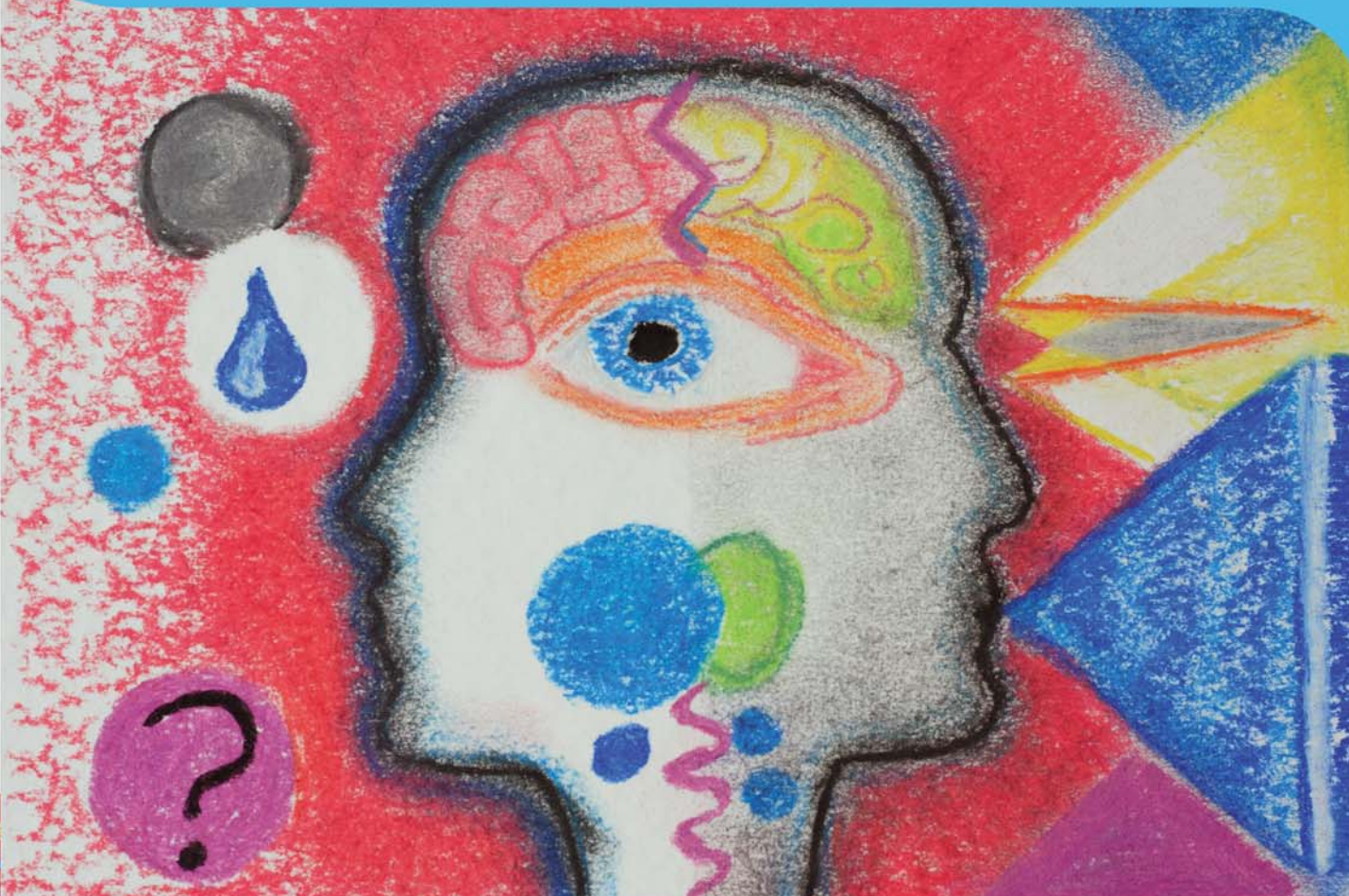


Neel Burton Psychiatry

SECOND EDITION

Foreword by Robert Howard

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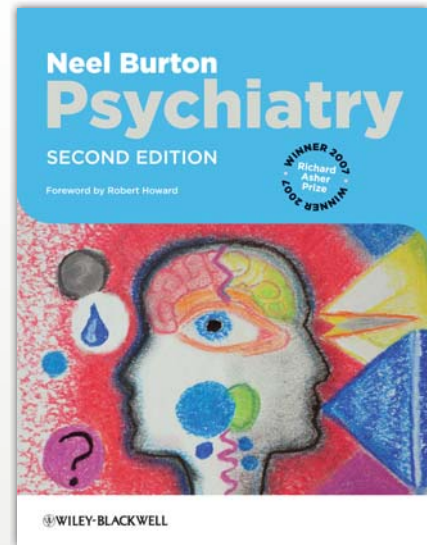
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Schizophrenia and other psychotic disorders

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Key learning objectives

- First rank symptoms of schizophrenia
- Aetiological factors in schizophrenia, including the dopamine hypothesis
- Clinical features of schizophrenia: positive symptoms, disorganised symptoms, negative symptoms
- Differential diagnosis of schizophrenia
- Management of schizophrenia
- Prognostic factors in schizophrenia

The story of VL

A 23-year-old female anthropology student of Afro-Caribbean descent, VL, is brought to Accident and Emergency by one of her colleagues. She is very agitated and difficult to assess. The psychiatry SHO on nights is able to make out that she is hearing three or four male voices coming from outside her head. These voices are talking together about her, making fun of her, blaming her for her family's financial problems, and commenting on her thoughts and behaviour. She is convinced that they are the voices of SAS paratroopers engaged by her parents to destroy her. It seems that the paratroopers are trying to put harmful thoughts, such as the thought of cutting off her fingers one by one, into her head. Any further questioning only serves to make her more agitated. When the SHO leaves the interview room she screams, 'I've seen your belt, they've sent you, they've sent you to distract me. I can't... I can't fight them anymore!'

VL's colleague reports that she has been behaving oddly for the past six months, and that she has not been to lectures since the beginning of last term. The SHO phones VL's house and one of her flatmates tells him that she has been locking herself in her room for hours on end. The flatmate thinks that she

started hearing the voices about 10 days ago after she discovered that a close childhood friend recently died from leukaemia.

The SHO ascertains that VL hasn't taken any drugs and that she doesn't have a psychiatric or medical history. He makes a provisional diagnosis of acute schizophrenia-like psychosis as, according to ICD-10, her symptoms have not been present for long enough to make a diagnosis of schizophrenia. After speaking to his registrar, he prescribes 1 mg of lorazepam to sedate her and arranges to admit her for further assessment and treatment. Three days later she is started on the antipsychotic drug risperidone by her consultant psychiatrist.

Issues raised by this case history are:

- Was the SHO justified in phoning VL's house to talk to her flatmate?
 - Should VL be detained under the Mental Health Act? Can the SHO do this?
 - If the patient refuses to take the risperidone, can it be enforced under the Mental Health Act?
- Refer back to Chapter 3 for the answers.

A brief history of schizophrenia

If you talk to God, you are praying. If God talks to you, you have schizophrenia.

Thomas Szasz

Although the oldest description of schizophrenia can be traced back to the second millennium before Christ, it is not until 1887 that **Emil Kraepelin** first recognised it as a distinct entity, distinguishing it from manic-depressive psychosis and naming it **dementia praecox** ('dementia of early life') to differentiate it from other forms of dementia such as Alzheimer's disease. Further to his credit, Kraepelin distinguished at least three clinical varieties of schizophrenia: catatonia, hebephrenia, and paranoia. Although he repeatedly stressed the diversity of signs and symptoms occurring in dementia praecox, he found a chronic course and a poor outcome to be its characteristic defining features.

In 1911 **Eugen Bleuler** (1857–1939) coined the term *schizophrenia* (Ancient Greek, 'splitting of the mind') because, unlike Kraepelin, he did not think that the illness inevitably lead to mental deterioration (*dementia*) nor that it inevitably affected young people (*praecox*). Although Bleuler's term of 'schizophrenia' has been upheld by history, it has led to much confusion about the nature of the illness. The term does *not* refer to 'split personality' but to the splitting of an individual's thinking and feeling processes ('split personality' or multiple personality disorder is a very rare condition classified under dissociative disorders, see Chapter 8). Bleuler's description of schizophrenia put more emphasis on thought disorder and on negative symptoms than on the more florid positive, or psychotic, symptoms. He described the primary symptoms of the illness as **ambivalence**, **autistic behaviour**, **abnormal associations**, and **abnormal affect** (the so-called 'four As').

In 1959 the German psychiatrist **Kurt Schneider** defined the first rank symptoms of schizophrenia, symptoms supposed to be specific to, and therefore pathognomonic of, schizophrenia (Table 4.1). Schneider's first rank symptoms largely consisted of florid psychotic symptoms



Figure 4.1 Double exposure photograph (1895) of Richard Mansfield, who played the roles of both Dr Jekyll and Mr Hyde. People with schizophrenia do not suddenly change into a different, unrecognisable person.

such as thought insertion, thought broadcasting, and third person auditory hallucinations – symptoms held together by a common theme of loss of control over thoughts, feelings, and the body. Unfortunately these symptoms are common to many psychotic disorders and are therefore not as useful as originally thought at differentiating schizophrenia from other psychotic disorders. They are also absent in about 20% of schizophrenia sufferers.

Case studies of Schneider's first rank symptoms

Echo de la pensée

A 32-year-old housewife complained of a man's voice, speaking in an intense whisper from a point about two feet above her head. The voice would repeat almost all the patient's goal-directed thinking – even the most banal thoughts. The patient would think, 'I must put the kettle on' and after a pause of not more than one second the voice would say, 'I must put the kettle on'. It would often say the opposite, 'Don't put the kettle on'.

Thought insertion

A 29-year-old housewife said, 'I look out of the window and I think the garden looks nice and the grass looks cool, but the thoughts of Eamonn Andrews come into my mind. There are no other thoughts there, only his ... He treats my mind like a screen and flashes his thoughts on to it like you flash a picture'.

Thought withdrawal

A 22-year-old woman said, 'I am thinking about my mother, and suddenly my thoughts are sucked out of my mind by a phrenological vacuum extractor, and there is nothing in my mind, it is empty ...'

Thought broadcasting

A 21-year-old student said, 'As I think, my thoughts leave my head on a type of mental ticker-tape. Everyone around has only to pass the tape through their mind and they know my thoughts'.

Passivity of affect

A 23-year-old female patient reported, 'I cry, tears roll down my cheeks and I look unhappy, but inside I have a cold anger because they are using me in this way, and it is not me who is unhappy, but they are projecting unhappiness onto my brain. They project upon me laughter, for no reason, and you have no idea how terrible it is to laugh and look happy and know it is not your, but their, emotions'.

Passivity of volition

A 29-year-old shorthand typist described her actions as follows, 'When I reach my hand for the comb it is my hand and arm which move, and my fingers pick up the comb, but I don't control them ... I sit there watching them move, and they are quite independent, what they do is nothing to do with me ... I am just a puppet who is manipulated by cosmic strings. When the strings are pulled my body moves and I cannot prevent it'.

Passivity of impulse

A 26-year-old engineer emptied the contents of a urine bottle over the ward dinner trolley. He said, 'The sudden impulse came over me that I must do it. It was not my feeling, it came into me from the X-ray department, that was why I was sent there for implants yesterday. It was nothing to do with me, they wanted it done. So I picked up the bottle and poured it in. It seemed all I could do'.

Somatic passivity

A 38-year-old man had jumped from a bedroom window, injuring his right knee which was very painful. He described his physical experience as, 'The sun-rays are directed by a US army satellite in an intense beam which I can feel entering the centre of my knee and then radiating outwards causing the pain'.

Delusional perception

A young Irishman was at breakfast with two fellow-lodgers. He felt a sense of unease, that something frightening was going to happen. One of the lodgers pushed the salt cellar towards him (he appreciated at the time that this was an ordinary salt cellar and his friend's intention was innocent). Almost before the salt cellar reached him he knew that he must return home, 'to greet the Pope, who is visiting Ireland to see his family and to reward them... because our Lord is going to be born again to one of the women... And because of this they [all the women] are born different with their private parts back to front'.

C. S. Mellor, First rank symptoms in schizophrenia. *British Journal of Psychiatry* (1970), 117, 15–23

Table 4.1 Schneider's first rank symptoms.**Auditory hallucinations**

Third person	Voices discuss or argue about the patient
Running commentary	Voices comment on the patient's thoughts and behaviour
<i>Gedankenlautwerden</i> and <i>echo de la pensée</i>	The patient's thoughts are heard as, or shortly after, they are formulated

Delusions of thought control

Thought insertion	Alien thoughts are put into the patient's mind by an external agency
Thought withdrawal	The opposite; thoughts are removed from the patient's mind by an external agency
Thought broadcasting	The patient's thoughts are overheard by, or otherwise accessible to, others

Delusions of control (passivity phenomena)

Passivity of affect, volition, and impulses	The patient's affect, impulses, and volition are under the control of an external agency
Somatic passivity	The patient's bodily sensations are under the control of an external agency

Delusional perception

The patient attributes delusional significance to normal percepts

Reminder of some important definitions

Delusion: an unshakeable (fixed) belief that is held in the face of evidence to the contrary, and that cannot be explained by culture or religion.

Hallucination: a percept that arises in the absence of a stimulus, and that is not subject to conscious manipulation.

Gedankenlautwerden: thoughts are 'heard' as they are being formulated.

Echo de la pensée: thoughts are 'heard' shortly after they have been formulated.

Febrile illnesses such as malaria had been observed to moderate psychotic symptoms, and in the early 20th century fever therapy became a popular form of treatment for schizophrenia. Psychiatrists tried to induce fevers in their patients, sometimes by means of injections of

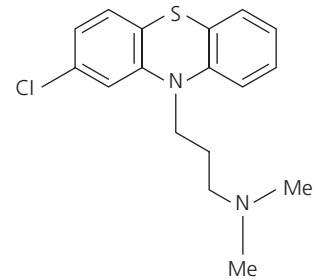


Figure 4.2 Chemical structure of the first anti-psychotic, chlorpromazine, stumbled upon by Henri Laborit in 1952. The French surgeon had been looking for a drug for the treatment of surgical shock.

sulphur or oil. Other popular but unsatisfactory treatments included electroconvulsive treatment, lobotomy, and sleep therapy. The first antipsychotic drug, **chlorpromazine**, first became available in the 1950s. Described as a 'chemical lobotomy', it controlled the positive symptoms of schizophrenia and made patients 'indifferent'. The side-effects of chlorpromazine and related drugs included tremors, restlessness, loss of muscle tone, and postural disorders, and this earned the drug class the name of **neuroleptic** (Ancient Greek, 'nerve seizure').

Although discovered in 1959, the second-generation or 'atypical' antipsychotic **clozapine** did not receive regulatory approval until the 1990s because of its potentially fatal side-effect of agranulocytosis. Like other atypical antipsychotics that came after it, clozapine had, overall, fewer side-effects than chlorpromazine, and controlled both the positive and the negative symptoms of schizophrenia. Today atypical antipsychotics such as risperidone, olanzapine, and quetiapine have become the first line of treatment for schizophrenia. Clozapine, because of its potentially fatal side-effect of agranulocytosis, is reserved for treatment-resistant cases and requires close monitoring (see later).

Epidemiology

- **Prevalence:** figures for the lifetime prevalence of schizophrenia depend on the diagnostic criteria used but are usually quoted at around 1%.
- **Sex ratio:** unlike many other mental disorders such as depression and anxiety disorders, which tend to be more common in women, schizophrenia affects men and women in more or less equal numbers. However, the illness tends to present at a younger age in men, and

also tends to affect them more severely. Why this should be so is at present unclear.

- **Age of onset:**
 - Any age, but rare in childhood and early adolescence and uncommon after the age of 45 (suspect organic causes)
 - Mean age of onset in men is 28 years
 - Mean age of onset in women is 32 years, with peak incidence in the 20s and 40s (bimodal distribution).
- **Geography:** generally speaking, lifetime prevalence is similar across populations and stable over time, despite the reduced reproductive fitness of affected individuals. Prevalence and severity tend to be greater in urban areas than in rural areas.
 - *Drift hypothesis:* prevalence is higher in urban areas because schizophrenia sufferers drift from rural areas to urban areas as a consequence of their illness or its prodromal symptoms.
 - *Breeder hypothesis:* prevalence is higher in urban areas because the stress of urban living actually plays a part in the aetiology of the illness.
- **Migration:** prevalence is higher in immigrants, and especially in second-generation Afro-Caribbean immigrants to the UK (about a 10-fold increase). This may reflect such factors as poor integration, socioeconomic deprivation, or diagnostic bias amongst

psychiatrists (although recent studies seem to rule out this factor).

- **Seasonality of births:** increased lifetime prevalence (+5–10%) if born from January to April in the northern hemisphere or July to September in the southern hemisphere. Seasonality of births may reflect a viral aetiology.
- **Socioeconomic status:** observed differences are likely accounted for by social drifting, as the socioeconomic status of the fathers of schizophrenia sufferers are normally distributed.

Aetiology

Genetics

Several candidate genes for schizophrenia have been identified, and it seems likely that any given individual has a complement of genetic variations that make him or her more or less vulnerable to developing the disorder. Individual genes identified so far include *dysbindin* (chromosome 6p), *neuregulin 1* (8p), and *G72* (13q). A concordance rate of about 50% in monozygotic twins suggests that genetic and environmental factors are more or less equally involved in the expression of the disorder.

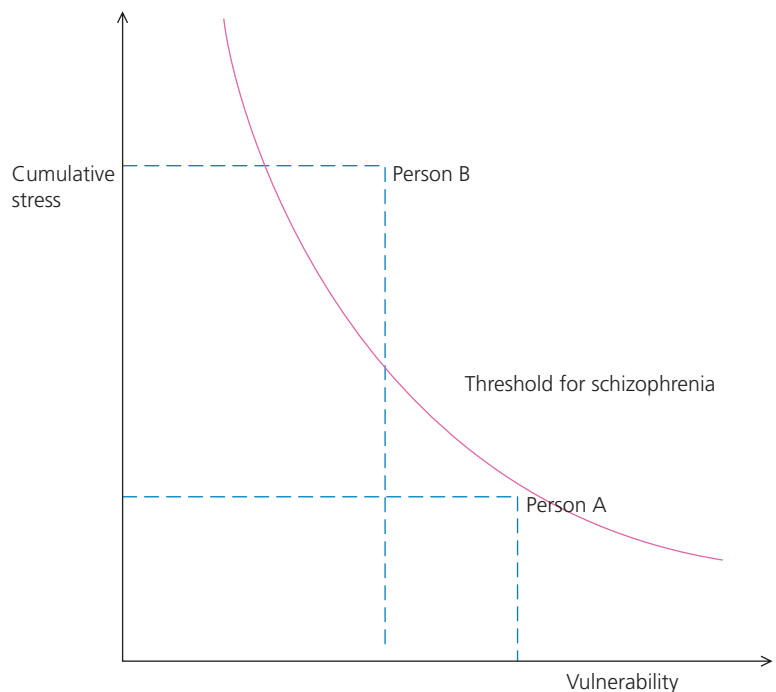


Figure 4.3 The stress–vulnerability or stress–diathesis model for schizophrenia. A person develops schizophrenia when the stress that he or she faces becomes greater than his or her ability to cope with it. Person A is highly vulnerable to developing schizophrenia but does not develop it because he or she is subjected to only moderate amounts of stress. On the other hand, person B is only moderately vulnerable to developing schizophrenia but does develop it because he or she is subjected to unusually high amounts of stress.

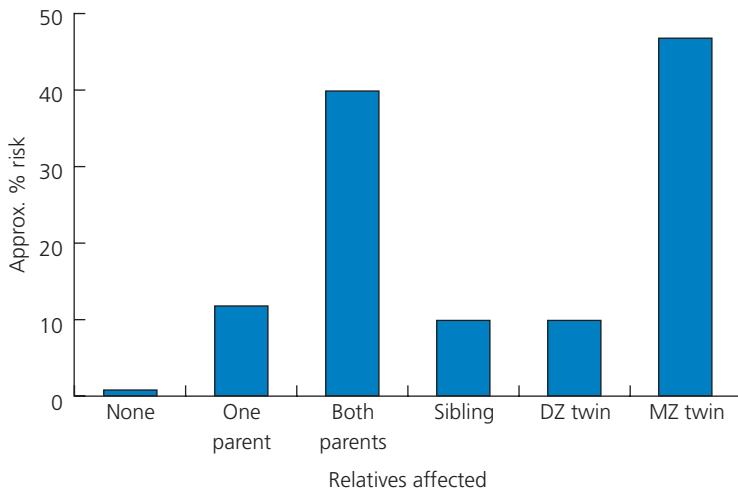


Figure 4.4 Lifetime risks of schizophrenia according to relative(s) affected.

Family studies

The lifetime risks of schizophrenia according to relative(s) affected are illustrated in Figure 4.4.

Adoption studies

Biological offspring of schizophrenic parents adopted by non-schizophrenic parents maintained their increased risk, but biological offspring of non-schizophrenic parents adopted by schizophrenic parents did not have an increased risk at all.

Neurochemical abnormalities

The **dopamine hypothesis of schizophrenia** states that schizophrenia results from increased levels of dopamine in the brain.

Snyder formulated the dopamine hypothesis of schizophrenia in 1976 based on four sets of findings:

- Amphetamines increase dopamine release and in high doses can induce a schizophrenia-like psychosis ('amphetamine psychosis')
- Amphetamines and other dopaminergic agents exacerbate the symptoms of schizophrenia
- Dopamine antagonists, specifically phenothiazines such as chlorpromazine and butyrophenones such as haloperidol, are effective in the treatment of schizophrenia

- The clinical potency of these typical antipsychotics is correlated to their affinity for dopamine (D_2) receptors. In addition:

- Amphetamine psychosis responds to antipsychotics
- Antipsychotics used in the treatment of schizophrenia can result in parkinsonian side-effects
- L-dopa used in the treatment of Parkinson's disease can result in schizophrenia-like symptoms
- Post-mortem studies suggest that there are increased levels of dopamine and dopamine receptors in the brains of schizophrenia sufferers (although this finding may have resulted from antipsychotic treatment and not from the disease process itself).

Unfortunately, not all findings support the dopamine hypothesis of schizophrenia. In particular:

- Schizophrenia is a relapsing and remitting disorder that presents in a variety of clinical pictures. Such a complex disorder is unlikely to be accounted for by the dopamine hypothesis
- The clinical effects of antipsychotics are only apparent several days after starting treatment, and then only in about 70–85% of schizophrenia sufferers
- Studies of brain tissue, cerebrospinal fluid, and plasma levels of dopamine and its metabolites (homovanillic acid, HVA) do not uniformly support an increase in dopamine levels. In some cases, levels of HVA in the cerebrospinal fluid have even been found to be *decreased*
- Increased levels of dopamine can transiently improve the negative symptoms of schizophrenia.

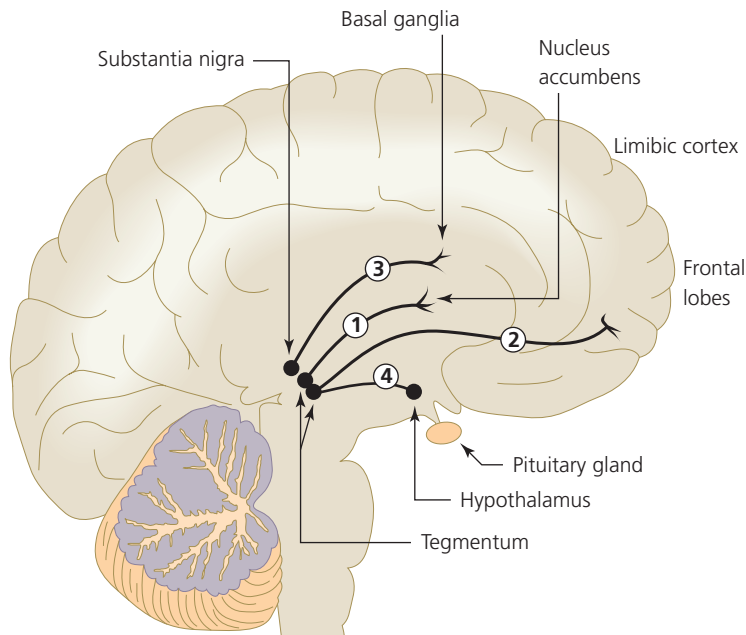
In light of this conflicting evidence, Davis and colleagues revised the dopamine hypothesis by theorising that the positive symptoms of schizophrenia resulted from dopamine overactivity (hyperdopaminergia) in the **mesolimbic system**, as previously thought, but that the negative symptoms of schizophrenia resulted from dopamine *underactivity* (hypodopaminergia) in the **mesocortical system** (Figure 4.5).

One of the strengths of this revised dopamine hypothesis is its ability to account for the effects of antipsychotic treatment. Typical antipsychotics such as chlorpromazine are unselective in their blocking effect at the dopamine D₂ receptor, and thus decrease the positive symptoms and increase the negative symptoms of schizophrenia (thus making patients 'indifferent'). Atypical antipsychotics such as clozapine, on the other hand, are less likely to increase the negative symptoms of schizophrenia or cause extrapyramidal side-effects because they are selective for the D₂ receptor subtype in the mesolimbic system or because they act primarily on serotonergic receptors.

Indeed, other neurotransmitters such as serotonin (5-HT), glutamate, noradrenaline, and gamma aminobutyric acid (GABA) also seem to play a role in the aetiology of schizophrenia.

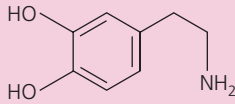
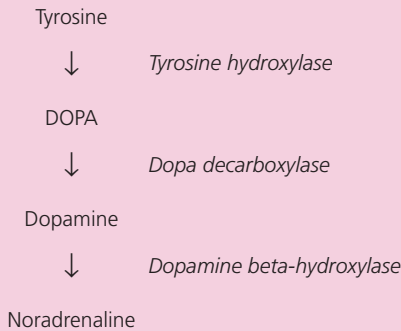
- Findings in support of a role for serotonin:
 - LSD (lysergic acid diethylamide), a 5-HT receptor agonist, can induce a schizophrenia-like psychosis
 - Clozapine, a combined dopaminergic and serotonergic antagonist, is more effective than any other antipsychotic in treatment-resistant schizophrenia.
- Findings in support of a role for glutamate:
 - NMDA antagonists such as phencyclidine hydrochloride (PCP, 'angel dust') and ketamine can induce a schizophrenia-like psychosis
 - Certain studies have reported increased levels of glutamate receptors in the brains of schizophrenia sufferers.

It is probable that altered levels of dopamine and other neurotransmitters such as serotonin and glutamate are interrelated, once more raising the age-old problem of the chicken and the egg.



- ① Mesolimbic tract – positive symptoms of schizophrenia
- ② Mesocortical tract – negative symptoms of schizophrenia
- ③ Nigrostriatal tract – extrapyramidal side-effects of antipsychotic medication
- ④ Tuberoinfundibular tract – endocrine side-effects of antipsychotic medication

Figure 4.5 Dopamine projections in the brain.

Psychopharmacology: Dopamine in the brain**Figure 4.6** Chemical structure of dopamine.**Figure 4.7** Synthesis.

Inactivation is through reuptake through the dopamine transporter and either repackaging into vesicles or enzymatic degradation by monoamine oxidase (MAO, in the pre-synaptic terminal) or catechol-O-methyltransferase (COMT, in synapses).

Receptors

There are five types of dopamine receptor, all of them G-coupled metabotropic receptors. They are divided into the D1-like family (D1 and D5) and the D2-like family (D2, D3, and D4). Activation of D1-like receptors results in increases in cAMP and is typically excitatory. In contrast, activation of D2-like receptors results in decreases in cAMP and is typically inhibitory. D1-like receptors are principally postsynaptic, whereas D2-like receptors are both pre- and post-synaptic.

Functions

Arousal, motivation, desire, pleasure, sociability (mesolimbic and mesocortical tracts), motor control (nigrostriatal tract), release of prolactin from the anterior pituitary (tuberoinfundibular tract), and vomiting (chemoreceptor trigger zone).

Other neurological abnormalities

As structural abnormalities are evident at first presentation and may also be observed in unaffected relatives, they are more likely to be the consequence of a developmental anomaly than that of chronic illness or of its treatment. Structural abnormalities include:

- Reduction in brain mass and size by about 3%, principally affecting the frontal and temporal lobes and medial temporal lobe structures such as the hippocampus, parahippocampus, and amygdala. Reduction in brain mass and size seems to result from a decrease in neuronal size rather than from a neurodegenerative process
- Ventricular enlargement of about 25%, although the distribution of ventricular volumes in schizophrenia sufferers overlaps with that of normal control subjects
- Cytoarchitectural abnormalities.

Functional abnormalities include:

- 'Hypofrontality', that is, poor performance on tests of frontal lobe function

- Soft neurological signs such as abnormalities of stereognosis and proprioception
- Abnormal eye-tracking performance
- Electroencephalograph (EEG) changes, e.g. increased theta activity, fast activity, and paroxysmal activity, and decreased alpha activity.

Developmental factors

The cytoarchitectural abnormalities and lack of gliosis in the brains of schizophrenia sufferers point to a neurodevelopmental rather than a neurodegenerative pathological process. Studies of the children of schizophrenic parents and of pre-schizophrenic children have suggested subtle manifestations of the schizophrenic genotype characterised by cognitive, motor, and social impairments, and quasi-psychotic symptoms (the so-called pre-morbid phase).

Further support for a neurodevelopmental pathological process comes from the 'season of birth effect' (see

previously). Some studies have suggested that this effect may result from prenatal exposure to the influenza virus or to other viruses. In this respect, it is interesting to note that a season of birth effect has also been reported for autism, depression, and bipolar affective disorder.

Obstetric complications, childhood head injury, and childhood encephalitis have also been suggested as aetiological factors in schizophrenia.

Life events and background stressors

Studies have found that schizophrenia sufferers experience more adverse life events in the month prior to the onset of acute symptoms of the illness. Although this finding suggests that schizophrenia is precipitated by adverse life events, the opposite might also be true. It is also important to remember that most of the stress that a person experiences on a daily basis does not come from life events, but from seemingly smaller 'background' stressors such as tense relationships, painful memories (especially memories of physical or sexual abuse), isolation, discrimination, poor housing, and unpaid bills.

Expressed emotion

Expressed emotion can be thought of as a specific type of stress. It refers to the amount of critical, hostile, or emotionally over-involved attitudes directed to the schizophrenia sufferer by his or her relatives and carers. Such attitudes often originate in a misunderstanding that the schizophrenia sufferer is actually in control of his or her illness and 'choosing' to be ill. Alternatively, over-involvement can result from an unjustified sense of guilt about the schizophrenia sufferer's illness, and a desire on the part of the relative or carer to 'share out' the burden of the illness. A number of studies have demonstrated that high expressed emotion is an important risk factor for relapse in schizophrenia, and that it can increase the risk of relapse by up to four times. As with the relationship between life events and schizophrenia, the relationship between high expressed emotion and schizophrenia is far from being a simple one: in some cases, high expressed emotion may reflect legitimate feelings of anxiety and distress at the illness of a loved one.

Cannabis and other drugs of abuse

Research has found that people who smoke cannabis are up to six times more likely to develop schizophrenia, and that people with schizophrenia who smoke cannabis have more frequent and severe relapses in the illness. Other drugs that have been associated with schizophrenia include stimulant drugs such as amphetamines, ecstasy, and cocaine.

Clinical features

F20 Schizophrenia

The schizophrenic disorders are characterized in general by fundamental and characteristic distortions of thinking and perception, and by inappropriate or blunted affect. Clear consciousness and intellectual capacity are usually maintained, although certain cognitive deficits may evolve in the course of time. The disturbance involves the most basic functions that give the normal person a feeling of individuality, uniqueness, and self-direction. The most intimate thoughts, feelings and acts are often felt to be known to or shared by others, and explanatory delusions may develop, to the effect that natural or supernatural forces are at work to influence the afflicted individual's thoughts and actions in ways that are often bizarre. The individual may see himself or herself as the pivot of all that happens. Hallucinations, especially auditory, are common and may comment on the individual's behaviour or thoughts. Perception is frequently disturbed in other ways: colours or sounds may seem unduly vivid or altered in quality, and irrelevant features of ordinary things may appear more important than the whole object or situation. Perplexity is also common early on and frequently leads to a belief that everyday situations possess a special, usually sinister, meaning intended uniquely for the individual. In the characteristic schizophrenic disturbance of thinking, peripheral and irrelevant features of a total concept, which are inhibited in normal directed mental activity, are brought to the fore and utilized in place of those that are relevant and appropriate to the situation. Thus thinking becomes vague, elliptical, and obscure, and its expression in speech sometimes incomprehensible. Breaks and interpolations in the train of thought are frequent, and thoughts may seem to be withdrawn by some outside agency. Mood is characteristically shallow, capricious, or incongruous. Ambivalence and disturbance of volition may appear as inertia, negativism, or stupor. Catatonia may be present ...

ICD-10

The onset of schizophrenia is often preceded by an insidious prodromal phase. This prodromal phase can last several years and consists of subtle and non-specific problems in language, cognitive ability, and behaviour that result in a **loss of function**. (Compare this with schizotypal disorder below.)

The symptoms of schizophrenia have traditionally been divided into positive symptoms and negative symptoms, but more recently factor analytical studies have identified a third cluster or dimension of symptoms referred to as ‘disorganised symptoms’ (Table 4.2). Positive symptoms consist of delusions and hallucinations (‘psychotic symptoms’), and are usually most prominent in the acute phase of schizophrenia. Disorganised symptoms involve various cognitive difficulties and are sometimes referred to as ‘thought disorder’. They are often detectable in the prodromal phase before the onset of positive symptoms and, although less evident than positive symptoms, they can be just as distressing and disabling. Whereas positive symptoms can be thought of as an excess or distortion of normal functions, negative symptoms can be thought of as a diminution or loss of normal functions. Compared to positive symptoms, negative symptoms tend to be more subtle and less noticeable, but also more persistent. Indeed, they can remain even through periods of remission, long after the positive symptoms have burnt out or faded into the background. During such periods of remission, the severity of any residual negative symptoms is an important determinant of the schizophrenia sufferer’s quality of life and ability to function. Unfortunately, negative symptoms are often misconstrued by the general public – and sometimes also by relatives and carers – as laziness or obstreperousness. They can also be difficult to differentiate from symptoms of depression, which are

common in schizophrenia sufferers, and from the motor side-effects of antipsychotic medication (see later).

Schizophrenia and creativity

Some highly creative people have suffered from schizophrenia, including Syd Barrett (1946–2006), the early driving force behind the rock band Pink Floyd; John Nash (born 1928), the father of ‘game theory’; and Vaclav Nijinsky (1889–1950), the legendary choreographer and dancer. The cases of Barrett, Nash, and Nijinsky are exceptional, and most people with schizophrenia are intensely disabled by the disorder. Even highly creative people with schizophrenia such as Barrett, Nash, and Nijinsky tend to be at their most creative not during active phases of the disorder, but before its onset and during later phases of remission.

Many more highly creative people, whilst not suffering from schizophrenia themselves, have close relatives who do. This was, for example, the case for the physicist Albert Einstein (his son had schizophrenia), the philosopher Bertrand Russell (also his son), and the novelist James Joyce (his daughter). This is unlikely to be simple coincidence, and a number of studies have suggested that the relatives of people with schizophrenia do indeed have above average creative intelligence. According to one theory, both people with schizophrenia and their non-schizophrenic relatives lack lateralisation of function in the brain. Whilst this tends to be a disadvantage for the former, it tends to be an advantage for the latter who gain in creativity from increased use of the right hemisphere and thus from increased communication between right and left hemispheres. This increased communication between right and left hemispheres also occurs in people with schizophrenia, but their thought and language processes tend to be too disorganised for them to make creative use of it.

Table 4.2 Symptoms of schizophrenia.

Positive symptoms	Disorganised symptoms	Negative symptoms
Hallucinations	Disorganised thinking/speech	Affective flattening
Delusions	Disorganised behaviour	Apathy
	Inappropriate affect	Avolition
		Anergy
		Anhedonia
		Alogia
		Asociality
		Attentional impairment

Diagnosis and types

ICD-10 diagnostic criteria for schizophrenia

A minimum of one very clear symptom (and usually two or more if symptoms are less clear-cut) from groups (a) to (d), or symptoms from at least two of the groups (e) to (h).

These symptoms should have been present for most of the time during a period of one month or more. If present for less than one month, a diagnosis of acute schizophrenia-like psychotic disorder should be made.



Figure 4.8 Self-portrait of a schizophrenia sufferer with thought broadcasting, which is a symptom of the first rank. Courtesy of SANE/Bryan Charnley.

- (a) Thought echo, thought insertion or withdrawal, thought broadcasting.
- (b) Delusions of control, influence, passivity; delusional perception.
- (c) Hallucinatory voices of running commentary, third-person discussion, or other types of voices coming from some part of the body.
- (d) Persistent delusions of other kinds that are culturally inappropriate and completely impossible.
- (e) Persistent hallucinations in any modality if accompanied by fleeting or half-formed delusions that are not affective delusions, or by persistent over-valued ideas, or if occurring every day for months on end.
- (f) Breaks in the train of thought resulting in incoherence, irrelevant speech, or neologisms.
- (g) Catatonic behaviour such as excitement, posturing, waxy flexibility, negativism, mutism, and stupor.
- (h) 'Negative symptoms' such as apathy, paucity of speech, blunting or incongruity of emotional responses, social withdrawal not due to depression or neuroleptic medication.
- (i) Significant and consistent change in overall quality of some aspects of personal behaviour, manifest as loss of interest, aimlessness, idleness, a self-absorbed attitude, and social withdrawal.

ICD-10 types of schizophrenia

F20.0 Paranoid schizophrenia	F20.4 Postschizophrenic depression
F20.1 Hebephrenic schizophrenia	F20.5 Residual schizophrenia
F20.2 Catatonic schizophrenia	F20.6 Simple schizophrenia
F20.3 Undifferentiated schizophrenia	F20.8 Other schizophrenia
	F20.9 Schizophrenia, unspecified

Paranoid schizophrenia

Paranoid schizophrenia is the commonest type of schizophrenia. In paranoid schizophrenia, the clinical picture is dominated by relatively stable, often paranoid, delusions, usually accompanied by hallucinations and perceptual disturbances. Disturbances of affect, volition, and speech and catatonic symptoms are *not* prominent. Onset tends to be later than for hebephrenic or catatonic schizophrenia, and the course may be either episodic or chronic.

Hebephrenic schizophrenia

Hebephrenic schizophrenia is marked by prominent affective changes. Mood is inappropriate and often accompanied by giggling or self-satisfied, self-absorbed smiling, or by a lofty manner, grimaces, mannerisms, pranks, hypochondriacal complaints, and reiterated phrases. Thought is disorganised and speech rambling and incoherent. Behaviour is characteristically aimless and empty of purpose. Compared to paranoid schizophrenia, delusions and hallucinations are fleeting and fragmentary. Hebephrenic schizophrenia is normally diagnosed for the first time only in adolescents or young adults, and has a poor prognosis due to the rapid development of negative symptoms.

Catatonic schizophrenia

Catatonic schizophrenia or catatonia is diagnosed in the presence of prominent psychomotor disturbances that may alternate between extremes such as hyperkinesia and stupor, and automatic obedience and negativism (see Chapter 2 for a full description of catatonia). Catatonia has become rare in occidental and occidentalised societies, perhaps as a result of antipsychotic treatment, or perhaps because the clinical profile of schizophrenia is culturally determined and therefore mutable.

Undifferentiated schizophrenia

Undifferentiated schizophrenia is a diagnosis reserved for conditions meeting the general diagnostic criteria for schizophrenia but not conforming to any of the above subtypes, or exhibiting the features of more than one of them without a clear predominance of a particular set of diagnostic characteristics.

Postschizophrenic depression

A diagnosis of postschizophrenic depression can only be made if the patient has had a schizophrenic illness in the past 12 months, and if some schizophrenic symptoms are still present although no longer dominating the clinical picture. The depressive symptoms must independently fulfil the diagnostic criteria for a depressive episode.

Residual schizophrenia

For a diagnosis of residual schizophrenia to be made, there must have been a clear progression from an early stage (comprising one or more episodes with psychotic symptoms that meet the general criteria for schizophrenia) to a later stage characterised by long-term, although not necessarily irreversible, negative symptoms. For a confident diagnosis of residual schizophrenia to be made, this later stage should already have lasted for at least one year and conditions such as dementia, chronic depression, or institutionalisation should have been excluded.

Simple schizophrenia

Simple schizophrenia is characterised by the insidious but progressive development of oddities of conduct, an inability to meet the demands of society, and a decline in total performance. The characteristic negative symptoms of residual schizophrenia develop without having been preceded by any overt positive or psychotic symptoms. The diagnosis is difficult to make and unreliable.

NB: At this point it is interesting to note that, whilst the biological validity of all these types is questionable, para-

noid schizophrenia tends to be dominated by positive symptoms, hebephrenic schizophrenia by disorganised symptoms, and simple schizophrenia by negative symptoms (see Table 4.2).

DSM-IV diagnostic criteria for schizophrenia

A. **Characteristic symptoms:** two or more of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):

- Delusions
- Hallucinations
- Disorganised speech
- Grossly disorganised or catatonic behaviour
- Negative symptoms, i.e. affective flattening, alogia, or avolition.

NB: Only one Criterion A symptom is required if delusions are bizarre or hallucinations consist of a voice keeping up a running commentary on the person's behaviour or thoughts, or if there are two or more voices conversing.

B. **Social/occupational dysfunction:** for a significant portion of time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, or self-care are markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, failure to achieve expected level of interpersonal, academic, or occupational achievement).

C. **Duration:** continuous signs of the disturbance persist for at least six months. This six-month period must include at least one month of symptoms (or less if successfully treated) that meet Criterion A (i.e. active-phase symptoms) and may include periods of prodromal or residual symptoms. During these periods of prodromal or residual symptoms, the signs of the disturbance may be manifested by only negative symptoms or by two or more symptoms listed in Criterion A present in an attenuated form, e.g. odd beliefs, unusual perceptual experiences. (NB: ICD-10 differs from DSM-IV principally in that it does not specify a six-month period of continuous signs of the disturbance, and in that it does not require criterion B on social/occupational dysfunction.)

- D. Schizoaffective and mood disorder exclusion:** *schizoaffective disorder and mood disorder with psychotic features have been ruled out because either (1) no major depressive episode, manic episode, or mixed episode have occurred concurrently with the active-phase symptoms; or (2) if mood episodes have occurred during active-phase symptoms, their total duration has been brief relative to the duration of the active and residual periods.*
- E. Substance/general medical condition exclusion:** *the disturbance is not due to the direct physiological effects of a substance or a general medical condition.*
- F. Relationship to a pervasive developmental disorder:** *if there is a history of autistic disorder or another pervasive developmental disorder, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations are also present for at least one month (or less if successfully treated).*

DSM-IV types of schizophrenia

295.10	Disorganised type	Disorganised speech or behaviour; flat or inappropriate affect
295.20	Catatonic type	Catalepsy, excessive motor behaviour, rigid posture, mutism, posturing, grimacing, echolalia, or echopraxia
295.30	Paranoid type	Preoccupation with delusions or auditory hallucinations
295.60	Residual type	Absence of prominent positive symptoms but continuing evidence of disturbance
295.90	Undifferentiated type	Non-specific; used when criteria are not met for paranoid, disorganised, or catatonic types

The difficulty with diagnosing schizophrenia

The majority of medical conditions are defined by their cause ('aetiology') or by the damage to the body that they result from ('pathology'), and so are relatively easy to diagnose. For example, if a person is suspected of having malaria, a blood sample can be taken and examined under a microscope for malarial parasites of the genus *Plasmodium*. If a person is suspected of having had a cerebral infarction ('stroke'), a brain scan can be taken to look for evidence of obstruction of an artery in the brain. In contrast, mental disorders are concepts that so far can only be defined by their (supposed) predominant symptoms. For this reason, they are more difficult to describe and diagnose, and more open to misunderstanding and misuse. If a person is suspected of having schizophrenia, there are no laboratory or physical tests that can objectively confirm the diagnosis. Instead the psychiatrist must base his or her diagnosis solely on the symptoms manifested by the patient, without the help of any tests. If the symptoms tally with the diagnostic criteria for schizophrenia listed above, then the psychiatrist is able to make a diagnosis of schizophrenia.

The problem here is that the definition of schizophrenia is circular: the concept of schizophrenia is defined according to

the symptoms of schizophrenia, and the symptoms of schizophrenia are defined according to the concept of schizophrenia. Thus, it is impossible to be certain that either schizophrenia or its symptoms map onto any real or distinct disease entity. Given the 'menu of symptoms' approach to diagnosing schizophrenia, it is even possible to have two people with completely different symptoms, but one diagnosis of 'schizophrenia'. Perhaps for this reason, a diagnosis of schizophrenia is a poor predictor of either the severity of the disorder or its likely outcome or prognosis. It has also been argued that psychotic symptoms such as delusions or hallucinations of voices are a poor basis for making a diagnosis of schizophrenia, since psychotic symptoms occur in a number of mental disorders and are therefore a relatively non-specific indicator of mental disorder. Furthermore, most of the disability in schizophrenia is caused by cognitive and negative symptoms rather than by positive symptoms. For these reasons, making a diagnosis of schizophrenia based on psychotic symptoms may be akin to making a diagnosis of pneumonia or appendicitis on the basis of little more than a fever.

Clinical skills/OSCE: Enquiring about delusions

Begin with an introductory statement and general questions, such as,

I would like to ask you some questions that might seem a little bit strange. These are questions that we ask to everyone who comes to see us. Is that all right with you? Do you have any ideas or opinions that your friends and family do not share?

Then ask specifically about common delusions (make sure you tailor your questions to the individual patient you are speaking to, e.g. you don't need to ask a manic patient about nihilistic delusions):

- Delusions of control and passivity experiences:
Is someone or something controlling you?
Is someone forcing you to think/say/do certain things?
- Delusions of thought control:
Are you able to think clearly?
Are your thoughts being interfered with?
Are thoughts which are not your own being put into your head? (thought insertion)
Are your own thoughts being removed from your head? (thought withdrawal)
Are your thoughts being heard or otherwise accessed by other people? (thought broadcasting)
- Delusional perception:
Do things happening around you have a special meaning to you?
- Delusions of persecution:
How are you getting on with other people?
Is anyone deliberately trying to harm you or to make your life miserable?
- Delusions of reference:
Do people talk about you behind your back?
Do people drop hints about you/say things that have a special meaning for you?
- Delusions of misidentification:
Do you feel that people are not who they seem to be? For example, do you feel that they been replaced by imposters (Capgras delusion) or disguised to look like other people? (Fregoli delusion)
- Delusions of grandeur:
How do you see yourself relative to other people?
Do you feel you have a special mission?
Do you feel that you have any special abilities or powers?
- Religious delusions:
Are you a very religious person?
Are you especially close to God?
- Delusions of guilt:
Do you have any regrets?
Do you feel as though you have committed a crime/sinned greatly/deserve punishment?
- Nihilistic delusions:
Do you feel that something terrible has happened or is about to happen?
Do you feel that a part of your body has stopped functioning/been removed?
Do you feel as though you have died?
- Somatic delusions:
Are you concerned that you might have a serious illness?
- Delusions of jealousy
How are you getting on with your partner? Does he or she reciprocate your loyalty?

Explore any delusions and ask in particular about their onset, their effect on the patient's life, and the patient's explanation for them (degree of insight).

Clinical skills/OSCE: Enquiring about auditory hallucinations (voices)

Begin with an introductory statement and general questions, such as,

I gather that you have been under quite some pressure recently. When people are under pressure they sometimes find that their imagination plays tricks on them. Have you had any such experiences? Have you heard things which are unusual? Have you heard things which other people cannot hear?

Then ask more closed questions to determine:

- Content: whose voices are they, where are they coming from, and what are they saying? In particular, are they commanding the patient to do anything dangerous (command hallucination)?
- Type: do the voices speak directly to the patient (second person), speak about him (third person), comment on his every thought and action (running commentary), or repeat his thoughts (thought echo)? Differentiate between true hallucinations and pseudohallucinations. Exclude hypnagogic and hypnopompic hallucinations (see Table 2.7)
- Frequency and duration
- Onset and precipitating factors
- Effect on the patient's life
- The patient's explanation for them (degree of insight and, especially important, likelihood to act on any command hallucinations).

Differential diagnosis**Psychiatric disorders**

- Drug-induced psychotic disorder (e.g. amphetamines, cocaine, cannabis, alcohol, LSD, phencyclidine, glucocorticoids, and L-dopa). This differential diagnosis is an important one, as drug-induced psychotic disorders are very common
- Schizoaffective disorder (see p. 73)
- Psychotic depression (see Chapter 5)
- Manic psychosis (see Chapter 5)
- Other psychotic disorder such as schizotypal disorder, brief psychotic disorder, persistent delusional disorder, or induced delusional disorder (see later)
- Puerperal psychosis (see p. 96)
- Personality disorder

Organic disorders

- Delirium
- Dementia
- Stroke
- Temporal lobe epilepsy
- Central nervous system infections such as AIDS, neurosyphilis, herpes encephalitis
- Other neurological conditions such as head trauma, brain tumour, Huntington's disease, Wilson's disease
- Endocrine disorders, in particular Cushing's syndrome

- Metabolic disorders, in particular vitamin B12 deficiency and porphyria
- Autoimmune disorders, in particular systemic lupus erythematosus (SLE)

! Chronic or residual schizophrenia must be differentiated from the symptoms of depression and from the motor side-effects of antipsychotic medication (see later). Depressive symptoms are common in schizophrenia, and about a quarter of patients become depressed once their psychotic symptoms have resolved.

Investigations

Investigations for a first episode of psychosis should include full physical (including neurological) examination, a serum and/or urine drug screen, liver, renal and thyroid function tests, full blood count, fasting blood glucose (or HbA1c), and lipids. The aims of these investigations are principally to uncover possible organic causes of psychosis and to establish baselines for the administration of antipsychotic medication. Other, more specific investigations should be considered on a case-by-case basis, and might for example include brain imaging if there is a suggestion of a space-occupying lesion.

Management

Management is discussed under three principal headings:

- Antipsychotic drugs
- Other drugs and electroconvulsive therapy
- Psychosocial treatments.

Antipsychotic drugs

The following are guidelines to treatment.

- Patients are more likely to respond if treatment is started early, so it is usually best to start treatment soon after the diagnosis is established. Antipsychotic drugs are effective against positive symptoms in about 70–85% of patients, but it can be several days before they take effect and a benzodiazepine such as lorazepam may need to be prescribed in the interim if the patient is agitated or difficult to manage.
- Current treatment guidelines recommend using one of the atypical antipsychotics other than clozapine as a first line of treatment. Choice should be guided primarily by side-effect profile and patient choice (see Tables 4.4 and 4.5).
- If the patient has previously been on treatment, the choice of drug should also be guided by the patient's response to treatment and susceptibility to side-effects.
- The starting dose should be small to minimise side-effects and then increased according to clinical response to the **minimum effective dose**.
- If the patient fails to respond to the chosen antipsychotic or cannot tolerate its side-effects, an alternative antipsychotic from a different class should be tried.
- If the patient fails to respond to two or more antipsychotics after an adequate trial of each (6–8 weeks), clozapine should be considered. Clozapine is effective in about 50% of treatment-resistant patients – ‘treatment-resistance’ in this case being defined as failure to respond to an adequate trial of at least two antipsychotics.
- If a patient has improved on a particular drug, he or she should continue taking the same drug at the same dose for *at least* the next six months, **and preferably for the next 12–24 months**. Long-term antipsychotic treatment has been demonstrated to reduce rates of relapse and rehospitalisation in a substantial number of patients. Patients with chronic schizophrenia may remain on antipsychotic treatment for many years even though antipsychotics are not effective in the treatment of persistent negative symptoms.
- Depot preparations can be used to improve long-term compliance, but only one atypical antipsychotic (ris-

peridone) is available in depot form. The principal advantages and disadvantages of oral versus depot preparations are listed in Table 4.3. Upon converting a patient to a depot antipsychotic, it is usual to first administer a small test dose. The first treatment dose can be administered after about seven days if the patient does not suffer from any unacceptable adverse reactions during this time. The treatment dose can then be increased at regular intervals, as the oral antipsychotic is tapered off and stopped.

Typical antipsychotics (previously referred to as neuroleptics or major tranquillizers) used to be the first line of treatment for schizophrenia. They include chlorpromazine, fluphenazine, flupenthixol, zuclopenthixol, and haloperidol. The clinical antipsychotic efficacy of typical antipsychotics is related to their antagonism of the dopamine D₂ receptor. Their common side-effects are listed in Table 4.4. Extrapyramidal side-effects (EPSEs) are particularly common and can occur in up to 70% of patients. They include acute dystonias, akathisia, Parkinson-like symptoms, and tardive dyskinesia (see Table 4.5). Typical antipsychotics are effective once they reach a certain threshold of D₂ receptor occupancy, thought to be around 60%. Beyond a threshold occupancy of 80% there is little additional clinical efficacy and a significantly increased risk of EPSEs.

Table 4.3 Principal advantages and disadvantages of oral versus depot preparations.

	Advantages	Disadvantages
Oral medication	Short duration of action Flexibility	Variable absorption/ first-pass effect Potential for poor compliance Potential for misuse and overdose
Depot medication	Improved bioavailability Less potential for poor compliance Less potential for abuse and overdose Regular contact with CPN	Potential damage to therapeutic alliance Needle injections: pain, potential local complications, e.g. abscess formation Potential delayed side-effects Potential prolonged side-effects

CPN, Community Psychiatric Nurse.

Table 4.4 Side-effects of antipsychotic drugs according to receptor action.

Receptor action	Potential therapeutic effect	Potential side-effects
Antidopaminergic	Improvement in positive symptoms	Extrapyramidal symptoms (see Table 4.5), Hyperprolactinaemia* Neuroleptic malignant syndrome Weight gain
Serotonergic	Improvement in affective symptoms Improvement in negative symptoms	Anxiety Insomnia Change in appetite leading to weight gain Hypercholesterolaemia Diabetes [§]
Antihistaminergic	Unknown	Sedation (can be a benefit) Weight gain
Antiadrenergic	Unknown	Postural hypotension Tachycardia Ejaculatory failure
Anticholinergic	Unknown	Dry mouth Blurred vision Constipation Urinary retention

* Symptoms of hyperprolactinaemia include loss of libido, amenorrhoea, erectile dysfunction, galactorrhoea, gynaecomastia, and reduced bone density.

[§]The prevalence of type II diabetes is increased in schizophrenia sufferers, their relatives, and those on antipsychotic medication (most evidence for clozapine and olanzapine). This may be a result of weight gain or insulin resistance.

NB: Other side-effects of antipsychotics may include neuroleptic malignant syndrome (NMS – see box, p. 69), hypo- or hyperthermia, convulsions, cardiotoxic side-effects (increased QTc, myocarditis, cardiomyopathy), hepatotoxicity, blood dyscrasias, photosensitivity, and allergic reactions.

! Although drugs may be used to treat EPSEs, in the first instance it is often preferable to reduce the dose of the antipsychotic or change the antipsychotic to another (usually atypical) antipsychotic. The prophylactic use of anticholinergics to prevent certain EPSEs is a common practice which is best avoided.

Table 4.5 Extrapyramidal side-effects of antipsychotics.

1. Acute dystonias

Often painful spastic contraction of certain muscles or muscle groups most commonly affecting the neck, eyes, and trunk; for example, tongue protrusion, grimacing, torticollis. Acute dystonias may respond to anticholinergics

2. Akathisia (Greek, *not to sit*)

Distressing feeling of inner restlessness manifested by fidgety leg movements, shuffling of feet, pacing, and so on. Akathisia may respond to anticholinergics, propranolol, the antihistamine cyproheptadine, benzodiazepines, or clonidine

3. Parkinson-like symptoms

Triad of parkinsonian tremor, muscular rigidity, and bradykinesia. Parkinson-like symptoms may respond to anticholinergics

4. Tardive dyskinesia (TD)

Involuntary, repetitive, purposeless movements of the tongue, lips, face, trunk, and extremities that may be generalised or affect only certain muscle groups, typically orofacial muscle groups ('rabbit syndrome'). TD occurs after several months or years of antipsychotic treatment and is often irreversible. Risk factors for TD in patients receiving antipsychotic treatment are length and dose of antipsychotic treatment, increased age, female sex, prominent negative symptoms, head injury/brain damage, and organic brain disease. There is no consistently beneficial treatment and the condition may be *exacerbated* by anticholinergics. Although TD is typically thought of as an antipsychotic-related EPSE, it may also occur in untreated schizophrenia and in healthy elderly people

EPSE, extrapyramidal side-effects.

Strictly speaking, the definition of an atypical antipsychotic is a drug which does not produce catalepsy in rats despite having an antipsychotic profile in behavioural tests, that is, a drug which, unlike typical antipsychotics, has a high therapeutic index in relation to EPSEs. The reason postulated for this is that in contrast to typical antipsychotics, atypicals undergo 'fast dissociation' at the dopamine D₂ receptor. The group of atypical antipsychotics is considered to include clozapine, risperidone,

olanzapine, quetiapine, amisulpride, sertindole, ziprasidone, zotepine, and paliperidone. Atypicals are at least as effective as typical antipsychotics and treat positive symptoms, and, it has been argued, also affective symptoms and negative symptoms. As aforementioned, current treatment guidelines recommend using one of the atypical antipsychotics other than clozapine as a first line of treatment for schizophrenia. Clozapine causes agranulocytosis in about 1% of patients and for this reason patients on the drug must have their differential leucocyte counts monitored. Despite this inconvenience, clozapine is the drug of choice in treatment-resistant schizophrenia, in schizophrenia accompanied by marked suicidality, and in the management of disabling tardive dyskinesia.

Aripiprazole is a novel, third-generation antipsychotic that has been described as a **dopamine-serotonin system stabiliser**. The drug has partial agonist activity at D₂ and 5HT_{1A} receptors and antagonist activity at 5HT_{2A} receptors. It is purported to have good efficacy in treating positive symptoms, negative symptoms, and affective

symptoms, and to be better tolerated than other antipsychotics. Principal side-effects include headache, anxiety, insomnia, nausea, vomiting, and light-headedness, but *not* EPSEs, weight gain, or hyperprolactinaemia.

4

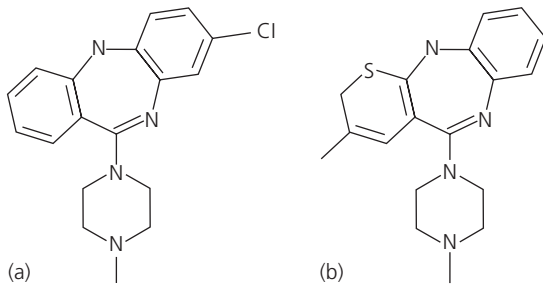


Figure 4.9 Chemical structures of the atypical antipsychotics clozapine (left; a dibenzodiazepine) and olanzapine (right; a thienobenzodiazepine). Compare to the structure of chlorpromazine (a phenothiazine) (Figure 4.2).



Figure 4.10 'Medication' by Philippa King. Philippa King explains, 'The side-effects I was experiencing on antipsychotic medication were tremors in my arms and hands (illustrated by the wavy line of the sleeve), a dry mouth (another reason for including a glass of water in the picture), and weight gain'.

Table 4.6 Comparison of the side-effect profiles of four commonly prescribed atypical antipsychotics (note that clozapine in particular is also associated with sialorrhoea (hypersalivation), tachycardia, myocarditis, cardiomyopathy, insulin resistance, increased risk of convulsions at higher doses, and agranulocytosis).

Atypical antipsychotic	EPSE	Hyperprolactinaemia	Sedation	Weight gain	Orthostatic hypotension	Anticholinergic side-effects
Risperidone	+	++	+	+	++	0/+
Olanzapine	0/+	+	++	+++	+	+ /+++
Quetiapine	0/+	0/+	++	++	++	0/+
Clozapine	0	0	+++	+++	+++	+++

EPSE, extrapyramidal side-effects.

! Neuroleptic malignant syndrome

Neuroleptic malignant syndrome (NMS) is a rare but underdiagnosed and potentially fatal idiosyncratic reaction to antipsychotic medication. NMS results from blockade of dopaminergic hypothalamospinal tracts that normally tonically inhibit preganglionic sympathetic neurons. It is characterised by a square of **hyperthermia, muscle rigidity, autonomic instability, and altered mental status**. Rhabdomyolysis, as reflected by a high creatinine phosphokinase (CPK) blood level, may lead to renal failure. Other complications include respiratory failure, cardiovascular collapse, seizures, arrhythmias, and disseminated intravascular coagulopathy (DIC). The mainstay of treatment involves stopping the drug and supportive measures such as oxygen, IV fluids, and cooling blankets, although drugs such as dantrolene and lorazepam may also be used to decrease muscle rigidity. **If left untreated, mortality is as high as 20–30%**. Differential diagnosis includes infection, catatonia, parkinsonism, and malignant hyperthermia. Note that atypical antipsychotics, antiparkinsonian drugs, antidepressants, and drugs of abuse such as cocaine or ecstasy can also cause NMS.

Other drugs and electroconvulsive therapy

There may a number of reasons why a patient has not responded to an 'adequate trial' of antipsychotic medication, including ongoing stressors, non-compliance, substance misuse, or an overlooked organic aetiology. If such factors have been excluded or addressed, a **benzodiazepine, lithium, or carbamazepine** may be added to the antipsychotic medication. These so-called adjunctive or augmentative treatments are *not* as effective as **clozapine** and should therefore only be used after an adequate trial of clozapine. Clozapine itself is sometimes augmented with **sulpiride** or **risperidone**, but never with carbamazepine which is also linked to agranulocytosis. Non-pharmacological strategies for distressing chronic hallucinations include an IP3 player, subvocal counting or singing, and a pair of earplugs. These strategies should be considered in all treatment-resistant cases, as they are cheap, simple, and empowering, and lacking in side-effects.

Benzodiazepines can also be used in the treatment of ancillary symptom complexes such as anxiety and agitation, and in the emergency treatment of acute psychosis ('rapid tranquillisation'). A typical regimen for rapid

tranquillisation is lorazepam 1 mg as required, up to 4 mg per 24 hours, delivered either orally or intramuscularly. The atypical antipsychotic haloperidol is also sometimes used for rapid tranquillisation, often in combination with lorazepam. However, this practice is best avoided, as it exposes the patient to a broader range of potential side-effects than lorazepam alone.

Antidepressants and electroconvulsive therapy can be used to treat depressive symptoms.

Treatment trials of EPA, an n3 fatty acid contained in fish oil, have so far proven inconclusive.

Clinical skills: Coffee and a cigarette

The vast majority of schizophrenia-sufferers smoke, and typically smoke more heavily than smokers in the general population. This could be because nicotine functions as a neuroprotective agent, or because it stimulates dopamine release in the prefrontal cortex and so alleviates symptoms and improves cognitive performance. Aside from the long-term effects of smoking, nicotine induces the hepatic microsomal enzyme CYP1A2. As clozapine and olanzapine are metabolised by CYP1A2, the levels of these antipsychotics are reduced in smokers. Caffeine is also metabolised by CYP1A2, for which reason smokers tend to drink more coffee than non-smokers. However, caffeine competes with clozapine and olanzapine for CYP1A2, and thereby *increases* the levels of these antipsychotics. *Work it out: you probably need an aspirin by now.*

Psychosocial treatments

The management of a patient is usually planned at one or several Care Programme Approach (CPA) meetings (see Chapter 3). These meetings are useful to establish the context of the patient's disorder, evaluate his or her current personal circumstances, assess his or her needs, and formulate a detailed care plan to ensure that medical, psychological, and social needs are met. Apart from ensuring that the patient takes his or her medication and that he or she is regularly seen by a member of the mental health-care team, the care plan should involve a number of psychosocial measures, possibly including supportive therapy, patient self-help groups, family education/therapy, cognitive-behavioural therapy (CBT), and rehabilitation (social skills training and sheltered employment programmes). **Although under-utilised, psychosocial**

Table 4.7 Summary of commonly used atypical, typical, and depot antipsychotics.

Antipsychotic	Trade name	Licensed daily dose range in adults under the age of 65
Atypical antipsychotics		
Risperidone	Risperdal	2–16 mg (rarely exceed 10 mg)
Olanzapine	Zyprexa	5–20 mg
Quetiapine	Seroquel	50–750 mg in two doses, up to 800 mg in mania (usual dose range 300–450 mg)
Amisulpride	Solian	400–1200 mg in two doses
Clozapine	Clozaril/Denzapine	25–900 mg (usual dose range 200–450 mg)
Aripiprazole	Abilify	10–30 mg
Typical antipsychotics		
Phenothiazines		
Chlorpromazine	Largactil	75–1000 mg
Fluphenazine	Modecate/Moditen	2–20 mg
Butyrophenones		
Haloperidol	Haldol/Dozic/Serenace	3–30 mg (18 mg if administered IM/IV)
Diphenylbutylpiperidines		
Pimozide	Orap	2–20 mg (ECG monitoring required)
Thioxanthenes		
Flupenthixol	Depixol	3–18 mg
Zuclopenthixol	Clopixol	20–150 mg
Substituted benzamides		
Sulpiride	Dolmatil/Sulpitil/Sulpor	400–2400 mg
Depot antipsychotics		
Risperidone	Risperdal Consta	Max 50 mg fortnightly
Fluphenazine decanoate	Modecate	Test dose 12.5 mg Max 100 mg fortnightly
Flupenthixol decanoate	Depixol	Test dose 20 mg Max 400 mg/week
Zuclopenthixol decanoate	Clopixol	Test dose 100 mg Max 600 mg/week
Pipotazine palmitate	Piportil depot	Test dose 25 mg Max 200 mg/4 weeks

NB: Prior to starting an antipsychotic, it is good practice to obtain at the very least: urea and electrolytes, liver function tests, fasting plasma glucose, blood lipids, and prolactin, as well as recordings of blood pressure and weight. Clozapine additionally requires registration with a monitoring service, baseline full blood count and ECG, and weekly full blood counts for the first 18 weeks. It also requires close monitoring of pulse rate, blood pressure, and temperature during the titration period.

measures are often cost-effective and their importance in the treatment of schizophrenia should not be underestimated.

Supportive therapy should be offered to the patient and his or her relatives by one or several members of the Community Mental Health Team. The patient and his or her relatives can additionally be referred to charitable organi-

sations which offer information and support, and which organise patient self-help groups. These enable patients to meet other schizophrenia sufferers, share their experiences, and thereby normalise and destigmatise them. Family education/therapy is useful as relatives invariably need explanation and support, and need to be involved in the patient's management plan. This can help to reduce

high expressed emotion and improve compliance with medication, amongst others. CBT can be useful if the patient continues to suffer from residual symptoms such as drug-resistant delusions, or from negative or depressive symptoms. CBT for drug-resistant delusions typically involves exploring the subjective nature of the delusions, challenging the evidence for them, and subjecting them to reality testing. CBT can also be useful to improve a patient's insight, and so his or her compliance with medication.

Some patients, especially those suffering from prominent negative symptoms, may need a period of rehabilitation. Areas that are considered during rehabilitation are accommodation, activities of daily living, occupational activities, leisure activities, and social skills. Several members of the multidisciplinary team, such as the Community Psychiatric Nurse, Occupational Therapist, and Clinical Psychologist, may be co-opted into the patient's care. Sheltered employment programmes use the place-and-train vocational model and significantly increase the patient's likelihood of re-entering competitive employment. Social skills training involves dividing complex social activities, such as making conversation and participating in recreational activities, into simpler steps that can then be learned and practised through role play. Despite a period of rehabilitation, some patients may remain unable to live independently, and may therefore require long-term supported accommodation. Such supported accommodation is often found in a sheltered home or group home – a house shared by several schizophrenia sufferers and supported by a group homes organisation.

Hospitalisation

Schizophrenia is increasingly managed in the community, but hospitalisation may sometimes be required. The possible functions of hospitalisation are listed in Table 4.8. In many cases, attendance at a day hospital or management by the crisis team (in the short-term) or assertive outreach team (in the longer-term) can prevent a hospital admission or decrease time spent in hospital.

Course and prognosis

The course of schizophrenia can vary considerably from one person to another, but it is often marked by a number of distinct phases. In the acute phase, positive symptoms come to the fore, while any cognitive and negative symptoms that may already be present appear to sink into the background. The patient typically reaches a crisis point, at

Table 4.8 Possible functions of hospitalisation.

-
- Establishment of a diagnosis
 - Stabilisation of medication
 - Management of acute exacerbations, e.g. severe psychotic symptoms, non-cooperation, lack of insight
 - Management of comorbid conditions
 - Safety of the patient and of the community
 - Carer respite
 - Substitute to community care if patient is lacking a social structure
-

which time contact with mental health services is made. Antipsychotic medication is started and the acute phase resolves, even though residual positive symptoms may still remain in the background for some time. As the acute phase resolves, the cognitive and negative symptoms may appear to return to the fore and dominate the clinical picture. This chronic phase, if it occurs, may last for a period of months or, in some cases, several years. In some cases it may be interrupted by relapses to the acute phase, particularly if the patient is not taking any antipsychotic medication. Common causes of relapse to the acute phase include reduction or discontinuation of antipsychotic medication, non-compliance with antipsychotic medication, substance misuse, high expressed emotion, life events, and child birth.

The prognosis of schizophrenia can be summarised by the 'rule of thirds' according to which, after an acute psychotic episode:

- About one-third of patients recover and lead normal or almost normal lives
- About one-third of patients improve but continue to experience significant symptoms
- About one-third of patients do not improve significantly and require frequent hospitalisation.

The suicide rate in schizophrenia is estimated to be 5%, but the rate of attempted suicide is significantly higher. Risk factors for suicide include being young, being male, being early in the course of the illness, having good insight into the illness, coming from a high socioeconomic family background, having high intelligence, having high expectations, being unmarried, lacking social support, and being recently discharged from hospital (Table 4.9). Other important causes of death in schizophrenia include accidents and cardiovascular disease. Overall, life expectancy is decreased by about 8 years.

Interestingly, the prognosis of schizophrenia is better in traditional societies than in industrialised ones. This may

be because people in traditional societies tend to be more tolerant of mental disorder and better able to pull together and support mentally disordered members of their community.

Table 4.9 Prognostic factors in schizophrenia.

Good prognosis	Bad prognosis
Acute onset	Insidious onset
Late onset	Early onset
Precipitating factors	No precipitating factors
Florid symptoms or associated mood disorder	Negative symptoms
Female sex	Male sex
No family history	Family history
No substance misuse	Substance misuse
Good premorbid occupational/social adjustment	Poor premorbid occupational/social adjustment
Good social support/adequate social stimulation	Poor social support/poor social stimulation
Married	Unmarried (including separated, etc)
Early treatment and compliance	Delayed treatment and non-compliance
Good response to treatment	Poor response to treatment

Other psychotic disorders

Psychosis is basically a generic term for a mental state involving a loss of contact with reality and manifested by delusions or hallucinations. Although psychosis can be a non-specific marker of a serious underlying disorder, it can also represent one end of a continuum of normal consciousness. Hallucinatory experiences in particular are very common; in a survey (M. M. Ohayon, Prevalence of hallucinations and their pathological associations in the general population. *Psychiatry Research* (2000), 97(2–3), 153–164) of representative samples of the general population in the UK, Germany, and Italy, as many as 38.7% of respondents reported having had hallucinatory experiences of some sort or other.

Thus, psychosis can be associated not only with schizophrenia and mood disorders such as depression and bipolar disorder, but also with other mental disorders such as ‘brief psychotic disorder’; with medical and neurological disorders such as temporal lobe epilepsy, brain tumour, stroke, and dementia; with drugs such as amphetamines, cocaine, cannabis, and LSD; or simply with intense, disturbing, or stressful experiences. This section describes some of the other mental disorders that are associated with psychosis.

ICD-10 and DSM-IV classification of schizophrenia and other psychotic disorders

ICD-10

F20 Schizophrenia
 Paranoid
 Hebephrenic
 Catatonic
 Undifferentiated
 Residual
 Simple
 Post-schizophrenic depression
 Other schizophrenia
 Unspecified schizophrenia

F21 Schizotypal disorder

F22 Persistent delusional disorder

F23 Acute and transient psychotic disorders
 Acute polymorphic psychotic disorder
 Acute schizophrenia-like psychotic disorder
 Other acute psychotic disorder

F24 Induced delusional disorder

F25 Schizoaffective disorder

F28 Other non-organic psychotic disorders

F29 Unspecified non-organic psychosis

DSM-IV

Schizophrenia
 Paranoid
 Disorganised
 Catatonic
 Undifferentiated
 Residual

Delusional disorder

Brief psychotic disorder
 Schizophreniform disorder

Shared psychotic disorder
 Schizoaffective disorder

Psychotic disorder not otherwise specified

Schizotypal disorder

Schizotypal disorder, also called latent schizophrenia, is a personality disorder characterised by eccentric behaviour and anomalies of thinking and affect similar to those seen in schizophrenia. First-degree relatives of schizophrenia sufferers are at an increased risk of schizotypal disorder. In DSM-IV schizotypal disorder is classified under personality disorders (see Chapter 8).

Persistent delusional disorder

An uncommon condition characterised by the development of a single delusion or set of related delusions, often persecutory, hypochondriacal, or grandiose in content. The delusions are of a fixed, elaborate, and systematised kind, and can often be related to the patient's life situation. Other psychopathology is characteristically absent, although intermittent depressive symptoms may be present in some cases. There may be occasional or transitory auditory hallucinations, especially in elderly patients, but these are unlike schizophrenic auditory hallucinations and form only a small part of the overall clinical picture. Eponymous examples include de Clérambault's syndrome and Othello syndrome (see Chapter 2). The condition sometimes responds to antipsychotic medication.

Brief psychotic disorder (DSM-IV)

Brief psychotic disorder resembles an acute episode of schizophrenia and is characterised by prominent positive symptoms such as delusions and hallucinations, a rapid onset, a short course of less than one month (by definition), and a complete recovery. In France, psychiatrists refer to such an episode as *bouffée délirante aiguë* and are apt to describe it as '*un coup de tonnerre dans un ciel serein*' – a thunder clap in a clear sky. Onset is rapid and typically preceded by acute stress. Substance misuse, mood disorders, and organic disorders should be excluded.

Schizophreniform disorder (DSM-IV)

Schizophreniform disorder is characterised by comparatively stable psychotic symptoms that fulfil the diagnostic criteria for schizophrenia and last for more than one month but for less than six months. Mood disorders, organic disorders, and substance misuse should be excluded. Onset is brief and symptoms are present at least most of the time.

Induced delusional disorder

Induced delusional disorder (*folie à deux*, *folie à trois*, or even *folie à plusieurs* and *folie à famille*), is a rare delusional disorder shared by two or more people in a close and dependent relationship (cf mass hysteria). The delusions are usually chronic and either persecutory or grandiose in content. There are several subtypes of *folie à deux*. In *folie imposée*, only A suffers from a primary psychotic disorder such that B's delusions disappear if he or she is separated from A. *Folie communiquée* is similar to *folie imposée*, except that B maintains his or her delusions even if he or she is separated from A. In *folie simultanée*, both A and B suffer from a primary psychotic disorder but happen to share the same delusions. In *folie induite*, both A and B suffer from a primary psychotic disorder, and transfer their delusions to each other. Note that induced delusional disorder is referred to as shared psychotic disorder in DSM-IV.

Schizoaffective disorder

Schizoaffective disorder is characterised by prominent affective and schizophrenic symptoms in the same episode of illness. Its relationship to affective disorders and schizophrenic disorders is still unclear, and care must be taken to differentiate it from post-schizophrenic depression, bipolar disorder, and recurrent depressive disorder. It appears that the prognosis for schizoaffective disorders is better than that for schizophrenia but not as good as that for mood disorders. Mood disorders are the subject of Chapter 5.

Late paraphrenia

'Late paraphrenia' is a term that is sometimes used to refer to late-onset schizophrenia, which is either an expression of schizophrenia in the elderly or an entity that is genetically distinct from schizophrenia. The term is not coded in ICD-10 and DSM-IV. Prominent hallucinations and delusions, particularly paranoid delusions, are typical, whereas disorganised, negative, and catatonic symptoms are extremely uncommon. Risk factors include brain disease, family history, female sex, social isolation, visual impairment, and hearing loss. Late paraphrenia responds to antipsychotics, and, whilst prognosis is variable, life expectancy is unchanged.

Schizophrenia and the abuse of psychiatry

Whilst a lack of scientific validity and reliability is a problem for all psychiatric disorders that are defined and diagnosed according to their clinical manifestations and symptoms, it is a particular problem for schizophrenia, which has a history of being abused for political motives. Beginning in the early 1970s, reports began appearing that political and religious dissidents in the Soviet Union were being incarcerated in maximum-security psychiatric hospitals. In 1989 the Soviet government authorised a delegation of psychiatrists from the United States to make site visits to selected hospitals and to conduct extensive interviews of 27 suspected victims of abuse, of whom 24 had at one time or another received a diagnosis of schizophrenia. This investigation provided unequivocal proof that psychiatry had been abused to imprison people who were not mentally disordered and whose only transgression had been the expression of political or religious dissent. In as many as 14 of the 27 cases, there was no evidence of mental disorder of any kind, let alone mental disorder of a nature and degree requiring hospital treatment. The living conditions in the psychiatric hospitals were found to be primitive and highly restrictive, with 'patients' unable even to keep books or writing materials. Physical restraints were used and high doses of antipsychotic and other drugs were administered by injection. In a paper that dates

back to 2002 (R. Bonnie, Political abuse of psychiatry in the Soviet Union and China: complexities and controversies. *Journal of American Academic Psychiatry and the Law* (2002), 30, 136–44), Richard Bonnie writes,

In some cases, abuse was undoubtedly attributable to intentional misdiagnosis and to knowing complicity by individual psychiatrists in an officially directed effort to repress dissident behaviour. In other cases, the elastic conception of mental disorder used in Soviet psychiatry was probably bent to political purposes, with individual psychiatrists closing their eyes to whatever doubts they may have had about the consequences of their actions.

In this respect, it is interesting to note that the prevailing diagnostic system in the Soviet Union accommodated a very broad concept of schizophrenia which included mild ('latent' or 'sluggish') and moderate forms supposedly characterised by 'personality changes'. Such blatant abuses of psychiatry are sadly not confined to the Soviet Union. China, for example, has established a system of maximum-security psychiatric hospitals ('Ankang') similar to that in the Soviet Union, in part for confining political offenders and Falun Gong practitioners who are deemed to be a 'social danger'.

4

Recommended reading

The Meaning of Madness (2009) Neel Burton. Acheron Press.
The Divided Self: An Existential Study in Sanity and Madness
 (1990) R. D. Laing. Penguin Books Ltd.
Madness Explained: Psychosis and Human Nature (2004)
 Richard P. Bentall. Penguin Books Ltd.

The Dialectics of Schizophrenia (1997) Philip Thomas. Free
 Association Books Ltd.
The Quiet Room: Journey Out of the Torment of Madness (1996)
 Lori Schiller and Amanda Bennett. Little, Brown & Co.

Summary

Epidemiology

- Prevalence of schizophrenia is around 1%, with males and females more or less equally affected.
- Onset can occur at any age but is typically in early adulthood and earlier in males than in females.

Aetiology

- Genetic factors and environmental factors are both involved in the aetiology of schizophrenia.
- The dopamine hypothesis states that positive symptoms of schizophrenia result from overactivity in the mesolimbic system, and negative symptoms from underactivity in the mesocortical system.

Clinical features

- The onset of schizophrenia is often preceded by an insidious prodromal phase that may last for several years and that consists of subtle and non-specific problems in language, cognitive ability, and behaviour that result in a loss of function.
- The symptoms of schizophrenia can be divided into positive symptoms, disorganised symptoms, and negative symptoms. Acute schizophrenia tends to be dominated by positive symptoms and chronic schizophrenia by negative symptoms. It can sometimes be difficult to differentiate chronic schizophrenia from depressive symptoms and from the motor side-effects of antipsychotic medication.
- Schneider's first rank symptoms are supposed to be specific to, and therefore pathognomonic of, schizophrenia. However, they are neither. They largely consist of florid psychotic symptoms held together by a common theme of loss of control over thoughts, feelings, and the body.

Differential diagnosis

- The differential diagnosis of schizophrenia is from other psychiatric disorders (including drug-induced psychotic disorder) and from medical/neurological disorders.

Management

- Antipsychotic drugs are effective against positive symptoms in about 70–80% of patients.
- Current treatment guidelines recommend using one of the atypical antipsychotics other than clozapine as a first line of treatment.
- Atypical antipsychotics such as risperidone, olanzapine, quetiapine, and clozapine are at least as effective as typical antipsychotics and treat the positive and, it has been argued, the affective and negative symptoms of schizophrenia. They are better tolerated than typical psychotics, principally because they cause considerably fewer extrapyramidal side-effects.
- Clozapine causes agranulocytosis in about 1% of patients and for this reason patients on the drug must have their blood counts monitored. Despite this, clozapine is the drug of choice in treatment-resistant schizophrenia.
- Psychosocial measures in the treatment of schizophrenia include family education, psychotherapy, cognitive-behavioural therapy, self-help groups, and rehabilitation.

Prognosis

- Although complete recovery is possible, schizophrenia tends to be progressive and punctuated by episodes of relapse and remission.
- Good prognostic factors include acute onset, late onset, precipitating factors, florid symptoms or associated mood disorder, female sex, no family history, no substance misuse, good premorbid occupational/social adjustment, good social support, early treatment, and good response to treatment.
- Common causes of relapse to the acute phase include reduction or discontinuation of antipsychotic medication, non-compliance with antipsychotic medication, substance misuse, high expressed emotion, life events, and child birth.

Self-assessment

Simply answer with true or false. Answers on p. 217.

1. Kraepelin's description of schizophrenia put more emphasis on thought disorder and on negative symptoms than on positive symptoms.
2. Schizophrenia tends to present earlier in women, and also tends to affect women more severely.
3. The season of birth effect is not mirrored in the southern hemisphere.
4. The lifetime risk of schizophrenia if one parent has been affected is about 12%.
5. According to the revised dopamine hypothesis, the negative symptoms of schizophrenia result from dopamine overactivity in the mesocortical system.
6. The subtle and non-specific problems in language, cognitive ability, and behaviour that form part of the prodromal phase of schizophrenia do not result in a loss of function.
7. Strong religious beliefs can be classified as delusions.
8. Pseudo-hallucinations tend to point towards a diagnosis of personality disorder rather than of schizophrenia.
9. Pseudo-hallucinations typically differ from true hallucinations in that they are perceived to arise from the mind rather than from the sense organs, and in that they are less vivid or less distressing.
10. Second person auditory hallucinations are one of Schneider's first rank symptoms.
11. Third person auditory hallucinations are one of Schneider's first rank symptoms.
12. ICD-10 criteria for the diagnosis of schizophrenia are based on Schneider's first rank symptoms.
13. According to ICD-10, for a diagnosis of schizophrenia to be made, symptoms should have been present for most of the time for a period of one month or more.
14. The main differences between the ICD-10 and DSM-IV criteria for schizophrenia is that ICD-10 specifies a six-month period of continuous signs of disturbance, and has an additional criterion specifying social/occupational dysfunction.
15. About 10% of schizophrenia patients become depressed once their psychotic symptoms have resolved.
16. 'Deficit syndrome' refers to prominent and enduring negative symptoms of schizophrenia.
17. Oculogyric crisis and akathisia are side-effects of both typical and atypical antipsychotics.
18. Tardive dyskinesia can occur in up to 10% of patients on typical antipsychotics.
19. The antiadrenergic side-effects of atypical antipsychotics include postural hypotension and tachycardia, but not ejaculatory failure.
20. Olanzapine typically causes more hyperprolactinaemia than either risperidone or quetiapine.
21. Risperidone typically causes more weight gain than either olanzapine or quetiapine.
22. Patients on clozapine must have their blood counts monitored because they are at risk of thrombocytosis.
23. Symptoms of neuroleptic malignant syndrome include hyperthermia, tremor, autonomic instability, and altered mental status.
24. Anticholinergics are used to treat tardive dyskinesia.
25. Psychotic experiences are common in the general population and, for the most part, do not constitute mental disorder.
26. In DSM-IV schizotypal disorder is classified under personality disorders.
27. Brief psychotic disorder has a short course of less than one week.
28. Brief psychotic disorder is often precipitated by acute stress.
29. *Folie à famille* describes a delusional disorder that is shared by several members of the same family.
30. In *folie imposée*, B maintains his or her delusions even if he or she is separated from A.
31. Schizophreniform disorder is a condition characterised by the development of a single delusion or set of related delusions. The delusions are of a fixed, elaborate, and systematised kind, and can often be related to the patient's life situation.
32. The prognosis for schizoaffective disorders is better than that for schizophrenia.
33. If a patient has improved on a particular drug, he or she should continue taking that same drug at the same dose for at least the next three months.
34. Acute onset is a poor prognostic factor in schizophrenia.
35. Florid positive symptoms and associated mood disorder are good prognostic factors in schizophrenia.
36. Suicide is the most common cause of death in schizophrenia.
37. Having good insight into the illness is a protective factor against suicide in schizophrenia.
38. Having high intelligence is a protective factor against suicide in schizophrenia.

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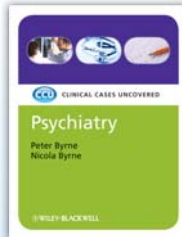
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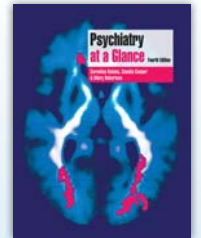
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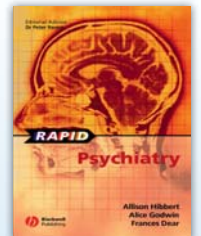
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