Abstract

Recent followup studies establish that schizophrenia, however defined, is a disease of very variable outcome. Many patients recover completely and improvements can occur after many years. This is difficult to reconcile with the concept of slowly progressive brain disease, and it seems increasingly likely that the neuropathology observed in chronic schizophrenia largely precedes the onset of symptoms. The adverse effect of a hostile or overprotective emotional environment on the prognosis of schizophrenia, and recently reported differences in family environments between industrial and Third World countries, may account for the relatively good outcome of schizophrenia in the latter. Despite its many imperfections, the concept of schizophrenia is unlikely to be abandoned until we have radical new insights into the etiology of the "functional psychoses."

The main findings of both the American and the European long-term followup studies reviewed by McGlashan (1988) and Harding (1988) are all fairly similar, so confident conclusions can be drawn from them. First, it is clear that, however the syndrome is defined, the outcome of schizophrenia is remarkably variable. Not only does it vary from full and permanent recovery to a state of lifelong invalidism, but surprising improvements or deteriorations can occur even after many years of apparent stability. Despite this variability, the average outcome is a good deal worse than that of affective disorders (Samson et al. 1988). On the whole, patients do not deteriorate after the first 5 years of illness, and some make important and sustained improvements, on psychological test performance as well as on social functioning, even after many years of invalidism. Although the outcome of schizophrenia has improved considerably since the early years of the century, the cause of this improvement is obscure and the long-term followup studies reviewed here tell us remarkably little about the long-term effects of treatment. McGlashan's Chestnut Lodge study strongly suggests that intensive, analytically oriented psychotherapy does not improve the prognosis, and although the short- and medium-term benefits of neuroleptics are incontrovertible, there is much less evidence that they have any major influence on long-term outcome. Certainly, the outcome of schizophrenia had started to improve before these drugs were introduced, and many of the patients with the best long-term outcome are found to have taken no neuroleptic drugs after the earliest stages of their illness.

Etiological Implications

This evidence of striking variability in outcome and unpredictable fluctuations in course makes it very difficult to regard schizophrenia as the progressive brain disease we once imagined it to be. Only a minority of patients have a progressive downhill course, and even schizophrenia but worse than that of affective disorders. This variability suggests that the etiology of schizophrenia is complex and multifactorial, involving genetic, environmental, and developmental factors.
in them the disease process appears to arrest after the first 5 or 10 years. When enlargement of the lateral ventricles was first reported in schizophrenia, it soon became apparent that it was more often present, or at least easier to demonstrate, in chronic than in relatively recent illnesses, and this led to the assumption that ventricular enlargement was evidence of some slowly progressive pathological process, atrophic or infective. But no one has yet succeeded in demonstrating progressive enlargement of the ventricles of a cohort of patients followed over 5–10 years, or indeed in demonstrating any other progressive neuropathological changes in schizophrenia. We therefore have to take more seriously the possibility that these chronic patients may have had enlarged ventricles all along, at least from the early stages of their illness and perhaps since early childhood, and that the neuropathology we observe is a relatively stable predictor of chronicity rather than evidence of a slowly and inexorably progressive brain disease. Indeed, the season of birth effect (Bradbury and Miller 1985) and the evidence that intrauterine infection (Mednick et al. 1988) or perinatal injury (McNeil and Kaij 1978) can play a part in the etiology of schizophrenia raise the possibility that the ventricular dilatation and other evidence of neuropathology observed in chronic schizophrenic patients and postmortem schizophrenic brains may be largely the result of brain damage in utero or around the time of birth rather than of any schizophrenic disease process. Although the season of birth effect and evidence from several other lines of enquiry would be focusing interest on the early neurological development of schizophrenic patients in any case, and prompting us to think of schizophrenia as the delayed manifestation of a developmental disorder produced by an interaction between early brain damage and genetic influences, this shift in outlook is undoubtedly encouraged by the results of these outcome studies and the difficulty of reconciling them with any conventional kind of progressive brain disease.

The Better Outcome in Third World Countries

The outcome of schizophrenia does not only vary from one patient to another; it varies strikingly from one setting to another. In particular, it is better in so-called underdeveloped countries than in industrial countries, despite the latter’s far more extensive therapeutic services. As Lin and Kleinman (1988) remark, this unexpected and embarrassing discovery “has received more consistent support than almost any other finding in cross-cultural psychiatry” (p. 561) and “is arguably the single most important finding … in cross-cultural research on mental illness” (p. 563). Lin and Kleinman go on to discuss several possible explanations for this difference in outcome, including the “egocentric” nature of contemporary industrial society as opposed to the “sociocentric” nature of traditional agrarian societies and differences in their family structure, occupational provisions, and attitudes to mental illness. They also discuss potentially confounding issues such as a differential survival of vulnerable individuals and differences in the ratio of acute to chronic patients presenting for treatment in different settings. Although they discuss the possible effects of relatives’ attitudes and behaviors toward schizophrenic family members, and cross-cultural differences in these, they do not in my view do justice to the potential importance of this key influence on relapse rates.

Vaughn and Leff and their colleagues have now shown in a series of carefully designed prospective studies in London, California, and India that living with relatives who are critical, hostile, or emotionally overinvolved (a so-called high “expressed emotion” environment) has a major influence on the risk of relapse (Brown et al. 1972; Vaughn and Leff 1976; Vaughn et al. 1984; Leff et al. 1987). They find that if patients are simply dichotomized into those living in high and low expressed emotion (EE) environments after leaving hospital, the risk of relapse in the first 9–12 months ranges from 3 percent to 16 percent in those living in a low EE environment and from 31 percent to 60 percent in those in a high EE environment. In all four studies, the difference between the two groups was at least threefold and statistically significant. It has also been shown that lowering the relatives’ EE scores is associated with a significant reduction in relapse rates (Leff et al. 1982).

The available evidence also suggests that there may be systematic differences in mean EE ratings in different parts of the world. In India the proportion of schizophrenics’ relatives achieving a high EE rating was only 8 percent in the rural villages of Ambala and 30 percent in the modern capital city of Chandigarh (Wig et al. 1987). In London and in Aarhus in Denmark, the corresponding proportion was 54 percent. In southern California, it was 67 percent and high hostility scores (the major component of EE) were significantly more common.
than in London (Vaughn et al. 1984). Although Leff and his colleagues took great pains to try to ensure that identical criteria were used in these very diverse settings, it is obviously impossible to be sure that they succeeded. In any case, their samples were relatively small. Even so, their results raise the fascinating possibility that there may be major differences in relatives’ attitudes and behaviors toward schizophrenic patients in different parts of the world, and that these differences may be the main reason why the prognosis of schizophrenia is so much worse in industrial countries than it is in the Third World. Although Lin and Kleinman do not comment on the fact, the major differences in outcome revealed by the International Pilot Study of Schizophrenia concerned the proportion of schizophrenic patients suffering from recurrent psychotic relapses during the followup period. This was much higher in the industrial countries than in Colombia, India, and Nigeria, whereas the proportion of patients pursuing a steadily deteriorating course was much the same. It seems, therefore, that the component of outcome largely responsible for the overall difference in outcome between industrial and Third World countries may be the component most susceptible to the attitudes and behavior of family members.

Although existing data concerning the cross-cultural distribution of high EE settings are still rather sparse, and in obvious need of extension and replication, it is tempting to visualize a spectrum of attitudes and behaviors ranging from those characteristic of a Third World village (Ambala) to the competitive sophistication of southern California, with Third World (Chandigarh) and European (Aarhus and London) cities in between. It is most unlikely that the crucial difference is specific to schizophrenia, either as a cause or an effect. There is accumulating evidence that a high EE environment may have baneful effects on the prognosis of other mental disorders besides schizophrenia (Vaughn and Leff 1976; Szmukler et al. 1985); and it is likely that people’s attitudes to their mentally ill relatives are determined, or at least strongly influenced, by their attitudes toward much broader issues. It is possible, for example, that what might loosely be called “fatalism” might be a crucial influence. The Indian peasant may philosophically accept his son’s or his wife’s psychotic illness and subsequent incapacity just as he accepts the failure of the monsoon or the death of a newborn child. All are the impositions of an inscrutable Providence and an inevitable part of man’s lot. There is therefore no point in protesting, and certainly none in blaming or upbraiding the afflicted relative; the situation simply has to be borne—with patient and dignified resignation. His urban cousin in the city of Chandigarh, with its banks, cinemas, and prosperous merchants, is less likely to share these attitudes but does at least find them comprehensible. The outlook of the Anglo-Californian could hardly be more different. He expects to be comfortable and prosperous, to be healthy and happily married, and assumes that if these various desiderata are not forthcoming it is either because he himself has not been making enough effort or because someone is letting him down. If his job or his house are unsatisfactory, he changes them, and if his wife is unsatisfactory, he first tells her to shape up and, if that fails, he changes her.

This is, of course, mere speculation and perhaps rather fanciful. But the basic proposition that fundamental attitudinal differences between one culture and another may contribute toward the observed cross-cultural differences in attitudes to psychotic relatives is not at all implausible. They may also contribute to the better outcome of schizophrenic illnesses in rural than in urban areas—exemplified to McGlashan (1988) by the striking difference in outcome between the Vermont State Hospital and Boston State Hospital studies—though other factors like the greater occupational and residential stability available in a rural setting are almost certainly also involved. We can at least be confident of one thing: that further exploration of the reasons why schizophrenic illnesses have so much better a prognosis in Third World countries will be one of the most interesting and important areas of schizophrenia research in the next decade.

The Concept of Schizophrenia

The term schizophrenia has now survived for nearly 80 years and the underlying concept is even older, going back at least to the 5th (1911/1971) edition of Kraepelin’s Lehrbuch. It is probably better known and more widely used than any of our other diagnostic terms and, with the passage of time, has become one of our most basic concepts. Even so, it remains a provisional and purely clinical concept, and few psychiatrists would be prepared to predict confidently that the term will still be in use in 50 years’ time. Several contributors to this issue refer to the scientific
and conceptual problems posed by our inability to agree how the term should best be defined, and to the folly of using long-term outcome as a validator when chronicity (the 6 months' duration criterion in the Feighner and DSM-III definitions) is built into the definition. In fact, this latter criticism is not entirely justified, for it has been shown that the predictive power of the Feighner and DSM-III definitions is only partly dependent on the 6-month criterion (Helzer et al. 1983). The more important problem is that of definition. Our understanding of its etiology is so incomplete that schizophrenia can still only be defined by its clinical syndrome. Family and adoption studies strongly suggest that what is genetically transmitted is a "spectrum" of disorders including schizotypal and other related personality deviations rather than schizophrenia itself. There is nothing sufficiently distinctive about either the course or the treatment response of the syndrome for these to provide a validating criterion; and the clinical syndrome merges imperceptibly with that of the affective disorders as well as with the personality deviations of the schizophrenia spectrum. In the face of this confusion and uncertainty, it is hardly surprising that disenchanted researchers should start to consider alternative approaches. Angst (1988), for example, after referring with some exasperation to "the unbridgeable gaps between the current diagnostic concepts," advocates a return to "a multiaxial or multidimensional description" in place of arbitrary categorical definitions. In a similar vein, Carpenter and Kirkpatrick (1988) conclude that in the absence of any evidence that schizophrenia is a "valid disease entity," it is probably more useful to compare different "domains of psychopathology"—acute versus insidious onsets, persistent versus remitting psychoses, and good versus bad long-term outcomes—than to continue to study unitary populations of schizophrenic patients in the hope that Kraepelin will eventually be vindicated. There is much to be said for alternative approaches such as these. Indeed, very few of psychiatry's diagnostic terms have yet been sufficiently validated for enduring decisions to be made about their status and definition (Kendell, in press). It is therefore sensible for researchers to experiment with dimensional classifications and novel ways of defining the syndrome, and to use two or more of these alternatives simultaneously so that their implications can be compared. For all its imperfections, however, the concept of schizophrenia remains invaluable and indispensable to the ordinary clinician. It is woven into his ingrained ways of thinking, and much of his professional expertise and knowledge would be lost without it. He will only be prepared to abandon the term if he acquires some dramatic new therapy which renders old diagnostic distinctions irrelevant, or if the concept is undermined by radically new insights into the origins of the "functional psychoses," and such developments are probably still some way off.

References


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