young girl who wants to act elegantly – speaks in correct German, presents herself as proper, coquettish and droll, and flirts with the officer.

We are justified in speaking here of a splitting of personality, a breaking away of the 'assortment' from the dominant personality, since the patient recognises the assortments for what they are at the moment when she experiences them. This means that her own true personality is present at the same time as the assortment, and even when 'the girl' is substituting as the dominant personality it too is apart from and yet simultaneous with the remainder of the assortment.

References

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Eugen Bleuler (1857–1939)

Eugen Bleuler was born in a Swiss village near Zurich, of farming stock. He studied medicine in Zurich and after a short period in Paris with Charcot and a brief stay in London to study neuropathology he returned to Zurich where he remained for the rest of his life, becoming director of the internationally renowned Burghölzli Clinic.

Bleuler first introduced the term schizophrenia in 1908, in the following article which has never been translated into English before. He suggested that this term was more appropriate than dementia praecox. His monograph on schizophrenia published in 1911 was only translated into English in 1950 and is now out of print. The current extract therefore provides a useful summary of his views on schizophrenia.

The prognosis of dementia praecox: the group of schizophrenias

E. Bleuler (1908)
(Die Prognose der Dementia Praecox – Schizophreniegruppe. *Allgemeine Zeitschrift für Psychiatrie* **65**, 436–64)

Introduction

In using the term dementia praecox I would like it to mean what the creator of the concept meant it to mean. To treat the subject from any other point of view would serve no purpose, but I would like to emphasise that Kraepelin's dementia praecox is not necessarily either a form of dementia or a disorder of early onset. For this reason, and because there is no adjective or noun that can be derived from the term dementia praecox, I am taking the liberty of using the word *schizophrenia* to denote Kraepelin's concept. I believe that the tearing apart or splitting of psychic functions is a prominent symptom of the whole group and I will give my reasons for this elsewhere.

With the help of two doctoral students, Wolfsohn and Zablocka, I have taken the records of 647 schizophrenics admitted to Burghölzli over a period of eight years and studied them with particular reference to prognosis . . .

Two points must first be emphasised: not only does prognosis

depend on the illness which we diagnose and name according to its symptoms, but it is also determined by a large number of other factors, the effects of which are interwoven and may even cancel each other out. We must therefore make up our minds to consider separately the individual factors and their influences.

The concept of prognosis itself includes many and varied sub-concepts, contained in the following questions:

> Will the patient deteriorate intellectually?
> What form will this deterioration take (hebephrenic, catatonic, paranoid)?
> How far will it progress?
> How quickly will it progress?
> Will the symptoms remit? Which symptoms? To what extent? How soon?
> Will the illness recur, or will there be new and progressively deteriorating attacks?

General and specific prognosis

There is also the question of the difference between a *general* prognosis for the condition as a whole, and a *specific* prognosis for each individual patient. If each sub-group had its own precise, definite prognosis, then there would be no need to consider specific, individual prognoses, because by knowing to which sub-group a patient belonged one would know the patient's precise prognosis. Unfortunately, except for sub-groups such as chronic catatonia, where patients have been chronically ill for years, the general prognosis of a sub-group is as vague as that of the whole illness. It is a general truth that, whatever form the illness may take initially, catatonic symptoms may later supervene, though catatonic disorders, provided they are not acute and transitory, tend to remain catatonic throughout. The same is true of the paranoid sub-group and subjects with delusions of influence. The ideal situation would, of course, be to understand fully the general prognosis of every sub-group.

The general prognosis of dementia praecox is clear to anyone who has grasped Kraepelin's concept. It deals only with qualitative or directional aspects and points towards a specific kind of state to which we give the vague and far too general name of dementia. The general prognosis does not specify how long the disease process takes or how severe the final dementia will be. There is so much individual variation that a 'specific' prognosis is required in virtually every case.

In the present state of our knowledge this specific prognosis amounts virtually to a temporal prognosis, one which would estimate the duration of the illness before the onset of dementia. This quantitative prognosis cannot be too sharply distinguished from the qualitative, directional prognosis, and one has to be content with what Kahlbaum in his day established for catatonia: the process can come to a standstill and equally well resume its progress at any stage of the disease.

The incurability of schizophrenia

If we understand this clearly, then the contentious question of recovery in dementia praecox loses much of its significance. If the dementia comes to an early standstill the patient may seem both to the layman and to many psychiatrists to have recovered. Indeed, many patients can be regarded as cured in the social sense of the term. If no acute attack has occurred, then such cases must remain latent, and latent schizophrenia is quite common, to judge by those patients who are admitted briefly to hospital only on account of random and transient disturbances caused by some unfavourable life event. The large number of schizophrenics who marry after a spell in hospital, and are regarded as healthy, points in the same direction. In my view many of Magnan's 'degenerates' and many paranoid personality disorders described by other authors are misdiagnosed schizophrenics.

It is a matter of individual opinion whether these stationary and improved states, in which the patient is capable of resuming his former life, should be regarded as cures. The decision depends on many factors. One of the most important is the time which the doctor has available for determining the state of the patient on discharge. My own experience leads me at all events to agree with Aschaffenburg's view that in schizophrenia there is no cure in the sense of *restitutio ad integram*. Although the large majority of schizophrenics who have passed through an institution may live permanently outside it and are regarded for the most part as healthy, whenever I have been able to examine any of those who have been pronounced cured I have found a residue of the illness. The diagnosis of a cure has often been rash, in that the discharged patients, without having changed in any way, have speedily had to be readmitted to an institution. Personally I have never treated a patient who has proved on close examination to be entirely free of signs of the illness.

I know patients whose achievements in life have been outstanding, but even these I would not regard as cured. They include business men who independently build up large and successful businesses, civil servants, parsons, a poet and a scholar of international renown. The last of these had suffered two attacks of catatonia before writing a new scientific work. It was a pleasure to discuss scientific matters with him even when he was still suffering from genuine delusional ideas. But when I finally considered him completely ready to resume work, he was still making crude logical mistakes when one spoke to him about the complexes which had played a part in his illness. I would not like to accept as a genuine cure a state in which some parts of the mental apparatus are permanently inaccessible to logic. I can appreciate, however, how physicians who are not used to looking for such phenomena attest many cures.

In the psychological sphere we find that when there is an exacerbation of the illness the ideas from an earlier attack regularly return in some form or other, which also shows that residual mental symptoms of the illness cannot have been completely overcome.

When the disease process flares up, it is more correct, in my view, to talk in terms of deteriorating attacks, rather than its recurrence. Of course the term recurrence is more comforting to a patient and his relatives than the notion of progressively deteriorating attacks.

Time-course of the disease

Although a precise prognosis of the dementing process is impossible at the present time, one may still identify the factors which govern its time-course.

These factors include the important but as yet unknown personal characteristics that can be grouped together under the term disposition. Heredity and the level of mental energy – the only constituents of disposition which are at present recognised – are irrelevant so far as prognosis is concerned.

I have already pointed out that we do not know whether or not the time-course depends upon the sub-group of the illness.

Random external influences must, however, also be relevant and, other things being equal, the patient who from inclination or necessity leads an irregular life which exposes him to many emotional ups and downs is more likely to become ill again than one who lives quietly. In two of my cases a definite recurrence was associated with a chance encounter with a former sweetheart. I have no doubt, too, that treatment counts for a good deal. The sooner the patients can be restored to an ordinary life and the less they are allowed to withdraw into the world of their own ideas, the sooner do they become socially functional. I have not yet observed that deprivations and excesses contribute to a poorer prognosis, but alcohol has an indirect effect, in that it makes it impossible for even mild cases of schizophrenia to function in society.

All these factors must be the subject of detailed investigation, but before committing ourselves to an individual prognosis of the time-course we must first make a precise assessment of the *degree of dementia* or, in other words, of the *aspects of a case which are no longer susceptible to remission*.

The nature of the dementia

The assessment of the dementia is no easy task. Those who think they can diagnose or exclude or even gradually identify a schizophrenic dementia by means of an intelligence test lasting a few hours or even days must have a concept of dementia praecox that is completely different from that of the Kraepelinian school. Such investigators proceed as if all that the patient has earlier known and understood has been lost, forgetting that this form of dementia cannot be considered in a cross-sectional perspective. What we call dementia can fluctuate in time between very wide limits. External circumstances often have a decisive influence. The same patient who in the institution behaves in a completely unreasonable or demented manner can participate at home in all domestic functions and can be regarded by his relatives as healthy in every respect. The patient can show an absolute defect in logic when it comes to appreciating his own position vis-à-vis his environment. He is often quite incapable of reaching the simplest conclusion, e.g. 'If I behave in an antisocial way in the institution then they will not discharge me', even though he is daily confronted with evidence supporting this conclusion and understands the individual components of the argument as well as someone who is deemed to be healthy. He believes, however, that he has good grounds for behaving badly in so far as he is trying hard to get out of the institution. The same patient is capable, however, of presenting a well-reasoned, hour-long lecture in which he convinces most of his audience that he is of sound mind, in which he very deliberately omits everything that is contrary to his argument and embroiders, or even changes, whatever favours his theme. He can carry out complicated tasks, can show scholastic know-

ledge which his doctor might well envy, can understand that knowledge and make correct use of it. He can make refined plans for flight, or for hoodwinking and deceiving clever people. He can at a given moment appreciate everything that is explained to him, but an hour or even a few seconds later he can be entirely lacking in understanding of the very same matter. Over a given period he can furnish an excellent account of his life history, but at a certain point he may become blocked and seem demented. The schizophrenic, however, is no simple dement; he is demented in respect of certain issues, certain periods of time, or certain complexes only.

Acute stages of the disease

It is generally recognised that it is better to diagnose the dementing process early. Unfortunately the various empirical rules which have been put forward for achieving this are not helpful. This is probably because there are two sets of phenomena which occur in the early stages of the illness. One set does permit us to reach certain conclusions about the nature and strength of the schizophrenic process, for example certain disturbances of association, but other phenomena also occur in other illnesses and tell us nothing about the nature of the process or about its severity, for example manic and depressive symptoms. Our task is therefore to look beyond these general phenomena, seek out what is specifically schizophrenic and then assess, according to their severity, those symptoms which we can say with certainty or even with some degree of probability will persist.

Where the first attacks are manic or, as is more common, melancholic in nature, it is relatively easy to distinguish the specific symptoms of schizophrenia, namely the disturbance of association and of affect. The more pronounced these are, the worse the prognosis, because these are precisely the disturbances which show least improvement. Catatonic symptoms are significant, too, since they reveal, behind the affective fluctuations, a schizophrenic process, the seriousness of which corresponds by and large to the overt severity of these phenomena. We must, however, be cautious in drawing conclusions, since as long as the illness is in its acute stage these symptoms usually show some improvement and the significance of a depressed mood is notoriously unreliable. It has been asserted that catatonic symptoms occur frequently in manic-depressive illness. I have so far never observed in this illness any symptom that could be regarded on analysis as catatonic in the same sense as the catatonic symptoms of

schizophrenia. This may be because we have admitted exceptionally few manic-depressive patients in the last few years.

In oneiroid (dream-like) states it is important to assess the genuinely schizophrenic symptoms and to base our prognosis on them, but marked confusion and stupor are usually difficult to analyse. It is rare, however, for such syndromes to persist for long in an extreme form, and periods of greater lucidity eventually make it possible to form some judgement on the presence or absence of the characteristic schizophrenic symptoms.

Chronic stages of the disease

In chronic states a considerable improvement is always possible. A transfer of location, for example, which has proved ineffectual on a dozen occasions may on the thirteenth bring about a kind of recovery. Failure to take this into account has been the rock upon which most attempts to draw up reliable principles of prognosis have foundered. At the same time it is in fact rare for recovery to be extensive once the dementia has reached a certain point and the patient has shut himself off from the outside world. I have, however, lost touch with hardly any of our admissions of the last ten years, and when patients are transferred from clinic to institution I feel regret that the improvement that might have taken place was not achieved under our care.

Distinction between primary and secondary symptoms

In analysing the power of individual symptoms to determine outcome, I lay great weight on a distinction between the primary symptoms, which are part of the disease process, and the secondary symptoms, which arise as reactions of the ailing psyche to environmental influences and to its own strivings.

To take an analogy, it is possible for osteoporosis to reach a very advanced stage without obvious symptoms, until the patient suffers a physical trauma. The real disease, and with it the prognosis, lies in the brittleness of the bones which can, depending on external circumstances, produce symptoms or not. Further, it is not always possible to reach conclusions about the severity of the illness by relating the symptoms to the seriousness of the event that caused them: a severe trauma, for example, can break a normal bone. In osteoporosis brittleness is the primary symptom, the break resulting from a slight trauma is the secondary. Similarly an abductor paralysis

is a primary symptom, while the contracture of the internus and the ocular spasms are secondary symptoms. The latter have nothing to do with the prognosis of the illness. They depend on circumstances which are external to the illness, for example on whether one or both eyes are covered.

To find analogous differences in the phenomena that manifest themselves in schizophrenia, there is already a point of reference, namely the symptoms which are triggered by external influences, for example attacks of abusive rage which are the patient's psychological reaction to external influences, even when they last a long time and begin with hallucinations. The same significance can be attached to states of altered consciousness, including the Ganser syndrome. Here it is a question of a waking dream, a dream of desire or fear, such as are also seen in hysterical patients who have encountered an emotionally disturbing event, and in others who are not demented, but whose nervous system is labile. Thus a female schizophrenic patient with hallucinations and delusions may develop false ideas about marriage, pregnancy and motherhood. This is not the illness itself, but only an inessential symptom of the illness, triggered by some external or inner emotion. Thus states of altered consciousness have a very good prognosis. Among 18 patients with this condition only one was not restored more or less to his earlier condition. There is no question here of progressive deteriorating attacks of the illness, merely random episodes.

The disease process does not actually produce the complicated symptoms which we are accustomed to see. Particular delusions or hallucinations are not generated by the process itself. The disease process can only create certain elementary psychological disturbances, on the basis of which hallucinations and delusional ideas arise, governed by the joint effect of determinant and releasing factors. It was not the disease process which made a former patient, a widow, experience lengthy and unendurable pains in her abdomen: it was the fear of being pregnant and, finally, the conviction of pregnancy after she had had sexual relations with her neighbour. Prior to this she was a latent schizophrenic, quite capable of regular employment. The pains are a reaction of the sick mind to certain emotional events and have nothing to do with the disease process and therefore with the prognosis. Similarly, the behaviour of the patients – whether they withdraw into themselves, whether they work or not, whether or not they smear excrement – does not constitute the primary symptomatology of the

illness, but rather the result of several psychological processes and external influences.

The most striking support for the idea that what we call dementia is in the main a secondary symptom comes from the sudden improvement that can take place in apparently hopeless cases as a result of psychological influences. In one institution a female patient had been kept in a cell for years because she was so dangerous. When taken along to a Christmas celebration she behaved impeccably, and on Berchtold's Day she appeared complete with couplets and after a few months we were able to discharge her fit for work. She managed to maintain herself outside the institution for many years. In her case the 'dangerousness' was not the direct consequence of her cerebral disorder, but a reaction to the precautions and compulsion used in her environment. Another female patient who was undergoing a mild phase of depression developed the idea of taking her own life and kept on trying to do herself harm. All the measures were taken that are customary in such cases; the only procedure we did not try was to ignore her behaviour, since this was too dangerous. In the end she had to be strapped to her bed, with the result that the patient, realising that all attempts were now useless, gave up trying to kill herself, and after a few days was set free. Later she became well enough to be certified as fit to manage her own affairs.

I would also include affective flattening among the secondary symptoms, recognising that this view goes against the general opinion. Admittedly it is uncommon for the disturbance of affect to remit if it is very prominent in chronic cases. In certain circumstances, however – for example, if we touch upon a patient's complexes, or if schizophrenia is complicated by senile dementia – the emotional life may suddenly revive after years of comparative dormancy. I have so far never seen disturbed associated functions restored in this way.

Catatonic symptoms are regarded by most psychiatrists as primary phenomena. I do not believe that this is correct. Stereotyped speech, for example, may represent an abbreviated symbol of a loved one; if there were no loved one, then there could be no stereotypy. I would not even consider cataleptic phenomena to be primary symptoms, since psychological influences can cause them to disappear completely and to reappear. The same applies to negativism, mutism and command automatism, all of which can have various origins.

I do regard certain bodily symptoms, such as fibrillary twitchings, muscular excitability and pupillary disequilibrium, as primary

symptoms worth studying in detail as direct signs of the involvement of the nervous system. Other physical symptoms are much less easy to understand. Disturbances of the vasomotor system are very complicated and their genesis can be varied. The same applies to anomalies of diet, secretion, temperature and menstruation. Oedema occurs in the early stages of the disease, and cannot be entirely attributed to idiosyncratic postures. An increase in body weight, unaccompanied by psychological improvement, distinguishes the illness from manic-depressive insanity, but an increase in deep reflexes is a feature which the illness shares with many functional and organic disorders. The same is true of sleep disturbances and headache. All these symptoms are transient, with the exception of the twitchings and the muscular and pupillary excitability. Other frequent symptoms – dry coated tongue, loss of appetite, rapid loss of weight and energy and coarse tremor – are usually regarded as signs of an intercurrent infection.

On the psychological side the most fundamental disorder appears to be a change in associations. In schizophrenia it is as if the physiological inhibitions and pathways have lost their significance. The usual paths are no longer preferred, the thread of ideas very easily becomes lost in unfamiliar and incorrect pathways. Associations are then guided by random influences, particularly by emotions, and this amounts to a partial or total loss of logical function. In the acute stage associations are broken up into little fragments, so that in spite of constant psychomotor excitement, no kind of action is possible because no thought is followed through, and because a variety of contradictory drives exist side by side and cannot be synthesised under one unitary affective or intellectual point of view.

There is, in addition, a kind of stupor and a slowing down of all mental processes which cannot be explained as arising from secondary sources. The impression made by these symptoms is that they have an organic basis. They may be linked with the severe cerebral oedema which we usually find in fatal cases of catatonia. Many catatonic attacks also present an organic appearance: they begin with loss of consciousness and convulsions and in some circumstances are even followed by slight paresis.

Confusional states call for more detailed study. Confusion is not a unitary symptom but a phenomenon which occurs in many mental disorders, once the disturbance becomes so severe that the observer is no longer able to follow the patient's thoughts. Some forms of confusion can be attributable to the primary cerebral disorder, in that they mark the progressive attacks of the illness and reflect the disorder of

association. Other forms arise because the patients' various hallucinations prevent them from being in touch with reality, though closer examination usually shows some connective thread in the ideas. We have to be careful to distinguish between schizophrenic confusional states and those which occur in mania.

Stupor is another symptom that has many different roots. It can be the outward sign of several basic disturbances which severely hamper inner activity or the expression of such activity. It is a notoriously difficult phenomenon to study.

Manic and depressive mood swings occupy a very special position, their significance varying from case to case. In rare instances they give the impression of being a chance combination of manic-depressive symptoms in a schizophrenic illness. Sometimes they seem to be triggered by the schizophrenic disease process, a view which is supported by the frequency with which the manic-depressive syndrome occurs in schizophrenia in general, and in the progressive attacks in particular. A third possibility is that states of anxiety and depression take on the role of secondary symptoms, with their origin in the basic schizophrenic thought process.

Remission of individual symptoms

So far, we have established some practical, empirical rules which can guide us in predicting outcome. An experienced psychiatrist often has a 'nose' for prognosis and may make a better prediction than one which is logically deduced from these rules. Nevertheless I believe that it is helpful to know how each individual symptom tends to remit, and how this affects the general prognosis. I also believe that a knowledge of whether a symptom is primary or secondary is the most useful guide to this. A few examples of which symptoms and syndromes tend to remit will illustrate this principle.

Acute syndromes do, of course, often die away, sometimes in a way which recalls the pattern seen in infectious delirium. Mania, depression, alterations in consciousness and rage behave in this manner. Confusion and stupor are not always part of an acute syndrome, and the hallucinations and delusional ideas which may accompany confusion and the withdrawal from the external world that occurs in stupor have an inherent tendency to become chronic. Catatonic syndromes, if acute, have no particularly sinister significance; the outlook changes, however, if they persist in a state of clear consciousness.

It is rare for the disturbance of association to improve once it has become chronic, and affective flattening will persist too in most, but not all, cases. It would seem self-evident that symptoms that are determined purely psychologically should be capable of remitting. Yet in the individual case this does not necessarily happen. Even in a mentally healthy person some experiences can leave behind a small scar; religious or superstitious ideas acquired in youth can come to the fore later, even if they have for decades been overthrown by logical reasoning. In schizophrenia we hardly ever find that delusional ideas which have receded completely lose their influence on logic and emotions. They are never entirely corrected, in the way that a normal person corrects an error that is not affected by emotion. Other symptoms, too, can become stereotyped. Thus I accept the existence of what Wernicke rightly called residual symptoms, and we can never be entirely certain that purely functional psychological symptoms will recover. An ominous sign is the patient's withdrawal from the external world.

Mechanism of remission

The disease process, which as far as we know destroys only a few cerebral elements, can recede to a certain extent. The study of cerebral pathology has made us familiar with the phenomenon of 'insult', in which a damaged organ accommodates very well to changed circumstances. It may also be that a poison arising from an infection or from a metabolic change operates for a certain period of time, after which one has to deal only with the established damage.

Conditions which are triggered by emotions, for example altered states of consciousness and attacks of rage, are self-limiting because in the end every emotion exhausts itself. This applies to the mentally ill and to the mentally healthy alike. An individual weeps or storms or rages until the tears and tempers are spent.

What seems to happen is that the mental symptoms that remain when a disease process has run its course are split off from the personality. For example, delusional ideas may continue to linger in 'cured' patients without being corrected. The patient has only 'forgotten' them; when questioned directly about them, or when another attack supervenes, he summons them forth again in undiminished strength. External circumstances contribute greatly to this process of splitting. The more external stimulation the patient receives and the more capable he is of accepting such stimulation, the sooner can this

splitting take place. Therein lies the value of occupation and of early discharge, which so greatly influence prognosis.

We thus see that schizophrenic dementia is capable of receding not only in respect of individual symptoms, but also as a whole. Examples of late remission in schizophrenia are no longer curiosities; one has even come to expect them. It is no longer necessary to regard late recovery from schizophrenic dementia as proof of a wrong diagnosis.

It would represent an important achievement if the intensity of the disease process could be estimated. As long as we are ignorant of its nature we can only surmise. If, for example, cerebral oedema does play a part in severe catatonia, then the effect of raised intracranial pressure would make the clinical outlook particularly severe. However, it is still unclear whether such oedema is an expression of the schizophrenic cerebral process or not. Meanwhile, we can only assume that the severity of the associative disorder or stupor is in some way related to the severity of the disease process. Similarly, there is a strong impression that catatonic phenomena and pupillary differences are particularly prominent features of severe cerebral affection.

Problems in the estimation of prognosis

A few other points concerning the difficulty in assessing prognosis should be mentioned.

First of all, there is the question of the case material itself. Only a very small proportion of all schizophrenics come under observation in our institutions, and when it comes to the individual groups of the illness we see only a selective sample. For example, patients who recover after one attack are observed only during that initial attack; chronic patients are observed only in the late stage of the disease. With regard to simple schizophrenia, only the worst, most deteriorated cases are brought to medical attention. Mild cases of schizophrenia are admitted because of an intercurrent attack of rage, forensic complications, a burst of manic excitement, a suicidal attempt, or a bout of pathological drinking. The prognosis of such different cases may be unrepresentative of the average case.

The case material observed is also very much influenced by the admission and discharge policies of an institution. Where patients are discharged early, only the very serious cases remain on the books. Where a large institution for the care of the mentally ill is available, the inhabitants of the district may become accustomed to ridding themselves even of the more mildly ill. If an institution admits only the most

severe cases, then acute cases will predominate there because, as we have seen, if they do not 'recover' they tend to develop more severe end-states than patients whose course is more gradual. Moreover, such an institution will soon fill up with chronic cases. Prognostic findings from different institutions can therefore never be directly compared.

The truth of this was made clear to me by the difficulties encountered in allocating to discharged patients a favourable or unfavourable outcome. Better criteria than those which I employed should be sought. It was fairly easy simply to divide the patients into those who were socially adequate, that is to say who could live more or less independently outside the institution and maintain themselves in the community, and those who were quite demented and needed to be looked after away from their families. Patients who fell into neither of these extreme categories were put into a special group of 'moderately demented' cases. However, this rating could not be used within the institution because hardly any patients came into the first category. Instead, those who were able to do useful work and with whom it was possible to maintain social contact were deemed to have had a favourable outcome. All remaining patients were classified as actively or passively asocial.

In the course of establishing these ratings I made the interesting observation that in Neu-Rheinau the patients with a 'favourable' outcome were more severely ill than those allocated to the same category in Burghölzli or in Alt-Rheinau. As I had known most of the patients personally for years, it seemed unlikely that this was due to an error. The observation was also confirmed by the physicians in Alt-Rheinau. The explanation for this fact, I concluded, was that Neu-Rheinau was run in accordance with new ideas and, from the start, had been organised so as to be filled by patients from every quarter of the globe. Here it was possible to create a new spirit which allowed the transformation of a number of severe and chronic catatonics into good workers. This difference between different parts of the same psychiatric service is very striking.

If the degree of dementia is used as a measure of outcome, then we encounter the problem that we have no satisfactory method of quantifying this. A true measure would only apply to the severity of the basic disturbance, whereas what we call dementia has no clear connection with the basic disturbance. Further, the fluctuating performance of the schizophrenic psyche raises the question of whether there is a

true end state of the condition, i.e. a stage at which the illness neither progresses further nor recedes.

Summary

Our present knowledge in regard to the progress of schizophrenia may be summed up as follows.

The mental debility which follows an acute attack, no matter of what kind, usually continues to increase for some time after the acute symptoms have subsided and then reaches a relatively stable state, with longer or shorter periods of fluctuation. It is, however, quite rare to encounter cases who do not deteriorate. Among patients who remain in the institution the most frequent symptom is a gradual increase in the degree of dementia, hardly perceptible to those who are in day-to-day contact with the patients. It is noticeable if we examine such patients after ten or more years. It manifests itself in a reduced performance capacity, a greater apathy, and in physical and mental deterioration. The decline can be caused psychologically or by a progression of the disease process. In chronic cases the illness is less likely to come to a standstill, but in my experience there is no essential difference between chronic and acute forms.

In the majority of patients, however, severe dementia can be excluded, since they maintain themselves in the outside world, and even among institutionalised patients there are some who hold their own for decades, maintaining a certain standard throughout their time in hospital.

There is therefore no doubt that, on the evidence of our present material, the disease process can come to a standstill; that this point can occur at any stage of the illness; that many patients who are apparently stable can very gradually deteriorate with the passage of time; and, finally, that progressively deteriorating attacks can keep recurring. These new attacks may take place at any time, even after decades of quiescence. The menopause seems to be a period of vulnerability, but even much later in life acute exacerbations may still occur; there is no time limit that can be established with any certainty. It has been said that an interval of three or five years between attacks constitutes a chance of lasting recovery, but I do not believe this has been proved. As far as I am aware, there are no good statistics that provide information about the frequency of new attacks in the course of subsequent years. Such statistics would have to take into account the different forms of

the illness and would also have to allow for the age structure of the material studied . . .

I do not like having to complicate rather than simplify the question of the prognosis of dementia praecox, but there is nothing else I can do. Only when all these matters have been cleared up will we be able to simplify our findings and speak with authority. Progress will be made only when the research worker looks separately at each of the many factors involved and carefully evaluates their significance in the different circumstances which pertain to each case.

Karl Kleist (1879–1960)

Karl Kleist was born in Alsace and studied under the most influential neurologists of the turn of the century – Ziehen, Wernicke and Anton. He was in charge of a neurological team caring for brain-damaged soldiers during the First World War, and then became professor of neurology and psychiatry in Frankfurt and then Leipzig.

Kleist was committed to a neurological interpretation of psychiatric symptoms, always looking for comparisons between functional disorders in psychosis and a corresponding deficit in brain-damaged subjects. This extract illustrates his attempt to explain thought disorder in schizophrenia in terms of the disturbance in language and thought which occurs in aphasic subjects with recognisable brain damage.

Alogical thought disorder: an organic manifestation of the schizophrenic psychological deficit

Karl Kleist (1930)
(Zur hirnpathologischen Auffassung der schizophrenen Grundstörungen. Die alogische Denkstörung. *Schweizer Archiv für Neurologie und Psychiatrie* **26**, 99–102)

In my search for the cerebral pathology of schizophrenic disturbances I was able to isolate in 1914, from the many varieties of confused speech which one sees in that condition, several particular disturbances which could be regarded as based on cerebral pathology. In doing so I confirmed Kraepelin's hypothesis that some schizophrenic disorders of speech depend, like similar phenomena that are found in dream speech, on functional disturbances in the temporal speech area. In addition to disturbed sentence formation and paragrammatisms, we found in some patients unmistakable and sometimes literal verbal paraphasias and word amnesias, particularly in their designation of general and abstract concepts. Another of my colleagues succeeded in showing that the understanding of speech was also disturbed: in his study he demonstrated beyond doubt the sensory aphasic nature of certain schizophrenic disorders of speech. As a counterpart to these findings in schizophrenics, I was able to show that patients with brain disease and war wounds in the left temporal lobe also demonstrated