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-
The role of the limbic system in schizophrenia has been suggested in previous postmortem studies. Organic processes such as encephalitides, 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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