Alzheimer’s disease is a progressive degenerative disorder of the cerebral cortex, especially the frontal lobe. It affects approximately 5 million Americans; by 2030, that figure may reach 7.7 million. It’s the seventh-leading cause of death in the United States.

AGE ALERT
Alzheimer’s disease typically affects adults older than age 65, but some cases have been reported in individuals as young as in their late 30s.

The disease has a poor prognosis. Typically, the duration of illness is 8 years, and patients die 2 to 5 years after the onset of debilitating brain symptoms.

Causes
- Exact cause unknown
Possible contributing factors
- Genetic patterns
- Beta-amyloid plaque development
- Inflammatory and oxidative stress processes
- The role of estrogen in the brain

Pathophysiology
The brain of a patient with Alzheimer’s disease has three characteristic features: neurofibrillary tangles (fibrous proteins), neuritic plaques (composed of degenerating axons and dendrites), and neuronal loss (degeneration).

Neurofibrillary tangles are bundles of filaments found inside neurons that abnormally twist around one another. Abnormally phosphorylated tau proteins accumulate in the neurons as the characteristic tangles and ultimately cause neuronal death. In a healthy brain, tau provides structural support for neurons, but in patients with Alzheimer’s disease this structural support collapses.

Neuritic plaques (senile plaques) form outside the neurons in the adjacent brain tissue. Plaques contain a core of beta-amyloid protein surrounded by abnormal nerve endings or neurites. Overproduction or decreased metabolism of beta-amyloid peptide leads to a toxic state causing degeneration of neuronal processes, neuritic plaque formation, and eventually neuronal loss and clinical dementia.

Tangles and plaques cause neurons in the brain of the patient with Alzheimer’s disease to shrink and eventually die, first in the memory and language centers and finally throughout the entire brain. This widespread neuron degeneration leaves gaps in the brain’s messaging network that may interfere with communication between cells, causing some of the symptoms of Alzheimer’s disease.

Complications
- Injury from wandering, violent behavior, or unsupervised activity
- Pneumonia
- Malnutrition and dehydration
- Aspiration

Signs and symptoms
Mild
- Disorientation to date
- Impaired recall
- Diminished insight
- Irritability
- Apathy

Moderate
- Increased disorientation (time and place)
- Fluent aphasia
- Difficulties with comprehension
- Impaired recognition
- Poor judgment
- Trouble performing activities of daily living (ADLs)
- Aggression
- Restlessness
- Psychosis
- Sleep disturbances
- Dysphoria

Severe
- Unable to use language appropriately
- Memory only to the moment
- Needs assistance with all ADLs
- Urinary and fecal incontinence

Diagnostic test results
- Neuropsychologic evaluation shows deficits in memory, reasoning, vision-motor coordination, and language function.
- Magnetic resonance imaging or computed tomography scan reveals brain atrophy at later stages of the disease.
- Positron emission tomography scanning shows decreased brain activity.
- EEG shows evidence of slowed brain waves at later stages of the disease.

Treatment
- Cholinesterase inhibitors, such as tacrine, donepezil, rivastigmine, and galantamine
- Memantine (Namenda), and N-methyl-D-aspartate receptor antagonist
- Behavioral therapy
- Nonsteroidal anti-inflammatory drugs
- Cholesterol-lowering drugs
- Estrogen
**TISSUE CHANGES IN ALZHEIMER’S DISEASE**

- **Granulovascular degeneration**
- **Neurofibrillar tangles in the neuron**
- **Neuritic plaques outside neurons**
- **Alzheimer’s disease**
- **Normal**

- Vacuoles
- Tangles
- Neurofilament
- Dendrites
- Amyloid in blood vessel
- Beta-amyloid protein core
- Neurites
- White matter
- Cerebral cortex (gray matter)
- Neuron cell body
- Axon
- Message
- Neurotransmitter (acetylcholine)
- Receptor site
- Synapse
- Granules containing neurotransmitter
- Axon
- Dendrite of receiving neuron