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Dissociative amnesia: Disproportionate retrograde amnesia, stressful experiences and neurological circumstances

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ABSTRACT

Dissociative amnesias have been reported in neurological episodes mild enough to not cause any visible lesions on morphological examination. Disproportionate retrograde amnesia with or without identity loss happens in the context of psychological trauma (known or not). In metabolic imaging studies, some authors have reported functional alterations, particularly in the bilateral hippocampus, right temporal regions and inferolateral prefrontal cortex, despite normal morphological imaging. To avoid the presumption of an organic, psychogenic or mixed origin for such changes, De Renzi et al. suggested the term 'functional amnesia' to describe the condition. Patients have sometimes recovered during events similar to those preceding the amnesia in either a spectacular fashion or never. Also, in some cases, distraction or sedation may trigger the start of recovery. During psychotherapy, one patient remembered seeing a car on fire when he was a boy, and his amnesia started when his house was on fire. This suggests control by the frontal cortex, with repression blocking amnesic traces in the new emotional and biological context.

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1. Introduction

Dissociative amnesia is characterized by retrieval blockade of episodic autobiographical memories arising in the context of psychological trauma (whether known or not), with no evidence of brain damage on structural imaging [1–4]. Patients experience a disruption of self [5]. Put simply, the common characteristic of all these conditions — regardless of cause or mechanism — is that they are clinical disorders involving disproportionate retrograde amnesia. The definition of dissociative amnesia is given in the 2017 French International Classification of Diseases and Related Health Problems (CIM-10) as "a disorder characterized by a retrospective gap in memory of important personal information, usually of a traumatic or stressful nature; the memory loss far exceeds ordinary forgetfulness and is not the result of substance use or the consequence of a medical condition". The term 'psychogenic amnesia' is considered an approximate synonym. According to the 2013 Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) [6], amnesia can be lacunar or total with or without amnesia of identity.

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2. Disproportionate retrograde amnesia

This rare clinical condition can be described according to its extent as lacunar, massive or total amnesia; its duration as transitory with partial or long-lasting recovery; its profile as either isolated episodic biographical amnesia, global biographical amnesia (episodic and semantic) or biographical amnesia (isolated or global); and by whether it is accompanied by other disorders, such as collective amnesia (specifying the sector involved, such as famous people, public events) and, more rarely, procedural amnesia. The two main symptoms of dissociative amnesia are loss of identity and whole-life amnesia. Through distraction or sedation [7], however, physicians may be able to detect some preservation of memory as the patient's attention is directed elsewhere. The particular severity of a patient's amnesia could reflect the phenomenological representation of human memory that such patients have. Amnesia conversion will then need to include a role for social influence and symbolic amnesia [5].

However, the clinical pattern cannot easily differentiate amnesia patients with or without a mild neurological context. When Kritchevsky et al. [8] studied 10 patients, seven had loss of identity, eight had one or more neurological abnormalities and a further eight had significant premorbid psychiatric histories, including conversion symptoms in two cases. It is important to note that the literature shows wide variability in recovery: when it happens, it usually takes a few days to 'lift', although it may sometimes convert in spectacular fashion. This was the case with three patients, reported by Stracciari et al. [9], all of whom recovered in less than a week. Recoveries have also been reported following situations involving a similar context: patient P.N. [10] recovered after seeing a funeral scene on television (a similar episode had preceded the amnesia); while playing tennis, patient M.M. [11] saw himself playing an earlier match; and patient G.R. [11] re-experienced an earlier instance of anesthesia during subsequent anesthesia.

Nevertheless, a number of patients fail to recover. Patient A.M.N. [2] developed amnesia after discovering a fire, yet did not recover despite remembering a traumatic childhood incident. However, the fact that some patients recover suggests there may be a 'blocking' of access to past memories, probably caused by a disturbance of control phenomena, although the underlying mechanism remains unknown. Motivational and emotional control processes could be blocking the more executive control processes involved in seeking out memories, as it appears these three types of control can be described independently at a neuronal level [12].

The 'lifting' of the disorders experienced by patient F.F., the associations made by the patient and the fact that nothing had been forgotten (ecmnesia) all point towards a control problem and the possibility of resolving it by 'working' on the symptoms or removing the traumatic stress [7,13]. However, disorders may still not disappear when access, convergence or storage systems are destroyed and in cases where traumatic stress can only be overcome by definitive distancing, just as it is possible for neurological disorders to not recover when diffuse lesions disrupt the system of access or destroy memory stores. In addition, the fact that some highly intense levels of oxidative stress can cause neuronal destruction cannot be ignored [1]. All medical disorders, including psychiatric ones, are organic or somatic and, thus, our medical language needs to be updated across the international literature and in clinical practice. If a dichotomy is to be maintained, the terms 'psychiatric disorder' and 'non-psychiatric disorder' are preferable to 'organic disorder' and 'non-organic disorder' [14].

3. Stressful experiences and neurological precipitants

The loss of autobiographical memory may arise after traumatic or stressful experiences. Retrograde amnesia is the result of changes in activation of executive functions [15], and inhibition of memory retrieval has been linked to functional changes [2,16–18]. The term 'functional amnesia', proposed by Markowitsch et al. [2], refers to cases following a neurological condition precipitating even minor illness, with no brain abnormality detected by conventional electrophysiological investigations and high-resolution structural neuroimaging, and/or emotional precipitants that may yet be unknown. Patient A.M.N., in an unclear neurological context (calling the fire brigade because of a fire in his attic) with no suspicion of anoxia, and a psychological context suggesting that a traumatic memory from early childhood may have helped to trigger the condition (during psychotherapy, he remembered seeing a car in flames on the motorway with the driver trapped by the fire, a scene that he suppressed, but which was confirmed as true by his mother), underwent a positron emission tomography (PET) scan, which showed a significant reduction in cerebral blood flow involving the hippocampus, but which was, in fact, more widespread than that.

The term 'functional amnesia', proposed by De Renzi et al. [1], suggests that the diagnosis may be non-organic. The neurological precipitant could be a biological or psychological precipitant ('irritative' symptoms). Dissociative amnesia has been variously qualified as 'hysterical', 'conversive' and 'psychogenic' but, in fact, it corresponds to functional amnesia [17,19]. In 2014, Staniloiu and Markowitsch [20] concluded that ''dissociative amnesia is characterized by functional impairment. Additionally, preliminary data suggest that affected people have an increased and possibly underestimated suicide risk. The prevalence of dissociative amnesia differs substantially across countries and populations. Symptoms and disease course also vary, indicating a possibly heterogeneous disorder. The accompanying clinical features differ across cultural groups. Most dissociative amnesias are retrograde, with memory impairments mainly involving the episodic–autobiographical memory domain''.

While few neurological cases have been reported in the literature, it should be emphasized that most observations involved diffuse pathological conditions, apart from one exceptional case of a lesion in the fasciculus uncinatus in the context of benign traumatic brain injury (TBI) [21]. The main clinical situations associated with pure retrograde amnesia are TBI [8,21–23], stroke [10], colloid cyst [24], meningioma treated with surgery and radiotherapy [25], encephalitis [26–29] and vasculitis [30], and three general

hypotheses have been proposed to explain this type of amnesia: frontal impairment; temporary disruption of the medial temporal lobe; and impairment of the neocortical network [30].

Thus, patient G.R. [11] presented with a left thalamic and capsular stroke, but also had an ''unusual'' psychological context that evoked psychogenic disorders superimposed on the lesion-based neurological condition. The neurological episode in such cases served as a triggering mechanism. The case of M.M. [11] is another of these highly evocative cases. This patient presented with benign trauma and no psychological context of note, yet his presentation and spectacular recovery during a tennis match, when he ''saw himself again'' in an earlier match, is suggestive of such a context [31]. Also noteworthy are the cases of P.A. [32], whose amnesia developed following a poorly documented loss of consciousness and who has never recovered, and S.M. [33], who developed amnesia following a benign TBI and quickly recovered. The present author herself has reported on the case of F.F. [7,13], a 41-year-old married man with two daughters (aged 14 and 10 years) and three step-daughters (aged 33, 30 and 28 years) who, while talking on the phone, fell from his chair. After being found on the floor, he regained consciousness and initially presented with pure retrograde amnesia involving his entire life and a certain amount of collective knowledge. There was a transitory element related to his identity. He was monitored regularly and his amnesia lifted after 9 months. His memory recovery was progressive, with preserved autonoetic awareness and sometimes even instances of ecmnesia. Again, in such a case, the neurological episode acted as a triggering mechanism.

This suggests that certain circumstances (benign TBI [33], a fall and blow to the head while on the phone [7,13], with the onset of amnesia that 'prolongs' the brief loss of consciousness) may either cause patients to develop a coping attitude or simply interrupt the activity without ruling out the possibility that biological phenomena (excitatory neurotransmitters) can predispose to 'blockages' in the system. When F.F. recovered, he could once again remember a personal traumatic memory that had happened several days prior to his amnesia. In some cases, there is a history of childhood trauma, as in the case of A.M.N., who recalled a traumatic scene from his childhood (the burning car) during psychotherapy while going through a particularly stressful period of his life (conflict with his father) [2].

4. What can we learn from changes in functional imaging?

While the absence of any structural lesion clearly does not rule out any microscopic lesions (particularly diffuse axonal lesions in benign trauma), the fact that a change in functional imaging is observed still does not necessarily mean a 'neurological' diagnosis. It is widely accepted that changes in blood flow are observed in a large number of psychiatric conditions, including conversion disorders, and that these images only show a change away from 'normal' functioning of the brain. In 2006, Ghaffar et al. [34] published their observations, using functional MRI (fMRI), in three patients with unilateral sensory loss that matched DSM-IV diagnostic criteria for conversion disorder. These patients had no activation in the contralateral somatosensitive cortex in response to vibrotactile stimulation of the affected side, whereas activation was observed with bilateral stimulation. The authors suggested that double stimulation may serve as a 'distractor' to overcome any inhibition phenomenon.

Recently, the present author published a study highlighting a metabolically dysfunctional cerebral network involving the bilateral medial temporal lobes [4]. Patient N.N. (a rare case of a 'traveller without luggage') showed changes in cerebral blood flow on resting fMRI [16]. The author then used functional imaging to perform further studies that showed the unusual way in which the patient undertook to recover personal episodic events vs controls and which included changes to blood flow in the frontotemporal area. Indeed, other studies have emphasized dysfunction involving these regions [2,7,17] as well as the prefrontal, ventrolateral or orbitofrontal [16,17,35], anterior temporal and posterior/visual neocortical regions, especially in the right hemisphere [36]. These regions all play an important role in generating processes relating to self (frontal areas), personal semantic (anterior temporal) and visual mental imagery (posterior/visual areas). The right prefrontal cortex is critically important for synchronizing emotional and factual components of personal events for their successful retrieval, thereby allowing a sense of self-awareness. Hypometabolism in this region has been reported in one case (C.L.) [35] and recently in 14 patients with dissociative amnesia [17]. The medial temporal lobe is part of the limbic circuitry involved in episodic memory encoding and retrieval. Sellal et al. [33] found right temporal single-photon emission computed tomography (SPECT) abnormalities in one patient, whereas Kopelman [36] demonstrated abnormalities on electroencephalography (EEG) of the right frontal region, but also in the temporal region, in a patient with amnesia and fugue. Such hippocampal hypometabolism was also observed in two out of 14 patients [17]. Functional neuroimaging studies of dissociative amnesia, with prevailing retrograde memory impairments, show changes in the network that subserves autobiographical memory [20].

5. Implicit and explicit attitudes

Several patients have reported difficulty with abilities such as writing, reading, speaking a language, riding a bike, tying shoelaces, using a pen or other common tools, driving and playing the piano [37,38]. A 33-year-old woman with dissociative amnesia presented with whole-life autobiographical amnesia and loss of identity. She forgot how to play the flute, drive, speak English and do her job, and also changed her taste in food (unpublished case). However, she did not modify her lifestyle (values, beliefs, home, friends), and quickly learned how to play the saxophone, paint and sew. In summary, she showed a change in preferences and aims, but the components of her self, which form the bases of her implicit attitudes and internal coherence, did not change.

Thus, dissociative amnesia only modifies autonoetic consciousness and explicit attitudes. Amnesia may, in fact, in some cases favor the performance of highly automatic

behaviors, particularly ambulatory automatism. Indeed, Maldonado and Spiegel [39] reported that certain forms of dissociation, such as suspension of critical thought, might even enhance the performance of coordinated and complex motor acts in athletes. It may be conjectured that the transfer of artistic and manual competence in the above case is a similar phenomenon.

6. Discussion

Amnesia with loss of identity may be compared to a repressive psychological mechanism designed to protect the patient, although this offers no explanation as to why these patterns of focal retrograde amnesia remain rare and why their outcomes vary so much [17,20]. One perspective of dissociation assumes that these alterations of consciousness and encoding arise because of a massive release of hormones in reaction to stress and of neurotransmitters during trauma, thereby leading to extreme levels of activation of the sympathetic nervous system [2].

Dissociative phenomena were identified more than 100 years ago by French psychiatrist Pierre Janet (1859–1947), who proposed that the intense emotion aroused during trauma might be interfering with the assimilation and integration of perceptions, thoughts and experiences. In some cases, neurological circumstances as a triggering mechanism may precede the onset of amnesia [36]. Not infrequently, dissociative amnesia may arise after minor accidents (neurological precipitants) such as mild head injury or loss of consciousness. However, the precise mechanisms underlying the process are as yet unknown. Dissociative amnesia is frequently diagnosed in young adults in their third or fourth decades of life. While such patients are able to acquire and store new long-term memories, there is an impairment (block) of episodic autobiographical memory, whereas impaired procedural memory is rare and usually transient.

The episodic memory system is thought to allow mental time travel through subjective time from the present to both the past and future. For Conway and Pleydell-Pearce [40], episodic memory maintains and supports current aspects of the self while acquiring future goals consistent with goals of the current self and its system of beliefs. Thus, episodic memory may be indispensable for our sense of personal identity and self.

Preservation of the self may be explained by the fact that an inability to retrieve past memory is associated with only explicit loss of self, with no changes in beliefs, values, personality, emotions and character [1,2,7–11,15–19,35]. Indeed, these latter components of self reside in associative processes, which build the basis of what researchers call 'implicit attitudes'. This means that these patients generally know how to behave in social or affective situations, and this integrity of implicit attitudes explains how patients can face their autobiographical history as novel and yet live 'normally' while 'staying themselves' to their family members. Dissociation between implicit and explicit attitudes may lead to the frequently encountered 'la belle indifférence' [41]. Nevertheless, with dissociative amnesia and even in the absence of autonoetic consciousness, patients may still retain their internal coherence of life.

There is a growing trend towards discussing inhibition of memory retrieval in relation to functional changes in right prefrontal or medial temporal lobe activity. Many clinicians believe that patients use dissociation as a defensive coping strategy to protect them in the face of overwhelming horror and traumatic stress [42].

7. Conclusion

The clinical condition of dissociative amnesia can be described in terms of its extent, duration and profile, and whether it is accompanied by other disorders. The presence of an identity-related factor should be specified and, likewise, any circumstances indicative of stress, conversion or history of psychological events (personality, pathology, traumatic stress during childhood…). This approach should simplify the task of clinicians, while providing better help to patients themselves by observing them globally and increasing the future possibility of ultimately understanding the biological and neuronal mechanisms common to all these conditions. Identifying traumatic events (or neurological triggering mechanisms) should also help to improve the quality of patient monitoring. Furthermore, when patient interviews are performed, it is worth remembering that it may sometimes be more useful to not attempt to eliminate those instances of amnesia created by the patient's mental state as a means of self-protection.

Disclosure of interest

The author declares that she has no competing interest.

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