
Geriatrics

Academic Half Day

DEMENTIA

Pre-Reading Module

2011

In previous decades, medical students or physicians could cope (sometimes barely) without sound knowledge about cognitive impairment. Now, with ever changing demography this is no longer the case. Dementia is a syndrome of progressive deterioration in multiple cognitive domains, resulting in loss of function.

DEMENTIA

Alzheimer's disease (AD) is a chronic neurodegenerative disorder and the most common cause of dementia. It is one of the principle causes of disability and decreased quality of life among older adults. Progress and our clinical knowledge of AD has led to more reliable diagnostic criteria and accuracy, and research efforts are expanding to uncover the earliest manifestations and even the presymptomatic phases of the disease.

The diagnosis of AD is primarily one of exclusion and usually can be made using standard clinical criteria. There is currently no cure for AD. Current treatment focuses on establishing an early accurate clinical diagnosis, early institution of cholinesterase inhibitors and/or NMDA receptor targeted therapy. Treating medical comorbidities and dementia related complications, ensuring that appropriate services are provided, and addressing the long-term well being of caregivers and treating behavioral and psychological symptoms with appropriate non-pharmacologic and pharmacologic interventions are also important. The initiation and propagation of pathologic processes and the anatomic location of the earliest changes will become new targets of research and therapeutic development. A possible precursor of AD, mild cognitive impairment (MCI) is under investigation as a possible therapeutic starting point for disease modifying interventions.

Facts and Figures:

Among Canada's population of just over 30 million people, 3.7 million (12%) are over the age of 65. This figure is projected to rise to 5 million (14%) by 2011. Presently, Canada is second only to Japan in current life expectancy at birth, 76 years for men in Japan and 75 years for men in Canada; 83 years for women in Japan, 81 years for women in Canada.¹ At age 65, life expectancy is 16 years for men and 20 years for women; at age 80 men can expect just over 7 more years of life and women just over 9. While it is estimated that approximately 7% of the population aged 65 and over lives in some form of long term care institution (Statistics Canada, 1999), approximately 35% of those aged 85 years and over are residents in long term care. These figures, however, vary significantly across Canada.²

In Alberta, 10.6% of its population is over 65, close to 340,000 people. This year, an estimated 20,000 Albertans will turn 65 and become senior. The largest group of seniors is under the age of 75, but the most rapidly growing segment are those 65 to 74.

¹ McPherson BD. Aging as a Social Process (3rd Edition). Toronto: Harcourt Brace; 1998

² Dumas J, Belanger A. Report on the demographic situation in Canada. Ottawa: Statistics Canada 1996

In Alberta, the proportion of seniors is expected to grow to 13.9% by 2016. It will be 25% by the year 2031.³

The prevalence of dementia in Canada is approximately 8% for those over the age of 65 in Canada, with the most common cause being Alzheimer's disease. The incidence of dementia is 21.8 (women) and 19.1 (men) per 1,000 per year.⁴ Of the factors studied thus far, exercise appears to be highly protective for all forms of late-life cognitive impairment. The same statement probably applies to the treatment of systolic hypertension. The prevalence of dementia is 35% in people over the age of 85 (26% Alzheimer's disease).

AD unassociated with other pathology ("pure AD") is found in between 50 and 60% of most unbiased autopsy samples, although such figures do vary from study to study. The incidence rises to 80% if AD is used in conjunction with other pathological lesions. Vascular dementia, dementia with Lewy bodies, and frontal temporal dementia account for most of these. Cerebral vascular disease is the second most common cause of acquired cognitive impairment and dementia, and often contributes to the cognitive decline in the neurodegenerative dementias. Hence, overlap is quite common among AD, VAD, and DLB.

AD reduces life expectancy by half. The figures significantly associated with reduced survival at diagnosis are: (1) increased severity of cognitive impairment, (2) decreased functional level, (3) history of falls, (4) physical examination findings of frontal release signs, (5) abnormal gait. The prognosis of AD is that of inexorable decline and eventual death.

The annual treatment cost of AD in the US is approximately 100 billion dollars; approximately \$18,408 per patient per year for mild AD, \$30,096 per patient per year for moderate AD, and \$36,132 per patient per year for severe AD. The economic impact of AD is expected to worsen with the demographic, epidemiological and technological and economic transitions going on world wide.

Neuropathologic Changes of AD

The neuropathologic hallmarks of AD include amyloid rich senile plaques, neurofibrillary tangles of phosphorylated tau protein, neuronal degeneration, and synaptic loss. Synaptic loss is the best pathological correlate of cognitive decline, and synaptic dysfunction is evident long before synapses and neurons are lost. Once synaptic function fails, even in the setting of surviving neurons, there may be little chance of effectively interfering with the disease process.

Mutations in three genes are known to cause autosomal dominant forms of familial, early onset AD. These include the amyloid precursor protein gene located on chromosome 21 and the genes for presenilin 1 and presenilin 2 located on chromosome

³ AHCIP Population Registry File for 2004/2005.

⁴ The Canadian Study of Health and Aging Working Group. The incidence of dementia in Canada. *Neurology*; 55:66-73. 2000

14 and 1 respectively. Studies of these mutations have provided strong support for the amyloid cascade hypothesis of AD. Within that, the amyloid precursor protein is cleaved enzymatically and result in secretions of the toxic form of amyloid A-beta-42 these aggregate readily into highly insoluble amyloid fibrils, which form the major components of senile plaques.

The APO-E gene type is associated with increased AD risk. The APO-E2 allele may be protective while the APO-E4 alleles is associated with a definitely increased risk. The precise role of APO-E4 and the pathogenesis of AD nonetheless is still unclear. APO-E is found in A-beta plaques and neurofibrillary tangles and may affect protein-protein interactions. Although the amyloid cascade hypothesis is currently considered by many researchers as the key contributor to the pathogenesis of AD, some researchers have challenged this assertion, that AD occurs secondary to neuron stress and functions as a protective adaptation to the disease rather than causing the cell death.

AD is also characterized by disruption in multiple major neurotransmitters in which cholinergic abnormalities are most prominent. There are reduced numbers of cholinergic neurons in late AD (particularly in the basal forebrain) and there is selective loss of nicotinic receptor subtypes in the hippocampus and cortex. Hence, acetylcholine (ACH) is an important neurotransmitter in areas of brain involved in memory function and loss of ACH activity correlates with the severity of AD. Presynaptic nicotinic receptors control the release of neurotransmitters important for memory and mood such as ACH, glutamate, serotonin, and norepinephrine. It has been shown that the reduction in the number of ACH receptors precedes the pathologic changes. Inhibition of the down regulation of ACH is a strategy for the treatment of AD because it might augment ACH levels within synaptic clefts. In this context, cholinesterase inhibitors which improve cognitive function are currently approved for the treatment of AD. Pathologic stimulation of glutamate receptors results in abnormally high levels of intracellular calcium and may ultimately lead to cell death. This probably explains the beneficial effects of moderate to low affinity NMDA receptor antagonist memantine on cognitive and functional measures compared with placebo in trials of patients with moderate to severe AD.

Mild Cognitive Impairment:

An interesting finding from the CSHA I was that the prevalence of cognitive impairment that did not meet dementia criteria (CIND) was almost twice as large as all causes of dementia combined.⁵ Forgetfulness used to be viewed as an inevitable consequence of aging, arising from progressive neuron loss. Postmortem studies showing a steady decrease in brain weight after age 40 were felt to support this view.

Benign senescent forgetfulness, as it was called in the 1960's, implied a complacent although likely inaccurate view of this syndrome. New developments are challenging these old assumptions. The scientific community now believes that mild cognitive

⁵ The Canadian Study of Health and Aging: Study methods and prevalence of dementia. Can Med Assoc J; 150:899-913. 1994

impairment is a variable transition stage between normal aging and Alzheimer's disease. It is estimated that 10 - 15% of MCI patients per year cross the line into probable Alzheimer's disease. Unfortunately, double-blind trials of cholinesterase inhibitors showed no positive response over placebo. The usual MCI symptom is short-term memory loss that progresses gradually. Frequently, such seniors have insight into their memory deficit with preservation of the other domains of cognition such that they can continue to function quite independently, barring physical illnesses. Presently, research projects are underway to scrutinize mild cognitive impairment with a multi-modal approach featuring serial genetic testing, neuropsychological evaluation, and magnetic resonance imaging of the hippocampus in the brains of these individuals. However, some cases remain stable and a small number improve. The aspiration is that, 5-10 years downstream, a physician will be able to predict which of the mild cognitive impairment individuals are most in danger of progressing to Alzheimer's disease. Treatments soon might be targeted to this group much "earlier in the game". Is MCI a pathologic entity? Some consider it to be incipient AD, promoting the idea that MCI is a pathologic process causing progressive cognitive decline, and not a normal consequence of aging. In American centres, some clinicians do treat E4 positive MCI with cholinesterase inhibitors but in Canada, this step is not common.

The best predictors of conversion from MCI to AD are function in every day situations requiring judgment and problem solving, presence of depression, and hippocampal atrophy on neuroimaging. Without exhaustive questioning, MCI is often very difficult to detect clinically. The MMSE scores of patients with MCI are typically normal (24 to 30) but they often do poorly on the memory component of the test which requires the recollection of three words. Referral to a neuropsychologist is needed to accurately differentiate MCI from the cognitive impairment associated with normal aging. Patients suspected to have MCI should be counseled regarding AD's risk reduction strategies, such as mental and physical exercise, continuing social engagements, stress reduction, proper nutrition and aggressive treatment of cardiovascular risk factors. Finally, the MOCA is a newly developed screening tool particularly sensitive in MCI.

Definition and Diagnostic Criteria:

Dementia is a clinical syndrome involving loss of memory and intellectual function of sufficient severity to cause dysfunction in daily living. Its key features include:

- a gradually progressive course (usually over months to years)
- no disorder of alertness (i.e. normal conscientiousness)

The DSM IV Criteria for dementia are:

- memory impairment plus one of the four:
 - aphasia
 - apraxia
 - agnosia
 - disturbance in executive function and planning
- which impairs independent daily function and/or social interactions

Definition and Clinical Course:

The operational definition of Alzheimer's Disease is as follows:

- progressive neurodegenerative disorder
- characteristic clinical and pathological features
- individual variations
- multiple etiologies converging to neuronal loss

There is usually insidious onset and continuing cognitive decline, impairment in social or occupational functioning, not due to other CNS disorders and these deficits do not occur exclusively during the course of a delirium.

The average course of Alzheimer's disease is 8 - 12 years from the first onset of memory loss. Of course, the MCI phase can lengthen the two measurements. Obviously, there can be large variations in disease diathesis, with some individuals progressing extremely rapidly to death within two years, while others may survive as long as 20 years. The appearance of short-term memory loss is usually very insidious, and progression is gradual. With the passage of considerable time, one gradually observes erosion of the domains of cognition other than memory, including speech and language, visual/spatial function, judgment and insight, and executive function and planning abilities. Later, apraxia and difficulty in recognition become apparent. In typical cases, it usually is not until the 4th or 5th year of progression that one begins to observe changes in behavior and personality, and motor features. These include withdrawal, loss of initiative with apathy, agitation or temper outbursts, with paranoid delusions. Late stage motor features can include Parkinsonism, gait apraxia with falls (often due to poor motor planning) and, occasionally, myoclonic jerks.

Some individuals with incipient AD are aware of their declining abilities, but most patients with evolving AD will not acknowledge that they have memory dysfunction. Eventually, recognition may occur because of an apparent sudden crisis, such as getting lost, an accidental fall, or discoveries by neighbors or relatives of an unsafe, messy home environment, or acute confusion (delirium) during illness, after surgery or hospitalization, or environmental stress. Careful questioning at that juncture will usually reveal that cognitive impairment and dysfunction have been present for several years before the acute crisis. A decline in calculation abilities is one of the hallmark cognitive features of AD. Other clinical presentations can be psychosis, depression, and agitation/behavioral disturbances.

The functional decline seen in this disease is particularly distressing to patients and caregivers. Not surprisingly, it progresses in a reverse-hierarchical fashion, with the earliest shortfalls entailing such activities as chess playing, balancing a complicated financial portfolio, completing income tax forms or utilizing a computer. With the passage of time, functional impairment begins to include progressively less sophisticated activities and eventually erodes to the ability to perform basic ADLs. At any stage of this progressive deterioration, the critical detail is the presence of another family member to function as caregiver. Ongoing support will often compensate for the

functional deficits that are developing and will postpone significantly eventual placement into a long-term care facility. This fact heightens the importance of family physicians' vigilance for caregiver stress in the spouses or children of dementia patients. Ensuring that caregivers have regular breaks in their responsibility from others, as well as periods of respite care, are critically important to maintaining the dementiform patient in the community.

The procedural steps in evaluating cognition must be learned by students and residents alike. The approach to cognitively impaired individuals is a mandatory skill for health care practitioners in the new millennium. A suggested approach follows:

- I. **Seek out a reliable caregiver.** The clues and insights provided by a caring spouse, daughter or son, sibling or even well meaning neighbors are invaluable. It is also particularly useful to contact staff in an apartment building or a lodge, or friends that are particularly well known to the individual. By its very nature, dementia frequently deprives seniors of insight, to the extent that their own account, though far from useless, lacks the necessary scope to be completely reliable.
- II. **Map Out Carefully the Course of the Confusional State.** This points to the necessity to take a complete history of the cognitive decline, including duration, onset, and particularly events that have affected it's course. A useful question can be "how far back must we go to the point when this senior was mentally completely normal?" In neurodegenerative conditions, often it is impossible to pin down the onset of symptomatology, although conditions of vascular etiology or those triggered by head injury obviously will be different. Lewy Body dementia would frequently commence with an episode of prolonged delirium. Normal pressure hydrocephalus could present with gait changes, urinary urge incontinence and cognitive decline all in fairly close proximity to each other.
- III. **Explore All Domains of Cognition.** While indeed memory is the key domain to evaluate when considering dementia, speech and language function decline will be revealed by a history of the patient having difficulty with word finding or completion of sentences, or becoming much less conversational in recent months or years. Insight and judgment commonly are impaired, but seldom is the patient involved able to recount shortfalls in this area, so that caregiver reports are invaluable. Agnosia can be addressed by inquiring about difficulty in recognizing people, places, or things that should be familiar.

Apraxia is easily recognizable when it affects basic ADLs, but deterioration also can be identified by erosion of higher functions (see above). Visual/spatial decline will be characterized by difficulties in navigation with becoming lost in unfamiliar locales and also by confrontational testing with reproduction of intersecting pentagons or clock drawing. Abstraction is extremely difficult to elicit by history, but is detected by asking patients word similarities or differences, or proverb interpretation. Inquiries about alterations in personalities should be made and typical changes include withdrawal from previous more energetic, outgoing

pattern of behavior or becoming less compliant and more argumentative. Executive function erodes when the senior can no longer initiate activity, plan effectively or organize her actions in a meaningful, self-directed way to achieve a definite goal. These patients have difficulty with medication compliance, handling finances, driving or planning future events. Finally, neurobehavioral difficulties are vital to identify, so that questions about agitation, temper outbursts, suspiciousness, nocturnal wandering, verbal or physical aggression, hallucinations and delusions should be posed. Agitation is a common component of dementia; an acute episode may present a psychiatric emergency. Rapid treatment and resolution protects both the patient and caregiver from potential injury.

- IV. Perform an Objective Screening or Evaluation or Scale.** The Folstein Mini Mental Status Examination has been validated and standardized and functions as an objective screening probe for cognitive impairment.⁶ **It is not a gold standard for dementia, delirium, or depression**, but can be abnormal in all of these conditions. As a screening test, it can single out individuals with hitherto unrecognized cognitive decline, and does serve as a benchmark in time, so that its repetition in the ensuing months or year provides some input about the progression about the underlying condition. Its outcome should be weighted according to the patient's background, language, sophistication, and formal education.

The Folstein, in particular, focuses on certain domains, including orientation (10 points), registration (3 points), recall (3 points), attention and calculation (5 points), language (8 points), and construction (1 point). Bear in mind that the Folstein does **not** test for visual spatial capacity, agnosia, apraxia, executive function, insight and judgment, and personality or behavior. Hence, there are severe limitations to its scope. The learner should neither overrate the value of the Folstein Mini-Mental Status Examination, nor disdain it to the point where he/she does not use it at all.

- V. Compare Closely the Cognitive Loss to Functional Loss.** Documenting the functional decline and the exact level of patient function in terms of the instrumental and basic ADLs is another important step in this process. Have the cognitive changes affected the patient's ability? Are hobbies, special skills and socialization ongoing? Knowing the exact levels of independence, and the amount of support required are key steps in the evaluation of cognitive impairment.
- VI. Examine the Patient as Thoroughly as Possible, Especially the CNS.** The physical examination will involve a thorough general exam as well as a painstaking neurological review and some bedside neuropsychological testing as well. These findings can help sort out the differential diagnosis. Generally

⁶ Folstein MF, Folstein SE, McHugh PR. "Mini-Mental State": a practical method for grading the cognitive state of patients for the clinician. J. Psych Res; 12:189-198. 1975

speaking, uncomplicated Alzheimer's disease, especially stages I and II usually discloses a normal neurological examination without lateralizing pyramidal track findings and a normal gait and balance pattern. Advanced Alzheimer's disease (late stage II and III) often will reveal motor changes mentioned above obviously, it is not uncommon to observe lateralizing CNS findings with vascular dementia, or Normal Pressure Hydrocephalus (NPH) while Lewy Body dementia commonly reveals extrapyramidal changes as well. The frontal-temporal dementias may or may not disclose frontal release changes. Probing for medical issues, sensory deficits (in hearing or vision) EKG findings and concomitant medications that may adversely affect the cognitive performance are also worthwhile strategies.

VII. Communicate the Diagnosis, and Counsel the Caregivers (and patient) with Meaningful Information. This pertains to disclosure of the diagnosis and describing the future evolution of events within the short and medium term which is described later in this document. Community support measures should be included at this point, particularly, referral to the Alzheimer Society and its "first step" program.

Basic Laboratory Tests:

Extensive investigations for potential reversibility are no longer justified unless there are features in the presentation that would suggest an alternate diagnosis such as delirium due to a particular reversible cause. Hence, a few basic tests are suggested for general use.

Compared to previous days, a relatively short battery of investigation is recommended for the patient with advancing cognitive decline. All such patients should undergo the following blood tests:

- CBC
- TSH
- Electrolytes
- Calcium
- Glucose

In selected cases, it may well be desirable to pursue:

- B12, Folate
- BUN, Creatinine
- Liver Function Test
- VDRL
- HIV

Neuro Imaging in Dementia:

Neuroimaging (most commonly computerized axial tomography, CAT Scanning) has a role in detecting certain causes of dementia such as vascular dementia, cerebral tumor, normal pressure hydrocephalus or subdural hematoma. It is currently less effective in distinguishing AD or other cortical dementias from normal aging. Exaggerated cerebral atrophy may be present in advanced AD, but not as a diagnostic feature. Patchy, white matter lucencies occur in up to 12% of cognitively intact older individuals and are of uncertain significance.⁷ In primary care settings, some have stated that CT scanning could be limited to atypical cases, but others have recommended routine scanning.

The Canadian Consensus Conference on Dementia (1998) limits CT scanning to individuals who meet the following criteria:

- age less than 60
- rapid deterioration (over one or two months)
- unexplained decline in cognition or function
- "short" duration of dementia (less than 2 years)
- recent significant head trauma
- unexplained neurological symptoms (e.g. new onset of severe headache or seizures)
- history of cancer, especially in sites and types that metastasize to the brain
- use of anticoagulants or history of bleeding disorder
- history of urinary incontinence and gait disorder early in the course of dementia (as in NPH)
- any new localizing or lateralizing neurological findings (e.g. hemiparesis or a Babinski reflex)
- unusual or atypical cognitive symptoms or presentations (e.g. progressive aphasia)
- gait disturbance⁸

We must remember that AD starts at a molecular level, possibly decades earlier than can be detected by neuropsychological tests. Neuropathologic and neuroimaging data suggest that amyloid accumulation precedes the clinical onset of AD. Disease modifying agents would have to be used early to alter the course of AD. Therefore, preclinical diagnosis is necessary. Neuroimaging with MRI and PET can provide objective measures of preclinical disease and, when measured serially, the rate of change. Such information eventually can be used in prevention trials. Because there is a high frequency of clinically unrecognized CVD among patients who present with AD, neuroimaging with CT or MRI probably should be part of the routine dementia assessment.

⁷ Mark K, Lewis T, Wilcock G, Scott M, Box R. The Relationship Between White Matter Low Attenuation on Brain CT and Vascular Risk Factors: A Memory Study Clinic. *Age Ageing*; 24(5):411-415. 1995

⁸ Patterson CJ, Gauthier S, Bergman H, Cohen CH, Feightner JW, Feldman H, Hogan DB. The recognition, assessment and management of dementing disorders and conclusion of the Canadian Consensus Conference on Dementia. *Can Med Assoc J*; 160(suppl 12):5-15, 1999

Non-Routine Laboratory Testing

Determination of APO-E4 genotype is not currently recommended for use in diagnosis because the genotype is found in many elderly people without dementia, and is not found in many patients with AD. The most extensively evaluated biological markers of sporadic AD are AD protein levels and levels of both total and phosphorylated microtubular associated protein. These markers assessed in CSF or plasma, alone and in combination, are being studied to clarify their potential clinical applicability including sensitivity in the diagnosis of AD and mild cognitive impairment (MCI), specificity and discriminating AD from other dementias, and correlations with disease progression as well as APO-E genotype. At present, CSF screening for these potential markers is not yet recommended.

Classic Patterns of Presentation; Differential Diagnosis:

Regardless of numerous advancements and therapy, the diagnosis of dementia still depends heavily upon a careful history and gathering of information from cooperative sources, as alluded to above. History, together with physical neurological and cognitive examinations, a limited number of laboratory investigation and selective neuroimaging continue to constitute the mainstay of appropriate diagnosis.

Alzheimer's Disease is characterized by a very insidious onset, continuing decline of memory and at least one additional cognitive domain, not explained by other neurologic or systemic disorders. This most common cause of dementia in Canada accounts for approximately 60% of cases. Its progression and evolution eventually leads to a significant decline of other domains of cognition, as well as the loss of independence in function and decision-making. Later downstream, changes in personality and behavior can transpire as well. Support for such individuals is mandatory for them to remain in the community for an extended period of time.⁹

Vascular Dementia Exists as a Number of Syndromes Typically Associated with Cerebral Vascular Disease:

The long-standing tradition has been to characterize these by abrupt onset, step-wise decline, impaired executive function, gait disorder and emotional ability, with clinical or neuroimaging evidence of cerebral vascular disease. A temporal relationship between a vascular event and ensuing cognitive change always should be sought but is not always present. However, in recent years, experts have come to regard pure vascular dementia as quite uncommon, accounting for a total prevalence of 1 to 2% of all dementiform conditions. Furthermore, there has been an over focus on the classical step-wise features of large vessel cortical infarctions to constitute vascular dementia. This excludes other varieties

⁹ Gautier, S

of vascular dementia that finally have become included in its new definition.¹⁰ This new grouping also can include intracerebral hemorrhages, familial types of repetitive vascular injury, and small strategic infarctions. Commonly, in vascular dementia, some aspects of cognition remain well-preserved and the erosion of cognition is patchy. Lateralizing CNS pyramidal track signs such as atypical deep tendon reflexes or extensor plantar responses tend to lend clinical credence to this diagnosis. The risk factors of hypertension, atrial fibrillation, past CVA, diabetes mellitus and smoking are similar to many seen with Alzheimer's Disease.¹¹ Frequent is the concurrence of small vascular infarcts or lacunes associated with the typical Alzheimer-like pathology (so called mixed dementia).

Fronto-Temporal Degenerative Dementia is characterized by an insidious onset and slow progression of behavioral changes such as loss of social awareness, disinhibition, mental rigidity, inflexibility, preservative behavior and alarming loss of insight. The condition has a predilection for males in late middle age. Frequently, family members notice a striking decline in hygienic standards, prominent language changes, particularly a reduction in verbal output. At times, this pattern is dominated by an apathetic and withdrawn form, with individuals losing initiative and spending long hours of the day dosing in their favorite chair in the home. Initiative, insight and self-monitoring in particular is eroded. Besides this lethargic, apathetic presentation, one can see aggressive, disinhibited behavior in other cases of this disorder, frequently in psychiatric clinics.

Mixed Dementia - The association of AD with VaD is complex. Both conditions increase with age, they frequently occur concomitantly, and considerable overlap occurs in their symptomatology, pathophysiology, and co-morbidity. In the past, the co-existing of these two conditions in mixed dementia has been under-recognized. As already stated, memory and language dysfunction prevail in AD, while frontal lobe features (executive function) are commonly seen in VAD. Mixed dementia can be seen in a variety of combinations of the two conditions. Pure AD without cardiovascular pathology is seen in only 20% of suspected cases. Cholinergic deficits result from vascular lesions. The key point is that Mixed Dementia has unique presentation especially progression and response to treatment.¹²

Dementia with Lewy Bodies is a progressive cognitive decline with fluctuating symptoms, recurrent visual hallucinations, and spontaneous Parkinsonism with extrapyramidal signs. The diagnosis is supported by a presentation with prolonged delirium, repeated falls, hypersensitivity to neuroleptics, hallucinations, delusions, and syncope or transient losses of consciousness. In recent years, further scrutiny has revealed striking REM sleep abnormalities within which the LBD patient does not become atonic during REM sleep but rather "acts out his

¹⁰ Davies

¹¹ Nyemhuis DL, Gorelick PB. Vascular Dementia: A contemporary review of epidemiology, diagnosis, prevention, and treatment. JAGS 1998; 46:1437-1448

¹² Langa KM, Foster NL and Larson EB. Mixed Dementia: Emerging Concepts and Therapeutic Indications. JAMA, December 2004; Vol 292: No. 23, 2901-2988

dreams" in a manner that he can become somewhat violent during dreams, a pattern distinctly uncommon for normal individuals. The cholinergic deficits in lewy body dementia individuals are actually more profound than those found in Alzheimer's disease, making this condition quite responsive to cholinesterase inhibitors, particularly Rivastigmine (Exelon). The pathological changes include the curious admixture of Alzheimer's and Parkinson's disease pathology.

Normal Pressure Hydrocephalus is an uncommon disorder involving a relatively rapid progressing dementia associated with gait disturbance and urinary urge incontinence. Frequently, it is the gait apraxia that appears earliest. The features of this dementia are hardly unique, in that any advanced dementia can feature advanced cognitive decline, urinary incontinence and impairment of gait and balance. However, when these symptoms appear in close temporal relationship to each other, this diagnosis should be considered strongly and can be suspected when the CT Scan or MRI Scan show ventricular enlargement out of proportion to the degree of cortical atrophy. There is still some controversy about the precise diagnosis of this condition and the indications for and the efficacy of surgical treatments, but it is known that surgical intervention is most effective when applied early in the natural history of this disease. This form of dementia usually exhibits the clinical features of a subcortical dementia, including the abnormality of posture and gait, and the appearance of depression.

Alcohol-Related Dementia. One should not confuse this condition with the classic presentations of Wernicke-Korsakoff syndrome. In Wernicke's Disease, features include a classic ophthalmoplegia impairing lateral gaze, an associated truncal ataxia and mental confusion. In the Korsakoff syndrome, striking anterograde amnesia is seen with confabulation and poor temporal sequencing. In Korsakoff's, the striking memory decline often is not associated with erosion of other domains of cognition.

True alcohol-related dementia tends to resemble Alzheimer's disease quite closely, with an insidious onset and gradual progression. Not uncommonly, apathy and/or irritability predominate. Frequently, visuo-spatial difficulties are apparent with preservation and intact language abilities which differentiate it quite significantly from the typical Alzheimer's presentation. It is usually seen in the context of heavy, long-term ingestion of alcohol.

The Sub-Cortical Dementias include a number of disorders characterized by parkinsonian features with extra-pyramidal findings such as bradykinesia with difficulty with transfers. However, bradyphrenia (mental slowness) is prominent as is lack of initiative and executive dysfunction. Mood disturbances such as depression are common as well. The classic Parkinson's disease dementia often is subcortical in nature, although those individuals also have an increased risk of Alzheimer's disease when compared to age and sex match controls. Parkinson's-Plus syndrome not uncommonly are characterized by presentations of subcortical dementia.

Parkinsonism and Dementia. This constitutes one area where, the increased scrutiny of skilled scientific observers has rendered the waters muddier rather than clearer with the passage of time. It has been known for years that patients with Stage III Alzheimer's disease frequently will develop Parkinsonian features such as bradykinesia and extrapyramidal rigidity, albeit without the classic resting tremor. As stated above, the prevalence of Alzheimer's disease in Parkinson's disease patients is higher than in the age and sex matched population. A recent study by Emry has revealed that the neuropathology behind Parkinson's disease with dementia is identical to that seen in Lewy Body dementia.¹³ Hence, the clinical effort to separate out these two by the one year period in between the motor symptoms and the first appearance of dementia may be a somewhat futile effort, since both conditions will eventually prove to be identical under neuropathological review. Seniors with vascular dementia may have lacunes or infarctions involve the basal ganglia, and develop vascular Parkinsonism with a mixture of pyramidal and extrapyramidal CNS findings. The arrival of Lewy Body dementia on the scene, with its extrapyramidal CNS findings and sensitivity to neuroleptics has intensified the complexity of this combination. Finally, the Parkinson's-Plus syndrome and the entity of Multiple system atrophy characterized by dementia, Parkinsonism and dysautonomia round out this bewildering group.

Etiology and Pathogenesis of AD:

Although the picture is far from clear, undoubtedly there is more insight into the pathophysiology of AD than the other dementias. It is now understood that genetic factors play a crucial role in the risk of developing AD. Rare mutations in at least three genes are responsible for early onset familial AD. A common polymorphism in the apolipoprotein E gene (chromosome 19) is the major determinant of risk in families with late onset AD as well as in the general population. The apo E gene on chromosome 19 has three alleles- 2, 3, and 4. In the general population, the presence of apo E 4 genotype is associated with an increased risk of AD. For example, a population-based perspective study of individuals over age 75, revealed a relative risk for developing AD of 3.24 (95% CI, 1.67-6.25) in those possessing apo E4. However, the sensitivity (approximately 50%) and specificity (approximately 75%) for the presence of the apo E4 genotype in diagnosing AD is insufficiently high to guide diagnosis or accurately quantify genetic risk.¹⁴ The place of genetic testing and genetic risk assessment remains unclear at present.

Advanced age, however, remains the major established risk factor for AD although environmental variables (such as pesticides) may also have some role in disease expression. Other risk factors for AD include gender (female sex), head trauma, low education, systolic hypertension, and Down's syndrome. Diabetes and atrial fibrillation are best termed "probable risk factors" for AD. There are also protective factors against

¹³ Emre IM. *Lancet Neurol* 2003; 2:229-237

¹⁴ Roman GC, Tatemichi TR et al. Vascular Dementia: diagnostic criteria for research studies. Report of the NINDS-AIREN International Workshop. *Neurology*; 43(2):250-260. 1993

AD, and these include apolipoprotein E2 or E3, high education, long term use of anti-inflammatory drugs and long term use of estrogen in females.

Pathology and Structural Abnormalities of AD:

In terms of pathogenesis, it is now understood that many pathological mechanisms are operative in the development of AD. It is best regarded as a convergence syndrome, akin to scarring of the brain, which can result from different simultaneous disease activities. Although cerebral atrophy is a typical manifestation of AD, it does not distinguish normal aging from AD accurately enough to be diagnostic; this applies to neuroimaging as well as gross inspection at post mortem. However, microscopic examination reveals the critical features of the disease - a cerebral cortex peppered with neurofibrillary tangles and senile plaques.

Neurofibrillary tangles consist of aberrantly phosphorylated fibrillary proteins aggregated within the neuron cytoplasm. Their presence signifies the failure of the neuron to properly maintain its cytoskeleton, which is required to support the extraordinarily complex branching shape of its numerous processes. A small number of such tangles are a universal consequence of aging. However, it is the increased number and architectural distribution of the tangles that promote the cardinal pathology.

Senile plaques are more complex; they consist of extracellular deposits of amyloid material and are associated with swollen, distorted, neuronal processes called dystrophic neurites. The specificity of cerebral amyloid is provided by its major peptide component, beta amyloid, a short 40 or 42 amino acid fragment derived from the much longer transmembrane protein, beta-amyloid precursor protein (B-APP). The latter large molecule is a component of normal human brains and believed to play a role in maintaining neuronal integrity.

Starting in the 5th decade of life, progressively greater proportions of individuals develop cortical senile plaques until the 8th decade, when approximately 75% of the population is also affected. Plaques start as innocuous deposits of nonaggregated, putatively non-neurotoxic beta-amyloid. However, they can undergo an orderly sequential transformation into the **mature** senile plaques that are associated with the development of AD. The plaques and tangles bear a relation to dementia similar to that of atherosclerosis and infarction - as with atherosclerosis the primary lesions are common in aging, but clinical manifestations will appear after a certain density of these lesions is reached. In AD, this level will vary among individuals and will depend on genetic and environmental risk factors, as well as comorbid brain pathology. Readers must bear in mind that **only 50% to 60% of individuals fulfilling the neuropathological diagnosis of AD have dementia or significant cognitive decline during life. Moreover, no neuroimaging or laboratory markers now exist for reliable pre-symptomatic diagnosis AD, which is a scourge for health care workers trying to solve the Alzheimer mystery.** Although not a structural change, it is worthwhile to include the striking neurotransmitter changes that result in the Alzheimer disease process. Most prominent of these is the glaring deficiency of acetylcholine (ACH) but others include Dopamine, Serotonin, Norepinephrine, Somatomedin, as well as the changes (usually

excesses) in glutamate. Increased levels of the latter, in combination with beta-amyloid, are exceedingly toxic to brain tissues.¹⁵

Ethical Issues in Dementia:

Loss of insight, decline in capacity to make reasonable decisions and risk to others must be carefully balanced against preservation of autonomy. Such difficult ethical issues include the following:¹⁶

- participation in research
- decision making: respecting individual choice

- quality of life
- behavior control
- use of restraint
- advanced directives
- end of life decisions

Several publications have looked at these difficult issues. The reader is particularly encouraged to read the document produced by the Alzheimer's Society of Canada entitled "Tough Issues".

Disclosure of diagnosis:

The case for informing an individual of the diagnosis rests upon the patient's right to know (principle of autonomy). Knowledge of the diagnosis can allow for future planning (e.g. Advanced Directives, Power of Attorney, planning for future living arrangements). Disclosure allows for consent to treatment and participation in research. It facilitates the dialogue between patient and caregiver, avoiding the conspiracy of silence that might otherwise exist. Arguments against disclosure include the risk of depression, and, in rare instances, of suicides, concern about diagnostic uncertainty and the lack of effect of disease modification. Most senior and caregivers of AD state that they wish to be told their diagnosis.

Care Plan:

It is of paramount importance that the disclosure of diagnosis is accompanied by an effort to broker discussion with patient and family such that a care plan is designed. Caregivers and patients must be informed about the valuable educational activity of the local Alzheimer's Society, and provided access to helpful literature on the topic. If necessary, support for patients and caregivers should be instituted. This usually involves a dynamic connection between the family physician and home care case managers in the community. When necessary (but not before) services related to dementia care should be set in motion, after some discussion with family members and

¹⁵ Desai AK, Grossberg. Diagnosis and Treatment of Alzheimer's _____. Neurology 2005; 64(Suppl 3):S34-S39

¹⁶ Munoz DG, Feldman H. Causes of Alzheimer's Disease. Can Med Assoc J; 162(1):65-71. 2000

home care workers. As well, caregiver needs must be addressed. The cessation of driving in dementia frequently is a painful process for physicians, patients, and caregivers. These individuals have multiple roles in caring for individuals with dementia. Their report is often reliable as objective measures of cognitive decline and may alert health care professionals to the presence of dementia. They play a vital role in promoting direct care for dementia patients. Specialists rely on caregivers to monitor change in status and symptoms and need to include them in treatment plans. Absence of caregivers is a major predictor of earlier institutionalization of individuals with dementia. Higher perceived caregiver burden also leads to earlier institution.

Up to 50% of caregivers experience significant psychiatric symptoms during the course of their caregiving. Despite these negative consequences, many caregivers also report of sense of satisfaction with their role, particularly a sense of accommodation in keeping their loved ones at home.

Patients and caregivers should be strongly encouraged to articulate future directives. The optimal time and circumstances for such decisions are in a relaxed setting at home without urgency or stress involved. Then, the complicated issues of continuing to drive, personal directives, and enduring power of attorney can be discussed thoroughly and decided upon.

Care Plan/Treatment:

The treatment of the Alzheimer patient involves considerably more than simply providing the new medications that are now available for this disorder. Some prudent steps include:

- regular office visits (2-3 months) to optimize medically, all such patients
- correct sensory disturbance such as severely impaired vision and/or hearing when possible
- provide caregiver support and counsel as well as utilizing the local chapter of the Alzheimer's Society; future plans such as enduring power of attorney and driving issues should be discussed as well
- advise caregivers and patients clearly about changes of environment or upcoming hospitalization. It is extremely common for such individuals to become delirious with a hospitalization
- an exercise and activity program should be advocated to preserve patient mobility
- home care and respite care should also be utilized when indicated
- continual focus on reduction of medications used

After years of speculation and failed trials, finally some compounds have emerged as improving the symptoms of Alzheimer's disease. Thus far, these have been cholinesterase inhibitors, whose action raises the minimal levels of acetylcholine (Ach) in the synaptic clefts in the brains of AD patients. The earliest of these was Donepezil (Aricept) which came onto the market in September of 1997. It is probably, one quarter to one third of the group will show a noticeable improvement to caregivers. The other responders, while not exhibiting striking improvement, appear to deteriorate more slowly

in the future after the drug has been started. Aspects of improvement include alertness and vigilance, speech and language function, and abilities in day-to-day function.

Subsequently, it was shown in a Canadian study that patients with moderately advanced to severe Alzheimer's disease also benefit from exposure to Donepezil.¹⁷ An unexpected benefit was the discovery that cholinesterase inhibitors have a significant effect on behavioral changes seen in more advanced stages of AD. Features of anxiety, depression, agitation, frequently ameliorate after exposure to this class of medications. The duration of action of Aricept is prolonged, necessitating a once daily dosage that can be taken morning or evening, before or after meals.

Rivastigmine (Exelon) was the next cholinesterase inhibitor to appear. Besides inhibition of cholinesterase, this compound appears to also inhibit butyrylcholinesterase, an enzyme that becomes much more active in the metabolism of ACh as Alzheimer's disease progresses. Rivastigmine appears to inhibit both enzymes, and has also been demonstrated to have a robust effect on individuals with Lewy Body dementia. It has a low potential to interact negatively with other drugs. Some concern has arisen about the tolerability of Rivastigmine (Exelon), necessitating a slow and cautious titration.

The third such compound is Galantamine (Reminyl) which was released in August 2001. This agent also inhibits acetylcholinesterase, as well as modulating the nicotinic receptors of ACh in a positive way. By acting on these receptors, Reminyl actually increases the release of ACh. Both Aricept and Reminyl are taken once daily, and Exelon on a BID basis. Galantamine has been proven-effective in mixed dementia cases. All three cholinesterase inhibitors tend to produce symptomatic improvement in AD patients. Hard evidence shows benefit to large group of patients for up to a year; anecdotally, the effect often appears to continue for significantly longer than that, but this is speculative.

Treatment of AD focuses on establishing an early accurate diagnosis, early institution of AChEIs and/or NMDA receptor/targeted therapy, treating medical comorbidities and dementia related complications, ensuring that appropriate services are provided, addressing long term well-being of caregivers, and treating the behavior and psychological symptoms of AD. The cholinesterase inhibitors and Memantine provide modest but clinically relevant symptomatic benefits in cognition, function and behavior. Although they may have intrinsic disease modifying activity, this remains to be proven. Early institution of these medications may delay the onset of behavioral and psychological symptoms of AD by one year. Emerging data support beneficial effects of CHEIs in moderate to severe AD and in long term care situations. CHEIs may be helpful in DLB and VAD and Memantine has been proven helpful in VAD.

Memantine, a reversible inhibitor of the NMDA receptors, has emerged onto the scene for the treatment of later-stage AD. Glutamate is a compound that in regular amounts facilitates memory and learning. As AD pathology worsens, the glutamate levels in the

¹⁷ Feldman H, Gauthier S, Hecker J et al. A 24-week randomized double-blind study of Donepezil in moderate to severe AD. *International Psychogeriatrics*.

brain tend to rise, which can lead to calcium influx into neurons and their subsequent death. Memantine (EBIXA) reversibly blocks the NMDA receptor in such a way that memory and learning can still transpire while the toxic effect of high levels of glutamate does not cause cell death. This compound is shown to be effective in preserving a degree of cognitive function and inhibiting the behavioral changes with advanced Alzheimer's disease.

Experimental Work:

Numerous trials are testing new compounds in the treatment of AD. Beta and Gamma Secretase inhibitors are under scrutiny to see whether they can reduce the formation of beta amyloid peptide from amyloid precursor protein (APP). These compounds would mitigate against amyloid deposition and maturation. As well, the Alzheimer vaccine after failing because of meningo-encephalitis in six subjects treated in their Phase II trial, seems to be making a comeback. Modification of this approach using monoclonal antibodies will be tested rather than trying to stimulate the development of antibodies against A-beta in subjects. Hence, there is still hope for this approach. Research has developed ways to induce antibodies against the amino acid chain that predispose to beta amyloid peptide.

There are three main classes of disease modification approaches that can be defined:

- (1) one that is broadly neurotrophic or neuroprotective;
- (2) one that targets specific aspects of AD pathology; and
- (3) one that is based on epidemiologic observation.

Among these anti-amyloid treatment is now the most active area of investigation. Oxidative stress and cell cycle related abnormalities are early events in AD, occurring before any cytopathology can be identified and together may propagate disease pathogenesis. Therefore, antioxidants are an AD prevention strategy appropriate for investigation.

Behavioural Assessment Tool

Resident's Name _____

Date of Assessment _____

Describe the Behaviour(s) _____

Is this behaviour new? Yes No

When does this behaviour occur? _____

Names of the individuals involved in the assessment: _____

Identify possible causes of this behaviour

Consider these Psychiatric Influences (eg)	Summarize the Psychiatric Influences	Action Required
Dementia <ul style="list-style-type: none"> ◆ Memory ◆ Communication ◆ Planning ◆ Judgment ◆ Insight ◆ Self Care 		
Delirium <i>Is this a sudden change in cognition/ Behaviour?</i> <ul style="list-style-type: none"> ◆ Day/night Reversal ◆ Difficulty paying attention ◆ Agitated behaviour 		
Depression <ul style="list-style-type: none"> ◆ Mood ◆ Sleep ◆ Appetite ◆ Somatic complaints ◆ Lack of energy ◆ Suicidal thoughts 		
Psychosis <ul style="list-style-type: none"> ◆ Hallucinations ◆ Delusions 		

Other		
Consider these Physical Influences (eg)	Summarize the Physical Influences	Action Required
Acute Illnesses ◆ Infection		
Chronic Illness		
Pain		
Constipation		
Incontinence		
Sleep		
Appetite		
Dehydration		
Weight		
Medications ◆ New ◆ Change		
Mobility		
Hearing		
Vision		
Other		
Consider these Physical Environmental Influences (eg)	Summarize the Physical Environmental Influences	Action Required
Physical environment ◆ Noise Level ◆ Lighting Level ◆ Temperature		
Space to move around		
Access to outdoors		
Private space		

Personalized room		
Appropriate signage		
Social Environmental Influences (eg)	Summarize the Social Environmental Influences	Action Required
Communication abilities		
Decision-making opportunities		
Response to others <ul style="list-style-type: none"> ◆ Residents ◆ Staff ◆ Family 		
Participation in facility life		
Staff approach <ul style="list-style-type: none"> ◆ Personal Space ◆ Tone of voice ◆ Body language 		
Other		

Summary of Actions Required: *Further assessment, lab tests, care strategies, medications, consults, etc.*

Summary of Actions Required	By Whom	Date Completed

Date to review the outcomes of actions: _____

Psychiatric Influences (Cue questions)

(Consider the following)

Dementia

- ◆ Does the resident have a diagnosis of dementia?
- ◆ Does the resident have a chronic progressive memory loss?
- ◆ Is the resident chronically disoriented to: time, place, person?
- ◆ Does the resident have difficulty solving problems, making choices, making decisions?
- ◆ Does the resident have difficulty with ADL's which are related to cognitive impairment, not physical impairment?

Delirium

- ◆ Has there been a relatively recent onset of confusion (days to weeks)?
- ◆ Do the symptoms of confusion fluctuate throughout the day and night?
- ◆ Is the resident restless and awake during the night and sleepy during the day? (day/night reversal)
- ◆ Does the resident have hallucinations/illusions?
- ◆ Does the resident have a recent onset of difficulty with ADL's which are related to cognitive impairment, not physical impairment?
- ◆ Are there any signs/symptoms of acute illness or episodic chronic illness? (infections, CHF, COPD)
- ◆ Has there been a new or change in medication?

Depression

- ◆ Has the resident's mood been consistently depressed for at least two weeks?
- ◆ Is the resident irritable?
- ◆ Have you observed a decrease in appetite, weight, energy or sleep?
- ◆ Has the resident talked about wanting to die or to kill himself or herself?
- ◆ Is there a decrease in the resident's ability to think clearly or concentrate?

Psychosis

- ◆ Describe the hallucination or delusion.

Other

- ◆ Does the resident have a diagnosis of any of the following psychiatric disorders: Schizophrenia, Delusional Disorder, Bipolar Mood Disorder, Anxiety disorder, etc?

Briefly describe the behaviours that you observe in the resident that fit the syndrome of dementia, depression, delirium or other psychiatric disorder.

Identify the action to be taken such as other assessments, which are required. (MMSE, Geri-snap, behaviour logs, Geriatric depression scale, sleep logs, weight charts, food and fluid intake logs, Confusion Assessment Method [CAM]).

Physiological Influences (Cue questions) (Consider the following)

Acute Illness

Are there symptoms of any common acute illnesses?

- ◆ Infection (urinary tract infection, pneumonia, wound)
- ◆ Constipation
- ◆ Metabolic abnormalities (electrolyte imbalance, hypo/hyperglycemia, etc.)
- ◆ Dehydration
- ◆ Skin conditions (cellulitis, ulcers)
- ◆ Vascular conditions (MI, CVA)

Chronic Illness

Have any of the resident's chronic illnesses become unstable?

- ◆ Chronic Congestive Heart Failure, Chronic Obstructive Pulmonary Disease, Arthritis, Diabetes, etc.)

Pain

- ◆ Is there a new onset of pain?
- ◆ Is there a worsening of chronic pain?
- ◆ Have there been any unwitnessed falls?
- ◆ Is the resident able to report pain?
- ◆ Analgesia issues:
 - Is the resident receiving PRN pain medication? (how often is it being used?)
 - Does the resident require regular pain medication?
 - Is the form of medication appropriate for the resident and the degree of pain? (Pill, liquid, injection, patch)

Constipation

Incontinence

Sleep

Appetite

Dehydration

Weight

Any concerns of changes?

Medications

- ◆ Is the resident a new admission? Were they actually taking their medication at home?
- ◆ Have there been any changes in medication dose or frequency?
- ◆ Has the resident been refusing medication?
- ◆ Have any new medications been started?

Other Physical Influences including mobility, hearing and vision.

Briefly describe the symptoms that you observe in the resident.

Identify other assessments which are required. (physical exam, C&S, blood work, x-rays, intake & output logs, weight logs, BM records, movement charts, medication records, pain assessment)

Psychosocial Influences (Cue questions) (Consider the following)

Personal Routines

- ◆ Does the resident have specific preferences around their daily routine?
- ◆ Are there personal preferences that clash with facility routines?

Early Life Factors and Life Events

- ◆ Has the resident had an abusive/neglectful childhood? (could lead to lack of trust or disrespect for authority).
- ◆ Has the resident experienced any major life events? (war, economic depression, etc.)
- ◆ What was the resident's work history?

Significant Relationships

- ◆ Does the resident have a good social support system?
- ◆ Have the social supports changed since coming to the facility?
- ◆ What is the state of their current relationships?
- ◆ Is the primary support away or experiencing added stress in their life at the moment?

Personality Style

- ◆ How does the resident/family and friends describe the resident's personality style?
- ◆ In the past, how have they handled stress? Are they comfortable talking or do they withdraw when stressed?
- ◆ Do they tend to be more independent or dependent?
- ◆ Do they tend to have a more rigid/obsessive personality style?
- ◆ Do they tend to be quiet and self-absorbed or always looking after others?
- ◆ How has placement in the facility affected their sense of role, purpose, and self-esteem?

Losses

What recent losses has the resident experienced?

- ◆ Loss of independence, loss of autonomy?
- ◆ Loss of a loved one?
- ◆ Loss of their customary roles?

Interests (see attached Individualized Interest Chart)

- ◆ Give details as to what increases the stress/anxiety of the resident
- ◆ Also include what gives them pleasure (useful for care planning).

Cultural/Spiritual beliefs and values

- ◆ Are the resident's cultural/spiritual needs being met?
- ◆ Is there conflict between the environment and the resident's culture?
- ◆ Is there conflict between cultural expectations and how care is being delivered?

Briefly describe the symptoms that you observe in the resident.

Identify other assessments which are required. (SW interview, Activities assessment, Pastoral Care interview, etc.)

Environmental Influences (Cue questions)

(Consider the following)

Physical Environment

- ◆ Is the environment over-stimulating/not stimulating enough?
- ◆ Is it too hot/cold/bright/dark/noisy, etc?
- ◆ Are there private spaces?
- ◆ Are there assistive devices to encourage independence?
- ◆ Space to move around?
- ◆ Personalized room?
- ◆ Appropriate signage?
- ◆ Cues to reminisce about or connect with the past?
- ◆ Space to spontaneously interact with others?
- ◆ Access to outdoors?

Social Environment - Communication Abilities

- ◆ Does the resident have a cognitive, physical, vision or hearing deficit that will affect communication? Describe.
- ◆ Is English their first language?

Decision-Making Opportunities

- ◆ Resident/family input into decisions?

Response to Others

- ◆ Does the behaviour increase/decrease in the presence of others (in the dining room, group activities, in crowded areas, etc.)
- ◆ Is the resident responding to the behaviours of those around him/her?

Participation in Facility Life

- ◆ How does the resident participate in the events of the facility?
- ◆ Are they involved in meaningful activities? How often in the day?
- ◆ Do they initiate interaction with others? How?

Staff Approach

- ◆ Is there an approach that works well with the resident?
- ◆ Does my body language (touch, posture, how fast I move, how loud I speak, my facial expression, etc.) affect the way the resident behaves?

Briefly describe the symptoms that you observe in the resident.

Identify other assessments which are required (OT, physical exam including vision, oral hearing, and medication review).

Summary of Actions Required

Identify: the actions required as per the assessment sheets

Indicate: who will be responsible for the action (name and discipline)

Identify: the date to be completed

Other References

Websites for Dementia:

Alzheimer Society of Canada

<http://www.alzheimer.ca>

DASN Dementia Advocacy and Support Network

<http://www.dasninternational.org/index.html>

National Institute of Neurological Disorders and Stroke (NINDS)

<http://www.ninds.nih.gov/index.htm>

Alzheimer's Disease Education and Referral Center - ADEAR (NIA)

<http://www.alzheimers.org>

Family Caregiver Alliance

<http://www.caregiver.org>

Alzheimer's Association (US)

<http://www.alz.org>

Alzheimer's Society (UK)

<http://www.alzheimers.org.uk>

Other Readings:

Snowden D. (2001) *Aging with Grace. What the Nun Study Teaches us about Leading Longer, Healthier, and More Meaningful Lives*. Bantam Books: New York, New York

Laurence M. (1964) *The Stone Pony*. McClelland and Stewart: Toronto, Ontario