The Temporolimbic System Theory of Paranoid Schizophrenia

by Manuel F. Casanova

Abstract

The hippocampus serves as a funnel for heavily processed sensory information that has converged at the entorhinal cortex. Lesions of the hippocampus do not alter incoming sensory or motor information but, rather, alter their integration with our baggage of emotional experiences and social values. According to Bogerts, such a lesion would be ideally situated to result in laboriously processed sensory information that is out of context to our outside environment. In this regard, Bogerts describes the pathological findings of a patient with a gross delusional disorder. The salient finding at autopsy was a developmental lesion in the left posterior parahippocampal gyrus. Although a number of lesions have been described in the brains of patients with schizophrenia, Bogerts believes that those in the limbic system appear critical to the expression of paranoid symptoms.


The limbic system is a relatively recent attempt to explain a convoluted area of the brain intrinsically important to psychiatry. Anatomical definitions have varied greatly during the past few decades, a problem that has to do primarily with lack of cytoarchitectural homogeneity between different brain regions. The term limbic system is derived from the Latin word *limbus*, meaning hem—a term first used by Broca to define the gray matter of the brain folding itself like a hem over the choroid plexus and rostral brainstem (Isaacson 1982). In a broader sense, some researchers have added several adjacent anatomical structures and thus expanded the initial concept. In some instances, the question of which areas are designated as limbic is a matter of semantics. Thus, the hippocampus proper (also called Ammon’s horn), an unequivocally limbic structure, is only part of the hippocampal formation. The entorhinal cortex (a paralimbic or transitional type of cortex) is often termed limbic in origin, but it only expands the most anterior portion of the parahippocampal gyrus.

In the present article, Bogerts (1997, this issue) includes the entorhinal cortex within the limbic system to account for a hypothesis that relies on the elaboration of sensory processing and the integration of this information with drive, affect, and other aspects of mental content (Mesulam 1985). Faulty integration of this information appears crucial to schizophrenia as well as other psychiatric conditions.

According to the concepts of Jones and Powell (1970), later extended by Mesulam et al. (1977), the sequential flow of information through the brain follows connections from primary sensory cortices to modality-specific association areas and from there to sites of sensory convergence. These polymodal association areas direct their afferents to supramodal cortical sites and finally into the limbic system. Such projections allow the hippocampus to receive multimodal sensory information derived from both the external (association cortex) and internal (amygdala) environments.

The lack of connections with primary sensory and motor cortices would explain why lesions of the limbic system would not negate visual, auditory, or somatosensory information but would rather alter its processing. The malformed sensory clues are thought through laboriously by the patient and built into a delusional system, while other signs, such as hallucinations, remain absent. This process is the basis of the temporolimbic system theory of paranoid schizophrenia—a theory that attempts to place findings of limbic pathology in perspective in terms of the clinical signs observed in paranoid schizophrenia.

Kraepelin (1913/1919) believed that pure paranoia was a rare disorder characterized by stability of behavior and mood. Modern studies give credibility to the tem-
porolimbic system theory of paranoid schizophrenia and the fact that mood coloring in these patients is in keeping with the content of the delusion. Intracellular recordings in animals have shown that transmission of information through the entorhinal cortex and into the hippocampus proper is modified during different behavioral states, such as sleeping versus waking (Winson and Abzug 1978). Dahl and coworkers (1983) believe that the purpose of this modification is to adjust the hippocampal function according to requirements of the prevailing behavioral state. In an analogy to paranoid schizophrenia, the expression of limbic system dysfunction may therefore be modulated by, rather than extraneous to, the environment.

According to Bogerts (1997, this issue), a landmark case in favor of the temporolimbic system theory of paranoid schizophrenia is provided by pathological studies on the brain of Ernst Wagner. The clinical history of this case has vexed psychiatrists for several generations. The controversy has revolved around whether the case represents a form of schizophrenia (paranoid), or whether the lack of disturbances outside of Wagner's delusional system made his an independent disorder.

Ernst Wagner was a teacher with a family history of mental problems. At age 27, he committed an undisclosed sexual act with animals. The next day, and for the rest of his life, Wagner developed the idea that other people knew what he had done and were mocking him. This led to a sensational mass murder that included his wife, children, and eight others from his village (Gaupp 1974a, 1974b). Long-term followup revealed classical Kraepelinaan paranoia with little or no gravitation toward other schizophrenia-type traits.

Not surprisingly, a recent investigation of Wagner's brain by Bogerts has revealed a clear developmental abnormality within the left posterior parahippocampal gyrus of the temporal lobe. Contrary to patients with ablation of the temporal lobes or limbic lesions, Wagner retained his intellectual faculties, especially his memory. According to Bogerts, impairment of memory requires bilateral extensive damage of the limbic system, which was absent in this case. I would also like to add that the developmental nature of the lesion in Wagner's brain could have accounted for a certain amount of plasticity. As Bogerts carefully stipulates, it is doubtful that a disease characterized by a vast array of clinical manifestations is the sole consequence of a minute focus of pathology. It seems more likely that limbic pathology may account for some, but not all, of the clinical abnormalities seen in this patient.

The role of the limbic system in schizophrenia has been suggested in previous postmortem studies. Organic processes such as encephalitis, cerebrovascular accidents, tumors, and head injury leading to temporal-lobe damage may present with schizophreniform symptoms (Davidson and Bagley 1969; Trimble 1988). Topographical analysis of these abnormalities indicates that the incidence of psychosis is highest for those lesions of presumed developmental origin lying toward the medial aspect of the temporal lobes (Taylor 1975; Roberts et al. 1990).

In addition to experiments noted above, Bogerts summarizes a whole array of findings derived from the neuropathology, neurochemistry, and neuroimaging literature that suggest limbic pathology in schizophrenia. But, unfortunately, any claims suggesting a clinicopathological correlation to schizophrenia still remain extravagant. Most of the studies quoted present considerable methodological difficulties, including lack of corroboration, lack of clinical subtyping, and inadequate controls. The major criticism of the Bogerts' hypothesis remains that, given all of the varied findings described in the neuropathological, neurochemical, and neuroimaging literature, limbic pathology is only one of several constructs that can be associated with schizophrenia or paranoia itself. Neuropathological findings still remain subtle for such gross clinical manifestations.

Although dysfunction of the limbic system may provide a physiological basis for some of the salient symptoms observed in paranoid schizophrenia, by itself it is insufficient to explain the whole spectrum of manifestations characterizing the disorder. The rich connections of the limbic system, and the shared pathological vulnerability with other sites of the nervous system, make Bogerts believe that instead of an isolated focus of abnormality, there are a number of pathological foci whose aberrant function explains the variable clinical expression observed in schizophrenia. In this system, dysfunction of the limbic system appears critical to the expression of paranoid symptoms.

References


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