Perspective

Critical Evaluation of the Concept of Schizophrenia from A Historical Perspective

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INTRODUCTION

Brief descriptions of an illness resembling schizophrenia can be found hundreds of years ago but it was not until the middle of 19th Century that Morel\(^1\) first coined the term “dementia precoce” to describe a disorder starting in adolescence and leading first to withdrawal, odd mannerisms and self neglect then eventually to intellectual deterioration. Morel searched for specific entities and argued for a classification based on cause, symptoms and outcome. In 1868, Kahlbam described the syndrome of catatonia and 3 years later Hecker wrote an account of a condition he called hebephrenia.

The crucial development in defining this syndrome was achieved by Emil Kraeplin\(^2\) in 1893. Kraeplin argued against a single psychosis and proposed division into “dementia praecox” and “manic depressive insanity”. He originally divided the disorder into three subtypes (catatonic, hebephrenic and paranoid) but later added a fourth type (simple). It was Eugene Bleuler,\(^3\) a professor of psychiatry in Zurich, who proposed the name ‘Schizophrenia’ in 1911, to denote a splitting of psychic functions, which he thought to be of central importance. He believed that the fundamental symptoms were incongruity of affect, loosening of associations, ambivalences and autism (Four A’s). Although Bleuler’s concept of schizophrenia became very popular in United States, Kraeplin’s original concept of dementia praecox remained influential and dominant in other parts of the world.

EARLY HYPOTHESIS

Freud\(^1\) presented his theory of schizophrenia in 1911 and 1914. According to him, in the first stage libido was withdrawn from external objects and attached to the ego, resulting in exaggerated self-importance, making the external world meaningless. Langfeldt,\(^4\) in
late 1930’s studied the symptomatology in detail and distinguished between schizophrenia and schizophreniform psychosis, suggesting that both had a different outcome.

In the mid 20th Century Kurt Schneider⁵ tried to make the diagnosis more reliable by identifying groups of symptoms, which he believed to be characteristic of schizophrenia but were rarely found in any other disorder. On the basis of his work at the Medical Research Council, Crow ⁶ advanced his two-syndrome model. He proposed that there are two major dimensions of psychopathology in schizophrenia: Andreasen⁷ provided a broader concept of the negative syndrome, including affective blunting, alogia (impoverished thinking and speech), avolition/apathy, anhedonia/associality and disturbance of attention. Crow⁸ himself described the shortcomings of the two-syndrome concept when he stated that this approach attempted to correlate neurochemical and structural elements of the schizophrenic disease process with the positive and negative concepts of the symptoms. Liddle⁹ described three syndromes, psychomotor poverty, reality distortion and disorganization.

**WHAT CAUSES SCHIZOPHRENIA?**

As mentioned above, for a considerable period the psychodynamic approach, abnormal family interaction and stressful life events were in the spotlight as a causation of schizophrenia, causing enormous grief to parents. The concept of schizophrenogenic mother was suggested by From-Rchicmann and Lidz¹⁰ and described two types of abnormal family pattern one of them being marital skew and the other marital schism. Bateson et al¹¹ presented the idea of double bind, which is said to occur when an instruction is given overtly but contradicted by a second, more covert, instruction, leaving the child in an ambiguous and meaningless situation. Eminent sociologists like Laing, Szasz and Goffmann¹ opposed the medical models of schizophrenia and Szasz believed that schizophrenia was a myth. Goffmann was of the opinion that this was a role forced on an individual and Laing described it as a sane reaction to an insane world.

**IS SCHIZOPHRENIA A BRAIN DISEASE?**

The breakthrough in the understanding of the illness came in 1950, with the discovery of certain drugs that had a therapeutic effect on schizophrenic symptoms. It lead directly to the dopamine hypothesis of schizophrenia but cerebrospinal studies produced weak or negative findings. It has been known from the early years of the century that schizophrenia shows a tendency to cluster in families.¹² The most consistent abnormality found in the brains of people with schizophrenia is structural and takes the form of lateral ventricular enlargement, mainly restricted to the temporal horns on the left side as documented in a post mortem study by Crow et al.⁸ Other histological changes for schizophrenia claims include (a) reduced cell numbers in the hippocampus (b) reduced cell numbers in the entorhinal cortex (c) reduced hippocampus cell size and disturbed cytoarchitecture in the entorhinal cortex. It was found that impairment of performing tasks was reflected in a significantly smaller increase in blood flow to the pre-frontal cortex.¹³,¹⁴
NEUROHUMORAL AND ENVIRONMENTAL FACTORS

While the relationship between negative symptoms and abnormal cerebral structure has proven to be weak, as implied in earlier hypothesis by Crow and Andreasen, the relationship between negative symptoms and disordered cerebral function has proved robust. With regard to positive symptoms, definitive evidence for a neurohumoral mechanism is still lacking. Changes in symptom severity are associated with a complex pattern of changes in the metabolism of monamine neurotransmitters but many of the observed changes apply to both positive and negative symptoms. Furthermore, the partial success of atypical antipsychotics in alleviating all three of the major dimensions of schizophrenic symptoms shows that negative symptoms are subjects to pharmacological influence in certain circumstances.

So, is schizophrenia a neurodevelopmental disorder or a neurodegenerative disorder? Crow holds the idea of the continuum of psychosis, where schizophrenia is the most severe of the spectrum of psychosis. Advocates of neurodevelopmental disorder feel that the severe illness is caused by increased cerebral ventricular size. There is no certain answer as to why there is a high evidence of schizophrenia in the afro-Caribbean population in the UK when this is not the case in the Caribbean. It is most likely that environmental factors have a major role to play in this conundrum.

CONCLUSION
Schizophrenia is a heterogenous disorder and, on balance, there is good evidence to support for genetic causes, subtle brain abnormalities, hypofrontality and cytoarchitectural abnormality. In most cases, biochemical abnormalities are suspected, where dopamine plays the central role but serotonergic and glutamate system are likely to be responsible also. Non-specific stressful life events often provoke the disorder and play a part in the outcome. What is required is an integrative approach, as the conflicting notions about this disorder have greatly hampered the research and the progress in the understanding and management of this most disabling disorder.

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