A Comment on Late-Onset Schizophrenia

To the Editors:

"Late-Onset Schizophrenia: An Overview" by M. Jackuelyn Harris and Dilip V. Jeste (Schizophrenia Bulletin, 14:39-55, 1988) is an excellent review on the topic. The authors provided a fine integration of the material and a very appropriate analysis of the abundant methodological problems.

It is my opinion that much of the controversy about whether the onset of schizophrenia can occur over the age of 40 is more a matter of semantics than a disagreement about facts. If by schizophrenia one means a cluster of symptoms, then late-onset schizophrenia meets the criteria fairly well. However, if by schizophrenia one means the big picture of a unitary disease-like entity that includes family history, medical history, precipitants, etiology, demographic and other epidemiological factors, onset, prognosis, and response to treatment, then this late-onset condition meets the criteria in a less satisfactory fashion. I suspect that Harris and Jeste would be to a substantial degree in agreement with me by their inference that "The likelihood that schizophrenia is not a homogenous entity but a syndrome with clinically and biologically relevant subtypes . . ." (p. 53). They also said, "The main question is whether a syndrome that may be called 'schizophrenia' occurs with any regular frequency after age 40 (or 44)" (p. 52).

The matter of late-onset schizophrenia appears to be relevant to the "primary" versus "secondary" schizophrenia conceptualization of Templer and Cappelletty (1986). In this model the use of primary and secondary is rather similar to this distinction in epilepsy insofar as in both primary epilepsy and schizophrenia the etiology is more constitutional and the secondary forms are more a function of acquired brain injury or condition.

The primary versus secondary schizophrenia distinction is rather similar to the process versus reactive schizophrenia conceptualization heavily researched in the 1950's and 1960's except that the assaults to the brain in secondary schizophrenia are in contrast to the psychosocial assaults presumed in reactive schizophrenia. The reactive and secondary forms of schizophrenia are both described as being characterized by a later age of onset, more adequate psychosocial functioning, and a somewhat more favorable prognosis. Other characteristics of the secondary schizophrenic patient suggested by Templer and Cappelletty include likelihood of having a brain injury, more "positive symptoms" and fewer "negative symptoms" of schizophrenia, less genetic etiology, more of an affective component, greater seasonality of schizophrenic births (presumed to result from infection or some sort of harmful influence), fewer "kernel" or "nuclear" schizophrenics such as catatonics and hebephrenics, and more paranoid schizophrenics, more "atypical" schizophrenia, and more favorable response to antipsychotic drugs.

In the literature reviewed by Harris and Jeste (1988), late-onset schizophrenia tended to have more of an affective component; less dementia; several times as many women than men; olfactory, tactile, and visual hallucinations in addi-
tion to auditory hallucinations; fewer schizophrenic relatives; dominant paranoid symptoms; visual and auditory deficits; brain lesions; and a favorable response to antipsychotic drugs.

Both the late-onset schizophrenia perspective and the Templer and Cappelletty primary versus secondary schizophrenia conceptualization have distinct limitations. The late-onset perspective is limited by much of the material coming from the softer clinical rather than harder scientific literature. The Templer and Cappelletty conceptualization is limited by being theoretical and, although buttressed by research evidence, has gaps that need to be confirmed or negated by further research. Nevertheless, the Harris and Jeste article and the primary/secondary overview seem to provide mutual and encouraging, albeit limited, support.

References


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Reply to Templer

To the Editors:

We want to thank Dr. Templer for his kind comments about our review article (Harris and Jeste 1988).

Dr. Templer’s concept of “primary” versus “secondary” schizophrenia is quite interesting. As he states, his “secondary” schizophrenia differs from the older notion of reactive schizophrenia in that brain damage rather than a psychosocial stressor is believed to be the precipitating factor. The Templer-Cappelletty (1986) subtyping of schizophrenia is also distinct from Crow’s type I-type II classification of schizophrenia (Crow 1980). Whereas Crow’s type II schizophrenia with brain damage is associated with predominantly negative symptoms and poor response to neuroleptics, the “secondary” schizophrenia (with brain damage) is associated with positive symptoms and good response to neuroleptics.

There are, however, some problems with this interesting nosology. DSM-III-R (American Psychiatric Association 1987) has diagnostic categories of organic delusional disorder and organic hallucinosis, in which the psychotic symptoms are secondary to a known brain disorder. We presume that Dr. Templer does not include such patients under secondary schizophrenia. It may also be pointed out that the causative nature of a specific organic insult is usually well established in disorders such as secondary epilepsy. In contrast, demonstrating that a certain kind of brain damage produces secondary schizophrenia (rather than an organic mental syndrome) would be far more difficult. Indeed, it is highly likely that most of the schizophrenic syndrome is associated with brain damage, the exact nature and specificity of which still remain uncertain.

The concept of late-onset schizophrenia is a relatively young one. Whether it is a valid diagnostic entity would depend on satisfactory demonstration of face validity, descriptive validity, predictive validity, and construct validity (Spitzer and Williams 1985). In one recent study (Harris et al. 1988), we described clinical, neuropsychological, and magnetic resonance imaging characteristics of five patients diagnosed as having late-onset schizophrenia according to DSM-III-R. Conditions in the differential diagnosis such as mood disorders, delusional disorder, organic mental syndromes were specifically excluded. Another study (Jeste et al. 1988) reported common features among 36 late-onset schizophrenic patients from four different centers in the United States and Canada. Such features included bizarre delusions, usually of the persecutory nature, auditory hallucinations, chronicity of course, and symptomatic improvement with relatively low doses (typically under 200 mg chlorpromazine equivalent daily) of neuroleptics. The patients did not have clinical or neuropsychological evidence of dementia, and certain symptoms such as looseness of association and inappropriate affect were uncommon. These findings point to face validity and descriptive validity of late-onset schizophrenia. We are pursuing further work to test predictive validity.
and construct validity of this condition.

We do agree with Dr. Templer that it will be valuable to study the contribution of brain damage to the etiopathology of late-onset schizophrenia. In our own studies, we exclude patients with diagnosable and relevant structural brain lesions (e.g., cysts or tumors in temporohippocampal or prefrontal regions) from the category of late-onset schizophrenia. We are, however, following such patients in order to compare and contrast their course of illness with that of patients without similar lesions.

References


Spitzer, R.L., and Williams, J.B.W. Classification of mental disorders.


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