An Atlas of
SCHIZOPHRENIA

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Preface

There have been major changes in attitudes towards schizophrenia in recent years. In clinical practice, more effective pharmacological and psychological treatments for schizophrenia have helped regenerate a sense of therapeutic optimism. In research, progress in a range of basic disciplines has opened up new avenues which promise to help unravel the abnormalities of brain development, structure and function which are at the core of the disorder. These have been complimented by advances from epidemiology which remind us that schizophrenia is not just a brain disorder, and that social and psychological factors can have a profound impact on its onset and outcome. Research in schizophrenia has never been more exciting. This Atlas is our attempt to put together a visual overview of this fascinating and challenging territory.

We have included many of the more familiar landmarks and monuments, but also some informative images of the most interesting new developments. Inevitably, because of the vast volume of new developments, our compilation has been somewhat selective. However, we hope this Atlas reflects our sense that a cohesive clinical and theoretical understanding of this complex disorder is now within reach, and that we can now bring hope and better care to sufferers.

Martin Stefan, Mike Travis and Robin M. Murray
November 2001
Foreword

Schizophrenia is a puzzle. Emil Kraepelin considered that his life's work had resulted merely in progress in understanding the psychoses, not a solution. Gottesman and Shields regarded the causes of schizophrenia as being an epigenetic puzzle, an analogy that continues to serve us well. Research in this area seems to produce ever more pieces, rather than fitting them together.

More importantly, schizophrenia puzzles patients who have the syndrome, their families and, as often as not, the clinicians who try to help them. Martin Stefan, Mike Travis and Robin Murray are experts in this field, and have produced an excellent and highly readable overview of the clinical features of the disorder, the epidemiological context, possible causes, and the current status of drug treatment. Clinicians working in all aspects of services for people with schizophrenia will find this an accessible and clear reference. Many may consider recommending the book to some patients and carers who want rather more information than is contained in standard educational materials.

An atlas is probably not the most obvious format for a book on schizophrenia but the authors have succeeded in producing a useful and interesting one. Diagrams, tables and figures give contemporary views an immediate impact, with modern techniques of investigation, such as neuroimaging, being particularly well suited to this format. These are balanced by the paintings from the Bethlem Royal Hospital Archives and Museum that entertain and fascinate alongside the factual information: they give an impression of the human as well as scientific and psychiatric aspects of schizophrenia. Thus, the book provides a useful map for all.

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CHAPTER 1

Clinical features

HISTORY AND CLASSIFICATION

Schizophrenia is arguably the most severe of the psychiatric disorders. It carries a lifetime risk of around 0.5–1%, and its early onset and tendency to chronicity mean that its prevalence is relatively high. Disability results particularly from negative symptoms and cognitive deficits, features that can have a greater impact on long-term functioning than the more dramatic delusions and hallucinations which often characterize relapses. The social and economic impact of the illness is enormous, and its impact on sufferers and their families can be devastating.

Although descriptions of people who may have had schizophrenia-like illnesses can be found throughout history (Figure 1.1) the first comprehensive descriptions date from the beginning of the 18th century (Figures 1.2 and 1.3)\(^1\). The modern concept of schizophrenia was first formalized by the German psychiatrist Emil Kraepelin (Figure 1.4)\(^2,3\) at the turn of the 20th century. Kraepelin, who drew on contemporary accounts of syndromes such as catatonia and hebephrenia, was the first to distinguish between

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Figure 1.1 Sketch to Illustrate the Passions: Agony – Raving Madness, by Richard Dadd (1854). In this painting, Richard Dadd (1817–1886) alludes to a pre-Kraepelinian distinction between ‘raving madness’ and ‘melancholic madness’. Dadd himself was a patient at the Bethlem (Bedlam) Hospital, England’s oldest mental hospital, and at Broadmoor, the hospital for the criminally insane. A Victorian painter best known for his fairy paintings, Dadd developed his illness at around the age of 25, when he became suspicious and preoccupied with religion, and developed delusions relating to the Egyptian god Assyris, beliefs that thoughts and commands which he had to obey were put into his head, and delusions that he was persecuted by the devil. At the age of 27, in response to these beliefs, he attacked and killed his father. He spent the rest of his life in institutional psychiatric care. Figure reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK.
Figure 1.2 Frontispiece from Illustrations of Madness (1810), by John Haslam1, which provides a vivid description of psychosis in an individual patient early in the industrial revolution. James Tilley Matthews was admitted to the Bethlem Hospital in 1797, after writing a threatening letter to a senior official in the British Admiralty. Haslam, who was Matthews’ doctor at the Bethlem, wrote his book as a rebuttal of claims made in court that Matthews was not insane. Matthews believed that a ‘gang of villains, profoundly skilled in pneumatic chemistry’ were assailing him: ‘while one of these villains is sucking out the brain of the person assailed, to extract his existing sentiments, another of the gang will force into his mind a train of ideas very different from the real subject of his thoughts’. He experienced many other unpleasant experiences, including ‘sudden death squeezing, stomach skinning, apoplexy making with the nutmeg grater, lengthening of the brain, thought making and laugh making’. Figure reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK

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Figure 1.3 Air-loom, by James Tilley Matthews, circa 1810. This plate is included in Haslam's book[^1]. It is Matthews' own 'diagram or plan of the cellar or place where the assassins rendezvous and work, showing their own and their apparatuses' relative positions, as it has at all times appeared to me by the sympathetic perception'. As well as the 'air-loom', Matthews indicates the sources of various abnormal experiences: the voices of the King, Bill, the Middle Man, the Glove Woman, Augusta, Charlotte, St Archy, and assorted visitors, who are 'not half so distinct as when they advance to the Loom Table, especially the Middle Position', together with a 'door into a back room where I have not the least perception of them beyond the said door'. Figure reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK.

the two major poles of severe mental illness. He described one group of patients in whom the clinical picture was dominated by disordered mood and who followed a cyclical pattern of relapse and relative remission; he termed this ‘manic depressive insanity’. Others had a deteriorating illness characterized by florid onset, often in adolescence, with a prolonged course marked by profound social and functional disability. He called the latter ‘dementia praecox’, and saw it as ‘a single morbid process’, endogenous rather than acquired, starting in youth and with dementia as a common outcome. This concept has since been enormously influential in guiding our perception of the disorder, even though Kraepelin himself came to recognize many of its limitations: for example, the disease was not always confined to younger people, the progression to dementia was not inevitable and in some individuals a recovery would be seen4.

The Swiss psychiatrist Eugen Bleuler (Figure 1.5) coined the term ‘schizophrenia’ in 1911 and this rapidly displaced dementia praecox5. Unlike Kraepelin, who was strongly influenced by the successes of clinical pathology in the search for causative agents in diseases such as syphilis and tuberculosis, Bleuler thought of schizophrenia in psychological rather than neuropathological terms. For Bleuler, the florid but highly variable symptoms of psychosis, such as delusions and hallucinations, were secondary, ‘accessory’ phenomena. At the core of the illness, he believed, was a more generalized psychological deficit, characterised by a ‘loosening of associations’ in the form of language, by deficits in volition and attention, and by incongruity of affect, ambivalence and autism.

Although intellectually compelling as a model of schizophrenia, Bleuler’s core symptoms were difficult to define reliably. In particular, the limits
of ‘simple schizophrenia’ (schizophrenia uncomplicated by ‘accessory’ symptoms) and ‘latent schizophrenia’ (people with odd personalities said to share some of the characteristics of the full-blown disorder) were difficult to define. This confusion was compounded by the clinical heterogeneity of schizophrenia, the lack of clear prognostic features and the failure to discover any definitive pathological abnormalities, and led to an expansion of the concept of schizophrenia to the extent that it became a vague synonym for severe mental illness with different meanings in different countries. This was especially the case in the USA, where Bleuler’s concepts held great sway, and in the former Soviet Union, where different models (incorporating, for example, anorexia nervosa sufferers and political dissenters) had developed.

In Western Europe, where the Kraepelinian formulation remained dominant, diagnosis was more consistent, partly because the positive psychotic symptoms central to the Kraepelinian definition are more readily and reliably determined. In 1959, Kurt Schneider (Figure 1.6) set out a list of such symptoms, which would be most likely, in the absence of organic brain disease, to lead to the diagnosis of schizophrenia. These ‘symptoms of the first rank’ were selected because they were relatively easily elicited and reliably identified, rather than because of any central theoretical importance; nevertheless, they were highly influential in determining diagnostic practice.

The International Pilot Study of Schizophrenia (IPSS; Figure 1.7) which investigated the illness in several centers around the world, suggested a high degree of consistency in the clinical picture of schizophrenia when diagnosed using strict diagnostic rules. This appeared to hold true in rural and in urban areas, and both in Western European countries and in developing countries. The use of operationalized diagnostic rules in this, and in the earlier US/UK Diagnostic Project, greatly facilitated research into the epidemiology of the disorder. Since then, as a result of the development and use of operationalized definitions in the Diagnostic and Statistical Manual of Mental Disorders (DSM–IV) in the USA, and the International Classification of Diseases (ICD–10) worldwide, definitions of schizophrenia have been considerably narrowed.

**SCHNEIDER’S SYMPTOMS OF THE FIRST RANK**

- Audible thoughts
- Voices heard arguing
- Voices heard commenting on one’s actions
- The experience of influences playing on the body
- Thought withdrawal and other interferences with thought
- Diffusion of thought
- Delusional perception
- Feelings, impulses and volitional acts experienced as the work or influence of others

Figure 1.6  Kurt Schneider. In 1959 he listed the ‘first rank features’ of schizophrenia. One of these symptoms, in the absence of organic disease, persistent affective disorder, or drug intoxication, was sufficient for a diagnosis of schizophrenia.
Both ICD-10 and DSM-IV identify a number of subtypes of schizophrenia. In clinical practice, a distinction is also often made between acute and chronic schizophrenia. A third way of thinking about the varied clinical picture seen in schizophrenia is in terms of syndromes (Figure 1.9). Unlike subtypes, syndromes do not

Although diagnosis still depends on the presence of combinations of symptoms and the course of illness, and diagnostic practice may vary to some extent from country to country, there is a core group of patients who fulfill all definitions and would be diagnosed as suffering from schizophrenia anywhere in the world (Figure 1.8).

Figure 1.7 Geographical variations in the incidence of schizophrenia, (a) broadly defined and (b) narrowly defined. These data from the International Pilot Study of Schizophrenia were taken as evidence that, particularly when narrow operational criteria are used, the incidence of schizophrenia is remarkably constant between countries. Although this was very influential when it was published, subsequent studies have demonstrated that the incidence does indeed vary, with higher rates found in those born or brought up in cities and in certain immigrant groups.
Symptoms of schizophrenia appear to cluster into three syndromes: ‘positive’, or reality distortion, characterized by delusions and hallucinations; ‘negative’, consisting of the ‘deficit’ symptoms including ‘negative thought disorder’, such as poverty of speech and poverty of content of speech; and ‘disorganization’, in which ‘positive’ formal thought disorder, for example knight’s move thinking or loosening of associations are seen.
represent exclusive domains; any individual patient may have features of more than one syndrome. Initial syndromal studies distinguished between positive and negative symptoms, but more recent work using factor-analytic studies of associations between symptoms supports the existence of a third syndrome, characterized by positive thought disorder or ‘disorganization’. Thus, most clinicians would recognize the existence of psychotic symptom clusters in the following three domains:

1. Delusions and hallucinations, the so-called ‘positive’ symptoms.
2. Positive thought disorder or ‘disorganization’.
3. Social withdrawal, apathy, self-neglect, poverty of speech and of content of speech, and other broadly ‘negative’ symptoms.

**CLINICAL PRESENTATION AND SYMPTOMATOLOGY**

As in the rest of medicine, reliable diagnosis depends on accurate history taking and clinical examination. The family and personal history may offer important etiological clues. There may be a family history of schizophrenia, complications in pregnancy or birth, or a personal history of childhood problems. Recent studies, including the follow-up of the British 1946 birth cohort\(^\text{10}\), have suggested that children who later develop schizophrenia are more likely to have a history of developmental delay, as well as slightly lower IQ and educational achievements than other children (Figure 1.10). They are also more likely to have interpersonal and behavioral difficulties. Mothers may describe their premorbid personality as having shown emotional detachment; ‘preschizophrenic’ children may appear cold and aloof, avoiding play and engaging in solitary occupations. Some may be prone to temper tantrums, tend to avoid competition, have odd ideas and seek refuge in fantasy. These characteristics are also over-represented in the families of schizophrenics, although they do not reliably predict the development of schizophrenia.

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**Figure 1.10** Differences between children destined to develop schizophrenia as adults and the general population were found across a range of developmental domains. As with some other adult illnesses, the origins of schizophrenia may be found in early life. This figure illustrates the associations between adult-onset schizophrenia and childhood sociodemographic, neurodevelopmental, cognitive, and behavioral factors in a cohort of 5362 people born in one week in 1946. Thirty cases of schizophrenia arose between ages 16 and 43 years. Data with permission from Jones P, Rodgers B, Murray R, et al. Child development risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet* 1994;344:1398–1402
### Differential Diagnosis of Schizophrenia

**‘Functional’**
- Schizotypal disorder
- Persistent delusional disorders
- Acute and transient psychotic disorders
- Schizoaffective disorders
- Induced delusional disorder
- Mania
- Other nonorganic psychotic disorders

**‘Organic’**
- Drug/substance-induced psychosis (e.g., alcohol withdrawal, amphetamines, crack cocaine, LSD, cannabis, PCP, and also steroids, dopamine agonists and some heavy metals)
- Epilepsy – in particular, the fits of temporal lobe epilepsy may resemble an acute psychotic episode
- Tumors, either primary or secondary
- Stroke
- Early dementia
- Long-term sequelae of head injury
- Endocrine causes (e.g. Cushing’s disease; rarely hyper- and hypothyroidism)
- Infections (e.g. encephalitis, meningitis, neurosyphilis)
- Multiple sclerosis
- Autoimmune disorders such as systemic lupus erythematosus (SLE)
- Metabolic disorders (e.g., hepatic failure, uremia, hypercalcemia, acute intermittent porphyria)

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### Hallucinations

Hallucinations are defined as false perceptions in the absence of a real external stimulus. They are perceived as having the same quality as real perceptions and are not usually subject to conscious manipulation.

Hallucinations in schizophrenia may involve any of the sensory modalities. The most common are auditory hallucinations in the form of voices, which occur in 60–70% of patients diagnosed with schizophrenia. Although voices in the second person are most common, the characteristic ‘Schneiderian’ voices are in the third person and provide a running commentary on the patient’s actions, arguing about the patient or repeating the patient’s thoughts. Voices may be imperative, ordering the patient to harm himself or others. Visual hallucinations occur in about 10% of patients, but should make one suspicious of an organic disorder. Olfactory hallucinations are more common in temporal lobe epilepsy than schizophrenia, and tactile hallucinations are probably experienced more frequently than is reported by patients.

No single type of hallucination is specific to schizophrenia, and the duration and intensity are probably most important diagnostically.
### CATATONIC SYMPTOMS

These mainly motor symptoms may occur in any form of schizophrenia but are particularly associated with the catatonic subtype

<table>
<thead>
<tr>
<th>Ambitendence</th>
<th>Alternation between opposite movements.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echopraxia</td>
<td>Automatic imitation of another person’s movements even when asked not to.</td>
</tr>
<tr>
<td>Stereotypies</td>
<td>Repeated regular fixed parts of movement (or speech) that are not goal directed, e.g. moving the arm backwards and outwards repeatedly while saying ‘but not for me’.</td>
</tr>
<tr>
<td>Negativism</td>
<td>Motiveless resistance to instructions and attempts to be moved, or doing the opposite of what is asked.</td>
</tr>
<tr>
<td>Posturing</td>
<td>Adoption of inappropriate or bizarre bodily posture continuously for a substantial period of time.</td>
</tr>
<tr>
<td>Waxy flexibility</td>
<td>The patient’s limbs can be ‘molded’ into a position and remain fixed for long periods of time.</td>
</tr>
</tbody>
</table>

### THOUGHT DISORDERS

Usually a disorder of the form of thought, such that the speech is difficult to follow or incoherent and follows no logical sequence.

**Knight’s move thinking** (or derailment) occurs when the patient moves from one train of thought to another which has no apparent connection to the first – it takes its name from the chess piece that moves two steps forward and one to the side. A less severe form is called **loosening of associations** which merges into **tangential thinking** and **loss of goal**.

Some patients may invent **neologisms** (new words), exhibit **verbal stereotypy** (repetition of a single word or phrase out of context), or use **metonyms** (ordinary words given a special personal meaning).

Negative thought disorder includes **poverty of speech** (limited quantity of speech), and **poverty of content of speech** (limited meaning conveyed by speech).

### DISORDERS OF THOUGHT POSSESSION

Disorders of thought possession in schizophrenia are sometimes called thought alienation. The patient has the experience that his thoughts are under the control of an outside agency or that others are participating in his thinking.

**Thought insertion** The patient believes that thoughts that are not his own are being put into his mind by an external agency.

**Thought withdrawal** The patient believes that thoughts are being removed from his mind by an external agency.

**Thought broadcasting** The patient believes that his thoughts are being ‘read’ by others, as if they were being broadcast.

**Thought blocking** Involves a sudden interruption of the train of thought, before it is completed, leaving a ‘blank’. The patient suddenly stops talking and cannot recall what he has been saying or thinking.
**Delusions**

A delusion is a fixed, false personal belief held with absolute conviction despite all evidence to the contrary. The belief is outside the person’s normal culture or subculture and dominates their viewpoint and behavior. Delusions may be described in terms of their **content** (e.g. delusions of persecution or grandeur). They can be **mood congruent** (the content of delusion is appropriate to the mood of the patient), or **mood incongruent**. Delusions are described as **systematized** if they are united by a single theme.

A **primary delusion** arises fully formed without any discernible connection with previous events (also called autochthonous delusions), e.g. “I woke up and knew that my daughter was the spawn of Satan and should die so that my son could be the new Messiah”. **Secondary delusions** can be understood in terms of other psychopathology, for example hallucinations: “The neighbors must have connected all the telephones in the building; that’s why I can hear them all the time”.

The term delusional mood is slightly confusing in that it does not describe an abnormal belief, but refers to an ill-defined feeling that something strange and threatening is happening which may manifest as perplexity, uncertainty or anxiety. This may precede a primary delusion or a delusional perception, which involves a real perception occurring almost simultaneously with a delusional misinterpretation of that perception, e.g. “I saw the traffic lights change from red to green and knew that I was the rightful heir to the throne of England”.

**Overvalued ideas** are unreasonable and sustained intense preoccupations maintained with a strong emotional investment but less than delusional intensity. The idea or belief held is demonstrably false and not usually held by persons from the same subculture.

**Delusions may be classified in terms of their content, for example delusions of...**

**Persecution** An outside person or force is in some way interfering with the sufferer’s life or wishes them harm, e.g. “The people upstairs are watching me by using satellites and have poisoned my food”.

**Reference** The behavior of others, objects, or broadcasts on the television and radio have a special meaning or refer directly to the person, e.g. “A parcel came from Sun Alliance and the radio said that ‘the son of man is here’, on a Sunday, so I am the son of God”.

**Control** The sensation of being the passive recipient of some controlling or interfering agent that is alien and external. This agent can control thoughts, feeling and actions (passivity experiences), e.g. “I feel as if my face is being pulled upwards and something is making me laugh when I’m sad”.

**Grandeur** Exaggerated belief of one’s own power or importance, e.g. “I can lift mountains by moving my hands, I could destroy you!”.

**Nihilism** Others, oneself, or the world does not exist or is about to cease to exist (often called Cotard’s syndrome), e.g. “The inside of my tummy has rotted away. I have no bowels”.

**Infidelity** One’s partner is being unfaithful (also known as delusional jealousy or the Othello syndrome).

**Doubles** A person known to the patient, most frequently their spouse, has been replaced by another (also known as Capgras’ syndrome or, confusingly, ‘illusion’ of doubles).

**Infatuation** A particular person is in love with the patient (also known as erotomania or de Clerambault’s syndrome).

**Somatic** Delusional belief pertaining to part of the person’s body, e.g. “My arms look like they’ve been melted and squashed into a mess”.
THE ACUTE ILLNESS

The onset may be rapid, or slow and insidious. In the latter situation in particular, there may be a prolonged period of undiagnosed illness in which the affected person slowly becomes more withdrawn and introverted. They may develop unusual interests, particularly of a religious or philosophical kind, and drift away from family and friends. They may also begin to fail in their occupation or schoolwork. This process can take weeks to years but eventually, often with a seemingly precipitating event, the symptoms of florid illness appear.

The acute symptoms are variable but usually include delusions, hallucinations, abnormal thought processes and passivity experiences (Figures 1.11–1.16). In addition, there may be formal thought disorder, and flat or inappropriate affect. Abnormal motor signs, sometimes termed catatonic, used to be common but now are much less so in Western countries. At this stage of the illness, positive symptoms tend to dominate the clinical picture.
Figure 1.12  Grey self-portrait, by Bryan Charnley. This painting illustrates aspects of Charnley's psychotic symptoms, including that of hearing voices. Reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK.

Figure 1.13  Puppeteers, by Phyllis Jones, 1936. This patient was a talented artist, who was admitted to a psychiatric hospital at the age of 22 years complaining of hearing voices, and convinced her food was being poisoned. The clinical description suggests a florid psychotic illness, of acute onset, accompanied by severe affective disturbance. Reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK.
Figure 1.14 Cordron, by Gilbert Price. This patient was admitted at the age of 22 years. His extreme shyness and eccentric behavior culminated in his arrest for 'suspicious' conduct. Neologisms, elaborated into complex descriptive systems of pictures, chimneys and other objects, dominated his psychopathology. Reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK.
NEGATIVE SYMPTOMS

**Poverty of speech**  Restriction in the amount of spontaneous speech and in the information contained in speech (alogia).

**Flattening of affect**  Restriction in the experience and expression of emotion.

**Anhedonia–asociality**  Inability to experience pleasure, few social contacts and social withdrawal.

**Avolition-apathy**  Reduced drive, energy and interest.

**Attentional impairment**  Inattentiveness at work and interview.

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**The Chronic Illness**

Eventually, even without treatment, the acute symptoms of schizophrenia usually resolve. Unfortunately this does not always mean that the patient will fully recover. Over 50% of patients diagnosed as suffering from schizophrenia will show evidence of a significant degree of negative symptomatology. Furthermore, in chronic schizophrenia, positive symptoms also frequently remain, although they tend not to predominate.

Negative symptoms may also be seen in the acute episode, and their onset can often precede (as one form of ‘schizophrenic prodrome’) the development of typical positive symptoms. Negative symptoms are multifactorial in origin. Primary negative symptoms may be difficult to distinguish from those secondary to florid positive symptoms.

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**Figure 1.15**  Cats, by Louis Wain (1860–1939). Wain was a British artist who became famous for his drawings of cats. He was a patient at the Bethlem Hospital in the 1920s. Paintings such as these, which are suggestive of disorganization, visual perceptual disturbances and abnormalities of affect, have been taken as illustrative of his psychological decline, although more recent scholarship suggests that they were not out of keeping with contemporary design practice. Reproduced with kind permission of the Bethlem Royal Hospital Archives and Museum, Beckenham, Kent, UK.
psychotic symptomatology, while others may represent side-effects of antipsychotic drugs. It is often difficult to differentiate between the negative symptoms of schizophrenia and the symptoms of a depressive illness. Depression is common in schizophrenia, and often becomes evident as the acute episode resolves.

True primary negative symptoms are often described as ‘deficit’ symptoms. Their frequency is markedly increased in chronic schizophrenia and is related to poor prognosis, poor response to antipsychotic drugs, poor premorbid adjustment, cognitive impairment and structural brain abnormalities (sometimes called ‘type 2’ schizophrenia). These symptoms are not easy to treat, are often very distressing to families and carers, and are the most important cause of long-term disability.

**COURSE AND OUTCOME OF SCHIZOPHRENA**

The in-patient psychiatric population has fallen dramatically since the 1950s in Western countries, when effective antipsychotic drug treatments first became available (Figure 1.17). However, the outcome of schizophrenia, even with treatment, remains variable (Figure 1.18 and Table 1.1). Longitudinally, the typical course of chronic schizophrenia is described in Figure 1.19. Schizophrenia also carries a high mortality. Rates of suicide in follow-up studies vary from about 2%
Figure 1.17  There was a steady increase in the in-patient mental hospital population in England and Wales during the hundred years from 1860. This was due to a combination of factors, including increased urbanization and changes in mental health legislation. The sharp decline in this population coincided with the introduction of effective antipsychotic medication, together with changes in health policy and legislation.

Figure 1.18  Course of schizophrenia. Four typical patterns in the course of schizophrenia are described by Shepherd and colleagues who followed up a cohort of patients with an operational (CATEGO-defined) diagnosis of schizophrenia who were admitted to a UK hospital over an 18-month period. Figure reproduced with permission from Shepherd M, Watt D, Falloon I, et al. The natural history of schizophrenia: a five-year follow-up study of outcome prediction in a representative sample of schizophrenics. Psychol Med Monogr 1989;15 (Suppl.):1–46.
to 10% and the overall rate of suicide in schizophrenia is estimated to be in the region of 10% (Figure 1.20).

**FACTORS AFFECTING PROGNOSIS**

Certain clinical features are associated with a poor prognosis: early or insidious onset, male sex, negative symptoms\(^{19,20}\) (Figure 1.8), lack of a prominent affective component or clear precipitants, family history of schizophrenia, poor premorbid personality, low IQ, low social class, social isolation, and significant past psychiatric history.

Several studies have demonstrated an association between longer duration of untreated illness and poorer outcome. For example, Loebel and colleagues\(^{21}\) found that a longer duration of both psychotic and prodromal symptoms prior to treatment was associated with a lesser likelihood of remission. The longer the duration of pretreatment psychotic symptoms, the longer the time to remission. These data suggest that early detection and intervention in schizophrenia may be important in minimizing subsequent disability.

**Table 1.1 Summary of long-term clinical outcome studies in schizophrenia.**

Table reproduced with permission from Frangou S, Murray RM. Schizophrenia. London: Martin Dunitz, 1997

<table>
<thead>
<tr>
<th>Study</th>
<th>Years of follow-up</th>
<th>Number of patients</th>
<th>Good clinical outcome (%)</th>
<th>Poor clinical outcome (%)</th>
<th>Social recovery (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ciompi 1980(^{11,12})</td>
<td>37</td>
<td>289</td>
<td>27</td>
<td>42</td>
<td>39</td>
</tr>
<tr>
<td>Bleuler 1978(^{13})</td>
<td>23</td>
<td>208</td>
<td>20</td>
<td>24</td>
<td>51</td>
</tr>
<tr>
<td>Bland &amp; Orme 1978(^{14})</td>
<td>14</td>
<td>90</td>
<td>26</td>
<td>37</td>
<td>65</td>
</tr>
<tr>
<td>Salokangas 1983(^{15})</td>
<td>8</td>
<td>161</td>
<td>26</td>
<td>24</td>
<td>69</td>
</tr>
<tr>
<td>Shepherd et al., 1989(^{16})</td>
<td>5</td>
<td>49</td>
<td>22</td>
<td>35</td>
<td>45</td>
</tr>
</tbody>
</table>

**Figure 1.19** Breier and colleagues\(^{18}\) among others have suggested that, despite the heterogeneity of schizophrenia, a model of a common course of illness can be derived. Based on evidence from their own studies and other long-term follow-up studies they describe an earlier deteriorating phase usually lasting some 5 years. During this time there is a deterioration from premorbid levels of functioning, often characterized by frank psychotic relapses. After this phase much less fluctuation can be expected and the illness enters a ‘stabilization’ or ‘plateau’ phase. This period may continue into the fifth decade, when there is a third ‘improving’ phase for many patients. Figure reproduced with permission from Breier A, Schreiber JL, Dyer J, Pickar D. National Institute of Mental Health longitudinal study of chronic schizophrenia. Prognosis and predictors of outcome. Arch Gen Psychiatry 1991;48:239–46
Figure 1.20 A series of self-portraits by Bryan Charnley which vividly illustrate his experiences as he came off medication. His descent into paranoia, hallucinations and depression is graphically depicted and explained with reference to his diary entries. Sadly the series ended with his death from suicide. Figures reproduced with kind permission of Mr Terence Charnley.
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5 Chapter 5 Psychosocial management
