Review Article

Symptomatology of temporal lobe epilepsy

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Summary

The numerous and varied symptoms of temporal lobe epilepsy are reviewed in detail. Partial elementary, partial complex, secondary generalized, post-ictal and predisposing aetiological symptoms are examined.


Slater and Roth stress the immense variations in symptoms in temporal lobe epilepsy. They also remark on how 'fixed and stable' symptoms are in specific patients. Because of its protein presentation, symptoms of temporal lobe epilepsy (TLE) are difficult to classify. Broadly, they may have specific localizing value to the temporal lobes, or they may reflect lesions in closely allied areas such as the deep Sylvian insular area. As they are epileptic in nature, these features may be paroxysmal and sudden. On the other hand, there may be no specific localizing features, for example in tonic-clonic seizures without any aura ('warningless'). These would reflect a generalized seizure which may be ab initio, or may be secondary to a very rapid partial seizure, of which the patient may be amnesic.

Symptoms specifically referable to the temporal lobe and sometimes found in TLE will be discussed here. All these features may occur singly or in combination ('compound partial seizures'). The occurrence of compound seizures greatly increases the probability of localization to specific areas. For example, intense fear is a nonspecific psychopathological feature and its occurrence together with an unpleasant burning smell constitutes a 'small focal seizure', and as such must be construed as epileptic in nature. These features may be paroxysmal and difficult to classify. Broadly, they may have specific localizing value to the temporal lobes, or they may reflect lesions in closely allied areas such as the deep Sylvian insular area. As they are epileptic in nature, these features may be paroxysmal and sudden. On the other hand, there may be no specific localizing features, for example in tonic-clonic seizures without any aura ('warningless'). These would reflect a generalized seizure which may be ab initio, or may be secondary to a very rapid partial seizure, of which the patient may be amnesic.

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Elementary symptomatology with motor symptoms

Gastaut classifies aphasic and phonatory seizures under this heading; although temporal lobe aphasia usually signifies a receptive abnormality, it can be mixed. Ictal aphasia is a common symptom of TLE; Hécaen and Piercy (in Bingley) found it in 37% of 97 right-handed and 90% of 29 left-handed TLE patients. Many of these patients may have speech and not genuine language disturbances, and the incidence of receptive aphasia for the sample was about 13%. This figure compares with that obtained by Currie et al. in the London Hospital (1949 - 1967), based on clinical and EEG findings; they found a 22% rate of speech disturbance in TLE patients. Bingley found an incidence of 48% with dominant hemisphere TLE and 21% with bilateral foci; in his sample none of the patients with non-dominant TLE had ictal aphasia. The site of origin of temporal lobe aphasia can be in Wernicke's area. Rasmussen mentions that those ictal aphasia elementary seizures originating in the dominant parietotemporal speech zone (around Wernicke's area) are often followed by numbness of the face or arm.

Elementary symptomatology with special sensory or somatosensory symptoms

Somatosensory phenomena classically derive from the somatosensory cortex. More complex somatosensory phenomena derive predominantly from the parietal lobes, giving rise to agnosias of various kinds. Penfield further stressed a group of poorly defined sensations which at times derive from the anterior and mesial surfaces of the temporal lobe. These may take the form of head sensations (cephalalgia, the exact localizations of which are obscure) or
body sensations, and may involve sensations of thermal quality (heat or cold).³

**Visual ictal features.** Rasmussen⁴ divides visual illusions and hallucinations into the categories of simple and complex, and believes that elementary limitations are limited to the occipital lobe, involving spots or dots (coloured or black) or just 'lights'. These may be static, moving or rotating. He regards complex visual phenomena as deriving from the temporal lobe. Williams' elaborates on the description of complex visual hallucinations of temporal lobe origin; induction is frequently via a highly specific precipitator, the hallucination may recur in a stereotyped way, and it is almost always linked to other qualities of perception such as voices, emotions or time. Williams stresses, however, that hallucinations of the special senses are uncommon in temporal lobe lesions, and are of no further localizing value. Bingley³ found visual hallucinations in 18% of dreamy states (the same figure he found for auditory hallucinations). Currie et al.³ found that 18% (121) of his TLE sample had visual symptoms of some nature. His corresponding figure for auditory symptoms was 16% (107 cases). Blurred vision is also common, and was found in 16% of Feindel and Penfield's 1954 sample of 50 patients with dreamy states.⁹

**Auditory perceptual abnormalities.** The transverse gyrus of Heschl, situated in the middle of the superior temporal gyrus, and the adjacent areas, passing inward to posterior to the island of Reil, are devoted to crude auditory sensation.³ Stimulation of the primary area and the adjacent areas, passing inward to posterior to the island of Reil, are devoted to crude auditory sensation.³ Stimulation of Heschl, situated in the middle of the superior temporal gyrus, and are responsible for auditory hallucinatory qualities: these are more complex than just sounds, and in epilepsy would be complex partial seizures. Midtemporal lesions may therefore produce undeveloped sounds (e.g. whistles) or developed sounds (e.g. music), and these may be associated with a special meaning, relevance or significance. They are common concomitants in dreamy states; Bingley³ found them in 18% of 29 patients with dreamy states, with tendency (t = 2.0) to non-dominant localization.

**Olfactory hallucinations** are classically and also statistically usually of unpleasant and disagreeable nature.⁸ Ervin stresses the burning or rotting quality that is usual. They are rare, and may reflect an anteromedial lesion of the hippocampus.¹⁰ Williams points out that the olfactory cortex has not actually been localized, and that the previous association with the anterior hippocampus is being reviewed. Olfactory hallucinations have been considered by many authors to be an essential part of the 'uncinate fit' but Bingley³ points out their rarity and that Hughlings Jackson has been misquoted. Penfield¹² thinks that the rarity of the olfactory hallucination may put in question the localization of smell in man; olfactory hallucinations can at best be regarded as a poor localizer. Feindel and Penfield⁹ felt that olfactory hallucinations more commonly reflect tumours as opposed to atrophic lesions. Ictal olfactory hallucinations occurred in 2.3% of Lund's¹¹ 733 patients with a supratentorial tumour, of whom three-quarters had temporal lobe tumours. Fifty per cent of his 34 patients with 'dreamy states' had olfactory hallucinations. Currie et al.⁹ found that 12% (80) of TLE patients have olfactory hallucinations. It can be concluded that olfactory hallucinations of the paroxysmal kind suggest temporal lobe origin, and this becomes much more certain if they are part of a compound complex temporal lobe experience. On their own they are classified as an elementary partial seizure.⁸

**Gustatory hallucinations.** Currie et al.⁹ elicited gustatory hallucinations in 3% (20 patients) of his TLE series. These also occurred in 3% of Lund's series of 733 supratentorial tumour patients; two-thirds derived from temporal lobe tumours, and 10 of the 21 patients involved also had dreamy states.¹¹

The work of Penfield and Faulk¹² suggests that the sensation of taste can be evoked by stimulation of the insula below the superior opercula. Williams¹ points out that illusions or hallucinations of taste are rare in organic lesions. Also worth noting is the fact that olfactory disturbances are sometimes experienced as disturbances of taste.³ Rasmussen⁴ regards paroxysmal disturbances of taste as being elementary partial seizures of deep-lying Sylvian origin.

**Vestibular symptomatology.** Ictal vestibular features arise from the middle and posterior areas of the superior temporal gyrus. Anatomically, the primary vestibular area is posterior and postero-inferior to the auditory cortex.¹² The subjective vertigo is often described as dizziness producing a paroxysmal impression of 'turning' of self or the environment.⁸ This rotational component is symptomatic of true vertigo. Williams¹ points out that vestibular ictal features can also manifest as disequilibrium. Currie et al.⁹ found this to be a feature of 19% (126) of his TLE patients.

**Elementary symptomatology with visceral sensations or autonomic symptoms**

A great variety of autonomic phenomena derive from the periamygdaloid region, but none originate from the lateral temporal lobe.⁴ Feindel and Penfield⁹ found (in their 50 cases) that among the wide range of phenomena that initiate, accompany and follow automatisms, flushing or pallor of the face occurred in 22% and abdominal sensations (including nausea) in 34%. These were, of course, complex phenomena (part of the dreamy state) frequently occurring (72%) as compound symptoms. Viscero-motor symptoms may also involve sweating.¹³

**Alimentary symptoms.** Stimulation of the cortex of the insula produces sensations referring to some part of the alimentary tract from the mouth to the rectum. Motor responses such as borborygmi or the desire to defaecate may also occur.¹ Penfield and Faulk¹² found that stimulation of the anterior insula produced movement, and posterior insula, sensations; Currie et al.¹⁰ in their study found that visceral features occurred in 40% (264) of TLE subjects. Epigastric sensations, although deriving from the insula, are looked upon as rather classic features of TLE and are variously described. For example, 'sinking feeling' is Batchelor's description,¹⁶ 'rising' is Slater and Roth's,¹ and 'gripping' that of Penfield.¹⁷

**Complex partial seizures of temporal lobe origin**

These form the majority of the epileptic abnormalities occurring in the temporal lobes and 'complex partial seizures' is now frequently used as a synonym for TLE.¹⁸ In general, they are associated with defects in consciousness, which may only be transient, may be associated with partial recall and frequently have a mixed quality encompassing, for example, cognitive disturbances (depersonalization, dejà vu or déjà vécu) with emotional hallucinations.¹² Partial preservation of awareness is the special feature of focal epilepsy, particularly the temporal lobe attack.¹ Williams stresses that epileptic events are caricatures of the function of the executive or receptive primary or association areas.¹ These result in a 'disintegrated' picture. This description is particularly applicable to the complex partial seizure.
Complex partial seizures with impaired consciousness only

Wilder Penfield\(^7\) describes the 'amnesic seizure' which may or may not have a motor automatism component. The one constant characteristic is the subsequent amnesia, even though the patient may not appear unconscious. The alteration in consciousness itself may be severe or slight. The amnesia is due to the loss of the 'capacity of making a permanent record in the stream of consciousness', the basic defect being an inability to record present experience.\(^7\)

Anatomically, amnesia can be produced by stimulation of the area around the amygdaloid nucleus which, however, also causes automatism. As a sequel to this finding, Penfield,\(^1\) with Milner, was able to show that the hippocampal zone played an important role in the recording of present experience. This could be the anatomical site of the epileptic absence frequently seen in TLE.

A complex partial seizure with loss of consciousness involves an 'absence'. These absences are said to be differentiated from the typical petit mal generalized absence by the concentrated 'fixed stare', 'not blank eyes', which may be indiscriminate at some point, and last from 30 to 60 seconds.\(^7\) These must also be differentiated from aphasial attacks. Another feature found either alone or in combination in the 'dreamy state' is what Feindel and Penfield\(^9\) described as 'conscious confusion'. This symptom was described in 32% of their 50 cases. Its anatomical localization is also probably in the anterior mesial temporal area.

Complex partial seizures with cognitive symptomatology

These can take many forms. For example, a classic symptom is the 'play-back' in which there is a reliving of stereotyped auditory or visual memory as part of the ictal experience.\(^1\) The best known research is that of Penfield and Perot\(^20\) who in more than 1000 operations elicited visual and auditory experiential recall by stimulation of areas of the lateral temporal lobes. The most useful classification of cognitive complex temporal lobe experiences is that of Wilder Penfield\(^17\) who called these 'flashbacks' ('play-backs') 'psychical hallucinations' or 'experiential seizures'. These involve hallucinations of past experience, and reactivation of a strip of the stream of consciousness.

The 'interpreative seizure' is Penfield's second cognitive temporal lobe complex component. These involve alterations of the present and illusions (or false interpretations) of present perception. Penfield\(^1\) classifies these illusions into auditory illusions of distance, loudness and tempo, visual illusions of comparison involving familiarity, strangeness and unreality, and emotional illusions of fear, separation, sorrow and disgust. An overlap occurs here: interpretative illusions of comparison come under cognitive dysmnesic disturbances in the form of familiarity illusions, under which Penfield classifies \(d\)\(é\)\(j\)\(a\)\(\acute{v}\)u illusions of emotion fall into Gastaut's\(^4\) classification of ictal emotion; and illusions of auditory and visual kind fall under Gastaut's 'psychosensory illusions'. Penfield's 'experiential seizures' are largely synonymous with Gastaut's 'ideational disturbances'.

During both types of attack the patient continues to be conscious and he is usually capable of introspection.\(^1\) Penfield regards their origin from the same area (lateral and superior temporal lobe of both hemispheres, excluding primary auditory and visual areas, but including Wernicke's area) to be significant for localization of what is now regarded as memory engrams. Under the heading of dysmnesic disturbances, Gastaut\(^4\) has included conscious amnesias, \(d\)\(é\)\(j\)\(a\)\(\acute{v}\)u and \(d\)\(é\)\(j\)\(a\)\(v\)\(é\)\(c\)u. Also included for convenience, although their memory disturbance components may be disputed by individual authors, are the other illusions of comparison involving strangeness and unreality — namely \(j\)amai\(s\) \(v\)u, depersonalization and derealization.

Williams\(^7\) points out that disturbances of memory are associated with bilateral anterior temporal lesions, frequently linked to naturally occurring components such as sight, sound, smell and emotional tone. The crucial interplay of memory recall with subjective awareness of time is also stressed by Williams.\(^7\)

Gastaut\(^4\) includes forced thinking and dreamy states under 'ideational disturbances'. Dreamy states have been dealt with earlier and are also discussed later. Forced thinking involves the intrusion into consciousness of a thought that is resisted, but remains or repeats itself ('compulsive thinking'). Reef\(^2\) points out that it is usually unpleasant. Forced thinking is, of course, usually symptomatic of an obsessive-compulsive neurosis. Its value in the diagnosis of temporal lobe epilepsy is, therefore, only nonspecific.

Complex affective symptomatology

Williams\(^7\) regards these symptoms as being 'emotional hallucinations'; they are usually unpleasant, vivid and unrelated to the environment,\(^9\) and often autochthonous. In 100 epileptics with these he found 61 to be of anxiety quality, 21 depressive, 9 pleasant and 9 unpleasant. He also described feelings of anger and suspense. The pleasant and unpleasant feelings, he felt, had anterior temporal origin, while the anxiety had anterior temporal origins.

Ervin\(^7\) stresses that temporal lobe affect can present as depression which can take the form of acute despair, with or without suicidal ideation. On the other hand, pleasurable feelings can reach the level of elation, or the subject may experience serenity. The importance of the temporal lobes in these affective disturbances had been illustrated by the fact that operations on the amygdala, as well as medial temporal lobectomy and bilateral temporal lobectomy, induce change.\(^7\)

Currie \(^4\) found emotional disturbances in 19% (130) of their TLE sample.

Ictal anxiety: the frequent occurrence of 'fear which comes by itself' in the 'uncinate group of seizures' was noted by Hughsings Jackson.\(^23\) MacLean\(^24\) confirms that this is an epileptic phenomenon, out of the psychological context and not due to natural fear of the fit itself. Williams\(^7\) found ictal anxiety in 3% (61 out of 2000) of epileptics, 165 of whom had complex ictal symptoms (probably of temporal lobe origin). He attributed this emotional hallucination to the temporal cortex. (5 of the 61 had anterior TL foci, and 17 middle TL foci). Lund\(^1\) found that 60% (10 out of 17) of his sample with ictal anxiety had other autonomic disorders (e.g. epigastric sensation, flushing, sweating, palpitations). Williams\(^10\) found this association in 50% of his ictal anxiety cases. Bingley\(^7\) shows a close statistical correlation between the topographical and physiological relationship of ictal anxiety and epigastric sensations. According to Reef,\(^2\) fear is the commonest form of seizure with affective disturbance, and is usually associated with a hallucinatory experience. Ictal anxiety may also be experienced as panic symptoms.\(^15\)

Aggressive outbursts: The anatomical location of aggression has important connections in the amygdala and periamygdaloid areas.\(^25\) This has been supported by EEG findings. Thus, Williams\(^10\) points to EEGs with predominantly anterior temporal and frontotemporal abnormalities in 2000 subjects with habitual aggression. This finding is of nosological interest too, as the disturbed behaviour frequently found with anterior temporal dysfunction is often regarded as part of the antisocial personality. Despite all this, aggression or rage as an epileptic phenomenon of significant intensity is generally rare; Williams\(^10\) and Feindel and Penfield\(^4\) quote figures of the order of 0.5 - 1%. It is uncertain if these operationally refer only to epileptic furor states, which would be one extreme. Reef\(^2\) describes 'seizures with antisocial behaviour', involving moodiness, irritability, anger or other behaviour disturbances usually lasting only a few minutes but possibly persisting for
Complex partial seizures with psychosensory symptomatology

Depersonalization and derealization phenomena are frequently associated with illusions and hallucinations, according to Williams, and are just two striking manifestations that may compound psychosensory temporal lobe symptomatology. They may occur in combination with many other temporal lobe symptoms, as confirmed repeatedly in the literature (Feindel and Penfield, Currie et al., Bingley). These illusory and hallucinatory experiences may also be associated with a defect in consciousness, producing a 'dreamy state'. They involve complex seizures because of their experiential/interpretative, distorting or disintegrating quality. Their actual content may be protean and widely varied, but their frequent stereotyping and constancy within individual patients is noteworthy.

Psychosensory illusions may be visual or auditory. Penfield regards 'visual illusions of alteration' occurring as epileptic discharges, as being of predominantly non-dominant (for handedness, not speech) temporal hemisphere origin (10 out of 11 cases). He also elicited these features by only stimulating the minor hemisphere. If these results are generalized, it is possible that psychosensory hallucinations theoretically involve a reactivation of something previously in memory storage, they should differ markedly from the hallucinations found in the functional psychoses. However, a variety of other compound complex phenomena of cognitive, affective and other psychosensory kind may be associated with the reactivation of the stream. This may produce a picture very similar to an acute schizophrenic or manic episode.

Penfield points out that the epileptic is having the double experience of a re-creation of the past with the consciousness of the present time during the experiential hallucination. As a specific area may theoretically be involved in firing during a seizure, the epileptic will have the same psychosensory hallucination repeatedly. It may also not have the specific relevance, special meaning, auditory quality, and self-reference so frequently found in schizophrenia.

The psychosensory hallucination deriving from the temporal lobes can involve any of the sensory modalities or combination thereof, namely visual, auditory, olfactory, gustatory, vestibular, tactile or the 'indescribable' hallucinations described by Williams. The complexity of these hallucinations depends upon the specific area of firing. Williams' conceptualization of increasing qualitative complexity of each experience as the area of firing moves away from the primary sensory to the secondary, tertiary and more distant association areas, is again emphasized.

Williams describes TLE hallucinations of indescribable quality which may originate in the medial temporal cortex; their indescribable quality may be associated with pathways not usually brought into conscious awareness.

Psychomotor seizures and the 'dreamy state'

These have already been discussed. The major feature of the 'dreamy state' of Jackson is whole or partial withdrawal from the present. This involves a defect of consciousness of the environment, but not of consciousness of self. The 'dreamy state' emphasizes the subjective mental phenomena. The 'psychomotor seizure' emphasizes behavioural phenomena. Technically, the two are almost identical. As both terms are used to refer to a wide variety of combinations of perceptual and motor disturbance, these symptoms are best discussed individually, with the knowledge that compound partial seizures (usually complex) in many combinations make up the mental and behavioural components of the dreamy state or psychomotor seizure.

Automatisms are 'complex partial seizures with psychomotor symptomatology'. The automatism involves the motor component of the psychomotor seizure. Its usual origin is from the mesial temporal area, never the lateral temporal area. It may, however, have orbitofrontal, subcortical and other extra-temporal lobe origins. Further evidence as to its origin relates to the success of treatment of automatism by operative ablation of the amygdala and surrounding tissue, and production via stimulation of this area.
Automatisms have a stereotyped fixity. They may be behavioural, involving masticatory/salivatory, buttoning/unbuttoning or other simple, repetitive movements, and seldom last more than a few minutes, often far less. Alternatively, Ervin stresses the rich flow of associations achieved under hypnosis and the induction of automatism by this method. He points out that in the same way that normal dreams have symbolic significances so too do complex automatism. These may last hours or, rarely, days. A hazy or distorted memory may be epileptic. Epileptic automatism involve programmed motor activity set off by the attack and interference with this will cause resistance which may be dangerous. The attack often begins with a ‘dreamy state’ of some kind. Automatisms may take the form of fugue states, speech automatism or furor, and involve very complicated acts with normal or occasionally dyssocial behaviour. They may be differentiated from hysterical dissociation by their abruptness, the absence of precipitating factors, and the complete amnesia (Livingston and Pauli). Ervin stresses the absence of secondary gain, and the fact that even under hypnosis the amnesia persists. Nemiah points out that clinical dissociative features with other evidence of TLE should be suspected to be organic.

The electroclinical correlate may be forced normalization or an unchanged EEG, in which case depth electrography may clarify the picture. Depth electrography (using sphenoidal electrodes) with chemical activation of the patient’s habitual seizure recorded is suggested in cases of difficult diagnosis by Remick and Wada. Ervin differentiates the post-ictal twilight state, which commonly presents with automatic behaviour associated with a confusion, lasting often days, and with symmetrical, slow, non-paroxysmal EEG activity.

Bingley points out that in ictal speech automatism, the patient utters a mixture of words and sentences which may be linguistically correct but bear no appropriate relation to the present situation; this is less closely related to the dominant lobe than ictal aphasia and more closely related to other forms of automatism. Thirty-three per cent of his sample had speech automatism.

Rare psychomotor symptoms include orgasmoeypsy, in which the patient may experience orgasm as an aura, or in association with other symptoms e.g., laughter or weeping. Anatomically this may be associated with the limbic part of the temporal lobe, but may also occur with midline di-encephalic lesions. Gelastic epilepsy involves paroxysms of laughing, giggling, usually associated with pleasure. Quiristaure (or dacrocystic) epilepsy involves crying episodes. Cursive epilepsy involves actual running in paroxysms.

Gelastic, dacrocystic and cursive epilepsy are all highly unusual but well described epileptic symptoms. These are usually associated with amnesia and often involve the complex juxtaposition of temporal lobe symptoms of the visceral, expressive and affective kind. They may derive from the amygdaloid or hippocampal areas of the temporal lobe, but may also have origin in the posterior hypothalamus, basal ganglia and third ventricle.

Partial temporal lobe seizures, secondarily generalized

Bingley found that 83% of his sample of 90 neurological/neurosurgical patients had tonic-clonic seizures secondary to a primary temporal lobe focus. There was no significant difference between tumour and non-tumour cases, or in laterality of the lesion. This figure corresponds closely with others quoted for associated tonic-clonic seizures, the exact sample derivations of which are unknown: Gibbs and Anderson and Trethowan both indicate that in 80% of cases TLE is associated with grand mal seizures. The latter stress that almost all cases of aura followed by tonic-clonic seizure originate from the temporal lobe. It is very possible that the figures for generalization of seizures of temporal lobe origin are far lower in psychiatric populations. In any event, the presence of tonic-clonic seizure with auras of temporal lobe origin can be regarded as making the diagnosis of TLE almost definitive.

Post-ictal symptomatology

The presence of typical post-ictal features may alert the clinician to a diagnosis of epilepsy, although these may reflect epilepsy of generalized, partial or unclassifiable origins. Post-ictal features are common, and unless at least one post-ictal feature typically occurs with seizures which generalize themselves (particularly tonic-clonic seizures), an equivocal diagnosis of epilepsy should be seriously reviewed.

The most common features are listed by Slater and Roth as: confusion, clouded consciousness with disorientation for time, place and even person, often with rambling speech or disjointed behaviour, fidgeting or restlessness; post-epileptic automatism that may involve well-controlled behaviour or purposeless movements; sleep with fatigue; headache; often very severe, or waking from a post-ictal sleep or separately. Nausea with or without vomiting is also common.

Aetiology of temporal lobe epilepsy

It is important to summarize the most common causes of TLE, as the presence of a recognized aetiological factor in the history may alert the clinician to the diagnosis.

In the large London Hospital sample (about 600) of Currie et al., two-thirds of TLEs were of unknown aetiology. Tumours were found in 9.5%, head injury was associated with 7%, as was birth trauma. Infantile seizures had occurred in 5%, and 11% had a family history of epilepsy. Overall, 25% of cases were thought to have a definite cause. This study found no EEG temporal lobe focus in 8%, and in only 6% was the diagnosis purely electrographic. Of the EEG abnormalities, 52% were left-sided, 29% right-sided and 19% were bilateral. Routine activation procedures, often with sleep and sometimes with sphenoidal leads, were used.

Penfield and Jasper explained the frequency of mesial temporal lesions as due to ‘insular sclerosis’ which results from the bitemporal scarring associated with compression of the anterior choroidal artery due to herniation of the deepest portion of the first temporal convolution, uncus and hippocampus during birth trauma. The alternative explanation relates to infantile hypopryrexial convulsions which may affect this area, which is relatively primitive and highly sensitive to anoxia.

It is particularly important to elicit any history of birth trauma, infantile febrile convulsions, head injuries and constitutional predisposition to epilepsy. Other possible causes such as the presence of tumour, infection and prenatal and postnatal problems, should also be noted.

REFERENCES

Is *déjà vu* a symptom of temporal lobe epilepsy?

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**Summary**

The definition and conceptualization of the *déjà vu* phenomenon are interpreted in various ways. The common occurrence of *déjà vu* in the general population stresses the need for the development of specific qualitative features which will be valuable in the diagnosis of temporal lobe epilepsy.


**Definitions**

Exact definitions of the *déjà vu* phenomena differ from that of Warren,¹ who refers to an 'illusion of recognition', to others that do not require the recognition concept at all, such as Barton's² 'an abnormal feeling of familiarity'. Some definitions do not even stress the erroneousness of the experience, and that of Rycroft³ is probably the best example: 'The subjective sense that a present novel experience has been gone through subjectively'. Thus *déjà vu* is conceptualized differently, and at times contradictorily. One important theoretical contradiction involves part-whole recognition; Silverman¹ stresses how *déjà vu* illustrates 're-dintegration in which the same stimulus or stimuli has occurred at another time (and) elicits a pattern of remembering'. This implies that a 'part tends to reinstate the whole' and is equivalent to what Banister and Zangwill³ call a 'restricted paramnesia', being specifically differentiated from *déjà vu* which is 'the illusion of reliving a situation in its entirety'.

*Déjà vu* strictly implies an 'already seen experience', but in a broader sense can imply any feeling of familiarity with the past. In this study the concept of *déjà vu* is used in its broader sense. It therefore includes many other experiences of the past: *déjà entendu* (already heard), *déjà épruvé* (already experienced), *déjà fait* (already done), *déjà pensé* (already thought), *déjà voulu* (already desired), *déjà raccordé* (already recounted), *déjà senti* (already felt), and *déjà vecu* (already lived).

**Categorization**

The *déjà vu* phenomenon can be categorized as a paramnesia, as an ego-state disorder, or as a perceptual disturbance. All these...