A COMPARATIVE STUDY OF ALZHEIMERS DISEASE, MULTI INFARCT DEMENTIA AND THE AIDS DEMENTIA COMPLEX.

by

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ABSTRACT
Nursing homes and other long term health care facilities have traditionally been the home of the frail aged or younger people with profound disabilities. With increases in medical knowledge comes increased human longevity and diseases associated with longer life will lead to an increase in number of people with dementia. As a sufferer's condition worsens, the idea of placement may arise, and the person is placed in a nursing home where sufferers receive specialised care. As this happens expectations of quality of care in long term care facilities will rise, and certain questions will need to be addressed.

The aim of this study is to look at three of the more common forms of dementia - Alzheimers disease (AD), multi infarct dementia (MID) and the lesser known AIDS dementia complex (ADC).

ADC care is a relatively new concept to the majority of nursing homes mainly due to the fact that powerful anti-viral drugs are being used as treatment, resulting in an increase in the lifespan of sufferers. Due to the fact that placement in a long term care facility is significantly cheaper than specialised home care, it is eminently possible that the numbers of AIDS sufferers in nursing homes will also increase. This study will also address the contentious issues associated with the placement of ADC sufferers in nursing homes.

In the future studies such as this will be of utmost importance not only to the medical field but to all allied health teams providing care in a nursing home or other long term care facility.
INTRODUCTION

The extension of the average human lifespan has resulted in a rising prevalence of chronic disease in our population, and dementia is no exception to this. This study will look at a brief overview of dementia and discusses the diagnosis, pathology, clinical features, treatment and duration of Alzheimer's disease (AD), multi infarct dementia (MID) and acquired immune deficiency syndrome (AIDS) dementia complex (ADC). Finally, the feasibility of care of ADC sufferers in long term care institutions will be discussed.

In order to define dementia, it is important to realise that it is not merely a part of growing old and is not necessarily associated with wrinkles and grey hair. Dementia affects an estimated 5% of the population aged 65 and over, and 20% of the population aged 75 and over (McLean, 1987).

Dementia is a syndrome, characterised by a series of separate signs and symptoms which may be caused by underlying illnesses (Jacques, 1988). Each of these illnesses should have a clear, definable underlying pathology - organic or psychological. The presence of dementia may be the localising sign of an underlying pathology of some sort.

McLean provides this lengthy definition:

"dementia is.....an acquired decline in a range of cognitive abilities (including memory, learning, orientation and attention) and intellectual skill (including abstraction, judgement, comprehension, language usage and calculation) accompanied by alterations in personality and behaviour which impair daily functioning, social skills and emotional control." (1987, p149)
What McLean is suggesting is that dementia is a progressive and often irreversible loss of mental function, until the person is no longer able to perform basic functions such as eating or drinking. There are two main forms of dementia - that found in the younger population of 75 years and below, the so called "young old", and the other found in older patients of 75 and over - the "old old" (Council on the Ageing, 1994). Dementia may be further divided into two categories - cortical or subcortical, depending on the part of the brain affected. The distinction between the two is a basic difference in the pathological process causing the dementia and therefore the differing clinical outcomes (McLean, 1987). Clinical features are also different - subcortical dementia is characterised by a generalised mental slowing and apathy, while those suffering from cortical dementia will often display an impairment in the higher cerebral functions such as cognition.

Approximately 10% of dementia cases are caused by a number of other factors such as acute infective illnesses, metabolic disorders or depression, which may lead to a diagnosis of pseudodementia (Forsythe, 1990). Syphilis of a long standing nature may also cause dementia, as well as brain tumors or chronic subdural haematoma (Forsythe, 1990).

For many years it has been known that a severe affective disorder could lead to some form of cognitive impairment, hence the term pseudodementia. The syndrome of pseudodementia refers to a variety of conditions where the clinical features point to a form of dementia, but the underlying cause is a psychiatric illness. Interestingly enough, the clinical picture of profound, progressive cognitive decline may be indistinguishable from that seen in progressive, irreversible dementia. The depression must then be treated - if the dementia symptoms recede, then it is feasible to say that the problem is depression related. However, Gustafson (1975) reported that 30% and Liston (1977) 25% of people ultimately diagnosed as having dementia presented with a first symptom of depression (cited in Siegal and Gershon, 1986). Indeed, van Dujin
and Hoffman (1991) suggested that a history of medically treated depression was 80% more common in those with AD than in controls. As well as those described above, there are also many genetic disorders causing dementia such as Huntingtons chorea, Pick's disease and the rarer Jacob Creuzfeldt disease (Forsythe, 1990).

ALZHEIMERS DISEASE
Alzheimers disease (AD) is the most common form of dementia, accounting for almost 60% of all cases (McLean, 1987). AD is a cortical brain disease of unknown cause, which leads to memory loss and ultimately death. There are certain risk factors, the most important of which is old age. A family history of the disease puts a person at a definite risk. Also at risk are sufferers of Down's syndrome, who develop the brain changes of AD by the time they are forty years old, however the observable signs of the dementia do not present until the person is much older. The reason for this is not understood, however the genes on chromosome-21 (the gene affected in Down's syndrome) appear to be implicated in Alzheimer brain changes (Department of Health, Housing and Community Services (DHHCS), 1993). Other risk factors include head trauma which means AD is more likely in those who have received a blow on the head sufficient to cause unconsciousness (van Duijn and Hoffman, 1991). The environmental factor aluminium may also play a part in AD. Metals such as aluminium are known to have a damaging effect on nerve cells in the body, and it is feasible that AD may be caused by some form of toxic buildup (Jaques, 1988).

MULTI INFARCT DEMENTIA
Vascular dementias are the second most frequent form of dementia, occurring in 25 - 33% of people with diagnosed dementia. The most common of these is MID (Metter and Wilson, 1993). MID is characterised by certain parts of the brain dying due to tiny infarcts in the blood vessels. Once an area of the brain dies it no longer works, hence the loss of function. Where AD has an insidious onset, MID is characterised by an abrupt onset. Both show similar rates of decline, however there is a greater mortality
associated with MID (Metter and Wilson, 1993). There has been less research carried out on the risk factors of MID than AD, however since vascular dementia is due to tiny strokes it may be assumed that the risk factors are similar than those of stroke. Again, old age and family history are factors, and so is race and geographic location. For example, the incidence of vascular dementia is higher in Japan and China than in Australia, Europe or North America. (1) Diabetes mellitus predisposes a person to MID, as do high blood cholesterol levels. Sufferers of MID tend to have low levels of the good form of cholesterol, high density lipoprotein cholesterol. The major risk factor is hypertension, which on its own may have an adverse affect on the persons memory, unrelated to its role in producing infarctions (Jorm, 1990).

AIDS DEMENTIA COMPLEX

As at the end of March 1991, there were 2 527 cases of AIDS in Australia, (Kernutt, Price, Judd and Burrows, 1993) and the AIDS dementia complex (ADC) is an integral part of this disease. This is a less common form of dementia, however as people with HIV are living longer it is expected to become more common, (ADC used to be known as human immunodeficiency virus (HIV) encephalopathy, but ADC is now generally used). ADC is the most commonly reported psychiatric syndrome associated with HIV infection, with over two-thirds of the mental abnormalities in AIDS patients found to be signs of ADC (Pratt, 1991). Unlike AD and MID, ADC is a late manifestation of the later stage of HIV infection, usually occurring after one or more opportunistic infections (Farthing, Brown and Staughton, 1988). It is described as a distinct, relatively stereotyped neurological disorder, complicating HIV infection, and is characterised by a constellation of cognitive, motor and behavioural symptoms (Nurnberg, Prudic and Fiori, 1984). ADC affects approximately 4.2% of AIDS sufferers (Kernutt et al, 1993).

DEMENTIA INCIDENCE

Different studies report different rates of the prevalence of dementia in the community. The one common thread is that the prevalence of dementia increases with age (DHHCS,
1993). The risk of AD doubles with every 4.5 years of age after the age of 60, with the risk of vascular dementia doubling for every 5.3 years of age after the age of 60 (DHHCS, 1993). It is predicted that the incidence of dementia will continue to rise, along with the ageing of the Australian population, and that there are far more cases of dementia found in the "old-old" age group. Incidence figures are difficult to find concerning ADC, however since it was recognized as an AIDS defining condition, the number of cases has steadily risen from 1.1% of cases reported in 1987 to 4.2% in 1990 (Kernutt et al, 1993).

**CLINICAL FEATURES**

AD presents with memory disturbance, personality and behaviour change and general intellectual decline. La Rue, Watson and Plotkin (1993) found that initial symptoms included depression, confusion language and motor problems and getting lost. The effects of the disease on the cerebral cortex are shown by the development of aphasia, apraxia and agnosia in some cases, and the short term memory loss. They also suggest that sufferers may be anxious and aggressive, and have paranoid delusions.

MID sufferers typically show an uneven distribution of deficits in memory and a certain lability of emotions. This may lead to brief episodes of being sad, with outbursts of weeping or laughter. Personality changes may include apathy, or an accentuation of previous personality traits (DHHACS, 1993). Interestingly enough, it is possible for features of both AD and MID to be present in the one individual - this is known as mixed dementia, and it is very hard to determine which form of dementia presented first.

On a final note, a feature of AD and MID concerns Cerebral Blood Flow (CBF) and metabolic changes. Both have been noted to differ in these forms of dementia, with AD showing a more uniform reduction in both, whilst MID is characterised by patchy areas
of decreased CBF and metabolism. These changes may even precede the onset of clinical symptoms.

Cognitively, symptoms of ADC may include difficulty in concentrating, and generalised slowing (Gee and Moran, 1988, Kernutt et al, 1993). Motor function is affected, especially balance and co-ordination, with ocular motility and slowing of rapid successive and alternating movements of the fingers and wrists other features of the disease (Price and Brew, 1988). Generalised weakness of the legs and arms, leading to end stage ataxia, urinary and faecal incontinence and tremors are all neurological signs of dementia (Gee and Moran, 1988, Price et al, 1988)

PATHOLOGY
AD is characterised by a series of changes in the brain itself, including brain shrinkage and degeneration of neurones leaving plaques and tangles, and is therefore only positively confirmed at autopsy. Ventricles also become enlarged and sulci are widened (Thornton, Davies and Tinklenberg, 1986).

Over the course of the disease, the cerebral hemispheres start to shrink (Jacques, 1988, Forsythe 1990, Bloom, Lazerson and Hofstader, 1985). A Computer Tomography (CT) scan will easily show this process in younger populations with AD, but in older brains, age related changes make these more difficult to identify. Shrinkage occurs mainly in the cerebral cortex, the grey matter (neurones) and may effect the white matter (nerve fibres) (Jacques, 1988). It is not known why the death of these particular cells cause cerebral gray matter to shrink. The most significant damage appears to be centered on the frontal, parietal and temporal lobes (Jacques, 1988). Damage is to the hippocampal region of the temporal lobes - this is the centre for short term memory function, the loss of which is a feature of AD. As the disease progresses, the lobes of the brain become less convoluted and the surface flattens. At the same time, the sulci widen, and ventricles which circulate cerebro-spinal fluid (CSF) expand. It also seems
that neurones, especially the endings and connections, degenerate leaving plaques and tangles (Jacques, 1988). Plaques are microscopic changes in the brain that show up under magnification with silver stains containing deteriorating nerve endings. Their numbers are directly proportional to the degree of intellectual impairment in the sufferer, and are found in areas of greatest shrinkage (Jacques, 1988). (2) Neurofibrillary tangles meanwhile, are found inside the neuron (Forsythe 1990, Jacques 1988) and they appear to be made up of the remains of the damaged part of the neuron itself. Whatever their meaning, the mere existence of plaques and tangles indicates a reduction in the numbers of functional nerve cell connections. Neurotransmitters in the brain are used to transmit messages and acetyl-choline is known to be decreased in the brains of AD sufferers, particularly in the all important hippocampus (Bloom et al, 1985, Jacques, 1988, Forsythe, 1990). It is easy to see then that if the neurones die, or if chemical messengers are not available, then the message will not be sent. The presence of AD also effects other transmitters - noradrenaline, gamma-aminobutyric acid and 5-hydroxytryptamine show a lesser decline (Wesson Ashford and Zec, 1993, Jacques, 1988, Bloom et al, 1984). The losses of these transmitters however, may still be secondary to the agent causing the underlying neuronal destruction.

MID results from certain pathological changes occurring in the circulatory system. This form of dementia is caused by the occurrence of multiple cerebral infarctions and haemorrhage (Jacques, 1988). Indeed when one looks at the surface of the brain of an MID sufferer, the cerebral cortex is covered with multiple, miniature areas of damage, with the underlying brain tissue softened and the cells are seen to be dead or dying (Forsythe, 1990; Jacques, 1988). MID appears to result from one of two causes - the occlusion of blood vessels with resulting infarction, or by haemorrhage into the brain. Typically, the infarction is a result of thrombotic or embolic occlusion (Metter et al, 1993). Atherosclerosis, although not a direct cause of MID, may cause the arteries to develop lesions which render them more susceptible to occlusion. Reasons for this are not yet clear (Metter et al 1993; Jacques, 1993). Metter et al (1993) on the other hand,
suggests that MID patients with hypertension may develop lipohyalinosis in their arteries. This results in a loss in the muscular layers of the artery, and the formation of lacunes which may cause some of the damage.

MID is classified as a subcortical dementia, however there is a current push to divide it into being both cortical and subcortical. Cortical forms result from infarctions in the large vessels, whilst subcortical forms are from damage to medium or small vessels (Metter et al, 1993). Again, the degree of intellectual impairment is directly proportional to the degree of cerebral softening. Repeated infarcts continue to cause decline, however it is when larger infarcts occur that the stepwise decline occurs. A sudden infarct may cause the brain to become slightly oedematous, leading to acute confusion and disorientation (Jacques, 1988). Functional level may remain lowered for weeks or months, providing no further damage occurs. The area of cortex not damaged may go on working normally, however connection with damaged areas may mean that a person has decreased use of a particular function.

In ADC, the damage to the brain is considerable. It is generally believed that dementia is a direct result of central nervous system (CNS) infection by the HIV virus (Price and Brew, 1988). It seems that HIV penetrates the blood-brain barrier shortly following initial infection, thereby causing abnormalities in the white matter and subcortical areas with vaculocation (formation of vacuoles in the interior of the nerve cells), an increase in the number of astrocytes, inflammation of lymphocytes and macrophages and demyelination of white matter the most common findings (Price and Brew 1988; Gee and Moran, 1988; Kernutt et al, 1993). It appears that the cerebral cortex is generally spared in ADC. Other neurological findings include an almost universal result of cerebral atrophy, with widened cortical sucli and enlarged ventricles - both may be confirmed by either CT scan or Medical Resonance Imaging (MRI).
DIAGNOSIS

The primary requirement for a diagnosis of a dementia of any type is evidence of a decline in thinking sufficient to impair activity. This should be evident for a period of six months or more. It is vital that a diagnosis of dementia be made on a series of recognized guidelines such as the Diagnostic and Statistical Manual of Mental Disorders (DSM) (Oxford Dictionary of Nursing, 1994). This manual has now been revised and the current version is known as the DSM-III-R criteria. If diagnosis is made purely from a general interview with the person concerned, it is possible that vital information the DSM-III-R are designed to identify may be missed.

According to McLean, for a diagnosis of AD, the DSM-III-R requires the presence of:

1. the dementia syndrome
2. an insidious onset
3. a uniformly progressive, deteriorating course. (McLean, 1987)

The DHHCS (1993) adds to these, suggesting that there should also be absence of evidence (historically or clinically) that the dementia could be caused by any other cause, and that there be an absence of vascular impairment in the brain. This diagnosis may be further supported by progressive deterioration in cortical skills - primarily language, motor performance and perception (McLean, 1987). However, a positive diagnosis of AD can only be made at autopsy, although experienced clinicians can make a correct diagnosis in approximately 90% of cases. The diagnosis of MID again according to McLean, follows almost the same criteria, again set out by the DSM-III-R:

1. presence of the dementia syndrome
2. a stepwise course with patchy cognitive deficits
3. focal neurological symptoms and signs
4. evidence of contributing cardiovascular disease (McLean, 1987)
Neurological findings often go hand in hand with the above, depending on the location of the lesions caused by infarction. These may include sensory loss, agnosia, neglect or aphasia (Metter et al, 1993). Metter et al (1993) suggest that probable vascular dementia requires not only evidence of memory loss, but clinical (DSM-III-R) or radiological evidence of cerebrovascular disease, as well as evidence of two or more strokes, or a single stroke that may lead to the dementia.

As yet, DSM-III-R do not seem to have diagnostic criteria for ADC, however Pratt (1991) suggests that one would need "Clinical findings of disabling cognitive and/or motor dysfunction, a loss of behavioural developmental milestones, progressing over weeks and months in absence of concurrent illness." (p260) However it seems clinical evidence of HIV infection would also need to be confirmed. These results should be confirmed by CT scan or MRI for two reasons. Firstly, progressive multifocal leucoencephalopathy and cytomegalovirus encephalitis - both opportunistic viral infections - can mimic ADC. Secondly, ADC may vary in its presentation - it may be mild with only a few features, or profound, leaving a totally unresponsive patient. (Farthing, Brown and Staughton, 1988) The importance of a thorough assessment of a potential dementia patient is evidenced by the fact that ADC may often be misdiagnosed as AD (Kernutt et al, 1993) and that ADC may be the sole presenting feature of HIV infection. (Kernutt et al, 1993; Roth, 1989; Price and Brew, 1988; and Saura Walsh and Whipple, 1990).

**DURATION OF THE DISEASE**

Dementing diseases, due to their progressive decline, will shorten a person's life expectancy considerably. However the reasons for death differ. In AD, the cause of death is a gradual "fading away", whereas in MID it usually relates to disorders of arteries in other parts of the body so death often results from hypertensive problems, a
major heart attack or a major stroke (Jaques, 1988, p26). In ADC, the major cause of death is from opportunistic infections such as pneumonia (Pratt, 1991). From the point of medical detection, Schoenberg, Okazaki and Kokomen (1981) reported that 93% of general dementia sufferers survived one year, 14% survived 5 years and 16% survived ten years.

Taking AD, MID and ADC separately, it is difficult to provide a definitive disease duration, as there is little information on survival rates. The average life expectancy of a person with AD is three to ten years after recognition and diagnosis of the disease (Kernutt et al, 1993). Recent studies setting out to compare the survival rates of MID and AD sufferers have indicated that survival is far poorer for the sufferers of MID (Metter et al, 1993). There is surprisingly little information on the survival rates of MID sufferers, but it is safe to assume that they survive less than the ten years of AD sufferers. Early estimates of the survival of those with ADC, put it at one to nine months once diagnosis had taken place (Kernutt et al, 1993, Navia, Cho, Petito, Price, 1986) however this is lengthened in people being treated with Zidovudine (AZT). It is difficult to put a time frame on this population as individual differences in the speed of decline make accurate predictions difficult.

**TREATMENT**

No cure currently exists for these three forms of irreversible dementia. However, it is possible, through careful diagnosis and assessment to treat secondary signs and symptoms.

There is no known way to prevent, halt or slow the AD process. There have been many attempts, including mainly cholinergic neuro transmitter enhancement therapy however no large study has recorded clinically significant improvements in core dementia symptoms (Wesson Ashford et al, 1993). At one stage it was thought that people suffering from MID who were given cyclandate, isoxsuprine and dihydroergotoxine -
all of which appear to increase cerebral blood flow - helped to improve memory and intellectual capacity after several months continual treatment. Unfortunately, there appeared to be no correlation between degree of improvement in cerebral circulation observed in radio-circulograms and level of improvement in psychometric test scores. Naftidrofuryl, another substance, has also been tested, and it proved superior to placebo in two controlled studies (Silverstone and Turner, 1974).

To treat ADC is nigh on impossible without treating AIDS itself, and the drug AZT is primarily used for this. Of course it is eminently possible that one day we will be able to halt or even reverse the dementia process, however a fundamental difficulty with this type of treatment is that no therapy can reverse structural damage to the brain and by the time dementia has occurred it appears we can do little to alter the damage.

Management of the sufferer is all important while there is still no cure, and when a person presents with memory problems they should receive a full dementia evaluation (Wells, cited Wesson Ashford et al, 1993). This is generally carried out by a multidisciplinary team who will carry out a psychiatric and physical examination, and a social and environmental assessment prior to treatment. This is used to diagnose medical conditions that may be contributing to the memory problems, to determine whether the problem is treatable or untreatable and to determine the severity of the dementia if it exists. The team will then make recommendations to the family or guardian as to:

1. whether current medications should be stopped
2. whether current medications should be changed
3. whether a change in living situations is appropriate (ie institutionalisation).
4. modification of living situation
5. the feasibility of respite care. (Mace, 1988)
Treatment of clinical features of dementia is most often carried out by chemical means, but it is important to ensure that before a medication is added to a treatment program that there will be no unwanted side effects. There are many types of medications used to treat the features of AD such as antipsychotics to decrease paranoia, belligerence and agitation, however Thornton et al (1986) suggest that these should be used with care due to the fact that demented people are especially susceptible to orthostatic hypertension. Antidepressants are useful, however assessment to discount pseudodementia is important. Tranquillisers may also be useful to treat minor sleep disturbances over a short period of time.

For those with MID, medications described above may be useful, as well as drugs to treat hypertension. The referring medical officer must remember in a case like this that several of the medications used to treat hypertension may have an adverse affect on mental function. Beta blockers that cross the blood-brain barrier more slowly may be safer more because they affect cognitive function variably (Thornton, et al, 1986).

No specific treatment for HIV dementia currently exists, although there has been short improvement in patients whose neurological changes were treated with the drug AZT (Price and Brew, 1988, Grant and Heaton, 1990). This antiviral drug has certain properties allowing it to cross the blood-brain barrier. Preliminary reports show that AZT may be able to reverse some of the damage caused by ADC (Aggleton, Homans, Mojsa, Watson and Watney, 1989) One of the side effects of AZT however, appears to be amnesia, but the severity of this is not reported (The Albion St (AIDS) Centre, 1988). Other medication used to treat the myriad of infections of AIDS, while not treating the ADC, will help to make the person more comfortable. At present there are many newer anti-retro-viral drugs in various stages of development, with clinical trials to begin shortly.
INSTITUTION BASED CARE

A potential problem may occur for people suffering from AIDS and ADC in that at present hostels and nursing homes are primarily used for the care of people suffering from AD and MID. In the future, the introduction of newer drugs to battle the AIDS virus will mean that patients will be living longer (although their quality of life is questionable). As such, the incidence of ADC will rise and it may be that nursing homes will take the brunt of long term care, as many are specifically set up to deal with the management of dementia. Long term care facilities such as nursing homes may be a less attractive alternative to home care, but they are far less expensive than private nursing care twenty four hours a day.

At present, there are very few long term care facilities caring for people with AIDS and ADC, but this will need to change as the affected population grows. To do this, there are several obstacles that would need to be overcome first. There is fear or apprehension among staff in caring for an ADC patient, in institutions as well as in the field, along with staff ignorance and bias. Even if staff are registered, (and one would assume, therefore educated) there still may be problems as staff fear transmission. Staff turnover may well rise as a result of family members becoming anxious about the social stigma attached to HIV and related illness. This pressure may also result in greater stress levels among staff members. A general fear of AIDS may also be seen to be a deterrent in staff recruitment. Staff may also be generally unwilling to care for those residents with ADC. Of course for staff to be working with ADC, it is vital that they understand the disease, and therefore understand that transmission is very specific. If transmission occurs the effect is devastating - the question should be asked whether or not it is fair to expose anyone to that risk.

Level of resident population understanding of AIDS or ADC needs to be investigated, with resident concerns and reactions being taken into account (Linsk, Cich and Cianfrani, 1993). Public paranoia about AIDS and HIV is a very real issue - anyone
who has worked in a nursing home knows that news spreads like wildfire, and there may be residents moved out of the nursing home because of this. It is important that issues such as these be looked at by nursing home administration, management and owners, and strategies set up to deal with the problem sooner rather than later.

As was suggested, the incidence of dementia will increase as Australia's population ages and it is safe to suggest that as it does, the numbers of people with AD, MID and ADC will increase as well. As we learn more about the pathology of dementia it is possible that treatments will become more sophisticated and a cure may be found. It is vital for those administering and managing long term care facilities to start to think about the placement of ADC patients and to set up guidelines to govern these.

CONCLUSION

There are three types of dementia discussed in this paper - Alzheimers disease, multi infarct dementia and the AIDS dementia complex. Although all deal with the brain, each have different clinical features, pathology, duration and treatment and as such, behavioural and medical management will also differ. As yet, no cure is available for any of the three, but it is possible to treat the associated symptoms such as depression with careful diagnosis and assessment. This must then be followed by professional behavioural and chemical management of the person. The question of the placement of ADC sufferers in nursing homes is one that needs discussion and may require education of not only staff, but also other residents as well as their families and friends.
FOOTNOTES

(1) Whereas AD is the major cause of dementia in Australia, Europe and North America, MID is the major cause in Japan and China. This difference could well be diet as opposed to racially related - the traditional Japanese diet tends to produce more strokes than the traditional Western diet (Jorm, 1990).

(2) These changes are also found in the brains of undemented people, although in far smaller numbers (Jacques, 1988).
GLOSSARY

ACETYL-CHOLINE - a neurotransmitter released at the synapse of parasympathetic nerves and neuromuscular joints (Oxford Dictionary of Nursing, 1994).

AGNOSIA - Total loss of the ability to recognise familiar object or persons as a result of organic brain damage (Oxford Dictionary of Nursing, 1994).

APHASIA - an abnormal neurologic condition in which language function is defective or absent because of injury to certain areas of the cerebral cortex (Oxford Dictionary of Nursing).

APRAXIA - an impairment in the ability to perform purposeful acts or manipulate objects (Oxford Dictionary of Nursing, 1994).

CYTOMEGALOVIRUS ENCEPHALITIS - a herpes like virus causing inflammation of the brain (Mosbys Medical, Nursing and Allied Health Dictionary, 1990).

DIAPEDESIS - migration through the of blood capillaries into the tissue spaces. A reaction of tissue to injury (Oxford Dictionary of Nursing, 1994).

5-HYDROXYTRYPTAMINE - also known as serotonin, it is a central nervous system neurotransmitter (Mosbys Medical, Nursing and Allied Health Dictionatry, 1990).

GAMMA-AMINO BUTYRIC ACID - an ammino acid with neuro transmitter activity found in the brain (Mosbys Medical , Nursing and Alllied Health Dictionary, 1990).

HEMOSIDERIN - an iron rich pigment that is the product of red cell haemolysis (Oxford Dictionary of Nursing, 1994).

LACUNE - a small infarction or haemorrhage less than 1cm in diameter (Oxford Dictionary of Nursing, 1994).

LIPOHYALINOSIS - the end stage of subintimal accumulation of hyaline materials. The media becomes split and dissected and microaneurysms form. Diapedesis occurs through the wall of the vein, and hemosiderin filled macrophages accumulate. The lumen of the vessel becomes stenotic then occluded with lacunar infarcts developing (Mosbys Medical Nursing and Allied Health Dictionary, 1990).

MEDIA - the mid layer of the wall of a vein or artery (Oxford Dictionary of Nursing, 1994).
MULTI-FOCAL LEUCOENCEPHALOPATHY - a disease caused by a virus which spreads throughout the white matter of the brain. Deterioration is usually rapid with symptoms of confusion, gross short term and long term memory impairment as well as motor retardation (Albion St (AIDS) Centre, interview, 20 October, 1994).

ORTHOSTATIC HYPERTENSION - hypertension incurred when a person stands (Oxford Dictionary of Nursing, 1994)

SUBINTIMAL - the area beneath the membrane that lines a blood vessel (Oxford Dictionary of Nursing, 1994).

SUCLI - the gaps between the convolutions in the brain (Oxford Dictionary of Nursing, 1994).

STENOTIC - an abnormal narrowing of the opening of a blood vessel (Mosbys Medical, Nursing and Allied Health Dictionary, 1990).
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