Brit. J. Psychiat. (1983), **143**, 77–84

Discussion

Schizophrenic Deterioration

***John Cutting** Section Editor*

Psychological deterioration, defect state and psychological deficit are all terms introduced this century to replace Kraepelin's idea of dementia praecox. They all express the notion that schizophrenics have some global impairment in psychological functioning, which is different from the presenile dementias or recognised causes of cerebral dysfunction. At the beginning of the century it was generally regarded as organic in nature. In the 1960s the social revolution in psychiatry encouraged the view that the deterioration was psychosocial, the effect of institutionalisation. There has been a recent trend to revive the organic formulation.

The five articles which follow examine the idea from different points of view. They were commissioned because correspondence to the editor indicated that the issue was topical and controversial. It is hoped that they may be the first of a series of multi-author contributions on topical issues.

Manfred Bleuler presents his view that deterioration is not a progressive condition, but one of several outcomes, and even then liable to remission. Luc Ciompi discusses the relationship between organic and psychosocial factors in its cause, and concludes that it is the result of an interaction between the two. Tim Crow gives a clear account of the organic model. David Abrahamson draws attention to some methodological issues, which have led to a false clinical impression, and wrongly overemphasised the extent of deterioration. Digby Tantam analyses the complex effect of drug treatment.

Manfred Bleuler (Professor of Psychiatry, University of Zurich Bahnhofstrasse 49, CH–8702 Zollikon, Switzerland)

In this article the concept "schizophrenic psychosis" is to be understood as in DSM II. "Deteriorated schizophrenic psychosis" means a chronic schizophrenic psychosis in which not only single symptoms (e.g. delusions) continue to exist in the long term but also an altered cognitive and affective life.

The experience on which my findings are based is the everyday experience of 50 years, since I lived so many years under the same roof as schizophrenics. It is also based on many years of systematic investigation of the course of the disease, involving 556 selected schizophrenics and about 11,806 of their relatives. These investigations also enabled me to compare the course of the disease in 591 schizophrenic relatives.

The incidence of deterioration

About 10 per cent of schizophrenics sink into such a severe state of deterioration that they need permanent nursing care. About 35 per cent show long-lasting mild psychotic signs and live at home, in hostels or in open hospital wards. However, only about half of these chronic mild cases are deteriorated. The others show mainly single psychological signs or symptoms (e.g. passivity or delusions).

About 35 per cent of the patients appear cured and almost cured for long periods but are threatened by new acute psychotic phases.

About 20 per cent are stable and cured over many years.

The factors which influence deterioration

1) *Decade of onset.* The above figures are only valid for patients admitted after the 1940's. Schizophrenia in the first decades of this century was on average more severe, yet the numbers of the severely deteriorated and of the permanently cured remained the same.

2) *Family history.* Although schizophrenic psychoses of siblings and other blood relatives often run quite a different course, the course and result of schizophrenias of blood relatives are more alike than those of non-related patients.

3) *Phase of illness.* Of the illnesses which result in deterioration approximately one half occur after acute phases of the disease and the other half after a chronic course. Improvement and cures are not at all rare in cases in whom acute phases preceded the deterioration, but this can hardly be expected when the development has been purely chronic.

4) *Early upbringing.* In the history of schizophrenics one does not find broken homes or adverse conditions in childhood more frequently than in the history of addicts or many personality disorders. However, there exists a statistically significant correlation between adverse childhood conditions and schizoid personality in the history of schizophrenics.

Conclusions

The approximate figures which I quoted regarding the long-term development and final result of schizophrenic psychoses have general validity in the countries with western culture and in the second half of our century. At other times and in different settings the course of the schizophrenic disease is not the same. International experience has been that in many African and some Asian countries there is a more benign and phasic course. This might be due to a higher mortality of the severely ill in these countries.

It seems probable that the improvement in prognosis which has taken place since the middle of this century has something to do with the improvement of treatment, nursing care and efforts at rehabilitation. Yet it must be noted that even with modern treatment with neuroleptics the number of permanently cured individuals has not increased. According to my own statistics not one of the permanently cured patients has during the last few years been under continuous medication. Although modern treatment has caused the disappearance of severe permanent states immediately following a first acute attack it has not succeeded in reducing the number of severely deteriorated cases from the level of 10 per cent.

The symptomatology of patients who later recover can for a long period not always be distinguished from the symptomatology of those patients who remain permanently deteriorated. Benign and malignant cases often occur in the same family. Therefore, the concept "schizophrenic psychoses" covering both cases with and without permanent deterioration is still justified. Clinical experience shows that it is not possible to contrast the 'real' or malignant schizophrenia as a sharply defined disease entity from benign schizophrenia as another disease entity. Why do certain psychiatrists try to restrict the term schizophrenia or dementia praecox to that of a completely incurable disease, and exclude benign schizophrenias? The chief reason is probably the suggestive influence of the concept "disease entity". This supposes the existence of diseases with uniform cause, uniform symptomatology and uniform development. It is impossible in many medical conditions, and even more so in psychiatric, to carve out such clearly circumscribed disease entities.

According to the findings mentioned above there are two different family influences on schizophrenic events. One influence is the disposition to a schizophrenic illness, the other a disposition to the course of the illness. In neither of the two cases is the family influence decisive, nor is the family influence purely an inherited trait. The inherited tendency to personality development is closely linked with the family's effect on a subject's environment.

While adverse conditions in childhood cannot have a statistically visible influence on the onset of schizophrenia they can determine whether the prepsychotic personality is schizoid or not, and thereby increase the likelihood of chronicity and deterioration.

Luc Ciompi *(Professor of Psychiatry, Director of the Social-Psychiatric University Clinic, Murtenstrasse 21, CH–3010, Berne, Switzerland)*

1. *What is meant by "schizophrenic deterioration"?*

"Schizophrenic deterioration" is a very puzzling notion; it can even be considered as one of the central enigmas of schizophrenia. Does schizophrenia necessarily lead to a specific deterioration? What is its nature, phenomenology and aetiology?

When Kraepelin introduced the term "dementia praecox", he thought that he had isolated an "illness entity" which led relentlessly from an early age to a chronic dementia quite like the organic dementias of old age. In 1908 Eugen Bleuler criticized this concept, emphasizing that neither "praecox" nor "dementia" were essential. According to him, the main characteristic of the illness was a particular splitting of the personality, certain being parts deeply disturbed and deteriorated but others, including the intellectual skills, quite normal. Subsequently, the term "dementia" tended to be replaced by "defect", which is still used by many clinicians. Others, however, criticized even this notion for its flavour of irreversible organicity, and preferred the more neutral one of the "residual state".

In spite of such ambiguities, the phenomenological descriptions given for this particular "deterioration" show a high degree of concordance. Most of them include flatness of affect, indifference, social withdrawal and restriction; some mention resignation, stereotyped behaviour and mannerisms. Although close analysis reveals certain cognitive disorders, especially in focussing the attention and in forming correct logical classes and sequences, the basic intellectual functions are in fact preserved. In the tradition of Kraepelin's "state of feebleness" *(Schwächezustände)* several German authors reactivated the old notion of an overall "reduction of the energetic potential" as a common psychopathological denominator.

The described deterioration appears mainly during the *chronic* phases of the illness. Even in old age, it does not evolve towards a typical organic dementia. Certain authors who believe that it constitutes the very core of schizophrenia point to the fact that elements of it can sometimes be observed *prior* to the acute manifestations.

Two facts have recently reawakened interest in schizophrenic deterioration. First, several European long-term studies have shown that, in the long run, the evolution of schizophrenia is better than usually admitted. In some cases, late remissions occur even after long periods of severe deterioration. Hence, the described states are sometimes reversible. The second important fact is the increasing tendency to distinguish more sharply between two quite different (although often combined and overlapping) syndromes. The first one predominates in the *acute* psychosis and is characterized by so-called positive or productive symptoms, such as anxiety, agitation, confusion, thought-disorder, feelings of depersonalisation and derealisation, delusions and hallucinations. The second one is closely related to *chronic* states. It is characterized by the so-called negative or unproductive symptoms which we described as typical of schizophrenic deterioration. Crow has recently proposed to call the first picture syndrome I and the second one syndrome II. There are indications that they differ not only in phenomenology but also in psychophysiology and perhaps in aetiology.

2. *Aetiological theories*

Concerning aetiology, two opposing, and seemingly quite irreconciliable, theories have been proposed. The first one considers schizophrenic deterioration as predominantly biological and organic in nature. The second one is predominantly psychosocial. Among the arguments for the first hypothesis is the fact that quite similar states of reduction of the energetic potential occur after certain cerebral diseases (e.g. cerebral trauma). Furthermore, there is increasing evidence for a relationship between certain states of deterioration and an enlargement of the third cerebral ventricle. The cognitive disorders too can be interpreted as organic. Some authors also find a statistical association between genetic factors and chronic deterioration. Another important argument for organicity is based on the notion of irreversibility of these states. However, most

of these arguments are questioned by the defenders of the psychosocial hypothesis. They emphasize that the reduction of energetic potential sometimes reversible and is far from specific, that enlargement of the third ventricle is inconstant and that the cognitive disorders can also be related to defence mechanisms and disturbed communication. Furthermore, the recent long-term investigations, proving the existence of late remissions, found no evidence for a close statistical relationship between genetic factors and outcome. Contrasting with such negative findings, several studies detected a positive association between psychosocial variables and deterioration. Thus, British authors showed that the development of negative, unproductive symptoms was statistically related to prolonged social understimulation, for example in understaffed wards for chronic patients. This led to the important notion of institutionalism, and to reform of the whole system of institutional care. Other studies revealed that the long-term evolutions are also strongly influenced by family dynamics (e.g. emotional over-involvement, high expressed emotions) and by economic, cultural and several other psychological and psychosocial factors. The available institutional infrastructure too plays an outstanding role. Therefore, the question finally arises whether the chronic schizophrenic deterioration could not predominantly be a psychosocial artefact.

For the time being, neither of these hypotheses can be convincingly upheld. Both can however be combined in modern multi-causal concepts of schizophrenia which emphasize the many possible combinations and circular relationship between biological and psychosocial factors. This might explain the enormous variety of long-term evolutions. Another interesting bridge between biologic and psychosocial variables is the recently proved neuronal plasticity of the brain (dendritic atrophy or hypertrophy depending on the amount of external stimuli). This shows that psychosocial influences and the organic substratum are closely interdependent.

It seems likely that "schizophrenic deterioration" closely related to the diminution of the energetic potential of the Germans and to Crow's syndrome II is a multi-determined state which occurs mainly after, but sometimes before the acute productive schizophrenic manifestations. It can hardly be considered as specific. In its aetiology psychosocial factors play at least an equal, and perhaps even a greater, role than organic factors.

T. J. Crow, (Head, Division of Psychiatry, Clinical Research Centre, Northwick Park, London)

Many patients with schizophrenia go on having symptoms. Often they are not well tolerated by society, are isolated from their fellows, and subsist at a low economic level. A few survive only in long-stay institutions. A neglected truism of clinical practice is that a patient who has had previous schizophrenic illnesses will rarely appear entirely normal in work and personal relationships.

Yet schizophrenic illnesses vary in intensity with time, and often there is a response to neuroleptic drugs. Such variability encourages the notion that they are "functional" rather than "organic" psychoses, and that there is no part of the illness which, at least in principle, is not reversible. According to this view the schizophrenias differ from the affective disorders in symptoms and duration, but not in reversibility of process.

The view that I hold is that there is an irreversible component of the psychopathology of schizophrenia which is characteristic of this disease, even though it is not invariably present, particularly in the early stages of the illness. With colleagues at Northwick Park I have referred to this component as the type II syndrome and identified it with the negative symptoms, features which are pathological because some normal function is diminished or lost. Such symptoms are difficult to assess but include those referred to as poverty of speech (which may reflect a loss of the range of association of ideas) and flattening of affect (a restriction of the range of emotional response). They are to be distinguished from such negative behavioural features as lack of self-care and social withdrawal, which are less constant, and may be a secondary consequence of different pathological changes including the presence of positive symptoms.

True negative symptoms are assessed at clinical interview, may be related to the *schizophrenie gefuhl* (praecox feeling) of earlier authors, and seem to be closely associated with loss of intellectual function (manifest in an extreme form in the age disorientation phenomenon observed in in-patient populations). Sometimes the negative symptoms almost appear to have the status of a neurological sign. Indeed it may be that the abnormal involuntary movements, which are commonly, but I believe erroneously, attributed to long-term neuroleptic medication, are a manifestation of the type II syndrome.

The type II syndrome of negative symptoms is to be distinguished from the type I syndrome of positive symptoms. This includes delusions and hallucinations, and probably some forms of thought disorder. It is characteristic of acute episodes of psychosis, some

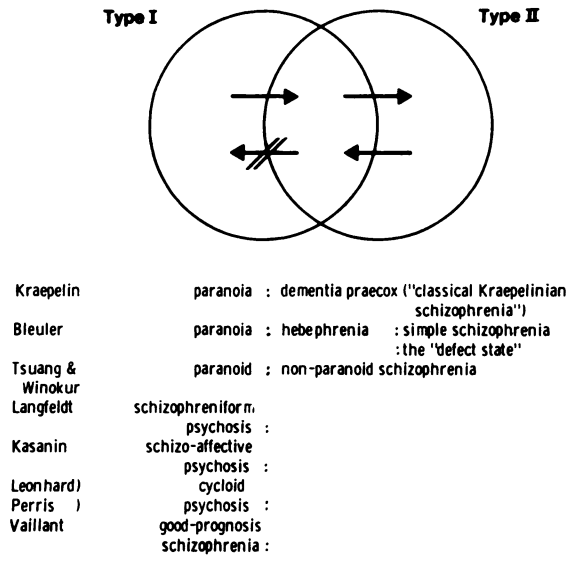
times remits spontaneously and, when it does not, often responds to neuroleptic medication. It is probable that this syndrome arises from a disturbance of neurohumoral function. There is a strong case that neuroleptic drugs exert their anti-psychotic effects by dopamine receptor blockade and a less compelling case that some schizophrenic patients (particularly those with persisting positive symptoms) have increased numbers of D2 dopamine receptors. Thus the type I syndromes may be the result of a disturbance of dopaminergic transmission.

I have argued that the type II, in contrast to the type I, syndrome is closely related to structural changes in the brain, for which there is now substantial neuroradiological and some post-mortem evidence. In Haug's air encephalographic study, ventricular enlargement was associated with clinical deterioration; in four of the CT scan studies increased ventricular size has been significantly associated with the presence of negative but not of positive symptoms, and in a number of studies has been associated with intellectual impairment. The evidence is not yet decisive on the question of whether these structural changes precede or are a consequence of the illness. Some observations are consistent with the possibility that sometimes they are precursors, but I predict that in many cases they will prove to be sequelae of episodes of illness.

Cerebral ventricular enlargement is no more than a gross index of structural change. Undoubtedly there are some patients with negative symptoms who do not have enlargement of the ventricles; they may have a more localised structural change. Enlargement may be the result of a diffuse process (e.g. a periventricular encephalitis), which could cause damage to certain critical connexions. Such connexions may include the stria terminalis, the output pathway of the amygdala, and structures such as the amygdala itself and hippocampus in the temporal lobe. There is evidence from recent post-mortem studies of a loss of cholecystokinin and somatostatin-containing neurones in these areas in patients with negative symptoms. Perhaps the amygdalo-hippocampal peptidergic connexions are particularly important in integrating emotional responses with ongoing cognitive activity.

The type I and type II syndromes are not separate diseases. Rather, they are overlapping constellations of symptoms which can be represented by a Venn diagram.

In the figure arrows indicate changes that occur with the passage of time. Some patients have only episodes of positive symptoms and may be described as suffering from good prognosis or reactive schizophrenia. However patients with more typical Kraepelinian illnesses acquire negative symptoms, and some of these may in due course lose their positive



FIG

symptoms to join the ranks of those with the pure defect syndrome. Some patients may reacquire positive symptoms. I suggest that is very unusual for patients who have once acquired true negative symptoms to lose them. This may be because they reflect the irreversible component which results from a structural brain change.

Why do only some patients develop the type II syndrome? Genetic factors may be important. Winokur and colleagues, who distinguish non-paranoid (i.e. illnesses with affective impairment or intellectual change) from paranoid illnesses (without such changes) find genes relevant to this distinction. Genes may predispose both to schizophrenia and to type of schizophrenic illness. Perhaps both the type I and II syndromes are caused by the same agent. There are grounds for believing this is a virus. The case of herpes simplex, a neurotropic virus which affects specific sets of neurones, establishes a latent infection and can become reactivated, is a thought-provoking model. Perhaps in patients with the genetic predisposition to schizophrenia the virus causes a neurochemical disturbance (e.g. by an affinity for a neurotransmitter receptor) corresponding to the type I syndrome. In a sub-group of these patients (again defined by genetic predisposition) the virus spreads more widely and causes destruction of critical neural structures in the temporal lobe. The results of this cell lost are seen in the negative symptoms and intellectual impairment of the type II syndrome.

David Abrahamson *(Consultant Psychiatrist, Goodmayes Hospital, Essex)*

The image of progressive deterioration that has for so long dominated thinking about chronic schizophrenia owes a great deal to the profound impact made by long-stay patients. The silent, immobile figures that form the nucleus of mental hospital populations produce an indelible impression of a state which it is feared other patients may recreate given sufficient time. Debate about its aetiology has diverted attention away from a re-examination of whether such patients do deteriorate progressively or not.

Relationship between individual and group deterioration

Studies in which groups of long-stay patients have been compared according to length of hospital stay have demonstrated statistically significant associations between time in hospital and the severity of negative handicaps such as poverty of affect and speech, underactivity and social withdrawal. A survey of almost 300 long-stay schizophrenic patients at Goodmayes, a typical catchment area hospital, produced the expected highly significant correlation between the length of stay and social withdrawal. The pattern throughout mirrored the one demonstrated by Wing and Brown in their classic study of three hospitals.

One need not suppose that such group patterns result from a homogeneous patient population. It might be that over a period of 50 years, different cohorts of patients will have had different degrees of initial deterioration. This would, in retrospect, produce a misleading impression of gradual *individual* deterioration.

Longitudinal course

The usual practice in assessing outcome is to group patients according to length of stay in hospital. This length of stay is divided into five year stretches. When I examined the social withdrawal scales when length of stay was divided into year stretches, I found much greater variation than expected, with no sustained worsening from year to year in the earlier decades. It was not until the end of the third decade that scores increased markedly and persistently to establish the overall correlation between length of stay and social withdrawal. This is inconsistent with individual deterioration, unless there was a cumulative effect which only manifested itself when a late threshold was reached.

However, the case notes of three samples of patients, with the most, an intermediate degree and minimal disability, disclosed courses with a surprising degree of long-term consistency. Levels of disability at the time of survey were strikingly similar to those recorded at or shortly after the index admission, even when this had taken place four or five decades earlier. In less than 10 per cent was the final state worse than the initial, and even here, the deterioration had been almost entirely confined to the first third of their admission. Improvement, which occurred in about 25 per cent of patients, had in contrast developed predominantly in the middle or last third of their admission. The pattern was thus the reverse of that predicted by either a progressive or threshold deterioration model.

These were particularly surprising findings. Patients with the greatest amount of disability accounted for the 'back-ward' stereotype, and were largely responsible for the statistical correlation between length of stay and social withdrawal in the population as a whole. Other studies have shown that such patients have disabilities which are maximal on admission or relatively soon afterwards, and the correlation appears to reflect the paucity of similar patients in the shorter stay groups, due to changes in the pattern of schizophrenia. In essence they appear to have been *selected* rather than *formed* by the institution.

The overall picture that emerged was remarkably similar to that described by Manfred Bleuler—whose findings were not known to me at that time. His thesis is that there is an approximate equilibrium over long periods, without precluding improvement even after decades. Such equilibrium he conceives as a plateau.

Discussion

One of the most striking features of schizophrenic 'deterioration' is that patients were frequently described as demented or institutionalised, according to the period, despite comments in the case notes that belied the terms. The misuse of these terms probably stemmed from the second class status of chronic patients. As the general feeling is that deterioration worsens with time, resources were concentrated on early cases.

This was particularly unfortunate in institutions that might have been well placed to develop long-term strategies to take advantage of stable plateaus and promote improvements, however late in the course.

Outside hospital the same misconception leads to continuing neglect of the rhythm of rehabilitation. Interventions are more commonly timed according to the short term availability of resources or enthusiasm, than tuned to the needs of the individual for as long as handicaps persist.

More is known about the determinants of acute relapse than of the plateaus that form the major part of most outcomes. Still less has been established about the relationship between plateaus and phases of change, or the varying effects of social and other influences at different stages.

Most fundamentally, attempts to explain chronic schizophrenia have been based on misconceptions about its course. The concept of institutionalisation depended on the assumption of fundamental differences between hospital and community courses. The disease process theory, derived from the work of Crow and colleagues, is again asserting itself over the psychosocial model, but I am concerned that it should not contain the same misconception about progression as Kraepelin's model did.

D. Tantam *(Lecturer, Social Psychiatry Unit, Institute of Psychiatry, London)*

The course of schizophrenia, and therefore the outcome, varies from individual to individual. Many factors are responsible for this. The symptomatology and apparent severity of the illness itself varies. The effects of the illness may be ameliorated or exacerbated, depending on the individual's personal resources; these reflect pre-morbid factors, for example the development of personality, and the acquisition of occupational skills, as well as current factors such as the individual's support system. Contingent factors, for example the availability of different forms of treatment, or the occurrence of adversity or opportunity, will also have an effect. It is not, therefore, surprising that the clinical stage of a patient at a single point in the course of illness turns out to be a very complex issue. Deterioration, therefore, is only one example of a particular outcome. This contribution will examine the effect of phenothiazines on deterioration.

When patients are followed up for reasonably long periods, it is apparent that some of their symptoms wax and wane whereas others change much more slowly, if at all. The changeable symptoms, delusions, hallucinations and other positive symptoms, have been well characterised, but the negative symptoms belong to a much vaguer grouping which includes such diverse entities as disturbances of non-verbal communication (flattening of affect, monotonous voice and unusual use of gaze), lack of initiative, poverty of speech and avoidance of social contact. It has been repeatedly demonstrated that positive symptoms respond immediately to neuroleptic drugs, but negative symptoms do not. The course of positive symptoms is also improved by long term neuroleptic drugs, at least as long as the symptoms remain ego-alien, with symptom-severity being reduced and new symptoms being prevented. Much less attention has been focused on the influence of neuroleptics on the course of negative symptoms, partly because not much is known about their causes.

In most patients it is likely that several causes act together. It is sometimes assumed that negative symptoms result from a toxic effect of the florid illness, especially if this is left untreated. However, there is no evidence for this. In fact the evidence points, if anything, to the contrary. In this respect schizophrenia is not like other relapsing medical conditions such as rheumatoid arthritis. However social isolation does exacerbate negative symptoms and this may result indirectly from the social disrepair caused by episodes of florid illness. Institutional confinement may result from florid uncontrolled illness as well as from some specific dangerous positive symptom such as a homicidal pre-occupation. Neuroleptic treatment by reducing positive symptoms may therefore reduce the risk of social isolation or institutionalism, and consequently improve the course of negative symptoms. However other factors may be more important; empirically, the severity or type of symptoms is only weakly associated with social adjustment at follow-up. It is also possible that prolonged neuroleptic treatment, through its extra-pyramidal side effects, may worsen some negative symptoms by increasing apathy and reducing social expressiveness. Obesity and Parkinsonism may also contribute to social stigma. The factors so far mentioned do not amount to a sufficient explanation of negative symptoms, even when taken in combination. It seems likely that at least in many cases, schizophrenia is associated with a specific social impairment, possibly of non-verbal expression, from its onset. The effects of this kind of impairment would vary in different situations depending on factors like emotional demand, and this does seem to be the case with some negative symptoms. It is the author's impression that social impairment does not respond to neuroleptic maintenance treatment, and can be best treated indirectly by helping the patient and his family to adapt to it.

From the foregoing it can be seen that maintenance neuroleptics can be expected to reduce positive symptoms but to have a much more equivocal effect on

negative symptoms. It is therefore important to note that there is a different proportion of positive and negative symptoms in different patients and, within the same patient at different stages in the course of illness. Patients with a schizo-affective type of illness, for example, show few negative symptoms, whilst patients with a disorder of insidious onset may have few positive symptoms. In the great majority of patients with both negative and positive symptoms the proportion varies as the illness progresses. In most patients progression is phasic: peaks of severity of positive symptoms alternate with troughs during which positive symptoms disappear or become much less obtrusive.

In time the peaks tend to become lower but the troughs may also become higher as positive symptoms become less ego-alien and therefore more persistent. Negative symptoms do not show phasic changes but tend to accumulate until a plateau is reached. Accordingly the later stages of the illness, when positive symptoms have become less salient, are often dominated by negative symptoms. This is sometimes attributed to 'burning out' of the illness, but may actually be due to the patient adapting to a socially sheltered environment in which positive symptoms are unlikely to be provoked.

As maintenance neuroleptics are most clearly of benefit in treating positive symptoms and as the proportion of positive to negative symptoms changes during the course of the illness, it is apparent that the effects of maintenance treatment on outcome will vary with the stage of the illness. A further complication is introduced by the fact that there are different dimensions of outcome, some of which may be improved by maintenance neuroleptics whilst others may not. The distinction between positive and negative symptoms is also useful in considering these different dimensions. For example, abnormal behaviour, usually in response to positive symptoms, increases family burden and may lead to social isolation. Maintenance neuroleptic treatment may therefore reduce isolation for this reason. Domestic task performance and sexual activity are however much more affected in patients with severe negative symptoms, and are unaffected by maintenance neuroleptic treatment. Although severe, distressing or burdensome symptoms obviously do impair social adjustment it is important to note that moderately ill patients can manage in spite of persistent positive symptoms if they have adequate personal or social resources. Most psychiatrists know of patients, usually with a history of continuous previous employment, who return to work despite being hallucinated and of others who, although apparently symptom free, may be unemployable. Negative symptoms are similarly likely to have much less effect on social outcome if resources are available to circumvent them. The effect of maintenance neuroleptic treatment on social outcome therefore depends not only on the relative proportion of positive and negative symptoms, but also on the extent to which the symptoms in a particular patient intrude into and disrupt his social environment.

In conclusion, the use of neuroleptics in the short term treatment of florid episodes of schizophrenia reduces suffering and distress caused by the illness, and probably improves social outcome by reducing social disrepair. The use of maintenance may also prevent florid episodes in many, but not all, patients. In some patients maintenance neuroleptics are also valuable because they reduce or abolish persistent positive symptoms, thereby reducing the risk of social isolation or institutionalism. However there are other factors, possibly due to specific social impairment, which cause social deterioration in many patients with schizophrenia and these may actually be made worse by maintenance neuroleptic treatment. These factors tend to become more salient as the illness progresses, and as positive symptoms become less obtrusive. The disadvantages of maintenance neuroleptic treatments, including the serious long term side effects which have not been considered here, may therefore outweigh the advantages of maintenance treatment during the later stages of schizophrenia. As this brief review has indicated there are many occasions when social intervention may reduce a patient's need for neuroleptic treatment. Systematic social treatment in conjunction with more selective use of both short term and maintenance neuroleptics may prove to be the best means of improving the outcome of schizophrenia in the future.