ACE CLINICAL GUIDELINE

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Management of mild and moderate dementia in older adults

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Objective

To enhance management of dementia, with the goal of maintaining patients' quality of life

Scope

Assessment and management of diagnosed mild or moderate Alzheimer's disease, vascular dementia, and mixed dementia (Alzheimer's disease and vascular dementia) in older adults aged 65 and over

Target audience

This clinical guideline is relevant to all healthcare professionals caring for patients with dementia, especially those in primary or generalist care

Dementia is a complex neurological condition that significantly impacts quality of life and daily functioning, with risk increasing substantially with age. In Singapore, where 1 in 4 people will be aged 65 years and above by 2030, its impact on the healthcare system and society is expected to grow.[1,2] Dementia is characterised by cognitive decline, loss of independence, and behavioural and psychological symptoms, leading to significant emotional, social, and economic challenges for patients, families, and carers.[3]

The progression of dementia is highly individualised, necessitating personalised care approaches. Aligning with the broader National Dementia Strategy, this ACE Clinical Guideline (ACG) aims to support healthcare professionals in navigating the complex landscape of dementia management. The ACG focuses on assessing and managing the cognitive, functional, and behavioural symptoms associated with dementia, with the goal of optimising quality of life for patients and carers and supporting ageing in place.

Statement of Intent

This ACE Clinical Guideline (ACG) provides concise, evidence-based recommendations and serves as a common starting point nationally for clinical decision-making. It is underpinned by a wide array of considerations contextualised to Singapore, based on best available evidence at the time of development. The ACG is not exhaustive of the subject matter and does not replace clinical judgement. The recommendations in the ACG are not mandatory, and the responsibility for making decisions appropriate to the circumstances of the individual patient remains at all times with the healthcare professional.

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Dementia management requires a comprehensive, goal-directed approach that evolves with disease progression, focusing on:

- Managing cognitive and functional decline to delay symptom progression
- Managing behavioural and psychological symptoms of dementia (BPSD) to support ageing in place, optimise patient quality of life, and reduce carer stress

This ACG supports healthcare professionals in managing mild or moderate dementia in patients aged 65 years and over, focusing on the three most common dementia subtypes: Alzheimer's disease, vascular dementia, and mixed dementia (Alzheimer's disease and vascular dementia).

Figure 1 (page 3) provides an overview of dementia management in primary care. Cognition, function, and behaviour are core domains affected by disease progression, informing management strategies and guiding shared decision-making.

A multidisciplinary approach is necessary for managing dementia. However, this is beyond the scope of this ACG. Broader support services and resources may be found in **Annex F**.

Primary care clinicians are encouraged to seek specialist input as required. Referral considerations may include complex behavioural symptoms, medication optimisation challenges, or when additional expertise would benefit patient care.

Assessment to inform management approach

Recommendation 1 **Assess cognition, function, and behaviour to prioritise treatment.**

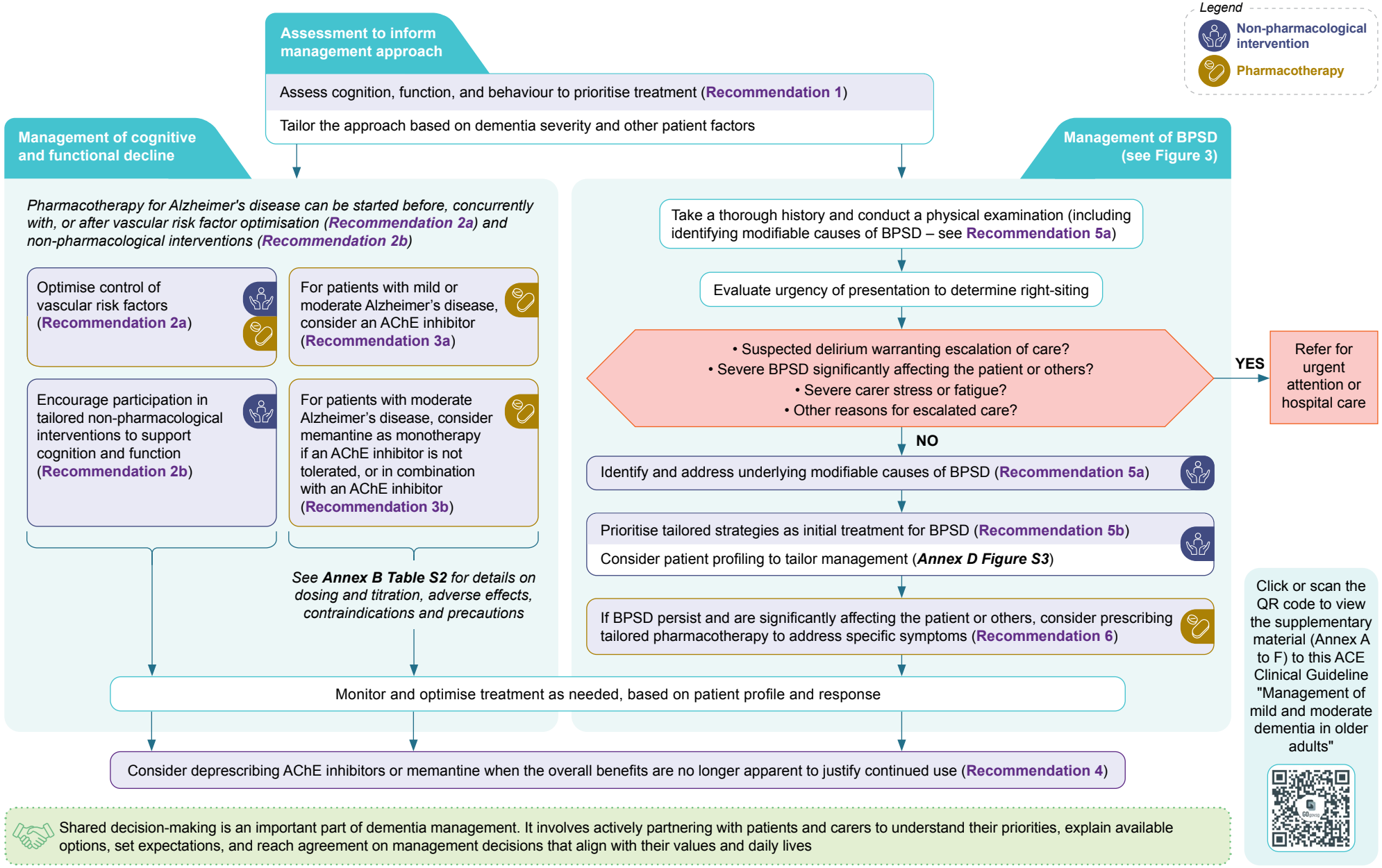
The rate of cognitive and functional decline varies widely among individuals due to differences in disease progression, age of onset, underlying aetiology, and overall health (e.g. comorbidities, frailty, polypharmacy).[4] Similarly, behavioural changes manifest differently, typically with increasing frequency and severity as the disease progresses.[5] These variabilities highlight the need for personalised and adaptive care plans. **Table 1** provides examples of the cognitive, functional, and behavioural changes typically observed at each stage of dementia.

Table 1. Examples of changes in cognition, function, and behaviour across mild, moderate, and severe dementia[a]

	Mild dementia	Moderate dementia	Severe dementia
Cognitive changes	• Difficulties with remembering personal history	• Increasing lack of orientation to time, date, or place	• Loss of verbal ability
Functional changes	• Increasing difficulty with complex tasks or instrumental activities of daily living (ADLs) (e.g. preparing meals, paying bills)	• Requires assistance with some basic ADLs (e.g. dressing, bathing, toileting) • Increasing urinary and faecal incontinence	• Requires assistance with basic ADLs (e.g. moving, eating, swallowing) • Urinary and faecal incontinence • Often bedbound
Behavioural changes	• Changes in mood or emotion (e.g. increasingly irritable, frustrated at lost abilities, easily upset, more anxious, frightened, or sad, more withdrawn, lack self-confidence, lose interest in hobbies or people)	• Changes in personality and behaviour (e.g. apathy, depression, anxiety, psychosis, agitation, obsessive behaviour, being repetitive, sleep disturbances, loss of inhibition, sundowning)	• Significant personality and behaviour changes

[a] Clinical changes mentioned in this table were sourced from the Functional Assessment Staging Tool and Global Deterioration Scale.[6,7]

Figure 1. Overview of mild and moderate Alzheimer's disease[b] management in primary care



AChE inhibitor, acetylcholinesterase inhibitor; BPSD, behavioural and psychological symptoms of dementia.

[b] The principles in this flowchart also apply to the management of vascular dementia and mixed dementia (refer to section on off-label use of AChE inhibitors and memantine under **Recommendation 3**).

Assessment of cognition, function, and behaviour for staging of dementia

Assessment of the three core domains (cognition, function, and behaviour) informs staging of the disease. This helps identify and guide priorities for management, as improvements in one or more domains can significantly improve quality of life.[8,9]

These assessments can be conducted using patient or carer input (e.g. observations, verbal reports, self-assessment) or standardised scales (e.g. Clinical Dementia Rating,[10] Functional Assessment Staging Tool,[6] or Global Deterioration Scale[7]). Using a consistent assessment approach allows the detection of changes in the three core domains over time.

Assessment beyond cognition, function, and behaviour

In addition to the core domains, regularly assess concomitant chronic conditions, diet, physical health status, and carer stress through routine tests, examinations, and patient or carer inputs as part of holistic management.

Review medications regularly, focusing on medications with anticholinergic properties that may impair cognitive function (and potentially trigger delirium). Systematic identification and appropriate deprescribing of these medications can help optimise cognitive function (see **Annex A** for guidance on managing anticholinergic burden).

Shared decision-making: a partnership approach to management

Shared decision-making empowers patients and carers to actively participate in care decisions while respecting their values, preferences, and daily routines. This collaborative approach can also reduce uncertainty, promote adherence, and facilitate continuity of care.

Engage the patient and their carer to:

- Understand what matters most in their daily lives
- Explain management options and their practical aspects in clear, relatable terms
- Clarify expectations before starting treatment
- Agree on management options and when to proceed

In addition, encourage patients and carers to discuss planning for future care early, ideally while the patient is still able to actively participate in such conversations. This includes discussing medicolegal aspects such as Advance Care Planning and Lasting Power of Attorney. Links to resources can be found in **Annex F**.

Management of cognitive and functional decline

Recommendation 2

a) Optimise control of vascular risk factors.

b) Encourage participation in tailored non-pharmacological interventions to support cognition and function.

Management of cognitive and functional decline involves a multifaceted approach, with pharmacotherapy (see **Recommendation 3**), optimisation of vascular health, and non-pharmacological interventions all playing a role.

- **Optimise vascular health** by actively managing risk factors through appropriate medication, lifestyle modifications, and regular monitoring (e.g. optimising management of hypertension, diabetes and dyslipidaemia; smoking cessation; minimising alcohol intake).[11–13]
- **Implement targeted non-pharmacological interventions** (**Table 2**) which promote cognition or function.[14,15] Using a combination of interventions is more beneficial than a single intervention.[16–19] Where multiple interventions are used, consider rotating them within a structured weekly programme to provide consistent engagement.[16–19]

Also, encourage patients to remain socially engaged within supportive, age-friendly community environments. This includes participating in community activities, maintaining social connections, and addressing practical barriers (e.g. hearing and vision loss) that may limit engagement.[20–22] Establishing consistent daily routines and providing memory aids (such as visual calendars, medication organisers, and keeping important information visible) are practical approaches that help orientate patients to their daily routine and surroundings, supporting independence as cognitive decline progresses.

Vascular dementia and management of modifiable vascular risk factors

Vascular dementia is aetiologically different from neurodegenerative Alzheimer's disease, as it is primarily caused by cerebrovascular disease.

To limit further cerebrovascular damage and disease progression in vascular dementia, mainstay treatment optimises vascular health through aggressive risk factor modification, including hypertension, diabetes mellitus, dyslipidaemia, smoking cessation, and atherosclerosis, as part of appropriate secondary stroke prevention measures.

Table 2. Examples of non-pharmacological interventions[c] which support cognition and function in patients with mild or moderate dementia

Non-pharmacological intervention	Implementation examples from the published literature and associated benefits
Cognitive stimulation therapy (CST)	Group sessions (typically 45 minutes, twice weekly, over 7 weeks) involving themed activities (such as discussion of current events, word games, number and money tasks, creative crafts, food-related activities, and reminiscence), delivered by trained staff or carers to a small group.[19,23,24] CST can also be provided on a one-to-one basis by trained carers, including at home.[25] Reported benefits: Improvements in global cognition and ability to manage complex daily tasks (instrumental ADL). Cognitive benefits are maintained with continued delivery.[25]
Reminiscence therapy (RT)	Guided recall of personal memories using photographs, music, familiar objects, or videos. Can be delivered individually or in groups.[26] Reported benefits: Improvements in global cognition and memory, with benefits sustained beyond the end of the intervention.[26]
Physical exercise	Moderate-intensity aerobic exercise (e.g. brisk walking, cycling) for 30–50 minutes, 3 or more times per week, sustained over at least 12–16 weeks (longer programmes show greater benefit).[27–29] Resistance training (e.g. weights, resistance bands) and Tai Chi are also supported by evidence.[27–29] Programmes should be tailored to the patient's physical capacity and delivered under appropriate supervision. Reported benefits: Improvements in global cognition (particularly in Alzheimer's disease); possible improvements in daily functioning (ADL).[27–29]
Cognitive rehabilitation	Personalised, one-to-one home-based sessions (typically 8–14 sessions over 3 months) with a trained therapist (occupational therapist or nurse). Specific everyday activities are identified for improvement; the therapist uses techniques such as spaced retrieval, memory aids, and environmental modifications to support goal achievement. Family members or carers are often involved in the sessions.[30] Reported benefits: Improvements in performance on patient-identified daily activity goals (goal attainment).[30] Benefits are specific to practised goals; evidence does not support generalised functional improvement.

[c] These interventions were selected based on reported benefits for cognition or function in the published literature and at least moderate certainty of evidence (Grading of Recommendations, Assessment, Development, and Evaluation, GRADE). See the Evidence-to-Recommendation Framework for the full evidence base and rationale.

Recommendation 3

a) For patients with mild or moderate Alzheimer's disease, consider an AChE inhibitor.

b) For patients with moderate Alzheimer's disease, consider memantine as monotherapy if an AChE inhibitor is not tolerated, or in combination with an AChE inhibitor.

There is limited evidence to guide when pharmacotherapy should be started in relation to optimising vascular health and implementing non-pharmacological interventions (before, concurrently with, after, or instead of). There are benefits to implementing these concurrently (see page 7, **Benefits of combining pharmacotherapy with non-pharmacological management**).[31]

Cognitive enhancers, i.e. acetylcholinesterase inhibitors and memantine, are the main pharmacotherapy options for managing symptoms of Alzheimer's disease. The role of other complementary medications and emerging therapies is not yet established (see **Annex B Table S4**).

AChE inhibitors in mild or moderate Alzheimer's disease

In mild Alzheimer's disease, AChE inhibitors (donepezil, galantamine, and rivastigmine) remain the preferred therapy over memantine for cognitive symptoms, with treated patients experiencing slower cognitive decline than untreated patients.[32,33] The three medications offer similar efficacy in supporting cognition, so choose based on access, cost, dosing frequency, route of administration, and adverse effects. Their benefits beyond cognition are limited: functional gains are small, and behavioural improvements are very modest and not clinically significant.[34] Long-term observational studies suggest extended use may lead to better cognitive outcomes and increased survival over time.[34–36]

In moderate Alzheimer's disease, continued use of AChE inhibitors provides symptomatic benefits for cognition and reduced mortality.[35,36]

Memantine in moderate Alzheimer's disease

In moderate (and severe) Alzheimer's disease, memantine improves cognition, function, behaviour, and survival rates.[32,37] It may be considered for worsening cognition, function, or behavioural and psychological symptoms. Currently, there is very limited evidence for its use in mild Alzheimer's disease, and it is only approved for moderate (and severe) Alzheimer's disease.[37]

Concurrent memantine and AChE inhibitor therapy has been reported to provide modest improvements in cognition, function, behaviour, and survival rates in moderate Alzheimer's disease without significantly increasing adverse events.[38,39]

Practical considerations when prescribing cognitive enhancers

Donepezil, rivastigmine, galantamine, and memantine are registered for use in Singapore, but galantamine is not commonly used and not available in some practice settings. **Annex B Table S2** provides details on dosing and titration schedules, adverse effects, and precautions for cognitive enhancers in Alzheimer's disease. Some practical considerations for prescribing cognitive enhancers are listed below.

Before prescribing	• Exercise caution in patients with cardiac conditions. In particular, AChE inhibitors may cause bradycardia due to vagotonic effects; consider ECG prior to prescribing. • For memantine, also exercise caution for patients with seizure disorders.
When prescribing	• Start at a low dose to reduce adverse effects (particularly gastrointestinal adverse effects for AChE inhibitors, and dizziness for memantine) and to facilitate adherence. • To minimise gastrointestinal adverse effects, administer oral rivastigmine or galantamine with food.
After initial prescribing	• Tailor titration rate to maintenance dose based on patient tolerance (refer to product information leaflets for details). For patients unable to tolerate maintenance doses of AChE inhibitors, lower doses may still provide benefit, albeit more modest.[40] While similar analyses for memantine could not be located, lower doses may still confer some benefit for patients unable to tolerate the standard daily dose. • For galantamine, monitor for skin reactions; discontinue if rash appears. • Assess response typically within three months; more frequent reviews may be clinically indicated.[41] • In patients with suboptimal response to an AChE inhibitor, consider adjusting the treatment approach (**Annex C Figure S2**), guided by careful assessment of cognition, function, medication suitability, and patient preferences.

Benefits of combining pharmacotherapy with non-pharmacological management

Evidence on combining pharmacotherapy with non-pharmacological therapy is currently limited to cognitive stimulation therapy (CST) combined with an AChE inhibitor.[19] However, this offers greater benefits than either option alone, suggesting that early implementation may be particularly beneficial.[19,33]

Clarifying expectations before starting a cognitive enhancer

Help the patient and carer to understand what the medication can and cannot do:

- Existing cognitive enhancers help manage the symptoms and do not provide a cure; they may provide modest improvement or 'stabilise' cognition, function, or both, but **do not prevent disease progression – cognitive decline will continue even with treatment**.
- Benefits and response can vary among individuals. A common pattern of response to AChE inhibitor treatment is initial improvement in cognition, followed by maintenance of cognitive gains above baseline for up to 1 year.
- The treatment is not life-long, and deprescribing may be appropriate after a while.

Discuss how the medication may affect the patient and other considerations:

- Benefits and potential harms, common adverse effects, and need for carers to monitor for evidence of benefits or adverse effects (**Annex B**)
- Expected titration schedule (**Annex B**)
- Importance of carer involvement in managing medication dosing to ensure patient adherence especially when new medications are started
- When to return for review and follow-up

Off-label use of cognitive enhancers in vascular dementia and mixed dementia

Due to the limited treatment options available for vascular dementia, the use of cognitive enhancers may be clinically appropriate in two scenarios:

- Patients with **mixed dementia (Alzheimer's disease and vascular dementia)**
- Patients in whom **Alzheimer's disease cannot be definitively excluded**

Evidence indicates modest cognitive benefits for donepezil and rivastigmine in vascular dementia.[42] Both medications have dose-dependent adverse effects, including nausea and diarrhoea. Memantine offers modest benefits in cognition and behaviour with a more favourable safety profile.

Before prescribing for an off-label indication:

- Discuss with the patient and carer the off-label nature of the prescription, expected benefits, limitations, potential risks, adverse effects, and alternative treatment options
- Obtain their consent
- Document the rationale for the off-label use and establish a monitoring plan (see section on monitoring and follow-up).

Recommendation 4 **Consider deprescribing AChE inhibitors or memantine when the overall benefits are no longer apparent to justify continued use.**

Deprescribing cognitive enhancers

The goals of cognitive enhancers are to manage progression of symptoms and support existing cognition and function in patients with dementia. At different time points, there may be different reasons to consider stopping these cognitive enhancers (see **Table 3**). **Figure 2** provides practical advice for deprescribing these medications.

Table 3. Reasons to consider deprescribing cognitive enhancers[43–45]

Any time after starting cognitive enhancers	When cognitive enhancers have been used for 12 months or more
• Decision by patient, family, or carer to discontinue treatment • Intolerable adverse effects • Unable to adhere to treatment • Drug-drug or drug-disease interactions that make continued treatment risky • Terminal illness	• Clinical deterioration over the past 6 months (excluding other medical causes) ▸ Sustained decline in cognition, function, or behaviour at a greater rate than previous • No benefit observed during treatment ▸ No improvement, stabilisation, or decreased rate of decline • Severe or end-stage dementia ▸ Dependence for most ADLs, inability to respond to environment, or limited life expectancy

Deprescribing: what is important to the patient and carer?

Consider the pros and cons of deprescribing an AChE inhibitor or memantine with the patient and their carers, based on what is important to them (e.g. ability to socially interact, recognise family, perform ADLs).

Examples of questions to discuss:

- What do they value most (e.g. preserving cognition, enjoying quality of life, remaining independent)?
- Has there been any improvement while on treatment?
- Are they experiencing any adverse effects?

AChE inhibitor or memantine in patients with BPSD[46]

- Delay deprescribing cognitive enhancers until BPSD have stabilised.
- If BPSD worsen after starting a cognitive enhancer, discontinue the medication.
- If BPSD worsen after increasing the dose of a cognitive enhancer, revert to previous dose.

Figure 2. Practical advice for deprescribing cognitive enhancers[43,44]

Reduce the dose slowly

- Halve the dose or step down through the available dose formulations to the lowest dose.
- Other alternatives:
  - Consider reducing the dose if the patient or carer is concerned about complete discontinuation (and reassess possible cessation after a suitable period)
  - Stop the medication immediately if clinically warranted (e.g. if the patient is experiencing severe adverse effects)

Involve the patient and carer

- Educate the patient and carer on what to expect and what to do if they have concerns

Monitor the patient regularly

- Review patient progress (e.g. every four weeks) and:
  - Check for withdrawal symptoms in the first week (e.g. severe agitation, hallucinations, or reduced consciousness)
  - Consider how cognition, function, behaviour, and carer burden have changed over the follow-up period
- Monitor the effect of deprescribing on other medications
- Restart the medication at the previous minimum effective dose if the patient experiences a noticeable decline (e.g. adverse medication withdrawal event, reversal of medication effect, or progression in condition) after stopping

Management of behavioural and psychological symptoms of dementia

Behavioural and psychological symptoms of dementia (BPSD) refer to neuropsychiatric symptoms that accompany dementia;[47] these symptoms are often a patient's non-verbal expression of unmet needs.[47,48] Arising from psychological, social, and biological factors,[49] BPSD occur in most patients across all stages of dementia, with symptoms typically worsening with disease progression.[5] BPSD often cause more distress to patients and carers than cognitive decline itself.[50] **Table 4** lists common BPSD and their typical presentation(s).

Table 4. Common presentations of frequently encountered BPSD[51]

Behaviour	Presentation
Agitation or aggression	Verbal (e.g. complaining, moaning, angry statements, threats) or physical (e.g. resisting carers, restlessness, spitting, hitting out)
Apathy	Lack of initiative, motivation and drive, aimlessness, and reduced emotional response. Reduced motivation can be a feature of depression, but apathy can be distinguished from depression by the absence of sadness and other signs of psychological distress
Anxiety	Restlessness, pacing, repetitive movements like hand-wringing or fidgeting, repeatedly looking for items or asking the same questions, or becoming clingy and following carers around constantly
Depression	Sadness, tearfulness, pessimistic thoughts, withdrawal, inactivity, or fatigue
Disinhibited behaviour	Inappropriate sexual, verbal or physical behaviour ordinarily considered 'rude'. May be exacerbated by impaired judgement, reduced awareness of environment, or lack of understanding of effect on others
Psychosis (hallucinations and delusions)	Delusions are usually reflective of the underlying memory loss or changes in perception (e.g. accusation of theft of personal items, infidelity of a spouse, or that family members are imposters). Vivid visual hallucinations are common; auditory hallucinations are less common
Sleep disturbance	May occur secondary to depression, anxiety, agitation, or pain and may cause other BPSD to be exacerbated at night (e.g. wandering)
Vocally disruptive behaviour	Usually considered collectively with agitation and poorly understood as an individual presentation of BPSD
Wandering	Sometimes related to agitation. Wandering may be circular, between two points, random, or direct to a location without diversion. May be challenging due to safety concerns
Sundowning	Not BPSD; rather it is the emergence or worsening of BPSD in the late afternoon or early evening

Adapted with permission from Best Practice Advocacy Centre New Zealand (bpac^{nz}). Managing the behavioural and psychological symptoms of dementia. April, 2020. Available from: bpac.org.nz/2020/bpsd.aspx.

Recommendation 5

a) Identify and address underlying modifiable causes of BPSD.
b) Prioritise tailored strategies as initial treatment for BPSD.

BPSD management in primary care focuses on identifying modifiable causes and implementing strategies, supported by a thorough history and physical examination to evaluate the urgency and guide the appropriate care pathway for the patient (see **Figure 3**).

Annex E Table S5 provides examples of strategies to address frequently encountered BPSD.

Figure 3. Assessing and managing BPSD presentations

Take a thorough history and conduct a physical examination

Document the patient's symptoms, impact on daily functioning, and safety concerns (e.g. falls)

- Note the onset and course of symptoms[d] and establish a baseline; carers can provide helpful information and insight (see **Annex E**)
- Cross-reference current assessment with medical records or previous assessments
- Consider validated scales (e.g. Neuropsychiatric Inventory) to measure symptom severity and frequency, and to monitor intervention effectiveness, especially in patients with multiple symptoms

Identify reversible exacerbating causes

- Medical causes, including
  - Pain or discomfort (e.g. joint pain, dental problems, discomfort from skin problems)
  - Chronic conditions (e.g. cardiovascular disease)
  - Delirium
  - Medications[e]
- Other contributing causes, including
  - Psychological (e.g. fear, confusion, frustration, communication challenges)
  - Environmental (e.g. issues with lighting, noise, or stimulation; change in surroundings or daily routine)

Conduct a physical examination to identify factors that may contribute to worsening BPSD

Physical findings (e.g. fever, hypoxia, abdominal tenderness, palpable faecal masses, retention of urine, fluid overload, inflammation, or localised neurological deficits) may point to an underlying condition causing the delirium (see below)

Laboratory tests or imaging are generally **not** necessary for gradually worsening BPSD, unless history, physical examination, or both suggest alternative causes

Evaluate urgency of presentation to determine right-siting

Is delirium suspected? Does it warrant escalation of care?

Refer for urgent attention if delirium is suspected and cannot be managed in primary care

Delirium presents as an acute disturbance in attention and cognition, representing a change from the patient's baseline that often fluctuates over the course of the day, with **hypoactive** delirium (sedate or withdrawn) more common but frequently overlooked compared to **hyperactive** delirium (over vigilant, agitated, often wandering).[53]

Possible causes of delirium in patients with dementia include:[53–55]

- Infections (e.g. urinary tract or respiratory)
- Cardiac events (acute myocardial infarction, heart failure)
- Stroke
- Fluid overload (from cardiac, renal and hepatic causes etc)
- Dehydration, constipation, urinary retention
- Hypo- or hyperglycaemia
- Polypharmacy (drug-drug interactions), adverse medication effects, sudden withdrawal of medication
- Cessation of regular alcohol intake
- Recent surgery or post-operative complications

Identify and address readily reversible causes where possible. If there is no improvement, the cause is unclear or possibly serious, or the behaviour is very challenging, refer the patient to the Emergency Department.

Are there other reasons warranting escalation of care?

Consider referring for hospital care

Hospitalisation may be warranted for patients with severe BPSD, for example if:

- The patient is endangering themselves or others
- The patient refuses to eat and is nutritionally compromised
- The patient refuses care
- The patient requires further comprehensive or urgent evaluation to identify and address underlying medical causes
- The carer is experiencing significant distress

None of the above?

Continue management in primary care

- **Address reversible exacerbating causes of BPSD**
- **Implement strategies (Annex E)**: Select and tailor strategies based on the patient's presentation, personal history, interests and capabilities, and patient and carer preferences. Consider tools (e.g. the Sunflower Tool in **Annex D**) to assist with selecting and tailoring strategies
- When BPSD persist despite these primary interventions and significantly affect the patient or others, pharmacotherapy may be required (see **Recommendation 6**)

Caring for the carers

Regularly check in on how carers are coping and share practical strategies to manage BPSD at home (**Annex E**). Where feasible, direct carers to resources for training and support, connection with other carers, and respite services (**Annex F**). Consider discussing the need to engage further assistance (e.g. live-in helper) to alleviate burden and prevent burnout.

[d] Nature and severity, frequency and timing, pattern and progression.

[e] Particularly medications known to contribute to BPSD: anticholinergic medications (e.g. amitriptyline, oxybutynin) (refer to **Annex A** on how to deprescribe); antipsychotics; antiepileptic medications (e.g. valproate, carbamazepine, phenytoin); high-dose corticosteroids; sedatives (e.g. benzodiazepines, non-benzodiazepine hypnotics such as zopiclone); anti-Parkinsonian medications. Note that in some patients, AChE inhibitors have been associated with neuropsychiatric events such as agitation, anxiety, confusion and disorientation, psychosis (hallucinations), and sleep disturbance.[52]

Recommendation 6 **If BPSD persist and are significantly affecting the patient or others, consider prescribing tailored pharmacotherapy to address specific symptoms.**

Non-pharmacological interventions (Annex E Table S5) are the foundation of BPSD management and should be used whenever possible. As the disease progresses, persistent BPSD – defined as symptoms that remain despite optimal management of underlying causes and tailored behavioural strategies[56] – may require pharmacotherapy when they cause significant distress, pose safety risks, or affect quality of life.[47] Earlier pharmacotherapy use may also be appropriate based on the patient's presentation and clinical judgement.

Selecting pharmacotherapy for BPSD

Pharmacotherapy for BPSD should target specific symptoms, as **not all BPSD will respond to medication**. For example, symptoms such as apathy, anxiety, disinhibited behaviour, psychosis, and wandering may be better managed through environmental modifications and behavioural strategies (**Annex E Table S5**) rather than pharmacotherapy.[57,58]

Multiple medications are used to manage BPSD, but benefits may not always outweigh the risks. While some medications have approved indications relating to dementia and BPSD, many treatments are used off-label in practice. This ACG focuses on medications commonly initiated and monitored in general practice.

Before starting pharmacotherapy for BPSD

Carefully discuss the potential benefits and risks with the patient and carer. Follow a measured approach with low initial doses, slow titration, clear treatment goals, and a stipulated timeline for review and follow-up. **Tailor the choice and sequence based on the individual presentation, considering factors such as urgency and severity of the symptoms, and expected time for therapeutic effects to be observed.**[f]

[f] Onset of therapeutic effects varies by medication class and individual patient response. The time to onset of effect listed here is based on approved indications: cognitive enhancers – within months; antidepressants – within weeks; antipsychotics and antiepileptics – within hours to weeks.[59–63]

Cognitive enhancers for BPSD

In practice, many patients presenting with BPSD may already be taking an AChE inhibitor or memantine for cognitive reasons (see **Recommendation 3**).[34,37]

AChE inhibitors are reported to have mixed efficacy for reducing overall BPSD, and memantine may provide modest benefits for agitation, aggression, and psychosis.[64,65]

Overall, if cognitive enhancers are not exacerbating BPSD symptoms (see note below), they may be continued primarily for their cognitive benefits rather than specifically for BPSD.[66,67]

Note: In some patients, AChE inhibitors have been associated with neuropsychiatric events (such as anorexia, agitation, anxiety, confusion and disorientation, depression, psychosis, and sleep disturbances).[52,68]

Second-generation antidepressants[g] for BPSD (off-label for dementia)

While evidence of efficacy varies between medications, the favourable safety profile of second-generation antidepressants in elderly patients, and lower anticholinergic burden (compared to antipsychotics) support their use in primary care settings.

Antidepressants do not demonstrate consistent class-wide effects for BPSD, with individual antidepressants reporting mixed efficacy for reducing agitation,[69–71] and poor efficacy for treating depression and anxiety in dementia.[72]

In clinical practice, selection often exploits known adverse effect profiles rather than primary antidepressant mechanisms – such as sedating properties for sleep disturbances or appetite-stimulating effects for loss of appetite, though evidence for these targeted applications remains limited.[73]

Annex B Table S3 provides guidance on selecting commonly used antidepressants for managing BPSD in primary care.

(If a patient with dementia also has a diagnosis of major depressive disorder, refer to the ACG on major depressive disorder for further information).

[g] Second-generation antidepressants include selective serotonin reuptake inhibitors, noradrenergic and specific serotonergic antidepressants, or multimodal serotonergic antidepressants; exclude tricyclic antidepressants and monoamine oxidase inhibitors.

Antipsychotics and antiepileptics for BPSD

Beyond cognitive enhancers and antidepressants, medications for BPSD are typically initiated in specialist care settings due to their potential adverse effects and need for close monitoring.[74,75]

Prescriptions should be for the **lowest** dose that is clinically effective, and for the **shortest** amount of time.

- **Antipsychotics** (e.g. risperidone – indicated for persistent aggression in moderate and severe Alzheimer's disease, brexpiprazole – persistent agitation associated with Alzheimer's dementia; aripiprazole, olanzapine, quetiapine – off-label for dementia) may be used for moderate or severe agitation (risk of harm to self or others), or aggression.[76]
- **Antiepileptics** (e.g. carbamazepine – off-label for dementia) have been reported to have uncertain efficacy in reducing agitation but may be of benefit.[76,77] There is no strong evidence that valproate is effective in reducing agitation, and it is associated with increased risk of adverse effects.[76,78] Hence, it should only be considered if significant agitation continues despite the use of safer, evidence-based pharmacotherapy.

Monitoring and follow-up

Monitor patients regularly to evaluate treatment effectiveness, ensure safety, and optimise outcomes across cognitive, functional, and behavioural aspects.

- For non-pharmacological interventions, follow-up allows evaluation of engagement and effectiveness of the chosen strategies.
- For pharmacotherapy, monitoring helps to assess medication benefits, adjust medication dosing and detect potential adverse effects.

Monitoring patients with concomitant common chronic conditions

Closer monitoring is advised for patients with dementia who have concomitant chronic conditions (e.g. diabetes mellitus, hypertension, or dyslipidaemia) as observational data suggest potential risks such as excessive blood pressure lowering and cardiovascular adverse effects.[79,80]

Patient reviews

Regular assessment of cognition, function, and behaviour (**Recommendations 1** and **5**) should be tailored to patient needs. As an example, review the patient once every 4 to 8 weeks during medication titration or changes, extending to once every 3 to 6 months when stable, with a minimum of two visits a year. Closer monitoring may be clinically indicated for patients with high cardiovascular risk, complex medical conditions, unstable symptoms, or significant carer stress.

Navigating care transitions and referrals

The moderate stage of dementia represents a critical transition period where patients may start to experience significant cognitive decline and require substantial assistance with ADLs. **Healthcare professionals in primary care may continue to manage the patient's care if it remains feasible, and they are confident and comfortable to do so.** Consider specialist referral based on:

- Symptom severity and complexity
- Risk level to patient and others
- Response to current management strategies
- Support system stability and carer stress
- Patient and family preferences
- Practice resources and expertise (including capacity for multidisciplinary care)

References

Click or scan the QR code for the reference list to this clinical guideline

Evidence-to-Recommendation Framework

Click or scan the QR code to view the Evidence-to-Recommendation Framework for the recommendations in this clinical guideline

Google Gemini was used to portray a collaborative dementia care consultation between a healthcare professional, patient, and carer in Singapore, with plain background and clean lines to allow for the future addition of a coloured background (Google Gemini, accessed 24 November 2025).

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About the Agency

Established by the Ministry of Health, the Agency for Care Effectiveness (ACE) is Singapore's national health technology assessment (HTA) and clinical guidelines agency. We support providers, patients, and payers make better-informed decisions about patient care. We achieve this by conducting health technology assessments (HTAs), developing clinical guidelines and technology guidance, and providing education.

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