Caring Sheet #11: Alzheimer’s Disease: A Summary of Information and Intervention Suggestions with an Emphasis on Cognition
By Shelly E. Weaverdyck, PhD

Introduction
This caring sheet focuses on Alzheimer’s Disease, with an emphasis on cognition.

Caring sheets #12 and #13 summarize information and intervention suggestions regarding Dementia with Lewy Bodies (caring sheet #12) and Frontotemporal Dementia (caring sheet #13). All three outline the brain changes in each type of dementia, the impact these changes have on cognition and behavior, and implications for effective intervention.

The three caring sheets (#11, #12, and #13) are written as companion pieces in outline form with virtually each line of one caring sheet corresponding with each line of the other two. The three can be laid out side by side, and compared almost line by line.

Other caring sheets describe the cognitive and behavioral changes and specific interventions for these dementias in more detail. Caring Sheet #2 in particular describes the characteristics of dementia, and the relationship between the brain changes and changes in cognition.

Dementia is a decline in a person’s cognition. This decline occurs because of changes in the brain.

If the cognitive decline is caused by treatable disorders such as a urinary tract infection, vitamin deficiency, reactions to medications, or depression, it is likely temporary and treatable (e.g., delirium).

In other cases the brain changes and resulting cognitive decline are irreversible and progressive (i.e., increasingly severe). They are caused by disorders such as Alzheimer’s Disease, vascular disorders (e.g., ministrokes), Creutzfeldt-Jakob Disease, Dementia with Lewy Bodies, or Frontotemporal disorders. There are over 80 different disorders that cause this type of progressive dementia. Alzheimer’s Disease is the most common.
ALZHEIMER’S DISEASE (AD)

CHARACTERISTICS
Brain disorder
Most obvious symptoms: memory loss and cognitive impairment
Progression: increasing severity of symptoms over time (a progressive dementia)
Onset: insidious; age 40-90, usually over age 65 (median age 73.5)
Duration: average 8 years from onset to death (may vary with time of diagnosis)
Cause unknown
Cure: no cure at this time, but there is treatment to reduce symptoms
Diagnosis verified at autopsy
Is the most common cause of dementia (60% of all dementia cases)
Affects 10% of all people over age 65
Risk factors: age, APOe4 gene, Down’s Syndrome, family history
Hereditary in 10% of cases
Course: gradual, steady decline, decline 2-4 points per year on Folstein Mini-Mental State Exam, no spontaneous improvement
Alois Alzheimer first described neuropathology in a 51-year-old woman in 1907

NEUROPATHOLOGY
Neuritic plaques outside of cells in brain
Neurofibrillary tangles inside of cells in brain
Atrophy (i.e., loss) of brain tissue; cell death
Acetylcholine reduction

LOCATION OF CORTICAL BRAIN CHANGES
Cortical refers to the cortex (i.e., the outer layer) of the brain
Changes (pathological abnormalities) occur in the cortex and in internal (subcortical) structures of the brain
Changes (pathological abnormalities) occur on both sides of the brain
Order of cortical brain structures affected, creating stages:
Hippocampus (subcortical)
Parietal lobes
Temporal lobes (posterior then anterior)
Frontal lobe
Cognitive Changes
Memory impairment first obvious symptom
Visuospatial perception and skill impairment:
  Difficulty recognizing distance between objects and from self
Difficulty arranging objects in space
Language impairment:
  Difficulty understanding and producing speech
  Difficulty reading and writing
  Difficulty understanding what is being read, when can read
Insight impaired
Judgment impaired
Disorientation
Concentration impaired
Abstract processing impaired
Attention impaired
Sense of time impaired
Ability to analyze, plan, organize impaired
No sensory loss (though may occur with normal aging)
No focal deficits
No disturbance of consciousness
Person becomes increasingly dependent on environment throughout course

Emotional Changes
Depression throughout course, especially in early stages
Emotional lability (switch quickly from one emotion to another)
Less expression of emotional intensity and switching in later stages

Behavioral Changes
Get lost occasionally in early stages; increasingly often throughout course
Loses objects occasionally in early stages; increasingly often throughout course
Distressing behaviors usually triggered by anxiety, confusion and misinterpretation of environment in middle stages
Distressing behaviors usually triggered by physical pain and discomfort in later stages
Gait and physical movements preserved until later stages
Incontinence only in later stages
Hallucinations when they occur, occur in later stages
INTERVENTIONS: Non-medicinal

Assess individual regularly over time as dementia progresses
Modify expectations and intervention as change occurs
Express warmth and affection for person, verbally and nonverbally
Respect person as adult; avoid treating person as a child
Stay calm and help the person feel relaxed
Compensate for the person’s cognitive impairment to help person feel good and comfortable and to prevent fatigue and embarrassment
Assess and modify environment, caregiver interactions and daily routines over time throughout course
Maintain appropriate stimulation that engages person
Avoid meaningless or confusing stimulation
Use touch as appropriate when communicating
Simplify daily routines and tasks
Provide information through cues in environment and when communicating
Ensure cues are understandable to person
Avoid arguing with the person
Address person’s feelings and then distract when person engaged in distressing behavior
Explain to person what caregiver is doing and intends to do
Use concrete requests and concrete cues
Compensate for sensory changes that occur with normal aging

MEDICAL TREATMENTS

Cure unknown
Reduce loss of acetylcholine (inhibit acetylcholinesterase)
   Cognex, Aricept, Exelon, Reminyl
   Most effective in mild-moderate stages
Memantine (regulates activity of glutamate)
   Can be effective in severe stages (for persons with MMSE score of 3-14)
Cholinergic medications for behavioral symptoms
Antioxidants
Anti-inflammatory agents
Neurotrophic compounds
Anti-amyloid deposition
Vitamin E in high doses (only under doctor’s care) (effectiveness is controversial)
To extent AD associated with cardiovascular risk factors, can prevent by reducing cardiovascular risk factors: Exercise, Diet, Cholesterol-lowering medications (statins) such as Lipitor

COMMENTS

In 1984 criteria for clinical diagnosis created
Red flags that suggest a disorder is probably not AD:
- Onset before age 60 years
- Sudden onset
- Rapid progression
- Symptoms that do not occur in the order of typical AD stages
- Behavior changes or hallucinations occur much earlier than memory impairment
- Incontinence occurs before later stages
- Seizures occur before later stages
  (Exception: Downs may cause earlier seizures)
- Abnormal neurological symptoms (impaired gait, falls, weakness) occur before later stages

RESOURCES

(National Institute of Neurological Disorders and Stroke NINDS)

http://www.alzheimers.org/ (Alzheimer’s Disease Education and Referral Center ADEAR)

http://www.alz.org/ (Alzheimer’s Association)

http://www.med.umich.edu/madrc/ (Michigan Alzheimer’s Disease Research Center MADRC)

© Copyright 2004 (Revised 2005) by S. Weaverdyck
This caring sheet focuses on Alzheimer’s Disease (AD). More details about the brain changes and resulting cognitive changes in AD are in caring sheet #2.

Alzheimer’s Disease is by far the most common cause of irreversible brain changes & dementia in persons over the age of 65 years.

Though these changes in behavior and cognition result from brain damage, they are often mistakenly viewed as intentional or manipulative.

In AD the four hallmark pathological changes to the brain are:

- **Atrophy**: the reduction in size of a structure. Atrophy due to death of nerve cells in AD causes much of the confusion and cognitive impairment. Atrophy occurs with normal aging, but is especially pronounced and is pathological in AD. The atrophy is visible on a CAT scan and at autopsy.

- **Neuritic plaques**: little patches or collections of debris in the brain. They are located outside of nerve cells. A protein called amyloid is at the core of the plaques. The number of neuritic plaques correlates with a person’s performance on cognitive tests (such as Intelligence or IQ tests).

- **Neurofibrillary tangles**: inside nerve cells, particularly in the axons of nerve cells. Axons are the protrusions from the nerve cell body, which carry information from one nerve cell to the next. Tiny neurofibrils (filaments or tubules) transport cell nutrients within the nerve cell. The neurofibrils become tangled in a very characteristic way (double helical) and therefore disrupt the cell maintenance processes, probably contributing to the cell’s death. The number of neurofibrillary tangles also correlates with a person’s performance on various cognitive tests.

- **Neurochemical changes**: a reduction in some of the neurotransmitters. Neurotransmitters (the chemical messengers) are neurochemicals which are transferred from one nerve cell to another as a method of communication with that nerve cell. This intercellular communication is essential to the brain’s maintenance and functioning. There are many different kinds of
neurotransmitters. One, which is particularly reduced in amount in Alzheimer’s Disease, is called acetylcholine.

**Vascular Dementia**

Vascular dementia is another common disorder. In Vascular dementia the brain has many tiny strokes on the cortex or surface of the brain (as well as other areas). Spots of softened dead tissue (lesions) occur throughout the cortex. These lesions can sometimes be seen on a CAT scan. A CAT scan can rule out the possibility of a major stroke as a source of the cognitive impairment. The cognitive changes are more varied than they are in AD.

**Other Dementias**

See caring sheet #12 for more information about Dementia with Lewy Bodies (DLB) and caring sheet #13 for more information about Frontotemporal Dementia (FTD). DLB is another more common disorder. FTD is less common but can be a particularly challenging disorder.