Epilepsy Research (2009) **85**, 162—171

journal homepage: www.elsevier.com/locate/epilepsyres

REVIEW

Delusions, illusions and hallucinations in epilepsy: 1. Elementary phenomena

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Received 31 October 2008; received in revised form 8 March 2009; accepted 15 March 2009
Available online 6 May 2009

KEYWORDS
Epilepsy;
Hallucinations;
Delusions;
Illusions;
SEEG;
Cerebral localisation

Summary The purpose of this paper and its pair is to provide a comprehensive review, from the different perspectives of neurology and neuropsychiatry, of the phenomenology and mechanisms of hallucinatory experience in epilepsy. We emphasise the clinical and electrophysiological features, and make comparisons with the primary psychoses. In this paper, we consider definitions and elementary hallucinatory phenomena. Regarding definition, there is a clearly divergent evolution in meaning of the terms delusion, illusion and hallucination in the separate traditions of neurology and psychiatry. Psychiatry makes clear distinctions between the terms and has focussed on the empirical use of descriptive psychopathology in order to delineate the various psychiatric syndromes, including those in epilepsy. These distinctions in psychiatry have stood the test of time and are useful in clinical descriptive terms, but do not help to understand the basic mechanisms. The focus of neurology has been to regard delusions, illusions and hallucinations in epilepsy as a result of localised or network based neuronal epileptic activity that can be investigated especially using intracranial stereoelectroencephalography (SEEG). The neurological approach leads to a more synoptical definition of 'hallucination' than in psychiatry and to the conclusion that there is little point in differentiating hallucination from illusion or delusion in view of the overlap in the physiological bases of the phenomena. The semiologically derived differentiation of these terms in psychiatry is not supported by similarly discrete electrophysiological signatures. However, as discussed in the second paper, some psychotic states are associated with similar electrophysiological changes. The wide range of hallucinatory symptoms occurring during epileptic seizures recorded during intracranial SEEG and brain stimulation are reviewed here, including: experiential and interpretive phenomena, affective symptoms, as well as auditory, olfactory, gustatory, somatic and visual hallucinatory phenomena. Several conclusions can be drawn. First, it is clear that there is only limited anatomical specificity of many hallucinatory states. Repeated seizures or stimulation of a single area, even within the same patient can produce different psychic responses, whilst stimulation of

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0920-1211/$ — see front matter
doi:10.1016/j.eplepsyres.2009.03.018

widely distinct areas (especially in the limbic system) within the same individual can produce remarkably similar phenomena. This lack of specificity applies particularly to psychic symptoms, including experiential phenomena, and complex hallucinatory states. The most anatomically specific areas from this point of view are the elementary hallucinations arising from primary visual and auditory cortices. Involvement of the limbic cortex is a pre-requisite for the occurrence of complex hallucinatory states. It is clear that on the basis of these findings, as has been recognised at least since the 1960s, that even apparently focal epileptic seizures, (especially in the mesial temporal lobe, insula and limbic cortices), must involve widely distributed neuronal networks.

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> 'If sensitive nerves are enough to make a poet I should be worth more than Shakespeare and Homer... I who have heard through closed doors people talking in low tones thirty paces away, across whose abdomen one may see the viscera throbbing, and who have sometimes felt in the space of a minute a million thoughts, images and combinations of all kinds throwing themselves into my brain at once, as it were the lighted squibs of fireworks.' Correspondence of Flaubert, (Lombroso 1891).

The term 'lighted squibs of fireworks' is a pertinent way of describing the disordered bursts of energy which underpin at least some of the hallucinatory experiences seen in epilepsy.

Historically, much of the research in this area derives from the field of psychiatry, with its emphasis on the empirical use of descriptive psychopathology in order to delineate the various psychiatric syndromes and determine to what extent the psychopathology (i.e., abnormalities of affect, thought, and perception) seen in epilepsy is similar to, or differs from that seen in the major mental disorders, for example schizophrenia.

By contrast, there has been an almost entirely separate tradition within neurology. The central theorem of this school is that hallucinations occur as a consequence of the activation of a localised group of neurones which can be investigated by cerebral recording and cerebral stimulation, and the 'Gold Standard' investigation has been intracranial stereoelectroencephalography (SEEG). Over the past 50 years this application has greatly clarified some of the details regarding the anatomical basis and physiological mechanisms underpinning hallucinations in epilepsy. Throughout this period psychiatry has tended to echo the view of Hughlings Jackson that compound mental states 'cannot be owing to an epileptic discharge' (Jackson, 1958), however, whilst it is true that complex psychic states as seen in the 'functional' psychoses have less often been correlated with abnormalities on SEEG, as Trimble (1991) points out 'specific Schneiderian phenomena have not been recorded but in all probability have not been examined for.' There is now a considerable body of SEEG evidence which calls this view into question and it appears, in at least some cases, that complex psychotic symptoms are directly due to the effects of non-convulsive epileptic activity (limbic status) or to the indirect after-effects of chronic epileptic discharges.

The purpose of this set of two papers is to provide a comprehensive review of the phenomenonology of hallucinations in epilepsy from a specifically neurological and neuropsychiatric standpoint, and to draw distinctions between this approach and that of clinical psychiatry. The term 'hallucination' is here taken to encompass a range of phenomena giving it a very different usage to that seen in psychiatry. Based as it is on electrophysiological findings, our main thrust is to present SEEG evidence which demonstrates that hallucinations often have an electrophysiological basis, usually involving widely disseminated limbic structures. In this first paper, we begin with a brief review of definitions and of the different meanings assigned to key terms such as 'hallucination' and 'psychosis' that have emerged as a result of the divergent evolution of very separate neurological and psychiatric traditions. We then consider the elementary hallucinatory states which are observed during brain stimulation, and during spontaneous brief epileptic seizures.

In the second paper we consider the more complex and prolonged states associated with complex partial status epilepticus, postictal and interictal psychosis. The similarity of these latter states to the primary psychoses raises interesting questions about the pathophysiology of psychosis.

It is important to recognise that SEEG does have a major limitation, namely sampling bias, which we would like to mention at the outset. The electrodes record activity from only a very small area in the vicinity of the electrode (measured in millimetres), and activity in areas beyond this will be overlooked. This is important as there are many indications that limbic functional (and dysfunctional) activity is often a widely distributed network phenomenon involving disparate interconnected neuronal areas. Gloor for instance, has shown repeatedly that the concept of a small discrete limbic epileptic focus is inaccurate and that limbic seizures (even with focal pathology, such as hippocampal sclerosis) may involve simultaneously a wide network of neuronal activation, not necessarily even contiguous. This