Introduction
Dementia is a chronic progressive neurodegenerative syndrome characterised by a decline in memory and at least one other cognitive function, resulting in loss of social independence. The word dementia is derived from the Latin words de (“out of”) and mens (“the mind”), but is now defined as a brain disorder that includes memory loss, deficits in cognition (thinking, planning and organising abilities), a decline in emotional control or motivation, and changes in social behaviour (such as increased irritation, apathy or problems interacting with other people). The dementias are a family of diseases defined in clinical terms, and the understanding of the dementia subcategories continues to evolve as the fields of imaging and neuropathology supply new levels of detail. About 40 years ago dementias were only classified as cortical or subcortical, while today a far more accurate classification exists of the different types of dementia.

Definition of dementia
Dementia is a chronic, global, usually irreversible deterioration of cognition. It is defined as an acquired deterioration in cognitive abilities that impairs the successful performance of activities of daily living. Memory is the most common ability lost with dementia (10% of persons over 70 years and 20% to 40% of individuals over 85 years have clinically identifiable memory loss). In addition to memory, other mental faculties are also affected in dementia, for example language, spatial orientation, calculation, decision making, judgement, problem solving and abstract reasoning. Neuropsychiatric and social deficits also develop in many dementia syndromes, resulting in depression, withdrawal hallucinations, delusions, agitation, insomnia and disinhibition. The most common forms of dementia are progressive, but some dementing illnesses are static and unchanging, while others fluctuate dramatically from day to day. Most diagnoses of dementia require some memory deficit, although there are many dementias, such as frontotemporal dementia (FTD), where memory loss is not a presenting feature. In contrast to patients with confusional states, dementia symptoms progress over months to years, and alertness is preserved until the very late stages of the disease.

Epidemiology of dementia
Dementia may occur at any age but affects primarily the elderly. Alzheimer’s disease is the most common cause of dementia in Western countries and accounts for more than 50% of cases. Dementia also develops in approximately 30 to 70% of people with Parkinson’s disease. Vascular disease is also a common cause of dementia, representing 10% to 20% of dementia cases in the United States. In populations with limited access to medical care, where vascular risk factors are undertreated, the prevalence of vascular dementia can be high. Vascular dementia is related to cardiovascular risk factors (for example, smoking, diabetes, hypertension and hyperlipidaemia), but the causes of other forms of dementia are unclear. Onset is rare in people under the age of 65 years (termed young- or early-onset dementia)
onset dementia as opposed to late- or senile-onset dementia), accounting for about 2% of cases. In patients under the age of 60 years, FTD rivals Alzheimer’s disease as the most common cause of dementia. Chronic intoxications (including those resulting from alcohol and prescription medicines) are an important and treatable cause of dementia.

The worldwide direct costs of dementia care were estimated to be $315 billion for 29.3 million demented in 2005, with 77% of the costs occurring in the more developed regions of the world. The cost (including $142 billion for informal care) was estimated to be $422 billion in 2009, based on a population of 34.4 million demented persons. Due to demographic changes, it is forecasted that a major increase in the prevalence of dementia will occur in developing countries. Dementia affects approximately 700 000 people in the United Kingdom (about 1.1% of the entire population), and is forecast to increase by 38% over the next 15 years. Dementia affects more than 4 million Americans and results in a total health care cost of more than $100 billion annually. It is therefore associated with high health care and social costs. About two-thirds of people with late-onset dementia live in private households, with the remainder in care facilities. Dementia is said to account for more than 50% of nursing home admissions.

A guideline was issued in November 2006 by the National Institute for Health and Clinical Excellence (NICE) and the Social Care Institute for Excellence (SCIE) that provides a framework on which the delivery of health and social care services should be based for the support of patients with dementia and their carers in England and Wales. Importantly, it makes recommendations to ensure that all people with dementia have fair access to assessment, care and treatment on the basis of need, irrespective of their age, gender and social or cultural background.

In countries with high HIV-infection rates, HIV-associated dementia (HAD), AIDS-related dementia or AIDS dementia complex (ADC), is an increasing problem. Prevalence of ADC is estimated to be between 10% and 24% in HIV-infected individuals in Western countries. In the United States, HIV-1 is the most common cause of dementia in adults under the age of 40 years. Researchers found AIDS-related dementia in 31% of HIV-patients in Uganda, and it is projected that more than 8 million people in sub-Saharan Africa suffer from HIV-associated dementia. Furthermore, neurocognitive impairment affects 10 to 60% of people living with HIV/AIDS, depending on the stage of the disease. The major causes of neurological impairment include opportunistic infections. With the advent of highly active antiretroviral therapy (HAART), the incidence of ADC has declined in developed countries. However, its prevalence is increasing (due to the lengthening of HIV-infected patients’ life expectancy).

**Aetiology/pathophysiology of dementia**

Dementia results from the disruption of cerebral neuronal circuits. Two factors combine to cause the specific disorder, namely the quantity of neuronal loss and the location of the affected regions. Behaviour and mood are modulated by noradrenergic, serotonergic and dopaminergic pathways, while acetylcholine seems to be particularly important for memory. Therefore, the loss of cholinergic neurons in Alzheimer’s disease may underlie the memory impairment, while in patients with non-Alzheimer’s dementias, the loss of serotonergic and glutaminergic neurons cause primarily behavioural symptoms, leaving memory relatively spared. Neurotrophins are also said to play a role in memory function, in part by preserving cholinergic neurons, and therefore represent a possible pharmacologic pathway toward slowing or reversing the effects of Alzheimer’s disease.

Dementia is a syndrome with many causes. The following is said to account for almost 90% of dementia cases:

- Alzheimer’s disease
- Multiple cerebral infarcts
- Dementia with Lewy bodies
- Alcoholism
- Normal pressure hydrocephalus
- Primary or metastatic central nervous system (CNS) neoplasms
- Frontotemporal dementia (FTD)
- Parkinson’s disease
- Huntington’s disease
- Pick’s disease
- Prion diseases (for example, Creutzfeldt-Jakob disease)
- Neurosphilis
- HIV infection
- Hypothyroidism
- Deficiency of vitamins B12, B6, B1 or niacin
- Chronic meningitis
- Subdural haematoma

Dementias have anatomically specific patterns of neuronal degeneration that dictate the clinical symptomatology. Alzheimer’s disease begins in the entorhinal cortex, spreads to the hippocampus, and then moves to the posterior temporal and parietal neocortex, eventually causing a relatively diffuse degeneration throughout the cerebral cortex.

**Multi-infarct dementia** (also known as vascular dementia) is associated with focal damage in a random patchwork of cortical regions. In this case dementia results from multiple infarctions in the territory of major cerebral vessels.

Diffuse white matter damage may disrupt intracerebral connections and cause dementia syndromes. Dementia, in this case, results from subcortical infarctions in the distributions of deep penetrating arteries (lacunar state, Binswanger’s disease, subcortical arteriosclerotic encephalopathy). There is usually a history of stepwise progression of neurologic deficits, focal signs of neurologic examination and multiple infarctions on brain imaging studies. Patients generally have a history of hypertension or other risk factors of atherosclerosis.

Whereas Alzheimer’s disease primarily presents as memory...
Evidence loss and is often associated with aphasia or other disturbances of language, in contrast, patients with frontal lobe or subcortical dementias such as FTD or Huntington’s disease are less likely to begin with memory problems and are more likely to have difficulties with attention, judgement, awareness and behaviour.5 Lesions of specific cortical-subcortical pathways have equally specific effects on behaviour.7 The dorsolateral prefrontal cortex has connections with the dorsolateral caudate, globus pallidus and thalamus. Lesions of these pathways result in poor organisation and planning, decreased cognitive flexibility and impaired judgement. The lateral orbital frontal cortex connects with the ventromedial caudate, globus pallidus and thalamus. Lesions of these connections cause irritability, impulsiveness and distractibility. The anterior cingulated cortex connects with the nucleus accumbens, globus pallidus and thalamus. Interruption of these connections produces apathy and poverty of speech and even akinetic mutism.

Chronic drug intoxication is often listed as a cause of dementia but actually produces a confusional state. The existence of alcohol-induced dementia is controversial. Although animal and cell culture studies provide evidence for a direct neurotoxic effect of alcohol, dementia in alcoholic patients also results from associated nutritional deficiency, from recurrent head trauma and (rarely) from acquired hepatocerebral degeneration, a complication of chronic hepatic insufficiency caused by alcoholic cirrhosis.

Diagnosis of dementia Currently only a third of people with dementia receive a formal diagnosis at any time during their illness, and when made it may be too late for those suffering from the illness to make informed choices.7 Accurate diagnosis of dementia is a prerequisite for optimal therapy. The general diagnostic criteria for dementia according to the DSM-IV are given in Box 1.3 Different tests can be conducted to determine the degree and type of dementia. Table I provides an overview of the evaluation of patients with dementia. The diagnostic process in dementia has three major components, namely the clinical diagnosis, a logical search for the cause, and the identification of treatable comorbid conditions and other contributing factors.13

A range of brief cognitive tests can be used for the diagnosis of dementia, for example the Montréal Cognitive Assessment, the DemTect, the 7-Minute Screen, the General Practitioner Assessment of Cognition and the Behavioural Neurology Assessment Short Form.13 These tests may be more accurate than the Mini-Mental State Examination (MMSE) in discriminating between dementia and the normal state. There is, however, insufficient evidence to recommend one test over

<table>
<thead>
<tr>
<th>Routine evaluation</th>
<th>Optional focussed tests</th>
<th>Occasionally helpful tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Psychometric testing</td>
<td>EEG</td>
</tr>
<tr>
<td>Physical examination</td>
<td>Chest x-ray</td>
<td>Parathyroid function</td>
</tr>
<tr>
<td>Laboratory tests</td>
<td>Lumbar puncture</td>
<td>Adrenal function</td>
</tr>
<tr>
<td>– Thyroid function (to rule out hypothyroidism)</td>
<td>Liver function</td>
<td>Urine heavy metals</td>
</tr>
<tr>
<td>– Vitamin B₁₂</td>
<td>Renal function</td>
<td>Red blood cell sedimentation rate</td>
</tr>
<tr>
<td>– Complete blood count (to rule out anaemia)</td>
<td>Urine toxin screen</td>
<td>Angiogram</td>
</tr>
<tr>
<td>– Serum electrolytes (to rule out hypernatraemia)</td>
<td>HIV</td>
<td>Brain biopsy</td>
</tr>
<tr>
<td>– Serum calcium (to rule out hypercalcaemia)</td>
<td>Apolipoprotein E</td>
<td>Single Photon Emission CT (SPECT)</td>
</tr>
<tr>
<td>– Serum fasting glucose (to rule out hyperglycaemia)</td>
<td>Rapid plasma regain (RPR) test or test for syphilis (VDRL)</td>
<td>Positron Emission Tomography (PET)</td>
</tr>
</tbody>
</table>
another, and these brief cognitive tests have not been developed to differentiate between dementia subtypes and should not be used for this purpose.13

Dementias can be classified in several ways, for example Alzheimer’s or non-Alzheimer’s type, cortical or subcortical, irreversible or potentially irreversible, or common or rare. Dementias may furthermore be primary neurodegenerative disorders or due to another condition. Table II indicates the differential diagnosis of dementia. Treatable causes are important to recognise and include hypothyroidism, vitamin $B_{12}$ deficiency, neurosyphilis, brain tumour, normal pressure (communicating) hydrocephalus and chronic subdural haematoma. In addition, although not curable, dementia associated with HIV infection may be slowed by antiretroviral treatment. Approximately 10% to 15% of patients referred for evaluation of dementia suffer from depression (“pseudodementia”, depression that mimics dementia), which may also respond to treatment.1

Table III provides an overview of how the major dementias can usually be distinguished by initial symptoms, neuropsychological, neuropsychiatric and neurologic findings, and by neuroimaging features.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Specific examples</th>
<th>Potentially reversible</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Most common causes of dementia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vascular dementia</td>
<td>Multi-infarct, diffuse white matter disease (Binswanger’s)</td>
<td></td>
</tr>
<tr>
<td>Alcoholism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug/medication intoxication</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Less common causes of dementia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin deficiencies</td>
<td>Thiamine (Vitamin B$\text{I}$), Wernicke’s encephalopathy, $B_{12}$ (pernicious anaemia), nicotinic acid (pellagra)</td>
<td>Yes</td>
</tr>
<tr>
<td>Endocrine and other organ failure</td>
<td>Hypothyroidism, adrenal insufficiency and Cushing’s syndrome, hypo- and hyperparathyroidism, renal failure, liver failure, pulmonary failure</td>
<td>Yes</td>
</tr>
<tr>
<td>Chronic infections</td>
<td>HIV, povavirus (progressive multifocal leukoencephalopathy), Prion (Creutzfeldt-Jakob and Gerstmann-Sträussler-Scheinker diseases)</td>
<td></td>
</tr>
<tr>
<td>Head trauma and diffuse brain damage</td>
<td>Dementia pugilistica, postanoxia, postencephalitis</td>
<td></td>
</tr>
<tr>
<td>Neurosyphilis</td>
<td>Chronic subdural haematoma, normal-pressure hydrocephalus</td>
<td>Yes</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Primary brain tumour, metastatic brain tumour</td>
<td></td>
</tr>
<tr>
<td>Toxins</td>
<td>Drug, medication and narcotic poisoning, heavy metal intoxication</td>
<td>Yes</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>Depression (pseudodementia), schizophrenia, conversion reaction</td>
<td>Yes</td>
</tr>
<tr>
<td>Degenerative disorders</td>
<td>Huntington’s disease, Pick’s disease, dementia with Lewy bodies, progressive supranuclear palsy (Steel-Richardson syndrome), multisystem degeneration (Shy-Drager syndrome), hereditary ataxias (some forms), motor neuron disease (amyotrophic lateral sclerosis (ALS) – some forms), FTD, cortical basal degeneration, multiple sclerosis, adult Down’s syndrome with Alzheimer’s disease, ALS-Parkinson’s-Dementia complex of Guam</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Sarcoïdosis</td>
<td></td>
</tr>
<tr>
<td>Vasculitis, acute intermittent porphyria, recurrent nonconvulsive seizures</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Additional conditions in children or adolescents</td>
<td>Hallervorden-Spatz disease, subacute sclerosing panencephalitis, metabolic disorders (e.g. Wilson’s and Leigh’s diseases, leukodystrophies, lipid storage diseases, mitochondrial mutations)</td>
<td></td>
</tr>
</tbody>
</table>
Overview of common forms of dementia

A concise overview is given below of some of the most common forms of dementia.

**Alzheimer’s disease**

Alzheimer’s disease is the most common cause of dementia and accounts for more than 50% of dementia cases. It is a slowly progressive disorder that runs a course of five to 10 years and typically begins with impairment of learning and recent memory. The pathology of Alzheimer’s disease is characterised by the presence of beta-amyloid plaques outside neurons and neurofibrillary tangles within neurons.2 Although these lesions may be present in any aging brain, these lesions in people with Alzheimer’s disease tend to be more numerous and accumulate in areas of the brain involved in learning and memory.2

The hallmark symptom of Alzheimer’s disease is difficulty in recalling new information.2 As Alzheimer’s disease progresses, memory loss disrupts daily life (for example, the person may get lost in a previously familiar neighbourhood). The patient may also experience a decline in cognitive ability (finding it hard to make decisions, solve problems or make good judgements), and may undergo significant changes in mood and personality (such as becoming more irritable, hostile or apathetic).2 Alzheimer’s disease is severely debilitating.

**Vascular dementia**

The second most common type of dementia in old age is vascular dementia, which develops after one or more ischaemic strokes (blockages of blood vessels in the brain depriving brain cells of oxygen and causing infarcts).2 Areas of dead tissue result. Symptoms at onset depend on the type of stroke that occurs. In the most common type of vascular dementia, brain damage results from a series of minor strokes (affecting small blood vessels) that may occur unobserved by the patient, family members or friends. The mental deterioration proceeds in a “stepwise” pattern, in which a person experiences a cognitive decline, seems to stabilise, then deteriorates further after another stroke. Specific symptoms may include confusion, slurred speech or impaired thinking. This type of vascular dementia is also known as multi-infarct dementia.

**Mixed dementia**

A patient with mixed dementia has dementia of the Alzheimer’s type plus another type of dementia (most often vascular dementia). The fact that Alzheimer’s disease and vascular dementia often co-occur makes sense, given that atherosclerosis and vascular disease contribute to the development of both types of dementia. Unfortunately, when both types occur together, the brain damage and resulting mental deterioration may be particularly severe. Mixed dementia is most likely in a patient with cardiovascular disease, such as someone with high cholesterol levels, hypertension, or evidence of atherosclerosis, who also develops dementia.

**Lewy body dementia**

Lewy bodies, named after the scientist who discovered them, are abnormal deposits of alpha-synuclein (a protein whose function is unknown) within neurons.2 These lesions are also found in some patients with Alzheimer’s disease and in those with Parkinson’s disease. They are the hallmark of Lewy body dementia and can be confirmed only through autopsy. Practitioners diagnose the condition on the basis of symptoms characteristic of Lewy body dementia, such as hallucinations, fluctuating levels of alertness during the day, and movement disorders, such as akathisia, muscle rigidity, unstable posture and tremor.2

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### Table III: Clinical differentiation of the major dementias

<table>
<thead>
<tr>
<th>Disease</th>
<th>First symptom</th>
<th>Mental status</th>
<th>Neuropsychiatry</th>
<th>Neurology</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s disease</td>
<td>Memory loss</td>
<td>Episodic memory loss</td>
<td>Initially normal</td>
<td>Initially normal</td>
<td>Entorhinal cortex and hippocampal atrophy</td>
</tr>
<tr>
<td>Frontotemporal dementia</td>
<td>Apathy, poor judgement/insight/speech/language, hyperorality (insertion of inappropriate objects in the mouth)</td>
<td>Frontal/executive, language, spares drawing</td>
<td>Apathy, disinhibition, hyperorality, euphoria, depression</td>
<td>Due to progressive supranuclear palsy/cortical basal degeneration overlap, vertical gaze palsy, axial rigidity, dystonia, alien hand</td>
<td>Frontal and/or temporal atrophy, spares posterior parietal lobe</td>
</tr>
<tr>
<td>Dementia with Lewy bodies</td>
<td>Visual hallucinations, REM sleep disorder, delirium, Capgras syndrome, parkinsonism</td>
<td>Drawing and frontal/executive, spares memory, delirium prone</td>
<td>Visual hallucinations, depression, sleep disorder, delusions</td>
<td>Parkinsonism</td>
<td>Posterior parietal atrophy, hippocampi larger than in Alzheimer’s disease</td>
</tr>
<tr>
<td>Creutzfeldt-Jakob disease</td>
<td>Dementia, mood, anxiety, movement disorders</td>
<td>Variable, frontal/executive, focal cortical, memory</td>
<td>Depression, anxiety</td>
<td>Myoclonus, rigidity, parkinsonism</td>
<td>Cortical ribboning and basal ganglia or thalamus hyperintensity on diffusion/flare MRI</td>
</tr>
<tr>
<td>Vascular</td>
<td>Often but not always sudden, variable, apathy, falls, focal weakness</td>
<td>Frontal/executive, cognitive slowing, can spare memory</td>
<td>Apathy, delusions, anxiety</td>
<td>Usually motor slowing, spasticity</td>
<td>Can be normal</td>
</tr>
</tbody>
</table>

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1. Evidence-Based Pharmacy Practice
2. SA Pharmaceutical Journal – August 2010
disorders similar to Parkinson’s disease (stiffness, shuffling gait, balance problems that cause falls and lack of facial expression).

**Frontotemporal dementia (FTD)**

This type of dementia has a younger age of onset than for Alzheimer’s disease. FTD develops when brain damage occurs in the front of the cerebral cortex (the frontal lobes) and the sides (the temporal lobes). The source of brain damage remains unclear, although autopsies have revealed abnormal lesions known as Pick bodies that develop in the brain (a subtype known as Pick’s disease). In most cases of FTD, the frontal lobes shrink as the disease progresses. This dementia has a rapid onset, then progresses steadily usually first causing changes in personality and behaviour (for example, a previously polite person may suddenly start making rude remarks). Language impairment is also prominent, but memory and awareness of surroundings are usually spared. No treatment is available to slow the progression of this type of dementia.

**Counselling approach to follow**

All new cases of dementia should be referred by the pharmacist so that a correct initial diagnosis can be made. A thorough medical history should be taken, a comprehensive physical examination should be performed and, if appropriate, laboratory/diagnostic tests may be conducted to confirm the diagnosis and to exclude serious disorders.

The history will typically include the onset, duration and tempo of progression of the dementia. An acute or subacute onset of confusion may represent delirium and should trigger the search for intoxication, infection or metabolic derangement. An elderly person with slowly progressive memory loss over several years is likely to suffer from Alzheimer’s disease.

**Available treatment options for dementia**

There is a range of available treatment options for dementia depending on the cause. Management of patients with dementia has to be individualised, and tailored to each patient’s responses and preferences.

**Therapeutic objectives**

The major therapeutic objectives are to treat any correctable causes of the dementia and to provide comfort and support to the patient and caregiver(s).

**General measures**

Many approaches have been suggested to prevent and treat dementia, but there is no known way to prevent dementia (except possibly vascular dementia where risk can be reduced by maintaining a healthy lifestyle and by interventions targeting cardiovascular risk). Middle aged and older people should therefore be reviewed for vascular and other modifiable risk factors for dementia (for example, smoking, excessive alcohol consumption, obesity, diabetes, hypertension and hypercholesterolaemia) and treated where appropriate. Treatment of other underlying causes may include thyroid replacement for hypothyroidism, vitamin therapy for thiamine or vitamin B12 deficiency or for elevated serum homocysteine, antibiotics for opportunistic infections, ventricular shunting, and appropriate surgical, radiation and/or chemotherapeutic treatment for CNS neoplasms. Removal of sedating or cognition-impairing medicine is often beneficial. If the patient is depressed rather than demented (pseudodementia), the depression should be vigorously treated.

**Non-pharmacological interventions**

The primary goal is to make the demented patient’s life comfortable, uncomplicated and safe. Non-pharmacologic measures have an important place in the management of dementia, and include patient safety, environmental measures, caregiver assistance and end-of-life issues.

**Patient safety**

Safety is an important issue that includes not only driving but also safety in the kitchen, bathroom and sleeping area. These areas need to be monitored, supervised and made as safe as possible to prevent accidents (particularly falls). A move to a retirement home, assisted-living centre or nursing home can initially increase the confusion and agitation. Repeated reassurance, reorientation and careful introduction to the staff will help smooth the process.

Demented patients usually object to losing control over familiar tasks such as driving, cooking and handling finances. Attempts to help or take over may be greeted with complaints, depression or anger. Explanation, reassurance, distraction and calm statements are more appropriate responses than hostile or forced responses. Eventually, tasks such as finances and driving must be assumed by others, and the patient will conform and adjust.

**Environmental measures**

Preparing lists, schedules, calendars and labels can be helpful. It is also useful to stress familiar routines, short-term tasks, walks and simple physical exercises. For many demented patients, memory for facts is worse than that for routine activities, and they may still be able to take part in physical activities such as walking, bowling, dancing and golf. According to a Mayo Clinic study, even small amounts of moderate exercise are associated with a 30% to 40% lower risk of developing mild cognitive impairment. The provision of activities that are known to be enjoyable to the patient can be of considerable benefit.

**Caregiver assistance**

Attention should be paid to frustration and depression in family members and caregivers. Caregiver guilt and burnout are common, and day-care facilities and breaks can be helpful. Family members often feel overwhelmed and helpless and may vent their frustrations on the patient, each other or health care providers. Education and counselling about dementia are important. Support groups and support services can provide assistance.

**End-of-life issues**

Insight and judgement deteriorate in patients with dementia. The appointment of a family member, guardian or lawyer
to oversee finances may be necessary. Early in dementia, before the patient is incapacitated, the patient’s wishes about care should be clarified, and financial and legal arrangements should be made (for example, power of attorney).

Pharmacological treatment
Agitation, hallucinations, delusions and confusion are difficult to treat. These behavioural problems represent major causes for nursing home placement and institutionalisation. Before treating these behaviours with medicine, a thorough search for potentially modifiable environmental or metabolic factors should be sought. Hunger, lack of exercise, toothache, constipation, urinary tract infection or drug toxicity all represent easily correctable factors that can be treated without psychoactive medication. Medicines such as phenothiazines and benzodiazepines may ameliorate the behaviour problems but have untoward side effects such as sedation, rigidity and dyskinasias.

Only a few active ingredients are indicated for the treatment of dementia, and pharmacological treatment depends on the type of dementia. Five active ingredients have been approved for Alzheimer’s disease, but they alleviate symptoms only slightly when used alone. Three acetylcholinesterase inhibitors (AChIs) are available, namely donepezil, galantamine and rivastigmine.10 They inhibit acetylcholinesterase, increasing the acetylcholine level in the brain. Galantamine and rivastigmine are indicated for the treatment of mild-to-moderate dementia, while donepezil is indicated in South Africa for the symptomatic treatment of dementia of Alzheimer’s disease.11 Tacrine is also mentioned, but it is rarely used because of its hepatotoxic effects.2 The newest medicine is memantine (an N-methyl-D-aspartate (NMDA)-receptor antagonist) that blocks the action of glutamate, a neurotransmitter that usually activates neurons but in excessive amounts can destroy them.2,12 Memantine may help to slow the progression of moderately severe to severe dementia resulting from Alzheimer’s disease.4,7,13 Efficacy has not been established beyond six months.13 Rivastigmine is indicated for treatment of mild-to-moderate dementia in Parkinson’s disease.7 It has been suggested that combining a cholinesterase inhibitor with memantine may work better, although the benefits remain modest. Other medicines such as anti-inflammatory agents are being investigated in the treatment or prevention of Alzheimer’s disease.

Depression, if present, should be recognised and treated, usually initially with a low dose of a selective serotonin re-uptake inhibitor (SSRI), closely monitoring for efficacy and toxicity. Anxiety and agitation can also be treated if appropriate. Sometimes apathy, visual hallucinations, depression and other psychiatric symptoms respond to AChIs, obviating the need for other more toxic therapies.

Despite their unfavourable side effect profile, second-generation antipsychotics such as quetiapine are increasingly being used for patients with behavioural disorders (for example, agitation, aggression and psychosis). When patients do not respond to treatment, it is not recommended to advance to higher doses or to use anticholinergics or sedatives (such as barbiturates or benzodiazepines). The risk-benefit ratio of prescribing psychoactive drugs should always be considered, especially the use of atypical antipsychotics in people with Alzheimer’s disease. All antipsychotics, especially atypical agents, are associated with an increased risk of cerebrovascular accidents in patients with dementia and should therefore be used with caution in these patients.14

A proactive strategy has been shown to reduce the occurrence of delirium in hospitalised patients. This strategy includes frequent orientation, cognitive activities, sleep-enhancement measures, vision and hearing aids, and correction of dehydration.

Conclusion
Dementia, in most cases, is progressive and incurable. Dementia is a syndrome and many different subtypes can be classified. The single strongest risk factor for dementia is increasing age, but whether dementia is an inevitable consequence of normal human aging remains controversial. The most common form of dementia is Alzheimer’s disease, and in South Africa with its high prevalence rate of HIV, HIV-associated dementia is also increasing rapidly. There is no cure for dementia, but medication is available to control the symptoms and to slow down the progression of the condition. Most interventions are used to relieve symptoms and improve the quality of life of patients and their carers. It remains a complicated syndrome for which an accurate diagnosis by a specialist is crucial to ensure that optimal pharmacological and non-pharmacological treatment is provided. It is also a condition that not only affects the person, but also family and friends. There is generally a low level of public and professional understanding of dementia.7 The role of the pharmacist in dementia is supportive, consisting of information-sharing and counselling on the condition and on its treatment.

References