



## Australian and New Zealand Society for Geriatric Medicine

### Position Statement No 28

### Dementia in Older People

#### Key points

- Dementia is a chronic neurodegenerative illness with no disease modifying treatment currently available.
- Dementia is characterised by multiple cognitive impairments, severe enough to cause a decline in social or occupational function.
- Timely diagnosis allows for education, counselling and future planning of lifestyle and legal issues of older people and their families.
- Dementia has a major impact on the lives of those with the condition, their families and carer partners.
- Alzheimer's disease (AD), vascular cognitive impairment (VCI), vascular dementia (VaD), dementia with Lewy bodies (DLB) and frontal lobe dementia (FLD) account for the majority of cases.
- The prevalence overall is increasing with an aging population.
- Exclusion of conditions such as depression and delirium is important as they may mimic dementia.
- Behavioural and psychological symptoms of dementia (BPSD) occur in almost all people at some stage and are associated with significant carer stress.

*This Position Statement represents the views of the Australian and New Zealand Society for Geriatric Medicine. This Statement was approved by the Federal Council of the ANZSGM on 4<sup>th</sup> December 2017. Authors: Dr Shabana Ahamed and Dr Dina Logiudice.*

#### BACKGROUND

Dementia has a major impact on the lives of those with the condition, their families and carers. It is a disorder that presents many challenges to health professionals throughout the pathway of care, from early diagnosis to end of life.

Dementia is not a normal part of ageing, but predominantly affects older people. The main underlying causes of dementia in older people include Alzheimer disease (AD), vascular cognitive impairment (VCI)/vascular dementia (VaD) and dementia with Lewy bodies (DLB).

In 2012, dementia was declared the ninth Australian National Health Priority Area, and a national framework for action (2015-2019) has highlighted a number of areas to address, These include: increasing awareness and reducing risk; need for timely diagnosis; and accessing care and support throughout including during hospital admissions and at end of life with guidelines from the NHMRC Clinical Practice Guidelines and Principles of Care for People with Dementia also having been endorsed by the ANZSGM in 2016.

#### DEFINITION

**Dementia** is currently defined as an acquired condition of the brain characterised by multiple cognitive impairments, severe enough to cause a decline in social or occupational function, and not better accounted for by delirium or depression.<sup>1</sup> Recent Diagnostic and

Statistical Manual of Mental Disorders, Fifth Edition (DSM V) <sup>2</sup> criteria describe cognitive impairment and dementia in the realm of a neurocognitive disorder.

### **MILD COGNITIVE IMPAIRMENT (MCI)**

Mild cognitive impairment (MCI) is thought to be twice as common as dementia. It is a syndrome defined as objective cognitive decline greater than expected for age and education that does not interfere notably with activities of daily living (ADL's).

In the DSM-5, mild neurocognitive decline (NCD), which is a new and separate entity, is comparable to mild cognitive impairment (MCI) and to prodromal dementia.

MCI can progress to dementia in 17% or revert to normal in 40%.<sup>3</sup> Those with amnesic MCI have an increased rate of conversion to dementia (predominantly AD).<sup>4</sup> Other predictors of progression to dementia include: age, hypertension, lower education, worse verbal and executive dysfunction, depression and subtle changes in IADL function.<sup>5</sup> Therefore it is recommended that people with MCI are reassessed over time. <sup>6</sup>

### **EPIDEMIOLOGY**

It is estimated that 322 000 people in Australia and 48,000 people (1.1%) of the New Zealand population live with dementia, a number that is expected to triple by 2050.<sup>7, 8, 9</sup> Prevalence and incidence of dementia increases with age, with an estimated 20% of people over 85 years affected. The rate of dementia doubles for every 5 years of life after the age of 60 years and continues to rise exponentially even in the oldest-old.<sup>10</sup> Five percent have an onset before the age of 65 years.<sup>11</sup> However, there are some recent European studies showing the decline of dementia cases.<sup>12</sup> Dementia is thought to be more prevalent in Indigenous people <sup>13,14,15</sup>, and rates are projected to increase in the future.

Dementia is the leading cause of non-fatal burden of disease in those over 75 years, and accounts for 6% of all deaths.<sup>16</sup> Estimates of median survival from onset of symptoms vary

but are estimated as 4.1 years for men and 4.6 years for women, influenced by age and disability.<sup>17</sup> Older people with dementia admitted to acute hospitals comprise a vulnerable group – with higher rates of morbidity and mortality, increased length of stay and greater likelihood of experiencing adverse events or admission into residential care.<sup>18</sup>

Both depression and dementia account for significant years of life lost to disability. Of those with moderate to severe dementia, 43% live in residential care accommodation and 57% live in households.<sup>7</sup> Dementia is projected to be the greatest cause of health expenditure by 2060.<sup>7</sup>

### **TYPES OF DEMENTIA**

Over 70 diseases can cause dementia or chronic cognitive impairment. Alzheimer's disease (AD), vascular cognitive impairment (VCI), vascular dementia (VaD), dementia with Lewy bodies (DLB) and frontal lobe dementia (FLD) account for approximately 90% of cases of dementia. Mixed pathology is very common in people over the age of 85 years.<sup>19</sup>

#### **Alzheimer's disease**

One underlying pathogenesis for AD has been hypothesised to be due to amyloid cascade. The neurofibrillary tangle composed of hyperphosphorylated tau is also a pathological hallmark of AD, and tau pathology is noted early in the enterorhinal and hippocampal regions.<sup>20</sup>

Alzheimer's disease (AD) is the most common form of dementia. It is a neurodegenerative condition that is insidious and progressive, predominantly affecting the temporoparietal cortical areas of the brain and resulting in loss of memory function and language abilities.<sup>21</sup> Impaired new learning and recall (amnesic loss not improved with prompting) displayed as pervasive forgetfulness is an early feature. Communication changes are noted, with shrinking vocabulary, expressive or word-finding difficulties, and repetitive conversations. Visuoconstructional deficits, spatial disorientation and dyspraxia become evident. As the condition progresses these

deficits become more prominent and involve impaired executive function, with impaired reasoning and planning abilities. Interference with everyday activities of cooking, cleaning and attending to finances become more prominent, with the person requiring increased assistance. With progression, agitation, altered sleep patterns, incontinence and swallowing disorders can become more evident, often leading to the need for residential care, but are not universal in advanced disease.

Although memory impairment is frequently a presenting symptom, it is not seen in all instances,<sup>22</sup> and plateauing in cognitive function can occur. Focal cortical syndromes describe atypical non-amnesic presentations of AD, and include the following: (a) language presentation, with prominent word-finding problems; (b) visual presentation (including difficulty with object recognition (agnosia), impaired face recognition and problems with reading (alexia); and (c) executive dysfunction. The prominent deficits may therefore begin in a biparietal or occipitotemporal manner.<sup>23</sup> In the older person comorbid presentation with VCI or DLB may alter the typical presentation of disease. Common risk factors include: age, diabetes, depression, ApoE4, hypertension for women and heart disease for men.<sup>24</sup>

Early onset AD (EOAD) is uncommon and more frequently associated with a genetic abnormality, such as presenilin genes. Those with EOAD are more likely to present with atypical features and progress rapidly.<sup>22</sup>

The McKhann criteria (1984) which initially emphasized clinical features has been revised recently (2011)<sup>25</sup> as there has been increasing neuropathological evidence. Although promising, the use of biomarkers are currently more for research rather than of clinical utility.

**Table: Main differences between the old and new criteria for diagnosis of AD**

<b>New guidelines NIA 2011</b>	<b>McKhann Criteria 1984</b>
Considers AD as a progressive disease on a spectrum in three	Recognised only one stage – Alzheimer’s dementia

stages: a) dementia due to ADs b) mild cognitive impairment due to AD and c) preclinical AD	
Considers memory loss and other aspects of cognition e.g. judgement, word finding difficulty etc	Memory loss is the main focus
Use of biomarkers measured in blood, fluid and neuroimaging	Relied on clinical features only

### Vascular dementia

This is the second most common type of dementia and accounts for 10 to 20% of cases. Age is the most consistent risk factor. Other risk factors, initially thought to predict VaD are also associated with AD (as above).<sup>24</sup>

The relationship between cognition and vascular damage is not completely understood. While vascular lesions have been found to be associated with vascular risk factors and the development of dementia, they also occur in many older people without dementia.<sup>26,27</sup>

The term Vascular Cognitive Impairment (VCI) encompasses a spectrum of disease that includes subtypes described on the basis of clinical and radiological findings, ranging from mild cognitive impairment, to Vascular Dementia (VaD) and mixed neurodegenerative dementia (e.g. AD and VCI). Underlying causes of VaD may include post-stroke dementia, multi-infarct dementia, subcortical dementia, strategic infarct (e.g. thalamic region), hypoperfusion and hereditary causes e.g. CADASIL.<sup>28</sup> Memory loss may not be an early feature of vascular dementia, with deficits in executive function, including attention, speed of processing, psychomotor and motor slowing and gait disturbance being more prominent. Language is generally preserved, and deficits in retrieval of memory (rather than encoding) are noted.

Cerebral small vessel disease, including subcortical infarcts, white matter

hyperintensities, lacunes, prominent perivascular spaces, cerebral microbleeds and atrophy, is a common cause of VaD as well as a significant contributor to mixed dementia.<sup>29</sup>

### **Lewy body dementias**

Lewy body dementias describe both Parkinson's Disease Dementia (PDD) and Dementia with Lewy bodies (DLB), and if both mental and motor symptoms appear within one year of each other, then DLB is more likely the cause.

DLB is characterised by the presence of Lewy pathology which are abnormal aggregates of the synaptic protein alpha-synuclein which form inclusion bodies (Lewy bodies) or Lewy neurites.<sup>30</sup>

DLB affects approximately 10% of elderly people with dementia and PDD affects 20-30% of people with Parkinson's Disease. It is associated with higher rates of morbidity, mortality, carer stress and a poorer quality of life than AD.

Characteristics include early visuospatial problems, executive dysfunction with prominent attentional impairment and less impaired episodic memory than in AD.

Core features of DLB include recurrent visual hallucinations, cognitive fluctuation and motor parkinsonism. Other common features include rapid eye movement sleep behaviour disorder, delusions, depression, anxiety, autonomic dysfunction and recurrent falls.<sup>30</sup>

The distinction between PDD and DLB is not always clinically clear. There appear to be some subtle differences that suggest presence of DLB, including more conceptual and attentional deficits, predominant and often florid hallucinations, and sensitivity to neuroleptics.<sup>31</sup> Relative preservation of memory is noted until later in the disease.

It is thought that many of these features in the spectrum of DLB are non-dopaminergic, with less prominence of tremor, rigidity and bradykinesia normally seen in PD<sup>32</sup> and

greater emphasis on postural instability and falls and lack of facial expression. As a result of striatal degeneration, L-dopa responsiveness is less likely.

Neuroleptic sensitivity is suggestive of DLB. Antipsychotics (useful for delusions or hallucinations) such as haloperidol and risperidone can worsen parkinsonism and precipitate fatal sensitivity reactions in up to 50% of people.<sup>31</sup> Quetiapine has demonstrated no significant increase in parkinsonism, although there may be associated functional decline if non-pharmacological strategies are ineffective.<sup>33</sup>

### **Frontotemporal dementia**

This accounts for 10 % of dementia cases with predilection for frontal lobe damage. It tends to occur in younger people and has a genetic link. FTD is clinically characterised by behavioural and language disturbances that may precede or overshadow memory deficits.

The underlying brain changes affect predominantly the frontal lobes (Behavioural-variant FTD). In contrast, other people show change in language proficiency, either in the form of a difficulty understanding the meaning of words (Semantic Dementia), or a difficulty using the correct words (Progressive Non-fluent Aphasia)<sup>34</sup>.

### **RISK FACTORS AND PREVENTION**

The major proven risk factors for dementia are age and genetic predisposition, including ApoE4.<sup>20</sup> Emerging research indicates that several risk factors, initially thought to predict VaD are also associated with AD.<sup>45,46</sup> These include hypertension, diabetes mellitus, atrial fibrillation, carotid artery disease, obesity, hyperlipidaemia and hyperhomocysteinaemia.

Other factors included: head injury associated with loss of consciousness,<sup>47</sup> depression,<sup>48</sup> traumatic stress disorder,<sup>49</sup> smoking<sup>50</sup> and use of anticholinergic medications<sup>51, 52</sup>.

Increased physical activity is associated with a decreased risk of AD, and randomised trials

have demonstrated cognitive benefits from physical activity in those at risk of dementia.<sup>53,54</sup>

Social engagement, higher education, promotion of cognitively stimulating activities lessens the risk.<sup>54</sup>

A public health approach is being considered to identify risk factors and optimise interventions to reduce the incidence of dementia.

## ASSESSMENT OF DEMENTIA

### Approach to diagnosis

The diagnosis of dementia should only be made after a comprehensive assessment that includes personal and informant history, cognitive assessment, physical examination, review of medications and neuroimaging, and should be undertaken within a framework of person-centred care.

There is often an element of therapeutic nihilism with respect to diagnosis of dementia, due to lack of an effective cure and stigma associated with the diagnosis.<sup>35</sup> There is evidence that the earlier identification of people with dementia and appropriate referral to support services and counselling can alleviate some of the stresses associated with caring for people with dementia and delay institutionalisation.<sup>36</sup> Benefits of timely diagnosis include planning for the future, particularly with respect to finances and lifestyle decisions. Monitoring risk factors, reviewing potentially harmful medications and being alert to the increased risk for depression and delirium may assist in the management of a person with dementia. One of the priorities stated in the recent National Framework for Action on dementia describes the need for a timely diagnosis, that reflects the important role of the primary care physician, supported by access to clinical specialists and teams described within accessible pathways of care.

### Clinical evaluation

Evaluation focuses on determining a pattern of

decline and potential factors that may contribute to the clinical picture. Alzheimer's disease is typically of insidious onset over a number of years, without predominant physical neurological features. Therefore the emphasis is obtaining a thorough collateral history from key informants. Vascular dementia may have a more abrupt onset with associated neurological signs, and dementia with Lewy bodies is frequently associated with fluctuations, prominent hallucinations and parkinsonian features. See Box 1.0 for clinical features that indicate alternatives to AD. Of course, mixed pathologies are very common and conditions such as VCI and DLB may present with a mixture of clinical cognitive findings.

#### Box 1.0 Clues to consider diagnoses other than AD

**Abrupt onset:** vascular dementia, delirium

**Prominent behavioural:** frontotemporal, vascular dementia

**Profound apathy:** frontotemporal, vascular dementia

**Gait disorder:** vascular dementia, Parkinson's disease dementia, normal pressure hydrocephalus

**Prominent fluctuations:** delirium, DLB, epilepsy, metabolic disturbances

**Hallucinations/delusions:** DLB, delirium

**Frequent falls:** DLB, vascular dementia, parkinsonian syndromes

**REM sleep disorder:** DLB, Parkinson's disease

Adapted from Kester & Scheltens 2009<sup>5</sup>

Clues that indicate possible underlying cognitive impairment include vague complaints, forgetting scripts and appointments, word-finding difficulties, irritability, recent decline in previously controlled symptoms, for example diabetes management, and increasing withdrawal from activities. All concerns regarding cognitive symptoms and change in function should be taken seriously and further investigations and evaluation of cognition are required.

## **COGNITIVE ASSESSMENT SCREENING TOOLS**

Although routine population screening is not recommended, medical practitioners should be aware that cognitive impairment is increasingly common with age and as such, investigate concerns raised by a person or their carer.

The amount and type of cognitive testing required is determined by the clinical situation and the level of cognitive impairment. In addition to informant history, the initial cognitive assessment utilises a short cognitive test. This determines the need to perform further testing to establish the diagnosis. However, clinicians commonly miss cases of dementia in their routine practice without the assistance of formal cognitive assessment.<sup>42</sup>

There are several cognitive screening tools commonly used, including Mini-Mental Status Examination (MMSE), Rowland Universal Dementia Assessment Scale (RUDAS), Addenbrooke's Cognitive Examination III, Montreal Cognitive Assessment and the Kimberley Cognitive Assessment Scales (for older Aboriginal and Torres Strait Islander Australians living in remote and rural areas). The GP Cog is commonly used in primary care.<sup>43</sup>

There are many other screening tools available, but have their limitations, the most obvious being that they test only limited domains of cognition. It is essential to gather collaborative history from an informant, typically a family member. The Australian-developed Informant Questionnaire for Cognitive Decline in the Elderly (IQCODE)<sup>43</sup> is well validated.

Neuropsychology assessment is useful for those who appear to be in a transition stage with mild cognitive changes or if further clarification or exclusion of deficits is required. Other allied health professionals such as speech pathologists and occupational therapists are helpful.

## **ASSESSMENT OF FUNCTION**

A diagnosis of dementia requires evidence of functional decline. This information is often forthcoming from family members who are aware of changes in the person's ability to cope with everyday things, reflecting initially in instrumental activities of daily living, followed by loss in ability to maintain personal care. In other people, particularly those of CALD backgrounds, determination of functional decline may be a challenge where families step into the role as part of their perceived duty, or else certain functions may have never been performed. Therefore there is a need to define what cannot be done compared to what is never done.<sup>44</sup> Assessment and understanding of function is also key in implementing appropriate care supports. Areas of functional decline include driving ability, accuracy in completing financial transactions, safety within the home environment, ability to cook and maintain adequate nutrition, and decision-making capacity around complex issues and tasks.

## **INVESTIGATIONS**

Laboratory investigations should be done to rule out the presence of a reversible or partially reversible condition. Routine tests include electrolyte and renal function, B12 and folate deficiency, thyroid function tests, calcium, glucose and selective testing for vasculitides, neurosyphilis or HIV antibodies. Currently there is no evidence of diagnostic utility of ApoE allele status.

It is recommended that some form of neuroimaging is required to exclude other organic pathologies and potentially help differentiate among various dementia subtypes.

Neuroimaging is most informative when performed as part of a comprehensive dementia evaluation. It can detect treatable causes of dementia (yield 1-10%)<sup>37,38</sup> and although CT imaging is cheaper and more accessible, whereas MRI can better assess medial temporal lobe atrophy, presence of vascular pathology and differential pattern of lobar atrophy.<sup>39</sup>

The sensitivity and specificity of hippocampal atrophy for detecting mild to moderate AD is 85% and 88% respectively.<sup>40</sup>

MRI changes in PDD and DLB are similar, with a key feature appearing to be preservation of the medial temporal area, the latter commonly involved in AD.

Functional imaging, such as SPECT or PET, may delineate hypometabolism or hypoperfusion in specific regions associated with disease pathology, although functional imaging is less specific than MRI and its clinical utility is still not determined.

Newer neuroimaging technologies are being developed, e.g. SPECT (DATScanTM) which is useful to differentiate DLB from AD.<sup>41</sup>

Use of EEG and CSF are less common and would be more useful to exclude other pathologies of cognitive decline, such as demyelination, vasculitis, neurosyphilis and prion diseases.

### **DIFFERENTIAL DIAGNOSIS**

An essential part of assessment is exclusion of diseases such as depression that may mimic dementia. It is difficult to make a diagnosis of dementia for the first time when there is coexisting delirium.

Depressive symptoms are noted in 20–40% of cases of dementia and more common in the earlier stages of dementia and VCI. Screening scales for depression such as Geriatric Depression Scale<sup>55</sup> or Cornell Depression Scale for Dementia<sup>56</sup> may be useful. The clinician needs to be aware that depression may present with features of cognitive impairment (i.e. pseudodementia) and may require treatment in the first instance. Serial assessment will help determine whether dementia coexists. As treatment of depression in older people is frequently effective, regular monitoring is required and the threshold for intervention should be low.

Drugs, particularly anticholinergic medications, may cause a delirium or other cognitive effects and should be reviewed. Despite the known

adverse effects, these medications continue to be prescribed and chronic anticholinergic use potentially may increase risk of dementia.<sup>51, 52</sup> Other conditions that may mimic dementia or contribute to confusion include sleep apnoea, temporal lobe epilepsy, space-occupying lesion, metabolic disease and others.

Delirium can have features similar to dementia however the onset is often acute and attention is impaired. The incidence of delirium particularly during hospital admissions is common and can be as high as 70% in those admitted to intensive care.<sup>57</sup> There is a high rate of functional decline, under/mis diagnosis of dementia in the setting of delirium.<sup>57</sup>

### **MANAGEMENT**

The post diagnostic management of dementia requires a holistic multidisciplinary approach, which aims to optimise the independence of the person with dementia and support their family and carers. Apart from the economic burden, the psychosocial stress encountered by those with the condition, their families and carers, is high. It is essential to involve the caregivers and families in the treatment plan. Management will also vary and evolve according to the stage of the dementia, including the timely and appropriate implementation of services and support, such as respite and packaged care.

Early personal and financial planning to discuss about Enduring Power of Attorney/Guardian and advance directives is important. Education about diagnosis can be obtained from memory clinics and Alzheimer's Australia.<sup>58</sup> Safety assessment should involve a multidisciplinary approach (occupational therapy, falls prevention). Carers can get assistance through the Dementia Helpline, Alzheimer's New Zealand, Dementia Behaviour Management Advisory Service (DBMAS)<sup>59</sup> Carers Link,<sup>60</sup> council services, My Aged Care website and respite services.

### **PHARMACOLOGICAL MANAGEMENT**

Medications play a limited but important part in management of dementia, providing symptomatic benefit with acetyl cholinesterase inhibitors (AChEI) (donepezil, rivastigmine and

galantamine) and memantine, which is a N-methyl-D-aspartate partial antagonist.

AChEI have shown modest symptomatic improvement in 40-50% of people in cognitive function, global outcomes and activities of daily living.<sup>61</sup> Evidence suggests positive benefits for non-cognitive symptoms, especially apathy, depression and anxiety and improved function compared with placebo.<sup>62</sup>

Side effects include nausea, vomiting, diarrhoea, dizziness, weight loss and bradycardia.<sup>63</sup>

Memantine is well tolerated, with main side-effects, including constipation, dizziness and headache and has a potential role in the management of agitation in more severe disease.

In Australia and New Zealand, subsidised funding for AChEIs is only available for those with Alzheimer's Disease. Restricted criteria are available for memantine in Australia and rivastigmine in New Zealand for those with AD. A combination of both drugs has been trialled in clinical studies, yet evidence of significant improvement is lacking.<sup>64</sup>

There is no evidence of benefit for vitamin E and other antioxidants, Gingko biloba, oestrogen, omega-3 polyunsaturated fatty acids and calcium channel blockers. The use of anti-amyloid therapies in established dementia has been ineffective.

Pharmacological treatment options of VaD have been disappointing. Aspirin has not been shown to be effective. AChEIs have been shown to provide modest improvement in outcomes, although likely confounding of associated AD limit interpretability. Memantine confers mild improvement in cognitive scores, but no substantial improvement in function. In Australia, neither drug is subsidised on pharmaceutical benefits scheme for VaD.

Cholinesterase inhibitors form the mainstay of treatment of cognitive and psychiatric symptoms in DLB with benefits observed in

cognitive fluctuation, delusions, hallucinations and sleep disturbance.<sup>65</sup>

As DLB is characterised by cholinergic loss, cholinesterase inhibitors have demonstrated improvement in fluctuations, hallucinations, apathy, anxiety and sleep disturbance.<sup>66</sup> Adverse effects of the cholinesterase inhibitors include worsening tremor and drooling.<sup>67</sup>

Neither rivastigmine nor other medications are approved on PBS in Australia for use in DLB and memantine may be of modest benefit.<sup>68</sup> Anti-parkinsonian medications may be helpful for the treatment of motor symptoms in some people with DLB. Those with DLB present at older age and are less responsive to L-dopa than are those with typical Parkinson's disease (PD).

Review and rationalisation of medications is important, as is the optimisation of other concurrent medical conditions and co-morbidity, as the risk of dementia has been shown to increase with the number of medications used.<sup>69</sup> Addressing sensory losses in addition to an early dental review is also of benefit.

**Non-pharmacological interventions** should be considered as strategies for the person and caregiver. Few research studies<sup>70,71</sup> have shown cognitive training (e.g. computerised brain training) to improve daily function, memory function and delay symptom progression.

The evidence is limited to particular domains and does not generalise, therefore the need for larger samples and randomised controlled trials.

## **BEHAVIOURAL AND PSYCHOLOGICAL SYMPTOMS OF DEMENTIA (BPSD)**

Every person needs to be regularly assessed for challenging behaviours or behaviours of concern.<sup>72</sup> BPSD is important as it occurs in 90% of people with dementia at some stage of their illness. It is associated with increased mortality, complications, institutionalisation, faster rate of cognitive decline and distress to the person as well as caregiver.<sup>73</sup>

BPSD refer to a spectrum of symptoms that include: physically aggressive behaviours (hitting, kicking, biting), physically non-aggressive behaviours (pacing, inappropriate touching), verbally non-aggressive agitation (repetitive phrases or requests, calling out) and verbally aggressive behaviours (cursing, screaming).<sup>74</sup> Psychological symptoms include depression, anxiety, hallucinations or delusions. Managing these symptoms can be extremely challenging, both at home and particularly in a hospital environment. Best practice guidelines recommend non-pharmacological interventions as the first approach, excluding delirium, addressing nutrition, constipation, sleep and sensory deficits.<sup>75</sup> Treatment of pain is also important.<sup>76</sup>

Reassurance, redirection distraction, single rooms and avoidance of inappropriate external cues (e.g. over-stimulating environment) may benefit. Therapies such as massage, animal-assisted therapy, music, aromatherapy and activity therapy has shown some benefit.<sup>77</sup>

Identifying triggers and aiming to meet unmet needs utilising psychosocial interventions, is often sufficient to improve BPSD symptoms.

Evidence for the benefit of use of psychotropic medications is small and outweighed by significant adverse effect profile that includes sedation, parkinsonism, gait disturbance, falls, chest infection, dehydration, cognitive decline, stroke and up to 60% greater risk of mortality.<sup>78</sup>

Judicious use of atypical antipsychotics may be required for targeted symptoms, such as psychosis and agitation in a small proportion, and low doses for 6–12 weeks with regular monitoring are required to document efficacy. This should be started by people with expertise in this area, and regular withdrawal of medication trialled.<sup>78</sup>

## **TERMINAL/END-STAGE DEMENTIA**

Dementia is characterised by high levels of disability throughout the last year of life, and there is increasing recognition of advanced dementia as a terminal illness requiring a

palliative approach.<sup>79</sup> End-of-life issues have implications for clinical prognosis, education and planning for families and carers.

## **CAPACITY**

Capacity assessments<sup>80</sup> may be required over the course of the illness where concerns have been raised regarding tasks that require specific cognitive abilities, such as driving, making a will, an enduring power of attorney, occupational safety etc. Lack of capacity in one area does not imply global incapacity.

## **DRIVING**

The ability to drive in someone with dementia needs to be considered at an early stage of diagnosis by all health professionals involved in ongoing management. A diagnosis of dementia does not preclude driving, but clinicians need to be aware of regional differences in legislative requirements. An on road physical driving assessment is often required to evaluate one's performance and safety, generally performed by an occupational therapist. Giving up driving can be difficult for people especially for those who feel they are giving up their independence.<sup>81</sup> Discussion around alternative travelling arrangements such as taxi cards, public transport, and community bus services should be encouraged.

## **DEMENTIA IN DIFFERENT SETTINGS**

There needs to be a shared care model for people with dementia and their carers' in supporting their needs in different settings (i.e. *cultural*, community, acute hospital setting and residential care).

Australia and New Zealand have led the way with regard to dementia policy, starting back in 2005. The 2012 Aged Care reforms provided funding to tackle dementia in terms of diagnosis and better care of people with dementia in hospitals, that includes implementation of national standards on safety and quality.<sup>8,82, 83</sup>

An integrated approach to delivery of services and care is required that is developed in partnership with the person with dementia and their carer and families and takes into account the changing needs of the person.

## CONCLUSION

Dementia is a condition that is common in older people and frequently contributed to by multiple pathologies and comorbidities, including delirium, depression and polypharmacy. The diagnosis of dementia relies greatly on clinical assessment that includes collaborative history and exclusion of contributing conditions. However, emerging technologies, including the development of biomarkers and novel neuroimaging techniques may supplement clinical assessment in the near future. Although pharmacological therapies have been largely unsuccessful in treating dementia, targeting potential risk factors aiming to decrease incidence of dementia is an important public health initiative.

## USEFUL LINKS

[www.dbmas.org.au](http://www.dbmas.org.au)

[www.alzheimers.org.au](http://www.alzheimers.org.au)

[www.alzheimers.org.nz](http://www.alzheimers.org.nz)

[www.carerslink.com.au](http://www.carerslink.com.au)

[www.myagedcare.gov.au](http://www.myagedcare.gov.au)

[www.fightdementia.org.au](http://www.fightdementia.org.au)

[www.safetyandquality.gov.au/publications/a-better-way-to-care-actions-for-clinicians](http://www.safetyandquality.gov.au/publications/a-better-way-to-care-actions-for-clinicians)  
[www.health.govt.nz/system/files/documents/publications/new-zealand-framework-for-dementia-care-nov13.pdf](http://www.health.govt.nz/system/files/documents/publications/new-zealand-framework-for-dementia-care-nov13.pdf)

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