Déjà Vu: Possible Parahippocampal Mechanisms

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Déjà vu experiences are common in normal subjects. In addition, they are established symptoms of temporal lobe seizures. The author argues that the phenomenon is the result of faulty and isolated activity of a recognition memory system that consists of the parahippocampal gyrus and its neocortical connections. This memory system is responsible for judgments of familiarity. The result is that a momentary perceived scene is given the characteristics of familiarity that normally accompany a conscious recollection. The normal functioning of other brain structures involved in memory retrieval—the prefrontal cortex and the hippocampus proper—leads to the perplexing phenomenological quality of déjà vu. The hypothesis accounts for many characteristics of déjà vu in healthy subjects and is well fitting with experimental findings in patients with epilepsy.

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Most people at some times experience déjà vu (DV). In a questionnaire study about paroxysmal psychic phenomena in a student population,[1] only 10% of the subjects denied that they ever had experienced DV. In the same study, 56% of participants reported that they had had DV experiences in the preceding month. Although DV experiences have no behavioral counterpart, most people seem to agree on their phenomenological characteristics, and the ubiquity of the phenomenon is reflected in numerous descriptions in poetry and fiction.[2] In addition, the DV experience has long been known as a seizure manifestation,[3] and it is an established symptom—usually in association with other symptoms—of partial seizures of temporal lobe origin.[4]

The issue of DV has most often been treated at a psychodynamic level,[5,6] and only few attempts have been made to explain DV at the level of brain–behavior relationship. One strictly somatic hypothesis was proposed by Efron,[7] who argued that material pertinent to time perception is received by the nondominant hemisphere and transferred to the hemisphere dominant for language. Functional or anatomical nondominant temporal lobe damage may delay transfer of information and might cause the left dominant hemisphere to receive the information twice, once directly and once delayed via the right hemisphere. Newly perceived material thus

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is rendered familiar by virtue of the near-simultaneous double presentation. Although ingenious, this idea does not seem to have received much independent support. Moreover, it is not clear why this mechanism should not lead to a feeling of having experienced a situation just before, and not somewhere in an undefined past.

Using holography as an analogy for memory processes, Sno and Linszen[5] have emphasized that DV experiences should not be thought of as pure memory or pure perceptive disturbances, but as a result of a disturbed interaction *between* memory and perception processes.

The results of electrophysiological studies in patients with epilepsy, together with recent advances in our knowledge about the functional architecture of memory processes, give us the opportunity to set up a more specific hypothesis about the cognitive mechanism of DV.

THE PHENOMENON TO BE EXPLAINED

DV experiences are subjectively inappropriate impressions of familiarity of the present with an undefined past.[8] They are not necessarily connected with significant events or deep emotion. No differences between the sexes have been found, but DV seems to be more frequent in young subjects,[2] and it can be triggered by drowsiness or fatigue.[9]

DV experiences are not vague feelings; reports typically include the term “exactly” or “to every last detail.”[2] We even use the idiom “it’s like a déjà vu” for a real remembrance, when we want to express its special vividness. The scene or episode appears to be not only exactly the same as what happened before, but also to be perceived in the same manner, from the same point of view and in the same state of mind, as when seemingly originally encountered. DV experiences have been described as photographic copies of an alleged experience in the past.[5] On the other hand, the immediate feeling of inappropriateness of the experience sharply distinguishes the phenomenon from hallucinations and from “false memories.”[10]

Typically no context, and especially no temporal context, can be retrieved.[11] It is this characteristic feature of DV experiences that has led many laypersons to interpret them as reminiscences from a former life.[12]

From exploration of DV as a seizure manifestation in temporal lobe epilepsy and after electrical brain stimulation, it is known that temporal lobe structures are heavily involved in DV.[13–16] Whereas earlier studies had emphasized the role of neocortical temporal stimulation for the generation of experimental phenomena,[13] later workers have found a preponderance of mesiotemporal limbic structures.[14,17] Furthermore, it has been suggested that DV experiences are reported only when both mesiotemporal and neocortical temporal areas are involved.[16]

In epilepsy research, however, the phenomenon of DV is usually discussed in a broader context—termed by Jackson “dreamy state”[3]—that does not include DV alone, but scenic,[16] auditory, and visual hallucinations[13] as well. Unlike hallucinations, typical DV as experienced by normal subjects is not accompanied by impaired reality testing. So the specific substrate for DV in its narrower sense, which has also been called “forme légère” or “minor form,”[18] has not been focused on.

THE ARCHITECTURE OF MEMORY RETRIEVAL

The importance of the mesial temporal lobe, and especially the hippocampus, for episodic memory processes has become firmly established, but there is agreement that the mesiotemporal memory system is not important for all types of memory.[19] Mesiotemporal memory structures do not seem to have an important role in the learning of skills,[20] in unconscious priming effects,[21] or in assessing knowledge that is not bound to a specific situation (semantic memory).[22,23] However, the mesiotemporal memory system is especially important when encoding or retrieval concerns autobiographical or episodic information.

The mesiotemporal memory system is not homogeneous. Not only the hippocampus proper, but also the entorhinal cortex and the neighboring perirhinal and parahippocampal cortices are important for memory processes. Moreover, the different structures seem to be important for different aspects or even subtypes of episodic memory. The perirhinal cortex receives strong projections from visual areas important for object recognition[24] and might therefore be highly important for visual memory for objects. The parahippocampal cortex gets strong input from regions of the parietal cortex that are important for visuospatial processing,[25] and it has been demonstrated in animal studies that the parahippocampal gyrus is involved in spatial aspects of memory.[19] In humans as well, lesion studies in neurological patients[26,27] and functional imaging studies[28] have shown that the parahippocampal gyrus is especially relevant for memory for the location of objects.

Specialization among the structures might not be restricted to the type of material to be encoded; it could reflect a more basic differentiation between different memory systems. In animal studies it has been shown that although learning and retrieval can readily be accomplished by the parahippocampal cortex alone, the hippocampus proper is needed to make these associations flexible, hence to make them applicable under changing conditions and to combine information acquired under different learning situations.[29] It seems that while the hippocampus might be needed for a deliberative remembrance of previous episodes, parahippocampal activation, as Gabrieli and colleagues have noted, "could reflect other memory processes that distinguish between familiar and unfamiliar stimuli."[30]

This view has been expressed more explicitly by Aggleton and Brown,[31] who differentiated between two memory systems partly situated in the mesial temporal lobe. The first, including the hippocampus, enables recall and conscious recollection of contextual elements. The second, which includes the parahippocampal cortex, is important for familiarity judgments or the "feeling of knowing." These authors also emphasize the distinction between viewer-independent allocentric and viewer-dependent egocentric spatial processing, with only the former being dependent on an intact hippocampus. Therefore, the parahippocampal recognition system might be sufficient for memorizing a specific view of a scene, but only by virtue of the hippocampus can one use this information for constructing and remembering a representation of a spatial layout as it is necessary for spatial orientation and navigation.[32] Simplistically, one could say that the representation of a place in parahippocampal terms is like a photograph of the scene, whereas for the hippocampus it is like a map. DV is much more like looking at a photograph than it is like looking at a map.

The mesial temporal lobe and interconnected diencephalic counterparts[31] are not the only structures involved in memory retrieval. During retrieval tasks in functional imaging studies, prefrontal areas are even more consistently activated.[33] While it has been suggested that the mesiotemporal structures are involved in actual recollection, prefrontal activation reflects strategic memory search[34] and the reconstruction of the general context of an event.[10] Furthermore, the frontal networks are especially important for remembering the time when an event took place and the temporal order among events.[35,36]

THE HYPOTHESIS

I argue that DV is the phenomenological result of a false activation of connections between mesiotemporal memory structures and neocortical areas directly involved in the perception of the environment. This false activation results in wrongly labeling a momentary perceived scene as familiar. I argue further that in DV this activation remains isolated. According to the presented hypothesis, DV experiences reflect an inflexible parahippocampal recognition memory system, responsible for feelings of familiarity,[37] working in isolation while the more flexible hippocampal recall system is not involved. This does not imply that the hippocampus does not work properly. To the contrary: a normally working hippocampus together with a normally functioning prefrontal system is a prerequisite to recognize the illusionary character of a DV experience and to remember it afterwards.

DV experiences reflect a memory system that is highly involved in spatial processing in an egocentric sense—and not in the way the hippocampus is needed for building up and holding an allocentric representation of space important for navigation.[32] During a DV experience one seems to recognize every single detail of a scene, as if comparing it to a photograph, and nevertheless one has no idea how it looks behind one's back or around the next corner. This stands in contrast to normal remembrances: when one comes to a place one has not been for a long time, everything seems to look a little bit different than remembered (smaller if one was last there as a child), and one is not entirely sure which details have changed since and which not. On the other hand, however, one at least vaguely remembers which way one would go to other formerly known places in the surroundings, and what kind of turn the road makes behind one's back.

EXPERIMENTAL EVIDENCE

Although electrophysiological data have convincingly demonstrated the contribution of mesiotemporal structures in epileptic DV,[14,16,38] clinical and methodological considerations have limited the systematic efforts to study the separate contributions of the different structures. Nevertheless, data from the existing studies are in agreement with the hypothesis. In one study,[16] different symptoms were included under the label "vivid recollection" or "dreamy states," and seizures were recorded from a wide area within the mesial and the neocortical temporal lobe. Nevertheless, in 4 of the 6 patients who reported DV, ictal activity was recorded in the parahippocampal gyrus, whereas this was the case in only 2 of the 10 patients with other symptoms such as hallucinations or isolated feelings of strangeness.

In a study of patients with strictly defined, spontaneously occurring epileptic DV experiences,[38] mesiotemporal seizure onset was found in all cases. Subdural strip electrodes were used that do not directly monitor the hippocampus, but rather the parahippocampal gyrus. The consistent finding of epileptiform activity is therefore compatible with the view that it was the relevant structure—the parahippocampal gyrus—that was monitored. As no stereotactic implanted electrodes were used, however, this study could not clarify the role of the hippocampus proper.

Experimental experiences can be evoked by stimulation not only of medial temporal structures, but also of temporal neocortex.[13,16] According to the hypothesis, neocortical areas that are directly connected to the parahippocampal gyrus should be preferentially involved in DV. Indeed, DV experiences have been documented after stimulation of the directly connected superior temporal gyrus,[14] and not after stimulation of the inferior and middle temporal gyri,[13] areas that are connected with the hippocampus via the perirhinal cortex.[24] In patients with mesiotemporal epilepsy who experienced DV as part of their seizures, parietal lobe hypometabolism has been demonstrated.[39] Because of their widespread brain damage, the sample in that study might not be representative. Nevertheless, the results were clear enough to allow the authors to argue that DV might stem from a mismatch between memory and perceptive processes. I certainly agree, but the data seem to allow a more specific hypothesis. Not all brain areas relevant for memory and perception were activated, only parietal ones. In view of the substantial anatomical projection from area 7 of the parietal cortex to the parahippocampal cortex,[19,25] this fits well with the hypothesis proposed here.

Even if one accepts the suggested mechanism and the similarity between DV as an ictal phenomenon and DV in normal subjects, the question remains open, why DV happens at all in normal subjects. Although DV might be an ictal phenomenon in normal individuals as it is in patients with epilepsy, the potential of drowsiness to elicit DV points to a more plausible explanation. Sleep is not accompanied by a global decrease of brain activity but by a selective change in the pattern of activity.[40] The role of sleep for consolidation of memory traces has been emphasized,[41] and it has been argued that this might be achieved by a more intensive functional connectivity between mesiotemporal and neocortical structures during sleep. This might be accompanied by weakened functional interconnections within association cortex. While enabling the reorganization of connectivity in favor of memory consolidation, as a by-product these alterations would lead to a higher susceptibility to DV experiences. In this context it might be interesting that DV experiences often have been compared with dreams.[42] So, DV experiences in normal individuals might—like dreams—be a by-product of a mechanism that is responsible for memory consolidation.

Despite the subjective and fleeting character of the phenomenon, the hypothesis is testable. First, it is based on the prevailing interpretation of the available data on the role of mesial temporal structures and neocortex for memory retrieval, which is far from settled. Every substantial change in our knowledge about the episodic memory system will thus either support or undermine the hypothesis to the extent that new experimental data confirm or disconfirm the underlying theories. More directly, electrophysiological studies using stereotactic implanted electrodes in patients with epilepsy could clarify the roles of the different parts of the mesiotemporal system in generating epileptic DV. Finally, a way to test the hypothesis would be to capture a DV event during $H_2{}^{15}O$ PET, as was done in migraine headache,[43] or during performance of another suitable functional imaging procedure. However, one would have to be very lucky to have DV occur by chance under these conditions, especially in normal subjects.

POSTSCRIPT

Cognitive neurosciences have demonstrated that cognitive processes can be dissociated, that they often work in parallel,[44] and that they do not converge on a "central observer."[45] Normally all of these processes work together in a way that gives us the impression of a unitary process. Only when something goes wrong does the distinctness of these processes become observable. Such a situation can be found in neurological disorders and is the starting point for cognitive neuropsychology. In healthy subjects as well, sometimes lapses allow glimpses into the cognitive machinery working in the background. Well-known examples are optical illusions.[46] DV experiences give us the rare opportunity of an immediate glance at the isolated performance of one of our systems relevant for remembering. Through an explanation of DV at a neuropsychological level, it may become possible to reconcile theoretical knowledge about the dissociations within the memory system with our genuine experience of an integral and unitary memory of the past.

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