

INTERPRETATION OF SCHIZOPHRENIA

Heredity and Constitution in Schizophrenia

SILVANO ARIETI MD

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Silvano Arieti, M.D.

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Heredity and Constitution in Schizophrenia

I

Introductory Remarks

The organic studies of schizophrenia outnumber by far the psychological ones. The quantity of these works and the variety of directions that they have taken reveal that no breakthrough has been made. Even in fields like genetics and biochemistry where some evidence of the organic nature of schizophrenia has been collected, the results are unclear and controversial. Some approaches popular in the 1920s and 1930s have been totally abandoned and would seem absurd today, for example, the studies that investigated the supposed tubercular origin of schizophrenia or an etiologic connection with rheumatic fever. The constitutional, cardiovascular, endocrinological, and neuropathological researches have lost ground. On the other hand, the genetic, biochemical, and neurophysiological approaches have gained support.

I have not engaged in research designed to find organic changes

in the somata of schizophrenics. Nor can I review even a large part of published works on the subject; there are simply too many of them. Only those works will be mentioned that retain a promising outlook or that had an important historical role at a certain period, since schizophrenia was recognized as a psychiatric syndrome. For a greater coverage of the somatic studies done on schizophrenia in the last fifty years the reader is referred to other works (Lewis, 1936; Beliak, 1948, 1957; Beliak and Loeb, 1969; SivaSankar, 1969; Weil-Malherbe and Szara, 1971; Cancro, 1971, 1972; Kaplan, 1972).

Many findings reported by numerous authors do not necessarily indicate a direct etiological connection with the disorder. They may represent a greater variability of the schizophrenic in his physiological functions, relative to the general population.

It could also be that rather than the organic origin of schizophrenia, some of the reported changes indicate the psychosomatic aspect of the disorder. In fact, the psychological changes occurring in the psychosis so disrupt the usual habits of life and have such repercussion on the individual that they can presumably produce alterations of the somatic functions. These

altered somatic functions in their turn may lead to organic pathology in certain cases. In other words, psychosomatic effects might be formed not only in milder psychiatric conditions but in psychoses too. In addition, some somatic changes may be compensatory mechanisms or reactions to previous changes. A chain of altered functions leading to clear-cut anatomical pathology may be engendered. Some of my studies concerning functional changes involving the nervous system have already been mentioned (Chapters 23, 24, 25). The role of the central nervous system will be further studied in Chapter 30. This chapter will be devoted to genetic studies.

Heredity has been studied in reference to schizophrenia since the early period of classic psychiatry. Genetic research is of two major kinds. The first consists of collecting statistical data from family studies and from surveys of total populations to determine whether the morbidity risk is higher than expected in certain families or other groups. By definition the disease expectancy of a person is “the risk of becoming ill during one’s lifetime, if one lives long enough to pass the period of risk” (Stromgren, 1950). The period of risk must be considered that part of life during which the disease may develop. According to Kallmann (1959) the expectancy of the schizophrenic

disorder in the general population is 0.9 percent. The specific genetic studies of schizophrenia aim at determining whether the incidence increases (or, theoretically, decreases) in certain families or other groups.

A second set of studies consists of investigating in schizophrenics special physical characteristics (typically, the chromosomes) that are connected with genetic characteristics.

II Statistical Studies

Statistical studies have generally consisted not only of collecting data, but also of determining whether these data could be interpreted in accordance with Mendel's laws (and their derivatives). On Kraepelin's request, Riidin was probably the first author to make significant attempts to prove the assumption that schizophrenia was determined by genetic Mendelian traits. Although he continued to believe in the genetic origin of schizophrenia, he had to abandon the idea that the disorder was transmitted by a single Mendelian trait. Later (1916) he advanced the theory that the disorder was caused by recessive genes.

Kallmann (1938, 1953) conducted two of the most important investigations into the genetics of schizophrenia; the first on the relatives of over 1,000 patients of a Berlin psychiatric hospital, the second on 953 twins from psychiatric hospitals of New York State. Although Rosanoff and co-authors (1911, 1931, 1934) had already studied the incidence of schizophrenia in twins, Kallmann's work was the one that inspired a large amount of similar research. Kallmann (1959) called the twin-study method a "quasi-experimental procedure." It permits the study of two genetically different types of twins—one-egg (monozygotic) twins and two-egg (dizygotic) twins. Monozygotic twins, who of course are always of the same sex, are supposed to have the same genetic sets. Kallmann concluded that single-factor inheritance of recessive type is the genetic cause of schizophrenia. However, the single recessive factor is modified by the action of other genes. Kallmann found that the full siblings of patients have a higher expectancy (11.5-14.3 percent) for the disorder than half siblings (7.1 percent), and half siblings have a much higher expectancy than step-siblings (1.8 percent). He found the highest incidence of schizophrenia in the children of two schizophrenic parents and in monozygotic twins.

Kallmann's work was criticized by Beliak (1948) on the grounds that he excluded from his studies "the schiziform psychoses," which lack hereditary traits, thus obtaining different statistical results; by Pastore (1949) on the grounds of his statistical analysis technique; and by Koller (1957) on the grounds that if simple recessivity is the hereditary basis of schizophrenia, the incidence should be higher among the sibs than the children of schizophrenics, whereas Kallmann's figures indicate the opposite.

Böök (1960), reviewing the work of many authors, as well as his own work, reports that the incidence of schizophrenia is higher in the family of schizophrenics than it is in the general population: in parents, approximately 12 percent; in siblings, 9-12 percent; in grandchildren, 3 percent; in first cousins, nephews, and nieces, 2 percent.

Slater (1951) found a concordance rate of schizophrenia to be 14 percent in dizygotic twins and 76 percent in monozygotic twins. In a recent presentation Slater (1968) reaffirmed his conviction that genetic factors play an important role in schizophrenia. According to him the increased incidence of the disorder in certain families is

consistent with a genetic hypothesis.

Other authors have reported different findings. In the case of identical (monozygotic) twins studied in Finland, Tienari (1968) found a concordance rate of schizophrenia inferior to that found by other authors: 6 percent of all the cases are taken into account; 10 percent if only the diagnoses made in hospitals are taken into consideration. Kringlen (1967, 1968, 1970) found the concordance rate of schizophrenia to be 25-38 percent in monozygotics and 4-10 percent in dizygotics.

Pollin and co-authors (1969) studied a series of 15,909 pairs of veteran twins. In this large series the concordance rate of schizophrenia for monozygotic twins was found to be 3.3 times greater than the dizygotic rate. In contrast the difference for psychoneurosis was only 1.3. These results suggested—according to the authors—the presence of a genetic factor in the pathogenesis of schizophrenia and its relative absence in psychoneurosis. However, the role of the suggested genetic factor appeared to be a limited one: 85 percent of the affected monozygotic pairs in the sample were discordant for schizophrenia.

III

Studies of Specific Families

These studies have been very interesting and accurate but unfortunately inconclusive. Rosenthal (1963) reported on a set of quadruplets. All the members of the set were found to be schizophrenic, but their disorder presented various degrees of severity and different symptomatology. Swanson, Brown, and Beuret (1969) reported a family with nonschizophrenic parents and four schizophrenic children, possibly five if we include the controversial diagnosis of another child, an alcoholic. The authors feel that the occurrence of schizophrenia in at least four out of six children suggests a heterozygous or dominant transmission; but contradicting this hypothesis is the fact that neither parent was afflicted. One would have to assume that “reduced penetrance of the dominant factor could exist in one or both parents.” The authors prefer to attribute the most important role to psychological environmental factors.

Scott and Ashworth (1969) have reported their studies of schizophrenics coming from families where schizophrenia had already appeared, but they give a psychodynamic interpretation to their findings rather than a predominantly genetic one. They explain a new

way “in which schizophrenia may be visited upon the third and fourth generation.” The authors feel that “if the parent of a schizophrenic had an experience in earlier life of psychotic illness in a close relative, this psychotic ancestor may act as a powerful aetiological factor, operating primarily through intrafamilial expectations and relationships.” The authors found several cases in which a parent experienced the patient as he experienced once his mentally ill relative. In some cases the parent literally saw the patient as the “same as” the mentally ill relative and destined for the same fate. The image of the ill relative had been present as a secret fear; the parents were looking for a duplication of such image both in themselves and in their children. The parents, however, deny such fear, and this denial produces a state of depersonalization and unrelatedness that increases the psychopathogenic effect of the family on the prospective patient.

IV

Chromosomal and Other Physical Data

Barr and Bertram’s (1949) accidental discovery of “chromatin satellite bodies” in the neurons of males and females was a breakthrough for many genetic studies concerning chromosomes. In

1956 new techniques (Tjio and Levan) permitted the visualization of forty-six chromosomes in men. Since then much research on chromosomes of schizophrenic patients has been carried out. Money and Hirsch (1963) studied 1,700 mentally defective patients. Among them they found two males who were the XXXY configuration with forty-eight chromosomes, and three females who were XXX with forty-seven chromosomes. Two of these five patients (one male and one female) were schizophrenic and had schizophrenic relatives. Raphael and Shaw (1963) studied cytogenetically 210 patients who were schizophrenic but not mentally defective. In this group they found one XXY male with forty-seven chromosomes and one XXX female with forty-seven chromosomes. They concluded that specific abnormalities of sex chromosomes are more frequent among schizophrenics than in the general population. They could not exclude, however, a chance association.

Kaplan and Cotton (1968) found an unusual incidence (more than 0.5 percent) of mosaicism involving X-chromosome aneuploidy in 986 female patients diagnosed as schizophrenics and institutionalized in Ohio State Hospital.

In an unpublished work quoted by Norman Brill (1969), Judd and Brandkamp (1969) failed to find “any significant or constant chromosomal abnormalities” in a series of forty adult schizophrenics, half of whom had family history of schizophrenia. As a result of their study of sixteen patients suffering from childhood schizophrenia, as well as of similar studies by other authors in both adults and children, Siva Sankar and Saladino (1969) concluded that no chromosome anomalies can be correlated with schizophrenia with any statistical significance. Sankar and Saladino pointed out that the situation in childhood schizophrenia is quite different from that of Turner’s, Klinefelter’s, and Down’s (or mongolism) syndromes, where chromosomal anomalies were found. The authors postulate that “a molecular lesion at the ultrastructural level of the chromosome” may be responsible for the disorder.

Kallmann (1959) visualized various studies of discordant monozygotic twins (one schizophrenic and the other nonschizophrenic) to determine the factors responsible for the discordance. These studies are being carried out now by many authors. A multidisciplinary study of discordant monozygotic twins is being conducted at the Clinical Center, National Institute of Mental

Health (NIMH), Bethesda, Maryland. Investigation includes psychiatric, psychological, physiological, and biochemical variables studied in each of the monozygotic twins. In one of such studies, Stabenau and others (1969) found that the lactate-pyruvate ratio and particularly the lactate production were higher for the schizophrenic than the nonschizophrenic twins. The authors thought that their findings permit distinguishing between schizophrenic and nonschizophrenic populations. They felt, however, that it was not clear as to whether the biochemical distinction derived from the presence of schizophrenia, from some metabolic process directly related to the schizophrenic process, or was influenced by circumstantial factors, such as medication, hospitalization, or diet.

V Review of Additional Studies and Interpretations

Altshuler (1957) summarized the findings of various observers up to 1957 and came up with the following expectancy rates of schizophrenia: general population, 85 percent; half siblings, 7-8 percent; full siblings, 5-15 percent; parents, 5-10 percent; children of

one schizophrenic parent, 8-16 percent; children of two schizophrenic parents, 53-68 percent. Taking into consideration specifically twin studies, Altshuler (1957) reported the following expectancy rate: concordance in dizygotic twins, 3.3-16.7 percent; concordance in monozygotic twins, 66.6-86.2 percent. These statistics confirm the impression held even by laymen for centuries that psychoses run more frequently in certain families.

I feel that additional conclusions can be drawn:

1. No Mendelian trait for the transmission of schizophrenia has been found.
2. The available data do not permit explanation of the incidence of schizophrenia solely on the basis of some Mendelian laws.
3. The findings obtained in the genetic studies of well-known hereditary diseases, such as hemophilia, muscular dystrophy, Huntington's chorea, and others, could not be repeated for schizophrenia.

We must, of course, keep in mind that in the statistical studies of schizophrenia we encounter special difficulties that do not exist in studying the hereditary diseases that we have mentioned.

Schizophrenia is not so clearly definable or diagnosticable as, let us say, hemophilia or Huntington's chorea. The less typical cases can be confused with other personality disorders. Moreover, the various methods or facility of hospitalization and the various degrees of environmental toleration or ambulatory treatment make statistics less reliable. Even after making allowances for all these possibilities of error, which various authors have tried to correct in various ways, it seems obvious that schizophrenia cannot be explained with the traditional Mendelian laws. To find a possible explanation, some authors have advanced the idea that the hereditary genes can be altered by different degrees of "penetrance" or "expressivity." Other authors, for instance, Kallmann (1959), believe that genes, other than the one that carries the disease, may modify that gene and confer a greater or lesser degree of resistance upon the individual. Other authors believe that a set of several genetic factors is necessary to determine the disorder. Denber and Teller (1963) hypothesized a primary chromosomal failure, and alteration in nucleotide sequences in both DNA and RNA and other factors.

As a result of his statistical studies, Karlsson (1966) believes that his data are consistent with "a modified dominant inheritance" of

schizophrenia. A dominant gene, associated with a thought deviation, and the recessive gene, “associated with a state of nervous tension,” could together determine an incompatible situation, usually leading to schizophrenia.

Manfred Bleuler (1968) believes that heredity plays an important role but not a specific one. He cannot believe that hereditary disposition to schizophrenia “consists of the morbidity of one or two or three genes which are altered by mutation. ’ ’ First of all, no Mendelian law could be determined in families of schizophrenics; secondly, “the fertility rate of schizophrenics is very much reduced and, in spite of this, schizophrenia remains a very frequent psychosis.” He adds, “If we assert that mutation of a gene is the main background of schizophrenia, we must conclude that the mutation rate is very high. As a matter of fact it would have to be much higher than any known mutation rate.” Bleuler believes that the hereditary background of schizophrenia is most probably a disharmony, an insufficient interplay of different predispositions of the personality. In other words, the genes would transmit physical disharmonies that run parallel to disharmonies of personality. These genetic disharmonies of personality would interact with environmental factors and the

combination would result in schizophrenia.

Gottesman and Shields (1966), after reviewing genetic studies, including their own, conclude, “It seems reasonable to postulate that genetic factors are largely responsible for the specific nature of most of the schizophrenics and that these factors are necessary but not sufficient for the disorder to occur.” Even Slater (1968), who, as we have already seen, is one of the strongest advocates of the genetic nature of schizophrenia, states that the genetic factors are “largely responsible but not wholly responsible.” Other authors, like Kringlen, believe that the genetic factors are somewhat responsible, but not largely responsible.

There are, of course, other factors to be considered. Families may transmit schizophrenia not genetically, but psychodynamically. We have seen in detail how some authors like Jackson, Lidz, and Wynne interpret this psychological transmission and how I interpret it (see Chapters 5 and 8). With particular reference to identical twins, we cannot exclude that they have an additional psychological problem—an identity problem that may predispose them to a schizophrenic decompensation (Kringlen, 1967, 1968).

In my long psychiatric career I have had the opportunity of examining accurately and treating only three pairs of monozygotic twins. Coincidentally, all three pairs involved female patients. I have also treated many other patients who were twins, males and females, concordant by sex, but not monozygotic.

The first pair was treated by me in 1943, and I reported their cases in the literature (Arieti, 1944*b*). At that time I was very much influenced by the hypothesis of the antischizophrenic defense mechanisms advanced by Kallmann and Barrera and tried to interpret why one of the sisters, Magda, made a recovery, whereas Selma remained delusional, withdrawn, and required indefinitely long hospitalization. In agreement with the theory of Kallmann and Barrera (1941) I reported that the recovery occurred in the patient who was constitutionally the more athletic and less asthenic. I added that the recovery occurred in the patient whose prepsychotic personality was definitely more extroverted, who had always shown better ability to cope with the problems of life, and whose symptomatology was somewhat atypical because of the presence of many “psychoneurotic symptoms.” I also saw a connection between Magda’s mild hypochondriacal symptoms and Selma’s somatic delusions. Actually

there was much of psychodynamic significance in this history of the patients, which I was not able to evaluate fully at that time. There were identity problems between the two girls. Until the age of 12 they were always dressed alike. They were so similar that they were often misidentified. The mother always tried to emphasize their similarity by insisting upon dressing them alike. Once I asked the mother why she wanted to do so, and she answered, "I was proud of them. I liked to see them alike." However, the two girls did not like to dress identically and always objected to this wish of their mother, although they did so unsuccessfully. They resented the fact that they were the object of everyone's attention on account of their similarity. Magda, in particular, began to consider her being a twin as something of which she was ashamed. After the age of 12, the sisters succeeded in dressing differently. At that time, according to the mother, they started to be jealous of each other, each of them being especially jealous of the other's dresses, which by now were different. Then, too, the first differences in their personalities became apparent at that time. Up to the age of 12, the patients were both vivacious and sociable children. From that age Selma became more seclusive and reserved. She preferred to be alone; when in company she was considered shy and

bashful. In the major areas of life the two sisters continued to show similarities of interest and actions, Magda retaining the role of leadership. In their teens they both started to work as domestics; Magda had her first boy friend when she was 16; Selma when she was 18. Magda was the first one to leave the parental home and became pregnant without being married. Selma always repeated Magda's events of life one or two years later. But one event she did not duplicate. At the age of 22 Magda got married, but Selma remained single. She could not relish her sister's marriage and started to hate her brother-in-law. She became delusional, withdrawn, had to be hospitalized, and although treated with sixty-three insulin treatments, she remained delusional. For details of her illness, the reader is referred to the original report (Arieti, 1944b).

As soon as Selma was admitted to the mental hospital, Magda started to express somatic complaints. Subsequently she revealed her fear of becoming insane and repeatedly stated that because her twin sister was insane, she would become insane also. She soon presented paranoid trends against her husband, accusing him of jealousy, although there was no foundation for such belief. The episode lasted a few months. Magda made an apparent recovery, was discharged from

the hospital, and there were no relapses.

The second pair consists of Edith and Barbara, identical twins who lived together for twenty years, that is, from the time they left the parental home to the time Barbara got married. Edith felt betrayed and was unable to adjust to the separation. She could not understand how the sister who had been “part of her” could become so disinterested in her now that she had a husband. Edith had always been afraid throughout her life that the day would come when Barbara would get married. The day finally came, and Edith considered Barbara cruel, sadistic. Barbara had always been the dominant one when they lived together, but now she was much worse. Edith started to express feelings of unreality and somatic delusions and had periods of intense panic and disorientation. Her saying to herself, “You are not only half you. You are you and must live independently,” did not help much. Her work declined in quality, but she was able to maintain her position. Edith underwent intense psychotherapy, and hospitalization was not necessary. Barbara did not present any symptoms.

The third pair (Henriette and Paulette) has already been mentioned in Chapter 20 in relation to Henriette’s artwork. Both

Henriette and Paulette exhibited a quasi-schizophrenic symptomatology since their late teens. But whereas Paulette improved after her marriage, Henriette, who remained single and alone, became much worse and eventually developed a full-blown psychosis after separation from her sister.

These three pairs of twins, although not enough to be statistically significant, show very well that the state of being an identical twin engenders additional problems for the patient. At times both siblings may be affected by the disorder, or only one, but in all cases the twin who was the dominant one in the relation is also the one who suffers less from the separation and the one who is less likely to have the psychosis. If he, too, develops the psychosis, it is the milder of the two. The psychodynamic development of twins differs from that of nontwins but it acquires pathological features in disturbed families only. The Oedipal situation is altered, the dominant sibling assuming a parental role. The submissive sibling tends to remain dependent and, in pathological cases, unable to face life if deprived of the assistance and approval of the twin.

Other authors have described how the experience of being a

monozygotic twin may pervade the patient's entire life. Rosenthal (1974) wrote:

Identification with another person reaches its strongest point in such genetically identical individuals. The literature on twins is replete with all kinds of experiences and lore that bear on this intense communality or psychological bond. If one twin develops an illness, the other is likely to do so as well. This sharing of fates is an ongoing, integral part of their development and learning. Therefore, if one twin develops a schizophrenic illness, the likelihood of the second twin's developing that illness is increased inordinately, and the concordance rate for monozygotic pairs should be appreciably higher than that of dizygotic pairs.

These hypotheses are put in doubt by the work of Allen and Pollin (1970). In a sample of 31,818 male veteran twins, 1.14 percent were found to be schizophrenic. This incidence of the disorder was thus similar to that in the general population, suggesting that there is no clear difference in incidence of schizophrenia between twins and nontwins. The incidence of the psychosis in monozygotic twins was no greater than in dizygotic twins. Their data thus did not support the hypothesis that the experience of being an identical twin increases the occurrence of the disorder.

In order to separate the effect of the family as a psychodynamic factor from that of the family as a genetic factor, the incidence of schizophrenia in identical twins reared apart was studied. Slater (1968) collected data about sixteen twin pairs (monozygotic) who were separated early in life and one or both were schizophrenic. He found that of the sixteen twin pairs, ten were concordant and six discordant.

Rosenthal (1974), after having evaluated all the evidence, concludes that a hereditary factor in the etiology of schizophrenia has been sufficiently demonstrated. However, he believes that environmental factors are also important. If schizophrenia were engendered only by heredity, all monozygotic pairs would be concordant for schizophrenia, whereas in half or more of the pairs, the twin sibling, although having exactly the same genes, is not clinically schizophrenic.

Rosenthal (1963) divided the genetic theories of schizophrenia into two main groups: (1) the monogenic-biochemical, which assumes that a single pathological gene (responsible possibly for a specific metabolic error) is either necessary or sufficient to cause

schizophrenia; (2) diathesis-stress theories, according to which what is inherited is a predisposition to develop the illness. A neural integrative defect may be at the basis of the diathesis.

Even such psychodynamically oriented authors as Rado attributed an important role to genetics in the etiology of schizophrenia. Rado and co-workers (1956) accepted the distinction between genotypes and phenotypes in relation to schizophrenia. Genotype is the inherited cause of development; phenotype the final outcome. The genotype, through interaction with the environment, causes the schizophrenic phenotype (called also the schizotype) to develop an abnormal organization. Similar interactions are well known in the field of pathology. For instance, an allergy, let us say, to hay may be based on a hereditary factor, but is transformed in actuality by the presence of a particular antigen in the environment: for instance, a pollen. However, environmental situations psychological in nature presumably act with more complex mechanisms than a simple antigen.

VI Conclusions

In Part Two of this book we have seen that psychodynamic factors are prerequisites to schizophrenia. No schizophrenic who was examined psychodynamically was found to come from a well-adjusted, harmonious family and to have had no serious conflict. However, the findings reported in this chapter indicate that a genetic predisposition is necessary to channel the psychogenic factors along certain patterns and to provide the additional elements necessary to that interplay of causes and effects that eventually leads to the disorder. If this point of view is correct, we can draw the following conclusions:

1. At least two sets of factors of different origins (biological and psychodynamic) are necessary to engender schizophrenia.
2. It is sufficient to prevent or remove one set of these factors in order to avoid schizophrenia.
3. Genetic factors cannot be altered. We must thus try to alter or prevent psychodynamic or environmental factors.

There are some authors, however, who, perhaps with some justification, are alarmed about the increasing danger of genetic factors. Erlenmeyer-Kimling et al. (1966) and Rainer (1966) report that the reproductivity rate for schizophrenic women in the period

from 1934-1936 to 1954-1956 has increased from 58 percent to 87 percent. The more effective the therapeutic procedures will be, the higher will be the reproductivity rate of patients who had been diagnosed as schizophrenics. Consequently the larger will be the number of children to whom this genetic factor can be transmitted.

VII Constitutional Factors in Schizophrenia

In 1921 Kretschmer published *Physique and Character* (the American edition in 1925), a work that remained popular in psychiatric circles for about two decades. Advancing his theories concerning personality and constitution, he divided body build into three types: (1) the leptosomatic (or asthenic), characterized by angular profile, narrowness of muscles, small shoulders, flat chest, relatively long extremities, and little muscular development; (2) the athletic type, with strong skeletal and muscular development; and (3) the pyknic type, characterized by pronounced deposits of fat on the trunk, short extremities, and gently curved profile.

Kretschmer saw a certain correlation between schizophrenia and body type. According to his studies, two-thirds of schizophrenics are

leptosomatic or athletic and one-tenth dysplastic.

Sheldon, Stevens, and Tucker (1940) and Lindegarde (1953) refined Kretschmer's rather crude technique, and many authors have adopted their method generally to confirm that there is a greater incidence of asthenic constitutions among schizophrenics. Rees (1957) and several other authors, including myself, however, unlike Kretschmer, have not confirmed a lower incidence of pyknic constitutions among schizophrenics.

Kline and Tenney (1950) studied 455 hospitalized patients with the Sheldon method. Mesomorphy was generally correlated with good prognosis and paranoid type; ectomorphy with hebephrenic type and poor prognosis. Many clinicians have noted a change in the constitutional appearance of the patient, in accordance with the improvement made. Many patients tend to become more mesodermic when they get better. This is a rule of thumb and is by no means true in every case.

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