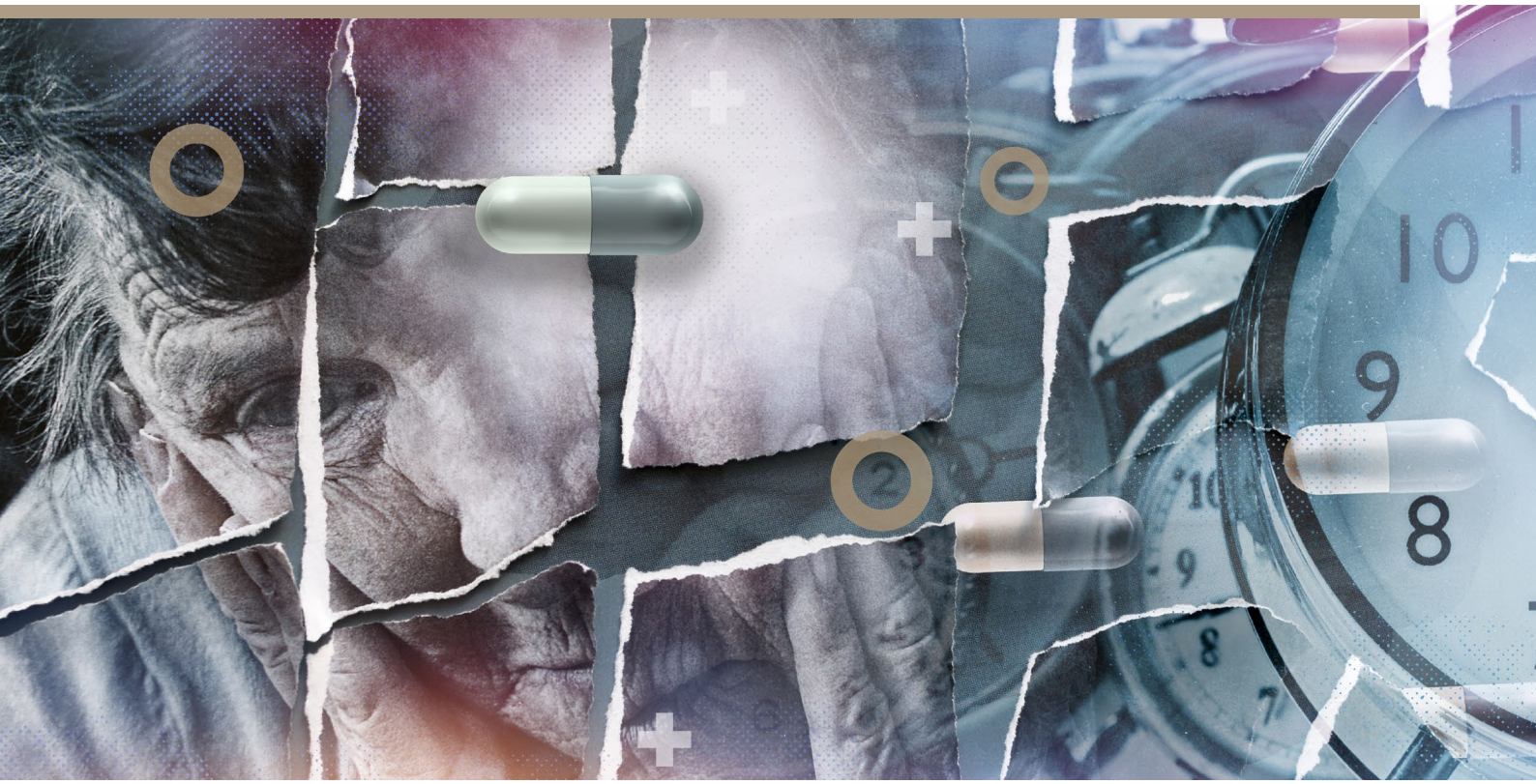


Managing the **behavioural and psychological symptoms** of dementia

Most people with dementia develop behavioural and psychological symptoms (BPSD) at some point during their journey, which can be distressing for patients, family/whānau and other carers, and challenging for health professionals to manage. In many cases, BPSD can be improved with modifications to the patient's environment and behavioural interventions. Antipsychotics are often over-used in this situation. Their use is associated with significant risks in people with dementia, and they are only effective for specific behaviours, such as psychosis and agitation.

This is the third article in a series on cognitive impairment and dementia in older people. The final article in the series will focus on palliative care for people with dementia.

KEY PRACTICE POINTS:

- Behavioural and psychological symptoms of dementia (BPSD) are often an attempt by the patient to communicate, therefore understanding why the behaviour or symptom is occurring is the key to management, e.g. are they in pain or frustrated by an aspect of their surroundings?
- Environmental and behavioural interventions should be considered first in patients with BPSD; an understanding of the patient's previous vocation, interests, abilities, social and family roles, cultural background and spirituality helps to individualise interventions.
- Antipsychotic medicines have limited evidence of benefit for BPSD and are associated with significant risk. If an antipsychotic is required, it should be appropriate for the target behaviour requiring modification, and frequent monitoring of treatment response and adverse effects should occur. Trial withdrawal of the antipsychotic within three months, except in patients with long-term psychiatric illness, e.g. bipolar disorder.
- Antipsychotics should not be used as a routine method to sedate patients with dementia who are difficult to manage or as a routine alternative to benzodiazepines

Part 1: Understanding the symptoms and trialling non-pharmacological interventions

The term "Behavioural and Psychological Symptoms of Dementia (BPSD)" refers to the spectrum of non-cognitive and non-neurological symptoms of dementia, such as agitation, aggression, psychosis, depression and apathy.[1, 2] At least 80% of people with dementia experience BPSD.[2] Depression and anxiety can be among the first symptoms of dementia, while other BPSD such as agitation and aggression more commonly occur later, especially as the person's ability to communicate and influence their environment diminishes.[3] These behaviours can be extremely stressful for the person and their family/whānau and carers, and are often a reason for people with dementia being admitted into residential care.[2] Appropriate treatment of BPSD can significantly improve the quality of life of the patient and their families.

Non-pharmacological approaches are the first-line intervention

There is concern that antipsychotics are over-prescribed to control behaviours in people with dementia that family, whānau or carers may find challenging.[4] The first approach to managing BPSD is to try to understand why the behaviour is occurring, and, where possible, resolve these underlying factors.

Do you know who I am?

Having an understanding of the patient's background can provide greater insight into the potential causes and solutions for BPSD, e.g. knowing their life story, their culture and religion/spirituality, their previous vocation, interests, routines, family/whānau role, sexuality and significant life events.[3] It is also important to understand the nature of the relationship between the person with dementia and their carer and the stresses that the condition is placing on both.[3]

Individualise interventions and monitor the patient's response

Although the evidence base for non-pharmacological treatments of BPSD is not strong, in part because person-centred treatment approaches are difficult to study, there are generally fewer risks associated with these interventions and they should always be considered first. Non-pharmacological interventions should be tailored to the individual patient and the target behaviour(s) and the response monitored.

Identify target behaviours

Managing BPSD requires a targeted approach, i.e. focus on a specific behaviour or symptom and plan individualised interventions.

Identify the target symptoms or behaviours that require modification (Table 1) and document this in the patient's notes, including the timing, frequency, pattern and severity.[5] If a patient has numerous or severe BPSD consider quantifying them using a tool so the response to interventions can be more easily assessed (see: "Quantifying BPSD with a tool"). A request for a home risk assessment may be necessary to assess any danger the patient may present to themselves or others.[3]

Assess for underlying causes and contributing factors

"Why is this behaviour occurring?"

Consider factors that may be causing or exacerbating the behaviour; Table 2 lists some common examples:

- Are the symptoms explained by another psychiatric condition such as depression or delirium?
- Is the patient taking any medicines that may be causing or contributing to the symptoms?
- Is the patient in otherwise good physical health? Is there a possibility of undetected pain (see: "Untreated pain may be a cause of BPSD"), infection, constipation or discomfort?
- Are there any factors in the patient's living environment, i.e. their home/care facility, or unmet personal needs which may be exacerbating behaviours?

Quantifying BPSD with a tool

Quantifying BPSD with a tool such as the Neuropsychiatric Inventory (NPI) questionnaire provides a baseline for monitoring the effectiveness of interventions. The NPI can be completed by a family member or carer in less than five minutes. The questions relate to behavioural changes that have occurred since the onset of dementia. The NPI provides a severity score (three point scale) and carer distress ratings (five point score) for each symptom and total severity and distress scores.[6] These relative scores are useful in determining if the patient's behaviour is improving or worsening over time, rather than providing an objective assessment of severity. The total score is not useful if the BPSD is isolated to one subtype.

The NPI questionnaire is available from: **https://download.lww.com/wolterskluwer_vitalstream_com/PermaLink/CONT/A/CONT_21_3_2015_02_26_KAUFER_2015-10_SDC2.pdf**

Table 1: Common presentations of frequently encountered BPSD and non-pharmacological management strategies[1–3]

Behaviour	Presentation	Non-pharmacological management strategy
Agitation and aggression	Occurs in approximately 60% of people with dementia. Can be verbal, e.g. complaining, moaning, angry statements, threats, or physical, e.g. resistiveness to carers, restlessness, spitting, hitting out.	May be due to underlying depression, unmet needs, boredom, discomfort, perceived threat or violation of personal space. Make environmental or management modifications to resolve these issues. Non-specific calming and positive experience interventions may be beneficial such as music or touch therapy, e.g. hand massage, a mechanical pet or a twiddle muff (sleeve or glove with attached materials, buttons, etc, for sensory stimulation).
Apathy	Estimated to occur in 55–90% of people with dementia; most frequently vascular, Lewy body and frontotemporal dementia. Presents as lack of initiative, motivation and drive, aimlessness and reduced emotional response. Reduced motivation can be a feature of depression, but a pure apathy syndrome can be distinguished from depression by the absence of sadness and other signs of psychological distress.	Reading to the person and encouraging them to ask questions, small group and individual activities, e.g. puzzles, games, sensory stories may all be helpful. Music, exercise, multi-sensory stimulation with touch, smell and sound, and spending time with pets can also be effective. The key is to provide enriched prompts and cues to overcome the apathy and generate positive behaviour.
Depression	Occurs in approximately 20% of people with dementia but is more prevalent in early stages. May present as sadness, tearfulness, pessimistic thoughts, withdrawal, inactivity or fatigue.	Recommend exercise, social connection and engaging activities. Cognitive behavioural therapy (CBT) may be helpful in early stages. Severe depression requires input from a clinician with experience in managing patients with dementia.
Anxiety	Estimated to occur in 16-35% of people with dementia. One of the most disabling BPSD. In later stage dementia this may be an exaggerated response to separation from family, a different setting or a reduced capacity to make sense of the environment.	Focus on identifying and eliminating the trigger, rather than symptom control. Maintain structure and routine and reduce the need for stressful decision-making. Assess if sensory overstimulation may be contributing. Music and CBT have the greatest amount of evidence showing benefit.
Psychotic symptoms	Approximately 25% of people with dementia will experience psychosis, causing delusion or hallucinations. In dementia, 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, rather than delusions normally associated with mania or schizophrenia. Vivid visual hallucinations are common, particularly in Lewy Body dementia, but auditory hallucinations are less common.	Often causes more distress for the carer/family than the patient. Potentially reversible causes of psychosis include sensory or vision loss, over-stimulation, delirium, initiation of a new medicine or substance misuse. Confirm that the patient's claims are not occurring, e.g. items are not being stolen. Use memory aids, e.g. photographs to cue the person to reality. Distraction can sometimes be effective.
Wandering	Sometimes related to agitation. Wandering may be circular, pacing between two points, random or direct to a location without diversion. Often one of the most challenging and problematic BPSD due to safety concerns.	Wandering can have positive effects via exercise, e.g. improving sleep, mood and general health, and may prevent the person from feeling confined. Consider how to make wandering safe; supervised walks, secured space to roam, exercise equipment, GPS watch. Try to determine if there is a purpose to the wandering, e.g. trying to return home, looking for a person, escaping a perceived threat.
Nocturnal disruption	Sleep disturbance can occur secondary to depression, anxiety, agitation or pain and may cause other BPSD to be exacerbated at night, e.g. wandering. Occurs more frequently in people with Lewy Body dementia. Sundowning, i.e. increased agitation in the late afternoon, is also common. A sleep/wake reversal can sometimes be the cause; a form of sleep phase-shift.	Assess for underlying cause, including thirst or hunger. Restrict caffeine in the evening, limit fluid intake in the hours before bed, establish a night-time routine, minimise light and noise intrusion, ensure adequate stimulating activities during daytime.
Disinhibited behaviour	Typically occurs due to reduced impulse control. May be exacerbated by impaired judgement, reduced awareness of environment or lack of understanding of effect on others. Inappropriate sexual behaviour or verbal or physical behaviour ordinarily considered "rude" can occur. Reduced privacy, lack of personal affection, absence of sexual partner, misinterpretation of assistance provided by carers, and dopaminergic medicines, e.g. to treat Parkinson's disease, delusions or hallucinations may contribute to the behaviour.	Avoid reflexive responses that may humiliate the patient. People with dementia can often learn what is appropriate and what is not with clear messages, but they may take longer to do so. Identify triggers, e.g. a carer performing a particular task, and where possible modify environmental factors, e.g. temperature control to avoid overheating. Use distraction and redirection techniques to divert the patient's focus, e.g. provide a craft activity or puzzle. Ensure the patient has privacy if sexual behaviours are prominent.

Table 2: Factors that may cause or contribute to BPSD[3,7]

Medical	Pharmacological	Environmental or social
■ Depression ■ Anxiety ■ Delirium; may be due to infection, metabolic disturbances, medicine toxicity, substance withdrawal ■ Untreated pain ■ Infection, especially of the urinary tract or pneumonia ■ Dehydration or hyponatraemia ■ Constipation or urinary retention ■ Fatigue ■ Hearing/visual impairment	■ Medicines with anticholinergic action, e.g. amitriptyline, oxybutynin ■ Anticonvulsants, e.g. carbamazepine, phenytoin ■ Systemic corticosteroids, especially at high doses ■ Medicines with a sedative action, e.g. opioids, benzodiazepines and zopiclone, centrally acting antihistamines ■ Anti-Parkinsonian medicines	■ Unfamiliar environment ■ Separation from family ■ Noise ■ Crowding ■ Lack of privacy ■ Difficulty finding facilities, e.g. toilet ■ Difficulty accessing outdoors ■ A lack of space to move around ■ A perceived lack of security, e.g. living quarters that cannot be locked ■ Glare from sunlight or artificial lighting or poor lighting ■ Under or overstimulation ■ Withdrawal from alcohol or another drug ■ Loneliness ■ Difficult relationship with carer, family member or another resident in care

Non-pharmaceutical interventions for patients with BPSD

Non-pharmacological treatments and interventions should be trialled first for managing BPSD.[2] There are limited clinical trials supporting non-pharmaceutical approaches, and due to variations in study design and methodology it is difficult to compare the efficacy of non-pharmacological treatments, however, there is a substantial body of clinical experience justifying this approach.[7]

Once a non-pharmacological intervention has been introduced, the patient should be monitored to determine the effect of the intervention.[3] An assessment tool may be useful for this; see: "Quantifying BPSD with a tool".

Environmental interventions to prevent BPSD

Problems in the physical surroundings and amenities of the patient's home or residential care facility can cause or exacerbate restlessness, frustration, anxiety and disorientation. Changes that could be discussed with the patient's carer, family/whānau or residential care manager may include:[1]

- Reducing levels of noise and negative distraction, e.g. television at high volume
- Setting an ambient temperature and considering the patient's proximity to heaters or cold drafts
- Ensuring adequate lighting, avoiding glare from artificial light or sunlight
- Using labels and memory aids for objects the patient frequently uses
- Ensuring easy access to the toilet – regular prompting can help, signage may be appropriate
- Providing privacy and avoiding over-crowding, e.g. from groups of visitors to the home or other residents in a care facility
- Having a prominent clock, calendar and daily schedule to improve orientation to time and to prompt activity
- In residential care facilities, make the patient's surroundings as "home-like" as possible with personal belongings, pictures, photos and items of cultural significance
- Having consistent carers, where possible

Behavioural interventions to reduce BPSD

Specific behavioural interventions for BPSD will depend on the target behaviour that requires modification (Table 1). In general, encourage people with dementia to participate in activities that they find enjoyable and meaningful and are appropriate to their level of function, e.g. board games, cards, console games, artwork, craftwork, kapa haka or kaumātua

group, spectating or participating in sport, listening to music, using exercise equipment, supervised walking, social groups, interaction with animals.[2]

Local branches of Alzheimers New Zealand or Dementia New Zealand organise group activities: **www.alzheimers.org.nz** and **www.dementia.nz**

Age Concern provide an accredited visiting service where a younger person may visit an older person for approximately an hour each week: **www.ageconcern.org.nz**

Structured therapies

There is some evidence that structured interventions including music therapy, cognitive behavioural therapy and sensory therapy are beneficial for managing some BPSD including apathy, agitation, vocally disruptive behaviour, depression and anxiety.[3] Enquire with your local DHB or PHO as to the availability of specialised therapy services for patients with dementia.

Untreated pain may be a cause of BPSD

It is estimated that at least half of people with dementia regularly experience pain due to causes such as osteoarthritis and other musculoskeletal conditions, falls, pressure ulcers, infections, neuropathy, urinary retention, constipation, dental abscesses, cerumen (ear wax) or other co-morbidities.[7,8] Pain may explain BPSD such as agitation, calling out or aggression.[8]

Pain is often poorly recognised and undertreated due to the patient's difficulty in communicating their needs. Regularly enquire about pain with simple questions, e.g. "*Does it hurt?*" or "*Is it sore?*". Look for non-verbal indicators of pain such as body language, breathing patterns, facial expressions or negative vocalisation.

The Abbey Pain Scale is an example of a useful tool for assessing potential pain in patients with dementia: **www.apsoc.org.au/PDF/Publications/APS_Pain-in-RACF-2_Abbey_Pain_Scale.pdf**

Part 2: Pharmacological interventions for patients with BPSD

Pharmacological interventions have a limited role in the management of BPSD as they are associated with a range of serious adverse effects in older people and the indications for which they are effective is relatively limited.[9]

Medicines for BPSD should be:[1,3,9–11]

- Prescribed for target symptoms or behaviours for which there is evidence of effectiveness, i.e. they should not be used for other indications or to sedate patients who are difficult to manage
- Only considered once potentially reversible causes have been excluded and non-pharmacological interventions have been trialled; unless there is an immediate risk to the patient or others, or the patient is very severely distressed
- Always used in combination with non-pharmacological interventions
- Only initiated after informed consent has been obtained (and documented) from the patient or their legal representative
- Initiated as a trial and not prescribed indefinitely without need; review response to treatment, dose and adverse effects at least every three months
- Routinely withdrawn, slowly, after three months of improved symptoms unless symptoms were severe or due to a co-morbid psychiatric disorder, e.g. bipolar disorder or major depression; this is often possible without symptom re-emergence
- Re-started at the lowest effective dose, if symptoms return following a withdrawal, and schedule a further trial withdrawal in three to six months

Acetylcholinesterase inhibitors may be beneficial in mild to moderate dementia

Acetylcholinesterase inhibitors may be considered in people with Alzheimer's-type dementia, vascular dementia where subcortical ischaemic changes are prominent and dementia associated with Parkinson's disease/Dementia with Lewy Bodies (unapproved indication).[12] Donepezil (funded), rivastigmine (transdermal patch funded with Special Authority approval) or galantamine (not funded) may improve apathy, delusions and hallucinations, and less commonly improve aggression, depression, disinhibited behaviours, irritability or nocturnal disruption in patients with mild to moderate dementia.[3,7] There are a range of potentially significant adverse effects associated with acetylcholinesterase inhibitors, including gastrointestinal and neurological symptoms and bradycardia.

For further information on prescribing acetylcholinesterase inhibitors, see: "Recognising and managing early dementia", **https://bpac.org.nz/2020/dementia.aspx**

Selective serotonin reuptake inhibitors for depression, anxiety or agitation

Selective serotonin reuptake inhibitors (SSRIs) are effective for the management of depression and anxiety in people with dementia that cannot be managed by non-pharmacological interventions alone.[3] For people with Alzheimer's disease, citalopram has been shown to reduce agitation, thereby causing less caregiver distress.[13] There is also evidence that citalopram may improve other symptoms of BPSD, such as delusions, suggesting it may have antipsychotic effect.[14] A two-month trial of citalopram may be considered, although the dose-dependent risk of increased QT-prolongation and worsening cognition needs to be balanced against the benefit of treatment.[2, 13]

Tricyclic antidepressants should generally not be prescribed to patients with dementia as the anticholinergic effects may further disrupt cognition.[2]

Antipsychotics for aggression, delusions and hallucinations

Antipsychotics are only appropriate for patients with BPSD if **aggression, agitation or psychotic symptoms are causing severe distress or an immediate risk of harm to the patient or others** or if the patient has a pre-existing, co-morbid mental illness where antipsychotics are indicated.[15]

Antipsychotic medicines are only modestly effective in managing BPSD, and the level of effectiveness varies between patients. Antipsychotics are unlikely to be beneficial for wandering, calling out, social withdrawal or inappropriate sexualised behaviour in people with dementia.[1, 16] They are also less likely to be effective for intermittent but challenging behaviours that are closely related to clear environmental triggers, e.g. aggression that only occurs during personal cares.

Even short courses of antipsychotics can cause significant adverse effects in people with dementia, e.g. sedation, increased risk of falls, extrapyramidal effects, pneumonia, stroke, cardiovascular events and increased mortality (see: "Stroke and mortality risk with antipsychotic medicines"), therefore the potential benefit of treatment needs to be weighed against the risks and discussed with the patient or their representative.[15]

Antipsychotics should be avoided in patients with Lewy body dementia or Parkinsons disease with dementia, as they can cause severe adverse reactions, particularly extrapyramidal symptoms.[2] Specialist advice should be sought before initiating an antipsychotic for these patients; quetiapine, aripiprazole or clozapine may sometimes be indicated.

Selecting an antipsychotic medicine

Risperidone and haloperidol are currently the only antipsychotics approved for use in BPSD in New Zealand.[15] However, haloperidol is not a first-line choice due to increased adverse effects compared to atypical antipsychotics; it is, however, still used for the treatment of delirium in some patients with dementia.[7] Other atypical antipsychotics such as olanzapine and quetiapine are not approved for the treatment of BPSD and treatment is off label; written consent should be obtained following a discussion of the risks and benefits of treatment.[1, 15]

It is not possible to definitively recommend a single, safest and most effective antipsychotic medicine for BPSD.[18] The patient's co-morbidities, other medicines and the adverse effect profile of the medicine (Table 3) are used to determine the most appropriate treatment option. Risperidone is usually trialled first as it has strong evidence of effectiveness for BPSD, including psychosis, agitation and aggression.[2, 15] It may also be less sedating than other antipsychotics, and therefore associated with a lower risk of falls.[18]

Fewer studies have been conducted on other atypical antipsychotics

There is moderate evidence that aripiprazole is effective for the treatment of aggression and agitation in people with BPSD, but not psychosis.[2]

There is currently insufficient evidence to support the use of the newer atypical antipsychotics, amisulpride and ziprasidone for the treatment of older patients with BPSD.

Start low, go slow, with frequent monitoring

Antipsychotic medicines for the management of BPSD should be initiated as a trial and should not be prescribed indefinitely; treatment should ideally not exceed three months.[15] Initiate at the lowest dose likely to provide therapeutic benefit (Table 4), e.g. half the adult dose or less, depending on body weight, co-morbidities and concurrent medicine use.[16] Consider timing the dose in relation to the target behaviour, e.g. lunchtime for patients with agitation in the late afternoon.

Monitor patients during treatment and reduce dosing where possible

Prior to initiating an antipsychotic the patient's body weight, blood pressure and HbA_{1c} should be recorded so changes can be compared to baseline.[16] An ECG may be needed in patients at high cardiovascular risk to record their QT interval and monitor for any significant rise with treatment.[16]

The adverse effects associated with antipsychotics are generally dose-related and the risk can be minimised by regularly reviewing treatment, reducing the dose where possible and withdrawing treatment once the target behaviour is well-controlled.[16]

Table 3: A comparison of the adverse effect profile of atypical antipsychotic medicines most commonly prescribed to older patients with BPSD[19–22] *

	Risperidone	Quetiapine	Olanzapine
Adverse effect profile			
Anticholinergic effects	–	++	+
Extrapyramidal effects	++	–	+
Falls and fractures	+	++	+
Postural hypotension	+	++	+
Prolonged QT interval	–	+	–
Sedation	+	++	++
Stroke risk†	++	++	++

* Antipsychotics are associated with a wide range of adverse effects, some of which, e.g. hyperprolactinaemia and dyslipidaemia, may be less relevant to older patients. A more extensive list of adverse effects is available from: **https://bpac.org.nz/BPJ/2013/December/dementia.aspx**

† See: "Stroke and mortality risk with antipsychotic medicines"

Table 4: Recommended starting and maintenance doses for antipsychotic medicines in older people with dementia[16]

Medicine	Dose
Risperidone	Initially 250 micrograms, twice daily, increased according to response in steps of 250 micrograms, twice daily, on alternative days; usual dose 500 micrograms twice daily (up to 1 mg, twice daily, has been required). Once daily dosing is sometimes used due to the long half-life.
Quetiapine	Initially 12.5 mg, twice daily, titrated to a maximum of 100 mg per day (other patients may tolerate up to 800 mg, daily). The dosing frequency is determined by the purpose of the medicine; four times daily may be required to achieve 24-hour coverage due to the short half-life.
Olanzapine	Initially, 2.5 mg, daily, titrated to 5–10 mg, daily. There is no evidence that doses higher than 10 mg are beneficial and some evidence that they can be harmful. Once daily dosing at night is preferred.

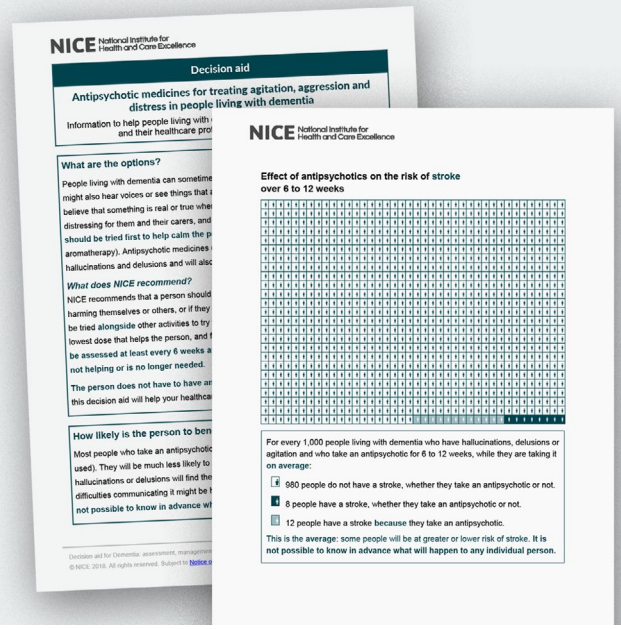
Stroke and mortality risk with antipsychotic medicines

There is significant concern that antipsychotics may cause strokes, cardiovascular events and death for some older people, particularly those with dementia. At this stage, there is insufficient evidence to state with certainty if some antipsychotics are safer than others for the management of patients with BPSD.

In older patients, all antipsychotic medicines are associated with an increased risk of stroke, cardiovascular events and excess mortality over a relatively short time frame. For example, it has been estimated that for every 1,000 patients with dementia who take an antipsychotic for six to 12 weeks, 12 additional people will have a stroke (eight other people will have a stroke whether they have taken the medicine or not).[9] Over the same period, an additional eleven people who have taken an antipsychotic will die (22 other people will die whether they have taken the medicine or not).[9]

The most common causes of death in older people taking antipsychotic medicines appears to be pneumonia, stroke and cardiac arrest.[17] Pneumonia may be an indirect result of the sedative properties of antipsychotics increasing the likelihood of aspiration.

A visual tool demonstrating the increased stroke and mortality risk associated with the use of antipsychotics in people with dementia is provided by the National Institute for Health and Care Excellence (NICE), available from: **www.nice.org.uk/guidance/ng97/resources/antipsychotic-medicines-for-treating-agitation-aggression-and-distress-in-people-living-with-dementia-patient-decision-aid-pdf-4852697005**



Close monitoring is required, especially in patients who are taking medicines with the potential for interactions with antipsychotics, including for:[16]

- Central nervous system depression, including sedation, increased confusion or cognitive impairment. Benzodiazepines, zopiclone, opioids, antihistamines, anticholinergic medicines of any kind, anti-Parkinson medicines and alcohol may exacerbate these symptoms and can also precipitate delirium.
- Peripheral anticholinergic effects, including dry mouth, constipation, urinary retention and blurred vision. Tricyclic antidepressants, opioids, oxybutynin and many other anticholinergic medicines may exacerbate these symptoms.
- Postural hypotension, dizziness and falls risk. Antihypertensives, benzodiazepines, zopiclone and antidepressants may exacerbate these symptoms.
- Extrapyramidal effects, including akathisia (restlessness), dystonia (abnormal facial and body movements), tardive dyskinesia (involuntary movements of tongue, face and jaw) and Parkinson symptoms (mainly akinetic-rigidity, but sometimes tremor)
- Metabolic changes, including weight gain, diabetes and hyperosmolar hyperglycaemic syndrome
- Infection, particularly urinary tract infections and aspiration pneumonia
- Deep vein thrombosis, pulmonary embolism and stroke

The medicine should be withdrawn immediately if significant adverse effects occur.[15]

A tool for estimating the anticholinergic burden of medicines in patients aged over 65 years is available from: **www.acbcalc.com**

Regularly review the need for antipsychotic medicines

Many patients with BPSD can be withdrawn from antipsychotics following three months of stable or improved behaviour.[1] In patients with severe symptoms it may be necessary to continue treatment long-term, e.g.:[21]

- When there are no alternative treatment options
- When the potential consequences of symptom relapse are unacceptably high
- When serious withdrawal symptoms have occurred in the past

If the patient does not respond to pharmaceutical treatment, confirm with their carer that they have been adherent to treatment and consider if the dose could be optimised and if treatment has continued for an adequate length of time, e.g. four to six weeks.[16, 23] The antipsychotic should be withdrawn if there has been no improvement in the patient's symptoms

following this timeframe.[5] If another antipsychotic is trialled, this should be done sequentially, rather than concurrently prescribing multiple antipsychotics.[15]

Withdraw antipsychotics gradually if treatment has been long-term

If a patient has been taking an antipsychotic long-term, e.g. for a year or more, the dose should be gradually tapered over weeks to months to prevent acute withdrawal and rapid symptom relapse.[23] The risk and potential severity of relapse is likely to be more severe in patients who have taken an antipsychotic for a long period or in those who have previously experienced a relapse following treatment withdrawal.[21]

In summary: assess the risk versus benefit carefully before prescribing antipsychotics

The increased risk of stroke, cardiovascular events and death associated with antipsychotic medicines in older people, and particularly those with dementia, is a significant clinical concern. It is currently unclear whether this risk is a class effect of if the risk is higher with specific medicines. What is certain is that the use of any antipsychotic medicine in people with dementia requires a careful risk versus benefit assessment. If it is decided that the patient is likely to benefit from an antipsychotic medicine, an appropriate consent process is essential, as is limiting the duration of pharmacological treatment to the minimum time period that is clinically necessary.

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