Affective disorders

Affective disorders are psychiatric diseases with multiple aspects, including biological, behavioural, social, and psychological factors. These are mental disorders that can be associated with mood disturbance and psychotic symptoms, which include depression, anxiety and mania. Persons with an affective disorder may or may not have psychotic symptoms such as delusions or hallucinations. This article highlights the main types of affective disorder, the clinical features and the treatment options in older people.

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Affective disorder is a mental disorder characterised by a consistent, pervasive alteration in mood, and affecting thoughts, emotions, and behaviours. It may include manic (elevated, expansive, or irritable mood with hyperactivity, pressured speech, and inflated self-esteem) or depressive (dejected mood with disinterest in life, sleep disturbance, agitation, and feelings of worthlessness or guilt) episodes, and often combinations of the two.

Persons with an affective disorder may or may not have psychotic symptoms such as delusions, hallucinations, or other loss of contact with reality. Old age depression is often mistreated, or undertreated, and also under-diagnosed. There are several reasons for that, such as older people tend to reduce their social relations, depression is often presents as a comorbidity with organic diseases (that cover and mask depressive symptoms); and patient may believe that a depressive state is a normal course of life in old age.[1]

Clinical features

Affective disorders can result in symptoms ranging from the mild and inconvenient to the severe and life-threatening; the latter account for more than 15% of deaths due to suicide among those with one of the disorders.[1] Patients often have a hard time getting through the day without experiencing symptoms. The depressive symptoms in old age depression are similar to those in adults; however the following aspects require special care, in order to ensure a correct diagnosis despite the presence of comorbidities:[1]

- The mood: Old people often do not complain about their low mood
- Psychotic and hypochondriacal symptoms are often present
- Anxiety symptoms are often present together with neuro-sensory symptoms
- Somatic symptoms and comorbidity with organic diseases can mask and overlap the depressive state
- The reduction of cognitive functioning is quite frequent, and it is essential to make a differential diagnosis from "pseudo-dementia" and "dementia".

Types

Major depressive disorder, bipolar disorders, and anxiety disorders are the most common affective disorders.

Major depressive disorder

Major depressive disorder (MDD), also known as uni-polar affective disorder, is a common, severe, and sometimes life-threatening psychiatric illness. It is characterised by having five or more depressive symptoms that last for two weeks or longer; these symptoms include a feeling of worthlessness, a pessimistic attitude, sadness, hopelessness and new sleeping or eating habits.[2] MDD is further subdivided into five subtypes, melancholic depression, atypical depression, catatonic depression,

Box 1: Main criteria for seasonal pattern "specifier"

1. Presence of a regular temporal relationship between the onset/recurrence of major depressive (or manic) episodes and a particular time of the year (excluding cases in which there is an obvious effect of seasonal-related psychosocial stressors, such as regularly being unemployed every winter).
2. Full remissions (or change from depression to mania or hypomania) also occur at a characteristic time of the year (eg. depression disappears in the spring).
3. Two major depressive episodes in the previous two years that demonstrate the temporal seasonal relationships, and no non-seasonal major depressive episodes have occurred during that same period.
4. Seasonal major depressive episodes substantially outnumber the non-seasonal major depressive episodes that may have occurred over the individual's lifetime.

post-partum depression, and seasonal affective disorder. MDD causes prolonged periods of emotional, mental, and physical exhaustion, with a considerable risk of self-destructive behaviour and suicide. Major studies have identified MDD as one of the leading causes of work disability and premature death, representing an increasingly worldwide health and economic concern.[1,2]

Bipolar affective disorder

Bipolar affective disorder constitutes one pole of a spectrum of mood disorders including bipolar I, bipolar II, cyclothymia (oscillating high and low moods), and hypomania disorder. Bipolar I disorder is also referred to as classic manic-depression, characterised by distinct episodes of major depression contrasting vividly with episodes of mania, which lead to severe impairment of function. In comparison, bipolar II disorder is a milder disorder consisting of depression alternating with periods of hypomania. Hypomania may be thought of as a less severe form of mania that does not include psychotic symptoms or lead to major impairment of social or occupational function. Bipolar disorders are chronic and recurrent affective diseases that may have degrees of severity, tending however to worsen with time if not treated. Severe crises can lead to suicidal attempts during depressive episodes or to physical violence against oneself or others during manic episodes. In many patients, however, episodes are mild and infrequent.

Anxiety disorders

Anxiety disorders are common psychiatric disorders, and are considered one of the most under-treated and overlooked health problems. Among their common manifestations are panic syndromes, phobias, chronic generalised anxiety disorder, obsessive-compulsive disorder (OCD), and post-traumatic disorder. They are important contributors to other diseases such as hypertension, digestive and eating disorders, and cardiac arrhythmia.

Seasonal affective disorder

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) categorises seasonal affective disorder (SAD) not as a unique mood disorder, but as a specifier of major depression.[3] Thus, patients with SAD experience episodes of major depression which tend to recur at specific times of the year. These seasonal episodes may take the form of major depressive or bipolar disorders (table 1).[4] Like major depression, SAD probably is under-diagnosed in primary care settings.[4] Although several screening instruments are available, such screening is unlikely to lead to improved outcomes without personalised and detailed attention to individual symptoms.

In general, standardised screening instruments for SAD probably are not sensitive enough to be used for routine screening.[5] Physicians should be aware of comorbid factors that could signal a need for further assessment. Specifically, some emerging evidence suggests that SAD may be associated with alcoholism and attention-deficit/hyperactivity disorder.[4]

Demography

Affective disorders, particularly depression, are common in older people and may have a fatal outcome. Depression presents as

a common problem in older people, even though its incidence may peak in the two decades before age 65 years.[6,7] Gurland and associates found a point prevalence of 13% of significant depression, with a figure of 2.5% for "severe" or major affective disorders.[8] Suicide remains as common in the elderly as in other age groups with depression.[8] A study of the prevalence of depression among older patients in general practice showed that major depression (DSM-III-R) affected 22.4%, dysthymic disorder (depressive disorder with chronic course and insidious onset) 6.3% and not otherwise specified depression 7.1%.[9] Studies indicate differences in lifetime prevalence estimates for bipolar disorders: 1.0% for bipolar I disorder, 1.1% for bipolar II disorder, and 2.4–4.7% for sub-threshold bipolar disorders.[10] The overall lifetime prevalence of SAD ranges from 0 to 9.7%.[5]

Aetiology

Affective disorders in late life may begin prior to and then persist into old age or may have their very first onset later in life. Structural and functional brain change has been implicated in the genesis of late-life affective illness both by direct investigation and by implication from observed associations of affective disorder with organic conditions. A number of age-related processes may be important in late-onset affective disorders, including reduced neurotransmitter levels [eg. 5-Hydroxytryptamine (5-HT) and noradrenalin (NA)],[11,12] increasing vascular burden,[13] early degenerative processes,[14-16] increasing vulnerability to oxidative stress[17] and disordered neuro-endocrine function, particularly of the hypothalamo-pituitary-adrenal (HPA) axis.[18] These biological factors occur within a changing psychosocial milieu as individuals' age and there may be important interplay between them. Also, there are contrasts between the aetiology of early- and late-onset disorders with regard to both biological and psychosocial factors. A helpful view is that of Baldwin suggesting that depression in the elderly is a condition that is modified by factors that are not present in younger individuals, rather than being an illness subtype in its own right.[19]

Biological factors that appear to be of importance in depression in older people may at first seem disparate—cortisol hyper-secretion, hippocampal

atrophy, white matter changes, vascular disease—but there are important interactions between these such that none can be considered mutually exclusive.[18] SAD is associated with serotonergic dysregulation and possibly with noradrenergic mechanisms.[20] SAD may overlap with other diagnoses that share similar mechanisms, including generalised anxiety disorder, panic disorder, bulimia nervosa, late luteal phase dysphoric disorder, and chronic fatigue syndrome.[20] A pattern of seasonal alcohol use may be associated with SAD. Recent studies concluded that some patients with alcoholism may be self-medicating an underlying depression with alcohol or manifesting a seasonal pattern to alcohol-induced depression.[21] In general, cultural influences and social pressures in achievement-oriented societies are important risk factors in affective disorders symptoms.

Box 2: Non-pharmacological therapy in affective disorders

Therapy	Indications and value
Psychotherapy	Psychotherapy alone is rarely sufficient for the treatment of affective disorders, as the existing neuro-chemical imbalance impairs the ability of a person with an affective disorder to respond. However, psychotherapy is important in helping to cope with guilt, low self-esteem, and inadequate behavioural patterns once the neurochemistry is stabilised and more normal levels of neurotransmitters are at work.
Cognitive behaviour therapy (CBT)	CBT has some effectiveness in improving dysfunctional automatic thoughts and attitudes, behaviour withdrawal, low rates of positive reinforcement, and ruminations in patients with major depression. CBT is being used increasingly to reduce anxiety in older persons and it may involve relaxation training, cognitive restructuring (replacing anxiety-producing thoughts with more realistic, less catastrophic ones) and exposure (systematic encounters with feared objects or situations). However, few studies have assessed its effectiveness in the treatment of SAD.
Light therapy	Among susceptible persons, decreased seasonal exposure to light may mediate SAD through phase shifts in circadian rhythms, with resulting alterations in several aspects of serotonin metabolism. Thus, light replacement has been the most widely studied treatment for SAD.

Diagnosis

Well-known sets of clinical characteristics associated with MDD, bipolar diseases, SAD, or anxiety disorders provide the physician the necessary data for an initial diagnosis of affective disorder.[22] Practitioners analyse the person's pattern of mood, behavioural, and cognitive symptoms, along with the family history and environmental-contributing factors.

The diagnosis of bipolar I disorder requires the presence of a manic episode of at least one week's duration that leads to hospitalisation or other significant impairment in occupational or social functioning. The episode of mania cannot be caused by another medical illness or by substance abuse. These criteria are based on the specifications of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR).[22]

Investigations

Certain baseline studies, such as thyroid function tests and serum electrolytes, may be considered to role out any underlining impairment that

would necessitate adjusting the dosage of medications. Pre-treatment ECG may be considered as tricyclics and some of the antipsychotics can affect the heart and cause conduction problems. Lithium also can lead to changes such as reversible flattening or inversion of T waves. The total value of performing brain imaging or electroencephalogram in a patient with these disorders remains unclear.

Treatment

There are several treatment options for patients with affective disorders, including psychotherapy, light therapy, cognitive behaviour therapy, pharmacotherapy and electroconvulsive therapy (table 2). For SAD, treatment options should include light therapy, in addition to cognitive behaviour therapy and pharmacotherapy. Each option has been proven beneficial in treating SAD, but no large studies have found any treatment to be superior.[1]

Pharmacological therapy

Depressive disorder

Selective serotonin reuptake inhibitors (SSRIs), tricyclics and monoamine oxidase inhibitors, are among the medications used to treat depression. The effects of antidepressants are to some extent superior to those of psychotherapy, although in short-term trials more patients—especially those with milder forms of depression—cease medication than cease psychotherapy. This is most likely due to adverse effects from the medication and to patients' preferences for psychological therapies over pharmacological treatments.[23] The SSRIs are the primary medications prescribed owing to their effectiveness, relatively mild side effects, and because they are less toxic in overdose than other antidepressants.[24] Patients who do not respond to one SSRI can be switched to another antidepressant, such as venlafaxine, and this results in improvement in almost half of cases.[25]

Antidepressants are usually continued for 16 to 20 weeks after remission, to minimise the chance of recurrence, and even up to one year of continuation is recommended.[24] People with chronic depression may need to take medication indefinitely to avoid relapse.[24] Most of antidepressants can cause hyponatraemia and it has been reported more often with SSRIs.[26] Many older patients resist the use of antidepressants due to fear of dependence; resistance to viewing depressive symptoms as a medical illness; concern that antidepressants will prevent natural sadness; and prior negative experiences with medications for depression.[27]

Bipolar disorder

Although lithium is the mainstay in treatment of manic symptoms in younger bipolar patients, there are no double-blind studies of its effectiveness in older people as they have difficulty tolerating lithium neurologic side effects, even at low serum concentrations (0.4 to 0.6mEq/L).[28] Side effects include ataxia, subjective memory problems, polyuria, and tremor, and these are more pronounced in older patients, who often have concomitant medical or neurologic illnesses.[29] Carbamazepine and valproates have been found to be as effective as lithium in the treatment of mania in younger patients and are better tolerated in the elderly.[30]

Lamotrigine and gabapentin are two other anticonvulsants that have been reported to be effective in the treatment of bipolar disorder, and gabapentin has been shown to be effective in treatment-resistant bipolar disease for both manic and depressed phases.[31] Older bipolar patients have been found to have a high rate of tardive dyskinesia (approaching 20%), and the prevalence of tardive dyskinesia increases with age.[32] In addition, older patients are more sensitive to the anti-cholinergic and orthostatic hypotension associated with low-potency antipsychotics as well as the extrapyramidal symptoms (EPS) of high-potency neuroleptics.[32] The traditional antipsychotics are, therefore, infrequently used as the sole therapy of manic patients.

Neuroleptics are often used as adjunctive medication in the treatment of bipolar disorder.[33] Atypical anti-psychotics, such as risperidone, clozapine and olanzapine, can be used in mania and they exert their antipsychotic effect as mixed dopamine-serotonin antagonists. The primary advantage of these atypical antipsychotics is a marked reduction or elimination of EPS and the potential for tardive

JANUVIA®▼sitagliptin

PRESCRIBING INFORMATION

Refer to Summary of Product Characteristics (SPC) before prescribing

Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard. Adverse events should also be reported to MSD (tel: 01992 467272).

PRESENTATION

25 mg film-coated tablet containing 25 mg of sitagliptin

50 mg film-coated tablet containing 50 mg of sitagliptin

100 mg film-coated tablet containing 100 mg of sitagliptin.

USES

For adult patients with type 2 diabetes mellitus 'Januvia' is indicated to improve glycaemic control:

as monotherapy

- in patients inadequately controlled by diet and exercise alone and for whom metformin is inappropriate due to contraindications or intolerance

as **dual oral therapy** in combination with

- metformin when diet and exercise plus metformin alone do not provide adequate glycaemic control
- a sulphonylurea when diet and exercise plus maximal tolerated dose of a sulphonylurea alone do not provide adequate glycaemic control and when metformin is inappropriate due to contra-indications or intolerance
- a PPARγ agonist (i.e. a thiazolidinedione) when use of a PPARγ agonist is appropriate and when diet and exercise plus the PPARγ agonist alone do not provide adequate glycaemic control

as **triple oral therapy** in combination with

- a sulphonylurea and metformin when diet and exercise plus dual therapy with these medicinal products do not provide adequate glycaemic control.
- a PPARγ agonist and metformin when use of a PPARγ agonist is appropriate and when diet and exercise plus dual therapy with these medicinal products do not provide adequate glycaemic control.

Januvia is also indicated as add-on to insulin (with or without metformin) when diet and exercise plus stable dosage of insulin do not provide adequate glycaemic control.

DOSAGE AND ADMINISTRATION

One 100 mg tablet once daily, with or without food. When sitagliptin is used in combination with metformin and/or a PPARγ agonist, maintain the dosage of metformin and/or PPARγ agonist, and administer sitagliptin concomitantly. When used in combination with a sulphonylurea or with insulin, consider a lower dose of sulphonylurea or insulin, to reduce risk of hypoglycaemia. If a dose of Januvia is missed, take as soon as the patient remembers. Do not take a double dose on the same day.

Renal impairment: when considering use in combination with other anti-diabetic products, check conditions for use in patients with renal impairment. No dosage adjustment required for mild renal impairment (creatinine clearance [CrCl] ≥50 ml/min). For patients with moderate renal impairment (CrCl ≥30 to <50 mL/min), the dose of 'Januvia' is 50 mg once daily. For patients with severe renal impairment (CrCl <30 mL/min) or with end-stage renal disease (ESRD) requiring haemodialysis or peritoneal dialysis, the dose of 'Januvia' is 25 mg once daily. 'Januvia' may be administered without regard to the timing of dialysis. Because there is a dosage adjustment based upon renal function, assessment of renal function is recommended prior to initiation of 'Januvia' and periodically thereafter. Hepatic impairment: no dosage adjustment necessary for patients with mild to moderate hepatic impairment. Januvia has not been studied in patients with severe hepatic impairment. Elderly: no dosage adjustment necessary. Exercise care in patients ≥ 75 years of age as there are limited safety data in this group. Children: not recommended in children below 18 years of age.

CONTRA-INDICATIONS

Hypersensitivity to active substance or excipients.

PRECAUTIONS

General: do not use in patients with type 1 diabetes or for diabetic ketoacidosis.

Pancreatitis: Post-marketing experience - spontaneously reported adverse reactions of acute pancreatitis. Inform patients of the symptom of acute pancreatitis: persistent, severe abdominal pain. Resolution of pancreatitis has been observed after discontinuation of sitagliptin, but very rare cases of necrotizing or haemorrhagic pancreatitis and/or death have been reported. If pancreatitis is suspected, 'Januvia' and other potentially suspect medicinal products should be discontinued.

Hypoglycaemia when used with other anti-hyperglycaemic agents: Rates of hypoglycaemia reported with sitagliptin were generally similar to rates in patients taking placebo. When sitagliptin was added to a sulphonylurea or to insulin, the incidence of hypoglycaemia was increased over that of placebo; therefore consider a lower dose of sulphonylurea or insulin to reduce the risk of hypoglycaemia. Renal impairment: 'Januvia' is renally excreted. To achieve plasma concentrations of 'Januvia' similar to those in patients with normal renal function, lower dosages are recommended in patients with moderate and severe renal impairment, as well as in ESRD patients requiring haemodialysis or peritoneal dialysis (see section 'Dosage and administration' above and section 4.2 and 5.2 of the SmPC). Hypersensitivity reactions: Serious hypersensitivity reactions have been reported, including anaphylaxis, angioedema, and exfoliative skin conditions including Stevens-Johnson syndrome. Onset occurred within the first 3 months after initiation of treatment with some reports occurring after the first dose. If suspected, discontinue 'Januvia', assess for other potential causes and institute alternative treatment for diabetes.

Drug interactions

Low risk of clinically meaningful interactions with metformin and ciclosporin. Meaningful interactions would not be expected with other p-glycoprotein inhibitors. The primary enzyme responsible for the limited metabolism of sitagliptin is CYP3A4, with contribution from CYP2C8.

Digoxin: sitagliptin had a small effect on plasma digoxin concentrations, and may be a mild inhibitor of p-glycoprotein in vivo. No dosage adjustment of digoxin is recommended, but monitor patients at risk of digoxin toxicity if the two are used together.

Pregnancy and lactation: Do not use during pregnancy or breast-feeding.

SIDE EFFECTS

Refer to SPC for complete information on side effects

Sitagliptin monotherapy: *Common (≥ 1/100 to <1/10)*: upper respiratory tract infection†, nasopharyngitis†, osteoarthritis†, pain in extremity† hypoglycaemia‡, headache; *Uncommon (≥ 1/1,000 to <1/100)*: dizziness, constipation. Combination with metformin: *Common (≥ 1/100 to <1/10)*: hypoglycaemia§, nausea, flatulence, vomiting; *Uncommon (≥ 1/1,000 to <1/100)*: somnolence, constipation, upper abdominal pain, diarrhoea, blood glucose decreased. Combination with a sulphonylurea: *Common (≥ 1/100 to <1/10)*: hypoglycaemia§. Combination with metformin and a sulphonylurea: *Very common (≥ 1/10)*: hypoglycaemia§; *Common (≥ 1/100 to <1/10)*: constipation. Combination with a PPARγ agonist (pioglitazone): *Common (≥ 1/100 to <1/10)*: hypoglycaemia§, flatulence, peripheral oedema, blood glucose decreased. Combination with a PPARγ agonist and metformin: *Common (≥ 1/100 to <1/10)*: upper respiratory tract infection†, headache, diarrhoea, vomiting, hypoglycaemia§, peripheral oedema, cough†; *Uncommon (≥ 1/1,000 to <1/100)*: fungal skin infection†. Combination with insulin with/without metformin: *Common (≥ 1/100 to <1/10)*: headache, hypoglycaemia§, influenza; *Uncommon (≥ 1/1,000 to <1/100)*: dry mouth, constipation.

Adverse events with sitagliptin alone in clinical studies, or during post-approval use alone and/or with other diabetes medicines where frequency is not known: hypersensitivity reactions including anaphylactic responses (see section 4.4)‖,‡, interstitial lung disease‡, vomiting‡, acute pancreatitis‡ fatal and non-fatal haemorrhagic and necrotizing pancreatitis‡,¶, angioedema‡,¶, rash‡,¶, urticaria‡,¶, cutaneous vasculitis‡,¶, exfoliative skin conditions‡,¶ including Stevens-Johnson syndrome‡,¶, arthralgia‡, myalgia‡, impaired renal function‡, acute renal failure‡.

† Based on incidence regardless of causal relationship.

‡ Adverse reactions were identified through postmarketing surveillance.

§ 54-week time point.

‖ See precautions.

PACKAGE QUANTITIES AND BASIC NHS COST

28 Tablets: £33.26

Marketing Authorisation Number

EU/1/07/383/002 – **Januvia** 25 mg tablets

EU/1/07/383/008 – **Januvia** 50 mg tablets

EU/1/07/383/014 – **Januvia** 100 mg tablets

Marketing Authorisation Holder

Merck Sharp & Dohme Limited

Hertford Road, Hoddesdon, Hertfordshire EN11 9BU, UK

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MSD

dyskinesia. Electroconvulsive therapy (ECT) may be the ideal treatment for bipolar disorder and the response rate to ECT is approximately 80% for depressive and manic symptoms.[34] ECT therefore has an advantage over the traditional mood-stabilising treatments in providing effective treatment for both phases of the illness as well as a faster response compared with lithium.[34] However, patient acceptance of the procedure has limited ECT to a third-line treatment in bipolar disorder.

Seasonal affective disorder

SAD often can be treated with light therapy, which appears to have a low risk of adverse effects, with effect sizes similar to those for antidepressant medications in treating depression.[35] Light therapy is more effective if administered in the morning. The total daily dosage should be approximately 5,000 lux, administered in the morning over 30 to 120 minutes.[35] It remains unclear whether light is equivalent to drug therapy, whether drug therapy can augment the effects of light therapy, or whether cognitive behaviour therapy is a better treatment choice. However, cognitive behaviour therapy should be considered as an alternative to light therapy in the treatment of SAD.[36]

Several trials showed that patients with SAD had significantly better response on several measures of depression after eight weeks of sertraline therapy compared with control patients.[37] It is also possible that pharmacotherapy may preserve an initial therapeutic response to light therapy. Among 168 patients who had a positive response to light therapy, citalopram was found to be no more effective than placebo at preventing relapse; however, it was superior in terms of some secondary measures of depression.[38] In general, current evidence does not provide clear guidance as to whether antidepressant treatment is superior to light therapy, or whether antidepressants are useful as an adjunct to light therapy in SAD.

Anxiety disorder

Both medication and psychosocial therapies are used to treat anxiety in older patients, although clinical research on their effectiveness is still limited. Anti-depressants such as SSRIs, rather than anti-anxiety medication such as the benzodiazepines, are the preferred medication for most anxiety disorders. Antidepressants become the drugs of choice in the treatment of anxiety disorders, particularly the newer agents that have a safer adverse effect profile and higher ease of use than the older tricyclic agents; however, benzodiazepines often are used as adjunct treatment.

It has been well established that sleep disturbance, cognitive difficulty, impairment in activities of daily living, motor vehicle crashes, and problems with gait are potential negative side effects of benzodiazepines on older adults.[39] Drug–disease interaction and chronicity of use are two other common problems associated with benzodiazepine use in this population.[40] The SSRIs are helpful in a variety of anxiety disorders, including generalised anxiety disorder, panic disorder, obsessive–compulsive disorder, and social phobia.

Conclusion

Affective disorder is a mental disorder characterised by a broad set of psychiatric symptoms and disorders that are sometimes under-estimated among older persons. Structural and functional brain change has been implicated in the genesis of late-life affective illness both by direct investigation and by implication from observed associations of affective disorder with organic conditions. Major depressive disorder, bipolar disorders, and anxiety disorders are the most common affective disorders.

Diagnosis relies on analysing patients' pattern of mood, behavioural, and cognitive symptoms, along with the family history and environmental-contributing factors. Several treatment options are used, including psychotherapy, light therapy, cognitive behaviour therapy, pharmacotherapy and electroconvulsive therapy.

Conflict of interest: none declared

References available on online version at: www.gmjournal.co.uk