CHAPTER 8

Depression in physically ill older patients

GRAHAM MULLEY

Introduction

Depression is the commonest mental illness in old age. In 2001–02, there were 9.8 million visits by older people to office-based physicians in the USA for depression.[1] In the UK, one in six older medical inpatients are depressed (Geriatric Depression Score – GDS – of 7 or more out of 15).[2] The mean prevalence in over sixty-fives living at home is about 12%. In the UK, the National Service Framework (Standard 7) for Older People recognised that 10–15% of people over 65 have depression. It recommended that primary care trusts should ensure that every general practice uses an agreed protocol to diagnose, treat and care for patients with depression.

Although there are few community studies large enough to tell us the precise incidence in old age, it is apparent that the incidence differs markedly in different cities and countries. High rates of depression are found in Amsterdam, Berlin, London, Munich and Verona. Lower rates occur in Dublin, Liverpool, Iceland and Zaragoza.[3] Depression is commoner in women and Asians, and less prevalent in those with a sense of spirituality. It does not increase in prevalence with age, though its severity and associated suicide risk may worsen with advancing years.

The causes are heterogeneous: there are probably interactions between susceptibility (gene products) and a precipitant (in the external environment).[4] Social factors such as loss of status, wealth, intimacy and life opportunities can be important. There may also be developmental, psychological and biological factors. It can be difficult to recognise the disease, particularly if the patient has medical comorbidity. The biological signposts to the disease are less helpful than in younger,

healthier people: disturbances of sleep, appetite, weight, libido, and bowel motility can have a variety of physical causes. The non-biological features are therefore usually more helpful: low mood, lethargy, anxiety, irritability, hypochondriasis, regression, crying and pessimism. Geriatricians miss the diagnosis in a half of patients admitted to an acute geriatric unit (in one study, the consultant in old age psychiatry diagnosed depression in 67 out of 155 consecutive admissions, the geriatrician 29 of them).[5]

There is no sharp demarcation between a 'case' and a 'non-case'. Depression is a continuum, ranging from feeling sad to experiencing major depression. The word 'depression' is used for the understandable demoralisation in the face of adversity; grief; persistently negative thoughts and the full-blown clinical syndrome of depression.

Depression can be reversed by treatment (though some cases of severe depression do not always respond so well). However, many people with the disease may receive no treatment. In others, the therapy may be delayed and adherence to the drug regimen may be poor.

The effects of depression are legion: it can cause disability and handicap; increase mortality, exacerbate coexisting illnesses, place a burden on the family and causes much unnecessary suffering. It has a huge healthcare cost, with readmissions to hospital, prolonged lengths of stay and an overuse of services.

In this chapter, I will focus on loss of health and depression.

Why is depression important in sick old people?

Depression that warrants intervention occurs in 10% of people over the age of 60. Nearly a third of older people admitted to hospital for acute care experience depression. The lifetime experience of depression is 15–20%. Unipolar depression is the fourth leading cause of temporary and permanent disability worldwide.[4]

Physical illness can cause or be the result of depression. Depression worsens the prognosis of many medical disorders. Those with depression have a two-fold increase in cardiac mortality. It is a risk factor for non-suicidal mortality. Suicide in old age is associated with multiple physical illnesses and a history of hospital-treated depression.[6] Yet many people do not receive therapies that are of proven effectiveness – even up to eight years after the onset of their condition.

The relationship between depression and physical illness

The hospital and care home environment

Most hospitals were not designed with the welfare of older patients in mind. Hospital routines are not always conducive to well-being and few older patients after discharge wished that they had spent more time in hospital. Interviews of hospitalised patients in Australia revealed that all of them felt depressed. They were demoralised, feeling unable to cope. They disliked having to rely on others

and felt that they were a burden. They perceived themselves as being helpless and hopeless, with a low sense of self-esteem.[7]

Many residents in residential and nursing homes are depressed. The diagnosis is underestimated and few residents are referred for specialist psychiatric care.

A high prevalence of suicidal ideation occurs in medically ill nursing home residents – particularly those with severe depression, medical comorbidity and little social support.[8]

Cardiovascular disease

There is a semantic association between grief and a broken heart. In some cases, depression precedes the clinical manifestations of cardiovascular disease. Major depression can predict first cardiac events.[9]

Depression is independently associated with coronary heart disease (as are social isolation and lack of social support).[10] The increased mortality seems to be related to the *severity* rather than the *presence* of depression – in a study of 763 patients who had sustained a myocardial infarction, depression *per se* was not an indicator of death. There was a higher death rate on those with more marked depression who had non-Q wave myocardial infarction.[11]

In heart failure, depression is related not only to the degree of physical impairment but also to how the individual copes with the impairment.[12] Koenig *et al.*[13] found that less than 50% of patients with heart failure who had major depression had it treated and few had psychiatric consultations.

Conversely, cardiovascular disease is a risk factor for late-onset depression. The mechanisms whereby depression and heart disease affect each other are uncertain. Part of the reason may be that depressed subjects tend to have poor concordance with their drug therapy – not surprisingly, health status is worse if people do not take their drugs.[14] Altered autonomic function may also explain the relationship between depression and cardiac mortality.[15] In depression, heart rate recovery after exercise is slower. Heart rate variability (a measure of autonomic function) is less in those with major depression than controls. An acutely depressed mood can trigger life-threatening cardiac events. It is speculated that biological responses involving inflammatory cytokines and platelet activation may be contributory.[16]

Chronic obstructive airways disease (COPD)

Thirty per cent of people with COPD are depressed. Depression is also a factor in the frequency of hospital readmissions for acute exacerbations.[17] Risk factors for depression in COPD include disease severity, lack of carer support, inadequate pulmonary rehabilitation, being underweight, use of psychotropic drugs and being a smoker. In COPD, depression and anxiety are significantly related to negative quality of life outcomes.

Their depressive symptoms improve if inpatient rehabilitation results in symptom relief and reduced disability.[18]

Depression is also common during recovery from acute respiratory failure.

Stroke

Stroke is the major cause of serious disability and its incidence increases exponentially with age. Depression is common, with about 50% of survivors being depressed at six months – especially those with poor mobility or limitations in activities of daily living. A history of depression is also associated with low mood after stroke. In a pan-European study of 626 non-disabled older people,[19] depressive symptoms were associated with white matter ischaemia on CT scan. The presence of lacunes (small, deep infarcts) was not associated with the development of depression.

After stroke, there is usually a reduction in social interaction over time.[20] Not surprisingly, the GDS score rises between the third and six month post-stroke.

Some stroke patients have difficulty controlling the expression of their emotions (emotionalism). They may have a low threshold for crying or laughing. The usual triggers are sadness, sentimentality and discussion of this symptom. Some people cry for no obvious reason. Emotionalism can distress the patient, who may feel that they are losing control. It can also be disturbing for the family, who might worry that their loved one is being badly treated in the hospital or care home. It is therefore wise to explain emotionalism to the patient and the family. Antidepressants can considerably reduce the frequency of crying or laughing episodes.[21] The improvement is not specific to a particular type of drug.[22]

Communication difficulties as a result of stroke are the strongest predictor of the severity of depression and its prognosis. The presence of depression can impede stroke rehabilitation.[23]

Depression in stroke is often undiagnosed and inadequately treated. A study of seven trials with 780 participants[22] found that antidepressants are associated with a reduction in scores on mood-rating scales. However, there was no reduction in the frequency of clinically diagnosable depression. Treating non-depressed stroke patients with sertraline in the first two weeks does not reduce the incidence of depression at 24 weeks.[24] Two small trials of psychotherapy showed no benefit. Physical exercise may improve post-stroke depression: a three-month programme was associated with less depression and an improved quality of life.[25]

There have been no studies of ECT in patients with stroke.

Cancer

People will be understandably depressed on being told that they have cancer. Depression can be the presenting feature of occult cancers – especially pancreatic cancer. Breast cancer and gastrointestinal malignancies are particularly likely to be associated with depression. Depression is also a manifestation of non-metastatic malignancy.[26] Patients with cancer who are also depressed have a significantly increased risk of death.[27] Some cancer patients date their depressive symptoms to the onset of treatment with chemotherapy or deep X-ray treatment.

Bone and joint disease

Antidepressants double the risk of falls and people who are depressed are more likely to break their hips. Almost a half of patients with a range of traumatic orthopaedic conditions are depressed. Open fractures can increase the rate of depression.[28] Patients with rheumatoid arthritis may be depressed because of the physical impact of the disease (pain, disability) or such psychological factors as low self-esteem and perceived impact of the disease.[29]

Neurodegenerative disease

Old people who are depressed are more likely to develop Alzheimer's disease, vascular dementia and Parkinson's disease. Depression may also be associated with mild cognitive impairment: this is independent of underlying cerebrovascular disease.[30]

Dementia may be accompanied by depression but it is not known whether drug treatment is effective or whether the balance of benefits versus adverse effects is favourable. The clinician should therefore be cautious in prescribing antidepressants to patients who are demented.[31]

Recurrent depression is a particular risk factor for dementia. Depression can mimic dementia, but depressive pseudo-dementia is not so common nowadays. Depression may be secondary to dementia – particularly in the early stages, when the patient has insight into their cognitive decline. Of course, depression and dementia may coexist – they are both common conditions.

Depression is also common in Parkinson's disease. Apathy may be a core feature of this illness and may occur in the absence of depression. Because of symptom overlap, there may be diagnostic difficulties.[32] There are no studies of ECT or behavioural therapy in Parkinson's disease. There are insufficient data from the few trials to help us make informed choices on whether, when or with which drug to treat depression in Parkinsonism.[33]

Endocrine disorders

Depression in diabetes mellitus is three times more common than in the general population. Those with diabetes may forget to log their blood glucose measurements, not manage their disease well and not attend for clinic appointments. Only a third of those with diabetes mellitus have their major depression identified and treated, partly because some of the somatic symptoms of depression (sleep disturbances, weight gain or loss, fatigue) may be attributed solely to the diabetes.

Depression occurs in most people with Cushing's disease. It also should be considered in hypothyroidism and hyper-parathyroidism.

Sensory impairment

Depression is likely in old people who are deaf or visually impaired. In age-related macular degeneration (the commonest cause of blindness in old people in Western

countries, where it affects 30% of those over 75) is associated with depression – especially in those with impaired physical function.[34]

Other disorders

Depression can occur in a range of clinical disorders. Depression is common in old people with genito-urinary diseases and injuries that require admission to hospital.[6] Elderly Chinese men with moderate-to-severe lower urinary tract symptoms are at increased risk of having clinically relevant depression.[35] Depression is associated with impaired immune activity and altered inflammatory response. Depression can therefore be instrumental in causing physical illness or result from it. It should be considered in any older person, but especially those with disability, pain, chronic disease as well as those who do not take their drugs regularly or fail to attend for clinic appointments.

Why is depression under-recognised in medically ill old people?

Patients

Old people tend to under-report or deny depression. This may be a cohort effect of a generation of stoical individuals who often minimise their symptoms. The old person may attribute constipation, weight loss and chronic aches and pains to normal ageing. The dominant symptoms might be anxiety or irritability and they may not feel particularly sad. There may be inadequate privacy on the ward or at the clinic consultation. The patient may fear the stigma that still accompanies psychiatric disease. Or they simply may be unaware that they are depressed. The diagnosis may be difficult if the patient is deaf, dysarthric or dysphasic, or if their first language is not English. The general public has a poor understanding of antidepressant drugs, confusing them with tranquillisers and regarding them as addictive.

The family

Relatives may be unaware that their loved one is depressed. If the depression is associated with memory impairment, they may fear that the person is becoming demented. Depression may also be overlooked in informal caregivers, especially those who are under strain. Common causes of caregiver stress include sleep disturbance, faecal soiling and behavioural disorders.

How to improve the recognition of depression in medically ill patients

We should consider the possibility of depression in the following circumstances:

- sadness – this is the commonest presentation
- poor physical health
- weight loss
- alcohol abuse
- not eating properly or refusal to eat

- living in squalor
- poverty
- isolation
- poor concordance with medication
- not exercising
- not rehabilitating successfully
- living in a care home
- living at home with chronic illness or bereavement
- not attending for medical appointments.

Conversely, frequent attendances at the general practitioner's surgery (after controlling for physical illness, depression was found in those who attended the primary care physician more than once a month for the past six months).[36]

Not attending for influenza vaccination – this has been seen in depressed caregivers who are looking after an elderly person with dementia.[37]

Physical signs that should raise the possibility of depression include:

1. a slowed-up patient
2. flattening of affect
3. lack of eye contact
4. taciturnity
5. unkempt clothing or poor personal hygiene
6. an aura of heaviness or gloom.

Behavioural features that should alert the physician to the diagnosis include:

- agoraphobia
- anhedonia – little relish for life, not enjoying things that once gave pleasure
- anxiety or being worried for no obvious reason
- bad moods
- dark thoughts
- crying
- importuning
- being argumentative
- being suspicious about others, including friends and family
- complaining of feeling tired all the time
- expressing repeated concerns about death and dying
- feeling worthless or hopeless
- fearing that they have an incurable disease.

We should also consider depression if the patient has several volumes of case notes. Some patients have somatisation, or their symptoms may not be readily diagnosable and these patients can present to different specialists. They may have undergone numerous investigations before the possibility of depressive illness is considered.

As depression is so common and may present in so many ways, it is wise to administer screening tests as a routine part of the assessment of old people.

Which tests for depression?

There is a bewildering array of tests, reflecting the fact that there is no ideal instrument for measuring depression. Some screen for the presence, others assess the degree of depression. The busy physician wants assessment tools which are easy and quick to administer, do not upset the patient and are valid.

The Geriatric Depression Scale (GDS) is probably the most useful in older patients. In patients referred to a specialist geriatric outpatient memory clinic, an abbreviated form consisting of five questions has been found to rapidly identify those with depression (positive predictive value of 97%).[38]

The questions are as follows.

- Do you often feel downhearted or sad (blue)?
- Do you often feel helpless?
- Do you feel that your life is empty?
- Do you feel happy most of the time?
- Are you basically satisfied with your life?

The key questions to pose to all elderly patients are therefore on mood and loss of interest or pleasure in activities.

Investigating depression

It is important to consider underlying medical conditions which are associated with depression as the treatment may improve the patient's affect. Examples include the following.

1. Vitamin B12 and folate deficiency – there is a two-fold increase in depression in people with these deficiencies.
2. Subclinical hypothyroidism (in which the T4 level is normal but the TSH is elevated) is associated with a four-fold increase in depression – more common than in established overt hypothyroidism.[39]

We should also be aware that many drugs can cause depression. The older antihypertensive drugs such as reserpine and methyldopa are rarely used now, but beta-blockers and nifedipine can cause depression. Steroids are another recognised cause. Centrally acting drugs which might be incriminated include barbiturates, benzodiazepines and phenothiazines.

The management of depression in physically ill old people

To the non-specialist, the treatment of depression can be vexing. There are so many drugs as well as physical and herbal remedies. In recent years, there have been

a number of clinical trials and literature reviews which have made management decisions a little easier for the physician.

The fact that there are so many therapies tells us that no single treatment is uniformly effective. The decision on whether to treat, with what treatment and for how long must be based on the individual's wishes, degree of depression, suicide risk, previous response to antidepressants, other drugs taken and concomitant medical problems.

Antidepressant drugs

About two-thirds of those with severe depression will respond to drug therapy (compared with a third who will respond to placebos). In recent years, there has been a big increase in the number and type of drugs available. We will briefly consider those that might be considered by the physician treating older patients and review the evidence for effectiveness and safety.

There are several classes of drugs for depression. All of them make various neurotransmitters more available in the brain but their precise mode of action is still not fully understood.

Tricyclic antidepressants (TCAs)

These were the first group of antidepressants to be developed and have been in use for over 50 years. They have effects on serotonin as well as noradrenaline (and perhaps on other neurotransmitters). Examples include amitriptyline, clomipramine, dosulepin (dothiepin), trazodone and trimipramine (which have sedative effects) and imipramine, nortriptyline and lofepramine (which are less sedating). Amitriptyline and dosulepin are dangerous in overdose. Lofepramine is safer but can cause hepatic toxicity. These drugs should be used with caution in patients with epilepsy. Older people are prone to syncope and falls when taking these drugs, which also cause hyponatraemia in some elderly users.

Tricyclics should be started with low dosage, which is then gradually increased. They usually begin to work within a few weeks. Patients taking these drugs should be advised to avoid alcohol, as the combination can result in drowsiness.

They are particularly useful in severely ill depressed patients. They improve sleep patterns. They are generally given once each evening (they usually have a long half-life). These often cause anti-cholinergic side-effects (dry mouth, constipation, confusion, urinary hesitancy and dribbling). They can also cause orthostatic hypotension and falls. They are best avoided in subjects with ischaemic heart disease or cardiac rhythm disturbances.

Newer drugs in this class have fewer side-effects than earlier compounds. Because they are much cheaper than SSRIs, they should be considered as first line treatment – unless there are medical contraindications (which is often the case in geriatric patients).

Physicians have been criticised for administering sub-optimal doses of TCAs. A systematic review of 39 studies found that doses between 75 and 100 mg per

day (and even lower doses) resulted in a better response than placebo. Interestingly, one study found there to be no evidence that standard doses of TCAs were more effective than lower dose schedules.[40]

Selective serotonin reuptake inhibitors (SSRIs)

SSRIs are front line therapy in patients at risk of overdose, patients with diabetes mellitus and cardiac problems. In general, they have low toxicity and good tolerability. Which SSRI should be prescribed? There are claims made for the advantages of individual drugs but we have no good large trials to inform us when to prescribe individual SSRIs.[41]

This group of drugs includes citalopram, escitalopram, paroxetine, sertraline and fluoxetine. Fluoxetine has been shown in a well-conducted placebo-controlled study to be efficacious in minor depression. However, is less used now because of its long half-life and potential for drug–drug interactions.[42] It can also occasionally reduce blood glucose levels and make it difficult for people to sit still. Escitalopram is a new SSRI, being the S-enantiomer of citalopram. It has been promoted as being superior to citalopram. However, there is no compelling evidence that this drug is more effective or has a faster onset of action than its parent compound.[43] Tachycardia is a side-effect of citalopram. SIADH also occurs in patients on this drug and untoward effects with the two drugs are similar. Paroxetine is associated with tiredness. It has a short half-life and patients should be advised not to stop taking it suddenly. Sertraline can cause diarrhoea.

These drugs should be introduced gradually and the dose slowly increased. There is a risk of increased anxiety in the early days of treatment. Soon after starting these drugs, patients may become nauseated or anxious. If the drugs are stopped suddenly, there is a risk of a withdrawl syndrome. These unpleasant symptoms occur in the first five days of discontinuation of the drug. Patients may experience stomach upsets, anxiety, flu-like symptoms, vivid dreams and sensations of electric shocks. They are more likely with paroxetine, which has a short half-life.

Other adverse effects of SSRIs are falls and fractures, gastrointestinal bleeding and hyponatraemia. Hyponatraemia is three times more common in patients taking SSRIs than in those taking other antidepressants.[44] Hyponatraemia is potentially life-threatening. It should be suspected in all patients who become drowsy, delirious or who have seizures while taking an anticonvulsant. It usually occurs in the first few weeks of treatment and is probably secondary to inappropriate antidiuretic hormone (ADH) secretion. The syndrome of inappropriate ADH is characterised by a low serum sodium (<135 mmol /L), a high urinary osmolality (>200 mOsm/kg), a urinary sodium of over 20 mmol per litre and a serum osmolality of less than 280 milliOsmoles per kilogram.[44] It is commoner in older people, women, those of low body weight and those with a low baseline sodium concentration. It is also more common if diuretics are used concomitantly. Patients receiving multiple medications may also be at increased risk. Treatment is with fluid restriction, severe cases requiring a loop diuretic and isotonic saline.[45]

Despite a large catalogue of possible adverse effects, few users get serious side-effects with SSRIs.

If a patient cannot tolerate a specific SSRI, or there is no remission of symptoms after about three months' therapy, one in four will respond to a second step choice.[46]

Serotonin and noradrenalin reuptake inhibitors (SNRIs)

These include venlafaxine (and duloxetine) which is used for both anxiety and depression and there is some evidence that venlafaxine might be more effective than SSRIs for major depression. Two thirds of patients with anxiety respond to this drug. It should be avoided in patients with renal impairment, uncontrolled hypertension, or cardiac disease. The treatment effect can be delayed. This drug can cause tremor, raised cholesterol, increased blood pressure (which may be sustained with the slow-release preparation) as well as gastrointestinal disturbances and tiredness. Patients on this drug should have their blood pressure monitored. There is a risk of overdose especially in patients with conduction disturbances or ventricular dysrhythmias. Up to a third of users who stop taking these drugs experience withdrawal symptoms (the same proportion as in those who stop SSRIs). They should therefore be tapered rather than stopped suddenly. The SNRIs are second line drugs best prescribed and monitored by an old age psychiatrist.

Lithium

This drug is efficacious in both the treatment and prevention of bipolar depression. Lithium reduces the suicide risk in bipolar disorder.[47] It is also used in acute mania. This drug is inexpensive and can be used as a second line drug for maintenance treatment of depression. It has a narrow therapeutic window and it is therefore important to monitor serum levels 12 hours after the last dose taken the previous evening. In older patients, the recommended level for prophylaxis is 0.4–0.6 mmol/L and for treatment of an acute episode 0.8–1.0 mmol/L. Before starting the drug, it is wise to measure renal and thyroid function (the drug can induce hypothyroidism). Lithium levels are decreased by acetazolamide and theophylline and increased by some antibiotics, anti-hypertensives and several diuretics (e.g. thiazides, spironolactone and metolazone).

Mono-amine oxidase inhibitors (MAOIs)

These drugs are rarely used now because they are less effective than TCAs and SSRIs. There is a risk of hypertension with foods containing tyramine and therefore the need for observing strict dietary limitations.

Noradrenergic and specific serotoninergic antidepressants (NaSSAs)

Mirtazapine, a pre-symptomatic alpha antagonist, causes initial sedation and can cause oedema, dizziness, weight gain and GI disturbances. It is used for major depression and is best prescribed by experts.

Tryptophan

Hydroxytryptophan and tryptophan are so-called 'natural' alternatives to conventional antidepressants. They are recommended for dysthymia and unipolar depression. Only two clinical trials are good enough to be reliable: they suggest that these drugs are better than placebo at relieving depression, but more studies are needed on their safety and efficacy.[48] A rare but serious complication is eosinophilic-myalgia syndrome, characterised by myalgia, arthralgia, dyspnoea, neuropathy and an increased eosinophil count.

A Cochrane review of 17 studies involving TCAs, SSRIs and MAOIs administered to 2000 patients showed that they were effective in the treatment of older community patients as well as inpatients.[49]

Another Cochrane review scrutinised 32 studies of different classes of drugs used in the treatment of depression in old people.[50] Tricyclics (TCAs) and selective serotonin reuptake inhibitors (SSRIs) were equally efficacious. However, TCAs are less well tolerated, with a higher withdrawal rate because of side-effects. There appear to be no differences between discontinuation rates (either because of adverse reactions or lack of efficacy) between the SSRIs and other second-generation antidepressants (e.g. venlafaxine, mirtazapine, bupropion) in patients with major depression. The number needed to treat with paroxetine to prevent one recurrence of depression is four.

The Royal College of Psychiatrists[51] recommends that antidepressants should be taken for six to nine months after the depressive symptoms have abated. The British National Formulary suggests 12 months' continuance after remission for elderly people, with five years' (or even lifelong) therapy for recurrent depression. Certainly, depression is less likely to recur if the drug is given for two years.[46] In practice, 50% of GPs discontinue these drugs after six months.[42]

A more detailed discussion of antidepressant use in older people can be found in Chapter 4.

Herbal and other remedies

Many people do not wish to take powerful synthetic antidepressants, fearing habituation and side-effects. Eighty-two per cent of Americans over the age of 65 use complementary or alternative therapies in the course of a year. Those with self-reported depression or anxiety are more likely to use natural remedies, relaxation techniques or spiritual practices.[52]

St John's wort (*Hypericum perforatum*) has long been used for depression in Germany where it is also licensed for anxiety and sleep disorders. Extracts of the herb contain many different chemical classes, so the active agent is uncertain. Evidence of effectiveness is inconsistent (perhaps in part because of the differing formulations of the drug) but a Cochrane review of 37 trials[53] suggested that *Hypericum* may be as effective as standard antidepressants

in treating mild to moderate – but not severe – symptoms (but other studies suggest it may have only limited benefit (*see* Chapter 4). Moreover, this preparation seems to have fewer side-effects than conventional drugs. However, it should not be taken in conjunction with antidepressant drugs. It can induce drug metabolising enzymes. Drug interactions include warfarin, digoxin, and anticonvulsants.

Long-chain omega-3 fatty acids might help relieve depression when given in addition to existing antidepressant medication but there is no good evidence justifying treating depression with these drugs alone.[54]

Physical activity

In a literature review of randomised controlled trials of exercise in depression, one study[55] found that it produced short-term gains in both major and minor depression. Another[25] found that exercise was helpful in treating post-stroke depression.

Psychological therapy

Historically, there was a bias against psychotherapy – largely because Freud believed that older people were too rigid to benefit from this form of treatment. Nowadays, most lay people consider that counselling therapy is the best way to treat depression. It helps people review how they see themselves, the world and other people.[51] GPs are more likely to offer counselling or psychotherapy than physicians.[1] Cognitive behavioural therapy is as effective as antidepressants for moderate (but not for severe) depression but takes longer to begin to work. There is an argument for using both concurrently but access to trained staff may make this unrealistic. Psychotherapy does not seem to be effective as maintenance treatment for major depression.[46]

Other approaches include behavioural activation (with scheduled restructured daily activities recorded in a diary), anxiety-management and problem-solving strategies (learning how to deal with retirement, bereavement or relocation). Family therapy is also helpful in helping people face up to unresolved intergenerational conflict, or deal with guilt or unrealistic expectations.

Support groups can be helpful, especially in dissipating the fears of dementia or inevitable institutionalisation.

Electroconvulsive therapy (ECT)

A recent Cochrane review[56] found only three trials which compared real ECT to simulated ECT and to antidepressants. Each study had serious methodological flaws. Despite the fact that this treatment has been in use for decades, it is not yet possible to say whether ECT is more effective than antidepressant medication. NICE[57] recommends that this treatment is used when the depression might be life-

threatening or to achieve rapid and short-term improvement when other options have been ineffective.

A more detailed discussion of ECT use in older people with an affective disorder can be found in Chapter 6.

When to prescribe antidepressants

For most people who are depressed in the context of an acute physical illness, it is perhaps best to treat the physical illness first and review the patient in two to three weeks to see if the depression has resolved (watchful waiting). Once the pain has been controlled or the heart failure treated, the patient's spirits will often lift. Similarly, the patient is likely to be less depressed if their physical independence improves. If the depression persists but is mild, there is no need to prescribe an SSRI – this group of drugs is no better than placebo in the management of mild depression. In severe depression, antidepressants are more effective than placebo.

Given the paucity of studies and the poor quality of the published trials of antidepressant therapy, it is difficult to make many firm recommendations about the optimum treatment for depression. The older TCAs are best avoided in many old people, as these drugs often have unacceptable side-effects. Whether to prescribe a newer TCA or an SSRI as first line therapy will be a decision made on cost, potential drug interactions and patient preference.

The patient should be asked if they would like to consider medication. They should be reassured about the low risk of habituation. It is important to treat depression – if not, there is a two-fold increase in overall mortality. If in doubt, it is wise to treat the depression: better to treat the potentially reversible than to deny effective therapy.

When to refer to an old age psychiatrist

If a first line drug is not working, it is prudent to ask for specialist help. Decisions about whether to increase the dose to higher levels than physicians are comfortable with, to switch to another agent in the same class (e.g. another SSRI), to switch to another class of drugs ('out of class switch'), to use a dual agent (e.g. a drug which also inhibits the uptake of 5-hydroxytryptamine *and* noradrenaline) or to augment therapy with a different class of drug (such as lithium) are best made by consultants in old age psychiatry. Referral should also be considered if the patient is refusing to eat or drink – in severe cases, ECT may be considered.

Where do we go from here?

The priority is to do large high quality randomised controlled trials in older populations – it is unfortunate that studies still focus on younger subjects. These should be based in primary care as well as including hospital patients. They

should include economic evaluations. We also need to find ways of improving the recognition of depression in old people by the patients themselves, their informal caregivers, nurses, therapists and physicians.

KEY POINTS

- Depression is common in old people but often unrecognised and untreated.
- It occurs in many medical conditions, particularly if there is pain, disability or prolonged or serious illness.
- It should always be considered in those who are bereaved and residents of nursing and residential homes.
- In mild depression, counselling is useful but drugs are not usually indicated.
- In moderate to severe depression, antidepressants should be considered.
- There is little difference in effectiveness between tricyclics and SSRIs.
- The newer tricyclics are sometimes suitable but should not generally be prescribed for old people with heart disease, constipation, glaucoma or those who fall.
- SSRIs are less cardiotoxic and safer in overdose and are generally well tolerated, but are more expensive and can cause unpleasant effects if they are suddenly withdrawn.
- When prescribing SSRIs, always check the blood urea and electrolytes after two weeks.
- The drugs should be taken for at least six to nine months after the depressive symptoms after the depression has resolved (some advocate a two-year course).
- In recurrent depression, lifelong treatment may be necessary.

REFERENCES

1 Harman JS, Veazie PJ, Lyness JM. Primary care physician office visits for depression by older Americans. *J Gen Intern Med.* 2006; **21**: 926–30.
2 Cullum S, Tucker S, Todd C, *et al.* Screening for depression in older medical inpatients. *Int J Geriatr Psychiatr.* 2006; **21**: 469–76.
3 Copeland JR, Beekman AT, Braam AW, *et al.* Depression among older people in Europe: the EURODEP studies. *World Psychiatry.* 2004; **3**: 45–9.
4 Rubinow DR. Treatment strategies after SSRI failure: good news and bad news. *NEJM.* 2006; **354**: 1305–6.
5 Pepersack T, De Breucker S, Mekongo YP, *et al.* Correlates of unrecognised depression among hospitalised geriatric patients. *J Psychiatr Pract.* 2006; **12**: 160–7.
6 Koponen HJ, Viilo K, Hakko H, *et al.* Rates and previous disease history in old age suicide. *Int J Geriatr Psychiatr.* 2007; **22**: 38–46.
7 Clarke DM, Cook KE, Coleman KJ, *et al.* A qualitative examination of the experiences of 'depression' in hospitalized medically ill patients. *Psychopathology.* 2006; **39**: 303–12.
8 Raue PJ, Meyers BS, Rowe JL, *et al.* Suicidal ideation among elderly homecare patients. *Int J Geriatr Psychiatr.* 2007; **22**: 32–7.
9 Bremmer MA, Hoogendijk WJ, Deeg DJ, *et al.* Depression in older age is a risk factor for first ischemic cardiac events. *Am J Geriatr Psychiatr.* 2006; **14**: 523–30.

10 Arthur HM. Depression, isolation, social support, and cardiovascular disease in older adults. *J Cardiovasc Nurs.* 2006; **21**(5 Suppl. 1): S2–7.
11 Sorensen C, Brandes A, Hendricks O, *et al.* Depression assessed over 1-year survival in patients with myocardial infarction. *Acta Psychiatr Scand.* 2006; **113**: 241–4.
12 Turvey CL, Klein DM, Pies CJ. Depression, physical impairment, and treatment of depression in chronic heart failure. *J Cardiovasc Nurs.* 2005; **21**: 178–85.
13 Koenig HG, Vandermeer J, Chambers A, *et al.* Comparison of major and minor depression in older medical inpatients with chronic heart and pulmonary disease. *Psychosom.* 2006; **47**: 296–303.
14 Morgan AL, Masoudi FA, Havranek EP, *et al.* Difficulty taking medications, depression, and health status in heart failure patients. *J Card Fail.* 2006; **12**: 54–60.
15 Hughes JW, Casey E, Luyster F, *et al.* Depression symptoms predict heart rate recovery after treadmill stress testing. *Am Heart J.* 2006; **151**: 1122.el–6.
16 Steptoe A, Strike PC, Perkins-Porras L, *et al.* Acute depressed mood as a trigger of acute coronary syndromes. *Biol Psychiatr.* 2006; **15**: 837–42.
17 Cao Z, Ong KC, Eng P, *et al.* Frequent hospital readmissions for acute exacerbation of COPD and their associated factors. *Respirology.* 2006; **11**: 188–95.
18 Alexopoulos GS, Sirey JA, Raue PJ, *et al.* Outcomes of depressed patients undergoing inpatient pulmonary rehabilitation. *Am J Geriatr Psychiatr.* 2006; **14**: 466–75.
19 O'Brien JT, Firbank MR, Krishnan MS, *et al.* White matter hyperdensities rather than lacunar infarcts are associated with depressive symptoms in older people: the LADIS study. *Am J Geriatr Psychiatr.* 2006; **14**: 824–41.
20 Kwok T, Lo RS, Wong E, *et al.* Quality of life of stroke survivors: a 1-year follow-up study. *Arch Phys Med Rehabil.* 2006; **87**: 1177–82.
21 House AO, Hackett ML, Anderson CS, *et al.* Pharmaceutical interventions for emotionalism after stroke. *The Cochrane Database of Systematic Reviews.* 2004; **2**. Available from: http://www.cochrane.org/reviews/en/ab003690.html
22 Hackett ML, Anderson CS, House AO. Interventions for treating depression after stroke. *The Cochrane Database of Systematic Reviews.* 2004; **3**. Available from: http://www.cochrane.org/reviews/en/ab003437.html
23 Thomas SA, Lincoln NB. Factors relating to depression after stroke. *Br J Clin Psychol.* 2006; **45**: 49–61.
24 Almeida OP, Waterreus A, Hankey GJ. Preventing depression after stroke: results from a randomised placebo-controlled trial. *J Clin Psychiatr.* 2006; **67**: 1104–9.
25 Lai SM, Studenski S, Richards L, *et al.* Therapeutic exercise and depressive symptoms after stroke. *J Am Geriatr Soc.* 2006; **54**: 240–7.
26 Katona CE. *Depression in Old Age.* Chichester: John Wiley and Sons; 1994.
27 Onitilo AA, Nietert PJ, Egede LE. Effect of depression on all-cause mortality in adults with cancer and differential effects of cancer site. *Gen Hosp Psychiatr.* 2006; **28**: 396–402.
28 Crichlow RJ, Andres PL, Morrison SM, *et al.* Depression in orthopaedic trauma patients: prevalence and severity. *J Bone Joint Surg Am.* 2006; **88**: 1927–33.
29 Covic T, Tyson G, Spencer D, *et al.* Depression in rheumatoid arthritis patients: demographic, clinical, and psychological predictors. *J Psychosom Res.* 2006; **60**: 469–76.
30 Barnes DE, Alexopoulos GS, Lopez OL, *et al.* Depressive symptoms, vascular disease, and mild cognitive impairment: findings from the Cardiovascular Health Study. *Arch Gen Psychiatr.* 2006; **63**: 273–9.
31 Bains J, Birks JS, Dening TD. Antidepressants for treating depression in dementia. *The Cochrane Database of Systemic Reviews.* 2002; **4**. Available from: http://www.cochrane.org/reviews/en/ab003944.html
32 Kirsch-Darrow L, Fernandez HH, Marsiske M, *et al.* Dissociating apathy and depression in Parkinson's disease. *Neurology.* 2006; **67**: 33–8.

33 Ghazi-Noori S, Chung TH, Deane K, *et al.* Therapies for depression in Parkinson's disease. *The Cochrane Database of Systematic Reviews.* 2003; **3**. Available from: http://www.cochrane.org/reviews/en/ab003465.html

34 Berman K, Brodaty H. Psychological effects of age-related macular degeneration. *Int Psychogeriatr.* 2006; **18**: 415–28.

35 Wong SY, Hoing A, Leung J, *et al.* Lower urinary tract symptoms and depressive symptoms in elderly men. *J Affect Disord.* 2006; **96**: 83–8.

36 Menchetti M, Cevenini N, De Ronchi D, *et al.* Depression and frequent attendance in primary health care patients. *Gen Hosp Psychiatr.* 2006; **28**: 119–24.

37 Thorpe JM, Sleath BL, Thorpe CT, *et al.* Caregiver psychological distress as a barrier to influenza vaccination among community-dwelling elderly with dementia. *Med Care.* 2006; **44**: 713–21.

38 Molloy DW, Standish TI, Dubois S, *et al.* A short screen for depression: the AB Clinician Depression Screen (ABCD). *Int Psychogeriatr.* 2006; **18**: 481–92.

39 Chueire VB, Romaldini JH, Ward LS. Subclinical hypothyroidism increases the risk for depression in the elderly. *Arch Gerontol Geriatr.* 2007; **44**: 21–8.

40 Furukawa T, McGuire H, Barbui C. Low dosage tricyclic antidepressants for depression. *The Cochrane Database of Systematic Reviews.* 2003; **3**. Available from: http://www.cochrane.org/reviews/en/ab003197.html

41 Cipriani A, Brambilla P, Furukawa T, *et al.* Fluoxetine versus other types of pharmacotherapy for depression. *The Cochrane Database of Systematic Reviews.* 2005; **4**. Available from: http://cochrane.org/reviews/en/ab004185.html

42 Fitch K, Molnar FJ, Power B, *et al.* Antidepressant use in older people: family physicians' knowledge, attitudes, and practices. *Can Fam Physician.* 2005; **51**: 80–1.

43 Drug and Therapeutics Bulletin. New drugs from old. *DTB.* 2006; **44**: 73–7.

44 Covyeou JA, Jackson CJ. Hyponatraemia associated with Escitalopram. *NEJM.* 2007; **356**: 94–5.

45 Jacob S, Spinler SA. Hyponatraemia associated with selective serotonin-reuptake inhibitors in older adults. *Ann Pharmacother.* 2006; **40**: 1618–22.

46 Reynolds CF III, Dew MA, Pollock BG. Maintenance treatment of major depression in old age. *NEJM.* 2006; **354**: 1130–8.

47 Freeman MP, Freeman SA. Lithium: clinical considerations in internal medicine. *Am J Med.* 2006; **119**: 478–81.

48 Shaw K, Turner J, Del Mar C. Tryptophan and 5-Hydroxytryptophan for depression. *The Cochrane Database of Systematic Reviews.* 2001; **3**. Available from: http://www.cochrane.org/reviews/en/ab003198.html

49 Wilson K, Mottram P, Sivanranthran A, *et al.* Antidepressants versus placebo for the depressed elderly. *The Cochrane Database of Systematic Reviews.* 2001; **3**. Available from: http://www.cochrane.org/reviews/en/ab000561.html

50 Mottram P, Wilson K, Strobl J. Antidepressants for depressed elderly. *The Cochrane Database of Systematic Reviews.* 2006; **1**. Available from: http://www.cochrane.org/reviews/en/ab003491.html

51 Royal College of Psychiatrists. *Antidepressants.* Available from: http://www.rcpsych.ac.uk/mentalhealthinformation/mentalhealthproblems/depression/antidepressants.asp (accessed 2007).

52 Grzywacz JG, Suerken CK, Quandt SA, *et al.* Older adults' use of complementary and alternative medicine for mental health: findings from the 2002 National Health Interview Survey. *J Altern Complement Med.* 2006; **12**: 467–73.

53 Linde K, Mulrow CD, Berner M, *et al.* St John's wort for depression. *The Cochrane Database of Systematic Reviews.* 2005; **4**. Available from: http://www.cochrane.org/reviews/en/ab000448.html

54 Drug and Therapeutics Bulletin. Do omega-3 fatty acids help in depression? *DTB.* 2007; **45**: 9–12.

55 Sjosten N, Kivela SL. The effects of physical exercise on depressive symptoms among the aged: a systematic review. *Int J Geriatr Psychiatr.* 2006; **21**: 410–18.
56 Stek M, Van der Wurff FB, Hoogendijk W, *et al.* Electroconvulsive therapy for the depressed elderly. *The Cochrane Database of Systematic Reviews.* 2003; **2**. Available from: http://www.cochrane.org/reviews/en/ab003593.html
57 National Institute for Clinical Excellence. Technology Appraisal: electro-convulsive therapy. In: *Compilation: summary of guidance issued to the NHS in England and Wales.* London: National Institute for Clinical Excellence; 2003. pp. 133–4.