

7 Depression

Robert Baldwin

*Classification • Epidemiology • Clinical presentation • Assessment •
Differential diagnosis • Aetiology • Management • Prognosis •
Conclusion*

Functional disorders are nowadays seen almost as often, in many old age psychiatric services, as dementia. Depression is a common, sometimes the most common, reason for referral.

Classification

'Depressive illness' is synonymous with depressive episode in ICD-10 (World Health Organization, 1993) (Box 7.1) and major depressive episode in DSM-IV (American Psychiatric Association, 1994).

Epidemiology

The prevalence of depressive symptoms far exceeds that of depressive illness. Classic research from Newcastle (Kay *et al*, 1964) found a prevalence of 10% for depression in community residents, although less than 2% met criteria for what would now correspond to depressive illness. In a cross-national comparison, Gurland *et al* (1983) found levels of 'pervasive depression' in 13% of New York elderly residents and 12% of Londoners. Point (past month) prevalence of manic depressive or depressive disorder, akin to depressive illness, was found to be 1.3 and 2.5%, respectively. The rates are double this in general practice attenders, and considerably higher in hospital settings (Jackson & Baldwin, 1993).

Clinical presentation

Clinical features normally regarded as more common in elderly depressed patients include: neuro-vegetative symptomatology, somatic preoccupation, agitation, forgetfulness and delusions. However, these stereotypic descriptions of depression in later life derive, in the main, from the study of in-patients and may reflect illness severity. Blazer *et al* (1986), using an approach which controlled for symptoms that may increase fortuitously with age, found:

Box 7.1 ICD-10 classification of depression (World Health Organization, 1993)

Mild depressive episode (at least two from (b) and at least four from (c)). Moderate depressive episode: at least six of the symptoms under (c). Severe depressive episode: all three from section (b) and at least five from section (c) (at least eight symptoms in total)

- (a) The syndrome of depression must be present for at least two weeks, no history of mania, and not attributable to organic disease or psychoactive substance
- (b) At least two of the following three symptoms must be present:
 - (i) Depressed mood to a degree that is definitely abnormal for the individual, present for most of the day and almost every day, largely uninfluenced by circumstances, and sustained for at least two weeks
 - (ii) Loss of interest or pleasure in activities that are normally pleasurable
 - (iii) Decreased energy or increased fatigability
- (c) An additional symptom or symptoms from the following (at least four):
 - (i) Loss of confidence or self-esteem
 - (ii) Unreasonable feelings of self-reproach or excessive and inappropriate guilt
 - (iii) Recurrent thoughts of death or suicide; suicidal behaviour.
 - (iv) Complaints or evidence of diminished ability to think or concentrate, such as indecisiveness or vacillation;
 - (v) Change in psychomotor activity, with agitation or retardation (either subjective or objective)
 - (vi) Sleep disturbance of any type
 - (vii) Change in appetite (decrease or increase) with corresponding weight change

- (a) Older depressed subjects reported more somatic symptoms.
- (b) Older people had more thoughts about death and had a greater preoccupation with the wish to die than younger people, although not significantly so.
- (c) Surprisingly, it was the younger rather than the older people with depression who reported more memory problems.
- (d) There was no support for the notion of masked depression in the elderly.

More recently, Musetti *et al* (1989) could find no symptoms which reliably differentiated older community dwelling patients with major depression from younger ones. Against expectations, the group over 65

years, exhibited more retardation than agitation. In terms of clinical presentation, the adage 'depression is depression at any age' has some truth to it. However, the pathoplastic influences of age and ageing alter the presentation of depression among the elderly. The main influences are shown in Box 7.2.

Coexistence with physical illness

A common difficulty arises from the coexistence of lowered mood and physical illness. For example, symptoms such as weight loss, fatigue and insomnia may overlap in someone with rheumatoid arthritis. Diagnostic sensitivity is increased if attention is paid to the following.

- (a) The history may indicate a change in symptoms at a time of static physical health.
- (b) Appropriate questions must be asked, e.g. 'Do you feel tired even when at rest?', not 'Have you no energy?'
- (c) Particular care must be directed towards uncovering anhedonia and depressive ideation (self-deprecation, guilt, etc.).

Complaining of depression

In the elderly a complaint of depression may be denied or minimised (Georgotas, 1983). In fact, DSM-IV recognises this and allows anhedonia, instead of low mood, for a diagnosis of major depression. Depression has only recently been an acceptable reason for consulting a doctor. Instead, elderly people more frequently develop excessive physical preoccupation in the form of hypochondriasis (Gurland, 1976). There may be an actual pain for which no organic cause can be uncovered or, more commonly, disproportionate complaining about known organic pathology.

Box 7.2 Factors which may alter or obscure presentation

Overlap of physical and somatic psychiatric symptoms
 Reduced expression of sadness
 Somatisation or disproportionate complaints associated with physical disorder
 Neurotic symptoms of recent onset
 Medically 'trivial' deliberate self-harm
 Pseudodementia
 Depression superimposed upon dementia
 Behavioural disorder
 Accentuation of abnormal personality traits
 Late-onset alcohol dependency syndrome

Other neuroses

Neuroses such as obsessive–compulsive disorder, hypochondriasis and hysteria are rare as primary disorders among the elderly. When neurotic symptoms occur for the first time in an older person they are usually secondary (sometimes referred to as ‘symptomatic’) with depression being the most common underlying cause. Likewise, anxiety symptoms may dominate the clinical picture with the danger that an underlying depression is missed.

Deliberate self-harm

Perhaps the most serious error is to dismiss an act of deliberate self-harm in an older person because in medical terms it was ‘trivial’. Likewise, depressive illness is a very frequent accompaniment of any act of deliberate self-harm. Any older individual who deliberately harms themselves should always have a full psychiatric assessment.

Other behaviours

These behaviours may indicate an underlying depressive illness:

- (a) Behavioural disorder: the sudden occurrence of food refusal, aggression or inappropriate micturition, usually in a residential facility and within a context of resented dependency.
- (b) Shoplifting.
- (c) Alcohol dependence arising for the first time in later life.
- (d) An accentuation of premorbid personality traits: such individuals are often theatrical and importuning, and a depressive illness may easily be overlooked.

In these instances a thorough history from the patient and an informant often clears up uncertainty. Evidence of recent change is crucial and a comprehensive past personal and family psychiatric history must be obtained, along with a full drug and alcohol history. Depressive cognition may have been noticed by relatives, e.g. ‘I wish the Lord would take me’.

Pseudodementia

Another important, if controversial, presentation is depressive pseudodementia. The term is unsatisfactory. Other terminology such as ‘the dementia of depression’ (Pearlson *et al*, 1989) has been proposed, but pseudodementia seems here to stay. Perhaps it is best used as a reminder that severe depression can mimic dementia. The term has been used in at least three ways:

- (a) Depressed patients who perform poorly on a bedside screening test for dementia. Since dementia is defined clinically and not on the basis of a test result, this use should be discouraged.
- (b) Depressed, usually elderly patients who have cognitive impairment which does not meet the diagnostic criteria for dementia. These patients have a general impairment in effortful tasks, especially those concerned with memory, rather than an impairment in cortical function (language, praxis, construction) (Weingartner *et al*, 1981). After reviewing the literature, Reynolds *et al* (1988a) suggest a prevalence of between 10 and 20% of depressed patients. There is evidence that these cognitive deficits arise from subcortical dysfunction, suggesting that depressive illness may cause a subcortical dementia. However, since most elderly patients with depressive illness have some degree of cognitive impairment the term pseudodementia used this way may be rather meaningless.
- (c) To define a characteristic, if dramatic, presentation of dementia. Post (1982) provides a vivid account. The patient appears perplexed and may be inaccessible at interview, although non-verbally may convey considerable distress. The history is months at most and poor memory can often be dated accurately, unlike the insidious onset and course of dementia. Patients with dementia try their best but are inaccurate and are often unaware of their errors when asked questions concerning cognitive function. Pseudodementia patients become irritable and complain vociferously of bad memory. Such patients are usually described as giving 'don't know' answers. Post makes the point that the term 'pseudodementia' should really be reserved for those patients who can be tested, rather than those who are inaccessible and not really testable.

Assessment

History

A detailed history is required from the patient and an informant. Evidence suggesting recent change is very important and areas such as a family and personal history of depression, and a full drug and alcohol history must be covered. Treatments and responses in previous depressive episodes should be clarified. Information about major adverse life events, for example bereavement and other losses such as ill-health, should be documented, along with previous capacity to cope with stressful situations and difficulties. This covers aspects of personality. Personality traits may be difficult to assess but they are important in setting a realistic target to therapy. For example, an individual with long-standing dysthymia may become severely depressed and require treatment, but restitution to a former state of relative gloom may be more realistic than attempting to

help the patient attain perfect happiness. Availability and the quality of support from family, friends, neighbours, and statutory and voluntary sectors are important, as they may influence prognosis.

Mental state examination

Care must be taken not to overlook suicidal ideation and delusional phenomena. Evidence of cognitive impairment should be carefully documented so that comparisons can be made after recovery. Routine neuropsychological testing is not usually warranted unless dementia is suspected, and even then the findings are likely to be equivocal. However, it is useful to include a screening measure such as the Mini-Mental State Examination (Folstein *et al*, 1975) (see Chapter 1).

Physical examination

A physical examination is important because of the close association between physical illness and depression (Box 7.3). As well as possibly causing depression, physical illness is a poor prognostic factor. Cases of diagnostic difficulty usually need admission to hospital for skilled psychiatric nursing observation.

Investigations

Laboratory investigations include haemoglobin and red blood cell indices which may indicate B12 deficiency or excessive alcohol intake. B12 estimation should be undertaken in a first episode. Folate should be measured if severe depression has been present for some weeks, as a state of under-nutrition may have developed. For similar reasons urea and electrolytes are important. A low serum potassium may delay electroconvulsive therapy (ECT). An elevated calcium is occasionally associated with depression, in primary hyperparathyroidism and metastatic

Box 7.3 Causes of organic ('symptomatic' or 'secondary') depression

Occult carcinoma: lung, pancreas

Metabolic or endocrine: hypothyroidism, hypercalcaemia, Cushing's disease

Drugs: steroids, beta-blockers, methyl dopa, clonidine, nifedipine, digoxin, L-dopa, tetrabenazine

Infections: post-viral, myalgic encephalomyelitis, brucellosis, neurosyphilis

Organic brain disease: space occupying lesion, dementia

cancer. Thyroid function tests may reveal hypothyroidism or an 'apathetic hyperthyroidism', both of which can be mistaken for depression. The main clinical use of an electroencephalogram (EEG) is to help differentiate depression from an organic brain syndrome. Abnormal investigations may be responsible for depression, or the result of depression (Table 7.1).

Imaging

Imaging is not routinely carried out in depression in the elderly, but only if indicated by other factors such as a rapid onset or the presence of neurological symptoms or signs. However researchers are looking at ways in which the newer imaging techniques may be clinically useful in diagnosis or treatment. For example, O'Brien *et al* (1994) have found that measures of temporal lobe atrophy on magnetic resonance imaging discriminate between depression and Alzheimer's disease (see Chapter 12).

Biological markers

More contentious is the role of markers of depressive illness. The best known of these is the dexamethasone suppression test (DST). The rationale is that there is hyperactivity of the hypothalamic-pituitary-adrenal axis in depression, which leads to a failure to suppress cortisol after ingestion of dexamethasone. Following initial encouraging reports (Carroll *et al*, 1981) a deluge of publications ensued, all enthusiastically supporting the DST as a 'diagnostic tool' for major depression, only to be followed by doubt

Table 7.1 Investigations

	First episode	Recurrence
Full blood count	Yes	Yes
Urea and electrolytes	Yes	Yes
Calcium	Yes	If symptoms indicate
Thyroid function	Yes	If symptoms indicate, or > 12 months previously
B12	Yes	If symptoms indicate, or > 2 years previously
Folate	Yes	If indicated by nutritional state
Liver function	Yes	If indicated (e.g. alcohol intake)
Syphilitic serology	Yes; if atypical presentation	If not already done
CT (brain)	If clinically indicated	If neurologically indicated
Electroencephalogram	If clinically indicated	If neurologically indicated

and uncertainty. Clearly the DST is less specific for depressive illness than was first thought. For old age psychiatry the situation is even less promising, as a consensus seems to be emerging that DST non-suppression gradually increases with age, particularly after 75 years. Furthermore the DST does not appear to differentiate between dementia and depression (Spar & Gerner, 1982).

Assessment schedules

Another approach to diagnosis concerns schedules. These are useful in improving detection in areas such as residential and nursing home facilities, where the prevalence of depression is known to be high. The Geriatric Depression Scale (GDS) which is available in either a 30-item and a 15-item version (Yesavage, 1988) is probably the most widely used.

Differential diagnosis

ICD-10

The main differential diagnoses in ICD-10 are:

- (a) organic depressive disorder, the aetiology of which may be systemic or cerebral;
- (b) dysthymia, a form of chronic 'low grade' depression;
- (c) delusional disorder, in which the judgement is whether delusional symptoms are mood-congruent, in which case psychotic depression is most likely; and
- (d) adjustment disorder, with either a brief (one to six months) or prolonged (more than six months) depressive reaction, adjustment disorder is sometimes secondary to a bereavement in older people.

Bereavement

Some of the symptoms of bereavement overlap with those of depressive illness. Certain features point more clearly towards depressive illness and to a consideration of antidepressants:

- (a) progress over the first few months only to slip back for no apparent reason;
- (b) suicidal thoughts;
- (c) pervasive guilt (not merely remorse over what more might have been done to prevent death);
- (d) disability; and
- (e) 'mummification', maintaining grief by keeping everything unchanged.

Aetiology

Predisposing factors

Genetic

The genetic contribution to depressive illness decreases with age. Hopkinson (1964) reported that the risk to first degree relatives of probands with depressive illness was 20% in early onset but only 8% in late onset. Others have confirmed this difference (Mendelwicz, 1976).

Gender

Depression in all age groups is more common in women and this is true in later life. The ratio is approximately seven to three, female to male. Some studies have reported higher rates of depression in those widowed or divorced.

Neurobiology

Amine changes may predispose to depression, or be altered as a consequence of it. Perhaps not surprisingly, no clear consensus has emerged in respect of the amine theory in later life depression. However, some biological changes associated with ageing are similar to those seen in depression. Both ageing and depression are associated with decreased brain concentrations of serotonin, dopamine, noradrenaline and their metabolites, and increased MAO-B activity (Veith & Raskind, 1988).

Neuroendocrinology

A variety of neuroendocrine changes are associated with ageing (Veith & Raskind, 1988). However, the site of action is not clear and may involve changes at cortical, limbic, hypothalamic or pituitary level. Depression in all age groups is associated with hyperactivity and dysregulation of the hypothalamic-pituitary-adrenal axis. The TSH response to thyroid-releasing hormone is less age dependent than cortisol non-suppression, but is not specific for depression, and there has been very little research on depressed subjects in later life. Schneider (1992) concluded that none of the neurochemical markers or neuroendocrine challenge tests is sufficiently sensitive or specific to be of clinical use in depression of old age.

Electroencephalogram

Variables measured during sleep show similar changes in depression and ageing (Veith & Raskind, 1988). These include night time wakefulness and decreases in slow wave sleep, total rapid eye movement sleep and rapid eye movement latency. Using discriminant function analysis,

Reynolds *et al* (1988b) found that four EEG sleep variables correctly differentiated depressed elderly patients from those with dementia in 80% of cases.

Structural brain changes

Over a quarter of a century ago, Felix Post (1968) wrote “subtle cerebral changes may make ageing persons increasingly liable to affective disturbance”. Recently several factors have renewed interest in this hypothesis:

- (a) The attenuation of genetic risk, an important factor in ‘younger’ depression, ought to lead to a reduced prevalence of depression in later life. Since this has not been demonstrated, other factors such as subtle brain damage may be active.
- (b) Biological and clinical markers have been described which may distinguish early from late-onset depressions. These include a lower sedation threshold to barbiturates, both before and after treatment; latency in auditory cortical evoked responses, midway between patients with dementia and controls; and a higher than expected rate of death from vascular causes (Murphy *et al*, 1988).
- (c) There is growing evidence that structural and functional brain changes occur in depression in old age. Findings include (see Chapter 12): ventricular enlargement on computerised tomography (Jacoby & Levy, 1980); magnetic resonance imaging changes (white matter change of possible vascular aetiology) (reviewed by Baldwin, 1993); single photon emission tomography changes in blood flow (Sackheim *et al*, 1990); and focal abnormalities in cerebral blood flow with positron emission tomography (Bench *et al*, 1992).

Physical health

Diseases, some with occult presentation, may predispose to severe depression (Box 7.3). Some neurological illnesses, such as stroke and idiopathic Parkinson’s disease, may have specific organic links with depressive illness.

Personality

Surprisingly little has been written about this (see Chapter 11). Roth (1955) believed that those with late-onset depressive illness had more robust personalities than those with depression arising earlier in life. Post (1972) noted that severe depression was associated with less premorbid personality dysfunction than milder depression. However, differentiating current illness from premorbid personality is very difficult, and even informants often provide biased accounts. Even so, Bergmann (1978) and Post (1972) note that patients with predominantly neurotic symptom

profiles of depression have often been categorised anxiety-prone individuals. Bergmann (1978), basing his argument on the influential work of Bowlby, suggested that satisfactory attachment behaviour in early life was necessary to adaptively cope with the real threats of old age. Post (1972) noted that obsessional traits were over-represented in his in-patient group. Abrams *et al* (1987) found an association of 'avoidant' and 'dependent' types with late-life depression. Murphy (1982) found that a life-long lack of a capacity for intimacy, a personality variable, seemed to be a risk factor for depression in later life.

Social supports and intimacy

Most of the vulnerability factors for depression, identified by Brown & Harris (1978), are not relevant to the elderly. One potentially relevant one, loss of mother before aged 11, was not confirmed by Murphy (1982) in her study of depressed elderly patients. Murphy's research suggested that a confidant may buffer social losses. The increased realisation in recent years that childhood sexual abuse often has severe and long-lasting damaging effects, including depression, should not be overlooked when treating elderly patients. One woman in her seventies who presented to me with a late-onset depressive illness after a difficult orthopaedic procedure, revealed for the first time (to the female nurses) that she had been sexually abused by her father many years earlier.

Precipitating factors

Life events

As with depression in younger age groups, a recent adverse life event is an important precipitant. Post (1962) reported these in two-thirds of severely depressed in-patients. Murphy (1982) found that 48% of depressed in- and out-patients had experienced threatening or loss events in the preceding year, compared to 23% of controls. In addition, major chronic

Box 7.4 Aetiology

Genetic (reduced)
 Gender (female)
 Neurobiology
 Neuroendocrinology
 Structural brain changes
 Physical health
 Personality
 Social supports

social difficulties (as distinct from abrupt events) lasting at least two years were associated with depression. These findings are comparable to the life event studies of Brown & Harris (1978), except for one difference: in the elderly a recent grave physical illness or a chronically disabling disorder assumed a far more conspicuous role (Murphy, 1982).

Perpetuating factors

Social factors

There is evidence that, on the whole, social factors exert less influence on the course of depressive illness in later life. Nevertheless, it is important to examine the social factors which may influence outcome for the better. Oxman *et al* (1992) examined the effect of social networks and supports on depressive symptoms, in people seen three years apart. Using multiple regression analysis, most variance was explained by disability, but there were important social variables such as loss of spouse, adequacy of emotional support, presence of 'tangible' support and the role of family and confidantes. Although the evidence is meagre and at times contradictory, poor support is likely to be a maintenance factor for depression.

Carers

Hinrichsen & Hernandez (1993) found three factors which associated with poorer outcome at one year from major depression in a cohort of patients with a mean age of just over 70 (see Chapter 18): psychiatric symptoms in the carer, reported difficulties and poorer carer health.

Management

Biological treatments

Antidepressants

How antidepressants are prescribed is perhaps as important as knowing what to give. Some elderly people are somewhat in awe of their doctors. Rather than causing offence, because they do not consider tablets to be the answer to depression, they will discreetly bin their antidepressants or retain them in their bottles or sealed packages. Start with an explanation that depression which warrants treatment with tablets is an illness, and that it is common, treatable and not a sign of moral weakness. Many patients need reassurance that the tablets are not addictive and that depression is not 'senility' or a harbinger of dementia. Involve the patient in an agreed treatment plan. They need to be told, in layman's terms, why they should not expect immediate results. A keyworker from the old age

psychiatric team should coordinate the care and act as a point of contact for the patient and family (for a fuller account see Chapter 18).

Electroconvulsive therapy

ECT remains the most effective treatment for depressive illness at any age, with recovery rates of around 80%. It is the treatment of choice for patients exhibiting profound disability, food or fluid refusal and suicidal behaviour. It is often considered the treatment of choice in delusional depression. It is tolerated well by elderly people (Benbow, 1989). The factors which predict a good response to ECT are the same at all ages and, in general, encompass the symptomatology of severe depression. The elderly, unlike younger patients seem to respond well when anxiety dominates the clinical picture (Benbow, 1989). Memory impairment is measurably worse in older patients given ECT, which is why clinicians often prescribe unilateral electrode placement. However, this may be counterbalanced by arguments that this memory effect may be of negligible clinical relevance, and that bilateral placement may be more effective therapeutically (Benbow, 1989).

All the contraindications to ECT are relative (Box 7.5). It may be given to a patient who is desperately ill with depression, even though their physical health is also poor. A careful physical examination is the most important way to screen for problems (Abramczuk & Rose, 1979), with subsequent investigations (ECG, chest radiograph, pulmonary function testing, etc.) and referral to specialists as appropriate. ECT can be given to patients with a pacemaker provided the patient is insulated and no-one touches him or her during the passage of electricity.

Patients detained under Section 3 of the 1983 Mental Health Act can be given ECT without consent, but a doctor appointed by the Mental Health Act Commission must agree with the decision. Under such circumstances it is worth remembering that the 'standard' six treatments given to younger depressives is probably insufficient for elderly patients (Baldwin & Jolley, 1986). It is reasonable to specify a course of up to 12 treatments when giving ECT under Section 3.

Treatment-resistant depression

When faced with a patient whose depression has not shown worthwhile improvement after six weeks treatment with an adequate dose of antidepressant, the options are as follows (see review, Baldwin, 1996):

- (a) Extend the trial beyond the 'traditional' six-week period, ensuring the dose is optimal, for between three and six more weeks. There is some evidence that this helps up to 50% of patients (Georgotas & McCue, 1989).

Table 7.5 Contraindications to ECT (all relative)

Myocardial infarct within three months
 Stroke within three months
 Markedly compromised respiratory reserve
 Uncontrolled hypertension
 Uncontrolled heart failure
 Predisposition to dysrhythmia
 Aortic or carotid aneurysm
 Traditional monoamine oxidase inhibitor within 10 days

- (b) Change to an antidepressant of another class, a popular strategy for which there is remarkably little convincing evidence.
- (c) Use an augmentation strategy such as lithium.

Lithium augmentation

Flint (1995) reviewed the literature on lithium augmentation and was unable to draw firm conclusions. There have only been four prospective studies of lithium augmentation in elderly patients. Zimmer *et al* (1988) and Flint & Rifat (1994) found response rates of around 20%, much lower than open studies of younger adults. Parker *et al* (1994) compared a group treated with a single antidepressant with a lithium augmented group, in a prospective study. The lithium augmentation group were significantly less depressed at follow-up. However, this study did not use random allocation, or blinded measures, so the results are encouraging but preliminary. There have been no double-blind, placebo-controlled trials of augmentation therapy in elderly patients, to date.

Psychological treatments

Psychotherapy

This has not been as extensively evaluated as drug treatment in the elderly. Jarvik *et al* (1982) treated elderly depressives in either a psychodynamic group, or with group cognitive-behavioural therapy. Both did better than a third placebo group. Thompson *et al* (1987) reported similar results in elderly depressives, who were randomly allocated to behavioural, cognitive or psychodynamic individual therapy (see Chapter 17).

Cognitive-behavioural therapy

Interest in this therapy has accelerated in recent years. Yost *et al* (1986) provide a comprehensive guide for those wishing to work with the elderly.

However, the major role of cognitive-behavioural therapy may be as an adjunct to pharmacotherapy, especially in the acute and continuation phases of depression (Reynolds *et al*, 1992, 1994).

Family therapy

Old age psychiatrists are well aware of the potency of the family to promote or disrupt recovery of an elderly depressed member. Attention to communication, explanation and a consistent approach to management, which excludes no relevant family member, are clearly important. Interventions with families vary in complexity. Blazer (1982) has summarised some of the important roles adopted by family members: others are beginning to modify systems theory in order to give formal family therapy to elderly depressed patients and their families (Benbow *et al*, 1990). The effectiveness of such interventions have yet to be evaluated over usual practice with a multi-disciplinary team.

Marital therapy and bereavement counselling

These are further skills which may be offered with benefit to selected depressed patients, after resolution of the more severe symptoms.

Social interventions

Social aspects of treatments should not be overlooked. For example, poor housing, poverty, high local crime rates and other indices of deprivation are important in the prognosis of depression in old age (Murphy, 1983). Also, evidence that elderly depressed people report a lack of diffuse (as opposed to intimate) contacts in the community (Henderson *et al*, 1986) suggests possible treatment strategies, such as the imaginative use of a day centre.

Prognosis

Although the recovery rate for an individual episode of depression with energetic treatment is good (around 75–80% (Flint & Rifat, 1996)), the longer-term prognosis is poorer. Murphy (1983) applied modern investigational techniques to a cohort of depressed patients and found that only 35% had good outcomes. The short follow-up period (one year) and possible under-treatment (judged by the ECT data offered) might partially explain these gloomy findings. More recent studies have been less pessimistic (Baldwin & Jolley, 1986; Burvill *et al*, 1991). In a review of 10 studies, Cole (1990) calculated that 60% of patients (with a mean follow-up time of 32 months) had either remained well, or had suffered

relapses which had been successfully treated. Of the remaining 40%, between 7 and 10% remain unremittingly depressed, and around a third have been characterised as having 'depressive invalidism'; in other words, some recovery but residual symptoms, such as anxiety and hypochondriasis.

Outcome compared to younger patients

Depressive illness is prone to relapse, recurrence and chronicity irrespective of age. However, several studies have shown a better outcome in elderly depressed patients than younger ones (Murphy, 1983; Baldwin & Jolley, 1986; Meats *et al*, 1991). Other studies have found outcome to be at least as good (Hinrichsen, 1993; Hughes *et al*, 1993; Reynolds *et al*, 1994; Alexopoulos *et al*, 1996). This impressive weight of evidence is clearly contrary to the received wisdom, still evident in some textbooks.

Mortality

Another outcome is death. A number of studies point to a higher than expected death rate, in the short term, for elderly depressives (Murphy, 1983; Rabins *et al*, 1985; Burvill *et al*, 1991; Kivela *et al*, 1991). However, in the longer term the death rate for older depressives may not be significantly higher than the base population (Robinson, 1989). Whereas Ciompi (1969) reported an excess of deaths due to suicide, recent studies have not confirmed this, either in the medium term (Murphy *et al*, 1988) or the long term (Robinson, 1989). The obvious answer, that the excess is accounted for by greater physical morbidity in the depressed group, is also probably incorrect. Murphy *et al* (1988) controlled for physical illness severity but still found an excess of deaths in the depressed group compared to non-depressed controls, especially from cardiovascular causes.

Cognitive impairment

It is uncertain whether depression predisposes to dementia. Sometimes depression is a harbinger of what, with the passage of time, is clearly dementia. Several other neurodegenerative disorders may present in this way. There is evidence that depressed patients who present with pseudodementia have an increased risk of developing true dementia. For example, an often quoted study is Kral & Emery (1989), who reported that 89% of patients previously diagnosed as having pseudodementia went on to develop unequivocal dementia. Alexopoulos *et al* (1993) found the same. Abas *et al* (1990) noted that despite successful treatment of mood, the cognitive impairment that they had detected (which did not amount to dementia) persisted in around a third of those patients who had it at

the outset. On the other hand, long-term naturalistic studies have not confirmed these findings. It seems that when a depressed patient presents with marked confusion, this may herald dementia, so careful follow-up is required. In general, however, depression in old age is not a risk factor for dementia.

Suicide

Elderly people are over-represented in the suicide statistics and the relationship between depression and suicide is particularly strong. Barraclough (1971) found that nearly 90% of successful suicides had clear-cut evidence of depression. Worryingly, 90% had been seen by their general practitioners within the previous three months, half within the preceding week. Furthermore, unlike parasuicide in young people, approximately two-thirds of elderly attempters have a psychiatric disorder. Manipulative overdoses are uncommon in elderly attempters.

There are three interrelated risk factors for suicide:

- (a) Psychiatric disorder – most closely associated with severe depression. The clinical picture is of agitation, guilt, hopelessness, hypochondriasis and insomnia and, contrary to expectation, the illness is often a first-onset, non-psychotic depression of only moderate severity.
- (b) Chronic – often painful, physical health problems.
- (c) Social isolation – however, many more elderly people live alone than ever attempt suicide. It may be then that better social conditions serve to reduce a vulnerability to suicide rather than removing a cause of it (Lindesay, 1986).

Predictors of poor outcome

General factors associated with poorer outcomes (Box 7.6) are:

- (a) Initial and supervening serious physical illness, presenting acutely or chronically disabling (Murphy, 1983; Baldwin & Jolley, 1986).
- (b) Cerebral pathology, either coarse (Post, 1962) or subtle (Jacoby *et al*, 1981). Baldwin *et al* (1993) failed to replicate Post's findings, suggesting that modern antidepressants may be more effective in depression associated with cerebral organic disorder.

Predictors of good outcome

Adequate treatment

Late-life depression is undertreated in primary care settings (McDonald, 1986), and the more sparing use of physical treatments in specialist settings is associated with poorer prognoses (Baldwin, 1991). There is a large

Box 7.6 Factors associated with a poor prognosis

Presence of cerebral organic pathology
 Preceding severe physical health problems
 Supervening health events
 Slower recovery
 More severe initial depression
 Duration of symptoms of more than two years
 Two or more previous episodes in the last two years or three
 in five years

variation in the use of antidepressant drugs, lithium and ECT in naturalistic studies (Baldwin, 1991).

Planned after-care

Relapse is most likely in the first 18 months to two years after an initial episode (Godber *et al*, 1987; Flint, 1992). Since relapses are often undetected without planned after-care (Sadavoy & Reiman-Sheldon, 1983) it makes sense to have a clear plan of follow-up for at least 18 months. After-care can be by a member of a multi-disciplinary old age psychiatric team or the primary care team.

Prophylaxis

The Old Age Depression Interest Group (1993) found that of 219 patients with major depression, 69 recovered sufficiently to enter a two-year, double-blind, placebo-controlled trial of dothiepin. Dothiepin (75 mg daily) reduced their relative risk of relapse by a factor of two and a half.

Prevention

There is a case for indefinite prophylactic treatment of any new episode of major depression in an older patient. An elderly person in his or her 70s may have an expected life span of five years. Preventing six months morbidity due to the recurrence of a depressive illness is an important gain. However, blanket prophylaxis risks over-treatment. Furthermore there are difficulties with the long-term use of tricyclics, notably weight gain and dental decay. Clearly studies are needed which look at the efficacy of selective serotonin reuptake inhibitors, and other new antidepressants, in the prevention of relapse and recurrence of depression in older patients. Lithium carbonate should be considered for those who have had either two episodes of depression in the last two years, or three in the past five

years (Abou-Saleh & Coppen, 1983). To date there are no controlled evaluations of maintenance ECT in the prevention of relapse.

Drugs are not the only form of prophylaxis. Ong *et al* (1987) demonstrated that a simple weekly support group for recovered depressives significantly reduced both relapses and readmissions. The emphasis in prevention is increasingly on the combined use of antidepressant drugs and psychological interventions such as interpersonal psychotherapy. The early evidence is that this approach is as effective in older patients as younger ones (Reynolds *et al*, 1994).

Conclusion

Advances have been made in the aetiology and management of depression in the elderly. In spite of this, most depression remains undetected and untreated. Health professionals tend to see the older people who are most susceptible to depression, including the physically unwell or those living in residential accommodation. In these circumstances it may become tempting to consider depression as an inevitable part of ageing, and that little can be done. This is not the case.

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