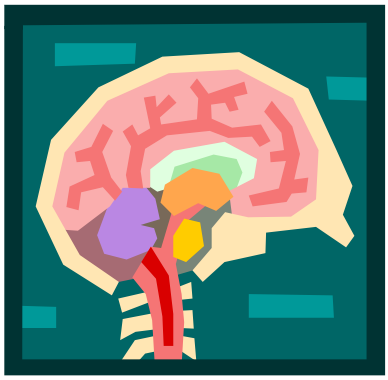
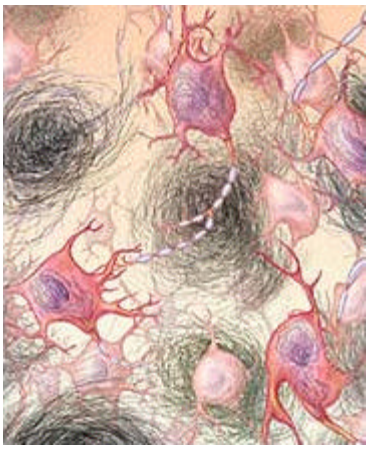


Table of Contents

Dementia and Ageing

Introduction

A number of changes in the brain are thought to occur as we age. Although not universally observed, some of the changes may include:



- Decreased volume and weight (2-3% decline per decade after the age of 50)
- Decreased in nerve cell number and size
- Lipofuscin accumulation (brown pigment left over from the breakdown and digestion of cells)
- Reduced cell nucleus volume
- Reduced length and number of dendrites
- Synapse reduction
- Plaques and tangles

Certain conditions seem to express an increased incidence and severity of these changes. For example, although normal ageing is often associated with a mild mental decline ("*benign forgetfulness"*), a more severe condition known as dementia may occur.

Dementia

Dementia can be defined as "A chronic deterioration of intellectual function and other cognitive skills severe enough to interfere with the ability to perform activities of daily living" (The Merck Manual of Diagnosis and Therapy).

Dementia is not a part of normal ageing, but rather a syndrome that is part of an illness, such as Alzheimer's disease (AD). Various organisations have created criteria for the diagnosis of dementia, with the two most popular being:

- International Classification of Disease (ICD-10) by the *World Health Organisation*
- Diagnostic and Statistical Manual (DSM-IV) by the *American Psychiatric Association*.
    - The development of multiple cognitive deficits that include memory impairment and at least one of the following:
        - Aphasia (difficulty in expressing thoughts as spoken words)
        - Apraxia (difficulty in carrying out simple, directed acts)
        - Agnosia (difficulty in interpreting familiar faces or other well known objects)
        - Disturbance in executive functioning (difficulty with planning and organisation of tasks)

Some of the *most common symptoms* exhibited are (Aging Vignette #39):

- 97% are confused or have trouble making decisions

- 97% forget names of people and places
- 87% have problems with physical ambulation
- 86% do or say things repeatedly
- 85% have problems completing tasks like housework or grocery shopping
- 85% act restless or agitated
- 83% sit doing nothing
- 73% are stubborn or uncooperative
- 63% talk to themselves or talk nonsense

Some *less common symptoms* observed include:

- 52% have disturbed sleep patterns
- 50% see things that aren't there
- 50% are fearful or suspicious of people
- 18% have antisocial behaviour
- 14% threaten to hurt themselves or others

For our purposes, we will distinguish between three types of dementia in the elderly:

- *Alzheimer's Disease*
  - Most common type of dementia, accounting for approximately two thirds of all dementia cases.
    - Gradual onset
    - Continuing decline of memory
    - At least one additional cognitive domain involved
    - Not explained by other disorders
- *Vascular dementia*
  - Dementia associated with problems in the circulation of blood to the brain (cerebrovascular disease). Vascular dementia usually results from damage to brain function from multiple strokes (also known as multi-infarct dementia). Vascular dementia is the second most common type of dementia in Canada, accounting for approximately 20% of all cases. In 1991, 1.5% of the population 65+ had some form of vascular dementia.
    - Associated with cerebrovascular disease
    - Abrupt onset
    - Stepwise decline following another ischemic event
    - Impaired executive functioning
    - Gait disorder
    - Emotional liability
- *Lewy body dementia*
  - Illness is associated with protein deposits called Lewy bodies, tiny spherical structures found in the cortex of the brain. Some of the common presentations include:
    - Progressive cognitive decline
    - Fluctuating symptoms

- Recurrent visual hallucinations
- Movement difficulties
- Hypersensitivity to neuroleptics (antipsychotic drugs)
- Repeated falls

Demographics

Although dementia can strike at any age, it is mostly a disease of the elderly, affecting more than 15% of persons over the age of 65 and as many as 40% of persons over the age of 80.

Canada[1]

In 1991, 8% of Canadians (± 250 000) over the age of 65 suffered from dementia, and the economic cost of dementia was estimated to be over $3.9 billion annually (± 5.8 % of total health care costs). It is estimated that dementia is present in

- 23% of seniors aged 85 to 89 years old
- 40% of seniors aged 90 to 94 years old
- 55% of seniors aged 95 to 99 years old
- 85% of seniors aged 100 to 106 years old

In 1991, women accounted for 68% of dementia cases. The greater number of women with dementia mainly reflects the greater number of women in older age groups where the likelihood of dementia is even higher. It is expected that the number of cases will triple by 2031, and costs will rise to upwards of 12 billion dollars per year. Approximately 50% of seniors with dementia live in the community while the rest reside in an institutional setting. The net economic cost of caring for a dementia patient in the community is $10,100, compared with $19,100 in an institution.

It is reported that as many as 10,000 deaths per year can be attributed directly to dementia, and up to 300,000 deaths per year may be caused by dementia to a varying degree (1985 data).

Table 1: Expected cases of dementia

YEAR	NUMBER OF DEMENTIA CASES IN CANADA
1991	253,000
2001	364,000
2011	475,000
2021	592,000
2031	778,000

[1] Source: Aging Vignettes, National Advisory Council on Aging (1991 data)

Caregivers[2]

Most patients with dementia have an informal *caregiver* (friend or family) to help them. The demands placed on these caregivers can be quite important. Caregivers of people with mild to moderate dementia put in 3.2 hours of care per day, while caregivers of people with severe dementia can put in as many as 8.06 hours of care per day. In addition, depression is twice as common in someone who cares for a dementia patient than in someone who cares for a person without dementia. The spouse often serves as a caregiver in the community setting, while a child usually serves this purpose in institutions. Furthermore, wives are most likely to be the informal caregiver of a dementia patient living in the community, while daughters are most likely to be the informal caregiver of a patient living in an institution. On average, informal caregivers spend 2.5 years providing care to a dementia patient.

Alzheimer's Disease (AD)

AD is the leading cause of dementia, and the number of people who have this disease is expected to triple over the next 20 years as the population ages. It is estimated that by the year 2025, as many as 22 million people around the world may suffer from Alzheimer's disease. Although not recognized as a unique form of dementia until the 1960's, the German neurologist Alois *Alzheimer* was the first to describe the syndrome at a meeting in Munich in 1906. During this meeting, he described the symptoms observed in a 51-year-old female patient (Auguste D) who died after years of progressive dementia. The symptoms described included progressive memory impairment, altered cognitive function, changed behaviour including paranoia, delusions, and loss of social appropriateness, and a progressive decline in language function. During an autopsy, he also observed that her brain tissue had abnormal clumps and irregular knots of brain cells. These signs remain the hallmark of the disease even today.

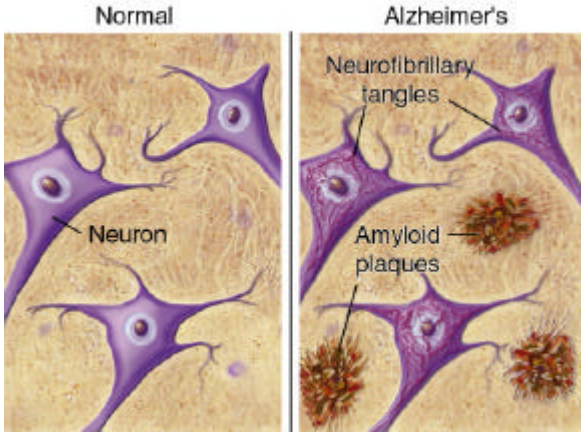


Figure 1: Lesions observed in AD

[2] Source: Aging Vignettes, National Advisory Council on Aging (1991 data)

Demographics

It is estimated that 8.5% of the world population 65+ and 28% of the world population 85+ suffer from AD. In 1991, it was estimated that 5.1 % of the Canadian population 65+ was suffering from Alzheimer's disease (± 161 000). This accounted for approximately two thirds of all dementia cases.

Causes

Although research is ongoing, the precise aetiology of AD has yet to be identified. Nonetheless, a number of theories do exist. Some of the most popular include:

- *Chemical Theories* (Deficiencies and Toxic Excesses)
  - *Biochemical Changes in Growth (Trophic) Factors*
    - Certain naturally occurring substances may affect the nervous system and that may contribute to the dysfunction or death of brain cells in AD. One possible reason for nerve cell death in Alzheimer's patients is a decline in growth-promoting factors that maintain the functioning of brain cells an increase in factors that are toxic to brain cells. One of the substances that scientist are currently investigating is *nerve growth factor* (NGF), a protein that promotes development of the sensory and sympathetic nervous systems and is required for maintenance of sympathetic neurons. Experiments in aged rats indicate that specific nerve growth factors can stimulate the growth of new synaptic connections in the hippocampus, restoring some memory loss. Methods of safely introducing NGF into the brain, possibly through the transplant of genetically engineered cells, are being researched. Other research is exploring whether changes or an imbalance in the metabolism of certain elements like calcium in brain cells may be part of the process by which the cells degenerate and die in AD.
  - *Chemical Deficiencies*
    - One of the ways in which brain cells communicate with one another is through chemicals called *neurotransmitters*. Studies of Alzheimer's disease brains have uncovered diminished levels of various neurotransmitters that are thought to influence intellectual functioning and behaviour. For example, reduced levels of the neurotransmitter acetylcholine (ACh) have been found in Alzheimer's disease. In fact, drugs whose side effects lower ACh levels in the brain can cause reversible memory problems. These findings have led to a number of drug studies employing pharmacological agents that affect ACh levels in patients. Tacrine hydrochloride (THA or tacrine, Cognex) is a drug that has been studied extensively.
  - *Toxic Chemical Excesses*
    - Although some researchers have found increased levels of aluminium, mercury, or other metals in the brains of Alzheimer's disease victims, others have not. It is possible that certain

substances only accumulate in the brain in response to the changes associated with AD, and are not related to the cause of the disease.

- *Genetic Theory*
    - The genetic component of AD is not well understood, and its contribution varies from 1-40% depending on the studies. Several connections between Alzheimer's disease and Down's syndrome led researchers to look for genetic factors in Alzheimer's disease on chromosome 21, the chromosome that is affected in Down's syndrome. At the present time, several genetic markers have been identified on chromosomes 21 and 14 in that small number of families where Alzheimer's disease has occurred with unusual frequency at relatively early ages. In families where the disease has tended to develop at later ages, other studies suggest that Alzheimer's disease is unusually frequent in persons who have a particular form of the apolipoprotein E (ApoE) gene found on chromosome 19. Only a minority of the general population show this version (ApoE4) of the gene, out of several variants that occur. Despite these findings, the extent of genetic and hereditary involvement in Alzheimer's disease remains unclear. There are a vast number of people affected with this disorder who are not part of a strong family pattern. Furthermore, the genetic factors associated with the disease clearly vary for different families. This has led some investigators to postulate that there may be a number of subtypes of Alzheimer's disease, with differing risk factors and causes.

- *Autoimmune Theory*
    - The body's immune system may begin to attack its own tissues, producing antibodies to its own essential cells. This autoimmune response may take place in the brain, and some scientists believe that certain late life changes in aging neurons might be triggering an autoimmune response that evokes symptoms of Alzheimer's disease in vulnerable individuals. Curiously, some *antibrain antibodies* (protein produced by immune system to destroy nerve cells) have been identified in the brains of those with Alzheimer's disease. Their significance, though, is not known, especially since some antibrain antibodies have also been identified in aging brains without Alzheimer's disease. Moreover, even if changes are occurring in brain neurons to trigger an autoimmune response, what originally induces these brain cell changes is not known.

- *Slow Virus Theory*
    - Because a slow-acting virus has been identified as a cause of some brain disorders that closely resemble Alzheimer's disease (for example, Creutzfeldt-Jakob disease – mad cow disease), some researchers have suggested that a virus may cause suspicious brain tissue changes in AD victims as well. However, to date a virus has not been isolated from the brains of those with Alzheimer's disease, and no immune reaction has been

found in the brains of Alzheimer's patients comparable to that found in patients with other viral dementias.

- *Blood Vessel Theory*
  - Potential defects in the blood-brain barrier, a protective membrane-like mechanism that guards the brain from foreign bodies or toxic agents circulating in the blood stream outside the brain, have been proposed as a possible cause of AD. There have been several reports of a possible association between serious head injuries involving a loss of consciousness and later onset of Alzheimer's disease. One theory as to why this connection might occur has to do with possible breaks in the blood-brain barrier as a result of these injuries to the brain.

Risk Factors

Although no definite cause of AD has been identified, a number of risk factors have been associated with a greater risk of developing the disease:

- History of head trauma
- Heredity
- Slow acting virus
- Environmental toxins
- Mitochondrial genetic defect
- Decreased blood flow to the brain (inadequate oxygen and glucose delivery)

Diagnosis

In the early stages of AD, some of the changes may be very subtle and we often do not recognize the problems immediately. Some of the symptoms can include:

- Repeating statements frequently
- Frequently misplacing items
- Trouble finding names for familiar objects
- Getting lost on familiar routes
- Personality changes
- Becoming passive and losing interest in things previously enjoyed

As the disease progresses, other symptoms may include:

- A decrease in knowledge of recent events
- Forgetting events in their life history, essentially losing awareness of who they are
- Problems choosing proper clothing
- Hallucinations, arguments, striking out and violent behaviour
- Delusions, depression, agitation

The average length of the disease is between eight and 12 years, but the progression of Alzheimer Disease varies from person to person and can span three to 20 years. The progression can be classified into seven stages (*Global Deterioration Scale*):

- Stage 1: No cognitive decline
  - Experiences no problems in daily living.
- Stage 2: Very mild cognitive decline
  - Forgets names and locations of objects.
  - May have trouble finding words.
- Stage 3: Mild cognitive decline
  - Has difficulty travelling to new locations.
  - Has difficulty handling problems at work.
- Stage 4: Moderate cognitive decline
  - Has difficulty with complex tasks (finances, shopping, planning dinner for guests).
- Stage 5: Moderately severe cognitive decline
  - Needs help to choose clothing.
  - Needs prompting to bathe.
- Stage 6: Severe cognitive decline
  - Needs help putting on clothing.
  - Requires assistance bathing; may have a fear of bathing.
  - Has decreased ability to use the toilet, or is incontinent.
- Stage 7: Very severe cognitive decline
  - Vocabulary becomes limited, eventually declining to single words.
  - Loses ability to walk and sit.
  - Becomes unable to smile.

There is no single diagnostic test that can detect if a person has Alzheimer's disease. Standard clinical methods combine physical and neuropsychological testing with caregiver input and the physician's judgment. A large part of the diagnosis remains the *exclusion* of other illnesses that may cause intellectual impairment (brain tumours, thyroid problems, etc.) where the evaluator has come to the conclusion that symptoms are most likely the result of Alzheimer's disease: ***probable diagnosis***. Current clinical diagnostic tools can accurately diagnose AD in about 90% of cases. A typical battery of tests during the diagnostic process may include:

- *Medical history*: information about current mental or physical conditions, prescription drug intake, and family health history.
- *Mental status evaluation*: assesses sense of time and place and ability to remember, understand, communicate, and do simple calculations.
- *Physical examination*: includes the evaluation of nutritional status, blood pressure, and pulse.
- *Neurological examination*: tests the nervous system (brain and spinal cord) for evidence of other neurological disorders. A magnetic resonance imaging (MRI) study of the brain is used to search for other possible causes of dementia (e.g., stroke).

- *Laboratory tests*: blood and urine tests provide additional information about problems other than Alzheimer's that may be causing dementia.
- *Neuropsychological evaluations*: test memory, reasoning, vision-motor coordination, and language function. May provide the only evidence of dementia, especially in the early stages.
- *Psychiatric evaluation*: provides an assessment of mood and other emotional factors that could mimic dementia or may accompany Alzheimer's disease.

AD is associated with changes in neurotransmitters (i.e. loss of acetylcholine and serotonin) and structural changes within the brain. A ***definite diagnosis*** of AD can only be done through an autopsy at the time of death. During the autopsy, three distinct structural abnormalities are observed in AD: i) loss of neurons, ii) amyloid plaques, and iii) neurofibrillary tangles.

i) A ***loss of neurons*** is observed in the cerebral cortex, which is involved in reasoning, memory, language and other important thought processes, as well as in the hippocampus, an important centre for memory. It was observed that many of these neurons were *cholinergic* (they communicate using the neurotransmitter acetylcholine). Some drug therapies that inhibit the degradation of acetylcholine (*acetylcholinesterase inhibitors*) have some success in slowing the progression of AD (i.e. Cognex).

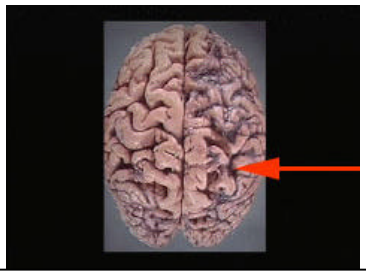


Figure 2: Prominent atrophy of the cerebral cortex and the widened sulci (arrow).

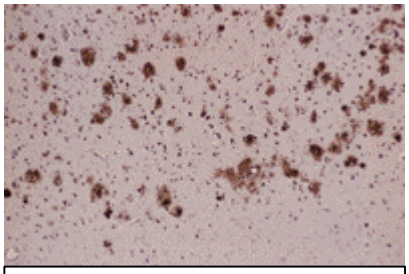


Figure 3: Light microscope picture of amyloid plaques

ii) ***Extracellular neuritic plaques*** result from the degeneration of axons and axon terminals as well as the fibrils of a protein called beta amyloid. These plaques have been observed in the cerebral cortex, hippocampus, basal ganglia, thalamus, and cerebellum. *Beta amyloid* is a protein fragment of a membrane *glycoprotein* (carbohydrate linked to protein; part of cell membranes) called *beta amyloid precursor protein* (bAPP). In a healthy brain, these protein fragments would be broken down and eliminated. In Alzheimer's disease, the fragments accumulate to form hard, insoluble plaques. The protein fragments disrupt sodium, potassium, and calcium channel function, and this can result in hydrogen peroxide accumulation (strong oxidizing agent) and increased free radical formation (highly reactive, disrupts membrane structures) that ultimately lead to cell destruction. The immune system's inflammation response to the cell damage may also further complicate matters by increasing the levels of glutamate, an excitatory neurotransmitter also released in response to injury, that can be toxic to nerve cells in large quantities.

There is a strong link between gene mutations on chromosomes 1, 14, and 21 and *early-onset AD* (onset before the age of 60-65; ± 10 % of AD cases). A gene on chromosome 21 codes bAPP, and genes on chromosomes 1 and 14 code for *presenilins* 1 and 2. Mutations in bAPP and presenelins modify the activity of *secretase* enzymes that process the cleavage of beta amyloid from bAPP.

Late-onset AD (after age 65) has been linked to mutations in the gene coding for Apolipoprotein E (apoE), a protein that helps transport cholesterol in the blood. Cholesterol is involved in the repair of nerve cells during development and after injury. The gene that code for this protein is found on chromosome 19 and has three alleles (apoE2, apoE3, apoE4). ApoE4 is linked to a greater incidence of AD, and may lack the ability to effectively remove beta amyloid as well as apoE2 and apoE3. The impact of the genetic component of late-onset AD varies greatly, and estimated can vary anywhere from 1-40 %.

iii) ***Intracellular neurofibrillary tangles*** (NFT) are abnormal bundles, resembling pairs of threads wound around each other in a helix, of various protein filaments inside the cells in affected brain regions. It has been observed that the density of neurofibrillary tangles in AD is related to the severity of the dementia. Tangles contain A68 protein (also found in older Down syndrome patients). A68 is thought to be an altered version of *tau*, and may be involved in neuron death. Tau is a protein normally associated with the microtubules of the *cytoskeleton*. The *cytoskeleton* is a series of rods that provide cellular structure and movement. There are three types of rods:

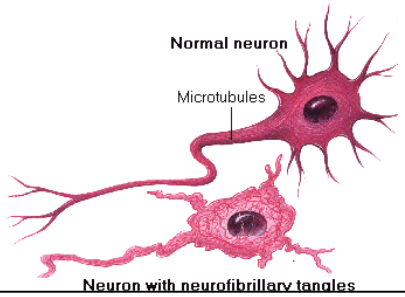


Figure 4: Neurofibrillary tangles

1. *Microtubules*: cylindrical, made of tubulin, support the cell and give it shape, involved in intracellular and cellular movement, form centrioles.
2. *Microfilaments*: fine filaments of the movement associated protein actin.
3. *Intermediate filaments*: various stable proteins that resist mechanical forces acting on the cell.

Tau is involved in the polymerisation of tubulin and stabilizes the microtubules. Disruption of the function of the microtubules would contribute to the destruction of nerve cells.

Treatment

The *progression* of AD is variable (2-20 years), and can be influenced by such factors as age of onset, environment, and concomitant pathologies (strokes, Parkinson's, etc.). Although there is presently no known cure, proper *management* may slow down the progression of the disease. Proper management strategies include:

- Control of medication

- Maintenance of normal wake/sleep cycles
- Insured adequate nutrition
- Optimization of physical health
- Minimization of social isolation.

Drug therapy in AD seeks to improve memory (cholinergic drugs) and slow the progression (nerve growth factor, calcium channel blockers) of the disease. Some of the more common medications include:

- *Tacrine (Cognex).* Tacrine can improve mental abilities in about 30 percent of people with mild to moderate Alzheimer's disease by slowing the breakdown of neurotransmitters in the brain. However, the drug has been linked to liver complications.

- *Donepezil (Aricept).* This medication also decreases mild to moderate symptoms of Alzheimer's by improving levels of neurotransmitters in the brain. Its side effects, which include nausea, diarrhoea and fatigue, are usually mild and don't last long.

- *Rivastigamine (Exelon).* Like tacrine and donepezil, rivastigamine blocks the breakdown of neurotransmitters in the brain, lessening symptoms. Side effects may include nausea and vomiting.

- *Other*. Medications that seek to improve behavioural symptoms that often accompany Alzheimer's are also often prescribed. These symptoms include sleeplessness, wandering, anxiety, agitation and depression.

End-of-Life Care[3]

Although often a difficult subject to discuss, end-of-life care is an important consideration for all seniors, not just those with dementia. Health Canada funded a *Guide to End-of-Life Care for Seniors* to address the unique issues facing seniors. The following are excerpts from this guide.

Terminology

End-of-life care: "End-of-life care for seniors requires an active, compassionate approach that treats, comforts and supports older individuals who are living with, or dying from, progressive or chronic life-threatening conditions. Such care is sensitive to personal, cultural and spiritual values, beliefs and practices and encompasses support for families and friends up to and including the period of bereavement."

Palliative care: "a special kind of health care for individuals and families who are living with a life threatening illness that is usually at an advanced stage. The goal of palliative care is comfort and dignity for the person living with the illness as well as the best quality of life for both this person and his or her family. A "family" is whoever the person says his or family is. It may include relatives, partners and friends. An important objective of palliative care is relief of pain and other symptoms. Palliative care is planned to meet not only physical needs but also the psychological, social, cultural, emotional and spiritual needs of each person and family. Palliative care may be the main focus of care when a cure for the illness is no longer possible. Palliative care services help people in later life who are ill to live out the remaining time in comfort and dignity."

Geriatric care: "typically viewed as the care of persons who are aged 65 and over. Geriatric care encompasses a wide range of treatment from intensive care to palliative care. It can work together with palliative care to meet the needs specific to the senior. Such care, which is proper and appropriate for older people who are dying, includes symptom control, care within seniors' facilities, continuity of care, flexibility and multidisciplinary teamwork. Today, however, many palliative care programs are delivering care which encompasses geriatric care by using an interdisciplinary approach." Many seniors may not have a health care and/or service provider who are specialized in geriatric care.

Quality of Life

Quality of care at the end of life mean ensuring that the right care is being provided at the right time in the right way. Problems in the following areas can arise:

- Overuse of care
- Under use of care
- Poor interpersonal performance

[3] Source: A Guide to End-of-Life Care for Seniors

For improving end-of-life care by health care and social service providers at the bedside, three main elements should be included:

- Comfort: control of pain and other symptoms. No senior should die in pain or with the other treatable symptoms.
- Decision-making: the use of life-sustaining treatments. The older person and family should be able to choose the care the individual will receive in the last days of life.
- Support: of those who are dying and their families. Support needs are unique to the senior and family and can be met by an interdisciplinary health care team.

Dying well

Seniors' abilities to age well are partly shaped by how they experience earlier stages of life. The prime consideration in measuring if someone is dying well is whether the person is dying in the way he/she prefers. Individual preferences are shaped by:

- Personality
- Gender
- Ethnic background
- Social class
- Culture
- Spiritual needs

A good death may include such elements as:

- Being pain-free
- Operating at the highest possible level of functioning
- Resolving long-standing conflicts
- Satisfying final wishes

Symptom Management

Many seniors have multiple chronic disease states, which can cause numerous distressing physical symptoms. A symptom is a physical or mental phenomenon, circumstance or change of condition arising from and accompanying a disorder. It is a subjective indicator as perceived by the individual, and patient self-report must be the primary source of information. Symptom assessments should occur at regular intervals, and consists of the following steps:

- Identify the cause of the symptom.
- Measure the quality and intensity of the symptom.
- Recognize and assess the multi-dimensional aspects of the symptom:
    - Physical: including impact on functional ability.

- Psychosocial (psychological distress, use of alcohol and other non-medical drugs, cognitive status, previous history of depression, coping patterns with previous life stressors).
- Cultural.
- Spiritual.

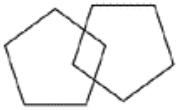
Some of the more common symptoms experiences by patients with progressive illnesses include:

- Pain
- Dyspnoea (shortness of breath)
- Confusion/delirium
- Anorexia (decreased or complete loss of appetite)
- Depression
- Constipation
- Nausea/vomiting
- Anxiety
- Pressure sores

Symptoms can be managed with appropriate use of medications and non-pharmacological interventions as well as complimentary therapies.

Numerous assessment tools are available. For example, the *Mini-Mental State Questionnaire* is a tool that is commonly used by various health care professionals to test for possible dementia. There are questions designed to test orientation, registration, attention, calculation, recall, language and spatial skills.

	Score	Points
Orientation		
1. What is the		
Year?	_____	1
Season?	_____	1
Date?	_____	1
Day?	_____	1
Month?	_____	1
2. Where are we		
State?	_____	1
County?	_____	1
Town/city?	_____	1
Floor?	_____	1
Address/name of building?	_____	1
Registration		
3. Name three objects, taking one second to say each. Then ask the patient all three after you have said them. Repeat the answers until the patient learns all three.	_____	3
Attention and Calculation		
4. Ask for serial sevens. Give one point for each correct answer. Stop after five answers. Alternative: Spell *world* backward.	_____	5
Recall		
5. Ask for names of three objects learned in question 3. Give one point for each correct answer.	_____	3

	Score	Points
Language		
6. Point to a pencil and a watch. Have the patient name them as you point.	_____	2
7. Have the patient repeat "No ifs, ands, or buts."	_____	1
8. Have the patient follow a three-stage command: "Take the paper in your right hand. Fold the paper in half. Put the paper on the floor."	_____	3
9. Have the patient read and obey the following: "Close your eyes."	_____	1
10. Have the patient write a sentence of his or her own choice. (The sentence should contain a subject and an object and should make sense. Ignore spelling errors when scoring.)	_____	1
11. Enlarge the design printed below to 1 to 5 cm per side and have the patient copy it. (Give one point if all the sides and angles are preserved and if the intersecting sides form a quadrangle.)	_____	1
		
Total	_____	30

Bereavement

Three closely related components make up the bereavement process: loss, grief and recovery.

- *Loss*: the separation from a part of one's life to which one was emotionally attached.

- *Grief*: the complex emotional, cognitive and perceptual reactions that accompany loss. It involves the painful separation from someone or something we have loved very much, and it hurts deeply. Grief may occur before death as well as after death.

- *Recovery*: the final component is surviving the death of a loved one and finding meaning and purpose in life after the death. While time may help to heal the wounds left by death, in recovery what really counts are the circumstances with which survivors must deal as time goes by: the nature of the losses one has experienced, the social and economic resources upon which one can call, and the ability to reconstruct one's life.

Practice Guidelines

Here is a brief summary of some practical guidelines for the health care professional dealing with end-of-life care:

- Respect seniors as unique individuals with particular beliefs, values and preferences.
- Facilitate open and timely discussion of decisions related to treatment, care, and planning for families, preparing wills and other issues of significance to seniors.
- Assess symptoms in a systematic and multi-dimensional fashion.
- Recognize that seniors may describe symptoms in a non-specific manner.
- Respond sensitively to seniors' pain, fears and anxieties.
- Observe for, and respond to, signs of burnout in family caregivers, including emotional distress, fatigue, depression, work-home conflict, and financial difficulties.
- Facilitate the family's coming to terms with the impending death.
- Extend support into the period of bereavement.

Conclusion

Dementia is a difficult time for both the patient and caregiver. As the population ages, health care professionals in numerous disciplines will be asked to deal with the unique challenges of this condition.

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