Review Article

Dissociation Following Traumatic Stress

Etiology and Treatment

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Abstract. We postulate that the cascade "Freeze-Flight-Fight-Fright-Flag-Faint" is a coherent sequence of six fear responses that escalate as a function of defense possibilities and proximity to danger during life-threat. The actual sequence of trauma-related response dispositions acted out in an extremely dangerous situation therefore depends on the appraisal of the threat by the organism in relation to her/his own power to act (e.g., age and gender) as well as the perceived characteristics of threat and perpetrator. These reaction patterns provide optimal adaption for particular stages of imminence. Subsequent to the traumatic threats, portions of the experience may be replayed. The actual individual cascade of defense stages a survivor has gone through during the traumatic event will repeat itself every time the fear network, which has evolved peritraumatically, is activated again (i.e., through internal or external triggers or, e.g., during exposure therapy).When a parasympathetically dominated "shut-down" was the prominent peri-traumatic response during the traumatic incident, comparable dissociative responses may dominate responding to subsequently experienced threat and may also reappear when the traumatic memory is reactivated. Repeated experience of traumatic stress forms a fear network that can become pathologically detached from contextual cues such as time and location of the danger, a condition which manifests itself as posttraumatic stress disorder (PTSD). Intrusions, for example, can therefore be understood as repetitive displays of fragments of the event, which would then, depending on the dominant physiological response during the threat, elicit a corresponding combination of hyperarousal and dissociation. We suggest that trauma treatment must therefore differentiate between patients on two dimensions: those with peritraumatic sympathetic activation versus those who went down the whole defense cascade, which leads to parasympathetic dominance during the trauma and a corresponding replay of physiological and dissociative responding, when reminded. The differential management of dissociative stages ("fright" and "faint") has important treatment implications.

Keywords: dissociation, complex trauma, PTSD, sexual abuse, borderline personality disorder, tonic immobility, fainting

A coherent mental structure requires organized and interconnected representations of salient external and internal events, including sensory perceptions, affective and behavioral responding, and the conscious implications of a given context in terms of meaning (Marmar, Weiss, & Metzler, 1998; Van der Hart, Nijenhuis, Steele, & Brown, 2004; Schauer, Neuner, & Elbert, 2005). Experience of overwhelming threat may interfere with the process of integrating active elements of sensation, emotion, and cognition into the particular declarative memory of the event and thus result in disorders of the trauma spectrum (Brewin, 2001; Conway & Pleydell-Pearce, 2000; Ehlers & Clark, 2000; Elbert, Rockstroh, Kolassa, Schauer, & Neuner, 2006; Schauer et al., 2005; Van der Kolk, McFarlane, & Weisaeth, 1996). When later confronted with trauma reminders, survivors typically "replay" their original response of the traumatic event (e.g., Keane, Zimering, & Caddell, 1985). When a parasympathetically dominated "shut-down" was the prominent peritraumatic response to the traumatic incident, comparable dissociative responses may dominate responding to subsequently experienced threat and may also reappear when the traumatic memory is reactivated, such as during script-driven imagery (Lang, Bradley, & Cuthbert, 1998) or trauma-focused treatment (Schauer & Elbert, 2008). Strong dissociative reactions that may even include fainting obviously prevent the success of therapeutic measures that attempt to integrate the trauma memory into the autobiographic narrative and hence pose a serious obstacle to successful treatment of disorders of the trauma spectrum. Instead of trauma-focused therapy, these patients therefore typically receive skill-training, for example, how to identify and avoid potential triggers that induce detachment or how to end dissociative responding once it has been triggered. Current clinical practice adds to a varying degree elements from dialectic behavior therapy (Hunter et al., 2005; Linehan, 1993). Unfortunately, these strategies are not sufficient remedies for patients with trauma-related dissociative symptoms (Dyer, Priebe, Steil, Krüger, & Bohus, 2009) and clinical trials have identified dissociative symptoms as predictive for a negative treatment outcome (Spitzer, Barnow, Freyberger, & Grabe, 2007). This is not surprising, since dissociation prevents emotional processing and learning (Ebner-Priemer et al., 2009) due to the "shut-down" symptomatology typically characteristic of dissociative states (Simeon, Guralnik, Knutelska, Yehuda, & Schmeidler, 2003, p. 93).

The current concept of posttraumatic stress disorder (PTSD) does not distinguish whether the reminder of the traumatic experiences results in a fight-flight alarm response

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or in a dissociative block of sympathetic arousal with a subsequent excessive vagal tone. However, Foa, Riggs, and Gershuny (1995) noted that different PTSD symptoms such as intense hyperarousal versus numbing may represent distinct pathological processes and that grouping PTSD subjects with different symptom patterns in the same diagnostic category may interfere with our understanding and treatment of post-trauma psychopathology. Bremner et al. (1999) suggested that there might be two subtypes of trauma responses, one characterized by intrusive memories and hyperarousal and the other predominately dissociative. Results reviewed by Lanius, Bluhm, Lanius, and Pain (2006) indicate that comparable patterns may indeed be seen in the neuroimaging laboratory: Key brain areas involved in the hyperarousal response include the anterior cingulate and surrounding medial prefrontal cortex, the amygdala and possibly the thalamus, while higher-order sensory association cortices are active during dissociative responses. Based on our own observations (Kolassa & Elbert, 2007; Kolassa et al., 2007; Ray et al., 2006), we assume that these abnormal activation patterns in parietal and occipital cortex might be driven by abnormalities in the temporal cortex and possibly limbic structures. In a meta-analysis of psychophysiological findings in PTSD, Pole (2007) acknowledges the weak consistency of psychophysiological responding and suggests that greater consideration be given to subtyping of PTSD.

Understanding Dissociation Within an Evolutionary-Based Framework of Mental Disorders

Dissociation was introduced as a term into psychiatry already at the end of the 19th century (Janet, 1889; Putnam, 1989), however, a conclusive, coherent conceptualization has not yet been eved. Experts note that "conceptual clarity regarding trauma-related dissociation is urgently needed" (Van der Hart, Nijenhuis, Steele, & Brown, 2004). Despite the appreciated clinical significance of dissociation, there is an ongoing controversy about its theoretical foundation. It "lacks a single, coherent referent . . . that all investigators in the field embrace" (Cardena, 1994), and it still does today: "there is no consistent agreement about precisely what dissociation 'is' . . . dissociation represents a semantically open term leading to conceptual confusions which – in turn – might restrict its value" (Spitzer, Barnow, Freyberger, & Grabe, 2006). The field is obviously hampered by a missing etiological model (Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009). We propose that an evolutionary-based understanding of mental disorders (Brune, 2002; Jones & Blackshaw, 2000; Wakefield, 1992, 1999) permits to sort different theoretical approaches and thus facilitates the etiological modeling of such behavior. Such progress would especially be helpful for the development of effective therapeutic interventions; it would guide the therapist's measures when confronted with a client with the potential of severe dissociative responding, and it thus would overcome the dissociation-related obstacles during the course of trauma-focused therapy.

An evolutionary perspective suggests that dissociation, albeit hazarding the consequences of physical injury, is an adaptive, and when strike is close, final remaining survival response to specific types of life-threats that include nearness of a superior perpetrator or other situations dominated by helplessness. Dissociation enables survival in the following situations:

- when the organism is in direct and close encounter with a dangerous perpetrator, for example, when there is skin contact;
- in the presence of body fluids with danger of contamination, for example, blood or sperm;
- when bodily integrity is already injured, for example, invasion, penetration, sharp objects (e.g., teeth and knife) at the skin.

These situations require physiological adaptations, including immobility, pain tolerance and with it "switches" in consciousness, information processing, and behavior that are perceived as strange, because they are outside the range of ordinary experiences.

Evolution has equipped us with a defense armament to imminent threat. A coherent sequence of defense responses that escalate as a function of proximity to danger and threat has been established by evolutionary biology and psychophysiology alike. It seems that the defense process is a chain-linking sequence of steps that build on each other like a cascade. Obviously the animal and human organism is able to orchestrate its survival strategy according to the specific situation and along a staged process, called "defense cascade" (Lang, Davis, & Öhman, 2000). During life-threat, not every stage of the defense cascade is necessarily passed through by the individual. The actual sequence of trauma-related response dispositions carried out in an extremely dangerous situation is dynamic and depends on several factors. It is known that defensive reflex reactivity is organized sequentially to allow optimal responding depending on the proximity of the threat (Bradley, Codispoti, Cuthbert, & Lang, 2001; Fanselow & Lester, 1988) and with it its speed of approach. In addition, a particular response may be classically conditioned and thus depends on previous experiences with threatening events (Adenauer, Catani, Keil, Aichinger, & Neuner, 2009). Moreover, we propose that defense reactivity is organized to account for battlesomeness (chances to win a fight) of the threatened individual, that is, the appraisal of the threat by the organism in relation to its own power to counteract (age, gender, physical condition, defensive abilities, etc.) and, not least, for the threat-specifics (type of threat, type and speed of approach, context, threat involving blood loss, etc.). Whereas the dynamics of the defense cascade progresses in gradients of alternating ascending and descending activation, the various defense responses can be categorized into two general forms, namely active defense and immobility (Vila et al., 2007).

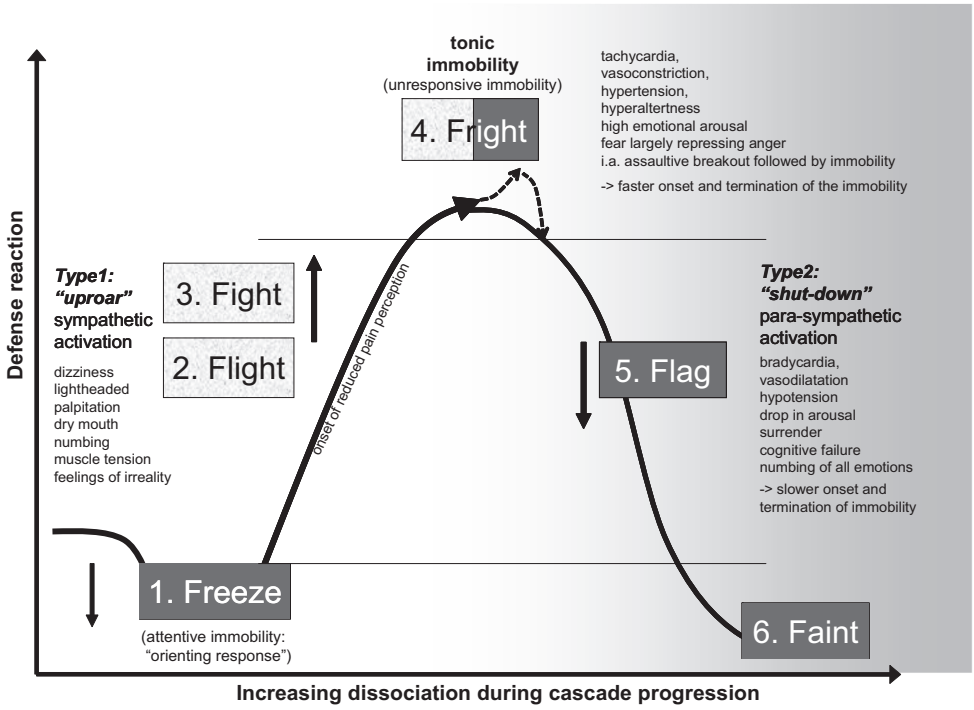

Figure 1. Schematic illustration of the defense cascade as it progresses along the 6-F course of action. The "uproar" sympathetic arousal reaches a maximum at the fright stage, eventually superseded by the onset of dissociative "shut down" (gray area).

The different suggestions for a defense cascade model consist of the following components: "freezing" in case of a large distance between subject and threat; "flight" when the distance is reduced; "fight" when the distance is eliminated; and "immobility" in case of prolonged elimination of distance (Ogden, Minton, & Pain, 2006). More recently, a model has integrated "faint" as another response possibility (Alboni, Alboni, & Bertorelle, 2008; Bracha, 2004; Lang et al., 2000; Moore & Amstey, 1962).

First, the freezing (orienting response – OR) facilitates a "stop-look-listen" perception of the threat. Pavlov (1927) referred to it as "Shto Eta/What is it?" – reflex that turns the sensory systems to the source of stimulation. This can be viewed as a collection of bodily responses that assist in processing the stimulus further, such as pupillary dilation, and a transient drop in heart rate. Then, having identified the danger and calculated the best means to escape, the mammal typically flees. If it is unable to successfully flee and the predator is upon it, it will initially fight. However, when there is clearly no chance of successfully fleeing or fighting, the animal may resort to immobility, in the hope that the predator will lose interest. Should the opportunity then present itself, the animal might make a final attempt to escape.

"Dissociation" as a defense disposition, however, has not been systematically included in the model of the defense cascade, although it accounts in large parts for an aggravated symptom spectrum of human survivors of life-threat and has immense treatment implications. Peri-traumatic dissociation is a set of subjective experiences, which includes alterations in perception of time, place, and self during and immediately after trauma exposure. Evidence has shown the predictive value of reported peritraumatic dissociation for the development of mental health problems including PTSD (Birmes et al., 2003; Bryant, 2007; Breh & Seidler, 2007), especially peritraumatic tonic immobility is a risk factor (Fusé, Forsyth, Marx, Gallup, & Weaver, 2007; Fiszman et al., 2008).

Our model wants to fill the missing link and suggests six defense responses, notably *Freeze, Flight, Fight, Fright, Flag*, and eventually *Faint*, whereby during the two Fs *Flight* and *Fight* bodily responses are mainly regulated via the sympathetic branch ("uproar reactions") and the following three Fs, the second half of the cascade, are dominated by parasympathetic arousal, determining the spectrum of dissociative responding ("shut-down" reactions: *Fright, Flag,* and *Faint*). We thus may arrange the stages of the defense cascade in form of an inverted u-shaped arousal function with the alarming flight-fight responses on the ascent of the curve and the set of dissociative variants on the descent (see Figure 1). In the next section, we discuss this evolutionary repertoire of armament step by step.

Stage 1 = *Freeze* (*Attentive Immobility*) – The OR

"Fear is often preceded by astonishment, and is so far akin to it, that both lead to the senses of sight and

hearing being instantly aroused. In both cases the eyes and mouth are widely opened, and the eyebrows raised. . . .” (Charles Darwin, *The Expression of the Emotions in Man and Animals*).

The fight or flight response is preceded by a response with a varying period of time known in ethology as freezing behavior or in psychophysiology as OR. It is characterized by information gathering (Bracha, 2004; Gray, 1987; Pavlov, 1927) and the activation of a set of bodily responses that assist in processing the stimulus and may prepare for further stages (Rockstroh et al., 1987), sometimes referred to as hypervigilance or hyperarousal: being on guard, watchful, alert, ready to respond. Contrary to the classic stress response model and in line with the defense cascade perspective (Adenauer et al., 2009), studies have consistently found that the initial reaction to aversive stimuli is characterized by a decrease rather than an increase of heart rate and is accompanied by an inhibition of the startle response (Bradley et al., 2001; Lang et al., 2000). This “fear bradycardia” (attentional or alerting response) includes motor inhibition, focused attention to the threat, and decelerating heart rate (Campbell, Wood, & McBride, 1997). A few seconds after the onset of a threatening stimulus, the direction of the physiological response reverses toward cardiac acceleration and an increase of the startle reflex. The transient pause before action mobilization is associated with heightened sensory perception and processing of contextual details and has been interpreted as the human counterpart of the freezing state in animals (Graham, 1979; Sokolov, 1963).

Ethological research has demonstrated that prey that remain “frozen” during potential threat are more likely to avoid detection because the visual cortex and the retina of mammalian carnivores primarily detect shapes through moving objects rather than color (Nesse, 1999; Page, 1994). This freezing might play a role in survival if it helps a hunted animal to blend in with its surroundings by remaining as motionless as an inanimate object, thereby allowing the shift of attention of predators to other moving or noisy stimuli in the neighborhood. Interestingly, in human subjects, who have survived several traumatic events and suffer from PTSD with hyperarousal as a consequence, the OR is passed by in the defense cascade (Adenauer et al., 2009) and the alarm response is immediately initiated, that is, the defense cascade is adjusted to allow for a rapid flight response to threatening cues without any further exploration of the stimulus. This finding is consistent with the observation that PTSD patients react to threatening cues and trauma reminders with a hypersensitive alarm system (Rauch et al., 2000). In contrast to fright, as a measure to eliminate aggression and prevent attack, “freezing” actually means the initial orienting response at the beginning of the defense cascade, which is important to achieve concentrated attentiveness to focus on the signs of threat. This attentive immobility is primarily a transient defense strategy, preliminary to fighting or fleeing, in which the animal stops moving to avoid detection and shifts resources to perceptual and executive functions to better locate the predator and plan escape.

Stages 2 and 3 = *Flight* and *Fight*: Toward the Arousal Peak of the Alarm Response

“. . . The heart beats quickly and violently, so that it palpitates or knocks against the ribs . . .. That the skin is much affected under the sense of great fear, we see in the marvellous manner in which perspiration immediately exudes from it . . .. The hairs also on the skin stand erect; and the superficial muscles shiver. In connection with the disturbed action of the heart, the breathing is hurried. The salivary glands act imperfectly; the mouth becomes dry, and is often opened and shut . . .” (Charles Darwin, *The Expression of the Emotions in Man and Animals*).

The long known “fight or flight” response, also called the “acute stress response,” physiologically detailed by Walter Cannon in 1929 as a theory suggests that animals react to threat with a general discharge of the sympathetic nervous system, and has subsequently been advanced by evolutionary psychologists (Bracha, 2004). If a stimulus is perceived as a major threat, a more intense and prolonged discharge of the locus coeruleus activates the sympathetic division of the autonomic nervous system (ANS). The initial OR is followed by attempts to flee, and, if not successful, by attempts to fight. The organism becomes extremely aroused. An “alarm response” is initiated, with sympathetic arousal and sympathetically modulated adrenal release that enables the organism to counterstrike (heart rate acceleration, blood pressure elevation, and vasoconstriction). At the central level endorphins are released to reduce pain and with it somatosensory perception and awareness is dampened.

The activation of the sympathetic branch at this point enables a reorganization of blood supply (vasoconstriction of peripheral vessels – cold skin, dilatation of certain organ supply vessels) in order to increase blood flow to the heart and muscles while decreasing the likelihood of blood loss after injury through peripheral vasoconstriction. Blood pressure is rising through increased cardiac output. Preparedness for aggressive engagement increases. Faster and deeper breath facilitates increased oxygenation of important organs and muscles and increased perspiration can cool the body. Moist palms at the same time allow a better grip to flee, lowering the chances for injury. Activity not acutely needed is halted, for example, digestion reduces, bowel mobility halts to a point of emptying out.

At the height of arousal, the adaptivity to flee or fight begins to turn: General fear symptoms are experienced, including dizziness, nausea, palpitation, drowsiness, lightheadedness, tension, blurred vision, feelings of irreality, numbing, and tingling appear (prodromal period before “shut-down”/pre-syncope; see European Society of Cardiology guidelines, 2009). At the extreme apex, when the organism experiences a climaxing activation, parasympathetic

activation sets in. The acute response to extreme stress becomes a peritraumatic "panic-like" dual autonomic activation. Stage 4 is reached, which we detail below. Moreover, a "*shut-down*" reaction and with it the various forms of dissociation appear. At this stage, sensation, perception, motor abilities, and speech behavior are dramatically altered. Before we continue in the characterization of the stages, we therefore discuss principles of the shut-down.

The Onset of Dissociative Shut-Down: Functional Sensory Deafferentation, Motor Paralysis, and Loss of Language Functions

DSM-IV characterizes dissociation as disruption of the usually integrated functions of consciousness, memory, identity, or perception of the environment. ICD-10 acknowledges that it may also involve the sensory and motor systems, leading to symptoms, which are subsumed under the term of conversion: "partial or complete loss of the normal integration, … immediate sensations, and control of bodily movements".[1] In general, dissociation can be understood in three distinct ways: (1) as a lack of integration of mental modules or systems, (2) as an altered state of consciousness, and (3) as defense mechanism. All of these are partial or even complete failures to deliberately control processes and take actions that can normally be influenced by an act of volition, for example, the ability to bring accessible information into conscious awareness or move voluntary muscles. Furthermore, memory retrieval deficits appear (e.g., amnesia, conversion, somatoform disorders, "detachment," out-of-body/depersonalization, and derealization) as subjective experiences of an altered state of consciousness characterized by alienation of oneself or the external world, with a flattening of emotional experiences (Allen, 2001; Holmes et al., 2005). It is well known that dissociation involves dysregulation of the visual and somatosensory modalities (Bernstein and Putnam, 1986; Bremner et al., 1998). Nijenhuis et al. have also found a loss of normal integration of somatoform components of experience, bodily reactions and functions (Nijenhuis, Spinhoven, Vanderlinden, Van Dyck, & Van der Hart, 1998). The fact that dissociative symptoms also pertain to functions of movement, sensation, and perception has been largely overlooked for quite some time (Van der Hart et al., 2004). Previously, this was called "deficit symptom": loss of somatic function that has no evident organic basis, for example, numbness, or loss of sensory function like "hysterical blindness" (Nijenhuis, Van Engen, Kusters, & Van der Hart, 2001; Nijenhuis et al., 1998). Accordingly, we have found that in stages of shut-down as well as during later reappearance of dissociative states, reality construction becomes more difficult. These typical behavioral features include that threatened individuals cannot hear, see, or perceive well any more. They move less – obviously due to excessive muscle tension – until motility stops. Furthermore, they have difficulties to understand language and are unable to produce speech. Functional interruption of afferent and efferent nerve impulses is taking place. Thoughts and reasoning no longer make sense. The mind is confused. Time perception changes dramatically.

To summarize, we postulate that dissociation is adaptive and manifests itself behaviorally on three dominant levels as life-threat escalates: (1) rise of and finally complete functional sensory deafferentation, (2) decline of and finally absence of efferent motor commands, and (3) decline of and finally absence of speech perception and production. If motionlessness of a human organism is of crucial importance in such moments of life-threat, nature needs to take care of systems that might corrupt adaptive behavior – sensation, movement, and speech. As long as the victim can feel pain and anger and is able to act, he or she will attempt to move away from the aversive stimulus or fight it off. When the organism is about to be attacked, immobility tends to be combined with analgesia, which is functional in that perception of pain would divert attention of the prey from defensive concerns (Bolles & Fanselow, 1980). Furthermore, tonic immobility is combined with numbing for anger affect, whilst at the same time fear emotions reach their maximum. In order to enable a maximal defensive and "dead" appearance ("as if dead," "playing possum"), which provides survival advantage by complete giving in, and cessation of fighting, and moving, perceptions and later emotions need to be switched off or deactivated. To guarantee motionlessness in these highly perilous situations, the organism should be unable and unwilling to use voluntary muscles and should feel neither anger nor pain, be finally emotionally numb, as if anesthetized. During this cascade numbing of sensations, analgesia and anger inhibition are followed, at a later stage, by numbing of fear as well. These effects inhibit reactions, which would compromise optimal protection in this stage of imminence. For example, grooming and licking wounds would attract attention and would elicit further attack (Siegfried, Frischknecht, & Nunez de Souza, 1990). It is biologically important at that point not to show any recuperative behavior or to care for injuries. This is only possible through reduction of panic and fear (Siegfried et al., 1990; Van der Kolk, 1994), suppression and delay of emotional and panic escape behavior, and inhibition of the production of sounds (Kalin, 1993; Kalin & Shelton, 1989). Suarez and Gallup describe an inability to call out and scream as a consequence of suppressed vocal behavior (Suarez & Gallup, 1979). The loss of capacity to communicate, sometimes described as mutism (Porges, 1995), or "*et vox faucibus hæsit*" (and my voice stuck in my throat) as Vergil has put it, seems adaptive at this stage. Whereas movement and vocalization must be put on complete hold during contact with the offender, on termination of threat imminence, pain perception returns and instigates

[1] World Health Organization. The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines. Geneva: World Health Organization, 1991.

recuperative behavior (Bolles & Fanselow, 1980), including behaviors directed at injuries, grooming behavior, and resting behavior serving to promote healing. The recuperative stage may be considered mainly as a response to nociceptive stimulation arising from injury and tissue damage. While stimuli predicting aversive events come to elicit analgesia, the presentation of a safety signal produced an abrupt change in pain sensitivity, essentially "switching off" conditioned analgesia (Wiertelak, Watkins, & Maier, 1992). The finding that (conditioned) safety signals produce acute anti-analgesic effects in animals is supportive of this view (Fanselow & Lester, 1988). Just as (conditioned) signals for danger elicit analgesia, (conditioned) signals for safety inhibit (conditioned) analgesia. Nociception, subsequently, evokes recuperative behavior.

The thalamus is the principal synaptic relay for information reaching the cortex (Kandel & Schwartz, 1991) and thus the gateway to awareness. All sensory information, except for olfaction, is routed through the thalamus to the cerebral cortex. The thalamus thus plays a major role in regulating arousal, the level of awareness, activity, and consciousness. The adaptive response at this stage, namely *functional sensory deafferentation* on the thalamic level, enables to turn or shut down vision, audition, and somatosensory and proprioceptive information. Changes in hearing, seeing, feeling occur. The thalamus has also been suggested to be involved in mediating the interaction between attention and arousal (Portas et al., 1998), both of which are relevant to the phenomenology of traumatic stress syndromes. Thalamic dysfunction in PTSD has previously been shown by Bremner et al. (1999) and Liberzon, Taylor, Fig, and Koeppe (1996/1997), and also in the laboratory of Lanius et al. (2001, 2003). High levels of arousal during traumatic experiences have been hypothesized to lead to altered thalamic sensory processing (Krystal, Bennett, Bremner, Southwick, & Charney, 1995), which in turn results in a disruption of transmission of sensory information to the cortex, cingulate gyrus, amygdala, and hippocampus. Hence, Krystal, Bremner, Southwick, and Charney (1998) have suggested that this mechanism may enable dissociative symptoms. It is interesting to note that in contrast to those with dissociative responses, persons who showed flashback/reliving/hyperarousal responses exhibited significantly less activation of the thalamus when confronted with trauma scripts (Lanius et al., 2003). Survivors of traumatic events who have experienced peritraumatic dissociative shut-down (e.g., skin contact with offender, presence of contaminating body fluids, and body penetration) often do not report immediate intrusive reexperiencing after the abuse; the emergence of involuntary sensory information about the events can be delayed for years or decades. In the study of Lanius et al. (2002) PTSD subjects who experienced dissociative responses to traumatic script-driven imagery reported that "I was looking down at myself from above," "I was detached from my body," "I was completely zoned out and floating," or "I was emotionless" during the script-driven imagery procedure. All PTSD patients included in this study had chronic histories of emotional, physical, and sexual abuse beginning in childhood, often continuing to the time of investigation. They reported that dissociation had been a defense they had used throughout their lives to escape overwhelming experiences. The lack of autonomic response often observed in PTSD patients during dissociative states is consistent with previous findings on depersonalization responses. Patients even showed a decrease in heart rate when they became depersonalized (Kelly & Walter, 1968; Lader, 1975; Lader & Wing, 1966; Sierra et al., 2002).

In addition, peritraumatically, during life-threat, the organism becomes insensitive to internal or external stimuli, that is, emotions (first anger, and only later fear) and pain. It is not just touch-insensitivity, but reduced nociception (analgesia) and numbing of feelings. A large majority of Albach's (1993) subjects who reported childhood sexual abuse experienced analgesia and kinesthetic anesthesia (insensitivity for touch). In fact, analgesia was the most commonly reported "hysterical" symptom. Laboratory studies with nociceptive stimulation found elevated pain thresholds in dissociative patients (Ludäscher et al., 2007; Schmahl et al., 2006) and demonstrated fear-induced hypoalgesia (Rhudy, Grimes, & Meagher, 2004). Reduced pain sensitivity under conditions of high stress can be central for adaptation and survival (Mayer & Fanselow, 2003; Millan, 2002). There is ample anecdotal evidence from soldiers or survivors of disasters that they could still run and take survival actions, although they were severely injured, bleeding, etc. Both pathways of pain processing seem to be progressively shut down along the defense cascade: the sensory-discriminative and the affective-motivational component (Schmahl et al., 2006). During life-threat victims do not feel pain and, probably most important, it does not matter to them. This rising cognitive, bodily, emotional indifference for the self, when being driven through the defense cascade, is the requirement to ensure survival. When the victim is about to be attacked, analgesia and numbing is functional: Perception of pain (nociception) or fear should not initiate recuperative behavior or draw attention and lead to defensive behavior.

Stage 4 = *Fright* (*Tonic Immobility – Unresponsive Immobility*) – Inhibition of Aggression Through Muscle Restraining When Being Overwhelmed by Threat

> "The frightened man stands like a statue motionless … the heart beats quickly and violently, … for the skin instantly becomes pale, as during incipient faintness. This paleness of the surface however, is probably in large part, or exclusively, due to the vasomotor centre being affected in such a manner as to cause the contraction of the small arteries of the skin … and the superficial muscles shiver … the breathing is hurried … the mouth becomes dry … one of the bestmarked symptoms is the trembling of all the muscles of the body … " (Charles Darwin, *The Expression of the Emotions in Man and Animals*).

Fright can be understood as the turning point at which the change from the rising to the falling phase occurs, being essentially uninterrupted as it proceeds into the faint. The imminent stage is characterized by coactivation of the sympathetic and parasympathetic system (Löw, Lang, Smith, & Bradley, 2008; Ogden et al., 2006). The high dual autonomic tone is known as a risk for sudden cardiac death (Skinner, 1988, 1985).

The heightened muscle tonus, which enables better action performance, causes the muscles to be overly tense beyond a certain threshold, as well as rigid, and movements to become slow and difficult. At this stage overt behavioral actions are not an option; skeletal muscles tense to a stage of tonic immobility. "Playing dead" in the early ethological literature, or the technical term "fright," best captures the Kraepelinean concept of "*Schreck*" as in "*Schreckneurosen*" (1896). Tonic immobility has also been used to describe the paralysis, which often immobilizes animals such as rodents or birds and a similar state is known from invertebrates when they feel threatened by a predator. The immobility of the organism during the fright stage reminds the observer of spastic paralysis. It looks as if the organism is "frozen like ice" (temporary gross motor inhibition) because of the stiffness involved. This metaphor is used in much of the trauma literature referring to "freezing" as immobility of the body. (Note that the term "*freezing*" in this context should be avoided, as it is already reserved for the OR; see above). In tonic immobility, the organism is emotionally aroused and full of fear, yet unresponsive to even painful stimulation and the posture, which may be held for long periods of time, often appears bizarre (Gallup & Rager, 1996; Ratner, 1967). Memory functions are mainly intact since learning is not disrupted (Gallup, Boren, Suarez, Wallnau, & Gagliardi, 1980) at this stage. While the efferent pathways are already inhibited, the afferent pathways and central processing capabilities remain still intact until a later stage. Tonic immobility or "unresponsive immobility" has been defined by Marks (1987) as "the sudden onset of prolonged stillness and decreased responsivity in a previously active animal in the face of threatening stimulation" (p. 60). The organism appears dead or unresponsive to exteroceptive stimuli, internally the animal is highly alert (Marx, Forsyth, Gallup, Fusé, & Lexington, 2008). This unresponsive immobility is elicited most easily during intense fear and some form of physical restraint or perception of entrapment (Marx et al., 2008; Moskowitz, 2004). In human beings, tonic immobility can be understood as a defensive strategy, developed during an extended period of evolution in which human beings had to face predators in much the same way many animals do today, and designed to maximize the individual's chances of surviving a potentially lethal attack, for example a murderous killing. An immobile prey animal is less likely to be killed and eaten (Marx et al., 2008). Tonic immobility or "thanatosis" is a process by which mammals feign death in order to evade unwelcome attention (Miyatake et al., 2004; Pasteur, 1982). Such catatonia can be understood as an evolutionary-based fear response (Moskowitz, 2004), with features such as immobility, decreased vocalization, analgesia, "waxy flexibility," and evidence of alertness as well as the fixed eye gaze (Dixon, 1998), reducing visual input from the offender and thereby decreasing distress and arousal. At a later stage, a "waxy flexibility" is present.

Thus, the core catatonic symptoms of stupor, mutism, and immobility are directly linked to tonic immobility. In the past, this was for a long time seen as a consequence of "very severe physical or mental stress . . . [such as] a very terrifying experience" (Kahlbaum, 1874/1973, p. 31). The patient remains entirely motionless, without speaking, devoid of any will to move or react to any stimuli. The general impression conveyed by such patients is one of profound mental anguish or an immobility induced by severe mental shock. All in all, these patients give the impression of the deepest mental pain, of being paralyzed after a great fright. Finally, Krystal (1993) argued that catalepsy or catatanoid reactions seen in human beings under conditions of profound helplessness were akin to animal immobility, both of which he characterized as submissive responses in the face of unavoidable danger.

The lack of movement in unresponsive immobility has a fast on and off switch, but can last from a few seconds to many hours (Gallup & Maser, 1977) depending on the species and the situation. Marks (1987, pp. 68–69) conveys that during extreme fear, human beings may be "scared stiff" or "frozen with fear." A paralyzed conscious state with abrupt onset and termination is reported from survivors of attacks by wild animals, by shell-shocked soldiers, and by rape victims (Rocha-Rego et al., 2008; Suarez & Gallup, 1979). Rape-induced tonic immobility was first noted by Suarez and Gallup: inability to move, inability to call out or scream, no loss of consciousness/recall of details of the attack, apparent analgesia, that is, numbness and insensitivity to pain. The researchers noted as well a sensation of feeling cold and shivering. We assign the feeling of cold and trembling to the consequences of adrenaline and vasodilatation whereby body warmth is lost fast and leads to shivering (medical "shock"). Tonic immobility guarantees negative or quiescent behavior even in the presence of massive aversive stimulation (Foley, 1938), a stilled organism that makes no attempt to struggle for freedom or fight. Fear plus the physical prevention of flight seem to bring on the response named catatonic trance, action inhibition, or "death feigning," which appears suddenly (Moore & Amstey, 1962; Pavlov, 1923).

In the PTSD literature tonic immobility has been referred to as peritraumatic panic-like symptoms. The peritraumatic panic reaches its maximum in the fright response, which may enhance survival when there is no longer a perceived possibility of escaping or winning a fight or when there is direct physical contact with the perpetrator (Bracha, Williams, Ralston, Bracha, & Matsukawa, 2004). The clinical relevance of immobility as a survival response may best be illustrated in relation to the behavior of certain victims of violence or sexual assault who exhibit extreme passivity during the assault. Here again, an understanding of the genetically prepared nature of the response might help to ameliorate this dimension of the painful memories that plague some victims who wonder why they did not put up more of a fight. For a long time clinicians, researchers, and legal advocates wondered why only a minority

of battered women who report childhood sexual and physical abuse actively resisted the perpetrator (Albach, 1993; Draijer, 1990). Barlow (Barlow, 2004; Burgess & Holmstrom, 1974, 1976) argues that immobility behavior had been overlooked since motor inhibition as evidenced in animals had not been thought to occur in human beings. But since the 1970s the parallel to animal immobility behavior became obvious. For example, Suarez and Gallup (1979) described similarities between tonic immobility and rape-induced paralysis as follows: Changes in body temperature, numbness, analgesia, suppressed vocal behavior, and vivid recall of details. In a later stage another form of immobility (flaccidity) will dominate (see below).

Since tonic immobility is elicited by extreme fear and helplessness in animals and human beings, younger and weaker organisms (i.e., children and women) will show this type of reaction with higher likelihood and frequency compared to adults or males. Tonic immobility is most useful when a slow-moving vulnerable organism (like the opossum) is confronted with a life-threatening situation involving mobile, large predators (Nesse, 1999). On the other hand, proximity of danger plays a critical role for the onset of immobility. In primates, man included, "fright" with tonic immobility is known to occur on seeing conspecifics that are being threatened, frightened, or mutilated, as a cue for live threat in the surroundings (Azevedo et al., 2005; Facchinetti, Imbiriba, Azevedo, Vargas, & Volchan, 2006; Hebb, 1946). We have many reports of child soldiers showing tonic immobility (not flaccid immobility) when witnessing man slaughter. As accurately expressed in Goethe's *Faust* however, "blood is a juice of very special kind." From our recent data we conclude that opposite to females it can act as a rewarding cue in male human hunters (Elbert & Weierstall, in press). In a fighter, fainting would be fatal or result in a drop in social hierarchy as key fitness indicator. In noncombatants, however, fainting in response to the sight or smell of blood may have evolved as an adaptive stress response that aided survival of victims during combat. For a noncombatant woman or preadolescent child, however, an approaching sharp object, experiencing skin penetration, or spilled fresh blood may be a crucial turning point from which onwards continued fight with its elevated sympathetic arousal may be an ineffective survival strategy, as opposed to fainting (Bienvenu & Eaton, 1998; Salazar, 2000). Epidemiological data support this view as syncopal response to blood, injection, and injury is significantly less common among adult men, with puberty as a turning point (Agras, Sylvester, & Oliveau, 1969; Costello, 1982; Trouern-Trend, Cable, Badon, Newman, & Popovsky, 1999). Tonic immobility is almost always displayed when the person is overwhelmed by threat and not allowed and not able to act aggressively against the threat. Thus immobility functions to supress anger in the victim and acts bidirectionally to inhibit aggression in a number of ways:

a) "Submission" after other strategies (like escaping, screaming, and fighting back) have failed in order to stop aggression (Marx et al., 2008): for example, a toddler shows a fright reaction as the adult displays rising aggression.
b) "Holding still" to reduce risk of tissue damage: for example, a penetrated rape victim or a victim having sharp objects touching the surface of the skin. When the skin is contacted by sharp objects (a knife, teeth) or penetrated at an orifice of the body, heavy movements, struggling, or pulling away with a sudden move will cause even more tissue damage and damage to mucous membranes, as well as deeper wounds. A more recent study showed that childhood sexual abuse involving attempted or completed penile/vaginal penetration was more likely to be associated with increased experiencing of tonic immobility, and reports of tonic immobility were associated with greater current psychological impairment (Heidt, Marx, & Forsyth, 2005).
c) "Eliminating cues" for predatory behavior: stiffening of the voluntary muscles can occur during direct physical contact with the carnivore or the human predator. Tonic immobility increases survival chances even when physical contact has been made, because movement cues are critical releasing stimuli for predatory behavior, and immobility eliminates these cues. Surviving through "motionless pretending to be dead" has its evolutionary roots as a defense mechanism against predators. Predation tests demonstrated that prey, which struggles or moves when attacked, is killed and eaten, but if prey remains immobile, feigning death, the predator loses interest and the prey survives (Miyatake et al., 2004).
d) "Escape preparedness" in case there is a chance to resume flight: captured prey that becomes tonically immobile rather than struggling and fighting may increase its chance of escaping when the predator temporarily loosens the grip under the assumption that its prey is indeed dead. In mammals, struggling prey is only held on to until resistance subsides. It is also a response that may be adaptive in human beings when there is no possibility to escape or win a fight (Perry, Pollard, Blakely, Baker, & Vigilante, 1995) and this behavior is in fact recommended by park authorities for encounters with a grizzly bear. An organism in tonic immobility is immobile but is markedly tachycardic, vasoconstricted, hyperalert, and prepared to flee in a moment of opportunity, a state that also characterizes human catatonia. It is often followed by assaultive behavior, at times directly out of a stuporous state (Fink & Taylor, 2003).

When the aggressor approaches rapidly and comes close, the organism can for a short time again dramatically change its behavior (Fanselow & Lester, 1988) in that it suddenly displays an explosive escape response (Hirsch & Bolles, 1980) in conjunction with aggressive behavior (Kalin, 1993). This fact demonstrates that high aggressive arousal (anger) is inhibited at that stage but present. If these explosive responses do not eliminate contact, immobility may return, reducing the likelihood of continued attack (Fanselow & Lester, 1988). This assaultiveness, well known

in catatonia of human beings, is only present during catatonic states of immobility. Further on in the cascade, once the vagal tone has increased and tonic immobility has changed to flaccid immobility (see next chapter), the ability of acting out is no longer present.

A person's sensory processing (sight, hearing, smell, taste, and touch), of kinesthetic (perception of movement and muscular sense) and somesthetic (sensory data derived from skin, muscles, and body organs) stimuli normally continuously serves the perceiving self as evidence that it resides "in" the physical body: Somatic processing ordinarily is integrated with a sense of *self*. Some patients describe that they leave the body during the traumatic event. Out-of-body experiences (depersonalization) seem to be a dissociation between sensory processing of somatic events and the sense of self or identity. If all sensory and physiological information, emotions, and pain perceptions are shut down at a certain stage of the defense cascade and if there is no longer the usual continuous feedback, the self inevitably must interpret its own physical position as outside of the body.

There have been discussions about the question whether dissociation exists because biology has equipped us with the gift of a gracious death. However, "good dying" does not underlie selection pressure in evolution. Those who have the ability to die with ease do not pass on this attribute to their offspring through natural selection. On the contrary, the shut-down reaction is solely a matter of surviving and it is important to look at its survival indicators to come up with an evidence-based model for its treatment.

Stages 5 and 6 = *Flag* and *Faint*: Facing Contamination and Penetration/Invasion May Result in *Flaccid Immobility* (*Unresponsive Immobility*) or Even *Fainting*

Stage 5 of the defense armament encompasses forms of dissociation and is characterized by reduced sympathetic arousal and passivity or a "shut-down" peripherally dominated by vagal activity. The survival value of these massive threat-induced changes in CNS and ANS regulatory control as well as its evolutionary roots are still under debate. Some have argued that threat-induced flaccid immobility with its vasovagal shut-down may be a uniquely human response that only evolved during middle paleolithic conspecific violence (Bracha, 2004) while others note that it involves similar physiological mechanisms as in other vertebrates, pointing to a much earlier and common evolutionary root in mammals (Alboni et al., 2008), the "alarm bradycardia" as a decrease in heart rate during fear-induced immobility. Probably, both views hold parts of the truth: during a long history of violent intraspecific attacks and violence, evolution will have reshaped the pan-mammalian defense repertoire specifically in homo sapiens.

Threat-induced flaccid immobility up to fainting, a response also seen in blood-injection-injury phobia, could have evolved as a guard against the danger of cardiac failure during inescapable attacks. The inhibition of the sympathetic system in conjunction with a marked activation of the parasympathetic system will lead to vasodilation and an extraordinary drop in heart rate and blood pressure. A satisfactory theory for this vasovagal syncope, however, is still absent (Bizios & Sheldon, 2009; Van Dijk & Sheldon, 2008). A widely held misbelief regarding bloodletting-related fainting assumes that fainting increases the probability of survival through minimizing blood loss. This neglects the fact that the major loss of blood in survivors results from venous injury and is thus unaffected by pressure in the arteries. On the contrary, vasodilation will lead to a greater loss of blood in response to injury than during the Flight-Fight stages. Vasoconstriction and tachycardia rather than vasodilatation and bradycardia are the adaptive initial responses to blood loss (Bracha, Williams, Haynes, et al., 2004).

Vasovagal reactions are known to be caused by excess vagal tone and decreased sympathetic skeletal muscle response to central nervous system stimulation (for example, fear), with the outcome of a hypotensive-bradycardic reflex syndrome: Inadequate vasoconstriction with an abrupt drop in systolic blood pressure, followed shortly by profound bradycardia and a decrease in cardiac output (Marchiondo, 2010). Threat-induced flaccid immobility up to fainting, a response also seen in blood-injection-injury phobia, could therefore have evolved as a guard against the danger of cardiac failure during inescapable attacks to reduce myocardial oxygen consumption when cardiac strain is (expected to be) excessive ("heart defense hypothesis"; Alboni et al., 2008). In humans, hypotension and bradycardia are finally responsible for loss of consciousness as a result of global cerebral hypoperfusion (Brignole et al., 2004). A horizontal position secures blood supply to the brain.

Fainting seems to be mediated through disgust (Curtis & Biran, 2001), which enables emotional responses to potentially infectious or noxious material in advance of actual contact with such material, to avoid pathogens and their toxins or invasive procedures (Marchiondo, 2010). Across cultures, disgust is universally elicited by disease-salient contacts such as bodily secretions, viscous substances, vermin, and sick or dirty people (Curtis, Aunger, & Rabie, 2004). Experiencing, witnessing, or listening to situations perceived as "disgusting" (e.g., mucus/stool, suctioning, wounds, colostomies) can trigger vasovagal reactions (Marchiondo, 2010). Even chimpanzees avoid faeces and show disgust reactions (Goodall, 1976). Vasovagal syncopes as a consequence of strong emotional stimuli such as fear, pain, and disgust are a common problem in emergency aid and critical care (Dennin & Haupt, 2009). The finding that blood-injection-injury phobics as well as healthy people may faint in the presence of blood or injury (e.g., Kleinknecht, Thorndike, & Walls, 1996; Kroeger, Atkinson, Marcuse, & Pickering, 2006) also points to the possible involvement of disgust, as strong and unopposed (parasympathetic) disgust reactions may promote fainting. Indeed, vasovagal syncopes, triggered by "blood" as a cue, are controlled by the same pan-mammalian physiological mechanism that regulates disgust (Marks, 1988; Page, 1994). Sexual violence and forced penetration provoke an intense

disgust reaction, as does the sight of blood (Berntson, Cacioppo, Quigley, & Fabro, 1994; Merckelbach, Muris, de Jong, & de Jongh, 1999). Disgust and the associated phenomena of nausea and vomiting are thought to have evolved to prevent physical contact with disgusting stimuli (Cisler, Olantunji, & Lohr, 2008) and protect omnivores from the risk of ingesting pathogen-laden food. Spontaneous vomiting is seen as an automatic reflex of involuntarily rejecting material that is disgusting to see, taste, smell, or feel around orifices of the body. Withdrawal and avoidance are likely to increase survival. Page (1994) noted that "when the (parasympathetic) processes underlying disgust combine with a homeostatic increase in parasympathetic activity (which counteracts the initial sympathetically mediated fight or flight response), the joint effect may produce a pattern of vascular and vagal responding responsible for fainting" (p. 452). People with blood-injection-injury phobia exhibit a unique biphasic response pattern of typical sympathetic followed by massive parasympathetic activation. This biphasic process may account for the fainting that is observed in up to 75% of people with this condition when they are exposed to phobic stimuli. Disgust has become implicated in the vasovagal syncope response because it is associated with parasympathetic activity and reductions in diastolic blood pressure leading to fainting sensations (Page, 2003). Although the disgust-fainting relationship in blood-injection-injury phobia has not been a consistent finding (Olatunji et al., 2006), there is consistent evidence that people with this phobia report more intense disgust than controls.

While a trauma reminder may produce strong post-traumatic anxiety and arousal in a patient who had responded with flight/fight during the traumatic stressor, a patient with flag conditioning would be highly unresponsive and disengaged. In contrast to tonic immobility, flaccid immobility has a slow onset and slow termination. Only over the course of several minutes dissociative reactions begin to dominate (an abrupt onset is observable only, once once the response is conditioned). The muscle stiffening changes to flaccidity, voluntary movements stop, so does speech. Even if there is effort from the survivor, language production fails; the mouth might open, but there is no vocal sound. The central skeletal muscles are flagging, so that the posture subsides. The patient reports that perception of internal or external stimuli becomes attenuated, sounds and voices become distant, visual stimuli fade or become unreal (derealization). Kinesthetic, somesthetic, nociceptive stimuli no longer seem to reach the central processing units, causing changes in body awareness and loss of control (depersonalization). Numbness prevails. During reactivation of flaccid immobility, there is often a more or less intense feeling of nausea present as well. Conscious processing of the events becomes limited, making meaning seems irrelevant. The crucial difference between flaccid and tonic immobility (or catatonic states) is, that in a more progressed flag state, emotional involvement fades away, that is, no action dispositions are assembled and memory consolidation becomes weak and later rehearsal more difficult. It may require minutes to hours for a patient to be oriented in reality.

Reenactment of the Defense Stages When the Fear Network Is Triggered Due to Reminders or During Exposure Therapy

Learning associations between cues in the environment gives organisms the ability to predict impending danger. The last few decades of research have established that these stimulus-danger or fear associations are formed in the amygdala (Schiller & Johansen, 2009). Repeated experience of traumatic stress forms a fear network (see Figure 2) that can become pathologically detached from contextual cues such as time and location of the danger, a condition, which manifests itself as PTSD (Brewin, 2001; Elbert & Schauer, 2002; Schauer et al., 2005). Thus, survivors of life-threatening events with PTSD become "stuck" in the trauma. Intrusions

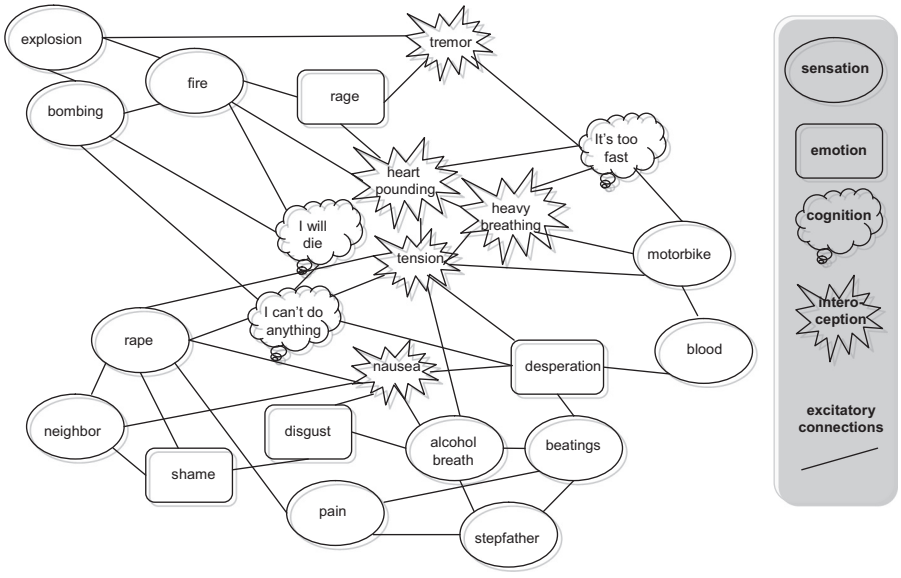

Figure 2. Illustration of a fear network in response to multiple traumatic stressors.

can therefore be understood as repetitive displays of parts/fragments of the events, which then elicit a corresponding response set that can be dominated by hyperarousal (Stages 2 and 3) or by dissociation (Stages 5 and 6), depending on the history of conditioning of physiological responses during the actual life-threatening experiences in the past.

Trauma-related emotional experiences are coded in memory as networks of mutually exciting chunks or information units. When one or several units are activated by cues, the activity spreads to adjacent units. Depending on the strength of activation, the entire structure may be engaged. The post-traumatic "fear network" may also be activated through narrative techniques, for example, when the survivor reports parts of his or her experiences. This will also activate the related support physiology of behavioral responses that have been engaged during the traumatic experience (Lang, 1979, 1984; Lang et al., 2000). Hence, we postulate that any activation of the trauma-related fear network will facilitate action replay of the defense behaviors the survivor has undergone peritraumatically. Repeated experience of traumatic stress may therefore produce at least two major subtypes of clinical symptom profiles, observable in the physiological responding during post-trauma exposure (Schauer & Elbert, 2008): Patients who experienced sympathetic activation (only) in response to the stressor show few signs of (shut-down) dissociation and passive avoidance during exposure treatment (Type 1), whereas those who went down the entire 6-F defense cascade, ending in parasympathetic dominance during the trauma, produce a corresponding replay of physiological responding when reminded by a cue (Type 2). The actual individual cascade of defense stages a survivor has gone through during the traumatic event will repeat itself every time the fear network – which has evolved peritraumatically – is activated again (i.e., through internal or external triggers or during exposure therapy). In relation to Figure 1, the two PTSD types may therefore be placed on the upstroke (uproar) (Type 1) or the downfall (shut-down) (Type 2) of the curve. A third type, positioned at the top, would manifest itself clinically among other symptoms in catatonia when the fear network is reactivated.

Dissociative responses are easily subject to conditioning (Bolles & Fanselow, 1980). In addition to classical conditioning, instrumental learning may reinforce the likelihood of dissociative responding. Survivors of trauma, who were rewarded with survival as a result of their "shut-down" during the offense, will also show a strong vasovagal dissociative response when confronted with trauma reminders, even decades later (Pitman, van der Kolk, Orr, & Greenberg, 1990; Van der Kolk, Pitman, & Orr, 1989).

In our recent studies we could confirm the two subtypes of responding. In survivors of organized violence, including sexual abuse and torture, we measured plasma and salivary cortisol levels and found that physical entrapment, body and mucosa contact with the offender and penetration by the perpetrator (especially rape vs. not being raped) during the traumatic event, modulated later cortisol responses to trauma reminders (Gola et al., 2010).

When survivors of severe human rights violations first speak about the trauma, for example, during a hearing or during exposure therapy, we can literally see that their hearts race, their hands become sweaty, and their breathing is heavy. Those who experienced multiple and extreme trauma, however, may soon stop to respond physiologically and report that they feel numb and unreal. This more clinical and anecdotal observation suggests that, just as the mind has a way of turning off strong emotions in overwhelming situations, the body can also turn off some of its stress responses, if feelings of terror and helplessness were strong. In another study (Ray et al., 2006) we observed that dissociative experiences are reflected in slow abnormal brainwaves, with generators residing in left ventro-lateral frontal cortex, an area that contributes to our ability to verbalize and to executive functioning, that is, the ability to plan, prepare, and be disposed for actions. In addition, these brain regions mirror and reflect the behavior of others and oneself (mirror neuron system). Focally generated slow waves as observed in this study often appear around structural or functional brain lesions. Correspondingly, we interpreted our findings of this left frontal brain activity as a sign of decoupling of these brain regions from both sensory experience and action – the only response that seems possible during the extreme helplessness induced by serious torture but that has devastating consequences later in life, as this brain reorganization is maintained even when the torture is over.

To summarize these findings we can conclude that those patients who merely underwent repeated sympathetic arousal during events show activation during exposure ("uproar-PTSD – Type 1"; typically called "simple trauma") and those who, at least once, reached the flag state will show dissociative shut-down reactions when reminded of the trauma ("shut-down-PTSD – Type 2"; typically called "complex trauma"). Therefore, a trauma-focused approach, which is necessary to overcome the trauma spectrum disorders, requires the management of dissociative stages (fright, flag, and faint) and this has obviously important treatment implications.

Self-Injury as a Means to Induce Vasovagal Shut-Down

Dissociative symptoms are common in borderline personality disorders (BPD) and the majority of patients meet the criteria for dissociative disorder diagnoses (e.g., Korzekwa, Dell, Links, Thabane, & Fougere, 2009). Characteristic for BPD is self-harming behavior including severe forms, such as cutting and burning. By self-injurious behavior patients may receive tension reduction and mood elevation from stress-induced aversive states or anxiety (Bohus et al., 2000; Klonsky, Oltmanns, & Turkheimer, 2003; Russ et al., 1992). Hence self-injurious behavior usually occurs in response to emotional stress or anxiety (Bierer et al., 2003; Yates, 2004). Self-injurious behavior appears to rapidly lower an escalating heart rate (Novak, 2003).

Based on the present model, we suggest that self-mutilating behavior facilitates tension relief through setting in motion the *flag* state. Self-mutilating behavior serves to initiate dissociation in the form of flaccid immobility. When mutilating the skin surface (i.e., cutting), a vasovagal reaction is launched. During this "shut-down" blood pressure and heart rate down, together with a fading emotional

tension and a termination of ruminating thoughts, worries, and troubles, etc. The potentially reinforcing effects of self-injurious behavior may account for the failure of some treatment regimens. We conclude that borderline patients induce their own relaxing dissociative shut-down response (type 2) as a learned response.

Management of Dissociative *Fright, Flag*, and *Faint* Reactions During Trauma Treatment

Emotional engagement in traumatic memory is a necessary condition for successful processing of the event and resultant recovery (Breuer & Freud, 1895; Foa & Kozak, 1986; Horowitz, 1986; Jaycox et al., 1998). Fear activation during exposure therapy is usually positively related to treatment outcome (Borkovec & Sides, 1979; Foa & Kozak, 1986; Foa, Riggs, Massie, & Yarczower, 1995). In trauma survivors, however, exposure may reactivate the flag state or even result in fainting. When the survivor responded peritraumatically with vasovagal shut-down or has even fainted during the event, a replay of these responses and emotions during treatment will quickly result in therapeutic failure. The patient cannot properly hear and see the therapist any longer, is not capable to narrate and react, disabled to respond and move, and incompetent to reprocess emotion or cognition. During treatment, patients with the dissociative shut-down form of PTSD, so called "complex trauma" patients, need assistance to actively fight the replay risk of vasovagal dominance (i.e., loss of blood pressure, bradycardia, vasodilatation, and eventually fainting). Activation (active motoric engagement) may help to maintain arousal and prevent immobility. Relaxation techniques are at this point contraindicated since they would aggravate the cardiovascular problem and support fainting. During the first times when confronting such a fear network, recall of the trauma material should be paralleled with forced reality testing and therapist's active engagement in directive guidance the very moment sings of flagging or fainting appear (for specific intervention, please see Table 1).

In most of the trauma-focused techniques, information about the traumatic material is available before the beginning of the session, no matter whether the events are worked through in chronological biographical order (Narrative Exposure Therapy – NET, Schauer et al., 2005), or in hierarchical order (i.e., EMDR, prolonged exposure). Characteristics of events that may elicit dissociative fright, flag, or faint are:

a) imminence of threat/aggressor and total helplessness, for example, direct body contact with the perpetrator, fixation (constraint), skin contact, and danger of skin penetration (specially sharp objects, for example, teeth and knife);
b) rapid arousal peak, often with startle, due to unexpected and sudden proximity of threat or aggressor;
c) presence of fresh blood, mutilated bodies;
d) being contaminated, contact to infectious material such as mucosa and body fluids, including sperms, urine, and feces;
e) anal, vaginal, or oral penetration of the victim;[2] and
f) severe pain being inflicted on the victim.

How to Notice the Onset of Dissociative Shut-Down During Exposure?

"I have also noticed that under fear there is a strong tendency to yawn." (Charles Darwin, *The Expression of the Emotions in Man and Animals.*)

When confronted with traumatic material, there is – at least initially – a brief increase in sympathetic arousal, the "uproar" branch is activated even in survivors with "shut-down" PTSD types. During a period of agitation, heartbeat (palpitation) and blood pressure are rising, vasoconstriction (cold hands and pale face) and elevated emotional and physiological arousal can be observed. However, bodily numbing along with slight paralysis mainly in the legs are signs that dissociative shut-down stages will be entered soon. Sudden yawning in the middle of the arousing exposure or tinnitus may indicate that hypotension has already set in (yawning can support blood pressure and heartbeat frequency elevation (Platek, Mohamed, & Gallup, 2005)). The hands become warm and the skin may change slowly from pale to red. The patient reports dizziness, blurred vision, and weakness of the muscles. The three main areas of behavioral changes that characterize a shut-down reaction peritraumatically can also be observed later, when recalling the traumatic experiences:

1. Functional sensory deafferentation: incoming stimuli seem not to reach beyond the gates in spinal cord and thalamus, they are perceived as weak, distant, and unreal. During treatment, the patient may become unresponsive, with unfocused gaze.
2. Reversible palsy leads to a visible decrease of bodily movements and immobility, posturing and waxy flexibility also during treatment.
3. Inhibition or disconnection of areas responsible for language processing and production of speech (unclear/confused speech, fragmentation of sentences, inability to speak, and almost no or belated response to acoustic stimuli).

We assume that any of these areas can be instrumentally conditioned in isolation and that respective states, like functional blindness or palsy of a limb, may become tonic. Consequently, ICD-10 classifies conversion disorders as dissociative disorders.

[2] Fainting through penetration is not only associated with sexual penetration, it can also occur when instruments are inserted in the body. For instance, the aspiration of lungs in a newborn baby may cause reflexive bradycardia.

Table 1. Therapeutic intervention

	Therapeutic intervention when "uproar" is dominant during the exposure session	Therapeutic intervention when "shut-down" is dominant during the exposure session
	Activation of elements of the fear network in relation to their autobiographical context (space and time when event has happened).	*Activation of elements of the fear network in relation to their autobiographical context, supported and interrupted by continuous reality testing and* *- cardiovascular activation* *- sensory stimulation* *- encouragement of speech production* *until trauma material can be processed without shut-down.*
Sensory-afferent	Emphasize the exploration of sensory details of the *past* traumatic event, comparing/contrasting with sensations and emotions in the present during recall.	Emphasize the exploration of sensory details of the *past* traumatic event, comparing/contrasting with sensations and emotions in the present during recall. + stimulation of the senses in the *here and now*, for example: present positive fragrances (e.g., lemon) or tasting samples (peppermint oil or chili gum), switch on bright light, present tactile information (e.g., texture, ice-packs), lead attention to auditory stimuli in the here and now.
Motor-afferent		Emphasize activation of the skeletal muscles, enhance blood pressure and muscle tonus (above all, use applied tension, physical exercises, leg crossing, physical counter pressure maneuvers) and use body balancing tasks.
Language processing	Emphasize the narration of the *past* traumatic scene.	Emphasize the narration of the *past* traumatic scene, supported by the facilitation of continuous narrative engagement in the *here and now* (e.g., active communication; enhance speech production).
Emotional processing	Support the full emotional expression.	Support the full emotional expression, allowing and promoting specifically anger affect that has been inhibited peritraumatically.
Nutritional demands		Pay attention to adequate nutrition (caution for malnutrition/eating disorders); increase dietary salt and fluid intake in daily life; advise to drink water (approx. 500 ml, if tolerated) just before exposure to trauma material.
	Counteract and abstain from:	
	• Termination of exposure before contextualization and integration could take place (i.e., avoidance).	• Termination of exposure before contextualization and integration could take place. • Disengagement from the here and now. • Relaxation (instead of activation). • Sensory similarities between the trauma context and the therapeutic setting. • Stimuli that are associated with disgust or similar to body fluids and feces (i.e., ammoniac). • Threat cues in the here and now (instead: present safety signals). • Semi-darkness in the room and objects for hiding behind (e.g., large plants, furniture).

Measures to Counteract Dissociative Shut-Down During the Exposure Therapy Session

Before starting an exposure procedure with a "shut-down" prone, that is, dissociative patient, it is important that the blood sugar level is in the normal range, that is, that the patient has sufficient nutrition intake including salt and minerals. Shut-down reactions like fainting and bradycardia may also be aggravated by low fluid volume such as a lack of water, low sodium intake and dehydration (Kreipe & Birndorf, 2000; Pritts & Susman, 2003). On the other hand, vasovagal reactions could be shown to decrease when subjects drank water just before being exposed to sharp objects like needles or orthostatic testing (Newman et al., 2007; Claydon, Schroeder, Norcliffe, Jordan, & Hainsworth, 2006).

Following explicit education on the causes of "shut-down" reactions during recall, the therapeutic measures, which are likely to be applied during the exposure session, must be explained and consented to by the patient. At the first signs of a shut-down process, active mobilization and explicitly induced sensory contrasting of time and place contexts (past vs. presence) should be initiated by the therapist in parallel to the encouragement of recall of the traumatic event. Feared stimuli can be briefly presented, in the human being always interrupted by reality testing, so that a vasovagal syncope will not be elicited and a massed series of brief exposure trials can bring about fear extinction, as demonstrated in basic research (Grey, Rachman, & Sartory, 1981; Martasian, Smith, Neill, & Rieg, 1992; Renfrey & Spates, 1994). For survivors with shut-down PTSD we suggest continuous shifting of attention between trauma-related material and the present context by recalling reality, sensory stimulation in the here and now and motor activation (using applied muscle tension and physical counterpressure maneuvers). The therapist responds to prodromal fainting symptoms by engaging in context-contrasting and muscle tension techniques to counteract the incipient syncope. Active muscle tension appears as the new frontline treatment of reflex syncope inducing significant blood pressure increase due to clinical trials to avoid shut down (Krediet et al., 2002; France, France, & Patterson, 2006; European Society of Cardiology, 2009). Slowing the rhythm of the attentional shifts between the present and the past will allow the patient to process the trauma narrative unhindered by prior *Flag* or *Faint* responses and thus allow to inhibit the fear network via contextualization of the experience in the past. Moving in and out of the trauma scene according to the response of the client's ANS modulation needs active monitoring and fast intervention by the therapist. Practical examples to counter dissociation are presented in Table 1.

Outlook and Further Research

Dissociation is seen as a state of human beings who are engaged in response to acute threat. Dissociative behavior can be classically and instrumentally conditioned and thus appear when appropriately cued. Subsyndromes, like functional deafferentation or limited paralysis, may thus become tonic like in conversion disorders. Dissociative shut-down reactions following traumatic stress can be successfully treated when usual exposure techniques are complemented by methods that counteract flag and faint reactions. The etiological model can help to understand the survivor's psychophysiological state and provides guidelines for practical intervention techniques, depending on the particular stage in the cascade. The prevention of dissociative shut-down during exposure sessions not only facilitates the required processing of the traumatic events, it also results in a speedy decrease from dissociative responding in everyday life and hence a considerable relief for the patient. Certainly more empirical evidence is needed to further specify the model and to test its clinical utility for trauma-related psychopathology.

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