeling “spaced out”), and even the subjective sense of detachment that accompanies depersonalization disorder, are not dissociative per se, as they do not reflect a dysfunctional organization or division of personality (e.g., as is the case with dissociative identity disorder [22]). Brown [24], on the other hand, argues that such states are in fact dissociative but agrees with the need for greater conceptual precision (see below).

Several studies suggest that only a subset of patients with PNEs show dissociative tendencies in general and/or during their nonepileptic events (at least when these tendencies are measured using self-report questionnaires) [5,25]. In this subgroup of patients, the visible manifestations referred to as “seizures”—because they include behaviors such as shaking, spasms, or attentional lapses—may indeed be dissociative. If so, their episodes of dissociation could be a response to intolerable panic, anger, frustration, guilt, fatigue, or other

experiences. In addition, the attacks themselves may provide relief not only from aversive emotional experiences but also, paradoxically, from the aversive experience of anticipating the attack itself [21]. Alternatively, as discussed later, a range of emotional stimuli and experiences may serve as direct triggers for PNEs without necessarily invoking dissociative processes. Study designs comparing patients with PNEs with those experiencing dissociative states and other forms of psychopathology—in addition to the typical comparisons with patients with epilepsy and healthy controls—may help to explore these hypotheses.

1.3. Defining and measuring dissociation

The *Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5)* defines dissociation as “a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior” [26] (p. 291). Evidence has been mixed as to whether patients with PNEs do, in fact, show greater dissociation tendencies (see discussion in [27]). Dissociation is typically assessed as a single construct using self-report measures, most commonly the Dissociative Experiences Scale (DES [28]) or Dissociation Questionnaire (DIS-Q [29]). Evaluating dissociation as a single construct has been raised as problematic by many theorists and researchers [22,24,30,31] and may obscure answers regarding PNE mechanisms and differential diagnosis. For example, patients with PNEs reported more dissociative symptoms than those with epilepsy based on an overall dissociation score [20,25]. However, such reports were accounted for by general psychiatric distress [32]. On the other hand, somatoform dissociation, which focuses on symptoms suggesting lack of integration of sensory and motor functioning in particular [33], was greater in patients with PNEs than those with epilepsy; this relationship held even after accounting for general psychiatric distress [32]. Similarly, Alper and colleagues found that overall reports of dissociation did not differ between patients with PNEs and those with epilepsy [34], but a depersonalization/derealization dimension of dissociation (which may be conceptually distinct from somatoform dissociation; see below) was greater in patients with PNEs. Finally, dissociation (measured using the DES and a clinical interview), hypnotizability, and absorption all failed to differentiate patients with PNEs from those with epilepsy, whereas demographic and seizure variables (e.g., age at onset) did show diagnostic differentiation [27]. Therefore, if “dissociative seizures” are in fact dissociative in nature, other methods or conceptualizations—going beyond self-report and including a more systematic examination of emotional and physiological processes—may be needed to capture this.

1.4. Two types of dissociation and PNEs

Brown has proposed two types of dissociation: *detachment*, which involves psychological distancing from one’s environment and includes symptoms such as depersonalization in response to a traumatic event, and *compartmentalization*, which involves a compromise in function, as in paralysis or other somatoform conditions including PNEs [31,35]. An empirical study examining this distinction in PNEs offered equivocal results, however [36] (see also discussion in [7]). Patients with PNEs endorsed compartmentalization (measured as somatoform dissociation) to a greater extent than those with epilepsy (similar to Kuyk and colleagues’ findings [32]), but this difference was not retained when statistically controlling for anxiety scores. Results also suggested a trend toward greater detachment among patients with PNEs than those with epilepsy (similar to Alper and colleagues’ findings [34]). The authors discuss that the use of a single measure of detachment and compartmentalization was a limitation of the study—although even the use of multiple self-report measures may not have provided a clearer insight into the processes of dissociation and the physiological mechanisms underpinning these processes, due to the limitations

inherent in the use of self-report questionnaires in this area. We note that with respect to Brown's dissociation subtype distinction, models of dissociation focusing on depersonalization (discussed below) offer evidence that may apply to patients with PNEs, and that it may therefore be useful to consider detachment and not just compartmentalization processes in PNEs.

1.5. Relevance of emotion to consciousness and dissociation

One particularly important construct that intersects with consciousness, dissociation, and their neural underpinnings is emotion. At its broadest conceptualization, emotion is an adaptive system that involves changes in physiology, behavior, and/or subjective experience (see Fig. 1) [37,38]. Through emotion regulation, automatic and effortful processes are employed to alter aspects of emotional experience or behavior [39]. Emotion is implied, if not stated explicitly as a contributing factor, in most theories of PNEs [20,40–42] and most theories of conversion or dissociation. Conversion or dissociative responses are thought to occur when emotional arousal becomes overwhelming (e.g., in response to strong anxiety or trauma) and must be suppressed—or, in the case of medically unexplained symptoms, expressed somatically—in order to cope [43]. For example, in PTSD, the emotions that accompany a traumatic event (i.e., feelings of fear, helplessness, or horror upon witnessing or experiencing severe injury or death, or threat of severe injury or death [44]) may be met with a “shutting down” of emotions in general [45,46]. This has been described more recently as “emotional overmodulation” [47,48] and may be accompanied by a shutting off of other internal sensations and/or awareness of external stimuli. As Nijenhuis and van der Hart [22] describe, trauma can be considered a “psychobiological ‘wound’” that limits “integrative capacity as revealed through dissociation, affect dysregulation, and persistent avoidance of traumatic memories” (p. 419). Dissociation may take the form of a disconnect between different aspects of emotion processing or a lack of integration of emotional information into one's sense of self and awareness. A host of neurobiological and behavioral studies of PTSD and some studies with patients with PNEs offer evidence for such a model.

Studies that systematically measure emotional awareness, gut-level feelings, preattentive or “nonconscious” processing, and other physiological or behavioral signals that precede or occur apart from subjective (conscious) awareness may inform discussions of PNEs and consciousness. Below, we offer examples of such studies conducted with patients with PNEs or other relevant populations, including individuals with PTSD and/or dissociative tendencies. Several of these studies include physiological measures or present neurobiological models relevant to conscious awareness, dissociation, emotion, and their intersection. We believe that a key conclusion arising from neurobiological studies of dissociation is the relevance of emotion processing and emotion dysregulation, which may have implications for understanding and, perhaps, defining consciousness.

1.6. Relating emotional arousal, dissociation, and awareness

Depersonalization is conceptualized as a component of dissociation whereby individuals feel detached from themselves, their emotions, and their surroundings—including a “profound inhibition of emotional responses” [49] (p. 903)—while maintaining an awareness of this sense of detachment [49,50]. In a study that highlights intersections of dissociation, emotion, and conscious awareness, Felmingham and colleagues [51] tested a neurobiological model of depersonalization (described in [49]) in patients with PTSD who were high and low in dissociation. Participants were identified as high and low in dissociation based on a clinical interview administered after they viewed fearful facial expressions in an MRI scanner. Participants who reported feeling *numb, unreal, or outside of myself* while viewing the faces were classified as high in dissociation [51]. To assess the role of conscious awareness in emotion processing among the groups with high and low dissociation, participants were presented with fear stimuli that were unmasked (targeting “conscious” awareness) and masked (targeting “nonconscious” awareness [51]). In response to conscious (unmasked) fear stimuli, participants who were classified as having high-dissociative PTSD showed greater left-sided medial prefrontal cortex activation but did not show amygdala activation. In response to nonconscious (masked) fear stimuli, these participants showed the opposite pattern, namely

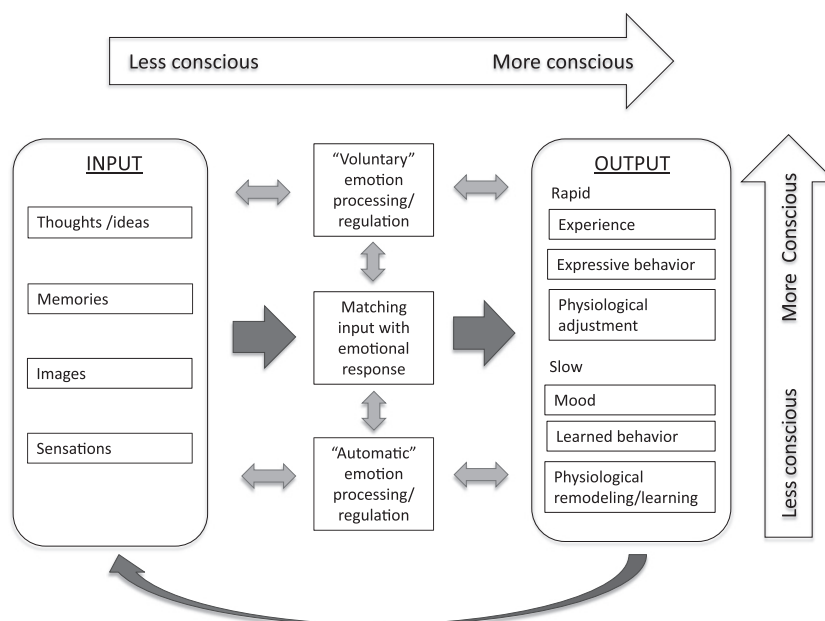


Fig. 1. Overview of emotion processing, whereby inputs are appraised—often with relatively low levels of conscious awareness—and matched to a “prototype” for triggering an emotional response. Emotion-related immediate and longer-term changes, including autonomic and immunological changes, are activated and also provide feedback to input sources. Output responses and input triggers of these responses may be controlled (relatively more conscious) or automatic (relatively less conscious), with corresponding differences in level of subjective awareness and regulation of these processes. Adapted from Levenson's core emotional system and biocultural model of emotion [73–75]. This model is not incompatible with current theories suggesting that emotion and emotion regulation may happen simultaneously [76], that behavior may precede emotion [77], or that there may be other ways that emotion may be organized based on neural networks [78].

greater amygdala activation in the absence of greater prefrontal activation (although right-sided prefrontal activation was expected [51]). Based on these patterns and previous research, the authors suggest that individuals with PTSD and greater dissociative tendencies may attempt to regulate emotions to a greater extent than those without dissociative tendencies, while at the same time, they are less well equipped to process threatening stimuli (i.e., have “impaired cognitive resources available”) [51] (p. 1778). Indeed, greater efforts to regulate emotions may not necessarily translate into success in doing so. Further, even if such efforts are successful from a short-term behavioral perspective, “success” may come at a cost, either in the moment or with respect to future adaptive functioning, as a host of research suggests that there are adverse consequences of suppressing or overcontrolling emotions [52–54].

In sum, within PTSD, there appears to be a subgroup of “high dissociative” individuals who are more reactive to threatening stimuli in their environment. These individuals respond to such stimuli by overcontrolling their emotions—their response does not necessarily involve any conscious awareness of the stimuli. Although the direction of causality cannot be determined with respect to whether such dissociative tendencies precede or result from trauma, some neurobiological evidence (see below) suggests that this subset of individuals may already have had tendencies toward emotional distancing or disengagement prior to experiencing traumatic events. The differentiation of patients with PTSD into “high” and “low dissociators” is now so widely accepted that it is reflected in the DSM-5 criteria for PTSD as a specifier [26,55]. Given the considerable etiological overlap between PTSD and PNEs, such a typology may inform future distinctions of PNE subtypes as well.

1.7. Emotional arousal, awareness, and avoidance in PNEs

A pattern of greater emotional intensity, coupled with maladaptive patterns of regulating this intensity, is evident in several studies of patients with PNEs. Bakvis and colleagues conducted a series of studies to examine preconscious attention to emotional stimuli in this patient group. Such stimuli may be perceived without conscious awareness and still alter cognition, mood, and/or behavior. In one study, patients with PNEs and healthy controls viewed angry, happy, or neutral facial expressions that were presented briefly and then followed by a “masked” stimulus (scrambled faces); in pilot testing, this masking procedure obscured the ability to consciously detect (i.e., report) that faces or emotions had been displayed [56]. Patients with PNEs but not controls showed greater preconscious attention to angry faces versus happy faces, as indicated by longer reaction times to name the color of ink in which masked anger faces were presented, suggesting greater attunement or vigilance to social threat [56]. This pattern was only apparent when participants were resting before a stress task and not immediately after this task [56]. This is notable in light of the baseline or “resting state” neural patterns in PNEs discussed below. The greater attentional bias for angry faces was also associated with higher baseline cortisol levels [57] and, for patients with PNEs, with reports of sexual trauma [56]. Therefore, subtypes of patients with PNEs, such as those with comorbid basal hyperreactivity or trauma histories, may be particularly prone to heightened threat detection, perhaps akin to the group with high-dissociative PTSD described earlier [51].

Preconscious emotional biases not only have repercussions in their own right—such as the uneasiness that can stem from greater attunement to social or interpersonal threats—but are also problematic because they can interfere with cognitive performance. In addition to the studies described above, Bakvis and colleagues [58] found that patients with PNEs showed greater working memory difficulties compared with controls after viewing emotional faces (either happy or angry) and also showed greater working memory difficulties after a stressful task, even without distraction from emotional faces. Higher

cortisol was also associated with working memory impairment, again pointing to the relevance of resting-state physiological processes [58].

Finally, patients with PNEs were slower to “approach” social threat stimuli (angry faces) than controls, based on a task where they used arm movements to signal “approach” versus “avoidance” [59]. This, again, is consistent with models of dissociation, suggesting that, paradoxically, baseline states of hyperarousal and hypervigilance to threat may result in an increased desire to avoid emotional stimuli. In addition to potentially heightened emotional intensity—which again, may occur in the absence of awareness of such arousal—an inability to regulate emotional arousal could exacerbate cognitive and behavioral difficulties and ultimately trigger motor programs that take the form of PNEs (described below). As shown in Fig. 1, emotion processing involves a number of components reflecting varying degrees of conscious awareness; accordingly, a number of different emotion processing problems could result in PNEs.

Echoing the above findings, patients with PNEs who were studied in our laboratory reported greater emotional intensity to neutral and pleasant pictures, showed less emotional intensity (based on observable behavior) to pleasant pictures, and showed physiological and self-report indicators of emotion regulation difficulties [60]. We included comparison groups of seizure-free, trauma-exposed individuals with low and high levels of posttraumatic stress symptoms (PTS-low and PTS-high, respectively). The pattern of patients with PNEs reporting greater emotional intensity was apparent even compared with participants showing comparably elevated levels of posttraumatic stress symptoms that exceeded a clinical cutoff for PTSD (PTS-high) [60]. Patients with PNEs and seizure-free participants high in posttraumatic stress symptoms did not differ in self-reported emotion regulation difficulties or in the extent to which respiratory sinus arrhythmia (RSA) was diminished (a low RSA indexes a low parasympathetic tone associated with increased levels of autonomic arousal) [61,62]. Both groups, however, showed greater dysregulation than seizure-free participants low in posttraumatic stress symptoms. Therefore, patients with PNEs were characterized by disconnects in terms of intense emotional experience, deficits in ability to regulate these experiences, and blunted behavioral responses. The similarities discovered in study participants without PNEs but with high levels of posttraumatic stress symptoms demonstrate the importance of including appropriate comparison groups (i.e., not only patients with epilepsy) to identify overlapping versus unique patterns of dysfunction in PNEs.

Similarly, an in-depth emotion assessment of five patients with PNEs revealed that those with PNEs tended to disengage emotionally when the stimuli were personally meaningful and reached a certain threshold of intensity (Roberts, unpublished results). For example, when asked to generate memories regarding past events evoking happiness, anger, or shame, individuals with PNEs reported greater difficulty reliving the anger and shame memories than trauma-exposed controls. Additionally, one patient with PNEs was unable to generate a shameful memory and became unresponsive, probably reflecting a nonepileptic attack. This is consistent with qualitative data suggesting that “unspeakable dilemmas”, particularly with respect to one’s family and often involving shame, may be associated with PNEs and related syndromes [63]. Many types of emotions, therefore, such as shame, guilt, or anger—and attempts to suppress or avoid these emotions—may trigger or exacerbate PNE symptoms. It is important to point out that seemingly neutral situations may evoke these emotions as well; however, idiosyncratic negative emotional triggers appear to be embedded within these “neutral” contexts (e.g., describing the routine of doing laundry may evoke memories of childhood maltreatment; Roberts et al., unpublished data). This can occur with or without the patient’s conscious awareness, and, therefore, identifying such triggers may have therapeutic utility [64–66].

1.8. Neural underpinnings of PNEs, dissociation, and emotion

Studies examining functional connectivity (connections among neural regions) in PNEs and dissociative states more generally offer further evidence of heightened emotional intensity during baseline states (i.e., in the resting or “interictal” state), and suggest that neural patterns in response to this intensity may underlie PNE pathology. In a review of the literature on functional connectivity and dissociative states, van der Kruijs and colleagues [67] identified that individuals higher in dissociative ability or “the ability to obtain distance from reality” show distinct physiological patterns as evidenced by EEG, heart rate variability, and functional MRI (p. 1). For example, neuroimaging studies reveal that individuals higher in hypnotic suggestibility, or with dissociative conditions such as dissociative identity disorder, dissociative amnesia, or high-dissociation PTSD, show less frontal synchrony and less frontal activation (summarized in [67]).

One study looking at functional connections in patients with PNEs did not find connectivity differences between patients with PNEs and those with epilepsy; however, among patients with PNEs, a greater seizure frequency was associated with reduced interictal synchronization in prefrontal regions and reduced synchronization in parietal regions [68] (also discussed in [67]). Van der Kruijs and colleagues [69] also studied functional connectivity in PNEs by examining resting-state fMRI data. Based on their findings, they proposed that greater connectivity between frontal motor and limbic areas and reduced connectivity between supplementary motor and prefrontal areas may result in emotions triggering motor symptoms while bypassing higher control centers [67,69]. Considering that the “control” aspect of their model may be a proxy for conscious awareness, this offers a potential neural mechanism for the idea that patients with PNEs may be “less conscious” of their emotional state and may resist the idea that emotional problems could be the cause of their symptoms [70].

Although much of the present paper has focused on the notion that disruptions in consciousness in PNEs reflect dissociation, nondissociative mechanisms certainly may occur instead or in addition (see Fig. 2). For example, high levels of emotional intensity or particularly aversive and self-threatening emotions such as shame (even if experienced at a lower intensity) may overwhelm the emotion processing system and produce experiences and behaviors which would be interpreted as PNEs without a dissociative process. In these cases, PNEs would be a direct expression of emotions such as anger or despondency. Patients may be (at least partially) aware of such emotions, although they may

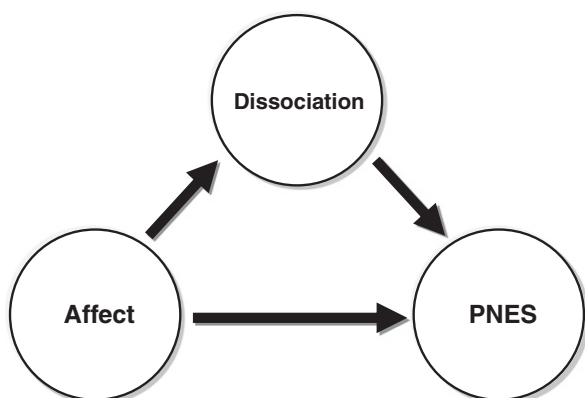


Fig. 2. Depicts the idea that affect can directly trigger PNEs (reactive/nondissociative path) or can lead to dissociation and, in turn, a PNE episode (inhibited/dissociative path). Affect that triggers PNEs (directly or indirectly) may be overwhelming (e.g., strong intensity of emotions, self-threatening emotions), consistent with a traditional model of conversion or dissociation; alternatively, the affect may be relatively neutral or lower in intensity, with PNEs and/or dissociation triggered through conditioning, a lowered threshold of responding, or emotion regulatory failures.

not be able (or willing) to share these emotions with health-care professionals. This would parallel van der Kruijs's model [69], whereby emotions trigger motor symptoms directly (although the model does not specify whether it is unexpressed or expressed emotions, and/or simply emotional awareness, that may be particularly likely to trigger motor symptoms). Clinical experience suggests that PNE behaviors can also be triggered by apparently neutral stimuli or very modest emotional fluctuations through conditioned responses (or other preconscious processes)—neither the stimuli nor the emotions need to be overwhelming. This would be consistent with the fact that there were more patients with PNEs who endorsed the statement that they “frequently”, if not “always”, have PNE attacks “out of the blue” than there were who agreed that their seizures occurred in association with emotional stress [5]. Although attacks without an identifiable (e.g., stress-related) cause are often thought to reflect a denial of affect (supported by the generally avoidant coping strategies [71] and avoidance of emotional experience [72] of patients with PNEs), avoidance processes may or may not be relevant in any particular attack.

2. Conclusions and recommendations

Psychogenic nonepileptic seizures present a conundrum in many regards. Consciousness in PNEs presents an even greater conundrum, as patients appear to lose consciousness or at least experience reduced awareness of their surroundings and inner world, yet current investigational techniques have so far failed to reveal the underlying neurobiological mechanism for this. One way to reconcile the discrepancies between subjective reports and objective observations may involve the examination of emotion processing. This should include the study of sensory, situational, and emotional triggers of PNEs, emotional (and physiological) changes during the attacks, and premorbid (trait-based) styles of emotional reactivity and regulatory capacity. Neural mechanisms underlying these processes have begun to be explored in PNEs, and relevant models have been developed more fully in related states or conditions such as PTSD, dissociation and depersonalization, and borderline personality disorder.

We suggest that emotion processing, when empirically studied, offers an important perspective regarding PNEs and consciousness. Our interpretation of the available evidence suggests that conceptualizations attributing impairment of consciousness in all types of PNEs to the same kind of “dissociation” are likely to be too simplistic. We think it is likely that there are a number of different mechanisms (in isolation or combined) which may impair consciousness in PNEs. Firstly, consciousness may become impaired as a collateral effect of excessive inhibition of emotion processing—either in reaction to overwhelming emotions or as a separate mechanism (e.g., based on early learning from coping with traumatic experiences, biologically-based tendencies, or other processes). This mechanism would come closest to the conventional dissociative interpretation of PNEs. Secondly, PNE behaviors and experiences of reduced control/awareness may be the direct behavioral manifestation of overwhelming emotions (triggered by internal processes or external stimuli). Thirdly, minor emotional fluctuations or relatively neutral stimuli may trigger PNEs through conditioning or other preconscious processes. In addition, different problems of emotion processing may predominate in the same patient at different points of their disorder. Future studies exploring the neurobiological mechanisms underpinning PNEs are likely to be more fruitful if researchers bear in mind that it is unlikely that all PNEs result from the same processes in the brain.

Our current understanding of PNEs suggests that future research on consciousness in PNEs could benefit from focusing on emotion processing and its relationship to “interictal” patterns of neural activity in PNEs and on exploring the physiological underpinnings of emotion regulation in patients with other types of psychopathology (e.g., pathology involving the frontal–limbic regions or autonomic

nervous system, patients with PTSD or other dissociative disorders). We recommend adopting a nuanced approach to emotion that examines variations in both the type and intensity of emotions and goes beyond anxiety and threat to measure other specific negative emotions, self-conscious emotions, and positive emotions. A better understanding of the different types of emotion processing problems underpinning PNEs would not only be of academic interest but would also be likely to have direct relevance for the most appropriate management of individual patients.

Conflict of interest

The authors declare that there are no conflicts of interest.

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