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Lecture

Different Causative Factors in Different Forms of Schizophrenia

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Definite conclusions about the origin of schizophrenia concerning hereditary as well as psychosocial factors can be obtained when different forms of schizophrenia are investigated separately.

We still know much too little about schizophrenia. This applies particularly to its origin. Being an older psychiatrist, I am well aware of the extensive and intensive research work performed since the time of Kraepelin and Eugen Bleuler. Unfortunately, though, there has not been much progress. After the First World War, when Kraepelin and Bleuler were still alive, most psychiatrists believed they could regard schizophrenia as an inherited disease. This was generally accepted in Germany where the doctrine of Rüdin dominated research on the genetics of schizophrenia. However, he was influential not only in Germany, but in many other countries, too. For example, Kallman in the USA worked with Rüdin for several years. Rüdin and his pupils knew the extent of the gap which existed between practical observations and theoretical expectations according to Mendel's laws, and tried again and again to bridge the gap. They did so in vain. Many hypotheses were established. Some authors suggested a dominant mode of inheritance, others suggested a recessive heritability. Neither of the contrasting views held up to scrutiny. Nevertheless the belief that heredity was crucial continued, and this had disadvantageous effects on psychiatric research for, in consequence, the existence of other causative factors was completely disregarded. The result in Germany was horrible: from the alleged exclusive heritability of the endogenous psychoses Hitler derived the foundation of his laws by which he forcibly sterilised and even killed so many patients.

Partly as a reaction to these crimes, afterwards heredity was disregarded. Psychoanalytical and psychosocial theories now became completely dominant. When the North-Americans came to Germany after the Second World War they attempted to convince us that schizophrenia would occur if a baby had been frustrated and had not been given his mothers milk in good time. This was the era of the "schizophrenogenic mother". Such psychoanalytical theories could not be upheld for long, though some of the psychosocial views arising subsequently were psychoanalytically oriented. The theories established by Lidz, Bateson, Wynne, Singer are generally known and very often cited. There has been much discussion of these theories, but after some attempts at replication, again, they did not hold up. Now many psychiatrists have returned to genetic theories and regard inheritance as decisive in causing schizophrenia. You can again find attempts at revealing a Mendelian mode of genetic transmission though all such attempts failed as long as 50 years ago. The wheel has turned full circle.

In my opinion, this regrettable state is due to the fact that schizophrenia is taken as a single entity, even though Eugen Bleuler did not speak of schizophrenia—but of "a group of schizophrenias". Indeed, definite results can be obtained concerning heredity as well as psychosocial factors when different forms of schizophrenia are investigated separately.

During the 15 years after I retired, I was able to devote myself entirely to research and was in a position to complement our knowledge of schizophrenia (through clinical and genetic examinations of more than 1400 patients). The classification on which my investigations are based have been made known in the English language from the publications by Fish, (1958, 1962, 1964), Astrup (1962), Perris (1960, 1974); and as far as my differentiation between the monopolar and the bipolar depressions is concerned, from many other publications. My classification has been presented in detail by Ban

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et al (1981). And my book *The Classification of Endogenous Psychoses* has also appeared in English (Leonhard, 1979).

Here, the "systematic" schizophrenias correspond to the "typical" schizophrenias, especially described by the late Japanese psychiatrist, Mitsuda. They constitute the "nuclear" schizophrenias and in almost all cases run an insidiously progressive course. The "non-systematic" schizophrenias correspond to the "atypical" schizophrenias and are predominantly remitting or even periodic. Also in their symptoms pattern they differ from the systematic forms (see Fish, 1958; 1962).

Among the endogeneous psychoses, 5 groups have to be differentiated. First: the systematic schizophrenias; second: the non-systematic schizophrenias; third: the cycloid psychoses must be separated from schizophrenia. They never lead to mental deterioration, and each phase is followed by complete recovery. The fourth and fifth groups comprise monopolar depressions and manic-depressive illness. These two forms are now generally accepted. In my opinion, there are also monopolar manias, the existence of which is mostly denied. The schizo-affective psychoses are not among the five groups. I cannot regard them as an independent group because their prognosis is indefinite. Some of these with schizo-affective psychoses recover from every phase and, in my opinion, have cycloid psychoses. Others tend to deteriorate, more and more, from shift to shift, and belong to the non-systematic schizophrenias. Whether the one psychosis is present or the other, can be identified from the distinct psychiatric picture. Thus the schizo-affective psychoses have to be separated into these two different groups (Leonhard, 1983).

The five groups of endogenous psychoses, especially the two schizophrenias and the cycloid psychoses, have quite different aetiologies. First, different inheritance: 604 systematic schizophrenics whom we examined had 1403 siblings-only 29 of them, i.e., 2.1% were ill; our 429 non-systematic schizophrenias had 744 siblings—117, i.e., 15.7% were ill. In these figures Weinberg's correction has been used. Individuals under 17 years have not been counted, individuals aged from 17 to 40 have been counted half, individuals over 40 have been fully counted.

So, in the severest forms an extremely small family history of psychotic disorder is found. In these forms psychossoal factors must be looked for. Genetic loading is also low in the cycloid psychoses, but not to so large an extent as in the systematic schizophrenias. Our 221 cycloid psychotic patients had 393 siblings—16 of them, i.e., 4.1 per cent were ill. Thus family history of psychotic disorder is low in the severest as well as in the mildest forms of the so-called schizophrenias. It is striking that our absolute figures differ so greatly in size. This is due to the fact that we undertook our investigations in hospitals where chronic patients were prevalent. In hospitals with predominantly acute psychoses the cycloid forms are as common as the systematic and as the non-systematic schizophrenias.

I found a low hereditary loading in the systematic schizophrenias when I started my investigations into the endogenous psychoses at the beginning of my psychiatric life; but over the years I could not detect any external influences. On the basis of the examination of twins I ultimately succeeded. I have personally examined 45 monozygotic twins and 25 ill co-twins, and, in addition, 47 dizygotic twins. Among the latter all forms of endogenous psychoses occurred. By contrast, in the monozygotic twins systematic schizophrenias were completely lacking. This was extremely surprising and totally unexpected. The finding applied to the 45 index-twins and the 25 ill co-twins as well. It seems that we encountered an external factor involved in the causation of the systematic schizophrenias. The finding gives rise to the conclusion that identical twin-ship can protect the individual from falling ill with this special kind of schizophrenia. How can this be explained? I think we must give consideration to the fact that monozygotic twins being identical in their somatic and psychical constitution understand one other to so complete a degree that they cannot become entirely autistic. And we know since Eugen Bleuler that autism is a central symptom of schizophrenia. The close communication with the co-twin seems to compensate sufficiently for the little contact which may exist with other people. Following our explanation we can assume that, in turn, lack of communication, lacking even one single friend, can encourage the development of schizophrenia.

Our findings contradict the psychosocial theory according to which identical twins have difficulties in finding their identity and therefore both of them are prone to the development of schizophrenia. On the contrary they are liable to schizophrenia to a lesser degree. However, I must add that other forms of endogenous psychoses accumulate in twins. I will return to this later.

Many authors have discussed the significance of isolation and lack of communication in the origin of schizophrenia. They could not obtain definite results as they took schizophrenia as a unity. Moreover, they predominantly deal with the concept isolation as it is present in adults, whereas, according to our observations lack of communi-

cation is only predisposing towards schizophrenia when operating in childhood.

Communication between twins is psychosocial in nature, lack of communication is also a psychosocial factor; but the effect on the patient seems to be organic, for the systematic schizophrenias, which in most cases terminate in serious deterioration, certainly have an organic basis. This kind of influence is possible in childhood and adolescence when the nervous system has not matured. Isolation can result in such consequences. Let me quote the brain pathologist Seitelberger (1980):

> "The maturation not only of the sensory system and the motor apparatus, but also of all parts of the brain which of necessity work as a unit, is dependent on the external demands made. It has been shown that systems which are kept from functioning by means of isolation do not attain their morphological maturation and remain non-functional. On exposure to external influences after the critical maturation period has been passed the deficit can no longer be compensated. The processes of morphological differentiation are to a continually decreasing degree demonstrable in the nerve-cells and their associative fibres until the end of the second decade of life. So, these changes accompany the processes of mental maturation in the individual."

So, the organic effect of isolation can be demonstrated morphologically. Such an influence was observed in the children described by Spitz (1945) in whom lack of communication resulted in severe somatic disturbances; many of the children died though they had been well fed, and children who survived became severely mentally retarded.

In the cycloid psychoses nothing is suggestive of an organic basis. Accordingly their origin has nothing in common with that of the systematic schizophrenias. As regards heredity, surprising results appeared when we differentiated between two sub-forms, anxiety-happiness-psychosis and motility psychosis. In both these cases the family history of psychiatric disorder is small, in siblings 4.1% and 4.2% respectively. Nevertheless the concordance rates in identical twins differ greatly. Of our six pairs with anxiety-happiness-psychosis only one was concordant; this conforms with the low hereditary loading. But of our 11 pairs with motility psychosis no less than 9 had also fallen ill. From this latter result the geneticists would derive an extremely great heritability which, in fact, does not exist. In motility psychosis no more siblings were ill than in anxiety-happiness-psychosis. In the parents, there were fewer ill individuals in motility psychosis (5.1% versus 6.3%). The old rule established by Galton and thereafter being an important guideline for geneticists, loses its validity with the cycloid psychoses. Indeed, in spite of the extraordinarily high concordance rate in identical twins the heritability of motility psychosis is low. I will give an interpretation of this finding later. In our twins with monopolar depression the concordance rate was also high though relatively few parents and siblings were ill.

Alarmed by the finding in our twin studies we focused on sibships, their sizes, and the position of the patient among the siblings. In this regard many investigations have been carried out by other authors, again considering schizophrenia as a whole. Various and conflicting results have been obtained. We again differentiated between different forms and many definite results became apparent. I came to the conclusion that siblings by mutual influences contribute to the development of the individual in childhood and adolescence and do so not only psychologically but also biologically. On this basis the influence of siblings can prevent or, on the contrary, encourage the appearance of schizophrenia. Childhood friends may play the same role as siblings.

Let me consider cycloid psychoses in this respect. Striking results appeared when I compared motility psychosis with another sub-form, confusion psychosis. There were relatively many siblings in the first illness and relatively few in the latter. However, the difference applied only to the older siblings. In our series, 100 patients with motility psychosis had 172 older siblings, and 100 patients with confusion psychosis had only 78. The number of younger siblings differed little (127:108). This latter difference is not statistically significant. I conclude that the alleged mutual influence of siblings occurs in the first years of life when younger siblings either do not exist or are so helpless that they cannot exert any influence on older siblings. This fits into the psychoanalytic view about the first years of life. I do not discuss the question why it applies just to these cycloid psychoses whereas in other endogenous forms the external influence is operating during later childhood. And I am not able to answer the question why the existence of many siblings contributes to the origin of motility psychosis and the lack of siblings to the origin of confusion psychosis. However, I point out an interesting parallel. Motility psychosis often occurs in twins. Of our 92 twins 22 (11 monozygotic, 11 dizygotic) suffered from this cycloid psychosis. Perhaps the close relationship existing in twins corresponds to the increased relationship resulting from a greater number of siblings and thus either condition enhances the morbidity risk for motility psychosis. Probably siblings can stimulate each other too much so that the nervous system can be overstrained.

At this point I should like to return to the question of why there are some different concordance rates in identical twins with anxiety-happiness-psychosis and, on the other hand, with motility psychosis. In the latter case the influence is mutually exerted, both twins are involved. In contrast, in anxiety-happiness-psychosis there is only a one-sided influence. From other findings, I came to the conclusion that tensions in childhood can create a disposition to anxiety-happiness-psychosis and jealousy of the inferior twin toward the partner can represent the basis of such tensions. In this case for the dominant twin there is no reason to be jealous and he will remain well. So the influence is one-sided. One of our identical twin pairs gives support to this interpretation. The index twin sustained a fracture of the vertebral column at the age of 12 and had to lie on a plaster bed for several years. Moreover her father favoured the co-twin. The mother tried to compensate for this, but the father was entirely dominating in the family so that his affection was of greater importance than that of the mother. The index-twin fell ill with anxiety-happiness-

psychosis while the co-twin remained healthy. She became jealous of her sister who was somatically healthy and was favoured by the father. She continuously fought her sister in an attempt to gain ascendancy.

Concerning the non-systematic schizophrenias, I must first point out the high hereditary loading. For one of these forms (periodic catatonia) I could suggest a dominant inheritance pattern. The gap existing between the Mendelian estimates and the empirical findings could be bridged by examining many of the allegedly healthy parents. In fact, many of them were seriously abnormal. We found that in periodic catatonia 22.0% of the parents were ill and approximately as many were abnormal to a high degree. Thereby the Mendelian estimate is nearly matched. However, when the psychosis manifests itself completely external factors must operate in addition.

We found that periodic catatonics have relatively few siblings. This contrasts with the findings in motility psychosis though there are certain similarities between the two psychoses. As in periodic catatonia, many of the parents were psychotic—the time for propagation was possibly limited. But it turned out that healthy parents had only a few more children than psychotic parents. It seems then that the manifestation of periodic catatonia decreases when many siblings exist. Direct evidence of this emerged when we examined how many ill siblings came from large sibships and how many from small sibships. Many more ill siblings were found in small sibships. The figures were 29.4:17.6 in relation to the number of healthy siblings.

Some families seemed to confirm that large sibships can prevent the manifestation of periodic catatonia. This applied to the following family which contained no less than 15 siblings. Two of them had children with periodic catatonia. Thus it can be seen that the hereditary predisposition for periodic catatonia existed in the family. Nevertheless all of the 15 siblings remained healthy. I think the large sibship protected them from falling ill.

What factor might be operating to produce this effect? I suggest that a child feels protected and sheltered when living in companionship with several siblings so that he is more likely to grow up free from anxieties. Such a view fits into some psychosocial assumptions. With respect to periodic catatonia, I can confirm this view. One of our observations seems at first to contradict our interpretation, but on the contrary it supports it when the family is more completely examined. Of 10 siblings, no less than four fell ill with periodic catatonia. The existence of so many children was by no means preventive. The Mendelian estimates were nearly reached; half of the children are expected to fall ill in a dominant inheritance.

But let me describe this family thoroughly: the father was an excessive drinker and most of the time he used to sit in pubs. He used to beat his children brutally. When a little child cried he beat him or he beat an older child whom he declared responsible for the crying. As soon as the father returned home the whole family became silent; and the children made themselves scarce. The mother tried to intervene, but was rejected and was often beaten herself. The children in this family grew up under continuous anxieties; the large sibship could not prevent this.

Another psychosocial theory was not confirmed. Some psychiatrists believe that the abnormal behaviour of a schizophrenic mother can cause an illness in the child. The assumption again has been related to schizophrenia as a whole, but periodic catatonia represents the schizophrenic form in which most frequently parents as well as children are ill, so that the validity of the theory can be tested. Heston (1966) opposed the assumption after he had examined children of schizophrenic mothers who had grown up in a home. Our patients who had catatonic mothers did not have more ill siblings than patients with healthy mothers; on the contrary they had fewer. But in spite of this the index-patients relatively often had psychotic mothers, when there was no older sister. This is astonishing and indicates that an older sister is qualified or even biologically designed to replace the mother who has been lost or is mentally incapable. Now we understand why index-patients with ill parents have not more, but fewer ill siblings than index-patients with healthy parents. In the first case the siblings could remain healthy because most of them had older sisters, for example the index individual who later fell ill. In the event of a healthy mother, the presence or absence of an older sister is without significance. If we are surprised that the existence of an older sister can reduce the risk for periodic catatonia we must remember how unselfishly older sisters often devote themselves to the care of younger siblings after the mother has been lost. According to my findings the above psychosocial theory must be corrected as follows: it is not the abnormal behaviour of a psychotic mother, but loss of the mother in association with absence of an older sister which may be important. However, in any case, the external influence does not cause periodic catatonia which is a hereditary illness, but only increases its manifestation.

Another non-systematic schizophrenia with a hereditary basis is affective paraphrenia. In this psychosis a recessive mode of inheritance is suspected because, on the one hand, we found relatively many ill siblings (13.3%) and very few ill parents (2.2%). On the other hand, I could describe a large family containing 18 affective paraphrenics of whom no less than 13 descended from consanguineous marriages. Both these findings suggest a recessive inheritance. The probability of manifestation is increased in females; in our series 95 affective paraphrenics were females and only 25 males. As far as affective form of paraphrenia is concerned, we may assume that affective lability, representing a personality trait of the women, can promote the onset of the illness.

As regards psychosocial factors, some psychiatrists believe that children become liable to schizophrenia if they cannot acquire the role belonging to them by age and sex. In most forms of schizophrenia I did not find anything fitting this theory, but in affective paraphrenia I did. Repeatedly, I was able to confirm psychosocial theories about the genesis of schizophrenia, but in no case concerning schizophrenia as a whole. Each theory applied only to one of the different forms of schizophrenia.

The affective paraphrenics are frequently the youngest of the siblings. Among our 120 affective paraphrenics there were 36 youngest and only 17 oldest siblings. We know that the youngest children are often kept in the role of a child by their parents when they should be allured to

mature. Observations in identical twins are suggestive in this sense. In one case the maturation of both twins had been retarded because the rigorous and harsh father had suppressed the activity and affectivity of the girls. This could have applied even more to the second born twin who fell ill seven years earlier than her sister. In another case the girls were never able to live an independent life. They were not allowed to undertake any activity without asking permission from their mother. In adolescence they were not allowed to make date with a young man. An incident demonstrates the high degree to which the mother dominated her daughters. When one of them was over 30 years old and had been divorced she wanted to marry another man whom her mother did not approve of. As she attempted to resist her mother she was beaten by her. Later she fell ill with affective paraphrenia. Her identical co-twin was even more submissive and fell ill with this psychosis 22 years earlier than her sister. I did not make similar observations in other forms of schizophrenia.

Maturation is retarded to the same extent when overprotection from the mother makes independent life impossible. I observed this in an identical twin recently. The mother loved one of her children very much and always wanted to keep the girl at home. While the co-twin was with friends the index-twin was with her mother. When, in adolescence, the girl wished to go out with her sister, the mother tried to prevent this and sometimes even shed tears in grief. In contrast, the co-twin had much personal liberty and was always with friends. The different attitude of the mother toward her children had developed because the index-twin was less lively and more submissive. She fell ill with affective paraphrenia at the age of 58. The co-twin remained well until the age of 74. Then she also expressed some ideas of reference; but it was difficult to determine whether she had now become psychotic or she was only susceptible to suggestion in consequence of her old age and had accepted some of her sisters ideas. In any case, the index-twin, who was overprotected by her mother, was much more severely ill. Her maturation had been hampered.

As in this case, identical twins not infrequently have different temperaments. I particularly found this with our affective paraphrenics. In three of our seven cases the one twin was lively, the other calm. In all the three cases the twin with the calm temperament fell ill. Of the partners, apart from the co-twin just mentioned, one remained healthy, the other was much milder ill than her sister. In all cases I saw both twins and personally observed that the co-twin was much more lively. One of them was even markedly hypomanic. We may enquire whether, in the patient described overprotection and submissiveness were crucial, or, perhaps the underlying temperament alone evoked affective paraphrenia. The other two twins had not been overprotected, but were submissive to their co-twins, one of them to a high degree.

Let me now turn to the third non-systematic schizophrenia, the cataphasia, which in its excited pole corresponds to the schizophasia described by Kraepelin. Here we found that the patients relatively often originated from big cities (over 100 000 inhabitants) and relatively rarely from villages (below 2000 inhabitants). The central symptom of cataphasia is represented by a disorder of thought. Thus we might suspect that the function of thinking can be damaged in big cities where there is often an excess of mental stimulation. Cognitive functions development in childhood and adolescence may be overstrained. This is a very interesting aspect which arouses the idea that in our modern time the occurrence of cataphasia must have increased, for technical development has so rapidly progressed and thinking is much more strained than in former times. Indeed, we found the incidence of cataphasia had increased. We examined our 1400 patients during the course of 15 years. We made three groups and separated patients examined in the first five years, in the second five years, in the third five years and then in each group we compared the numbers of patients suffering from the different forms of schizophrenia. The number of cataphasics had increased from group to group, relative to the other forms.

Finally, I should like to discuss manic-depressive illness and "pure" depressions. The hereditary loading of these two forms is more different than other authors have found. When differentiating according to course (monopolar versus bipolar) many manic-depressives are erroneously assigned to the pure depressions as they by chance had only gone through depressive phases, which generally are much more common. The cross-sectional picture is also different in the two conditions. We have differentiated on this basis and found 20.0% ill siblings and 18.3% ill parents with manic-depressive illness; but only 3.1% ill siblings and 5.8% ill parents with pure depressions. In the first case a dominant hereditability can be assumed though external factors must contribute to the manifestation of the illness. In this psychosis the abnormal behaviour of a psychotic parent seems to be significant, for probands with psychotic parents had 28.1% ill siblings, and probands with healthy parents only 15.5%, though these "healthy" parents had the full hereditary predisposition to the illness.

Among our pure phasic psychoses the euphoric form showed still fewer ill relatives than the depressive form. The figures just mentioned (3.1% and 5.8%) include the euphoric form. After separating the two psychoses we found 4.5% ill siblings and 7.8% ill parents in the depressives, whereas in the manias there were 1.2% ill siblings and 2.1% ill parents. In the euphoric form family history is less prominent. As noted above, the existence of pure manias is still predominantly denied. The low figures demonstrate that these patients belong even less than depressives to manic-depressive illness with its high hereditary loading. So monopolar mania is an independent entity, too.

Pure manics have relatively many siblings, pure depressives relatively few. However, just as we found in two of the cycloid psychoses, this difference concerns only the older siblings, so that I again conclude that a mutual influence operates in early childhood. As siblings stimulate each other we can envisage the possibility that too much stimulation in childhood may promote the occurrence of mania, while too little stimulation may promote the occurrence of depression. If this is the case, not only siblings, but also other people (above all parents) can stimulate the child too much or, on the other hand, not enough. Seven

purely depressive probands had a depressive parent (six females and one male), one purely euphoric patient had an ill mother who was euphoric, too. Perhaps manic parents stimulate their children too much, depressive parents too little. Hereditary influences are not decisive, since nearly all of the ill parents were mothers (seven of eight parents).

The finding that manics have many siblings and that depressives have few has an important transcultural aspect. In developing countries mania is relatively common, in industrial countries depression much more frequently occurs. This difference was noted by Kraepelin after he visited psychiatric institutions in Java, There is a parallel, in developing countries families are large, in industrial countries families are small. Various causes may account for the different incidence rate of manias and depressions, but a dependence on family sizes also has to be taken into account. I do not know whether in industrial countries mania was more common in former times when families were larger. In this connection, the question may be raised whether the incidence of schizophrenia has increased in our century. My opinion is: some forms of schizophrenia have increased, others have decreased. The insidiously progressive schizophrenias, i.e., the systematic forms, in my classification, are certainly more common at the present time. Some remitting and periodic forms occurred more frequently in former times.

A similar difference in the occurrence of schizophrenia seems to exist between industrial and developing countries as many authors have described. See Jilek (1974) and Jilek & Jilek-Aall (1970). Motility psychosis is usually assigned to the schizophrenias, and runs a markedly periodic course and probably occurs more frequently in developing countries. Since in these countries the families are larger, this fits with our finding that patients with motility psychosis have relatively many siblings.

Let me close with a general remark. You may ask why I still speak of endogenous psychoses though I presented evidence of many external influences. Why do I not add "so-called"? The external events create a disposition to psychoses in childhood and adolescence. From this time onwards, the individual has an internal propensity from which later the psychosis originates. No external influence is usually operative at the onset of this illness. In the exogenous psychoses the illness immediately follows the external influence.

References

ASTRUP, Ch. (1962) *Schizophrenia. Conditional Reflex Studies*. Springfield, Illinois: Charles C. Thomas.

BAN, THOMAS, A., WILLIAM GUY & WILLIAM, H. WILSON (1981) Psychopharmacology and Leonhard's classification of chronic schizophrenias: IIIrd World Congress of Biological Psychiatry. Stockholm, June 28—July 3.

FISH, F. (1958) Leonhard's classification of schizophrenia. *Journal of Mental Science* **104,** 943–971.

—— (1962) Schizophrenia. Bristol: John Wright.

—— (1964) The cycloid psychoses. *Comprehensive Psychiatry* **5,** 155–169.

HESTON, L. L. (1966) Psychiatric disorders in foster home reared children of schizophrenic mothers. *British Journal of Psychiatry* **112,** 819–825.

JILEK, W. G. (1974) In *Biological Mechanisms of Schizophrenia and Schizophrenia-like Psychoses*. (eds H. Mitsuda & T. Fukuda) Tokyo: Igaku Shoin.

—— & JILEK-AALL, LOUISE (1970) Transient psychoses in Africans. *Psychiatria Clinica* **3,** 337–364.

LEONHARD, K. (1979) *The classification of endogenous psychoses*. New York: John Wiley.

—— (1983) Is the concept of "schizo-affective psychoses" prognostically of value? *Psychiatria Clinica* **16,** 178–185.

PERRIS, C. (1960) A study of bipolar (manic-depressive) and unipolar recurrent depressive Psychoses. *Acta Psychiatrica Scandinavia*. Suppl. 194.

—— (1974) A study of cycloid psychoses. *Acta Psychiatrica Scandinavica*. Suppl. 253.

SEITELBERGER, F. (1980) Das Gehirn und das Nervensystem in psychosomatischen Geschehen. *Universitas*, **35,** 33–40.

SPITZ, R. A. (1945) Hospitalism: An inquiry into the genesis of psychiatric conditions in early childhood. *Psychoanalytic Study of the Child*, **1,** 55–74.

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