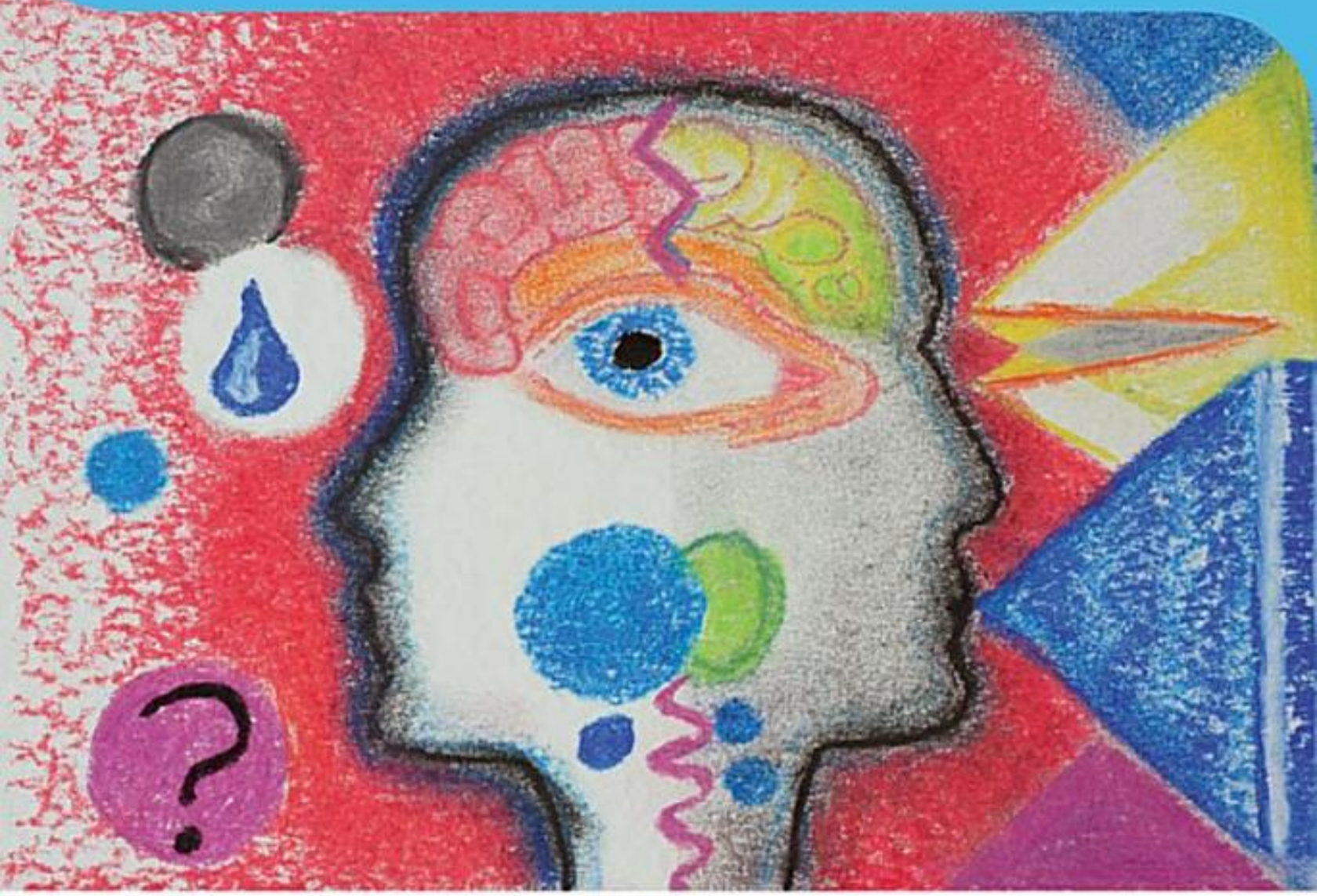
Neel Burton

Psychiatry

SECOND EDITION

Foreword by Robert Howard



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Contents

The psychiatric history

In psychiatry the history is of special importance as physical examination and investigations are seldom of diagnostic value. Mention of the psychiatric history often prompts deep sighs from medical students, but **the psychiatric history is actually not very different from any other medical or surgical history:** its structure is the same, except that 'past medical history' is divided into 'past psychiatric history' and 'past medical history', and that there is an additional section for 'personal history'. 'Drug history', 'family history', 'social history', and 'personal history' are accorded a lot of importance in the psychiatric history because of their strong bearing on the aetiology, treatment, and prognosis of mental disorders.

Thus, the psychiatric history can be carried out under 10 main headings:

1. Introductory information
2. Presenting complaint and history of presenting complaint
3. Past psychiatric history
4. Past medical history
5. Drug history/current treatments
6. Substance use
7. Family history
8. Social history
9. Personal history
10. Informant history

Keep in mind that the aim of the psychiatric history is not so much to rattle through a long list of headings, as it is to facilitate the patient's telling of his or her story. A rigid and inflexible approach may damage your rapport with the patient, who may perceive you as cold and uninterested, and lacking in tact, judgement, and understanding.

The mental state examination

The mental state examination (MSE) is, strictly speaking, **a snapshot of the patient's behaviour and mental experiences at or around that point in time.** Just as an abdominal examination is used to seek out the signs of gastrointestinal disorders, so the MSE is used to seek out the signs of psychiatric disorders. In addition, the MSE is also used to seek out the **symptoms** of psychiatric disorders, and in this respect it also resembles the functional enquiry of a medical history. Being as it is part examination and part functional enquiry, the MSE relies on a firm grasp of the signs and symptoms of psychiatric disorders (descriptive psychopathology).

Figure 2.1 A good historian is not one who practises textbook psychiatry, but one who listens for the sake of listening, with interest, respect, and understanding. This is in itself a powerful form of therapy.

The MSE is usually carried out after the psychiatric history. Alternatively, it can be carried out during the psychiatric history, immediately after 'presenting complaint and history of presenting complaint' (an approach that often makes more sense). The MSE can be carried out under seven main headings:

1. Appearance and behaviour
2. Speech
3. Mood, plus anxiety and risk assessment
4. Thoughts
5. Perception
6. Cognition
7. Insight

The MSE's role is to ensure that all important signs and symptoms of mental disorder are screened for and fully explored. It can be considered as a 'core and module' questionnaire: simple screening questions

the patient is unable to give a clear or full history, is lacking in insight, is cognitively impaired, or is under the influence of drugs or alcohol. Informant histories are also opportunities for assessing the attitudes of relatives and carers to the patient and to involve them in the treatment plan. Remember to seek consent from the patient before taking an informant history.

The mental state examination

In this section, descriptive psychopathology is integrated into the MSE in an effort to encourage direct reference to its subject: the signs and symptoms of mental disorder. Note that common and important signs and symptoms are in *italics*; the rest are included mainly for interest.

Recall that the MSE can be carried out under seven main headings:

1. Appearance and behaviour
2. Speech
3. Mood, plus anxiety and risk assessment
4. Thoughts
5. Perception
6. Cognition
7. Insight.

Appearance and behaviour

How are you? You have been in Afghanistan, I perceive.
Sherlock Holmes, *A Study in Scarlet*

Note the following:

- Level of consciousness, e.g. hyperalert/vigilant, alert, somnolent
- Appearance: body build, posture, general physical condition, grooming and hygiene, dress, physical stigmata such as scars, piercings, and tattoos. Remember that scars result not just from accidents and surgical operations, but also – and importantly – from deliberate self-harm
- Behaviour and attitude to the examiner. In particular note: facial expression, degree of eye contact, quality of rapport
- Motor activity/disorders of movement (Table 2.2). Disorders of movement that affect induced movements and posture tend to be associated with catatonic schizophrenia, which is relatively rare.

Clinical skills: Differentiating between catatonia, catalepsy, and cataplexy

Catatonia	A motor syndrome diagnosed by the presence of two or more of the following: • Motor immobility • Motor excitement • Negativism or mutism • Posturing, stereotypies or mannerisms • Echolalia or echopraxia.
Catalepsy	A feature of catatonia in which the limbs can be placed in any posture and maintained thereafter for unusually long periods of time. Catalepsy is also referred to as waxy flexibility or *cerea flexibilitas*.
Cataplexy	A sudden loss of muscle tone that leads to collapse. Cataplexy is a feature of the sleep disorder narcolepsy, and is thus completely unrelated to either catatonia or catalepsy.

Speech

Guard your roving thoughts with a jealous care, for speech is but the dealer of thoughts, and every fool can plainly read in your words what is the hour of your thoughts.
Alfred, Lord Tennyson (1809–1892)

A person's speech mirrors his or her thoughts, but under 'speech' you should limit yourself to recording the technical aspects of speech. The **content** of speech is more appropriately described under 'thoughts'.

Note the following:

- Amount, rate, volume, and tone of speech (Table 2.3)
- **Form** of speech (Table 2.3).

Mood

Record the following:

- Current mood and severity. Good screening questions for depression are,
  Have you been keeping reasonably cheerful?
  Are there times when you feel low-spirited or tearful?
  Good screening questions for mania are:
  Have you been feeling particularly cheerful?
  Have you been feeling on top of the world?
  If there is the suggestion of a mood disorder, this should be explored further (see Chapter 5). Note that it

Thought

Record the following:

- Stream of thought
- Form of thought
- Content of thought:
  - Phobias. For a phobia, record the stimulus, its psychological and physiological effects, and the nature and extent of any avoidance behaviour. A good screening question might be,
    Do you have any special fears, like some people are afraid of spiders or snakes?
  - Preoccupations, ruminations, obsessions. For an obsession, determine the underlying fear, the degree of resistance to the intrusive thoughts, and their effect on everyday life. Is the obsession perceived as being senseless? Is it accompanied by compulsive acts? A good screening question for obsessions might be,
    Do certain things keep coming into your mind, even though you try hard to keep them out?
    and for compulsive acts,
    Do you ever find yourself spending a lot of time doing the same thing over and over again, even though you've already done it well enough?
  - Delusions and overvalued ideas. For obvious reasons, you cannot easily ask directly 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 that your friends and family do not share?
    Then, if you feel that this is necessary, ask specifically about common delusional themes (Table 2.5 and see box, p. 64). For example, for delusions of control ask,
    Is someone or something trying to control you?
    Is someone or something trying to interfere with your thoughts?

Disorders of thought are listed in Table 2.6.

Reflecting upon the definition of a 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 …'

There are fictions when the society supports you, there are fictions when nobody supports you. That is the difference between a sane and an insane person; a sane person is one whose fiction is supported by the society. He has manipulated the society to support his fiction. An insane man is one whose fiction is supported by nobody; he is alone so you have to put him in the madhouse.

Reportedly said by Bhagwan Shree Rajneesh (1931–1990), quoted from Anthony Storr, *Feet of Clay: A Study of Gurus* (1997), HarperCollins

The antipsychiatry movement

The antipsychiatry movement took hold in the 1960s and early 1970s. Spearheaded by Thomas Szasz (1920–, author of *The Myth of Mental Illness*) and others, it claimed that severe mental disorder, especially schizophrenia, was little more than an attempt to medicalise and thereby control socially undesirable behaviour. Attractive though it may originally have seemed, this claim has been seriously undermined by the increasing evidence for a biological basis of severe mental disorder.

Table 2.7 Continued

Specific types of hallucination	
Hypnopompic hallucination	Visual or auditory hallucinations on awakening
Hypnogogic hallucination	Visual or auditory hallucinations on going to sleep
Extracampine hallucination	Hallucinations outside the limits of the sensory field, e.g. hearing voices from Antarctica
Functional hallucination	A hallucination triggered by an environmental stimulus in the same modality, e.g. hallucinatory voices triggered by the sound of a running tap
Reflex hallucination	A hallucination triggered by an environmental stimulus in a different modality, e.g. a *visual* hallucination triggered by the sound of a running tap
Synaesthesia	In synaesthesia sensations in one mode produce sensations in another mode; for example, a piece of music is experienced as a concert of colours. The French poet Arthur Rimbaud's 'Voyelles' is a poem about synaesthesia. Other artists to experiment with synaesthesia include Charles Baudelaire (poet), Wassily Kandinsky (painter), and Alexander Scriabin (composer)
Palinopsia	The persistence or recurrence of an image long after its stimulus has been removed
Pseudo-hallucinations	A pseudo-hallucination may differ from a true hallucination in that: • It is perceived to arise from the mind (inner space) rather than from the sense organs (outer space) • It is less vivid • It is less distressing • The patient may have some degree of control over it
Depersonalisation	An alteration in the perception or experience of the self, leading to a sense of detachment from one's mental processes or body processes or body
Derealisation	An alteration in the perception or experience of the environment, leading to a sense that it is strange or unreal

Clinical skills: Speaking to a patient about his psychotic symptoms

In speaking to a patient with psychotic symptoms, you should avoid challenging his or her delusions and hallucinations, but at the same time you should not validate them either. This difficult balance is best achieved by explicitly recognising that the patient's delusions and hallucinations are important to him or her, yet implicitly making it clear that you (personally) regard them as symptoms of mental disorder. For example,

Patient: *The aliens are telling me that they are going to abduct me tonight.*
Doctor: *That sounds terribly frightening.*
Patient: *I've never felt so frightened in all my life.*
Doctor: *I can understand that you feel frightened, although I myself cannot hear the aliens that you speak of.*
Patient: *You mean, you can't hear them?*
Doctor: *No, not at all. Have you tried ignoring them?*
Patient: *If I listen to my iPod then they don't seem so loud, and I don't feel so frightened.*
Doctor: *What about when we talk together, like now?*
Patient: *That's very helpful too.*

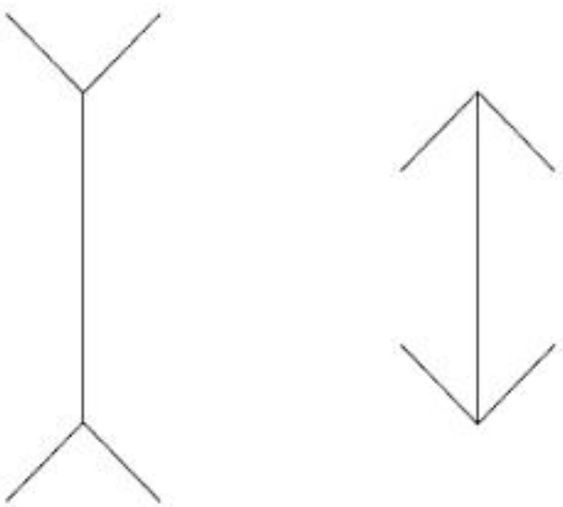


Figure 2.4 The Müller–Lyer illusion arises from the misinterpretation of a stimulus: both lines are in actual fact the same length. In contrast, a hallucination arises in the absence of a stimulus.

Table 2.10 Some possible measures for the management of mental disorder.

	Biological	Psychological	Social
Short-term	Drugs ECT Detoxification	Counselling Psychoeducation	Family education Carer support
Medium/long-term	Maintenance treatment Depot antipsychotic Mood stabiliser Addictions counseling Genetic counselling	Self-help guides Cognitive behavioural therapy Psychodynamic psychotherapy Family therapy	Patient groups Charities Benefits Housing Rehabilitation Power of attorney

Both clinical experience and research into the aetiology of psychiatric disorders suggest that many of the categorical concepts listed in classifications of mental disorders, such as schizophrenia and affective disorders, may not in fact map onto distinct disease entities, but instead lie at different extremes of a single spectrum of mental disorders.

ICD-10 classification

The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines, published in 1992, is chapter V of the *Tenth Revision of the International Classification of Diseases* (ICD-10) and is used in all countries. Other than simply listing and coding the names of diseases and disorders like other chapters in ICD-10, Chapter V provides clinical descriptions, diagnostic criteria, and diagnostic criteria for research. These are based on scientific literature and international consultation and consensus. The principal aims of the classification are to serve as a reference for national classifications and to facilitate international comparisons of morbidity and mortality statistics. ICD-10 comes in four different versions: (1) clinical descriptions and diagnostic guidelines; (2) diagnostic criteria for research; (3) primary care version; and (4) multiaspect (axial) systems.

The broad categories of mental and behavioural disorders in ICD-10 are:

F0–F9 Organic, including symptomatic, mental disorders
F10–F19 Mental and behavioural disorders due to psychoactive substance use
F20–F29 Schizophrenia, schizotypal and delusional disorders
F30–F39 Mood (affective) disorders
F40–F48 Neurotic, stress-related and somatoform disorders
F50–F59 Behavioural syndromes associated with physiological disturbances and physical factors
F60–F69 Disorders of adult personality and behaviour
F70–F79 Mental retardation
F80–F89 Disorders of psychological development
F90–F98 Behavioural and emotional disorders with onset usually occurring in childhood and adolescence
F99 Unspecified mental disorder

Note that these are organised in a loose diagnostic hierarchy (see above).

DSM-IV classification

The first classification of mental and behavioural disorders appeared in the sixth edition of *International Classification of Diseases*, published in 1948. The American Psychiatric Association (APA) did not lose much time in publishing an alternative classification, the *Diagnostic and Statistical Manual of Mental Disorders (DSM)*, for use in the USA. The fourth revision of the DSM, published in 1994 (Text Revision in 2000), is broadly similar to the ICD-10 classification. However, unlike the ICD-10 classification, which is available in four different versions, there is only one, multiaxial, version of DSM-IV.

Next steps:

1. Appointment made with Dr A (SHO) next Monday at 3 pm to monitor response to amisulpiride, monitor symptoms, and support and educate the patient and family
2. Weekly home visits by CPN to monitor symptoms, and support and educate the patient and family
3. CPN to provide the patient with the contact details of a local schizophrenia support group.

Prognosis

In the short-term he is likely to respond to amisulpiride and make a good recovery. In the long-term, he is likely to suffer from further relapses, particularly if he comes under stress, uses drugs, or stops taking his medication. Compliance with anti-psychotic medication is likely to be a major issue.

Positive prognostic factors, at least initially, include acute onset, presence of precipitating factors, florid symptoms, good premorbid occupational/social adjustment, good social support, early treatment, and good response to treatment.

Negative prognostic factors include male sex, family history, history of substance misuse, history of poor compliance with medication, and lack of a confiding relationship.

Recommended reading

Symptoms in the Mind: An Introduction to Descriptive Psychopathology (1997) Andrew Sims. W. B. Saunders Co Ltd.

Clinical Psychopathology: Signs and Symptoms in Psychiatry (1985) Frank Fish and Max Hamilton (eds). Butterworth Heinemann.

Psychiatric Interviewing: The Art of Understanding (1998) Shawn Shea. W. B. Saunders Co Ltd.

The Present State Examination (1974) J. K. Wing. Cambridge University Press.

Pocket Guide to the ICD-10 Classification of Mental and Behavioural Diseases (1994) J. E. Cooper. WHO.

Patient assessment: Checklist and summary

Psychiatric history

1. Introductory information.
2. Presenting complaint/history of presenting complaint: use open questions first, record patient's complaints verbatim, use a logico-deductive approach.
3. Past psychiatric history, including previous episodes of mental disorder, treatments, admissions, and history of self-harm and harm to others.
4. Past medical history, including history of epilepsy or head injury and vascular risk factors.
5. Drug history/current treatments, including psychological treatments, recent changes in medication, adverse reactions, and alternative remedies.
6. Substance use: alcohol, tobacco, and illicit drugs.
7. Family history, including quality of relationships and recent events in the family.
8. Social history: self-care, support, housing, finances, typical day, interests and hobbies, premorbid personality.
9. Personal history: pregnancy and birth, developmental milestones, childhood problems, educational achievement, occupational history, psychosexual history, forensic history, religious orientation.
10. Informant history/histories.

Mental state examination

1. Appearance and behaviour: level of consciousness, appearance, behaviour, motor activity/disorders of movement.
2. Speech: especially amount, rate, and form.
3. Mood: subjective and objective mood, affect, ideas of self-harm and suicide, ideas of harm to others, anxiety.
4. Thoughts: stream, form, content: delusions, overvalued ideas, preoccupations, ruminations, obsessions, phobias.
5. Perception: sensory distortions, illusions, hallucinations, depersonalisation and derealisation.
6. Cognition: orientation, attention and concentration, memory, grasp.
7. Insight.

Formulation

1. Case summary or synopsis.
2. Further information required: full psychiatric history and mental state examination, informant histories, old records, physical examination, laboratory investigations, brain imaging, psychological tests and inventories.

Continued...

psychiatric services, including admission to a psychiatric hospital. Patients in a crisis are referred to the Crisis Team from a variety of places and agencies, most commonly GPs, A&E, and CMHTs. A member of the team (often a community psychiatric nurse) promptly assesses the patient in conjunction with a psychiatrist to determine if a hospital admission can be avoided by providing short-term intensive home care. If so, the Crisis Team arranges for a team member to visit the patient's home up to three times a day, gradually decreasing the frequency of visits as the patient gets better. Other than simply providing support, the Crisis Team can assist in implementing a care and treatment plan and in monitoring progress. If a patient has already been admitted to hospital, the Crisis Team can get involved in expediting and facilitating his discharge back into the community. The key features of the Crisis Team are summarised in Table 3.3.

One Flew Over the Cuckoo's Nest

Vintery, mintery, cutery, corn,
Apple seed and apple thorn;
Wire, briar, limber lock,
Three geese in a flock.
One flew east,
And one flew west,
And one flew over the cuckoo's nest.

Popular nursery rhyme

The film *One Flew Over the Cuckoo's Nest*, adapted from Ken Kesey's popular 1962 novel of the same name, is directed by Milos Forman and stars Jack Nicholson as the spirited R. P. McMurphy ('Mac') and Louise Fletcher as the chilly but softly-spoken Nurse Ratched. When Mac arrives at the Oregon state mental hospital, he challenges the stultifying routine and bureaucratic authoritarianism personified by Nurse Ratched, and pays the price by being drugged, electro-shocked and, ultimately, lobotomised. Nominated for nine Academy Awards, the film is not only a (belated and contentious) criticism of the institutional model of psychiatric care, but also a metaphor of total institutions – institutions that repress individuality to create a compliant society. It is such criticism of the institutional model of psychiatric care that, in the UK and other countries, led to the development of community care.

It seems that utopias are much more easily achieved than we once thought. Today we are faced with a different and more agonising question: How do we prevent them from being finally achieved?... Utopias can be achieved. Life moves on towards utopia. And perhaps a new age is beginning, an age when intellectuals and cultured people will dream up a way of avoiding utopias, and returning to a society that is not utopian, with less 'perfection' and more freedom.

Nicolas Berdiaeff, translated from the foreword to *Brave New World* by Aldous Huxley, Longman Edition

Assertive Outreach Team

Some people with a severe mental disorder are reluctant to seek help and treatment, and as a consequence only appear in times of crisis. Paradoxically, these so-called 'revolving door patients' often have the most complex mental health needs and social problems. For this reason, the responsibility for their care is sometimes transferred to the Assertive Outreach Team (AOT), a specialised multidisciplinary team dedicated to engaging them in treatment and supporting them in their daily activities.

Early Intervention Service

Like the AOT, the Early Intervention Service (EIS) may also operate from the CMHT base. Its role is specifically to improve the short- and long-term outcomes of schizophrenia and other psychotic disorders through a three-pronged approach involving preventative measures, earlier detection of untreated cases, and intensive treatment and support in the early stages of illness.

Hospital and day hospital

If a patient requires admission to a psychiatric hospital, this is usually because care in the community is not an option. Commonly this is because:

- The patient is a danger to himself and/or to others
- The patient requires specialised care or supervised treatment
- The patient is lacking a social structure
- Carers can no longer cope and are in need of respite.

The vast majority of patients who are admitted are done so on an informal, voluntary basis. This is either because they are happy to take the advice of their psychiatrist or carers or because they are frightened of their symptoms and feel that the hospital is a relatively safe place for them

Mental capacity

Doctors and especially psychiatrists may be called upon to give an assessment of capacity and competence to decide on the ability of a patient to give informed consent or enter into another contract.

The terms 'capacity' and 'competence' are often used interchangeably, but strictly speaking:

- 'Capacity' is a **legal presumption** that adult persons have the ability to make decisions
- 'Competence' is a **clinical determination** of a patient's ability to make decisions about his or her treatment.

Issues about capacity arise in three groups of patients: children and adolescents, patients with learning difficulties, and patients with mental illness. A person has capacity so long as he or she has the ability to understand and retain relevant information for long enough to reach a **reasoned** decision, **regardless of the actual decision reached.** An adult person should be presumed to have the competence to make a particular decision until a judgement about capacity can be made. This judgement can only be made about present capacity, not about past or future capacity, and it should only be made for a specific decision, as different decisions require different levels of capacity. If capacity is lacking or cannot be established (e.g. in an emergency situation), treatment can be justified under the common law *Principle of Necessity*, as established by the case of *Re F* (1990). The doctor in charge has the responsibility to act in the best interests of the patient and in accordance with a responsible and competent body of opinion, as established by the case of *Bolam v Friern Hospital Management Committee* (1957) (the 'Bolam test'). Nevertheless, it is good practice for him or her to involve colleagues, carers, and relatives in the decision-making. In difficult situations or if there are differences of opinion about the patient's best interests, the doctor should consult a senior colleague or seek expert or legal advice. In England and Wales, the Mental Capacity Act 2005 provides for a Court of Protection to help with difficult decisions. Note that, in some cases, a child (a person under the age of 16) can be competent to consent to treatment if he or she fully understands the treatment proposed. This is sometimes referred to as 'Gillick competency', because it relies on a ruling of the House of Lords in the case *Gillick v West Norfolk and Wisbech Area Health Authority* (1985).

Having deemed Mr AB to be lacking in capacity on the grounds of mental illness, the casualty officer proceeded to act in his best interests by breaking confidentiality and disclosing information to and seeking information from Mrs AB (this information turned out to be of vital importance in Mr AB's assessment and management). Similarly, the psychiatrist proceeded to act in his best interests by admitting him as an involuntary patient and starting him on antidepressant therapy.

Re C (1994)

Mr C was a patient in a psychiatric secure hospital who had chronic paranoid schizophrenia with grandiose delusions of being a world famous doctor. When he developed gangrene in his right foot, he refused to consent to a below-knee amputation, as a result of which he was granted an injunction preventing such an operation. In granting such an injunction, Justice Thorpe held that Mr C sufficiently understood the nature, purpose, and effects of the proposed amputation, and that he retained sufficient capacity to consent to, or refuse, medical treatment. The case of *Re C* helped to establish the 'Re C criteria' or legal criteria for capacity.

Clinical skills/OSCE: Assessing mental capacity and obtaining consent

1. Ensure that the patient understands:
   - What the intervention is
   - Why the intervention is being proposed
   - The alternatives to the intervention, including no intervention
   - The principal benefits and risks of the intervention and of its alternatives
   - The consequences of the intervention and of its alternatives.
2. Ensure that the patient retains the information for long enough to weigh it in the balance and reach a reasoned decision, whatever that decision might be.
3. Ensure that the patient is not subject to coercion or threat.

It is important to bear in mind that a patient's capacity can and should be enhanced by, for example:

- Making your explanations easier to understand, e.g. adapting your language, using diagrams
- Seeing the patient at his or her best time of day
- Seeing the patient with a friend or relatives of his
- Improving the patient's environment, e.g. turning off the television, finding a quiet side-room
- Adjusting the patient's medication, e.g. decreasing the dose of sedative drugs.

Adapted from *Clinical Skills for OSCEs*, 3e (2009), by Neel Burton, Scion Publishing

on the functions which were performed by the Approved Social Worker (ASW, renamed 'Approved Mental Health Professional') and the Responsible Medical Officer (RMO, renamed 'Responsible Clinician')
- The ability for a person to make an application to displace (or change) his or her nearest relative, and the ability for civil partners to be the nearest relative
- The replacement of supervised discharge with 'Supervised Community Treatment', with a power to recall a person into hospital if he does not comply with certain conditions
- Statutory advocacy for all detained persons
- New safeguards for electroconvulsive therapy, which may no longer be given to a person who has capacity to refuse consent to it, and may only be given to a person without such capacity if it does not conflict with an advance directive, decision of a donee or deputy, or decision of the Court of Protection.

Clinical skills: Mental disorders and driving

The following advice applies to mania, schizophrenia and other schizophrenia-like psychotic disorders, and more severe forms of anxiety and depression.

You should stop driving during a first episode or relapse of your illness, because driving while ill can seriously endanger lives. In the UK, you must notify the Driver and Vehicle Licensing Authority (DVLA). Failure to do so makes it illegal for you to drive and invalidates your insurance. The DVLA then sends you a medical questionnaire to fill in, and a form asking for your permission to contact your psychiatrist. Your driving licence can generally be reinstated if your psychiatrist can confirm that:
- Your illness has been successfully treated with medication for a variable period of time, typically at least 3 months
- You are conscientious about taking your medication
- The side-effects of your medication are not likely to impair your driving
- You are not misusing drugs.

NB: People who suffer from substance misuse or dependence should also stop driving, as should *some* people who suffer from other mental disorders such as dementia, learning disability, or personality disorder.

Further information can be obtained from the DVLA website at www.DVLA.gov.uk. Note that the rules for professional driving are different from those described above.

Introduction to psychological or 'talking' treatments

> *He* [the mystical physician to the King of Thrace] *said the soul was treated with certain charms, my dear Charmides, and that these charms were beautiful words.*
>
> Plato (428–347BC), *Charmides*

Although drug treatments are the most readily available treatment option for mental disorders such as anxiety and depression, psychological or 'talking' treatments can in many cases be more effective. Many people prefer psychological treatments to drug treatments, because they consider (often correctly) that psychological treatments address underlying problems, rather than simply mask superficial symptoms. Of course, drug treatment and psychological treatment are not mutually exclusive and, although psychological treatments can have a role to play in mental disorders such as schizophrenia, bipolar disorder, and severe depression, they are no substitute for treatment with antipsychotic, mood-stabilising, or antidepressant medication. The type of psychological treatment that is chosen, if any, depends not only on the patient's diagnosis but also on his personal circumstances, his preferences, and – sadly all too often – on the funding and human resources that are available in his local area.

At its most basic, psychological treatment involves little more than explanation and reassurance. Such 'supportive therapy' should form an important part of treatment for all mental disorders, and in mild anxiety or depression is often the only treatment that is necessary or, indeed, appropriate. Counselling is similar to supportive therapy in that it involves explanation, reassurance, and support. However, it is more problem-focused and goal-oriented than supportive therapy, and also involves the identification and resolution of current life difficulties.

In contrast to supportive psychotherapy, exploratory psychotherapy such as cognitive-behavioural therapy (CBT) and psychodynamic psychotherapy aims to delve into the person's thoughts and feelings. Although CBT and psychodynamic psychotherapy are both forms of exploratory psychotherapy, CBT is principally based on learning and cognitive theories, whereas psychodynamic psychotherapy is principally based on psychoanalytical theory. Psychodynamic psychotherapy is similar to psychoanalysis but is briefer and less intensive; it aims to bring unconscious feelings to the surface so that they can

Part 2

That he's mad, 'tis true, 'tis true 'tis pity,
And pity 'tis 'tis true – a foolish figure,
But farewell it, for I will use no art.
Mad let us grant him then, and now remains
That we find out the cause of this effect,
Or rather say, the cause of this defect,
For this effect defective comes by cause:
Thus it remains, and the remainder thus.

Shakespeare, *Hamlet*, Act II, Scene 2

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

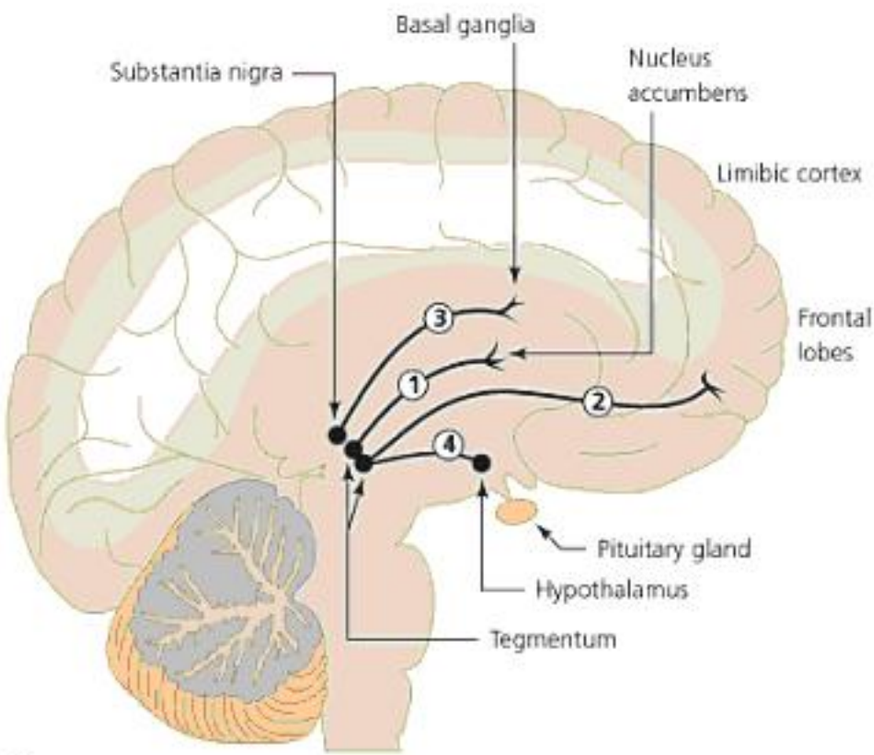
In light of this conflicting evidence, Davis and colleagues revised the dopamine hypothesis by theorising that the positive symptoms of schizophrenia resulted from dopamine overactivity (hyperdopaminergia) in the **mesolimbic system**, as previously thought, but that the negative symptoms of schizophrenia 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 D_2 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 D_2 receptor subtype in the mesolimbic system or because they act primarily on serotoninergic 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 Mesocorticol 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.

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, hypochrondriacal 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 hyperkinesis 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.

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 hypnogogic 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).

4

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.

! 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 transquillisation'). 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**

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.

Affective (mood) disorders 5

Key learning objectives

- Epidemiological factors in mood disorders
- Aetiological factors in mood disorders, including monoamine hypothesis of depression
- Clinical features of depression, mania, and hypomania
- Differential diagnosis of depression, including organic causes
- Be able to assess a patient with low mood
- Be able to talk to a patient about starting an antidepressant or mood stabiliser
- Be able to talk to a patient about electroconvulsive therapy (ECT)

Testimony of a bipolar sufferer

I have been high several times over the years, but low only once.

When I was high, I became very enthusiastic about some project or another and would work on it with determination and success. During such highs I wrote the bulk of two books and stood for parliament as an independent. I went to bed very late, if at all, and woke up very early. I didn't feel tired at all. There were times when I lost touch with reality and got carried away. At such times, I would jump from project to project without completing any, and did many things which I later regretted. Once I thought that I was Jesus and that I had a mission to save the world. It was an extremely alarming thought.

When I was low I was an entirely different person. I felt as though life was pointless and that there was nothing worth living for. Although I would not have tried to end my life, I would not have regretted death. I did not have the wish or the energy to do even the simplest of tasks. Instead I withered away my days sleeping or lying awake in bed, worrying about the financial problems that I had created for myself during my highs. I also had a feeling of unreality, that people were conspiring to make life seem normal when in actual fact it was unreal. Several times I asked the doctor and the nurses to show me their ID because I just couldn't bring myself to believe that they were real.

Psychiatry, 2e. By Neel Burton. Published 2010 by Blackwell Publishing.

Aetiology

Genetics

The prevalence rate for major depression in first-degree relatives is about 15%, compared to about 5% in the general population. Although first-degree relatives of a depressed patient are at increased risk of depressive disorders, they are *not* at increased risk of bipolar affective disorder or schizoaffective disorder. **The concordance rate for major depression in monozygotic twins is 46%, compared to 20% in dizygotic twins.** There is thus an important genetic component to the aetiology of depressive disorders. The inheritance pattern is no doubt polygenic, but more research is needed to identify the genes involved.

Neurochemical abnormalities

The monoamine hypothesis of depression suggests that depression results from the depletion of the monoamine neurotransmitters noradrenaline, serotonin, and dopamine. In its revised version the monoamine hypothesis of depression recognises that depression may actually result not from a depletion of the monoamine neurotransmitters, but from a change in their receptors' function.

Support for the original monoamine hypothesis of depression comes from several findings, notably:

- Antidepressants increase the levels of the monoamine neurotransmitters:
  - Monoamine oxidase inhibitors (MAOIs) inhibit the degradation of monoamines presynaptically;
  - Tricyclic antidepressants (TCAs) inhibit the reuptake of noradrenaline from the synaptic cleft;
  - Selective serotonin reuptake inhibitors (SSRIs) inhibit the reuptake of serotonin from the synaptic cleft
- Amphetamines and cocaine increase the levels of monoamines in the synaptic cleft and can elevate mood
- Reserpine decreases the levels of monoamines presynaptically and can depress mood
- CSF levels of 5-hydroxyindoleacetic acid (5-HIAA), a serotonin metabolite, are decreased in depression sufferers.

5

Psychopharmacology: Serotonin in the brain

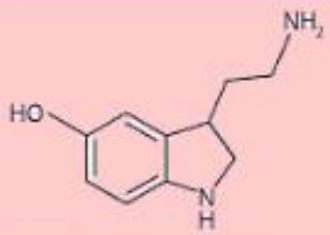


Figure 5.7 Chemical structure of serotonin.

Tryptophan
↓ *Tryptophan hydroxylase*
5-hydroxy tryptophan (5-HTP)
↓ *5-HTP decarboxylase*
Serotonin

Figure 5.8 Synthesis.

Inactivation is by reuptake from the synapse into the presynaptic neuron through the 5-HT reuptake transporter, which can be inhibited by serotonin-selective reuptake inhibitors, tricyclic antidepressants, cocaine, and ecstasy. Degradation is by monoamine oxidase (MAO), which converts serotonin to 5-hydroxyindole acetaldehyde, which is then to converted 5-hydoxyindole acetic acid, the major excreted metabolite of serotonin.

Receptors

The raphe nuclei are the principal source of serotonin in the brain: they are grouped in pairs and distributed along the entire length of the brainstem. There are at least seven families of serotonin receptors. They are all G-coupled metabotropic receptors except for the 5-HT_3 receptor, which is a ligand-gated ion channel.

Functions

Mood, anxiety, sleep, appetite, sexuality, vomiting, regulation of body temperature.

Figure 5.9 A drawing by an inpatient suffering from severe depression. She is drowning, and each time she struggles up near the surface, she is pushed back down.

Dysthymia

Dysthymia is characterised by mild chronic depressive symptoms that are not sufficiently severe to meet the criteria for mild depressive disorder. Although dysthymia has sometimes been regarded as a 'depressive personality', genetic studies suggest that it is in fact a chronic, mild form of depressive disorder. If it develops into a depressive disorder, it is then referred to as 'double depression' (Figure 5.10). Its lifetime prevalence is about 3% and, as it is a very chronic condition, its point prevalence is not significantly different. Dysthymia may respond to drug treatment and to psychological treatments, although there is no firm evidence base for the latter.

Figure 5.10 Dysthymia and double depression.

Diagnosis

ICD-10 criteria for depressive episode

In typical depressive episodes of all three varieties described in ICD-10 (mild, moderate, and severe), the individual usually suffers from depressed mood, loss of interest and enjoyment, and reduced energy leading to increased fatigability and diminished activity. Marked tiredness after only slight effort is common. Other common symptoms are:

- *Reduced concentration and attention*
- *Reduced self-esteem and self-confidence*
- *Ideas of guilt*
- *Pessimism*
- *Ideas of self-harm or suicide*
- *Disturbed sleep*
- *Poor appetite.*

Mood varies little from day to day and is often unresponsive to circumstances. In some cases, anxiety, distress, and motor agitation may be more prominent than depressed mood. For depressive episodes of all three grades of severity a duration of at least ***two weeks*** *is usually required for diagnosis,* ***but shorter periods may be reasonable if symptoms are unusually severe and of rapid onset****. The categories of mild, moderate, and severe depressive episodes should only be used for a single (first) depressive episode, and further episodes should be classified under one of the subdivisions of recurrent depressive disorder.*

- Although it is true that antidepressants are not a solution to life's problems, they can lift your mood and give you a better chance of addressing them
- Antidepressants are effective in over 60% of patients but it can be 10–20 days before you start noticing an effect. Better sleep is often the first sign of improvement
- Antidepressants may have troublesome side-effects, but these do tend to resolve in the first month of treatment. (List the common and potentially dangerous side-effects of the main alternatives)
- Antidepressants should not be stopped suddenly once treatment is established.

If a patient fails to respond to an adequate trial of an antidepressant (i.e. the antidepressant has been prescribed at its therapeutic dose for a period of at least one month), check compliance. If the patient has been compliant, the diagnosis is not in doubt, and there are no significant perpetuating factors (e.g. hypothyroidism, alcoholism, social factors), increase the dose to the recommended maximum or tolerated dose. If the patient still fails to respond, try another drug from the same class or from a different class. If the patient still fails to respond, this is referred to as 'treatment-resistant depression'. A third antidepressant can be tried, although it is important to remember that antidepressants are not the only form of treatment for depression (see later).

Serotonin-selective reuptake inhibitors

Serotonin-selective reuptake inhibitors (SSRIs) such as fluoxetine, fluvoxamine, paroxetine, sertraline, citalopram, and escitalopram (the pharmacologically active S-enantiomer of citalopram) selectively inhibit the reuptake of serotonin. SSRIs have replaced TCAs as the first line of treatment, notably because of their lesser need for dose titration and their safety in overdose. They are particularly useful in the elderly and the physically ill, in mixed anxiety–depression, and in suicidal patients. The response rate to SSRIs is 55–70%, but improvement in mood may be delayed for 10–20 days. Side-effects include dry mouth, nausea, vomiting, diarrhoea, dizziness, sedation, sexual dysfunction, agitation, akathisia, parkinsonism (rare), and convulsions (rare). As fluoxetine, fluvoxamine, and paroxetine in particular are potent inhibitors of the cytochrome P450 isoenzymes, they can also cause important pharmacokinetic drug interactions.

The **SSRI discontinuation syndrome** consists of headache, dizziness, shock-like sensations, paraesthesia, gastrointestinal symptoms, lethargy, insomnia, and changes in mood (depression, anxiety/agitation), and occurs most frequently after the abrupt discontinuation of paroxetine, which has a comparatively short half-life. Note that the fact that a discontinuation syndrome has been described does not mean that SSRIs are 'addictive' in the sense that people do not experience a 'high' from them, and do not seek or crave them as they might a drug of abuse such as cocaine or heroin.

! The serotonin syndrome

The **serotonin syndrome** is a rare but potentially fatal acute syndrome resulting from increased serotonin (5-HT) activity. It is most often caused by SSRIs but can be caused by other drugs too, e.g. TCAs or lithium.

Symptoms include:

- Psychological symptoms: agitation, confusion
- Neurological symptoms: nystagmus, myoclonus, tremor, seizures
- Other symptoms: hyperpyrexia, autonomic instability.

The principal differential of serotonin syndrome is from neuroleptic malignant syndrome. Management involves the discontinuation of the drug and institution of supportive measures.

Recent doubts about the efficacy of SSRIs

Doctors often tell people starting on an SSRI that they have a 55–70% chance of responding to their medication. However, a recent paper (E. H. Turner *et al*, Selective publication of antidepressant trials and its influence on apparent efficacy. *New England Journal of Medicine* (2008), 358(3), 252–260) suggested that the effectiveness of SSRIs is greatly exaggerated as a result of a bias in the publication of research studies. Of 74 studies registered with the United States Food and Drug Administration (FDA), 37 of 38 studies with positive results were published in academic journals. In contrast, only 14 of 36 studies with negative results were published in academic journals, and 11 of these were published in such a way that they conveyed a positive outcome. Thus, whilst 94% of published studies conveyed a positive outcome, only 51% of all studies actually demonstrated one.

Continued...

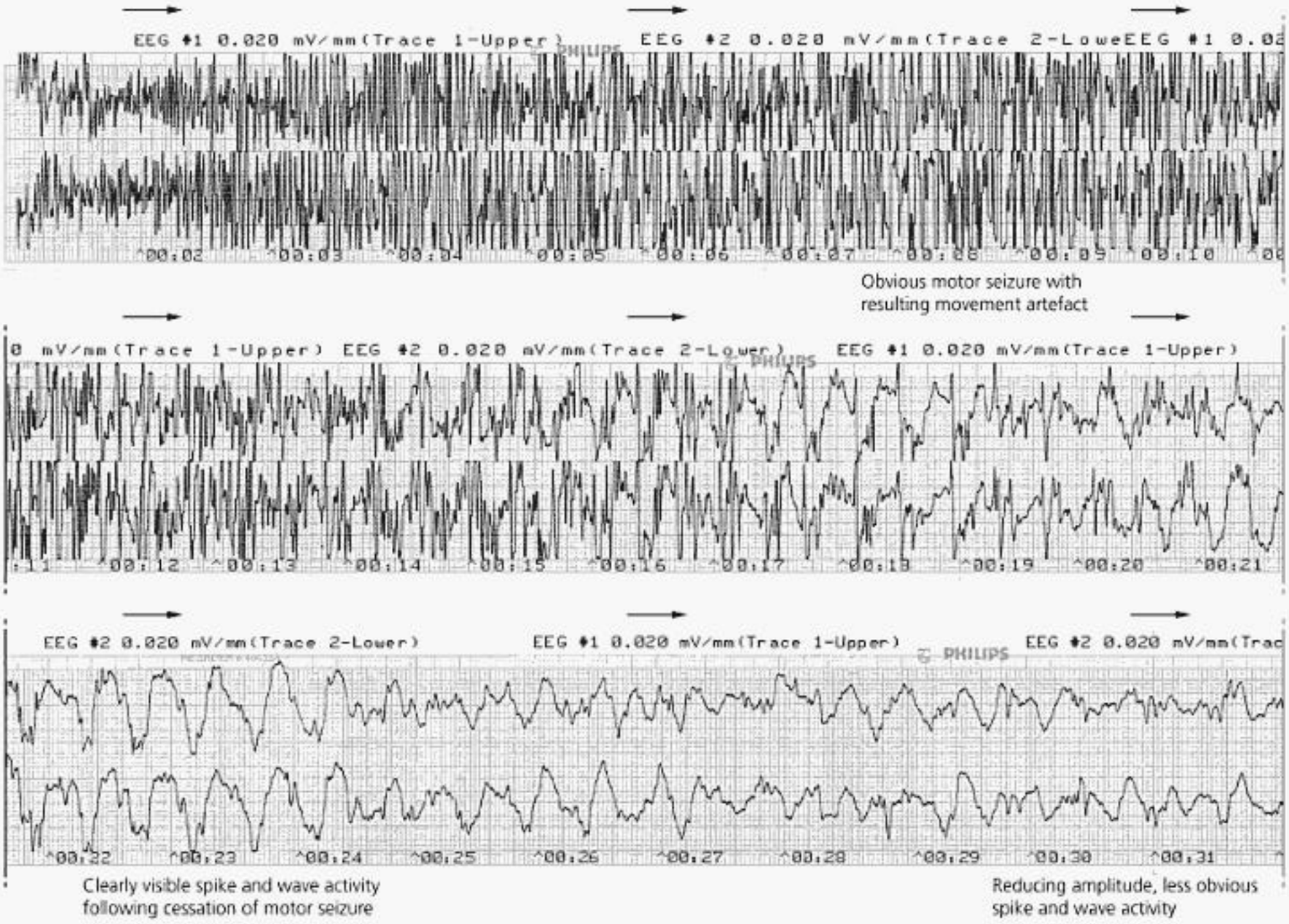


Figure 5.12 EEG activity following the delivery of bilateral constant current, brief-pulse ECT at a voltage above the patient's seizure threshold.

Epidemiology

The lifetime risk for bipolar affective disorder (BAD) ranges from 0.3% to 1.5% and because it is a chronic disorder, the prevalence rate is fairly similar. All races and both sexes are equally affected, although a recent study found that the incidence rates of BAD in ethnic minority groups in London, Bristol, and Nottingham are several times higher than those in comparison Caucasian groups (T. Lloyd *et al*, Incidence of bipolar affective disorder in three UK cities: results from the AESOP study. *British Journal of Psychiatry* (2005), 186, 126–131). The mean age of onset is 21 years and, although the age of onset is variable, a first episode of mania after the age of 50 should lead to an investigation for a primary cause such as organic brain disease or an endocrine or metabolic disorder. Interestingly, the prevalence rate of BAD is higher in higher socioeconomic groups. This may be because genetic susceptibility to BAD tends to favour unaffected relatives and sometimes even bipolar sufferers themselves, such that they are more creative, and hence more successful, than average. In *Touched by Fire: Manic Depressive Illness and the Artistic Temperament*, Professor Kay Redfield Jamison estimates that the prevalence of BAD is 10–40 times higher amongst artists than amongst the general public. Artists who suffered (or are thought to have suffered) from bipolar disorder include the authors Hans Christian Andersen, Honoré de Balzac, F. Scott Fitzgerald, Ernest Hemingway, Victor Hugo, Edgar Allan Poe, Mary Shelley, Mark Twain, and Virginia Woolf; the poets William Blake, Emily Dickinson, T. S. Eliot, John Keats, Robert Lowell, Sylvia Plath, Alfred Lord Tennyson, and Walt Whitman; and the composers Ludwig van Beethoven, Hector Berlioz, George Frederic Handel, Gustav Mahler, Sergei Rachmaninoff, Robert Schumann, and Peter Tchaikovsky.

Aetiology

Genetics

First-degree relatives of a bipolar sufferer have a 10% lifetime risk of BAD, and also have increased risks of unipolar depression and schizoaffective disorder. The concordance rate for BAD in monozygotic twins is 79% – higher than in either depressive disorders or schizophrenia – as compared to only 19% in dizygotic twins. Furthermore, children of bipolar sufferers remain at increased risk of affective disorders even after adoption by unaffected foster parents. There is thus a strong genetic component to the aetiology of BAD, stronger, in fact, than in any other psychiatric disorder. The inheritance pattern is most likely to be polygenic, but more research is needed to identify the genes involved.

Neurochemical abnormalities

The monoamine hypothesis of depression suggests that mania results from increased levels of noradrenaline, serotonin, and dopamine, and it has been observed that stimulant drugs such as cocaine and amphetamines can exacerbate mania. Unfortunately, neurochemical abnormalities in mania have not been as extensively studied as in depression.

5

That fine madness

I am come of a race noted for vigor of fancy and ardour of passion. Men have called me mad; but the question is not yet settled, whether madness is or is not the loftiest intelligence – whether much that is glorious – whether all that is profound – does not spring from disease of thought – from moods of mind exalted at the expense of the general intellect. They who dream by day are cognizant of many things which escape those who dream only by night. In their grey visions they obtain glimpses of eternity… They penetrate, however ruderless or compassless, into the vast ocean of the 'light ineffable'.

Edgar Allan Poe, *Eleonora*

But if a man comes to the door of poetry untouched by the madness of the Muses, believing that technique alone will make him a good poet, he and his sane companions never reach perfection, but are utterly eclipsed by the performances of the inspired madman.

Plato, *Phaedrus*

others, or to require hospitalisation to prevent harm to self or others, or there are psychotic features.

E. *The symptoms are not due to a substance or a general medical condition.*

DSM-IV criteria for mixed episode

A. *The criteria are met both for a manic episode and for a major depressive episode (except for duration) nearly every day during at least a one-week period.*

B. *The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to require hospitalisation to prevent harm to self or others, or there are psychotic features.*

C. *The symptoms are not due to a substance or a general medical condition.*

Differential diagnosis

Psychiatric disorders

- Mixed affective states (simultaneous manic and depressive symptoms)
- Schizoaffective disorder
- Schizophrenia
- Cyclothymic disorder
- Attention-deficit hyperactivity disorder
- Drugs such as alcohol, amphetamines, cocaine, hallucinogens, antidepressants, L-dopa, steroids

Medical/neurological disorders

- Organic brain disease of the frontal lobes such as cerebrovascular accident, multiple sclerosis, intracranial tumours, epilepsy, AIDS, neurosyphilis
- Endocrine disorders, e.g. hyperthyroidism, Cushing's syndrome
- Systemic lupus erythematosus
- Sleep deprivation

Management

Clinical skills: Investigations in mania

Laboratory investigations should include a serum and/or urine drug screen, liver, renal and thyroid function tests, full blood count, ESR, and a urine test (including pregnancy test). The aim of these investigations is to rule out drug abuse, establish baselines for the administration of mood-stabilising medication, and uncover possible medical causes for the patient's symptoms. Other, more specific, investigations such as antinuclear antibody and urine copper level should be considered on a case-by-case basis. A pretreatment ECG is important prior to starting lithium and some other drugs. If the patient is already on lithium, a lithium level should be taken.

Treatment

Methods of treatment for mania and BAD include:

- Mood stabilising and other drugs
- Electroconvulsive therapy (rarely used)
- Psychosocial treatments.

The choice of medication in BAD is largely determined by current symptoms. In a manic episode, the treatment most often prescribed is antipsychotic medication. In a depressive episode, the treatment most often prescribed is antidepressant medication, often in conjunction with a mood stabiliser to avoid 'manic switch', that is, overtreatment into mania. In rare instances, a depressive episode may be so severe or unresponsive to medication that ECT might be indicated. ECT might also be indicated for mania that cannot be treated by medication, either because it is unresponsive to medication or because medication is contraindicated. Finally, in the long term the patient should be prescribed a mood stabiliser to prevent further relapses into mania and depression. Although medication plays a central role in the management of BAD, there are a number of psychological and social interventions that can play a major role not only in improving outcome, but also in improving quality of life.

Recommended reading

Darkness Visible: A Memoir of Madness (2001) William Styron. Vintage.

The Noonday Demon (2002) Andrew Solomon. Vintage.

Churchill's Black Dog and other Phenomena of the Human Mind (1997) Anthony Storr. HarperCollins.

An Unquiet Mind (1997) Kay Redfield Jamison. Picador.

Touched with Fire: Manic Depressive Illness and the Artistic Temperament (1996) Kay Redfield Jamison. Simon & Schuster.

Summary

Depressive disorders

Classification

- In ICD-10 depressive disorders are classified according to their severity into mild, moderate, severe, and psychotic depressive disorder. In DSM-IV the term 'major depression' is used instead of depressive disorder. Major depression is simply sub-classified as 'single episode' or 'recurrent'.

Epidemiology

- The lifetime risk of depressive disorders is about 15%. The point prevalence is about 5%.
- Females are more affected than males by a ratio of about 2:1. Peak prevalence in males is in old age, but in females it is in middle age.

Aetiology

- Genetic factors and environmental factors are both involved in the aetiology of depressive disorders.
- The monoamine hypothesis of depression suggests that depression results from underactivity of monoamine projections.
- Organic causes of depression include neurological conditions, endocrine conditions, metabolic abnormalities, infections, and drugs.

Clinical features

- The clinical features of depression can be divided into core features, other common features, and somatic features.
- Dysthymia is characterised by mild chronic depressive symptoms that are not sufficiently severe to meet the criteria for mild depressive disorder.

Differential diagnosis

- The differential diagnosis of depression is from other psychiatric disorders and from secondary depression (depression due to medical or organic causes).

Management

- Methods of treatment include antidepressants, other drugs, electroconvulsive therapy, and psychological and social treatments.
- Psychological and social treatments are often preferred by patients because they are seen to address underlying problems rather than simply treating symptoms.

Prognosis

- The average length of a depressive episode is about six months. After a first depressive episode, about 80% of patients have further depressive episodes.

Disorders of the puerperium

- Maternity blues occurs in about 50% of mothers on the third or fourth day postpartum.
- Postnatal depression occurs in about 10–15% of mothers in the first month postpartum.
- Puerperal psychosis occurs in about 0.2% of mothers at about 7–14 days postpartum.

Mania and bipolar affective disorder

Classification

- In DSM-IV a single episode of mania is sufficient to meet the criteria for bipolar disorder. Bipolar I consists of episodes of mania and major depression, bipolar II of episodes of hypomania and major depression.

Epidemiology

- The lifetime risk for bipolar disorder ranges from 0.3% to 1.5%. Mean age of onset is 21 years. All races and both sexes are equally affected.

Aetiology

- Although genetic factors and environmental factors are both involved in the aetiology of bipolar affective disorder, genetic factors play an especially important role.
- The monoamine hypothesis of depression suggests that mania results from overactivity of monoamine projections.

Clinical features

- The frequency and severity of episodes is very variable, as is the proportion of manic to depressive episodes.

Continued...

Suicide and deliberate self-harm

6

Key learning objectives

- The definitions of the terms 'deliberate self-harm', 'parasuicide', 'attempted suicide', and 'suicide'
- Sociodemographic and clinical risk factors for suicide
- Assessment and management of suicidal risk

Introduction

And so it was I entered the broken world
To trace the visionary company of love, its voice
An instant in the wind (I know not whither hurled)
But not for long to hold each desperate choice.
Hart Crane (1899–1932), quoted from *The Broken Tower*

This poem was written not long before the poet committed suicide by jumping from the steamship SS Orizaba into the Gulf of Mexico.

Suicide is a neologism coined from *sui caedes*, Latin for 'murder of oneself', and has been defined by the sociologist Emile Durkheim as applying to 'all cases of death resulting directly or indirectly from a positive or negative act of the victim himself, which he knows will produce this result'. Suicide can simply be defined as the act of intentionally killing oneself, although sometimes intentionally killing oneself with the primary aim of saving or helping others is seen as self-sacrifice rather than as suicide. Thus suicide might best also be defined as the act of intentionally killing oneself, **with the primary aim of dying**. In some acts of suicide, the primary aim is not entirely clear. For example, in 1941 Virginia Woolf killed herself by walking into the River Ouse with a large rock in her pocket. In her suicide note to her husband she mentions both that her manic depressive illness is deteriorating *and* that she is spoiling her husband's life (see p. 104).

Suicide should also be distinguished from assisted suicide, the making available to a person of the means to end his or her life; and from voluntary euthanasia, the deliberate ending of the life of another person who has requested it and is physically unable to commit suicide. The act of suicide itself should be distinguished from other forms of self-harm, and particularly from attempted suicide and parasuicide. Attempted suicide is the act of intentionally trying to kill oneself but failing to do so, and parasuicide is an act that looks like suicide but that does not result in death. The intention of parasuicide may have been to kill oneself, but this is not necessarily so – it may have been a means of attracting attention, a 'cry for help', an act of revenge, or an expression of despair. Suicide, attempted suicide and parasuicide are all forms of deliberate self-harm, which is the act of intentionally injuring oneself, irrespective of the actual degree of injury sustained.

In summary then:

- **Suicide** is the act of intentionally killing oneself with the primary aim of dying

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