The impact of dissociation on the development and maintenance of posttraumatic stress disorder

The impact of dissociation on the development and maintenance of posttraumatic stress disorder

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op het gebied van de Sociale Wetenschappen

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Chapter 1

General Introduction

Introduction

The syndrome of posttraumatic stress disorder (PTSD) was first introduced in the Diagnostic and Statistical Manual of Mental Disorders in 1980 (DSM-III, American Psychiatric Association [APA], 1980). Although PTSD had not been described as such earlier, similar trauma-related syndromes had been recognized, for instance 'the soldier's heart' (Da Costa, 1871), 'traumatic neurosis' (Freud, 198; Oppenheim, 1889), 'shell shock' (Myers, 1940), and concentration camp syndrome (Bastiaans, 1957). The latest edition of the DSM (DSM-IV-TR, APA, 2000) stipulates the conditions that need to be met for PTSD to be diagnosed. First, exposure to a traumatic event must have taken place in which the individual reacted with extreme fear, horror or terror (criteria A1 and A2). Second, as a result of trauma exposure, current symptoms should include symptoms of reexperiencing (criterion B), avoidance and numbing (criterion C), and hyperarousal (criterion D). Third, the full symptom picture must be present for at least one month (criterion E) and finally, the disturbance should cause clinically significant distress, or impairment in important areas of functioning (criterion F; see Table 1.1 for the exact DSM-IV-TR PTSD criteria).

Numerous people experience traumatic events, such as sexual assault, natural disasters, life-threatening accidents, or war during their lifetimes. Lifetime prevalence rates vary widely though, ranging from 21.4% (both men and women; Perkonigg, Kessler, Storz, & Wittchen, 2000) to 28% (both men and women; Hepp, 2006), 60%-51% (men and women respectively; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), or even 69% (women only; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). Although these proportions are disturbingly high, most people are resilient and do not develop PTSD. An estimated 7.8% of the people that have been exposed to a traumatic event are thought to eventually develop PTSD (Perkonigg et al., 2000). The lifetime prevalence in the general population ranges from 1% to 14%, whereas the lifetime prevalence in high-risk individuals ranges from 3% to 58% (APA, 1994). Mentioned ranges indicates that it is far from clear how many people actually develop PTSD, but even the smallest percentages indicate many individuals do suffer from PTSD, and are in clear need of efficient and effective treatment. Therefore it is highly important to understand the mechanisms involved in the development and treatment of PTSD.

Many studies have tried to identify risk factors for the development of PTSD. Although many variables were investigated, only a few stable risk factors have been identified, among which peritraumatic dissociation (Ozer, Best, Lipsey, & Weiss, 2003). Dissociation is also suggested to play a role in the maintenance of PTSD. More specifically, it is thought to elicit the formation of unique memories in PTSD by hindering trauma information from being integrated into autobiographical memory (e.g., Van der Kolk & Van der Hart, 1991). Moreover, in the DSM-IV-TR (2000) dissociative symptoms such as amnesia and emotional numbing are even listed as PTSD symptoms. The aim of this dissertation was to study the association between dissociation and PTSD in more detail and to contribute to a better understanding of their relationship.

Table 1.1 *Diagnostic criteria for posttraumatic stress disorder (PTSD), fourth edition TR (APA, 2000)*

A. The person has been exposed to a traumatic event in which both of the following were present:
(1) the person experienced, witnessed or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
(2) the person's response involved intense fear, helplessness, or horror.
Note: in children, this may be expressed instead by disorganized or agitated behavior.

B. The traumatic event is persistently experienced in one (or more) of the following ways:
(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, and/or perceptions.
Note: In young children, repetitive play may occur in which these or other aspects of the trauma are expressed.
(2) recurrent distressing dreams of the event.
Note: In young children, there may be frightening dreams without recognizable content.
(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and/or dissociative flashback episodes, including those that occur on awakening or when intoxicated).
Note: In young children, trauma-specific re-enactment may occur.
(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
(1) efforts to avoid thoughts, feelings, and/or conversations associated with the trauma.
(2) efforts to avoid activities, places, and/or people that arouse recollections of the trauma.
(3) inability to recall an important aspect of the trauma.
(4) markedly diminished interest or participation in significant activities.
(5) feeling of detachment or estrangement from others.
(6) restricted range of affect (*e.g.*, inability to have loving feelings).
(7) sense of a foreshortened future (*e.g.*, does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two of the following:
(1) difficulty falling or staying asleep.
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

E. Duration of the disturbance (symptoms in criteria B, C, and D) is more than one (1) month.

F. The disturbance causes clinically significant distress and/or impairment in social, occupational, and/or other important areas of functioning.

Theoretical background

Theoretically, dissociation is thought to contribute to the development of PTSD by impeding adequate information processing. The dual-representation theory (Brewin, 2001; Brewin, Dalgleish, & Joseph, 1996), for instance, posits that adequate encoding of trauma-related information requires relatively conscious information processing and therefore dissociation would lead to an inadequate encoding of such trauma-related information. As a result, the information is stored in situationally accessible memory (SAM), instead of (mainly) in the verbally accessible memory (VAM) from which it can be retrieved voluntarily. SAM contains mostly sensory, visuospatial information without temporal context, which is retrieved automatically and involuntary, like intrusive images or flashbacks,. Similarly, Ehlers and Clark (2000) have suggested that rather than conceptual processing (processing the meaning of the situation and placing it into context), data-driven processing (i.e. processing sensory impressions) is associated with later intrusions. In their cognitive model of PTSD, the authors propose that the nature of the trauma memory is determined by the degree to which conceptual processing occurs during the trauma. If mainly data-driven and no or negligible conceptual processing takes place, then the trauma memory will be difficult to retrieve intentionally. The authors further hypothesize that some types of dissociation (depersonalization, derealization and numbing) may impede the integration of the trauma into the autobiographical memory knowledge base.

Animal models have also been used in order to comprehend the underlying mechanisms of PTSD and dissociation. For example, stress-induced analgesia in animals exposed to uncontrollable and unpredictable shock has been suggested to be analogous to numbing symptoms in PTSD in humans (Foa, Zinbarg, & Rothbaum, 1992).

Dissociation

The term dissociation is used for a variety of phenomena, which makes research in this field both complicated and challenging. For the sake of a clear discussion of the relationship between dissociation and the development and treatment of PTSD, it is necessary to first organize and delineate the different phenomena that are referred to as dissociation. Several authors have tried to categorize the different phenomena that are considered to be dissociative (Allen, 2001; Holmes et al., 2005; Kennedy et al., 2004; Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1998), which resulted in several distinct categorizations. It is beyond the scope of this dissertation to discuss them all. Instead, we will discuss one possible categorization that targets two different forms of dissociation, because this particular categorization is based on distinct underlying mechanisms and not just descriptive (see also Figure 1.1 for a graphic representation of the categorization used in the following chapters).

Several authors distinguished these two forms of dissociation (Allen, 2001; Brown, 2002; Holmes et al., 2005; Putnam, 1997, p. 71, 87) that have been referred to as detachment and compartmentalization (Allen, 2001; Holmes et al., 2005).

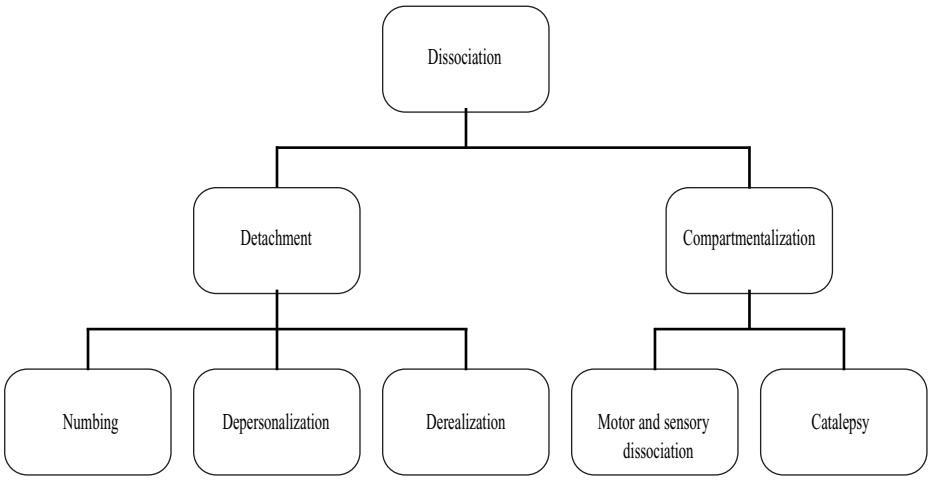


Fig. 1.1 *Schematic representation of the distinct dissociative symptoms studied in the current dissertation*

Detachment refers to experiences that concern an altered state of consciousness in which one feels detached from one's body, sense of self, or the world around one. Examples of detachment states are depersonalization, derealization, and emotional numbing. Detachment is thought to be part of a biological defense mechanism that minimizes anxiety during extreme threat. The neurophysiological profile of detachment is assumed to be characterized by left prefrontal (top-down) inhibition of the amygdala, along with an activation of right prefrontal attentional systems, resulting in a lack of emotional coloring and increased alertness, respectively (Sierra & Berrios, 1998).

Compartmentalization refers to experiences involving 'a deficit in the ability to deliberately control processes that would normally be amenable to such control' (Holmes et al., 2005, p. 7). The individual experiences an inability to bring normally accessible information into conscious awareness (like in dissociative amnesia), to deliberately move (like in conversion paralysis), or to stop movement (like in conversion seizures). Other examples of compartmentalization are sensory loss or pseudohallucinations, and forms of somatoform dissociation, i.e., a term that is used for dissociative symptoms that are manifested in somatic variables and concern a disintegration of somatoform components of experience, and bodily reactions and functions; Nijenhuis et al., 1998). Both empirical and case studies indeed suggest a deficit to deliberately control otherwise controllable processes (Bryant & McConkey, 1989; Kihlstrom, 1992; Kuyk, Spinhoven, & Van Dijk, 1999), like for instance the ability to control retrieval processes to get access to otherwise accessible information. Because of its many distinct symptoms, it is unclear whether compartmentalization – like detachment – is characterized by one core neurophysiological profile. Studies on neural correlates of hypnotically induced compartmentalization symptoms (mainly analgesia and pain perception) suggest an interference with frontal integrative functions that involve the awareness of perceptual stimulation (Kallio, Revonsuo, Hämäläinen,

Markela, & Gruzelier, 2001). Moreover, there is some evidence that hypnotically induced catalepsy, the form of compartmentalization that is used in this dissertation, causes an alteration in mental motor representations (Roelofs, Hoogduin, & Keijsers, 2002). Also, a PET study showed a state of total body catalepsy to be related to a deactivation of primary visual and (less significantly) primary auditory areas, which may reflect a shift in selective attention away from external stimuli and towards internal sensations (Grond, Pawlik, Walter, Lesch, & Heiss, 1995).

In sum, this distinction between detachment and compartmentalization concerns the nature of the dissociative symptoms as well as the hypothesized cause of these symptoms, i.e. an altered state of consciousness versus an inability to deliberately control otherwise controllable processes, respectively). Both forms of dissociation have been associated with PTSD or the development of PTSD. Consider for instance some of the typical PTSD symptoms: Amnesia due to a retrieval deficit could be classified as compartmentalization, whereas numbing symptoms could be classified as detachment. With respect to PTSD development peritraumatic dissociation, i.e. dissociation at the time of the trauma, is thought to play a key role. Most studies on peritraumatic dissociation have focused on detachment, partly because the items of the instrument that is used most often (the Peritraumatic Dissociative Experiences Questionnaire, PDEQ; Marmar, Weiss, Metzler, & Delucchi, 1996) typically measure detachment. Peritraumatic dissociation was found to be associated with PTSD in many retrospective studies (Bernat, Ronfeldt, Calhoun, & Arias, 1998; Engelhard et al., 2002; Kaufman et al., 2002; Marmar et al., 1994; Punamäki, Komproe, Quota, Elmasri, & De Jong, 2005; Tichenor, Marmar, Weiss, Metzler, & Ronfeldt, 1996; Weiss, Marmar, Metzler, & Ronfeldt, 1995) and also in some prospective studies (Birmes et al., 2001; Birmes et al, 2003; Ehlers, Mayou, & Bryant, 1998; Engelhard, Van den Hout, Kindt, Arntz, & Schouten, 2003; Griffin, Resick, Mechanic, 1997). In addition, compared to other variables peritraumatic dissociation had a relatively large predictive power in a review study on predictors of PTSD (Ozer, Best, Lipsey, & Weiss, 2003). However, many of these studies suffer from methodological shortcomings. In the retrospective studies these problems are obvious, since memory is known to be affected by many factors, such as forgetting, attribution of symptoms, over-reporting and malingering (Candel & Merckelbach, 2004). Recall of peritraumatic dissociation has shown to be unstable over time, and changes in PTSD symptoms proved highly correlated with changes in recall of peritraumatic dissociation (Marshall & Schell, 2002; Zoellner, Sacks, & Foa, 2001). But also the prospective studies suffer from methodological problems. For one, peritraumatic dissociation is still measured days to weeks after the traumatic event. Furthermore, some studies failed to control for initial PTSD symptoms or trait dissociation (a relatively stable tendency to experience dissociative symptoms), making it unclear to what extent peritraumatic (state) dissociation might act as an independent predictor of PTSD (Candel & Merckelbach, 2004). Lastly, most research on peritraumatic dissociation involves clinical field studies, and only very few studies used an experimental design to directly manipulate dissociation.

The first three studies described in this dissertation were designed to overcome these various problems and contribute to the dissociation debate by focusing on

some aspects of dissociation that have not been systematically studied yet. For example, a prospective study (Chapter 2) considers both detachment and compartmentalization in predicting PTSD development after a traumatic event. We additionally controlled for initial numbing symptoms and included cognitive interpretations as an alternative predictive factor. In another study (Chapter 4) an experimental design was used to investigate the impact of dissociation (compartmentalization, namely catalepsy) on PTSD, more specifically, on intrusion development. In a separate study, it was investigated whether catalepsy would be a suitable technique to evoke compartmentalization symptoms, such as subjective paralysis and other sensory-motor dissociative epiphenomena (Chapter 3).

Trauma memory and the maintenance of PTSD

Fear network theories propose a cognitive model for information processing of fear. In the emotional processing theory (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989), based on Lang's bio-informational theory (1977, 1979), trauma memory is represented as a fear structure that includes representations of trauma-related stimuli, responses and their meaning. In PTSD, this fear structure is characterized by a large number of stimulus representations associated with danger and strong response elements. The PTSD trauma memory itself is not unique, but more like an 'ordinary' memory with a particular structure and intensity. To recover from PTSD, Foa et al. (1989) propose that this strong fear memory must be integrated into 'ordinary' autobiographical memory by creating associations between elements of the fear structure and new information that is incompatible with the fear memory. Other theories, in contrast, suggest that PTSD trauma memories are represented qualitatively differently than ordinary memories. These theories hypothesize that the traumatic event is, as it were, split off from the general associative and autobiographical memory (e.g., van der Kolk & Van der Hart, 1991). It is suggested that peritraumatic and ongoing dissociation prevent adequate information processing, thereby leading to incoherent and fragmented memories, or even amnesia for parts of the traumatic event (Van der Kolk & Fisler, 1996).

To study whether trauma memories are indeed distinct from other memories, we compared the memories of several distinct populations: trauma memories of PTSD patients, memories of a severe panic attack of patients with panic disorder and agoraphobia, and trauma memories or memories of extremely anxious events of healthy controls; Chapter 5). We also analyzed whether there were any between group differences in peritraumatic dissociation (detachment) that could account for the possible differences between the groups.

Finally, dissociation is not only considered to play a key role in PTSD development, it is also thought to have an effect on PTSD treatment. It is assumed that dissociation hinders adequate fear activation, essential for an exposure treatment to be effective (Foa & Kozak, 1986; Hembree, Marshall, Fitzgibbons, & Foa, 2001; Jaycox & Foa, 1996). To verify this assumption we investigated the impact of dissociation on the efficacy of prolonged (imaginal) exposure. Again, several forms

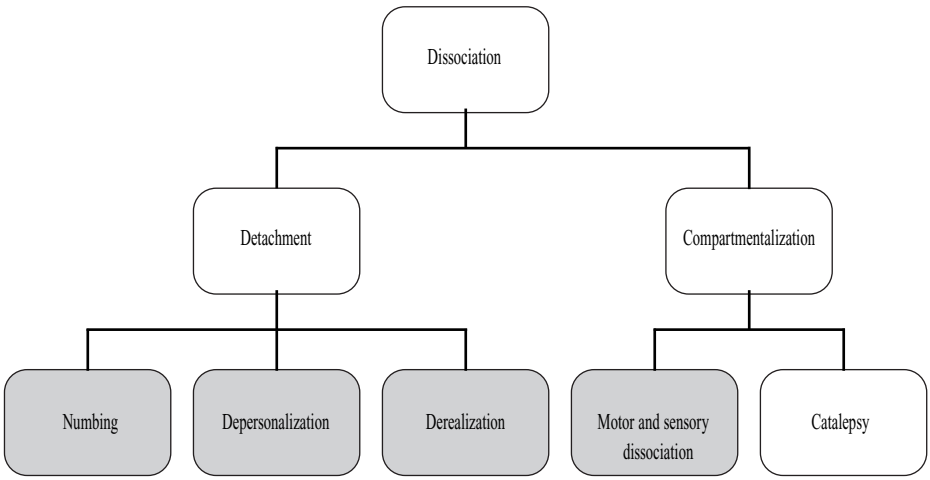
of dissociation were evaluated, including trait dissociation, depersonalization, and emotional numbing.

Brief outline of the dissertation

The subsequent chapters focus on the association between dissociation and PTSD or, more specifically, PTSD development, memory characteristics assumed unique for PTSD, and PTSD treatment. In Chapter 2 the impact of dissociation on PTSD development is explored in a prospective, controlled design. In Chapter 3 catalepsy is examined as a new paradigm that could prove to be useful in research on somatoform dissociation. This catalepsy paradigm is exploited in an experimental study described in Chapter 4 to investigate the impact of peritraumatic dissociation on intrusion development. Then, in Chapter 5, assumed PTSD-specific memory characteristics are compared in three distinct populations, i.e. PTSD, panic disorder and healthy controls. In the last study, presented in Chapter 6, the impact of dissociation on the efficacy of prolonged (imaginal) exposure treatment for PTSD is investigated. Finally, results of all studies are summarized and discussed, and directions for future research are given in Chapter 7.

Chapter 2

Peritraumatic Dissociation and PTSD Symptoms: A Prospective Study



Hagenaars, M.A., Van Minnen, A., & Hoogduin, C.A.L. (2007). Peritraumatical psychological and somatoform dissociation in predicting PTSD symptoms: A prospective study. *Journal of Nervous and Mental Disease, 195, 952-954.*

Abstract

Objective: The present prospective study evaluates the predictive value of two different types of dissociation (psychological and somatoform peritraumatic dissociation), and dysfunctional cognitions on symptoms of posttraumatic stress disorder (PTSD) at six months. *Method*: Assessment of dissociation, PTSD symptoms and dysfunctional cognitions took place in forty-nine participants approximately three weeks after a traumatic event. Six months later PTSD symptoms were assessed again. *Results*: The effect of both psychological and somatoform peritraumatic dissociation disappeared after controlling for initial PTSD numbing symptoms. Dysfunctional cognitions predicted PTSD at 6 months after controlling for initial numbing symptoms. *Conclusions*: The present study indicates that peritraumatic dissociation may not be a predictor of PTSD. In contrast, maladaptive posttraumatic coping behavior, like persistent dissociation and dysfunctional cognitions may be predictors.

Keywords: peritraumatic dissociation, posttraumatic stress disorder, cognitions, numbing.

Introduction

People's reactions during the time of a traumatic event have been studied intensively in the last decade, suggesting an important predictive role of peritraumatic variables in the development of PTSD. In a meta-analysis, peritraumatic dissociation was strongly related to later PTSD (Ozer, Best, Lipsey, & Weiss, 2003). Theoretically, peritraumatic dissociation is thought to interfere with adequate processing of trauma information, leading to poorly integrated representations of the trauma in autobiographical memory, which subsequently leads to PTSD intrusions and flashbacks (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000).

However, many of the studies in the meta-analysis were retrospective or non-controlled. An increasing body of literature is now questioning the usefulness of peritraumatic dissociation in the prediction of PTSD. Indeed, some recent controlled studies found the relationship between peritraumatic dissociation and PTSD disappeared after controlling for initial PTSD symptoms (e.g., Marshall & Schell, 2002). This may be the result of the overlap between dissociative and PTSD symptoms, more specifically numbing symptoms. Moreover, several studies successfully induced peritraumatic dissociation in an experimental context, but failed to find a relationship with trauma-related intrusions (Hagenaars, Van Minnen, Holmes, Brewin, & Hoogduin, 2006; Holmes, Brewin, & Hennessy, 2004; Holmes, Oakley, Stuart, & Brewin, 2006).

Some studies have shown that not peritraumatic dissociation but persistent dissociation predicts PTSD development (Briere, Scott, & Weathers, 2005; Murray, Ehlers, & Mayou, 2002). Unlike peritraumatic dissociation, an automatic reaction evoked by threat, persistent dissociation indicates a dysfunctional coping strategy. This is in line with the cognitive model of PTSD (Ehlers & Clark, 2000), which states that it is not the traumatic event itself, but one's coping with the event and its

sequelae that predicts PTSD. The cognitive model also posits that dysfunctional cognitions about the trauma or one's own reactions during or after the trauma predict the development of PTSD symptoms. Persistent dissociation is seen as a maladaptive coping behavior that prevents the person to change negative appraisals of the trauma, therefore maintaining PTSD. Indeed, some studies found that posttrauma dysfunctional cognitions and dysfunctional strategies like avoidance or numbing were related to the onset and maintenance of PTSD (Dunmore, Clark, & Ehlers, 1999).

In contrast with the large number of studies on peritraumatic psychological dissociation, mostly using the Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar, Weiss, & Metzler, 1997), peritraumatic somatoform dissociation has been underreported. However, this somatoform reaction to threat seems rather common in both animals and humans (Moskowitz, 2004). For example, it was found that two third of the rape victims froze and/or was unable to move during the assault (Galliano, Noble, Puechl, & Travis, 1993). This peritraumatic tonic immobility was related to an increased passive attitude after the assault. In a recent review, it was argued that two qualitatively distinct forms of dissociation can be distinguished, namely detachment and compartmentalization (Holmes et al., 2005). PDEQ items typically refer to detachment. Somatoform dissociation on the other hand typically refers to compartmentalization. In this respect, it would be interesting to study whether these two types of dissociation have different influences on PTSD development.

In conclusion, it is important to gain more insight in the impact of distinct forms of peritraumatic dissociation and confounding factors on PTSD development. The present study is set up to investigate prospectively the relationship between two types of dissociation - psychological and somatoform peritraumatic dissociation – and dysfunctional cognitions on PTSD symptoms 6 months posttrauma. Because of the substantial overlap between dissociative and numbing symptoms, and because numbing concerned persistent (and not peritraumatic) symptoms, we controlled for initial numbing symptoms.

Method

Forty-nine participants were recruited from three areas where a disaster had taken place in the home environment. In the first area (27 participants), the balconies of 5 apartments came down with a lot of noise and two people died under the wreckage. In the second area (8 participants) a gas explosion took place. Three houses were destroyed, but no one was injured. In the third area (14 participants) a truck drove into a supermarket, destroying the supermarket as well as the two houses next to it, and three people died. All participants had witnessed or were involved in the trauma. Participants were contacted if they were potential witnesses or potentially involved, that is, if their residence was close to the place where the disaster toke place. In addition, individuals that were in the supermarket (third area) were contacted via the owner of the supermarket. After receiving a written description of the

study, written informed consent was obtained for all participants. Three participants (all from the first area) withdrew their participation after the procedure was explained, leaving a total of 46 participants (25 men and 21 women, mean age 51.5 years, *SD* = 15.3), who completed the first assessment. The first assessment was approximately 20 days (*SD* = 5.9 days) after the trauma. Thirty-two participants completed the second assessment at 6 months posttrauma.

At the first assessment two types of dissociation were measured. Peritraumatic psychological dissociation was measured by the *Peritraumatic Dissociation Experiences Questionnaire-10-Self Report Version* (PDEQ-10-SRV), which is a reliable and valid instrument (Marmar, Weiss, & Metzler, 1997). The *Somatoform Dissociation Questionnaire – Peritraumatic* (SDQ-P; Nijenhuis, Van Engen, Kusters, & Van der Hart, 2001) was used to measure peritraumatic somatoform dissociation. Dysfunctional trauma-related cognitions were measured by the *Posttraumatic Cognition Inventory* (PTCI), which has good to excellent psychometric properties (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999;).

Both at the first assessment and second assessment the *Posttraumatic Symptoms Scale – Self Rating* (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993) was used to assess PTSD symptoms as defined by DSM-IV. Psychometric properties of the total scale and its subscales are good (Foa et al., 1993). As a dissociation-related construct, numbing was defined using the three numbing items of the PSS-SR avoidance subscale (e.g., Litz, 1992) that refer to diminished interest, detachment or estrangement, and restricted range of affect. Note that the assessment of numbing concerned persistent symptoms (e.g., symptoms in the week prior to the assessment), whereas assessments of psychological and somatoform dissociation concerned symptoms at the time of the trauma.

Regression analyses were used to predict PTSD symptoms at 6 months. In all analyses, the criterion for significance was .05.

Results

First, the effect of psychological and somatoform peritraumatic dissociation, and dysfunctional cognitions on PTSD symptoms at 6 months was studied. To establish the effect of these predictors without controlling for initial numbing symptoms, a stepwise regression was conducted with PSS-SR at 6 months as the dependent variable and peritraumatic psychological and somatoform dissociation, and posttraumatic dysfunctional cognitions as predictors (measured by PDEQ, SDQ, and PTCI respectively). Peritraumatic psychological and somatoform dissociation, and posttraumatic dysfunctional cognitions all uniquely predicted PTSD at 6 months (ΔR^2 = .42, $p < .001$, ΔR^2 = .11, p = .01, and ΔR^2 = .10, p = .01 respectively).

However, these results changed after controlling for initial PTSD numbing symptoms. For this purpose, initial PTSD numbing symptoms were entered in the first block of a hierarchical regression (R^2 = .57, β = .68, $p < .001$). Peritraumatic psychological and somatoform dissociation, and posttraumatic dysfunctional cognitions (PDEQ, SDQ, and PTCI respectively) were entered and stepwise analyzed in the

second block. Besides numbing symptoms, that continued to significant (β = .51, p = .001), only posttraumatic dysfunctional cognitions contributed to the prediction (ΔR^2 = .13, β = .40, p = .006). The two peritraumatic dissociation measures did not add to the prediction of later PTSD symptoms above initial numbing symptoms and dysfunctional cognitions.

Discussion

The present study investigated the influence of two forms of dissociation on PTSD symptom development. It was found that both psychological and somatoform peritraumatic dissociation failed to predict PTSD symptoms after controlling for numbing symptoms. Although peritraumatic (psychological) dissociation was strongly related to PTSD development in many studies (Ozer, Best, Lipsey & Weiss, 2003), recent controlled studies also failed to find this association (Marshall & Schell, 2002; Briere, Scott, & Weathers, 2005). In line with the cognitive model of PTSD (Ehlers & Clark, 2000), dysfunctional cognitions did predict later PTSD symptoms on top of initial numbing symptoms. Our findings are in line with other prospective studies, suggesting that sustained posttraumatic and not peritraumatic reactions are associated with PTSD development (Dunmore, Clark, & Ehlers, 1999).

The results have implications for theories on PTSD development, especially with regard to the uniqueness of trauma memory. It is often thought that peritraumatic dissociation, being a disruption of usually integrated processes of consciousness and restricting verbal encoding, leads to inadequate encoding of trauma information. This leads to information being stored as sensory, visuospatial fragments, without temporal context, i.e., intrusions (Brewin, Dalgleish, & Joseph, 1996). The absence of a relationship between peritraumatic dissociation and later PTSD symptoms may indicate that this mechanism of inadequate encoding during the trauma is not relevant with respect to PTSD development. Maybe this is the case because inadequate encoding can later be replaced by adequate information processing. It is possible that PTSD is developed if inadequate encoding is not followed by adequate information processing, leading to sustained inadequate information processing. Indeed, the association between persistent dissociation and PTSD development suggests that the absence of repeated processing of trauma information posttrauma plays a role in PTSD development. Numbing, and avoidance, may prevent the traumatic information to be repeatedly and emotionally processed. It is also possible that numbing prevents dysfunctional cognitions to be re-evaluated, which leads to the development and maintenance of PTSD (Ehlers & Clark, 2000). Indeed, a post-hoc analysis of our data showed that both initial active avoidance (β = .32, p =.04) and numbing (β = .50, p =.002) predicted later PTSD symptoms (ΔR = .53, p < .001), whereas reexperiences (β = .12, p =.44) and arousal (β = .16, p =.36) did not.

The uniqueness of trauma memory and processing of trauma information are complicated and intriguing issues. In this respect, future research focusing on the relationship between (peritraumatic and persistent) dissociation and information processing would be helpful. With respect to future research on dysfunctional

cognitions, it would be interesting to control for pretrauma dysfunctional thinking, or a concept like neuroticism, which is characterized by dysfunctional thinking. This would clarify if either a general tendency to dysfunctional cognitions that is sustained after the traumatic event, or dysfunctional cognitions that are developed merely as a posttrauma reaction, predict PTSD.

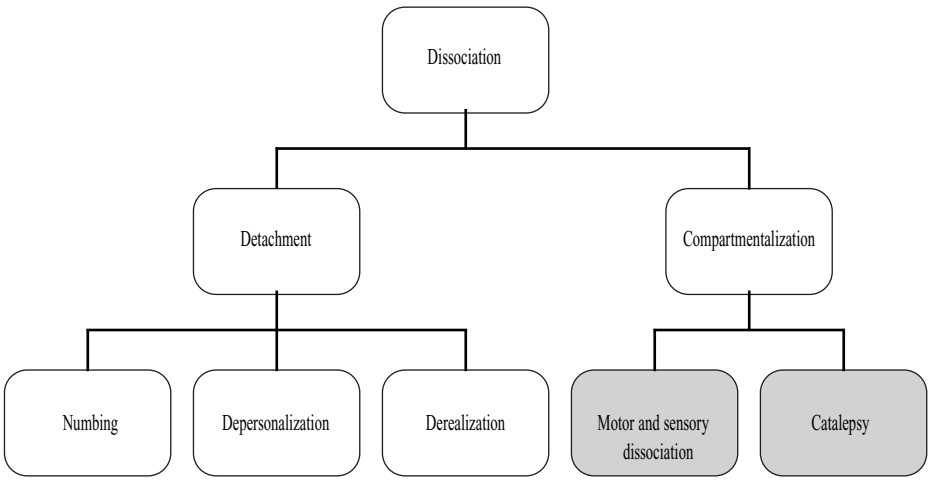
An important limitation of the study concerns the relatively small number of participants. However, the number of tests was in accordance with this small sample. On the other hand, the strength of the study is its prospective design, and the fact that it controlled for numbing symptoms at baseline. In addition, to our knowledge, it is the first to compare different forms of dissociation with respect to the development of PTSD symptoms. Especially somatoform peritraumatic dissociation has not been studied in a prospective design. However, the study merits replication with larger samples and different traumata.

Conclusions

In conclusion, we did not find that psychological or somatoform peritraumatic dissociation predicted PTSD. However, dysfunctional cognitions and numbing (an avoidant coping style) did predict PTSD. Our results suggest that sustained and not peritraumatic dissociation is of relevance. Our reactions during a traumatic event may be as they are, it is the way we cope with them that makes the difference.

Chapter 3

Motor and Sensory Dissociative Phenomena Associated with Induced Catalepsy



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Abstract

The purpose of the present study was to investigate dissociative symptoms that may occur as an epiphenomenon of tactile induced catalepsy. In 15 participants, catalepsy was induced in the right arm and dissociative symptoms were evaluated using a self-report questionnaire. In comparison with the left, non-cataleptic arm, the right, cataleptic arm was perceived different. In addition to increased rigidity, the cataleptic arm was characterized by the presence of paresthesias, a decreased perception of sense and a decreased awareness of the arm. Moreover, the self-reported changes in perception were significantly correlated to hypnotically induced arm immobilization, making part of the Stanford Hypnotic Susceptibility Scale. In conclusion, catalepsy induction elicits a variety of dissociative symptoms and provides a useful research paradigm for the study of motor-perceptual dissociative phenomena.

Introduction

Catalepsy refers to a state of waxy-flexibility and tonic immobility in one or more body parts. It has been observed to occur as a defensive reaction towards stress both in animals and humans (Gallup, & Maser, 1977) and is sometimes referred to as freezing (Kulikova, Kozlachkova, Maslova, & Popova, 1993) or somatoform dissociation (Nijenhuis, Spinhoven, Vanderlinden, Van Dyck, & Van der Hart, 1998). Apart from the stress-context, the phenomenon of catalepsy can be elicited in a hypnotic context (Diehl, Meyer, Ulrich, & Meinig, 1989; Grond, Pawlik, Walter, Lesch, & Heiss, 1995; Sacerdote, 1970a, 1970b), which might provide a useful research paradigm for the study of motor and sensory dissociative experiences. However, most studies in which hypnotic techniques are applied to elicit catalepsy, it remains unclear how catalepsy was induced. This makes it hard to replicate the results and to interpret the findings. Also, although catalepsy induction is meant to provoke stiffness and waxy-flexibility, other co-occurring dissociative symptoms have hardly been studied. The present study is set up to list dissociative symptoms that occur as an epiphenomenon after induced catalepsy. Once catalepsy induction is standardized and co-occurring dissociative symptoms are studied, catalepsy could become a useful research paradigm in provoking motor-perceptual dissociative phenomena.

Sacerdote (1970a, 1970b) was the first to apply the induction of catalepsy for therapeutic purposes. He described the technique as (conducted) inversed hand levitation. To achieve catalepsy, he placed the elbow of the person on the armrest of the chair the person was sitting in. He then took the wrist and lifted the arm in a vertical position. By alternately supporting and releasing the forearm, he induced catalepsy. Sacerdote (1970a, 1970b) discovered catalepsy could not only be used as a hypnotic induction technique, but the altered perception of the cataleptic hand could also diminish pain sensations.

In Kihlstrom's information processing theory dissociation is described as a disruption of the normal integrative functions of consciousness and sensory and motor

processes (Kihlstrom, 1992). When applying Kihlstrom's dissociation theory to catalepsy, one could assume this disruption concerns sensorimotor functions, resulting in a change in the perception of (parts of) the cataleptic body. For example, in arm catalepsy, there is no longer an explicit awareness that the arm can be moved. While objectively nothing is wrong with the arm, the person perceives the arm as stiff and is not able to bow it. Some evidence is also found for the idea that sensory information processing changes during catalepsy (Diehl et al., 1989). For example, a PET study found a state of total body catalepsy to be related to a deactivation of primary visual and (less significantly) primary auditory areas. This may reflect a shift in selective attention away from external stimuli and towards internal sensations (Grond et al., 1995).

Although Sacerdote noticed diminished pain perception after catalepsy induction and some evidence for a change of sensorimotor processing has been found in neuroimaging studies, co-occuring dissociative phenomena have never been closely studied. In the present study we investigated changes in self-reported motor and sensory experiences. Firstly, we hypothesized that phenomena other than stiffness will co-occur in the cataleptic body part. This means the cataleptic arm will be perceived as stiff and difficult to move, but there will also be symptoms like paresthesias, and the arm being gone, strange, unreal or not belonging to the person. A questionnaire was constructed to measure these co-occurring phenomena. Secondly, we expected participants with high hypnotic susceptibility to show more dissociative symptoms in the cataleptic arm than participants with low hypnotic susceptibility. Finally we expected dissociative symptoms after tactile catalepsy induction to be related to hypnotic suggestions of catalepsy and arm immobilization.

Method

Participants

A total of 15 right-handed female undergraduate university students, of whom most (12) were previously tested for hypnotic susceptibility in a Dutch normation study of the Stanford Hypnotic Susceptibility Scale form C (SHSS:C, Näring, Roelofs, & Hoogduin, 2001), participated in the present study. They were randomly selected and participated voluntarily. The mean age of the participants was 22 years and 1 month (*SD* 2 years and 2 months). The mean score on the SHSS:C was 6.67 (*SD* 2.02, range 4-10; $N = 12$), indicating that the hypnotic susceptibility was high following the norms for Dutch students (Näring, Roelofs, & Hoogduin, 2001).

Measures

Dissociative phenomena during catalepsy were measured by the *Catalepsy Questionnaire* (CQ; Roelofs & Hoogduin, 1999), a 22-item questionnaire that was constructed by two of the authors because no suitable measure was available yet (see Appendix A for the complete questionnaire). The CQ assesses changes in perception of both the right and the left arm. Ten items addressed the right arm, ten identical items addressed the left arm, and two items addressed both arms. Each

item could be rated on a 5 point Likert scale (1 'not at all' to 5 'yes totally'). The scores of items 1, 5, 9, 10, 13, 15, and 22 had to be reversed, so scores on all items indicated more presence of that particular symptom. Examples of items are 'It felt as if it was hard to bow the right arm' and 'It felt as if the left arm was heavy'. The 22 items were clustered in 3 categories: 'Both right and left arm' consisted of items 7 and 22, 'Catalepsy' consisted of items 1, 3, 8, and 15, and 'Dissociative symptoms' consisted of the remaining 16 items.

The *Stanford Hypnotic Susceptibility Scale, Form C* (SHSS:C; Weitzenhoffer & Hilgard, 1962) is designed to measure hypnotic susceptibility, classified into four categories: ideomotor actions, response inhibitions, cognitive distortions, and post-hypnotic suggestions. After a standard hypnotic induction, 12 items of progressively greater item difficulty are given. The scale showed good reliability (Hilgard, 1965).

Procedure

The participants sat in a comfortable chair with steady arm rests. They had to put both arms on an arm rest. The experimenter took place at the right side of the participant and took her right arm, after having asked permission to touch that arm. Then, he induced catalepsy in that arm following the catalepsy protocol (see Appendix B for the complete protocol), based on the reversed hand levitation described by Sacerdote (1970). It took only a few minutes for each participant to reach a right arm cataleptic state. After a minute mental rotation task (not making part of the present study, for details about that study, see Roelofs, Hoogduin, & Keijsers, 2002), the participant was told to shake her arms and hands so the catalepsy would disappear. All participants reported the arm being perceived as usual after they had shaken their arms. Then, the participants had to fill out the CQ, which was presented by a different experimenter. All experimental sessions were recorded on videotape and a random check was done afterwards, in order to control if suggestions were made during the catalepsy induction that could interfere with the research question. This was not the case. The SHSS:C was assessed at least 4 weeks earlier, being part of a different, independent study. As a result, the assessment of the SHSS:C, the induction of the catalepsy and the presentation of the CQ were all done by different persons.

Results

First, paired t-tests were conducted to check whether the participants perceived the arm as cataleptic (see Table 3.2). A significant difference in reported sensations of catalepsy was found between the left and the right arm (t (14) = -8.42, $p < .001$). A significant difference in perception of the right versus the left arm was found (t (14) = -12.79, $p < .001$), demonstrating that in addition to stiffness and a feeling of rigidity, dissociative symptoms occurred more in the cataleptic arm than in the non-cataleptic arm. The total score on the two items that represent perceived difference between both arms (M = 3.46, SD= 1.39, range 2-10) indicates that the right arm was indeed perceived different than the left arm.

Table 3.1 *Means (SDs) on each item of catalepsy questionnaire (N = 15)*

Item	Right arm	Left arm
Easy to bend	1.93 (1.10)	4.40 (1.18)
A tingling feeling	4.00 (1.36)	1.07 (0.26)
Hard to bend	3.93 (1.10)	1.40 (0.63)
As if the arm was light	3.00 (1.56)	2.33 (1.18)
As if the entire arm belonged to you	2.67 (1.18)	3.53 (1.51)
As if the arm felt strange	4.13 (0.83)	1.13 (0.35)
A normal feeling in the arm	1.80 (1.21)	4.80 (0.41)
As if (a part of) the arm was gone or unreal	3.60 (0.99)	1.00 (0.00)
A numb feeling	4.00 (0.93)	1.07 (0.26)
As if the arm was heavy	3.00 (1.41)	1.53 (0.74)
As if both arms felt different from normal	1.73 (1.03)	
Ao differences experienced between the left and the right arm	1.69 (1.18)	

Note. The range of each item is 1 ‘not at all’ to 5 ‘completely.

Table 3.2 *Means (SDs) of cataleptic and dissociative symptoms in both arms (N = 15)*

	Left arm	Right arm
Catalepsy*	3.00 (1.46)	8.00 (1.46)
Dissociative symptoms**	11.80 (2.11)	29.27 (4.96)

*Catalepsy range 0-10. **Dissociative symptoms range 8-40.

To check whether hypnotic susceptibility was related to changes in perception of both the left and the right arm, correlations between the total SHSS:C score and the total score on the Catalepsy Questionnaire were calculated. Interestingly, there was no significant correlation between the total SHSS:C score and the CQ score for both the normal left ($r = .17$, $p = .61$) and cataleptic right arm ($r = .40$, $p = .20$).

Finally, the third hypothesis was addressed: the relationship between tactile catalepsy induction and hypnotic induction of catalepsy (item 5 of the SHSS:C) and arm immobilization (item 8 of the SHSS:C). Analysis of the relation between CQ-scores (right arm) and the two motor items of the SHSS:C (arm immobilization and catalepsy) separately, demonstrated a significant correlation between CQ-score and SHSS:C-arm immobilization ($r = .63$, $p < .05$), but not between CQ score and SHSS:C-catalepsy ($r = -.16$, $p = .62$). No correlation was found for the CQ scores of the left arm ($r = -.13$, $p = .69$ and $r = .35$, $p = .27$ for arm immobilization and catalepsy respectively).

Discussion

The present study showed tactile induced catalepsy could be induced effectively, as was shown by the fact that participants reported difficulty in bending their cataleptic arm and perceiving it as being stiff. Although not mentioned in the catalepsy induction, other dissociative symptoms besides stiffness – like the cataleptic arm being perceived as 'strange' and 'as if the arm was not there or unreal' – were present as well. This change in perception was not present in the non-cataleptic left arm. Secondly, a high but nonsignificant correlation was found between the SHSS:C and the Catalepsy Questionnaire, but only for the cataleptic arm. Finally, a significant correlation was found between the CQ and the arm immobilization item of the SHSS:C, again only for the right arm only. Strangely, this correlation was not present between the CQ and the catalepsy item of the SHSS:C. This may be explained by the fact that the CQ measures not only catalepsy, but also motor and sensory dissociative epiphenomena. As a result the score on the CQ could be related to immobilization much stronger than to waxy-flexibility only. Besides that, both questionnaires are scored on different scales (a binary scale and a five point Likert scale for the SHSS:C and the CQ respectively), which may have affected the results.

A few remarks should be placed in general. Only 15 persons participated in the study. Nevertheless, the differences between the cataleptic and the non-cataleptic arm were very high (d = 3.42), making it likely that the results are solid and represent a general mechanism.

Another limitation of the present study concerns the fact that, because catalepsy was induced in only the right arm, this arm gets more attention that the left arm. The participants are focused only on their right arm, because this is where they expect the changes and this is where they are hold. So, one could argue it still is not proved that the changes are completely due to the induction of catalepsy and not to the attention paid to that particular arm. With regard to the research question in the present study this is of minor importance, because it investigates the effects of induced catalepsy and not the mechanisms that bring the arm in a cataleptic state. In a way, attention could very well be a valuable ingredient in order to change the perception of a particular body part. It would be interesting to use a control group just holding their arm upright in future research though.

We tried to control for demand characteristics by only suggesting stiffness and confusion of the muscles, and not mentioning either the purpose of the study or any other possible effects of a catalepsy induction (see also Appendix B for the exact wording of the catalepsy induction). Also, to keep the catalepsy induction and the measurement of symptoms independent from each other, the questionnaire was not presented to the participant by the person that had induced catalepsy. Furthermore, the assessment of the SHSS:C took place at least 4 weeks earlier by yet another person and in perspective of another, independent study, which makes it unlikely that this assessment has revealed something about the topic of the present study.

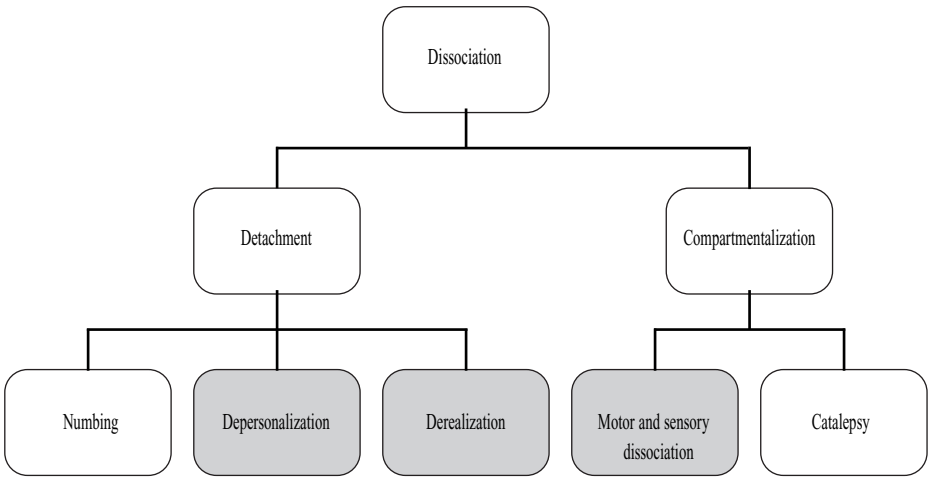
A dissociative state is assumed to be "an altered state of consciousness in which ordinary perceptual, cognitive or motor functioning is impaired" (Brewin & Andrews, 1998, pp. 951). In this paper evidence was found for the presence of dissociative

phenomena after catalepsy induction. Ordinary perceptual and motor functioning was indeed impaired as a result of a catalepsy induction. In an earlier study evidence was found that an alteration in mental motor representations had taken place after a catalepsy induction (Roelofs, Hoogduin, & Keijsers, 2002). Compared to healthy arms, subjects were slowed in mental rotations of the cataleptic arms, especially for larger arm rotations.

In this stage research needs a paradigm in which motor-perceptual dissociative phenomena are provoked. The present study showed that induced catalepsy provoked changes in self-reported motor and sensory experiences, stiffness as well as other dissociative phenomena. Also, the total of dissociative symptoms reported after the tactile induced catalepsy seems to be strongly related to responses to hypnotic suggestions for arm immobilization. To conclude, at least on self-report measures catalepsy leads to an altered perception of the cataleptic body part. This interesting field of study needs further exploration though, taking into account physiological markers on catalepsy.

Chapter 4

The Effect of Hypnotically-Induced Somatoform Dissociation on the Development of Intrusions after an Aversive Film



Hagenaars, M.A., Van Minnen, A., Holmes, E.A., Brewin, C.R., & Hoogduin, C.A.L. (in press). The effect of hypnotically-induced somatoform dissociation on the development of intrusions after an aversive film. *Cognition and Emotion.*

Abstract

Peritraumatic dissociation is thought to effect trauma information encoding, leading to PTSD symptoms like intrusive memories. Most studies have focused on peritraumatic psychological dissociation. The present experiment studies the impact of hypnotically-induced somatoform dissociation (dissociative non-movement) versus deliberate non-movement during an aversive film on intrusion development. Seventy-nine participants were randomized into three conditions: dissociative non-movement (catalepsy), deliberate non-movement, and non-restricted control. Participants recorded their intrusions of the film in a diary for one week. In the dissociative non-movement condition, catalepsy effectively provoked somatoform dissociation. Spontaneous somatoform dissociation across conditions was positively related to implicit bias to film-related words and negatively related to explicit recall, but was not related to intrusion frequency. Dissociative non-movement and deliberate non-movement conditions combined had more intrusions than controls. However, the dissociative non-movement group did not have more intrusions than deliberate non-movement and control groups combined. The implications of these findings are discussed.

Keywords: Peritraumatic dissociation, PTSD, tonic immobility, intrusion, memory.

Introduction

The Effect of Hypnotically-Induced Somatoform Dissociation on the Development of Intrusions after an Aversive Film

Peritraumatic dissociation has been highlighted in an increasing body of literature addressing the impact of peritraumatic reactions on later PTSD symptoms. However, findings are contradictory, the information processing mechanisms are not well understood, and most studies investigated psychological dissociation. This study investigates the impact of aspects of peritraumatic somatoform dissociation on intrusion development in a controlled experimental setting.

Proposed Effects of Dissociation on the Encoding and Retrieval of Trauma Memories

The dual-representation theory of PTSD (Brewin, 2001; Brewin, Dalgleish, & Joseph, 1996), posits that relatively consciously processed information is stored in verbally accessible memory (VAM), from which it can be retrieved voluntarily. When information is not consciously processed, it is stored in situationally accessible memory (SAM). Dissociation disrupts conscious processing and restricts verbal encoding, so information is stored in SAM. SAM contains mostly sensory, visuospatial information without temporal context, which is retrieved automatically and involuntary, e.g. intrusive images or flashbacks. Similarly, Ehlers and Clark's (2000) cognitive model of PTSD suggests data-driven processing (processing sensory impressions) rather than conceptual processing (processing meaning and context) is associated with later intrusions. The nature of trauma memories is determined by the degree of conceptual processing during the trauma, so if mainly data-driven and no conceptual

processing occurs, then the trauma memory is difficult to retrieve intentionally. Depersonalization, derealization and numbing may also impede the integration of the trauma into the autobiographical memory knowledge base.

Peritraumatic Dissociation and PTSD Development

Peritraumatic dissociation refers to dissociation (e.g., numbing, derealization, detachment) occurring during or shortly after trauma. Both retro- and prospective studies report a relationship between peritraumatic dissociation and development or severity of PTSD (e.g., Birmes et al., 2003) or flashbacks (Bremner & Brett, 1997). According to meta-analysis results (Ozer, Best, Lipsey, & Weiss; 2003), peritraumatic processes, specifically dissociation, are stronger predictors of PTSD than pre- or posttraumatic variables. However, many studies are limited by methodological shortcomings. Retrospective studies measure peritraumatic dissociation at the same time as PTSD symptoms, which is problematic as memory is influenced by many factors, like forgetting, attribution of symptoms, over-reporting, and malingering (Candel & Merckelbach, 2004). Indeed, recall of peritraumatic dissociation can be unstable over time, and changes in recall of peritraumatic dissociation are highly correlated with changes in PTSD (Marshall & Schell, 2002; Zoellner, Sacks, & Foa, 2001).

Prospective studies can overcome these problems, but some fail to control for initial PTSD symptoms or trait dissociation (a relatively stable tendency to experience dissociative symptoms), so the extent to which peritraumatic dissociation is an independent predictor of PTSD is unclear (Candel & Merckelbach, 2004). Indeed, some studies did not find a relationship between peritraumatic dissociation and later PTSD after controlling for related factors like initial PTSD symptoms (Marshall & Schell, 2002; Marx & Sloan, 2005), and event-related fears about death and losing control (Gershuny, Cloitre, & Otto, 2003).

To study peritraumatic dissociation more closely, experimental, laboratory based studies are useful. Dissociation can be elicited relatively easily by nonpharmacological induction techniques, in persons with or without a history of psychiatric disorder (Leonard, Telch, & Harrington, 1999; Miller, Brown DiNardo, & Barlow, 1994). Unfortunately, to our knowledge, only a small number of experimental studies have examined the relationship between peritraumatic dissociation and traumatic intrusion development. In two experiments (Kindt, Van den Hout, & Buck, 2005), spontaneously occurring dissociation during an aversive film (measured by the Peritraumatic Dissociative Experiences Questionnaire, PDEQ; Marmar, Weiss, Metzler, & Delucchi, 1996) was related to increased intrusion frequency 4 hours and one week later. However, this relationship was not found in another study using the same design (Kindt & Van den Hout, 2003), and in all experiments intrusion frequency was assessed retrospectively using a one-dimensional visual analogue scale (VAS). Stuart, Holmes and Brewin (2006) found that an intrusion frequency reduction was not accounted for by associated changes in state dissociation (measured by the dissociative states scale, DSS; Bremner et al., 1998). Instead, it was accounted for by a visual-spatial grounding task, hypothesized to use mechanisms that are also used for encoding information into an image-based (or SAM) memory system.

Only two studies have experimentally induced dissociation. Holmes, Brewin and Hennessy (2004) instructed participants to stare at a dot during an aversive film. Interestingly, there were no differences in intrusion frequency in the next week between participants and controls. Across conditions, however, spontaneous increases in state dissociation (measured by DSS scores) were related to more reported intrusions. In a second study (Holmes, Oakley, Stuart, & Brewin, 2006) participants watched an aversive film in a hypnotically induced dissociative state. During half the film depersonalisation and derealization-like suggestions were given. DSS scores showed that dissociation was successfully induced, but participants reported no more intrusive memories of film sections in which they experienced higher levels of dissociation.

Somatoform Dissociation and Trauma

Dissociation is a complicated concept that takes different forms. Studies described thus far have focused on (peritraumatic) 'psychological dissociation', i.e. dissociation manifested in psychological variables, like disruptions in memory, consciousness, and identity. Although already described by Janet (1907), interest in physical dissociative symptoms reappeared only recently in the concept of 'somatoform dissociation' (Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1998). Somatoform dissociation is manifested in somatic variables and concerns a disintegration of somatoform components of experience, and bodily reactions and functions, for example analgesia, and psychogenic seizures. Research has shown psychological and somatoform dissociation to be related but distinct constructs (Maaranen et al., 2005).

Another distinction was made in a recent review study (Holmes et al., 2005). Here, it was theorized that two forms of dissociation can be distinguished: detachment and compartmentalization (Allen, 2001). Detachment refers to an altered consciousness in which the person experiences a sense of separation, for example depersonalization and 'spacing out'. Compartmentalization refers to the separation of mental systems, characterized by "a deficit in the ability to deliberately control processes or actions that would normally be amenable to such control" (Holmes et al., 2005, p. 7), including dissociative amnesia and conversion paralysis. The PDEQ (Marmar et al., 1996), used to operationalize peritraumatic dissociation in the studies described thus far, typically measures detachment. Compartmentalization, incorporating somatoform dissociation, has hardly been studied with respect to PTSD development. This is remarkable because one component of somatoform dissociation, tonic immobility or 'freezing', occurs as a defensive reaction towards life threatening stress in both animals and humans (Gallup, & Maser, 1977; Moskowitz, 2004). Tonic immobility is thought to have developed because many predators lose interest when their prey remains motionless. Tonic immobility during trauma is considered a somatoform dissociative reaction in that it concerns the involuntary disruption of sensory and motor processes (Nijenhuis et al., 1998). Galliano, Noble, Puelch and Travis (1993) found that one third of rape victims reported freezing and were (subjectively) unable to move during the assault. Threat to life was also found to induce peritraumatic somatoform dissociative reactions like analgesia (Pitman, Van der Kolk, Orr, & Greenberg, 1990). To our knowledge, very few studies in-

vestigated the association between somatoform dissociation and PTSD. In one study, inmates who experienced child sexual abuse were found to have elevated somatoform dissociation levels compared to inmates who did not (Dietrich, 2003). In another study, somatoform dissociation was found to be strongly related to current or past PTSD (El-Have, Darves-Bornoz, Allilaire, & Gaillard, 2002). Clearly, the effect of compartmentalization-dissociation on information processing during trauma is still unclear and merits more research.

A previous study showed that a hypnotic technique successfully induced catalepsy, a state of waxy-flexibility and tonic immobility. Both sensory and motor (i.e., somatoform) dissociative symptoms were elicited by this technique, like the cataleptic body part feeling numb, unreal, or not belonging to them, and involuntary tonic immobility (Hagenaars, Roelofs, Hoogduin, & Van Minnen, 2006). Neuroimaging studies also provide evidence for changes in sensory information processing during catalepsy (Grond, Pawlik, Walter, Lesch, & Heiss, 1995). In a total body cataleptic state, the occipital areas (visual and paravisual cortex) became deactivated, while metabolic recruitment was found in the sensori-motor brain structures, corresponding to a shift of attention away from normal sensory input. Furthermore, studies using positron emission tomography (PET) showed hypnotic paralysis to be an effective way to generate a feeling of subjective paralysis (Halligan, Athwa, Oakley, & Frackowiak, 2000) and this has a different neural basis to simulated paralysis (Ward, Oakley, & Frackowiak, 2003). The altered information processing during subjective paralysis or catalepsy may have an effect on encoding other information at that time. Moreover, Bryant and Barnier (1999) found that hypnotic suggestions can alter memory and attributional processes.

Explicit and Implicit Memory in PTSD

Although a detailed review of explicit and implicit memory in PTSD is beyond the scope of this paper, related findings will be discussed briefly. Recent PTSD theories predict enhanced priming, and thus enhanced implicit bias for trauma-related content, resulting from inadequate processing of trauma information (Brewin, 2001; Brewin et al., 1996; Ehlers & Clark, 2000). Combat veterans with PTSD indeed exhibited an implicit bias for combat words on the word stem completion test, whereas veterans without PTSD did not (Zeitlin & McNally, 1991). During dissociation information is processed mainly in the SAM (where memories are difficult to regulate and triggered without voluntary control), leading to enhanced implicit reactions towards trauma-reminders. As less information processing occurs in Brewin's VAM (similar to declarative memory involving representations of facts and events that are subject to conscious recollection; 2001), dissociation during trauma would result in less access to voluntary, explicit memories.

Conclusions and Goal of the Present Study

In conclusion, although peritraumatic dissociation often is considered a strong predictive factor in PTSD development, naturalistic studies are methodologically flawed and findings are at best ambiguous. Experimental studies may provide a good alternative, though previous studies of induced dissociation failed to find the

predicted increase in intrusion. In addition, research has focused on psychological dissociation and detachment rather than, for example, somatoform compartmentalization (akin to the freezing response). In previous investigations, catalepsy successfully induced somatoform dissociative experiences (Hagenaars et al., 2006). However, non-movement alone may be an important factor.

This study aimed to investigate effects of dissociative non-movement and deliberate non-movement during an aversive film on intrusion development. Dissociative non-movement (catalepsy) participants were predicted to experience more intrusive recollections of the film than the other participants. We also explored differences in explicit recall and implicit cognitive bias. Dissociative non-movement participants were expected to show less adequate explicit recall of aversive film details, but enhanced implicit cognitive bias towards film-related words. Following Holmes et al. (2004) we also examined the impact of state dissociation, memory performance and implicit cognitive bias across conditions.

Method

Participants

Eighty-nine students were recruited through university campus advertisements. Nine students were excluded for meeting criteria of a DSM-IV disorder: blood phobia (with or without fainting, $n = 6$), depressive disorder (current or past, $n = 2$), and drug abuse ($n = 1$). One student failed to comply with the instructions during the experiment so was excluded. In sum, 79 students participated: 27 in the control condition (Co), 25 in the non-movement condition (NM), and 27 in the dissociative non-movement condition (DNM). Eleven participants (13.9%) were male and 68 (86.1%) were female. The age range was from 18 to 29 years ($M = 21$ years, $SD = 2.2$). All participants received a token reimbursement of 25-euro.

Material

To model a traumatic experience, we used a ten minute film depicting four traumatic scenes of real-life footage of the horrible aftermath of road traffic accidents, such as dead bodies being moved, injured victims, and car wrecks. A brief commentary introduced each scene, providing background information about the accident and people involved. To prevent fatigue from sitting still too long, one scene (having the lowest distress ratings as judged by independent raters) was removed from the original 5 scenes compiled by Steil (1996). A DVD recorder was used, and a projector (3M, type MP8745) to project the film on a 113 x 88 cm screen.

Measures

Diagnosis

Psychiatric symptoms were assessed by the Structured Clinical Interview for DSM-III-R (SCID-I; Spitzer, Williams, Gibbon, & First, 1992). The SCID-I is a

standardized, semi structured, diagnostic interview for diagnosing DSM-IV psychiatric disorders, which has good reliability (overall kappa's were .61 for current and .68 for lifetime diagnosis). The 12 screening questions of the SCID-I were used initially. If participants endorsed symptoms, then diagnoses were established using the relevant SCID-I sections. SCID-I interviews were conducted by licensed clinical psychologists.

Pre-film measures

Trait dissociation was measured by the Dissociative Experiences Scale – revised version (DES-II; Bernstein & Putnam, 1986; Carlson & Putnam, 1993), a 28-item questionnaire that reflects severity of dissociative experiences like amnesia, depersonalization/derealization, and absorption. Respondents rate what percentage of time (0 to 100) they have each experience. Convergent validity has been found to be very good (overall combined correlation = .67). Internal consistency has also been found to be good (mean alpha = .93; Van IJzendoorn, & Schuengel, 1996). In the present experiment, we controlled for trait dissociation because it is related to both peritraumatic dissociation (Tichenor, Marmar, Weiss, Metzler, & Ronfeld, 1996) and initial PTSD severity (Feeny, Zoellner, Fitzgibbons, & Foa, 2000).

State dissociation. Like Holmes et al., (2004) we used the 19 self-report items from the Clinician Administered Dissociative States Scale (Bremner et al., 1998) as the Dissociative State Subscale (DSS). Items are rated on a 5-point scale from 0 (*not at all*) to 4 (*extremely*). Like the PDEQ, DSS items typically refer to psychological dissociation, mostly addressing depersonalization and derealization. Participants indicate how much they are experiencing each dissociative symptom item 'right now, at this moment'. The total score ranges from 0 to 76. The 19 self-report items show good internal consistency (Cronbach's α = .94; Bremner et al., 1998).

Mood. Participants rated happiness, anxiety, horror, sadness, and anger on an eleven-point scale from 0 (*not at all*) to 10 (*extremely*) to show the film's impact on emotional state. Happiness was reversed scored.

Post-film measures

Mood (peri-film: during and immediately after the film) and *State dissociation* (DSS) were assessed again post-film.

Attention. Attention paid to the film was rated on a visual analogue scale from 0 (*none at all*) to 10 (*extremely*).

Catalepsy. The Catalepsy Questionnaire (CQ; Hagenaars et al., 2006) is a 22 item self-report questionnaire, originally designed to measure catalepsy and co-occurring sensory-motor (i.e., somatoform) dissociative symptoms in both arms. Items are scored from 1 (*not at all*) to 5 (*extremely*). The present version is adapted to reflect experienced symptoms in the entire body instead of the left and right arm, reducing the number of items to 12. The total score ranges from 12 to 60, higher scores indicating more catalepsy and more somatoform dissociation. In the original study (Hagenaars et al., 2006), internal consistency was not calculated because of the small sample size. In the present study, the CQ showed good internal consistency (Cronbach's α = .91).

State somatoform dissociation. The Somatoform Dissociation Questionnaire – Peritraumatic (SDQ-P; Nijenhuis, Van Engen, Kusters, & Van der Hart, 2001) is an 11 item self-report questionnaire designed to measure somatoform dissociation during and/or directly after a traumatic experience. Items are rated on a 5 point scale (from 1 = *did not occur* to 5 = *occurred to a great extent*). In the present study, the SDQ-P had satisfactory internal consistency (Cronbach's $\alpha = .85$).

Demand question. An open ended question was asked about the study's purpose in order to check for demand characteristics.

Follow up measures: Intrusions and Memory

A. Intrusions

Intrusion frequency. For seven days after the film, participants recorded every intrusion of the film using a tabular diary (see also Holmes et al., 2004; Brewin & Saunders, 2001; Davies & Clark, 1998). To check the intrusive character, participants had to describe the content of each intrusion, whether it was image or thought based, and how spontaneous it was. Verbal and written instructions were given about the nature of involuntary intrusions and how to keep the diary. Intrusive images were described as 'spontaneously occurring' rather than deliberate memories of the film. The importance of recording every intrusion was emphasized. They were instructed to carry the diary with them and check whether they had completed their diary at regular times each day. The total number of intrusions was calculated by the experimenter (blind to group membership), by adding up all intrusive images (not thoughts). Three images concerned not the actual film but instead what might have happened. These were included in the total, as intrusive images in PTSD are not necessarily a 'copy' of the original situation (Hackmann, Ehlers, Speckens, & Clark, 2004). Following Davies and Clark (1998) to check diary compliance at follow up, participants rated how often they forgot or were unable to record intrusions from 0 (*not at all true*) to 10 (*extremely true*).

B. Memory characteristics

Explicit memory: cued recall. Following Holmes et al. (2004), a cued recall test of twelve open-end questions assessed explicit memory of the aversive film. The experimenter and an independent second rater (both blind to group membership) rated whether the answer was correct by comparing the participants' answer to a list of correct answers that were established beforehand.

Cognitive bias. Cognitive bias was measured by the implicit Word Stem Completion test (WSC). Participants were told they would be completing a 'free association task'. Their task was to complete 30 word stems as quickly as possible with the first word that came to mind. Later, the experimenter and an independent second rater (both blind to group membership) rated whether the word was related to the film or not. The inter rater agreement between these two raters was 100%. The total score consisted of the number of completed words that were related to the film. Previous research has suggested that the WSC test is a 'truly' implicit memory test (Roediger, Weldon, Stadler, & Riegler, 1992).

Design

Participants were randomly assigned to one of three experimental conditions: dissociative non-movement (DNM), deliberate sitting still (non-movement, NM), or a free to move control condition (Co). All measures were the same across conditions.

Procedure

Randomization occurred before screening. For ethical reasons the aversive nature of the film was described and participants were told that they could terminate the experiment at any time. The 12 SCID-I screening items were used to screen for psychiatric disorder. All participants gave written informed consent.

Participants who passed screening completed the pre-film questionnaires and were told that the experiment was filmed to increase compliance. Then, participants were instructed about the manipulation:

Dissociative non-movement (DNM). Participants were told a special condition would make them stiff and unable to move. If they stopped feeling stiff during the film, they had to focus on this stiffness again. Following Hagenaars et al (2006), one of two researchers, licensed and experienced in inducing catalepsy in psychiatric patients and healthy individuals, put participants in total body catalepsy. First, both arms were put in a cataleptic state by pushing the forearm slightly and slowly up and down. Then, the upper part of the body was made cataleptic by pushing the shoulders slightly forwards and backwards. The feet were firmly placed on the ground, under the knees, so it was difficult to move them. Finally, the head was put in a cataleptic state by pushing the forehead slightly forwards and backwards. All participants entered catalepsy easily.

Non-movement (NM). Participants were told to sit as still as possible and they were not allowed to move. If they noticed they were moving during the film, they had to sit still again. Participants were helped to sit upright, with both feet on the ground, and both arms on the arm rests of the chair.

Control (Co). Participants were told they could sit however they wanted to and move as much as they wished.

All participants were given standardized instructions on how to watch the film (including 'Imagine you are present at the scene', 'Do not close your eyes', and 'Do not look away'). They then watched the film sitting 2 meters from the screen. To check for non-movement, a video camera recorded the participants discreetly.

Two NM participants and one DNM participant were taken out of the room before the film ended because they felt dizzy. These three participants were not excluded, but at follow up they answered only the questions on the explicit memory measure about the scenes they had seen. Because this measure was calculated in percentages, this had no effect on the total score.[1]

After the film had ended, DNM participants were instructed to shake their arms and legs to get out of their cataleptic state. The NM participants were told they were allowed to move again. The Co group was told the film had ended. All participants went to another room to complete post-film measures. They then were debriefed using a standardized debriefing form, which explained that they may or may not experience intrusions. It was emphasized there was no 'optimal reaction'

and the experiment was about what happened to them with respect to intrusions. For ethical reasons, all participants received a phone number of a psychiatrist (fifth author) in case they became too distressed.

One week later, participants completed the follow up measures. The explicit memory test was done at the end of the follow up, in order to avoid contaminating the WSC test. This way, the cognitive bias would be due to the film and not due to recall of the film. At the end all participants were told that experiencing intrusions is a normal reaction to a distressing film. For ethical reasons, they were instructed to contact one of the researchers if they were distressed at any point after the experiment had ended. None did this.

Statistical analyses

The number of participants was predetermined by power analyses using a previous study using this paradigm (Holmes et al., 2005), which showed that respectively 20 and 17 participants per condition were needed to obtain a power level of 80%. To prevent missing effects because of a sample size that was too small, and because of possible dropout or exclusion, we included 10 extra participants per condition.

In analyzing the data, two sets of contrast analyses were conducted: One with the DNM versus the two other groups, and one with the two non-movement groups (NM and DNM combined) versus the free to move control group. This was done because we were especially interested in the additional effect of somatoform dissociation above non-movement. Following Tabachnick and Fidell (2001) general ANOVA effects were not performed and only contrast effects are mentioned for the analyses that concern the specific hypotheses mentioned in the introduction. General ANOVA's are reported in case of explorative analyses or if no specific hypothesis was formulated. Bonferroni adjustments were made if more than one variable was used to test a hypothesis (e.g., in case of the post-film mood ratings). Hierarchical regression analyses were conducted to study predictors of intrusion frequency across groups. Statistical tests were two-tailed, and the criterion for significance was set at .05.

Results

The data were inspected for outliers based on median scores as variables were not normally distributed. Using box plots, four participants were outliers on intrusion frequency, scoring more than 4 times the third quartile above the median. Some of these four participants, but no others, were outliers on other variables as well (DES and pre-film DSS). Therefore, these four participants (one from the Co group, one from the NM group and two from the DNM group) were removed from further analyses.

Baseline differences

At baseline, there were no significant differences between experimental groups in age, DES, pre film DSS ($F < 1$ in all cases), or gender ($\chi^2(2, N = 79) = 1.73$, $p = .42$). No participant answered the demand question correctly, indicating the

purpose of the study was not clear to them. This was a positive finding with respect to minimizing the influence of demand characteristics.

Manipulation check

There was a difference in somatoform dissociative reactions on both the CQ ($F(2,72) = 30.71$, $p < .001$) and the SDQ-P ($F(2,72) = 6.45$, $p = .003$) (Table 4.1). Posthoc contrast analyses showed that the DNM group scored significantly higher than the NM and Co group on the CQ ($F(2,72) = 31.75$, $p < .001$) and SDQ-P ($F(2,72) = 6.45$, $p < .001$), both catalepsy and somatoform dissociation had successfully been induced in the DNM group. No differences were found between groups on the DSS post-film ($F(2, 72) = .22$, $p = .41$), nor on DSS increase from pre to post ($F(2, 72) = 0.68$, $p = .25$), suggesting the manipulation had no effect on psychological dissociation.

During the experiment, participants were observed by the experimenter for instruction adherence. A random selection of videos of the NM and DNM participants were checked by a second independent person. Results of this indicated that the NM and the DNM participants did not move during the film.

Mood ratings significantly increased from pre to post, indicating the film content was distressing ($t(78) > 5.3$ in all cases). In particular horror (PTSD criterion A2 in the DSM-IV) showed a large increase from pre ($M = .57$, $SD = 1.37$) to post ($M = 5.80$, $SD = 2.26$), indicating the film was distressing, and quite horrifying. Subjective ratings of diary compliance indicated that participants believed they recorded intrusions accurately ($M = 2.03$, $SD = 2.33$). There was no difference between groups in reported diary compliance ($F(2, 72) = 0.85$, $p = 0.43$).

Table 4.1 *Means (SDs) of dissociation and outcome measures (N = 75)*

Measure	Condition					
	Co $n = 26$		NM $n = 24$		DNM $n = 25$	
	M	*SD*	*M*	*SD*	*M*	*SD*
CQ**	24.96	7.06	29.50	10.14	43.40	8.80
SDQ-P**	14.38	4.78	14.63	3.92	18.84	5.89
DSS post	4.46	7.02	3.46	3.78	4.36	6.27
DSS change	2.00	3.35	1.33	2.97	2.60	4.81
Intrusive images*	1.04	1.40	1.92	2.17	1.88	1.51
Memory and attention:						
Attention**	8.58	0.80	8.90	1.12	7.60	1.65
Explicit recall[a]	52%	17%	51%	13%	50%	15%
Cognitive bias[b]	10.23	1.70	10.71	2.42	10.32	2.25

Note: Co = control; NM = non-movement; DNM = dissociative non-movement; CQ = Catalepsy Questionnaire; SDQ-P = Somatoform Dissociation Questionnaire - Peritraumatic; DSS = Dissociative State Subscale. [a]Percentage correct answers. [b]Number of film-related words on the word stem completion test (WSC) *$p < .05$. **$p < .001$.

Experimental group effects

Because specific hypotheses were formulated, planned contrast analyses were performed to test the hypotheses that (1) the DNM group experienced more intrusive images than the Co and the NM group combined (Co + NM); (2) the two non-movement groups combined (NM + DNM) experienced more intrusive images than the Co group.

Contrast analyses revealed a significant difference in intrusive images between the NM + DNM combined group versus the Co group ($t(72)= 2.07$, $p = .02$, see Table 4.1 for mean scores on outcome measures per group). Participants who did not move during the film (NM + DNM group) reported more intrusive images than participants who could move freely. There was no significant difference in frequency of intrusive images between the DNM group versus the Co + NM combined group ($t(72)= 0.96$, $p = .34$).

Because no specific hypotheses were formulated on the other variables, one-way ANOVA analyses were performed. Post-film mood ratings (mood during and directly after the film) showed no differences between groups ($F(2, 72) < 1$ in all cases). Interestingly, a significant effect appeared in the amount of attention participants paid to the film ($F(2, 72) = 7.36$, $p = .001$). Post hoc contrast analyses revealed that DNM participants had more difficulty paying attention to the film compared to the CO + NM combined group ($t(72) = -3.75$, $p < .001$). Contrast analyses between the NM + DNM combined group versus the Co group revealed no significant differences in attention ($t(72) = 1.10$, $p = .28$). No significant difference was found between groups in explicit memory ($F(2, 72) = .11$, $p = .90$), or cognitive bias (WSC test; $F(2, 72) = .35$, $p = .71$).

Dissociation and memory effects across all conditions

It is possible that, like in previous experimental studies, state somatoform dissociation occurred naturally, regardless of experimental condition. Regression analysis was used to study whether state somatoform dissociation had an effect on intrusion frequency if non-movement, experimentally induced dissociation and trait dissociation were considered. We controlled for these variables, because we wanted to make sure that, if an effect was found, this effect was indeed due to state somatoform dissociation and not to experimental condition or pre-film dissociation. In line with previous analyses, experimental condition was coded as paired contrasts, i.e., presence or absence of dissociation ('experimental dissociation'), and presence or absence of non-movement ('experimental non-movement'). Experimental non-movement was entered in the first block of the regression analysis, followed by experimental dissociation and DES in the second block, followed by SDQ-P in the third block. The total frequency of intrusive images was entered as dependent variable. In the final model, experimental non-movement predicted intrusive images ($\beta = .26$, $p = .02$), whereas SDQ-P, DES, and experimental dissociation did not (SDQ-P: $\beta = .01$, $p = .96$; DES: $\beta = .16$, $p = .19$; experimental dissociation: $\beta = -.01$, $p = .92$).

To study the effect of naturally occurring somatoform dissociation on memory measures, we computed two hierarchical regression analyses with explicit memory and bias to trauma related words as dependent variables. Besides experimental non-movement (first block) and experimental dissociation and trait dissociation (second

block), we also entered attention (third block, together with SDQ-P), because it could possibly have affected memory performance. In predicting recall, the model became significant only after the third block of variables was entered ($\Delta R^2 = .14$, $p < .01$). SDQ-P and attention made a significant contribution (SDQ-P: $\beta = -.28$, $p = .03$; attention: $\beta = .33$, $p = .01$), experimental non-movement, experimental dissociation and DES did not: Less somatoform state dissociation and more attention were associated with better explicit recall. In predicting bias to trauma related words, only SDQ-P made a significant contribution (SDQ-P: $\beta = .30$, $p = .02$), experimental non-movement, experimental dissociation, DES and attention did not: More somatoform state dissociation was associated with enhanced cognitive bias to trauma related words. However, there was only a trend in significance for the whole model ($\Delta R^2 = .07$, $p = .08$).

In addition, bivariate correlations were calculated to look at the relationship between different memory tests and intrusion frequency. Cognitive bias to trauma-related words was related to intrusion frequency ($r = .26$, $p = .03$), explicit recall was not ($r = -.06$, $p = .63$).

Discussion

The main finding of the present study is that the combined non-movement group (participants in the dissociative non-movement group who watched the film in a cataleptic state, and participants instructed to sit still deliberately) experienced more intrusive images than free to move control participants. Intriguingly, this suggests it may not be the dissociative state of not being able to move (dissociative freezing), but the non-movement itself that has an effect on the development of intrusive images. It is unlikely that the difference between the non-movement and free to move control participants is due to levels of attention, because only the cataleptic and not the deliberately non-moving participants stated that they paid less attention to the film. Instead, it may be that deliberate non-movement is related to feelings of uncontrollability (an important factor in the development of PTSD; Foa, Zinbarg, & Rothbaum, 1992) in a similar way that dissociative non-movement (catalepsy) is. Somatoform dissociation, evoked by the hypnotic technique catalepsy, may not add any predictive power to the effect of non-movement. Non-movement itself may be an evolutionary developed response to danger, strongly connected to fear and helplessness (Moskowitz, 2004). The film in combination with non-movement may have triggered this defense-reaction. In previous studies such a 'freeze' reaction was associated with peritraumatic dissociation (Nijenhuis et al., 1998), but it may be associated primarily with non-movement per se. It would be interesting to compare non-movement with dissociative non-movement with respect to heart rate, as decreased heart rate seems to be associated with the freeze reaction, and increases in intrusion frequency (Holmes et al., 2004; Moskowitz, 2004).

Until recently, it was commonly assumed that there is a relationship between peritraumatic dissociation per se and later intrusion development. However, studies of experimentally induced dissociation (Holmes et al., 2004; Holmes et al., 2006)

and controlled prospective studies (e.g., Marshall & Schell, 2002; Marx & Sloan, 2005) have failed to find this relationship. Because most studies examined psychological dissociation, the present experiment aimed to examine catalepsy, a form of somatoform dissociation often linked to trauma. Like some recent studies on psychological dissociation, induced catalepsy was not related to intrusion frequency. It is possible that experimentally induced analogues of peritraumatic dissociation differ in yet undetermined ways from spontaneous dissociation evoked by real traumatic events. However, considering recent well-controlled prospective studies, these findings may also indicate that the effect of peritraumatic dissociation is overestimated and possibly could be explained by the variance shared with other predictors, like early PTSD symptoms.

Our second hypothesis, that catalepsy participants would show different memory characteristics, was not confirmed by the data. No differences between groups were found on explicit recall or implicit bias to trauma related words. However, analyses across experimental conditions revealed state somatoform dissociation and attention both predicted explicit memory. The fact that poorer attention leads to poorer recall is not surprising, because less information would be stored in memory. The negative association between somatoform dissociation and explicit recall is in line with the dual representation theory (Brewin, Dalgliesh, & Joseph, 1996), which states that dissociation hinders access to voluntary, explicit memories.

Higher state somatoform dissociation across groups led to enhanced implicit cognitive bias to trauma related words. Enlarged bias to trauma related words was associated with an increase in intrusive images, although curiously state somatoform dissociation was not. The former finding is in line with the dual-representation theory (Brewin et al., 1996), as SAM is thought to be associated with both enhanced implicit memory and intrusive images. Similarly, Ehlers & Clark's (2000) model suggests that dissociation is associated with data-driven processing of trauma information, leading to perceptual priming for trauma-related stimuli and poor intentional recall of trauma information.

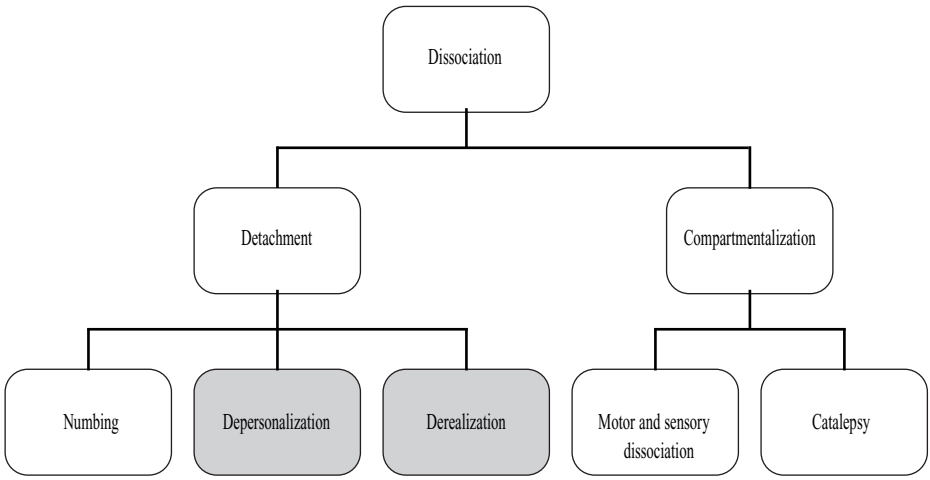
Although the study has methodological strengths, such as experimentally inducing somatoform dissociation and (unlike previous studies) controlling for trait dissociation and non-movement, there are several limitations. For example, participants were mostly female students. Intrusions, as in previous studies, were measured by potentially unreliable self-report, although it is unlikely the results were influenced by demand characteristics, as no participant guessed the purpose of the experiment correctly. Furthermore, these would be present across groups and could not explain differing effects. Future studies might improve the recording method, for example by contacting participants daily, and also assess somatic activity in order to verify non-movement. Furthermore, although the experimental design allowed us to study intrusion development in a highly controlled setting, the ecological validity remains uncertain. Although participants perceived the film as horrifying, the film may not have generated sufficient distress to approximate trauma. Clearly, for ethical reasons, truly traumatic event cannot be used, and therefore, it would be interesting to replicate the results in clinical studies. Finally, the manipulation used in the present

study induced catalepsy and evoked other symptoms of somatoform dissociation, but did not have an effect on psychological dissociation. The effects of psychological dissociation – a type of dissociation commonly measured in the earlier retrospective studies concerning peritraumatic reactions – may be different than the effects of somatoform dissociation. This raises the important question of what we mean by the term 'dissociation' in the trauma field, and the need to delineate different sub-types (Holmes et al., 2005). It would be interesting to compare experimental manipulations of psychological dissociation and somatoform dissociation. Because this study was the first to investigate the association between experimentally manipulated somatoform dissociation and intrusions, a replication of the present findings would be helpful.

In conclusion, the present study has brought us three important findings. First, an experimental catalepsy induction proved successful in inducing somatoform dissociation. Second and most importantly, we found evidence to suggest that the impact of somatoform dissociation on intrusion development may not be due to somatoform dissociation per se but to non-movement (akin to freezing). Third, state somatoform dissociation across groups was related to enhanced implicit cognitive bias towards trauma related words and to poorer explicit recall of trauma information.

Chapter 5

The Uniqueness of Trauma Memory: A Comparison of Autobiographical Memories in PTSD, Panic Disorder, and Healthy Controls



Hagenaars, M.A., Van Minnen, A., Hoogduin, C.A.L., & Verbraak, M. (2007). The uniqueness of trauma memory: A comparison of autobiographical memories in PTSD, panic disorder, and healthy controls. Manuscript submitted for publication.

Abstract

Trauma memory in patients with posttraumatic stress disorder (PTSD) is often thought to be unique, due to inadequate encoding of information during trauma. To test this hypothesis, trauma and panic memories were compared in two separate studies. In study 1, scripts of PTSD trauma memories (n = 21) were compared with scripts of panic memories of patients with panic disorder with agoraphobia (PDA; n = 25) using a narrative rating scale. The two groups did not differ in the intensity of reliving the event or in the level of disorganization of their memories. In study 2, PTSD trauma memories (n = 59) were compared with PDA panic memories (n = 58), and trauma memories of healthy controls (n = 135) on self-reported intrusion characteristics and disorganization. PTSD memories had more intrusion characteristics than memories of the other two groups, although PDA memories had more intrusion characteristics than the controls' trauma memories. Relative to the controls, PTSD and PDA memories were disorganized, and peritraumatic dissociation and current memory-associated dissociation were high. Implications of these results are discussed.

Keywords: Trauma memories, PTSD, Panic Disorder.

Introduction

An increasing body of literature addresses the question whether trauma memories are unique. In other words, are traumatic events processed and stored different than other events or is there just a quantitative difference and are trauma memories just at one extreme end of a continuum of memory processing and storage? The present paper describes two studies that were conducted to probe the uniqueness of trauma memories in patients suffering from posttraumatic stress disorder (PTSD) by comparing them to the memories of others, using a narrative rating and a meta-memory (perceived memory) method respectively. Each study compares the various memories as to two characteristics that are thought to be distinctive for PTSD trauma memories: 1) reliving or intrusive and sensory elements, and 2) disorganization.

Theories on the development of PTSD generally posit that the reexperiencing symptoms in PTSD are the result of trauma information not being processed adequately (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989). However, theories differ in whether they regard this process as unique for PTSD or whether it is a general process that is only more extreme in more stressful situations. For example, fear network models (e.g., Foa, Steketee, & Rothbaum, 1989) propose that a traumatic memory is an ordinary memory but with a particular structure. These theories elaborate on Lang's fear conditioning theory (1979) that postulated proposed that frightening events are represented in a fear memory, i.e., through interconnections between nodes representing information about the stimulus, response, and meaning of an event. In PTSD this strong fear memory can be integrated into 'ordinary' memory by creating associations between elements of the fear structure and new information that is incompatible with the fear memory

(Foa et al, 1989). The reverse is also hypothesized, namely that in PTSD trauma-related memories are represented qualitatively differently from ordinary memories. The traumatic event is, as it were, split off from the general associative and autobiographical memory (e.g., Van der Kolk & Van der Hart, 1991). The trauma memories are processed through distinct biochemical pathways, resulting in the trauma memory being disconnected from other autobiographical memories, being fragmented and disorganized, and lacking temporal context. Still other theories distinguish a memory for perceptual and a memory for verbal information (Brewin et al., 1996; Conway & Pleydell-Pearce, 2000). These latter theories assume a link between PTSD intrusions and perceptual memory, but do not necessarily predict a unique disorganization of the memory in PTSD.

Because the current paper addresses intrusion/sensory features and disorganization, we will briefly summarize the main findings pertaining to these memory characteristics, starting with intrusion/sensory features. Several studies exploring sensory impressions in the trauma memories of PTSD patients found that intrusions in PTSD typically consisted of sensory information, mainly images (e.g., Ehlers et al., 2002). PTSD trauma memories were also characterized by more self-reported sensory impressions than the trauma memories of subjects without PTSD (Berntsen, Willert, & Rubin, 2003). Michael, Ehlers, Halligan, and Clark (2005) found that intrusions in PTSD patients contained more sensory experiences than intrusions in participants without PTSD in one study, but not in another. Note that these studies concerned intrusive memories, which may have a different characteristics than deliberately recounted memories. Rubin, Feldman, and Beckham (2004) found self-reported sensory impressions of the trauma memory to be related to PTSD symptoms. The vivid perceptual content of PTSD intrusions has also been studied in relation to therapy. Vividness gradually faded in the course of therapy, although unfortunately, none of the studies used a control group without PTSD (Hackmann, Ehlers, Speckens, & Clark, 2004; Rauch, Foa, Furr, & Filip, 2004). Moreover, the change in self-reported vividness was not related to a reduction in PTSD symptom severity (Rauch et al., 2004). Furthermore, sensory impressions in initial PTSD memories did not predict PTSD one month later (Engelhard, Van den Hout, Kindt, Arntz, & Schouten, 2003), suggesting it may not be a PTSD-specific memory characteristic.

Whereas the association between sensory impressions, PTSD and trauma memory have frequently been studied, other factors have not received as much scientific attention. One study reported interesting results: distress caused by intrusions, and their 'here and now' quality ('nowness') and lack of context predicted PTSD severity six months after the trauma (Michael et al., 2005). In addition, in an effect study Hackmann et al. (2004) found that the distress and nowness of intrusive memories gradually faded during therapy.

In sum, there seems to be ample evidence to suggest that PTSD trauma memories are characterized by vivid intrusion elements. Yet, studies in student populations have found sensory components to be present in positive memories too (Porter & Birt, 2001), indicating that sensory impressions may be related to highly emotional and not solely to traumatic events. We clearly need more research that actually

compares PTSD trauma memories with memories of other high-impact events. For instance, studies of memory characteristics using other clinical populations that experienced highly emotional or fearful events are lacking.

A second line of research has concentrated on the disorganization and fragmentation of trauma memories. Because these two constructs are used interchangeable and probably denote the same concept, we will discuss the findings on the two together. The results on meta-memory judgment studies on disorganization and fragmentation are inconsistent. In a cross-sectional study self-reported disorganization was higher in trauma survivors with current PTSD than in those without PTSD, with intermediate scores for those with past PTSD (Halligan et al., 2003). In contrast, Berntsen et al. (2003) found no difference in the fragmentation of the trauma memories of PTSD and non-PTSD individuals. Rubin, Feldman, and Beckham (2004) found that self-reported fragmentation did not increase with higher trauma-relatedness of the event or PTSD severity. Initial self-reported fragmentation was not related to PTSD severity at six months in one study (Murray, Ehlers, & Mayou, 2002), but it mediated the relationship between peritraumatic dissociation and PTSD symptoms at six months in another study (Engelhard et al., 2003).

Disorganization and fragmentation have also been associated with the development of PTSD in narrative coding studies. Although initial self-reported fragmentation did not relate to later PTSD severity, initial rater-assessed fragmentation did (Murray et al., 2002). In their prospective study Halligan et al. (2003) found both self-reported and rater-assessed disorganization to predict subsequent PTSD. In addition, in their cross-sectional study rater-assessed disorganization was higher in both current and past PTSD trauma narratives than in non-PTSD trauma narratives. However, as the initial degree of narrative articulation (reading level) proved related to later PTSD severity (Amir, Stafford, Freshman, & Foa, 1998), it is possible that there is a relationship between intelligence, narrative disorganization, and PTSD development.

If disorganization is typical of PTSD memories, one would expect it to disappear after adequate treatment. Organization in PTSD narratives indeed improved after treatment (Foa, Molnar, & Cashman, 1995). In a replication study, however, it was shown that the changes in organization and fragmentation were more likely attributable to the repeated recounting of the trauma during treatment than the result of recovery (Van Minnen, Wessel, Dijkstra, & Roelofs, 2002). Moreover, fragmentation was not consistently related to therapeutic recovery, and it did not consistently discriminate between traumatized persons with and those without PTSD (Zoellner & Bittenger, 2004). Based on the above, one must conclude that findings on fragmentation and disorganization are at least inconsistent and that, hence, solid empirical evidence for PTSD trauma memories being disorganized and fragmented is lacking. Moreover, even though some findings point to elevated disorganization and fragmentation, this still does not imply these characteristics are unique to PTSD. They may, for instance, also or alternatively result from current fear experienced while recounting the event, or they may reflect general, cognitive features (e.g., low intelligence) or depressive symptoms typical of PTSD patients.

In conclusion, the results of research into the distinctive characteristics of

trauma memories in PTSD are ambiguous and sometimes even contradictory. In their review, Zoellner and Bittenger (2004) pointed out that in order to reliably establish the uniqueness of trauma memory in PTSD, it should be compared to and differ from memories of three control groups, i.e., (1) trauma memories of trauma victims without PTSD, (2) memories of other distressing, emotional but non-traumatic events, and (3) memories of events that are associated with the onset of other forms of psychopathology. Especially with regard to the last area, little research has been done. To fill this gap, we opted to include patients with panic disorder with agoraphobia (PDA) as a control group in the current studies. The rationale for that choice is that a panic attack resembles a traumatic experience in that it involves an unexpected, intensely fearful experience, during which many patients believe their lives are in danger. PDA also involves fear conditioning after the first panic attack. In addition, because meta-memory and rating procedures do not necessarily correspond, we used both procedures in our studies. If trauma memories of PTSD patients are special, they should feature more reliving, more intrusion- and sensory elements, and more disorganization than the panic memories of PDA patients.

Study 1

In our first study, we used a narrative rating method to establish the uniqueness of trauma memories in PTSD. Because the memories were recounted intentionally, it was impossible to assess intrusion characteristics like uncontrollability and intrusiveness. Instead, we assessed the intensity of reliving, which showed an overlap with certain intrusion characteristics like vividness and sensory impressions.

Method

Participants
Forty-six patients who were waiting to start cognitive behavior therapy at an outpatient clinic specialized in anxiety disorders participated voluntarily in the present study. Twenty-one patients met the DSM-IV criteria for PTSD without comorbid current or lifetime panic disorder with agoraphobia (PDA) and 25 patients met the DSM-IV criteria for PDA without comorbid current or lifetime PTSD. All patients had Dutch as their native language (see Table 5.1 for demographic and psychopathological variables). The two patient groups did not differ in age ($t(44) = -.76$, $p = .45$), or gender ($\chi^2(1, N = 46) = 2.49$, $p = .12$).

Material
In accordance with a standardized prolonged exposure procedure each patient was requested to close his/her eyes and to recollect the traumatic event (PTSD) or worst panic attack (which most often concerned the first; PDA patients) aloud, as vividly as possible, and in first person and present tense during several minutes. This exposure

Table 5.1 *Means and standard deviations of the demographic and psychopathology variables for PTSD and PDA patients in study 1 (N = 46)*

	Group	
	PTSD *n* = 21	PDA *n* = 25
Gender		
Male	9.5%	0%
Age		
M (years)	35	37
SD (years)	12	11
PSS		
M	25.57	-
SD	8.44	
MI		
M	-	2.24
SD		0.87

Note: PTSD = posttraumatic stress disorder; PDA = panic disorder with agoraphobia; PSS = PTSD Symptom Scale; MI = Mobility Inventory (alone and accompanied).

procedure was done by the two independent experimenters. The recollections were digitally recorded and one-minute fragments were selected, starting from the first perception of threat and panic, respectively. Because it was found that only the worst part of PTSD narratives was accompanied by flashbacks (Hellawell & Brewin, 2004), an independent rater checked whether the selected fragments indeed concerned the most fearful part of the narrative. This procedure was adhered to for both patient groups, resulting in 46 fragments of 21 trauma and 25 panic attack recollections. All one-minute fragments were then transcribed, including non-word utterances like 'uh', and the resulting scripts offered to eight independent raters (see Procedure section) in randomized order.

Measures

Mini-International Neuropsychiatric Interview (MINI). DSM-IV diagnoses were established by means of the MINI (Sheehan et al., 1998), a structured interview based on DSM-IV and ICD-10 criteria. Interrater reliability of the MINI is good (all diagnostic subscales > .75). In the current study all MINI interviews were conducted by trained psychologists.

PTSD Symptom Scale (PSS). The PSS (Foa et al., 1993) is a 17 item self-report questionnaire used to assess PTSD as defined in the DSM-IV. The total score (range: 0 - 51) is the sum of the subscales reexperiencing, avoidance and arousal, and reflects the severity of PTSD symptoms.

Mobility Inventory for Agoraphobia (MI). Severity of agoraphobic avoidance behavior was assessed using the selt-report MI (Chambless, Caputo, Jasin, Gracely, & Williams, 1985). Its 27 items are rated on a 5-point scale. The MI has a high

internal consistency (Cronbach's α = 0.91-0.97) and its concurrent as well as its construct validity have been shown to be good (Chambless et al., 1985).

Memory Characteristics–Rating Scale. We were forced to develop an instrument for the current study specifically because no existing validated scales measure the memory features that we target. Therefore, in the current study, memory characteristics were measured using a 20-item rating scale that addressed features that are thought to characterize trauma memories. The items probe various emotions, organization, vividness, and sensory impressions. All items were rated on an 11-point scale, ranging from 0 (*Completely absent*) to 10 (*Very much present*). Factor analyses showed the scale to measure two factors: *Reliving* (8 items: involvement, participation in the memory, vividness, sensory impressions, arousal, fear, helplessness, and physical symptoms); and *Disorganization* (6 items: logical order, fragmentation, containing essential information, coherence, organization, and completeness). The remaining 6 items did not load on any of the factors and were hence excluded from further analyses. Internal consistency of the 2 subscales proved excellent (Reliving: Cronbach's α = .85; Disorganization: Cronbach's α = .90).

Procedure

After having been interviewed to establish DSM-IV diagnoses, the patients gave their written informed consent and completed the questionnaires. They then recounted their traumatic event or panic attack. After one-minute sections of the recollections had been selected, eight independent raters, then evaluated the one-minute transcriptions using the Memory Characteristics rating scale. The raters were all female students who all studied psychology at the Radboud University Nijmegen. They had been told they were going to rate recollections of patients, but were naïve regarding their content. They practiced the procedure on two scripts that were not used in the present study. The actual scripts were offered to the raters group-wise, but they had to rate the scripts individually and they were not allowed to discuss their ratings. The scripts were offered one at the time, and each rater judged every script according to the following procedure: after having read the script, they proceeded rating it without being allowed to consult the script, thus leaving items blank when in doubt. When all raters had finished scoring the script, they could read it once more while completing the skipped items. To ensure optimal concentration the raters took a break every hour. The entire procedure took approximately eight hours.

Statistical analyses

Multivariate analyses were used to compare the two groups on the selected variables.

Results

Interrater reliability

The intraclass coefficient was high for the Reliving subscale (.84), and adequate for the Disorganization subscale (.72).

Group differences in memory characteristics between groups

The model proved not significant (Pillai's Trace, $F(2, 42) = .69$, $p = .51$, see Table 5.2). PTSD and PDA patients did not differ on reliving ($F(1, 43) = 1.09$, $p = .30$) or memory disorganization ($F(1, 43) = 1.16$, $p = .29$).

Table 5.2 *Means and standard deviations of the memory characteristics for PTSD and PDA patients in study 1 (N = 46)*

	PTSD $n = 21$		PDA $n = 25$	
	M	*SD*	*M*	*SD*
Reliving	51.75	.83	53.78	.78
Disorganization[a]	37.05	.69	38.44	.64

Note: PTSD = posttraumatic stress disorder; PDA = panic disorder with agoraphobia.
[a]Unlike in study 2, higher scores indicate less disorganization.

Discussion

Study 1 aimed to assess memory characteristics by narrative coding. No differences were found between PTSD and PDA memories in rater-assessed reliving intensity. This may mean that reliving is associated with recounting any highly emotional experience and not necessarily with recounting traumatic events only. Neither did the study uncover any differences between the PTSD and PDA memories as regards disorganization: PTSD memories were rated as equally coherent and (dis-) organized as PDA memories.

Because earlier studies have shown that meta-memory and actual memory do not necessarily correspond (Kindt & Van den Hout, 2003), we conducted a second study that used a meta-memory design instead of narrative-rating approach.

Study 2

The results of study 1 suggested that, compared to PDA panic memories, PTSD trauma memories may not be distinct in their level of reliving or disorganization during deliberate recounting. In study 2 we aimed to replicate these findings and to extend them by using an extra control group of healthy participants. In this second study we hence compared the memories of PTSD patients with those of PDA patients and those of healthy participants, by using a meta-memory judgment method. The memories were compared on two dimensions: intrusion characteristics, and disorganization. In addition, because the intrusive memories of PTSD patients include mainly visual and/or bodily sensations (Ehlers et al., 2002; Hackmann et al., 2004), and since memories of panic attacks may also contain bodily sensations, we decided to examine these two sensory qualities more closely in a separate analysis.

Peritraumatic and present dissociation are often associated with trauma and PTSD and with the formation of a distinct traumatic memory by impairing adequate encoding of information. Therefore, differences in (peritraumatic) dissociation between the groups under study are investigated.

Method

Participants

A total of 263 adult volunteers completed all questionnaires (see below). Participants consisted of three groups: 59 patients with PTSD, 58 patients with PDA, and 146 healthy participants (further referred to as 'controls'). The patients were again recruited from an outpatient clinic and inclusion was based on the same criteria as employed in the first study. Demographic and psychopathology variables are listed in Table 5.3. The controls were recruited at the university campus through advertisements. Controls were excluded if they met the DSM-IV criteria for PTSD or PD(A). Eleven controls were excluded based on this criterion, resulting in a total number of 135 controls and a total sample of 252 participants. There were significant differences between the three groups in age ($F(2, 249) = 72.17$, $p < .001$), education ($F(2, 248) = 132.39$, $p < .001$), and gender ($\chi^2(2, N = 252) = 11.83$, $p < .01$), with the controls being younger and more highly educated, and containing more women than the two patient groups. There was no difference in comorbidity between the PTSD and PDA patients ($t(113) = -.83$, $p = .41$).

Measures

DSM-IV diagnoses and PTSD symptoms were established by mean of the *MINI* (Sheehan et al., 1998) and the *PSS* (Foa et al., 1993) respectively.

Trauma exposure was assessed by the items of the *Clinician-Administered PTSD Scale* (*CAPS*; Blake et al., 1995) that address the PTSD criteria A1 and A2.

Panic and Agoraphobia Scale–Self-Administered (PAS). The PAS (Bandelow, 1995; Bandelow, 1999) was used to assess the severity of the panic disorder and agoraphobia. The 13 items are rated on a 5-point scale and measure panic attack frequency, agoraphobic avoidance, anticipatory anxiety, impairment in social and occupational functioning, and worries about health.

Memory Characteristics-Questionnaire. In accordance with Halligan et al. (2003) we gauged Intrusion characteristics and Disorganization using a self-report questionnaire that measures. Participants were first asked to write down their trauma (PTSD), their first or most frightening panic attack (PDA), or the most frightening situation they had ever experienced (Controls). Each participant then received the following written instructions: "Keep the *traumatic event* / *panic attack* / *frightening situation* [there were three versions of this questionnaire, MH] you have just written down in mind when answering the following questionnaire. Each item refers to your memory of this specific situation." Factor analyses showed the scale to comprise 2 factors: *Intrusion characteristics* (8 items: intrusiveness, vividness, fear, arousal, physical reactions, uncontrollability, nowness, and sensory impressions), and *Disorganization*

(6 items: logical order, fragmentation, containing essential information, coherence, organization, and completeness). Subsequent reliability analyses revealed an alpha of .68 and .80 for the Intrusion and Disorganization scales, respectively. In addition, one item assessed peritraumatic dissociation (following Ehlers, Mayou, & Bryant, 1998) and one item depersonalization and/or derealization during memory retrieval. The latter will further be referred to as 'memory-associated dissociation'.

Table 5.3 *The demographic and psychopathology variables for the PTSD and PDA patients and control in study 2 (N = 252)*

	Group		
Variable	PTSD *n* = 59	PDA *n* = 58	Controls *n* = 135
Gender*			
Male	35%	36%	16%
Age*			
M (years)	36	37	23
SD (years)	11	11	6
Education*			
Low (%)	25.4%	31.0%	1.5%
Medium (%)	41.7%	43.1%	0%
High (%)	31.7%	25.9%	98.5%
Comorbidity			
One or more comorbid DSM-IV disorder(s)	44%	38%	-
PSS*			
M	22.41	-	3.17
SD	8.44		3.27
PAS*			
M	-	26.79	3.63
SD		11.96	2.39
Trauma (*n*)			
Road traffic accidents	10.2%	-	13.3%
Other accidents	3.4%		11%
Witnessing death or serious accidents	3.4%		4.4%
Sexual assault	30.5%		3.7%
Nonsexual assault	30.5%		19.3%
Witnessing (sexual) assault	5.1%		-
War	3.4%		-
Death of a loved one	-		17.8%
Serious medical condition or surgery	-		9.6%
Miscellaneous	13.6%		20.7%

Note: PTSD = posttraumatic stress disorder; PDA = panic disorder with agoraphobia; PSS = PTSD Symptom Scale; PAS = Panic and Agoraphobia Scale. *$p < .01$.

All 16 items were rated on a 4-point scale ranging from 0 (*Never*) to 3 (*All the time*). Test-retest reliability was good for Intrusion characteristics (r = .86), Disorganization (r = .82), and the complete questionnaire (r = .86).

Procedure

The participants that met the study's inclusion criteria were asked to complete after having given their written informed consent. One week later, they again completed the Memory Characteristics Questionnaire to establish test-retest reliability.

Statistical analyses

Multivariate and univariate analyses of variance were used to compare the memories on intrusion characteristics and disorganization. Age, gender, and education were entered as covariates because these variables differed significantly between groups. Where significant effects emerged, pairwise post-hoc comparisons with Bonferroni correction were performed.

Results

Intrusion characteristic and disorganization

MANCOVA analyses showed a significant overall main effect for group, Pillai's Trace $F(4, 478) = 21.17$, $p < .001$). Univariate analyses showed significant group differences for Intrusion characteristics ($F(2, 239) = 45.91$, $p < .001$) and Disorganization ($F(2, 239) = 4.54$, $p = .01$; see Table 5.4). Pairwise post-hoc comparisons showed the PTSD trauma memories to feature more intrusion characteristics than both the PDA panic memories and the trauma memories of the healthy controls ($p < .001$ in both cases). Interestingly, the PDA panic memories showed more intrusion characteristics than the trauma memories of controls ($p < .001$). The post-hoc comparisons revealed unexpected differences on Disorganization: the PTSD trauma memories did not differ from the PDA panic memories on this factor ($p = 1.00$). Both types of memories (PTSD: $p = .04$; PDA: $p = .01$) were significantly more disorganized than the trauma memories of the controls.

In addition, two sensory qualities were submitted to univariate analyses: *images* and *bodily sensations*. Groups were found to differ with respect to both variables (images, $F(2, 244) = 19.59$, $p < .001$; bodily sensations, $F(2, 244) = 77.98$, $p < .001$). Interestingly, pairwise post-hoc comparisons showed the trauma memories of both the PTSD patients and the controls to include significantly more images than the PDA panic memories ($p < .001$ in both cases). The memories of the PTSD patients and the controls were comparable with respect to containing images ($p = .18$). As to the differences in bodily sensations, the memories of PTSD and PDA patients both included more such sensations than the trauma memories of the controls ($p < .001$ in both cases). There was no difference in bodily sensations between the memories of PTSD and PDA patients ($p = 1.00$).

Table 5.4 *Estimated means (SDs) of all memory characteristics for each group in study 2 (N = 252)*

Measure	Group					
	PTSD *n* = 59		PDA *n* = 58		Controls *n* = 135	
	M	*SD*	*M*	*SD*	*M*	*SD*
Intrusion**	1.63	.07	1.10	.07	.70	.05
Disorganization[a]**	.73	.08	.77	.08	.45	.06
Sensory impressions						
Images**	2.35	.12	1.35	.12	2.08	.08
Bodily sensations**	.99	.07	.92	.07	.11	.05
Present dissociation*	1.03	.14	.93	.14	.30	.10
Peritraumatic dissociation**	1.36	.16	1.49	.16	.73	.11

Note: PTSD = posttraumatic stress disorder; PDA = panic disorder with agoraphobia. [a]Higher scores indicate more disorganization. *$p < .05$. **$p < .01$.

Dissociation

Groups differed on peritraumatic dissociation ($F(2, 240) = 6.27$, $p < .01$). PTSD ($p = .02$) and PDA patients ($p < .01$) reported higher levels of peritraumatic dissociation than the controls, with these levels being similar in the two patient groups ($p = 1.00$). The three groups also differed as to the extend of their memory-associated dissociation ($F(2, 240) = 7.22$, $p < .01$). Again, both the PTSD and PDA patients experienced more dissociation while remembering the event than the controls ($p < .01$ in both cases), while the two patient groups did not differ from each other ($p = 1.00$). Thus, peritraumatic dissociation and memory-associated dissociation characterized both PTSD trauma and PDA panic memories.

Regression analyses across groups revealed that memory-associated dissociation was related to intrusion characteristics ($\beta = .54$ and $p < .001$), whereas peritraumatic dissociation was not ($\beta = .05$ and $p = .47$). Neither peritraumatic dissociation ($\beta = .11$, $p = .15$), nor memory-associated dissociation ($\beta = .07$, $p = .37$) predicted disorganization.

Discussion

PTSD trauma memories showed more intrusion characteristics than PDA panic memories. However, the results indicate a continuum of memory processing and storage, because the PDA panic memories in turn showed more intrusion characteristics than the trauma memories of the controls. Moreover, both PTSD trauma memories and PDA panic memories were characterized by disorganization compared to the trauma memories of the controls. In addition, sensory impressions were not unique to PTSD patients either. The trauma memories of the PTSD patients and the controls both included more images than the panic memories of the PDA patients. On

the other hand, both PTSD memories and PDA memories included more bodily sensations than the trauma memories of the controls. It is possible that the type of sensory impression is unique for the content of the memory (see also the overall discussion for an elaboration on this issue).

Another interesting finding concerns the fact that both PTSD and PDA patients experienced dissociation during the traumatic event or during the first panic attack. This is remarkable because, although in the DSM-IV it is mentioned among the symptoms of a panic attack, still, peritraumatic dissociation is often specifically linked to traumatic events (Van der Kolk, Van der Hart, & Marmar, 1996). Even the name 'peritraumatic' suggests the presence of a traumatic event. However, dissociation could just be a symptom people experience while having strong emotions. Indeed, it has also been found to occur during highly positive events (Candel & Merckelbach, 2004). Of course, in the current study peritraumatic dissociation was measured retrospectively, which should be kept in mind when interpreting the results because retrospective reports of peritraumatic dissociation may depend on present PTSD symptom severity (Marshall & Schell, 2002; Zoellner, Sacks, Foa, 2001). Besides peritraumatic dissociation, PTSD and PDA patients also experienced depersonalization or derealization while remembering the traumatic event or panic attack. To conclude, although dissociation is often connected with trauma and PTSD, the phenomenon may just be an indicator of strong emotions.

In contrast to Engelhard et al. (2003) we found no association between peritraumatic dissociation and intrusion characteristics. Instead, across groups, memory-associated and not peritraumatic dissociation was associated with increased intrusion characteristics. Dissociation occurring while remembering an event may have an immediate effect on the characteristics of that particular memory, i.e., dissociation may have an effect on the retrieval and not on the encoding of events into memory, and may cause the memory to be (perceived as) intruding, uncontrollable, vivid, and happening here and now. Alternatively, dissociation may simply be an epiphenomenon of strong emotions, and therefore memories featuring more intrusion characteristics and stronger emotions are associated with more dissociation. Neither peritraumatic nor memory-associated dissociation was associated with memory disorganization.

Study 2 is strong in that it included a large number of participants. Moreover, it compared the traumatic memories of adults with and without PTSD and the memories of patients diagnosed with another anxiety disorder that is thought to develop through conditioning processes. Nevertheless, some limitations need mentioning. For one, the traumas in the control group concerned less sexual and physical assault than the traumas in the PTSD group. Also, the educational level of the controls was higher, and they were younger and more likely to be female than was the case in the other two groups. Although we did control for these variables, the study still merits replication with matched groups that experienced similar kinds of trauma.

Overall discussion

The current studies tried to shed new light on the question whether trauma memories in PTSD are unique and thus contribute to the knowledge of traumatic memories. The results of both studies indicate that PTSD trauma memories may differ from PDA panic memories in intrusion characteristics, but not in the degree of disorganization or reliving while the memory is being recounted.

The finding that PTSD trauma memories contained high levels of intrusion characteristics (study 2) is at least partly in line with theories on PTSD (Brewin et al., 1996; Ehlers & Clark, 2000) as well as theories on memory in general (Conway, 2000). Conway (2000), for instance, posits that the 'working self' cannot adapt to the threat (to current plans and goals) that the trauma presents. Encoding of the information and integration into the autobiographical knowledge base cannot take place, and, as a result, the 'event-specific knowledge' escapes the control processes that are associated with the autobiographical knowledge base. The event-specific knowledge is automatically activated and without control processes, vivid, sensory-perceptual details intrude into awareness.

However, this still does not explain why the PDA panic memories contained fewer intruding sensory-perceptual details than the PTSD trauma memories, considering that their first or most intense panic attack also formed a 'threat (to current plans and goals)'. Perhaps the relative lack of sensory impressions in the PDA group is a consequence of the nature of the threatening event. A traumatic event typically occurs outside oneself, which suggests attention is drawn to the outer world. Panic attacks typically occur within oneself, drawing the attention to one's own physical symptoms. The fact that there was no difference in the intensity of reliving while recounting the threatening event indicates that although the PTSD trauma memories contained more sensory impressions, the PDA panic memories were equally vivid and real. Another explanation may be that PTSD patients typically appraise intrusions negatively and try to avoid them, whereas PDA patients do not, and, by not avoiding the memories themselves, offer the images a chance to be integrated into autobiographical memory. Indeed, initial appraisal of (intrusive) symptoms has been found to be related to later PTSD symptoms (Halligan et al., 2003; Hagenaars, Van Minnen, & Hoogduin, 2006).

The finding that both PDA panic memories and PTSD trauma memories were characterized by disorganization (study 1 and 2) has several implications for theories on trauma memory. It suggests disorganization, which has been specifically associated with PTSD memories (Van der Kolk & Fisler, 1995), can also characterize other types of memories. It is plausible that not the traumatic event but rather the strong emotions evoked by recalling the event are responsible for the disorganization, by disenabling adequate memory retrieval, for example. The healthy participants probably did not experience similar strong emotions during retrieval, and indeed, their memories were not disorganized. Hence, memory disorganization or fragmentation may just be an epiphenomenon reflecting anxiety or fear experienced during recounting (Zoellner & Bittenger, 2004, p. 155).

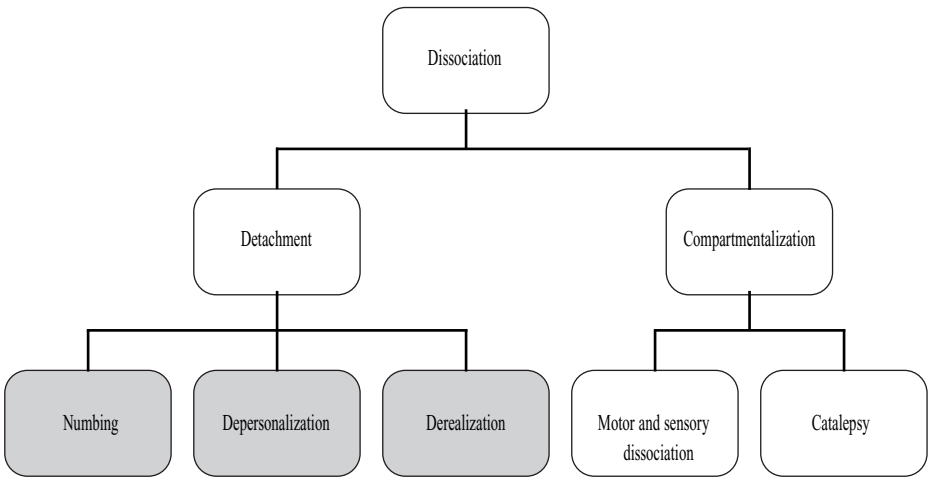
One of the strengths of this twofold study is its use of two distinct methods to

measure the memory characteristics in question, which allowed us to corroborate our initial findings on disorganization. The inclusion of another anxiety disorder that is thought to develop through conditioning processes, may also be called innovative. The present studies illustrated the usefulness of investigating symptoms across syndromes in enhancing our understanding of both the syndromes and the mechanisms underlying specific symptoms. The choice to include PDA patients as a control group has some advantages, in that a panic attack also involves a sudden, unpredictable, and frightening experience during which individuals often fear for their life. It has even been suggested that panic attacks may act like traumatic stressors and sometimes even provoke PTSD symptoms (McNally & Lukach, 1992). This would make the two disorders comparable concerning the development and specific features of the trauma and panic memories. The next challenge therefore is to study memory characteristics and memory development in both disorders in more detail. However, because it is possible that the original experience of a highly fearful event has caused the memories in both disorders to resemble each other, future research should also study so-called PTSD-specific memory characteristics in other disorders, like obsessive-compulsive disorder.

To conclude, the PTSD trauma memories were not distinct from the PDA panic memory on the majority of characteristics studied. The level of disorganization was comparable in both patient groups, in the narrative-rating as well as the meta-memory method, possibly resulting from the intenseness of the emotional experience. The intensity of reliving during recounting the event was also similar in both patient groups (study 1), as were peritraumatic and memory-associated dissociation (study 2). PTSD trauma memories only differed from PDA panic memories by their higher number of intrusion characteristics, although the PDA panic memories still featured more intrusion characteristics than the trauma memories of healthy controls. The current results suggest that traumatic events are not processed and stored qualitatively differently from other events. To increase our understanding of memory processes, we recommend more more research that compares memories across (anxiety) disorders using quantifiable, multimethod approaches.

Chapter 6

The Impact of Dissociation and Depression on the Efficacy of Prolonged Exposure Treatment for PTSD



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Abstract

This study investigates the impact of dissociative phenomena and depression on the efficacy of prolonged exposure treatment in 71 patients with posttraumatic stress disorder (PTSD). Diagnoses, comorbidity, pretreatment depressive symptoms, PTSD symptom severity, and dissociative phenomena (trait dissociation, numbing, and depersonalization) were assessed using semi-structured interviews and questionnaires. In a pretreatment behavioral exposure test, patients were imaginally exposed to (part of) their trauma memory for 9 minutes, during which subjective fear was assessed. At posttreatment and 6 months follow-up PTSD, depressive and dissociative symptoms were again assessed in the completers (n = 60). Pretreatment levels of dissociative and depressive symptoms were similar in dropouts and completers and none of the dissociative phenomena nor depression predicted improvement. Against expectations, dissociative phenomena and depression proves associated with enhanced rather than fear during exposure, although these effects disappeared after controlling for initial PTSD severity. Hence, rather than supporting contraindication, the current results imply that patients presenting with even severe dissociative or depressive symptoms may profit similarly from exposure treatment as patients with minimal dissociative or depressive symptoms.

Keywords: Posttraumatic stress disorder, dissociation, numbing, depression, treatment outcome, exposure.

Introduction

Exposure-based treatments have proven to be effective in reducing PTSD symptoms (Foa et al., 1999; Foa et al., 2005; Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Resick, Nishith, Weaver, Astin, & Feuer, 2002). Yet, despite its high efficacy, some patients do not or not sufficiently profit from exposure therapy (Bradley, Green, Russ, Dutra, & Westen, 2005). Both from a theoretical and a clinical point of view, i.e., to improve treatment indication and treatment efficacy, it is important to identify the patients whom this may concern. Thus far, few stable predictors of treatment outcome have been identified (Van Minnen, Arntz, & Keijsers, 2002). Remarkably, although in numerous studies (peritraumatic) dissociation has been associated with PTSD development (for a review see Ozer, Best, Lipsey, & Weiss, 2003), its impact on PTSD treatment has only been alluded to (Jaycox, & Foa, 1996; Shalev, Bonne, & Eth, 1996), but not systematically studied. The present study therefore investigates the impact of several dissociative phenomena on the efficacy of prolonged exposure treatment. The impact of depression is also studied, as dissociative phenomena like numbing seem to be related to depression (Monson, Price, Rodriguez, Ripley, & Warner, 2004).

First, it is important to understand how dissociation and depression may interfere with prolonged exposure treatment. Foa and Kozak (1986) developed the emotional processing theory, a theoretical framework that conceptualizes PTSD pathology and ways to correct this pathology in treatment. In this theory, it is proposed that

the traumatic memory can be represented as a fear structure that includes representations of trauma-related stimuli, responses and their meaning (Foa & Kozak, 1986; Foa & Rothbaum, 1998; Foa, Steketee, & Rothbaum, 1989). In PTSD, this fear structure is characterized by a large number of stimulus representations associated with danger and strong response elements. For a treatment to be effective, the pathological elements of the fear structure must be corrected. This can only be achieved if the fear structure is activated and if new information is introduced that is incompatible with the existing information in the fear structure. In addition to the emotional processing theory, cognitive models of PTSD have also emphasized the role of fear activation in effective treatment. For example, Ehlers and Clark (2000) underscore the need to relive the trauma so elaboration and contextualization of the trauma memory can take place and negative assumptions about recalling the trauma can be tested.

If fear activation is important with respect to successful treatment of PTSD, factors that impede fear activation should have a negative impact on recovery. In line with the emotional processing theory, some studies on PTSD treatment indeed found that higher (increase in) subjective fear during exposure was related to more improvement (Jaycox, Foa, & Morral, 1998; Van Minnen & Hagenaars, 2002). Also, factors that impede fear activation, like anger (Foa, Riggs, Massie, & Yarczower, 1995; Speckens, Ehlers, Hackmann, & Clark, 2006) and the use of benzodiazepines (Van Minnen et al., 2002), had a negative impact on treatment outcome in PTSD.

Several authors have suggested that dissociation might impede fear activation and therefore hinder adequate treatment of PTSD (Hembree, Marshall, Fitzgibbons, & Foa, 2001; Jaycox & Foa, 1996). In accordance, Ehlers and Clark (2000) suggested that depersonalization, derealization and numbing may interfere with recovery by impeding the elaboration of the trauma memory and its integration into the autobiographical memory knowledge base. One could argue that especially emotional numbing (also referred to as emotional analgesia) impedes fear activation, as it is characterized by emotional non-responsiveness and non-engagement in activities, feelings or other people. Numbing may make it difficult to recognize, describe and regulate emotions, including fear (Monson et al., 2004). This lack of fear may frustrate adequate fear activation, which is deemed essential for effective exposure treatment. In line with this, relative to low dissociators, high dissociators demonstrated suppressed autonomic activity when recounting their trauma (Griffin, Resick, & Mechanic, 1997), and lower resting heart rate (Bryant, Harvey, Guthrie, & Moulds, 2000), although Nixon and Bryant (2005) did not find any differences in psychophysiological responses during trauma recounting between these two patient types (Nixon & Bryant, 2005).

Also in clinical practice it is the prevailing view that dissociation negatively impacts PTSD treatment. Only a handful of studies have explored this notion though. Although it was not significant, Taylor et al. (2001) found a trend for partial responders to cognitive behaviour therapy (CBT) to have higher levels of pretreatment numbing and depressive symptoms after relative to full responders. Moreover, in the partial responders, numbing symptoms had not declined following treatment, whereas the other PTSD symptom clusters had. However, as more of the partial responders also suffered from comorbid major depressive disorder, it may well be possible that

their numbing symptoms were elevated as a result of their depression. Indeed, Monson et al. (2004) demonstrated numbing, and not the other PTSD symptom clusters, to be related to depressive symptoms. Another indication for numbing hindering effective treatment comes from Ehlers et al. (1998), who found that pretreatment alienation, a concept that resembles numbing symptoms like restricted range of affect and feelings of detachment from others, was related to poorer treatment outcome in PTSD after controlling for initial PTSD symptoms. In contrast, Jaycox et al. (1998) found no differences in trait dissociation between patients that did or did not engage in exposure treatment and did or did not habituate. In addition, Speckens et al. (2006) found that pretreatment dissociation did not affect the reduction of intrusions in CBT.

In the current study the effect of depression on treatment efficacy was explored in addition to dissociation because, as diminished interest and emotional numbing can also be found in depression, the latter may thus similarly hamper adequate fear activation and effective treatment. In a large prediction study, however, depression was not related to exposure treatment outcome (Van Minnen et al., 2002). Another study also found comorbidity (mostly depression) not to be associated with poorer outcome in PTSD treatment, although comorbid patients did receive more treatment sessions (Gillepsie, Duffy, Hackmann, & Clark, 2002). Note, however, that treatment in the latter study concerned cognitive and not prolonged exposure therapy. Depressive symptoms (BDI scores) tend to decline after treatment along with PTSD symptoms in prolonged exposure treatment studies (Foa et al, 1999; Foa, Rothbaum, Riggs, & Murdock, 1991; Tarrier, et al 1999). However, considering the substantial overlap between depression and PTSD, these may be PTSD related symptoms, decreasing after successful PTSD treatment.

Dissociation is a complicated construct that includes a variety of symptoms but is often studied as a general construct. Bryant (2007) correctly points out that this does not help to delineate the exact mechanisms that are involved. He suggests that to study dissociation, a deconstruction into more specific factors may be a better approach. Distinct dissociative symptoms may indeed have different effects on, in this case, exposure treatment efficacy. Therefore, in the present study several dissociative phenomena were investigated separately and not combined into one dissociation construct. Besides the theoretical importance to study dissociative phenomena separately, there is also some empirical evidence to support this approach. For example, general dissociative tendency, emotional numbing and depression were found to be distinct constructs with distinct effects on PTSD development (Feeney, Zoellner, Fitzgibbons, & Foa, 2000). The correlation between emotional numbing, anomalous body experience and alienation from surroundings was only moderate in patients suffering from depersonalization disorder, again suggesting distinct symptom domains (Sierra, Baker, Medford, & David, 2005). On the other hand, the moderate correlations also indicate that all constructs mentioned show substantial overlap and are somehow related. Depression seems to be a related construct as well, especially with respect to numbing, which also includes diminished positive affect. Several studies found the correlation between numbing and depression to be higher than the one between active avoidance and depression (e.g.,

Taylor et al., 2001), confirming a symptom overlap or relationship. However, other studies find that although related, numbing and depression are distinct constructs (Litz et al., 1997).

The present study investigates the impact of dissociation, i.e., trait dissociation, depersonalization, and numbing, and depression on the efficacy of exposure treatment for PTSD. We first examined the course of two dissociative phenomena, depersonalization and numbing, and depressive symptoms during treatment. Subsequently, we analyzed the predictive value of pretreatment dissociative phenomena and depression on improvement. Adhering to Foa and Kozak's (1986) conditions for effective exposure, and basing ourselves on the idea that dissociation leads to inadequate fear activation, we hypothesized that treatment would be less effective for patients with elevated levels of dissociation compared to those with lower levels. We additionally studied whether dissociation do indeed impede fear activation, expecting pretreatment elevated levels of dissociation to be associated with less fear during exposure. We applied the same hypotheses with regard to depression in order to stay consistent with the theoretical model.

Method

Participants

Of 95 patients referred for PTSD treatment to an outpatient clinic specialized in the treatment of anxiety disorders 74 met the inclusion criteria as they met the DSM-IV-TR criteria (APA, 2000) for PTSD according to the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) and the CAPS (Blake et al., 1995). Patients who were suicidal, or involved in ongoing traumatization, those fulfilling the DSM-IV-TR criteria for substance dependence or a psychotic disorder, and those that did not speak Dutch were excluded. Three patients (4%) refused to participate in the study, resulting in an intent-to-treat (ITT) sample of 71 patients.

Mean age of the ITT sample was 35.75 (*SD* 11.74; range 18 to 63). Twelve patients (17%) were male, 59 (83%) were female. Educational level was low in 5 (i.e. 6 years; 7%), low-extended in 11 (i.e. 8 years; 16%), medium in 29 (i.e. 10-12 years; 41%), high in 15 (i.e. 12-15 years; 21%), higher than a bachelor degree in 9 (i.e. 16 years or more; 12%), and unknown for 2 patients (2%). Thirty patients (42%) were employed, 20 (28%) employed but currently at home because of their PTSD symptoms, 8 (11%) were unemployed, 11 (16%) either attended university or took care of the housekeeping, and for 2 patients (3%) the occupation was unknown.

The patients had suffered mixed traumas, the index trauma being: sexual assault (n = 17, 24%), nonsexual assault (n = 21, 30%), both sexual assault and nonsexual violence (n = 12, 17%), accidents (n = 8, 11%), and miscellaneous (n = 13, 18%). Thirty-seven patients (52%) had been traumatized repeatedly, and 34 (48%) patients had experienced a single trauma. At the beginning of the treatment, the mean time elapsed since the traumatic event was 10 years and 4 months (*SD* = 11.07 years, range 6 months to 52 years). Comorbidity was high with many patients meeting the criteria of more than one comorbid disorder: 26 (37%) were diagnosed with panic

disorder with agoraphobia, 13 (18%) with social phobia, 8 (11%) with generalized anxiety disorder, 2 (3%) with obsessive-compulsive disorder, 16 (23%) with a current and another 12 (17%) with a past mood disorder, 4 (6%) with somatoform disorder, and 3 (4%) with an eating disorder. In addition, 12 patients (17%) were diagnosed with cluster C personality disorders, 1 (1%) with paranoid personality disorder, 1 (1%) with borderline personality disorder, and 1 (1%) with personality disorder NOS. In total, 49 patients (69%) had a comorbid axis I or II DSM disorder, and 22 (31%) did not.

Of the 71 ITT patients, 60 (84.5%) completed the treatment and 11 (15.5%) dropped out prematurely. The mean number of sessions for the dropouts was 5.82 (SD = 2.75). Dropouts and completers did not differ in age ($t(69) = -.51$, $p = .62$), educational level ($t(69) = .63$, $p = .53$), gender ($\chi^2(1, N = 71) = .57$, $p = .45$), comorbidity ($\chi^2(1, N = 71) = .08$, $p = .77$), multiple or single traumatization ($\chi^2(1, N = 71) = .03$, $p = .86$), or pretreatment PTSD symptoms (PSS-SR: $t(69) = .08$, $p = .94$).

Measures

Diagnostic measures

Mini-International Neuropsychiatric Interview (MINI). The MINI (Sheehan et al., 1998a) is a structured interview using closed-end questions based on DSM-IV and ICD-10 to establish DSM-IV psychiatric diagnoses. Its interrater reliability proved to be good (kappa values of all diagnostic subscales are above .75; Sheehan et al., 1997). In addition, comparison of the MINI with the SCID-I has shown that, in general, MINI-diagnoses are characterized by good or very good kappas (except for current drug dependence with a kappa below .50; kappa for PTSD = .78), good sensitivity (> .70 except for dysthemia, obsessive-compulsive disorder and current drug dependence), and high specificities and negative predictive values (> .85; Sheehan et al., 1998b).

Structured Clinical Interview for DSM-III-R (SCID-I and SCID-II). Both the SCID-I and SCID-II are standardized, semi-structured interviews for diagnosing DSM-IV psychiatric axis I (Spitzer, Williams, Gibbon, & First, 1992) and axis II disorders (First, Spitzer, Gibbon, & Williams, 1995). The reliability of the SCID-I in different patient samples was shown to be good with overall kappas of .61 for current and .68 for lifetime diagnoses. The reliability of the Dutch version of the SCID-II was shown to be good: in an outpatient population kappas ranged from .77 for obsessive-compulsive personality disorder to .82 for avoidant personality disorder. Weighted kappa for all personality disorders was .80 (Arnzt et al., 1992). The interrater agreement proved to be fair to excellent (Intraclass Correlation Coefficients (ICC) ranging from .41 to .88), except for the dependent personality disorder (ICC < .40; Weertman, Arntz, Dreessen, Van Velzen, & Vertommen, 2003).

Outcome measures

Clinician-Administered PTSD Scale (CAPS-1). The CAPS-1 is a structured interview designed to test for the presence of the 17 DSM-IV-TR criteria for PTSD and

to establish PTSD severity in the previous month (Blake et al., 1995). Each symptom is scored on two dimensions, i.e., frequency and intensity, using 5-point scales. The interrater diagnostic agreement proved excellent (Blake et al., 1990), and test-retest reliability for the three symptom clusters (r = .77 to .96) and total scale (r = .90 to .98) was good (Blake et al., 1995). The internal consistency for all CAPS-1 items proved to be high (β = .94; Blake et al., 1995) and the concurrent validity adequate (correlation with Mississippi Scale for Combat-related PTSD: r = .70 to .91, correlation with MMPI PTSD subscale r = .77 to .84; Blake et al., 1990; Blake et al., 1995). We used a Dutch version of the CAPS-1 (Hovens, Luinge & Van Minnen, 2005).

Posttraumatic Stress Symptom Scale–Self-Report (PSS-SR). The PSS-SR is a 17-item self-report questionnaire that measures the frequency of PTSD symptoms using 4-point Likert scales (Foa, Riggs, Dancu, & Rothbaum, 1993). Each item corresponds to one of the DSM-IV-TR criteria for PTSD, and has three symptom subscales: reexperiencing, avoidance and arousal. Analyses showed a high internal consistency (Cronbach's alpha for the total score was .91), and a good test-retest reliability of the overall severity (.74; Foa, Riggs, Dancu, & Rothbaum, 1993). The Dutch version also shows good internal consistency (β = .92; Mol et al., 2005).

Beck Depression Inventory (BDI). The BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report questionnaire assessing the severity of depressive symptoms (score range per item is 0 to 3). Its internal consistency for both psychiatric and nonpsychiatric samples was shown to be high (α coefficients range from .76 to .95 and .73 to .92 respectively). Its concurrent validity was also high in both psychiatric and nonpsychiatric samples (correlation with clinical ratings: r = .55 to .96, correlation with Hamilton Rating Scale for Depression: r = .61 to .86, correlation with Zung: r = .57 to .86, correlation with MMPI-D: r = .41 to .75; Beck, Steer, & Garbin, 1988). The Dutch version of the BDI also showed good internal consistency (α = .91; Schotte, Maes, Cluydts, De Doncker, & Cosyns, 1997).

Subjective Unit of Distress Scale (SUDS). Subjective fear was measured using the SUDS (Wolpe, 1973), a visual analogue scale on which the respondent indicated the degree of fear felt at that moment by placing a number from 0 (no fear) to 10 (panic) on the 10-cm horizontal line. To establish pretreatment fear levels, patients took a 9-minute behavioral exposure test, during which they were asked to indicate their current fear level every 3 minutes. Following Jaycox et al. (1998) a patient's level of fear activation was defined as his/her mean SUDS score during this test.

Dissociative phenomena

Trait dissociation was measured with the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986; Carlson & Putnam, 1993), a 28-item self-rating scale that measures the tendency to experience dissociative experiences in daily life (total range: 0 to 100). For each item the respondent states how often a specific dissociative symptom occurs. Compared to other instruments gauging dissociation, the convergent validity of the DES was shown to be very good and its reliability good (overall Cohen's d = 1.82 and mean alpha reliability = .93 respectively; Van

IJzendoorn, & Schuengel, 1996). In our sample, using the Dutch translation of the DES, reliability was similarly high (Cronbach's alpha = .92). Furthermore, it was found that the DES assesses a single dimension of dissociation (Holtgraves & Stockdale, 1997). Bremner et al. (1993) found some evidence for the DES measuring general dissociative tendencies and not state dissociation, as their Clinician-Administered Dissociative States Scale (CADSS), which measures dissociative states, was only moderately correlated to the DES.

Depersonalization was evaluated using by the 3 dissociation items of the associated features of PTSD from the CAPS (CAPS-D; reduced awareness, derealization, and depersonalization). We used the items' mean score in the analyses.

Numbing was defined based on the three numbing items from the PSS-avoidance subscale (PSS-SR-N; detachment from others, restricted affect, diminished interest in activities; see e.g., Litz, 1992). Like all PSS-SR items, the numbing items typically inquire after symptoms in the preceding week. The items' mean score was used in the analyses.

Procedure

The participants cooperated on a voluntary basis and all assessments were conducted by trained, independent assessors. Pretreatment screening (establishing diagnosis, comorbidity, and inclusion and exclusion criteria) comprised the SCID-I, and later MINI, and SCID-II. Within a week, included patients subsequently completed all pretreatment questionnaires and took part in the CAPS interview. Lastly, to establish pretreatment levels of distress they took the 9-minute behavioral exposure test which was delivered by two trained independent experimenters. Imaginal exposure during this test was consistent with the protocol for prolonged exposure treatment for PTSD the patients would be attending later (see next paragraphs; Dancu & Foa, 1993). The patients and the experimenter selected the first frightening (part of the) trauma from the exposure hierarchie for this purpose. Subjective levels of distress were rated by the patient at 0, 3, 6, and 9 minutes.

One week after the abovementioned assessments, patients entered a standardized prolonged exposure treatment program (Dancu & Foa, 1993) comprising 8 to 12 weekly sessions that lasted 45 minutes. Note, however, that in 10 cases treatment was ended before the 8th session because the patients concerned had already achieved (full) recovery: they no longer met the DSM-IV-TR PTSD criteria according to the CAPS, their PSS-SR total score had dropped below 10, and their SUDS scores recorded during the 3 last exposure sessions and the subsequent homework assignments were low (< 5). The mean number of session of these early completers was 4.38 (*SD* = 1.71).

The first therapy session included a presentation of the treatment rationale, education about the disorder and common reactions to trauma and information gathering. The subsequent sessions consisted of 30 minutes imaginal exposure: patients were asked to close their eyes and talk about the traumatic event in the first person and in the present tense, recollecting as many sensory details as vividly as

possible, i.e., as if the trauma was happening 'here and now'. Each imaginal exposure session was audiotaped and patients were instructed to listen to the tape at home five times a week. From the 4th session onwards in vivo exposure assignments were an integrated part of the treatment. These included exposure to fearful stimuli associated with the trauma, like visiting trauma-related places or listening to trauma-related sounds. Each session started with a review of the patients' homework and ended with homework assignment. At the start of each treatment session patients also completed the PSS-SR. Treatment fidelity was rated after each session and all therapists involved were supervised weekly by the second author. Following treatment conclusion, all patients again completed the questionnaires measuring state symptoms and participated in the posttreatment CAPS interview.

Follow-up assessments comprising all questionnaires measuring state symptoms and the CAPS were conducted six months after the posttreatment assessment.

Statistical analyses

Repeated measures analyses were conducted to analyze treatment effect for PTSD symptoms and dissociative phenomena using the intent to treat (ITT) sample. Because no posttreatment data were available for the dropouts, data were analyzed with the last observation carried forward (LOCF) to establish improvement for the entire ITT sample. As we wished to study the effect of dissociation and depression on improvement, we only included the data of the completers in our subsequent linear regression analyses. Although this may seem to provide a distorted image, it is in fact addressing the research question, which concerns the impact of dissociation and depression when someone completes treatment. These results are very important from a clinical point of view.

Several distinct statistical analyses were conducted to address the various research questions. To begin with, correlations between the DES, CAPS-D, PSS-SR-N, and BDI were calculated to check whether these indeed reflected associated but distinct constructs. As all correlations were indeed significant but moderate (Table 6.1) we did not use a composite variable but instead analyzed all dissociation and depression variables separately. Next, repeated measures analyses with PSS[1], CAPS-D, PSS-SR-N, and BDI as independent variables were used to analyze the effect of exposure treatment on PTSD, depersonalization, numbing and depressive symptoms.

The impact of dissociative and depressive symptoms on improvement was analyzed in two ways: 1) overall regression analyses and 2) comparing extreme symptom profiles. In the simultaneous entry regression analyses the three pretreatment dissociation measures (DES, CAPS-D and PSS-SR-N) and depressive symptoms (BDI) were entered as independent variables. We used PSS-SR residual gain scores as a dependent variable to reflect change in PTSD symptoms and control for initial PTSD severity

1. CAPS-data are not reported in this analysis or any of the following analyses, because the CAPS was introduced at a later stage of our study, resulting in CAPS-data of only 47 completers. However, all analyses were recomputed with the CAPS, yielding the same results.

Table 6.1 *Correlations between dissociative and depressive symptoms (N = 71)*

	BDI	DES	CAPS-D	PSS-SR-N
BDI		.53*	.31*	.49*
DES			.29*	.38*
CAPS-D				.44*
PSS-SR-N				

Note: BDI = Beck Depression Inventory, DES = Dissociative Experiences Scale, CAPS-D = Clinician-Administered PTSD Scale-Depersonalization, PSS-SR-N = Posttraumatic Stress Symptoms-Self Rating Scale-Numbing. *$p < .05$.

(Steketee & Chambless, 1992). Still, it was possible that overall analyses would not yield any effects because dissociation and depression have an impact on treatment efficacy only in patients with high levels of these symptoms. We hence divided the patients into high and low dissociation and depression groups, based on their pre-treatment DES, CAPS-D, or PSS-SR-N scores (mean plus or minus 0.5 SD). With this procedure we eliminated patients whose scores were in the middle range, thereby possibly neutralizing effects. It also allowed us to compare substantial groups of high- and low-scoring patients (about 50% of the total sample), minimizing losses of power. With regard to depression, patients were divided into 3 groups based on their DSM-IV-TR diagnosis: 1) current depression, 2) depressive episode(s) in the past but no current depression, 3) and no current or past depression. Improvement in these high- and low-scoring patients was subsequently compared using 2 (high versus low) x 3 (pre, post, follow-up) repeated measures MANOVAs and χ^2-tests.

Finally, to study whether dissociation and depression were associated with impeded fear activation correlations were calculated between DES, CAPS-D, PSS-SR-N, and BDI with mean SUDSs during the behavioral exposure test. The entire ITT sample (N = 71) was used in these analyses.

Results

Treatment Outcome

Treatment was successful in linearly decreasing PTSD symptoms from pretreatment to follow up in the ITT sample ($F(1, 70) = 126.84$, $p < .001$) and in the completers sample ($F(1, 59) = 154.37$, $p < .001$). Means, SDs and within subject effect sizes, controlling for repeated measurements, of the outcome measures are listed in Table 6.2. End-state functioning was defined as being at or below 20 on the PSS-SR and at or below 10 on the BDI, following Foa et al. (1999). Based on these criteria, 58% of the completers achieved good end-state functioning. Remarkably, there was a great discrepancy between patients achieving good end-state functioning using the criteria for the PSS and those for the BDI, with 90% of the completers achieving the PSS criterion and 57% the BDI criterion. Pretreatment PTSD-severity

and improvement were comparable to other PTSD studies evaluating prolonged exposure treatment (e.g., Foa et al., 1999). Considering the range of (multiple) traumas in our patient cohort, the improvement rate was quite high. The linear decrease of numbing symptoms, depersonalization, and depressive symptoms was also significant in the ITT sample (PSS-SR-N: $F(1, 70) = 33.77$, $p < .001$; CAPS-D: $F(1, 54) = 7.16$, $p < .01$; BDI: $F(1, 70) = 51.98$, $p < .001$) and in the completers sample (PSS-SR-N: $F(1, 59) = 40.84$, $p < .001$; CAPS-D: $F(1, 46) = 7.30$, $p < .01$; BDI: $F(1, 59) = 66.71$, $p < .001$). In sum, prolonged exposure treatment successfully reduced PTSD, including numbing symptoms, depersonalization and depressive symptoms.

Table 6.2 *Means (SDs) of outcome measures for the completers sample (N = 60)*

	Pretreatment	Posttreatment	Follow-up	Partial η^2	Cohen's *d*
PSS-SR*	25.52 (8.70)	10.95 (9.75)	9.20 (8.40)	.77	2.70
CAPS*	66.17 (16.81)	27.50 (26.24)	22.08 (22.08)	.82	3.07
BDI*	20.21 (10.50)	12.24 (10.05)	10.11 (9.23)	.53	2.18
DES	18.41 (13.44)	-	-	-	-
CAPS-D*	3.18 (3.88)	1.06 (2.39)	1.44 (3.27)	.28	.96
PSS-SR-N*	3.70 (2.50)	1.53 (2.22)	1.38 (1.97)	.47	1.81
Mean SUDS during the Behavior Exposure Test	7.58 (1.74)	-	-	-	-

Note: Effect sizes and significance values concern pretreatment to follow-up repeated measures analyses. PSS-SR = Posttraumatic Stress Symptoms-Self Rating Scale, CAPS = Clinician-Administered PTSD Scale, BDI = Beck Depression Inventory, DES = Dissociative Experiences Scale, CAPS-D = Clinician-Administered PTSD Scale-Depersonalization, PSS-SR-N = Posttraumatic Stress Symptoms-Self Rating Scale-Numbing, SUDS = Subjective Unit of Distress Scale. *$p < .01$.

Effect of dissociation and depression on treatment efficacy

As treatment efficacy concerns both improvement and dropout, we first tested whether dropouts showed more pretreatment dissociative and depressive symptoms than completers. Dropouts did not differ from completers on trait dissociation (DES: $t(69) = 0.09$, $p = .93$), depersonalization (CAPS-D: $t(69) = 0.20$, $p = .84$), numbing (PSS-SR: $t(69) = 0.90$, $p = .37$), depressive symptoms (BDI: $t(69) = 0.24$, $p = .81$), or in the presence of a current or past mood disorder ($\chi^2(1, N = 71) = 0.17$, $p = .68$). Means for dropouts and completers were respectively: DES: 18.80 (*SD* = 11.74) and 18.26 (*SD* = 13.09), CAPS-D: 3.45 (*SD* = 5.32) and 3.18 (*SD* = 3.88), PSS-SR-N: 4.46 (*SD* = 2.84) and 3.70 (*SD* = 2.50), and BDI: 20.82 (*SD* = 6.71) and 20.06 (*SD* = 10.19). Current or past depressions were present in 38% of the completers and 18% of the dropouts.

Regression analyses showed that none of the three dissociation variables, nor depressive symptoms predicted pre-to-posttreatment PTSD reduction ($\Delta R^2 = .13$ $p = .17$, all βs ns), indicating that neither pretreatment dissociation nor depression had affected improvement. Similarly, none of the dissociation variables, nor de-

pressive symptoms proved to predict pretreatment-to-follow-up PTSD reduction ($\Delta R^2 = .10$ $p = .25$, all β's ns). The results of this latter regression analysis must be interpreted with caution, though, because 7 (12%) of the patients from the completers sample did not take the follow-up assessment.

Next, extreme symptom groups were compared. The parallel lines Figure 6.1[2] indicate a similar pretreatment-to-follow-up decline in PTSD symptoms in patients with high ($n = 15$, M DES = 34.91, $SD = 9.59$) and low trait dissociation ($n = 21$, M DES = 6.19, $SD = 2.39$), high ($n = 25$, M CAPS-D = 7.82, $SD = 3.38$) and low depersonalization ($n = 17$, M CAPS-D = 0, $SD = 0$), high ($n = 22$, M PSS-SR-N = 6.53, $SD = 1.23$) and low ($n = 24$, M PSS-SR-N = 1.22, $SD = .85$) numbing, and current ($n = 12$, M BDI = 27.60, $SD = 13.33$), past ($n = 13$, M BDI = 23.92, SD = 7.33), or no current or past depression ($n = 35$, M BDI = 16.39, $SD = 8.22$). Note that a substantial number ($n = 11$, i.e., 18% of the entire completers sample) of the high DES patients were severely dissociative based on a cut-off score of 30. The 2 x 3 repeated measures MANOVA did not show an interaction effect for DES ($F(1, 30) = .18$, $p = .68$), or depression ($F(1, 51) = .32$, $p = .57$), indicating PTSD symptoms declined similarly in high and low DES and in current/past and no depression groups. There was an interaction effect for PSS-SR-N ($F(1, 36) = .14.35$, $p = .001$), but in a surprising direction: 'high numbing' patients showed a greater reduction in pre-to-follow-up PTSD symptoms than 'low numbing' patients. There was a similar trend for patients with high versus low levels of depersonalization ($F(1, 34) = 3.27$, $p = .08$) too. As expected, time also proved to be significant ($F(1, 36) = 122.75$, $p < .001$).

The parallel improvement curves and the absence of an interaction effect for the high and low DES and depression groups suggested that the high DES patients and those with a current/past depression would exhibit more PTSD symptoms at follow-up. This indeed proved the case; these two groups of patients met PTSD criteria at follow-up more often than patients with low DES ($\chi^2(1, N = 32) = 11.79$, $p = .002$) and those without depression ($\chi^2(1, N = 53) = 4.41$, $p = .04$). That is, 10% of the low versus 69% of the high DES group, and 18% of the non-depressed versus 45% of the past/current depressed met PTSD criteria at follow up. Interestingly, patients with a current or a past depression did not differ from each other at follow-up ($\chi^2(1, N = 20) = .04$, $p = .85$). There was no difference in meeting PTSD criteria between patients with high and low levels of depersonalization ($\chi^2(1, N = 37) = .02$, $p = .99$), and high and low numbing patients ($\chi^2(1, N = 39) = 1.37$, $p = .24$). That is, 27% of the patients with low levels of depersonalization versus 29% of those with high levels, and 20% of the low-numbing versus 31% of the high numbing patients met PTSD criteria at follow up.

2. Because the number of sessions varied between patients, the PSS-SR of the middle session was used in the graphs to show the course of PTSD symptoms during treatment.

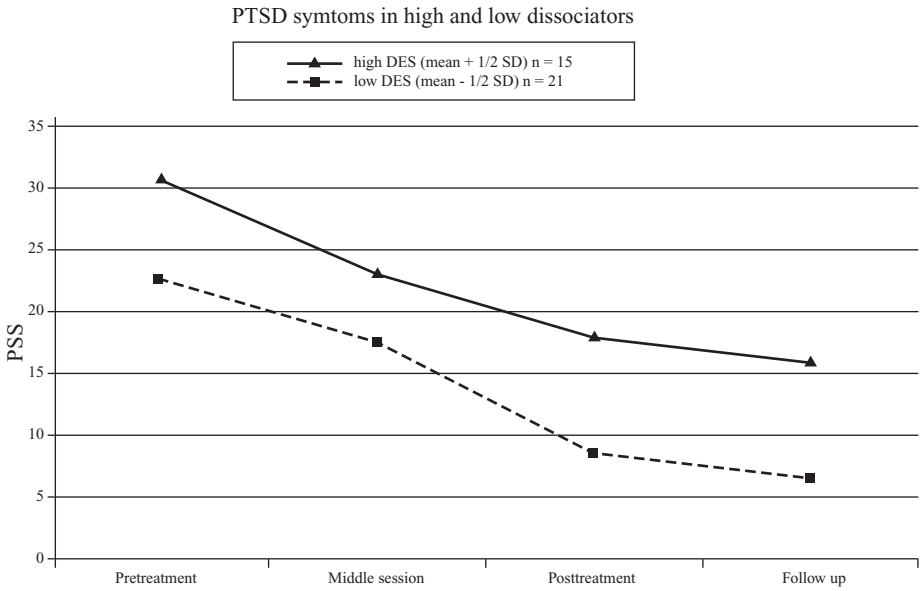

Fig. 6.1a *The course of PTSD symptoms in patients with high and low trait dissociation.*

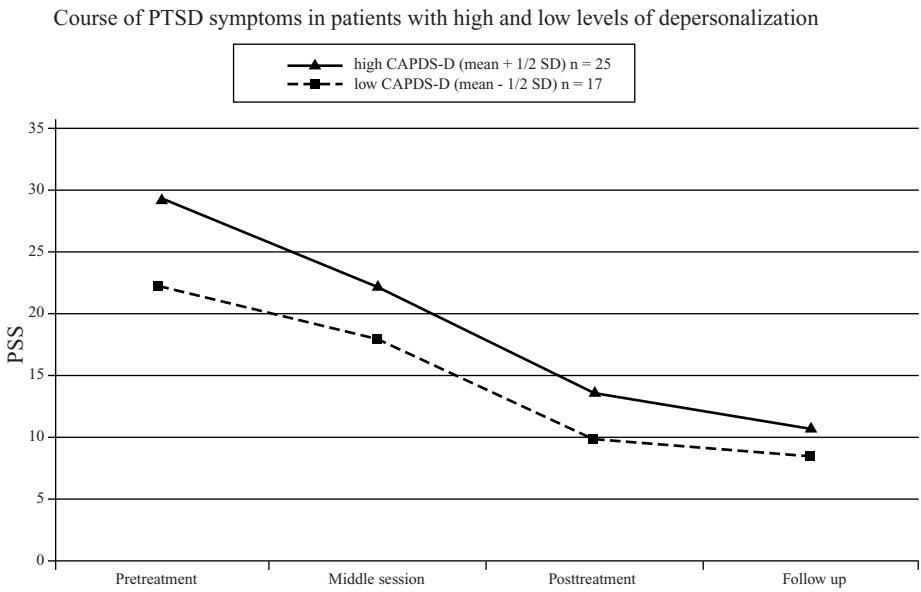

Fig. 6.1b *The course of PTSD symptoms in patients with high and low levels of depersonalization.*

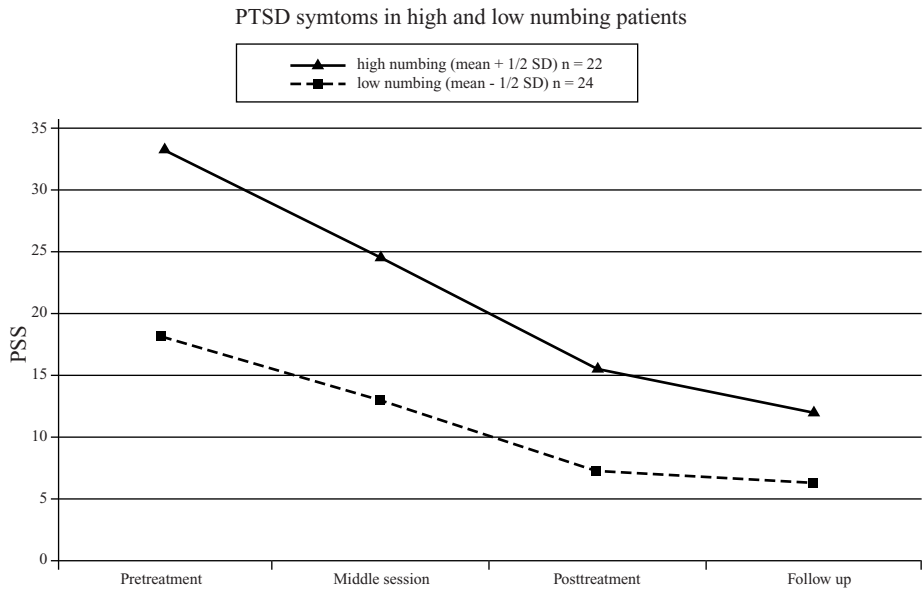

Fig. 6.1c *The course of PTSD symptoms in patients with high and low levels of numbing.*

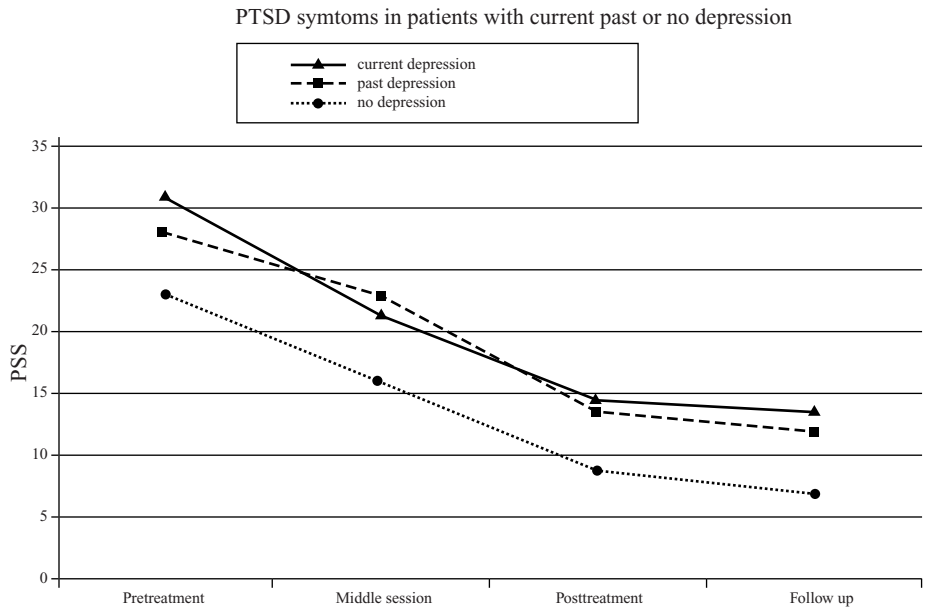

Fig. 6.1d *The course of PTSD symptoms in patients with current, past, and no depression.*

Dissociation and fear activation

DES (r = .38, p < .01), CAPS-D (r = .27, p < .05), PSS-SR-N (r = .37, p < .01), and BDI (r = .37, p < .01) were significantly related to mean SUDSs, albeit in the opposite direction as hypothesized: higher levels of dissociative and depressive symptoms were associated with higher levels of subjective fear during the behavioral exposure test. As the fear habituation curves during the behavioral exposure test were similar for high and low DES, high and low CAPS-D, high and low PSS-SR-N, and current or past versus no depression, Figure 6.2 only depicts the habituation curves for the high and low numbing patients (mean plus or minus 0.5 SD). Because the relationship between high levels of dissociative and depressive symptoms and elevated fear levels during the behavioral exposure test could be due to the fact that these were patients experiencing more severe PTSD symptoms to begin with, partial correlations were calculated while controlling for initial PTSD symptoms (pretreatment PSS-SR). Indeed, in these analyses all significant correlations between dissociative and depressive symptoms and fear during the behavioral exposure test disappeared (all ps ns).

In sum, neither the three pretreatment dissociative phenomena nor depression were associated with poorer improvement after exposure treatment. In addition, rather than impeding fear activation, numbing, depersonalization, trait dissociation, and depressive symptoms were related to higher fear levels during the behavioral exposure test, although this finding was explained by higher levels of pretreatment PTSD symptoms.

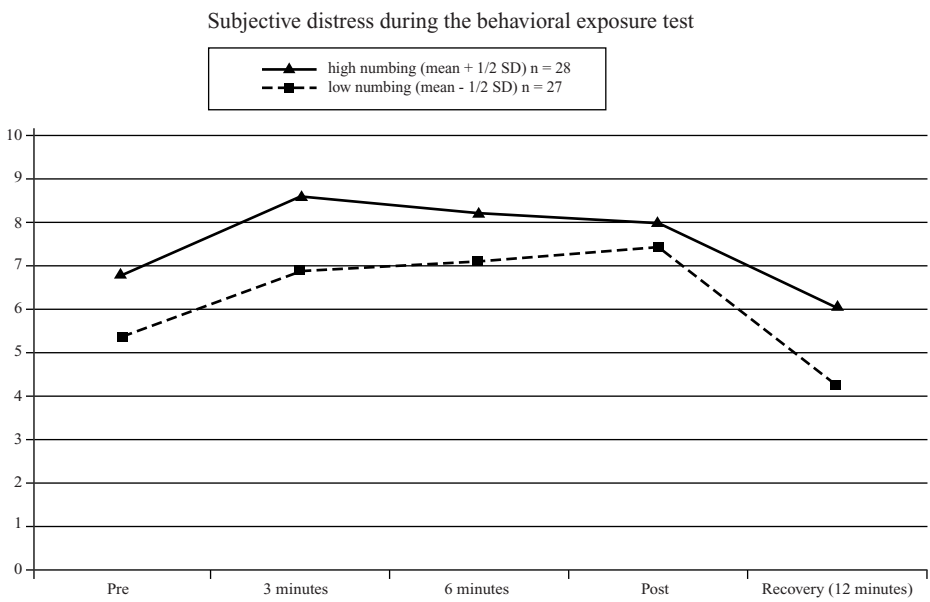

Fig. 6.2 *Pretreatment SUDS scores during the behavioral exposure test for patients with high and low levels of numbing.*

Discussion

The present study examined the impact of three dissociative phenomena and depression on exposure treatment efficacy in PTSD patients. The prolonged exposure treatment not only reduced PTSD symptoms, but also numbing, depersonalization and depressive symptoms. In contrast to our hypothesis, pretreatment trait dissociation, depersonalization, numbing, and depressive symptoms did not predict improvement or dropout. In fact, patients with high levels of trait dissociation, depersonalization, or numbing as well as patients with a past or current depression showed a similar reduction of PTSD symptoms from pre to posttreatment and pre to follow-up as patients with low levels of these dissociative phenomena or patients that had no (history of) depression. Patients with high levels of trait dissociation or depressive patients (current and past) showed more severe PTSD symptoms at posttreatment and follow-up, but also at pretreatment, thus showing similar improvement as patients with low levels of trait dissociation or no (history of) depression. Surprisingly, 'high numbing' patients (also having more severe pretreatment PTSD symptoms) even showed a greater reduction in PTSD symptoms from pretreatment to follow-up than 'low numbing' patients, and a trend in the same direction was found for patients with high levels of depersonalization. As a result, there was no difference between patients with high or low levels of depersonalization or numbing in the number of patients meeting PTSD criteria at follow-up. In sum, dissociation and depression had no predictive value with respect to improvement from treatment. With respect to depression, our results confirm earlier findings that depressive symptoms improve as a function of exposure treatment for PTSD (e.g. Foa et al, 1999); the findings on dissociation are quite novel.

The results have some important implications for clinical practice. Most importantly, PTSD patients with elevated levels of dissociation and those with a comorbid depressive disorder seem to improve similarly as a result of exposure treatment as patients without these symptoms. They are also not more likely to drop out of treatment. Thus, it would be ill-advised to exclude these patients from prolonged exposure treatment. Note that as suicidal patients were excluded from the present study, these conclusions should not be generalized to depressive PTSD patients with suicidal intent. The finding that patients with high levels of dissociation gained from treatment as much as others may also be relevant with respect to treating PTSD patients that suffered sexual abuse and subsequently have developed dissociative symptoms. The present study included childhood sexual abuse and dissociation did not affect improvement. This is consistent with an earlier study of Van Minnen et al. (2002), who found the type of trauma (childhood versus adulthood trauma) not to be a relevant predictor of exposure treatment outcome. Still, more research is required before any firm conclusions can be drawn about the efficacy of exposure therapy in victims of childhood (sexual) trauma.

The results also have some implications for current theories on exposure treatment for PTSD. Interestingly, not only did dissociation and depression not hamper effective exposure treatment, in fact, symptoms of depersonalization, numbing and depression even declined as a result of exposure therapy. This makes sense because

one of the aims of exposure treatment is to help patients engage and experience their fear, thereby reducing emotional numbing during exposure. This may have contributed to the reduction of numbing and depersonalization symptoms. Alternatively, as dissociation is no longer needed as a means of coping with anxiety (Elzinga, Bermond, & Van Dyck, 2002) it may have faded out when PTSD symptoms diminish. Similarly, Foa and Rauch (2004) found that exposure alone was just as effective in reducing negative cognitions as exposure plus cognitive restructuring was. Although DES is thought to be a trait measure, future research may administer this questionaire at posttreatment and follow-up assessments too to establish to what extend the DES-score reflects state dissociative symptoms related to PTSD severity.

Another interesting finding concerns the similar decline of PTSD symptoms in patients with higher levels of dissociation and (current or past) depression, compared to patients with lower levels of dissociation and no (history of) depression. At the 6-month follow-up, the number of patients meeting PTSD criteria was similar for patients with high and low numbing or depersonalization. As they started off with more severe PTSD symptoms, the patients with high levels of numbing or depersonalization may have needed more time to recover. However, because treatment was not controlled during the 6 months from posttreatment to follow-up, it is impossible to say what happened during this period. Patients with high trait dissociation levels and patients with a current or past depression improved like the others, but did meet PTSD criteria at follow-up more often than patients with low trait dissociation levels and patients without (a history of) depression. High trait dissociation may reflect a personality trait that makes one chronically more vulnerable to stress. These patients may hence chronically have higher stress levels chronically, perhaps also before the onset of their PTSD. Their end-state functioning is then limited to symptoms associated with a stress-related personality trait. Indeed, recent studies have shown trait dissociation to be related to neuroticism (Goldberg, 1999; Kwapil, Wrobel, & Pope, 2002) or even psychiatric symptoms in general (Spindler & Elklit, 2003). Moreover, in the current study, trait dissociation was related specifically to the PTSD arousal ($r = .41$, $p < .01$) and avoidance ($r = .40$, $p < .01$) symptom clusters, and not to the reexperiences cluster ($r = .23$, ns). Interestingly, there was no difference in meeting PTSD criteria between patients with a current and those with a past depression, again suggesting that an underlying stress-related trait is responsible for the maintenance of a somewhat increased and chronic symptom level. In this respect, it would be interesting to also address neuroticism in future studies, because it is possible that patients with high trait dissociation levels and those with a current/past depression show a similar end-state of PTSD symptoms as patients high on neuroticism.

Finally, trait dissociation, depersonalization, numbing, and depression did not impede fear activation during exposure. On the contrary, they were all three related to higher fear levels during exposure, although this association disappeared after controlling for initial PTSD severity. This suggests that dissociation may not protect the individual against experiencing distress but instead may be an epiphenomenon of high levels of distress. Other studies (Fikretoglu et al., 2006; Fikretoglu et al., 2007) showed similar results: high levels of peritraumatic dissociation were as-

sociated with high levels of peritraumatic distress. In fact, in the present study, initial PTSD severity (indicating higher levels of distress) was responsible for the association between dissociative and depressive symptoms, and fear during exposure. An additional explanation may be that numbing is a coping reaction to reduce distress (Litz et al., 1997), which can be switched off during high stress. Because exposure therapy aims at inducing fear and distress, it thereby halts coping symptoms like numbing, thus allowing the patient access to the fear network. It has been suggested that numbing and hyperarousal are related symptom clusters (Litz et al., 1997; Yoshihama & Horrocks, 2005), which is consistent with our finding that the 'high numbing' patients reacted with higher distress during the behavior exposure test (hyperarousal) than the 'low numbing' patients. More explicitly, in the absence of numbing, the patients experienced new, and thus extra, distress. Maybe numbing symptoms should not be interpreted as an inability to *experience* emotions (i.e., fear during exposure), but instead as an inability to adequately *regulate* emotions (in this case anxiety).

Although dissociation is believed to negatively affect the efficacy of exposure treatment in PTSD, to our knowledge, the present study is the first to address this issue directly. The study is strong in that it assesses three well-defined types of dissociation (trait dissociation, depersonalization, and numbing), and the related construct of depression, thereby acceding that dissociation covers a wide range of symptoms. Moreover, it included a relatively large sample and numerous types of traumas (including sexual abuse), allowing the results to be generalized to exposure treatment of a broad range of traumas. Nevertheless, not all traumas were represented (e.g., war trauma) and our results hence warrant replication in other treatment studies that include these other trauma populations. Although a substantial number of patients were severely dissociative (Carlson et al., 1993), the study also merits replication in patients with dissociative disorders included in the sample. Perhaps a randomized control trial would be an elegant design for this purpose. Other sorts of dissociation, like amnesia, must also be studied with respect to treatment efficacy. It would furthermore be interesting to not only administer the DES at pretreatment but also following treatment cessation because, even though the DES is thought to measure trait dissociation and therefore considered to be stable, it is possible that part of the DES score depends on the level of PTSD symptoms. The DES-score may therefore also decline after treatment. Finally, previous studies on fear activation in PTSD usually used SUDS measures derived from exposure treatment sessions. However, as exposure duration may vary and the exposure treatment is conducted by the patient's own therapist, we used a behavioral exposure test in order to control for any resultant confounders. Moreover, all behavioral exposure tests were conducted by the same two independent experimenters.

In conclusion, although the prevailing view in clinical practice is that dissociation and depression have a negative impact on exposure treatment for PTSD, we found no evidence to support this belief. Conversely, we found depersonalization, numbing and depressive symptoms to have declined after exposure treatment. There was no difference in dissociative and depressive symptoms between dropouts and completers. Furthermore, relative to patients with low levels of dissociation (trait dis-

sociation, depersonalization and numbing) and no depression, fear activation was not impeded in patients with high levels of dissociation and those with a current or past depression, these latter patients showed a similar decline of PTSD symptoms during therapy, and did not relapse after 6 months. These findings have clear clinical relevance as they indicate that PTSD patients with serious comorbid dissociative or depressive symptoms are just as likely to profit from effective treatment programs like exposure as those with low dissociative and depressive symptom levels.

Chapter 7

General Discussion

Discussion

This dissertation aimed at investigating the association between posttraumatic stress disorder (PTSD) and dissociation. Several areas were investigated in which this association is thought to play a role. The first line of enquiry concerned the development of PTSD and the impact of peritraumatic dissociation on this process. The second area of interest concerned the nature of PTSD trauma memories, which are considered to be different from other memories as a result of inadequate encoding during the traumatic event because of peritraumatic dissociation. The third and final area concerned the impact of dissociation and depression on the efficacy of exposure treatment for PTSD. Throughout the different studies we targeted several distinct dissociative phenomena, reflecting both detachment and compartmentalization. Detachment, in brief, refers to experiences that concern an altered state of consciousness, in which one feels detached from one's body, one's sense of self, or the world around one. Compartmentalization entails experiences involving an inability to deliberately control otherwise controllable processes (for a more detailed description, see the introduction chapter).

Dissociation and PTSD development

Most studies on peritraumatic dissociation focused on symptoms of detachment and not compartmentalization. Yet, their impact on the development of PTSD could be quite distinct. With this in mind, we studied hypnotically-induced catalepsy as a research paradigm for compartmentalization-dissociation (see Chapter 3). Induced catalepsy proved to be associated with several sensori-motor dissociative symptoms and therefore, we deemed it useful for future research of compartmentalization (see also the discussion of Chapter 4 below).

The research reported in Chapters 2 and 4 focused on the association between peritraumatic dissociation and PTSD development but none of the studies found proof for this relationship. Although in a prospective study (Chapter 2) the two forms of dissociation, i.e. detachment and compartmentalization, seemed to predict PTSD symptoms 6 months after the trauma, their predictive power was no longer significant after controlling for initial numbing symptoms. In an experimental study (Chapter 4), compartmentalization-dissociation in the form of hypnotically-induced catalepsy while watching a highly aversive film did not predict the frequency of intrusions about that film.

Although these findings seem to contradict previous research on peritraumatic dissociation and PTSD development (see Ozer et al., 2003, for a review of PTSD predictors), this is not really the case. That is, as noted in the Introduction of this dissertation, many of these earlier studies suffer from methodological problems, like retrospective designs and a lacking to control for initial PTSD symptoms. In fact, our findings are in line with other, more recent studies that also found no association between peritraumatic dissociation and later PTSD after having controlled for dissociation-related factors like initial PTSD symptoms (Marx & Sloan, 2005; Marshall

& Schell, 2002), and event-related fears about death and loss of control (Gershuny, Cloitre, & Otto, 2003).

Interestingly, in the prospective study (Chapter 2) an alternative predictive factor, i.e. inadequate cognitions about the traumatic event, oneself and the world in general, did predict later PTSD symptoms, even after initial numbing symptoms had been controlled for. This suggests that the interpretation of initial symptoms may be highly relevant in predicting who will develop PTSD after trauma. There already is some evidence supporting this contention. For example, several studies found that not peritraumatic but sustained dissociation predicted PTSD development (Briere, Scott, & Weathers, 2004; Murray, Ehlers, & Mayou, 2002). Again, these findings seem to indicate that not automatic peritraumatic reactions, but post-trauma coping strategies are a risk factor for PTSD. Accordingly, future research should address these coping strategies and the interpretation of early symptoms. It would be interesting to experimentally manipulate the interpretation of symptoms and study the effect of these appraisals on symptom maintenance. When educating or counseling people that have undergone a traumatic even, it may also be important to emphasize that experiencing dissociation during the trauma or shortly after is a perfectly normal reaction and does not indicate psychopathology.

In the experimental study (Chapter 4) another interesting alternative factor emerged as a possible predictor of PTSD: Non-movement per se proved to be associated with an increased intrusion frequency. Interpretation of this finding is difficult, though. It is possible that non-movement itself, regardless of whether it is deliberate or involuntary, is related to feelings of uncontrollability, an important factor in the development of PTSD (Foa, Zinbarg, & Rothbaum, 1992). Indeed, it has been hypothesized that non-movement may be an evolutionary-based response to danger that is strongly connected to fear and feelings of helplessness (Moskowitz, 2004). Maybe such a defensive non-movement or 'freeze'-response is not necessarily associated with dissociation but primarily with non-movement per se. Of course, this subject merits further research. Only very few experiments have been conducted with respect to dissociation provocation and PTSD and intrusion development, and our results merit replication and extension. More experimental studies are needed to control for several confounding factors in clinical studies, and to investigate alternative explanations for PTSD and intrusion development. In this light, it would be interesting to investigate and manipulate different forms of dissociation because each may a have distinctive impact on PTSD and intrusion development. Of course, using catalepsy means that the results of the experimental study concern compartmentalization only and cannot be generalized to detachment symptoms. Also, cognitive factors, like controllability, predictability, and interpretation of (dissociative) symptoms merit further research. In doing so, one must think of other ways than a diary to assess intrusions.

In conclusion, neither detachment nor compartmentalization was associated with later PTSD symptoms, indicating that peritraumatic dissociation may not play the important role in PTSD development as is often assumed. To our knowledge, the prospective study described in Chapter 2 was the first to investigate both detachment and compartimentalization. It was also strong in controlling for initial numbing

symptoms, which show an overlap with detachment-dissociation. In addition, as far as we know, Chapter 4 describes the first experimental study to evoke compartmentalization. The fact peritraumatic dissociation proved unrelated to later PTSD symptoms may imply that peritraumatic dissociation is a common response during threat which does not necessarily lead to later PTSD. Indeed, although a minority of people do develop PTSD after trauma, peritraumatic dissociation occurs quite frequently during trauma. This is true for both detachment (Engelhard, Van den Hout, Kindt, Arntz, & Schouten, 2003; Laposa & Alden, 2003; Ursano et al., 1999), and compartmentalization (e.g., involuntary immobility; Galliano, Noble, Puelch, & Travis, 1993).

PTSD trauma memory

The second subject of investigation concerned the uniqueness of trauma memory in PTSD. More specifically, we compared trauma memories of PTSD patients with panic attack memories of patients with panic disorder with agoraphobia (PDA), and trauma memories of healthy participants (controls) who had experienced a traumatic or highly anxious event (Chapter 5). PTSD trauma memories differed from PDA panic memories and trauma memories of controls regarding intrusion characteristics (e.g., intrusiveness, controllability, vividness, and sensory impressions). That is, PTSD trauma memories were perceived as having more intrusion characteristics than memories of the other two groups, although PDA panic memories still were more intrusive than trauma memories of controls. This suggests that trauma memory may not be qualitatively distinct from other memories but instead suggests a continuum. Furthermore, the memories of PTSD and PDA were similarly disorganized (more than the memories of controls), and the intensity with which they relived the event during intentionally recounting was rated as equally high in both patient groups. Finally, both peritraumatic dissociation and memory-associated dissociation were higher in both PTSD and PDA patients, relative to the healthy controls.

Thus, trauma memory in PTSD may not be as special as it is sometimes thought to be. Although some theories posit that traumatic memory is stored in fragments, and as a result not integrated in autobiographical memory (e.g., Van der Kolk & Fisler, 1995), recent research on fragmentation and organization of PTSD trauma memories do not always confirm this view (e.g., Geraerts et al., 2007). It has also been posited that highly emotional and important events help to keep the autobiographical memory integrated instead of leading to disintegration. In this view, these events form reference points for the organization of other, less important and less emotional, events (Rubin & Kozin, 1984; Shum, 1998). These by itself functional mechanisms, would be dysfunctional in case of trauma, because the traumatic event would bias the interpretation of other events. Interestingly, our study showed that fragmentation also characterized PDA memories. It is possible that strong emotions may be responsible for the fragmentary, disorganized nature of recounted memories, for example by disabling adequate memory retrieval. Memory fragmentation could just be an epiphenomenon reflecting anxiety or fear experienced

during recounting (Zoellner & Bittenger, 2004, pp155). The healthy participants probably did not experience such strong emotions during memory retrieval, and indeed they did not perceive their memories as fragmented. Similarly, decreases in fragmentation and disorganization during exposure treatment for PTSD were not related to PTSD symptoms or treatment effects, but instead they were epiphenomena of repeatedly recollecting the trauma memories (Van Minnen, Wessel, Dijkstra, & Roelofs, 2002). The fact that in our study there was no difference in reliving (including vividness, fear, and heart beat during recounting the memory) also showed that PTSD trauma memories were as vivid and as real as PDA panic memories.

Peritraumatic dissociation is often thought to be responsible for the fragmentary storage of information during the trauma, and for the intrusive character of PTSD memories (e.g., Van der Kolk & Fisler, 1995). However, we found neither peritraumatic or nor current dissociation (i.e., detachment) to be related to memory disorganization. In addition, current, memory-associated dissociation was related to intrusion characteristics, peritraumatic dissociation was not. Thus, we may need an alternative explanation for the differences in intrusion characteristics in the memories of PTSD and PDA patients. One possibility would be that a traumatic event typically occurs outside oneself, drawing one's attention to external sensory impressions, whereas panic attacks typically occur within oneself thereby drawing attention to one's own physical sensations. Yet another explanation may be that the appraisal of the panic or trauma memory is different in PDA and PTSD patients. In PDA patients, it is not the memory itself but the physical symptoms experienced during the panic attack that are appraised negatively, thus there is no need to avoid the memory of the panic attack, allowing the initial memory to be integrated into autobiographical memory. In PTSD patients, having the memory itself may be appraised negatively and hence avoided, which can hinder adequate processing of the memory. Indeed, initial appraisal of (intrusive) symptoms has been shown to contribute to the maintenance of the symptoms in PTSD patients (Chapter 2; Halligan, Michael, Clark, & Ehlers, 2003). Finally, vivid, intrusive memories do not occur in PTSD only. On the contrary, they even occur after positive events (Berntsen, 2001), suggesting that vivid, intrusion-like memories are the result of normal memory processes, where factors like high intensity, distinctiveness and importance of the event and subsequent rehearsal (rather than negative affect *per se*) result in the formation of vivid memories (Berntsen, 2001).

The study described in Chapter 5 was strong in using a narrative rating as well as a meta-memory method to investigate memory characteristics. Indeed, meta-memory and actual memory have been shown to be different (Kindt & Van den Hout, 2003). Moreover, to our knowledge, it is the first study to compare PTSD with another psychiatric disorder that involves fear conditioning after an emotionally intense event. Yet, one limitation of our study was the fact that the first study, using a narrative coding method, concerned intentional memories and therefore, intrusion characteristics could not be assessed. Reliving while deliberately recounting the memory in the narrative coding showed some overlap the Intrusion characteristics in the meta-memory study but nevertheless, a direct comparison is not possible. Another limitation was the fact that peritraumatic dissociation had to be measured

retrospectively, thus interpretations must be made with caution. Unfortunately, dissociation was not assessed in the narrative coding study, thus dissociation findings of the meta-memory study remain to be replicated. It would indeed be interesting to assess dissociation and investigate differences in dissociation between psychiatric disorders in a narrative coding design.

In sum, our study suggested that the trauma memory of PTSD patients does not have a unique structure comprising dissociation, fragmentation and disorganization, and reliving. Moreover, PTSD and PDA patients reported similar levels of both peritraumatic and memory-associated dissociation. In addition, peritraumatic and memory-associated dissociation were not related to memory disorganization, and only memory-associated dissociation (and not peritraumatic dissociation) was associated with increased intrusion characteristics.

Dissociation and PTSD treatment efficacy

In Chapter 6 of this dissertation, the impact of three sorts of dissociative phenomena (trait dissociation, depersonalization and numbing) and depression on exposure treatment was studied. Note that depersonalization and numbing can both be categorized as manifestations of detachment, whereas trait dissociation reflects a tendency to experience symptoms of both detachment and compartmentalization. It was found that numbing, depersonalization, and depressive symptoms decreased through treatment. In addition, none of the three dissociative phenomena nor depression negatively affected treatment efficacy. Patients with severe trait dissociation, depersonalization, and numbing, and those with a current or past depression benefited from treatment similarly as patients with minimal trait dissociation, depersonalization and numbing, and patients without (a history of) depression. Higher posttreatment PTSD symptom levels proved due to higher pretreatment PTSD symptoms levels. Remarkably, the reduction of PTSD symptoms from pretreatment to follow-up was even higher in patients with high numbing levels than they were in those with minimal numbing symptoms, and a similar trend was found for patients with high levels of depersonalization. Patients high on trait dissociation and those with a current or past depression experienced more PTSD symptoms at all assessments (pre, post, and follow up) than patients with low trait dissociation and those without a (history of) depression. As several studies showed trait dissociation and neuroticism to be related (Goldberg, 1999; Kwapil, Wrobel, & Pope, 2002), patients with high trait dissociation may have a personality trait that makes them chronically more vulnerable to stress, resulting in a chronically relatively high level of arousal.

Dissociation has been hypothesized to hinder effective exposure treatment because it impedes fear activation during exposure. However, in our study none of the three dissociative phenomena nor depression did impede fear activation during exposure. In fact, all these variables were related to higher levels of fear during exposure, although this association disappeared after controlling for initial PTSD severity. This may mean that dissociation does not protect the individual against experiencing high levels of distress, but it may rather be an epiphenomenon of high

stress. Indeed, initial PTSD severity (indicating higher levels of distress) was responsible for the association between dissociative and depressive symptoms, and fear during exposure. Other studies also found high levels of distress to be related to high levels of peritraumatic dissociation (Fikretoglu et al., 2006; Fikretoglu et al., 2007). An alternative explanation may be that numbing is a coping reaction to reduce distress (Litz et al., 1997), which can be switched off, which in return results in new, and thus extra high, stress.

The findings of the study described in Chapter 6 have some important implications for clinical practice. First of all, dissociation and depression do not seem to be prohibitive for exposure treatment for PTSD. In fact, dissociative symptoms, i.e. numbing and depersonalization, and depressive symptoms even decreased during treatment along with (other) PTSD symptoms. Of course, our study merits replication and extension, but at least it suggests that PTSD patients with dissociative symptoms or depressive patients should not be denied exposure treatment. Possibly, dissociative symptoms in PTSD are just a way of coping with anxiety (Elzinga, Bermond, & Van Dyck, 2002), which is no longer needed in case of a reduction or absence of PTSD symptoms. If dissociation is used as an avoidance strategy (Foa & Hearst-Ikeda, 1996; Litz, 1992; Spiegel, 1997), exposure may even be specifically indicated.

In conclusion, because this was the first study to investigate the impact of dissociation on the efficacy of exposure treatment in PTSD, future research on this issue is needed to see if our findings can be replicated. Although already several dissociative phenomena were investigated, it would be interesting to investigate the impact of still other dissociative phenomena (like dissociative amnesia) on treatment efficacy. As yet, based on the treatment study described in Chapter 6, there are no indications that dissociation is hindering the efficacy of PTSD exposure treatment.

General conclusion

The studies described in this dissertation used quite distinct methodologies, and yet their outcomes were rather stable. It was consistently found that dissociation was not related to PTSD development or to memory formation, nor did it affect the efficacy of prolonged exposure treatment. By using diverse, controlled, prospective, and experimental designs we tried to overcome the methodological problems of previous studies in this area. For instance, we controlled for initial PTSD symptoms, distinguished between various forms of dissociation (including compartmentalization) and included alternative explaining factors like dysfunctional cognitions (Chapter 2) and non-movement (Chapter 4) in the predictive models. Several studies described in this dissertation found that dissociation had no impact anymore after controlling for (specific) PTSD symptoms (Chapter 2 and 6). In fact, some of our studies (Chapter 5 and 6) showed that when anxiety and stress decreased, so did dissociative symptoms, which seems to support the notion that dissociation is a normal response in coping with high levels of stress and anxiety. Despite these

consistent findings and our best efforts to ensure effective designs, several shortcomings must be noted.

The most important factor that may complicate the interpretations of the studies is the construct of dissociation. The categorization in the Introduction (Chapter 1) was meant to structure the often blurred use of the term dissociation. It was also tried in each chapter to carefully describe what form of dissociation was addressed. Yet, it still proved extremely difficult to clearly and consistently define and measure the various forms of dissociation. In one study (Chapter 5), we even measured peritraumatic dissociation retrospectively, despite the potentially confounding effects of such method. Also, our investigations are by no means exhaustive as certain forms of dissociation have not been looked at. For instance, some expressions of compartmentalization (e.g., amnesia) have not been studied. It may very well be that different forms of dissociation affect the development and maintenance of or the recovery from PTSD differently. Obviously, more research is needed that does not handle dissociation as a total concept but rather probes its various manifestations.

Although experimental designs have the advantage that they allow the study of processes in a highly controlled setting, their ecological validity remains uncertain. For instance, in our experimental study on intrusion development (Chapter 4), a highly aversive film was used as an analogue to trauma as ethics preclude the use of a genuinely traumatic film. Therefore, interpretations and generalizations must be made with caution. Only a series of experimental studies on PTSD development will allow more definitive conclusions to be drawn. On the other hand, in non-experimental studies it proved difficult to control for the different factors that could be of importance with respect to PTSD development, and to achieve adequate power. Accordingly, our prospective study (Chapter 2) typically lacked power due to the limited number of participants and the relatively high level of dropouts and missing data.

In conclusion, regardless of the various limitations, the findings of the studies described in this dissertation had quite a stable pattern that suggests that the role of dissociation in the development of and treatment of PTSD may be overrated. The current findings merit replication and extension, especially in experimental studies that exploit different methods of inducing different forms of dissociation in order to study the impact of peritraumatic dissociation on trauma processing. Equally important, besides dissociation other factors, such as interpretation and attribution of symptoms and evolutionary-based responses like non-movement, must also be included to grant a more complete understanding of trauma processing and the development and maintenance of PTSD.

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Appendix A

Catalepsy Questionnaire

Indicate to what extend you experienced the feelings mentioned in the questionnaire *during catalepsy*:

		Not at all				Completely
1.	as if the right arm was easy to bow	1	2	3	4	5
2.	a tingling sensation in the right arm	1	2	3	4	5
3.	as if the left arm was hard to bow	1	2	3	4	5
4.	as if the right arm was light	1	2	3	4	5
5.	as if the entire left arm belonged to you	1	2	3	4	5
6.	as if the left arm felt strange	1	2	3	4	5
7.	as if both your arms felt different than usual	1	2	3	4	5
8.	as if the right arm was hard to bow	1	2	3	4	5
9.	a normal feeling in the right arm	1	2	3	4	5
10.	a normal feeling in the left arm	1	2	3	4	5
11.	as if (a part of) the right arm was gone or unreal	1	2	3	4	5
12.	numb feeling in the right arm	1	2	3	4	5
13.	as if the entire right arm belonged to you	1	2	3	4	5
14.	as if the right arm felt strange	1	2	3	4	5
15.	as if the left arm was easy to bow	1	2	3	4	5
16.	as if de left arm was light	1	2	3	4	5
17.	as if the right arm was heavy	1	2	3	4	5
18.	a numb feeling in the left arm	1	2	3	4	5
19.	as if the left arm was heavy	1	2	3	4	5
20.	a tingling sensation in the left arm	1	2	3	4	5
21.	as if (a part of) the left arm was gone or unreal	1	2	3	4	5
22.	no differences experienced between the left and the right arm	1	2	3	4	5

Appendix B

Protocol for catalepsy

1. Information
Before the procedure starts general information is given to the participant and the participant is reassured and told that it concerns a simple experiment without any negative consequences. The participant is also told that there is nothing to worry about, it is perfectly safe and no risks are involved.
The explanation is as follows:

> "What I am going to do in a minute, is bring your arm in a catatonic state. It is called catalepsy. You may know it from snakes. They are often seen to be standing stock-still for a long time. There is also these 'living statues', people that pretend to be a statue and are able not to move for a long time. They can do that because they bring themselves in a total body catalepsy, the same kind of stiff state that I am about to bring your arm in. The mechanism is the following. Normally there is gravity and your muscles constantly react to that. Your arm normally will be pulled down by gravity. So your muscles have a basic tension to compensate gravity. They are adjusted to the gravity condition that is always there. What I am going to do is confuse the muscles of your arm. I am going to make the muscles 'feel' as if gravity is no longer there. That is, I am going to provide information that is not consistent with gravity. As a result the muscles in your arm will get a different tonus. This is something that happens automatically, you don't have to do anything yourself. As a result of that changed muscle tonus, you will feel a stiffness in that particular arm. It will also be easy to keep that arm in the same position. In order to achieve this state, I will move your forearm up and down and at the same time I will block that movement. It is nothing special, really. The stiffness will disappear after you have shaken your hands a bit."

2. Procedure
The person that induces the catalepsy (E) takes the wrist of the participant (P) in his hands. The elbow of P rests against the arm of a chair. E takes the forearm from its horizontal position and lifts it a bit higher. Then, E pushes the forearm of P slightly and slowly up and down, alternatively supporting and releasing the forearm. E pushes the upper muscles down as the arm goes up and pushes the under muscles up as the arm goes down, just opposite of the natural movement muscle contractions. In other words, E pushes the forearm a little bit up and a little bit down and blocks this movement at the same time. E pushes the forearm always until a point where E feels some sort of resistance in it. No suggestions about dissociation are given at all. Only suggestions of stiffness of the arm are given. P does not have to do anything. Because people tend to find it hard to do nothing and ignore what E is doing, one could distract P by small talk for example. After 1 to 5 minutes of pushing the forearm up and down in decreasing amplitude, the arm will be in catalepsy. E checks if catalepsy is indeed present by softly pushing the arm down. Catalepsy is present if the arm returns to its elevated position, as is seen in tonic immobility. If P is trying to 'help', by moving the forearm actively, no catalepsy will be attained. In that case, E has to emphasize that P does not have to do anything, because the effect will occur naturally.

3. Debriefing
E asks P what he/she has felt. E suggests that catalepsy is more easily reached as P has done it more often. It's like 'the arm has learned what it has to do'. E also normalizes the procedure and states that nothing out of the ordinary has happened.

Summary

The focus of this dissertation is the impact of dissociation on the development and treatment of posttraumatic stress disorder (PTSD), and the uniqueness of traumatic memory in PTSD. Dissociation is a complex concept that includes a wide range of symptoms. There is some debate about whether distinct dissociative symptoms reflect one underlying dimension, or whether they in fact reflect different factors and, thus, distinct forms of dissociation. One categorization distinguishes two different forms of dissociation with distinctive underlying mechanisms: detachment and compartmentalization. Detachment refers to experiences that concern an altered state of consciousness, in which people feel detached from their bodies, their sense of self, or the world around them. Depersonalization, derealization, and emotional numbing are examples of detachment. Compartmentalization refers to experiences that imply a deficit in the ability to deliberately control processes that would normally be controllable. Dissociative amnesia, conversion paralysis, sensory loss or pseudo-hallucinations, and forms of somatoform dissociation are examples of compartmentalization. In this dissertation the association between PTSD and both detachment and compartmentalization was investigated.

First, in Chapter 2, it was investigated whether peritraumatic dissociation, i.e. dissociation occurring during the traumatic event, is related to the development of PTSD. Although some studies suggest such an association, they suffer from several methodological shortcomings such as using retrospective designs and omitting to control for initial PTSD(-related) symptoms. Furthermore, most previous studies investigated detachment and not compartmentalization. The current study therefore investigated the impact of both peritraumatic psychological dissociation (detachment) and somatoform dissociation (compartmentalization), while controlling for early numbing symptoms. This was done because numbing symptoms are part of PTSD but also reflect detachment, and therefore, the effect of peritraumatic dissociation could be tautologuous. Indeed, after controlling for initial numbing symptoms, neither of the two forms of peritraumatic dissociation predicted PTSD development at 6 months. Interestingly, dysfunctional cognitions did still predict PTSD, suggesting that the interpretation of the trauma and any ensuing symptoms is an important factor in the development of PTSD. In contrast, the original predictive power of dissociation may be due to the overlap between dissociation and PTSD.

In Chapter 3, the usefulness of catalepsy as a research paradigm was examined. Catalepsy in the right arm of healthy controls proved to change the perception of that arm. Compared to the non-cataleptic left arm, it not only showed signs of increased rigidity, but also of paresthesias, a lowered perception of sense and a diminished awareness of that arm. This change in perception was related to the item of the Stanford Hypnotic Susceptibility Scale that addresses hypnotically induced arm immobilization. Based on the results it was concluded that catalepsy indeed is a useful paradigm for the study of motor-perceptual dissociative pheno-

mena because it elicits a variety of dissociative symptoms.

In Chapter 4, catalepsy was used to induce dissociative non-movement in healthy participants in an experimental study that explored the impact of peritraumatic dissociation on one specific PTSD symptom, namely intrusion development. Similar to what was found in the study reported in Chapter 2, dissociation (in this case somatoform dissociation induced by catalepsy) was not associated with increased intrusion frequency. Instead, non-movement per se (dissociative non-movement and deliberate non-movement conditions combined) was related to increased intrusion frequency, as compared to a free-to-move control condition. Spontaneous dissociation across groups proved positively related to an implicit bias to trauma-related words and negatively related to explicit recall, but not related to intrusion frequency. In conclusion, dissociation during an aversive film, used as an analogue for peritraumatic dissociation, was not associated with intrusion development.

In conclusion, the presumed association between peritraumatic dissociation and PTSD was not found after controlling for early numbing symptoms (Chapter 2), or non-movement per se (Chapter 4), suggesting peritraumatic dissociation might be an epiphenomenon of PTSD, or even a byproduct of strong emotions, and not an independent predictor.

In Chapter 5, the uniqueness of trauma memory in PTSD was explored. Using a narrative rating method no differences were found in the intensity of reliving the event or in the disorganization of the memory between PTSD patients (while recounting their trauma) and patients with panic disorder with agoraphobia (PDA, while recounting their first or worst panic attack). The subsequent meta-memory study revealed that PTSD trauma memories were characterized by more intrusion characteristics (e.g., controllability, intrusiveness, sensory impressions) than PDA panic memories and trauma memories of healthy controls. However, the memories of the PDA group had more intrusion characteristics than the memories of the controls, suggesting a continuum of memory processing rather than a qualitatively different process in PTSD. The memories of both PTSD and PDA patients were disorganized relative to the memories of the controls. Peritraumatic and memory-associated dissociation were high in both patient groups and not in the controls. Furthermore, neither peritraumatic dissociation nor memory-associated dissociation was related to memory disorganization, and only memory-associated dissociation was related to intrusion characteristics. The results suggest that perhaps dissociation affects memory-retrieval and not necessarily the encoding of information into memory.

The study described in Chapter 6 investigated the impact of three dissociative phenomena (trait dissociation, depersonalization, and numbing) and depression on the efficacy of exposure treatment for PTSD. Neither of the three dissociative phenomena, nor depression predicted improvement. Quite the reverse: dissociative and depressive symptoms all decreased during therapy. Patients with a current or past depression and those with severe dissociative symptoms benefited equally from the treatment as non-depressed patients and those with minimal dissociative symptoms. Moreover, pretreatment dissociative and depressive symptoms seemed to be positively related to fear during exposure, but this association disappeared after pretreatment PTSD severity had been controlled for. This may indicate that

dissociation is just an epiphenomenon of high stress. Based on these results it was concluded that PTSD patients with high trait dissociation, depersonalization, and numbing, as well as depressive patients, should not be denied exposure treatment as often is the practice today.

The theoretical implications of the five studies reported in this dissertation concern the association between dissociation and PTSD. The current findings are in line with the results of other recent, controlled studies, and suggest that the role dissociation plays in the development of PTSD, or in memory formation in PTSD, may not be as crucial as previously assumed. Moreover, dissociation did not have an impact on treatment efficacy, did not impede fear activation during exposure, and even declined after treatment. This suggests that dissociation may be an epiphenomenon of fear which diminishes when PTSD symptoms decline.

The clinical implications of the study discussed in Chapter 6 are obvious, but nevertheless extremely important: PTSD patients with even severe dissociative symptoms should not be denied exposure treatment. In addition, as peritraumatic dissociation does not seem to predict PTSD development, trauma victims and PTSD patients should be explained that such dissociative phenomena are perfectly normal reactions to a traumatic event such as they have experienced.

Samenvatting

De studies in deze dissertatie richten zich op het effect van dissociatie op de ontwikkeling en behandeling van posttraumatische stress stoornis (PTSS) en de uniekheid van het trauma geheugen in PTSS. Dissociatie is een uitermate complex concept dat naar een veelheid van symptomen verwijst. Er is veel discussie over of verschillende dissociatieve symptomen uitingen zijn van één onderliggende dimensie of dat ze een uiting zijn van verschillende factoren en er dus te onderscheiden vormen van dissociatie bestaan. Verschillende malen is getracht orde te brengen in de verschillende manifestaties van dissociatie. Een van deze categorisaties onderscheid twee verschillende vormen van dissociatie met eveneens twee verschillende onderliggende mechanismen: vervreemding of onthechting ("detachment") en compartimentalisatie. Onder vervreemding vallen ervaringen die op de een of andere manier een veranderde manier van bewustzijn betreffen, waarbij men zich vervreemd voelt van het eigen lichaam, van zichzelf of van de omgeving. Voorbeelden van vervreemding zijn depersonalisatie, derealisatie en emotionele verdoving. Onder compartimentalisatie vallen ervaringen die een gebrek suggereren in de vaardigheid om willekeurige processen te controleren die gewoonlijk controleerbaar zijn. Voorbeelden zijn dissociatieve amnesie, conversieve paralyse, verlies van zintuiglijke waarneming, pseudo-hallucinaties en vormen van somatoforme dissociatie. De studies in deze dissertatie onderzochten het verband tussen PTSS en zowel vervreemding als compartimentalisatie.

In Hoofdstuk 2 werd onderzocht of peritraumatische dissociatie (dissociatie tijdens de traumatische gebeurtenis) is gerelateerd aan de ontwikkeling van PTSS. Hoewel sommige studies een dergelijk verband suggereren, is het de vraag of dit er wel echt is. De resultaten van veel van deze studies zijn namelijk moeilijk te interpreteren vanwege een aantal methodologische tekortkomingen zoals het gebruik van retrospectieve designs en het niet controleren voor initiële PTSS(-gerelateerde) symptomen. Bovendien onderzochten de meeste studies dissociatieve symptomen die een uiting zijn van vervreemding en niet van compartimentalisatie en het is mogelijk dat deze verschillende soorten dissociatie een verschillend effect hebben op het verwerken van een traumatische gebeurtenis. De huidige studie onderzocht daarom het effect van zowel peritraumatische psychologische als somatoforme dissociatie (respectievelijk vervreemding en compartimentalisatie) terwijl er gecontroleerd werd voor initiële symptomen van emotionele verdoving. Dit laatste werd gedaan, omdat het vervreemdings-dissociatie symptoom emotionele verdoving een symptoom is van PTSS, waardoor het effect van peritraumatische dissociatie tautologisch zou worden. Inderdaad voorspelden geen van de beide vormen van dissociatie de ontwikkeling van PTSS meer, nadat gecontroleerd was voor initiële symptomen van emotionele verdoving. Opmerkelijk genoeg bleven disfunctionele cognities wel PTSS voorspellen, hetgeen suggereert dat de interpretatie van het trauma en de gevolgen ervan een belangrijke factor is in het ontstaan van PTSS. De aanvankelijke

predictieve power van dissociatie zou daarentegen het gevolg kunnen zijn van de overlap tussen dissociatie en PTSS.

In Hoofdstuk 3 werd de bruikbaarheid van katalepsie als een onderzoeksparadigma bestudeerd. Gezonde proefpersonen bij wie de rechter arm in een kataleptische staat was gebracht rapporteerden een veranderde perceptie van die arm. Vergeleken met de linker, niet-kataleptische arm, was er in de kataleptische arm sprake van toegenomen rigiditeit, maar daarnaast was er ook sprake van paresthesieën, een verminderde perceptie van gevoel en een verminderd bewustzijn van de rechter arm. Deze verandering in perceptie was gerelateerd aan het item van de Stanford Hypnotic Susceptibility Scale dat hypnotisch geïnduceerde arm immobiliteit meet. Er werd geconcludeerd dat katalepsie inderdaad een bruikbaar paradigma vormt voor onderzoek naar motor-perceptuele dissociatieve fenomenen, omdat het naast rigiditeit ook verschillende dissociatieve symptomen oproept.

In Hoofdstuk 4 werd een experimentele studie beschreven waarin katalepsie gebruikt werd om dissociatieve bewegingloosheid te induceren in gezonde deelnemers teneinde de impact van peritraumatische dissociatie op één specifiek PTSS symptoom, intrusies, te onderzoeken. In overeenstemming met de resultaten van Hoofdstuk 2, was ook hier dissociatie (in dit geval somatoforme dissociatie geïnduceerd door katalepsie) niet gerelateerd aan een grotere frequentie van intrusies aan het trauma. Een aversieve film was gebruikt als analoog voor trauma. Verrassend was dat bewegingloosheid op zich (de dissociatieve bewegingloosheid en opzettelijke bewegingloosheid condities samen) wel gerelateerd was aan een toename van het aantal intrusies vergeleken met de controle conditie die naar wens mocht bewegen. Spontane dissociatie over condities heen hing positief samen met een impliciete bias voor trauma-gerelateerde worden en hing negatief samen met expliciete recall, maar niet met intrusie frequentie. Kortom, dissociatie tijdens een aversieve film ('peritraumatische dissociatie') was niet gerelateerd aan intrusie frequentie, bewegingloosheid wel.

In de eerste paar hoofdstukken werd de relatie tussen peritraumatische dissociatie en PTSS niet gevonden nadat er gecontroleerd was voor aanvankelijke symptomen van emotionele verdoving (Hoofdstuk 2) of bewegingloosheid op zich (Hoofdstuk 4). De resultaten suggereren dat peritraumatische dissociatie een epifenomeen van PTSS is, of een bijproduct van sterke emoties, en niet een onafhankelijke predictor.

In Hoofdstuk 5 werd de uniekheid van het PTSS trauma geheugen onderzocht. Gebruik makend van het scoren van narratieven werd geen verschil gevonden in de intensiteit van het herbeleven van de gebeurtenis of in de mate van disorganisatie van de herinnering tussen PTSS patiënten (die hun trauma herbeleefden) en patiënten met een paniekstoornis met agorafobie (PSA, die hun eerste of ergste paniekaanval herbeleefden). In de daaropvolgende studie, gebruik makend van meta-beoordeling van de herinnering door de deelnemers zelf, werd gevonden dat PTSS trauma herinneringen gekarakteriseerd werden door meer intrusiekenmerken (zoals controleerbaarheid en intrusiviteit van de herinnering en sensorische impressies) dan PSA paniek herinneringen en trauma herinneringen van een gezonde controlegroep. Echter, de herinneringen van de PSA groep hadden ook meer intrusiekenmerken dan de trauma herinneringen van de controlegroep, hetgeen eerder

een continuüm in geheugen processen suggereert dan een qualitatief ander en uniek geheugen proces in PTSS. De herinneringen van zowel PTSS als PSA patiënten waren gedesorganiseerd vergeleken met de herinneringen van de controlegroep. Peritraumatische en herinnering-gerelateerde dissociatie waren hoog in zowel PTSS als PSA patiënten en niet in de controlegroep. Bovendien waren deze beide vormen van dissociatie niet gerelateerd aan disorganisatie van de herinnering en alleen herinnering-gerelateerde dissociatie was positief gerelateerd aan intrusiekenmerken in de herinnering. De resultaten zouden kunnen betekenen dat dissociatie het ophalen van herinneringen beïnvloed en niet het opslaan van informatie in het geheugen.

De studie die beschreven wordt in Hoofdstuk 6 onderzocht de impact van drie dissociatieve fenomenen (trait dissociatie, depersonalisatie en emotionele verdoving) en depressie op de effectiviteit van prolonged exposure therapie bij PTSS. Geen van de dissociatieve fenomenen of depressie voorspelden verbetering. Het omgekeerde was zelfs het geval: dissociatieve en depressieve symptomen namen af tijdens de therapie. Patiënten met een huidige depressie of depressie in het verleden en patiënten met ernstige dissociatieve symptomen profiteerden evenveel van de behandeling als niet-depressieven en patiënten met minimale of geen dissociatieve symptomen. Dissociatieve en depressieve symptomen leken samen te hangen met meer angst tijdens exposure, maar deze samenhang verdween nadat er gecontroleerd werd voor de ernst van de PTSS. Dit kan wederom betekenen dat dissociatie een epifenomeen of symptoom is van een hoog stressniveau. Op basis van de resultaten werd geconcludeerd dat PTSS patiënten met een hoog niveau van trait dissociatie, depersonalisatie en emotionele verdoving en depressieve patiënten niet uitgesloten moeten worden van exposure therapie, zoals dat nu vaak wel gebeurt.

De theoretische implicaties van de vijf studies die in deze dissertatie gerapporteerd worden betreffen de associatie tussen dissociatie en PTSS. De huidige bevindingen komen overeen met andere recente, gecontroleerde studies en suggereren dat de rol van dissociatie in de ontwikkeling van PTSS en in de vorming van een trauma geheugen in PTSS niet zo cruciaal is als voorheen aangenomen werd. Bovendien had dissociatie ook geen effect op een exposure behandeling en belemmerde het niet een angstactivatie tijdens exposure. Dissociatieve symptomen namen zelfs af tijdens de behandeling. Dit alles doet vermoeden dat dissociatie een epifenomeen of symptoom is van angst, dat vermindert wanneer PTSS symptomen verminderen.

De klinische implicaties van de studie die in Hoofdstuk 6 beschreven wordt liggen voor de hand, maar zijn desalniettemin van groot belang: PTSS patiënten met ernstige dissociatieve symptomen zouden niet uitgesloten moeten worden van exposure behandeling. Aangezien peritraumatische dissociatie niet de ontwikkeling van PTSS lijkt te voorspellen, is het daarnaast van belang trauma slachtoffers en PTSS patiënten uit te leggen dat dergelijke dissociatieve fenomenen normale reacties zijn op een extreem emotionele situatie.

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Curriculum Vitae

Muriel Hagenaars was born on October 21, 1971 in Tilburg. She completed secondary education (gymnasium β,) in 1989 at the Mill Hill College in Goirle. She then studied Clinical Psychology (with specialisation Neuropsychology) at the Radboud University Nijmegen and graduated in 1996. From 1996 onwards, she worked as a psychologist in several outpatient clinics (HSK Group, the Ambulatorium of the Radboud University Nijmegen, and an Outpatient clinic for anxiety disorders of the GGZ Nijmegen) while in a post-doctorate training for psychotherapist. In the meantime she had become interested and involved in research and in 2000 she started a PhD project at the Department of Clinical Psychology at the Radboud University Nijmegen, carrying out the studies described in this dissertation. She also still worked at the Outpatient clinic for anxiety disorders during this period and became a registered Psychotherapist in August 2005. Since October 2007 she is employed as assistant professor at the department of Clinical, Health and Neuropsychology at Leiden University.