



THE CONTINUING PROFESSIONAL DEVELOPMENT PROGRAMME



This module is suitable for use by pharmacists as part of their continuing professional development. After reading this module, complete the learning scenarios and post-test at www.pharmacymag.co.uk and include in your CPD portfolio. Previous modules in the Pharmacy Magazine CPD Programme are also available to download from the website

MODULE 189

Welcome to the one hundred and eighty ninth module in the *Pharmacy Magazine* Continuing Professional Development Programme, which looks at supporting people with dementia and their carers. It is valid until June 2014.

Continuing professional development (CPD) is a mandatory requirement for pharmacists. Journal-based educational programmes are an important means of keeping up-to-date with clinical and professional developments and form a significant element of your CPD. Completion of this module will contribute to the nine pieces of CPD that must be recorded a year.

Before reading this module, test your existing understanding of the topic by completing the pre-test at www.pharmacymag.co.uk. Then after studying the module in the magazine, work through the six learning scenarios and post-test on the website. Record your learning and how you applied it in practice using the CPD report form, available online and on p viii.

Self-assess your learning needs:

- Describe the general early symptoms of dementia
- Name the different types of dementia likely to be encountered in primary care
- What are the main pharmacological agents used to treat the condition?

Warning: The content contained in this module is the copyright of Pharmacy Magazine and cannot be reproduced without permission in the form of a valid written licence granted after July 1, 2011

CURRENT THINKING ON...

SUPPORTING PEOPLE WITH DEMENTIA AND THEIR CARERS

Contributing author: Dr Denise Taylor, Senior Teaching Fellow in Clinical Pharmacy, Department of Pharmacy and Pharmacology, University of Bath

Introduction

In 2001, the National Institute for Clinical Excellence approximated the number of people with dementia in England and Wales to be 700,000, of which 400,000 would have had Alzheimer's disease (AD)¹.

Definition

Dementia has been defined as "a syndrome consisting of progressive impairment in two or more areas of cognition: (memory; language; visuospatial and perceptual ability; thinking and problem-solving; personality), sufficient to interfere with work, social function or relationships"².

General symptoms

Dementia is neurodegenerative so there is gradual onset, often noticed at times of stress

or change (e.g. when admitted to hospital with an infection or when there is a change in environment). The presentation of symptoms is influenced by pre-morbid personality. Early symptoms include:

- Memory loss, especially for recent events
- Difficulties with learning and/or retaining new information; being more repetitive; or misplacing objects (e.g. car keys or spectacles)
- Having trouble with complex tasks such as cooking, driving or dealing with finances
- Reduced ability to reason and problem-solve
- Impairment of spatial and visuospatial awareness (e.g. bumping into objects, getting lost in a familiar place)
- Language problems: inability to find the right word or difficulty following conversations
- Behavioural changes: more irritable, passive, withdrawn or suspicious.

FOR THIS MODULE

pharmacy MAGAZINE
FIRST IN PROFESSIONAL & BUSINESS DEVELOPMENT

GOAL: To enable pharmacists to provide proactive medicines management services to people with dementia and their carers to enhance quality of life.

OBJECTIVES: After completing this module you should be able to identify:

- Early signs and symptoms associated with a possible dementia
- Medicines which may be harmful for a person with dementia
- Information sources that would support patients and their carers.



Reflection exercise 1

Would you be able to create a list of referral or signposting links for people with dementia and their families that are local to you? The Alzheimer's Society website (www.alzheimers.org.uk) or www.dementiaweb.org.uk can be used to create your local list.

N.B. For access to a memory clinic people may need referral by their GP.

Community pharmacists can help recognise early signs and symptoms of neurodegeneration, and signpost or refer people appropriately. It is important to remember that at each stage of dementia there is an associated decline in physical functioning, which may affect caring and require support from social services.

Diagnosis

There is no definitive laboratory or tissue marker for the diagnosis of any dementia, so the diagnosis is one of exclusion requiring a full medical history and multi-faceted assessment to exclude pseudodementias (which account for about one per cent of all dementias), as these are potentially treatable and therefore reversible.

The vast majority of dementias are irreversible, with Alzheimer's disease (AD) accounting for 50 per cent of all cases, vascular dementia (VaD) 25 per cent (with an estimated 25 per cent of these having mixed AD & VaD), Lewy body dementia (LBD) accounting for 15 per cent, and all others (such as Creutzfeldt-Jakob disease, Pick's disease, Huntington's disease, AIDS dementia complex) accounting for 10 per cent³.

It is increasingly common that people with a mixed diagnosis including AD will receive an acetylcholinesterase inhibitor (AChEI).

Alzheimer's disease

In 2009, the National Dementia Strategy (NDS) stated that in England there were 400,000 people with AD⁴ and of these 250,000 had mild to moderate disease. The NDS highlighted that the majority of people with a dementia remain undiagnosed and therefore ineligible for treatment and/or support.

Reflection exercise 2

How many people with dementia are diagnosed in your region? Check out the diagnostic mapping link at www.alzheimers.org.uk/dementiamap and estimate the number of people (diagnosed and undiagnosed) that could be registered with your pharmacy.

Probable AD

Diagnosis is one of exclusion and is established by clinical and neuropsychological examination and the presence of deficits in at least two areas of cognition with progressive worsening of memory. There is no disturbance of consciousness and absence of other disorders to account for dementia. Onset can be between 40 and 90 years of age.

Risk factors

■ **Increasing age:** age reflects the passage of time, therefore there is more time for genes to express themselves; an increasing inability to repair cell damage; and/or more time to be exposed to environmental agents that may be involved in the onset of AD

■ **Genetic predisposition:** specific genes link age at onset, duration and disease severity. Genetic risk is an autosomal dominant inheritance. Each person with an affected gene had one parent with the gene, and offspring of a person with an affected gene has a 50:50 chance of inheriting the gene. It does not skip generations and men and women are equally affected

■ **Head injury:** deposition of amyloid plaques triggering neurodegeneration

■ **Gender:** studies suggest slightly higher incidence in women than men, but this may be because women live longer

■ **Depression:** now thought to be a prodromal symptom

■ **Older maternal age**

■ **Education:** maintaining intellectual functioning is protective

■ **Exercise:** a healthy cardiovascular system equates to a healthy cerebrovascular system

■ **Other factors include:** epilepsy, herpes zoster/simplex, alcohol, smoking.

AD is most likely the sum of external factors (an environmental trigger) plus inherent host factors (genetic predisposition). The genetic predisposition influences whether AD occurs as early onset (<65 years) or late onset (>65 years).

Proposed disease pathway

Post mortem brains of people who had AD show high levels of amyloid plaque deposition, which leads to abnormal tau binding (tau is a protein that maintains the stability of the microtubules that serve as a transport system within brain cells) and the formation of neurofibrillary tangles (NFTs). This process ultimately leads to poor cerebral perfusion and brain cell death.

There is dramatic loss in cholinergic neurones, which causes a reduction in neurotransmitter levels (especially acetylcholine, which is involved in memory pathways). There may be atrophy at the frontal, parietal and temporal lobes, and brain volume loss of 10-15 per cent. This links the behavioural changes exhibited by people with dementia to areas of the brain where the neurodegeneration is present. Explaining this to carers may help them understand that challenging behaviour is not deliberate but actually caused by brain degeneration.



Support for carers is a vital element in the management of dementia patients

Reflection exercise 3

One of your patients with newly diagnosed dementia has had a few minor accidents while driving. His carer is very concerned. What advice would you give and what are your responsibilities in terms of protecting road safety for other users by informing the DVLA?
[See www.dvla.gov.uk/medical.aspx]

Vascular dementia

Vascular dementia (VaD) usually has sudden onset and then follows a step-wise progress (periods of stability followed by sudden decline) and is the result of cerebral ischaemic events. There are focal neurological signs and symptoms, and relative preservation of personality. Often there is nocturnal confusion, the presence of depression and patchy cognitive impairment.

VaD is potentially preventable by improving cerebral perfusion and preventing ischaemia by the use of an antiplatelet such as aspirin. Underlying risk factors such as hypertension, cardiovascular disease and diabetes should be treated. Statins have a role in the lipid regulatory mechanism which may contribute to the pathogenesis of dementia and could have a role in reducing the incidence of the disease.

Lewy body dementia

Lewy body dementia (LBD) typically follows a progressive, fluctuating course and is delirium-like with fluctuating periods of confusion and variations in attention and alertness. Visual hallucinations are common, as are Parkinsonian-like signs such as rigidity, bradykinesia with repeated falls. LBD is associated with the histological presence of Lewy bodies and widespread reduction of choline acetyltransferase in the neocortex and dopamine in the caudate nucleus.

People with LBD exhibit an extreme sensitivity to antipsychotics, leading to a three-

fold increase in mortality as they exacerbate both motor and cognitive disability – so these medications should be avoided completely if possible. If there is an absolute need to use, they should be started at one-quarter of the usual dose, effects should be monitored carefully and they should be withdrawn as soon as possible.

Lewy bodies are also found in the brains of people with Parkinson's disease (PD) and 80 per cent of people with PD will go on to develop PD dementia over time and exhibit the same sensitivity to antipsychotics.

Assessment tools

A variety of tools are used in the assessment of dementia and include scales which assess severity of the disease, levels of cognitive functioning, activities of daily living, problem-solving and the impression of the prescriber and/or the carer of the person with dementia. Previous NICE guidance¹ recommended the Folstein Mini Mental State Examination (MMSE) for the grading of the severity of dementia, where the score (marked out of a possible 30) indicates:

- Mild Alzheimer's disease: MMSE 21-26
- Moderate Alzheimer's disease: MMSE 10-20
- Moderately severe Alzheimer's disease: MMSE 10-14
- Severe Alzheimer's disease: MMSE less than 10.

However the NICE Technology Appraisal (TA) 217⁵ (www.nice.org.uk/guidance/TA217) recommends that the score should be interpreted alongside other outcome measures so that a global picture of the person is seen. The carer's perceptions of improvement should also be included. Measurement scales include the:

- Clinicians' Interview-Based Impression of Change (CIBIC): a measure of global outcome
- Alzheimer's Disease Assessment Scale: Cognitive subscale (ADAS-cog)

■ Progressive Deterioration Scale (PDS): a measure of functional/quality of life in later stages

■ Behavioural Psychological Scale for Dementia (BPSD): measures the incidence and severity of behavioural changes

■ Neuropsychiatric Inventory (NPI): measures the presence of neuropsychiatric symptoms.

The NHS aims to improve diagnosis through service improvements and also by raising public awareness that concerns about memory loss are worth checking out. Early diagnosis helps the individual and his/her family to plan for future care, allows for personal affairs to be put in order while the individual has insight and allows early access to support groups (e.g. Alzheimer's Society).

Pharmacological treatments for dementia

There are two pharmacological treatment options for people with AD – acetylcholinesterase inhibitors (AChEIs) and the NMDA-receptor antagonist memantine. The NICE Technology Appraisal (TA) 217 states that AChEIs are recommended options for managing mild and moderate AD, and memantine for managing moderate Alzheimer's disease for people who cannot take AChEIs, and as an option for treating severe AD⁵.

Acetylcholinesterase inhibitors

In the normal brain, acetylcholine (ACh) is rapidly inactivated by acetylcholinesterase to acetate and choline. The acetylcholinesterase inhibitors (AChEIs) prevent this breakdown to increase ACh at the synapse.

Three AChEIs are licensed for the symptomatic treatment of mild to moderately severe Alzheimer's dementia. These are: donepezil (Aricept), rivastigmine (Exelon) and galantamine (Reminyl). Rivastigmine also has a licence for the symptomatic treatment of mild to moderately severe dementia in patients with idiopathic Parkinson's disease.

AChEIs are not cures for dementia but there is evidence that they can improve personal, social and cognitive functioning, and delay institutionalisation. The cost is about £950 a

Differential diagnosis of the common dementias

Alzheimer's dementia	Vascular dementia	Lewy body dementia
Insidious onset Progressive decline	Stepwise progression History of stroke History of CVD Preserved insight CT areas of infarct	Impaired attention & visuospatial skills Preservation of recent memory Fluctuating confusion & consciousness Persistent visual hallucinations Spontaneous parkinsonism Increased sensitivity to antipsychotics



Reflection exercise 4

What are 'advance directives' and where can people find help in putting their legal and financial affairs in order?

year. However Aricept comes off patent in 2012 and the others in 2015, so costs should fall dramatically. See Table 1 for a comparison of the three AChEIs.

Adverse effects

There are two forms of cholinesterases. Acetyl is found mainly in the brain and butyryl is found outside the CNS, mainly in the heart and small intestine. By blocking these enzymes the side-effects are predominantly cholinergic including diarrhoea, nausea, possible vomiting and nasal rhinitis. Others include muscle cramps, fatigue, insomnia and dizziness. These agents may cause syncope and any falls or faints should be referred immediately.

Care should be taken when prescribing these agents in co-morbid asthma, COPD or peptic ulcer disease, and the latest Summary of Product Characteristics (SPC) should always be checked for possible adverse effects (www.medicines.org.uk/EMC/default.aspx).

It is important to note that an individual may experience relatively severe adverse effects with one agent, but not another. It is always worth trying another drug, and to remember that memantine may also be an alternative.

Efficacy

One-third of patients respond significantly; one-third respond somewhat; and one-third seem not to respond. Non-response with one does not mean non-response with all, and an alternative should be tried. The agents follow linear dose pharmacokinetics (the most effective response is seen at the highest tolerated dose) and the titration process may take three months or longer to reach this point (see Table 1).

Stopping, starting and switching

Switching to another AChEI may be indicated if there is poor tolerance to adverse effects or poor response and may also be useful if compliance is an issue and once-daily dosing is preferable.

Co-morbidity is also important; if people have parkinsonian signs, then rivastigmine would be the agent of choice and, due to its very short half life, may be the agent of choice if co-prescribed

for someone with asthma or COPD. If an exacerbation or deterioration in the primary condition occurs, then when stopped the drug will be rapidly cleared. Memantine is also an option if an AChEI is contraindicated or not tolerated.

Long-term efficacy

As the disease progresses the amount of ACh produced will be less than at pre-treatment and individual patient performance will decline, eventually to a stage where the agent will seem to have little clinical effect. However long-term studies for these agents show continued benefit at four to seven years for some individuals.

The decision when to stop an AChEI in the later stages of dementia is complex, as even though it may seem there is no response, there may be a rapid deterioration in mood and behaviour on discontinuation. In these instances clinical advice would be to restart as soon as

Reflection exercise 5

Access the NICE TA217 guidance (www.nice.org.uk/guidance/TA217) and identify the four key cognitive enhancers licensed for people with dementia.

- How would you explain the differences between these four agents?

Table 1: Comparison of acetylcholinesterase inhibitors

	Donepezil	Rivastigmine	Galantamine
Activity	Reversible	Pseudo-irreversible	Reversible AChEI + enhances action of ACh on muscarinic receptors
Dosing	Start at 5mg once daily and increase to 10mg after four weeks Evening dosing, just before retiring; If insomnia and vivid dreaming, change to morning (N.B. Takes three weeks to get to steady state – see below)	Starting dose is 1.5mg twice a day with morning and evening meals. If well tolerated at two weeks increase to 3mg twice a day. Subsequent increases to 4.5mg and then 6mg twice a day should be based on good tolerability with a minimum of two weeks' treatment before a dose increase	Initially 4mg twice daily for four weeks increasing to 8mg twice daily for four weeks to maintenance dose of 12mg twice daily
Forms available	5mg tablet (£63.54 for 28) and 10mg (£89.06 for 28) Orodispersible tablets 5mg (£63.54 for 28) and 1mg (£89.06 for 28)	Capsules 1.5mg, 3mg or 6mg all costing £798.12 a year at twice daily dosing Oral solution (equivalent dose to capsule) costing £99.14 for 240mg Patches 4.6mg or 9.5mg/24 hours costing £77.97 for 30 = £935.64 a year	Tablets 8mg (£68.32 for 56) or 12mg (£84 for 56) Oral solution 4mg/ml 100ml = £120 Modified release capsules: 8mg (£51.28 for 28); 16mg (£64.90 for 28) and 24mg (£79.80 for 28)
General	Dose dependent activity Greater selectivity acetylcholinesterase Once-daily dosing Low side-effect profile (nausea 14 per cent, vomiting 8 per cent and diarrhoea 12 per cent)	Dose dependent activity Greater selectivity acetylcholinesterase Twice daily dosing, also available as daily patch Low side-effect profile but may cause severe anorexia in some and weight loss (nausea 47 per cent, vomiting 31 per cent and diarrhoea 19 per cent) Also licensed for use in PD dementia	Inhibits acetylcholinesterase and enhances response of nicotinic receptors to ACh Dose dependent activity Twice daily dosing liquid or once daily capsules Low side-effect profile (nausea 24 per cent, vomiting 13 per cent and diarrhoea 9 per cent)
Pharmacokinetics	Peak concentration three to four hours post=dose T _{1/2} = 70 hours (steady state in three weeks) Linear pharmacokinetics Food does not affect absorption 95 per cent protein-bound Metabolised in liver CYP450 system and may need to modify dose in mild to severe hepatic impairment Renally excreted	T _{1/2} approximately two hours Inhibition lasts 10 hours Renal excretion of rivastigmine and inactive metabolites Major cytochrome P450 isoenzymes are minimally involved in rivastigmine metabolism C max levels increase in moderate hepatic and renal impairment – dose adjustment may be necessary; avoid in severe liver disease Caution in renal impairment Little protein binding 40 per cent	Serum max 0.5 to 1 hour T _{1/2} seven to eight hours Protein binding 18 per cent Food affects absorption but does not affect extent of absorption Reduce dose in moderate hepatic impairment and avoid in severe impairment Avoid if eGFR <9ml/minute/1.73m ²
Interactions with other medication	Always check in the latest Summary of Product Characteristics as this is more explicit than the BNF		

Note: Prices taken from BNF 59 and information from the latest Summaries of Product Characteristics available at www.medicines.org.uk/EMC/default.aspx

possible, but definitely in less than two weeks. However, in end-stage care, when the process is one of palliation, all medication should be reviewed and withdrawn as appropriate.

Memantine

Memantine is a voltage-dependent, moderate affinity uncompetitive N-methyl D-aspartate (NMDA) receptor antagonist that blocks excessive calcium ion influx into the neuron to prevent associated neuronal death. It is licensed for the treatment of people with moderate to severe AD.

Studies demonstrate significant improvement in cognition, functioning (activities of daily living) and global outcomes. They also show that fewer people taking memantine develop agitation⁶.

The starting dose is 5mg once daily, increasing by 5mg per week until a maximum of 20mg daily. It needs to be taken at the same time each day and is available as film coated tablets or oral drops. Other NMDA antagonists, such as amantadine or dextromethorphan, should be avoided; the latter being important if the individual has a cold or cough.

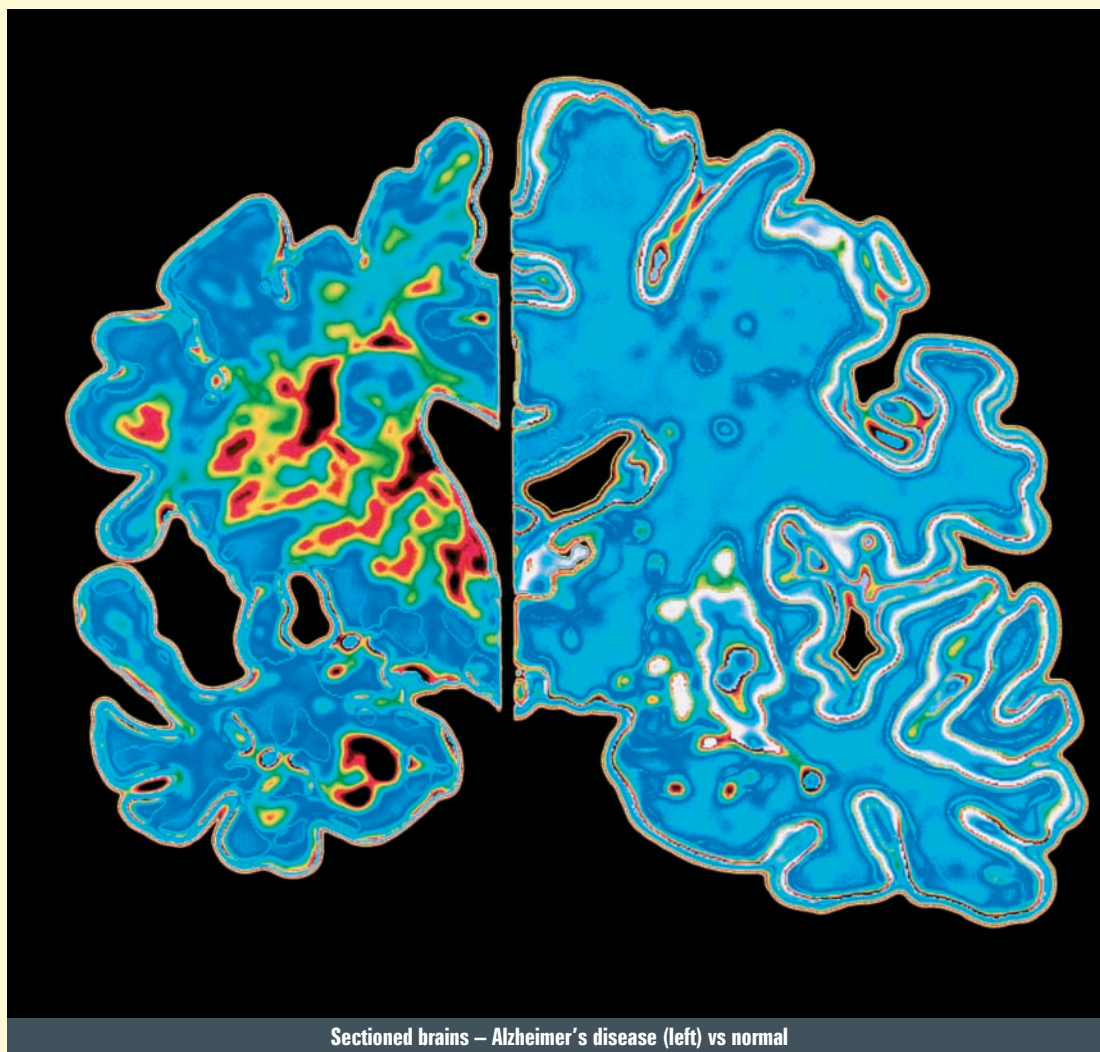
Common adverse effects include constipation, hypotension, confusion (1.3 per cent), dizziness (5 per cent), headache (5 per cent) and tiredness (1 per cent). If tiredness is present, evening dosing could be considered to promote sleep.

Other treatments

People with dementia and their families may turn to OTC and herbal supplements to try and prevent and/or delay the onset of dementia.

■ Ginkgo biloba

The proposed mechanism of action is to increase blood supply by dilating cerebral blood vessels and decreasing blood viscosity. There is a theoretical interaction with aspirin or warfarin possibly resulting in increased bleeding, but there are just two case reports^{7,8}. The prescriber should be consulted before use in these circumstances. Evidence for improvement is inconsistent with three studies showing equivalence to placebo and one showing a substantial positive effect⁹.



Sectioned brains – Alzheimer's disease (left) vs normal

Ginkgo seems safe to use and is well tolerated, but it is unknown whether long-term use has any effect on the onset (or not) of dementia.

■ Vitamin E

A Cochrane Review in 2008 stated that there was no evidence for the efficacy of vitamin E in the prevention or treatment of people with AD or mild cognitive impairment¹⁰.

■ Folic acid, B12 and iron

Deficit is linked with cognitive dysfunction (confusion and/or memory problems). Older people may not eat sufficient fresh green vegetables or dietary protein to sustain normal levels (and active transport mechanisms for B12 absorption are also often impaired). However if a deficiency has not been proven, supplementation will generally not have any clinical effect¹¹.

■ Light (Lux) therapy

Small studies have shown a benefit of people receiving light therapy in winter months having reduced behavioural problems such as agitation¹¹.

Managing behavioural problems

Behaviour may change in response to the area of the brain where the neurodegeneration occurs and, as the disease progresses, the reason for the person feeling sad or happy may not be remembered but the emotion will be. This inability to remember or communicate appropriately with others can cause much frustration and lead to behavioural changes.

Behavioural and psychological symptoms of dementia (BPSD) occur in up to 95 per cent of



people with the condition. These symptoms include the expression of delusions, hallucinations, agitation or aggression, depression, anxiety, elation or euphoria, apathy or indifference, disinhibition, irritation or lability, aberrant motor behaviour (wandering), night-time behaviour (sundowning), or a change in eating.

As can be seen, there is a wide variety of displayed behaviours and in general these are not harmful to anyone. However they can be difficult to control and are a major reason for carer stress and admission to long-term care.

What is agitation?

This has been defined as behaviour that is seen as disruptive but usually non-aggressive (e.g. moaning, pacing, crying, arguing), ranging to aggression in its severe forms where the person may endanger others or themselves (e.g. kicking, screaming, throwing objects, self-injury, scratching). These behaviours can be very distressing for carers and are the ones most likely to lead to institutionalisation. As a consequence of this, support of the carer is extremely important.

It has been shown that educational programmes for carers using behavioural techniques are more effective than most pharmacological treatments¹². However anti-psychotics are commonly prescribed for people with dementia, although there is a major drive to reduce such prescribing.

Supporting carers by providing information

The physical and psychological support of carers is very important, as evidence shows that lack of support leads to the caring process breaking down and early institutionalisation. Useful information includes:

- Financial and social benefits
- Legal issues such as power of attorney; advance directives; patient advocacy; and driving
- Respite services, which include people who provide a sitting service so a carer can have an afternoon or morning away from home or provide one or two weeks' care to give the carer a break. In many places the responsibility and finance for this has devolved down to the Alzheimer's Society, so it is a very useful first contact. Check the Alzheimer's Society website for details of your local branch – www.alzheimers.org.uk/Your_local_branch/index.htm
- Occupational therapy for living aids such as stairlifts; stair gates; shower and bathing aids; walking and dressing aids
- Support services that are available (e.g. washing and dressing services). Often these services are not offered proactively but only once an emergency or irretrievable situation has occurred. Early access can prevent carer stress and distress, and delay institutional admission
- Memory aids – local memory clinics and branches of the Alzheimer's Society can help with tools, prompts and activities for improving cognitive and memory function. Contact your local organisation for current brochures and services
- Carer (and healthcare professional) education groups on dementia are often provided by the Alzheimer's Society or the local memory clinic. They help carers to understand changing behaviour and learn how to cope with it. This greatly increases the support mechanism for the caring process and also ensures people with dementia are cared for appropriately
- Carer support services may also be run from the Alzheimer's Society, local memory clinics or GP surgeries. Many people are completely unaware of any services and appreciate actually knowing that they are a carer. There is a Carer Rights Act, which aims to support all carers appropriately. A carer website which outlines services is available at: www.direct.gov.uk/en/CaringForSomeone/CarersRights/DG_4001078
- The NICE-SCIE Dementia Guidelines is a very lengthy document that sets out a holistic care process for people with dementia and those that care for them. The summary document (www.scie.org.uk/publications/misc/dementia/index.asp) is a useful resource for problematic issues and also for referring healthcare professionals and carers to.

Sundowning

As the dementia progresses, people can lose a sense of whether it is day or night and will disrupt carers' sleep as they get up and get dressed in the middle of the night in order to get outside. Behavioural treatment and carer support are priorities – minimising catnapping by the patient during the day (to increase sleep at night), regular exercise, and establishing a day and night routine are important in the management of patients.

Use of bright light (Lux)¹¹ in the morning can reduce incidence of agitation in the evening. People with dementia tend to be very sensitive to any centrally acting medication and the prolonged action of sedatives increases risk of falls – so, if absolutely necessary, the smallest possible dose should be used for the shortest period of time.

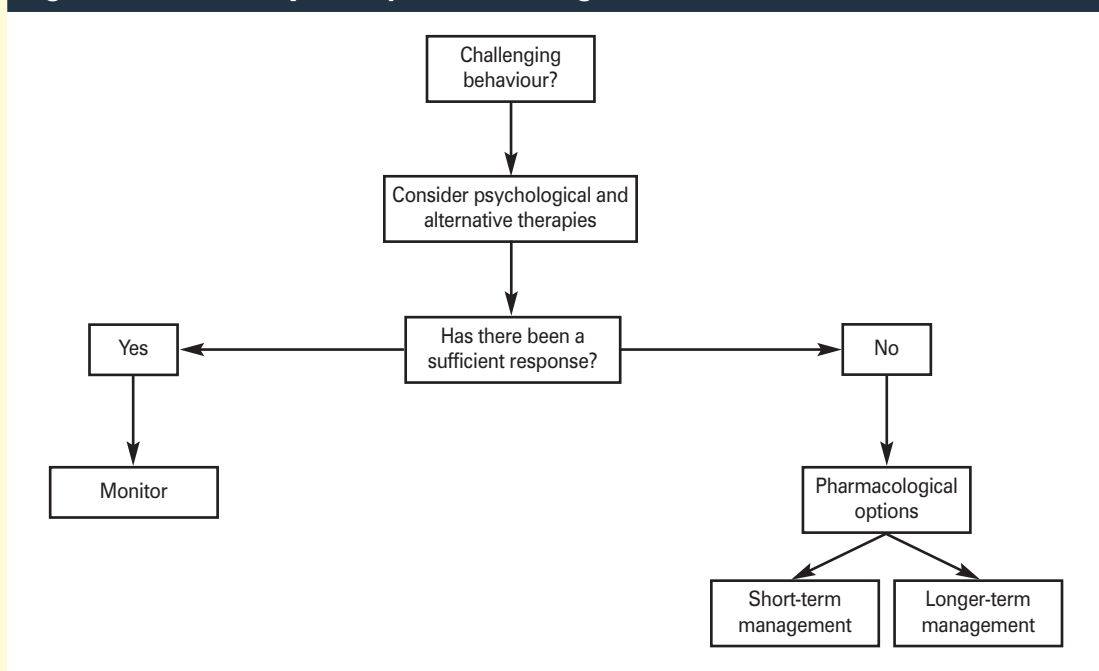
Treatment strategy for BPSD

Professor Clive Ballard, director of research at the Alzheimer's Society, has proposed a treatment pathway for managing BPSD (see Figure 1). He proposes that psychological and alternative therapies should be tried first and given sufficient time to exhibit a response.

Preferred non-pharmacological methods include:

- Environmental: lighting, space to walk, colour schemes, access to outdoors
- Behavioural interventions: distraction, reality orientation, occupational activities, reminiscence, sensory stimulation
- Exploring any possible underlying causes: e.g. pain, anxiety, depression, a recent change or upsetting event.

Figure 1: Treatment pathway for the management of BPSD



Reproduced by kind permission of Professor Clive Ballard

Evidence suggests that improving staff knowledge (e.g. in care homes) of what BPSD are and how to manage these using non-pharmacological means resulted in a sustained 50 per cent reduction in the use of antipsychotics over a year^{13,14}. Just improving social interaction also resulted in reduced rates of prescribing^{15,16}.

The use of lavender oil (aromatherapy and massage)^{17,18} has demonstrated significant improvement in agitation¹⁹ and a 34 per cent reduction in motor agitation²⁰. Aromatherapy with Melissa oil showed a significant reduction in CAMI scores²¹ (Confusion Assessment Method) and oral Melissa resulted in a significant reduction in agitation versus placebo¹⁹.

Short-term pharmacological management of behavioural symptoms in AD

Sometimes an antipsychotic may be appropriate if all other avenues have been tried and the individual is a danger to themselves or others and/or is extremely distressed.

Top tips to explore behavioural change

- Is the person depressed?
- Is the person in pain? (Remember patients often bump into things)
- Is there a superimposed acute confusional state (delirium) caused by infection (chest and urinary tract infections most common)?
- Has a new medication been started or one stopped?
- Is it a side-effect or withdrawal effect?
- Are there communication problems? (check glasses – correct prescription/clean; hearing aids – battery/turned on; teeth – in)
- Progression might mean the person wants to communicate but cannot and gets frustrated – be patient and calm; think of writing things down
- Has there been a recent change in environment/staff/routine?
- Is there always the same trigger (Antecedent); what Behaviour is produced; and what Changes the behaviour (e.g. distraction)?

CPD competences

This module supports the following community pharmacy competences:

Competence	Where this module supports competence development
C3b, C3e	This module highlights how pharmacists can provide pharmaceutical care to people with a chronic condition, such as dementia, and their carers
C4b-d, C4f-k	How pharmacists could potentially work with other healthcare professionals and across sectors to optimise both medicines use and provide a holistic care approach to people with dementia is highlighted. It also looks at how other healthcare and social provision may be accessed by carers as appropriate
C6a-c, C6g	Pharmacists are asked to consider optimising the dispensing and repeat process for people with dementia and their carers to ensure effective medicines management

Risperidone is licensed for a maximum of six weeks for the treatment of severe aggression in AD, which has not responded to other treatments. The Royal College of Psychiatrists recommends that the treatment decision is made by a specialist in dementia after discussion with the person's carer and/or family and the multidisciplinary team, and the reason recorded in the shared record²¹. The lowest dose producing the desired effect should be used and the drug's continued need should be reviewed regularly. Both typical and atypical antipsychotics have large side-effect profiles, with numbers needed to harm ranging from four to 13.

Longer-term antipsychotic prescribing

If a challenging behaviour continues and requires long-term management, then an alternative to an antipsychotic must be considered, as rates of mortality are increased in comparison to those who do not take long-term antipsychotics.

The DAART-AD trial showed that at 24 months the survival rate for those receiving an antipsychotic was 46 per cent compared to

71 per cent in the placebo arm; at 36 months survival was 30 per cent compared to 50 per cent; and at 42 months it was 26 per cent compared to 53 per cent²². Potential alternatives include:

■ AChEIs: Meta and pooled analyses suggest a 1.5-2 point advantage on total Neuropsychiatric Inventory (NPI) score over six months²³

■ Memantine: A meta-analysis suggests significant benefit for "behaviour" (2.76 points on NPI)²⁴. Promising *post hoc* pooled analysis shows improved behavioural symptoms in patients with moderate to severe AD²⁵

■ Citalopram: Two small four to six-week randomised controlled trials focusing on agitation/aggression, both with positive outcomes²⁶

■ Carbamazepine: A meta-analysis showed significant benefit on CGIC (Clinical Global Impression of Change) scores and BPSD^{27,28}. Care is required regarding interactions and hepatic enzyme induction.

References for the citations listed in this module are available from the Editor on request.



Pharmacy Magazine's CPD modules are now available on Cegedim Rx's PMR systems, Pharmacy Manager and Nexphase. Just click on the 'Professional Information & Articles' button within Pharmacy KnowledgeBase and search by therapy area. Please call the Cegedim Rx helpdesk on 0844 630 2002 for further information.



ASSESSMENT QUESTIONS

SUPPORTING PEOPLE WITH DEMENTIA

- 1. What diagnostic test is readily available for Alzheimer's disease?**
 - a. A computerised tomography (CT) scan
 - b. A magnetic resonance imaging (MRI) scan
 - c. Presence of tau in the cerebrospinal fluid
 - d. None – diagnosis is one of exclusion
- 2. People with mild Alzheimer's disease may have the following diagnostic indicators:**
 - a. A MMSE score between 17 and 20
 - b. Visual hallucinations
 - c. A MMSE score between 21 and 26
 - d. A space occupying lesion
- 3. Rivastigmine is licensed for use in:**
 - a. Parkinson's disease dementia
 - b. Vascular dementia
 - c. Alzheimer's disease only
 - d. Parkinson's disease dementia and Alzheimer's dementia
- 4. Which of the following can be a sign of agitated behaviour?**
 - a. Having visual hallucinations
 - b. Somebody who continually asks the same question
 - c. Someone who is pacing up and down the room, cannot settle and is getting increasingly anxious
 - d. Someone who is pacing up and down the room
- 5. Memantine is potentially beneficial for:**
 - a. Reversing the process of dementia
 - b. Helping with behavioural problems
 - c. People with mild dementia
 - d. People with vascular dementia
- 6. Which statement is FALSE? Benzodiazepines:**
 - a. Are associated with dependence and hangover effects
 - b. May increase the risk of falls in older people
 - c. Should usually be avoided when treating agitation
 - d. Can improve memory function
- 7. Which statement is TRUE about acetylcholinesterase inhibitors?**
 - a. They all work the same way
 - b. If one does not prove clinically effective, none of the others will
 - c. If severe side-effects occur with one agent, they will occur with each of the others
 - d. If severe side-effects occur with one agent, it is always worth trying a second agent
- 8. Which statement is FALSE? Vascular dementia:**
 - a. Has a sudden onset
 - b. Features periods of stability
 - c. Has a slow onset
 - d. Is potentially preventable

PHARMACY MAGAZINE CPD RECORD – JULY 2011

USE THIS FORM TO RECORD YOUR LEARNING AND ACTION POINTS FROM THIS MODULE ON SUPPORTING PEOPLE WITH DEMENTIA AND THEIR CARERS OR DOWNLOAD FROM WWW.PHARMACYMAG.CO.UK AFTER COMPLETING THE ONLINE LEARNING SCENARIOS

Activity completed. (Describe what you did to increase your learning. Be specific) (Act)

Name/date:

Time taken to complete activity:

What did I learn that was new in terms of developing my skills, knowledge and behaviours? Have my learning objectives been met? (Evaluate)

How have I put this into practice? (Give an example of how you applied your learning. Why did it benefit your practice? How did your learning affect outcomes?) (Evaluate)

Do I need to learn anything else in this area? (List your learning action points. How do you intend to meet these action points?) (Reflect)

* If as a result of completing your evaluation you have identified another new learning objective, start a new cycle – this will enable you to start at **Reflect** and then go on to **Plan, Act** and **Evaluate**. This form can be photocopied to avoid having to cut this page out of the module. Complete the learning scenarios at www.pharmacymag.co.uk

MODULE 189 ANSWER SHEET

ENTER YOUR ANSWERS HERE Please mark your answers on the sheet below by placing a cross in the box next to the correct answer. Only mark one box for each question. Once you have completed the answer sheet in ink, return it to the address below together with your payment of £3.75. Clear photocopies are acceptable. You may need to consult other information sources to answer the questions.

- | | | | | | | | | | | | | | | | |
|----|-----------------------------|----|-----------------------------|----|-----------------------------|----|-----------------------------|----|-----------------------------|----|-----------------------------|----|-----------------------------|----|-----------------------------|
| 1. | a. <input type="checkbox"/> | 2. | a. <input type="checkbox"/> | 3. | a. <input type="checkbox"/> | 4. | a. <input type="checkbox"/> | 5. | a. <input type="checkbox"/> | 6. | a. <input type="checkbox"/> | 7. | a. <input type="checkbox"/> | 8. | a. <input type="checkbox"/> |
| | b. <input type="checkbox"/> | | b. <input type="checkbox"/> | | b. <input type="checkbox"/> | | b. <input type="checkbox"/> | | b. <input type="checkbox"/> | | b. <input type="checkbox"/> | | b. <input type="checkbox"/> | | b. <input type="checkbox"/> |
| | c. <input type="checkbox"/> | | c. <input type="checkbox"/> | | c. <input type="checkbox"/> | | c. <input type="checkbox"/> | | c. <input type="checkbox"/> | | c. <input type="checkbox"/> | | c. <input type="checkbox"/> | | c. <input type="checkbox"/> |
| | d. <input type="checkbox"/> | | d. <input type="checkbox"/> | | d. <input type="checkbox"/> | | d. <input type="checkbox"/> | | d. <input type="checkbox"/> | | d. <input type="checkbox"/> | | d. <input type="checkbox"/> | | d. <input type="checkbox"/> |

Name (Mr, Mrs, Ms) _____

Business/home address _____

Town _____ Postcode _____ Tel: _____ GPhC/PSNI Reg no.

--	--	--	--	--	--	--	--	--	--

I am a PM subscriber I confirm the form submitted is my own work (signature): _____

Please charge my card the sum of £3.75 Name on card _____ Visa Mastercard Switch/Maestro

Card No. _____ Start date _____ Expiry date _____

Date _____ Switch/Maestro Issue Number _____

Processing of answers
Completed answer sheets should be sent to Precision Marketing Group, Precision House, Bury Road, Beyton, Bury St Edmunds IP30 9PP (tel: 01284 718918; fax: 01284 718920; email: cpd@precisionmarketinggroup.co.uk), together with credit/debit card/cheque details to cover administration costs. This assessment will be marked and you will be notified of your result and sent a copy of the correct answers. The examiners' decision is final and no correspondence will be entered into.