## Levels of evidence and grades of recommendation

### Levels of evidence

<table>
<thead>
<tr>
<th>Level</th>
<th>Type of Evidence</th>
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<tbody>
<tr>
<td>1++</td>
<td>High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias.</td>
</tr>
<tr>
<td>1+</td>
<td>Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias.</td>
</tr>
<tr>
<td>1</td>
<td>Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias</td>
</tr>
<tr>
<td>2++</td>
<td>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</td>
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<tr>
<td>2+</td>
<td>Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal</td>
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<td>Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal</td>
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</tr>
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<td>4</td>
<td>Expert opinion</td>
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### Grades of recommendation

<table>
<thead>
<tr>
<th>Grade</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>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</td>
</tr>
<tr>
<td>B</td>
<td>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+</td>
</tr>
<tr>
<td>C</td>
<td>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++</td>
</tr>
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<td>D</td>
<td>Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+</td>
</tr>
<tr>
<td>GPP (good practice points)</td>
<td>Recommended best practice based on the clinical experience of the guideline development group.</td>
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Dementia
Statement of Intent

These guidelines are not intended to serve as a standard of medical care. Standards of medical care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge advances and patterns of care evolve.

The contents of this publication are guidelines to clinical practice, based on the best available evidence at the time of development. Adherence to these guidelines may 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. Each physician is ultimately responsible for the management of his/her unique patient in the light of the clinical data presented by the patient and the diagnostic and treatment options available.
Foreword

Dementia is the progressive decline in cognitive function due to damage or disease in the brain. Essentially, dementia is a problem that makes it hard for a person to remember, learn and communicate. With progression, it becomes difficult for the person to take care of himself or herself. The disease extracts its toll not only on its victims but also on the family and caregivers in terms of physical, emotional and economic cost that translates into caregiver’s stress. It is estimated that 44% of the total cost of dementia is due to informal care.*

According to the WHO data, nearly five million DALYs are lost each year due to dementia in the Asia-Pacific Region. In Singapore, neuropsychiatric illnesses are the leading cause of DALYs loss and dementia ranks 4th among all neuropsychiatric illnesses. Dementia accounts for approximately 26,000 DALYs lost each year in Singapore.* As Singapore’s population is ageing rapidly, it is projected that the prevalence (22,000 in 2005) of dementia would double by 2020.*

The Ministry of Health released its first guidelines on Dementia in 2001 with the aim of upgrading the skills of practitioners to conduct proper clinical, functional and social assessment of the patients with and/or suspected of having dementia. Since then, further evidence have emerged in the area of therapeutics namely the role of cholinesterase in the treatment of dementia; non-pharmacological methods for the management of behavioral and psychological symptoms of dementia. It is thus timely to update this set of guidelines.

Apart from updating the guidelines in the area of management, the guideline also addresses ethical issues involved in the care of patients suffering from dementia and the utility of genetic tests.

I hope this set of guidelines will assist all doctors involved in the care of patients with dementia.

PROFESSOR K SATKU
DIRECTOR OF MEDICAL SERVICES

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Executive summary of recommendations

Details of recommendations can be found in the main text at the pages indicated.

Screening and assessment of dementia

C There is currently insufficient evidence for routine screening for dementia in older adults. Individuals who should be evaluated for dementia include those with progressive cognitive or behavioural complaints suggestive of dementia, as well as patients who arouse the physician’s or caregiver’s suspicion of cognitive impairment despite absence of complaints (pg 11).

   Grade C, Level 2+

GPP Assessment of dementia should be done via a comprehensive evaluation. This approach will aim to diagnose dementia early, assess for complications of dementia and establish the cause of the dementia (pg 11).

   GPP

B In individuals with suspected cognitive impairment, diagnosis can be made using the DSM-IV criteria for dementia with history from a reliable informant. This can be supplemented by an objective approach with cognitive tests (ECAQ/AMT/CMMSE) and/or neuropsychological assessment (pg 12).

   Grade B, Level 2++

B The complications of dementia can be broadly divided into behavioural and psychological symptoms, functional problems and social problems. These should be evaluated in all patients with dementia as these issues are the major causes of stress on the caregiver and assessment would enable the clinician to target subsequent management effectively (pg 15).

   Grade B, Level 2++

D The aim of determining dementia aetiology is to rule out potentially reversible causes of dementia and selecting appropriate treatment strategies for the irreversible dementias. This is done via clinical history and physical examination, followed by laboratory investigations and neuroimaging (pg 18).

   Grade D, Level 4
A number of well-validated clinical criteria for the two most common types of dementia (Alzheimer’s disease and Vascular dementia) have been developed over the years. These can be used in the specialized dementia clinics for the definition of Alzheimer’s disease and Vascular dementia (pg 20).

**Grade B, Level 2++**

**Pharmacological management of dementia**

**GPP** Pharmacotherapy should be part of a multi-pronged strategy to dementia management that encompasses a well-established diagnosis, education of patient and caregiver, non-pharmacological measures and comprehensive caregiver psychosocial intervention (pg 25).

**Grade B, Level 1++**

Although high dose vitamin E (2000 IU per day) may have a modest effect in delaying disease progression in moderately severe Alzheimer’s disease, doses of vitamin E in excess of 400 IU a day should be avoided for the treatment of Alzheimer’s disease until there is further data on its safety, especially in patients with cardiovascular disease (pg 26).

**Grade B, Level 1+**

Anti-inflammatory agents (such as non-steroidal anti-inflammatory agents and cyclo-oxygenase 2 inhibitors) are not recommended for the prevention of cognitive decline in Alzheimer’s disease (pg 27).

**Grade A, Level 1++**

Prednisolone is not recommended for the prevention of cognitive decline in Alzheimer’s disease (pg 27).

**Grade B, Level 1+**

Oestrogen is not recommended for the prevention of cognitive decline in women with dementia (pg 27).

**Grade A, Level 1++**

Acetylcholinesterase inhibitors should be considered for the management of all patients with mild to moderate Alzheimer’s disease (pg 27).

**Grade A, Level 1++**
Acetylcholinesterase inhibitors can be considered for the management of moderate to severe Alzheimer’s disease (pg 28).

Grade B, Level 1+

Acetylcholinesterase inhibitors have been shown to be of clinical benefit and may be considered for use in the management of mild to moderate vascular dementia (pg 28).

Grade A, Level 1+

Acetylcholinesterase inhibitors can be considered for the management of dementia with Lewy bodies and Parkinson’s disease dementia (pg 29).

Grade B, Level 1+

All three available acetylcholinesterase inhibitors (donepezil, rivastigmine and galantamine) can be considered for the pharmacological management of dementia, since there is no definite evidence to support a difference in clinical efficacy between them (pg 30).

Grade B, Level 1+

Where tolerated, acetylcholinesterase inhibitors should be titrated to recommended doses (5-10 mg/day donepezil; 6-12 mg/day rivastigmine; 16-24 mg/day galantamine), which have been shown to confer greater benefit compared with lower doses (pg 30).

Grade A, Level 1++

N-methyl D-aspartate antagonists such as memantine can be considered for the management of moderate to severe Alzheimer’s disease, either alone or in combination with acetylcholinesterase inhibitors (pg 31).

Grade B, Level 1+

N-methyl D-aspartate antagonists such as memantine may be a treatment option for mild to moderate Alzheimer’s disease, if acetylcholinesterase inhibitor therapy is contra-indicated, not tolerated or if there is disease progression despite an adequate trial of acetylcholinesterase inhibitor (pg 31).

Grade B, Level 1+

N-methyl D-aspartate (NMDA) antagonists have been shown to be of clinical benefit and may be considered for use in the management of mild to moderate vascular dementia (pg 32).

Grade A, Level 1+
Practitioners who prescribe ginkgo for the treatment of dementia should be aware of the unestablished benefit, variability of active ingredient among preparations, and potential for drug interactions (pg 32).

Grade B, Level 1+

Selegiline is not recommended for the treatment of core or associated symptoms in Alzheimer’s disease (pg 33).

Grade A, Level 1++

Appropriate treatment of vascular risk factors is recommended for all patients. However, it should be noted that whilst promising observational data exists, it remains to be shown in a randomised controlled clinical trial if any prevention strategy such as blood pressure reduction or antiplatelet treatment for the secondary prevention of stroke, will reduce the incidence of vascular dementia (pg 33).

GPP

The decision to initiate costly symptomatic dementia treatment, such as acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists, should always be made in consultation with the patient and family after careful consideration of the expected magnitude of benefit, side effects, comorbidities and costs of treatment (pg 33).

GPP

Patients who are started on acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists, should be carefully monitored for side effects and response to treatment (pg 34).

GPP

Management of behavioural and psychological symptoms of dementia (BPSD)

Non-pharmacological methods to manage behavioural and psychological symptoms of dementia should be instituted, prior to consideration of pharmacological measures (pg 37).

GPP

Antidepressants may be used for the treatment of comorbid depression in dementia provided their use has been evaluated carefully for each patient (pg 39).

GPP
A Conventional and atypical antipsychotics may be used with caution, given their side effect profile, to treat neuropsychiatric symptoms of dementia (pg 39).

Grade A, Level 1+

B Trazodone may be considered for patients with depressive symptoms and dementia associated agitation (pg 40).

Grade B, Level 1+

A Routine use of mood stabilizers, such as carbamazepine and sodium valproate, is not recommended for treatment of behavioural symptoms associated with dementia (pg 41).

Grade A, Level 1+

GPP An individualized approach to managing behavioural problems in dementia patients is required (pg 41).

GPP

GPP Cholinesterase inhibitor therapy may be considered in treatment of patients with behavioural problems if antipsychotics are inappropriate (pg 41).

GPP

GPP The decision to start antipsychotic therapy to control behavioural problems in dementia patients should be made in consultation with the patient and family, after careful consideration of the benefit, adverse-effects and co-morbidities (pg 41).

GPP

B For patients with dementia with Lewy Body and behavioural problems, acetylcholinesterase inhibitors should be considered first for management of the behavioural problems (pg 42).

Grade B, Level 1+

GPP In all patients started on antipsychotic medication, they should be monitored carefully for side effects and response to treatment. In patients who are stable, antipsychotic withdrawal should be considered (pg 42).

GPP
Social and caregiver management of dementia and community resources

**A** Caregiver interventions via a multifaceted approach should be considered in the total management of the person with dementia (pg 44).

Grade A, Level 1+

**GPP** Where appropriate, respite care can be offered to relieve the burden of caregiving on the family caregiver (pg 45).

GPP

**GPP** Referral to community resources to meet the care needs of the person with dementia and his/her carer should always be considered (pg 46).

GPP
1 Guideline Development and Objectives

1.1 Guideline development

The guidelines have been produced by a committee of psychiatrists, neurologists, geriatricians and primary care physicians appointed by the Ministry of Health. They were developed by the adaptation of existing guidelines, by the review of relevant literature and by expert clinical consensus with consideration of local practice.

1.2 Objectives

The main aim of these guidelines is to provide an approach for healthcare professionals to assess, evaluate and manage dementia (using local evidence where possible).

1.3 Target group

These guidelines are mainly developed for all healthcare professionals involved in the care of patients with dementia.

1.4 What’s new in the revised guidelines?

Since the last MOH Clinical Practice Guidelines on Dementia in 2001, new research findings and developments have emerged. These have culminated in updated recommendations in the area of therapeutics, namely the benefit of cholinesterase inhibitors in vascular dementia and dementia with Lewy Body; the revision of recommendations for the use of selegiline and vitamin E for Alzheimer’s disease; the management of behavioral and psychological symptoms of dementia and ethical issues in dementia.

1.5 Review of guidelines

Evidence-based clinical practice guidelines are only as current as the evidence that supports them. Users must keep in mind that new evidence could supercede recommendations in these guidelines. The workgroup advises that these guidelines be scheduled for review 5 years after publication, or if new evidence appears that requires substantive changes to the recommendations.
2 Introduction and Epidemiology of Dementia

2.1 Introduction and epidemiology

Rapidly graying demographics characterize many economically developed, as well as developing countries and from this perspective, dementia and its attendant problems are on the agendas of many governments. An estimated 25 million persons in the world have Alzheimer’s disease (AD) – generally considered to be the commonest subtype of dementia - and these numbers are expected to increase to 63 million in 2030, and by 2050, an estimated 114 million will be afflicted with the illness.¹

The number of those with dementia will increase in Asia-Pacific region from 13.7 million people in 2005 to 64.6 million people in 2050.² Singapore has one of the fastest ageing populations in the Asia-Pacific region with 15-20% of total population consisting of persons aged 65 and above by the year 2030.³ The prevalence of dementia is expected to increase from 22,000 in 2005 to almost 53,000 in 2020 and 187,000 in 2050.²

The prevalence of dementia or cognitive impairment in local epidemiological studies using various screening instruments ranges from 2% to 14%,⁴-⁷ with age-related increases in prevalence of cognitive impairment from 0.8% in those aged between 60-64 years to 32.2% among those aged 85 years and older.⁷ These prevalence rates are comparable with other populations in the world in evidence-based estimates generated by Delphi Consensus Study⁸ (See Table 1) and other comparison studies.⁹
<table>
<thead>
<tr>
<th>Region</th>
<th>Consensus dementia prevalence (%) (60+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Western Europe – EURO A</td>
<td>5.4</td>
</tr>
<tr>
<td>Eastern Europe low adult mortality – EURO B</td>
<td>3.8</td>
</tr>
<tr>
<td>Eastern Europe high adult mortality – EURO C</td>
<td>3.9</td>
</tr>
<tr>
<td>North America – AMRO A</td>
<td>6.4</td>
</tr>
<tr>
<td>Latin America – AMRO B/D</td>
<td>4.6</td>
</tr>
<tr>
<td>North Africa &amp; Middle East EMRO B/D</td>
<td>3.6</td>
</tr>
<tr>
<td>Developed Western Pacific – WPRO A</td>
<td>4.3</td>
</tr>
<tr>
<td>China and developing Western Pacific – WPRO B</td>
<td>4.0</td>
</tr>
<tr>
<td>Indonesia, Thailand and Sri Lanka – SEARO B</td>
<td>2.7</td>
</tr>
<tr>
<td>India and S Asia – SEARO D</td>
<td>1.9</td>
</tr>
<tr>
<td>Africa – AFRO D/E</td>
<td>1.6</td>
</tr>
</tbody>
</table>

(Sources: Ferri CP et al, 2005; Kua EH, 1996)

Subgroups based on patterns of child and adult mortality from A (lowest) to E (highest).

WPRO = Western Pacific Region
WPRO A = Australia, Japan, Brunei Darussalam, New Zealand, Singapore
WPRO B = Cambodia, China, Lao People’s Democratic Republic, Malaysia, Mongolia, Philippines, Republic of Korea, Vietnam, Cook Islands, Fiji, Kiribati, Marshall Islands, Micronesia (Federated States of), Nauru, Niue, Palau, Papua New Guinea, Samoa, Solomon Islands, Tonga, Tuvalu and Vanuatu.

SEARO = Southeast Asian region
SEARO D = Bangladesh, Bhutan, Democratic People’s Republic of Korea, India, Maldives, Myanmar and Nepal
Ethnic variations in dementia prevalence have also been demonstrated with higher dementia prevalences among the Malays (9.4%) and Indians (8.8%) as compared to the Chinese elderly population (4.2%). With regards to dementia aetiology, Alzheimer’s disease was found to be more common in Indians and Eurasians while vascular dementia more common in Chinese and Malays. A separate study found that among the elderly Malays, the prevalence of vascular dementia was higher in women than men.

With this rising trend, the evaluation of dementia, especially at the early stage, becomes increasingly important because with early diagnosis, the affected patients are more amenable to treatment advances. Interventions to slow down the progression of cognitive decline (both pharmacological and non-pharmacological measures) can translate into prolongation of functioning in the community, delay in institutionalisation and lower healthcare expenditure. This may help reduce the heavy cost and burden of the illness, both to family and society, especially in the light of our rapidly ageing population.
3 Screening and Assessment of Dementia

3.1 Screening

There is currently insufficient evidence for routine screening for dementia in older adults. Individuals who should be evaluated for dementia include those with progressive cognitive or behavioural complaints suggestive of dementia, as well as patients who arouse the physician’s or caregiver’s suspicion of cognitive impairment despite absence of complaints.

Grade C, Level 2+

The evaluation of dementia should be targeted at patients in whom there is some suspicion of cognitive impairment. They include:

- subjects with memory and other cognitive complaints, either reported by the patient himself or a significant other\(^{15}\)
- progressive forgetfulness\(^{16}\)
- subjects who arouse the physician’s suspicion of cognitive impairment during interview despite absence of memory or cognitive complaints
- those who are at increased risk for dementia (such as those with a strong family history of dementia)
- elderly patients who need to make important decisions (such as the sale of a house or making a will) and in whom mental competency is questionable.

3.2 Assessment of dementia

Assessment of dementia should be done via a comprehensive evaluation. This approach will aim to diagnose dementia early, assess for complications of dementia and establish the cause of the dementia.

GPP
In evaluation patients who present with forgetfulness or confusion, it is important to exclude delirium (acute confusional state) if this is of an **acute nature**. If the forgetfulness or confusion is of a **subacute nature** (weeks to few months), potentially reversible neurological conditions and depression have to be excluded.

(I) **Diagnosis of dementia**

B In individuals with suspected cognitive impairment, diagnosis can be made using the DSM-IV criteria for dementia with history from a reliable informant. This can be supplemented by an objective approach with cognitive tests (ECAQ/AMT/CMMSE) and/or neuropsychological assessment.

Grade B, Level 2++

**Subjective approach**

a) The **Diagnostic and Statistical Manual of Mental Disorders - IV (DSM-IV) criteria**\(^{17}\) are very often used as the gold standard for the clinical diagnosis of dementia.\(^{18-20}\) The criteria require memory impairment to be present, as well as deficits in one other cognitive domain (aphasia, apraxia, agnosia and executive dysfunctioning). Moreover, these cognitive **declines** must be of sufficient severity to cause perceptible impairment in social or occupational functioning (Table 2).
Table 2   DSM-IV clinical criteria for diagnosis of dementia\textsuperscript{17}

<table>
<thead>
<tr>
<th>Cognitive domain</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amnesia</td>
<td>Any forgetfulness? Did it start gradually or suddenly? Is it progressively worse? And if so, is it smoothly declining or showing a step-wise/ fluctuating decline? Is it over short-term or long-term matters?</td>
</tr>
<tr>
<td>AND declines in one of the following domains:</td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>Any word-finding difficulty or other difficulties with communication?</td>
</tr>
<tr>
<td>Apraxia</td>
<td>Any problems with buttoning or dressing? Any difficulties with using utensils during mealtimes?</td>
</tr>
<tr>
<td>Agnosia</td>
<td>Any problems recognising familiar faces or familiar items?</td>
</tr>
<tr>
<td>Executive dysfunctioning</td>
<td>Any problems handling money (loose change)? Any change in general problem-solving abilities? Is one’s work getting to be more disorganised?</td>
</tr>
<tr>
<td>OF sufficient severity to cause significant impairment in social or occupational functioning</td>
<td>AS a result of the above, is he becoming less independent in the - community? - home-care? - self-care level?</td>
</tr>
</tbody>
</table>

(Source: American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 1994).

b) The Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE)\textsuperscript{21,22} is a 26-item test that enquires about the subject’s memory, cognition and language ability for the last 10 years (see Appendix 1).

There is recent evidence of the Brief Informant Screening Test\textsuperscript{15} consisting of a single-item informant report of memory problem and a 4-item Instrumental Activities of Daily Living, as a useful screening instrument for patients with early cognitive impairment.
Objective approach

This is an observer-based approach using either performance-based instruments, such as mental status test (brief screening instruments), or a more detailed neuropsychological tests, which is usually administered by clinical psychologists.

a) The Elderly Cognitive Assessment Questionnaire (ECAQ)\textsuperscript{23,24} is a 10-item test screening for cognitive impairment assessing memory and information-orientation (Appendix 2).

b) The 10-item Abbreviated Mental Test (AMT) and

c) 28-item Chinese Mini Mental State Examination (CMMSE) (Appendix 3 and 4) have also been validated locally with age and education-adjusted cut-off values available.\textsuperscript{25} The CMMSE is more useful in those with higher educational attainment as the AMT may not be as sensitive.

d) Neuropsychological testing is usually administered by clinical psychologists. It is useful in detecting subtle cognitive difficulties which are not picked up by the brief screening instruments. They should be performed on subjects:

- who have memory complaints but do not yet satisfy criteria for dementia;
- depressed subjects who present with memory complaints to help in determining whether the memory complaints are due solely to the depression or whether they have concomitant dementia;
- Subjects in whom decision-making capacity is being assessed.

Psychometric testing can be a useful adjunct in the latter scenario. They are also useful in aetiologic differentiation of dementia.

Neuropsychometric batteries have been validated locally in the elderly Chinese\textsuperscript{26} and the Vascular Dementia Battery test has also been validated in the Singapore population.\textsuperscript{27}

Neuropsychological tests are also useful in individuals in whom the diagnosis of dementia is inconclusive and serial monitoring for performance decline over time may be useful in establishing the diagnosis.
(II) Assessing complications of dementia

The complications of dementia can be broadly divided into behavioural and psychological symptoms, functional problems and social problems. These should be evaluated in all patients with dementia as these issues are the major causes of stress on the caregiver and assessment would enable the clinician to target subsequent management effectively.

**Grade B, Level 2++**

a) Behavioural problems

Behavioural problems occur frequently in dementia patients; they affect 10% to 75% of all patients at some stage of their dementia. They can present severe problems to all who interact with them, leading to premature institutionalization, increased costs of care and significant loss in quality of life for the patient and family. Most of the major behavioural problems are amenable to treatment and are capable of reducing the suffering of the patients and their caregivers.

Different behavioural problems present at the different stages of the dementia process; anxiety and depression being common in early stages while aberrant motor behaviour is more common during the later stages of illness. In the clinic setting, the physician should *routinely ask about depression, anxiety, agitation, paranoia, hallucinations and sleep problems*, as these are potentially amenable to treatment, by way of either psychosocial interventions or pharmacologic agents.

These behavioural problems can also be rated objectively using behaviour scales which may be either self-rated, caregiver-based or observer reports. The Behavioural Pathology in Alzheimer’s Disease Rating Scale (BEHAVE-AD) and Neuropsychiatric Inventory (NPI) are examples of behaviour scales, but they are often used only in research settings.

Depression in the elderly may not show the classical features indicated in the DSM-IV and, in this regard, clinicians may find the brief depression scales useful in practice. Locally, the single-question test for depression, Geriatric Depression Scale (GDS) and Even Briefer Assessment Scale for Depression (EBAS-DEP) have been validated in cognitively intact, community-dwelling Chinese elderly. The Cornell Depression Scale in Dementia specifically
assesses depression in dementia\textsuperscript{33} and has been shown to be a useful screening instrument in our local population.\textsuperscript{34}

b) Functional difficulties

Functional difficulties can be assessed at three levels: community functioning, home functioning and self-care (See Appendix 5)\textsuperscript{35}. They are generally affected with the progression of dementia in a descending order and also allow these functional deficits to serve as markers of dementia severity. It is also important to make sure that these difficulties result from cognitive difficulties and not physical disabilities. The severity of dementia can be staged using the Diagnostic and Statistical Manual of Mental Disorders - 3\textsuperscript{rd} revised edition (DSM-III-R)\textsuperscript{36} or other formal functional assessment scales which include Clinical Dementia Rating Scale (CDR)\textsuperscript{37,38}, Functional Assessment Staging (FAST), Barthel Index and Blessed Dementia Scale (BDS).

c) Social problems

As a result of dementia, there is also loss of social role of the patient as a parent, spouse, friend and member of the larger society and these often result in caregivers’ coping difficulties. Identification of caregiver’s stress can allow targeted family education and counselling at tertiary dementia care centres, or even dementia day-care centres, and this has been shown to reduce institutionalisation.\textsuperscript{14} Elder abuse may also occur when the frustrated caregiver directs his or her stress at the vulnerable patient whose care is proving to be difficult.\textsuperscript{39} Likewise, financial difficulties should be asked for and if problems are identified, families can be referred to a social worker.

(III) Determining the aetiology of Dementia

Having determined the cognitive impairment to be chronic and having met clinical criteria for dementia, as well as assessing for the complications of dementia, the final step of the clinical evaluation involves determining the dementia aetiology. The types of dementia can be broadly divided into 2 categories – irreversible and reversible causes.

The more common causes of dementia can be classified into irreversible and potentially reversible causes.
• The irreversible causes include degenerative causes (Alzheimer’s disease, fronto-temporal dementia and dementia with Lewy body), cerebrovascular disease (Vascular dementia), prion-associated disorders (Creutzfeldt-Jakob disease) and neurogenetic disorders.

• The potentially reversible causes include infectious disorders (meningitis and encephalitis), toxic or metabolic encephalopathies (hypothyroidism, vitamin B$_{12}$ deficiency, and alcohol-related syndromes), neoplastic causes and hydrocephalus (obstructive or normal pressure hydrocephalus).

Alzheimer’s disease

Studies in many countries have shown that Alzheimer’s disease is the most common cause of dementia. Familial cases are rare and usually present as early onset dementia (less than 60 years). Sporadic cases usually present after the age of 60 with insidious, gradual deterioration of cognitive function. Short-term memory loss is the most common early symptom. However, other aspects of higher cognitive function may become affected over the course of time. Thus, patients may experience increasing difficulty with activities of daily living, changes in personality, behavioural and psychiatric problems, language dysfunction, loss of visuo-spatial and executive function.

Vascular dementia

Vascular dementia remains a controversial entity as there are disagreements over the validity of clinical criteria. Nevertheless, cognitive impairment and dementia is common in patients with cerebrovascular disease and it is often reported as the second most common cause of dementia in many studies.

Vascular dementia can be suspected in patients who have an abrupt onset of symptoms, stepwise deterioration, focal neurological signs and symptoms or a history of stroke. However, neuropathological studies have shown that many patients diagnosed with vascular dementia have Alzheimer’s disease pathology in addition to cerebrovascular lesions giving rise to doubts whether “pure” Vascular dementia exists. Due to the high variability of cerebrovascular pathology and its causative factors, no validated neuropathological criteria exist for vascular dementia. Nevertheless, in a large unselected, community-based neuropathology study of an
elderly population with prospectively evaluated dementia status, Alzheimer’s disease-type and vascular pathology were the major pathological correlates of cognitive decline but most patients had mixed disease\textsuperscript{40}, emphasising the important role of vascular pathology in dementia.

**Dementia with Lewy Body (DLB)**

DLB has been found in some studies to be the second most common form of degenerative dementia, accounting for up to 20% cases in the elderly. It is characterized by fluctuating cognitive impairment, spontaneous parkinsonism and recurrent visual hallucinations. Recognition of DLB is clinically important in view of the high incidence (60%) of adverse and life-threatening reaction to antipsychotics.

**Fronto-temporal dementia**

Fronto-temporal lobar degeneration produces 3 clinical syndromes:
- The most common is fronto-temporal dementia, characterized by personality change and profound alteration in social conduct and associated with bilateral atrophy of the frontal and anterior temporal lobes.
- Progressive non-fluent aphasia characterized by difficulty in verbal expression in the presence of relative preservation of comprehension, and
- Semantic dementia where there is fluent speech with semantic errors and severely impaired comprehension and naming, together with a visual associative agnosia.

The aim of determining dementia aetiology is to rule out potentially reversible causes of dementia and selecting appropriate treatment strategies for the irreversible dementias. This is done via clinical history and physical examination, followed by laboratory investigations and neuroimaging.\textsuperscript{41-44}

**History**

It is important to ask for the nature of the cognitive decline (sudden or gradual), progression – either gradually progressive (more suggestive of Alzheimer’s disease) or stepwise/fluctuating course (suggestive of Vascular dementia). A history of significant alcohol
ingestion and medication use (such as antipsychotics, antidepressants, anticholinergic agents and sedative-hypnotic agents) and history of medical, neurological and psychiatric illness is important.

**Physical examination and mental state examination**

A targeted physical examination should be performed, looking for evidence of depression, focal neurological deficits (such as visual field defects, hemiparesis, hemisensory loss, asymmetric deep tendon reflexes or unilateral extensor plantar responses). It is also important to examine for extrapyramidal signs such as rigidity and bradykinesia, movement disorders and gait abnormalities as these may point to certain etiologic diagnosis.

**Diagnostic tests to rule out metabolic and structural causes of dementia**

Dementias which are related to metabolic abnormalities are thought to be reversible. The haematological tests include **full blood count, urea and electrolytes, serum calcium, serum glucose, thyroid function tests and vitamin B\textsubscript{12} levels.**

Routine testing for neurosyphilis is problematic given the difficulties in interpretation of test results. It is best done when patients exhibit clinical features of neurosyphilis.

Other biomarkers which can help in establishing dementia diagnosis include apolipoprotein-E \textepsilon 4 allele, CSF-tau and \beta-amyloid for Alzheimer’s disease, CSF 14-3-3, neuron-specific enolase and electroencephalogram for Creutzfeld-Jakob disease. However, these are not performed routinely.

**Neuroimaging**

Neuroimaging is useful in the differential diagnosis of dementia and also necessary in the diagnostic criteria in Alzheimer’s disease and vascular dementia. It is also useful in detection of very early dementia as the functional and structural brain changes takes place before clinical manifestation of cognitive deficits. It consists of either

- Structural imaging techniques (computed tomography (CT) scan of head and magnetic resonance imaging (MRI)), or
• Functional neuroimaging techniques (Positron emission tomography and single-photon emission tomography).

Whether all patients with dementia require a structural imaging is an important clinical question, for which there is no consensus. The value of neuroimaging is in the identification of cerebral infarcts and clinically important surgical brain lesions (SBLs) such as subdural haematomas, cerebral tumors and normal pressure hydrocephalus. The Canadian Consensus Conference on the Assessment of Dementia (CCCAD)\(^{45}\) (Appendix 6) has outlined the criteria for undertaking a CT scan of the head, only if certain clinical conditions are met. In a patient with advanced dementia of a long duration (>2 years based on CCCAD’s recommendations), we believe a brain scan is not warranted to detect potentially reversible SBLs. Conversely, if the patient’s dementia is only mild to moderate (even after 2 years), it is still advisable to request for an initial CT scan of the brain.\(^{46}\) If the clinician is not inclined to perform a brain scan, there is immense value in discussing the matter with the caregivers and in securing their agreement not to order a neuroimaging procedure.

Neuroimaging is also useful for aetiologic differentiation of the different dementias.

\[B\] A number of well-validated clinical criteria for the two most common types of dementia (Alzheimer’s disease and Vascular dementia) have been developed over the years. These can be used in the specialized dementia clinics for the definition of Alzheimer’s disease and Vascular dementia.

\[\text{Grade B, Level 2++}\]

In Alzheimer’s disease, the common clinical criteria used include DSM-IV criteria for DAT, National Institute of Neurologic, Communicative Disorders and Stroke-Alzheimer’s disease and Related Disorders Association (NINCDS-ADRDA).\(^{47}\) In VD, the common clinical criteria used include DSM-IIIR, National Institute of Neurologic Disorder and Stroke and the Association Internationale pour la Recherche et l’Enseignement en Neurosciences (NINDS-AIREN)\(^{48}\), State of California AD Diagnostic and Treatment Centers criteria (ADDTC)\(^{49}\), Hachinski Ischemic Score (HIS)\(^{50}\), modified Rosen scale\(^{51}\) (the latter two being clinical scales).

Clinical Criteria for dementia with Lewy bodies\(^{52}\) and frontotemporal dementia\(^{53}\) have been developed.
## Appendix 1  Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE)\textsuperscript{21,22}

<table>
<thead>
<tr>
<th>Item</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Recognising the faces of family and friends</td>
<td>Rated on 5-point scale from 1. Much improved, A bit improved, Not much change, A bit worse, Much worse [1→5]</td>
</tr>
<tr>
<td>2. Remembering the names of family and friends</td>
<td></td>
</tr>
<tr>
<td>3. Remembering things about family and friends e.g. occupations,</td>
<td></td>
</tr>
<tr>
<td>birthdays, addresses</td>
<td></td>
</tr>
<tr>
<td>4. Remembering things that have happened recently</td>
<td></td>
</tr>
<tr>
<td>5. Recalling conversations a few days later</td>
<td></td>
</tr>
<tr>
<td>6. Forgetting what he/she wanted to say in the middle of a conversation</td>
<td></td>
</tr>
<tr>
<td>7. Remembering his/her address and telephone number</td>
<td></td>
</tr>
<tr>
<td>8. Remembering what day and month it is</td>
<td></td>
</tr>
<tr>
<td>9. Remembering where things are usually kept</td>
<td></td>
</tr>
<tr>
<td>10. Remembering where to find things which have been put in a different place from usual</td>
<td></td>
</tr>
<tr>
<td>11. Adjusting to any change in his/her day-to-day routine</td>
<td></td>
</tr>
<tr>
<td>12. Knowing how to work familiar machines around the house</td>
<td></td>
</tr>
<tr>
<td>13. Learning to use a new gadget or machine around the house</td>
<td></td>
</tr>
<tr>
<td>14. Learning new things in general</td>
<td></td>
</tr>
<tr>
<td>15. Remembering things that happened to him/her when he/she was young</td>
<td></td>
</tr>
<tr>
<td>16. Remembering things he/she learned when he/she was young</td>
<td></td>
</tr>
<tr>
<td>17. Understanding the meaning of unusual words</td>
<td></td>
</tr>
<tr>
<td>18. Understanding magazine or newspaper article</td>
<td></td>
</tr>
<tr>
<td>19. Following a story in a book or on TV</td>
<td></td>
</tr>
<tr>
<td>20. Composing a letter to friends or for business purposes</td>
<td></td>
</tr>
<tr>
<td>21. Knowing about important historical events of the past</td>
<td></td>
</tr>
<tr>
<td>22. Making decisions on everyday matters</td>
<td></td>
</tr>
<tr>
<td>23. Handling money for shopping</td>
<td></td>
</tr>
<tr>
<td>24. Handling financial matters, e.g. pension, dealing with bank</td>
<td></td>
</tr>
<tr>
<td>25. Handling other everyday arithmetic problems e.g. knowing how much food to buy, knowing how long between visits form family or friends</td>
<td></td>
</tr>
<tr>
<td>26. Using his/her intelligence to understand what’s going on and to reason things through.</td>
<td></td>
</tr>
</tbody>
</table>

(Source: Jorm AF et al, 1991; Lim HJ et al, 2003)
### Appendix 2  Elderly Cognitive Assessment Questionnaire (ECAQ)

<table>
<thead>
<tr>
<th>Items</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Memory</strong></td>
<td></td>
</tr>
<tr>
<td>1. I want you to remember this number. Can you repeat after me (4517). I shall test you again in 15 min.</td>
<td>1</td>
</tr>
<tr>
<td>2. How old are you?</td>
<td>1</td>
</tr>
<tr>
<td>3. When is your birthday? OR in what year were you born?</td>
<td></td>
</tr>
<tr>
<td><strong>Orientation and information</strong></td>
<td></td>
</tr>
<tr>
<td>4. What is the year?</td>
<td>1</td>
</tr>
<tr>
<td>5. date?</td>
<td>1</td>
</tr>
<tr>
<td>6. day?</td>
<td>1</td>
</tr>
<tr>
<td>7. month?</td>
<td>1</td>
</tr>
<tr>
<td>8. What is this place called? Hospital/Clinic</td>
<td>1</td>
</tr>
<tr>
<td>9. What is his/her job? (e.g. nurse/doctor)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Memory Recall</strong></td>
<td></td>
</tr>
<tr>
<td>10. Can you recall the number again?</td>
<td>1</td>
</tr>
</tbody>
</table>

Total

(Source: Kua EH & Ko SM, 1992)

### Appendix 3  Abbreviated Mental Test (AMT)

<table>
<thead>
<tr>
<th>Items</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the year?</td>
<td>1</td>
</tr>
<tr>
<td>What is the time? (within 1 hour)</td>
<td>1</td>
</tr>
<tr>
<td>What is your age?</td>
<td>1</td>
</tr>
<tr>
<td>What is your date of birth?</td>
<td>1</td>
</tr>
<tr>
<td>What is your home address?</td>
<td>1</td>
</tr>
<tr>
<td>Where are we now?</td>
<td>1</td>
</tr>
<tr>
<td>Who is our country’s Prime Minister?</td>
<td>1</td>
</tr>
<tr>
<td>What is his/her job? (show picture)</td>
<td>1</td>
</tr>
<tr>
<td>Memory phrase “37 Bukit Timah Road”</td>
<td>1</td>
</tr>
<tr>
<td>Count backwards from 20 to 1</td>
<td>1</td>
</tr>
<tr>
<td>Recall memory phase</td>
<td>1</td>
</tr>
</tbody>
</table>

Total score

(Source: Sahadevan S et al, 2000)
Appendix 4  Chinese Mini Mental State Examination (CMMSE)\textsuperscript{25}

<table>
<thead>
<tr>
<th>Items</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>What day of the week is it?</td>
<td>1</td>
</tr>
<tr>
<td>What is the date today?</td>
<td>1</td>
</tr>
<tr>
<td>What is the month?</td>
<td>1</td>
</tr>
<tr>
<td>What is the year?</td>
<td>1</td>
</tr>
<tr>
<td>Where are we now?</td>
<td>1</td>
</tr>
<tr>
<td>What floor are we now?</td>
<td>1</td>
</tr>
<tr>
<td>In which estate are we?</td>
<td>1</td>
</tr>
<tr>
<td>In which country are we?</td>
<td>1</td>
</tr>
<tr>
<td>Repeat the following words: “Lemon, Key, Balloon”</td>
<td>3</td>
</tr>
<tr>
<td>Subtract $7$ from $100$ and make 5 subtractions</td>
<td>5</td>
</tr>
<tr>
<td>Can you recall the three words</td>
<td>3</td>
</tr>
<tr>
<td>What is this? (show a pencil)</td>
<td>1</td>
</tr>
<tr>
<td>What is this? (show a watch)</td>
<td>1</td>
</tr>
<tr>
<td>Repeat the following:</td>
<td></td>
</tr>
<tr>
<td>a) “No ifs, ands or buts” (English)</td>
<td>1</td>
</tr>
<tr>
<td>b) “Forty-four stone lions” (Chinese)</td>
<td></td>
</tr>
<tr>
<td>Follow 1 3-stage command:</td>
<td></td>
</tr>
<tr>
<td>“Take this piece of paper, fold it in half, and put it on the</td>
<td>3</td>
</tr>
<tr>
<td>floor</td>
<td></td>
</tr>
<tr>
<td>Say a sentence of your choice</td>
<td>1</td>
</tr>
<tr>
<td>Read and obey what is written on this piece of paper</td>
<td></td>
</tr>
<tr>
<td>“Raise your hands”</td>
<td>1</td>
</tr>
<tr>
<td>Copy this drawing on a piece of paper</td>
<td>1</td>
</tr>
</tbody>
</table>

Total score

(Original source: Sahadevan S et al, 2000)
Appendix 5  Functional complications of dementia\textsuperscript{35}

<table>
<thead>
<tr>
<th>Variable</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community functioning</td>
<td>Can patient find his way around in unfamiliar surroundings, manage his finances, do shopping or marketing?</td>
</tr>
<tr>
<td>Home-care functioning</td>
<td>Can he prepare his own food, help in housework and cooking? Is he able to choose proper attire to dress himself? Is he safe to be left at home alone?</td>
</tr>
<tr>
<td>Self-care functioning</td>
<td>Is he able to bathe, dress himself? Is he able to go to toilet, transfer or feed himself? Is he continent of bladder and bowels?</td>
</tr>
</tbody>
</table>

(Source: MS Chong et al, 2003)

Appendix 6  Canadian Consensus Conference on the Assessment of Dementia (CCCAD) for performing Cranial CT in patients with dementia\textsuperscript{45}

Criteria:
- < 60 years old
- Rapid (e.g., over 1-2 months), unexplained decline in cognition or function
- Dementia of relatively short duration (<2 years)
- Recent, significant head trauma
- Unexplained neurologic symptoms (e.g., new onset of severe headache or seizures)
- History of cancer, especially of a type or at a site associated with metastasis to the brain
- Use of anticoagulants or history of bleeding disorder
- History of urinary incontinence and gait disturbance early in the disease (suggestive of normal pressure hydrocephalus)
- Presence of any new localising signs on physical examination (hemiparesis, Babinski’s sign)
- Unusual or atypical cognitive symptoms or presentation (e.g., progressive aphasia)
- Gait disturbance
- CT is recommended if one or more of these criteria are present.

(Source: Patterson CJ et al, 1999)
CT: computed tomography
4 Pharmacological Management of Dementia

4.1 Overview

Pharmacotherapy should be part of a multi-pronged strategy to dementia management that encompasses a well-established diagnosis, education of patient and caregiver, non-pharmacological measures and comprehensive caregiver psychosocial intervention.

All dementia patients should be evaluated for suitability of pharmacological strategies to:
- Reverse or stabilize the underlying disease
- Improve cognitive symptomatology
- Treat behavioural, mood or psychiatric symptoms associated with dementia

4.2 Pharmacological interventions to reverse or stabilize the underlying disease

Pharmacological strategies to address the underlying disease include treating identifiable reversible causes, reduction of established risk factors, and disease modifying measures to slow the rate of disease progression (Table 3).

Table 3 Possible pharmacological strategies to address underlying disease

<table>
<thead>
<tr>
<th>1. Treating identifiable reversible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Replace deficiency states (e.g. B&lt;sub&gt;12&lt;/sub&gt; deficiency, hypothyroidism)</td>
</tr>
<tr>
<td>Correct metabolic abnormalities (e.g. hypercalcemia, hypoglycemia)</td>
</tr>
<tr>
<td>Treat infections (e.g. neurosyphilis)</td>
</tr>
<tr>
<td>2. Reduction of vascular risk factors</td>
</tr>
<tr>
<td>Treatment of hyperlipidemia, hypertension, diabetes mellitus, and smoking cessation</td>
</tr>
<tr>
<td>Anti-platelet agents for secondary stroke prevention</td>
</tr>
<tr>
<td>Anti-coagulation for atrial fibrillation and cardioembolic strokes</td>
</tr>
<tr>
<td>3. Slowing rate of disease progression (disease modifying)</td>
</tr>
</tbody>
</table>
When significant neuronal damage has occurred, treatment of potentially reversible causes often arrests the underlying pathophysiology but may not reverse the dementia. Only a small percentage of potentially reversible abnormalities are completely reversible, and the more common of such conditions are hypothyroidism and vitamin B₁₂ deficiency. An increasing body of evidence suggests that vascular risk factors are putative not only in vascular dementia (VaD), but also in Alzheimer’s disease (AD), thus, vascular risk factors (such as hyperlipidemia, hypertension, diabetes mellitus, atrial fibrillation, smoking) should be sought for and managed in all dementia cases.

4.2.1 Disease-modifying strategies

Although high dose vitamin E (2000 IU per day) may have a modest effect in delaying disease progression in moderately severe Alzheimer’s disease, doses of vitamin E in excess of 400 IU a day should be avoided for the treatment of Alzheimer’s disease until there is further data on its safety, especially in patients with cardiovascular disease.

Grade B, Level 1+

The reported (and as yet unreplicated) benefit of high dose vitamin E (2000 IU per day) is at best a modest benefit in retarding progression in moderately severe Alzheimer’s disease. A recent meta-analysis examined vitamin E supplementation (alone and in combination with other vitamins and minerals) in doses up to 2000 IU a day, and reported a slight but significant risk for all-cause mortality with vitamin E dosage ≥ 400 IU a day (risk ratio 1.04, 95% CI 1.01-1.07). Of note, seven of the eight high-dosage studies in the meta-analysis that showed harmful effects of vitamin E involve participants with vascular risk factors or established cardiovascular disease. However, interpretation of the results of the meta-analysis is mitigated by methodologic concerns, including a possible type I error as the meta-analysis excluded vitamin E trials that reported fewer than 10 deaths and did not adjust for mortality over different follow-up periods.
Anti-inflammatory agents (such as non-steroidal anti-inflammatory agents and cyclo-oxygenase 2 inhibitors) are not recommended for the prevention of cognitive decline in Alzheimer’s disease.\textsuperscript{60, 61}

\textbf{Grade A, Level 1++}

Prednisolone is not recommended for the prevention of cognitive decline in Alzheimer’s disease.\textsuperscript{62}

\textbf{Grade B, Level 1+}

Oestrogen is not recommended for the prevention of cognitive decline in women with dementia.

\textbf{Grade A, Level 1++}

Contrary to evidence from epidemiological studies which suggests a protective role of oestrogens in Alzheimer’s disease, evidence from randomized controlled trials supports the ineffectiveness of estrogen for the treatment of Alzheimer’s disease.\textsuperscript{63-65} In addition, there are concerns about increased risk for heart attacks, strokes, breast cancer and thromboembolism with combination (oestrogen plus progestin) therapy.\textsuperscript{66}

There is insufficient evidence to support the use of other agents such as supplemental omega 3 polyunsaturated fatty acid,\textsuperscript{67} folate supplementation with or without B\textsubscript{12} (in the absence of a deficiency state),\textsuperscript{68} and statins\textsuperscript{69} for the prevention of cognitive decline in dementia.

\section*{4.3 Pharmacological interventions to improve cognitive symptomatology}

\subsection*{4.3.1 Acetylcholinesterase inhibitors}

Acetylcholinesterase inhibitors should be considered for the management of all patients with mild to moderate Alzheimer’s disease.

\textbf{Grade A, Level 1++}

There are currently three acetylcholinesterase inhibitors (AchEI) regularly used for the symptomatic treatment of dementia: donepezil, rivastigmine and galantamine (Table 4). Clinical trials (the majority
lasting one year or less in duration) involving the use of donepezil, rivastigmine or galantamine that are conducted in patients with mild to moderate Alzheimer’s disease consistently demonstrate modest improvement in (1) cognition and global functioning (on average, a 3-point difference on the 70-point Alzheimer’s disease assessment scale over a 6-month period), (2) activities of daily living and (3) neuropsychiatric symptoms (delay in emergence of symptoms, improvement in apathy, and variable patterns of improvement for milder degrees of anxiety, depression and hallucination). It is unclear whether AchEI therapy confers benefit in terms of reducing time to institutionalisation. The duration of benefit may persist as long as three years in some patients.

Patients in whom the start of AchEI treatment is delayed may demonstrate slightly reduced benefits as compared with those started on AchEI early in the course of disease. Although the placebo group did show improvement when switched to AchEI during open-label extensions of double-blind pivotal trials, they did not “catch up” with the group that received AchEI since trial inception. This suggests that AchEI may provide greater benefit when started as soon as dementia is diagnosed, rather than waiting until symptoms deteriorate further and become more prominent. In addition, drug holidays should be discouraged as they can be associated with clinical deterioration that may not revert to baseline even on resumption of therapy.

**B** Acetylcholinesterase inhibitors can be considered for the management of moderate to severe Alzheimer’s disease.

**Grade B, Level 1+**

There is evidence to suggest that the cognitive, functional and behavioural benefit of AchEI may extend to the more severe stages of Alzheimer’s disease. A recent Swedish study found that donepezil improves cognition and preserves function in individuals with severe Alzheimer’s disease (Mini Mental State examination score 1-10) who were living in assisted care nursing homes.

**A** Acetylcholinesterase inhibitors have been shown to be of clinical benefit and may be considered for use in the management of mild to moderate vascular dementia.

**Grade A, Level 1+**
Deficient cholinergic neurotransmission has been postulated to contribute to the cognitive impairment of vascular dementia. Two randomized, double-blind, parallel-group, placebo-controlled trials with a total of 1,219 people suffering from mild to moderate probable or possible vascular dementia have been published.\textsuperscript{80,81} Donepezil, at doses of 5 or 10 mg a day was compared with placebo for 24 weeks. A meta-analysis that includes these studies showed that the donepezil treated patients had a statistically significantly improvement in cognitive outcome compared to placebo treated patients on the cognitive subscale of the Alzheimer's Disease Assessment Scale (ADAS-Cog) as well as on the Mini-Mental State Examination (MMSE) at 12 and 24 weeks at both doses. However, in terms of global function and activities of daily living, patients showed a less uniform dose response.\textsuperscript{82}

Donepezil was well tolerated; most of the side effects were transient and were resolved by stopping the medication.

Moreover, galantamine showed statistically significant improvements in cognition (ADAS-cog), global functioning (CIBIC-plus), activities of daily living (DAD) and behaviour (NPI) in patients with vascular dementia diagnosed according to recognised criteria combined with a population of patients with Alzheimer's disease and coincidental radiographic findings of cerebrovascular disease.\textsuperscript{83}

Thus, AchEI have been shown to be effective and safe for the treatment of cognitive symptoms in vascular dementia. However, no AchEI has been licensed for the treatment of vascular dementia due to concerns as to the aetiological heterogeneity of patients included in the trials as well as lack of consistent effects on domains other than cognition.

\textbf{Acetylcholinesterase inhibitors can be considered for the management of dementia with Lewy bodies and Parkinson’s disease dementia.}

\textbf{Grade B, Level 1+}

There is growing appreciation that dementia with Lewy bodies (DLB) and Parkinson’s disease dementia (PDD) may actually represent different clinical manifestations of the spectrum of Lewy body synucleinopathies: the initial presentation is predominantly neuropsychiatric in DLB and motor in PDD.
Consistent with the neurobiochemical profile of cholinergic deficit in DLB, a study of 120 DLB patients reported that rivastigmine significantly reduced the core psychiatric symptoms of apathy, anxiety, delusions and hallucinations while enhancing cognitive performance in tasks with a substantial attentional component, without worsening the motor symptoms of Parkinsonism.\textsuperscript{84,85} Similarly, in Parkinson’s disease dementia, a rivastigmine study of 541 patients reported modest improvements in cognition mirroring the degree seen in Alzheimer’s disease, as well as benefits in activities of daily living and neuropsychiatric features.\textsuperscript{86} Although generally well tolerated, there were significantly more cholinergic-mediated adverse events such as nausea, vomiting and tremors in the treated group.\textsuperscript{86}

Clinical experience suggests that similar benefits in DLB and PDD may be observed with other AchEI, although more robust data are needed.\textsuperscript{87-89}

\textbf{B} All three available acetylcholinesterase inhibitors (donepezil, rivastigmine and galantamine) can be considered for the pharmacological management of dementia, since there is no definite evidence to support a difference in clinical efficacy between them.

\textbf{Grade B, Level 1+}

With regards to the three currently used AchEI (donepezil, rivastigmine and galantamine), there is very little to choose between them in practice in terms of core efficacy. The few head-to-head comparative studies are all industry sponsored, small, inconsistent in results, and offer little basis to make a clinical choice.\textsuperscript{78,90} The choice of AchEI therapy depends on the experience of the clinician, tolerance to side effects, ease of use, and the clinical profile of the individual to be treated (such as weight, co-morbid diseases and drug interactions). For instance, where medication compliance is an issue, once-daily formulations would be helpful. For patients who require medications to be crushed due to swallowing difficulties, the capsule formulations should be avoided (Table 4).

\textbf{A} Where tolerated, acetylcholinesterase inhibitors should be titrated to recommended doses (5-10 mg/day donepezil; 6-12 mg/day rivastigmine; 16-24 mg/day galantamine), which have been shown to confer greater benefit compared with lower doses.

\textbf{Grade A, Level 1++}
To reduce intolerability to gastrointestinal adverse effects, AchEIs are often started at lower doses (donepezil 2.5 mg/day; rivastigmine 1.5 mg twice daily; galantamine 8 mg/day) (Table 4). Studies have consistently shown that patients who received recommended doses of AchEIs exhibited better outcomes than those who received placebo or lower doses. Thus, where tolerated, AchEIs should be gradually titrated to recommended doses (5-10 mg/day donepezil; 6-12 mg/day rivastigmine; 16-24 mg/day galantamine) over 4-8 weeks.

4.3.2 N-methyl D-aspartate (NMDA) antagonists

N-methyl D-aspartate antagonists such as memantine can be considered for the management of moderate to severe Alzheimer’s disease, either alone or in combination with acetylcholinesterase inhibitors.

Grade B, Level 1+

Memantine appears to be beneficial alone or in combination with donepezil for moderately advanced Alzheimer’s disease. A study of moderate to severe Alzheimer’s disease patients on stable doses of donepezil (5-10 mg a day) reported that the addition of memantine 20 mg a day slightly improved cognitive, functional and global scores in comparison with patients taking placebo. Further analysis of the same study revealed that memantine reduced agitation/aggression in patients who were agitated at baseline and delayed its emergence in those who were free of agitation at baseline.

N-methyl D-aspartate antagonists such as memantine may be a treatment option for mild to moderate Alzheimer’s disease, if acetylcholinesterase inhibitor therapy is contra-indicated, not tolerated or if there is disease progression despite an adequate trial of acetylcholinesterase inhibitor.

Grade B, Level 1+

The efficacy of memantine in mild-to-moderate Alzheimer’s disease (alone or in combination with AchEIs) has yet to be firmly established. A recent study of memantine use in mild-to-moderate AD reported a small beneficial effect on cognition and behaviour, but not function. Taken together, memantine may provide a treatment option if AchEI treatment is contra-indicated, not tolerated, or if there is disease progression despite an adequate trial of AchEIs.
The initial dose of memantine is 5 mg once a day, with 5 mg increments at intervals of at least one week until a maximum of 10 mg twice a day is achieved (Table 4). It should be used with caution in patients with epilepsy and renal impairment, and the clinician should be aware of interactions involving commonly prescribed medications such as dextromethorphan and L-dopa.

**A** N-methyl D-aspartate (NMDA) antagonists have been shown to be of clinical benefit and may be considered for use in the management of mild to moderate vascular dementia.  

**Grade A, Level 1+**

There have been 2 clinical trials of memantine in mild to moderate vascular dementia.\(^{96,97}\) A Cochrane review\(^{98}\) of these 6-month studies showed that whilst memantine improved cognition and behaviour the clinical global measures did not show a significant difference. There was no difference in drop-out or adverse event rates between treatment and placebo groups.

Thus, memantine has been shown to be effective and safe for the treatment of cognitive symptoms in vascular dementia. However, memantine has not been licensed for the treatment of vascular dementia due to concerns as to the aetiological heterogeneity of patients included in the trials as well as lack of consistent effects on domains other than cognition.

### 4.3.3 Other treatment modalities

**B** Practitioners who prescribe ginkgo for the treatment of dementia should be aware of the unestablished benefit, variability of active ingredient among preparations, and potential for drug interactions.

**Grade B, Level 1+**

There is insufficient evidence to support that there is positive benefit of *Ginkgo biloba* on cognition and function in the treatment of dementia.\(^{99}\) There are two negative studies\(^{100,101}\) and the magnitude of benefit is less potent compared with AchEI.\(^{102}\) Practitioners need to be mindful that the dose of the active ingredient is not standardised and may differ among the different preparations. There are also clinically relevant drug interactions involving ginkgo, such as increased bleeding risk when combined with warfarin and antiplatelet
agents, and the antagonism of thiazides and anticonvulsants (valproate and carbamazepine).\textsuperscript{103}

\textbf{A} Selegiline is not recommended for the treatment of core or associated symptoms in Alzheimer’s disease.\textsuperscript{104} \hspace{1cm} \textbf{Grade A, Level 1++}

\textbf{GPP} Appropriate treatment of vascular risk factors is recommended for all patients. However, it should be noted that whilst promising observational data exists, it remains to be shown in a randomised controlled clinical trial if any prevention strategy such as blood pressure reduction or antiplatelet treatment for the secondary prevention of stroke, will reduce the incidence of vascular dementia.

\textbf{4.3.4 Treatment considerations}

\textbf{GPP} The decision to initiate costly symptomatic dementia treatment, such as acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists, should always be made in consultation with the patient and family after careful consideration of the expected magnitude of benefit, side effects, co-morbidities and costs of treatment.

\textbf{GPP} Despite the evidence of efficacy from clinical studies, there is debate over whether AchEI and NMDA-antagonist therapy are cost effective, because the treatment effects are small and not always apparent in practice.\textsuperscript{73,94,105,106} Cost-effectiveness issues become increasingly relevant in the advanced stages of dementia, since the magnitude of treatment benefit becomes less obvious and there are often competing demands on the available financial resources with regards to provision of care and placement. Until definitive findings are available, practitioners should continue to individualise treatment decisions for each patient.\textsuperscript{54} For instance, where financial resources are limited, the opportunity cost of employing a maid to look after a patient requiring help with activities of daily living may override the modest benefits of symptomatic therapy.
For many, the diagnosis of dementia can be devastating and thus, individuals with dementia and their family may have high, sometimes unrealistic, expectations of any treatments offered. It is therefore important to communicate with the patient and his caregiver/family from the onset that:

- The medications are not a cure.
- The medications may not work for everyone.
- Although there may be a response in terms of modest improvement or stabilization of symptoms, symptomatic therapy ultimately does not prevent progression of disease and cognitive decline will continue even with treatment.
- The medication may be discontinued if the patient does not respond after an adequate trial of 3-6 months.

**GPP** Patients who are started on acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists, should be carefully monitored for side effects and response to treatment.

**GPP**

Although generally well tolerated, dose-related gastrointestinal side effects (nausea, vomiting, diarrhea, anorexia) are common with AchEI use. These are transient and often circumvented to a large extent by a slower titration and taking the medication with food. Great caution should be exercised in those with bradycardia, sick sinus syndrome or cardiac conduction disturbances, in view of possible adverse effects of symptomatic bradycardia and syncope. Other less common side effects that have been reported include muscle cramps, insomnia, vivid dreams and weight loss. DLB and PDD patients commenced on AchEI should be carefully monitored for worsening of motor symptoms. Compared with AchEI, gastrointestinal-related side effects are uncommon with memantine use. Common adverse events of memantine include dizziness, headache, fatigue, hallucinations and confusion, but these tend to be transient.
Stabilisation or modest improvement above baseline may be observed in the first 6-9 months, which can be monitored by the use of clinical methods, via assessment of cognitive, functional and behavioural domains through interview with the patient and caregiver; and/or

(ii) standardized rating scales, which involves either:

a. brief mental status tests such as the Chinese Mini Mental State Examination (CMMSE), Abbreviated Mental Test (AMT) and Elderly Assessment Cognitive Questionnaire (ECAQ), or

b. more detailed psychometric testing. After 6-9 months, a lesser decline can be observed, which can be documented by patient and caregiver interview for cognitive, functional and behavioural (emergence of neuropsychiatric symptoms) features.

A trial of withdrawal of symptomatic treatment should be considered when the harm outweighs the benefit, and should be undertaken only after careful discussion with the patient and caregiver. Examples include intolerable or serious side effects, and progression of disease to the severe stages despite optimising treatment. When attempting withdrawal, it is important to monitor closely for any deterioration so that therapy can be quickly reinstated to regain the same level of symptomatic effect.
Table 4  Dosing information of dementia drugs in clinical use

<table>
<thead>
<tr>
<th>Medication</th>
<th>Forms</th>
<th>Dosing interval</th>
<th>Starting dose</th>
<th>Recommended dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Cholinesterase inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Donepezil (Aricept®)</td>
<td>Tablet (5 mg, 10 mg)</td>
<td>Once daily</td>
<td>2.5-5 mg once daily</td>
<td>5-10 mg/day</td>
</tr>
<tr>
<td>Rivastigmine (Exelon®)</td>
<td>Capsule (1.5 mg, 3 mg, 4.5 mg, 6 mg)</td>
<td>Twice daily</td>
<td>1.5 mg bid after meals</td>
<td>6-12 mg/day</td>
</tr>
<tr>
<td>Galantamine (Reminyl®)</td>
<td>PR Capsule (8 mg, 16 mg and 24 mg)*</td>
<td>PR capsules: Once daily Oral solutions: twice daily</td>
<td>8 mg once daily after meals</td>
<td>16-24 mg/day</td>
</tr>
<tr>
<td></td>
<td>Solution (4 mg/ml; 100 ml bottle)†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2) NMDA antagonists</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Memantine (Ebixa®)</td>
<td>Tablet (10 mg)</td>
<td>Twice daily</td>
<td>5 mg once daily</td>
<td>CCT &gt;60: 20 mg/day</td>
</tr>
<tr>
<td></td>
<td>Solution (10 mg/g oral drops)‡</td>
<td></td>
<td></td>
<td>CCT 40-60: 10 mg/day</td>
</tr>
</tbody>
</table>

(Source: British National Formulary; Geriatric Dosage Handbook, 11th Ed)

* PR: prolonged release once-a-day formulation. The immediate-release formulation has been phased out.
† Solution can be mixed with non-alcoholic beverage, but must be consumed immediately.
‡ 10 drops = 5 mg
CCT: Creatinine clearance
5 Management of Behavioural and Psychological Symptoms of Dementia (BPSD)

5.1 Non-pharmacological therapies (NPT)

GPP Non-pharmacological methods to manage behavioural and psychological symptoms of dementia should be instituted, prior to consideration of pharmacological measures.

Behavioural problems are a major cause of caregiver stress and often lead to premature institutionalisation of the patient. Factors such as pain or environmental triggers can be identified or manipulated and use of other non-pharmacological methods to manage behavioural problems prior to as well as in conjunction with pharmacological methods.

Problematic behaviours may respond to non-pharmacological therapies (NPT). NPTs are based on theoretical frameworks that conceptualize these behaviours, and four frameworks are particularly relevant.

First, the direct impact model whereby the behaviours are seen to be the direct result of brain pathology. Second, the unmet needs model\textsuperscript{108}, where unmet physical, emotional, spiritual and idiosyncratic needs underlie challenging behaviours. Behaviour is seen as a means of communication, a “cry for help” to fulfill a basic human need. Third, the behavioural model\textsuperscript{109,110} assumes connection between Antecedent, Behaviour and Consequence of the behaviour (ABC model), where antecedents operate through stimulus control, and the consequences reinforce behaviour. Modification of the behaviour would thus entail changing the antecedents and/or the consequences. Last, the concept of progressively lowered stress threshold\textsuperscript{111} (PLST) holds that dementia results in greater vulnerability to the environment, where minor stressors can precipitate anxiety, agitation and even a catastrophic reaction because the person with dementia is more susceptible and less able to cope with stressors or triggers in the environment compared to cognitively normal elderly individuals. Often, more than one model is needed to explain the problematic behaviour in question.
NPT are multifaceted and varied, and one form of intervention can bring about a broad range of effects. The appropriate NPTs are instituted when a good understanding of the issues behind the behaviour is procured. The following categories of NPT are noteworthy but this list is not exhaustive:

1) Medical/nursing care interventions, e.g. pain management, relief of fecal impaction and urinary retention, removal of restrainers, enhanced care methods such as person-centred showering and towel bath.

2) Environmental interventions, e.g. dementia safe and friendly environments, wandering paths, natural or enhanced environments, merry-walker.

3) Activities, e.g. structured activity programmes, physical rehabilitation and physical exercises.

4) Social contact, e.g. one-on-one interaction, pet therapy, simulated presence.

5) Timalation (interaction in which the senses are the main focus for engagement rather than interactions which involve an intellectual or emotional component), e.g. music, aromatherapy, massage, dance and movement, multi-sensory approaches such as snozelen.

6) Standard psychological therapies, e.g. behavioural therapy, validation, resolution, reality orientation, reminiscence.

7) Alternative therapies, e.g. art therapy, bright-light therapy.

8) Staff training.

Although the research evidence for these therapies is varied, some interventions being more evidenced-based than others, the reason for considering NPT first and as an enduring endeavour in addressing difficult behaviour is two-fold. First, NPTs guided by an understanding of behaviour in the frameworks elaborated above, address the underlying reasons for the behaviour. Second, medications carry adverse side-effects and often mask and suppress the behaviour that actually serves to communicate the need of the person with dementia. Therefore, the appropriate approach must entail trying to understand the etiology of the behaviour and addressing the problem at its root cause.
5.2 Pharmacological interventions to manage BPSD

5.2.1 Antidepressants

**GPP** Antidepressants may be used for the treatment of comorbid depression in dementia provided their use has been evaluated carefully for each patient.

The use of antidepressants for patients with dementia and depressive symptoms is common, however, their efficacy on depression and cognitive function is weak.\textsuperscript{112,113}

Evidence for use of older tricyclic antidepressants (clomipramine and imipramine) is weak.\textsuperscript{112} Selective serotonin reuptake inhibitor (SSRIs) which are commonly used to treat depression in the elderly does not appear effective in the treatment of neuropsychiatric symptoms of dementia other than depression.\textsuperscript{112,113} No current evidence is available on the newer antidepressants for treatment in dementia (e.g. newer classes of antidepressants such as selective noradrenergic reuptake inhibitors).

5.2.2 Antipsychotics

**A** Conventional and atypical antipsychotics may be used with caution, given their side effect profile, to treat neuropsychiatric symptoms of dementia.

**Grade A, Level 1+**

**Conventional antipsychotics**

Conventional antipsychotics have been used to treat behavioural problems associated with dementia. There is evidence for slight benefit of haloperidol over placebo for treatment of aggression. However, adverse events of extrapyramidal side effects and somnolence may limit its routine use.\textsuperscript{113} Low-potency antipsychotics like chlorpromazine should be used with caution in view of postural hypotension and anticholinergic side-effects.

**Atypical antipsychotics**

The atypical antipsychotics, olanzapine and risperidone have been shown to be modestly effective in the management of behavioural
problems in patients with moderate to severe dementia at doses of olanzepine at 5-10 mg/day and risperidone 1.0 mg/day. There is some evidence of quetiapine at doses of 25-100 mg/day showing improvement in agitation scores. In frail elderly patients, titration of dosage should be based on individual responses, starting at the lowest possible dose with gradual increments.

However, adverse effects of somnolence (5-8 times greater) and gait disturbance is 7.5-11 times more common in olanzepine-treated group compared to placebo. Serious adverse events occurred in 16.8% of risperidone versus 8.8% of placebo group, including 5 strokes and 1 transient ischaemic attacks, all in Risperidone group. Meta-analysis of adverse events performed showed 3-fold statistically increased risk of cerebrovascular adverse events with Risperidone and Olanzepine (no statistically significant increase in mortality) while another meta-analysis comparing risk of death with atypical antipsychotics (Aripiprazole, Olanzepine, Risperidone and Quetiapine) with placebo showed increase risk of death (OR 1.54, 95% CI 1.06-2.23) with number need to harm = 100 (95% CI 50-250). Other serious adverse events reported included somnolence and metabolic complications of hyperglycemia and weight gain.

Comparison between typical and atypical antipsychotics
Comparing the efficacy of typical and atypical antipsychotics, there is little good quality evidence to suggest one group is superior.

A recent retrospective cohort study had shown increased mortality among subjects using conventional antipsychotics compared to atypical antipsychotics.

Thus, despite the risks of atypical antipsychotics, there is currently no evidence to suggest that typical antipsychotics are better.

5.2.3 Trazodone

Trazodone may be considered for patients with depressive symptoms and dementia associated agitation. Grade B, Level 1+
One small randomised controlled trial showed reduction in agitation when accompanied by depressive symptoms in patients with dementia.\textsuperscript{122}

5.2.4 Mood stabilisers

Routine use of mood stabilizers, such as carbamazepine and sodium valproate, is not recommended for treatment of behavioural symptoms associated with dementia.

\textbf{Grade A, Level 1+}

There is no improvement in behavioural symptoms with mood stabilizers (carbamazepine and sodium valproate).\textsuperscript{113}

5.2.5 Benzodiazepines

There is no evidence of the efficacy of benzodiazepines in the treatment of behavioural problems associated with dementia.

There are no systematic reviews or randomised controlled trials of the use of benzodiazepines in the management of behavioural symptoms of dementia.

5.2.6 Treatment considerations

\textbf{GPP} An individualized approach to managing behavioural problems in dementia patients is required.

\textbf{GPP} Cholinesterase inhibitor therapy may be considered in treatment of patients with behavioural problems if antipsychotics are inappropriate.

\textbf{GPP} The decision to start antipsychotic therapy to control behavioural problems in dementia patients should be made in consultation with the patient and family, after careful consideration of the benefit, adverse-effects and co-morbidities.
In view of the potential adverse effects associated with antipsychotic therapy, non-pharmacological interventions and identification of pain and other environmental factors should be assessed and managed accordingly.

If the above fails and the behavioural problems are assessed to be significant causing difficulty in caring process (medically and functionally) with significant amount of caregiver distress, there has to be a discussion with the family members with regards to antipsychotic therapy, with the attendant risks of adverse effects (extrapyramidal side effects, somnolence, stroke, metabolic complications), especially in those patients with risk factors for cerebrovascular disease.

A recommended algorithm for management of neuropsychiatric symptoms of dementia is available (see Figure 1).

**B** For patients with dementia with Lewy Body and behavioural problems, acetylcholinesterase inhibitors should be considered first for management of the behavioural problems.

*Grade B, level 1+

Antipsychotic medication should be used cautiously in patients suspected to have dementia with Lewy Body as these patients have marked sensitivity to neuroleptic agents, including life-threatening neuroleptic malignant syndrome.\(^{123}\)

There is evidence of improvement of neuropsychiatric symptoms with Rivastigmine therapy in dementia with Lewy Body patients (see earlier section on acetylcholinesterase inhibitors treatment on dementia with Lewy Body).

**GPP** In all patients started on antipsychotic medication, they should be monitored carefully for side effects and response to treatment. In patients who are stable, antipsychotic withdrawal should be considered.

*GPP*
Figure 1  Algorithm for management of neuropsychiatric symptoms of dementia

Patient with dementia and a behaviour problem

Evaluate for and manage delirium, pain, other medical and environmental causes of behaviour

Evaluate the behaviour problem (Antecedent, Behaviour and Consequence of Behaviour)

Begin non-pharmacological management directed at specific behaviour. Caregiver education

Behaviour problem improved?

Does patient have symptoms of depression or anxiety?

Yes  Monitor for recurrence

No

Is patient receiving a cholinesterase inhibitor?

No

Begin trial of cholinesterase inhibitor with or without Memantine

Yes  Begin antidepressant (e.g. SSRI)

Behaviour problem improved?

Yes  Monitor for recurrence and adverse drug events

No  Begin trial of atypical antipsychotic medication

Behaviour problem improved?

Yes  Monitor for extrapyramidal symptoms and sedation. Attempt to taper medication every 6 months

No  Consider trial of mood stabilizers (valproate/carbamazepine). If still uncontrolled, consider referral to specialist.

Behaviour problem improved?

Yes  Monitor for recurrence and adverse effects

No

SSRI = selective serotonin reuptake inhibitor

Adapted with permission from KM Sink et al. JAMA 2005;293: 596-608. Recommended algorithm for management of neuropsychiatric symptoms of dementia.
Social and Caregiver Management of Dementia and Community Resources

Psychosocial interventions are targeted at both the person with dementia as well as the family caregiver. The goal is to enable appropriate care to be given according to the needs of the person with dementia and his caregiver, and these needs vary depending on the type and stage of dementia, as well as the systems in their ecological space. As the person with dementia is, by nature of the disease, largely unable to help himself, most interventions are directed at the family caregiver to indirectly help the person with dementia. These interventions are largely provided for by the community.

6.1 Family caregivers – rationale and role in dementia management

Most patients with dementia are cared for in their own homes by family members. Caregiver management is important for the following reasons:
1) Family caregivers need to be empowered with the necessary knowledge and skills, and psychosocial support to facilitate them in their task.
2) Family caregivers who face much negative consequences as a result of long-term caregiving need to be helped and supported.

Caregiver intervention can take several forms and they include:
1) Education sessions to impart knowledge on dementia and caregiving skills such as communication and behavioural techniques
2) Individual and family counselling
3) Regular caregiver support group meetings
4) Continuous availability of health care professionals and counsellors to provide support and help with crises and the changing nature of the patient’s symptoms
5) Respite care
6) Technology-based interventions

A Caregiver interventions via a multifaceted approach should be considered in the total management of the person with dementia.

Grade A, Level 1+
Several caregiver interventions have yielded promising results, the NYU spouse-caregiver intervention\textsuperscript{14,124,125} and the REACH\textsuperscript{126} (Resources for Enhancing Alzheimer’s Caregiver Health) initiative are noteworthy. They have been shown to reduce caregiver burden and depression, ease caregiver reaction to behavioural problems and delay nursing home placement. A meta-analysis\textsuperscript{127} also found that counselling and training caregivers in how best to care had moderate, statistically significant results. Multi-component interventions have a greater effect than narrowly focused ones. A support system that can meet the individual needs of different caregivers, and be able to provide on-going support that is responsive and continuous is most beneficial.

In addition, technology-based interventions such as web-based learning, on-line discussions and support groups, and telecommunications systems have burgeoned in recent years. Improvements in caregiver outcomes like depression and strain have been demonstrated in interventions involving technology.\textsuperscript{128,129} Caregivers also report they find the on-line resources useful and appreciate the ability to communicate with each other and health care professionals.\textsuperscript{130} Given the rapid advancement and easy availability of technology, it should be harnessed more fully to make help available to more family caregivers in a continuous, timely and economical way.

\textbf{GPP} Where appropriate, respite care can be offered to relieve the burden of caregiving on the family caregiver.

\textbf{GPP} Respite care can take place in the home of the person with dementia or a daycare centre. It may also vary in terms of who provides care; trained staff, relatives of the patient or volunteers. The care provided can also differ in duration, ranging from a couple of hours to weeks. Caregivers often express the need for respite to allow them sometime to rest, rejuvenate and have some moment to themselves. Although the few trials performed have not shown improvements in caregiver burden, improved caregiver satisfaction is reported.\textsuperscript{131,132} A systematic review\textsuperscript{133} failed to show any benefit in caregiver outcomes but as the review was only based on 3 studies that met inclusion criteria, this reflects the lack of high quality research rather than an actual lack of benefit.
6.2 Community resources

Referral to community resources to meet the care needs of the person with dementia and his/her carer should always be considered.

Integral to a comprehensive management plan is the referral of people with dementia to appropriate community resources. This assists in providing care and improving quality of life of both the patient and the caregiver. Generic eldercare services such as meal services, home help, befriender services, case management, home medical, home nursing and social day care services have already been established and some agencies are able to accommodate people with dementia on an ad hoc basis. However, referrals to specialised dementia services may be more suitable as such facilities have trained staff and special programmes for dementia. At present, dementia day care centres and nursing homes form the core of dementia services locally. It has been shown that the structured, social and modestly stimulating environment provided by a dementia programme, often offered in dementia day care centres and some nursing homes, can improve the well-being of people with dementia. The benefits of caregiver intervention and respite have been elucidated above. Other services such as home based dementia care and home respite care are still not well developed. Finally, organisations providing dementia care can also act as a resource centre that provides information, support, training and education for caregivers. The range of dementia services currently available are shown in Annex 1.
7 Ethical Issues in Dementia

7.1 Preamble

Dementia is a clinical syndrome characterized by global cognitive decline and impairments, which result in a loss of a patient’s individuality and autonomy. At different stages, the disease causes different extents of functional decline, and different severities in behavioural and psychiatric symptoms. The disease also results in varying extents of impairments in decision-making capacity, and at some point, a patient becomes mentally incompetent and ceases to be an autonomous agent.

Nevertheless, the dignity of the patient should be respected at all stages irrespective of the patient’s functional status. Therapeutic goals and treatment options that are coherent and appropriate to patient’s functional status should be individually tailored and adjusted at different stages of dementia, with an emphasis on quality for life of the patient, as well as the caregiver. The important question to ask for each individual patient is therefore what value of life holds for the patient, and directed at how life is experienced by the patient under the contextual circumstances specific to the patient. It is important to distinguish such evaluations from morally inappropriate social-worth evaluations, which base treatment decisions on an individual’s ability to contribute to society.\textsuperscript{135} The general ethical imperative must be to delay, prevent, stabilise dementia, but not to protract life and morbidity artificially in the severe stages of the disease.\textsuperscript{136}

7.2 Decision-making

1. Respect for autonomous decision making is a fundamental ethical and legal right of a mentally competent individual. This right of self-determination should be respected to the fullest possible extent, even in dementia or conditions associated with cognitive impairment.

2. When affected by dementia, the key to a patient’s right of autonomy is the presence of adequate decision-making capacity. As a patient’s cognition and hence functional abilities for decision making is impaired in dementia, a patient may or may
not possess adequate decision making capacity to make an informed choice.

3. A diagnosis of dementia per se, however, does not automatically imply a loss of decision making capacity, which is specific to each patient and to each medical decision. Therefore, those who cannot comprehend complex situations may still possess the capacity to make simple decisions, or to convey their opinions regarding the burdens and benefits of ongoing treatments.

4. In deciding if a patient with dementia possesses adequate capacity with respect to making a particular decision, a clinical evaluation of the following functional abilities should be made:
   a. Ability to express a choice
   b. Ability to understand information provided
   c. Ability to appreciate significance of information and relevance to self
   d. Ability to manipulate information rationally before arriving at a decision

5. If the patient lacks adequate decision making capacity, the ethical imperative switches to one that aims to protect the patient from his or her own harmful decisions or actions. Patients must not be under-treated nor forced to receive inappropriate treatment just because they lack decision making capacity or legally appointed guardian(s). Consideration of the patient’s functional status and quality of life is vital in making treatment decisions for the patient.

6. In Singapore, treatment decisions for a patient who is incapable of giving a valid consent will be made:
   a. in immediate, life threatening emergencies by doctors, based on the principle of medical necessity
   b. in less emergent situations, by:
      i. legal guardian (Committee of Person) appointed under the Mental Disorder and Treatment Act (Cap 178, 1985 Rev Ed)
      ii. the doctor, in accordance to principle of best interests (in the absence of any legally appointed guardian). Nevertheless, this does not exempt the doctor from communicating with patient’s family members and caregivers, so as to incorporate patient’s known values and established preferences in the determination of patient’s best interests. The opinions and sentiments of
the patient’s family ought to be sought, but they are not legally binding.\textsuperscript{139}

7. For patients adjudged clinically to have permanent and global decisional incapacity as a result of dementia, decisions about health care will be recurrent over their remaining lifetime. Therefore, when such issues arise, the attending physician may wish to consider a formal application should be made to the High Court for the declaration of mental incompetence and for the formal appointment of guardianship (committee of person(s) and committee of estate) under the provision of the Mental Disorder and Treatment Act (MDTA).

7.3 Genetic testing

1. As with any medical test, the decision regarding the utility of a genetic test should take into consideration the benefits to the individual patient and the ways that a test result would modify the care that the patient would receive.

2. As in any genetic testing, especially in pre-symptomatic susceptibility testing, individuals must be clearly informed\textsuperscript{140}:
   a. potential for severe psychological complications of testing positive for an incurable, devastating illness
   b. potential ramifications in the area of employment and medical insurance
   c. probabilistic implications of a positive test on genetically related family members, who may not have participated in any counselling or consented to testing

APOE ε4

3. There is a body of evidence that APOE ε4 is strongly associated with late-onset Alzheimer’s Disease (AD) and that when present may represent an important risk factor for the disease. However, at the present time, it is not recommended for use in routine clinical diagnosis nor should it be used for predictive testing.
   a. APOE genotyping does not provide sufficient sensitivity or specificity to be used alone as a diagnostic test for AD.\textsuperscript{141} It is therefore not recommended as a diagnostic tool in routine clinical evaluation of patients for sporadic early- and late-onset AD.\textsuperscript{136,141-147}
   b. Based on presently available data, APOE genotyping is not established as a predictive marker of AD. Furthermore,
APOE testing does not provide any medically useful information linked to treatments that are effective in preventing or delaying the onset of disease. Therefore, susceptibility testing in asymptomatic individuals is not recommended and may be associated with potential psychological harm.¹³⁶,¹⁴²-¹⁴⁴

### 7.4 Driving

1. Driving a motor vehicle is a complex task that requires the simultaneous and coordinated application of different cognitive abilities including ability to recall and apply traffic rules, sound judgement, attention span, and quick responses. A person’s ability to drive safely can therefore be affected in dementia,¹⁴⁸,¹⁴⁹ with consequently higher risk of accidents.¹⁵⁰,¹⁵¹

2. Driving also represents independence, freedom and mobility. The issue of driving in dementia requires therefore the balancing of individual freedom and patient confidentiality on one side versus public and patient safety on the other.¹⁵²

3. Dementia adversely affects driving performance even in its mild stages, although some persons with late-onset Alzheimer’s disease DAT appear capable of driving safely for some time after disease onset.¹⁴⁸ A diagnosis of dementia therefore does not automatically mean that a person is incapable of driving.¹⁴⁸,¹⁵³,¹⁵⁴ The decision should be based on dementia severity or a demonstration of impaired driving competence.¹⁴⁸

4. Longitudinal data suggests that driving performance deteriorates as the severity of dementia progresses over time,¹⁵⁵ primarily in early dementia. In one study, drivers with AD at a severity of CDR 1 were found to pose a significant traffic safety problem both from crashes and from driving performance measurements.¹⁴⁸

5. In patients with mild to moderate dementia, physicians find it difficult to identify which individuals should not drive. Performance-based measures of driving skills, such as on-road driving tests, are recommended as a means of assessing driving competency.¹⁵⁶,¹⁵⁷ A traffic-interactive, performance-based road test that examines cognitive behaviours provides an accurate and reliable functional assessment of driving ability.¹⁴⁸

6. Even if a driver with early dementia passes a road test, progression of the disease is expected to lead to deterioration in driving skills.¹⁵⁵ Therefore, repeat road testing at regular
intervals, usually 6 to 12 months, or earlier if suggested by significant cognitive decline, is important.

7. For patients who are assessed to be unsafe for driving, doctors should enlist the help of family members to persuade patient to stop driving. To encourage such patients to surrender their driving licences, alternative forms of transport should be arranged, where possible.\textsuperscript{158} If the patient is absolutely inflexible and insists on driving, thereby creating a reasonable risk to public safety, it is then ethically and professionally permissible for the doctor to breach doctor-patient confidentiality and file a report to the relevant licensing authorities.\textsuperscript{136} Other measures deemed to be ethical include hiding the patient’s car keys or immobilising the car if necessary.\textsuperscript{159}

\section{7.5 Truth-telling of diagnosis to patient}

1. Although patients generally would like to know the truth about their own medical condition, the rights of those who do not want to know should also be respected. Health care professionals should therefore seek to understand their patients' preferences with respect to the diagnosis of dementia and act appropriately according to their choice.\textsuperscript{160}

2. Studies have shown that the vast majority of patients with mild dementia wish to be fully informed.\textsuperscript{161,162} Therefore, unless a patient suffering from dementia explicitly declines to be informed of the diagnosis, the default mode should be to inform truthfully as it will enable the patient to:
   a. plan for optimal life experiences in remaining years of intact capacities
   b. designate and appoint a surrogate decision maker to take over the making of treatment decision upon eventual incompetence
   c. settle personal financial and legal matters
   d. participate in treatment decisions
   e. consider possible enrolment in research programmes and participate in informed consent process

3. When informing the diagnosis, the doctor needs to take into account the patient and family’s prior knowledge and their perception of the problems. The doctor should also be mindful of the impact the diagnosis can have on the patient’s life and family relationships. The communication should therefore be conducted sensitively and empathically, and should include a
discussion on treatment options and available support services both in the hospital and the community.

4. After the diagnosis has been communicated, the patient and family should be given time to process the information and to come to terms with it. They should be given ample opportunity to ask questions and seek clarification from the doctor.

5. The objectives of truthful disclosure of diagnosis to patients with dementia are to empower the patient with: the courage to request for information, the cognition to understand information and the strength and resources to cope with the burden of information.

7.6 Restraints

1. Restraints, whether environmental, physical or pharmacological, are used in the management of patients with dementia to restrict or control the patient’s movement or behaviour that may compromise the safety of the patient and/or others.

2. However, the use of restraints is not without potential problems:
   a. Risk of harm and injury
   b. Decrease in ability to perform cognitive and physical activities, thereby resulting in cognitive and functional decline, and ultimately loss of independence
   c. Loss of freedom, leading to loss of confidence and self-esteem

3. The preferred choice should therefore be to avoid the use of restraints. Restraints should not be a substitute for a proactive search for reversible precipitating factors for patient’s behavioural problems, or for good communication with the patient.

4. If restraints have to be used on a patient, it should be seen as a temporary means and should be stopped as soon as the indication is no longer present. Excessive use of restraints should be avoided - any restraint should therefore be instituted for the minimal period of time and at the minimal strength or degree needed to achieve the intended outcome for the patient. The patient should also be carefully monitored while on restraints.
7.7 Living alone

1. Many patients diagnosed to have dementia continue to insist on living alone. In some of these patients, cognitive decline arising from dementia leads to poor compliance with medical treatment, lack of safety awareness and poor judgement. All these can pose a risk to the safety and well-being of the patient. The patient may also be exposed, as a result, to mistreatment, fraud and exploitation by others.

2. A diagnosis of dementia does not automatically mean that the patient is incapable of living alone. This decision should be based on an assessment of the patient’s decision making capacity with respect to placement, and ability to continue living alone in the community without posing too much risk to self and to neighbours. Considerations should also be given to the potential negative social and physical impact of moving from a familiar environment to institutional care.

3. If the patient is assessed to have adequate decision making capacity and insists on living alone, the health care professionals should then support the decision by simplifying the daily tasks at home and using available community resources. The patient should also be reassessed as the dementia progresses and erodes both his decisional capacity and ability for self-care.

4. If the patient has doubtful decisional capacity, then there should be an assessment, preferably with the occupational therapist, on the following: safety to self and to neighbours when carrying out activities of daily living and tasks such as food preparation, handling of monies, laundry and compliance to medication.

5. The final objective is to provide the patient with dementia with a safe, familiar and comfortable living environment, and to avoid premature institutionalisation.
There are currently no local data on cost effectiveness of pharmacological agents, non-pharmacological methods and complex caregiver intervention programmes in dementia.

Due to the different healthcare funding mechanisms in other countries, we are thus unable to extrapolate from cost-effectiveness studies from overseas countries.
Clinical Quality Improvement

The following clinical audit parameters, based on recommendations in these guidelines are proposed:

1. Percentage of patients whose caregiver report subjective memory complaints underwent cognitive evaluation with subsequent appropriate management.

2. Percentage of patients newly diagnosed with dementia who had subsequent multi-pronged strategy to dementia management that encompasses education of patient and caregiver, non-pharmacological measures and comprehensive caregiver psychosocial intervention.

3. Percentage of patients newly diagnosed with dementia, institution of cognitive enhancers such as acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists, was made in consultation with the patient and family after careful consideration of the expected magnitude of benefit, side effects, co-morbidities and costs of treatment.

4. Percentage of patients newly diagnosed with dementia with significant behavioural problems, appropriate non-pharmacological management was instituted prior to consideration of pharmacological agents.

5. Percentage of patients newly diagnosed with dementia with significant behavioural problems despite non-pharmacological management, institution of antipsychotic therapy was made in consultation with the patient and family, after careful consideration of the benefit, adverse-effects and co-morbidities.

6. Percentage of patients newly diagnosed with dementia who had a referral to community resources to meet the care needs of the person with dementia and his caregivers.
## Diagnosis and treatment

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<td>Alexandra Hospital Geriatric Medicine Clinic</td>
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<td>2</td>
<td>Changi General Hospital Memory Clinic</td>
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<td>Institute of Mental Health Psychogeriatric Clinic</td>
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<td>4</td>
<td>National University Hospital Neuroscience Clinic</td>
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<td>Singapore General Hospital Dept of Neurology Geriatric Medicine Unit Clinic</td>
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## Dementia day care centres

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<tr>
<th></th>
<th>Alzheimer’s Disease Association (New Horizon Centre – Toa Payoh) (Has an Early Dementia Programme)</th>
<th>Blk 157, Toa Payoh Lor 1 #01-1195 Singapore 310157</th>
<th>Tel: 63538734 Fax: 63538518</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Alzheimer’s Disease Association (New Horizon Centre – Bukit Batok) (Has an Early Dementia Programme)</td>
<td>Blk 511, Bukit Batok St. 52 #01-211 Singapore 650511</td>
<td>Tel: 65659958 Fax: 65652257</td>
</tr>
<tr>
<td>3</td>
<td>Alzheimer's Disease Association - (New Horizon Centre- Tampines)</td>
<td>Tampines Polyclinic (Level 3) 1 Tampines Street 41 Singapore 529203</td>
<td>Te. 67865373 Fax: 67849587</td>
</tr>
<tr>
<td>4</td>
<td>Apex Harmony Lodge</td>
<td>10 Pasir Ris Walk Singapore 518240</td>
<td>Tel: 65852265 Fax: 65852982</td>
</tr>
<tr>
<td>5</td>
<td>Sunlove Dementia Day Care Centre</td>
<td>70 Buangkok View Singapore 534190</td>
<td>Tel: 63873593 Fax: 63863716</td>
</tr>
<tr>
<td>6</td>
<td>Sunshine Welfare Action Mission (SWAMI) Dementia Day Care Centre</td>
<td>5 Sembawang Walk Singapore 757717</td>
<td>Tel: 62576117 Fax: 67548443</td>
</tr>
<tr>
<td>7</td>
<td>Thong Teck Home for Senior Citizens (Day Care Centre)</td>
<td>91 Geylang East Avenue 2 Singapore 389759</td>
<td>Tel: 68460069 Fax: 68460396</td>
</tr>
<tr>
<td>8</td>
<td>Yong-en Dementia Day Care Service</td>
<td>Blk 335A Smith Street, #03-57 Singapore 051335</td>
<td>Tel: 62251002 Fax: 62255218</td>
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## Nursing homes with dementia facilities

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<tr>
<td>2</td>
<td>Ju Eng Home for Senior Citizens</td>
<td>205 Jalan Kayu</td>
<td>Tel. 64846891</td>
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<tr>
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<tr>
<td>3</td>
<td>Ling Kwang Home for Senior Citizens</td>
<td>156 Serangoon Garden Way</td>
<td>Tel. 62875466</td>
<td>Fax: 62843567</td>
</tr>
<tr>
<td></td>
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<tr>
<td>4</td>
<td>Lions Home for the Elders</td>
<td>41 Toa Payoh Rise</td>
<td>Tel. 62529900</td>
<td>Fax: 63535725</td>
</tr>
<tr>
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<tr>
<td>5</td>
<td>Peacehaven Nursing Home for the Aged</td>
<td>9, Upper Changi Rd North</td>
<td>Tel. 65465678</td>
<td>Fax: 65461831</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>6</td>
<td>Sunshine Welfare Action Mission</td>
<td>5 Sembawang Walk</td>
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<td>Fax: 67548443</td>
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<tr>
<td></td>
<td>(SWAMI) Home</td>
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## Carers support groups

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<tr>
<td>1</td>
<td>Alexandra Hospital Geriatric Centre Support</td>
<td>378 Alexandra Road</td>
<td>Tel. 63793420</td>
<td>Fax: 64714508</td>
</tr>
<tr>
<td></td>
<td>Group (in English)</td>
<td>Singapore 159964</td>
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<tr>
<td>2</td>
<td>Alzheimer’s Disease Association Support</td>
<td>Blk 157, Lorong 1, Toa Payoh</td>
<td>Tel. 63538734</td>
<td>Fax: 63538518</td>
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<tr>
<td></td>
<td>Group in English, Mandarin and Malay</td>
<td>#01-1195, Singapore 310157</td>
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<tr>
<td>3</td>
<td>O’Joy Care Services Support Group (in Mandarin)</td>
<td>701 Geylang Road</td>
<td>Tel. 67490190</td>
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<td>Teambuild Centre #03-03</td>
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### Training in dementia

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<td>1</td>
<td>Alzheimer’s Disease Association (including domestic helpers)</td>
<td>Blk 157, Lorong 1, Toa Payoh #01-1195 Singapore 310157</td>
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</tr>
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<td></td>
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<td>2</td>
<td>TSAO Foundation</td>
<td>5, Temasek Boulevard, #12-06 Suntec Tower Five Singapore 038985</td>
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### Home-based care

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<tr>
<td>1</td>
<td>Aged Psychiatry Community Assessment and Treatment Service Institute of Mental Health Department of Geriatric Psychiatry</td>
<td>10 Buangkok View Singapore 539747</td>
<td>Tel: 63892175</td>
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<td></td>
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References


3. Inter-Ministerial Committee on Health Care for the Elderly 1999.


119. UK Committee of Safety of Medicines (CSM) 9th March 2004.


Self-assessment (MCQs)

After reading the Clinical Practice Guidelines, you can claim one CME point under Category III (Self-Study) of the SMC Online CME System. Before you login to claim the CME point, we encourage you to evaluate whether you have mastered the key points in the Guidelines by completing this set of MCQs. This is an extension of the learning process and is not intended to “judge” your knowledge and is not compulsory. The answers can be found at the end of the questionnaire.

*Instruction: Choose either “True” or “False”.*

1. Dementia assessment and evaluation should be performed in the following group of patients:
   - A) Subjects with memory and other cognitive complaints (either self-report or caregiver-report)
   - B) Routine screening for all older subjects
   - C) Subjects with progressive forgetfulness
   - D) Subjects who arouse the physician’s suspicion of cognitive impairment during interview despite absence of memory or cognitive complaints
   - E) Subjects with strong family history of dementia

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2. Diagnosis of dementia
   - A) Dementia can be diagnosed clinically using DSM-IV criteria for dementia.
   - B) The patient can be diagnosed with dementia if he has memory impairment and deficits in one other cognitive domain (agnosia, aphasia, apraxia and executive functioning) although these cognitive declines do not impair social or occupational functioning.
   - C) Brief bedside screening instruments (such as ECAQ, AMT, CMMSE) may be used to aid in the diagnosis of dementia.
   - D) Neuropsychological testing is useful in detecting subtle cognitive difficulties not picked up by brief screening instruments.
   - E) Potentially reversible causes of dementia should be excluded in all patients who meet DSM-IV criteria for dementia.

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3. Complications of dementia
   A) Patients with dementia may present with anxiety and depression in the early stages of their illness.
   B) Caregivers of dementia patients with significant behavioural problems may experience coping difficulties and caregiver stress.
   C) Patients with dementia first experience difficulties in home functioning.
   D) Behavioural problems occur uncommonly in patients with dementia.
   E) Depression, agitation, anxiety, paranoia, hallucinations and sleep problems should be asked for routinely in patients with dementia.

4. The following are common causes of dementia
   A) Alzheimer’s disease
   B) Normal pressure hydrocephalus
   C) Dementia with Lewy Body
   D) Creutzfeld-Jakob disease
   E) Vascular dementia

5. Pharmacological management of dementia
   A) Pharmacotherapy is the most important aspect in dementia management.
   B) A multi-pronged strategy is required for dementia management, which consists of patient and caregiver education, non-pharmacological measures, pharmacotherapy and comprehensive caregiver psychosocial interventions.
   C) Oestrogen therapy should be considered for prevention of cognitive decline in women with dementia.
   D) Acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists for symptomatic treatment of dementia should only be started after careful consideration of expected magnitude of benefit, side-effects, co-morbidites and costs of treatment.
   E) Patients started on acetylcholinesterase inhibitors or N-methyl D-aspartate antagonists do not require regular monitoring and follow-up for side effects and treatment response.
6. Management of behavioural and psychological symptoms of dementia
   A) Pharmacological measures should be instituted initially for management of behavioural and psychological symptoms of dementia. ☐ ☐
   B) Non-pharmacological methods to manage behavioural symptoms of dementia entails understand the aetiology of the behaviour and addressing the problem at its root cause. ☐ ☐
   C) Antipsychotics (conventional or atypical) should be used as first line in pharmacological treatment of dementia. ☐ ☐
   D) Antidepressants may be used to treat comorbid depression in dementia. ☐ ☐
   E) Typical antipsychotics is more efficacious compared to atypical antipsychotics. ☐ ☐

7. Social and caregiver management of dementia
   A) Psychosocial interventions in dementia are targeted mainly at patients with dementia. ☐ ☐
   B) Family caregivers who face much negative consequences as a result of long-term caregiving need to be helped and supported. ☐ ☐
   C) Respite care can be offered to relieve the burden of caregiving on the family caregiver. ☐ ☐
   D) In meeting the care needs of the person with dementia and his/her carer, referral to community resources should be considered. ☐ ☐
   E) Dementia daycare centres are useful in patients in dementia in keeping them active, improve their sleep and aid in reducing caregiver stress. ☐ ☐
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## Workgroup members

The members of the workgroup, who were appointed in their personal professional capacity, are:

**Chairman**

Dr Chong Mei Sian
Consultant
Department of Geriatric Medicine
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<table>
<thead>
<tr>
<th>Name</th>
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<tbody>
<tr>
<td>Dr Christopher Chen Li-Hsian</td>
<td>Senior Consultant</td>
<td>Dept of Neurology NNI (SGH Campus)</td>
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<td>Consultant</td>
<td>Dept of Geriatric Medicine TTSH</td>
</tr>
<tr>
<td>Prof Kua Ee Heok</td>
<td>Head, Dept of Psychological Medicine,</td>
<td>Yong Loo Lin School of Medicine, NUH</td>
</tr>
<tr>
<td>Dr Philip Yap Lin Kiat</td>
<td>Consultant</td>
<td>Dept of Geriatric Medicine AH</td>
</tr>
<tr>
<td>Dr Chiam Peak Chiang</td>
<td>Chief &amp; Senior Consultant</td>
<td>Dept of Geriatric Psychiatry WH/IMH</td>
</tr>
<tr>
<td>Dr Francis Ngui Tet Shin</td>
<td>Medical Director</td>
<td>Adam Road Hospital</td>
</tr>
<tr>
<td>Dr Lim Wee Shiong</td>
<td>Consultant</td>
<td>Dept of Geriatric Medicine TTSH</td>
</tr>
<tr>
<td>Dr Tan Kok Leong</td>
<td>Director</td>
<td>Singhealth Polyclinics-Outram</td>
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Subsidiary editors

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Assistant Director (Health Technology Assessment)
Health Services Research & Evaluation Division
Ministry of Health

Dr Rajni Gupta
Executive (Health Technology Assessment)
Health Services Research & Evaluation Division
Ministry of Health

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Senior Evidence-Based Medicine Analyst
and
Co-Director of the Singapore Branch, Australasian Cochrane Centre
Clinical Trials & Epidemiology Research Unit