Chapter-I

INTRODUCTION

Brain is as essential a part of the body as is air for “Prana”. Each and every system including heart and lungs is controlled by the brain. Sensation, perception, motor movements and even cognition is controlled and coordinated by brain.

Human brain is like a computer, having both the hardware and the software systems. Brain organs are the hardware and the cognitive processes are the software. The intact human brain may be considered to be a highly programmable system of “hardware”, and psychosocial experience in both its developmental and current aspects are functionally equivalent to “software”. Such an analogy has widespread implications throughout the domain of abnormal psychology as for example in posing a problem for exclusively biological interpretations of the “functional” (non organic) psychoses – but it will focus on so called organic mental disorders. Using the analogy, these disorders, by definition, have hardware defects as their primary cause. In other words, in such situations the brain cannot perform the physical operations called for in its “design” by virtue of a breakdown in one or another (or several) of its components. The direct “symptoms” of such a breakdown should be both limited in extent and, within reason, predictable from knowledge of how these components “work”. In fact they should be similar in general character to the symptoms, such as memory defects.

A breakdown in the brain’s hardware will necessarily have pervasive effects on the processing of software. Indeed, in the case of extensive hardware damage, much or perhaps most previously loaded information may be lost because the structural components in which it had been encoded are no longer operative, new information for the same reason fails to be adequately loaded. Such a condition is known clinically as dementia (Carson, Butcher and Mineka, 1998).

In the text revision of the fourth edition of Diagnostic Statistical Manual of Mental Disorders (DSM-IV-TR) three groups of disorders – delirium, amnestic disorders, and dementia– are characterized by the primary symptoms common to all the disorders, which is an impairment in cognition (as in memory, language, or attention). Although
DSM-IV-TR acknowledges that other psychiatric disorders can exhibit some cognitive impairment as a symptom, cognitive impairment is the coordinal symptom in delirium, amnestic disorders, and dementia. Within each of these diagnostic categories, DSM-IV-TR delimits specific types.

**DSM-IV Classification of Cognitive Disorders:**

For each of the three major groups – delirium, amnestic disorders, and dementia – there are subcategories based on etiology. They are summarized as follows:

**Delirium:**

Delirium is marked by short term confusion and changes in cognition. There are four subcategories based upon several causes: (i) general medical condition, e.g. infection; (ii) substance induced, e.g. cocaine, opioids, phencyclidine (PCP); (iii) multiple causes, e.g. head trauma and kidney disease; and (iv) delirium not otherwise specified, e.g., sleep deprivation.

**Amnestic Disorders:**

Amnestic disorder is marked by memory impairment and forgetfulness. There are three subcategories: (i) caused by medical condition (hypoxia); (ii) caused by toxin or medication, e.g., marijuana, diazepam; and (iii) amnestic disorder not otherwise specified.

**Dementia:**

Dementia is marked by severe impairment in memory, judgment, orientations, and cognition. There are six subcategories: (i) dementia of the Alzheimer’s type, which usually occurs in persons over 65 and is manifested by progressive intellectual disorientation and dementia, delusions, or depression; (ii) vascular dementia (including multi-infarct dementia), caused by vessel thrombosis or hemorrhage; (iii) dementia due to other general medical conditions, e.g. human immunodeficiency virus (HIV) disease, head trauma, Pick’s disease, Parkinson’s disease, Huntington’s disease, Crutzfeldt – Jakob disease (caused by a slow growing transmittable virus); (iv) substance induced persisting dementia, caused by toxin or medication, e.g., gasoline fumes, atropine; (v) dementia due to multiple etiologies; and (vi) dementia not otherwise specified (if cause is unknown).
The term *dementia* is used in different ways by neurologists, psychiatrists, and geriatricians. Many psychiatrists employ the term to describe only these patients with progressive and irreversible diffuse structural brain damage. Used in this sense dementia is synonymous with untreatable progressive disintegration of the brain, with the consequence that the application of the term precludes any hope of a treatable cause or therapy. Yet, in practice, the term dementia is applied to describe a syndrome of generalized, global loss of higher mental function, irrespective of its cause.

As mentioned by Carson, Butcher and Mineka (1998) *Dementia* has as its essential feature a noteworthy decrement, or deterioration, in intellectual functioning occurring after the completion of brain maturation, that is after about 15 years of age.

The word “dementia”, is derived from the Latin word “*demens*”, which means ‘being out of one’s mind’ (Mahendra, 1984).

“The word “dementia” denotes a progressive decline in mental function, including acquired intellectual skills. It can be caused by many abnormal progresses and therefore, usually lacks diagnostic specificity. Some of the potentially treatable causes of dementia are infections of the brain and the meanings, nutritional disease (vitamin deficiency), endocrine and metabolic disease, intracranial mass lesions, chronically increased intracranial pressure, normal pressure hydrocephalus, and cerebrovascular disease (Cote, 1981).

In terms of psychology, *Dementia* is a gradual deterioration of brain functioning that affects judgment, memory, language, and other advanced cognitive processes caused by several medical conditions or by the abuse of drugs or alcohol that cause negative changes in cognitive functioning (Scharfetter, 1980; Kolb and Brodie, 1982; Heston, 1983; Durands and Barlow, 2000; Martin, Elaine, and David, 2000).

In the text revision of the fourth edition of Diagnostic and Statistical Manual of Mental Disorder (DSM-IV-TR), *dementia* is “characterized by multiple cognitive defects that include impairment in memory” without impairment in consciousness. The cognitive functions that can be affected in dementia include general intelligence, learning and memory, language, problem solving, orientation, perception, attention and concentration, judgment, and social abilities. The person’s personality is also affected.
Early in the course of this disorder, an individual is alert and fairly well attuned to events in the environment. Episodic (memory for events), but not necessarily semantic (language and concept), memory functioning is typically affected in the early stages, especially memory for recent events (Arie, 1973; Cummings and Benson, 1983). Patients with dementia also show increasingly marked deficits in abstract thinking, the acquisition of new knowledge or skills, visuospatial comprehension, motor control, problem solving, and judgment (Strub and Black, 1988). Personality deterioration and loss of motivation accompany these other deficits. Normally, dementia is also accompanied by impairment in emotional control and in moral and ethical sensibilities (Albert, Feldman, and Wills, 1974).

**Diagnosis and Symptoms:**

There are many bases of diagnosis of *dementia*. As already described Essential features of dementia are a decrement in the intellectual functioning occurring after brain maturation of sufficient severity to interfere with occupational or social performance or both. The cognitive deficit always involves memory and usually there is also marked evidence of impairment associated with thinking, learning new skills, problem solving, and adjustment. There are often also personality changes or impairment and impulse dyscontrol (Sakles and Balis, 1978).

Somewhat varying diagnostic standards are described in the Diagnostic and Statistical Manual, Vol. 4 (DSM-IV) and by the National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer’s Disease and Related Disorders Association, NINCDS-ADRDA (McKinnon, Drachman, Folstein, Katzman, Price, and Stadlan, 1984).

Following is the table depicting diagnostic criteria for dementia as given by DSM-IV and NINCDS/ADRDA:
### Table 1: Showing the diagnostic criteria for Dementia.

<table>
<thead>
<tr>
<th>Criterion</th>
<th>DSM-IV</th>
<th>NINCDS/ADRDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory impairment</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>Impairment of additional area of cognition (language, construction, praxis, or executive functioning).</td>
<td>R</td>
<td>D</td>
</tr>
<tr>
<td>Confirmed on mental status tests</td>
<td>NS</td>
<td>R</td>
</tr>
<tr>
<td>Impaired/decline in social occupational function</td>
<td>R</td>
<td>NS</td>
</tr>
<tr>
<td>State of consciousness unclouded</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>Evidence of specific organic factor etiologically related to the disorder or absence of condition other than organic mental syndrome</td>
<td>R</td>
<td>NS</td>
</tr>
</tbody>
</table>

R = Required, D- Desirable but not required, NS = Not specified.
Source: Adapted from Rebok and Folstein (1993).

So there are some common agreements about diagnosis of dementia (Arie, 1973). These are the loss of cognitive and intellectual functioning that is acquired and it involves multiple area of cognitive impairment.

A diagnosis of dementia, according to DSM-IV-TR, requires that the symptoms result in significant impairment in social or occupational functioning and that they represent a significant decline from a previous level of functioning.

Accordingly the **core criteria** for dementia may be summarized as follows:

1. Memory impairment (inability to learn new information and recall previously learnt material).
2. One or more of the following cognitive disturbances:
   a. Deterioration of language function (speech becomes empty or vague; comprehension of spoken or written language may deteriorate).
   b. Impaired ability to execute motor activities despite intact motor function, sensory function, and comprehension of the required task (e.g. impaired ability to pantomime using an object, such as combing hair).
c. Failure to recognize or identify objects despite intact sensory function (e.g. loss of the ability to recognize objects such as chairs or pencils despite normal visual acuity).

d. Disturbance in executive functions such as planning, organizing, sequencing and abstracting (executive dysfunction includes inability to recite the alphabet or state as many animals as possible in 1 minute).

**Categorization of Dementia based on etiological factors:**

There are six types of dementia according to DSM-IV-TR:

1. **Dementia of the Alzheimer's Type:**

   In 1907, Alois Alzheimer first described the condition that later assumed his name. It is a most common type of dementia. It is caused by a loss of brain cells. In this disease, cells in the areas of the brain that control memory and mental functions are destroyed by abnormal protein deposits in the brain. The brain of a person with Alzheimer disease is also clogged with two abnormal structures (called neurofibrillary tangles and senile plaques). However, why these structures develop is still unknown. People with Alzheimer disease also have lower-than-normal levels of brain chemicals called neurotransmitters that control important brain functions. Two neurotransmitters seem to play a role in Alzheimer's disease: acetylcholine and glutamate. Acetylcholine (ACh) activates muscles and helps with arousal, short-term memory, and learning. Individuals with AD have low levels of ACh. Some research suggests that plaques may be one of the reasons for low levels of ACh because they increase the activity of a chemical called acetylcholinesterase, which is involved in breaking down ACh. Too much acetylcholinesterase has the overall effect of decreasing ACh levels, which contributes to the characteristic symptoms of AD. Glutamate is the most common neurotransmitter in the brain and is involved in learning and memory. As the brain cells of someone with Alzheimer's disease die, they release excess amounts of glutamate. The excess glutamate becomes harmful because it over-stimulates healthy brain cells (a phenomenon called excitotoxicity), causing them to become damaged or to die (Carrie and Natalie, 2008).
DSM-IV-TR Diagnostic Criteria for Dementia of the Alzheimer's Type

A is same as given in core criteria 1 and 2.

B. The cognitive deficits cause significant impairment in social or occupational functioning and represent a significant decline from a previous level of functioning.

C. The course is characterized by gradual onset and continuing cognitive decline.

D. The cognitive deficits in Criteria A1 and A2 are not due to any of the following:
   1) other central nervous system conditions that cause progressive deficits in memory and cognition (e.g., cerebrovascular disease, Parkinson's disease, Huntington's disease, subdural hematoma, normal-pressure hydrocephalus, brain tumor)
   2) systemic conditions that are known to cause dementia (e.g. hypothyroidism, vitamin B12 or folic acid deficiency, niacin deficiency, hypercalcemia, neurosyphilis, HIV infection)
   3) substance-induced conditions

E. The deficits do not occur exclusively during the course of a delirium.

F. The disturbance is not better accounted by another Axis 1 disorder (e.g., major depressive disorder, schizophrenia).

(2) Vascular Dementia:

It is the second most common type of dementia. It is caused by poor circulation of blood to the brain. In this type of disease there occur lots of mini strokes (or infarcts) which cut off the blood supply to part of the brain. The mini strokes that cause vascular dementia are after so slight that they cause no immediate symptoms, or they may cause same temporary confusion. However, each stroke destroys a small area of cells in the brain by cutting off its blood supply and the cumulative effect of a number of mini strokes is often sufficient to cause vascular dementia.

Vascular dementia and Alzheimer's disease frequently occur together and they may often act in combination to cause dementia.

DSM-IV-TR Diagnostic Criteria for Vascular Dementia:

A and B are same as criteria for Alzheimer's disease.
C. Focal neurological signs and symptoms (e.g., exaggeration of deep tendon reflexes, extensor plantar response, pseudobulbar palsy, gait abnormalities, weakness of an extremity) or laboratory evidence indicative of cerebrovascular disease (e.g., multiple infarctions involving cortex and underlying white matter) that are judged to be etiologically related to the disturbance.

D. The deficits do not occur exclusively during the course of a delirium.

There are a number of different types of vascular dementia. But two most common of them are:

(a) **Multi-infarct dementia**: It is the most common form of vascular dementia. It is caused by a number of small strokes called mini-strokes or Transient Ischaemic Attacks (TIA). The strokes cause damage to the cortex of the brain (the area associated with learning, memory and language). A person with Multi-infarct dementia is likely to have better insight into their condition in the early stages than people with Alzheimer’s disease. Parts of their personality may remain relatively intact for longer. Symptoms may include severe depression and epilepsy.

(b) **Binswanger’s disease (Subcortical vascular dementia)**: This was thought to be rare, but is now being reassessed, and may be relatively common. ‘White matter’ deep within the brain is affected in this type of vascular dementia. It is caused by high blood pressure, thickening of the arteries and inadequate blood flow. Symptoms often include slowness and lethargy, difficult walking, emotional ups and downs, lack of bladder control early in the course of the disease, gradually progressive dementia developing later, most people with this disease have, or have had, high blood pressure.

(3) **Dementia due to other general medical condition:**

DSM-IV-TR lists six specific causes of dementia. These include:

**Dementia due to HIV disease**, because the virus has been found in the brain of the people with HIV as early as two days after initial infection.
**Dementia due to Head Trauma**, caused due to damaged brain cells in accidents, falls, assaults, gunshot wounds or beatings, or from activities such as boxing without protective gear.

**Dementia due to Parkinson's disease**, which may develop late in some patients and is a disease of basal ganglia.

**Dementia due to Huntington's disease**, cause wasting of certain types of brain cells that control movement as well as thinking.

**Dementia due to Pick’s disease**, due to damages of cells in the front part of the brain. Behavior and personality changes usually precede memory loss and language problems.

**Dementia due to Creutzfeldt-Jakob disease**, caused by infection agents called prions, invade and kill brain cells. It leads to behavior changes and memory loss.

A seventh category is also given. It allows the clinicians to specify other non-psychiatric medical condition associated with dementia.

**DSM-IV-TR Diagnostic Criteria for Dementia Due to other general medical conditions:**

A and B are same as criteria for Alzheimer’s disease.

C. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct physiological consequence of any of the above described general medical condition other than Alzheimer’s disease or cerebrovascular disease.

D. The deficits do not occur exclusively during the course of a delirium.

(4) **Substance induced persisting dementia:**

The specific substances that DSM-IV-TR cross-references are alcohol; inhalant; sedative, hypnotic, or anxiolytics; and other or unknown substances.

**DSM-IV-TR Diagnostic Criteria for Substance-induced Persisting Dementia:**

A and B are same as criteria for Alzheimer’s disease.

C. The deficits do not occur exclusively during the course of a delirium and persist beyond the usual duration of substance intoxication or withdrawal.
D. There is evidence from the history, physical examination, or laboratory findings that the deficits are etiologically related to the persisting effects of substance use (e.g., a drug of abuse, a medication).

(5) **Dementia due to multiple etiologies:**

Below table is showing the diagnostic criteria for Dementia due to Multiple Etiologies:

**DSM-IV-TR Diagnostic Criteria for Dementia due to Multiple Etiologies:**

A and B are same as criteria for Alzheimer’s disease.

C. There is evidence from the history, physical examination, or laboratory findings that the disturbance has more than one etiology (e.g., head trauma plus chronic alcohol use, dementia of the Alzheimer’s type with the subsequent development of vascular dementia).

D. The deficits do not occur exclusively during the course of a delirium.

(6) **Dementia not otherwise specified:**

Below is the table describing the criteria for dementia not otherwise specified category:

**DSM-IV-TR Diagnostic Criteria for Dementia Not Otherwise Specified:**

This category should be used to diagnose a dementia that does not meet criteria for any of the specific types described in this section but the memory impairment and cognitive disturbances or deficits are observed.

An example is a clinical presentation of dementia for which there is insufficient evidence to establish a specific etiology.

The incidence of the dementia indicates that the two types of dementia are very common, i.e. *Alzheimer’s dementia* and *Vascular dementia*. These two types are having a large portion of cases of dementia.

Recent estimates have suggested that over 24.3 million people live with dementia worldwide, with 4.6 million new cases every year (one new case every 7 seconds). The number of people affected will be double every 20 years to 81.1 million by 2040. Most people with dementia live in developing countries (60% in 2001, rising to 71% by 2040). Rates of increase are not uniform; numbers in developed countries are forecast to increase by 100% between 2001 and 2040, but by more than 300% in India (Ferri, Prince,
Alzheimer’s disease is the most common type of dementia and it accounts for 50%-60% of all cases. It affects about 3% of all people between ages 65 and 74, about 19% of those between 75 and 84, and about 47% of those over 85. It predominantly affects the elderly. Slightly more women than men are affected with Alzheimer’s disease (Liston, 1979; and Johansson, 1991).

The second most common form of dementia is vascular dementia, including multi-infarct type dementia. It is accounting for about 40% of all cases. It usually affects people between ages 50 and 75. It is slightly more common in men than women (Kay, 1995).

Some major differences between the two above mentioned forms are highlighted in the following table:

**Table 2: Showing the difference between Alzheimer’s disease and Vascular dementia:**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Alzheimer’s disease</th>
<th>Vascular dementia (including Multi-infarct dementia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>65-90</td>
<td>50-75</td>
</tr>
<tr>
<td>Sex most affected</td>
<td>Possibly females</td>
<td>Males</td>
</tr>
<tr>
<td>Nature of onset</td>
<td>Gradual</td>
<td>Abrupt</td>
</tr>
<tr>
<td>Course of disease</td>
<td>Progressive</td>
<td>Stepwise</td>
</tr>
<tr>
<td>Physical impairment</td>
<td>Few, appear late in life</td>
<td>Frequent</td>
</tr>
</tbody>
</table>

In the Vascular dementia, Multi-infarct dementia is the most common form. It is estimated that 10 to 20% of all dementias are caused by blood vessel narrowing (Hachinski, Lassen, and Marshall, 1974).

After viewing the incidence, it becomes very interesting to study with the most common forms of dementia. So, the present study is concentrated on the senile dementia of Alzheimer type (SDAT) and Multi-infarct type (MIT).
Cognitive changes associated with aging

Although epidemiological studies estimate that 1 to 5% of the population between 60-70 years old suffers from dementia, the incidence increases exponentially with age, so that by age 85 the prevalence of dementia in the population is estimated at between 18% and 85% (Johansson, 1991; Kay, 1995).

As mentioned by Comer (1996), dementia is age related and since the average life-span continues to increase, it can be expected the prevalence eventually to reach catastrophic proportions in the society unless preventive and curative treatments are developed soon. In the 1980, over 1.5 million persons displayed dementia. New cases during that year were around 3, 50,000. By the year 2020, the incidence will exceed 600,000 and the total number of persons with dementia is projected to be about 3.5 millions (Schoenberg, Kokmen, and Okazaki, 1987).

The literature on the psychology of aging is immense. All of it is complicated, much of it controversial (Shaie, 1974; Horn and Donaldson, 1976). There is now general agreement that aging produces gradual deterioration of many intellectual functions, including memory. When this impairment is moderate or severe, it is called “dementia”. The type of memory impairment that accompanies dementia is an overall reduction of memory. The general cause of memory difficulties in aging is apparently two kinds of processes. First, there is a normal deterioration of most function related to aging; second, there is a marked increase in other neuropathological conditions related to aging.

Old age presents many challenges. Socially, older people may feel increasingly isolated with adjustment to retirement and death of friends or a spouse. Physically, age brings the threat of increased ailments and chronic illness (Critchley, 1981). Cognitively, older people are often concerned with the possibility of memory problems and mental slowing. The quality of life of the elderly people is also affected negatively (Savage, Britton, Boton, and Hall, 1973; and Corsellis, 1982).

Although the positive effects of psychosocial variables on healthy aging have not been heavily researched, but some studies indicate that curiosity, social support, optimism, and a sense of control over one’s life contribute strongly to successful aging (Ory, Abeles, and Lipman, 1992).
Cross sectional studies (Kendrick and Post, 1967; Whitehead, 1975; and Inglis, 1979) indicated that stored knowledge in memory (long-term memory) and habitual ways of solving problems (Crystallized intelligence) resist the effects of aging. However, novel reasoning and the efficiency of solving new problems or responding to abstract ideas (fluid intelligence) decline with advanced age. The most reliable decline in age group comparison studies shows up in three areas of intellectual activity, all of which are considered fluid markers of intelligence: (i) new learning, (ii) abstract and complex new problem solving, and (iii) behavioural speed.

The aging brain undergoes visually apparent structural changes such as diminution in size, flattening of the cortical surface, and increasing amount of intracranial space (Berlin and Wallace, 1996). Concomitant changes take place at the neuro-anatomical and biochemical levels. Researches have reported some loss of neurons, changes in neuronal size, altered dendritic processes and an increased frequency of neurofibrillary tangles and senile plaques with normal aging (Cote, 1981).

Thus, brain—truly the master organ degenerates with the time. The above discussed mental disorders that sometimes accompany this brain degeneration and occur in old age are traditionally called senile dementias. Disorders associated with such earlier degeneration in life, of the brain are known as presenile dementias. Not only is the age of onset different in the presenile dementias, but they are also distinguished from the senile dementias by their different behavioral manifestations and tissue alterations. Etiologic factors in dementia are many and varied. They include degenerative processes that usually, but not always, affect older individuals.

**Quality of Life**

There is a lack of consensus as to how quality of life should be defined and measured (Arnold, 1991; and Lawton, 1991).

In 1995, the World Health Organization defined Quality of life as "the individual's perceptions of their position in life in the context of the culture and value system in which they live, and in relationship to their goals, expectations, and standards".

Quality of life is conceptualized as a composite of physical, psychological and social well-being of an individual, as perceived by the person or the group. A very important aspect of Quality of Life is the happiness, satisfaction or gratification
subjectively experienced which is often called as well-being (Diener, Diener, and Diener, 1995).

Quality of life provides a validated approach for expanding the definition of health to include other domains of physical, mental, and social well-being (Lawton, 1991; Patrick and Deyo, 2005; and Dixit, Mehrotra, and Singh, 2007).

It is believed that old age is a period of cumulative stress, deprivation and loss. The old people are unhappy than all the other age groups. The aged persons encounter several stresses including their deteriorating faculties, the high cost and increased need of medical care, and loneliness or alienation. All these affect the quality of life of the aged persons (Ramamurti, 1990). It becomes very horrible if the person is suffering from dementia. The person is unable to do anything without the help by other family members.

There have been many studies on the dimensions of quality of life in elders (Abbey and Andrews, 1986; Clark, 1988; Lawton, 1991; Moss, Lawton, and Glicksman, 1991; and Pearlman and Uhlmann, 1991). Lawton (1994) identified four overarching dimensions that contribute to quality of life in dementia: psychological well being (e.g. positive and negative affect), behavioral competence (e.g. cognitive and functional abilities), objective environment (e.g. caretakers and living situation), and perceived quality of life.

Dementia is an enormous public health issue, affecting 24.3 million people in the world and at least half of residents in long-term care facilities have dementia. Quality of life issues for this population are varied, which also include the non-cognitive issues of apathy, depression, agitation, sleep difficulties, loss of autonomy and social isolation. It has been said that “patients with Alzheimer’s disease do not die of the disease, they die with it from some other cause” (Boller and Duyckaerts, 1997). Across the spectrum of dementing illnesses from vascular dementia to Parkinson’s and Alzheimer’s diseases, depression is both a manifestation of the dementing process (Cummings, 1992) and the consequence of social isolation. In the view of some investigators, depression itself may be responsible for the precipitous declines in clinical course (Absher and Cummings, 1993).

There are a number of overlapping factors that may influence the quality of life in the elderly persons. These factors include physical, emotional, intellectual, and social
functioning, life satisfaction, health perceptions, economic status, leisure activities, cognitive functioning, and vitality and energy (Arnold 1991; Lawton, 1994; and Ranzijn and Luszcz, 2000). Stewart and King (1994) suggest that each domain needs content or subject area as well as a response dimension consisting of both behavioral states and subjective evaluations.

The most difficult population in which very bad quality of life issues is individuals with dementia, because the cognitive changes affect one's subjective experience and subsequent verbalization of quality of life issues. Several diseases cause development of progressive dementia, characterized by memory deficits and other cognitive dysfunctions. The most common of these diseases are Alzheimer's disease, vascular dementia (including Multi-infarct type), frontotemporal dementia (including Pick's disease), and dementia with Lewy bodies (Klein and Kowell, 1998).

Dementia has been found to be highly associated with agitation, depression, anxiety, dis-inhibition, and irritability (Banerjee, Smith, Lamping, Harwood, Foley, Smith, Murray, Prince, Levin, Mann, and Knapp, 2005). The consequences of agitation and sleep difficulties may also be dramatic. The need for sedating medications during the daytime leads to poorer social interaction and exacerbates a reversal of the sleep-wake cycle. This, in turn, leads to a greater use of hypnotics at bedtime, contributing to the steady decline into the vegetative stage of many dementias (Pearlman, and Uhlmann, 1991).

**Treatment of Dementia**

**Aims of Treatment:**

i)  **To improve memory and concentration:**

The memory difficulties seen in dementia of Alzheimer's type can be relieved for a short time in some people with drugs that prevent the breakdown of acetylcholine in the brain. These drugs are called 'cholinesterase inhibitors' and are through to work by increasing the levels of acetylcoline in the synapses. The drugs may be given that reduce the inactivation of the neurotransmitter acetylcholine and thus potentiate the cholinergic neurotransmitter, which in turn produces a modest improvement in memory and goal-directed thought. These drugs are most useful for persons with mild to moderate memory...
loss who have enough preservation of their basal forebrain cholinergic neurons to benefit from augmentation of cholinergic neurotransmission (Heston, 1983).

The serotonergic system is impaired and may contribute both to cognitive and non-cognitive symptoms in senile dementia of Alzheimer’s type (SDAT). Porter, Lunn, and O’Brien (2003) suggested that compromised serotonergic function may be an important contributor to cognitive decline in SDAT. Strategies targeting specific 5HT receptors may be helpful in SDAT.

ii) To reduce psychiatric problem:

Dementia is a complex syndrome with many symptoms other than memory loss or the loss of functional abilities. These symptoms include depression, anxiety, agitation and occasionally aggression, altered sleep and psychotic symptoms (include visual hallucinations) hearing voices or false smells or taste (Balas, 1999).

iii) To improve quality of life:

The aim of treatment in dementia is also to improve the negatively affected quality of life. There are so many techniques which can help in improving the quality of life of the patients of dementia. These include psychosocial therapies, physical therapies, drug therapies, and other psychological techniques.

Techniques of Treatment:

i) Psychosocial Approach:

Recent memory is lost before remote memory in most cases of dementia and many patients are highly distressed by clearly recalling how they used to function while observing their obvious deterioration at the most fundamental functioning. Patients identities begin to fade as the illness progresses, and they can recall less and less of their past. Emotional reaction ranging from depression to serve anxiety to catastrophic terror can stem from the realization that the sense of self is disappearing.

Zarit, Orr, and Zarit (1985) provided an excellent guide to psychological intervention for families affected by dementia. This approach combines psychotherapeutic services to caregivers with training designed to improve the skills of caregivers in managing problem behaviors.

Patients often benefit from a supportive and educational psychotherapy in which the nature and course of their illness are clearly explained. They may also benefit from
assistance in grieving and accepting the extent of their disability and from attention to self-esteem issues. Clinicians can also help patients find ways to deal with the defective ego functions, such as keeping calendars for orientation problems making schedules to help structure activities and taking notes for memory problems (Coffey, Cummings, Lovell, and Pearlson, 1994). There are several psychological techniques to help people cope with dementia. These include reality orientation which involves regularly reminding patients of information such as the day, date, season and place where they are. As the memory of distant events is rarely impaired, reminiscence therapy which encourages people to talk about the past may also help by bringing past experience into consciousness. Aromatherapy and music therapies are also thought to be beneficial, though there is no scientific evidence to support this (Wolfe and Herzberg, 1996; and Holmes and Ballard, 2004).

The general treatment approach to patients with dementia is to provide supportive medical care, emotional support for the patients and their families or caregivers, and pharmacological treatment for specific symptoms, including disruptive behavior.

ii) Drug Treatment Approaches:

Four drugs have shown at least modest benefit for Alzheimer’s disease: donepezil (Aricept), rivastigmine (Exelon), galantamine (Reminyl), and cognex. These medications usually produce a modest improvement in the treatment of moderate or severe Alzheimer’s disease by increasing the duration of acetylcholine. However, more research is needed to establish whether these are suitable for everyone and how serious are side-effects (Browning, 2004).

Tacrine treatment is an effective treatment of Alzheimer if used in the proper content of clinical guidelines. Lykotos, Corazzine, Steel, and Koraus (1996) studied the positive effect of tacrine in Alzheimer patients in a double-blind placebo study.

Other drugs tested for cognitive enhancing activity, are general cerebral metabolic enhancers, calcium channel inhibitors, and serotonergic agents. Some studies have shown that selegilive (Eldepryl), a selective type B monoamine oxidase (MAO_B) inhibitor, may slow the advance of this disease.
Memantine (Akatinol) protects neurons from excessive amounts of glutamate, which may be neurotoxic. Ondanstron (zofran), a 5-HT₃ receptor antagonist, is under investigation.

Ascorbic Acid is also found having cognitive enhancing properties in a study (Parle and Dhingra, 2003).

Besides this the drug treatment is used to alleviate or manage the associated psychotic symptoms. Some patients with dementia require medications to control severe psychiatric and behavioral problems. Clinician may prescribe benzodiazepines for insomnia and anxiety, antidepressants for depression, and antipsychotic drugs for delusions and hallucinations, but they should be aware of possible idiosyncratic drug effects in older people (such as paradoxical excitement, confusion, and increased sedation).

Other psychiatric drugs help with aggression and agitation, as many certain antiepileptic drugs (Charatan, 1985). The psychotic symptoms may be controlled by anti-psychotics (especially the newer atypical agents, olanzapine and quetiapine).

The depression associated with dementia can be alleviated with antidepressants. The psychotic symptoms may be controlled by anti-psychotics like olanzapine and quetiapine (Semke, 2006).

iii) Herbal Treatment Approach:

All the synthetic compounds like Donepezil, Rivastigmine, Galantamine, Tacrine DuP996, Ketanserin, Tryptophan, Velnacrinemaleate, Memantine, L- deprenyl, Cerebrolysin etc. are being practiced to treat dementia are having ill effects in the long run. These side effects include slower heart rates, fainting episodes, dizziness, nausea, sedation, drowsiness, sleep disturbance, and weight loss (Bartus, Dean, Beer, and Lippa, 1982). An alternative that is claimed to be safer is the discipline of Ayurveda, which has existed in India from millennia. One of the practices of ayurveda is to treat poor health with medicines obtained from herbal plants. These medicines are prepared from the leaves, roots, or some other parts of certain plants. Below are some herbal medicines which are being used for treating dementia:-

**Bacopa (Bacopa monnieri):** leaf extract, called Brahmi, is used in Ayurvedic or Indian medicine to improve brain function and learning. However, no scientific studies
have looked at bacopa to see whether it might help lessen symptoms of dementia. One study found that 300 mg per day for 12 weeks seemed to improve cognition in healthy people (Limpeanchob, Jaipan, Rattanakaruna, Phrompittayarat, Ingkaninan, 2008).

**Ginkgo biloba:** is the world’s oldest living tree species. It has been found to be helpful in the symptoms of cerebral vascular insufficiency including short-term memory loss and depression. Ginkgo extract enhances oxygen utilization and increases the uptake of glucose to maintain brain balance. It promotes the nerve transmission rate, improves the synthesis of brain neurotransmitters, and normalizes acetylcholine receptors in the hippocampus, the area of the brain most affected by Alzheimer’s disease (Pizzorno and Joseph, 1999). It shows the best evidence for treating early Alzheimer’s disease and other forms of dementia. It may be taken in a standardized extract of 40-50 mg three times per day. It is derived from the leaves of the ginkgo tree and contains several substances called ginkgolides (Ernst, and Pittler, 1999; and Diamond, Shiflett, and Feiwel, 2000).

**Huperzine A:** is a chemical made from the plant *Huperzia serrata*. It may improve memory in both vascular (including infarct dementia) and Alzheimer’s dementia. The usual dose is 200 mg twice a day (Jiang, Luo, and Bai, 2003; Zhang, Zheng, Yan, Wang, Tang, Gao, and Tang, 2008).

**Vinpocetine:** is derived from leaves of common periwinkle (Vinca minor) as well as the seeds of various African plants. It is also used for treating memory loss (Szatmari, and Whitehouse, 2003).

**Hawthorn (Crataegus monogyna):** It is another cognitive enhancer. It is also a circulatory tonic (Ameenah, 2005).

**Siberia ginseng (Eleutheracoccus senticosus) and American ginseng (Panax quinquefolium):** have also been shown to increase endurance and improve cerebral circulation (David and Traci, 2003).

One another popular herbal medicine used to treat impaired cognitive functioning is **Lemon balm (Melissa Officinalis):** Studies showed that it helped improve cognitive function in people with mild to moderate Alzheimer’s dementia. The dose is 60 drops per day.

Besides these, there are some other herbs having cognitive enhancement properties. These include Shankhapushpi, Jatamansi, Ashvagandha, Vacha and so on.
Shankhapushpi has been found having somewhat surprising effects on cognitive functioning. For this, it is also called as a neural tonic.

About Shankhapushpi

Shankhapushpi, belongs to the family of convolvulaceae. Its botanical name is Convolvulus Pluricaulis.

Convolvulaceae is a family of 55 genera and 1650 species. Most are annual or perennial, a few shrubs and trees. Genera include Ipomea (500 spp.), Pharabits Ipomea, Argyseia (91 spp.), Cuscutta (170 spp.) and convolvulus (250 spp.). One member of this family i.e. Convolvulus pluricaulis chois (Syn. convolvulus microphyllus Sanskrit/Hindi name – Shankhapushpi) is a well known Ayurvedic drug that’s called as a brain tonic, laxative and alterative (Jadavji Trikamji, 1935; and Govindji, 1948).

It has been considered as the best Medhya Rasayana in Charak Samhita, Susruta Samhita, Astanga Sangrah, Astang Harida, Bhavprakash Nighantu, Nighantu Aadarsh, Kaidele Nighantu, Dravyaguna Vighan, and other popular books of Ayurveda.

The synonyms of Shankhapushpi are Shankini, Shankhahuli, Manglya Kusuma, Kaharpushpi etc., and its use as Medhya Rasayana many plants are used in the name of Shankhapushpi in different parts of India. They are as follows:
1. Convolvulus Pluricaulis Choisy (family convolvulaceae).
2. Evolvulus alinoids Linn (family convolvulaceae)
3. Clitoria ternatea Linn (family Leguminaceae)
4. Canscova Decaussata Schutt (family Gentianaceae)

These four varieties have different chemical properties and different effects but convolvulus is the most popular due to its larger availability and chemical properties. Chemical analysis of convolvulus pluri showed that the whole plant contains shankhapushpine, volatile oil, ceryl alcohol, β-sitosterol, potassium chloride, organic salts, 3-D-glucoside, 3-4-dihydr oxycinammic acid, carbohydrates such as glucose, fructose, rhamose, sucrose and starch.

The effort to understand the composition of plant Shankhapushpi was made by Basu and Bhan (1951). They noted the presence of $C_{17}H_{23}NO_3$ which is soluble in chloroform. They also observed two other water soluble bases: first was acetone – insoluble base $C_3H_{14}NO_6$, having m.p. $84^0$, hydrochloride m.p.$214^0$, picrate m.p. $176^0$,
and the second base was an acetone soluble base. But this was not obtained in crystalline form. Two more derivatives were also prepared by them which named as base hydrochloride, having m.p. 272° and base oxalate having a relatively lower m.p. 154°.

Rakhit and Basu (1958) extracted all the solvents and analyzed left over ashes in the powder form. Total ash was 19.25% containing 12.2 of acid insoluble ash and 5.9% of water soluble ash. They noted the presence of iron, calcium, potassium, nitrate, and carbonate in the ash. They also analyzed further and found the two crystalline substances showing positive colour reactions for sterols I and II having formula C28H50O2, m.p. 124-25° and C40H60O5, m.p. 64-65° respectively. The melting points of the acetate of sterol I and sterol II were 17°<88-89°. The substances formulation of insoluble digitonides has been stated of β-type configuration of the –OH group at 3 position of the sterols.

In another study Deshpande and Srivastava (1969) investigated the presence of scopoletin, D-glucose, and maltose in 1% HCl soluble portion and β-sitosterol and ceryl alcohol in nonsaponifiable part.

The plant Shankhapushpi contains alkaloids: betaine, Shankhapushpine and evolvine. Fresh plant contains volatile oil. It also contains a yellow neutral fat, an organic acid and saline substances. An unidentified compound has been isolated (Goyal and Singh, 2005). Scopoletin, scopolin, umbelliferone, 2-methyl-1,2,3,4-butanetetrol, ferulic acid esters with alcohols C14-C17 and palmitic, oleic, 8-methyldecanoic and heptadecanoic acids have been reported (Cervenka, Koleckar, Rehakova, Jahodar, Kunes, Opletal, Hyspler, Jun, and Kuca, 2008). 2,3,4-trihydroxy-3-methylbutyl 3-[3-hydroxy-4-(2,3,4-trihydroxy-2-methylbutoxy)-phenyl]-2-propenoate (1) and 1,3-di-O-cafeoyl quinic acid methyl ester, caffeic acid, 6-methoxy-7-O-β-glucopyranoside coumarin, 2-C-methyl erythritol, kaempferol-7-O-β-glucopyranoside, kaempferol-3-O-β-glucopyranoside and quecetine-3-O-β-glucopyranoside were reported from n-BuOH soluble fraction from the ethanol extract of *E. alsinoides* (Gupta, Siripurapu, Ahmad, Palit, Arora, and Maurya, 2007).

In summing up, it can be said that Shankhapushpi contains small amount of sulphates, picrate, ceryl alcohol, β-sitosterol, glucose and maltose, and other components having OH as their components, iron, calcium, potassium, nitrate, carbonate etc. the
presence of these contents enhances the possibility to be a neural tonic for Shankhapushpi and thus it enhances energetic level in the organism.

In regard to Medhya effect of Shankhapushpi very little work has been done on convolvulus pluricaulis pharmachologically and clinically. Very little pharmacological work has been reported also on conscora decaussatta and Evolvulus alsinoids. Some clinical work have been reported, on Evolvulus alsinoids, Conscorea decussata and clitoria ternatea in regard to their Medhya activity.

Convolvulus pluricaulis choisy being a species convolvulaceae is often confused with Evolvulus alsinoids (Hindi name – Vishnukranta) that belongs to the same natural order. Both plants grow together in waste lands and look alike although the flower of Evolvulus alsinoids are usually blue, while those of convolvulus pluricaulis are white to light pink. Plant of Shankhapushpi is a procumbent shrub.

Shankhapushpi is quoted in Charaka Samhita to be the single greatest herb enhancing all the three aspects of mind power – learning (Dhi), memory (Dhriti) and recall (Smriti) among all four Medhya Rasayana i.e. Convolvulus pluricaulis, Evolvulus alsinoids, Clitoria ternatea and canscora decaussata. Thus it is called the greatest Madhya Rasayana (that which enhances the mind). It is also used as the best herb for beauty, stating that it achieves the goal of beauty, which is auspicious in all parts of the body. It also helps to nourish all layers of the skin (Twachya effect). It enhances all three pillars of Ayurvedic beauty, known as outer beauty, inner beauty and lasting beauty. Shankhapushpi enhances the quality of bone marrow and nerve tissue (Majju Dhatu). In all chikitsa grantha such as Charak Samhita, Sushruta Samhita, Nighantu etc. the use of Shankhapushpi are more or less the same. In most of the preparations it is used in the form of extract, paste or powder of whole plant along with ghee, honey, and other drugs like Brahmi, Vacha and Jatamansi. It is reported to have the qualities of promoting intellect in these granthas.

Shankhapushpi is commonly used as a clinical medicine. It was very early i.e. in 1927, when Shankhapushpi was referred as a plant that is useful in increasing the level of intelligence, in treating mania, high fever, dysentery and even for relaxation to sleep (Shah, 1927). He also claimed it as a brain tonic which may enhance memory, but also
reported that there is a lack of experiments in this area. Further he recommended memory experiments in this area on animals and human beings.

Many herbal formulations, popular as memory enhancers, have Shankhapushpi as major ingredient. Some formulations are Baidyanath Shankhapushpi, Dabur Shankhapushpi, Shankhapushpi by Unjha, Zandu’s Brento, Ceilo herbal’s Focus Gelcaps, and the like.

It has been mentioned in the literature available with the medicine that by removing the brain fatigue and establishing normally in weak memory the drug has been found very much useful for students, professors, lawyers, merchants, and such intellectual brain workers affected with mental tension and forgetful nature. Regular use of this drug refreshes the system, brings energy and calmness of the brain.

Shankhapushpi appears to possess anti-anxiety effect, which leads to improved mental functions and provides significant symptomatic relief in patients of anxiety neurosis.

The anti-anxiety effect may be responsible for its anti-hypertension action (Chatruvedi, Sharma and Sen, 1966; Sharma, Barar, Khanna and Mahawar, 1965; Mudgal, Srivastava, Singh and Udupa, 1972). Shah (1982) reported that convolvulus pluricaulis is a brain tonic to enhance memory. He also quoted that the students use it during examination to enhance memory. Ali (1998) claimed that the drug is used as brain tonic in hypertension as tranquilizer.

Handa and Kapoor (1998) noted the alkaloid shankhapushpine as a main chemical constituent of this plant. They reported that the fresh juice of shankhapushpi is used as nerve tonic in cases of epilepsy, insanity and nervous debility.

The herb appears to produce its actions by modulation of neuro-chemistry of the brain (Czukles, 2008). Further, this herb is non-toxic and its use does not produce any side effect. The efficacy of Convolvulus pluricaulis choisy was found to be comparable to that of imipramine and fluoxetine (Prozac) administered for 10 successive days.

In this way it becomes very interesting to see the surprising effect of the herb Shankhapushpi on cognitive deterioration and impairment such as in dementia, but before conducting the research, it is important to go through the research work already done in this area.