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FUNCTIONAL BRAIN IMAGING AND THE INDUCTION OF TRAUMATIC RECALL: *A Cross-Correlational Review Between Neuroimaging and Hypnosis*

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Abstract: The behavioral and psychophysiological alterations during recall in patients with trauma disorders often resemble phenomena that are seen in hypnosis. In studies of emotional recall as well as in neuroimaging studies of hypnotic processes similar brain structures are involved: thalamus, hippocampus, amygdala, medical prefrontal cortex, anterior cingulate cortex. This paper focuses on cross-correlations in traumatic recall and hypnotic responses and reviews correlations between the involvement of brain structures in traumatic recall and processes that are involved in hypnotic responsiveness. To further improve uniformity of results of brain imaging specifically for traumatic recall studies, attention is needed for standardization of hypnotic variables, isolation of the emotional process of interest (state), and assessment of trait-related differences.

In the last 10 years, there has been a rapid increase in our understanding of the brain processes that are involved in processing of traumatic events (see Stern & Silbersweig, 2001). Much of this research is related to the processing of stress, memory, and emotion (see reviews of Armony & LeDoux, 1997; Baddeley et al., 2000; Bremner, Krystal, Southwick, & Charney, 1995; Bremner & Narayan, 1998; Cahill, 2000; LeDoux, 1993; McGaugh, Cahill, & Roozendaal, 1996; Nijenhuis,

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Van der Hart, & Steele, 2002; Packard & Cahill, 2001; Phillips, Drevets, Rauch, & Lane, 2003a, 2003b; and the special issues of the *International Journal Clinical and Experimental Hypnosis*, April and July 2003). Among the factors that contributed to this increase are the availability of high-quality functional brain imaging techniques, cross-fertilization of different disciplines (e.g., cognitive and developmental psychology, nuclear medicine, pharmacology, molecular biology, psychiatry), and the increasing specificity of induction procedures for traumatic recall in study protocols. (For a description of the characteristics of two of the main neuroimaging methods, functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), see the Appendix). Innovative experimental designs in the field of neuroimaging have increased our understanding of basic processes of memory storage and emotion processing, lesion studies have pointed to brain regions that are critically involved, and clinical studies in a variety of patient populations have revealed a neural circuitry that is involved in different disorders. These developments have also further contributed to our current understanding of brain processes involved in pain, the phenomenology of consciousness, and emotional processing in general and have led to an understanding of regional patterns of activation and deactivation.

Still, little is known about this neural circuitry that underlies (alteration of) perceptual processing in patients with psychopathology in which emotive processing is challenged in relation to exposure to traumatic events. This alteration of perceptual processing can be challenged by using visual, acoustic, or other sensory stimuli, or with personalized narratives that induce recall of traumatic events. The pattern of metabolic changes in the brain can be measured and correlated with the subjective emotional response. This emotive process may be paralleled by a set of involuntary/automatic processes that occur: effects in heart rate, perceptual and emotional alteration, time distortion, and analgesia (Hull, 2002), upon which patients may be selected for assessment of their regional blood flow patterns (Lanius et al., 2002). These studies have started to appear in scientific journals in the last 8 years but are still scarce.

Despite promising study results, the field of hypnosis has not fully used the momentum that arose from these developments. Several imaging studies in healthy populations have demonstrated differences in the neural circuitry that is involved in response patterns across hypnotic states, e.g., alterations of pain affect and pain modulation (Faymondville et al., 2003; Rainville, Duncan, Price, Carrier, & Bushnell, 1997), alteration of visual processing (Kosslyn, Thompson, Costantini-Ferrando, Alpert, & Spiegel, 2000), or hypnotic alteration of acoustic perception (Szechtman, Woody, Bowers, & Nahmias, 1998). Most of these studies have used high and low hypnotizable subjects to gain

insight in the neural mechanisms of perceptual alteration by measuring alteration in regional brain blood flow. From these studies, it appears that high hypnotizables are capable of modifying their brain metabolism in response to a specific set of instructions to alter affect, pain, or other experiences and have pointed out that subjects can differentially alter (block or stimulate) certain perceptual functions, e.g., ''taking the color out of a picture'' that is presented in front of them. To a considerable extent, high hypnotizables are capable of modifying the circuitry with which their brains process stimuli. To date, few of these studies have used the cumulative power of combining these knowledge-based resources in neuroimaging studies in patient populations.

It has been a decade since studies by Stutmann and Bliss (1985), Spiegel, Hunt, and Dondershine (1988), and Frischholz, Lipman, Braun, and Sachs (1992) empirically confirmed Janet's early notions (1889) that there is an overlap between the phenomena that are typically related to hypnosis and the phenomena occurring in emotional recall in posttraumatic stress disorder (PTSD). These patients have demonstrated enhanced susceptibility to ''hypnotic'' situations, which traumatic recall can be considered to be. Hypnotic induction can mobilize a wide spectrum of responses, varying from increased anxiety to flashbacks that can occur with or without feelings of detachment to other dissociative experiences, such as numbing or freezing, feelings of involuntariness, and loss of self-agency.'' Moreover, classic hypnotic responses such as time distortion, analgesia, and derealization can occur along with these memories. The content of the emotion is also widespread and can change rapidly depending on the focus of attention: e.g., anger, shame, guilt, or disgust. These responses can have bimodal effects, such as enhanced attention versus lowering of attention or out of body experiences versus detailed focus on details, and can also be reflected on the level of psychophysiological alteration, e.g., increased versus decreased heart rate. Although these may be related to hypnotic virtuosity, this has not been studied yet.

Within a general framework of identification, production, and regulation of emotional recall (see Phillips et al., 2003), hypnotic response patterns are related to the involvement of different brain correlates (Lanius et al., 2002). We pose that insight in these hypnotic response patterns needs to be taken into account when analyzing brain correlates of traumatic recall in trauma disorders, e.g., in PTSD but also in dissociative identity disorder (DID) and borderline personality disorder (BPD). Moreover, hypnotic paradigms can provide additional information regarding the involvement of involuntary mechanisms in traumatic recall. In addition, we feel that by cross-correlating the phenomenology and neurophysiology of traumatic recall and hypnosis similarities in parameters, results can be found that improve our

understanding of hypnosis and basic elements of consciousness and emotion. To explore this relation, we will review the imaging results in these studies.

Trauma as a Hypnotizing Agent

It is a known fact that traumatic stress can mobilize responses that have hypnotic features. These can be seen in a variety of situations, e.g., in the battered and abused child who creates an invisible identity so as not feel the pain and humiliation (*identity alteration*, *amnesia*, R. Loewenstein, personal communication, November 2000), in journalists when watching an execution as an eyewitness (*dissociation*; Freinkel, Koopman, & Spiegel, 1995), in survivors of the Estonia ferry disaster who attempted to rescue other survivors (*numbing*; Ericksson & Lundin, 1996), in people who witnessed victims jumping from the World Trade Center on September 11, 2001 (*verbal inhibition*, Spiegel, personal communication, 2001), or the responses in orphaned Rawandan children (*stupor*). Traumatic experiences can mobilize hypnotic responses that resemble the *hypnotic state* during which intense absorption in the hypnotic focal experience (Tellegen & Atkinson, 1974) can be achieved by means of a dissociation of experience (Hilgard, 1977; Spiegel et al., 1988, p. 301). It was Janet who described the splitting of consciousness that occurrs in response to traumatic stress and the consequences of trauma on memory and identity. Janet described a constellation of symptoms that we now categorize as PTSD or dissociative disorders, including dissociative amnesia and fugue, with a central assumption that different aspects of the traumatic experience are not actively available to consciousness, although they may have an influence on behavior (Loewenstein, 1993; Spiegel & Cardeña, 1991).

The psychological processes that were captured as core components of the hypnotic experience as described by Spiegel (1997) are: (a) *absorption*; (b) *dissociation*, and (c) *automaticity*. These three factors have been postulated because they explain the phenomena best in a hypnotic situation (Spiegel & Cardeña, 1991). Hypnosis has been best defined as an altered state of consciousness. Recently, a new definition of hypnosis has been coined in which the use of the word *hypnosis* as part of the hypnotic situation is not necessary for the induction nor description of the state of hypnosis (A. Barabasz, personal communication, September 2003).

Absorption is defined as a narrowing and intensification of attention, a disposition for having episodes of single total attention that fully engage one's representational resources (Tellegen & Atkinson, 1974). Physiological arousal can produce this narrowing of attention, which is directed more to central aspects of the traumatic experience than to

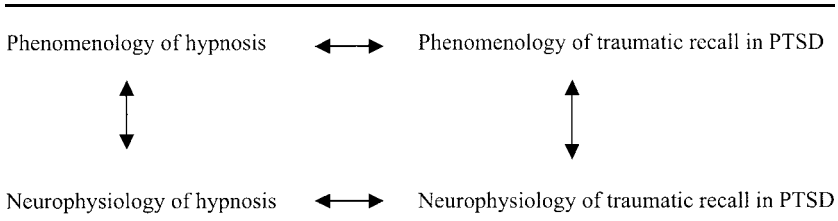

Figure 1. **Hypothesized similarities between hypnosis and traumatic recall. This diagram may be especially true for high hypnotizable subjects, which is usually the case in patients with PTSD. (Adapted from P. Rainville, personal communication, 2002).**

peripheral aspects (Christianson, 1992). Narrowing of attention can be functional in that all attention can be devoted to essential threat stimuli and defensive concerns. *Dissociation* can be described as a kind of divided or parallel access to awareness where several systems may have some independence. It refers to a compartmentalization of experience, which can be considered complementary to absorption. The term refers both to its origins, i.e., the splitting of consciousness that may occur during trauma, and to the subsequent process of associating or assigning experiences, as they occur over and over in time, to specific states of consciousness, ego centers, or affective states (Crabtree, 1992). Dissociation can also be part of an autohypnotic process (e.g., ''I am invisible; I have no feelings''), which is applied to reduce the perception of pain and the personal implications of trauma (Van der Kolk & Van der Hart, 1989). *Automaticity* may be defined as the tendency to automatically develop a belief in a suggested reality or the nonvolitional transformation of a suggested idea to a suggested effect (Van Der Hart & Van Der Kolk, 1991). Hypnotic automaticity reflects an altered sense of self-agency consistent with a modification of the property of *mineness* of self-generated intentions and voluntary actions. The involuntariness is captured in this description as well, representing the recognition of one's own volition and capacity to act (P. Rainville, personal communication, September 25, 2002; cf. Krystal, 1988). A diagram illustrating the shared neurophysiology of hypnosis with the neurophysiology of traumatic recall situations in highly hypnotizable subjects is illustrated in Figure 1.

HYPNOTIC SUSCEPTIBILITY IN TRAUMA-RELATED PSYCHOPATHOLOGY

A central theme in trauma-related psychopathology is that physical, emotional, or sexual trauma can play a major role in the shift of this control function manifesting psychological dysfunctions and/or bodily or somatic problems (Van Der Kolk et al., 1996). This can be viewed as a

disembodied process with an emphasis on the information processing analysis of attention mechanisms but also as a state of engagement of the body-self in the interaction with an object of consciousness, with emphasis on the biological substrate for the representation of self (Damasio, 1999) and the property of selfhood (Metzinger, 2000). This disembodiment can also be seen as a disengagement strategy that serves a natural defensive function (Gilbert, 2000).

Hypnotizability has been described as the fundamental capacity to experience dissociation in a structured setting. It underlies the ability to enter trance; it involves the ability to segregate and idiosyncratically encode experience into separate psychological or psychobiological processes (Janet, 1898). Like dissociation, hypnotizability can be related to a lack of agency or control versus loss of control over psychological and sometimes also physical functions. It is a dispositional term that points to its manifestation under certain circumstances, e.g., hypnotic induction. The critical alteration in these processes occurs in what Damasio called ''feeling of knowing,'' which is a fundamental aspect of self-reflective consciousness that can be separated in hypnosis (p. 280, 1999). Self-representation is a derivative of this fundamental function of consciousness. It is thought that in hypnosis, and also in traumatic situations, these representations can be disrupted or processed in separate streams of information. Self-representation is a hierarchically organized function with activity in some first-order maps in the brain that are necessary (but not sufficient) for higher-order representation of self (e.g., autobiographical self), regulation of cognition and behavior, and other more extended forms of consciousness.

From these notions, hypnotic capacity can be considered to be both a liability and an asset; from the perspective of a defense strategy, it serves a protective purpose (e.g., not remembering or not feeling), however it can also become maladaptive and lead to dysfunctions (e.g., time gaps, estrangement from inner feelings, flashbacks) and (psycho)pathology, like PTSD and dissociative or other trauma spectrum disorders. The disposition itself does not change but can be considered ''sensitized.'' The symptoms of the dissociative and post-traumatic states have been hypothesized to fit in a diathesis-stress model that views pathological dissociation as originating from an interaction between innate hypnotizability and traumatic experience (Butler, Duran, Jasiukaitis, Koopman, & Spiegel, 1996). If traumatic experiences involve a hypnotic process or induce a hypnotic state, then we should expect traumatized patients to show higher hypnotizability, in particular while still suffering from their trauma-induced disorder. One would expect that they have higher scores on classical hypnotizability scales than other psychiatric patient groups and healthy or trauma controls. Indeed, several studies supported the hypothesis that trauma-spectrum-disorder patients demonstrate

higher scores on classic hypnotic susceptibility scales than other psychiatric patient groups and normal control subjects (Frischholz et al., 1992; Spiegel et al., 1988; Stutman & Bliss, 1985). Their attention and arousal systems are altered, rendering them prone to entering hypnotic states, with a *relative* decoupling between irrelevant external events and mental (emotional) states during hypnotic states. It is not the experience of trauma; it is the psychopathology that accounts for the difference in hypnotic susceptibility. What happens with their hypnotic susceptibility after successful treatment is largely unknown. Although Janet observed that recovered patients became less hypnotizable (Janet, 1898), this finding still awaits testing in systematic research.

Recall of Traumatic Memories

The field of trauma spectrum disorders (consisting of PTSD, dissociative disorders, (DD)), and perhaps also borderline personality disorder (BPD, see Schmahl, McGlashan, & Bremner, 2002) has received a great deal of interest in brain imaging studies. The phenomenology of theses disorders is at the heart of the interface between memory and emotion.

Reexperiencing, Traumatic Recall, Flashbacks, and Flashbulb Memories

One of the most intriguing aspects of trauma disorders is the reexperiencing phenomena. Numerous labels and descriptions have been applied to this phenomenon (vanOyen Witvliet, 1997). In earlier days traumatic recall was also described as 'flashback', the reliving of the traumatic event with strong emotional involvement (Frankel, 1994). Flashback can lead to sleeping problems, irritability, feeling worse with traumatic reminders, and secondary avoidance. For a long time flashbacks were assumed to lack a recognizable neurophysiological correlate – therefore they were thought to be at least as likely to be the product of imagination as it is of memory (Frankel, 1994). However, in a recent study in 62 PTSD patients comparing flashbacks with ordinary autobiographical memory performance on cognitive tasks demonstrated that flashback periods were associated with a specific decrement in visuospatial processing, not specific with decrements on a verbal processing task. Flashback periods were found to be associated with increases in a wide range of autonomic and motor behaviors (Hellawell & Brewin, 2002).

Flashbacks share a phenomenology with what has been described by Brown and Kulik in 1977 as *flashbulb memory*, to refer to the vivid recollections that humans may have of events considered to be of particular significance to the individual. These memories were described as having a photographic quality and as being accompanied

by a strong apparel of contextual information (weather, background music, clothes worn, etc.) pertaining to the time and place where the event was first known. From a memory point of view we now know that these memories are not perfectly accurate and are subject to decay, but what does not necessarily decay is their capacity to evoke emotions similar to the ones felt upon when first exposed (Conway et al., 1994). We feel that flashbulb memories are formed by the activity of evolutionary old brain mechanism evolved to capture emotional and cognitive information relevant to the survival of the individual. In the modern neuroimaging era some of the original assumptions made by Brown and Kulik have since been challenged, but the phenomenon in question has remained an important area of research (Davidson & Glisky, 2002; Sierra & Berrios, 1999). The experiences share clinical features such as involuntary paroxysmal repetition, sensory vividness, and a capacity to trigger emotions like anxiety, shame, or anger.

We prefer to use the term '*traumatic recall*'. This can be defined as imaginary (or virtual) re-exposure to a traumatic event in which the person experienced, witnessed, or was confronted by death or serious injury to self or others, and responded with intense fear, helplessness, or horror, in which a re-experience of similar emotional responses occur. They usually differ from usual/normal (autobiographical) memories in their emotional involvement (Van Der Kolk & Van Der Hart, 1991). Their nature is that a recall of the helplessness and uncontrollability of the situation at that time, co-occurs with narrowing of the attention so that 'it feels like being back there' (i.e., when and where the traumatic event occurred). There can be a sense of loss of control or of self-agency (''That's not who I am'' or ''It is not me to whom that happened''). There can be an autonomic response (such as tachycardia, tachypnea, and diaphoresis) that can induce a feeling of panic (''I'm not going to make it''). The recall may be activated by a variety of trauma-related stimuli, thoughts about the trauma, the context, information about the trauma, and trauma-related images, sounds, or smells, all factors of which the person does not have to be aware. Veterans can reveal this effect potently when they are exposed to darkness and demonstrate augmented startle reflexes (Grillon, Morgan, Southwick, Davis, & Charney, 1996).

Storage and Retrieval of Traumatic Memories

With long-term storage, memories are shifted from hippocampus to neocortical areas, where the sensory impressions take place (Kim & Fanselow, 1992; Phillips & LeDoux, 1992). This shift in the process of memory storage to the cortex may represent a shift from conscious representational memory (explicit memory) to unconscious memory processes (episodic and implicit memory) that indirectly affect

behavior (Wallenstein, Eichenbaum, & Hasselmo, 1998). The cognitive neuroscience perspective (see Brewin, 2001) favors a dual representational model of traumatic memories that proposes separate memory systems underlying vivid reexperiencing versus ordinary autobiographical memories of trauma. These two can be separated in hippocampally-dependent and non-hippocampally-dependent forms of memory, and are differentially affected by extreme stress. Within this system, the strength of traumatic memories relates, in part, to the degree to which certain neuromodulatory systems, particularly catecholamines and glucocorticoids, are activated by the traumatic experience (Cahill, 1997; Hasselmo, 1995). Both the quantity of release of these stress hormones, and the functional availability of the target brain areas (e.g. hippocampus) modulate the encoding of memories of the stressful event; ineffectiveness of the system may be responsible for breakdown in the stream of events and changes in the central and peripheral processing of the events. This can lead to the wide spectrum of memory symptoms, ranging from hypermnesia, amnesia, deficits in declarative memory, delayed recall of abuse, and other memory alterations or distortions in trauma disorder patients.

It should be kept in mind that traumatic memories are not fixed or indelible, but can change over time. Enhanced memory for the gist of emotional events seems to be a dominant theme. What is encoded depends on what was perceived, and what is encoded determines what will be retrieved (Tulving & Thomson, 1973). Neuroimaging may shed a light on the retrieval aspect of memory and its emotional involvement by investigating brain processes that are occurring during traumatic recall (Baddeley et al., 2000; Bremner, Krystal, Charney, & Southwick, 1996; Sara, 2000; Zola, 1998). In PTSD patients '*traumatic cues*', such as a particular sight or sound reminiscent of the original traumatic event, typically can induce a cascade of anxiety and fear-related symptoms, sometimes without conscious recall of the original traumatic event. This traumatic stimulus may not always be easy to identify; it may be that through exposure to a movie, a smell, or more subtle, a gesture or voice, a memory is metaphorically '*reawakened*' – traumatic memories can remain indelible for years or decades and can be recalled by a variety of stimuli and stressors. A model of extinction to explain this does not seem to qualify in these cases; a better model seems to be the failure of successful inhibition of traumatic memories.

Traumatic recall may not always be processed in an integrated mode of consciousness. This may be a discontinuous experience with amnesic gaps. Zimbardo, LaBerge, and Butler (1993) compared the emotional, cognitive, and physiological responses of subjects experiencing induced physiological arousal with and without awareness of the source of their

arousal. When subjects received posthypnotic suggestions for arousal (increases in heart and respiration rate) with and without amnesia for its source only hypnotizable subjects were expected to differ between conditions. Indeed, for the hypnotizable subjects, unexplained arousal produced significant and dramatic effects when compared with explained arousal, including misattributions (Zimbardo et al., 1993). These experiments demonstrated that '*discontinuous experiences*' can contribute to the development of psychopathological symptoms in normal persons. But recall can also be hypnotically blocked, e.g. by posthypnotic suggestion. Here a disruption of retrieval like in post-hypnotic amnesia or posthypnotic suggestion refers to a subjects difficulty in remembering, after hypnosis. This is not a state-dependent memory, but it does involve a disruption of retrieval processes similar to the functional amnesias observed in clinical dissociative disorders. In a situation like this implicit memory, however, is largely spared, and may underlie subjects' ability to recognize events that they cannot recall (Kihlstrom, 1997).

Induction Procedures of Traumatic Recall

Recall of traumatic events in imaging studies is usually embedded in a so-called ''activation paradigm'' of re-experiencing traumatic events. In this paradigm, the patient is asked to briefly (for 1 to 2 minutes) recall a memory that is induced by a personal narrative, smell, picture, or sound with different traumatic load (traumatic vs. neutral). For the purpose of this paper, we focus on the recall induced through emotional or cognitive induction. For the purpose of this paper, we focus on the recall induced through emotional or cognitive induction.

Traumatic Recall Through Emotional and Cognitive Induction

Typically in a traumatic-script procedure, the patient writes a narrative of his or her two most traumatic personal events some days before the scanning. Usually two neutral texts are made at that time for the no-activation scan. This text is edited for length (30–40 seconds) and content. The script is audio taped or can be read during the scan procedure. The script can then be presented in first or second person, usually present tense. Immediately before each scan the participant is instructed to ''close your eyes, listen carefully to the audiotape or voice and imagine the described events as vividly as possible, as if you were actually participating in the event again'' (cf. Lanius et al., 2001; Osuch et al., 2001; Shin et al., 2000). The participant is then usually scanned 6 to 12 times with a 10-minute interval between scans. When the patient is lying in the scanner, and the radioactive ligand is administered intravenously, a trauma script (prepared by a participating patient)

similar to the one below (B. Elzinga, personal communication, July 2000) can be read:

> Listen carefully to the script, and try to imagine as vividly as possible the experience:
>
> *My mom is taking a shower. Dave comes up to me in the living room, where I am standing. He is whispering in my ear, ''I would prefer to kiss your private part.'' I think he is saying that as he presses my breast. Soon his hands sweep down to my private area and he begins to massage it. His touch is not welcoming; his pressing my breast hurt me and so does his touching my private area. I am confused and afraid. Mom can come out of the bathroom any minute. I want to tell him ''stop,'' but I don't. It seems as if I can't find my voice. Eventually, I make gestures that imply I don't want any more touching. He eventually stops, after calling my name a couple of times. I am relieved, and I seek some quiet corner of the apartment, just as my mom comes out of the shower.*
>
> Now, continue to imagine the experience from the beginning to the end, until I ask you to stop.

When applying the model of induction of emotional memories in a trauma population, some points need to be considered:

(1) A prerequisite for successful implementation of a recall paradigm and completion of the task in neuroimaging research is the ability of the participating subjects to have reasonable control over their emotional response in recalling traumatic events. In a PET paradigm, they need to be able to return to a normal state within approximately 10 minutes. Subjects may – even though they are informed and have given informed consent – become tearful, panicked, and emotionally overwhelmed during the recall and feel an urge to suppress these responses. Sometimes this fails and leads to termination of the scan (Osuch et al., 2001).

(2) Extreme stress, high or low arousal, and fatigue are distinct psychological factors that can separately and interactively affect how information is processed – rendering it especially influential because it is not submitted to critical reality testing in a calm, relaxed, and rested state. This is what Bowers described as a situation in which type II unconscious influences occur. These describe how information is processed outside normal awareness, initiative, and volition, speaking of *dissociated experience* and *dissociated control* as two complementary aspects of hypnotic responsiveness (Bowers, 1973). Low-level monitoring of the process when exposed to traumatic slides and sounds and calling this to a halt will typically occur in the trauma-control subject; the situation is different in patients with PTSD. Their dissociated experience refers to the fact that the (induced) state of affairs seems to occur nonvolitionally, – which means here that the effort involved is not well presented in conscious experience. These observations contribute to a framework in which brain correlates of traumatic recall can be understood as dissociated control. Upon asking subjects to voluntarily start recalling

a situation (''Now continue to imagine the experience from the beginning''), some will anticipate becoming stressed and voluntarily control the situation, and some will become upset and may not be able to stop recalling (involuntary response).

(3) An important aspect in recall inductions is the content of intrusions. Research studies suggest that they are not random fragments of the experience. Typically, they represent stimuli that were present shortly before the moments with the largest emotional impact (Ehlers et al., 2002). They need not be sensory per se. Reynolds and Brewin described elaborations of the original experience as the most intrusive, linked to preoccupations with appraisals of the trauma and its sequelae, rather than presenting trauma memories (Reynolds & Brewin, 1998). This needs to be taken into account when preparing a narrative script.

(4) Of importance in the induction of traumatic recall for brain imaging studies is the theme of general versus specific induction of trauma-related memories. Typically, in a general paradigm a standardized set of images or words is presented, and the response pattern in the target population can be calculated by averaging the responses. In a trauma-specific paradigm, an individual induction is prepared before the brain imaging procedure. In this paradigm, the surprise effect of the inductions is somewhat diminished since the subject will recognize his or her specific elements. Ehlers provides examples of the specific (sensory) nature of the traumatic events from which it appears that traumatic triggers are specific for both nature and content of the trauma-related stimulus. In designing an experiment using olfaction as a trauma-related cue in combat-related PTSD, we were to choose a traumatic smell that could either be specific for each person or a smell that all veterans reported as a trigger for traumatic memories. All veterans had been exposed to diesel during their combat experience, and diesel was present throughout the war. This smell therefore seemed to qualify as both a generic and specific trauma-related smell in the population (Vermetten, Schmahl, Southwick, & Bremner, 2003). The same can be applied to trauma-related words and other types of sensory stimulation.

(5) Laboratory studies have demonstrated that central cues of a traumatic event are usually well remembered, whereas memory for peripheral details is poor (Christianson, 1992). The *narrowing of attention* is often used as an explanation for this finding. High anxiety and arousal are thought to focus the attention on central aspects, such as the weapon used, and hinder a full processing of the situation. It is thought that changes in the perfusion of limbic brain structures that coincide with the high arousal and/or anxiety, such as the amygdala and the hippocampus, can lead to fragmented memories and personality fragmentation (Spiegel, 1989; Van Der Kolk, Burbridge, & Suzuki, 1997). Narratives should be written according to these notions.

(6) In all imaging studies in traumatic recall, the patient anticipates the presentation of trauma (-related) material, and some researchers have performed a dry run with the patient. Then the subject is not ''cold'' to

the trauma cue. It needs to be taken into account that this may dampen the activation of the brain when exposed to the challenge.

(7) Last, in addition to the first observation of this section, many clinicians have described a ''dissociative'' or ''hypnotic'' blocking of perceptual aspects as an adaptive response to trauma. Pain in recall can be blocked, time processing can be distorted, or processing of the perception of emotions like threat cannot be adequately processed. Patients may dissociate during the experience and unless this is assessed at each between-scan interval (to assess whether this is a positive or negative phenomenon, see Lanius et al., 2002; Nijenhuis et al., 2002) it may explain a difference in participant responding. In case patients do dissociate, a systematic procedure needs to be administered to help reorient them to the common environment and enable them to continue with the scanning procedure reliably. In PET protocols, this is especially important since the production of radioactive material is delivered in a time-wise manner, and typically each interscan interval is set to 10 minutes.

Functional Brain Imaging Results in Traumatic Recall in Trauma Disorders

To date, 12 imaging studies that used a symptom provocation paradigm in PTSD have been published. Seven studies used PET (Bremner, Narayan, et al., 1999; Bremner, Staib, et al., 1999; Osuch et al., 2001; Pissiota et al., 2002; Rauch et al., 1996; Shin et al., 1997, 1999), three used fMRI (Lanius et al., 2001, 2002; Rauch et al., 2000), and two used SPECT as imaging technique (Liberzon et al., 1999; Zubieta et al., 1999). The design, patient population, induction method, measure of recall, psychophysiological coregistration, and changes in brain metabolism are tabulated in Table 1. These studies have used various challenge models, exposing the subject – at varying levels of complexity – to perceptual stimulations that range from exposing patients to slides and sounds, smells of trauma-related experiences, to reading narrative scripts, to the administration of pharmacologic agents like yohimbine (see reviews by Bremner, 2002; Hull, 2002). Reexperiencing of traumatic events typically coincides with heightened attention, lack of awareness for the surroundings, and loss of perception of time. At the same time, emotions of fear, shame, disgust, anger, and sadness, may occur and sometimes coincide with dissociation, freezing, and other psychophysiological arousal phenomena (Nijenhuis et al., 1998).

The first PET studies in traumatic recall used combat slides and sounds and script-driven imagery in PTSD patients. The results suggested that symptoms associated with traumatic recall were mediated by the limbic and paralimbic systems within the right hemisphere. Activation of visual cortex corresponded to the visual component of PTSD reexperiencing phenomena (Rauch et al., 1996). When generating

Table 1
Overview of Designs of Neuroimaging Assessments in PTSD Studies – Up to 2002

Study	Imaging Method	Population				Trauma Type	Activation	No Activation	Scan Data Acq	Add'l Data Acq	INC PTSD-TC or ACTIV-NO ACTIV	DEC PTSD-TC or ACTIV-NO ACTIV
Rauch et al. (1996)	$[^{15}O]H_2O$ PET	males	$N = 8$	PTSD	–	combat	listening to trauma script	neutral script	during exposure	HR, SUDS	ri limbic, paralimbic and visual areas	le inferior frontal and middle temporal cortex
Shin et al. (1997)	$[^{15}O]H_2O$ PET	males	$N = 14$ (7/7)	PTSD	TC	combat	watching combat related and negative pictures	neutral pictures	generating visual mental images	–	ventral anterior cingulate gyrus, ri amygdala	Broca
Bremner et al. (1999)	$[^{15}O]H_2O$ PET	males	$N = 20$ (10/10)	PTSD	TC	combat	watching combat related slides and sounds	neutral slides, nonverbal music	during exposure	HR, SUDS, PTSD symptom scale, PASS, CADDS, VAS fear	cerebellum, ri inf front gyrus, midbrain	prefront cortex (25), le ant cingulate, le thalamus, le vis assoc cortex, sup temp lobe, le mid temp cortex
Bremner et al. (1999)	$[^{15}O]H_2O$ PET	females	$N = 22$ (11/11)	PTSD	TC	childhood sexual abuse	listening to personalized script of trauma	neutral script	during listening	SUDS, PTSD symptom scale, CADDS, fear VAS	ant prefront cort (6, 9); post cingulate (31), motor cortex, (alt in med prefront cortex)	subcall gyrus (25); ant cingulate (32); ri hipp; inf temp gyrus, sup.marg. gyrus, vis assoc cortex

(*continued*)

Table 1
(continued)

Study	Imaging Method	Population				Trauma Type	Activation	No Activation	Scan Data Acq	Add'l Data Acq	INC PTSD-TC or ACTIV-NO ACTIV	DEC PTSD-TC or ACTIV-NO ACTIV
Shin et al. (1999)	[^{15}O]H_2O PET	females	$N=16$ (8/8)	PTSD	TC	childhood sexual abuse	listening to traumatic script	neutral script	during recall and imagination	BDI, STAIS, VVIQ, HR, BP, emot state	orbitofrontal cortex, anterior temporal lbes	insular cortex, ant cingulate gyrus, ant front regions, le inf front gyrus
Liberzon et al. (1999)	[^{99m}Tc] HMPAO SPECT	males	$N=39$ (14/11/14)	PTSD	TC/HC	combat	listening to combat sounds	white noise	60 min after tracer injection	HR, GSR, SUDS	ant cingulate/mid prefr gyrus; amygdala/acc,	le retrosplenial region
Zubieta et al. (1999)	[^{99m}Tc] HMPAO SPECT	males	$N=34$ (12/11/12)	PTSD	TC/HC	combat	listening to combat sounds	white noise	during exposure	GSR, SUDS, EMG, HR	med prefr cortex, cingulate	–
Rauch et al. (2000)	fMRI	males	$N=16$ (8/8)	PTSD	TC	combat	watching masked fearful faces	masked happy faces	during viewing	Subj report	amygdala	–
Lanius et al. (2001)	fMRI	males, females	$N=18$ (9/9)	PTSD	TC	sexual abuse/ assault, MVA	listening to traumatic script	baseline	during reading 30 sec	HR	–	ri/le thalamus, ri/le med front gyrus (10/11), ri/le ant cing qyrus (32), ri occ lobe (19)

Osuch et al. (2001)	PET	females, males	$N = 11$	PTSD	no control	mixed	listening to traumatic script plus flashback experience	no flashback	during listening	HR, SUDS	le inf frontal, le hipp, le ant insula, ri insula, ri putamen, le somatosensory, le cerebellum, lingula, brainstem	Bilat dorsolat prefrontal, ri fusiform and ri med temporal cortices
Lanius et al. (2002)	fMRI	females, one male TC	$N = 17$ (7/10)	PTSD (dissociative responses)	TC	sexual, physical and emotional abuse	listening to traumatic script	baseline	during reading 30 sec	HR, CADSS	ri sup/mid temp gyri (38), inf frontal gyrus (47), occ lobe (19), ri parietal lobe (7), ri med frontal gyrus (10), ri med prefr cortex (9), ri ant cingulate (24, 32)	–
Pissiota et al. (2002)	$[^{15}O]H_2O$ PET	males	$N = 8$	PTSD	no control	combat	listening to sounds of combat	simple tones	during exposure	HR, STAIS, SUDS, panic VAS	ri sensorimotor cortex (4, 6), primary sensorimotor cortex (1, 2, 3), cerebellar vermis, PAG, ri amygdala	ri retrosplenal cortex (26, 29, 30)

Note. TC = trauma controls, HC = healthy controls, HR = heart rate, GSR = Galvanic Skin Response, SUDS = Subjective Units of Distress, PAG = periaquaductal gray, ri = right, le = left, act = n accumbens, VAS = visual analog scale, CADSS = Clinician Administrated Dissociative Symptom Scale, STAIS = State-Trait Anxiety Inventory Trait Test, MVA = Motor Vehicle Accident, VVIQ = vividness visual imagery questionnaire.

mental images of combat-related pictures, increased regional cerebral blood flow (rCBF) in the ventral anterior cingulate cortex (ACC) and right amygdala was seen; when viewing combat pictures, subjects with PTSD showed decreased rCBF in Broca's area (Shin et al., 1997). These first PET studies of traumatic recall in PTSD have since led to a rapid increase in similar studies modifying the experimental condition and/or study population.

There is overlap but also considerable diversity in various traumatic recall studies. The ACC, middle and superior temporal, middle frontal, right orbitofrontal, occipital, hippocampal, parahippocampal, anterior temporal, and inferior frontal cortices have all been implicated in different studies, demonstrating either increases or decreases in perfusion depending on the study conditions and sample population (Phillips et al., 2003a, 2003b). In general, in comparison to trauma-control subjects, these studies reveal an exaggerated response activation in the right (Rauch et al., 1996; Shin et al., 1997) or left (Liberzon et al., 1999) amygdala, and in the sensorimotor cortex (Bremner, Narayan, et al., 1999; Shin et al., 1997) and attenuated responses within the medial prefrontal cortex (mPFC) (Bremner, Narayan, et al., 1999; Shin et al., 1999) in patients with PTSD. In line with this, imaging studies of normal autobiographical memory (i.e., no emotional activation) in healthy subjects compared to memory-control tasks have pointed to mPFC and (left) hippocampus that are just particularly responsive to such memories (Conway et al., 1999); other studies point to right frontal cortices, medial parietal cortex, and cerebellum (Nyberg, Forkstam, Petersson, Cabeza, & Ingvar, 2002).

Current studies support a model of PTSD in which (a) the amygdala is hyperresponsive to threat-related stimuli, and (b) interconnected areas may provide insufficient ''top-down'' inhibition by mPFC and ACC of amygdala response. This relative dysfunction of mPFC and ACC is thought to lower the threshold of amygdala response to fearful stimuli and is central to symptom mediation (Pitman, Shin, & Rauch, 2001; Villarreal & King, 2001). Thus, dysfunction of the mPFC areas may provide a neural correlate of a failure of extinction of fearful stimuli in PTSD.

Recall induction of emotion specifically activated the ACC. This brain structure is critically involved in cognitive induction of emotional responses and processes attention, executive functions, and semantic and episodic memory. ACC activation represents a normal brain response to traumatic stimuli that serves to inhibit feelings of fearfulness when there is no true threat. Failure of activation in this area and/or decreased blood flow in the adjacent subcallosal gyrus (area 25) may lead to increased fearfulness that is not appropriate for the context, facilitating exaggerated emotional and behavioral responses (hyperarousal) to conditioned stimuli (Hamner, Lorberbaum,

& George, 1999). Posterior cingulate cortex (PCC) and motor cortex and anterolateral prefrontal cortex are also known to modulate emotion and fear responsiveness (Bremner, 2002). PCC plays an important role in visuospatial processing and is therefore an important component in the preparation for coping with a physical threat. PCC also has functional connections with the hippocampus and adjacent cortex.

In a meta-analysis of PET and fMRI studies of general emotional activation reviewing 43 PET and 12 fMRI activation studies spanning almost a decade of research, Phan, Wager, Taylor, and Liberzon (2002) describe brain areas that are involved in emotion induction with cognitive demand, typical paradigms of the recall of autobiographical elements or visual imagery:

• mPFC	*general role in emotional processing*
• amygdala	*specific role in fear processing*
• mPFC/subcallosal gyrus (area 25)	*involved in the processing of sadness*
• occipital cortex (OC) and amygdala	*activation by emotional induction by visual stimuli*
• ACC (area 32) and insula	*induction of emotional recall/imagery and induction of emotional tasks with cognitive demand*

There are inconsistencies across studies that may be attributable to methodological differences. Imaging studies inducing memories of traumatic reminders and/or PTSD symptoms with scripts and combat-related slides and/or sounds have not all consistently resulted in increased amygdala activation in PTSD (Rauch et al., 1996). In order for increased amygdala function to be observed, it is necessary to utilize the appropriate task. Because most of these studies involved enteroceptive or internally generated emotional states, they may not actually correspond to classic states of fear. An explanation may be that induction of traumatic memories through techniques such as traumatic scripts represent a different condition than the acquisition of conditioned fear responses, as is seen in the pairing of a US (shock) with a CS (bright light) (Liberzon et al., 1999; Rauch et al., 2000; Semple et al., 2000).

There are a number of emotional states that characterize PTSD in addition to exaggerated fear responses to threat. As reviewed earlier, these include symptoms of dissociation, loss of self-agency, feeling worse with traumatic reminders, amnesia, and flashbacks upon visual imagery of the traumatic event that plays back like a movie. These states can be very subtle but can be discerned when the patient reflects on his or her emotional response after the traumatic recall; just compare the following statements: ''*I was back there, saw through my own eyes. And felt how it felt at that moment*,'' versus ''*I observed myself, I was afraid to go*

into the experience too deep, and I held back a bit how it felt to really have been there'' or ''*Every time I go there, I space out, it is as if my mind becomes black. I don't feel anything until [he] leaves the room,*'' or ''*This time was different from a lot of other times I recalled the experience. I felt more pity for the man and despite my fear, could relax more during the whole experience,*'' or ''*I felt distracted and could not focus on the situation; my legs hurt but I could not move them*.''

These subjective reports may have implications for activation of brain areas, mPFC, amydala, hippocampus, and ACC. All patients may have reported that they were fearful in the same range after the traumatic recall. In the study of Osuch et al. (2001), regional cerebral perfusion correlated directly with flashback intensity in the visual association areas, which confirmed the hypothesized activation of these regions but not the hypothesized involvement of the amygdala. In this study, rCBF also correlated directly with left hippocampal/perihippocampal areas, inferior frontal, left anterior insula, left somatosensory and left cerebellar cortices, the brainstem, right putamen, and right insula. Inverse correlations with flashback intensity were prominent in the right and left superior frontal, right fusiform, and right medial temporal cortices. These findings are consistent with the studies that demonstrated superior frontal rCBF decreases with symptom evocation (Liberzon et al., 1999; Rauch et al., 1996, 2000; Shin et al., 1999) and hippocampal or parahippocampal involvement with PTSD (Bremner, Narayan, et al., 1999; Rauch et al., 1996; Southwick, Morgan, Charney, & High, 1999), but not with those studies implicating the anterior cingulate, superior and anterior temporal lobe, or amygdala.

In their review study, Phan et al. (2002) discuss how different ways of recall lead to different areas of brain activation. The recall of memories or imagery of personally relevant, affectively laden events required explicit and intensive cognitive effort. The recollection or recall-induction method specifically activated the anterior cingulate: 50% of studies report ACC activation as compared to 31% and 0%, respectively, of visual and auditory induction studies. ACC appears to have cognitive functions including the modulation of attention and executive functions, and interconnects with subcortical limbic structures. This is not surprising as subjects are instructed to recall or imagine an emotionally laden personal event and then self-induce or internally generate intense target emotions (Teasdale et al., 1999). This is in line with the observation of Lanius when conducting recall studies that some patients demonstrate spontaneous out of body experiences with ''dissociative'' lowering of heart rate (R. Lanius, personal communication, September 2003). In a study with abuse-related PTSD patients, approximately 70% of patients relived their traumatic experience and showed an increase in heart rate while recalling the traumatic memory.

The other 30% of patients had a dissociative response with no concomitant increase in heart rate. PTSD patients in a dissociative state showed more activation in the superior and middle temporal gyri (BA 38), the inferior frontal gyrus (BA 47), the occipital lobe (BA 19), the parietal lobe (BA 7), the medial frontal gyrus (BA 10), the medial cortex (BA 9), and the ACC (BA 24 and 32).

Despite a variety in rCBF in application of the traumatic recall paradigm, we now can describe a model of the neural circuitry in traumatic recall. In this model, emotional involvement and memory dysfunction implicate limbic brain regions, including the amygdala, hippocampal formation, and limbic cortex, such as the orbitofrontal and anterior cingulate areas. Additional key brain structures are thalamus (relaying incoming perceptual input), mPFC (planning execution, working memory, attention), and ACC/PCC (attention, affect, and affective control) (see Figure 2).

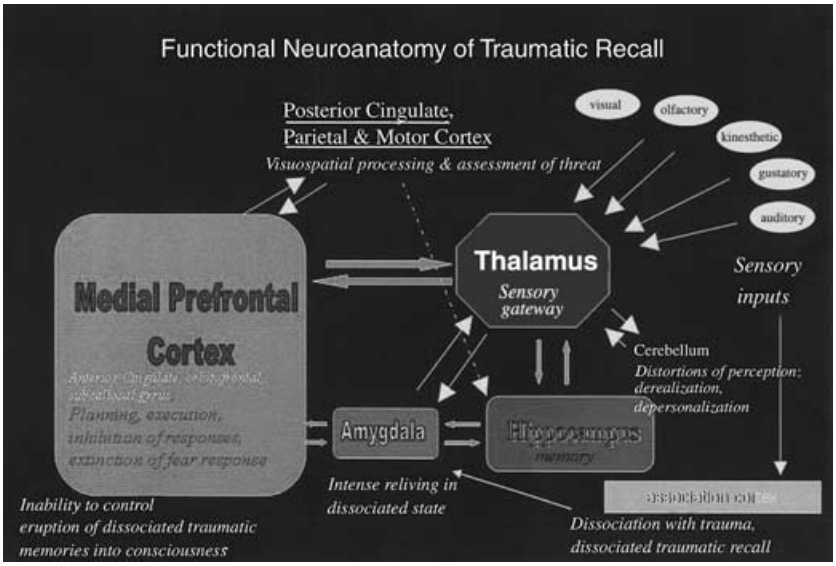

Figure 2. **Schematic working model of the functional neuroanatomy of traumatic recall. Key players are the thalamus (sensory gateway); the hippocampus, which is critically involved in learning and memory; the amygdala, which contributes to information adding emotional significance. The medial PFC (OFC) together with the ACC is involved in the planning and execution, inhibition of responses and in the extinction of fear responsivity. The model is also intended to serve as a working model for hypnotic responsiveness, perhaps with a key role for the ACC. Failure of inhibition by the mPFC, in conjunction with alterations in ACC and amygdale processing is considered the pivotal element in traumatic recall. (Adapted and modified from van der Kolk, 1996).**

HYPNOSIS AND BRAIN IMAGING: QUESTIONS

In the last 5 years, nearly 20 imaging studies using hypnosis as a parameter have been performed. These studies were aimed at the study of, for example, processes of regulation of consciousness (Rainville, Hofbauer, Bushnell, Duncan, & Price, 2002), imagination of exercise (Thornton et al., 2001; Williamson et al., 2002), pain (Faymonville et al., 2000; Hofbauer, Rainville, Duncan, & Bushnell, 2001; Laurent, Peyron, Garcia Larrea, & Mauguiere, 2000; Rainville et al., 1997; Rosén, Willoch, Bartenstein, Berner, & Røsjø, 2001; Wik, Fischer, Bragee, Finer, & Fredrikson, 1999; Willoch et al., 2000), paralysis (Halligan, Athwal, Oakley, & Frackowiak, 2000), color perception (Kosslyn et al., 2000), and auditory processing (Szechtman et al., 1998). This has provided a great deal of insight in regulatory principles. ''The imaginary is real,'' ''seeing is believing'' and ''the strain in pain lies in the brain'' are statements derived from some of these studies. Hypnosis is not role playing, but it may involve a reinterpretation of the perceptual experience, influence the verbal mediation of suggestions, and integrate input with contextual information, memory, and affect.

Results are not uniform and depend on the paradigm used: In a study by Maquet et al. (1999), the hypnotic state (revivication of pleasant autobiographic memories vs. instructed color hallucination) is related to the activation of a widespread, mainly left-sided, set of cortical areas, without a specific brain region involved in the state. Brain activity associated with hypnosis in high versus low hypnotizables (after mental relaxation) is characterized by a decrease in cortical arousal and a reduction in cross-modality suppression (disinhibition). Rather than providing a unique brain ''hypnotic state,'' they provide a framework for understanding the neurobiology of consciousness and the modulatory effect of perceptual and emotional events by hypnosis.

No studies have yet been performed looking at the effects of hypnotizability or of a trance state on (traumatic) recall in brain blood flow. Does the subject have affective control in an induced hypnotic state? A role for the ACC may well be the case, analogous to the result in the Rainville et al. study (1997) in pain processing. Several questions remain unanswered, e.g., does it occur automatically? What is the difference in highs versus lows? This may have therapeutic implications if it shows that high hypnotizable subjects can ''learn'' to control affective brain states. Hypnosis may then be understood to help engage in self-regulatory mechanisms that originally (at the time of trauma) evolved to support orientation to the episodic environmental context, which later was integrated within attachment and imprinting instincts (the hippocampus and ACC/PCC). At the same time, hypnosis can be understood

to modulate a suspension of other self-regulatory mechanisms that normally serve to disengage the person from the episodic context (of the amygdala and its input into the ACC). This suspension could well be found when subjects comply with suggestions without genuinely experiencing their responses as nonvolitional (Zamansky & Ruehle, 1995).

Discussion and Summary

When reviewing these neuroimaging studies, several types can be distinguished: (a) studies that basically investigate phenomena, e.g., the processing of phenomena like fear, attention, pain, consciousness (*fear-no fear*) (Paradiso et al., 1997); (b) studies that are aimed to experimentally modify emotional responses within subjects (*alert-hypnosis*) (Rainville et al., 1999); (c) studies that compare different spontaneous responses to traumatic reminders (*dissociators-nondissociators*) (Lanius et al., 2002); (d) studies that take the results of one to compare across different cohorts or populations (*patient-control*) (e.g. Bremner, Narayan, et al., 1999); (e) studies that longitudinally follow up on conditions within subjects or groups (*Week 1–Week 6*) (Fernandez et al., 2001). Several studies overlap in this matter, e.g., modification of emotional responsivity can be investigated with pharmacological stimulation but can also be accomplished by task-driven induction of emotional states. These task-driven inductions can be administered in a variety of designs, varying from a design in which the subject is alert and conscious, to one in which the subject is invited to engage him or herself in an altered state of consciousness, e.g., hypnosis. This is of particular interest for the purpose of this paper that focuses on the interplay between neuroimaging, behavioral states, and psychopathology.

Affected brain regions and the direction of change are not always the same across studies. There is some agreement in the findings of these functional imaging studies of trauma disorders despite differences in experimental variables: (a) study design, (b) imaging techniques, (c) experimental variables, and (d) study populations. The study design and imaging techniques have been described earlier in this paper. Different experimental variables including (a) type of eliciting stimulus, (b) order of neutral and symptom evocation conditions, (c) type of traumatic event to which subjects were initially exposed, (d) length of time since the traumatic event, and (e) whether or not physiologic nonresponders to traumatic stimuli were included in the sample population. These all need to be carefully looked at (Osuch et al., 2001). Optimal diagnostic criteria may be able to define a subset of patients with PTSD who are pathophysiologically homogeneous. Many neuroimaging studies of PTSD have focused on increasing the homogeneity of their subject population. For example, some studies

have selected for physiologic responders those who have both a subjective and physiologic response to traumatic reminders and are likely to experience different patterns of reaction in the scanner over time. When nonphysiologic responders are included, it becomes even more apparent that there can be highly varied subjective responses and autonomic dissociations to a single auditory script paradigm.

The role of hypnosis in studying traumatic recall is a caveat and at the same time a promise for imaging studies in patients with trauma-related disorders. Traumatized individuals with trauma-related psychopathology like PTSD or other trauma-related disorders can alternate between states of consciousness in which they experience their trauma over and over again as if it were happening on the spot, with the same vividness and psychophysiologic changes, and episodes in which they are apparently unaware of it or on first sight seem relatively undisturbed. This is a population that has been shown to be on average highly hypnotizable and can use their hypnotic capacity to block pain, alter time perception, or modify their affective response in an experimental situation of traumatic recall. Usually in brain imaging studies, a careful description of the conditions to which an individual is exposed is omitted (Phan et al., 2002). Individual patient variables may add to the brain metabolism as well as by a description of their psychopathology. These patients may be anxious at baseline, and some may be easily overwhelmed, whereas others stay in control throughout the procedure. One patient may perceive intrusive memories, whereas another is able to successfully block these memories while perceiving them. These responses may be involuntary, and, as the patient may not even be aware of them, they may add to the variability of the data acquired. In addition, from a subject's perspective, a he or she usually does not want to fail and disappoint the experimenter and so may not disclose his or her uneasiness at baseline for fear of being thrown out of the study and losing the reimbursement. The subject may also be intimidated by the experimenter (who never before appeared in a white coat).

Implications for Future Research Studies in Traumatic Recall

The past decade has seen an exciting expansion of research in the field of fear and anxiety, stress and memory. These studies are beginning to map out a neural circuitry of PTSD. Future research will need to continue to apply findings from this ''revolution'' in neuroscience to further understanding of emotional processing, anxiety, and intrusive memories. This is a challenge not only for patients but also for researchers; rather than experimentally induce trauma in the lab, there is an opportunity to overcome the disparity in laboratory analogues of

traumatic reminders and those that are based on traumatic events that have been present in real life (Bower & Sivers, 1998).

The effect of individual differences in brain activation now would need to be examined by parametric or factorial design or by personality and temperament measures (Howe, 1998), or psychophysiological assessment (Phan et al., 2002), e.g., investigation of flashback intensity and rCBF in subjects who have an autonomic response needs to be compared with those who do not so as to separate the neural substrates of autonomic aspects of flashbacks from the more nonautonomic, internal experience (Osuch et al., 2001) or the relation of peripheral psychophysiological changes in different emotional states (Shin et al., 1999). Specific activation could be further isolated through network analyses or event-related designs. It may be insightful to move beyond single time points to temporal dynamic patterns, enabling insight in the ''chain of events,'' enabling a description of the patterns of activation, and focusing on how one can use patterns of activation.

Detailed research into human brain function may further contribute to our understanding of the central mechanisms of attention, memory, arousal, anxiety, aversion, and, in general, hypnosis (Chen, 2001). Research in the core symptoms of human consciousness may also help to understand the core problem of human consciousness. Brain measures of traumatic recall can also help to model the body-brain, brain-mind, and mind-matter duality in the triad physics (stimulus), physiology (brain activity), and psyche (perception) (Chen, 2001). Understanding of the human mind may occur when delineating the arousal-attentions system, the emotion-motivation and perception-cognition neural networks of emotional processing. As Kosslyn nicely put it, ''When asking the right questions, given the fine interplay between state-dependent processes, behavior, and psychopathology, neuroimaging will be able to provide the answers'' (p. 1289, 1999). Therefore, we are also in favor of studies that ask for uniform, standard stimuli and design activation paradigms with careful and exact isolation of the emotional process of interest; and, in addition to state, consider assessing trait variables to rule out differences in, for example, hypnotic capacity, as well as trait and state dissociation, before and during scanning intervals (Howe, 1998; Phan et al., 2002). These parameters will enhance uniformity of the results of brain imaging studies in the traumatic recall paradigm and will further improve our knowledge of relationship between hypnotizability and traumatic recall.

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APPENDIX: PRINCIPLES OF FUNCTIONAL BRAIN IMAGING

The major neuroimaging techniques used in neuroscience research are Positron Emission Tomography (PET), Single Photon Emission Computed Tomography (SPECT), and functional Magnetic Resonance Imaging (fMRI), along with electro-encephalography (EEG), an earlier technique for monitoring brain activity. These are all noninvasive procedures that can measure biological activity and reveal the human brain at work. Each technique has its own advantages and each provides different information about brain structure and function. Advances in all these techniques have enabled us to produce detailed computer-screen images of brain structures, and observe neurochemical changes that occur in the brain as it processes information or responds to various stimuli, such as memory recollections of traumatic events.

Positron Emission Tomography (PET)

The development of PET permits a relatively direct way to investigate changes in cerebral activity patterns as a function of specific task performances. The basic idea behind the application of PET is that a short-lived radioactive tracer (e.g., $[^{15}O]H_2O$) is used to measure rCBF during a 60-second period (half life of radioactive water is about 2 minutes). Other tracers that are used are radio-labeled glucose, $[^{18}F]$2-fluoro-2-deoxyglucose, or FDG. SPECT is used to measure brain blood flow with technetium, $[^{99m}Tc]$HMPAO. The radiotracer is injected into the bloodstream, which carries it to the brain. A scan, which represents a three-dimensional map of the CBF distribution in the entire brain during that time, is then reconstructed with a spatial resolution on the order of

5–8 mm, or as much as 2 mm with newer brain-dedicated cameras. Typically, three to four scans with an active task and three to four scans with a control task are obtained. Data from a group of subjects may then be averaged, after appropriate stereotaxic normalization is applied to correct for differences in brain size, shape, and orientation.

In PET, the short scan duration (60–120 seconds) and interscan interval (8–10 minutes) permit multiple studies in rapid succession, allowing comparisons between consecutive functional states, including the resting state. Data interpretation is based on statistical comparisons of rCBF values obtained in two conditions, often labeled the *activation* and *control* conditions. Averaged rCBF data from both conditions may then be compared by superimposition of the relevant scans, and application of a pixel-by-pixel subtraction algorithm that detects significantly different areas of CBF in one condition as compared to another. The assumption is that the different image reflects areas of cerebral activity specifically related to the task in question, relative to the baseline condition (which typically represents an attempt to control for certain aspects of the task), e.g., recall versus no recall. Structural images (MRI) are also obtained for each subject, and are coregistered to the PET images, thus allowing for accurate anatomical localization of the brain activation. An increase in function of neurons in a specific area is reflected by an increase in metabolism and a shunting of blood flow toward the area that can be measured with these imaging techniques (Stern & Silbersweig, 2001).

Functional Magnetic Resonance Imaging (fMRI)

The fMRI process relies on the magnetic properties of blood; it is based on changes in the blood oxygenation level dependent (BOLD) signal, which reflects local CBF changes, and variations in deoxyhemoglobin content simultaneously. Results from fMRI studies/experiments have been found to be strongly correlated with PET-CBF in identical paradigms. Changes in brain activity can be monitored as patients perform various tasks or are exposed to various stimuli. In fMRI image acquisition, a large cylindrical magnet creates a magnetic field around the head of the subject, and radio waves are sent through the magnetic field. When brain protons are placed in this magnetic field, they become capable of receiving and transmitting electromagnetic energy. This information is processed and a computerized image is constructed. With fMRI, both surface and deep brain structures can be imaged with a high degree of anatomical detail, and minute changes that occur over time in these structures can be detected.

fMRI scans can produce images of brain activity as frequently as every second, whereas PET usually takes longer to image brain activity. Thus, with fMRI, it can be determined with greater precision when brain regions become active and how long they remain active. As a result, it can be seen whether brain activity occurs simultaneously or sequentially in different brain regions as a patient responds to experimental conditions. An fMRI scan can produce high-quality images that can pinpoint in detail which areas of the brain are being activated. For example, fMRI can produce an image with a spatial resolution of 1 millimeter or less (Detre & Floyd, 2001).

Methodological Considerations of PET and fMRI

PET has a number of limitations: (a) low temporal resolution due to signal averaging during 1 minute; (b) the need for group analysis pooling the data of at least 5 to 8 subjects to obtain meaningful results; (c) the need for a nearby cyclotron to prepare short-lived radioactive tracers; and (d) the need to give intravenous injections to subjects (Peyron, Laurent, & Garcia-Larrea, 2000). Compared to PET, fMRI provides superior image clarity along with the ability to assess blood flow and brain function in seconds. On the other hand, PET has fewer technical problems related to noise and claustrophobia (due to a larger opening of the PET gantry). In addition, another drawback of fMRI is the requirement of MRI-compatible equipment, restricting the ability to perform simultaneous psychophysiology measurements, as well as creating the need for strict timing between stimuli and acquisition in rapidly alternating conditions. Finally, studies using fMRI are limited by artifact from cavernous structures (e.g., the petrous bone), which can limit the ability to image medial temporal structures such as the amygdala. All of these add technical constraints, making some experiments more difficult to conduct than with PET. More important, fMRI remains limited to activation studies and is not able to provide information on the resting state or, as said earlier, on neurotransmitters or receptors that may represent a shortcoming in future studies in this field (Peyron et al., 2000). To date, however, PET retains the significant advantage of superior sensitivity in the picomolar/nanomolar versus the micromolar range, which allows measurement of neuroreceptors. So far, data obtained with fMRI and PET have been very similar.

Funktionelle Bildgebung und die Induktion traumatischen Wiedererlebens; Ein kreuz-korrelationaler Überblick über Bildgebung und Hypnose

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Zusammenfassung: Die verhaltensbezogenen und psychophysiologischen Änderungen während des Wiedererlebens bei Patienten mit Traumastörungen gleichen häufig Phänomenen, die in Hypnose beobachtet werden. Bei Studien zum emotionalen Wiedererleben wie auch bei bildgebenden Studien hypnotischer Prozesse sind ähnliche Gehirnstrukturen beteiligt: Thalamus, Hippocampus, Amygdala, medialer präfrontaler Cortex, anteriorer cingulärer Cortex. Dieser Artikel konzentriert sich auf Zusammenhänge zwischen traumatischem Wiedererleben und hypnotischen Reaktionen und gibt eine Übersicht über Korrelationen zwischen der Beteiligung von Gehirnstrukturen beim traumatischen Wiedererleben und Prozessen, die auch bei der Ansprechbarkeit auf Hypnose beteiligt sind. Um die Einheitlichkeit der Ergebnisse bildgebender Verfahren speziell für Studien zum traumatischen Wiedererleben zu verbessern, sollte auf die Standardisierung von hypnotischen Variablen, die Isolierung der relevanten emotionalen Prozesse (state) sowie auf die Erhebung von trait-bezogenen Unterschieden geachtet werden.

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Imagerie cérébrale fonctionnelle et induction d'un souvenir traumatique: Une revue de croisée correlationelle entre l'imagerie neurologique et l'hypnose

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Résumé: Les changements comportementaux et psychophysiologiques pendant le rappel chez les patients présentant des troubles de trauma ressemblent souvent aux phénomènes qui sont observés dans l'hypnose. Dans les études de rappel émotionnel comme dans les études de neuro imagerie des processus hypnotiques, des structures semblables de cerveau sont impliquées: thalamus, hippocampe, amygdale, cortex prefrontal médical, cortex antérieur de la cingulaire. Cet article focalise sur des corrélations croisées dans le rappel traumatique et les réponses hypnotiques, et passe en revue des corrélations entre l'implication de structures de cerveau dans le rappel traumatique et les processus mis en jeu dans la réponse hypnotique. Afin d'améliorer plus encore l'uniformité des résultats de l'imagerie cérébrale spécifiquement pour des études de rappel traumatique, une attention particulière est nécessaire pour l'étalonnage des variables hypnotiques, l'isolement de l'état émotionnel (en rappel traumatique) et l'évaluation des différences de trait-connexes.

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Imagenología cerebral y la inducción del recuerdo traumático: Una reseña correlacional entre la neuroimagenología y la hipnosis

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Resumen: Las alteraciones de comportamiento y psicofisiológicas durante el recuerdo en pacientes con desórdenes postraumáticos frecuentemente se asemejan a los fenómenos hipnóticos. Las mismas estructuras cerebrales están implicadas en los estudios de recuerdo emocional así como en los de neuroimagenología de la hipnosis: el tálamo, el hipocampo, la amígadala, la corteza prefrontal media, y la corteza anterior cingulada. Este trabajo se enfoca en las correlaciones entre el recuerdo traumático y los procesos subyacentes a la respuesta hipnótica. Para fomentar la uniformidad en los resultados de la imagenología cerebral, específicamente para el estudio de memorias traumáticas, es necesario estandarizar las variables hipnóticas, aislar el proceso emocional de interés (estado) y evaluar las diferencias en los rasgos de personalidad relevantes.

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