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Relationship between Presumed Etiological Factors and Clinical Picture in 100 Schizophrenic Males

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Abstract. The schizophrenic syndrome, in one form or another is a result of combinations of genetic, organic and psychosocial factors. 100 schizophrenic males were studied and etiological factors such as schizophrenia or affective illness in direct relatives, brain damage or temporal lobe epilepsy, an over-protective parent or latent homosexuality were isolated. The findings show a relationship between these etiological factors and the clinical picture and course and an attempt is made to use the etiological factor in classifying schizophrenia.

Introduction

The historic approaches to classification in the medical sciences are based on phenomenology, cause and response to therapeutic intervention (6). Classification in schizophrenia has been based either on the phenomenological-evolutive approach (*Bleuler's* prognosis classification (3)) or on the phenomenological approach alone (*Schneider's* or *Carpenter's* classification (4, 16)).

The attempt to use the etiological factor in classifying schizophrenia is problematic. Clinicians have not favored one etiological model exclusively, but rather utilized several complementary perspectives (generally chosen from the physical or psychosocial groups, but not from both) in order to synthesize their personal view of the nature or cause of schizophrenia (19).

In current diagnostic concepts, schizophrenia is referred to as an imperfectly defined syndrome that encompasses patients with different etiologies, divergent pathogeneses, various treatment requirements and heterogenous outcomes (5).

The schizophrenic syndrome, in one form or another, probably appears as a result of a combination of genetic, organic and psychosocial factors, serving as various codeterminants with etiological significance. The relative contribution of these factors varies from one type of schizophrenia to another, from person to person and from one episode of the same disease in the same person to another

(10). During the past decade a number of new studies (9, 15) have followed *Heston's* original work on the adopted-away offspring of schizophrenic mothers (8) and have focused on the role of heredity in the etiology of schizophrenia.

Another interpretation of schizophrenia claims that what is generally transmitted is a potentiality which is transformed in clinical actuality by special circumstances of life originating in the family environment (1). One explanation for the family abnormalities associated with schizophrenia is that abnormal communication between family members might represent the family's response to the disorganizing impact of the schizophrenic member (14).

The organic factors are considered in papers concerning symptomatic schizophrenia (13) or schizophrenia-like psychosis of epilepsy (18). The theoretical importance of these factors, in relation to schizophrenic etiology, lies in understanding the following parallelism: such symptoms as primary delusions and passivity feelings produced by amphetamine intoxication or by a temporal lobe lesion by the same mechanisms which are involved in the nuclear group of schizophrenias (12).

In the present study, I studied a group of schizophrenic patients in an attempt to find a relationship between presumed etiological factors (genetic, organic and dynamic-familial) and the clinical picture.

Methods

The study was conducted on 100 male schizophrenic patients hospitalized during the years 1970–1974 in Ness Ziona Psychiatric Hospital.

All patients had been diagnosed as schizophrenics and were hospitalized at least once. Follow-up evaluations were conducted, on the average, 4 years after initial admission. I have not included in the study patients whose first psychotic episode occurred after the age of 45 (so as to exclude involutional factors), patients with clinical findings that indicate organic brain damage and patients who lacked follow-up evaluations for at least two years after the first hospitalization. All patients were 20–50 years old when last examined.

The clinical records of these 100 patients were reviewed so as to determine presumed etiological factors, as well as diagnose the clinical syndromes and clinical course.

The presumed etiological factors which were considered are the following.

Genetic Factors

In order to study the genetic factors in our sample, I sought familial incidence of schizophrenic or effective illness in first-degree relatives.

Organic Factors

I located the cases of head concussion with loss of consciousness but without neurological or electroencephalographical (EEG) findings which occurred one to five years prior to the first schizophrenic outbreak.

I sought the testimony of relatives describing a history of minimal brain damage (MBD) in childhood, particularly a history of hyperactivity, learning disorders and distractibility, problems of impulse control and lack of response to reward and punishment.

The EEG of all these patients was recorded and these data are described elsewhere (17). Temporal lobe epilepsy (TLE) was diagnosed for the first time during these studies. None of the patients had a history of epileptic seizures. The criterion for the diagnosis of TLE was the finding of spikes or sharp waves in the temporal lobe on the EEG.

Dynamic and Familial Factors

Many of these patients were tested with a battery of psychological tests. We isolated those patients who showed signs of latent homosexuality in the Rorschach test, Thematic apperception test (TAT) and Human drawing test (HDT). I sought over-protective parents or even a symbiotic relationship between patients and their parents.

In diagnosing the clinical pictures, four clusters were employed, whose starting point was the brief psychiatric rating scale (BPRS):

(1) *Depression:* Somatic concern, anxiety, guilt feelings, depressive mood.

(2) *Thought disorder:* Conceptual disorganization, grandiosity, suspiciousness, hallucinatory behavior, unusual thought content.

(3) *Anergia:* Emotional withdrawal, motor retardation. blunted affect.

(4) *Activation:* Tension, excitement, disorientation.

The assessment of the clinical course was based on the mean of 4-year follow-up and a distinction was made between cyclic and continuous course. In the assessment of the outcome I distinguished between patients who suffered from personality deterioration (with defect), and those who did not (without defect).

Results

The principal findings of this study are presented in tables I and II. Table I shows the distribution of presumed etiological factors. One or more of the considered etiological factors have been found only in 75 patients of the sample. The most frequent factors are schizophrenia in first-degree relatives (27% – 27 patients), over-protective parent (16%) and head concussion (11%). These three factors frequently overlap. Less frequent factors are latent homosexuality (8%), affective illness in first-degree relatives (5%), MBD (5%) and TLE (3%).

Schizophrenia was found in first-degree relatives of more than half of the patients who suffered from head concussion (6 out of 11). Most of the patients with an over-protective parent (9 out of 16) have schizophrenics in their families. The over-protective mother is more common than the over-protective father (8 versus 1 in the genetic group). It should be noted that in all cases, the over-protective parent was the non-schizophrenic one. No combination between organic factors such as MBD and TLE, and schizophrenia in first-degree relatives, was found.

In order to compose table II one or two clinical clusters (usually two) were diagnosed, either concurrently or successfully. Of the 75 patients in whom etiological factors were found, 74.6% had thought disorder, 36% suffered from depression, 36% were in a state or anergia and 32% were hyperactive. The course of illness was cyclic in 65.3% and continuous in 34.6%. The outcome in 53.2% of the patients was with defect.

Table I. Presumed etiological factors in 100 schizophrenic males

Item	No. of patients	With genetic factor		With organic factor			With dynamic or familial factors	
		schizophrenia	affective illness	head concussion	minimal brain damage	temporal lobe epilepsy	latent homosexuality	over-protective parent
Genetic factors								
Schizophrenia in first-degree relatives	27	–	–	6	0	0	2	9
Affective illness in first-degree relatives	5	–	–	1	2	0	0	0
Organic factors								
Head concussion	11	6	1	–	–	–	1	1
Minimal brain damage	5	0	2	–	–	–	0	0
Temporal lobe epilepsy	3	0	0	–	–	–	0	0
Dynamic and familial factors								
Latent homosexuality	8	2	0	1	0	0	–	–
Over-protective parent	16	9	0	1	0	0	–	–
Total	75	17	3	9	2	0	3	10

Table II. Relation between presumed etiological factors and clinical picture in 100 schizophrenic males

Presumed etiological factors	No. of patients	Clinical clusters				Course		Outcome	
		depression	thought disorder	anergia	activation	cyclic	continuous	without defect	with defect
Genetic factors									
Schizophrenia in first-degree relatives	27	7	25	13	6	16	11	9	18
Affective illness in first-degree relatives	5	3	3	0	4	5	0	4	1
Organic factors									
Head concussion	11	5	5	2	8	9	2	6	5
Minimal brain damage	5	5	0	2	0	4	1	3	2
Temporal lobe epilepsy	3	2	1	0	0	3	0	3	0
Dynamic and familial factors									
Latent homosexuality	8	4	8	0	2	5	3	7	1
Over-protective parent	16	1	14	10	4	7	9	3	13
Total	75	27	56	27	24	49	26	35	40

An analysis of table II suggests that some etiological factors influence the nature of the clinical picture. The cluster of depression with cyclic course and outcome without defect, is correlated with MBD (5 out of 5) and TLE (2/3). On the other hand, there is a weak relationship between this diagnostic cluster and the head concussion (2/11) and no correlation at all with TLE, latent homosexuality and affective genetic factor.

The cluster of activation is well related to head concussion (8/11) and does not exist in MBD and TLE. The negative results concerning MBD and TLE have little importance because of the small number of cases in these groups.

The data in columns dealing with the clinical course of table II show a high percentage of cyclic course in most cases, especially in affective genetic factor (5/5), TLE (3/3), MBD (4/5) and head concussion (9/11), as well as a good outcome (without or with minimal defect) in TLE (3/3), latent homosexuality (7/8) and affective genetic factor (4/5). A continuous course and outcome with more defect is found in patients with an over-protective parent (13/16) and schizophrenia in first-degree relatives (18/27) which are usually overlapping.

Discussion

In summarizing these findings it should be recalled that the etiological factors studies in this work are assumptions. No causal relationship between these factors (except perhaps the hereditary factors) and the schizophrenic syndrome, has been proven to date.

Considering the findings, an overlapping between schizophrenia in first-degree relatives, an over-protective parent and head concussion should be noted. The relationship between schizophrenia in first-degree relatives and head concussion could be explained either by the fact that head concussion functioned as a trigger in individuals with genetic vulnerability, or that such individuals are more prone to accidents and head concussion. The highly over-protectiveness in families of schizophrenic patients, especially the non-schizophrenic over-protective parent, may result from the fear of the healthy parent of the genetically transmissible disease.

The absence of combination between organic factors such as MBD and TLE, and schizophrenia in first-degree relatives suggests the existence of subgroups with organic etiology in the schizophrenic syndrome. One of these subgroups may be the anxiety endogenic syndrome, a main manifestation of adult brain dysfunction, i.e. adults who had MBD as children (2, 11). Another subgroup may be the schizophrenia-like psychoses of temporal lobe epilepsy (7, 18).

In patients with schizophrenia in first-degree relatives and an over-protective parent, there is a clear tendency to a continuous course, with defect in personality and a predominance of anergia. On the other hand, patients, in whose

families affective illness was found, tend more towards a cyclic course and outcome without defect; the clinical picture is varied, without an anergia cluster. Organic factors are clearly associated with a cyclic course. One should specifically note that the patients with head concussion tend more towards activation, whereas patients with TLE and MBD tend more to depression. Our TLE patients also always had a cyclic course and the outcome was without defect.

Findings of latent homosexuality are accompanied by more thought disorders without anergia. In these patients there was only slight incidence of personality deterioration.

One should be cautious when dealing with the findings of this pilot study, particularly since it was carried out by one person, who served both as the therapist and the evaluator. It would therefore seem desirable to repeat the work in a larger, better controlled study.

However, some interesting questions have been raised which might serve as working hypotheses for further research. The personality, with its particular psychophysiological specificity resulting from the interaction between genetic and environmental factors, colors the specific psychotic activity. The exacerbation of the psychotic activity is the result of overactivity of one or more brain systems. Different abnormalities in one or more brain systems are responsible for the different clinical pictures, which are combinations of mood, kinetic and thought disorders, each of which is dominated by a different brain system. The clinical course is influenced by the reactivity of the whole brain; periodicity is correlated with good reactivity, chronicity with bad reactivity.

The outcome is a result of both the premorbid personality and the stimulus situation, i.e. a good outcome (without defect) is a result of a good psychophysiological state and a therapeutic stimulus situation.

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