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Chapter 36

Functional (dissociative) retrograde amnesia

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Abstract

Retrograde amnesia is described as condition which can occur after direct brain damage, but which occurs more frequently as a result of a psychiatric illness. In order to understand the amnesic condition, content-based divisions of memory are defined. The measurement of retrograde memory is discussed and the dichotomy between "organic" and "psychogenic" retrograde amnesia is questioned. Briefly, brain damage-related etiologies of retrograde amnesia are mentioned. The major portion of the review is devoted to dissociative amnesia (also named psychogenic or functional amnesia) and to the discussion of an overlap between psychogenic and "brain organic" forms of amnesia. The "inability of access hypothesis" is proposed to account for most of both the organic and psychogenic (dissociative) patients with primarily retrograde amnesia. Questions such as why recovery from retrograde amnesia can occur in retrograde (dissociative) amnesia, and why long-term new learning of episodic-autobiographic episodes is possible, are addressed. It is concluded that research on retrograde amnesia research is still in its infancy, as the neural correlates of memory storage are still unknown. It is argued that the recollection of episodic-autobiographic episodes most likely involves frontotemporal regions of the right hemisphere, a region which appears to be hypometabolic in patients with dissociative amnesia.

INTRODUCTION

The Greek/Latin word *hysteria* refers to suffering in the uterus. It was used to describe women who had excessive emotional reactions of fear or panic without a direct organic basis. Patients with a condition of hysteria usually became unconscious or semiconscious in anxiety-provoking situations and later lacked remembrances related to the circumstances of the hysteric attack – that is, they were retrogradely amnesic (Fig. 36.1).

Jean-Martin Charcot at the Salpêtrière Hospital in Paris studied and described patients with hysteria in the second half of the 19th century (see the review of Bogousslavsky, 2011). The concept became very prominent at the beginning of the 20th century, popularized not only by French authors, but also, for example, by Sigmund Freud, who had studied this condition at the Salpêtrière (e.g., Breuer and Freud, 1895; for reviews see Markowitsch, 1992a; Markowitsch and Staniloiu, in press). During the First World War hysteria was described in soldiers ("war trembler," "*Kriegszitterer*") and consequently lost its connotation as a "female" disease (Peckl, 2007). The name hysteria has nevertheless not only remained prominent in everyday language, but also in scientific communications up to today (e.g., Stone et al., 2006; Bell et al., 2011). It was listed as a disease category only in the early editions of the *Diagnostic and Statistical Manual* (DSM-II: American Psychiatric Association, 1968) and is used in association with (retrograde) amnesia up to today (e.g., Iglesias and Iglesias, 2009; Thomas-Antérion et al., 2010).

More recent versions of the DSM, including the present one (DSM-5: American Psychiatric Association, 2013), replaced the term and introduced (among others) the term "dissociative amnesia" (cf. Spiegel et al., 2011, who also criticize differences between DSM and the

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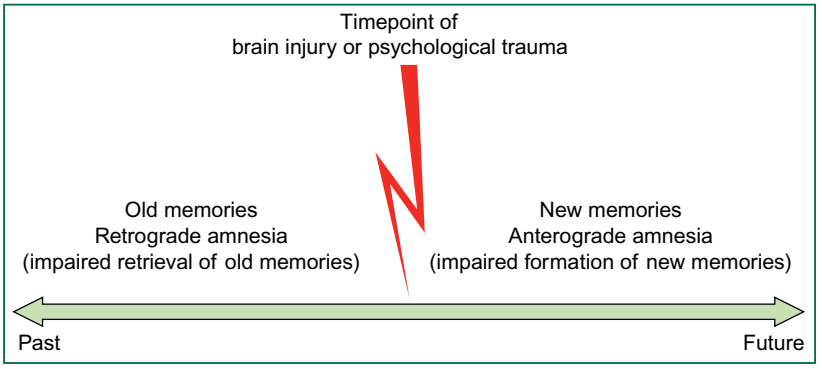

Fig. 36.1. After brain injury or a psychotraumatic event, memory may be impaired with respect to the remembering of old information (which was stored prior to the injury or the event), or with respect to the long-term acquisition of information with which the individual was confronted after the event or injury. The flash symbolizes the event or injury. Already Ribot (1882) noted that there is a gradient in retrograde amnesia: information dating back a long time is better preserved (can be retrieved more easily) than recently acquired information ("Ribot's law" or "law of regression"). (Reproduced from Staniloiu and Markowitsch, 2014.)

International Classification of Diseases, 10th edition (ICD-10: World Health Organization, 1992), and the lack of a proper definition of dissociative disorders). Spiegel et al. definite dissociation on p. 826 as:

> *a disruption of and/or discontinuity in the normal, subjective integration of one or more aspects of psychological functioning, including – but not limited to – memory, identity, consciousness, perception, and motor control. In essence, aspects of psychobiological functioning that should be associated, coordinated, and/or linked are not.*

"Dissociative amnesia" is seen as that subcategory of dissociative disorders in which memory and identity problems are pre-eminent. In a review we defined "dissociative amnesia" as an "inability to consciously recall autobiographical information in the absence of significant brain damage (as detectable by conventional structural neuroimaging)" (Staniloiu and Markowitsch, 2014, Panel 1, p. 2). The term is *a priori* theoretically loaded, since it assumes dissociation to be the primary or only pathogenetic mechanism. To avoid this, or to suggest alternative connotations, a number of additional terms are still widely used (cf. Panel 1 in Staniloiu and Markowitsch, 2014); among them are psychogenic amnesia, which of course emphasizes the psychic nature and origin of the amnesia and distinguishes it from "organic amnesia"; functional amnesia, which suggests that the amnesia serves a function for the affected individual; and mnestic block syndrome, which implies that the amnesia is potentially reversible (Markowitsch, 2002).

As dissociative amnesia can be seen in the tradition of the work of Charcot (1892), Janet (1893), Souques (1892), and other workers (see Markowitsch and Staniloiu, in press) of the 19th century, it can be regarded as more intensely studied than organic amnesic conditions prominent in this epoch (e.g., Korsakoff's disease: Markowitsch, 2010). In spite of its long tradition, dissociative amnesia remains an enigma, as it demonstrates that a primarily psychic condition – (most likely) induced by an adverse environment – can have effects on memory which outweigh those of severe brain injuries.

Before discussing dissociative amnesia in more detail, we will briefly introduce memory divisions as they are relevant for dissociative amnesia (having already defined that dissociative amnesia affects the autobiographic memory domain).

CONTENT-BASED MEMORY SYSTEMS

That memory is not a unity can best be inferred from clinical cases with disorders of memory and was consequently already investigated and described more than a century ago (Ribot, 1882; Markowitsch, 1992a; Markowitsch and Staniloiu, in press). In 1882 Ribot introduced and specified the distinction between anterograde and retrograde amnesia (Fig. 36.1). After some forms of brain damage or psychiatric disease, memory formation or memory retrieval may be impaired. A central question is whether this impairment is permanent, as is the anterograde memory impairment after certain kinds of brain damage (e.g., bilateral medial thalamic or hippocampal damage; cf. Markowitsch, 2008; Markowitsch and Staniloiu, 2012a), or whether it may be better seen as a time-limited blockade of retrieval (Markowitsch, 2002) which can be treated therapeutically.

Retrograde amnesia in principle has to be differentiated from forgetting, which occurs in everyday life situations and is therefore not a pathologic process (Markowitsch and Brand, 2010; Roediger et al., 2010); there is, however, an overlap between nonpathologic and pathologic forms of forgetting (Harris et al., 2010)

and – as Sigmund Freud wrote already in 1901 – we never have a guarantee that our memories are correct (Freud, 1901a). In fact, false memories and memory distortions are more common than generally assumed (Loftus, 2006; Kühnel et al., 2008; Risius et al., 2013), and may play a role especially in forensic situations (Markowitsch and Staniloiu, 2011c).

Though predecessors existed 100 years ago (cf. Markowitsch, 1992a), the content-based division of memory, as used herein, distinguishes between a short-term and five long-term memory systems (Fig. 36.2). Of the five long-term memory systems shown in Figure 36.2, the first two (procedural memory, priming) are considered to be anoetic – that is, they lack or do not require conscious processing; the third and fourth memory systems (perceptual memory, semantic memory) are termed noetic – that is, they require conscious reflection, and the last memory system – the episodic-autobiographic one – is named autonoetic, which means that it requires self-reflection, self-awareness, and reconstructive processes. Interestingly, most neurologic and psychiatric patients manifest impairments principally in the episodic-autobiographic memory system, which indicates that this system requires a complex synchrony of neuronal assemblies (e.g., circuits for emotion and cognition) in order to function properly (cf. Figure 8 in Markowitsch, 2013, and the accompanying description).

MEASUREMENT PROBLEMS OF RETROGRADE AMNESIA

It is still much easier and much more valid to measure anterograde than retrograde memory functions (Markowitsch, 1992c). This problem is inherent in the deficit structure: It is always possible to apply stimulus material which is unknown to the patient, ask the patient to learn it, and measure learning progress. It is, however, much less reliable and valid to assess information which might or might not have been acquired properly or which might have got lost, or been suppressed or repressed with passing time. All patients have a very individual background determining what they learned, paid attention to, or neglected. Aside from a few stereotypically learned facts (own birthday, own name, names of parents, place of birth, and the like), knowledge depends on personal interests and intellectual background. Furthermore, episodic-autobiographic retrieval usually is accompanied by the feeling of autonoesis – of self-experience and emotional colorization (Staniloiu et al., 2010; Markowitsch and Staniloiu, 2011a, 2012a, b, 2013;

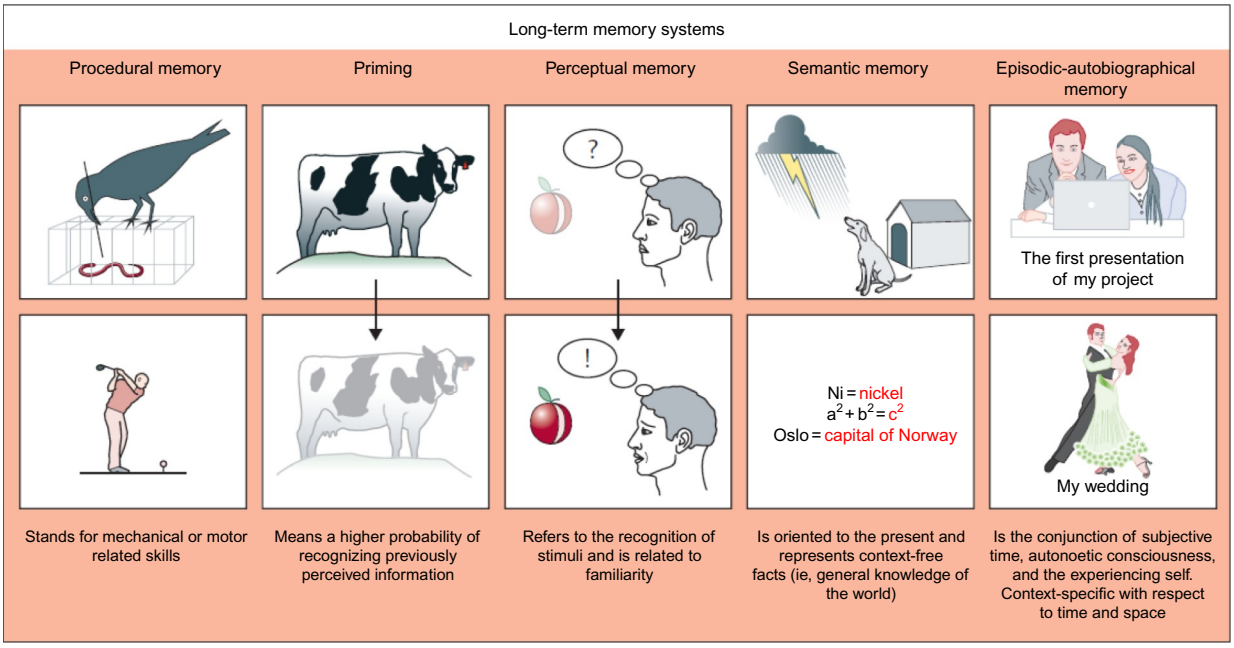

Fig. 36.2. The five long-term memory systems, based on Tulving's terminology and ideas (e.g., Tulving, 2005). These systems are assumed to develop from simple to complex (from left to right). Therefore, the simple systems, such as procedural memory and priming, exist in human beings from early childhood onward and in simple forms of animals, while the complex ones are only available in older children or in more advanced species. Tulving and others postulate that the episodic-autobiographic memory system exists in its full form only in (healthy) human beings. Also based on Tulving's work is the "remember–know" distinction: remembering occurs with a context and with conscious reflection ("reliving the event") and therefore refers to the episodic-autobiographic memory system, while knowing simply refers to yes or no distinctions without further connotations. (Reproduced from Staniloiu and Markowitsch, 2014, with permission from Elsevier).

Staniloiu and Markowitsch, 2012a, b, 2015; Markowitsch, 2013). For patients with dominant retrograde amnesia there is always the possibility that they relearned information about themselves which then is processed similarly to information learned about a third (unknown) person (Markowitsch et al., 1997b; Staniloiu and Markowitsch, 2012c; Markowitsch, 2013).

If a patient denies remembering anything from his or her personal past, one either can believe that, and not apply any autobiographic retrograde memory tests (cf., e.g., Fujiwara et al., 2008 or Markowitsch and Staniloiu, 2013 for descriptions), or try to apply symptom validity tests which again are not central, as they tap on anterograde memory abilities. Furthermore, the patient may still have stored his or her personal past, but is not aware of that (Mayes, 1988; Prigatano and Schacter, 1991; Schacter and Prigatano, 1991). This in fact may be the case in most patients with idiopathic amnesia, that is, amnesia of an unknown or uncertain cause (Markowitsch, 2002; see Panel 1 of Staniloiu and Markowitsch, 2014), or with so-called focal (as opposed to widespread cerebral) brain damage. For these cases indirect measures such as priming tasks (Damasio and Tranel, 1990; von Cramon et al., 1993) or application of galvanic skin response measures (Tranel and Damasio, 1985; Markowitsch et al., 1986; Damasio et al., 1991) might be used. Other problems are the possibility of increased emotional bluntness, especially after right-hemispheric lesions (Cimino et al., 1991; Schore, 2002; Moriguchi et al., 2006; Seidl et al., 2006; Anderson et al., 2011; cf. also Kihlstrom et al., 2013, who suggested that hypnosis might be mediated by the right hemisphere alone, and Quirin et al., 2013, who found electrophysiologically that emotions such as love are related to the right hemisphere). If there are psychiatric concomitants, a so-called overgeneral memory effect may be found; that is, the patients provide only very general information about their past life events and fail to show adequate emotional engagement (Williams et al., 1996; Watkins et al., 2000; Valentino et al., 2009). That variables such as vividness, contents, detail, and emotional colorization affect individual memories, especially if they stem from different time periods of the patient's life, was remarked already several decades ago (Squire and Cohen, 1982).

RETROGRADE AMNESIA: ORGANIC OR PSYCHOGENIC?

Since the early times of clinical brain research, relations between certain forms of brain damage and memory disturbances have been discussed. Interestingly, this research seemed to be more fruitful for anterograde than for retrograde memory, since for patients with anterograde amnesia distinct forms of brain damage were found (Markowitsch, 2008; Markowitsch and Staniloiu, 2012a). First two, and later three, types of brain damage leading to anterograde amnesia (with in part differing concomitant behavioral deficits) were established – medial temporal-lobe amnesia, diencephalic amnesia, and basal forebrain amnesia (cf. Table 1 in Markowitsch and Staniloiu, 2012a). It also was found that left-hemispheric damage leads to more severe deficits in the verbal, and right-hemispheric damage in the nonverbal, domains (Jokeit et al., 1997).

For retrograde amnesia such clearcut relations were much less obvious initially. Instead, especially patients with traumatic brain injury (TBI) were found to suffer from long-standing memory loss (Schlesinger, 1916; Russell and Nathan, 1946; Deelman et al., 1990; Rees, 2003; Anderson, 2004). Diffuse brain injury, including white-matter damage (Gale et al., 1995), and coma duration were seen as predictors of memory disturbances in the retrograde direction (Stuss and Richard, 1982; Markowitsch, 1999a). Already in 1899 Paul stated that "the degree or extent of amnesia is to a certain degree proportional to the duration of coma" (p. 264).

By far most cases with retrograde amnesia were, however, initially attributed to be of hysteric nature (Markowitsch, 1990a, b, 1992a). As mentioned above, hysteria was a very popular concept at the turn of the 20th century and was fostered from psychodynamic as well from other schools (Charcot, 1892; Breuer and Freud, 1895; Ganser, 1898, 1904; Janet, 1907; Matthies, 1908). Hysteria in those days meant emotional changes that lead to symptoms in sensory, motor, or mental domains. Aside from psychogenic blindness or paralysis, amnesia was especially often diagnosed and interpreted as a mechanism of protection against an adverse environment. As hysteria was considered to be a psychogenic illness, no underlying organic changes were assumed to exist. However, already in the 19th century Bennett (1878) had published a "case of cerebral tumour-symptoms simulating hysteria" and had questioned the dichotomy between organic and psychogenic illnesses. On page 120 he wrote about "Miss A., a young lady aged 16" (p. 114):

> *In conclusion, there appear to me to be at least two points of interest in this case: 1st, the anomalous symptoms of pressure caused by the tumour; and 2nd, that symptoms of what is called hysteria may co-exist with organic disease of the brain – whether independent of it or the result, being in this patient doubtful. Under any circumstances it serves to indicate what caution should be exercised in diagnosing, and more especially in treating, as hysteria, any nervous affection in women which may appear indefinite or mysterious.*

(A case with related symptomatology after brain disease was reported by Savage in the same year and in the same journal.)

While in later times many case reports appeared showing intermingling of brain disease or damage and psychic disturbances such as posttraumatic stress disorder or dissociative amnesia (Osnato, 1930; Silver et al., 1997; Joseph and Masterson, 1999; O'Neill of Tyrone and Fernandez, 2000; Kim et al., 2007; Mishra et al., 2011; Sehm et al., 2011; Pommerenke et al., 2012; Staniloiu and Markowitsch, 2014; Toussi et al., 2014), as long ago as 1870 a well-known medical doctor, Henry Maudsley (after whom a London hospital was named), wrote "Mental disorders are neither more nor less than nervous diseases in which mental symptoms predominate, and their entire separation from other nervous diseases has been a sad hindrance to progress" (p. 41). Maudsley's statement is remarkable particularly in light of the fact that neuropsychiatric societies were established only in the 1980s and 1990s.

The discussion on brain correlates of psychiatric diseases is, of course, continuing (cf. Pietrini, 2003) and – as Pietrini (2003) remarked – there is evidence for changes in glucose metabolism, in volume of limbic structures, and in white-matter changes in patients with dissociative disorders (Markowitsch et al., 2000a; Vermetten et al., 2006; Tramoni et al., 2009). Nevertheless, it should be emphasized again that extensive retrograde amnesia – aside from in patients with severe dementia (Piolino et al., 2003; Jetten et al., 2010) – seems more frequently to be a psychiatric than a neurologic disease. Already in 1911, Heine reviewed possible amnesic states and listed many with a psychologic background (Table 36.1). Similarly, a few years later, Schneider in 1928 formulated on p. 520 that there is evidence against a sharp distinction of organic and functional amnesic states.

In 2014 we reviewed the current understanding of dissociative amnesia, its epidemiology, clinical and psychologic features, and hypotheses for its occurrence. We view dissociative amnesia as a condition which is (in most cases) stress-related and is based on negative past experiences with which the patient could not cope adequately (Staniloiu and Markowitsch, 2014; Table 36.2). Patients with dissociative amnesia usually either had experienced a major negative event (such as in a life-threatening war situation), or, much more frequently, a number of negative events, the last of which led to dissociative amnesia (e.g., Markowitsch et al., 1999c). Because of this, we proposed the "two-hit hypothesis" as a likely cause for its occurrence, with two hits meaning "an additive or synergistic interaction between psychological and physical incidents" (p. 231) of a negative, adverse nature (see also Roberts et al., 2013).

Table 36.1

Conditions leading to memory disturbances according to Heine (1911, p. 55f)

1. Epileptic somnolence
2. Hysteric somnolence
3. States of unconsciousness and of mnestic activity after traumatic damage of the brain:
   1. Commotio cerebri
   2. Attempt to hang oneself
   3. Reanimation after hanging
4. States of somnolence with a relation to physiologic sleep
5. Hypnotic states
6. Migraine-based somnolence
7. Affect-based somnolence
8. Toxic somnolence, or disturbance of mind:
   1. Complicated states after intoxication
   2. Disease of the mind after carbon monoxide inhalation
9. Vasomotoric states of somnolence:
   1. Congestive (transitory mania)
   2. Angiospastic (raptus melancholicus)
10. Transitory disturbances of mind after infectious diseases
11. Paralytic attacks
12. Retrograde amnesia without previous disturbances of consciousness
13. Korsakoff's psychosis

SHORT REVIEW OF RETROGRADE AMNESIA AFTER STRUCTURAL BRAIN DAMAGE

As the topic here is functional retrograde amnesia, directly organic-based retrograde amnesia will only be briefly summarized. Etiologies for cases with predominant retrograde amnesia after brain diseases or brain injuries are: (1) TBI/minor brain injury; (2) viral infections such as herpes encephalitis; (3) degenerative brain diseases (e.g., Alzheimer's disease); (4) brain infarcts; (5) severe hypoxia (e.g., carbon monoxide poisoning, attempted hanging); and (6) Korsakoff's syndrome. Usually milder forms of retrograde amnesia are found in patients with transient epileptic amnesia and in transient global amnesia (TGA), in which it lasts by definition less than 24 hours. (TGA refers to an amnesic condition of sudden onset, usually affecting old people. It is triggered by sudden physical or psychic changes (e.g., considerable temperature change, unusual physical exercise, or an unexpected psychologic stress situation) and results in usually complete anterograde and partial retrograde amnesia in the episodic-autobiographic domain. It passes away within 24 hours.)

Brain infarcts and vascular brain damage

Brain infarcts usually lead to a combination of anterograde and retrograde memory disturbances with a higher

Table 36.2

Sequence of possible changes in brain–behavior interrelations induced by stress-conditions

Psychological or biological stress or trauma situations, especially in childhood
↓
Biological priming
Change in receptor structure
↓
Supersensitivity for excitatory neurotransmitters
↓
Latency phase
↓
(Re-)activation
via
Psychological mechanisms or biological events
(e.g., conflict, deprivation, accident, infection)
↓
Absence of adequate emotional-cognitive processing
↓
Second latency phase
↓
Dissociation between cognition and emotion
Psychobiologic stress reaction
↓
Depressive tendencies
↓
Dissociative amnesia / mnestic block syndrome

After Aldenhoff (1997) and Markowitsch (2000).

proportion of anterograde deficits (Markowitsch, 1988, 2008). Nevertheless there are case descriptions of severe and lasting retrograde amnesia also after damage to very focal regions such as the medial diencephalon (Hodges and McCarthy, 1993; Markowitsch et al., 1993b; for reviews, see: van der Werf et al., 2000; Carlesimo et al., 2011). In most of these patients retrograde amnesia follows Ribot's law, which states that old memories from childhood and youth are better preserved than recent memories from the last years (Ribot, 1882). (e.g., Markowitsch et al., 1993b). This clearly distinguishes brain-damaged patients from those with a dissociative amnesic condition where such a gradient is absent. It indeed seems that more widespread brain damage or a long-standing disease condition such as epilepsy can lead to more extensive retrograde amnesia, while restricted hippocampal damage may lead to more time-limited episodic-autobiographic memory loss (or inaccessibility). However, it needs to be emphasized that there may frequently be a difference between visible brain damage and existing brain damage, as was demonstrated, for example, for brain damage after heart attack (Markowitsch et al., 1997d).

Degenerative and metabolic brain diseases

Generally, most of the classic forms of dementia are subsumed under degenerative diseases. However, also metabolic, toxic, and viral diseases and continuing epilepsy-caused neural hyperactivity (excitotoxicity) may lead to brain degeneration. Therefore we will subsume Korsakoff's disease and herpes simplex encephalitis under this heading (even tuberculous meningitis might be added here, as Kapur mentioned in 1993).

Many of the group of dementia diseases lead in their more advanced stages to retrograde amnesia. This is due to a disintegration of cerebral networks involved in the storage of memories (Markowitsch, 2013). Seidl et al. (2006), for example, found that the more the condition of Alzheimer's disease progresses, the less detailed, less complete, and less comprehensive were reports of patients about their past. Furthermore, the number of reported events shrank with advancing disease. As an exception to the usual temporal gradient (Ribot's law) in dementias, in semantic dementia a reversed gradient seems to exist. Possible reasons for this were given by Kopelman (2002).

Korsakoff's disease is a thiamine deficiency-related degeneration of medial diencephalic nuclei, nowadays affecting mainly patients with severe alcohol abuse. Korsakoff's patients probably have been studied the longest among groups with different etiologies (Markowitsch, 1992a, 2010). Already in 1852 Huss wrote a book of roughly 600 pages on *Chronische Alkoholskrankheit oder Alcoholismus chronicus* [*Chronic Alcohol Disease or Alcoholismus chronicus*], in which he emphasized its negative effects on mental capacities, stating that memory becomes weak (p. 356). Markowitsch (2010) concluded from reviewing the available data on patients with Korsakoff's symptomatology that their memory deficits are principally "in the domain of (anterograde) episodic-autobiographic memories, and much less so in the domains of the other memory systems currently defined. With respect to semantic retrograde memories, the deficit is less pronounced, as can be inferred from a superior retrieval capacity under conditions of recognition compared to free recall." (p. 133). (cf. Markowitsch et al., 1984, 1986).

Epilepsy-related amnesic conditions are even found in patients with transient epileptic amnesia (Milton et al., 2010; Butler and Zeman, 2011; Soper et al., 2011) and are usually of anterograde nature (Bartsch and Butler, 2013). Long-lasting epilepsy may be accompanied by remote memory problems (Viskontas et al., 2000, 2002; Lah et al., 2006, 2008). Whether temporal extent and content-based broadness of retrograde amnesia vary with the extent of temporal-lobe damage still seems uncertain (Gold and Squire, 2006; Noulhiane et al., 2007; Insausti et al., 2013; Gregory et al., 2014).

Hypoxia

Hypoxic-ischemic brain lesions regularly result in cognitive disturbances (Anderson and Arciniegas, 2010). It is known that severe hypoxic conditions can lead to retrograde amnesia, probably due to reduced hippocampal volumes (Allen et al., 2006) or due to volume reductions in other brain regions (Hokkanen et al., 1995, 1996a, b; Markowitsch et al., 1997b; Kopelman et al., 2003). Especially cases with developmental amnesia demonstrate that hypoxia at birth may lead to medial temporal-lobe degeneration (reduced hippocampal volumes) and severe retrograde amnesia for the episodic-autobiographic domain (Staniloiu et al., 2013). For one of those patients it was detected that he had congenital absence of the mammillary bodies (Rosenbaum et al., 2014). These patients, however, constitute an exception within the category of patients with hypoxic-ischemic brain damage, as they probably were unable from early life on to consolidate episodic-autobiographic events. But also patients suffering from sudden hypoxia (e.g., after attempted hanging) (or even chronic hypoxia, as in sleep apnea patients) have long been found to suffer from partial (time-limited) retrograde amnesia (Boedeker, 1896; Markowitsch, 1992b; Reinhold et al., 2008).

A particularly interesting case with a background of hypoxia was published many decades ago by Grünthal and Störring. These authors investigated (1930) and followed up (Störring, 1931, 1936; Grünthal and Störring, 1933, 1956) a case of carbon monoxide intoxication with particular deficits in the anterograde memory range, but with massive retrograde amnesia as well. The patient's behavior with respect to memory performance, emotions, will, spontaneous activity, and ability to think and reflect consciously was documented over more than 120 pages in Störring's publication of 1931.

The patient's retrograde amnesia followed typically Ribot's law (the more recent the information is, the more likely it was lost, while, in contrast, the longer it had been stored, the more likely it was retained). He had a good ability to remember events from his youth, but practically no knowledge of his recent past. Grünthal and Störring (1930) speculated on the morphologic substrate of his amnesia and negated the existence of diffuse brain damage, but acknowledged the possibility "that the more refined physical-chemical processes of large brain areas might have suffered so differently in their dynamics or quality that especially the correlates of mnestic functions are affected" (p. 368). They preferred, however, to assume that distinct brain portions such as the mammillary bodies might have been damaged.

In 1933, the patient married his fiancée (who had already been mentioned in the 1930 report) and lived at home. He was still markedly amnesic and introduced his wife consistently as his fiancée. He was always happy to see her, as if he had just fallen in love. He showed appropriate behavioral stereotypes, such as taking off his hat when entering church or when being greeted, and was able to behave well during meals and to explain industrial drawings he had made about 10 years previously. But he used external help to memorize. For instance, he once explained that it must be Sunday because he was wearing a suit or that he would not be traveling on a train, as he was not dressed appropriately. He also assisted his wife in climbing a mountain as he remembered from the time before his accident that she had difficulties on such occasions.

It is interesting that, when asked about the present date, he always said "the last day of May, 1926," and in fact his accident had occurred on May 31 in 1926. His response resembles that of an amnesic patient who had had a stroke affecting the diencephalon and who always gave the year as 1981 – the year of his stroke – when asked in 1990 (Markowitsch et al., 1993b).

The case of Grünthal and Störring was revisited by Craver et al. (2014a). These authors discussed the pros and cons of the case with respect to being a true amnesic or a faker, citing also all the later work published on this case (e.g., Grünthal and Störring, 1954). A scientist, criticizing the description of Grünthal and Störring, also mentioned, according to Craver et al. (2014a), that the patient – Franz Breundl – at times gave implausible answers to questions, suggesting some kind of hysteric pseudodementia. If true, this might speak of features of Ganser's syndrome, a psychiatric disease characterized by *vorbeireden* (giving approximate answers, such as "3," in response to the question, "How many legs does a cat have?) and disturbed consciousness, and which for many years was subsumed under the category of psychogenic amnesic states (cf., e.g., the case given in Staniloiu et al., 2009).

Craver et al. (2014a) pointed out that philosophers might see in Breundl's case the continuity of personality in the absence of memory. One of us (H.J.M.) had a related case with carbon monoxide poisoning (due to a suicide attempt). This patient, however, seemed to have changed in personality due to his anterograde as well as retrograde memory loss. He usually appeared to be joyful and gregarious, demonstrating this also when he was invited to a TV talk show, where he immediately asked all women watching TV to make a date with him. From analyzing case descriptions of patients with retrograde dissociative amnesia, it seems that, though character traits may persist, a number of features of the self may be altered (Fradera and Kopelman, 2009; Rathbone et al., 2009, 2015; Staniloiu et al., 2010; Arzy et al., 2011; Markowitsch and Staniloiu, 2011a; Markowitsch, 2013).

Both the patient Breundl and this patient demonstrate that carbon monoxide poisoning as an etiology may be

particularly prone to a mixture of organic (hippocampal degeneration) and psychogenic causes of retrograde as well as anterograde amnesia. (See also the descriptions of the anterograde psychogenic amnesic case Q. in Markowitsch and Staniloiu, 2013, of case T.A. in Markowitsch et al., 1999b, and of case F.L. in Smith et al., 2010.)

Traumatic brain injury/minor head trauma

TBI, accompanied by retrograde memory impairment, constitutes a very broad category ranging from minor head concussions to severe brain tissue damage (Russell, 1935, 1971; Russell and Nathan, 1946; Fisher, 1966; Lucchelli et al., 1995, 1998; Dean and Sterr, 2013). Temporary cardiac arrest may be added as related in its consequences to TBI. In war time, shot and shrapnel injuries were common (Kleist, 1934). Interestingly, the duration of retrograde amnesia varies considerably after TBI and is not always predictable from available variables (though coma duration is to some extent a predictor) (Markowitsch and Calabrese, 1996). Also a relation between posttraumatic amnesia duration and long-term cerebral atrophy was established (Wilde et al., 2006). Common to most forms of TBI is an inability to remember the immediate time period before and after the injury.

Another feature of patients with TBI is that TBI "seems to make patients particularly susceptible to depressive episodes, delusional disorder, and personal disturbances" (Koponen et al., 2002, p. 1315). (The same, at least with respect to depression and personal disturbances, holds true for patients with dissociative amnesia.) Such psychiatric changes may add to memory disturbances as it is known that there exists a strong relation between depression, personality disorders, and memory problems (Markowitsch et al., 1999c; Staniloiu and Markowitsch, 2014, 2015). A more refined analysis of the brain of TBI patients, for example, with diffusion tensor imaging or magnetization transfer ratio, may reveal microstructural changes that could account for continuing behavioral deficits (e.g., Back et al., 1998; Bendlin et al., 2008; Sidaros et al., 2008; cf. also Grafman et al., 1988; Markowitsch and Calabrese, 1996). Also the coma state may alter the usual biochemical inflow between neuronal assemblies representing stored information (Markowitsch, 1988). Prolonged disuse – also a consequence of coma and concussions – may enhance such detrimental effects.

Cases with severe amnesia after major TBI – relation to functional amnesia?

One of the best-known cases with both severe anterograde and retrograde amnesia is patient K.C., who had a motor cycle accident damaging several portions of his brain (Rosenbaum et al., 2005, 2009; Craver et al., 2014b).

Since the 1990s several papers, reporting complete or nearly complete retrograde amnesia in the episodic-autobiographic domain after combined temporopolar and frontal brain damage have been published (e.g., Kapur et al., 1992; Markowitsch et al., 1993a; Calabrese et al., 1996; Kroll et al., 1997; Markowitsch and Ewald, 1997; Levine et al., 1998) (Fig. 36.3). That is, such patients apparently had forgotten their whole life, and did not remember their partner or their profession. However, they could still read, write, and calculate or were able to relearn these skills quickly. Likewise, as our patient demonstrated, social skills, priming, and procedural memory were principally preserved (Markowitsch et al., 1993a). Similarly, case reports with mainly left-hemispheric cortical damage and severe retrograde amnesia in the semantic memory domain appeared from the late 1980s (De Renzi et al., 1987; Grossi et al., 1988; Markowitsch et al., 1999a). (The patient of De Renzi et al., however, did not have TBI, but herpes encephalitis; cf. also the encephalitis patient of Hokkanen et al., 1995, for whom the authors, however, excluded a psychogenic etiology.) These patients remembered their relatives, but were unable to recognize prominent politicians or actors.

It was assumed that the retrograde amnesia was "focal" or "isolated," that is, an isolated symptom that was the consequence of either the cortical damage or of some other mechanisms (e.g., Goldberg et al., 1982; Kapur et al.,

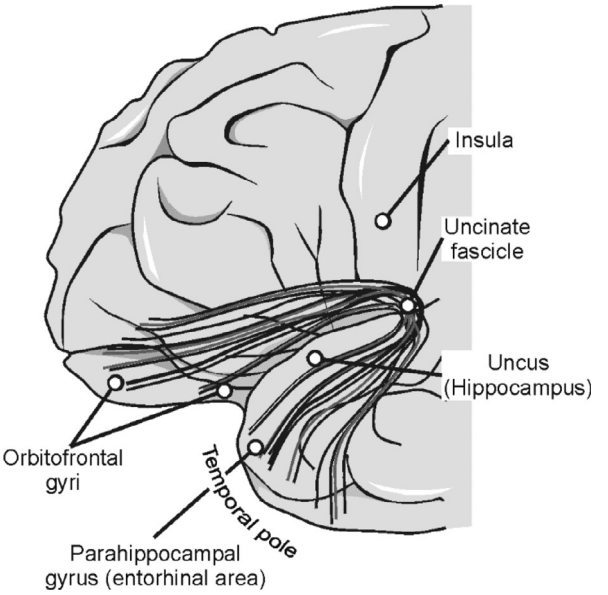

Fig. 36.3. The region of the temporofrontal cortex containing the uncinate fasciculus. It is assumed that this region is engaged in triggering the retrieval of consciously processed information – primarily semantic memory in the left hemisphere, and primarily episodic-autobiographic memory in the right hemisphere.

1989, 1992; Yoneda et al., 1992; Kapur, 1993; Hunkin et al., 1995; Hokkanen et al., 1995; Parkin, 1996; Levine et al., 1998, 2009; Fast and Fujiwara, 2001; Miller et al., 2001; Yamadori et al., 2001; Teramoto et al., 2005; Stracciari et al., 2008; Sehm et al., 2011). Sometimes also the expressions "disproportionate retrograde amnesia" (Kapur et al., 1996; Thomas-Antérion et al., 2014) or "permanent global amnesia" (Kritchevsky and Squire, 1993), or "pure retrograde amnesia" (Lucchelli et al., 1998) were used. This assumption was, however, questioned by Kopelman (2002), who also provided various different interpretations. As there were other – even earlier – cases with TBI (and other etiologies; e.g., Roman-Campos et al., 1980) and retrograde amnesia (e.g., Goldberg et al., 1981, 1982), the idea that at least some of the brain-damaged patients had dissociative amnesia or a combination of dissociative (psychogenic) and brain-organic amnesia was suggested (De Renzi et al., 1995, 1997; Markowitsch, 1996a, b) and the term "functional amnesia" was proposed for these cases (Lundholm, 1932; Schacter and Kihlstrom, 1989; De Renzi et al., 1997; Brandt and Van Gorp, 2006), implying that the amnesia served a function in their life. However, even in more recent times case reports appear which point to the "same faulty mechanism in the neural circuitry" (Ouellet et al., 2008, p. 27) in cases with brain damage (in the "organic" case of Ouellet et al., a wound in the right temporal lobe, caused by a nail gun) and after an intense emotional trauma in the other case of Ouellet et al. A similar view was already held by early researchers such as Syz (1937) and Maudsley (1870), and was also formulated in an editorial by Pietrini (2003) (see above).

DISSOCIATIVE AMNESIA WITH AND WITHOUT TBI

Nearly 150 years ago the first case reports of patients with so-called hysterical amnesia occurred and a number of them had a combination of minor TBI and a "hysterical amnesic state" (Markowitsch, 1992a). There is nearly always a problem when a minor head trauma leads to lasting retrograde amnesia (cf. Ruff and Jamora, 2009). If the patient is young and therefore probably of an immature personality structure, the likelihood for a psychiatric condition is even higher (Staniloiu and Markowitsch, 2014). While it is assumed that a long duration of the amnesia and the lack of compensation claims may speak for an organic origin (e.g., Hunkin et al., 1995), this cannot be seen as a rule. We have, for example, a patient whose variant of dissociative amnesia has lasted now for over 20 years – since 1994 (the case was first published in Markowitsch et al., 1999b).

There are a number of case descriptions of patients under 20 years of age, most of them still attending school (Reinhold and Markowitsch, 2007), who after minor accidents developed retrograde amnesia in the autobiographic domain. Lucchelli et al. (1998) described a 15-year-old boy who became retrogradely amnesic after a minor head bump; Markowitsch and Staniloiu (2013) described another one, who bumped against the opening door of a cigarette machine; Barbarotto et al. (1996) discussed a 38-year-old woman who slipped and fell in her office, resulting in pure retrograde amnesia, and a considerable number of patients were involved in motor vehicle accidents (De Renzi and Lucchelli, 1993; Stracciari et al., 1994; De Renzi et al., 1995, 1997; Mortati and Grant, 2012). For most of these patients, no brain injury could be detected on the basis of neuroimaging. In a few there was minor evidence for metabolic alterations, as inferred from single-photon emission computed tomography (SPECT) or positron emission tomography (PET) using radioactive water (Markowitsch et al., 1997b) or glucose (Markowitsch et al., 1998, 2000a; Brand et al., 2009; Thomas-Antérion et al., 2010, 2014). Some of the patients recovered from their amnesia after varying time periods (Lucchelli et al., 1998). Sometimes, it remained obscure whether any specific event had happened (Dalla Barba et al., 1997) and sometimes "purely" psychic conditions seemed likely (Kessler et al., 1997; Markowitsch et al., 1999c; Kritchevsky et al., 2004). A 9-year-old boy was diagnosed with TGA after suffering from retrograde and anterograde amnesia (Meijneke et al., 2014); there might have been a minor emotional trigger for his amnesia, as his new watch broke that day. As Bartsch and Deuschl (2010) remarked, an emotional – and therefore psychologic – trigger can be found in 30% of patients with TGA. While the authors did not consider a dissociative condition, there may in fact have been features of this, though the patient remembered facts like his name and address.

These and related cases (e.g., Reinhold and Markowitsch, 2009; Staniloiu and Markowitsch, 2012a, b, c; Markowitsch and Staniloiu, 2013) provoke the question on possible mechanisms of retrograde amnesia induction, maintenance, and resolution (Markowitsch and Staniloiu, 2012c). These will be discussed below.

THE PUZZLE OF RETROGRADE AMNESIA OCCURRENCE

The occurrence of retrograde amnesia is a puzzle, because, much more so than anterograde amnesia (Markowitsch, 2008; Markowitsch and Staniloiu, 2012a), it cannot be linked to a distinct neuropathology (cf. Kopelman, 2000). However, one of the distinct differences between patients with a clear organic cause and

those with probable or likely psychogenic origin is the usually found gradient ("Ribot's law") in the organic, but not in the psychogenic, cases (Brown, 2002; Staniloiu and Markowitsch, 2012a–d, 2014, 2015). McKay and Kopelman (2009) even propose a reversed gradient for psychogenic amnesia. Furthermore, the cases with primarily psychogenic origin usually show some distinct personality features, namely a poor childhood or youth, an insecure personality profile as adults, a heightened vulnerability towards stress and stressful events, a lack of appropriate coping strategies against surprising or threatening situations, and a heightened susceptibility towards suggestions from others (Markowitsch, 2009).

In Tables 36.3 and 36.4 certain features accompanying severe and lasting retrograde amnesia of primarily organic and psychogenic origin are listed. For retrograde amnesia of direct organic origin, extent and locus of brain damage are considered to be responsible for the deficit. For retrograde amnesia with less obvious brain damage, as in mild head trauma and concussion, a number of factors were listed as possibly leading to a usually more transient or less severe retrograde amnesia. Among them are *contrecoup* damage, rotational forces leading to axonal and synaptic injury, gliosis, and biochemical changes at the microstructural level (Schoenfeld and Hamilton, 1977; Walker and Tesco, 2013). Demyelination and accompanying frontal-lobe dysfunctions have been added (Craver et al., 2014a), as well as intracranial microbleeding, hypoxia, and the formation of plaques with time.

Some of these aforementioned changes may more likely be seen with advanced neuroimaging techniques such as glucose PET, diffusion tensor imaging, magnetic resonance spectroscopy, or magnetization transfer imaging. This is clearly exemplified in a study of Ruff et al. (1994), who examined 9 patients suffering minor TBI with little or no evidence of computed tomography- or magnetic resonance imaging-proven brain damage, but with deficient neuropsychologic performance. PET examination on the other hand confirmed for all 9 patients the neuropsychologic evidence. On a similar line, Markowitsch and coworkers tried to prove an organic basis for primarily psychogenic forms of amnesia since 1997, mainly on the basis of PET investigations (Markowitsch, 1999d; Markowitsch et al., 1997a–c, 1998, 2000a; Brand et al., 2009). Other workers followed this line of research (Hennig-Fast et al., 2008; Stracciari et al., 2008; Tramoni et al., 2009), which revealed significant changes, most consistently in frontotemporal regions of the right hemisphere (Reinhold et al., 2006; Staniloiu and Markowitsch, 2010; Staniloiu et al., 2011). An overview of such studies is given in Table 4

Table 36.3

Distinct features of dissociative compared with direct organically based amnesias*

	Dissociative amnesias	Organically based amnesias (neurocognitive disorders)
Age at the time of diagnosis (years)	20–40	Variable
Course	Acute or chronic	Acute or chronic
Episodic-autobiographic anterograde amnesia	Less common	Most common
Episodic-autobiographic retrograde amnesia	Most common	Uncommon and rarely without anterograde amnesia
Loss of personal identity	Common	Uncommon
Preservation of learning of new facts	Usual, but not always	Rarely reported
Onset related to trauma or psychologic stress or conflicts	Common	Uncommon
Precipitants	Psychologic stress with or without physical events	Neural tissue damage (but also emotional precipitants in transient global amnesia)
Reversal of memory loss with hypnosis	Sometimes	No
Improvement with sedative hypnotics (e.g., pharmacologically facilitated interview)	Sometimes	No, or may worsen
Brain damage	In most cases not detectable by conventional neuroimaging techniques or postmortem	Usually structurally detectable or postmortem
Affected brain regions (metabolic or tissue damage)	Prefrontotemporal areas/limbic system	Variable, usually limbic areas

*Some criteria have been adapted from Table 4 of Spiegel and colleagues (2011).

Table 36.4

Similarities and differences between cases with retrograde amnesia due to organic or psychogenic causes

	Direct organic causation	Psychogenic causation
Initiating event	Traumatic brain injury, infarct, etc.	Psychic stress, mild head trauma*
Cognitive impairment beyond memory	Frequently and frequently severe	Usually limited, though existent
Factual self-knowledge	Usually preserved	Not preserved
Reversibility	Full reversibility infrequent	Full reversibility possible and sometimes quick
Gradient ("Ribot's law")	Usually existent	Nonexistent
Congruence of brain damage and degree of amnesia	Usually given	Usually not given
History of subtle personality disorder or premorbid psychiatric disease	Usually nonexistent	Usually more likely to exist

*It should be noted that, in transient global amnesia, about one-third of cases are triggered by an emotional stressor (Bartsch and Deuschl, 2010). Partly after Reinhold and Markowitsch (2009).

of Staniloiu and Markowitsch (2014). Interestingly, while older studies emphasized the transience of dissociative amnesia, more recent work points to chronic conditions in a number of cases (Coons and Milstein, 1992; Markowitsch and Staniloiu, 2013; Staniloiu and Markowitsch, 2014).

On the other hand, visible and proven organic brain damage – contrary to classic descriptions – does not preclude the formation of dissociative amnesia (Lucchelli et al., 1995; Markowitsch, 1996a, b; Mishra et al., 2011; Pommerenke et al., 2012) (though this diagnosis is not always considered: see Wilson et al., 2015). The existence of cases with organic brain damage and dissociative amnesia indicates – similarly to TGA (Bartsch and Deuschl, 2010; Markowitsch and Staniloiu, 2012b) – that emotional stress as well as somatic-physical alterations may result in a similar symptomatology. Furthermore, in TGA the symptomatology is by definition transient and short, and in dissociative amnesia apparently rather complete recovery from the symptom of episodic-autobiographic amnesia has been found as well. While recovery seems always to be spontaneous in TGA, in dissociative amnesia it is more likely triggered by various mechanisms such as hypnosis, amytal injection, electric stimulation, drug medications, or – of course – conventional forms of psychotherapy (Naef, 1897; Bumke, 1924; Krarup, 1924; Schneider, 1928; Stuss and Guzman, 1988; Iglesias and Iglesias, 2009; Lee et al., 2011), though more or less spontaneous recovery has been reported as well (Lucchelli et al., 1995). Such findings encourage the postulation of an "inability of access" hypothesis for retrograde amnesia, thus leading to the idea that organic and psychogenic amnesia are similar insofar as both represent a frequently temporary (and partial or selective) memory loss, primarily affecting the episodic-autobiographic domain (Markowitsch, 1996a, b, 2002) (Fig. 36.2).

INABILITY OF ACCESS HYPOTHESIS

This hypothesis states that (episodic-autobiographic) memory still is stored in the brain, but that, due to an interruption in communication between brain network systems engaged in memory storage and in memory retrieval, a successful conscious recollection of episodes is blocked. Klein (2015) stated:

> *Recollection consists in two separate but interdependent parts. First, to count as an act of recollection (= memory) a mental state must be causally linked to an experience the individual formerly enjoyed. Second, memory is not simply* from *the past; it is a special way of being* about *the past … To qualify as an act of memory, the content present in awareness must present itself as a re-experience of an experience previously had. This feeling of re-experiencing is directly given to consciousness, rather than the product of an act of inference or interpretation.*

There are two – at first glance – opposite ideas: one, that direct brain tissue damage is responsible for the blockage of old personal memories, and the other, that a psychic disturbance causes the observed retrograde amnesia (sometimes even just headache seems to result in persistent retrograde amnesia: Reinvang and Gjerstad, 1998). In fact, however, these two approaches are not mutually exclusive: they have a common denominator. Assuming that all psychic phenomena have an organic basis, the question is only how to measure the basis and how to find out about its plasticity (reversibility, stableness).

As mentioned before, the idea of "organicity" is not new (Maudsley, 1870; Flechsig, 1896a, b; Syz, 1937; Freud, 1954; Markowitsch, 1996a, b; Pietrini, 2003), though the separation between neurologic and psychiatric diseases seems to have even widened in the last decades (Markowitsch, 1999c). There are two prominent examples from the beginning of the 19th century, Freud and von Monakow. Freud started his career in neurology, publishing on fiber tracing and aphasia (see Markowitsch, 1992a) and worked from 1895 on an *Entwurf einer Psychologie* [Project of a Scientific Psychology], which was published only after his death (Freud, 1954; cf. Peper and Markowitsch, 2001). Freud was trained in neurobiologic subjects as a student of Ernst Brücke at the Physiological Institute of Vienna's University. Brücke was also a mentor to Exner, who took over Brücke's chair in 1891. Freud was consequently well acquainted with the work of Exner (1894), who was a pioneer in modeling brain circuits for affective and cognitive behavior (see Peper and Markowitsch, 2001). Freud (1954) assumed that ultimately a brain basis would be found for his psychoanalytic theories. He described psychologic phenomena as the routing of nervous energy in a neuron system and assumed that cerebral lesions and mental disorders have a common physiologic mechanism (cf. Jacobson, 1995).

A complementary approach came from von Monakow (1914) (see also Markowitsch and Pritzel, 1978; Engelhardt and da Mota Gomez, 2013). As explained in Markowitsch (1988), von Monakow divided shock into four types: (1) the shock of the surgeon (wound shock, traumatic shock); (2) psychic shock; (3) apoplectic shock (following a concussion of the brain); and (4) diaschisis (a usually sudden functional interruption in distinct, widely distributed central functional circuits). von Monakow in general favored a more holistic ("anti-localizationistic") approach in interpreting the consequences of brain damage. With his four forms of shock he acknowledged that both somatic-physiologic and psychic conditions can alter the functioning of the nervous system. And therefore he also at this early phase of brain research emphasized that intellectual functions can be suddenly disrupted by a variety of conditions.

Modern neuroimaging methods allow the investigation of structural as well as functional interruptions of the brain's circuitry and therefore narrow the gap between neurologic and psychiatric findings (Markowitsch, 1999c). The so-called trauma model seems to fit the findings best (Dalenberg et al., 2012; see also Vermetten et al., 2007; it is also in accordance with the writing of Freud, 1893, who considered hysteria to be caused by incompletely abreacted psychic traumata). Functional imaging findings in normal subjects attribute a role to the right-hemispheric anterior temporal lobes and the right inferolateral prefrontal cortex for triggering the recollection of episodic-autobiographic episodes (Fink et al., 1996). These regions furthermore contain emotion-processing structures such as the amygdala (Markowitsch and Staniloiu, 2011b) and are interconnected by various branches of the uncinate fascicle (Fig. 36.3), a fiber system which seems to be more expanded in the right hemisphere (Highley et al., 2002), and which seems to grow with advancing age of the individual (Lebel et al., 2008). Children reared in a deprived, neglectful environment show microstructural changes of the uncinate fascicle (Govindan et al., 2010) that may be accompanied by the so-called overgeneral memory effect, a phenomenon characterized by reduced specificity, detail, and emotional colorization of reported autobiographic episodes. A similar effect can be found in patients with Alzheimer's disease (Seidl et al., 2006). While normal individuals, requested to recollect autobiographic information from their past, demonstrate increased frontotemporal activity, particularly in the right hemisphere (Fink et al., 1996), patients with dissociative amnesia show a decreased activation in this area (Markowitsch et al., 1997b; Brand et al., 2009), or a very selective right anterior temporal increase only, which corresponds with reporting affect-related excitation towards past events which cannot be consciously narrated (Markowitsch et al., 1997c).

A major question is how the dysfunction of this combination of areas is initiated and maintained. As stress is acting, and has acted, nearly universally in patients with dissociative amnesia (Arrigo and Pezdek, 1997; Markowitsch, 1999b, 2006; Bremner, 2005, 2010; Igwe, 2013; Wabnitz et al., 2013; Magnin et al., 2014), an overflow of stress hormones (glucocorticoids; O'Brien, 1997; de Kloet et al., 2005a, b; Lupien et al., 2005, 2009; de Kloet and Rinne, 2007) and an altered activity of the hypothalamic–pituitary–adrenal axis (Heim et al., 2008) is postulated to occur, blocking the retrieval of stress-related memories and possibly also generalizing to other memories of an emotional nature (Markowitsch et al., 1999c; Markowitsch, 2002; Fujiwara and Markowitsch, 2006a; cf. Wolf, 2009; Brand and Markowitsch, 2010; O'Brien, 2011; Wingenfeld and Wolf, 2014; Staniloiu and Markowitsch, 2015). Stress, induced by traumatic events during childhood or youth, seems to result in a long-term change in the brain's response to further stress situations later in (adult) life (Markowitsch, 1999b, 2000; Spiegel et al., 2013).

Hippocampal formation and the amygdala play central roles in emotion-related memory processing (Heim et al., 2008; Lupien et al., 2011; Markowitsch and Staniloiu, 2011b; Markowitsch, 2013; cf. also the

discussion between Anderson, 2004, and Rees, 2003, on the interaction between brain damage, stress, and cognitive consequences). Amygdala and hippocampal formation possess the highest density of stress hormones in the brain. Consequently, several studies reported damage to brain structures after stress and psychic trauma conditions (e.g., Sapolsky, 1996a, b, 2000; Bremner, 2005). Memory problems accompanying stress are consequently a common occurrence (Markowitsch, 1999b, 2006; Lupien and Maheu, 2000; Valentino et al., 2009; Quesada et al., 2012). And, as a number of studies found a functional lateralization, with the right hemisphere processing emotionally laden information and the left one neutral facts (Markowitsch et al., 1999a; Schore, 2002, 2005; Moriguchi et al., 2006; Gregory et al., 2014), it makes sense that functional imaging studies principally confirmed the idea of right-hemispheric episodic-autobiographic memory (see Fink et al., 1996; Markowitsch et al., 2000a, b; LaBar and Cabeza, 2006).

A relation to conscious memory suppression has been proposed as well (cf. Kikuchi et al., 2010; Staniloiu and Markowitsch, 2014). In a number of more recent articles, mechanisms of willful memory suppression were analyzed (e.g., Paz-Alonso et al., 2009, 2013; Benoit and Anderson, 2012; Detre et al., 2013; van Schie et al., 2013), a skill which might also be used to conceal guilty knowledge (e.g., Bergström et al., 2013) and which is related to prefrontal activation (Anderson et al., 2004). We have studied patients who first willfully pretended to be amnesic, but later apparently had dissociative amnesia which they no longer could control willfully. Such patients therefore demonstrate that there may be brain mechanisms which, as a consequence of stress, lead to a block of the possibility to retrieve subjectively problematic memories (cf. Fig. 15.2 of Fujiwara and Markowitsch, 2006a, and the corresponding explanation in the text). There also seems to be a relation between prefrontal activity and susceptibility to hypnosis and hysteria (Bell et al., 2011); posthypnotic amnesia has been discussed as a model for dissociative amnesia (Cox and Barnier, 2003).

The probable mechanism acting in dissociative amnesia may be comparable to when one is trying to reconstruct the contents of a dream after having been awake for some time: there may be very vague schemes, but it is not possible to arrange a united scene which includes a "what, where, when" trilogy and a first-person perspective (Markowitsch and Staniloiu, 2013). This idea has similarities to the index theory of memory (Teyler and Rudy, 2007), which assumes that there has to be an interaction between limbic regions – such as the hippocampus – and neocortical ones for conscious retrieval of information (Llewellyn, 2013). Similarly, the lack of identity feelings has parallels to sleep conditions or to conditions of dementia (Jetten et al., 2010; Stickgold and Walker, 2013), where the corticolimbic network is so broken and unstable that a coherent resonance pattern, creating identity, cannot be established. Both the unstable identity and memory conditions are preserved as long as under conditions of psychic effort stress hormones are released and block retrieval. There may be a process of prolonged continuity which then leads to alterations in the neuronal network so that with time also the connectivity between brain structures becomes damaged so that the ability to re-establish a united self (Staniloiu et al., 2010; Markowitsch and Staniloiu, 2011a; Markowitsch, 2013) and access to the personal past remains chronically impaired.

Such a condition may also follow or be strengthened by direct tissue damage in regions which usually are involved in accessing stored episodes and in preparing them for conscious reconstruction. For instance, brain damage that includes frontotemporal regions of primarily the right hemisphere can trigger the blockade of retrieving episodic-autobiographic episodes (Calabrese et al., 1996; Kroll et al., 1997). Of special interest, and somewhat puzzling, is why at least some patients with organic brain damage and retrograde amnesia are able to acquire new episodic-autobiographic information long-term, while they remain unable to retrieve old information. This issue was discussed in Kroll et al. (1997); alternative retrieval paths were suggested for the newly acquired episodes or the possibility

> *that the storage of memory content is composed according to landmarks (e.g. around an important event such as the Second World War). This could then result in the inability to recall events which occurred prior to the landmark of the brain damage, while not affecting those stored thereafter (Treadway et al., 1992; Hodges and McCarthy, 1993) (Kroll et al., 1997, p. 1396).*

It was also referred to Wolpaw's (1971) hypothesis "that brain damage which is especially traumatic … may disrupt the association between memories due to the 'missing link' (temporofrontal junction area) which is necessary for the organized triggering of (frontal portion) and access to (temporal portion) the engrams" (Kroll et al., 1997, p. 1396). A related hypothesis was put forward by Lucchelli et al. (1995), who suggested the existence of a reversible distortion of "neuronal pattern matrices." And, finally, one can mention the work of Mace, who

argued for involuntary memory chaining in autobiographic memory recall and that events always consolidate in the same conceptual class or network (Mace et al., 2010, 2013; Mace, 2014). His ideas imply that memories may be consciously activated via spreading activations. In contrast, this could mean that, if one or a few critical mnemonic events are suppressed, this can in principle spread to all mnemonic events.

HOW CAN RECOVERY OCCUR?

Even in cases with direct organic amnesia (thalamic stroke), sudden recovery may occur (Lucchelli et al., 1995). In case G.R. of Lucchelli et al. (1995), this occurred when he was in a special, somewhat uncomfortable and unusual situation – similar to a very related one that had occurred some 25 years before. The second case of Lucchelli et al. (1995) – patient M.M., who had had a car accident, but no visible structural brain damage – recovered from his severe autobiographic retrograde amnesia 1 month after the accident, triggered by the fact that he had made the same error while playing tennis as he had done years before. One of our patients (patient D.F.) showed partial memory recovery after being confronted with a slaughter scene 10 months after the onset of her amnesia (Reinhold and Markowitsch, 2009). This slaughter scene involved putting her hands in pig blood; it triggered an event of homicide in China after which she had become amnesic. She still felt guilty that she did not intervene or help and this negative feeling may have prolonged her partial autobiographic amnesia. One of our dissociative amnesic patients was extensively documented in a book entitled *Der Mann, der sein Gedächtnis verlor* [*The Man, who Lost his Memory*] (Kruse, 2010). The journalist Kuno Kruse, who followed his life for more than 5 years, found that certain confrontations with emotion-laden loci and comrades from his past triggered a few memories of his childhood, while others – for an outsider, similar situations – failed to do so. Also listening to music or playing the piano (which the patient had done in childhood) evoked a few broken memories.

These cases demonstrate the importance of the concept of state dependency of memory, originally proposed by Semon in 1904 – together with the concept of "ecphory" (Markowitsch and Staniloiu, in press). Tulving in 1983 reintroduced both concepts and brought them to general attention. Semon also stated in his 1904 monograph that engrams are rarely lost after brain damage – there is just an inability to access (to ecphorize) still fully intact memories. Tulving described ecphory as the process by which retrieval cues interact with stored information so that an image or a representation of the information in question appears. Retrieval cues may occur as other thought associations or as cues from the environment. If the retrieval cues are very different from those existing during encoding, distortions in remembrance may occur – a phenomenon taken up by Sigmund Freud in a number of variations (Breuer and Freud, 1895; Freud, 1901a, b, 1910), and named "encoding specificity principle" in modern literature (Tulving and Thompson, 1973).

Though the encoding specificity principle is in general considered a valid hypothesis, Naime (2002) opposes it in his critique. Naime assumes that it is not so much the match between states of encoding and of retrieval, but cue distinctiveness. He proposes that retrieval is successful if there is a highly distinctive (though possibly only minimal) overlap between the encoding and retrieval conditions ("relative diagnostic match"). Memory is characterized by him as an active process of discrimination. A more recent study by Goh and Lu (2012) tested and supported Naime's proposal. Also Naime's ideas could explain the retrieval impairment of patients with retrograde episodic-autobiographic amnesia: Such patients cannot recognize and discriminate proper cues that would trigger the respective memories. An analogy to this hypothesis might be when a certain word or scene occurring during the daytime triggers the remembrance of a dreamwhich otherwise would have been "forgotten" (or inaccessible).

It seems that only a kind of reconnection between new events similar to the blocked ones triggers memory recovery – by unblocking the pathways to the autobiographic engrams. This phenomenon can be found in very old anecdotic texts in which it is stated that one should cure a shock condition by introducing a similar shock. Some of the present therapeutic approaches for patients with dissociative amnesia may have a related rationale for unblocking memories, namely hypnosis or the sodium amytal abreaction procedure ("truth drug").

WHY IS ANTEROGRADE LEARNING OF AUTOBIOGRAPHIC EPISODES USUALLY UNIMPAIRED OR MUCH LESS IMPAIRED THAN RETROGRADE MEMORY?

After onset of a memory-blocking event patients are in a different setting, compared to their life before – their life is split into an inaccessible personal past

and a new, accessible present. There is frequently the observation that the new present differs emotionally from the past. Many patients – whether with structural brain damage or with dissociative amnesia – live in a very different emotional condition compared to that prior to the amnesia-triggering event (Reinhold and Markowitsch, 2009; Staniloiu et al., 2010; Staniloiu and Markowitsch, 2012a–c). Already before the turn of the 20th century, Janet (1893) and then Breuer and Freud (1895) named one of these altered conditions "*la belle indifférence*"; it describes a flattening of emotions. In a survey based on 11 reports, Stone et al. (2006) found that only 21% of patients with conversion disorder and 29% with organic disease showed *la belle indifférence*. However, there was a considerable variance between studies (0–54% in 356 patients with conversion disorder and 0–60% in 157 patients with organic disease). Whether *la belle indifférence* is indeed rarer than noted in the literature (e.g., Kleist, 1918; Kiersch, 1962; Reinhold and Markowitsch, 2007, 2009; Serra et al., 2007; Pommerenke et al., 2012) is a still open issue. Staniloiu and Markowitsch (2014) wrote that many patients with dissociative amnesia "report feeling distressed by their amnesic syndromes" (p. 229). An observation we made is that patients with retrograde amnesia encode new information likely in an emotionally flat manner (Markowitsch et al., 1993a; Reinhold and Markowitsch, 2007, 2009; Staniloiu and Markowitsch, 2012b; Markowitsch and Staniloiu, 2013) and may show impaired somatic responses to emotional stimuli (Reinhold and Markowitsch, 2009; Tramoni et al., 2009). In patients with dissociative (conversion) amnesia heart rate variability is lower than in healthy participants (Tramoni et al., 2009; van der Kruijs et al., 2014). Furthermore, patients with dissociative amnesia frequently show signs of depression and alexithymia (Markowitsch et al., 1998, 1999c, 2000b; Maldano and Spiegel, 2008; Moriguchi et al., 2009; Staniloiu et al., 2010). All this impairs theory of mind functions and foresight, and reduces the patient to an extended noetic present (Suddendorf et al., 2009) with resignation and lack of concern. And in fact, this is a not uncommon observation in patients with retrograde amnesia: they can and do learn new information, but they do this in a neutral, unengaged manner – they are unable to resonate with their social and biologic environment (Markowitsch, 1998).

It is obvious that there are also patients pretending to be amnesic or exaggerating their deficit – for example, in legal or forensic situations – and that proper assessment has to be performed in order to preclude faking in such cases (Bass and Halligan, 2007; Jenkins et al., 2009; Markowitsch and Staniloiu, 2011c; Boone, 2013). Already in 1943 Lennox wrote that feigned amnesia may accompany both organic and psychogenic amnesia. He suggested that some patients may manifest a combination of three types of amnesia ("pathological" [organic], "psychological," and "feigned"; Lennox, 1943, p. 741). Similarly, Barbarotto et al. later (1996) described a case under the heading "A case of simulated, psychogenic or focal pure retrograde amnesia: Did an entire life become unconscious?" (A similar case with a similar title was published by Weusten et al., 2013.) We had several patients who started with conscious memory suppression or faking of amnesia and apparently ended with true dissociative amnesia. Changes in the brain's circuitry therefore may occur as a consequence of certain patterns of thinking and acting – a phenomenon that may have relations to the concepts of embodiment (Pfeifer and Bongard, 2007; Campbell and Garcia, 2009; Dove, 2011) and extended mind (Clark and Chalmers, 1998; Clark, 2008).

CONCLUSIONS

Aside from cases with major cortical degeneration such as in most forms of dementia, the brain correlates of retrograde amnesia are still unclear. Reasons for this most likely have to do with the uncertainty about how memories – especially episodic-autobiographic episodes – are stored (and retrieved) in the brain. For memory storage, ideas exist which assume distinct storage places, based on findings that some patients showed an inability to retrieve specific categories of information after circumscribed brain damage (e.g., Warrington and Shallice, 1984; Damasio, 1990; De Renzi and Lucchelli, 1994), gnostic units (John, 1975; Quiroga, 2013), grandmother (Gross, 2002), and concept cells (Quiroga et al., 2005, 2008; Quiroga, 2012), statistically distributed (John, 1972), and holistic representations (Pribram, 1971; Deacon, 1989), or a compromise between such ideas (Markowitsch, 1985, 2013; Mesulam, 1990, 2000). This variety of approaches demonstrates an *ignoramus* – we do not know. Similarly, there are at present only speculations on how stored memories are accessed and how access is blocked. This is a continuing issue, especially for dissociative or psychogenic amnesia, though the expression "psychogenic amnesia" can already be found as the headline of a commentary in the *Lancet* in 1935 in which the writer warns of the "incompleteness of purely psychological explanations of amnesia, and the

occasional practical risks of accepting them as final" (Anonymous, 1935).

Regions of the frontotemporal cortex have, however, frequently been associated with retrograde amnesia. They are also most commonly affected in TBI, the etiology most closely associated with retrograde amnesia. Damage to these regions – in addition to memory – also affects social-emotional processing, inhibitory processes, attention, and consciousness (e.g., Feuchtwanger, 1923; Damasio, 1999; Eluvathingal et al., 2006; Fujiwara and Markowitsch, 2006b; LaBar and Cabeza, 2006; Sturm et al., 2006; Schulte-Rüther et al., 2007, 2011; Marinkovic et al., 2011; Vandekerckhove et al., 2014). Furthermore, they are intimately interconnected (e.g., Horel, 1978; Sarter and Markowitsch, 1984; Ebeling and von Cramon, 1992; Kier et al., 2004; Eluvathingal et al., 2006; Diehl et al., 2008; Phan Luan et al., 2009; Staniloiu and Markowitsch, 2012e). It is therefore likely that the complex network of emotion-embedded memory functions and autonoetic consciousness becomes disturbed both after direct brain damage and after major biochemical alterations (Markowitsch, 1996a, b, 1998; O'Brien, 1997; Lupien et al., 2005, 2009). Attentional dysfunctions and processes of increased inhibition may strengthen and maintain the block of information retrieval (or, more generally, of consciously forming representations of events), as delineated in the model depicted in Figure 15.2 of Fujiwara and Markowitsch (2006a).

Another, related model was proposed by R.J. Brown (2004) to account for medically unexplained symptoms. It proposes body-focused attention as psychologic defense. The model assumes "that traumatic events such as physical, sexual, and emotional abuse often lead to the use of body-focused attention as a means of avoiding the affect and cognitive activity associated with experiences of this sort" (p. 806). Also Chadda and Raheja (2002) argue with a narrowed attention in patients with dissociative amnesia. Support for such ideas comes from findings showing a forgetting-related downregulation of neural synchrony mediated by the prefrontal cortex (Hanslmeyer et al., 2012) and from the study of Brand et al. (2009). These authors combined brain glucose PET data from 14 patients with dissociative amnesia in order to detect brain regions with changed activity patterns. They found a significant metabolic reduction in the right ventromedial prefrontal cortex, extending in principle to the right anterior temporal cortex (Fig. 36.4). Their data are in agreement with previous findings demonstrating increased PET activation (radioactive water PET) in individuals retrieving autobiographic episodes from their past (Fink et al., 1996) and with data from patients with damage to the right frontotemporal brain failing to retrieve memories from their personal past (Markowitsch et al., 1993a; Kroll et al., 1997; Levine et al., 1998, 2009).

Further support for the involvement of the prefrontal cortex in memory retrieval comes from a study of Kunii et al. (2012). These authors conducted a sequential cerebral blood flow (CBF) study with SPECT in an ex-convict with dissociative amnesia. They carried out CBF-SPECT measurement during memory retrieval 10, 50, 86, 114, and 146 days after admission, while the patient gradually recovered from his amnesia during this time period. A regions-of-interest analysis revealed a continuous increase in frontal cortex regional CBF during the process of recovery (increased retrieval) and suggested, in the eyes of the authors, that the frontal cortex might be inhibited (less active) during dissociative amnesia. Alternatively, or in addition to this function, a heightened activation could reflect "an active mental defense against unwanted memories of which the patient was not aware due to strong repression" (p. 624). This last remark they made with reference to the findings of Anderson et al. (2004) on neural systems underlying the suppression of unwanted memories.

So, at least with respect to recollecting past personal episodes, it seems that there is evidence for a crucial involvement of the right temporofrontal cortex. What, however, remains to be unraveled are especially the conditions for recovery from retrograde episodic-autobiographic amnesia. From the sparse results available (see above), it seems that even full recovery can occur in less severely brain-damaged patients and in principle in most patients with dissociative amnesia. Supportive or triggering factors for recovery have to be established, especially for more severe cases, in addition to the conventional forms of therapy (McKay and Kopelman, 2009; Staniloiu and Markowitsch, 2014). The neuropathology of retrograde amnesia is still much more a riddle than that of anterograde amnesia. Probably, more subtle forms of brain damage, occurring in a considerable proportion of patients with retrograde compared to anterograde amnesia, have to be investigated (changes in biochemistry, in the neuropil) (Markowitsch and Staniloiu, 2012a). Findings of electric brain stimulation (Doty, 1970; Bancaud et al., 1994) and electroconvulsive therapy (Hihn et al., 2006; Sackeim et al., 2007; Fraser et al., 2008) point in the direction that circuits and association fibers seem to play a particular role in evoking conscious memories.

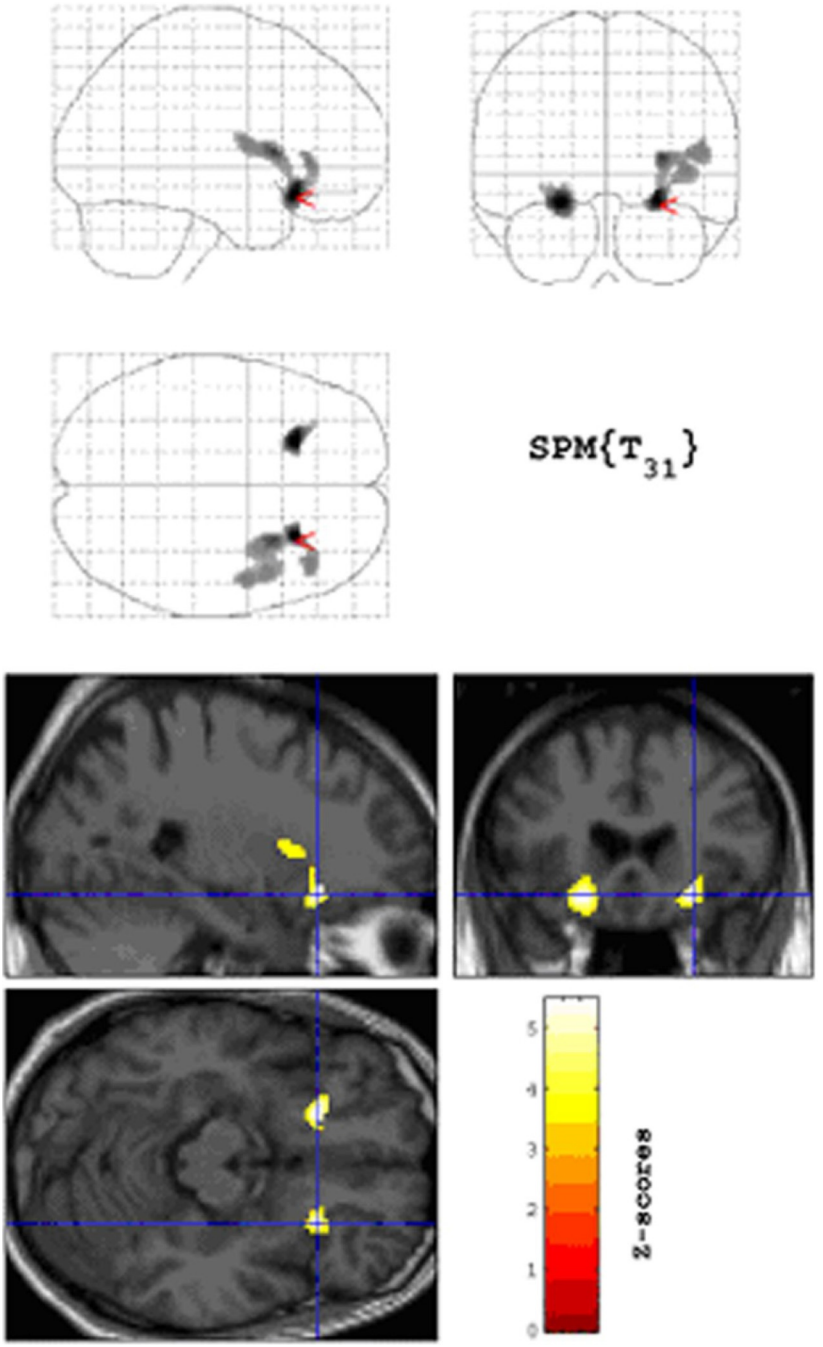

Fig. 36.4. Relative decreases in regional cerebral glucose metabolism in 14 patients with dissociative amnesia relative to 19 control individuals (sagittal, frontal, and horizontal views as "glass brains" and superimposed on magnetic resonance imaging (MRI) sections: MRI template). The blue cross indicates the locus of the only significantly deactivated spot in the right inferolateral prefrontal cortex ($p_{\text{corrected}} < 0.001$, $x = 26$ mm, $y = 24$ mm, $z = -14$ mm). The homologous hypometabolic region within the left inferolateral prefrontal cortex failed to reach significance ($p_{\text{corrected}} < 0.083$, $x = 22$ mm, $y = 24$ mm, $z = -14$ mm). (Reproduced from Brand et al. (2009), Figure 1, with permission of Elsevier.)

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