

THE “DEJA-VU” PHENOMENON – FORENSIC AND PSYCHIATRIC IMPLICATIONS

Andrei Scripcaru*, Cristina Furnică, Gabriela Crăciun, Călin Scripcaru

Institute of Legal Medicine, Iași, Romania

Abstract: The “déjà-vu” phenomenon is very frequent in normal individuals but also in some psychiatric diseases [most often in epileptic disease]. The non-pathological “déjà-vu” state may appear spontaneously in individuals without psychiatric symptoms and that is why people couldn’t understand the specific behavioral effects. According to the cognitive psychology, déjà-vu represents any subjective feeling of false familiarity which is given by a present event towards a past one. The new stimulations of “here and now” generate a strong feeling of recognition of objects or events which we perceive as already lived. The déjà-vu state may appear and disappear suddenly, being experienced like a strange feeling or an illusion that our actual experience was lived in an identical way before. It represents a pure subjective experience associated with hard to explain feelings, even if one realizes that they are in that place, situation or state for the first and only time. The déjà-vu state is perceived by several senses, not only by sight but also by hearing, taste, touch or proprioceptive sensations. The result is an extremely detailed perception which may last from one second to a few minutes. Even if forensic consequences are rare, sometimes the déjà-vu state may accompany some psychiatric diseases [epileptic psychosis, schizophrenia], determining the amplification of delirium and the focus on a certain dominant direction which becomes obsessive for the patient.

Keywords: déjà-vu, epilepsy, memory.

INTRODUCTION

The déjà vu phenomenon was mentioned for the first time in 400 AD by Saint Augustine as “falsae memoriae” [25]. Although it was previously mentioned in the literature, medical attention was not focused on the phenomenon until 1844, when it was referred to as “the feeling of preexistence” [24]. The strange feeling of déjà vu is due to the inability to identify in one’s autobiographical memory the currently perceived situation, which contributes to intensifying the sense of conflict between the lack of memory and the feeling of familiarity [23]. In this paper we aim to provide a brief overview of what has been found so far about déjà experiences from neuropsychological, neuroscientific and psychological research, as well as its medico-legal and psychiatric implications.

MATERIALS AND METHODS

Temporal lobe epilepsy is the most well-known neurological condition associated with the déjà vu phenomenon, which also appears in the context of

several psychiatric disorders. Thus, characteristics of the déjà experience that may indicate the substrate of a neurological or psychiatric pathology will be discussed, along with some neuroanatomical and psychological models involved in the genesis of déjà vu [25].

RESULTS

The déjà vu sensation appears as a conjunction of two streams of consciousness: the phenomenological experience of recognizing a current situation, simultaneously with the awareness that this feeling of recognition is inadequate [26]. Probably due to the fact that this sensation gives rise to such contradictory experiences, over time the phenomenon has even been explained as a parapsychological one, in terms of reincarnation, telepathy or clairvoyance [5].

A large number of studies have been conducted attempting to identify the prevalence of déjà vu experiences in the general population and patient groups, but considering the nature of this experience, all studies face problems of patient selection and definition. Between 31% and 96% of “normal”

*Correspondence to: Andrei Scripcaru, Institute of Legal Medicine, Iași, Romania, E-mail: calinscripcaru@yahoo.com

respondents reported experiencing *déjà vu*, and in two direct comparisons of normal subjects with psychiatric or neuropsychiatric patients, *déjà vu* was less common in the normal groups. Younger age, education, and socioeconomic status are thought to be associated with increased rates of *déjà vu*, but no significant differences were found for gender or race [22].

What is *déjà vu*? It is described by the general population as “if I have seen it before”, in reality the area of the phenomenon is much wider. *Déjà vu* can literally mean “already seen,” but also “already heard”, “already met”, “already visited”, and numerous other forms of “already.” It doesn’t mean “I’ve had this experience before and I know exactly when, I recognize it’s happening again”, because in this case the recognition results from an actual familiarity, whereas in *déjà vu* there is this defining sense of the inappropriate familiarity of the present experience [10]

In 1983 Neppe proposed a definition of *déjà vu* as “any subjectively inappropriate impression of familiarity of a present experience with an undefined past” [13]. According to Jessen, *déjà vu* occurs when an adequate sense of recognition of a small part of a scene is inappropriately extended to the scene as a whole [8]. It has been suggested that there may be two qualitatively distinct forms of *déjà vu*: a pathological and a non-pathological form. They differ in the frequency and duration of the experience, in the first case the phenomenon being called reduplicative paramnesia, with gradual onset and long duration, and in the second, with rapid onset and short duration. A longer experience of *déjà vu* suggests an illness, be it neurological or psychological [25]. There are also definitions that differentiate between the experiences of *déjà vu* and *déjà vécu*, *déjà vu* defining “false familiarity” and *déjà vécu* “false memory” [7]. It is assumed that on the basis of the patient’s description of the *déjà vu* experience, the differentiation between these two forms can be achieved, allowing the identification of the underlying disease [3].

A classification into four specific subtypes of *déjà vu* has been proposed, namely: *déjà vu* in temporal lobe epilepsy [qualitatively different from the situation of other epileptic patients or with temporal lobe dysfunction, who show the same *déjà vu* qualities as the “normal” population], psychotic *déjà vu* [identified in a “schizophrenic” population subgroup], *déjà vu* within subjective Psi experiences [in those who, using specific subjectively validated criteria, claimed to have “psychic” experiences, but neither confirmed nor denied the objective validity of their experience], and associative

déjà vu [which in its most frequent form occurs in an “apparently normal” group, where *déjà vu* is associated with perplexity and generally not with a deep degree of intensity].

Neuroanatomy

One area of relative consensus in *déjà vu* research is that of lateralization, with early suggestions that *déjà vu* was primarily a function of the non-dominant temporal lobe for language [11]. At the same time, it was also hypothesized that handedness rather than language dominance appears to be a more consistent predictor of ictal *déjà vu* lateralization, suggesting the value of *déjà vu* as a localization and lateralization feature in epilepsy.

The temporal region appears to be the origin of *déjà vu* in both healthy individuals and individuals with psychiatric and neurological conditions, but the precise mechanism and anatomical basis of this phenomenon still remain unknown. However, what is certain is the involvement of three brain structures: hippocampus, parahippocampal gyrus and temporal neocortex [16]. The most serious of the conditions associated with *déjà vu* is certainly temporal lobe epilepsy, in which prolonged duration and a higher frequency of occurrence have been indicators of said pathophysiology, compared to the otherwise unaffected brain [18].

It was also hypothesized that lateral stimulation caused *déjà vu* by spreading medially from the temporal lobe to the amygdala and hippocampus, studying 16 temporal lobe epilepsy patients with pre-surgically implanted electrodes. They were simultaneously stimulated and measured in the temporal lobe, amygdala and hippocampus. It was found that spontaneously occurring “dream states” always resulted in activation of all three areas, and that this dream state could be experimentally evoked by stimulation of any of them, but stimulation of deep structures was 10 times more likely to evoke such states. Thus, it is suggested that the key to the experience of *déjà vu* lies in the hippocampus and amygdala, with the temporal neocortex playing an important but secondary role [1].

Martin *et al.* [1994] proposed that the brain’s “time succession” processing is conducted in the hemisphere dominant for handedness. Damage to this nondominant hemisphere thus may result in time delays in sending the information to the dominant hemisphere. Currently perceived information could be received twice, so the novel material may be “felt” as previously known when received for the second time [12].

De Nayer proposed a “tape recorder” hypothesis of *déjà vu*. He suggests that, as in a tape recorder, perception is converted into memory by a neurological “recording head” and recall of events is performed by a “reproducing head.” In *déjà vu*, sensory information is somehow simultaneously recorded and reproduced, and therefore simultaneously perceived and remembered [6].

It has also been proposed that the brain’s “time succession” processing is carried out in the dominant hemisphere for handedness. Damage to this non-dominant hemisphere can therefore lead to delays in sending information to the dominant hemisphere. Information currently perceived could be received twice, so that new material can be “felt” as previously known when received a second time [23].

Typical case studies report increased incidence of *déjà vu* experiences in neuropsychiatrically affected individuals [epilepsy, migraines, dementia, sometimes drug use, etc.]. Also, there was reported the case of an adolescent who, following amygdectomy, showed resolution of seizures, *déjà vu*, and olfactory auras that he presented at admission, which led to the hypothesis that amygdala pathology, independent of other structures of the mesial temporal lobe, is capable of generating auras of this nature [16]. Also, in the treatment of hemidystonia with deep brain stimulation [DBS] of the globus pallidus, comparing [by single photon emission CT imaging] DBS leading to *déjà vu* with DBS not leading to *déjà vu*, DBS evoked *déjà vu* was found to be associated with hyperperfusion in right hemisphere mesial temporal structures. Thus, in both cases, the selective involvement of the medial temporal regions in the generation of *déjà vu* is reported [16].

Contemporary memory theory posits that two separable processes are associated with separate neural structures [26]. In short, these two processes are related to two different phenomenological states: recollection and familiarity. In the case of *déjà vu*, the subject experiences an inappropriate sense of familiarity [realizing that it is inappropriate], while for *déjà vécu*, the sensation derives from false feelings of recollection [not necessarily accompanied by awareness that it is inappropriate]. In this experience, instead of a vague sense of familiarity with some material, the subject feels a more complete sensation of information retrieval, including contextual details, a sense of mental time travel, and a sense that he or she knows what is going to happen [14].

Another mechanism proposed to explain the phenomenon of *déjà vécu* is that by which an erroneous

sense of recollection might be experienced for situations that should be interpreted as new [15]. This mechanism is based on two sets of findings: first, that the hippocampus is involved in signaling both recollection and novelty, and second, that, depending on whether the cells primarily encode (during novelty) or retrieve information (during recall), they signal at various mean phases of the hippocampal theta oscillation. According to this mechanism, regions downstream of the hippocampus are disrupted so that regions normally responsive to retrieval become responsive to signals with an average theta phase similar to that associated with the encoding signal. Thus, in *déjà vécu*, novel stimuli that elicit false memories should at some point in the functional network become associated with a dephased theta signal that has a similar mean theta phase to that signaling the recollection.

Temporal lobe epilepsy [TLE] is epilepsy in which seizures begin in the temporal lobe of the brain. Currently, there are two different types of TLE, based on the anatomical division of their points of origin: mesial temporal lobe epilepsy [MTLE] and lateral temporal lobe epilepsy [LTLE]. The first type originates in the medial parts of the temporal lobe [structures included: hippocampus, parahippocampal gyrus, and amygdala], while the second type, as the name suggests, originates in the neocortex of the temporal lobe. Furthermore, mesial temporal lobe epilepsy is the most common type of epilepsy encountered and can be divided into three different subtypes, according to different symptoms: simple partial seizures [SPS], complex partial seizures, and secondary generalized tonic-clonic seizures; simple partial seizures refer to most *déjà* experiences [20].

Pathophysiological hypotheses of *déjà vu* in epilepsy have implicated either the limbic regions of the temporal lobe, the temporal neocortex, or both. A new hypothesis of the corticolimbic network has emerged, suggesting that *déjà vu* may result from an abnormal synchronization between the rhinal cortices and the hippocampus. Thus, it was studied the role of the perirhinal and entorhinal cortices in *déjà vu* in patients with epilepsy, and analyzed the symptoms evoked by direct electrical stimulation of the above-mentioned regions in comparison with stimulation of the amygdala and hippocampus, and the results demonstrated that stimulation of the perirhinal and entorhinal cortices produced *déjà vu* responses more often, the entorhinal cortex being the most sensitive site. Thus, it was pointed out that the findings of previous studies reporting *déjà vu* after stimulation of the amygdala or hippocampus could be explained by evoking *déjà vu* by spreading

the discharge from those regions to the adjacent rhinal cortex [2].

On the other hand, it appears that only amygdala pathology may be involved in pathological déjà vu [along with olfactory auras], as described in the case of an adolescent who presented with seizures and déjà vu, as well as olfactory auras that resolved following amygdalectomy [9].

Cognitive explanations

Cognitive explanations involve processes that produce a sense of familiarity towards a perceived situation, the source of which is not accessible to memory or consciousness. It has been suggested [10] that déjà vu may occur when “a person glancing hastily across a street, preparing to cross it, is momentarily distracted by the contents of a shop window. Then, when crossing the street, the person glances again and experiences the false recognition as the feeling of having previously crossed the same street – a feeling of déjà vu”. This type of explanation involves a stimulus being perceived twice and an inattention mechanism that makes the person unaware of it.

Psychiatric implications

Déjà vu is a frequently occurring phenomenon in the general population, often being associated with neurological and psychiatric diseases, but also being compatible with neuropsychiatric normality. [22]

The meaning of déjà vu manifestations in psychiatry is not clearly established, but it is believed to be a symptom of many major psychiatric disorders. It is intuitively reasonable that déjà-vu is a lesser manifestation of the phenomena observed in frank psychiatric illness [25]. For example, states of anxiety and panic can result in feelings of impending doom; depression can cause distorted perception of time; depersonalization and derealization can cause disturbances of familiarity and sensory perception; psychosis could produce hallucinations or even delusions of precognition. The main discriminator in this setting is patient’s perception. Impaired reality testing, intrusiveness, and the incorporation of déjà vu into a delusional system may suggest psychopathology. Duration is also important: when déjà vu is instantaneous and self-terminating, it is less likely to be due to psychopathology than if it is prolonged and cannot be terminated [19].

Déjà vu is defined by the recognition of a situation at the same time as the awareness that this recognition is inadequate. Although some forms of

déjà vu resolve in favor of inadequate recognition and thus result in potential behavioral consequences, most experiences of déjà vu resolve in favor of awareness that the sensation of recognition is inadequate [4].

For a long time, clinical occurrences of déjà vu were often described interchangeably with depersonalization phenomena. However, the basic cognitive neuropsychiatric concept, dissociation, and the experience of déjà vu do not appear to be related in healthy populations. Also, anxiety disorder patients [with or without symptoms of derealization and depersonalization] experienced déjà vécu and déjà vu to equal degrees: between ½ and ½ of all patients studied [22]. In conclusion, déjà-vu may not be seen as a significant symptom in psychiatry, although the response to it by patients with schizophrenia may be more pronounced, and certain groups tend to experience it more often than healthy groups [patients with derealization/depersonalization, anxiety disorders]. Since there is a neurobiological model of depersonalization, invoking the same temporal limbic networks reviewed in the epilepsy section above, it seems that exploring the state of déjà in depersonalization might be a useful avenue for future research.

Medication

An element that could lead us closer to discovering the origins of the déjà vu phenomena could be the study of the effects of drugs and psychotropic drugs that are described as being associated with déjà vu. For example, *Salvia divinorum* was reported as a possible cause of déjà vu in a young man with a history of *Salvia* smoking [17], while Taiminen and Jääskeläinen reported repeated déjà vu experiences in a healthy 39-year-old man within 24 hours of co-initiation of amantadine-phenylpropanolamine treatment against influenza [21].

CONCLUSIONS

A diagnosis of epilepsy is not known to be associated with an increase in violent acts, even though it can be associated with complex behaviors outside ordinary conscious control, which may sometimes result in violence. Violence in people with epilepsy is usually due to other risk factors, and it is probably the association with these that was responsible for the previous view that epilepsy itself was a risk factor for violence. On the other hand, there is a direct association between a diagnosis of schizophrenia or schizoaffective disorder and violence, demonstrated in various studies.

The risk for violence is increased up to four times, and the association appears to depend on the presence of acute psychotic symptoms. In both cases, the violent acts may be a result of confusion, panic or fear caused by their symptoms, among which déjà vu could be counted.

We suggest the need for more experimentation and a more cautious interpretation of research results, especially since many techniques used to explore déjà vu are in early stages of development [16].

Conflict of interest

The authors declare that they have no conflict of interest.

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