

ALZHEIMER'S DISEASE

We are currently living in the age of technology. Our advancements in the past few decades overshadow everything learned in the last 2000 years. With the elimination of many diseases through effective cures and treatments, Canadians can expect to live a much longer life than that of their grandparents. In 1900 about 4% of the Canadian population was over the age of 65. In 1989 that figure tripled to 12% and the government expects that figure to rise to 23% by the year 2030 (Medical,1991,p.13). This increase has brought with it a large increase in diseases associated with old age. Alzheimer's dementia (AD) is one of the most common and feared diseases afflicting the elderly community. AD, once thought to be a natural part of aging, is a severely debilitating form of mental dementia. Although some other types of dementia are curable or effectively treatable, there is currently no cure for the Alzheimer variety.

A general overview of Alzheimer's disease including the clinical description, diagnosis, and progression of symptoms, helps one to further understand the treatment and care of patients, the scope of the problem, and current research.

The clinical definition of dementia is "a deterioration in intellectual performance that involves, but is not limited to, a loss in at least 2 of the following areas: language, judgement, memory, visual or depth perception, or judgement interfering with daily activities" (Institute,1996, p.4).

The initial cause of AD symptoms is a result of the progressive deterioration of brain cells (neurons) in the cerebral cortex of the brain. This area of the brain, which is the largest and uppermost portion, controls all our thought processes, movement, speech, and senses. This

deterioration initially starts in the area of the cortex that is associated with memory and then progresses into other areas of the cortex, then into other areas of the brain that control bodily function. The death of these cells causes an interruption of the electrochemical signals between neurons that are a key to cognitive as well as bodily functioning.

Currently AD can only be confirmed at autopsy. After death the examined brain of an Alzheimer victim shows two distinct characteristics. The first is the presence of neuritic plaques in the cerebral cortex and other areas of the brain including cerebral blood vessels. These plaques consist of groups of neurons surrounded by deposits of beta-amyloid protein. The presence of

these plaques is also common to other types of dementia.

The second characteristic, neurofibrillary tangles, is what separates AD from all other forms of dementia. Neurofibrillary tangles take place within the disconnected brain cells themselves. When examined under a microscope diseased cells appear to contain spaghetti-like tangles of normally straight nerve fibers. The presence of these tangles was first discovered in 1906 by the German neurologist Alois Alzheimer, hence the name Alzheimer's disease.

Although the characteristics listed above are crucial to the diagnosis of AD upon death, the clinical diagnosis involves a different process. The diagnosis of AD is only made after all other illnesses, which may have the same symptoms, are ruled out. The initial symptoms of AD are typical of other treatable diseases therefore doctors are hesitant to give the diagnosis of Alzheimer's in order to save the patient from the worsening of a treatable disease through a misdiagnosis. Some of the initial symptoms include an increased memory loss, changes in mood, personality, and behavior, symptoms that are common of depression, prescription drug conflict, brain tumors, syphilis, alcoholism, other types of dementia, and many other conditions.

The onset of these symptoms usually brings the patient to his family doctor. The general practitioner runs a typical battery of urinalysis and blood tests that he sends off to the lab. If the tests come back negative, and no other cause of the symptoms is established, the patient is then referred to a specialist. The specialist, usually a psychiatrist, will then continue to rule out other possible illnesses through testing. If the next battery of tests also comes back negative then the specialist will call on a neurologist to run a series of neurological examinations including a PET and CAT scan to rule out the possibility of brain tumors. A spinal tap is also performed to determine the possibility of other types of dementias. The patient will also undergo a complete psychiatric evaluation. If the patient meets the preliminary criteria for AD an examination of the patient's medical history is also necessary to check for possible genetic predispositions to the disease.

The psychiatric team finally meets with the neurological team to discuss their findings. If every other possible disease is ruled out, and the results of the psychiatric evaluation are typical to that of a person with AD, the diagnosis of Alzheimer's disease is given.

The initial symptoms of AD are usually brushed off as a natural part of aging. The myth that a person's memory worsens over time is just that - a myth (Myers, 1996, p.100-101). AD's victims are mostly over the age of 65 and many delay treatment by attributing their problems to

age. A victim might forget a well known phone number or miss an important appointment. These symptoms eventually escalate to the total disintegration of personality and all patients end up in total nursing care.

In descending order, the patient goes from (1) decreased ability to handle a complex job to (2) decreased ability to handle such complex activities of daily life as (3) managing finances, (4) complex meal preparation and (5) complex marketing skills. Next comes (6) loss of ability to pick out clothing properly, (7) or to put on clothing properly, followed by (8) loss of ability to handle the mechanics of bathing properly. Then (9) progressive difficulties with continence and (10) toileting occur, followed by (11) very limited speech ability and (12) inability to speak more than a single word. Next comes (13) loss of ambulatory capability. Last to go are such basic functions as (14) sit up, (15) smile and (16) hold up one's head (Brassard,1993,p.10).

The average time from diagnosis to inevitable death is 8 years. The family of the victim is usually able to care for the victim for an average period of about 4 years (Alzheimer's, 1996,p.44). During the progression of the disease between 10% and 15% of patients hallucinate and suffer delusions, 10% will become violent and 10% suffer from seizures (Alzheimer's,1996,p.46).

Once a person is diagnosed as having AD, an assessment is made of the disease's stage of progression and of the strengths and weaknesses of the victim and the victim's family. There are different types of assessments available to evaluate the level of dysfunction of the patient. Based on one of these assessments a care plan is put together by a team consisting of a family member, a paid or unpaid care provider, and the victim's physician. Throughout the progression of the disease, and depending on the needs of the patient, a wide range of expensive medication, such as psychoactive drugs to lift depression and sedatives to control violence, may be required.

Unfortunately, although a wide range of treatments have been tested, most prove to be ineffective. At the beginning of the disease the family is able to look after the patient without much effort. Frequently families will hire a care giver in order to alleviate some of the work.

Simple changes in the home can make life much easier for the sufferer, help them keep their self esteem, and prolong their stay at home. Examples of low-cost

modifications to the

environment include reducing the noise levels in the home (telephones, radios, voices, etc.); avoiding vividly patterned rugs and drapes; placing locks up high or down low on doors leading outside (AD sufferers are known to wander off); clearing floors of clutter; reducing the contents of closets in order to simplify choices (Alzheimer,1992, p.17). These costs are paid for by the victim's family. Many of these, and other more expensive modifications are introduced in long-term care settings. They help in maintaining the safety and security of the victim as well as reducing their confusion.

The patient's and the family's condition should be assessed every six months (Alzheimer,1992, p.21). In response to constantly changing needs, the aspects of care must be constantly modified. Other issues that usually arise during the care of the patient are assessment of the competence of the victim, power of attorney, and response to and prevention of abuse (Aronson,1988, p.124). Eventually the victim's condition deteriorates to the point where home care is no longer possible and they must be moved to a long-term care facility.

In Canada care, support and information for victims and their families comes from the health care system and the Alzheimer's Society of Canada. The care giver must obtain information and education about the disease in order to effectively care for the victim. During the course of the disease victims might wander, hallucinate, become suspicious. This behavior can place a large strain on the care giver as well as causing depression and deterioration of their own health (Aronson,1988, p.132). An AD support group is crucial to alleviating some of the stress on the care giver. Through a support group the care giver is given the emotional and practical help needed to accomplish the large task of looking after the victim for as long as possible.

Currently there are 300,000 persons in Canada with AD. This figure is more than that of Parkinson's disease, cancer and multiple sclerosis combined. With continuous growth in the percentage of Canadians over the age of 65, this figure could hit 700,000 by the year 2020 (Carlton,1996,p.17). These large and increasing figures translate into a large burden on the health care system.

Even when using the most conservative estimates of the average number of years spent in an institution and the number of afflicted Canadians, the costs to health care are immense. At \$33,000 (1989) per patient per year in an institution and with an average stay of three years until death, the cost of AD will amount to

\$3

billion over the next three years; and if the entry into the disease state remains constant, it will cost the Canadian taxpayer [an added] \$1 billion per year thereafter. (Brassard,1993,p.11)

There have been many studies that conclude that the number of incidences of AD is on the rise. A very high incidence was reported in a U.S. survey conducted in East Boston by the Harvard Medical school. It showed the incidence of AD to be 3% for people between the ages of 65-74, 18.7% for those between 75-84, and 47.2% for those over 84 (Evans,1989,p.4).

AD is a democratic disease. It affects persons of both sexes and all races and ethnic backgrounds. The major risk factors for AD are age and heredity. Persons with a high incidence of AD in their family history are most susceptible.

A specific subtype of AD exists that is solely connected to heredity. This subtype is known as Familial Alzheimer's disease (FAD). FAD is also known as Early Onset Alzheimer's disease, named so because its symptoms start to develop much earlier than in the regular sporadic type. Only 5%-10% of all cases are of this type. FAD is suspected when AD can be traced over several generations and there is a history of, among previously affected family members, a similar age of onset and duration of the disease (usually 4 years) . Approximately 50% of the children of an affected parent go on to develop the disease (Pollen,1993,p.89).

Much research has been conducted in an attempt to locate the gene that is responsible for FAD. Currently, researchers have isolated genes 1, 14, and 21 (Alzheimer's,1996,p.36), however, the evidence still remains inconclusive (Statement,1996, p.2). There is also a possibility that a specific genetic mutation merely puts a person at risk to the disease and AD is triggered by an external force e.g. a head injury.(Statement,1996,p.4). Finding the specific location of the gene will pave the way for a diagnostic or even predictive test for FAD. Similar genetic tests already exist for cystic fibrosis and muscular dystrophy. Locating the AD gene will also allow scientists to study why the particular gene is not functioning properly and may give clues to treatment and cure. The long term goal of this research is the same as that of any other genetic research and that is gene therapy - which is the possibility that science could one day alter our genetic make-up.

The other much more common type of AD is Sporadic Alzheimer's Disease (SAD). This includes all other types of the disease which are not linked to heredity. Genetic research is also playing a major role in the progress towards a diagnostic or predictive test for SAD. Recently, a gene involved in the transport of cholesterol has been identified to be associated

with AD. Apolipoprotein E is located on chromosome 19 and seems to contribute to the susceptibility of a person to AD (Statement, 1996, p.6). The gene exists in three different forms or alleles (Apo E 2,3,4) and each person has a combination of two of the three. Thus an individual can have any one of the following combinations: Apo E 2/2, 3/3, 4/4, 2/3, 3/4 or 2/4. Researchers have found a relationship between the number of copies of the 4 allele and the person's probability of developing the disease.

Source: Institute for Brain Aging

FIGURE 1 illustrates an analysis of the proportion of individuals remaining normal at increasing ages for two, one, or zero copies of Apo E 4. For example a 75 year old individual with the Apo E 4 genotype has approximately a 20% chance of remaining normal; Apo E 3/4 or 2/4, 40%; 2/2, 3/3 or 2/3, a 75% chance. For many years, scientists believed that aluminum was at the root of AD. High levels of aluminum were detected in the areas surrounding the beta-amyloid plaques associated with neural atrophy (Pollen, 1990, p.77). Recently, however, this theory has been abandoned. Scientists concluded that the build-up of aluminum was a direct result of the wrongful use of a particular test agent employed in the studies (Brown, 1992, p.6). Some of the current pursuits of research are in the areas of viral infection, malfunction of the immune system, and chemical imbalances. One of the hardest theories to disprove is that AD is the result of a slow acting virus present at birth (Carlton, 1996, p.13). Others believe that AD is an immune system disorder. Support for this theory comes from the presence of beta-amyloid plaques identical to those found in AD brains in the post mortem examinations of immuno-deficiency disease victims (Alzheimer's, 1996, p.22). The detection of lower neurotransmitter substances such as acetylcholine, serotonin, norepinephrine and somatostatin in AD sufferers forms the basis of another theory that says AD is brought on by a chemical imbalance in the brain. Treatment of patients with drugs

that block the
break down of neurotransmitter substances in the brain have been met with limited
success
(Brassard,1993,p.16).

AD is an enormous social and economic problem. As the population ages, the
number of
victims will steadily increase, imposing a massive burden on the health care
system. Until a cure
and effective treatment are found AD will remain a terrible disease that slowly
eats away at that
which is the very essence of a person, their mind, leaving in its wake a mere empty
shell of that
person. It takes away from all of us the insightful wisdom of society's most
prized possession -
the elderly.

References

- Alzheimer Society of Canada.(1992).Guidelines for Care.Toronto: Alzheimer Society
of Canada.
- Alzheimer's Disease Education and Referral Centre.
(1996).Internet.http:\\www.alzheimers.
org/adear.drct.txt
- Aronson, Miriam.(1988).Understanding Alzheimer's disease.New York: Scribner's.
- Brassard, Daniel.(1993).Alzheimer's Disease.Ottawa: Library of Parliament, Science
and
Technology Division.
- Brown, Phyllida.(1992, November 7).Alzheimer's May Not be Linked to Aluminum.New
Scientist
Supplement,p.6.
- Carlton University Department of Health Sciences Freenet.
(1996).Internet.http:\\www.nct.carlton
ca/fp/social.services/alzheimer/disease.dir
- Evans, Denis, et al.(1989).Prevalence of Alzheimer's Disease in a Community
Population of
Older Persons.Journal of the American Medical Association,272(15),1152.
- Institute for Brain Aging.(1996).Internet.http:\\www.128.200.55.17/aboutad.html
- Medical Research Council of Canada.(1991).Presidents Report 1989-1990.
- Myers, David.(1996).Exploring Psychology.New York: Worth.
- Pollen, Daniel.(1990).Hannah's Heirs: The Quest For the Genetic Origins of

Alzheimer's
Disease.London:Oxford University Press.

Statement on Use of Apolipoprotein E Testing for Alzheimer's Disease.
(1996).American College
of Medical Genetics/American Society of Human Genetics Working Group on ApoE and
Alzheimer's Disease.Internet.<http://www.faseb.org/genetics/asng/policy/pot>

