Déjà vu in neurology

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Abstract

The significance of déjà vu is widely recognised in the context of temporal lobe epilepsy, and enquiry about déjà vu is frequently made in the clinical assessment of patients with possible epilepsy. Déjà vu has also been associated with several psychiatric disorders. The historical context of current understanding of déjà vu is discussed. The literature reveals déjà vu to be a common phenomenon consistent with normality. Several authors have suggested the existence of a “pathological” form of déjà vu that differs, qualitatively or quantitatively, from “non-pathological” déjà vu. The features of déjà vu suggesting neurological or psychiatric pathology are discussed. Several neuroanatomical and psychological models of the déjà vu experience are highlighted, implicating the perceptual, mnemonic and affective regions of the lateral temporal cortex, hippocampus and amygdala in the genesis of déjà vu. A possible genetic basis for a neurochemical model of déjà vu is discussed. Clinical approaches to the patient presenting with possible déjà vu are proposed.

Introduction

To those that have experienced it first hand, déjà vu is a unique and unforgettable experience. To those that have not, its description may seem impenetrable or abnormal. It has eluded explanation and fascinated neurologists, psychologists, philosophers and the public for centuries. This article discusses a widely accepted definition of déjà vu; the history of its study; explanations of déjà vu from the fields of parapsychology, psychology, psychodynamics neuroanatomy and molecular genetics; and its clinical significance in terms of incidence, neuropathology and assessment. To a recent review by Warren- Gash and Zeman, it adds detail of theories of déjà vu and recent neuroanatomical and genetic work.

Definition

V M Neppe proposed a definition of déjà vu in 1983 as “any subjectively inappropriate impression of familiarity of a present experience with an undefined past” [39]. The definition is precisely worded and provides useful insights into the phenomenon. The word “any” is intended to convey aetiological neutrality, implying that the experience need not originate from any particular pathological entity, or indeed any cause at all. The “subjectively inappropriate” nature of déjà vu is critical to its understanding, as it implies insight into the unusual nature of the experience. The subject simultaneously seems to recognise a situation, yet knows that recognition to be impossible.Taking this further, the definition implies (though does not state) that the subject will try to explain the sense of familiarity and struggle to pinpoint its source but, frustratingly, cannot do so.

The term “impression” is a deliberately all-encompassing term for the experience that reinforces its holistic nature. Déjà vu is not confined to affect, thought process, perceptions or cognition but involves all of these aspects. Specifically, it is not confined to the visual domain. The alternative “deja vécu” has been suggested [39] as being less biased towards the visual; but the term “déjà vu” has such an established place in the literature (scientific and otherwise), as well as in colloquial

parlance, that there is little to be gained from changing it. An extended catalogue of “déjà” experiences – such as entendu (heard), pensé (thought), gouté (tasted) and rencontré (met) [39],while useful descriptively, does little to clarify matters of definition.

“Familiarity” is the cornerstone of déjà vu. Again, the term used is inclusive, encompassing both recognition – the scene seems literally to have been “seen before”– and acquaintance – which suggests a more intimate familiarity with every detail of the scene. Here, Neppe implies the tantalising feeling that the subject can predict what will happen next – despite which, such apparent prescience is of course misguided. Finally, the definition relates the experience to an “undefined past” – or perhaps more definitively, a nonexistent past. This hints at a subject’s often frantic and frustrating efforts to pinpoint the moment in question [47].

True déjà vu is to be distinguished from a number of similar phenomena that may mimic or be falsely described as déjà vu. Jamais vu is an inappropriate unfamiliarity with a situation that has been encountered before – the converse of déjà vu. Flashbacks include visual and auditory illusions and hallucinations, and give the sense that the subject has actually been transported to the midst of a prior experience. Depersonalisation is a dissociative phenomenon involving a sense of detachment from the self [2]. Colloquial déjà vu refers to the incorrect description of a genuinely recurring or unwanted phenomenon as evoking a sense of déjà vu [50]. The dreamy state is an experimental surrogate for déjà vu in epileptic patients: induced by extrinsic brain stimulation, it shares many features of déjà vu but with clouding of consciousness [4, 36].

Historical perspective

Déjà vu was first referred to by St Augustine in c 400 AD as “falsae memoriae”. Despite earlier appearances in literature, medical attention was not refocused on the phenomenon until 1844 when the psychiatrist Arthur Ladbroke Wigan referred to it as “the sentiment of pre-existence” [52]. The term “déjà vu” was coined by FL Arnaud in 1896, in an attempt to reflect its aetiological uncertainty (unlike other terms such as “fausse mémoire”) [3]. Despite erroneously implying that the experience is limited to visual perception, the term has become widely accepted and has passed into common parlance [47]. A number of subjective accounts of déjà vu are found in prose and poetry, including the writings of Sir Walter Scott [45], Charles Dickens [11],Leo Tolstoy [49],Marcel Proust [43] and Thomas Hardy [23].

Perhaps the most insightful account, touching on the subject’s attempt to predict and control the experience, can be found in Catch-22 by Joseph Heller: For a few precarious seconds, the chaplain tingled with a weird, occult sensation of having experienced the identical situation before in some prior time or existence. He endeavoured to trap and nourish the impression in order to predict, and perhaps even control, what incident would occur next, but the afflatus melted away, as he had known beforehand it would [25].

Explanatory theories

The question of what causes déjà vu is one that has intrigued both scientists and the public alike for many years. As an unusual, ephemeral and diffuse phenomenon, déjà vu lends itself to imaginative speculation but is difficult to define experimentally and study meticulously. Consequently, many of the theories of déjà vu, while interesting and persuasive, lack any foundation in evidence. More recently, plausible

neuroanatomical models of déjà vu based on neurosurgical and radiological work have been proposed, and molecular genetic findings in epilepsy may herald a neurochemical basis for the phenomenon.

Parapsychology

Parapsychological theories are mentioned here purely for interest. Such theories include: the suggestion that déjà vu arises from past life memories through reincarnation [8]; déjà vu as evidence of telepathy [31]; and déjà vu as astral transportation [6].

Psychology

Hughlings Jackson coined the term “mental diplopia” to describe the phenomenon of déjà vu in 1888 [27] and several theories evoke this notion of splitting of the brain in déjà vu.Wigan, Jensen and Maudsley proposed that the two cerebral hemispheres function separately but synchronously and suggest that déjà vu arises as a result of a loss of synchronicity between the two parts [28, 32, 52]. Myers proposed that each hemisphere contains a subliminal or supraliminal “self” and déjà vu 3 arises as a result of double perception of a scene by both selves simultaneously [37]. According to Jessen, déjà vu occurs when an appropriate sense of recognition of a small part of a scene is inappropriately extended to the scene as a whole [29]. Lalande and Berndt-Larsson suggested that déjà vu is a primary disturbance of time perception, such that events occurring a moment ago are interpreted as having been seen long before [5, 31]. This is in keeping with the suggestion of an association between déjà vu and psychiatric disorders involving distorted time perception [39].

Gestalt psychology proposes that perceptions and the affect they generate are organised by the brain into object- affect entities. According to this theory, déjà vu results when an object is encountered that stimulates a given affect. That affect inappropriately causes recall of a dissimilar object, resulting in an inappropriate feeling of familiarity [46]. De Nayer proposes a “tape recorder” hypothesis of déjà vu. He suggests that, as in a tape recorder, perception is converted to memory by a neurological “recording head”, and memory recall is carried out by a “reproducing head”. In déjà vu, sensory information is somehow simultaneously recorded and reproduced, and therefore perceived and remembered simultaneously [10].

Psychodynamics

Freud proposed that déjà vu is triggered by perception of a situation with some similarity to a suppressed fantasy, causing the fantasy to be awakened as a wish to improve the present situation. Thus, déjà vu is a manifestation of a wish for the turning back of time [14]. Oberndorf agreed that déjà vu is a defence mechanism and suggested that it is a psychic means of reassurance in situations of adversity: “you went through all this once before and came out well. The same will happen this time.” He proposed that déjà vu would be better called “encore vu”, meaning “seen again” rather than “seen before”[40]. His theory is consistent with the finding that some subjects find déjà vu pleasurable [42]. Finally, some psychoanalysts have suggested that déjà vu may be a form of waking dream, or a daytime residue of dreams. In support of this Zuger claims,based on small-scale studies, that some people neither dream nor experience déjà vu [54]. Freud went so far as to suggest that the source of many déjà vu experiences was “the genitals of the dreamer’s mother; there is indeed no other place about which one can assert with such conviction that one has been there before” [13].

Neuroanatomy

Over a century ago Jackson highlighted the association between temporal lobe epilepsy, with associated “mental diplopia” phenomena, and neuroanatomical malformations of the temporal lobe [27]. In 1959, Mullan and Penfield performed electrical stimulation and intracranial EEG recording during temporal lobe resection in epileptic patients, and elicited a “dreamy state” akin to déjà vu [36]. In 1978,Halgren obtained similar results by stimulating the deeper structures of the hippocampus and floor of the amygdala but could not reproducibly do so in the non-diseased hemisphere [22]. In 1982, Gloor and colleagues [18] used stereotactically implanted depth electrodes (consisting of long, thin electrode strands) to monitor “experiential phenomena” in epileptic patients, and sought to reproduce them by electrical stimulation. Déjà vu was experienced in 4 of 35 patients studied, though their neuroanatomical conclusions were based on the much broader pooled findings from all experiential phenomena, including such diverse entities as auditory and visual hallucinations, flashbacks, fear and thirst.

Like Halgren, Gloor and colleagues emphasised the importance of limbic rather than temporal neocortical structures (Fig. 1b), declaring that “unless limbic structures are activated . . . experiential phenomena do not occur”. Limbic activation, they propose, “may be essential for bringing to a conscious level percepts elaborated by the temporal cortex”. Though the majority of experiental phenomena observed were associated with activation of all four areas studied (amygdala, hippocampus, parahippocampal gyrus and temporal neocortex), they report 37 instances where the temporal neocortex was not activated. The authors further posit an anteroposterior gradient of excitability within the limbic system, the amygdala being most easily stimulated to generate experiential phenomena, followed decrementally by the hippocampus and parahippocampal gyrus. However, the authors admit,“ with our approach only a relatively narrow sector of neocortex was accessible to stimulation and recording”, which may have led to underestimation of neocortical involvement. Moreover, they do report one instance of déjà vu produced by temporal neocortical stimulation without electrical spread to the deeper structures [18].

Bancaud and colleagues in 1994 attempted to synthesise the theories of Mullan, Penfield and Halgren and hypothesised that lateral stimulation of the kind studied by Mullan and Penfield caused déjà vu through medial spread from the temporal lobe to the amygdala and hippocampus. They studied sixteen temporal lobe epilepsy patients with pre-surgical implanted electrodes and simultaneously stimulated and measured from the temporal lobe, amygdala and hippocampus. They found that spontaneously occurring “dreamy states” always resulted in activation of all three areas – and that the dreamy state could be experimentally evoked by stimulating any one of the three – but that stimulation of the deeper structures was ten times more likely to evoke such states. Bancaud suggests that the hippocampus and amygdala are thus key to the déjà vu experience, with the temporal neocortex playing a secondary but important role [4]. Bancaud’s theory is that a neural network in the association cortex and limbic areas encodes the holistic experience of an event. Perceptual information is encoded by the temporal neocortex and stored in the hippocampus, with emotional content added by amygdala (Fig. 1a).

According to this theory, the dreamy state, and by extension déjà vu, is caused by inappropriate activation of deep memory structures by superficial sensory structures (Fig. 1c). Effectively, this amounts to a “neuroanatomical tape recorder” model of déjà vu [4]. Returning to the topic of experiential phenomena in temporal lobe epilepsy in 1990, Gloor [16] maintained the importance, suggested by his earlier work [18], of limbic rather than neocortical structures, but incorporated more recent ideas about distributed parallel cortical networks [19, 20] into a theory of how such

events are triggered in epilepsy. Like Bancaud, he suggested that a neural network throughout the cortex and limbic system encodes an experience and is specific for it.

No single area or cell is indispensable in such a network, and the order of activation is less important than its spatial pattern. Stimulation of any part of the network can result in activation of the whole, and thus result in the evocation of a complete experience. He did not posit a specific neuroanatomical substrate for déjà vu, but his theories on the key role of the limbic system and the importance of parallel neural networks in encoding and evoking experiential phenomena,are complementary to Bancaud's tripartite neuroanatomical model of déjà vu [4] (Fig. 1c).

One area of relative consensus in déjà vu research is that of lateralisation. From their early work, Mullan and Penfield suggested that déjà vu was predominantly a function of the temporal lobe non-dominant for language [36]. In a report of a patient with ictal déjà vu and seizures of right hippocampal origin, Gloor concluded that déjà vu, if strictly defined, is sufficient to localise an epileptogenic focus to the right temporal lobe [17]. Questioning the neuroanatomical value of stimulation studies, Weinand and colleagues [51] used subdural strip electrocorticographic monitoring in 8 epileptic patients with preictal déjà vu. 6 were right-handed and 2 left-handed. Using intracarotid amytal testing, they established that all 8 patients were left hemisphere dominant for language. In all 8, seizures arose in the hemisphere non-dominant for handedness (that is, the hemisphere ipsilateral to the dominant hand). They concluded that "handedness rather than language dominance appears to be a more consistent predictor of ictal déjà vu lateralisation"and suggest the value of déjà vu as a localising and lateralising characteristic in epilepsy [51]. This contrasts sharply with the failure of Gloor and colleagues to find lateralising features when they examined "experiential phenomena" as a whole – including those in the visual and auditory domains and jamais vu [18].

Déjà vu is apparently exceptional in respect of its lateralising power. In 1999 Adachi et al. used positron emission tomography to study the functional anatomy of the déjà vu experience in 31 patients with temporal lobe epilepsy, with and without déjà vu as an ictal feature. In the patients with déjà vu, they found significantly reduced glucose metabolism in the mesial temporal and parietal cortex. They concluded that temporal lobe dysfunction is necessary but not sufficient for the generation of déjà vu, but that the presence of déjà vu in temporal lobe epilepsy was of no lateralising value [1]. Though, unlike the previous neuroanatomical work [4, 22, 36], this study has the advantage of being non-invasive, the recordings were not made during episodes of déjà vu.

Molecular genetics

Autosomal dominant lateral temporal epilepsy (ADLTE), also known as autosomal dominant partial epilepsy with auditory features, is a focal epilepsy syndrome first described in 1995 [41]. It usually begins in the early twenties, is relatively mild, and is characterised by recurrent seizures with aura consistent with temporal lobe onset. Auditory hallucinations predominate, but visual, olfactory and gustatory symptoms have been reported [53]. Though its presence is by no means invariable, déjà vu has been reported as a prominent feature in several cases [15, 53] and may be the sole ictal feature [9]. The syndrome demonstrates autosomal dominant inheritance with high penetrance [41]. The causative gene has been identified as LGI1/epitempin, mapped to chromosomal region 10q24 [12, 30, 35]. Mutations in this gene are invariably found in affected individuals, while no mutations were found in 123 unaffected controls [30]. The function of LGI1 is unknown, but it is expressed throughout the human brain in neuronal tissue, in what appears to be a highly

regulated manner,with three different protein products expressed differentially between cells and brain regions. A role in cell-cell communication has been proposed for the gene products, possibly as a regulator of cell migration or transmembrane receptor or channel [30, 34, 35]. LGI1 is certainly not a “gene for déjà vu”: not all those with LGI1 mutations experience ictal déjà vu, and the gene is apparently not mutated in normal individuals, some of whom may be expected to experience déjà vu (see Incidence below). However, given the association between ADTLE and preictal déjà vu, study of the gene and its role in brain development and function has the potential to shed light on the genetic and molecular basis of déjà vu. LGI1 demonstrates locus heterogeneity – that is, different mutations may produce different clinical syndromes [34]. Thus, the gene may be expressed or responded differently to in individuals who experience déjà vu. Further study of the structure and function of the LGI1 gene and its products in individuals who experience déjà vu, with and without epilepsy, is required to move towards a neurochemical explanation for déjà vu.

Clinical significance

Déjà vu is common in the general population. It can be associated with neurological and psychiatric illness but is compatible with neuropsychiatric normality [7, 21, 24, 26, 33, 38, 39].

Incidence

Numerous studies have attempted to discover the prevalence of déjà vu experiences in the general population and patient groups. Given the nature of the déjà vu experience, all such studies are faced with problems of patient selection and definition. Between 31% and 96% of “normal” respondents reported having experienced déjà vu [7, 24, 26, 33, 38]. In two direct comparisons of normal subjects with psychiatric or “neuropsychiatric”patients, déjà vu was less frequent in the normal groups (51 % vs 65 % [21] and 68% vs 73 % [39]).No significant differences have been found in gender or race, but there is some suggestion that younger age, education and socio-economic status are associated with increased rates of déjà vu [7, 21, 24, 26, 33, 38, 39].

Déjà vu in neuropathology

Given that déjà vu is anecdotally linked to pathological entities but consistent with normality, it is clear that it is not sufficient simply to ask a patient “Do you experience déjà vu?” when suspecting neurological or psychiatric pathology. It has been suggested that there may be two qualitatively distinct forms of déjà vu: a nonpathological form (referred to as déjà éprouvé, with rapid onset, short duration and full insight) and a pathological form (called reduplicative paramnesia, with gradual onset, long duration and impaired insight).

Distinguishing between these two forms is claimed to enable identification of underlying disease based on the patient’s description of their déjà vu experience [5]. Other authors hold that the differences between déjà vu in normality and pathology are qualitative and that scrutinising experiences of déjà vu cannot be used to rule out neuropathology [3]. Disease states associated with déjà vu, they suggest, should be recognised by the associated features of the experience – “the company it keeps” [39]. As first reported by Jackson, déjà vu in temporal lobe epilepsy is often distinguishable by its associated features. Jackson reported hallucinations; a “voluminous mental state” and “mental diplopia”, by which he probably meant dissociative phenomena; epigastric phenomena (the unpleasant sensation of rising or falling); fear; automatisms and, of course, seizures [27]. The significance of déjà

vu in psychiatry is less clearly established but it is thought to be a manifestation of many major psychiatric disorders [44]. It is intuitively reasonable that déjà vu may be a lesser manifestation of phenomena seen in frank psychiatric disease. For example, anxiety and panic states may cause feelings of impending disaster; depression can cause distorted time perception; depersonalisation and derealisation result in disturbances of familiarity and sensory perception; psychosis may cause hallucinations or even delusions of precognition. The key discriminant in this setting is the patient's insight. Impaired reality testing, intrusiveness and incorporation of déjà vu into a delusional system may suggest psychopathology. Duration is also of importance: 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 [47].

Assessment of déjà vu

Sno and colleagues have suggested a detailed and rigorous Inventory for Déjà vu Experiences Assessment [48]. In practice, an informal system of triage for the further evaluation of patients complaining of déjà vu is perhaps more useful. Warren-Gash and Zeman have proposed such a system. The first step is to establish that the patient is experiencing true déjà vu rather than similar phenomena such as depersonalisation, flashbacks or colloquial déjà vu. Next, the patient is placed into one of three categories according to the features associated with the déjà vu. If the experience is occasional, tran6 sient or isolated, the déjà vu can safely be interpreted as "probably normal". If there is evidence of depression, anxiety or psychosis, a psychiatric line of enquiry should be followed. If the experience is frequent, prolonged, associated with physical sensations or automatisms, and if there are seizures, the likely explanation is temporal lobe epilepsy [50].

Conclusion

Déjà vu is a common phenomenon consistent with both neuropsychiatric normality and pathology. Numerous theories have been suggested as to the cause of déjà vu; and a neuroanatomical substrate has been suggested, but all neuroanatomical studies to date have been carried out in patients with abnormal brains who may not have been experiencing true déjà vu. Study of the molecular genetics of autosomal dominant lateral temporal epilepsy shows promise for the elucidation of a neurochemical basis of déjà vu. In patients with frequent déjà vu, pathology is suggested by both qualitative and quantitative findings: a detailed history of exactly what the patient means by déjà vu is essential, and the features associated with déjà vu are the key to further elucidation.

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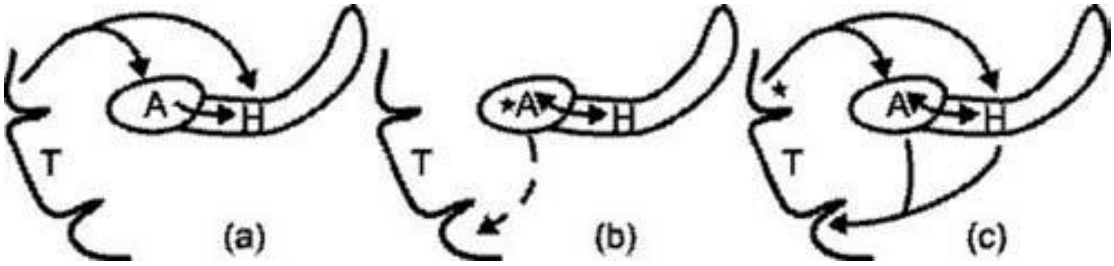

Fig. 1 Simplified schematic diagram of the neuroanatomical models of déjà vu. (a) Bancaud's theory of memory storage; (b) Gloor's and Halgren's early models of déjà vu, with amygdala pivotal, and neocortical activation relatively unimportant; (c) Neural network model incorporating Gloor's and Bancaud's theories, with mutual activation of limbic and neocortical areas. A amygdala; H hippocampus; T temporal neocortex, asterisk indicates proposed origin of activation in déjà vu [4, 16, 18, 22] 5