

CHAPTER 2

Literature review

2.1 INTRODUCTION

This chapter discusses the literature review conducted on nurses' views on pain, dementia and nursing patients with dementia. The purpose was to familiarise the researcher with research on the topic and to situate the current study in the context of what is known about the phenomenon under study. Polit et al (2001:121) state that a literature review provides a background for research.

The literature review covered definitions of pain; pain transmission; the pathophysiology of pain, which can be classified as nociceptive, neuropathic and idiopathic processes; changes in patients' in behaviour or feelings; the nurse's role in the assessment of pain; strategies that lead to the provision of treatment; misconceptions held by both patients and nurses that may lead to under-treatment of pain, and lastly, different types of dementia such as Alzheimer's disease (AD), Vascular, Lewy body and Pick's disease.

2.2 PAIN

There are various definitions of pain. McManus (2003b:502) defines pain as the sensation of acute physical hurt or discomfort caused by, amongst others, injury, illness, emotional suffering or mental distress. Briggs (2002:23) cites Merksy's (1986) definition of pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or discussed in terms of that damage". Andrews and Boyle (1999:283) refer to Morris' (1991) definition as "a sensation associated with real or potential tissue damage involving chemical disturbances along the neurological pathways. Pain is a private as well as personal experience and is influenced by cultural heritage. Culture has been recognised in nursing practice and research as a factor that influences a person's expression and reaction to pain."

Briggs (2002:23) states that pain does not occur in isolation but in a specific human being in psychological, economic and cultural context.

According to Griffith (2002:18), pain is “a complex phenomenon originating as sensory stimuli, which can be modified by individual emotions, expectations or memories”. The elderly may minimise the severity of the pain thus leading to under-diagnosis of potentially serious health conditions. The ability to manage pain effectively requires an understanding of normal pain mechanism.

2.2.1 Normal pain mechanism

According to the American Medical Association (AMA) (2004:7), the physiology of normal transmission involves basic concepts that are necessary to understand the pathophysiology of abnormal pain. These include the concepts of transduction of the first-order afferent neuron nociceptors. “(The nociceptive neuron has specific receptors that respond to specific stimuli if a certain amount of stimulus is applied to the peripheral receptors. If sufficient stimulation of the receptor occurs, the nociceptor neuron becomes depolarised.)”. Figure 2.1 depicts the normal pain mechanism.

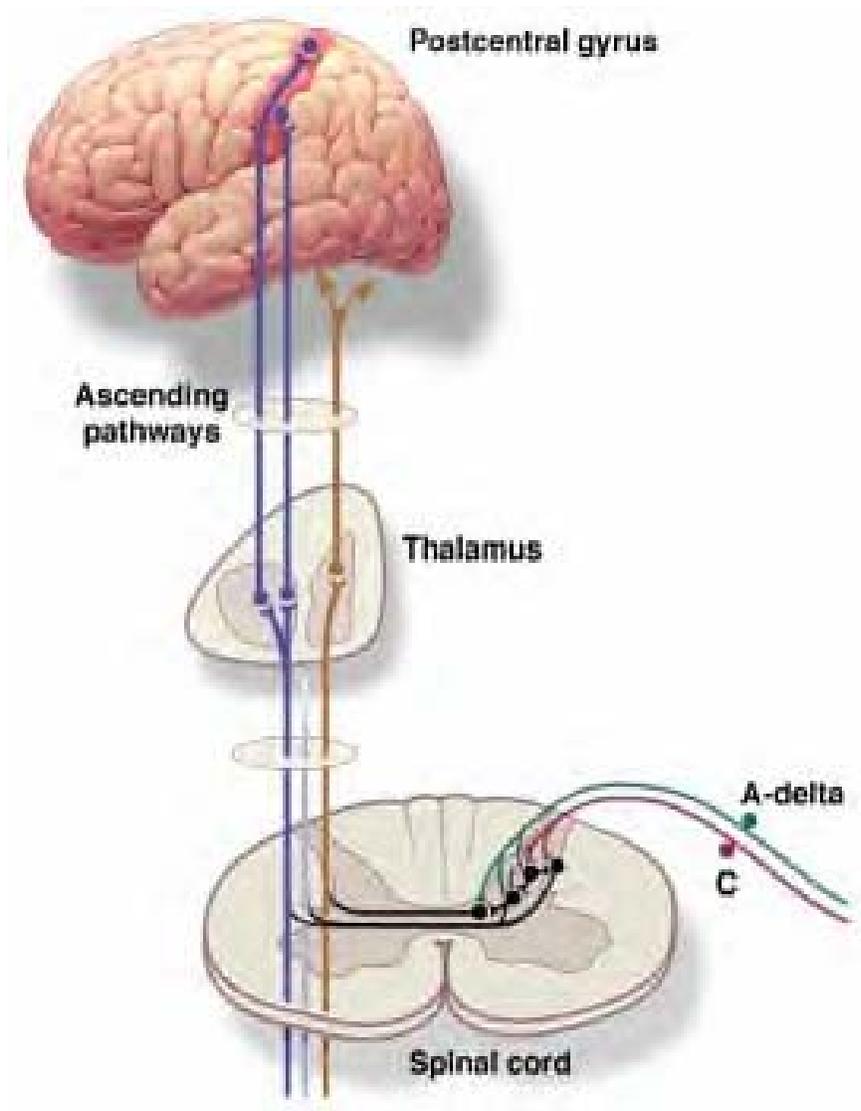


Figure 2.1
Normal pain mechanism

Source: American Medical Association (2004:1)

The nociceptor axon carries the impulse from the periphery to the dorsal horn of the spinal cord to make connection directly and indirectly through the spinal interneurons, with the second-order afferent neuron in the spinal cord.

The second-order neurons transmit impulses from the spinal cord to the brain. They ascend mostly via spinothalamic tract up the spinal cord and terminate in higher neural structures, including the thalamus of the brain.

Third-order neurons originate from the thalamus and transmit signals to the cerebral cortex.

There is evidence that numerous supraspinal control areas modulate pain. These include the reticular formation, midbrain, thalamus, hypothalamus, limbic system of the amygdala and cingulate cortex, basal ganglia and the cerebral cortex. Neurons originating from these cerebral areas synapse with neurons of the descending spinal pathways which terminate in the dorsal horn of the spinal cord (AMA 2004: 9).

2.2.2 Pathophysiology of pain

According to the AMA (2004:1), the pathophysiology of pain can be classified into nociceptive, neuropathic or idiopathic processes. The processes are complex and represent the interplay of a number of underlying mechanisms.

2.2.2.1 Nociceptive pain mechanism

The AMA (2004:1) cites Loeser's (2000) definition of nociception as "the detection of tissue damage by specialised transducers connected to the A-delta and C fibres".

Anatomically, this process occurs in the periphery and involves nociceptor neurons terminating in the dorsal horn of the spinal cord. Nociceptors are afferent neurons that have specific nerve endings able to distinguish noxious (thermal, chemical and mechanical) and innocuous events.

The International Association for the Study of Pain (IASP) in AMA (2004:3) describes pain as the "perception of a noxious stimulus that begins in the dorsal horn of the spinal cord and involves the entire spinal cord and brain".

Pain is described in terms of sensory, emotional and cognitive components. These three

components are reflected in the transmission and modulation of painful stimuli. Such mechanisms are intervened through the nociceptor neurons, the spinal cord process and the cerebral process (AMA 2004:3). Clinically, pain is termed nociceptive when it is sustained primarily by the nociceptive system. Nociceptive pain is the pain that is proportionate to the actual tissue damage. A more severe injury leads to pain that is perceived to be greater than that caused by a less severe injury. Such pain acts as a protective function. When sensing a noxious stimulus, a person behaves in a manner that will reduce pain and promote healing such as, pulling finger away from a hot object. Examples of nociceptive pain include burns and bone fractures (AMA 2004:4).

2.2.2.2 Neuropathic pain

According to the AMA (2004:5), neuropathic pain occurs through changes in the central nervous system, such as the process of “wind up” phenomenon and central sensitisation that occurs in patients with prolonged exposure to nerve injury or noxious stimuli or through peripheral changes such as neuroma formation. Pain is disproportionate to the degree of tissue damage. It can occur without nociception.

Pathologic pain can occur independently of the causative event. It serves no protective function. Examples include painful diabetic and other peripheral neuropathies such as laceration, compression as well as nerve inflammation (AMA 2004:6).

A patient may experience both nociceptive and neuropathic pain simultaneously, such as patients who suffer from leg and back pain following Lumbar-spine surgery (failed low-back- surgery syndrome) (AMA 2004:6). Some patients with complex regional pain syndrome (reflex sympathetic dystrophy or causalgia) may develop painful complications that are nociceptive, such as joint ankylosis, that coexist with underlying pain underlying neuropathic pain (AMA 2004:6).

2.2.2.3 Idiopathic pain

According to the AMA (2004:7), idiopathic pain is “pain that persists without any identifiable organic lesion or that is disproportionate to the degree of tissue damage”.

2.2.3 Pain modulation

AMA (2004:10) found that pain modulation is influenced by the endorphinergic system and other pain modulation systems. Exactly how this complex neural system works is not understood, but the principal mediation or modulation is endorphins (endogenous opioid compounds). In the endorphinergic system analgesia occurs due to the binding of the endorphins to subsets of receptors: mu, delta and kappa. Herr (2002:23) adds that endorphins are widely distributed and closely related to the system that regulate homeostasis, respond to pain and stress. Other neurotransmitters, such as serotonin or norepinephrine, also play a role in the endogenous pain modulating system.

Researchers at the University of Toronto discovered a gene involved in pain modulation that could lead to pain control (Easton 2002:2). In their study, genetically engineered mice lacking a gene called DREAM (downstream regulatory element antagonistic modulator) showed a dramatic loss of pain sensitivity compared to mice that had the DREAM gene. DREAM produces a protein that suppresses the genetic machinery that reads the DNA (deoxyribonucleic acid) code for dynorphin. Dynorphin, known as endorphin, is produced in the body in response to pain or stress. When the DREAM gene was absent in mice, the researchers discovered an increased production of dynorphin in the region of the spinal cord involved in transmitting and controlling pain messages.

People with well-managed pain have improved quality of life. They are likely to sleep better and have energy during the day. Being active reduces the complications of immobility such as pneumonia and thrombosis (Andrews & Boyle 1999:283).

2.2.4 Pain perception

Gaston-Johansson, Johansson and Johansson (1999:190) state that pain perception refers to whether individuals experience any change in their behaviour or feelings. It depends on complex interaction between nociceptive and non-nociceptive impulse transmission ascending in relation to the activation of descending pain-inhibiting systems in the nervous system.

Pain may cause people to cut back on their activities and social interactions, which can lead to loss of physical stamina and increased isolation. This combination of events can lead to depression. A vicious circle results when physical, social and emotional deterioration leads to more pain (Andrews & Boyle 1999:268).

Feldt (2001: 13) asserts that everyone has a unique response to pain. Something that is unbearably painful to one person might be only mildly painful to another. Attention to pain may also influence the opinion of that pain.

2.2.5 Nurse's role in the assessment of pain

Gaston-Johansson et al (1999:190) emphasises that pain assessment includes identifying the psychosocial factors that contributes to an individual's pain experience and that form motivational strategies for treatment. Horgas (2003:3) posits that there is neither a blood test for pain nor a scientific method for pain measurement. However, there are methods to assist in determining the cause of pain. Electromyography (thin needles inserted into the affected muscle or nerve); nerve conduction studies (electrodes that record electrical responses to identify nerve damage); X-rays, neurological examination and magnetic resonance.

The assessment of pain in the elderly may at times be different because of decreased cognitive capacity, illness, disease and disability. A decreased cognitive capacity may prevent the use of pain assessment tools leaving observation of the patient's bio-behavioural responses, a less accurate method of assessing pain, as the only procedure available for estimating pain intensity (Gaston-Johansson et al 1999:191). According to Middleton (2004:42), nurses often distrust the patient's self-report of pain because they

have their own benchmark of what is an acceptable pain level warranting intervention.

Behaviour is more related to pain than dementia. Bonifazi (2002:1) cites Furlow's three pain assessment sides, determined by patients' cognitive status: "You have to be very sensitive to the patient's look, breathing, the way the patient holds him/herself, rests or moves."

Andrews and Boyle (1999:295) explain three major objectives of pain. First, it allows the nurse to understand what the patient is experiencing. Second, it evaluates the impact the pain has on the patient. Third, it allows the nurse to determine the cause of pain. The first two objectives are described below.

2.2.5.1 Understanding the experience

For the nurse to understand what the patient is experiencing, it is vital to seek information regarding location, duration, intensity and the quality, onset and frequency as well as factors that relieve or exacerbate the pain. The nurse and client need to communicate about the pain experience.

Pain intensity can be assessed using assessment tools such as a 10-point scale, with zero representing no pain and 10 representing severe pain. Other assessment tools include a pictorial assessment scale and a visual analogy scale (Hiskisson 1974:1127-1131; Lynch 1999:388).

Pain expression varies among clients. Variations must also be acknowledged within cultures. It also varies among the same clients in different situations. For instance, clients experiencing chronic pain may display nonverbal behaviour in relation to the pain they feel other than clients experiencing acute pain (Andrews & Boyle 1999:297). Prkachin and Craig (1995) (cited in Biersdorff 2000:1) found that "most of what is known about experience comes from studying what people communicate about their own pain and their interpretation of others' pain".

2.2.5.2 Evaluating the effect

The most difficult aspect of pain assessment is evaluating what effect pain has on the patient, such as poor quality of sleep or loss of appetite. The patient should be allowed to express the effect of pain without the nurse imposing. Assessment of actual responses to pain should include gathering data on what a particular behaviour means. A baseline understanding of the patient's response to pain is essential. However, assessing pain in elderly clients with dementia poses a challenge due to the biological and psychological factors that influence their perception and experience of pain (Andrews & Boyle 1999:297).

The single most reliable indicator of pain existence and intensity is self-report. In practice, however, nurses tend to rely on factors such as facial expression, position and request for relief as useful in assessing pain (Andrews & Boyle 1999:297).

2.2.5.3 Strategies for pain assessment

According to Horgas and McLennon (2002:1), the following strategies aid in the assessment of pain in elderly patients:

- Review medical history, physical examinations and laboratory and diagnostic tests in order to understand sequence of events leading to pain.
- Assess pain regularly and frequently, at least every four hours. Pain intensity to be monitored after giving analgesia to evaluate effectiveness.
- Review medications, including current and previously used prescribed drugs, over-the-counter drugs, and home remedies. Previously effective pain control methods used by the patient should be determined.
- Assess the patient's attitudes and beliefs about the use of analgesia (Paracetamol), anxiolytics (propranolol), and nonpharmacological treatments (massage).

- Gather information from family members about patient's pain experiences. Ask about patient's verbal and nonverbal behaviour, particularly in patients who suffer from dementia.
- Use a standardised tool to assess self- reported pain. Choose from published measurement tools. The elderly and patients who suffer from dementia have difficulty using 10-point visual analogy scales; therefore faces scales may be useful with these patients. Figure 2.2 depicts a visual analogue scale.

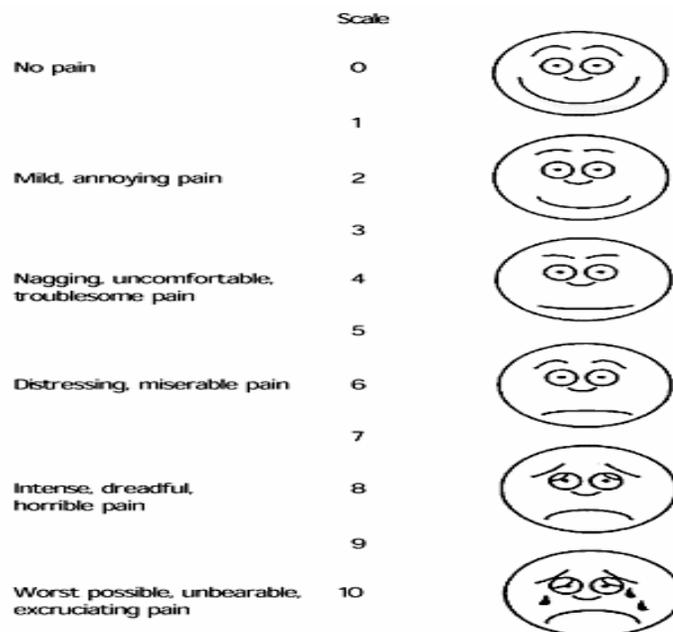


Figure 2.2
Visual analogue scale

Source: Horgas and McLennon (2002:2)

2.2.6 Causes of under-treatment of pain

Pain is subjective in nature. It is what patients state themselves to be experiencing at that particular time. However, due to numerous factors, such as language and cultural influence as well as misconceptions regarding pain, not all patients are able to report pain

experience (Huffman & Kunik 2000: 578). Both patients and nurses have misconceptions about pain.

2.2.6.1 *Misconceptions held by elderly patients*

According to Acello (2001:474), the elderly have misconceptions about pain such as that pain is the natural outcome of growing old or that pain sensation and experience decreases with age. Other misconceptions about pain held by elderly people include that acknowledging pain denotes personal weakness; chronic pain is punishment for the past; pain is an inevitable part of ageing; pain means death is near; serious disease will result in the need for expensive tests and/or the loss of independence.

2.2.6.2 *Misconceptions held by nurses*

Horgas (2003:2) list several misconceptions among nurses about pain and pain in patients who suffer from dementia, such as the following:

- Persons who do not complain of pain do not experience pain.
- Medications that treat pain have side effects that make them dangerous to use in the elderly, and if treated, patients will become addicted to the medication.
- However, if the medications are resumed in lower doses and adjusted as the pain progresses, the chances of addiction and harmful side effects can be avoided or lessened.

McManus (2003:2) states that there is no clinical evidence to confirm that the pain perception threshold is diminished as part of the aging problem. Attempts to help may cause anger and frustration for both clients and nurses. Health care professionals often fail to provide the best possible pain management and elderly patients are often at high risk of having their pain overlooked due to the beliefs or misconceptions held regarding pain.

Suffering may be prevalent in residents with dementia as self-report of pain is less; therefore patient behaviour may be the main indicator of distress. Pain prevalence in the elderly can be as high as 85% (Hicks 2000:5).

Pain is the most common reason for visits to physicians, health care clinics and pharmacies. Pain prevalence in dementia can impair the ability to function because of the effect on the physical and psychological aspects of health. Pain often reduces level of activity, comfort and quality of life. As pain is subjective, physician and nurses rely on self-report before evaluating and managing it. Herr and Mobily (1991) (cited in Briggs 2002:49) maintain that if older people are offered privacy to discuss their pain a more reliable pain assessment can be obtained.

Feldt (2002) (cited in Bonifazi 2002:1) asserts that pain assessment in residents with late stage dementia is more difficult than in less demented. Pain assessment in the more impaired should be made during movement or palpation of the suspected area when a patient is likely to perceive pain and react. Non-verbal vocalisation of pain includes holding or clutching furniture, clenched teeth, dropped jaw or distorted expression, screaming, grimacing, lack of activity or withdrawal.

The ability to identify the feelings of someone with dementia is significant to understanding the person and their behaviour. Loeser and Melzack (1999:1607) state that behaviours are more related to pain than dementia. Furlow (cited in Bonifazi 2002:1) emphasises that nurses need to be sensitive to patients' breathing, how they hold themselves, rest and move. Pain assessment should include family members as they have knowledge regarding the patient's history of pain, cultural meaning of pain as well as medication taken to alleviate pain. According to Eastman (2001:1), neurological assessment may shed light on the functioning of the pain-related areas. Tools are being developed to assess pain in cognitively impaired elderly patients as reliably as possible.

Gagicse and Melzack (1997:5) found that a significant majority of people who suffer from dementia experience pain which interferes with their normal function, but do not receive adequate pain management. Three factors contribute to this, namely lack of proper pain assessment, potential risks of pharmacology as well as the efficacy of non-pharmacological pain management, and the attitude of the elderly towards such treatment.

Koval (1997) (cited in Eastman 2001:1) asserts that "in some cases discomfort can be

resolved by meeting the patient's basic needs". The nurse who spends most time with the patient will be able to identify and resolve unmet needs immediately by assessing the following: the need for a working hearing aid, spectacles; need to use the toilet; a change in the weather, or physical discomfort due to infection, inflammation, acute or chronic illness. Challenging behaviour and behavioural disturbances often occur as a result of ineffective communication as well as unmet needs (Bush 2003:42).

A late stage dementia patient may have difficulty in detecting discomfort due to such disease manifestation such as inability to communicate, difficulty in eating and swallowing, reduced mobility, increasing need for help with personal help as well as increased susceptibility to infections and pneumonia (Sachdev, Brodaty & Looi 1999: 83).

2.3 DEMENTIA

Fares (1997:49) defines dementia as a set of symptoms where there is a decline in memory and capacity to solve problems of daily living, performance of learned perception and motor skills as well as the control of emotional reactions. Dementia is characterised by changing patterns of behaviour, disorientation to time, place and person, inappropriate responses and actions such as decision making, expressing thoughts or understanding what people are saying, repeating the same questions, becoming lost in familiar places, and inability to follow directions. Abilities are lost at different rates.

Dementia is caused by many conditions, some of which are reversible whilst others are not. Reversible conditions may be caused by fever, dehydration, vitamin deficiency and poor nutrition, minor head injury, adverse reaction to medications or problems with the thyroid gland (Melding 2002:2). Different types of dementia include Alzheimer's Disease (AD), Vascular dementia, Lewy body dementia (LBD) and Pick's disease.

According to Yanagihara (2000: 26), dementia affects different areas of the brain, therefore producing different symptoms. For instance the parietal lobe affects perception as in Pick's disease, frontal lobe affects movement, emotional behaviour, personality and speaking,

such as in AD, and the temporal lobe affects long term memory, smell and hearing as in vascular dementia, as reflected in figure 2.3.

2.3.1 Alzheimer's disease (AD)

According to Fares (1997:2), AD and Multi infarct dementia (vascular dementia) are two forms of irreversible dementia. AD is the most common disease and accounts for about fifty percent of all cases. AD was named after Alois Alzheimer; a German neurologist, who described the disease in 1906. He described the disease after the death of a 51-year woman from a mysterious progressive disease. The post-mortem revealed abnormal clumps (now called neuritic plaques) and tangled bundles of fibres (known as neurofibrillary tangles). Plaques are extracellular structures consisting of swollen degenerating nerve tissues. Tangles are intracellular structures, which consist of bundles of abnormal fibre. Kornstein (2002:1) adds that these plaques and tangles are the hallmarks of AD. Figure 2.3 depicts the anatomy of the brain and figure 2.4 represents a cross-section of a normal brain and one with AD.

According to Barrett (2004:1), from the First World War until 1970 there was no research into AD. Research into AD commenced in 1980s when the condition became familiar and the funding for biomedical research into brain disorders was granted. Whaley (cited in Reger 2002:1), states that before the discovery of AD in 1907, both scientists and the non-science community viewed dementia as “natural” and “senility was seen as part of aging”.

In AD, the nerve cell in certain parts of the brain undergoes change resulting in the death of large number of cells. AD has a short insidious beginning which progresses gradually. Progression of the disease results in mild forgetfulness to serious impairments in thinking, judgement and ability to perform activities of daily living (Fares 1996:2).

The American Association for Geriatric Psychiatry (2003:2) posits that scientists found brain changes in people who suffer from AD. Nerve cells die in areas of the brain that are vital to memory and other mental abilities. There are also lower levels of chemicals in the brain that transmit messages between the nerve cells. AD may impair thinking and memory

by disrupting these messages.

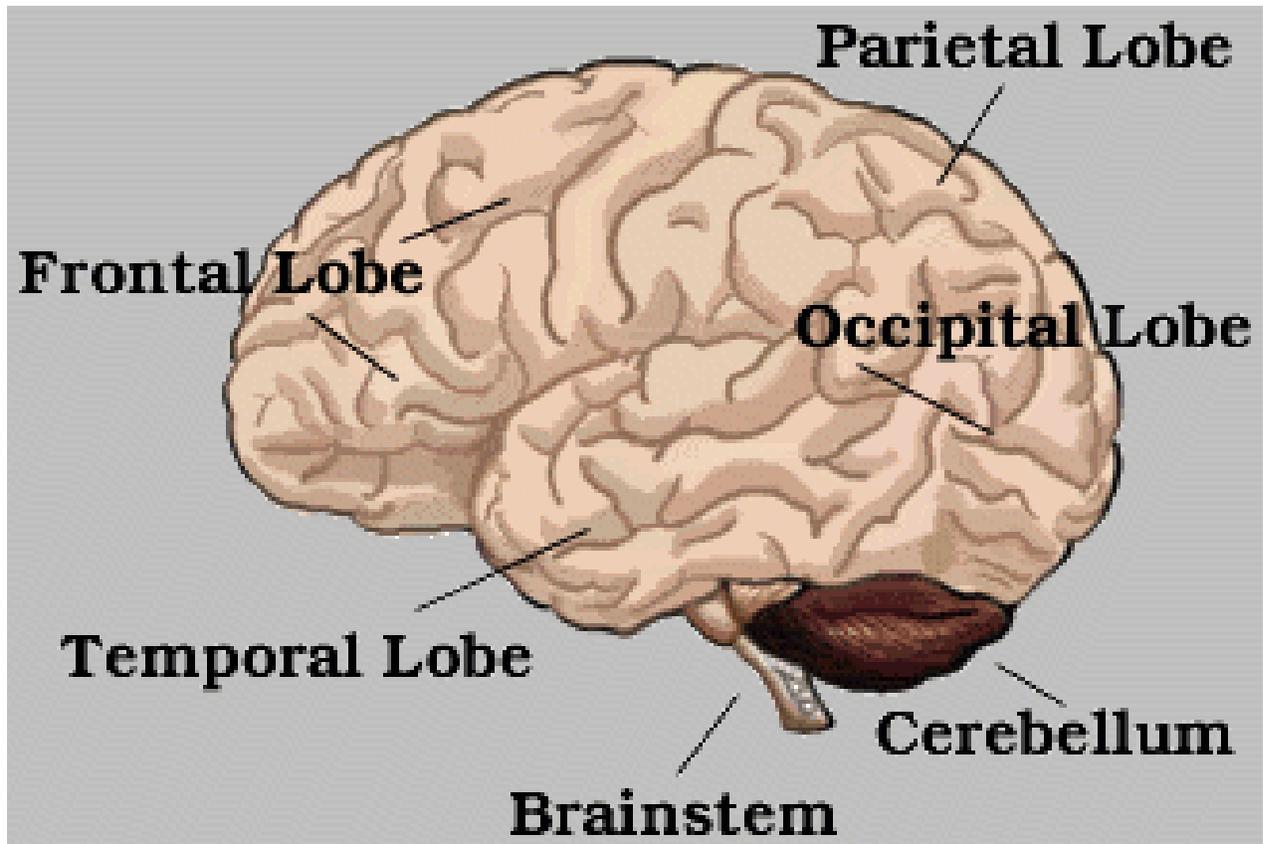


Figure 2.3
Anatomy of the brain

Source: Johnson (2004:1)

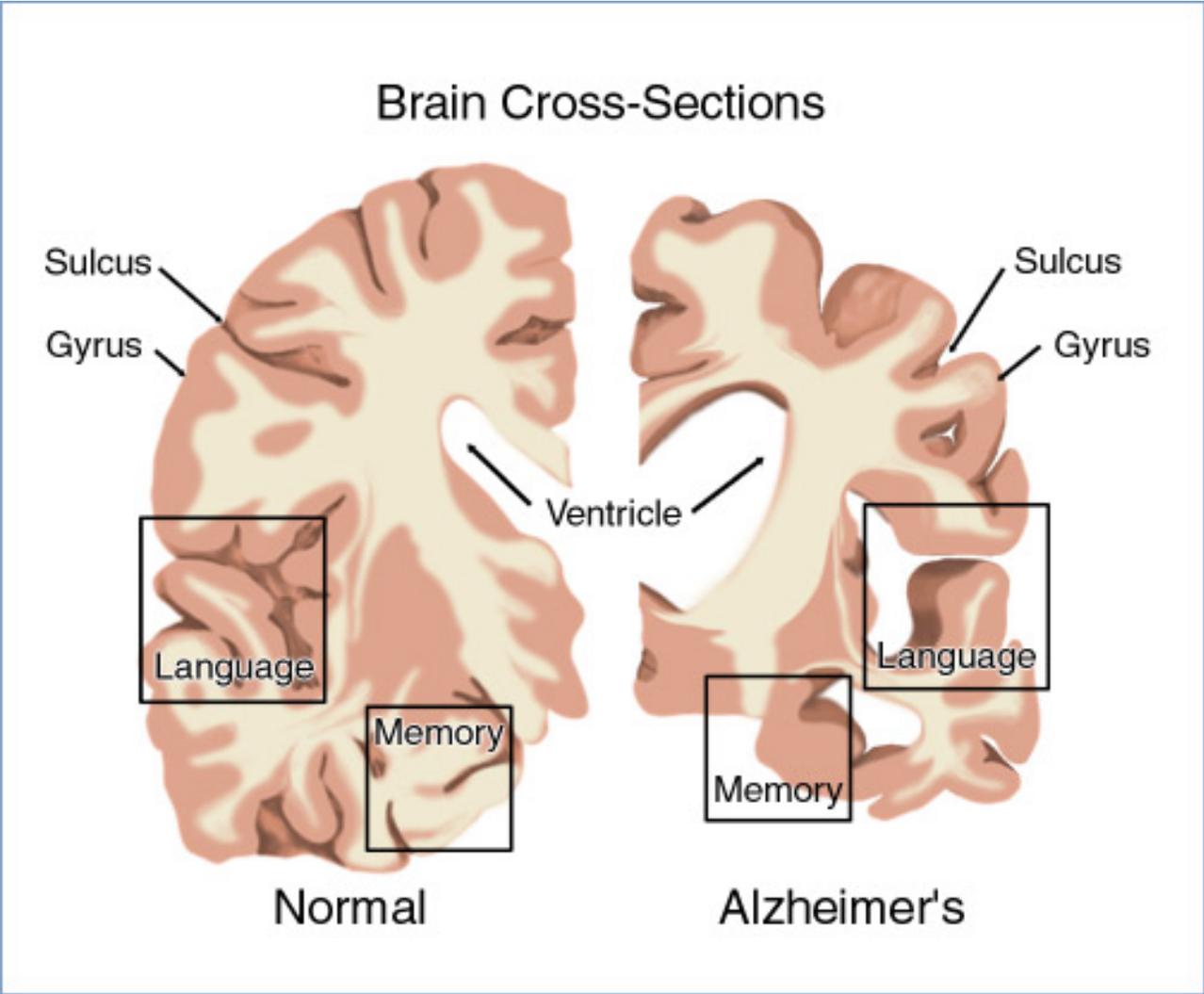


Figure 2.4 Cross section of brain (a) normal brain (b) Alzheimer's disease

Source: Strock (2004:1)

Watson (2004:1) is of the opinion that research has also identified that a defect in chromosome 21 may be responsible for the onset of AD. In the past aluminum levels in the blood were linked to the manifestation of the disease, but there is no conclusive evidence to cite it as a causative factor.

Antouno (2001:1) points out that as the disease progresses, the ability to perform calculations or planning activities as well as language skills diminish. The patient becomes dependent on the family for activities such as shopping and getting dressed.

2.3.1.1 Causes of AD

AD is not fully understood. There is probably no one single cause but several factors that affect each person differently (Watson 2004:2)

According to the Family Doctor Organisation (2004:1), an enzyme, protease, is responsible for the breaking down of proteins in the process of proteolysis. Breaking down of proteins is important for cell processes. However, inappropriate breaking down can be harmful because not only does it destroy the proteins, but abnormally high levels of fragments can also damage the cells.

Other scientists link family history as a risk factor to AD. Scientists suggest that genetics may play a role in the cause of AD. Familial AD, a rare form of AD that occurs between the ages of 30 and 60, is inherited. The most common form of AD is known as late-onset, namely it occurs late in life and there is no obvious pattern of inheritance. However, several risk factors may interact with each other to cause the disease. The only risk factor identified so far for late-onset AD is a gene that makes a form of protein called apolipoprotein (apoE), which helps carry cholesterol in the blood (Alexander 2002:2).

Researchers at the Gladstone Institute of Neuro Disease have identified processes that may explain how a key protein, apoE4, contributes to the development of AD. The findings include identifying where in the brain apoE4 is broken down into toxic fragments that can impair function and survival of nerve cells (Watson 2004:2). ApoE4 is the best-known genetic risk factor of AD, but until now the mechanism by which it increases that the risk

has remained a mystery. The key finding of the Gladstone study relates to apoE4's tendency to be broken down into toxic fragments when it is produced in the neurons, the brain cells responsible for cognitive function (Hanks, Bell, Halvey & Paice 2004: 1).

In the DREAM study (Ahmed 2001:50-58; Watson 2004:1) on genetically engineered mice, the researchers established that

- Only apoE4 produced by neurons is susceptible to fragmentation, unlike apoE4 produced by other brain cells.
- Fragmentation is correlated with age, occurring more frequently the older the animal, similar to the effect of age on AD risk in humans.
- Fragmentation of apoE4 occurs predominantly in the parts of the brain that are vulnerable to AD, the neocortex and hippocampus. In contrast fragmentation does not occur in the cerebellum, which is much less vulnerable to AD.
- ApoE4 fragments cause an abnormal change in the protein Tau, which is also affected in AD. Both apoE4 fragments and AD lead to abnormal attachment of phosphate group on the protein Tau that can end up distorting the shape of brain cells (Watson 2004:4).

This work may solve the riddle of apoE4, namely what happens at molecular level that makes this protein so active in causing neurodegenerative disease (Watson 2004:1).

According to De la Monte (1997:4), a research team at the Massachusetts General Hospital discovered a new gene that appeared to play a role in AD. The gene produced a protein called AD7c NTP in nerve cells, and excessive amounts of this protein were found in brain tissue and cerebrospinal fluid of people suffering from AD. Moreover, the accumulation of this protein in nerve cells caused changes similar to those seen in AD, including cell death.

De la Monte (1997:4) isolated the gene for AD7c-NTP and determined its sequence of nucleotides. She showed that the gene's protein was produced in brain tissue but not in tissue from other organs. The effect of this protein mimics the damage seen in AD patients. In addition, excessive levels of AD7c-NTP were seen much earlier in AD than other proteins associated with AD is known to appear.

According to Shiekhattar (2004:1), a study conducted at the Wistar Institute in the United states of America linked the genes responsible for neurofibromatosis to a protein that plays a role in ADThe protein shared by neurofibromatosis and AD is Kinesin-1, known to be significant in protein trafficking (a movement of various needed proteins from one part of a cell to another). The two genes linked to the disease called NF1, or neurofibromin, primary gene associated with neurofibromatosis. Another less common gene linked to the same disorder is called NF2 or merlin. Researchers used tools to identify NF1 and NF2 containing protein complexes in the nucleus and the cytoplasm of the cells. An analysis showed four subunits one of which was Kinesin-1. Finding Kinesin-1 in protein complexes that also contain NF1 and NF2 ties neurofibromatosis and Alzheimer's disease (Kenard 2005:1).

2.3.1.2 *Diagnosis of AD*

According to Hart (2004:1), early diagnosis helps patients who suffer from AD and their families plan for the future. It gives them time to plan care whilst the patient can still take part in decision-making. Early diagnosis offers them a chance to treat the symptoms of the disease.

Currently the only way to diagnose AD is to find out whether there are plaques and tangles in the brain. This is achieved when an autopsy is done. However, at specialised centres AD can be diagnosed correctly up to 90% of the time. Several tools are used to diagnose Alzheimer's diseases including CT scan.

Figure 2.5 illustrates a CT scan of a brain with dementia and a normal one.

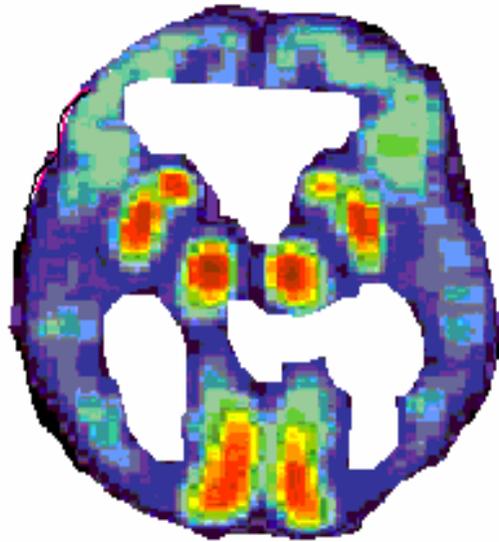
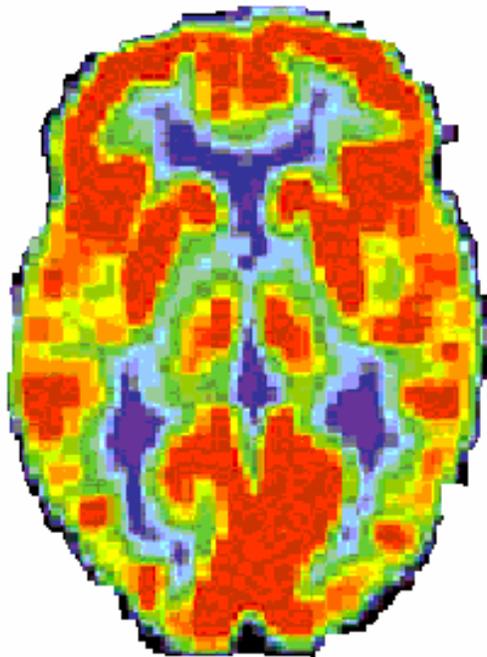


Figure 2.5

(a) CT scan depicting brain with dementia



(b) CT scan of normal brain

Source: Carson-DeWitt (2004:1)

Currently the efficacy of the Positron Emission Tomography (PET) scan in diagnosing AD is debated (Watson 2004:1).

Silverman and Small (cited in Lubieniecki 2000:1) explored the accuracy of PET in determining the aetiology of dementia non-invasively. The PET scans of 150 patients worldwide who underwent brain biopsy or autopsy to determine the specific cause of their dementing disorder were identified. An interim analysis found PET to have an accuracy rate of 87% for diagnosing AD specifically, and 93% for identifying neurodegenerative diseases overall.

According to Lubieniecki (2004:1) another study by Silverman and colleagues assessed the effect of anticholinesterase therapy (ACT) commonly used for AD, on the ability of PET to predict cognitive decline by reviewing 128 scans of patients who suffer from dementia. Scans considered positive were 85% accurate in showing clinical progression of dementia over the following three years or more of patients not on ACT with positive PET scans, 79% suffered clinical progression in the post-imaging period. In comparison, among those untreated patients with negative scans, only 9% suffered subsequent progression

The most recent advance involves a substance being developed at the University of Pittsburgh that can tag amyloid, a sticky substance that builds up into plaques surrounding the brain cells. This substance was tested by injecting into 16 people newly diagnosed with AD and other non-AD volunteers. The tagging substance crosses the brain and allows the team to identify the affected part of the brain. The most accepted theory of AD is that amyloid build-up is the trigger of cell death Watson (2004:1).

2.3.1.3 Treatment of Alzheimer's disease

The treatment of AD includes medications and non-medication treatment.

According Antuono (2001:2), there is no cure for AD, but over the years treatment of AD became symptomatic. Some medications treat behavioural symptoms such as agitation, restlessness and hallucinations. Others, such as Aricept (donepezil) and Exelon (rivastigmine), treat cognitive changes that affect memory and language, and some may

slow progression of the disease and maintain cognitive function for a longer period. However donepezil was found to be more effective and better tolerated than galantamine (Ebell 2003!).

According to Bayne (2004:1), in a study on mice, a team of researchers led by Yan identified one of the missing steps in how AD develops. They found that an enzyme “beta-amyloid peptide binds to a protein ABAD and accumulates inside the brain cells” and suggested developing a drug that would prevent the beta-amyloid peptides from binding with ABAD. A new approach involves administration of a vaccine that stimulate the immune system to prevent beta-amyloid being deposited in the brain.

There is controversy regarding the efficacy of hormone replacement therapy (HRT) in the treatment of AD. According to Shumaker (2003:1) studies show that postmenopausal women who receive HRT are 40 to 60% less likely to develop Alzheimer's disease. Oestrogen enhances memory.

However, Mercola (2003:2) posits that researchers are not certain how the combination therapy increased the risk of dementia but suggested that it increased the risk of blood clots and clogged blood vessels in the brain, which might injure brain cells and contribute to AD.

According to Maywood (2004), a new study found strong evidence that statin drugs are beneficial for AD. Statin drugs, such as Valsartan, are used for lowering cholesterol. It was found that taking cholesterol-lowering agents was associated with slower cognitive decline and suggested their efficacy by a mechanism independent from the cholesterol-lowering agent. Munzar (cited in Maywood 2004) adds that these drugs are safe, well tolerated and are widely available and therefore have a great potential for AD as they reduce The incidence of AD by 70%.

According to Jacques (2001), a Dutch research study published in 2001 found that non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen can delay or protect against the onset of AD. The supporting theory to the effectiveness of the drugs is that there is an inflammatory-type response in the brain to the cell damage and death that is caused by

AD. This inflammation is important and might contribute to the damage to the brain cells. The study suggested that, in addition to dampening down the inflammatory response, the NSAIDs might interfere with the process of cell damage through amyloid. The effect of the drug in reducing AD showed that people who took the drug for less than a month had 5% less chance of developing the illness while those who had been taking the drug for over two years were 80% less likely to develop AD. This could mean the onset of AD in people who might have developed it was delayed, but this was not clear.

Aldridge (2004:1) suggests that some drugs might have an adverse effect on the brain chemicals involved in AD. Acetylcholine is a brain chemical, which is decreased in AD and is linked to the memory and learning problems that typify the condition. Researchers at Newcastle Hospital, England presented a study that showed how drugs that block acetylcholine could worsen AD (Aldridge 2004). A group of drugs called muscarinic antagonists used in Parkinson's disease block acetylcholine. The study found that post-mortems of patients who had taken muscarinic antagonists revealed more plaques and tangles. The study recommended that muscarinic antagonists and drugs that block acetylcholine be prescribed with caution in elderly people. Conversely, the acetylcholine boosting drugs used in treating memory problems in AD may also be protecting the brain by inhibiting the development of plaques and tangles (Aldridge 2004:1).

Ginkgo biloba has recently been approved in Germany for the treatment of Alzheimer's disease (Shumaker 2003:2). Hoffman (2004:1) states that the herb industry and supplement manufacturers make dramatic claims for ginkgo biloba in the treatment of dementia and AD. Ginkgo biloba reduces vascular, tissue and metabolic disturbances as by increasing blood flow into the ischaemic tissue by

- Raising levels of glucose and ATP (Adenotriphosphate) in the cell, thus maintaining energy levels.
- Slowing the onset of dementia resulting from sclerosis of cerebral arteries.
- Ameliorating the effects of progressive cerebral circulatory insufficiency due to age.

Bren (2004:1) carried out a prevention trial to determine the efficacy of selenium and

vitamin E in preventing memory loss. The institute found that increased oxidative stress (from excess free radicals) may damage brain cells and is linked with AD. Increased oxidation of brain lipids, proteins, carbohydrates and DNA found in Alzheimer's disease. It is believed that oxidative stress contributes to damaging brain cells in AD. Animal and tissue culture studies of vitamin E and selenium suggest that they can protect the brain cells from damage.

Other drugs being tested for slowing the progression of AD include the combination of foliate B6/12 (Jacques 2001:3).

Non-drug treatment includes healthy diet, exercise, social activities, regular medical care and a safe environment. The use of memory aids such as calendars, lists and written directions can be helpful in the initial stages of AD (Kornstein 2002:4).

2.3.2 Vascular dementia

Moretti (2001:3) describes vascular dementia as decline in memory functioning leading to inability to perform routine activities. It is caused by poor circulation in the subcortical area of the brain resulting in stroke. In vascular dementia deterioration occurs step-wise and the symptoms worsen with each new stroke.

According to Alagiakrishnan (2004:3), as early as 1899 arteriosclerosis and senile dementia were described as different syndromes. In 1969, Mayer-Gross, described this syndrome, vascular dementia and suggested hypertension as the cause in 505 cases. In 1974, Hachinski coined the term "multi-infarct dementia". In 1985, Loeb used the broader term "vascular dementia". The term "vascular cognitive dementia" has been used to describe this particular form only recently by Bowler and Hachinski. According to Arvanitakis (2000:1), most authors propose the term vascular cognitive impairment (VCI) with the objective of broadening the term further.

Vascular dementia is responsible for twenty percent of all types of dementia in Western Europe. It occurs commonly in old age. Contributory factors correspond with those responsible for stroke and cerebrovascular disease, namely smoking, heart disease and

excessive alcohol intake. Vascular dementia is caused when small infarcts invade the cortical areas of the brain reducing oxygen content in the brain leading to death of those brain cells. (Fares 1997:2).

2.3.2.1 Causes of vascular dementia

Vascular dementia is caused by a series of strokes that interfere with blood flow as well as destroying brain tissue. Some of the causes include untreated hypertension, diabetes, high cholesterol and heart disease (Alzheimer’s Society 2004b). Therefore, causes are classified as reversible and non-reversible. Table 2.1 reflects reversible and irreversible causes of Vascular dementia.

Table 2.1 Causes of vascular dementia

NON-REVERSIBLE CAUSES	REVERSIBLE CAUSES
<ul style="list-style-type: none"> • Increasing age 	<ul style="list-style-type: none"> • Hypertension
<ul style="list-style-type: none"> • Genetic disposition 	<ul style="list-style-type: none"> • Coronary artery disease
<ul style="list-style-type: none"> • Prior strokes 	<ul style="list-style-type: none"> • Atrial fibrillations
<ul style="list-style-type: none"> • Low level of education 	<ul style="list-style-type: none"> • Diabetes mellitus
	<ul style="list-style-type: none"> • Hyperglycaemia
	<ul style="list-style-type: none"> • Hyperlipidimia
	<ul style="list-style-type: none"> • Smoking

Source: Alzheimer’s Society (2004b)

2.3.2.2 Pathophysiology of vascular dementia

According to Alagiakrishnan (2004:2), vascular dementia produces either focal or diffuse effects on the brain and causes decline in cognition. Focal cerebrovascular disease occurs secondary to thrombotic or embolic vascular occlusion. Common areas of the brain related to cognitive decline are the white matter of the cerebral hemisphere and the deep grey nuclei, especially the stratum and thalamus. Hypertension is the principal cause of diffuse disease and in many patients both form of the disease is present simultaneously. The three most common mechanisms of vascular dementia are multiple cortical infarcts, strategic

single infarct and small-vessel disease.

In multi-infarct dementia, the combined effects of different infarcts affect the neural nets thereby producing cognitive decline. In single infarct dementia different areas of the brain are affected resulting in cognitive impairment. This may be observed in cases of cerebral artery infarct and parietal lobe infarct (Black 2002:17).

2.3.2.3 Symptoms of vascular dementia

The symptoms include transient ischaemic attacks, loss of eyesight, falls, confusion, dysarthria and dysphasia (Alagiakrishnan 2004:2). Yanagihara (2000:25) adds the following symptoms:

- Memory loss
- Confusion
- Mood swings and personality changes
- Language problems
- Difficulty paying attention or following a conversation
- Impaired motor skills
- Difficulty planning or organising tasks
- Difficulty with calculations, making decisions, solving problems
- Depression-like behaviour

Various characteristics differentiate VaD from AD and an ischaemic score has been devised to indicate this (see table 2.2). The ischaemic score assesses the presence of thirteen clinical features out of eighteen. If the score is seven it is suggestive of VaD and a score of below four suggests AD (Arvanitakis 2000:3)

Table 2.2 Ischaemic score

• Abrupt onset	2
----------------	---

• Stepwise deterioration	1
• Fluctuating course	2
• Nocturnal confusion	1
• Relative preservation of personality	1
• Depression	1
• Somatic complaints	1
• Emotional incontinence	1
• History of hypertension	2
• History of strokes	1
• Associated atherosclerosis	2
• Focal neurological symptoms	1
• Focal neurological signs	1

Source: Arvanitakis (2003:4)

2.3.2.4 *Diagnosis of vascular dementia*

The following results are suggestive of vascular dementia on CT or MRI:

Bilateral multi infarcts located in the dominant hemisphere and limbic structures, multiple lacuna strokes, or periventricular white-matter lesion extending into the deep white matter (see figure 2.4). PET may be useful in differentiating vascular dementia from AD (Alagiakrishnan 2004:3).

Strub (2003:2) cites a study conducted at San Francisco VA Medical Center to distinguish between kinds of dementia, using a combination of MRI and a related technique known as magnetic resonance spectroscopy (MRS). According to Sachdev et al (1999:82) MRI was used to create three-dimensional images of the patients' brains to detect difference in structure, while MRS was used to look at the chemical signature of different brain regions. A point of interest was neurons because they carry electrical signals between different parts of the brain and produce a chemical known as N-acetylaspartate or NAA, which is not produced by other type of brain cells. The amount of NAA in a particular region of the brain is considered normal while abnormal amounts indicate neural loss or dysfunction. Less NAA was found in the region of the brain involved in short-term memory and decision making (frontal cortex) in patients who suffer from VaD than in patients who suffer from AD. The levels of NAA were nil in the area of the brain involved in language and spatial orientation (medial temporal lobe) of the patients with VaD while patients with AD had substantial NAA deficits. Figure 2.7 depicts multiple infarcts in a VaD brain.

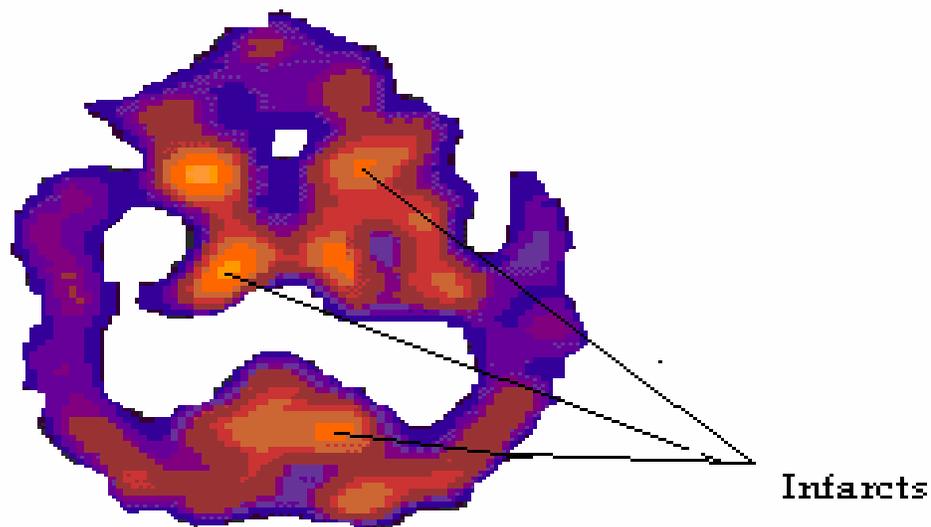


Figure 2.7
Multiple infarcts in vascular dementia

Source: Strub (2003:41)

2.3.2.5 Treatment of vascular dementia

General management includes referral to community services, addressing legal and ethical issues Such as driving competency and consideration of caregivers (Alagiakrishnan 2004).

According to Frank (2003:1305), drug treatment is used mainly to prevent further worsening of vascular dementia by treating the underlying diseases such as hypertension and diabetics. Antiplatelet agents such as aspirin are indicated. Neuroprotective drugs such as nimodipine are currently understudy and may be useful for vascular dementia.

2.3.3 Pick's disease

Pick's disease is a relatively rare brain illness that causes dementia (Lennox 2004:1). Arnold Pick first described it in 1892. Until recently Pick's disease was thought to be similar to AD during life. Consequently is has been little studied, much less is known about it than about AD.

Pick's differs from AD in two ways. First, the two diseases produce different abnormalities in the brain cells. Pick's bodies mark Pick's disease, rounded microscopic structures found within the affected cells. Neurons swell, taking on a "ballooned" appearance. Neither of these changes appears in AD. Plaques and tangles are not found in Pick's (Lennox 2004:1). Secondly, Pick's disease is confined to the front parts of the brain, particularly the frontal and anterior temporal lobes. This contrasts with AD, which is more widely distributed. The two diseases also produce different neurochemical changes in the brain. The two differences between Pick's and AD mean that the two produce different symptoms. In contrast to AD in which early memory loss predominates, Pick's symptoms include personality changes, both at work and home, in the form of apathy, indifference in social decorum and feelings of others. There is also poor social judgement, inappropriate sexual advances, or a coarse and jocular demeanour ma be seen. Function declines simply because the patient does little or may be confused or forgetful. Often times the patient performs well when directed but cannot do well independently. The ability to initiate, organise and follow through on simple plans and familiar activities is lost (Lennox 2004).

As the illness progresses, language becomes difficult, patients become quiet and when

they speak, speech is often slow and in brief sentences. There may be difficulty in making sounds of words. Speech becomes distorted. Some become apathetic (may sit for hours doing nothing at all, unless prompted to do so). Some patients are hypersexual; some, like a small child, will place anything they pick up in their mouth. There is glutinous eating and poor attention span, patients seem distracted instantly by anything they hear or see. Later they become mute. Restlessness leads to apathy and eventually to terminal vegetative state (Lennox 2004).

2.3.3.1 Onset of Pick's disease

Pick's disease usually begins after 40 years and is less common after the age of 50 years. It is a disease that invariably worsens. The average course is about five years but it ranges between two and fifteen years (Lennox 2004:2).

2.3.3.2 Causes of Pick's disease

Neither the cause of nor a cure for Pick's disease is known. According to Lennox (2004:2), studies suggest that Pick's has a genetic component, but most family members are unaffected.

2.3.3.3 Diagnosis of Pick's disease

Diagnosis of Pick's disease is difficult during life due to symptoms overlapping with AD. A CT or MRI scan may show patterns of atrophy that suggest Pick's (Lennox 2004).

2.3.3.4 Treatment of Pick's disease

According to Farmer and Dickson (2004:3), the treatment is the same as for AD and includes supervision and assistance aimed at maximising the patient's quality of life. Medications are symptomatic. Treatment also includes emotional and substantive support for the caregiver.

2.3.4 Lewy body dementia

According to Fares (1997:2), Frederick Lewy first described this type of dementia in 1912 as being the result of the cortical changes observed in Parkinson's disease. Lewy bodies are pink structures found in the cytoplasm of neurons. They occur in the brain stem and cortical areas of the brain. People who suffer from Lewy body dementia show marked impairment of parietal lobe functions, such as difficulties with hand and eye coordination tasks and dyspraxia. Loss of memory is uncommon. A patient presents with visual or auditory hallucinations, fluctuating episodes of confusion or lucidity and unexplained falls.

Crystal (2004:1) adds that Lewy body dementia is the second most common degenerative disease after AD. It is a progressive dementia marked by hallucinations and fluctuating levels of attention. Some rigidity and stiffness may be seen. Other symptoms include disturbed sleep patterns with nightmares and abnormal behaviour, cognitive decline, memory problems associated with various degrees of alertness and attention span, stiffness and rigidity contributing to problems with activities of daily living as well as repeated falls and visual hallucinations.

2.3.4.1 Causes of Lewy body dementia

The cause is unknown or uncertain. It is thought to be due to a faulty production of the protein, alpha-synuclein that builds up within the nerves of the brain. This protein is linked to Parkinson's' disease (Lantz 2004:2).

2.3.4.2 Diagnosis of Lewy body dementia

Diagnosis of Lewy body dementia is determined by the patient's history and symptoms. History of progressive cognitive decline deficits, visio-spatial may be especially significant (Roques 2004:1).

- There may be fluctuating cognition with pronounced variation in attention alertness.
- Recurrent visual hallucinations that is typically well formed and detailed.
- Spontaneous motor feature of Parkinson's disease.

Features supportive of the diagnosis include the following:

- Repeated falls
- Syncope or transient loss of consciousness
- Neuroleptic hypersensitivity
- Systematised delusions
- Visual hallucinations

2.3.2.4 Treatment of Lewy body dementia

According to Ferman (2004:2), treatment involves careful management and support. Neuroleptics given to control hallucinations and delusions are poorly tolerated.

2.4 NURSES' OPINION OF PAIN

The act of communication involves all the ways people send and receive messages. If the process becomes impaired a person's social capacity is reduced, resulting in isolation (Bush 2003:42). People who cannot communicate or communicate inappropriately are often marginalized by society.

As nurses have direct contact with the patient in long-term care they are in a position to notice subtle changes in the patient's condition. The point of view of the patient with dementia is frequently unknown, yet it is important to identify it in order to offer proper intervention (Cohen-Mansfield 2001:40). Middleton (2004:42) states that guesswork and assumptions are often used but are not an accurate basis for health care interventions.

McCaffery and Pasero (1999) (cited in Middleton (2004:42) state that institutions are responsible for managing patients' pain and patients should have access to the best level of pain relief that may be safely provided.

Research shows that pain is still under-reported and under-recognised by health care professionals. In the USA pain is considered the fifth vital sign (in addition to temperature, pulse, respiration and blood pressure) that is an early indicator of deterioration of condition.

Abram (2001:100) states that the Royal College of Surgeons and the Royal College of Anaesthetists (1990) recommend that pain become the fifth vital sign that needs to be assessed and documented accurately, regularly and appropriately. In the UK chronic pain teams are under-funded. Nurses complain that these teams are axed as a way of hiding waiting lists (Middleton 2004:43)

According to Buckley (2003:1) a study compared 216 dementia patients and cognitively intact adults who were hospitalised for either pneumonia or a fractured hip and examined their six months' survival. Both groups received almost identical care and therapeutic procedures. However, the patients with end-stage dementia received less analgesia than the cognitively intact adults. According to Mahoney (2004:1), "the under-treatment of pain in dementia patients likely resulted from the fact that these patients often did not communicate what they were feeling or the fact that they were in pain".

According to McManus (2003a:1), several factors influence a person's perception of pain including attitude, belief, coping ability, cognitive capacity, ethno-cultural factors, religion, health status and disease. Nurses need to recognise pain as a priority and play an active role in preventing and treating it. Gaston-Johansson et al (1999:190) assert that nurses and doctors depend on patient self-report for pain; inability to report is considered a lack of pain. In patients who cannot communicate, change in behavior is regarded as attention seeking. McLean and Higginbotham (2002:5) state that to nurses' change in behaviour may suggest attention seeking rather than entertaining the possibility of pain. Frequently the first line of approach is antipsychotic drugs or drugs that mask a pain problem or make it worse. Doctors are reluctant to prescribe opioids to the elderly for fear of side effects, constipation, falls or delirium.

Horgas (2003:15) asserts that many clients with dementia cannot verbalise pain while others are not taken seriously. Some clients with dementia communicate their pain verbally or admit to having pain if asked directly. Therefore it is important for nurses to rely on patients' self-report of pain in patients with dementia as they would those who are mentally alert. Unrelieved pain has been found to lead to decline in cognition. Nurses may lack the knowledge to manage pain adequately and may harbour attitudes that interfere with successful pain management (McLean & Higginbotham 2002:5).

Bonifazi (2002:2) affirms that in addition to loss of memory, judgement and communication skills, dementia characteristics may include confusion, aggression and agitation, anxiety and delirium. These are exacerbated by illness, increased temperature, disease dehydration, medication, tests, treatment, and changes in routine, unfamiliar hospital sites, sound, smell and staff.

A high prevalence of under-treatment of pain in the elderly has been documented both in national and international clinical settings. Both acute and chronic pain is a longstanding problem in this population (Caselli 2004:2). Under-treatment of pain leads to increased suffering, personal distress, immobility, decreased functional ability, poor sleep, anxiety, and depression, less social activity, isolation, loss of appetite, leading to malnutrition and also impacts on the patient's quality of life. Brignell (2004:1) adds that unrelieved pain can also result in an individual experiencing distressing cognitive impairment, such as disorientation, mental confusion, and reduced concentration ability. According to Macintyre and Ready (2001) (cited in Middleton 2003:31), the stressor effect of unrelieved pain therefore has the potential to increase anxiety levels and interfere with activities of daily living such as diet, work, leisure and exercise, resulting in insomnia.

According to Lane (2004:1), health care providers sometimes do not realise that these group of patients are in pain therefore analgesia, such as morphine, should be given even though the patient does not express the need.

2.5 CONCLUSION

This chapter discussed various definitions of pain, normal pain transmission and pain modulation in order to understand the pathophysiology of pain. Nurses' role in assessment of pain was described including the use of visual analogue scales that help nurses to understand the patient's level of pain. Various types of dementia and nurses' opinion of pain were also discussed. Chapter 3 describes the research design and methodology.

