Sluggish Schizophrenia in the Modern Classification of Mental Illness

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Abstract

The concept of latent schizophrenia was developed by E. Bleuler (1911) and was further promoted in the studies of a number of research psychiatric schools of Europe, the United States, Japan, and other countries. In Soviet psychiatry, there is a long-established tradition of studying “soft” forms of schizophrenia (Kerbikov 1933; Rozen- shtein 1933; Brukhanskii 1934). In the systematics of schizophrenia, developed by Snezhnevsky (1969) and his colleagues, sluggish schizophrenia is viewed not as an initial (prodromal) stage of schizophrenia, but rather an independent diagnostic category characterized by a slowly progressive course, subclinical manifestations in the latent period, overt psychopathological symptoms in the active period, and then by a gradual reduction of positive symptoms, with negative symptoms predominating the clinical picture during patient stabilization. Studies are reviewed examining the relationship of constitutional and genetic factors to the clinical manifestation of sluggish schizophrenia. Finally, the importance of methodological considerations and an examination of divergent factors in the U.S. and Soviet concepts of schizophrenia are presented.

The study of the hidden, latent forms of schizophrenia, which had its origin in the research of E. Bleuler (1911), was further developed by a whole group of psychiatric schools in Europe, the United States, Japan, and other countries. Descriptions of insidiously developing forms of the endogenous process, which are fully comparable to the Soviet concept of sluggish schizophrenia, were categorized under various designations—for example, abortive, ambulatory, and nonregressive schizophrenia (Gaupp 1938; Zilboorg 1956; Nyman 1978).

In American psychiatry, especially during the 1950’s and 1960’s, the problem of pseudoneurotic schizophrenia was intensively investigated (Hoch and Polatin 1949; Hoch and Cattell 1959; Hoch et al. 1963). During the last decade, research attention to this problem has been in connection with clinical genetic studies on the disorders of the schizophrenia spectrum (the concept of borderline schizophrenia of D. Rosenthal [1971], S.S. Kety [1971], and others).

In Soviet psychiatry there is a long tradition of the study of “soft” forms of schizophrenia by Kerbikov (1933), Rozen- shtein (1933), Sukhareva (1933), Brukhanskii (1934), Friedman (1934), Melekhov (1963), and others.

In the system of schizophrenia developed by A.V. Snezhnevsky (1969), and his colleagues, sluggish schizophrenia is an independent form (Nadzharov and Smulevich 1983). In a large sample of schizophrenic patients (Zharikov et al. 1973), 38.1 percent of patients in the sample were diagnosed as suffering from sluggish schizophrenia.

Statistics on the incidence of sluggish schizophrenia in the population of the U.S.S.R. range from 1:44:1000 (Gorbatevich and Boyarintseva...

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Within the framework of the schizophrenic disorders, the sluggish form is by no means an initial (prodromal) stage in the manifestation of the schizophrenic psychosis, but an independent diagnostic category (Smulevich 1987). The stereotypical development of the sluggish form, in keeping with the general nature of endogenous diseases, is characterized by a slowly progressive course. The course of the disease is characterized by subclinical manifestations in the latent period, overt psychopathological symptoms in the active period (a continuous course, an attack, or a series of attacks), and then a gradual reduction of positive symptoms, with negative symptoms predominating in the clinical picture during the period of stabilization.

It must be emphasized that one of the cardinal signs of sluggish schizophrenia is the gradual change in symptoms from the least differentiated in the sense of nosological specificity (the latent period) to more characteristic symptoms in the active period and the period of stabilization.

The pivotal symptoms—obsessions, somatoform symptoms, and disturbances of self-consciousness that occur in conjunction with symptoms of a defect—define the clinical picture and persist throughout the course of the disease despite the fact that psychopathological complexes (syndromes) are changing.

On this basis, within the framework of sluggish schizophrenia, clinical studies have defined several variants in which either pseudo-psychopathic or pseudoneurotic symptoms predominate (asthenic, hysterical, obsessive-phobic, delusionalizing, or cenestohypochondrial), as well as variants characterized by paranoid or negative symptoms (sluggish "simple" schizophrenia).

The classification of sluggish schizophrenia presented above is supported by the results of specially targeted clinical-genetic research (Dubnitskaya 1987). The endogenous, progressive course of sluggish schizophrenia reflects the basic features of its genealogical characteristics: in cases of sluggish schizophrenia, as in other endogenous forms, the families of probands show an increased incidence of schizophrenia spectrum disorders as compared to the general population. This statistically significant familial association between sluggish schizophrenia and other forms of schizophrenic psychoses makes it possible to consider all these forms within the framework of the general genetic system of which they form a part.

Sluggish schizophrenia may be viewed as a relatively independent form, intermediate between the schizophrenic psychoses and personality disturbances. The ratio of schizophrenic psychoses is significantly lower among the relatives of probands with sluggish schizophrenia than among the relatives of probands with more floridly psychotic forms of schizophrenia. However, the relatives of sluggish schizophrenic probands show a wider range of forms of personality disorders—of the schizoid type and all other major types. The frequency of personality disorder among first-degree relatives of the sluggish probands is higher than in the general population.

The contribution of constitutional personality genetic factors to the genesis of sluggish schizophrenia can be seen in the tendency of the disorder to run symptomatically "true to type." In the families of probands, one observes, in accordance with the clinical variation of the disease shown by the proband, a differentiated inclination toward monomodal psychopathies: schizophrenia with obsessions and compulsions—psychasthenia (compulsive personality); "hysterical schizophrenia"—psychopathy of the hysterical type; paranoid schizophrenia—paranoid personality disorder; nonsymptomatic form of schizophrenia—schizoidia, etc.

Thus, the combined evidence on the familial contribution to the expression of sluggish schizophrenia suggests that there are two types of genetic determinants—process related and constitutional (Dubnitskaya et al. 1988). In agreement with the findings of genetic-mathematic analyses of genealogical findings, sluggish schizophrenia is characterized by a specific genotypic structure, the separate components of which appear to be common for different clinical forms (overt schizophrenic psychoses on the one hand, and constitutional personality disorders on the other).

Intrinsic in its relation to the genetically determined group of schizophrenic variations under investigation is the nonsymptomatic form ("simple" sluggish) of schizophrenia, in which the structure of familial incidence is confined to disorders of the schizophrenia spectrum. The clinical characteristics of each of the remaining variants of sluggish schizophrenia are related to the influence of constitutional genetic factors of a nonschizoid nature (familial psychopathological predisposition to hysterical, psychasthenic, and paranoidal types).

In considering diagnostic principles used to define sluggish
schizophrenia, it is essential to emphasize that the development of diagnostic criteria in Soviet psychiatry has remained on the clinically descriptive level; quantitative methods are much less fully developed.

The diagnosis of sluggish schizophrenia demands an integrated approach that is based not on any distinguishing, differentiating pathognomonic symptom or symptoms, but on a complex of clinical indicators. Attempts to define the boundaries of sluggish schizophrenia that rely on models developed for fully developed psychotic forms of schizophrenia and primarily based on symptomatology criteria alone are likely to prove inadequate.

As part of the diagnostic process, information on the premorbid personality development in childhood, puberty, and adolescence must be taken into account. The appearance in the premorbid periods of atypical or bizarre interests (Lichko 1985), but also sharp characterological changes of limited duration, accompanied by a “breakdown of the professional standard” (Mundt 1983) and transformations in all aspects of the “life curve,” acquires great significance for the establishment of the endogenous nature of the subsequently appearing symptoms. Information on family incidence (cases of familial schizophrenia) and on impaired social adaptation is also significant. In contrast to borderline conditions, a gradual reduction of the ability to work, linked with a decline of intellectual activity and initiative, is noted in cases of schizophrenia.

Pathological manifestations that are used as clinical criteria in the diagnosis of sluggish schizophrenia are grouped into two basic categories: pathologically productive symptoms (positive psychopathological symptoms) and negative symptoms (the appearance of a defect state). From our point of view, for the recognition of sluggish schizophrenia, the latter is not merely obligatory, but also verifies the diagnostic verdict: The diagnosis of sluggish schizophrenia may be established only in the presence of definite symptoms of a defect state.

In this manner, provision is made for the exclusion of conditions which are defined not so much by the influence of the endogenic process (latent, residual) as by the “interaction” of personality and environment (Magnusson and Ohman 1987).

An integrated model of the schizophrenic defect state (Smulevich and Vorobyov 1988) forms the basis for the reliability of the diagnosis of sluggish schizophrenia. As part of this model, negative symptoms are not restricted to any isolated sphere of mental functions (e.g., affective, cognitive, volitional, or personality), but to one degree or another encompass all the levels of mental activity.

This approach can be distinguished in principle from several other concepts of the defect state, which confine the defect to relatively isolated spheres of mental activity. The first of these can be traced back to the study by J.H. Jackson (1958) and has been developed on the clinical level by H. Ey (1954), J. Mazurkiewicz (1980), and A.V. Snezhnevsky (1969). This concept implies the development of a defect in the initial stages or during the slow evolution of the process which affects the higher spheres of mental activity and is expressed by symptoms of personality disharmony, which are represented first of all by schizoidia.

The second concept, first expounded by K. Conrad (1958) with regard to the reduction of energetic potential on the clinical level, was more fully developed by G. Huber (1966). Huber proposed that in schizophrenia, the first or even the only areas to be affected involve elements of the highest level of neural activity, which are responsible for psychological activity (e.g., spontaneity, alertness, initiative, vitality, and concentration).

The concept of a multilevel system, analogous to the organization of highest neural activity (Bernstein 1947; Luria 1970; Pribram 1971; Polyakov 1976; Bekhtereva 1980; Bratus’ 1988), provides the theoretical basis for the integrated model of the schizophrenic defect. As in the close mutual bonds of separate substructures that form a “functional organ” (Leont’ev 1975), changes that occur as part of a disease process cannot exist in isolation and inevitably will affect other psychological functions. A change in one area will be reflected to one degree or another in the activity of all areas of the system.

On the psychopathological level, the integrated model of defect is represented by the combining of changes similar to psycho-sympathetic and pseudo-organic (Vnukov 1937) changes. In sluggish schizophrenia, disease-related changes, being inseparably linked, correlate among themselves

\[ \text{1 Asthenic defect and manifestations of a reduction of energetic potential are also related to the clinical appearance of the pseudo-organic defect together with intellectual decline.} \]
within the framework of two variations of the defect, which may also be regarded as diagnostic criteria. In one of these variations the defect is accompanied by hypotypical disintegration of personality, and in the other by hypotypical disintegration of personality\(^2\) (Luk’yanova, in press).

In cases of a defect with hypotypical disintegration of personality, one finds crude changes similar to psychopathy, which are revealed as a result of the decline in the level of personality (pseudo-organic defect).

The complication of disharmony of personality is evidenced by polymorphic psychopathological symptoms along with an increase of discordant psychological processes, eccentricity, pathological autistic activity, and many inadequately motivated actions that are linked closely to emotional blunting and a decline of insight. Cognitive disturbances take place as well as unexpected outpourings of ideas and a tendency toward paradoxical thinking and overvalued bizarre ideas.

In cases of a defect with hypotypical disintegration of personality, pseudo-organic changes are accompanied by impoverishment in all the spheres of mental function, and reflected in the structure of schizoid manifestations (withdrawal from social contacts, emotional impoverishment, flattening and toning down of individual personality traits).

The deepening of pseudo-organic defect features occurs due to ergic and cognitive manifestations—marked decline in intellectual activity, loss of initiative, apathy, reduction of intellectual functions and ability to think abstractly, difficulty in comprehension, and increased rigidity.

In diagnosing sluggish schizophrenia, the two following types of productive psychopathological manifestations are simultaneously noted: Type 1—symptoms predominantly typical for the endogenous process from the moment of its formation; Type 2—symptoms which display an endogenic process-related transformation during further stages of the disease.

The first group contains mild manifestations appearing as episodically occurring exacerbations: Verbal hallucinations of a commentative, imperative character; “calling out,” “sounding of ideas”; somatic hallucinations, tactile hallucinations; rudimentary ideas of being influenced, persecution, special meaning, etc.

The group of positive symptoms which display a transformation typical for endogenic process in later stages of the disease consists of the following:

- Hysterical conditions with a transformation of conversion and disassociative manifestations to the cenestohypochondrial, subcatatonic, pseudohallucinatory ones;
- Obsessive-phobic conditions with subsequent change of appearance from simple phobias to generalized ideo-obsessive conditions (“madness of doubts,” contrasting phobias) through ideo-obsessive delusion with ambidententious ritual conduct and abstract composition of symptoms;
- Hypochondriacal conditions with progression from neurotic and overvalued-type hypochondria to cenestohypochondrial, due to the appearance of somatoform symptoms and cenesthesias, essential cenestopathies, and of possession symptoms;
- Depersonalizations which change their appearance due to the development of persistent disturbances of id, manifestations of autopsychological depersonalization (alienation of higher emotions, awareness of personal psychological changes, of the inadequacy of the entire psychological life);
- Overvalued ideas with the changes of the clinical picture in stages: Overvalued ideas—overvalued delusions—systematic paranoid delusion lacking coherent composition with paradoxical, unrealistic content.

The breakdown in facial expression, which gives the features of the sick individuals the appearance of strangeness, oddness, and eccentricity has an auxiliary, though, in the opinion of modern European psychiatrists (Glatzel 1978, 1981; Weitbrecht and Glatzel 1978), extremely vital importance for diagnosis.

A disregard for the rules of personal hygiene, “neglectfulness,” inattention to grooming of one’s hair or clothes; characteristic avoidance of eye contact; awkwardness, jerkiness, “twitchiness” of body movements; and bombastic speech with exaggerated meanings but lacking in adequate intonation. The totality of these characteristics of the expressive sphere which gives a sense of unusualness or alienness was defined by H.C. Rümke (1958) as “Praecoxgefühl” (“praecox feeling” in English).

From materials presented above, the conflict between the clinical diagnostic criteria used to define sluggish schizophrenia in the U.S.S.R. edition and those of DSM-III-R (American Psychiatric Associa-
tion 1987) becomes obvious. If the diagnosis of schizophrenia according to DSM-III-R assumes the obligatory presence of typical psychotic symptoms (delusions of an outlandish nature, predominantly verbal hallucinations of a commentative character, incoherent thoughts, catatonic behavior, flattened or clearly inadequate affect), then in the U.S.S.R. system these symptoms are certainly not always necessary for the diagnosis of sluggish schizophrenia.

The indicated divergence represents, in the opinion of a number of Western psychiatrists, proof of the existence in the U.S.S.R. of an expanded concept of schizophrenia. However, in reality this divergence does not lead to disagreement about the limits of sluggish schizophrenia, but rather reflects the principal differences of approach to the qualification of groups of patients who are defined by this concept.

In DSM-III-R, wherein Axis I the criteria of syndromal diagnosis are introduced, the conditions that correspond in the understanding of Soviet psychiatrists to the different variations of sluggish schizophrenia are separated from the category of endogenous diseases and are included in the category of "personality disorders." It follows therefore that the cause of divergences between Soviet and American psychiatric schools (if one is to judge by DSM-III-R) lies not in the differences of approach to the nosological definition of conditions which are defined by the concept of "sluggish schizophrenia," but by the principal differences of approach to these conditions of formal classification. An analysis of DSM-III-R criteria for personality disorders demonstrates this. First of all, the category of schizotypal personality disorders includes conditions that in aggregate are unilaterally defined in Soviet psychiatry by the concept "sluggish schizophrenia." It is not by chance that in the previous edition (DSM-III; American Psychiatric Association 1980), latent, simple, borderline, and residual schizophrenia were listed as synonyms for schizotypal personality disorders.

Another category is that of borderline personality disorders. The insufficient preciseness of initial diagnostic criteria (and, above all, of the possibility of a diagnostic overlap between borderline personality disorders and schizotypal personality disorders as indicated in DSM-III-R as well) allows a range of intense, impulsive, unstable manifestations that belong to certain variants, from our point of view, of sluggish schizophrenia to fit within the boundaries of personality disorders.

Attempts to define the boundaries of sluggish schizophrenia using DSM-III-R diagnostic criteria do not appear to be correct. The model of schizophrenia as defined in DSM-III-R applies to manifest psychotic forms of this illness, which cannot be used in the identification of sluggish schizophrenia.

In conclusion, it must be emphasized that the grouping of non-manifested mental disorders of an endogenic-process nature with neurotic personality manifestations and deviations and transitory reactive conditions on a syndromal basis has been widely criticized, even by American psychiatrists (cf. Kendler 1985). The conditionality of this unification is reflected in DSM-III-R. This is shown in part by a large degree of overlap between sets of diagnostic criteria for the prodromal and residual phases of schizophrenia and for schizotypal personality disorders (Axis I of DSM-III-R). Axis II of DSM-III-R corresponds to the breakdown of ontogenesis and personality. In the section on "Differential diagnosis of schizophrenia," DSM-III-R points out the difficulties of distinguishing borderline and schizotypal personality disorders from the prodromal phase of schizophrenia.

It is evident that the problem of the relationship of slowly progressing endogenic diseases and the borderline mental conditions cannot be resolved by a "transfer" of psychopathologies from one of the nosological groups to another. Further study, with an objective comparison of the results of the studies of different national schools of psychiatric thought, of the role of factors which unite different forms of psychopathology (inheritance, similarity of psychopathological manifestations, level of social adaptation, treatment strategy, etc.) is essential, as well as a more accurate definition of the factors which reflect the heterogeneity of those forms (difference of the course, terminal conditions, and particular reactions to psychogenic, somatogenic, and other stresses).

References


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