SIGN

Scottish Intercollegiate Guidelines Network

NHS Quality Improvement Scotland

86

Management of patients with dementia

A national clinical guideline

February 2006

KEY TO EVIDENCE STATEMENTS AND GRADES OF RECOMMENDATIONS

LEVELS OF EVIDENCE

1^{++}	High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias
1^{+}	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
1^{-}	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
2^{++}	High quality systematic reviews of case control or cohort studies High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
2^{+}	Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2^{-}	Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
3	Non-analytic studies, eg case reports, case series
4	Expert opinion

GRADES OF RECOMMENDATION

Note: The grade of recommendation relates to the strength of the evidence on which the recommendation is based. It does not reflect the clinical importance of the recommendation.

A	At least one meta-analysis, systematic review of RCTs, or RCT rated as 1^{++} and directly applicable to the target population*; or* A body of evidence consisting principally of studies rated as 1^{+}, directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2^{++}, directly applicable to the target population, and demonstrating overall consistency of results*; or* Extrapolated evidence from studies rated as 1^{++} or 1^{+}
C	A body of evidence including studies rated as 2^{+}, directly applicable to the target population and demonstrating overall consistency of results*; or* Extrapolated evidence from studies rated as 2^{++}
D	Evidence level 3 or 4*; or* Extrapolated evidence from studies rated as 2^{+}

GOOD PRACTICE POINTS

☑	Recommended best practice based on the clinical experience of the guideline development group

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1 Introduction

1.1 THE NEED FOR A GUIDELINE

The first Scottish Intercollegiate Guidelines Network (SIGN) guideline on interventions for the management of behavioural and psychological aspects of dementia (SIGN 22) was published in February 1998.[1] The original guideline addressed assessment, non-drug interventions, neuroleptic drugs, use of other drugs, and consent.

This revision of that guideline is an opportunity to expand and update the evidence base supporting the recommendations and to incorporate advice on new treatments such as cholinesterase inhibitors. The principles of careful evaluation based on individual needs, frequent review and slow titration of exposure to any intervention described in SIGN 22 are retained.

The issue of capacity to consent for all treatments, pharmacological and non-pharmacological needs careful consideration in people with dementia. Referral to the Adults with Incapacity (Scotland) Act 2000 or local equivalent may be useful.

1.2 REMIT OF THE GUIDELINE

The guideline considers investigations and interventions in which direct benefit to the patient can be demonstrated. It covers all stages of dementia excluding mild cognitive impairment. The guideline does not address palliative care in advanced disease, risk or prevention.

1.3 THE EVIDENCE BASE

SIGN evaluates research using a hierarchy of evidence which gives greater weight to studies such as randomised controlled trials (RCTs), which reduce the possibility of bias. Some of the research into the management of people with dementia is qualitative and cannot be evaluated easily within this methodology. Additionally, much dementia research consists of studies of small numbers of patients. These do not form a reliable basis for contributing to an evidence based guideline. This guideline concentrates on studies of intervention against control.

1.4 DEFINITIONS

Dementia

A generic term indicating a loss of intellectual functions including memory, significant deterioration in the ability to carry out day-to-day activities, and often, changes in social behaviour.

Alzheimer's disease

The most common cause of dementia is Alzheimer's disease (AD). Symptoms include memory problems, a progressive deterioration in the ability to perform basic activities of daily living (ADL), and behaviour changes, mainly apathy and social withdrawal, but also behavioural disturbances. Alzheimer's disease causes abnormal function and eventual death of selected nerve cells in the brain. The average survival period for patients following diagnosis is eight to 10 years.[2]

Vascular dementia

The role of vascular disease in the aetiology of dementia is complex and controversial. In some cases there appears to be a direct chronological relationship between significant cerebrovascular events and the onset of dementia. Consequently patients may present with signs of stroke or other vascular problems, for example, ischaemic heart disease or hypertension. Onset may be abrupt or there may be periods of sudden decline followed by relative stability. Physical problems such as urinary incontinence, decreased mobility and balance problems are more commonly seen in people with vascular dementia (VaD) than in people with Alzheimer's disease.

Dementia with Lewy bodies

Characteristic features of dementia with Lewy bodies (DLB) are fluctuation of awareness from day-to-day and signs of parkinsonism such as tremor, rigidity and slowness of movement or poverty of expression. Visual hallucinations or delusions occur frequently. Falls are also common. DLB has a similar pathological basis to Parkinson's disease dementia and both are associated with progressive cognitive decline and parkinsonism. Approximately three quarters of older people with Parkinson's disease develop dementia after 10 years.[3]

Fronto-temporal dementia

Fronto-temporal dementia (FTD) is uncommon by comparison to Alzheimer's disease or vascular dementia but represents a significant proportion of people who present with dementia under the age of 65. Changes in behaviour such as disinhibition, lack of judgement, loss of social awareness and loss of insight are much more common than memory problems. Disturbance of mood, speech and continence are frequent. A positive family history of a similar disorder is not uncommon.

Mixed dementias

Mixtures of two or more of the active dementias can be found in the same person, with one or other usually dominating. Studies suggest that the interaction between vascular disease and the core features of Alzheimer's disease is extremely complex and that rigid boundaries between subtypes of dementia may be unduly artificial.[4,5] Response to treatment or side effects from treatment in people with mixed dementia may be different from those in people with a specific diagnosis.

Creutzfeldt-Jakob disease

Creutzfeldt-Jakob disease (CJD) is a very uncommon illness in which an abnormal protein accumulates in the brain and leads to rapid destruction of nerve cells. Tremor, impaired mobility and balance problems are common as are behavioural and mood disturbance. Death within one to two years of the onset of clinical symptoms is common.[2] The management of patients with sporadic CJD and variant CJD is not covered by this guideline.

1.5 STATEMENT OF INTENT

This guideline is not intended to be construed or to serve as a standard of care. Standards of care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns of care evolve. Adherence to guideline recommendations will not ensure a successful outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgement must be made by the appropriate healthcare professional(s) responsible for clinical decisions regarding a particular clinical procedure or treatment plan. This judgement should only be arrived at following discussion of the options with the patient, family and carers, covering the diagnostic and treatment choices available. It is advised, however, that significant departures from the national guideline or any local guidelines derived from it should be fully documented in the patient's case notes at the time the relevant decision is taken.

1.6 REVIEW AND UPDATING

This guideline was issued in 2006 and will be considered for review in three years. Any updates to the guideline in the interim period will be noted on the SIGN website: **www.sign.ac.uk**.

2 Diagnosis

Dementia is a clinical diagnosis made when acquired cognitive deficits in more than one area of cognition interfere with activities of daily living and represent a decline from a previously higher level of functioning. Dementia can result from a number of single or combined underlying aetiologies and is usually progressive. Dementia is not usually diagnosed in the presence of delirium, although the two can coexist. The accurate differential diagnosis of dementia subtypes has become increasingly important with the advent of licensed treatments for Alzheimer's disease and the recognition of the potentially serious side effects of antipsychotics in people with dementia with Lewy bodies.

2.1 HISTORY TAKING AND DIFFERENTIAL DIAGNOSIS

A detailed history is an important part of the assessment of someone with suspected dementia. Attention should be paid to mode of onset, course of progression, pattern of cognitive impairment and presence of non-cognitive symptoms such as behavioural disturbance, hallucinations and delusions. Sufficient information should be gathered to apply the diagnostic criteria discussed in this section. As a person with dementia may not be able to give a fully accurate history a relative or carer should also be interviewed.

Subjective memory complaints, especially in well educated people, should be taken seriously, as these have been shown to be predictive of dementia, although they are also associated with depression and anxiety.[6] 2^{++}

There is a body of evidence showing that diagnostic criteria for probable Alzheimer's disease, such as those based on definitions contained in the Diagnostic and Statistical Manual, 4th edition (DSM-IV criteria; *see Annex 1*) or the National Institute of Neurologic, Communicative Disorders and Stroke–AD and related Disorders Association Work Group (NINCDS-ADRDA criteria; *see Annex 2*) have reasonably good diagnostic accuracy with a sensitivity of up to 80%.[7,8] 2^{++}

There have been fewer studies examining the diagnostic accuracy of criteria for vascular dementia than for Alzheimer's disease. In addition vascular dementia is not a homogenous entity and it may be common for patients to present with both Alzheimer's and vascular pathology. None of the current diagnostic criteria perform well for mixed presentations.

There is evidence to suggest that the Hachinski Ischaemic Score can be used to discriminate AD from VaD (*see Annex 3*)[9] and that the National Institute of Neurological Disorders and Stroke-Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIRENS; *see Annex 4*) criteria may be useful.[7,10] 1^{++} 2^{++}

The clinical criteria for dementia with Lewy bodies (Consortium for DLB criteria; *see Annex 5*) and fronto-temporal dementia (Lund-Manchester criteria; *see Annex 6*) are not closely associated with neuropathological diagnoses but can still provide useful differentiating clinical features.[7,10,11] 2^{++} 2^{+}

B **DSM-IV or NINCDS-ADRDA criteria should be used for the diagnosis of Alzheimer's disease.**

B **The Hachinski Ischaemic Scale or NINDS-AIRENS criteria may be used to assist in the diagnosis of vascular dementia.**

C **Diagnostic criteria for dementia with Lewy bodies and fronto-temporal dementia should be considered in clinical assessment.**

2.2 INITIAL COGNITIVE TESTING

The extent to which clinicians assess cognitive function, and their choice of cognitive test, varies widely. The Mini-Mental State Examination (MMSE; see *Annex 7*) was developed as a screening instrument for dementia and is widely used.[12] The brevity of the MMSE results in superficial assessment of memory, language, visuoperceptual function. Processing speed and executive function are not tested.

Evidence from a systematic review has shown that the MMSE is suitable for the detection of dementia in individuals with suspected cognitive impairment.[13] 2++

The Addenbrooke's Cognitive Examination (ACE; *see Annex 7*) is a more comprehensive measure of cognitive function that incorporates the MMSE. It is a 100-point test battery assessing six cognitive domains.

B **In individuals with suspected cognitive impairment, the MMSE should be used in the diagnosis of dementia.**

☑ Initial cognitive testing can be improved by the use of Addenbrooke's Cognitive Examination.

The Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE; *see Annex 8*) is a short questionnaire filled out by someone who knows the patient and can be an adjunct to direct cognitive testing.

☑ A questionnaire, such as the IQCODE, completed by a relative or friend may be used in the diagnosis of dementia.

2.3 SCREENING FOR COMORBID CONDITIONS

It is good practice to screen for coexisting medical conditions that are common in older people and for potential causes of dementia at first presentation.

Reversible causes of dementia, for example, due to hypothyroidism and vitamin B_{12} deficiency are very rare (less than 1%) and very few cases of reversible or partially reversible dementia have been detected by batteries of routine physical investigations.[14-16]

There is no evidence that routine batteries of laboratory tests improve the accuracy of the clinical diagnosis of dementia, nor is there evidence for the routine use of genetic markers or syphilis serology to increase the predictive value of a diagnosis.[7,13,14] 2++

☑ Physical investigations including laboratory tests should be selected on clinical grounds according to history and clinical circumstances.

The relationship between depression and dementia is complex. A systematic review found that people with depression and cognitive impairment are highly likely to have dementia diagnosed during longitudinal follow up and that 12% of people with dementia were also depressed.[7] A cohort study found that depression is often part of a dementia prodrome.[17] 2++

B **As part of the assessment for suspected dementia, the presence of comorbid depression should be considered**.

2.4 THE USE OF IMAGING

The ability of clinical examination (for example, history-taking and physical examination) to predict a structural lesion has been reported as having sensitivity and specificity of 90%.[18,19]

Imaging can be used to detect reversible causes of dementia and to aid in the differential diagnosis of dementia. The choice of imaging technique varies widely, and includes computed tomography (CT), magnetic resonance imaging (MRI), single photon emission controlled tomography (SPECT) and positron emission tomography (PET).

A systematic review showed that clinical prediction rules which attempt to detect those patients who should undergo imaging have poor sensitivity and specificity,[20] and could result in patients with potentially reversible causes of dementia being missed. 2++

Measures of medial temporal lobe width on CT can help distinguish dementia from depression, but cannot discriminate between causes of dementia.[21] 2+

MRI indices such as hippocampal volumetry can support clinical diagnosis of early AD,[22-24] assist in differential diagnosis, for example, of VaD,[25,26] and diagnose sporadic and variant CJD.[27,28] 2++

In one study, MRI was found to be superior to PET and SPECT for aiding diagnosis of dementia, but none is as effective as neuropsychology.[29] Assessment of delayed recall is at least as good as volumetric MRI in distinguishing people with probable AD from controls.[26] 1++

A systematic review and several subsequent studies have shown the benefit of SPECT in the diagnosis of Alzheimer's disease.[30-33] While clinical criteria may be more sensitive at detecting AD than SPECT, SPECT provides greater specificity against other types of dementia than clinical criteria.[34] Its use in discriminating AD from VaD, dementia with Lewy bodies and FTD has been demonstrated.[35-37] 2++ 2+

Combining structural and functional investigations (for example, CT and SPECT) may lead to a more accurate diagnosis.[38,39] 2++ 2+

C **Structural imaging should ideally form part of the diagnostic workup of patients with suspected dementia.**

C **SPECT may be used in combination with CT to aid the differential diagnosis of dementia when the diagnosis is in doubt.**

2.5 THE ROLE OF CEREBROSPINAL FLUID AND ELECTROENCEPHALOGRAPHY

Preliminary diagnostic studies have shown that reduced levels of cerebrospinal fluid (CSF) beta-amyloid and increased levels of CSF tau can differentiate patients with Alzheimer's disease from patients with other dementias as well as from people without dementia. Although one study reported sensitivity of 92% and specificity of 89% for differentiating between patients with Alzheimer's disease and controls using CSF beta-amyloid and tau,[40] there is insufficient evidence to support routine use of CSF markers in the diagnosis of dementia. 2++

There is evidence that the presence of 14-3-3 protein in CSF is a predictor for sporadic CJD. One study found 53% sensitivity for diagnosis of CJD by CSF examination,[41] while other studies report sensitivities and specificities of above 90%.[42,43]

There is evidence to support only the limited use of electroencephalography (EEG) in the diagnosis of dementia, for example, in the diagnosis of sporadic CJD, with reported sensitivity of 65% and specificity of 86%.[40] 2+

B **CSF and EEG examinations are not recommended as routine investigations for dementia.**

☑ CSF and EEG examinations may be useful where CJD is suspected.

2.6 NEUROPSYCHOLOGICAL TESTING

Assessment of cognition is useful in both the initial and differential diagnosis of dementia.[26,44] The added value of neuropsychological testing in patients who have previously received simple but comprehensive cognitive testing has not been established.

Several studies have employed neuropsychology primarily to compare people with Alzheimer's disease, fronto-temporal dementia, dementia with Lewy bodies, vascular dementia and depression.

It is possible to detect even very early Alzheimer's disease using neuropsychological testing.[44] 2+

Neuropsychology is superior to imaging in discriminating people with AD from controls.[26,29] 1++ 2++

Neuropsychological testing also aids in the differential diagnosis of dementia:

- FTD is characterised by deficits of semantic memory and attention/executive function rather than the episodic memory deficit seen in AD [29,45,46] 1++ 2++
- dementia with Lewy bodies has more pronounced visuoperceptual and frontal impairment compared to AD [47-49] 2+
- vascular dementia exhibits executive dysfunction [50,51]
- depression shows a subcortical pattern of cognitive impairment [52]

B **Neuropsychological testing should be used in the diagnosis of dementia, especially in patients where dementia is not clinically obvious.**

☑ It may be useful to repeat neuropsychological testing after six to 12 months in patients where:

- the diagnosis is unclear
- measurement of the progression of deficits in a typical pattern supports a diagnosis of dementia and helps in differential diagnosis.

The provision of neuropsychology services is variable and in places non-existent.

3 Non-pharmacological interventions

Non-pharmacological interventions for the behavioural and psychological symptoms (neuropsychiatric symptoms) of dementia are used to ensure that underlying causes of behavioural disturbance are explored and to provide personalised approaches to presenting problems. This section examines the therapeutic interventions which have been assessed in clinical trials (*see Table 1*). Interventions are listed in alphabetical order.

Table 1: Index to core and associated symptoms and non-pharmacological interventions

Core symptoms	**Section**
Cognitive decline	3.3, 3.7
Functional decline	3.2, 3.7
Social decline	no robust evidence identified
Associated symptoms	
Agitation	3.5.1, 3.8
Aggression	no robust evidence identified
Depression	3.1
Psychosis	no robust evidence identified
Repetitive vocalisation	no robust evidence identified
Sleep disturbance	no robust evidence identified
Non-specific behaviour disturbance	3.2, 3.5.4, 3.8
Other	
Side effects	3.5.1

3.1 BEHAVIOUR MANAGEMENT

The term "behaviour management", is used to reflect structured, systematically applied and normally time-limited interventions usually carried out by carers or care home staff under the supervision of a professional with expertise in this area.

Four RCTs reported behaviour management as an intervention for patients living in a variety of residential settings, although how these relate to level of severity of dementia in individuals is not clear.[53-56] Each of the studies reported behavioural interventions with different levels of complexity. The largest study had the most complex intervention and the longest study period (12 weeks).[54]

Although affective symptoms associated with dementia, including facial expression, contentment and interest can be improved by behaviour management,[54,55] there was no evidence for significant reduction in disruptive behaviour in nursing home residents or those in the community.[53] 1+

There is evidence to support the use of behavioural management to reduce depression in people with dementia living in the community with a caregiver.[55] Depression in people with dementia receiving behavioural therapy either involving pleasant events or problem solving was compared to that in control groups. Patient depression was improved for up to six months after both interventions. 1+

B **Behaviour management may be used to reduce depression in people with dementia.**

Evidence suggests that reduction of repetitive verbalisations, management of aggression and management of eating behaviours in people with dementia have a positive effect on behaviour and well-being.[54,56] 1-

☑ Multilevel behavioural management interventions may be more effective than individual interventions at improving behaviour and well-being in people with dementia.

Behavioural management cannot be recommended for other symptoms associated with dementia due to a lack of good evidence. Further trials on effectiveness of behaviour management at differing levels of severity of dementia are required.

3.2 CAREGIVER INTERVENTION PROGRAMMES

Caregiver intervention ranges from the simplest reassurance to the most complex multi-faceted interaction with the person with dementia, including in one case, a caregiver residential programme. In this guideline only structured caregiver intervention programmes are assessed. These studies do not apply to subjects who are living alone.

There is evidence to support the use of comprehensive caregiver support in reducing institutionalisation. In one study, 65% of the intervention group were living at home after 30 months compared to 26% in the control group.[57] In a Finnish study the median time of residing in the community following a programme of systematic comprehensive support by a nurse or dementia family care coordinator was 647 days in the intervention group and 396 days in the control group.[58] The clinical impact of this treatment on the patient was minimal and time-limited, with greatest benefit to those with severe dementia. 1+

B **Caregivers should receive comprehensive training on interventions that are effective for people with dementia.**

The cost savings of delay to institutionalisation as a result of caregiver training are noted in one study.[57]

After three months of home environment support by their caregiver, one RCT showed evidence of a small improvement in the associated symptoms of the person with dementia.[59] There was a wide variation in the level of functional dependency on the carer, which may have influenced the outcome of the study. Ratings of patient change were undertaken by the carer giving the intervention and not independently. The study does not indicate significant benefits in improving instrumental activities of daily living. 1+

3.3 COGNITIVE STIMULATION

Cognitive stimulation may occur informally through recreational activities, or formally through: [60-62]

- a programme of memory provoking, problem-solving and conversational fluency activities
- the spaced retrieval method
- face name training.

Formal cognitive stimulation produced a positive clinical impact on cognitive function in people with dementia. Although memory of specific pieces of information was improved it did not produce general benefits to memory function. These studies did not generalise to overall neuropsychological function and had short follow up.[60,61] 1+

B **Cognitive stimulation should be offered to individuals with dementia.**

Cognitive stimulation training can be carried out at home by a caregiver, with no risk to the person with dementia and with minimal training/education of the carer.

3.4 ENVIRONMENTAL DESIGN

Residential unit design, such as corridor configuration, can influence restlessness, anxiety and disorientation in institutionalised people with dementia.[63] Given that people with dementia experience increasing memory impairment and cognitive decline it is important to have an environment that aids orientation.

A descriptive systematic review of the design of environments for people with dementia including cohort, quasi-experimental and longitudinal studies, cross-sectional surveys and one-off case studies showed that changes in environment can have a positive impact on associated symptoms of dementia.[64] Findings from the studies are impaired by the absence of comparison groups, comparison between non-equivalent groups and small sample size.

The studies examined:

- environmental comparisons
- design features
- environmental services and policies
- problem behaviours in people with dementia in different physical environments.

3

The most common outcome measures were impact on problem behaviours, on ADLs and on cognitive and social function. Physical environment interventions including simple modifications such as signage and homelike environments resulted in positive outcomes in patients' ADLs, behaviour, and orientation. Small scale group living also had a positive therapeutic impact.[64]

☑ Measures which should be considered when planning an environment for people with dementia include:

- incorporating small size units
- separating non-cognitively impaired residents from people with dementia
- offering respite care as a complement to home care
- relocating residents, when necessary, in intact units rather than individually
- incorporating non-institutional design throughout the facility and in dining rooms in particular
- moderating levels of stimulation
- incorporating higher light levels
- using covers over fire exit bars and door knobs to reduce unwanted exiting
- incorporating outdoor areas with therapeutic design features
- considering making toilets more visible to potentially reduce incontinence
- eliminating factors that increase stress when bathing.

3.5 MULTISENSORY STIMULATION AND COMBINED THERAPIES

It has been suggested that because dementia often results in an alteration of several sensory modalities, less is to be gained by an intervention designed to deal with a single sense than can be gained through multisensory stimulation. This approach uses a variety of equipment such as lighting effects, relaxing music, recorded sounds, massage cushions, tactile surfaces and fragrances to create a multisensory environment.

Other studies have used combinations of massage, aromatherapy with essential oils, such as lavender and lemon balm, and music.

3.5.1 AROMATHERAPY

Few RCTs exploring the use of aromatherapy in people with dementia were identified. *Melissa officinalis* (lemon balm) was shown to have a positive effect on agitation although patients in this study continued to receive neuroleptic medication with dose adjustments possible during the study period, confounding the results.[65]

1-

Use of *Lavendula officinalis* (lavender oil) has not been proven to reduce associated symptoms in people with dementia.[66,67]

There is insufficient evidence for the efficacy of aromatherapy in reducing the core and associated symptoms of dementia and more well controlled trials are needed. Aromatherapy is not well tolerated by all patients who receive it.[66] The non-randomised evidence demonstrates both benefit and harm for aromatherapy and the common assumption that aromatherapy does no harm may not be correct.

☑ In people with dementia who show behavioural disturbance despite the use of psychotropic medication, aromatherapy may influence behaviour but cannot be recommended as a direct alternative to antipsychotic drugs, nor for the reduction of specific behavioural problems.

☑ The use of aromatherapy to reduce associated symptoms in people with dementia should be discussed with a qualified aromatherapist who can advise on contraindications.

3.5.2 LIGHT THERAPY

Sleep disturbance in people with dementia can be particularly distressing for carers. Biological changes in the brain can disrupt the normal circadian rhythm and sleep/wake cycle. Bright light affects the production of melatonin, which may lessen these problems. Bright light therapy is a labour intensive intervention and there are problems in controlling the studies for staff interaction and in maintaining blinding.

Evidence from a series of small studies using bright light therapy shows its effect on cognitive function in patients with dementia is negligible (one of only six people).[68-71] 1-

Three RCTs were identified each using dim red light as a control.[69,70,72] Two studies used bright light either morning or evening and one a "dawn/dusk simulator".[70] No consistent benefit of bright light therapy is reported in the domains of sleep or agitation. 1-

☑ Bright light therapy is not recommended for the treatment of cognitive impairment, sleep disturbance or agitation in people with dementia.

3.5.3 MUSIC THERAPY

Evidence from a series of small under powered studies suggests that exposure to music, tailored to the individual's taste, can relieve agitation but not aggressive behaviour in people with dementia. It is not possible to determine whether the beneficial effect seen is the result of music therapy itself or other factors, such as the presence of the researcher.[73-77] 1- 2-

Music therapy is easy to implement, but more research is needed to determine whether it is beneficial to the person with dementia.

3.5.4 MULTISENSORY STIMULATION

Multisensory stimulation (MSS) is not tolerated by everyone. The differences in severity of dementia between the intervention and control groups in studies limit the conclusions that can be drawn.

Evidence from three RCTs showed some small benefit from using multisensory stimulation to relieve core symptoms in people with dementia.[78-80] Individuals exposed to multisensory environments showed less confusion and talked more spontaneously and in normal length sentences. 1+

Although no improvement was reported in psychotic behaviour, aggression and irritability, [79] in people with moderate dementia there was a small improvement in mood, apathy and restlessness for the duration of the session.[78,80] 1-

☑ For people with moderate dementia who can tolerate it, multisensory stimulation may be a clinically useful intervention.

☑ Multisensory stimulation is not recommended for relief of neuropsychiatric symptoms in people with moderate to severe dementia.

Evidence from one systematic review does not demonstrate a significant clinical effect in favour of the use of the commercially available sensory resource Snoezelen®.[81] 1+

Snoezelen® has a significant capital cost and is intensive with regard to staff time. Studies over a longer duration and with more carefully matched intervention and control groups would be required to assess any effect on concomitant resource utilisation such as medication or institutional care.

3.6 PHYSICAL ACTIVITIES

The suggested benefits of exercise programmes for people with dementia include improvements in ambulatory status, walking endurance and urinary continence, but there is a lack of good quality evidence to support this.

A well conducted meta-analysis showed that in people aged over 65 with dementia and cognitive impairment, exercise was associated with statistically significant positive outcomes.[82] The quality of the study was limited by small sample size and the absence of blinding. 1++

Evidence from patients in residential care suggests that a combination of conversation and exercise on a structured basis may reduce deterioration in mobility in people with dementia but there is no evidence to support the use of either intervention in isolation.[83] 1+

Overall the clinical impact of physical activities on core or associated symptoms of dementia is minimal.[83-86] 1-

☑ For people with dementia, a combination of structured exercise and conversation may help maintain mobility.

3.7 REALITY ORIENTATION THERAPY

Reality orientation therapy (ROT) is a psychosocial intervention widely used in the care of people with dementia. The purpose of ROT is to reorientate the person by means of continuous stimulation and repetitive orientation to the environment. This may be done formally in a daily group session, or informally using a way of communicating that is very individual to the person and involves orientation to time, place and person during every direct contact with the individual (24 hour method).

ROT may slow cognitive decline and delay nursing home placement.[87] The study found that therapy conducted over a long period using the 24 hour method had more benefits than the formal method. 2+

D **Reality orientation therapy should be used by a skilled practitioner, on an individualised basis, with people who are disorientated in time, place and person.**

3.8 RECREATIONAL ACTIVITIES

Recreational activities give an opportunity for people with dementia to engage in meaningful activity and are often used as a way of facilitating the individual's need for communication, self esteem, sense of identity and productivity. Activities used range from self expression through drawing, music, arts and crafts, to cooking, games and interacting with pets.

Evidence from one RCT suggests that recreational activities for people with dementia have a significant positive effect based on facial expression and body posture, although no effect on disruptive behaviour.[54] 1+

B **Recreational activities should be introduced to people with dementia to enhance quality of life and well-being.**

One study reported that there was a trend for individualised activities to have a positive effect on agitation.[88] The most successful activities for individuals with dementia utilised strengths from the individuals current functioning, and previous interests. 1-

☑ Individualised activities adapted to maximise the person's remaining abilities and based on previous interests may be more beneficial to people with dementia than generic activities.

3.9 SIMULATED PRESENCE

Simulated presence therapy attempts to keep the environment of a patient with dementia as familiar as possible to reduce anxiety and distress. It involves making a recording of a familiar person and playing it to the patient. The recorded voice is usually reassuring but the content can be varied depending upon the interests of the individual patient concerned.

For nursing home residents simulated presence therapy was associated with improved alertness but provided no clinical benefits compared to a placebo tape recording. [89] 1-

Simulated presence therapy is not effective for reduction of agitation in nursing home residents with severe dementia.

3.10 VALIDATION THERAPY

Validation therapy is an approach used to communicate with disorientated elderly people that involves acknowledging and supporting their feelings in whatever time and place is real to them, even if this may not correspond to their "here and now" reality. This differs from reality orientation therapy which aims to ground the person in the present reality.

Benefits claimed for patients through the use of validation therapy include: [90]

- restoration of self worth
- minimisation of the degree to which patients withdraw from the outside world
- promotion of communication and interaction with other people
- reduction of stress and anxiety
- stimulation of dormant potential
- help in resolving unfinished life tasks
- facilitation of independent living for as long as possible.

A systematic review of two RCTs and a further RCT showed no statistically significant or clinically relevant effects from using validation therapy with people with dementia. [90, 91] 1+

3.11 INTERVENTIONS LACKING EVIDENCE OF CLINICAL EFFECTIVENESS

The following non-pharmacological interventions lacked evidence of clinical effectiveness for the treatment of people with dementia:

- memory books
- reminiscence therapy.

4 Pharmacological interventions

The recommendations made in this section are based upon an assessment of the evidence for their clinical effectiveness. Issues of cost effectiveness have not been considered.

The central features of dementia are cognitive decline and impaired functional ability. A number of associated problems occur frequently, but not uniformly. These problems, sometimes referred to as behavioural and psychological symptoms of dementias or BPSD, can cause considerable distress for both patients and carers. The presence of symptoms such as agitation, irritability, sleep disturbance, delusions, hallucinations or aggression may precipitate admission to hospital or institutional care.

Traditionally these associated symptoms have been managed using antipsychotic medication, antidepressants or anxiolytic medication. Acetyl cholinesterase inhibiting drugs can also influence behaviour.

Table 2: Index to core and associated symptoms and pharmacological interventions

Core symptoms	Section
Cognitive decline	4.1.1-4.1.3, 4.3
Functional decline	4.1.1-4.1.3, 4.3
Social decline	4.1.2
Associated symptoms	
Agitation	4.4, 4.7
Aggression	4.6
Depression	4.5
Psychosis	4.1.1, 4.6
Repetitive vocalisation	no robust evidence identified
Sleep disturbance	no robust evidence identified
Non-specific behaviour disturbance	4.1.1-4.1.3, 4.3, 4.6
Other	
Side effects	4.4, 4.8.1, 4.8.4

4.1 CHOLINESTERASE INHIBITORS

Abnormalities in cholinergic neurones are prominent among the pathological changes in the brains of patients with AD. The impact of these abnormalities can be reduced by inhibiting the enzymatic breakdown of acetylcholine.[92]

Since the publication of SIGN 22,[1] several second generation cholinesterase inhibitors including donepezil, rivastigmine and galantamine have been introduced. Their relatively recent introduction means that there are few trials in which the efficacy of these drugs has been examined over a prolonged period of time.

One meta-analysis comparing the tolerability and the effect on cognition of donepezil, galantamine and rivastigmine in people with dementia concluded that the efficacy of the three drugs is similar.[93] Donepezil was associated with fewer study dropouts than either rivastigmine or galantamine, suggesting that donepezil may be better tolerated at therapeutic doses. The meta-analysis did not examine the effect of the drugs on either the function or the behaviour of people with dementia. 1++

At the case control study level, there is support for long term use of cholinesterase inhibitors to delay institutionalisation.[94] The cost of additional community services is not taken into account in this study, but savings in the cost of caring for patients in institutions may be substantial.

4.1.1 DONEPEZIL

There is a significant body of evidence to support the use of the cholinesterase inhibitor donepezil in people with mild to moderate Alzheimer's disease.[92,95,96] There is evidence to suggest that its efficacy may extend to the treatment of people with more severe forms of Alzheimer's disease.[97,98] 1++

B **Donepezil, at daily doses of 5 mg and above, can be used to treat cognitive decline in people with Alzheimer's disease.**

☑ Age and severity of Alzheimer's disease should not be contraindications to the use of donepezil.

A long term placebo controlled study confirms small benefits for donepezil over placebo in cognition and function despite being set up to involve periodic withdrawal of donepezil; an intervention designed to make patients deteriorate back to their baseline level.[99] No effect on time to institutionalisation or progression of disability in patients was noted. The magnitude of delay to institutionalisation and slowing of disease progression require further investigation. 1-

A systematic review of the use of donepezil in people with vascular dementia demonstrated some benefit to patients with mild to moderate cognitive impairment examined over a six month period.[100] 1+

There is a large body of consistent evidence indicating the effectiveness of donepezil in reducing psychotic symptoms and a limited number of behavioural problems in patients with mild to moderate dementia.[97,98,101-105] 1++ 1+

One small study of augmentation of perphenazine in patients with moderate dementia, with either open label donepezil or an increased dose of perphenazine suggests a possible antipsychotic effect of donepezil.[106] Longer term double blind controlled studies are required. 1+

B **Donepezil, at daily doses of 5 mg and above, can be used for the management of associated symptoms in people with Alzheimer's disease.**

Evidence for similar effects in patients with moderate to severe dementia is lacking, and for a diagnosis other than Alzheimer's disease is not available at the RCT level.

4.1.2 GALANTAMINE

Galantamine is effective for the maintenance of cognition in people with mild to moderate Alzheimer's disease.[95,107-112] There is evidence of some cognitive benefit to patients with mixed Alzheimer's disease and cerebrovascular disease.[107]

Higher doses of galantamine are more effective than lower doses, although there is no added benefit of doses in excess of 24 mg per day.[108,110] 1++

Slow dose escalation appears to improve tolerability to the drug.[109] One study suggests that the greatest benefit is achieved in patients with moderate dementia with an MMSE score of less than 18.[110]

B **Galantamine, at daily doses of 16 mg and above, can be used to treat cognitive decline in people with Alzheimer's disease and people with mixed dementias.**

☑ Galantamine should be used with slow escalation to doses of up to 24 mg.

Evidence from two large RCTs showed that galantamine has a significant positive impact on functional ability[113] and behaviour for people with Alzheimer's disease.[109] 1++ 1+

B **Galantamine, at daily doses of 16 mg and above, can be used for the management of associated symptoms in people with Alzheimer's disease.**

Evaluation of the efficacy of galantamine in people with moderate to severe dementia needs further research.

4.1.3 RIVASTIGMINE

In people with mild to moderately severe Alzheimer's disease, rivastigmine treatment showed significant benefits in cognitive and global function.[114-116] 1++

There is evidence from one study that the cognitive benefits of rivastigmine treatment were more robust in patients with moderately severe dementia.[117] 1++

B **Rivastigmine, at daily doses of 6 mg and above, can be used to treat cognitive decline in people with Alzheimer's disease.**

Rivastigmine is also effective in treating cognitive decline in people with dementia with Lewy bodies.[118] Discontinuation of rivastigmine leads to rapid decline in cognitive function within three weeks.[119] 1++ 1+

B **Rivastigmine, at daily doses of 6 mg and above, can be used to treat cognitive decline in people with dementia with Lewy bodies.**

In a trial examining the value of rivastigmine in patients with Alzheimer's disease and hypertension, the group of patients with hypertension showed more cognitive benefit than those without hypertension.[120] 1+

Rivastigmine may be effective in the management of associated symptoms in patients with AD.[114,121] 1++ 1+

In people with dementia with Lewy bodies, rivastigmine may be effective in reducing apathy, anxiety and hallucinations.[118] 1++

B **Rivastigmine, at daily doses of 6 mg and above, can be used for the management of associated symptoms in people with Alzheimer's disease and dementia with Lewy bodies.**

4.2 MEMANTINE

L-glutamate is the main excitory neurotransmitter in the central nervous system (CNS), implicated in neural transmission, learning, memory and neuronal plasticity. Enhancement of the excitory action of L-glutamate may play a role in the pathogenesis of Alzheimer's disease. Low affinity N-methyl-D-aspartate (NMDA) type receptor antagonists such as memantine may prevent excitatory amino acid neurotoxicity without interfering with the actions of glutamate that are necessary for learning and memory.[122]

The efficacy of memantine has been examined in people with moderate to severe Alzheimer's disease and mild to moderate vascular dementia.[123-127]

After six months of treatment with 20 mg of memantine per day, there was a small, although not clinically significant, benefit over a wide range of outcome measures in patients with mild to moderate vascular dementia. 1+

In patients with moderate to severe Alzheimer's disease there was a non-clinically significant positive effect from use of memantine on activities of daily living at six months.[122]

The Scottish Medicines Consortium (SMC) assessment of memantine concluded that the magnitude of any beneficial effect was small and the clinical importance unclear (www.scottishmedicines.org.uk).

There is currently insufficient evidence to recommend the use of memantine for the treatment of core or associated symptoms in people with dementia.

4.3 GINKGO

Products derived from the maidenhair tree, *Ginkgo biloba*, have been used in traditional Chinese medicines for centuries. Clinical trials have assessed its use for the treatment of cerebral dysfunction, age related cognitive decline and for slowing the progress of neurodegenerative disorders such as dementia.[128] Although a number of studies have looked at the potential benefit of *Ginkgo* the dose of the active ingredient is not standardised.

Ginkgo biloba is available in the UK without prescription.

A systematic review of data derived from the Cochrane Collaboration meta-analyses of the efficacies of *Ginkgo biloba*[128] and cholinesterase inhibitors [92,116,129] suggests that *Ginkgo* is less potent in establishing cognitive improvement than the cholinesterase inhibitors, though *Ginkgo* is tolerated as well as placebo.[130] | 1++

A number of studies demonstrate that using *Ginkgo* to treat dementia has a positive benefit on cognition and function.[128,131,132] | 1++ 1+

In patients with advanced AD the differences between *Ginkgo* and placebo are more pronounced.[132] | 1+

Patients may need to take *Ginkgo* for 52 weeks before there is an improvement in cognition and activities of daily living.[128] | 1++

Almost all studies show that *Ginkgo* is safe with few side effects, although two studies failed to demonstrate a clinical benefit.[133,134] | 1++

Ginkgo causes bleeding when combined with warfarin or aspirin, raises blood pressure when combined with a thiazide diuretic and possibly causes coma when combined with trazodone.[135] Further trials are required before a statement can be made about the effective dose of *Ginkgo* for the treatment of patients with dementia.[128]

☑ People with dementia who wish to use *Ginkgo biloba* should consult a qualified herbalist for advice and should be made aware of possible interactions with other prescribed drugs.

4.4 SALVIA

Historically, the herb *Salvia officinalis* is known for its soothing, calming effects and for its ability to improve cognition, especially memory.[136]

Salvia officinalis is available in the UK without prescription.

In one small RCT (39 participants), the effect of using *Salvia officinalis* to treat agitation in patients with Alzheimer's disease was small and non-significant[136] | 1+

Further trials are required before a statement can be made about the efficacy of *Salvia officinalis* for the treatment of agitation in people with Alzheimer's disease.

The benefits of *Salvia officinalis* over other pharmacological interventions have not been addressed.

☑ People with dementia who wish to use *Salvia officinalis* should consult a qualified herbalist for advice.

4.5 ANTIDEPRESSANTS

The use of antidepressants for patients with dementia accompanied by depressive symptoms is widespread, but their effect on depression and cognitive function is uncertain.[137]

Research into the use of antidepressants for patients with depression and dementia is lacking. Evidence for the use of older tricyclic antidepressants (clomipramine and imipramine) from a well conducted systematic review is weak.[137] The meta-analysis was based on a very small number of studies with small sample sizes investigating drugs not commonly used in clinical practice. One of the included studies evaluated sertraline, a selective serotonin reuptake inhibitor (SSRI). Although this study shows significant differences in favour of treatment, numbers are small. 1+

The systematic review found no evidence for the use of more recent antidepressants such as venlafaxine or mirtazapine in patients with clearly defined dementia and comorbid depression.[137]

D **Antidepressants can be used for the treatment of comorbid depression in dementia providing their use is evaluated carefully for each patient.**

4.6 ANTIPSYCHOTICS

Conventional antipsychotic drugs such as chlorpromazine and haloperidol have traditionally been used to treat behavioural problems associated with dementia. Side effects such as sedation, movement disorder and increased confusion are all recognised. Concern has been expressed that the use of these drugs accelerates decline in Alzheimer's disease but a causal effect has not been established.[138]

A meta-analysis of the use of conventional antipsychotics showed that they are very effective for treating behavioural disorders associated with dementia, with a number needed to treat (NNT) of four to five.[139] 1+

Of the older antipsychotics, haloperidol is the most commonly assessed drug. Evidence suggests that it is useful in the control of aggression in people with dementia. The development of side effects may curb its more widespread use.[140] 1+

A **If necessary, conventional antipsychotics may be used with caution, given their side effect profile, to treat the associated symptoms of dementia.**

The atypical antipsychotics, olanzapine and risperidone are useful in the management of psychotic symptoms, aggression and other behavioural problems associated with dementia. [141] There is no clear evidence from head-to-head studies which indicates a superior efficacy or adverse event profile by comparison to conventional antipsychotics. 1++

Although evidence supports the use of olanzapine and risperidone in the management of BPSD, particularly psychosis and aggression, these drugs are not currently recommended by the Medicines and Healthcare products Regulatory Agency (MHRA) due to concerns about serious adverse events, particularly stroke.[142]

☑ Atypical antipsychotics with reduced sedation and extrapyramidal side effects may be useful in practice, although the risk of serious adverse events such as stroke must be carefully evaluated.

Stroke risk associated with other atypical antipsychotics and conventional antipsychotics has not been clearly established.

Practitioners should be aware that up to 60% of patients with dementia with Lewy bodies suffer adverse reactions to antipsychotic drugs.[143]

In patients on stable antipsychotic regimens, who are free from behavioural disturbances, withdrawal of antipsychotic treatment may not be associated with relapse.[144]

☑
- An individualised approach to managing agitation in people with dementia is required.
- Where antipsychotics are inappropriate cholinesterase inhibitors may be considered.
- In patients who are stable antipsychotic withdrawal should be considered.

4.7 TRAZODONE

One small RCT of trazodone showed reduction in agitation when accompanied by depressive symptoms in patients with dementia.[145] The evidence is insufficient to recommend its use for other depressive symptoms.

☑ Trazodone may be considered for patients with depressive symptoms and dementia associated agitation.

4.8 CLINICALLY INEFFECTIVE INTERVENTIONS

4.8.1 ANTI-INFLAMMATORIES

Evidence suggests that endogenous inflammatory responses have a role in the pathogenesis of Alzheimer's disease.[146]

One systematic review showed that anti-inflammatories do not slow progression in cognitive decline and have significant side effects such as gastric ulceration, renal deterioration in patients with renal problems and respiratory problems in people with asthma.[147] 1++

A **Anti-inflammatories are not recommended for treatment of cognitive decline in people with AD.**

Evidence from one study into associated symptoms related to memory, disruptive behaviour and depressive behaviour in people with Alzheimer's disease revealed a clinical effect after treatment with hydroxychloroquine.[146] In the treatment group there was an insignificant decline in the progression of early/mild Alzheimer's disease. These findings were not applicable to the whole spectrum of disease severity. 1++

B **Hydroxychloroquine is not recommended for the treatment of associated symptoms in people with dementia.**

There is evidence to suggest that the glucocorticoid prednisolone is ineffective for the treatment of associated symptoms in people with Alzheimer's disease. There may be a trend towards more severe behavioural events in patients treated with prednisolone.[148] 1++

A **Prednisolone is not recommended for the treatment of associated symptoms in people with Alzheimer's disease.**

4.8.2 MELATONIN

Exogenous melatonin is not effective in reducing sleep disturbance associated with dementia.[149,150] Melatonin treatment does not affect total time asleep, median number of awakenings or sleep efficiency in patients with dementia.[149] 1+ 2++

4.8.3 OESTROGEN

Dementia, particularly AD, is more common in postmenopausal women than any other population subgroup. Treatment with oestrogen has been proposed as a possible therapeutic agent for treatment of AD in women.

There is evidence to suggest that oestrogen is ineffective for the prevention of cognitive decline in women with dementia.[151,152] | 1++ 1+

Although one small RCT (16 participants) showed a favourable effect of oestrogen on associated problems in people with dementia,[153] two further studies (170 participants) point to ineffectiveness of the treatment.[154,155] | 1++ 1+

B **Oestrogen is not recommended for the treatment of associated symptoms in women with dementia.**

4.8.4 PHYSOSTIGMINE

Compared to placebo, there was no clinical benefit in treating people with dementia with physostigmine. Physostigmine has a short half-life and significant adverse reactions.[156-158] | 1- 1+

4.8.5 SELEGILINE

Selegiline inhibits monoamine oxidation and its potential use as a neuroprotective agent in Alzheimer's disease was suggested after reports of elevated monoamine oxidase B (MAO-B) activity in patients with AD compared to healthy older people.[159] A large body of evidence revealed no clinically meaningful benefit from the drug selegiline in the treatment of Alzheimer's disease.[159,160] | 1++

A **Selegiline is not recommended for the treatment of core or associated symptoms in people with Alzheimer's disease.**

4.9 INTERVENTIONS LACKING EVIDENCE OF CLINICAL EFFECTIVENESS

4.9.1 ANTICONVULSANTS

No robust evidence was identified to suggest that valproate is effective in reducing associated symptoms in people with dementia.[161] | 1++

A **Valproate is not recommended for the treatment of behavioural symptoms associated with dementia.**

One small RCT suggested carbamazepine reduced behavioural problems associated with severe dementia.[162] | 1-

An open label study of gabapentin showed no statistical significance in outcome measures on completion of the study.[163] | 3

☑ Anticonvulsants may be considered for the symptomatic treatment of seizures or myoclonus associated with dementia but are not recommended for other symptoms of dementia.

4.9.2 ASPIRIN

Aspirin is widely prescribed for the secondary prevention of vascular diseases, for example, stroke, myocardial infarction and peripheral arterial disease.

A Cochrane systematic review identified no randomised controlled evidence that aspirin benefits patients with vascular dementia in a similar way. There is a risk that it may increase the frequency of intracranial haemorrhage.[164] 1++

Many people with a diagnosis of dementia (especially vascular dementia) may also have a history of stroke, myocardial infarction or peripheral arterial disease.

☑ Aspirin is only recommended in people with vascular dementia who have a history of vascular disease.

Further research is needed to address the possible effectiveness of aspirin in people with vascular dementia but no history of stroke, myocardial infarction or peripheral arterial disease.

4.9.3 BENZODIAZEPINES

No systematic reviews or RCTs examining the usefulness of benzodiazepines in the management of associated symptoms of dementia, including anxiety, were identified.

4.9.4 LITHIUM

No RCTs of the use of lithium in people with dementia were identified. Small open studies produced conflicting results probably related to the size of the study.

☑ In the absence of concurrent evidence of bipolar affective disorder lithium is not recommended for the reduction of behavioural problems in dementia.

4.9.5 OTHER INTERVENTIONS

The following pharmacological interventions lacked evidence of clinical effectiveness for the treatment of people with dementia:

- acetyl-L-carnitine[165-168]
- cerebrolysin[169]
- nicergoline[170]
- lecithin.[171]

5 Information for discussion with patients and carers

5.1 SUPPORTIVE INFORMATION FOR PATIENTS AND CARERS

The research literature does not provide a clear consensus regarding the type of information people with dementia and their carers need at different stages of their journey of care. There is a consensus that both people with dementia and their carers are entitled to receive relevant information.[172,173]

There may be a difference between the objective problems observed by clinicians and the patients' and carers' subjective need for information. Information should not only include issues considered relevant by clinicians, but be tailored to the needs of patients and carers.[172] 2+

C **Patients and carers should be offered information tailored to the patient's perceived needs.**

☑ Good communication between healthcare professionals, patients and carers is essential.

5.1.1 DISCLOSURE OF DIAGNOSIS

Most of the evidence regarding information sharing relates to the point of diagnosis and how diagnosis is disclosed to individuals and carers. Reasons to inform individuals tend to be based on respect for patient autonomy and the value of truth telling in familial and professional relationships.[173] 4

Evidence for diagnostic disclosure is inconsistent, limited and at variance with guidance regarding disclosure, with the perspective of people with dementia being largely neglected.[172] It was found to be more common for carers to be routinely given information than patients, regardless of who delivered the information. 2+

When their views and their reaction to their diagnosis were sought, most people with dementia identified positive opportunities for future planning, understanding what was happening to them and developing coping strategies with, for example, memory aids or social supports.[172] 2+

C **Healthcare professionals should be aware that many people with dementia can understand their diagnosis, receive information and be involved in decision making.**

Practitioners involved in assessing and diagnosing dementia are responsible for what people know of their diagnosis, or whether they know their diagnosis at all. Diagnosis disclosure can lead to mixed reactions, such as shock, distress, ambivalence or confirmation of existing suspicions. 2+

Uncertain diagnosis leaves questions unanswered and late diagnosis was reported by a group of 20 carers as being of little benefit to the person with dementia. [172]

C **Healthcare professionals should be aware that some people with dementia may not wish to know their diagnosis.**

Whilst people have the right not to know their diagnosis, inappropriate withholding of the diagnosis, was a source of distress for some.[172] 2+

Formal permission to disclose the diagnosis to carers should be sought.[172]

Arguments against disclosure often cite the lack of useful treatments and the uncertainty of diagnosis and prognosis. Disclosure to patients may also be considered unhelpful if cognitive deficits limit their ability to understand the meaning and implications of their diagnosis. A consensus that diagnosis disclosure is favoured appears to be emerging among professional organisations, although practice lags behind.[173] 4

D **Healthcare professionals should be aware that in some situations disclosure of a diagnosis of dementia may be inappropriate.**

☑ The wishes of the person with dementia should be upheld at all times.

☑ The diagnosis of dementia should be given by a healthcare professional skilled in communication or counselling.

☑ Where diagnosis is not disclosed there should be a clear record of the reasons.

5.1.2 INFORMATION AT OTHER STAGES OF THE PATIENT JOURNEY

Little evidence was identified regarding information sharing at any other time or for any other issue for patients. Further studies on the process and outcome of disclosure are needed. Research is required to determine a uniform understanding of the issues, although preferences for practice may be highly individual.

Information provision at other stages of the patient's journey of care is generally more focused on carer needs than that of the patient.

In decision making, people with mild dementia are more involved, largely in a collaborative role. Beyond that carers generally make final decisions.[174] 3

☑ Patients and carers should be provided with information about the services and interventions available to them at all stages of the patient's journey of care.

☑ Information should be offered to patients and carers in advance of the next stage of the illness.

Methods of disseminating information which may be appropriate for people with dementia and their carers include:

- written information
- individual education programmes
- group education programmes
- counselling
- telemedicine service
- communication workshops
- cognitive behaviour therapy (CBT)
- stress management
- combinations of the above.

Not everyone benefits from such interventions. Further studies are required to identify confounding factors so that practitioners can provide relevant information tailored to the needs of the person and their carer, to improve understanding and enable more informed decision making at all stages.

5.2 SOURCES OF FURTHER INFORMATION FOR PATIENTS AND CARERS

5.2.1 NATIONAL DEMENTIA ORGANISATIONS

Alzheimer Scotland – Action on Dementia
22 Drumsheugh Gardens, Edinburgh EH3 7RN
Tel: 0131 243 1453 • 24 hour freephone helpline 0808 808 3000
Website: www.alzscot.org.uk

Alzheimer's Society
Gordon House, 10 Greencoat Place, London SW1P 1PH
Tel: 020 73060606 • Helpline: 0845 300 0336
Website: www.alzheimers.org.uk

Alzheimer's Research Trust
Livanos House, Granhams Road, Cambridge CB2 5LQ
Tel: 01223 843 899
Email: enquiries@alzheimers-research.org.uk

Pick's Disease Support Group
Email: info@pdsg.org.uk • Website: www.pdsg.org.uk

Regional contacts are listed on the website.

5.2.2 NATIONAL GENERAL ORGANISATIONS

Age Concern Scotland
Causewayside House, 160 Causewayside, Edinburgh EH9 1PR
Tel: 0845 833 0200 • Fax: 0845 833 0759
Email: enquiries@acscot.org.uk • Website: www.ageconcernscotland.org.uk

Chest, Heart and Stroke Scotland
65 North Castle Street, Edinburgh EH2 3LT
Tel: 0131 225 6963
Website: www.chss.org.uk

Help the Aged in Scotland
11 Granton Square, Edinburgh EH5 1HX
Tel: 0131 551 6331
Email: infoscot@helptheaged.org.uk

Mental Health Foundation Scotland
5th Floor, Merchants House, 30 George Square, Glasgow G2 1EG
Tel: 0141 572 0125
Email: Scotland@mhf.org.uk • Website: www.mentalhealth.org.uk

5.2.3 SPECIALIST UNITS

Dementia Services Development Centre
Iris Murdoch Building, University of Stirling, FK9 4LA
Tel: 01786 467740
Website: www.dsdc.stir.ac.uk

National CJD Surveillance Unit
Western General Hospital, Crewe Road, Edinburgh EH4 2XU
CJD Support Helpline: 01630 673 973
Website: www.cjd.ed.ac.uk

A CJD Support Network providing help and support for people with CJD and their carers.

5.2.4 OTHER USEFUL RESOURCES

Carers Scotland
91 Mitchell Street, Glasgow G1 3LN
Tel: 0141 221 9141
Email: info@carerscotland.org • Website: www.carerscotland.org

Carers UK
Ruth Pitten House, 20-25 Glasshouse Yard, London EC1A HJT
Tel: 02074908818 • Carers Line: 0808 8008 7777
Website: www.carersuk.org

Crossroads (Scotland)
24 George Street, Glasgow G2 1EG
Tel: 0141 226 3793 • Fax: 0141 221 7130 • Helpline: 0141 353 6504.
Website: www.crossroads-scotland.co.uk

Crossroads is a Scottish charity with 49 local schemes providing care and short breaks for Scotland's carers. Crossroads provides practical help to any carer, regardless of the age, disability or illness of the person being cared for. The carers' information and support line offers advice, support and practical help.

DVLA
Drivers Medical Group
DVLA, Swansea, SA99 1DL
Tel: 0870 600 0301 (Monday to Friday, 8.15am to 4.30pm) • Fax: 0845 850 0095
Email: eftd@dvla.gsi.gov.uk • Website: www.dvla.gov.uk/drivers/dmed1.htm

Medical condition(s) can be notified by telephone, fax and email, quoting full name, date of birth and/or driver number (if known), and medical case number if available.

NHS Health Scotland
Woodburn House, Canaan Lane, Edinburgh, EH10 4SG
Tel: 0131 526 5500 • Textphone: 0131 536 5503 • Fax: 0131 536 5501
Website: www.healthscotland.com

NHS 24
Tel: 08454 24 24 24 • Textphone: 18001 08454 24 24 24
Website: www.nhs24.com

NHS 24 is a 24 hour nurse-led helpline providing confidential healthcare advice and information.

Office of the Public Guardian
Hadrian House, Callendar Business Park, Callendar Road, Falkirk FK11XR
Tel: 01324 678 300 • Fax: 01324 678 301
Email: opg@scotcourts.gov.uk • Website: www.publicguardian-scotland.gov.uk

Provides information and advice to guardians, attorneys or other authorised persons about the performance of functions in terms of the "Adults with Incapacity (Scotland) Act".

Public Guardianship Office
Archway Tower, 2 Junction Road, London N19 5SZ
Tel: 0845 330 2900 • Fax: 0870 739 5780 • Textphone: 020 7664 7755
Email: custserv@guardianship.gsi.gov.uk • Website: www.guardianship.gov.uk/

Princess Royal Trust for Carers (Scottish Office)
Campbell House, 215 West Campbell Street, Glasgow G2 4TT
Tel: 0141 2215 066
Email: infoscotland@carers.org • Website: www.carers.org

Samaritans
National Helpline Tel: 08457 909090

Offers support to those in distress/despair/suicidal who need someone to talk to. 24 hour service.

Scottish Independency Advocacy Alliance
138 Slateford Road, Edinburgh, EH14 1LR
Tel: 0131 455 8183

5.3 USEFUL PUBLICATIONS FOR PATIENTS AND CARERS

Are you worried about your memory?
NHS Health Scotland

A leaflet to help people decide whether they should seek medical advice. Copies are available by contacting 0131 526 5500 or can be accessed on the internet. www.hebs.com

Getting help from your doctor – a guide to people worried about their memory, people with dementia and carers.
Alzheimer Scotland

Copies are free to people with dementia and their families or can be accessed on the internet. www.alzscot.org//info/gettinghelpfromyourgp

The right to know? sharing the diagnosis of dementia.
Alzheimer Scotland

Copies are free to people with dementia and their families or can be accessed on the internet. www.alzscot.org//policy/rtksum

Dementia – money and legal matters a guide.
Alzheimer Scotland

Copies are free to people with dementia and their families or can be accessed on the internet. www.alzscot.org/info/mandlegchoice

Facing dementia – useful information for people with dementia.
NHS Health Scotland.

Copies available by contacting 0131 526 5500 or can be accessed on the internet. www.hebs.com

Don't make the journey alone.
Alzheimer Scotland

A booklet written by people with dementia for people with dementia. Single copies are free to people with dementia and their families or can be accessed on the internet. www.alzscot.org/info/dontmake

Keeping safe – a guide to safety when someone with dementia lives alone.
NHS Health Scotland

Copies available by contacting 0131 526 5500 or can be accessed on the internet. www.hebs.com

A positive choice – choosing long stay care for a person with dementia.
Alzheimer Scotland

Single copies are free to people with dementia and their families or can be accessed on the internet. www.alzscot.org/info/positive

Getting help – caring for someone with dementia.
Alzheimer Scotland

Single copies free to people with dementia and their families or can be accessed on the internet.
www.alzscot.org/info/gettinghelp

Safety in the home.
Alzheimer Scotland

Single copies are free to people with dementia and their families or can be accessed on the internet.
www.alzscot.org/info/safety

All about dementia.
Mental Health Foundation

Single copies are free to individuals or available on the internet.
www.mentalhealth.org.uk

Because you care.
Mental Health Foundation

Single copies are free to individuals or available on the internet.
www.mentalhealth.org.uk

The milk's in the oven.
Mental Health Foundation

Single copies are free to individuals or available on the internet.
www.mentalhealth.org.uk

Learning to speak Alzheimer's: the new approach to living positively with Alzheimer's disease.
Joanne Koenig-Coste, Vermillion

Alzheimer's disease and memory loss explained - a guide for patients and carers.
Alistair Burns, Sean Page and Jane Winter, Altman Press. 2001

6 Implementation, resource implications and audit

6.1 LOCAL IMPLEMENTATION

Implementation of national clinical guidelines is the responsibility of local NHS organisations and is an essential part of clinical governance. It is acknowledged that not every guideline can be implemented immediately on publication, but mechanisms should be in place to ensure that the care provided is reviewed against the guideline recommendations and the reasons for any differences assessed and, where appropriate, addressed. These discussions should involve both clinical staff and management. Local arrangements may then be made to implement the national guideline in individual hospitals, units and practices, and to monitor compliance. This may be done by a variety of means including patient-specific reminders, continuing education and training, and clinical audit.

Key areas to be considered for implementation are:

- the recognition of comorbid depression in dementia by primary care which will require significant training input
- the routine use of structural imaging which will require more access to imaging facilities given the nature of dementia and the prospect of treatment
- widespread availability of information for patients and carers. This needs to extend beyond GPs' surgeries and appear in areas where older people go, such as libraries, post offices or supermarkets
- clear strategies by NHS boards for the funding of cholinesterase inhibitors and associated infrastructure
- development of caregiver training programmes.

6.2 RESOURCE IMPLICATIONS

Group members identified 10 recommendations in the guideline which have resource implications for NHSScotland.

FROM SECTION 2.4:

C **Structural imaging should ideally form part of the diagnostic workup of patients with suspected dementia.**

There is wide variation in the use of imaging in the diagnosis of dementia. Implementing this recommendation is therefore likely to be associated with a significant increase in the number of scans performed per annum.

Practice Team information statistics from Information Services (ISD) NHS National Services Scotland provides an estimate of the incidence of dementia in people aged 45 and over (www.isdscotland.org/general_practice_info). For women this is estimated at 1.5/1,000 and for men at 0.6/1,000. Using Registrar General for Scotland population estimates, this equates to 650 new cases in men and 2,050 new cases in women per annum.

The resource implications of this recommendation will vary by NHS Board, but may have an impact on staff and equipment required and on the waiting time for such scans.

FROM SECTION 2.6:

B **Neuropsychological testing should be used in the diagnosis of dementia, especially in patients where dementia is not clinically obvious.**

Neuropsychological testing is carried out by clinical psychologists and is estimated to take between 90 minutes and three hours. The availability of such testing and clinical psychology services varies widely across Scotland.

It is estimated by the guideline development group that 10% of all patients would require neuropsychological testing. This equates to approximately 270 people across Scotland per annum. It is likely that this recommendation would require an increase in the availability of clinical psychology services in some areas.

FROM SECTION 3.2:

B **Caregivers should receive comprehensive training on interventions that are effective for people with dementia.**

Training of carers can take a variety of forms with the most common types being one to one observation by a community psychiatric nurse (CPN) and group training. The availability of such training varies across Scotland. It is likely that this recommendation would have some impact on CPN time.

FROM SECTION 4.1:

B **Donepezil, at daily doses of 5 mg and above, can be used to treat cognitive decline in people with Alzheimer's disease.**

B **Donepezil, at daily doses of 5 mg and above, can be used for the management of associated symptoms in people with mild to moderate dementia.**

B **Galantamine, at daily doses of 16 mg and above, can be used to treat cognitive decline in people with Alzheimer's disease and people with mixed dementias.**

B **Galantamine, at daily doses of 16mg and above, can be used for the management of associated symptoms in people with Alzheimer's disease.**

B **Rivastigmine, at daily doses of 6mg and above, can be used to treat cognitive decline in people with Alzheimer's disease.**

B **Rivastigmine, at daily doses of 6mg and above, can be used to treat cognitive decline in people with dementia with Lewy bodies.**

B **Rivastigmine, at daily doses of 6 mg and above, can be used for the management of associated symptoms in people with Alzheimer's disease and dementia with Lewy bodies.**

In Scotland in 2006 it was estimated that there are 62,000 people with dementia. During 2004/5 approximately 65,000 prescriptions for cholinesterase inhibitors were issued, equating to expenditure of £6.9 million. The guideline development group estimates that about 20% of new cases are currently prescribed these drugs and that implementation of these recommendations would increase this to 60% of new cases. An additional 40% of new patients per annum equates to around 1,080 people. On the other hand, the recommendations are anticipated to rationalise the long term use.

The National Institute for Health and Clinical Excellence (NICE) are reviewing their technology appraisal of the use and cost-effectiveness of cholinesterase inhibitors for people with dementia (www.nice.org.uk). The applicability to NHSScotland of this appraisal will be addressed by NHS Quality Improvement Scotland.

6.3 KEY POINTS FOR AUDIT

Key areas to consider for audit are:

- how many patients who undergo memory assessment are being referred for neuroimaging and/or neuropsychology?
- what mechanisms are in place in primary care to ensure early identification of patients with dementia?
- what is the frequency of use and effectiveness of non-pharmacological interventions?
- are complex caregiver intervention programmes being developed and implemented appropriately?
- are antipsychotic drugs being prescribed appropriately?

6.4 RECOMMENDATIONS FOR RESEARCH

The use of cholinesterase inhibitors for the treatment of dementia is relatively new and there is a need for research into their long term use and comparisons between therapies. Little has been described about the type and severity of dementia of the patients participating in clinical trials, the frequency of adverse events and the numbers of responders and non-responders to the drugs.

Other areas where evidence is lacking have been identified in the course of developing this guideline

- can matching of clinical and radiological appearances improve the identification of subgroups of people with Alzheimer's disease and those with mixed Alzheimer vascular dementia?
- does the availability of patient/carer information lead to earlier access to services?
- does contact with psychiatric services when a patient has early dementia lead to improved access to services as the patient deteriorates?
- what is the benefit from non-complex psychosocial intervention?
- what are the alternatives to drug treatment?
- what is the most effective method for treating pain in patients with dementia?
- what is the most effective way to prevent falls in patients with dementia?
- what is the most effective way to treat delirium in dementia, where patients are at greater risk of morbidity and mortality?
- what is the effectiveness of using antidepressants to treat depression in people with dementia?
- what are effective interventions for the treatment of repetitive vocalisation?
- the development of a set of brief tests which can enhance the diagnostic utility of tests such as the MMSE or clock drawing
- what is the effectiveness of caregiver interventions on the carer?
- what is the impact of the social context of living with dementia?
- are statins and antihypertensives effective treatments for people with vascular dementia with no history of stroke or myocardial infarction?
- what physical exercise programmes help ADL and maintain well-being?

7 Development of the guideline

7.1 INTRODUCTION

SIGN is a collaborative network of clinicians, other healthcare professionals and patient organisations and is part of NHS Quality Improvement Scotland. SIGN guidelines are developed by multidisciplinary groups of practicing clinicians using a standard methodology based on a systematic review of the evidence. Further details about SIGN and the guideline development methodology are contained in "SIGN 50; A Guideline Developer's Handbook", available at **www.sign.ac.uk**

7.2 THE GUIDELINE DEVELOPMENT GROUP

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The membership of the guideline development group was confirmed following consultation with the member organisations of SIGN. Declarations of interests were made by all members of the guideline development group. Further details are available from the SIGN Executive.

7.3 ACKNOWLEDGEMENTS

SIGN is grateful to the following former members of the guideline development group and SIGN Executive.

Dr Jennifer Borthwick	*Clinical Psychologist, Hartwood Hill Hospital, Shotts*
Mr Andy Lowndes	*Nursing Fellow, Nursing and Midwifery Research Centre, Glasgow Caledonian University*
Dr Jean Reid	*Consultant Old Age Psychiatrist, Glenkirk Centre, Drumchapel Hospital, Glasgow*
Mr Hugh Toner	*Clinical Psychologist, Stratheden Hospital, Cupar*
Dr Ali El-Ghorr	*Programme Manager, SIGN*
Ms Gemma Healy	*Information Officer, SIGN*

7.4 SYSTEMATIC LITERATURE REVIEW

The evidence base for this guideline was synthesised in accordance with SIGN methodology. A systematic review of the literature was carried out using an explicit search strategy devised by a SIGN Information Officer. Databases searched include Medline, Embase, Cinahl, PsychINFO, and the Cochrane Library. The year range covered was 1997-2004. Internet searches were carried out on various websites including the New Zealand Guidelines Programme, NELH Guidelines Finder, and the US National Guidelines Clearinghouse. The Medline version of the main search strategies can be found on the SIGN website, in the section covering supplementary guideline material. The main searches were supplemented by material identified by individual members of the development group. Each of the selected papers was evaluated by two members of the group using standard SIGN methodological checklists before conclusions were considered as evidence.

7.5 CONSULTATION AND PEER REVIEW

7.5.1 NATIONAL OPEN MEETING

A national open meeting is the main consultative phase of SIGN guideline development, at which the guideline development group presents its draft recommendations for the first time. The national open meeting for this guideline was held on 2 February 2004 and was attended by representatives of all the key specialties relevant to the guideline. The draft guideline was also available on the SIGN website for a limited period at this stage to allow those unable to attend the meeting to contribute to the development of the guideline.

7.5.2 SPECIALIST REVIEW

This guideline was also reviewed in draft form by the following independent expert referees, who were asked to comment primarily on the comprehensiveness and accuracy of interpretation of the evidence base supporting the recommendations in the guideline. SIGN is very grateful to all of these experts for their contribution to the guideline.

Mr Ian Anderson	*Ward Manager, Glen-O-Dee Hospital, Banchory*
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7.5.3 SIGN EDITORIAL GROUP

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Abbreviations

ACE	Addenbrooke's Cognitive Examination
AD	Alzheimer's disease
ADL	activities of daily living
BPSD	behavioural and psychological symptoms of dementias
CBT	cognitive behaviour therapy
CJD	Creutzfeldt-Jakob disease
CNS	central nervous system
CPN	community psychiatric nurse
CSF	cerebrospinal fluid
CT	computed tomography
DLB	dementia with Lewy bodies
DSM-IV	Diagnostic and Statistical Manual, 4th edition
EEG	electroencephalography
FTD	fronto-temporal dementia
IQCODE	Informant Questionnaire on Cognitive Decline in the Elderly
ISD	Information Services NHS National Services Scotland
MAO-B	monoamine oxidase B
MHRA	Medicines and Healthcare products Regulatory Agency
MMSE	Mini-Mental State Examination
MRI	magnetic resonance imaging
MSS	multisensory stimulation
NICE	National Institute for Health and Clinical Excellence
NINCDS-ADRDA	National Institute of Neurologic, Communicative Disorders and Stroke–AD and related Disorders Association
NINDS-AIRENS	National Institute of Neurological Disorders and Stroke Association Internationale pour la Recherche et l'Enseignement en Neurosciences
NMDA	N-methyl-D-aspartate
NNT	number needed to treat
PET	positron emission tomography
RCT	randomised controlled trial
ROT	reality orientation therapy
SIGN	Scottish Intercollegiate Guidelines Network
SMC	Scottish Medicines Consortium
SPECT	single photon emission controlled tomography
SSRI	selective serotonin reuptake inhibitor
VaD	vascular dementia

Annex 1
Diagnostic criteria for dementia of the Alzheimer's type

This abbreviated version of the DSM-IV criteria is reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. 1994, American Psychiatric Association.[175]

A. The development of multiple cognitive deficits manifested by both:

   (1) memory impairment (impaired ability to learn new information or to recall previously learned information)

   (2) one (or more) of the following cognitive disturbances:

      (a) aphasia (language disturbance)

      (b) apraxia (impaired ability to carry out motor activities despite intact motor function)

      (c) agnosia (failure to recognize or identify objects despite intact sensory function)

      (d) disturbance in executive functioning (ie, planning, organizing, sequencing, abstracting).

B. The cognitive deficits in Criteria A1 and A2 each cause significant impairment in social or occupational functioning and represent a significant decline from a previous level of functioning.

C. The course is characterized by gradual onset and continuing cognitive decline.

D. The cognitive deficits in Criteria A1 and A2 are not due to any of the following:

   (1) other central nervous system conditions that cause progressive deficits in memory and cognition (eg, cerebrovascular disease, Parkinson's disease, Huntington's disease, subdural hematoma, normal-pressure hydrocephalus, brain tumor)

   (2) systemic conditions that are known to cause dementia (eg, hypothyroidism, vitamin B_{12} or folic acid deficiency, niacin deficiency, hypercalcemia, neurosyphilis, HIV infection)

   (3) substance-induced conditions.

E. The deficits do not occur exclusively during the course of a delirium.

F. The disturbance is not better accounted for by another Axis I disorder (eg, Major Depressive Disorder, Schizophrenia).

Code based on type of onset and predominant features:

With Early Onset: if delirium is superimposed on the dementia

290.11 With Delirium: if delirium is superimposed on the dementia

290.12 With Delusions: if delusions are the predominant feature

290.13 With Depressed Mood: if depressed mood (including presentations that meet full symptom criteria for a Major Depressive Episode) is the predominant feature. A separate diagnosis of Mood Disorder Due to a General Medical Condition is not given

290.10 Uncomplicated: if none of the above predominated in the current clinical presentation.

With Late Onset: if onset is after age 65 years.

290.3 With Delirium: if delirium is superimposed on the dementia

290.20 With Delusions: if delusions are the predominant feature

290.21 With Depressed Mood: if depressed mood (including presentations that meet full symptom criteria for a Major Depressive Episode) is the predominant feature. A separate diagnosis of Mood Disorder Due to a General Medical Condition is not given

290.0 Uncomplicated: if none of the above predominates in the current clinical presentation

Specify if:

With Behavioral Disturbance

Coding note: Also code 331.0 Alzheimer's disease on Axis III

Annex 2

Diagnostic criteria for the clinical diagnosis of probable Alzheimer's disease

The NINCDS-ADRDA criteria are intended to serve as a guide for the diagnosis of probable, possible, and definite Alzheimer's disease. [176]

I. Criteria for the clinical diagnosis of PROBABLE Alzheimer's disease:

- dementia established by clinical examination and documented by the Mini-Mental Test; Blessed Dementia Scale, or some similar examination, and confirmed by neuropsychological tests
- deficits in two or more areas of cognition
- progressive worsening of memory and other cognitive functions
- no disturbance of consciousness
- onset between ages 40 and 90, most often after age 65
- absence of systemic disorders or other brain diseases that in and of themselves could account for the progressive deficits in memory and cognition.

II. The diagnosis of PROBABLE Alzheimer's disease is supported by:

- progressive deterioration of specific cognitive functions such as language (aphasia), motor skills (apraxia), and perceptions (agnosia)
- impaired activities of daily living and altered patterns of behavior
- family history of similar disorders, particularly if confirmed neuropathologically
- laboratory results of:
  - normal lumbar puncture as evaluated by standard techniques
  - normal pattern or non-specific changes in EEG, such as increased slow-wave activity
  - evidence of cerebral atrophy on CT with progression documented by serial observation.

III. Other clinical features consistent with the diagnosis of PROBABLE Alzheimer's disease, after exclusion of causes of dementia other than Alzheimer's disease, include:

- plateaus in the course of progression of the illness
- associated symptoms of depression, insomnia, incontinence, delusions, illusions, hallucinations, catastrophic verbal, emotional, or physical outbursts, sexual disorders, and weight loss
- other neurologic abnormalities in some patients, especially with more advanced disease and including motor signs such as increased muscle tone, myoclonus, or gait disorder
- seizures in advanced disease
- CT normal for age.

IV. Features that make the diagnosis of PROBABLE Alzheimer's disease uncertain or unlikely include:

- sudden, apoplectic onset
- focal neurologic findings such as hemiparesis, sensory loss, visual field deficits, and incoordination early in the course of the illness
- seizures or gait disturbances at the onset or very early in the course of the illness.

Annex 3
The Hachinski Ischaemic Scale

These criteria for vascular dementia are based upon the Hachinski ischaemic score, originally derived on the basis of cerebral blood flow patterns in people with dementia. [177]

On the weighted scale a score of 7 or more is taken to indicate vascular dementia while a score of 4 or less suggests that this is an unlikely diagnosis.

Hachinski ischaemic score	
Abrupt onset	2
Stepwise progression	1
Fluctuating course	2
Nocturnal confusion	1
Relative preservation of personality	1
Depression	1
Somatic complaints	1
Emotional incontinence	1
History of hypertension	1
History of strokes	2
Evidence of associated atherosclerosis	1
Focal neurological symptoms	2
Focal neurological signs	2

Annex 4

Diagnostic criteria for the clinical diagnosis of probable vascular dementia

Research criteria for the diagnosis of VaD from NINDS-AIREN, intended as a guide for case definition in neuroepidemiologic studies, stratified by levels of certainty (definite, probable, and possible). [178]

I. Criteria for the clinical diagnosis of PROBABLE vascular dementia include all of the following:

1. *Dementia* defined by cognitive decline from a previously higher level of functioning and manifested by impairment of memory and of two or more cognitive domains (orientation, attention, language, visuospatial functions, executive functions, motor control, and praxis), preferably established by clinical examination and documented by neuropsychological testing; deficits should be severe enough to interfere with activities of daily living not due to physical effects of stroke alone.

   Exclusion criteria: cases with disturbance of consciousness, delirium, psychosis, severe aphasia, or major sensorimotor impairment precluding neuropsychological testing. Also excluded are systemic disorders or other brain diseases (such as AD) that in and of themselves could account for deficits in memory and cognition.

2. *Cerebrovascular disease*, defined by the presence of focal signs on neurologic examination, such as hemiparesis, lower facial weakness, Babinski sign, sensory deficit, hemianopia, and dysarthria consistent with stroke (with or without history of stroke), and evidence of relevant CVD by brain imaging (CT or MRI) *including multiple large-vessel infarcts or a single strategically placed infarct* (angular gyrus, thalamus, basal forebrain, or PCA or ACA territories), as well as *multiple basal ganglia and white matter lacunes* or *extensive periventricular white matter lesions*, or combinations thereof.

3. A *relationship between the above two disorders*, manifested or inferred by the presence of one or more of the following: (a) onset of dementia within 3 months following a recognized stroke; (b) abrupt deterioration in cognitive functions; or fluctuating, stepwise progression of cognitive deficits.

II. Clinical features consistent with the diagnosis of PROBABLE vascular dementia include: thefollowing:

(a) early presence of a gait disturbance (small-step gait or marche à petits pas, or magnetic, apraxic-ataxic or parkinsonian gait)

(b) history of unsteadiness and frequent, unprovoked falls

(c) early urinary frequency, urgency, and other urinary symptoms not explained by urologic disease

(d) pseudobulbar palsy

personality and mood changes, abulia, depression, emotional incontinence, or other subcortical deficits including psychomotor retardation and abnormal executive function.

III. Features that make the diagnosis of vascular dementia uncertain or unlikely include

(a) early onset of memory deficit and progressive worsening of memory and other cognitive functions such as language (transcortical sensory aphasia), motor skills (apraxia), and perception (agnosia), in the absence of corresponding focal lesions on brain imaging

(b) absence of focal neurologic signs, other than cognitive disturbance

(c) absence of cerebrovascular lesions on brain CT or MRI.

Annex 5

Consensus criteria for the clinical diagnosis of probable and possible dementia with Lewy bodies

These operational diagnostic criteria for dementia of the Lewy body type (DLB), were developed at a consensus meeting bringing together proponents of the previous diagnostic criteria from healthcare professionals in Nottingham and Newcastle.[179]

1. The central feature required for a diagnosis of DLB is progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational function. Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression. Deficits on tests of attention and of frontal sub-cortical skills and visuospatial ability may be especially prominent.

2. Two of the following core features are essential for a diagnosis of probable DLB, and one is essential for possible DLB:
    a. fluctuating cognition with pronounced variations in attention and alertness
    b. recurrent visual hallucinations that are typically well formed and detailed
    c. spontaneous motor features of parkinsonism.

3. Features supportive of the diagnosis are:
    a. repeated falls
    b. syncope
    c. transient loss of consciousness
    d. neuroleptic sensitivity
    e. systematised delusions
    f. hallucinations in other modalities.

4. A diagnosis of DLB is less likely in the presence of:
    a. stroke disease, evident as focal neurologic signs or on brain imaging
    b. evidence on physical examination and investigation of any physical illness or other brain disorder sufficient to account for the clinical picture.

Annex 6
Clinical diagnostic features of fronto-temporal dementia

Clinical and pathological criteria for fronto-temporal dementia. [180,181]

Core diagnostics include:

1. *Behavioural disorder*
   - insidious onset and slow progression
   - early loss of personal awareness (neglect of personal hygiene and grooming)
   - early loss of social awareness (lack of social tact, misdemeanours such as shop lifting)
   - early signs of disinhibition (such as unrestrained sexuality, violent behaviour, inappropriate jocularity, restless pacing)
   - mental rigidity and inflexibility
   - hyperorality (oral/dietary changes, food fads, excessive smoking and alcohol consumption, oral exploration of objects)
   - stereotyped and preservative behaviour (wandering, mannerisms such as clapping, singing, dancing, ritualistic preoccupations such as hoarding, toileting and dressing)
   - utilisation behaviour (unrestrained exploration of objects in the environment)
   - distractibility, impulsivity and impersistence
   - early loss of insight into the fact that the altered condition is due to a pathological change of own mental state.

2. *Affective Symptoms*
   - depression, anxiety, excessive sentimentality, suicidal and fixed ideation, delusion (early and evanescent)
   - hypochondriasis, bizarre somatic preoccupation (early evanescent)
   - emotional unconcern (emotional indifference and remoteness, lack of empathy and sympathy, apathy)
   - amimia (inertia, aspontaneity).

3. *Speech Disorder*
   - progressive reduction of speech (aspontaneity and economy of utterance)
   - stereotypy of speech (repetition of limited repertoire of words, phrases or themes)
   - echolalia and preservation
   - late mutism.

4. *Spatial orientation and praxis preserved* (intact abilities to negotiate the environment)

5. *Physical Signs*
   - early primitive reflexes
   - early incontinence
   - late akinesia, rigidity, tremor
   - low and labile blood pressure.

6. *Investigation*
   - normal EEG despite clinically evident dementia
   - brain imaging (structural or functional, or both) predominant frontal or anterior temporal abnormality or both
   - neuropsychology (profound failure on frontal lobe tests in the absence or severe amnesia, aphasia or perceptual spatial disorder).

Supportive diagnostic features include:

1. onset before 65
2. positive family history of similar disorder in a first degree relative
3. bulbar palsy, muscular weakness and wasting, fasciculations (motor neuron disease).

Diagnostic exclusion features include:

1. abrupt onset with ictal events
2. head trauma related to onset
3. early severe amnesia
4. early spatial disorientation, lost in surroundings, defective localization of objects
5. early severe apraxia
6. logoclonic speech with rapid loss of train of thought
7. myclonus
8. cortical bulbar and spinal deficits
9. cerebellar ataxia
10. choreo-athetosis
11. early, severe, pathological EEG
12. brain imaging (predominant post-central structural or functional deficit, multifocal cerebral lesions on CT or MRI)
13. laboratory tests indicating brain involvement or inflammatory disorder (such as multiple sclerosis, syphilis, AIDS and herpes simplex encephalitis).

Relative diagnostic exclusion features include:

1. typical history of chronic alcoholism
2. sustained hypertension
3. history of vascular disease (such as angina, claudication).

Annex 7
The Mini-Mental State Examination

The Mini-Mental State Examination (MMSE) is a widely used screening instrument for dementia. Adapted from Folstein MF, Folstein SE, McHugh PR. Psychiatry Res, 1975.[12]

www.parinc.com

Addenbrooke's Cognitive Examination (ACE)

An example of a comprehensive, brief and reliable bedside instrument for early detection of dementia, comprising of a 100-point test battery that assesses six cognitive domains. [182] Reproduced by kind permission of Prof. John R Hodges, MRC Cognition and Brain Sciences Unit, Cambridge.

ADDENBROOKE'S COGNITIVE EXAMINATION - ACE-R

Final Revised Version A (May 2004)

Name :
Date of birth :
Hospital no. :

Addressograph

Date of testing: ...../...../.....
Tester's name: .....
Age at leaving full-time education: .....
Occupation: .....
Handedness: .....

ORIENTATION

➢ Ask: What is the	Day	Date	Month	Year	Season	[Score 0-5]
➢ Ask: Which	Building	Floor	Town	County	Country	[Score 0-5]

REGISTRATION

[Score 0-3]

➢ Tell: 'I'm going to give you three words and i'd like you to repeat after me: lemon, key and ball'. After subject repeats, say 'Try to remember them because i'm going to ask you later'. Score only the first trial (repeat 3 times if necessary).

Register number of trials .....

ATTENTION & CONCENTRATION

[Score 0-5] (for the best performed task)

➢ Ask the subject: ' could you take 7 away from a 100? After the subject responds, ask him or her to take away another 7 to a total of 5 subtractions. If subject fails, ask: 'did you mean __ ?' If subject still makes a mistake, switch to spelling. If subject corrects himself or herself, continue.

Stop after five subtractions (93, 86, 79, 72, 65). ....

➢ Ask: 'could you please spell **WORLD** for me? Then ask him/her to spell it backwards:

....

MEMORY - Recall

[Score 0-3]

➢ Ask: 'Which 3 words did I ask you to repeat and remember?'

....

MEMORY - Anterograde Memory

[Score 0-7]

➢ Tell: ' I'm going to give you a name and address and I'd like you to repeat after me. We'll be doing that 3 times, so you have a chance to learn it. I'll be asking you later'

Score only the third trial

	1st Trial	2nd Trial	3rd Trial
Harry Barnes	....	....	....
73 Orchard Close	....	....	....
Kingsbridge	.....	.....	.....
Devon	.....	.....	.....

MEMORY - Retrograde Memory

[Score 0 -4]

➢ Name of current Prime Minister .....
➢ Name of the woman who was Prime Minister .....
➢ Name of the USA president .....
➢ Name of the USA president who was assassinated in the 1960's .....

ATTENTION&ORIENTATION MEMORY

ADDENBROOKE'S COGNITIVE EXAMINATION - ACE-R *Final Revised Version A (May 2004)*

VERBAL FLUENCY - Letter 'P' and animals

➢ **Letters**

Say: 'I'm going to give you a letter of the alphabet and I'd like you to generate as many words as you can beginning with that letter, but not names of people or places. Are you ready? You've got a minute and the letter is P'

[Score 0 - 7] ☐

0-15 sec	16-30 sec	31-45 sec	46-60 sec		
				>17	7
				14-17	6
				11-13	5
				8-10	4
				6-7	3
				4-5	2
				3-4	1
				<3	0
				total	correct

➢ **Animals**

Say: 'Now can you name as many animals as possible, beginning with any letter?

[Score 0 - 7] ☐

0-15 sec	16-30 sec	31-45 sec	46-60 sec		
				>21	7
				17-21	6
				14-16	5
				11-13	4
				9-10	3
				7-8	2
				5-6	1
				<5	0
				total	correct

FLUENCY

LANGUAGE - Comprehension

➢ Show written instruction:

[Score 0-1] ☐ ☐

Close your eyes

➢ 3 stage command:
'Take the paper in your right hand. Fold the paper in half. Put the paper on the floor'

[Score 0-3] ☐ ☐

LANGUAGE - Writing

➢ Ask the subject to make up a sentence and write it in the space below:
Score 1 if sentence contains a subject and a verb (see guide for examples)

[Score 0-1] ☐ ☐

LANGUAGE

ADDENBROOKE'S COGNITIVE EXAMINATION - ACE-R *Final Revised Version A (May 2004)*

LANGUAGE - Repetition

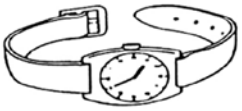
➢ Ask the subject to repeat:**' hippopotamus'; 'eccentricity; 'unintelligible'; 'statistician'**
Score 2 if all correct; 1 if 3 correct; 0 if 2 or less. [Score 0-2] ☐

➢ Ask the subject to repeat: **'Above, beyond and below'** [Score 0-1] ☐

➢ Ask the subject to repeat: **'No ifs, ands or buts'** [Score 0-1] ☐

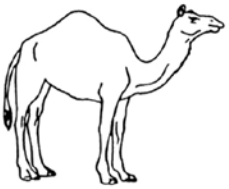
LANGUAGE - Naming

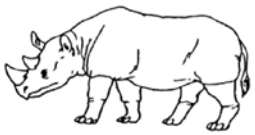
➢ Ask the subject to name the following pictures: [Score 0-2] pencil + watch ☐

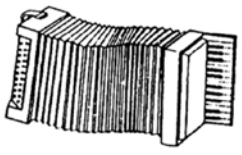

[Score 0-10] ☐











LANGUAGE - Comprehension

➢ Using the pictures above, ask the subject to: [Score 0-4] ☐

- Point to the one which is associated with the monarchy _____
- Point to the one which is a marsupial _____
- Point to the one which is found in the Antarctic _____
- Point to the one which has a nautical connection _____

LANGUAGE

ADDENBROOKE'S COGNITIVE EXAMINATION - ACE-R *Final Revised Version A (May 2004)*

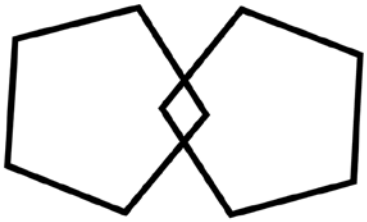
LANGUAGE - Reading

➢ Ask the subject to read the following words: [Score 1 only if all correct] [Score 0-1]

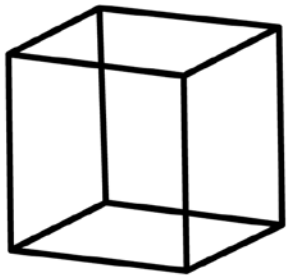
sew
pint
soot
dough
height

VISUOSPATIAL ABILITIES

➢ Overlapping pentagons: Ask the subject to copy this diagram: [Score 0-1]



➢ Wire cube : Ask the subject to copy this drawing (for scoring, see instructions guide) [Score 0-2]



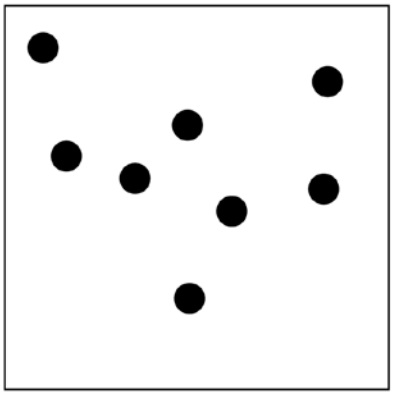
➢ Clock: Ask the subject to draw a clock face with numbers and the hands at ten past five. (for scoring see instruction guide: circle = 1, numbers = 2, hands = 2 if all correct) [Score 0-5]

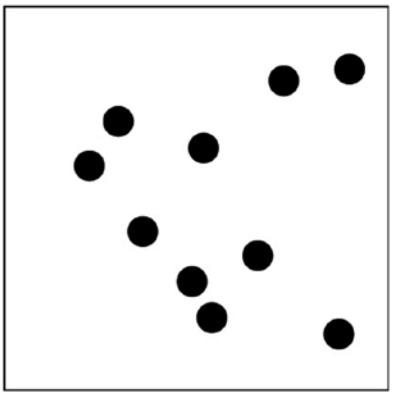
ADDENBROOKE'S COGNITIVE EXAMINATION - ACE-R *Final Revised Version A (May 2004)*

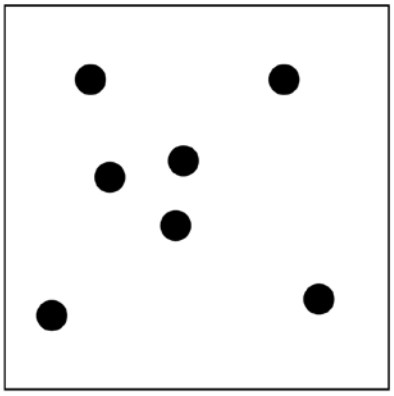
PERCEPTUAL ABILITIES

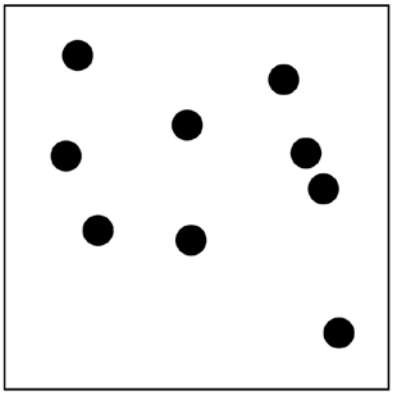
➢ Ask the subject to count the dots without pointing them

[Score 0-4]









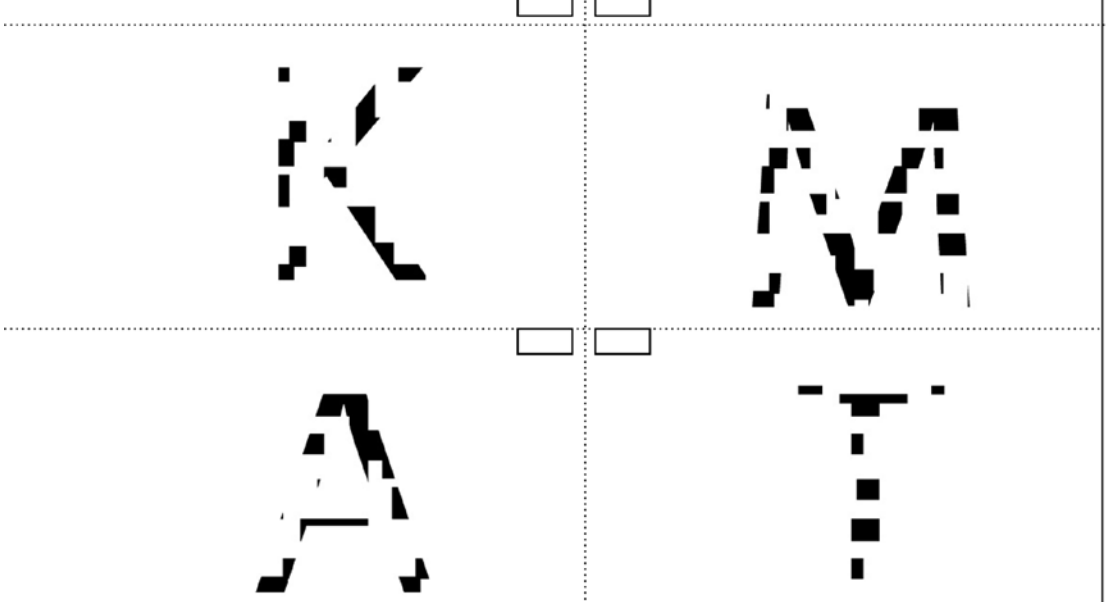
VISUOSPATIAL

ADDENBROOKE'S COGNITIVE EXAMINATION - ACE-R

Final Revised Version A (May 2004)

PERCEPTUAL ABILITIES

➢ Ask the subject to identify the letters [Score 0-4] ☐



RECALL

➢ Ask "Now tell me what you remember of that name and address we were repeating at the beginning"

Harry	Barnes	.....	.....
73	Orchard	Close	....
	Kingsbridge	.....	
	Devon	.....	

[Score 0-7] ☐

RECOGNITION

➢ Tick items recalled on the right hand side - shadowed column. For not recalled items, test recognition by reading the 3 alternatives 'was the name X, Y or Z?' and so on. Score 0-5 including items recalled and recognised. If all items were recalled, skip the recognition test, scoring 5 straight away (see instructions guide).

[Score 0-5] ☐

Jerry Barnes		Harry Barnes		Harry Bradford		recalled	
37		73		76		recalled	
Orchard Place		Oak Close		Orchard Close		recalled	
Oakhampton		Kingsbridge		Dartington		recalled	
Devon		Dorset		Somerset		recalled	

General Scores

MMSE	/30
ACE-R	/100

Subscores

Attention and Orientation	/18
Memory	/26
Fluency	/14
Language	/26
Visuospatial	/16

VISUOSPATIAL MEMORY SCORE

Annex 8

Short form of the Informant Questionnaire on Cognitive Decline in the Elderly (Short IQCODE)

Created by F. Jorm, Centre for Mental Health Research, The Australian National University, Canberra, Australia (www.anu.edu.au/iqcode).[183] In research studies the IQCODE is preceded by questions on the subject's sociodemographic characteristics and physical health. There is no copyright on the Short IQCODE, but please keep the author informed of research projects which make use of it.

Now we want you to remember what your friend or relative was like 10 years ago and to compare it with what he/she is like now. 10 years ago was in 19__. Below are situations where this person has to use his/her memory or intelligence and we want you to indicate whether this has improved, stayed the same or got worse in that situation over the past 10 years. Note the importance of comparing his/her present performance **with 10 years ago**. So if 10 years ago this person always forgot where he/she had left things, and he/she still does, then this would be considered "Hasn't changed much". Please indicate the changes you have observed by **circling the appropriate answer.**

Compared with 10 years ago how is this person at:	1	2	3	4	5
1. Remembering things about family and friends eg occupations, birthdays, addresses	Much improved	A bit improved	Not much change	A bit worse	Much worse
2. Remembering things that have happened recently	Much improved	A bit improved	Not much change	A bit worse	Much worse
3. Recalling conversations a few days later	Much improved	A bit improved	Not much change	A bit worse	Much worse
4. Remembering his/her address and telephone number	Much improved	A bit improved	Not much change	A bit worse	Much worse
5. Remembering what day and month it is	Much improved	A bit improved	Not much change	A bit worse	Much worse
6. Remembering where things are usually kept	Much improved	A bit improved	Not much change	A bit worse	Much worse
7. Remembering where to find things which have been put in a different place from usual	Much improved	A bit improved	Not much change	A bit worse	Much worse
8. Knowing how to work familiar machines around the house	Much improved	A bit improved	Not much change	A bit worse	Much worse
9. Learning to use a new gadget or machine around the house	Much improved	A bit improved	Not much change	A bit worse	Much worse
10. Learning new things in general	Much improved	A bit improved	Not much change	A bit worse	Much worse
11. Following a story in a book or on TV	Much improved	A bit improved	Not much change	A bit worse	Much worse
12. Making decisions on everyday matters	Much improved	A bit improved	Not much change	A bit worse	Much worse
13. Handling money for shopping	Much improved	A bit improved	Not much change	A bit worse	Much worse
14. Handling financial matters eg the pension, dealing with the bank	Much improved	A bit improved	Not much change	A bit worse	Much worse
15. Handling other everyday arithmetic problems eg knowing how much food to buy, knowing how long between visits from family or friends	Much improved	A bit improved	Not much change	A bit worse	Much worse
16. Using his/her intelligence to understand what's going on and to reason things through	Much improved	A bit improved	Not much change	A bit worse	Much worse

Annex 9
Further reading

The following is a list of publications addressing topics outwith the remit of this guideline, which may be useful in the management of patients with dementia. The quality of the evidence in these publications has not been assessed by the guideline development group.

An integrated care pathway for dementia: best practice for dementia care
Marian Naidoo and Roger Bullock
Harcourt Health Communications, London 2001

Lipids and the primary prevention of coronary heart disease (SIGN 40)
Scottish Intercollegiate Guidelines Network**,** Edinburgh 1999

Hypertension in older people (SIGN 49)
Scottish Intercollegiate Guidelines Network, Edinburgh 2001

Management of patients with stroke: rehabilitation, prevention and management of complications, and discharge planning (SIGN 64)
Scottish Intercollegiate Guidelines Network, Edinburgh 2002

Management of diabetes (SIGN 55).
Scottish Intercollegiate Guidelines Network**,** Edinburgh 2001

Obesity in Scotland: integrating prevention with weight management (SIGN 8)
Scottish Intercollegiate Guidelines Network**,** Edinburgh 1996

Clinical Standards: food, fluid and nutritional care in hospitals
NHS Quality Improvement Scotland, Edinburgh 2003

Clinical Standards: specialist palliative care
Clinical Standards Board for Scotland, Edinburgh 2002

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DIAGNOSIS

HISTORY TAKING AND DIFFERENTIAL DIAGNOSIS

B **DSM-IV or NINCDS-ADRDA criteria should be used for the diagnosis of Alzheimer's disease.**

B **The Hachinski Ischaemic Scale or NINDS-AIRENS criteria may be used to assist in the diagnosis of vascular dementia.**

C **Diagnostic criteria for dementia with Lewy bodies and fronto-temporal dementia should be considered in clinical assessment.**

INITIAL COGNITIVE TESTING

B **In individuals with suspected cognitive impairment, the MMSE should be used in the diagnosis of dementia.**

☑ Initial cognitive testing can be improved by the use of Addenbrooke's Cognitive Examination.

SCREENING FOR COMORBID CONDITIONS

☑ Physical investigations including laboratory tests should be selected on clinical grounds according to history and clinical circumstances.

B **As part of the assessment for suspected dementia, the presence of comorbid depression should be considered.**

THE USE OF IMAGING

C **Structural imaging should ideally form part of the diagnostic workup of patients with suspected dementia.**

C **SPECT may be used in combination with CT to aid the differential diagnosis of dementia when the diagnosis is in doubt.**

NEUROPSYCHOLOGICAL TESTING

B **Neuropsychological testing should be used in the diagnosis of dementia, especially in patients where dementia is not clinically obvious.**

ABBREVIATIONS

DSM-IV	Diagnostic and Statistical Manual, 4th edition
MMSE	Mini-Mental State Examination
NINCDS-ADRDA	National Institute of Neurologic, Communicative Disorders and Stroke–AD and related Disorders Association
NINDS-AIRENS	National Institute of Neurological Disorders and Stroke Association Internationale pour la Recherche et l'Enseignement en Neurosciences
SPECT	single photon emission controlled tomography

NON-PHARMACOLOGICAL INTERVENTIONS

BEHAVIOUR MANAGEMENT

B **Behaviour management may be used to reduce depression in people with dementia.**

CAREGIVER INTERVENTION PROGRAMMES

B **Caregivers should receive comprehensive training on interventions that are effective for people with dementia.**

COGNITIVE STIMULATION

B **Cognitive stimulation should be offered to individuals with dementia.**

MULTISENSORY STIMULATION AND COMBINED THERAPIES

☑ For people with moderate dementia who can tolerate it, multisensory stimulation may be a clinically useful intervention.

☑
- Multisensory stimulation is not recommended for relief of neuropsychiatric symptoms in people with moderate to severe dementia.
- Bright light therapy is not recommended for the treatment of cognitive impairment, sleep disturbance or agitation in people with dementia.
- In people with dementia who show behavioural disturbance despite the use of psychotropic medication, aromatherapy may influence behaviour but cannot be recommended as a direct alternative to antipsychotic drugs, nor for the reduction of specific behavioural problems.
- The use of aromatherapy to reduce associated symptoms in people with dementia should be discussed with a qualified aromatherapist who can advise on contraindications.

RECREATIONAL AND PHYSICAL ACTIVITIES

B **Recreational activities should be introduced to people with dementia to enhance quality of life and well-being.**

☑ For people with dementia a combination of structured exercise and conversation may help maintain mobility.

REALITY ORIENTATION THERAPY

D **Reality orientation therapy should be used by a skilled practitioner, on an individualised basis, with people who are disorientated in time, place and person.**

PHARMACOLOGICAL INTERVENTIONS

CHOLINESTERASE INHIBITORS

B **Donepezil, at daily doses of 5 mg and above can be used:**
- **to treat cognitive decline in people with Alzheimer's disease**
- **for the management of associated symptoms in people with Alzheimer's disease**

B **Galantamine, at daily doses of 16 mg and above can be used:**
- **to treat cognitive decline in people with Alzheimer's disease and people with mixed dementias**
- **for the management of associated symptoms in people with Alzheimer's disease.**

B **Rivastigmine, at daily doses of 6mg and above can be used:**
- **to treat cognitive decline in people with Alzheimer's disease**
- **to treat cognitive decline in people with dementia with Lewy bodies**
- **for the management of associated symptoms in people with Alzheimer's disease and dementia with Lewy bodies**

ANTIPSYCHOTICS

A **If necessary, conventional antipsychotics may be used with caution, given their side effect profile, to treat the associated symptoms of dementia.**

☑
- An individualised approach to managing agitation in people with dementia is required.
- Atypical antipsychotics with reduced sedation and extrapyramidal side effects may be useful in practice, although the risk of serious adverse events such as stroke must be carefully evaluated.
- In patients who are stable antipsychotic withdrawal should be considered.
- Where antipsychotics are inappropriate cholinesterase inhibitors may be considered.

ANTIDEPRESSANTS

D **Antidepressants can be used for the treatment of comorbid depression in dementia providing their use is evaluated carefully.**

☑ Trazodone may be considered for patients with depressive symptoms and dementia associated agitation.

HERBAL MEDICINES

☑
- People with dementia who wish to use *Ginkgo biloba* should consult a qualified herbalist for advice and should be made aware of possible interactions with other prescribed drugs.
- People with dementia who wish to use *Salvia officinalis* should consult a qualified herbalist for advice.