v. Discussion and Conclusions

By ELIOT SLATER and A. W. BEARD

The task now arises to indicate the significance which should be given to the findings described in earlier pages. There are three possible theories by which the combination of a schizophrenia-like psychosis with a pre-existing epilepsy might be accounted for. First the combination might be co-incidental, due purely to chance, in which case there would be no medical problem of any consequence. Secondly the occurrence of epileptic fits might tend to precipitate the psychosis in individuals genetically predisposed, who otherwise would have had better chances of escaping such a development. Thirdly the psychosis might be the direct consequence of the epilepsy, and therefore to be regarded as an epileptic and not a schizophrenic disorder in the aetiological sense. Fourthly it would be possible to suppose that in any given collection of cases representatives of more than one of these causal chains could be found. These theories may be discussed in turn.

Epilepsy and Schizophrenia as a Chance Combination

The proposition is advanced that the theory of chance combination is so improbable that it must be abandoned. This is on the following grounds:—

On a basis of chance combination one would expect the appearance of three to five fresh cases of epileptics becoming schizophrenic per annum in a population of ten million. If the incidence of such cases had been no greater than that, it is hardly thinkable that the authors would have been able to collect 69 cases in the course of a few years.

The theory of chance combination leads one to expect to find in our material the indications of a predisposition to schizophrenia in the same proportion of cases and to the same degree as if our patients had been schizophrenics of the ordinary kind. One of the fields in which these indications would have been found would be the family history. Specifically, the hypothesis leads to an expectation of finding a total of about 17 secondary cases of schizophrenia among parents and sibs. What was actually found was 2 such cases, the figure to be expected if the patients had been a sample of the general population, or epileptics of the ordinary kind. On the other hand, the incidence of epilepsy in the parents and sibs of our patients was approximately the number which might be expected from a group of "symptomatic" epileptics.

Indications of a predisposition to schizophrenia should also have been found in the pre-psychotic personalities of our patients. We found no excess of "schizoid" traits of personality before the onset of the psychosis. No good account was usually available of what the personality had been like before the onset of epilepsy. After that time and before the onset of psychosis, the abnormal traits which were found in excess were those known to arise in epileptics as a long-term effect.

Finally, the theory of chance combination leads one to expect in a material of schizophrenic epileptics a fairly normal representation both of epileptic and schizophrenic disorders. This expectation is not borne out. Though both hebephrenic and catatonic states are seen in these patients, the clinical picture in the great majority of cases tends to conform to a fairly typical paraphrenic form. The epilepsy is also of a special type: the great majority of patients have been found to have a focal epilepsy, and three-quarters of all the patients had a temporal lobe focus.

Epilepsy as a Precipitating Factor for Schizophrenia

We now turn to the second hypothesis, that we are observing genuinely schizophrenic psychoses, which have been facilitated by the pre-existing epilepsy.

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On this basis we can predict a rather more frequent appearance of the combination of the two conditions than on the hypothesis of purely chance combination. Thus on one theoretical model (Slater, 1958) it is estimated that about one in four of the individuals with adequate genetical equipment for schizophrenia actually develop the illness. Pre-existing epilepsy might increase the figure of one in four to one in one; and in that case we could expect in a population of ten million the annual appearance not of 3 to 5 schizophrenic epileptics but of 12 to 20. This still seems a very small incidence rate when put in conjunction with our 69 cases gathered over a few years in only two of the many hospitals which serve this region.

The hypothesis that a pre-existing epilepsy predisposes to the development of schizophrenia leads also to the expectation that one will find some indication of a relative abundance of schizoid traits in the pre-psychotic personalities of schizophrenic epileptics and some increase in the incidence of schizophrenia among their relatives. The finding that there is no shift at all towards a raised incidence of schizoid traits of personality, and no increase at all in the incidence of schizophrenia among first-degree relatives counts heavily against this hypothesis.

The hypothesis that a pre-existing epilepsy predisposes to the development of schizophrenia seems to be compatible with an unrepresentative and rather special group both of epileptic and schizophrenic disorders. Thus, on this hypothesis, it might be that a temporal lobe epilepsy was the one which specially favoured the development of schizophrenia, and that it was particularly a paraphrenic type of schizophrenia which was predisposed to. There is, however, nothing in the theory which would lead to such special features being hypothecated *a priori*; and when they are found, subsidiary hypotheses become necessary to explain them.

If the psychoses from which our patients suffered are to be regarded as schizophrenic in an aetiological and not merely descriptive sense, then they must be expected to conform to schizophrenic norms in their main features. While it is true that these patients have shown at times all of the cardinal features of schizophrenia, and indeed over two-thirds of them coming into the hands of independent psychiatrists had been diagnosed as suffering from schizophrenia, the psychoses observed deviate from schizophrenic norms in some interesting respects. During the psychosis itself, as has been emphasized by Pond, affective responsiveness tends to be preserved to an extent which is unusual in schizophrenia. The second principal difference from schizophrenia is in the later stages of development of the psychosis, which tends in the fullness of time to pass off, leaving a personality sometimes substantially undamaged, more often damaged by the personality changes we associate with organic disease of the brain and with long-standing epilepsy rather than with the post-schizophrenic state.

The Schizophrenia-like Illness Purely Epileptic in Origin

If, then, we exclude both the hypothesis of chance coincidence and the hypothesis of a genuine schizophrenia merely predisposed to by epilepsy, we are reduced to the third of our hypotheses, i.e. that the schizophrenia-like psychosis is not, aetiologically regarded, schizophrenic at all, but an epileptic psychosis. This hypothesis also has to be examined on its merits, and the question must be asked whether there is evidence to be brought either for or against it.

All the evidence we have considered so far, i.e. the data bearing on the frequency of schizophrenia-like illnesses in epileptics, on the pre-psychotic personality of these patients, on the incidence of both schizophrenia and epilepsy in their first degree relatives, on the predominance of paraphrenia-like psychotic pictures and of epilepsy of the temporal lobe focal variety, and even on the long-term follow-up results, all this evidence is compatible with the view that the psychoses observed are aetiologically different from ordinary forms of schizophrenia and constitute a special kind of epileptic psychosis. Some of these findings could have been predicted on the basis of that theory, e.g. that schizoid traits would not be found to any great extent in the pre-psychotic personality, and that schizophrenia would be no more frequent in first-degree relatives than in the general population; so that these particular findings can

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be regarded as supporting the theory. Other points, e.g. that temporal lobe foci should be so frequently found, could not easily have been predicted in advance, but once found seem to fall into a consistent picture of aetiological relationships. Their significance will be considered in due course.

An aetiological relationship could exist between the epilepsy and the psychosis in either of two ways. First, the epilepsy itself, i.e. the occurrence of epileptic fits, could be the adequate cause of the psychosis; secondly the basic disorder of function, which manifests itself in epileptic fits, could be the cause of the psychosis. In the first case we should expect the occurrence of a schizophrenia-like psychosis to be associated with special features of the epilepsy, with its long duration, its unusual severity or frequency of fits, with fits of a special clinical type, or occurring at special times such as during the night. Some of these expectations are fulfilled; others are not. These psychoses seem to arise in epileptics conforming to usual patterns in respect of frequency of fits, severity of epilepsy, amount of anticonvulsant drugs consumed, etc. But there is a relatively high correlation coefficient between the age of onset of epilepsy and age of onset of psychosis, with a mean interval of 14 years between the one and the other. Though there was no general tendency in this group of patients for the occurrence of nocturnal fits, such fits were found specially associated with a psychosis complicated by recurrent confusional episodes. The fits from which these patients suffered were also of a rather special type since, as temporal lobe fits, they involved a good deal of abnormal psychic experience. Twilight states with visionary experiences, for instance, were certainly much commoner in this group than in the general run of epileptics. On this basis, therefore, there is evidence to suggest that an aetiological relationship may exist between the epileptic fits as such and the psychosis.

If the psychosis and the epilepsy are related aetiologically in a less direct way, by the psychosis being a late product of a disorder of function of which the epilepsy is an earlier manifestation, then our expectations will be rather different. It is no longer surprising that there should be no relation between the psychosis and severity of epilepsy. The correlation between age of onset of epilepsy and age of onset of psychosis becomes an observation that could not have been predicted. Having been observed, it would suggest that underlying the epilepsy there was a process of some kind, with autonomous developmental tendencies, liable to produce fits at one stage and psychosis at a later one. But if the psychosis is caused by some factor underlying the epilepsy, then one would expect to find evidence of what that factor was. Here again we are rewarded. In the bulk of these patients there is evidence, convergent from a number of quarters, of a lesional basis for the epilepsy.

One such source of evidence was the past medical history of these patients, which showed a history of moderate or severe head injury in 11 cases. Commenting on this, Hill (1962), while accepting the lesional basis of the epilepsy, expressed doubts whether the history of head injury was significant. He remarked that head injury is a rare cause of temporal lobe epilepsy in the young, and that the lesions in our cases lie in the medial structures of the temporal lobe, which is not a site for injuries produced by head injury. This view may very well be correct.* Nevertheless, the history of definite head injuries in as many as 11 cases seems too important to be wholly disregarded. Closed head injuries are exceedingly common, and they are known to cause epilepsy in about 5 per cent. of cases. In a large and recent study, Hillbom (1960) reported that 20 per cent. of 2,047 patients with closed head injuries developed certain epilepsies, and a further 11 per cent. developed uncertain epilepsies and attacks of undetermined nature. Hillbom made a representative selection of 415 patients for special study. Of these 56 had suffered only concussions and contusions, and of them 18, or over 30 per cent., became epileptic, though none of them with temporal lobe attacks. If we bear such facts in mind in conjunction with the belief of

* There are differences of opinion. For instance Landolt (1960) regards the temporal lobes as particularly liable to damage, directly or by contrecoup, in closed head injuries and concussion. All parts, including the anterior poles, may be so affected. Relevant literature is quoted.

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many authorities that the occurrence of epileptic attacks over years may cause a temporal lobe lesion and temporal lobe attacks, then the frequent history of head injury in our cases may not be finally dismissed as irrelevant.

The frequent occurrence of head injury in the past was only one of the anomalous features of the medical history; this also included histories of birth trauma, middle ear and mastoid disease, encephalitis, etc. The lesional basis of the epilepsy was also supported by all the evidence of focal origins, the existence of minimal neurological changes in one-fifth of the patients, the evidence of organic personality changes, the findings of air encephalography indicating atrophic processes in 37 out of the 54 patients tested, the pathological findings in those patients submitted to a lobectomy, and the follow-up results showing a tendency for the psychosis to settle down into an organic deficit syndrome.

Taking all the evidence we have been able to collect, we find that a great part of it is in conformity with an aetiological relationship between the epilepsy, or a pathological process causing epilepsy, and the psychosis; and that there is no evidence against this hypothesis. The evidence is however ambiguous in value and insufficient to let us decide on the nature of the relationship, whether the epileptic fits themselves tend to cause the psychosis, or whether the cause of the epilepsy tends to cause the psychosis. The latter seems on the whole to be the more probable view, but there could be some validity in both.

Possibilities of Heterogeneity

There is finally the fourth hypothesis of heterogeneity. On this basis we would suppose that some of these psychoses were purely chance combinations of epilepsy and schizophrenia, some would be genuinely schizophrenic psychoses predisposed to by the epilepsy, some would be epileptic psychoses. This hypothesis is really a complex of hypotheses and so is undeserving of attention unless solid evidence can be brought for it. Is there any suggestion of heterogeneity in the material we have described?

The answer to this question is very largely in the negative. There are of course heterogeneities, but little suggestion that they are significant ones. Thus we find among the established pathological lesions both neoplastic and atrophic lesions; but these cases do not distinguish themselves from one another or from the rest of the material in any other way. The best we can do in the way of demonstrating heterogeneity is to draw attention to the differences between the three clinical groups A, B and C. It will be remembered that the A group was characterized by frequent occurrence of confusional episodes before the onset of the schizophrenia-like psychosis proper; the B group consisted of patients with predominantly paranoid and paraphrenia-like syndromes; the C group consisted of patients all of whom had hebephrenia-like illnesses. This method of classification naturally brought with it symptomatic differences. Thus the mode of onset was mainly episodic in group A and insidious in the other groups; and the incidence of such symptoms as schizophrenic thought disorder and flattening of affect was disproportionately heavy in the C group. Other differences between the groups, not directly caused by the mode of selection, were not very striking. Only one of the patients in group C had a history suggestive of brain trauma, and in this respect the difference between C and the other groups was highly significant. The age of onset of epilepsy was also significantly earlier in group C than in the other groups, at 9 years on average as against 20 and 16. Finally, nocturnal epilepsy was particularly common in group A.

In his discussion of our material, Hill observed that the cases now included in group C might well be different aetiologically and symptomatologically from the rest, and he suspected that the fortuitous combination of schizophrenia and epilepsy would be found, if at all, in this group. Unfortunately, the group is not large enough to provide evidence whether this is so or not. Thus an examination of the family histories, as suggested by Hill, does not show any difference between the relatives of patients in this group and the relatives of the patients in the other groups, either in incidence of schizophrenia or epilepsy; numbers are too small for an adequate material to be available. All we can say is that, while it is far from

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unlikely that there are one or two genuine schizophrenics in our total material, it is very improbable that there are more than a handful. It would seem to be a practical impossibility to distinguish them individually from the others.

The Pathogenesis of the Schizophrenia-like Psychosis

We now have to consider whether we can formulate any meaningful and testable conception of the pathogenesis of these states on an epileptic basis, and whether we can suggest any reason for their close similarity to schizophrenic states caused otherwise. These are two different questions, both of great importance.

Let us take the first of these problems. There are three outstanding clinical findings, which may be arranged in an order of reliability and significance. First is the focal origin, usually in the temporal lobes. This origin was demonstrated electrically in about three-quarters of the cases; but in the cases in which it was not demonstrated it seems very likely that temporal lobe functions, or limbic lobe functions, were disturbed. This may quite possibly have been the case with origins of fits shown in frontal or central subcortical regions. Thus in two cases, in which the EEG diagnosis was not one of temporal lobe epilepsy, tests of vestibular function showed an abnormality. The limbic lobe system is closely connected with the emotional life of the individual, and when it is disturbed he is liable in a state of clear consciousness to experience affects not connected with external reality, of a compelling and disquieting kind: *déjà vu*, depersonalization, fear, etc. There is also a liability to abnormal perceptual experiences, derealization, hallucination, etc., also occurring without detectable clouding of consciousness. Symonds (1962) rightly emphasized that there is hardly one of the most characteristic symptoms of schizophrenia, of a positive kind, which may not be experienced as an aural phenomenon in temporal lobe epilepsy. Such aural symptoms occurred in a large majority of our patients, and in a number of them were explained by the patient in a delusional way; the aural symptom in fact had entered into the content of the delusional psychosis. Furthermore, a whole group of our patients, group A, had recurrent confusional episodes, and some patients in groups B and C had occasional episodes of this kind. Many of these episodes could properly be called twilight states, since consciousness is dreamlike rather than obscured, and it is possible for the dream material to be retained into recollection the next day with great completeness. Those patients who had religious or mystical experiences of this kind were convinced of their reality and validity, and only regained partial insight years later. Thus one patient (*49*), who had had no fit and no mystical experience for four years, still felt that his religious revelations had been veridical and regretted that he had had no more. Another, who had heard God say "There is no such thing as nothing" (a rebuttal of existentialist philosophy!), felt that since then he had been given an understanding of why things exist.

There is accordingly much to be said for the point of view advanced by Pond (1962), that there is a causal relationship of a psychological kind between fits and psychosis. Pond suggests that "years of attacks of clouded consciousness might lead to a confusion of reality and autistic thinking and experience". The paranoid hallucinatory psychoses which are not uncommon in alcoholics and narcoleptics could be accounted for in this way. These conditions, he says, seem to have in common only disturbances of consciousness which are partial and not of the total kind seen in petit mal.

In its essence, this theory regards the epileptic schizophrenia-like psychosis as depending in the first place on abnormal emotional experiences physically caused, which are then integrated into the totality of psychic life as a psychodynamic process. For the development of a complex and lasting paranoid psychosis out of primitive single abnormal experiences a number of other conditions would probably be necessary. Thus the experience itself should have a strong reality value, so that at the time it occurs the patient does not easily recognize it as hallucinatory, but puts it down to a real event. It should have a strong affect accompanying it, so that it impresses the mind, and once remembered is not easily forgotten. It should occur in such a state of consciousness that it is not sub-

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sequently obliterated by an amnesic gap. It should not be easily dismissed by the patient as of trivial importance, nor seen through on reflection by subsequent use of powers of insight. It must be admitted that the aural symptoms of a temporal lobe epilepsy do tend to fulfil each and all of these conditions, whereas, for instance, the visual hallucinations accompanying a lesion of the visuosensory area do not.

On the other hand, while this theory provides a plausible explanation of the paranoid symptomatology, it seems powerless to explain such other symptoms as volitional disturbances, thought disorder and hebephrenic symptoms. Moreover it was not all of our patients who had aural symptoms or twilight states of a kind which might in time convert the mind to a totally distorted picture of the real world. A more comprehensive mode of explanation is offered by Symonds (1962). Discussing our material, this author wrote:

"If then neither the fits nor the temporal lobe damage can be held directly responsible for the psychosis, what is the link? It may be the epileptogenic disorder of function. . . . Epileptic seizures and epileptiform discharge in the EEG are epiphenomena. They may be regarded as occasional expressions of a fundamental and continuous disorder of neuronal function. The essence of this disorder is loss of the normal balance between excitation and inhibition at synaptic junctions. From moment to moment there may be excess either of excitation or inhibition—or even of both at the same time in different parts of the same neuronal system. . . . The EEG, though it only skims the surface, often records activity of one kind or another in the absence of seizures. The observation that epileptiform EEGs may be observed in 25 per cent. of schizophrenics is not without interest in this connection. What I have called the epileptogenic disorder of function may be assumed to be present continuously but with peaks at which seizures are likely to occur. This background disorder may cause symptoms other than seizures. The presence and nature of such interictal symptoms as do occur are related to the epileptogenic focus. From this focus the synaptic disorder tends to spread along lines that are functionally determined. Thus particular systems of neurons become involved so that the disorder of function may be highly selective. [The schizophrenic-like aural symptoms of temporal lobe epilepsy suggest that] the temporal lobe includes within its boundaries circuits concerned with the physiological basis of the psychological disorder which we call schizophrenia . . it is not loss of neurons in the temporal lobe that is responsible for the psychosis, but the disorderly activity of those that remain, and this disorderly activity is of the kind that is also likely to cause seizures."

So much may be said in connection with the predominantly temporal lobe origin of the fits in our patients. The second striking finding which emerges from the clinical analysis is the correlation between age of onset of epilepsy and age of onset of psychosis. This evidence that the duration of the epilepsy in itself plays a causative part in bringing on the psychosis is reconcilable with Pond's psychodynamic concept, but does not seem to be suggested by Symond's physiogenic hypothesis. Once there is an epileptogenic disorder of function involving temporal lobe circuits, with abnormalities occurring interictally as well as ictally, one might expect psychotic symptoms to appear at any time. It is true that in a few of our patients the psychosis did come on very quickly after the first epileptic fits. However, the evidence seems too strong to resist the conclusion that the chronic paranoid psychosis is a phenomenon which is specially likely to occur at a particular stage during the lifetime of the epilepsy, a stage in fact at which organic damage has been done.

This brings in the third of our clinical findings of central importance, the evidence of actual destruction of nerve tissue, as shown by the AEGs and by pathological examination in cases in which a lobectomy has been carried out. The AEG was done in 56 cases; in 17 the pictures were passed as normal; in 37 cases there was evidence of brain atrophy, and in 2 cases evidence of events of another kind. This evidence of a destructive lesion is paralleled and supported by clinical evidence of a dementing process. Even at the time when these patients first came under investigation four-fifths of them showed the psychiatric indications of organic personality change. As the disease continued these indications continued to multiply; and at the time of the follow-up, just about half the patients contacted were socially handicapped by organic deficits.

It would seem to the authors that, if the illuminating hypothesis proposed by Symonds is accepted, it would need some modification. He comments that temporal lobe damage by itself is unlikely to cause a schizophrenia-like psychosis. It may be then that in the atrophic processes that we have found, it is not the cells

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that have died that are causing trouble but those that are dying. This suggestion falls in both with a late onset of psychosis, and with the tendency of the psychosis in the fullness of time to pass off into an organic deficit.

Our final conclusion then must be a somewhat inconclusive one. Both the modes of explanation suggested by Pond and by Symonds have evidence in their favour and seem likely to account for at least a part of the observations. Our own tendency is to think the physiogenic causation of the psychosis the more important factor, and to regard it as predisposed to by a certain stage in what is in the long run a dementing process.

Significance of the Illness as a Symptomatic Schizophrenia

While the occurrence and the nature of these psychoses certainly raise issues important for the study of epilepsy, their import appears even greater when the pathogenesis of schizophrenia comes to be considered. The evidence shows that these psychoses are schizophrenic in form but not in aetiology. They are not endogenous but symptomatic. Their pathological basis is not entirely unknown but partly known. They occur in individuals lacking any special predisposition to endogenous schizophrenia, and the manifestation observed can be regarded as a phenocopy of a genetically determined condition.

Psychoses resembling schizophrenia have, of course, been repeatedly observed in the presence of gross pathological processes affecting the brain, such as neoplasm and syphilis, but in isolated cases for which so far no rhyme or reason has been found.* They have also been seen with chemical alterations such as amphetamine intoxication, and in this connection in a systematic way, as has been reported by Connell (1958). In this case, too, it was predominantly a paranoid form of schizophrenia which was mimicked by the organic psychosis. Features in which the amphetamine psychosis tended to differ from endogenous schizophrenia were the rapidity of onset, the dream-like quality of the experiences, the tendency towards visual hallucination, the brisk emotional reaction with emphasis on fear, and the rapid recovery on withdrawal of the drug. The resemblances to the epileptic psychosis we have discussed will not be missed. In this case it was hardly possible to argue, as had been argued with the psychoses accompanying gross brain lesions, that the illness was not only schizophrenia-like but actually schizophrenic, accompanying the other pathological events as a pure coincidence. But one might still argue, in the case of the amphetamine psychosis, that the peculiarities of an easily reversible toxic state lasting only a few days or weeks provided no useful model for the enduring changes that occur in schizophrenia. When we come to consider the schizophrenia-like psychoses of epilepsy, even this last argument cannot be applied. We are at last forced to consider what lessons we can learn from the symptomatic schizophrenias which will help towards a better understanding of the central group of schizophrenic cases, which we may call nuclear, endogenous or idiopathic.

One might take the view that with schizophrenia we are now approaching the stage of understanding which was reached half a century ago with epilepsy. It was customary then to classify the epilepsies into idiopathic and symptomatic types, and in the latter case to specify the underlying condition of which the epilepsy was symptomatic. This rather primitive stage in sub-classification may now have become possible in the syndrome we call schizophrenia. Very likely the bulk of the cases so diagnosed will prove to be "idiopathic", with genetical factors responsible for the specificities. But round this nucleus we would aim to define with increasing precision groups of cases in which the schizophrenia-like psychosis was traceable to known processes. A study of the mechanisms by which physical lesions and functional disturbances caused such symptomatic psychoses

* Of particular interest in connection with our case material are the schizophrenia-like manifestations of Huntington's chorea. According to Panse (1942) "schizophrenia" is by far the commonest mis-diagnosis, and the schizophrenic picture may be maintained for years. In his monograph he devotes 30 pages to the description and discussion of these cases. The schizophrenia-like state is most commonly of the chronic paranoid-hallucinatory type, but catatonic and hebephrenic pictures are also seen.

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to appear, would then become a tool in the investigation of the main problem.

A little earlier we asked the question whether any reason could be suggested for the close similarity between the epileptic psychoses we have described and schizophrenic psychoses otherwise caused. The reason which immediately suggests itself is that the nuclear schizophrenias are themselves organic psychoses, affecting the same functional systems of the brain, or ones closely integrated with them. It would not be the place here to go into the evidence on this problem, which is constantly being added to. Nevertheless brief reference may be made to the work of Huber (1961), showing in a well-controlled material of 212 cases of chronic schizophrenia, atrophic brain changes leading to enlargement of the ventricles, especially the third ventricle. Huber found a close parellelism between the degree of the pathological change shown radiographically and the nature and severity of the psychopathological findings.

As a working programme we have to use the principle that like effects are commonly due to similar causes. It is as a mock-up of the genuine schizophrenic that the schizophrenic-like epileptic is worth special study.

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References

Connell, P. H. (1958). *Amphetamine Psychosis*. London.

Hill, D., Pond, D., Symonds, C. (1962). "The schizophrenic-like psychoses of epilepsy" (Discussion), *Proc. roy. Soc. Med.*, 55, 311.

Hillbom, E. (1960). "After-effects of brain-injuries", *Acta psychiat. neurol. scandinavica*, Suppl. 142. Copenhagen.

Huber, G. (1961). *Chronische Schizophrenie: Synopsis klinischer und neuroradiologischer Untersuchungen an defektschizophrenen Anstaltspatienten*. Heidelberg.

Landolt, H. (1960). *Die Temporallappenepilepsie und ihre Psychopathologie*. Basel.

Panse, F. (1942). *Die Erbchorea*. Leipzig.

Pond, D. (1962). *See* Hill *supra*.

Slater, E. (1958). "The monogenic theory of schizophrenia", *Acta genet.*, 8, 50.

Symonds, C. (1962). *See* Hill *supra*.

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