# Altered Thought Processes: Dementia 4.0 CE Hours

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## Course Objectives

1. Define dementia.
2. Differentiate between reversible and non-reversible dementia.
3. List examples of causes, which can become the symptoms of reversible dementia.
4. List four common diseases associated with dementia.
5. Describe the clinical presentation, diagnosis, and treatment of Alzheimer's disease.
6. Identify the risk factors associated with vascular dementia (VaD).
7. Compare and contrast the disease progression of vascular dementia with Alzheimer's disease.
8. Compare and contrast the disease manifestations of Lewy body dementia with Alzheimer's disease.
9. Discuss how to diagnose Frontotemporal dementia.
10. Identify the most common symptoms identified with dementia.

## Defining Dementia

It would be nice if we had one standard definition for dementia, but unfortunately there is not one standard definition.  Dementia is not a disease. Dementia is a group of symptoms that are caused by a variety of diseases or conditions. Dementia could be defined as a group of diseases that cause a permanent and progressive decline in cognitive ability. Cognitive ability is the ability of a person to reason, think, and act appropriately in daily life.

Simply put, individuals with dementia are unable to do the

things they used to do because of the mental changes occurring in their brains. It involves a change in memory and intellectual function. It may involve deficits in language, manipulating objects, recognizing familiar objects, drawing, and movement. Dementia can be reversible or permanent.

## Risk Factors/Cause of Dementia

The causes of dementia are biological processes within the brain that are damaging brain cells. There are several things that can lead to dementia, including diseases that cause degeneration or loss of nerve cells in the brain, diseases that affect blood vessels, toxic reactions, nutritional deficiencies, infections, head injuries, or brain tumors and illnesses of other organs (such as kidney, liver, and lung diseases).

## Myths

There is a common misconception that dementia is a normal part of aging. There is a difference between forgetfulness, which can occur at any age, and dementia, which is a cognitive impairment of the brain. Although you typically see dementia more often in the older, or elderly, adult, this decline in cognitive function is an effect of other diseases and not an effect of the normal aging process.

## Reversible Dementia and True Dementia

Reversible dementia means a person has symptoms that mimic dementia. When the underlying cause is identified and treated, the symptoms of dementia most often resolve. These reversible or false dementias are highly treatable. A true dementia cannot be cured.

## Examples of Causes of the Symptoms of Reversible Dementia:

- Medications or combinations of prescribed medication
- Alcohol intoxication or withdrawal

- Illegal drug use
- Hormonal or vitamin imbalances
- Metabolic disorders: thyroid disease, vitamin B12 deficiency, hyponatremia, hypercalcemia, hepatic dysfunction, and renal dysfunction
- Depression
- Delirium
- CNS neoplasm
- Chronic subdural hematoma
- Normal pressure hydrocephalus_

## Types of Dementia

Dementia covers many different types of diseases. There are as many as 50 different causes of dementia. Each of these diseases has its own course of illness, different symptoms, different treatments and plans of care. This course will focus on four diseases associated with dementia: Alzheimer's disease (AD), vascular dementia (VaD, also known as multi-infarct dementia, or MID), Lewy body dementia (LBD), and frontal lobe dementia (FTD).

### Alzheimer's Disease (AD)

The most common form of dementia in the United States is Alzheimer's Disease (AD). AD causes 50-60% of all dementias. AD occurs when plaques (small pieces of proteins) form in the brain. These plaques interrupt the connections between the brain cells so the affected individual cannot save new information. As more plaques form in the brain, they destroy brain cells. The cause as to why the plaques start depositing in the brain is not definitively known. There is also no cure for AD at this time. Pharmacology agents currently on the market are designed to slow the progression of the disease when caught early on to keep patients as independent as possible. As the disease progresses, however, the individual will lose more memory, skills, abilities and functions. The survival time from the time of diagnosis is about 10 years.

## Cause and Risk Factors Associated with AD

The underlying cause as to why the plaques start to accumulate in the brain has not been definitively identified. Researchers are looking into the risk factors that may increase a person's risk of developing AD. Currently risk factors they are looking at include: age, genetics, nutrition, viruses, and environmental causes.

Older age is the single most important risk factor for the development of AD. AD is not an inherited disease, though researchers are looking at genetic factors that may contribute to the development of the disease. Another possible risk factor may be viral infections. Infections such as herpes zoster, herpes simplex, or viral infections of the brain might have an association with AD. Environmental factors that may contribute to AD include exposure to toxic substances or chemicals. Head injuries with a loss of consciousness have been associated with the development of AD in later years. Nutritional deficiencies of vitamin B12 have been reported among people with AD.

## Clinical Presentation/Signs and Symptoms

Signs and symptoms of AD are usually first noticed by significant others in the person's life such as a spouse, family and/or friends. The significant others typically notice signs such as: questions and comments being repeated, forgetting to take care of daily things like paying bills or returning phone calls, and taking their usual medication. As the disease progresses they become disoriented easily around once familiar places, become more forgetful, and have trouble with language and motor skills. They show signs of poor judgment and seem confused and/or restless. Personality changes are noticed, as well as a loss in interest in things that were previously interesting or fun to the person. Mood swings are noticed where they were never present before. They become more argumentative and accusatory, asking if

significant others have stolen things from them or lied to them. Eventually AD will completely destroy the individual's memory, personality, and the ability to function.

### Diagnosis of AD

MRI and PET scans can be used to identify the brain atrophy associated with AD. Clinical examination is the most common way to diagnose AD and is very accurate. Autopsy is a conclusive means of diagnosing AD.

### Treatment/Cure for AD

There is no cure for AD. Pharmacology options on the market work to slow the progression of the disease when caught early and to help the individual with AD to remain as independent as possible.

Nursing care involves helping the individual maintain cognitive and physical function as early as possible in the disease process to delay institutional care. Nursing care also involves helping the patient and family as the disease progresses while allowing as much dignity and independence as possible to the patient.

## Vascular Dementia (VaD)/Multi-Infarct Dementia (MID)

VaD is defined as a loss of cognitive function which was caused from ischemia, hypoperfusion, or hemorrhaging brain lesions. This is a result from cerebrovascular disease or cardiovascular pathologic conditions. VaD is also called "multi-infarct dementia," or MID, because it involves many small strokes (or transient ischemic attacks, TIAs). These series of small brain infarcts cause occlusions and blockages within the arteries of the brain and leads to brain cell death. The more infarcts there are, the quicker the rate of decline in a person's functional ability. It is estimated that

20% of all dementias have a vascular component. Although there can be improvement in a person’s functional ability over time from TIAs, there is never a full recovery.

## Risk Factors of VaD

Arteriosclerosis, blood dyscrasias, cardiac decompensation, hypertension, atrial fibrillation, cardiac valve replacements, systemic emboli, diabetes mellitus, peripheral vascular disease, obesity, smoking and TIAs are all risk factors for VaD.

## Clinical Manifestations/Signs and Symptoms

Whereas AD patients will show a steady decline, the usual progression of VaD follows more of a decline in a series of distinct stages. When patients with VaD have an infarct, there will be a decline in function, followed by a stabilized period. When another infarct happens there will be another decline in function, followed by another stabilized period. The functioning capabilities slowly decrease over time in this pattern.

Symptoms of VaD depend on the location of the infarct but they all interfere with the ability to work and social functioning. Symptoms may include: memory problems of being forgetful, not being able to concentrate, having problems counting money or making out a check, poor judgments, and having difficulty following instructions. Speech may be slurred; there may be arm or leg weakness, incontinence, inappropriate emotions, and dizziness. They may wander or get lost in familiar places.

## Diagnosing VaD

The only way to diagnose VaD is through neuroimaging and clinical examination. MRI and CT scans of the brain will reveal one or more areas of the brain affected by cerebral infarcts.

### Treatment

Although there are no treatments that can restore or repair brain cells, research is focused on drugs to improve cognitive and motor function in mild to moderate VaD. Treatment also includes preventing further episodes of infarctions by finding out the underlying cause and treating it. If the underlying cause was high blood pressure then medication for this condition should be started as soon as possible and lifestyle changes need to be initiated in order to keep the blood pressure within norms. Treatment also includes educating the caregivers on the importance of close monitoring of the patient and keeping them active. Physical therapy may be ordered if treatment is needed for physical conditions associated with the infarcts.

## Lewy Body Dementia (LBD)

Lewy body dementia is a progressive, degenerative brain disorder caused by a buildup of proteins called Lewy bodies in the brain. Over time, the proteins destroy brain tissue. It is not known what causes the proteins to form.

### Risk Factors of LBD

Advanced age is a risk factor for developing LBD as well as other disease processes such as Parkinson’s disease. Individuals with Parkinson’s disease have a six-fold increased risk for the development of Lewy body dementia compared to the general population.

### Clinical Manifestations/Signs and Symptoms

The signs and symptoms of LBD are similar to those of AD. Differences include: fluctuations in attention and the ability to communicate, psychiatric symptoms (particularly visual hallucinations), rigidity, bradykinesia (a slowness of movement found in Parkinson’s disease), flexed posture, shuffling gait, REM sleep disorder, excessive daytime

sleeping, difficulty arousing, and reduced concentration.

A person with LBD will slowly decline in function. The ability to think clearly, make decisions, or remember things will become more difficult. It is common for people with LBD to stare off into space and having trouble paying attention. They often have recurring visual hallucinations involving seeing things or people who aren't there. They develop similar symptoms to Parkinson's disease including a shuffling gait, slowness of movement, blank stare, and rigid muscles.

### Diagnosing LBD

No lab tests are available for the diagnosis of LBD. Clinical examination is the most common way to diagnose LBD.

### Treatment/Cure

There is no cure for LBD. There are no treatments that can reverse or stop the brain damage caused by the proteins. Treatment involves treating the symptoms. The same medication is used that treat AD with the addition of levodopa to combat movement problems. Medication is not often prescribed for the hallucinations because they tend to increase problems with movement.

## Frontal Lobe Dementia, or Frontotemporal Dementia (FTD)

Frontal lobe dementia is part of a larger set of brain diseases called frontotemporal dementia (FTD). FTD is relatively rare and includes non-AD dementias. It involves focal atrophy of the frontal and anterior temporal regions of the brain. FTD will usually be caused by abnormal formations of multifunctional proteins that are known as tau. There is either abnormal formation of tau or an imbalance- too much or not enough tau. This imbalance of tau leads to problems like tumors, brain atrophy, Pick bodies (abnormal substances inside

nerve cells in the damaged areas of the brain), or swelling/fluid in the brain.

## Risk Factors of FTD

FTD starts at a younger age than other dementias but the risk factors are not well understood or identified besides a family history of the disease.

## Clinical Manifestations/Signs and Symptoms

The two major signs include frontal or aphasic issues. Damage to the frontal lobe would cause changes in emotional responses and use of language. Examples include: progressive changes in personality, changes in social cognition, disinhibition, loss of empathy, changes in eating patterns, ritualized behaviors, and apathy. Aphasic issues include the loss of the ability to use language either by having difficulty speaking, having trouble finding the right word, or misunderstanding the meaning of words. A person with FTD typically would display changes in their personality such as overspending, overeating, drinking excessively, being hyperactive sexually, having poor personal hygiene, and having mood swings. They usually are unaware that their behavior has changed or is inappropriate.

A good example of a FTD is Pick’s disease. It is more common in Europe than in the United States. It often occurs around the age of 40-60. People with Pick’s disease have frontal lobe symptoms, especially behavioral problems.

## Diagnosing FTD

PET, CT, or MRI scans help diagnose FTD by looking for focal atrophy in the prefrontal or temporal regions, although sometimes this is not present. Clinical examination is the most common way to diagnose FTD.

## Treatment/Cure

There is no cure for FTD and currently no treatments for FTD

itself, although pharmaceuticals can be used to treat the behavioral abnormalities displayed due to the condition such as, selective serotonin reuptake inhibitors (SSRIs) and atypical antipsychotic agents (AAP).

**Summary**

- Dementia is not a disease. Dementia is a group of symptoms that are caused by a variety of diseases or conditions.
- Dementia could be defined as a group of diseases that cause a permanent and progressive decline in cognitive ability (the ability of a person to reason, think, and act appropriately in daily life).
- Dementia can be reversible or permanent.
- There are as many as 50 different causes of dementia.
- The cause of dementia is biological processes within the brain damaging brain cells.
- This course focused on risk factors, symptoms, diagnosis and treatment of: Alzheimer's disease, vascular dementia (multi-infarct dementia), Lewy body dementia, and frontal lobe dementia (frontotemporal dementia).
- Each of these diseases has its own course of illness, different symptoms, different treatments, and plans of care.

The most common symptoms seen with dementia include:

- Moodiness, getting upset easily, easily angered, and mood swings, and trouble completing everyday tasks
- Trouble remembering words or completing a sentence
- Losing interest in activities previously enjoyed
- Repeating the same story, comments, or questions during a conversation

**References:**

Profenno, L. A., Porsteinsson, A. P., & Faraone, S. V. (2010). Meta-analysis of Alzheimer's Disease Risk with Obesity,

Diabetes, and Related Disorders. *Biological Psychiatry*, *67*(6), 505-512.

Barnes, D. E., & Yaffe, K. (2011). The Projected Effect of Risk Factor Reduction on Alzheimer's Disease Prevalence. *The Lancet Neurology*, *10*(9), 819-828.

Dickstein, D. L., Walsh, J., Brautigam, H., Stockton, S. D., Gandy, S., & Hof, P. R. (2010). Role of Vascular Risk Factors and Vascular Dysfunction in Alzheimer's Disease. *Mount Sinai Journal of Medicine: A Journal of Translational and Personalized Medicine*, *77*(1), 82-102.

Rongve, A., Brønnick, K., Ballard, C., & Aarsland, D. (2010). Core and Suggestive Symptoms of Dementia with Lewy Bodies Cluster in Persons with Mild Dementia. *Dementia and Geriatric Cognitive Disorders*, *29*(4), 317-324.

Dolder C.R., Davis L.N., McKinsey, J. (2010). Use of Psychostimulants in Patients with Dementia. *Annals of Pharmacotherapy*, *44* (10). 1624-32.

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