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Art by Melissa Andrew

EDITORIAL BOARD

ON THE COVER

Dissociative Fugue, by Melissa Andrew

For many, the familiar wavers on the brink, often dipping into the confusion and unknown of what once seemed so close to reality. It is this unfamiliarity with the familiar that defines Alzheimer's in my thoughts.

I have chosen to represent this winding stream of consciousness that I envision in sound; a meandering path along the edge of a harsh cliff that separates what is dear and understood from the frightening depths of the nearly recognized but not wholly comprehended. Music and sound may be for some one of the last links to a real past, and thus to reality. A familiar melody or sound reunite a listener with a memory long set aside. I imagine this link to self and identity to be a saving grace amidst the tumult of a depersonalizing condition such as Alzheimer's.

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A Broad Spectrum of Issues

by Paul J. Coolican, MD, CCFP, FCFP

As family physicians, many of us are exposed to a wide array of symptoms, behaviours and thought patterns that we pile into a large basket and label "dementia." From the elderly widow whose daughter calls from far away complaining that her mother is confused, to the hospitalized man whose post-operative delirium exposes an underlying cognitive disorder, we try to assess the causes, treat the reversible ones, slow down the irreversible ones and help caregivers both at home and in "a home" deal with aberrant behaviours and psychological symptoms. This issue of the *Canadian Alzheimer Disease Review* covers a broad spectrum of issues relevant to family doctors that may help us in our diagnosis and care of dementia patients and their families.

Dr. Robert Frerichs, a clinical neuropsychologist, gives a down-to-earth description of the role of neuropsychological assessments in older adults (page 4) as a diagnostic tool, as an aid to determine competency issues and driving risk, and to evaluate other skills that might determine a patient's ability to remain independent. The information is given in a concise and straightforward manner.

In the first of two articles, Drs. Loy-English and Feldman provide a good review of the parkinsonian dementias (page 16), a spectrum of disease that includes dementia with Lewy bodies (DLB), the dementia of Parkinson's disease (PDD) and the advanced stages of Alzheimer's disease (AD). This article focuses on pathologic features, clinical features and clinical differentiation of these diseases. It includes criteria for diagnosing DLB and reviews the temporal differences between DLB, PDD and severe AD with parkinsonism. While a few years ago this might have been of academic interest only, new treatment options and symptom management of these conditions may make early diagnosis invaluable.

Between these articles (page 11) is a comprehensive review of delusions in AD by Dr. Corrine Fischer, a geriatric psychiatrist. Defined as "fixed false beliefs that are not culturally bound," there are seemingly as many difficulties classifying delusions as there are delusions themselves! From the famous Capgras syndrome to delusions of theft, of grandeur, of simple or complex persecution, phantom boarders and spousal infidelity, Dr. Fischer reviews the inconsistencies in the literature. She brings together their importance in AD—namely, they occur in one third of AD patients and are associated with increased caregiver burden and early institutionalization. It is a worthwhile read.

Personal Revelations, Experiences and Reflections of an AD Caregiver is a series of vignettes from Roberta Bedard about the experience of living with her husband Ray, an AD patient. This chapter (page 24), particularly the section titled "Laughing," is a poignant and elegant portrait of love and human strength that shocks me with its simple truth. We are privileged to have such a piece in this journal. As physicians, we are sometimes called upon to give hope where there is not much to find. This work will help me in that task.

Paul J. Coolican, MD, CCFP
Family Physician, St. Lawrence Medical Clinic
Morrisburg, Ontario
Active Staff, Winchester District Memorial Hospital
Winchester, Ontario

When Should An Older Adult Be Referred to Neuropsychology?

Neuropsychological assessments provide a systematic, evidence-based and comprehensive approach to assessing an individual's cognitive and emotional functioning, and can complement the results obtained from other investigations. Neuropsychological assessment is typically viewed as valuable by both consumers and referring agents, but is not appropriate for all older adults with either a known or suspected cognitive impairment.

by Robert Frerichs, PhD, CPsych

Changes affecting concentration, memory, communication, or even decision-making are not uncommon among older adults. Some of these cognitive changes are merely "slips," to which we are all vulnerable, some reflect normal aging, while others may be a clinically significant symptom. For example, cognitive symptoms manifest with psychiatric difficulties such as depression and anxiety, medication misuse, substance abuse and a variety of medical conditions (*e.g.*, hypertension, diabetes, hypothyroidism); they are also a defining feature of delirium and dementias, such as Alzheimer's disease (AD). Given the prevalence and non-specific nature of cognitive symptoms, it can be challenging to determine the significance of these symptoms in older adults. Yet this situation is often faced by healthcare professionals and will undoubtedly continue to occur with the rapid growth of this segment of our society.[1,2]

Neuropsychologists, as consultants to various health professionals, are in a position to provide valuable information to aid healthcare providers who work with older adults. The demand for neuropsychological assessments has grown over the years and this has been particularly noticeable in the field of geriatrics—where the contributions of neuropsychology have been recognized in the widely used research criteria for AD,[3] as well as in various published guidelines pertaining to geriatric assessment.[4-7]

The purpose of this article is to provide some background about neuropsychology and the role of geriatric neuropsychologists, describe the neuropsychological assessment process, and identify common questions that can be addressed by this type of evaluation. Suggestions are provided to help determine whether or not a neuropsychological referral may be appropriate, and what should be considered when referring to a neuropsychology service.

Dr. Frerichs is a clinical neuropsychologist with the Northern Alberta Regional Geriatrics Program at the Glenrose Rehabilitation Hospital, Edmonton, Alberta.

What is Neuropsychology and What is a Neuropsychologist?

Neuropsychology is the scientific study of the relation between brain-functioning and how a person thinks, feels and acts. It is concerned with understanding cognition, emotions and behaviours not only in the context of normal central nervous system development across the lifespan, but also with respect to compromised functioning that results from disease, disorder and injury. Clinical neuropsychology refers to the applied practice of neuropsychology in which knowl-

edge of brain-behaviour relations, assessment tools, and large databases of statistical information about normal and abnormal functioning are combined to assess an individual's mental abilities and emotional state, and/or to provide an intervention.

Neuropsychologists (or clinical neuropsychologists) are PhD-level clinical psychologists who have specialized training and experience in neuropsychology. Some neuropsychologists work in private practice and others work in hospital/clinic settings as part of multidisciplinary teams, or as consultants for primary healthcare providers. It is important to note that not all neuropsychologists are competent to practice with older adults, as their background may emphasize work with other demographics, such as children and adolescents.

What is a Neuropsychological Assessment?

Neuropsychological assessment involves the use of specialized tests, but is more than testing, *per se*. It is an evaluation that involves the integration of multiple sources of information about a patient, including data collected from an interview, collateral information (*e.g.*, informant reports, relevant medical data) and an individual's performance on standardized psychometric measures. A neuropsychologist selects tests from a broad array of cognitive measures designed to assess intelligence and global cognitive skill, attention/concentration, memory and learning, receptive and expressive language, academic skills, executive functioning (*i.e.*, problem solving, conceptualization, planning, organization, sequencing, mental flexibility), praxis, visuospatial and constructional abilities and perceptual and motor skills. Measures of mood state, behaviour and personality are also frequently included.

A neuropsychological assessment is an evaluation that involves the integration of multiple sources of information about a patient, including data collected from an interview, collateral information and an individual's performance on standardized psychometric measures.

Not all geriatric neuropsychological assessments employ the exact same measures or assessment methods. The selection of tests is determined by the neuropsychologist and may be influenced by several factors, including the specific referral question and the characteristics of the individual. The age and education of the patient, his/her culture, language facility, sensory/motor limitations and ability to tolerate testing (*e.g.*, due to pain, fatigue, comorbid medical conditions, and/or lack of motivation) may dictate the use of certain measures over others.

Given the demands and rigours of testing, brevity is an important consideration when working with older adults. The length of an evaluation varies depending on the individual and the referral question, though it is not uncommon for assessments to last from three to four hours. Persons who present with obvious and extensive cognitive impairment may complete relatively few tests, whereas those who are high-functioning will often complete more. Most assessments are completed during one visit, although a visit may be segmented in instances where fatigue is a factor. During testing, the patient works one-on-one with the neuropsychologist or a psychometrist (a specially-trained technician). Testing usually takes place in a room that is free from distractions and the individual completes most tests sitting at a table or, possibly, using a computer. In special circumstances, the tasks may be completed at bedside. An assessment typically involves having the examinee attempt to answer questions, solve problems and complete paper-and-pencil tests to the best of his/her ability.

Once testing is complete, the results are interpreted by comparing the individual's performance to a normative standard that accounts for the influence of age, education and gender on test scores. When interpreting a cognitive profile, neuropsychologists document strengths as well as impairments, in addition to patterns across cognitive domains and pathognomonic signs of cerebral dysfunction. By comparing an individual's performance to an estimated premorbid level of functioning (or to previous test data, when available), judgments can be rendered about the probability that cognitive change has occurred.

What Purpose Might a Neuropsychological Assessment Serve?

One should expect neuropsychological assessments to provide information about strengths and weaknesses in an individual's cognitive abilities and emotional state. This, in turn, may facilitate the diagnostic process, elucidate the impact of having a particular disease/injury, and/or facilitate treatment planning.

In the context of working with older adults, the most common aim of the evaluation is almost always diagnostic in nature. One basic issue is determining whether or not cognitive impairment is present. The established sensitivity of neuropsychological measures to cognitive dysfunction can help distinguish normal aging and benign cognitive change from pathological processes. An individual's cognitive profile, in addition to other information collected during the assessment, may be used to reliably determine whether he/she meets criteria for a dementia syndrome at the earliest possible stage,[8] or mild cognitive impairment[9] which, for at least some individuals, represents a prodromal dementia state. Neuropsychological assessment may also contribute to differential diagnosis, particularly distinguishing dementia from depression and other psychiatric causes of cognitive impairment. Cognitive profiles and assessment data may also help discriminate among various etiologies of dementias and cognitive impairment (AD, vascular dementia, dementia with Lewy bodies, frontotemporal dementias, alcohol-related dementias). However, there may be considerable overlap in these profiles due to the same brain regions being affected and the frequency of comorbidity (*e.g.*, AD and vascular changes). Baseline and follow-up neuropsychological evaluation increase the sensitivity to detecting progressive dementias,[10] often at early stages when the cognitive changes are subtle.

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Neuropsychological assessments may be used to describe the impact of various conditions on an individual's behaviour, mood and thinking. Evaluations may be used to gauge strengths and weaknesses due to acquired brain injuries, cerebrovascular accidents, medical conditions, seizure disorders, chronic substance use and exposure to toxins. The information may be used to gauge the relative severity of a dementia as cognition deteriorates over time. Finally, it may also be useful to track changes in cognition following interventions such as coronary bypass surgery, pallidotomy, transplant surgeries, rehabilitation or the implementation of a medication regimen.

Based on the data from a neuropsychological evaluation, recommendations can be detailed to guide treatment and management decisions and to enhance the

cognitive functioning of the older adult. For example, confirmation of a dementia or a depressive disorder can help clarify the most appropriate treatment avenue (*e.g.*, medication, psychotherapy, behavioural management and/or referral to other professionals or support agencies). Strategies and techniques to optimize cognitive functioning and minimize the impact of cognitive dysfunction can be identified. Test findings may clarify whether a person would likely benefit from counselling or other therapies to adjust to his/her cognitive or emotional changes. When cognitive problems are found on examination, they may highlight the need to address safety issues at home (*e.g.*, risk of forgetting stove burners on, difficulty remembering to take medications) or the need to increase support and structure (*e.g.*, home care, hired assistance, support from family and friends). At times, neuropsychological assessment data may contribute to decisions about moving the patient to a more supportive living setting, if required.

Neuropsychological assessments may also contribute to planning for the future by addressing concerns such as driving and decision-making. To be clear, neuropsychological tests were not specifically designed to assess driving skill or competence. A neuropsychologist may identify "red flags" based on a pattern of cognitive impairment that could compromise driving ability (*e.g.*, problems with divided and sustained attention, impulsivity and judgment and visuospatial deficits). In practice, an on-road evaluation remains the most reasonable test of an older adult's actual driving ability. Decision-making is an important and complex matter that frequently arises with older adults who have a known or suspected cognitive impairment. Guardianship and trusteeship are ultimately legal matters, but neuropsychologists may be asked to render opinions about an individual's ability to make decisions about healthcare, accommodation and finances (including the need for guardianship or trusteeship, or the need to enact an advance directive or an enduring power of attorney). Neuropsychological assessment can provide a way to investigate a person's comprehension and problem-solving skills, and insight into his/her difficulties.

Making a Referral for a Neuropsychological Evaluation

Suspicion of cognitive dysfunction or decline prompts most referrals to a neuropsychology service. The index of suspicion should be based on observations, cognitive screening, symptoms reported by the individual and changes noticed by family members and caregivers that might indicate a decline in cognition (*e.g.*, problems with memory, problem solving or language) or the person's ability to function independently at home (*e.g.*, difficulty with self-care, dressing, preparing meals, driving, taking care of finances, managing medication).

An individual's cognitive profile, in addition to other information collected during the assessment, may be used to reliably determine whether they meet criteria for a dementia syndrome at the earliest possible stage, or mild cognitive impairment which, for at least some individuals, represents a prodromal dementia state. Neuropsychological assessment may also contribute to differential diagnosis, particularly distinguishing dementia from depression and other psychiatric causes of cognitive impairment.

Prior to making a referral for neuropsychological assessment, physicians usually rule out medical causes that might account for a patient's cognitive symptoms. If medical conditions cannot be ruled out completely, the referrals should at least be delayed until the individual is medically stable (*i.e.*, no infections, untreated con-

Table 1

Examples of Geriatric Neuropsychology Referral Questions and Requests

- Does this person have cognitive impairment? Please assess the nature and extent of this individual's cognitive difficulties.
- Is there evidence of cognitive decline?
- Does he/she meet the criteria for a dementia?
- Can the patient's cognitive symptoms be explained by a condition or disease?
- Is the patient's presentation most consistent with a depression or a dementia?
- Is testing suggestive of a specific dementia (*e.g.*, AD, vascular dementia, dementia with Lewy bodies)?
- The patient has a diagnosis of "X" [or, is undergoing surgery for "X"]; a baseline evaluation of cognitive and emotional functioning would be appreciated.
- Has this individual's cognition improved/declined since his/her previous assessment?
- What strategies or treatments can be recommended to assist the patient?
- Is he/she able to make reasonable decisions about specific concerns (*e.g.*, medical treatment, living arrangements, finances)?
- Does the patient have the capacity to complete an enduring power of attorney? Personal directive, advance directive, or living wil?
- Is guardianship or trusteeship required?
- Will he/she require increased support? Be able to live independently?
- Is a driving assessment recommended?
- Are there any cognitive, emotional or behavioural concerns that might affect the patient's ability to participate in treatment?

ditions, intoxication/withdrawal, delirium or recent medication changes) in order to provide a fair and accurate picture of the person's abilities. Individuals with severe pain, pronounced amotivation, or problems focusing and sustaining their attention are not appropriate for referral. Assessments for medicolegal purposes are usually directed to neuropsychologists in private practice rather than those in hospital settings. With the exception of brief cognitive screening, assessments are not conducted when the person is actively abusing alcohol or other substances, or in the acute recovery stage following an injury, stroke, or surgery.

Neuropsychological assessments can play a valuable role in detecting dementia, but they are not necessary for all persons for whom this diagnosis is known or suspected. Physicians and specialists can render accurate diagnoses with procedures that are less demanding and time-consuming for the patient. Neuropsychological assessments tend to be most useful in patients with a strong educational background or above-average intelligence, persons with subtle deficits that may represent the early stage of a dementia, individuals with suspected cognitive impairment who have atypical presentations, and persons where traditional screening methods may be biased due to language barriers, culture or sensory/motor limitations. With regard to the latter, these biases may also affect neuropsychological testing but some tests may be less affected by these confounds, thereby allowing a more accurate picture of the individual to emerge.

A key issue when making a referral to a neuropsychological service is to have a clear and specific question to be addressed by the assessment (Table 1). It is also important to include sufficient information about a patient in the referral for the neuropsychologist to initiate the assessment (Table 2).

Since most people who are referred for neuropsychological assessment have never completed testing of this nature, it may be useful to provide prospective

Table 2

Information to Include When Referring to a Neuropsychologist

- Demographic data (*e.g.*, age, gender, education level, and living situation)
- Information about an individual's language preference, and whether an interpreter might be required
- Problems with uncorrected vision or hearing, motor limitations, or pain/fatigue that might pose a challenge to the assessment process
- Relevant developmental, medical, and psychiatric history, including knowledge of any conditions or medications that might affect cognition, mood, or behaviour
- Documentation of recent relevant concerns
- A specific referral question

patients with background information about the nature and purpose of the assessment. The neuropsychologist to whom a referral is made may have a letter or pamphlet designed for clients that describes their service.

Conclusions

Neuropsychological assessments can provide useful diagnostic and treatment information about an older adult that may not be easily uncovered using other clinical or laboratory investigations. It provides a systematic, evidence-based and comprehensive approach to assessing an individual's cognitive and emotional functioning, and can complement the results obtained from other investigations such as computerised tomography (CT) and magnetic resonance imaging (MRI). Neuropsychological assessment is typically viewed as valuable by both consumers and referring agents,[11-13] but it is not appropriate for all older adults with known or suspected cognitive impairment. Persons with subtle cognitive changes that may be difficult to detect with traditional screening tests and persons with atypical presentations are often best suited for this type of evaluation. The decision to refer to a neuropsychological service should take into consideration several factors, not the least of which are the patient's current state and his/her ability to tolerate testing. Formulating specific referral questions and providing some basic information about the assessment process to patients are two steps that healthcare providers can take to garner the most benefit from a neuropsychological assessment.

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Delusions in Alzheimer's Disease: A Literature Review

Delusions are common symptoms in AD. While much of the literature reveals inconsistencies, certain trends have emerged. Delusions occur in approximately one third of AD patients. They appear to be more common among older patients while the impact of other demographic variables is less clear. Certain types of delusions have been identified as being more common among AD patients. While some interesting associations exist, no compelling risk factors have been identified.

by Corrine Fischer, MD, FRCPC

Delusions are common symptoms in psychiatric practice and are often seen in illnesses such as schizophrenia, bipolar disorder and delirium. In Alzheimer's disease (AD), the literature suggests that these symptoms are not only common, but also associated with a number of adverse outcomes, including increased caregiver stress[1] and earlier institutionalization.[2-5] Despite the prevalence and severity of these symptoms in AD, the literature reveals many inconsistencies suggesting they are poorly understood. Some of the reasons may be that previous studies did not distinguish AD patients from patients with other forms of dementia and failed to separate delusions from hallucinations. This paper will review the existing literature on delusions in AD with respect to epidemiology, classification systems, etiology, neurocognitive findings and behaviour to familiarize the reader with what is known about these common symptoms.

Dr. Fischer is a staff geriatric psychiatrist at St. Michael's Hospital and a lecturer with the division of Geriatric Psychiatry, University of Toronto, Toronto, Ontario.

Epidemiology of Delusions in AD

Recent studies show delusions are present in approximately one third of patients with AD.[6-9] Previous studies examining the prevalence of delusions in dementia have quoted a prevalence range[10] from 10% to 73%, but most of these studies failed to separate delusions from hallucinations and were done in patients with different dementia diagnoses. It has been established that delusions occur more commonly in certain types of dementia, such as Lewy-body disease, and less commonly in other types, such as frontotemporal dementia,[11] although the mechanism of this is unclear. Paulsen and Salmon *et al*[12] showed the prevalence of delusions to increase 20% by year one and 50% by year four from the time of diagnosis, suggesting these symptoms may have to be evaluated over time.

The role of demographic variables including age, gender, education and race is not clear.

Most studies have shown AD patients with delusions and other psychotic symptoms are older than those without these symptoms,[6,13-15] but some have shown a lower prevalence[16] while others have shown no changes.[9,17] The role of gender is equally unclear, with some studies showing a higher prevalence of delusions and psychotic features among men with AD,[2,18-19] others showing a higher prevalence among women,[16,20] and still others showing an equal distribution among the sexes[9,17,21-23]. In terms of education, some studies have suggested an association between lower levels of education and the presence of delusions and psychotic features in AD,[24] while others have shown an association with higher levels of education[22-23] and still others have shown no difference.[9] The lack of consensus as to the role of demographic variables in the development of delusions in AD may reflect the poor differentiation between delusions and hallucinations, each of which may have separate associations.

Classification of Delusions in AD

Delusions are commonly referred to as "fixed false beliefs that are not culturally bound." Delusions exist in many psychiatric disorders, including bipolar disorder, schizophrenia and delirium. Classification systems exist for delusions in illnesses such as schizophrenia, where delusions are subtyped into persecutory, erotomanic, grandiose, etc. In AD patients, the challenge is to differentiate these symptoms from other memory-based symptoms such as confabulation or disorientation.

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There have been several attempts at developing classification systems for delusions in AD patients. One system[9] divided delusions into five different categories: paranoid, hypochondrical, Capgras syndrome, house misidentification and grandiosity. Capgras syndrome refers to the belief that someone close to the person is an imposter, and house misidentification refers to the belief that one's house is not one's home. Gormley *et al*[7] identified four common delusions in AD patients: delusions of theft (belief someone has stolen something), phantom boarders (belief an intruder is in the house), spousal infidelity (fear their spouse is having an affair) and delusions of abandonment (fear their caregiver is going to leave). Cummings[25] examined delusions in 20 patients with organic brain syndrome and found that four subtypes were most common, including simple persecutory delusions, complex persecutory delusions, grandiose delusions and delusions associated with a specific neurological deficit. He determined that simple persecutory delusions were most common in AD and that the other subtypes tended to occur more often in other types of organic brain syndromes.

While delusions of theft may be most common in patients with AD,[7] it has been suggested that multiple delusions may co-exist at one time.[9] Finally, there is some evidence that different types of delusions may emerge at different stages of the disease, with delusions of theft being most common and presenting early in AD and Capgras syndrome[26] being rarer and occurring late in the course of the disease.

Risk Factors for Delusions in AD

There is little consensus as to known risk factors for delusions in

AD. In terms of psychiatric symptoms, adverse life events[27] and premorbid personality[28] do appear to play a minor role, but the impact of depression is less clear. Other studies have looked at other variables and have found no relationship with apolipoprotein E (ApoE),[29] conflicting information around the role of extrapyramidal symptoms,[6,8,20,30,31] a possible link with antihypertensive medications[6] and a possible link with sensory changes.[32] In summary, no discrete risk factor has been identified to play a major role in delusion formation.

Etiology of Delusions in AD

There are three major theories regarding the etiology of delusions in AD. The theory of hypofrontality states that delusional symptoms are related to selective frontal lobe dysfunction. This theory has been substantiated by both neuroimaging and neuropathologic studies with Single-Photon Emission Computed Tomography (SPECT) scans showing diminished frontal lobe perfusion[22,33] and neuropathologic studies showing a higher density of senile plaques in the frontal lobes.[12] An alternative theory proposed by Flynn *et al* in 1991 is that delusions arise as an independent non-cognitive manifestation of AD, a theory substantiated by Sweet[34] who was able to demonstrate no difference in the density of neuritic plaques and tangles when comparing the brains of delusional and non-delusional AD patients. Finally, it has been proposed that delusions in AD patients may arise as a manifestation of the pathophysiology attributed to AD,[10,35] a theory substantiated by Farber[36] who examined 100 cases of AD post-mortem and found that patients with psychosis had twice the density of neurofibrillary tangles.

While delusions of theft may be most common in patients with AD, it has been suggested that multiple delusions may co-exist at one time. Finally, there is some evidence that different types of delusions may emerge at different stages of the disease.

Other investigators have found that delusions may be secondary to neuroanatomical changes such as isolated degeneration of the right frontal lobe[37] and temporal lobe asymmetry,[38] neurotransmitter changes—including a reduction in serotonin in the prosubiculum[39] and upregulation of postsynaptic muscarinic receptors,[40] genetic factors[41] and an altered sense of familiarity[42].

Neurocognitive and Behavioural Changes in AD Patients with Delusions

Several investigators have examined the relationship of cognitive function to the development of delusions in AD. The evidence has been conflicting, with some studies showing that AD patients with psychosis have an increased rate of cognitive decline,[12,16] some showing only mild differences[34] and others showing the preservation of intellectual function.[43-45] It has been speculated that a certain degree of cognitive function may be required to develop a delusion[46], although what aspect of cognitive functioning needs to be preserved is less clear. The fact that delusions tend to occur in the mid-range of cognitive impairment (Mini-Mental State Examination 17-23)[47] suggests that patients who are moderately affected may be more vulnerable to developing delusions.

More consistent findings have been documented in the area of frontal lobe dysfunction, supporting the theory of hypofrontality. AD patients with delusions have been shown to have more signs of frontal lobe dysfunction on neurocognitive testing,[12] although the relationship of this to poor insight has been questioned.[29]

There is substantial evidence that Alzheimer patients with delusions are more aggressive[7,14,15,47-50] with rates of verbal

aggression[51,52] outmatching rates of physical aggression.[53,54] Eustace *et al*[55] hypothesized that verbal aggression among delusional AD patients is likely linked to a perceived environmental threat. He also determined that delusions, unlike other behaviours such as wandering, may have only moderate persistence. Delusions among AD patients have been linked to other behaviours such as wandering[20] and adverse outcomes such as early institutionalization.[5]

Conclusion

Delusions are common symptoms in AD. While much of the literature reveals inconsistencies, certain trends have emerged. Delusions occur in approximately one third of AD patients. They appear to be more common among older patients while the impact of other demographic variables is less clear. Certain types of delusions have been identified as being more common among AD patients. While some interesting associations exist, no compelling risk factors have been identified. There is evidence neuropathologically, radiologically and neurocognitively that delusions may be linked to frontal lobe dysfunction. Patients appear to be most vulnerable to developing delusions when they are moderately ill. Finally, delusions are associated with a number of adverse outcomes, including increased caregiver burden, aggressiveness and earlier institutionalization. Future research needs to focus on clarifying areas of inconsistency, so as to create a better understanding of these complex and interesting symptoms.

The fact that delusions tend to occur in the mid-range of cognitive impairment suggests that patients who are moderately affected may be more vulnerable to developing delusions.

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The Emerging Spectrum of Parkinsonian Dementias

This is the first in a two-part article examining the emerging spectrum of parkinsonian dementias. The first part looks at pathology and neurochemistry, the clinical features and differences between dementia with Lewy bodies (DLB), the dementia of Parkinson's disease (PDD) and the advanced stages of Alzheimer's disease (AD). The second part of the article will appear in the next issue of the *Canadian Alzheimer Disease Review* and will examine the treatment of DLB, PDD and AD.

by Inge Loy-English, MD, FRCPC and Howard Feldman MD, FRCPC

There is increasing recognition of the group of dementias that are manifest with parkinsonism, behavioral symptoms and cognitive impairment. The clinical spectrum of such disorders includes: dementia with Lewy bodies (DLB), the dementia of Parkinson's disease (PDD) and the advanced stages of Alzheimer's disease (AD). Unifying this spectrum is an underlying mix of neuropathological lesions, including Lewy bodies, α-synuclein deposition and senile neuritic plaques.

Inge Loy-English, MD, FRCPC, Assistant Professor, Division of Neurology, Department of Medicine, University of Ottawa.

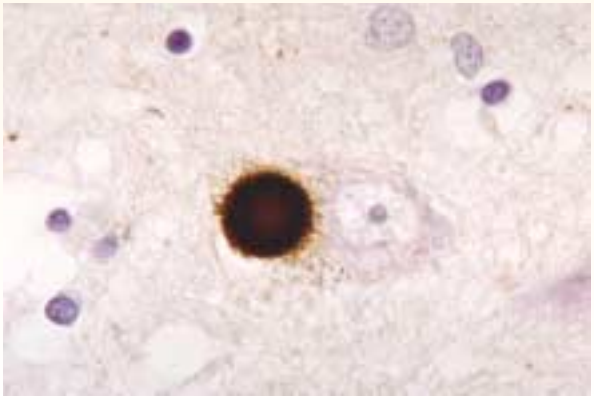
Howard Feldman, MD, FRCPC Professor of Medicine, Division of Neurology, University of British Columbia (UBC) Director, UBC Alzheimer Clinical Trials Unit Vancouver, British Columbia

DLB is an increasingly recognized form of dementia. In a recent study of individuals referred to dementia centres in Canada, the ACCORD study, DLB accounted for 1.9% of the primary diagnoses of dementia while 2.7% of dementia diagnoses were mixed AD and DLB.[1] There is a suggestion from the neuropathologic literature that DLB may be the second most common cause of dementia, accounting for up to 25% of cases at autopsy.[2] The average age of onset of DLB is 67 years, with males more affected than females (62% vs. 38%).[3] The average duration of illness is nine years,[3] though estimates of speed of progression vary considerably from study to study.[4] At the present time the neuropathological diagnosis is made more frequently than the clinical diagnosis. Further work is required to enhance the clinical diagnosis of this condition.

PDD is also becoming more recognized. Estimates of the prevalence of dementia in patients with Parkinson's disease (PD) vary widely, but usually range from 20% to 45% depending on the definition of dementia used.[5] In the ACCORD study,[1] PDD accounted for 0.6% of patients referred for dementia, but this estimate of its frequency was subject to a significant selection bias, as the ACCORD study was conducted in dementia research centres and not PD units. In one longitudinal study of the incidence of dementia in a community-based sample of patients with PD, the incidence of dementia was 95.3 per 1,000 patient years.[6] When the risk of dementia in PD patients was compared to that of control subjects, the risk of devel-

Figure 1

The Lewy Body: Spherical, Eosinophilic Intracellular Inclusions Located Within Neurons



oping dementia was 5.9 times higher in the PD group (OR 5.9, 95% CI, 3.9-9.1). Risk factors that predict the development of dementia in PD include age, age of onset of PD, depression and more severe motoric parkinsonian symptoms.[6,7,8]

In AD, extrapyramidal features typically occur in more advanced disease stages. In the Canadian study of Health and Aging, a population-based study of persons over 65 years of age, 9% of patients identified with AD had parkinsonism.[9] The reported rate of parkinsonism in AD ranges from 12% to 92% depending on the study.[10] Explanations for this wide variability in frequency have been linked to the definition of extrapyramidal features studied, inclusion of patients on neuroleptics, inclusion of patients with DLB in earlier studies, and the specific population of AD patients studied.[10,11]

Extrapyramidal symptoms (EPS) become more common with disease progression,[10] and are associated with increased mortality. This increased risk of mortality is independent of severity of cognitive impairment, age and residential status.[9] EPS occurring in AD predicts a worse outcome.

Pathology and Neurochemistry

Pathological findings. The Lewy body (LB) is the pathologic hallmark of DLB. LBs are spherical, eosinophilic intracellular inclusions located within neurons (Figure 1). They are composed of neurofilaments, crystallin, ubiquitin, and α-synuclein, a protein that aggregates and which is important in a number of neurodegenerative diseases[3,12] (Table 1). These diseases are all characterized by inclusions containing α-synuclein. With the exception of multi-system atrophy and its subtypes, all of these disorders also have LBs as part of their neuropathology.

Initially described in brainstem nuclei as one of the pathologic markers of PD,[2] LBs occur throughout the brain in patients with DLB. They have a predilection for certain areas of the brain: brainstem, subcortical nuclei, limbic cortex (especially cingulate, entorhinal, and amygdala) and neocortex (temporal > frontal = parietal).[13] LBs in DLB are more easily seen and better defined in the brainstem than the cortex, where they can be more easily missed if special staining methods are not used. They are best seen using antibodies to α-synuclein.[12]

AD pathology usually coexists with the typical pathology of DLB, though there rarely will be evidence of DLB pathology without evidence of ß-amyloid plaques and neurofibrillary tangles. The burden of the AD pathology in DLB patients is less than was found in a cohort of "pure" AD patients with similar or worse disability prior to death.[14]

In PD, LBs are classically found in the ventrolateral portion of the substantia nigra, and are associated with cell loss. These changes result in the loss of nigrostriatal dopaminergic projection neurons, and they are thought to be responsible for the extrapyramidal movement disorder that is seen.[5] To a lesser extent, the LBs can be

Table 1

The Synucleinopathies

- Lewy Body Diseases
  - Idiopathic Parkinson's disease
  - Dementia with Lewy Bodies
  - Incidental Lewy Body "disease"*
  - Rare sporadic syndromes associated with Lewy bodies
    - Pure Autonomic Failure
    - Lewy Body Dysphagia
  - Inherited Lewy body diseases
    - Mutations of the α-synuclein gene, PARK3 and PARK4
- Multiple System Atrophy
  - Olivopontocerebellar Atrophy
  - Striatonigral Degeneration
  - Shy-Drager Syndrome

* Incidental Lewy bodies found on brain autopsy but not associated with clinical disease.

found in other brainstem nuclei, and throughout the cerebral cortex. As in DLB, co-existent AD pathology is often seen in PDD, with ß-amyloid plaques, and neurofibrillary tangles in excess of that expected in age-matched normal controls, at times in sufficient quantity to meet criteria for AD.[15]

The pathology of the parkinsonian changes in AD is less clear. In 20% to 85% of autopsy-confirmed cases of AD with associated clinical parkinsonism, nigral degeneration with LBs is found.[10] This suggests a co-existent diagnosis of PD in at least some patients. Other patterns seen at autopsy include neurofibrillary tangles in the substantia nigra and cell loss without any pathological inclusions. Other patients who have parkinsonism during life do not have any demonstrable nigral pathology. Pathologic mechanisms suggested for this group include pathology in other dopaminergic pathways (*e.g.,* mesocortical), and increased ß-amyloid plaque in the striatum.[10]

Neurochemical abnormalities. There is significant neurotransmitter disruption in DLB. Neuropathologic studies have shown that there is a decrease in choline acetyltransferase (ChAT, the rate-limiting enzyme for the synthesis of acetylcholine) in DLB. This cholinergic marker deficit is even greater in DLB than in AD.[16,17] The loss of ChAT activity is paralleled by a loss of neurons from the nucleus basalis of Meynert, the primary source of cholinergic input to the cortex.[17] Low mid-frontal neocortical ChAT levels correlate significantly with low scores on the mini mental status exam (MMSE).[16]

There is disruption of the dopaminergic system in DLB. The mesolimbic, mesocortical, and striatonigral pathways show evidence of degeneration, largely due to involvement of the ventral tegmental area and the substantia nigra.[3] The disruption of the nigrostriatal pathway is responsible for the parkinsonian features seen in DLB.

The neurochemical abnormalities found in PDD are very similar to those found in DLB. There is disruption of cholinergic input to the cortex due to neuronal loss and LB formation in the nucleus basalis of Meynert. This loss is usually > 70% in patients with PDD and is significantly greater than in patients with PD without dementia.[5] As in DLB, the loss of cholinergic activity is purported to account for much of the cognitive decline seen in PDD. The loss of the nigrostriatal tract is responsible for the extrapyramidal movement disorder, but also may play a role in some cognitive dysfunction, through its connections with fronto-caudate feedback loops.[5]

The cholinergic hypothesis of AD states that the cognitive deficit seen is primarily due to the loss of cholinergic input to the

Table 2

Consensus Criteria for the Clinical Diagnosis of Probable and Possible DLB

1) Progressive cognitive decline, of sufficient magnitude to interfere with normal social or occupational function.

- may or may not have prominent memory dysfunction at onset but usually evident with progression
- may have prominent deficits on tests of attention, frontal-subcortical skills, and visuospatial ability

2) For Probable DLB 2 of (for Possible DLB 1 of):

- fluctuating cognition with pronounced variations in attention and alertness
- recurrent visual hallucinations that are typically well formed and detailed
- spontaneous motor features of parkinsonism

3) Features supportive of the diagnosis are:

- repeated falls
- syncope
- transient loss of consciousness
- neuroleptic sensitivity
- systematized delusions
- hallucinations in other modalities

4) Diagnosis of DLB unlikely in the presence of:

- stroke, as per focal neurologic signs or appropriate imaging
- evidence on physical examination and investigation of any physical illness or other brain disorder sufficient to account for the clinical picture.

Adapted from McKeith IJ, Galasko D, Kosaka, et al. Consensus guidelines for the clinical and pathological diagnosis of dementia with Lewy bodies (DLB): Report of the consortium on DLB international workshop. Neurology 1996: 47; 1113-24.

cortex, due to the degeneration of neurons in the nucleus basalis of Meynert. The neurochemical abnormality leading to the parkinsonism seen is felt to be either due to disruption of the dopaminergic nigrostriatal paths, mesocortical paths, or more "downstream" pathology leading to dopaminergic disruption in the striatum itself.[10]

Clinical Features

Dementia with Lewy bodies. The core features of DLB form a triad of cognitive impairment, neuropsychiatric symptoms (especially visual hallucinations), and parkinsonism. The current diagnostic criteria are listed in Table 2.[13]

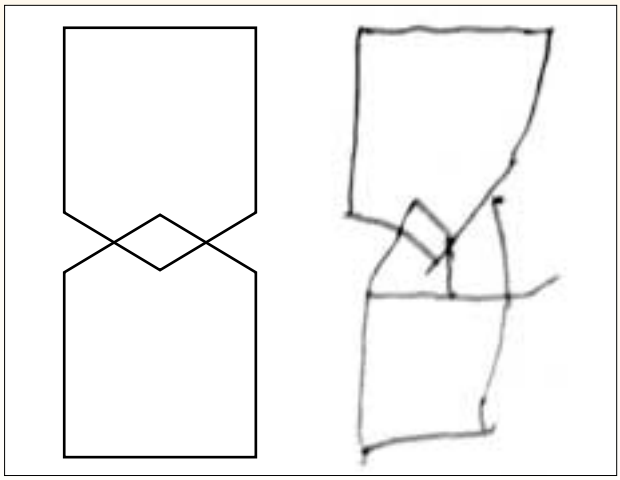
Cognitive impairment is often the presenting feature of DLB.[4] On testing, the pattern of dementia is a mixed cortical-subcortical dementia. Patients have prominent frontal-subcortical dysfunction with difficulty in attention, as well as executive function such as planning, sequencing and organization. A slowness of thought (bradyphrenia) is not uncommon.

In addition to the frontal-subcortical dysfunction, patients also exhibit marked visuospatial difficulties (Figure 2). A recent study compared AD patients to DLB patients on tests of visuospatial impairment.[18] Patients were matched for age, sex and severity of cognitive decline. This study showed that the DLB patients had more difficulty not only with complex visual tasks, but also with tasks designed to look at elementary visual perception.

Ballard et al investigated distinguishing DLB from AD and vascular dementia (VaD) with the use of simple bedside tests.[19] Using this battery of tests, DLB patients were significantly more

Figure 2

Visuospatial Dysfunction in DLB



Visuospatial dysfuntion can be exemplified when a patient is asked to recreate a shape such as the one shown on the left above (resulting in a skewed version such as the one shown on the right).

impaired than AD and VaD patients on tests of visuospatial praxis. On tests of recent memory, DLB patients were less impaired than their AD and VaD counterparts.

Decreased tracer uptake in the occipital lobe on single photon emission-computed tomography (SPECT) and positron emission tomography (PET) scanning is associated with DLB, though the pathological correlate of this has yet to be elucidated.[18,20] It is felt that this functional occipital lobe impairment is related to the visuo-perceptual difficulties seen in DLB.[18]

Attention in DLB is frequently impaired, and is often a component of the fluctuations in cognition that is one of the hallmarks of this disease. Fluctuations can be over minutes, days or even weeks to months, and can include variation in attention and alertness, as well as variation in performance on cognitive testing.[13] Fluctuations occur in 58% of patients at disease onset, and in 75% at some point during the course of the disease.[21] The fluctuations usually do not follow a clear diurnal pattern.[13] In a recent study evaluating tests of attention and reaction time, patients with DLB not only did worse than AD patients, they also exhibited a considerable variability in their reaction time consistent with second-to-second and minute-to-minute fluctuation.[22]

Neuropsychiatric symptoms are also very common, with visual hallucinations being the most frequent manifestation. These hallucinations are characteristically well formed, detailed and recurrent, often taking the form of small animals or people intruding in the person's home.[13] One study found that hallucinations are present in 33% of patients at onset, and in 46% of patients at some time over the course of their disease,[21] though rates as high as 80% have been reported.[4] Frank hallucinations often co-exist with perceptual difficulties such as misidentifications and visual agnosias. The degree of insight retained into the hallucinations is variable. Patients may have hallucinations in other modalities, most commonly auditory (19% of patients at presentation).[21] These tend to be well formed as well; for example, hearing the doorbell ring. Delusions can also happen, though they are less common. They tend to be bizarre and relate to recall of previous hallucinations and other perceptual disturbances.[13]

Parkinsonian symptoms. Parkinsonism (rest tremor, bradykinesia, ridgidity, postural instability) is a core clinical feature of DLB. Approximately 26% of patients present with parkinsonism alone and a further 19% present with parkinsonian features in combination with other symptoms, such as dementia.[3] By the end stages of the disease, only 4% to 25% of patients are free from parkinsonian motor symptoms.[4,3]

Dementia of Parkinson's Disease

Cognitive impairment. The pattern of cognitive impairment seen in PDD is similar to that seen in DLB. Patients have difficulties with attention, as evidenced by poor performance on tests of vigilance and cognitive reaction time.[23] There is also some clinical evidence for fluctuations of attention.[23]

Memory dysfunction is also similar to that found in DLB and different from that found in AD. It is generally less severe than in AD and, while patients may perform poorly on tests of free recall, they respond well to cues. Dysexecutive features are also prominent, with patients showing difficulties with problem-solving, set—shifting and maintenance. Patients also have difficulties with visuospatial tasks, especially "higher level" tasks such as visuospatial analysis and orientation judgement.[23] Language and praxis are relatively spared.

Neuropsychiatric symptoms are relatively common in PDD. Psychosis, including hallucinations and illusions, is found in 40% to 70% of patients with PDD.[23,24] These symptoms can be difficult to differentiate from drug-induced psychosis, as the majority of these patients are taking at least one dopaminergic agent. Depression is also common. While neuropsychiatric features of hallucinations, delusions and depression can be present in patients with PD both with and without dementia, they are more common in patients who have dementia.[25,26]

Alzheimer's Disease with Parkinsonism

Characteristics of the extrapyramidal disorder. The most common extrapyramidal features found in AD are bradykinesia and rigidity.[10] These features are usually bilateral in contrast to idiopathic PD, where an asymmetric and unilateral onset is most typical. It is important to differentiate this rigidity from paratonia, which is extremely common in any type of dementia. Gait disturbance is the next most common extrapyramidal feature, including shuffling gait and impaired turning. Resting tremor in AD is rare.[10] While extrapyramidal features can be found subtly in some patients in early AD, they are much more common as the disease progresses.[11]

Differentiation Between DLB, PDD and AD with Parkinsonism

The clinical differentiation between PDD, DLB and AD can be difficult, especially if a patient is presenting for the first time with a combination of dementia and parkinsonism. On a quite arbitrary basis, DLB is diagnosed if the dementia precedes the parkinsonian symptoms, or in the parkinsonism develops no more than one year prior to the cognitive impairment. If the parkinsonian symptoms have been present for more than 12 months prior to the onset of dementia, PDD is the preferred diagnosis. However, in most cases of PD with dementia, the motoric parkinsonian symptoms are typically present for many years prior to the onset of noticeable cognitive decline.[27] Other differences that would make one favor a diagnosis of DLB over PD include the decreased prevalence of resting tremor (55% vs. 85%),[3] the decreased responsiveness of motor symptoms to L-dopa, the decreased tolerability of L-dopa secondary to psychiatric side effects, and the occurrence of spontaneous (as opposed to drug-induced) visual hallucinations. It is felt that, given the similarity of pathology and clinical symp-

Approximately 26% of DLB patients present with parkinsonism alone and a further 19% present with parkinsonian features in combination with other symptoms, such as dementia. By the end stages of the disease, only 4% to 25% of patients are free from parkinsonian motor symptoms.

toms, PD and DLB likely lie upon a continuum, with clinical manifestations dependent on the extent and location of LB disease.[12]

The differentiation of DLB and PDD from AD is easier. Parkinsonism is usually a late feature in AD, preceded by cognitive decline for many years. The pattern of dementia is different, with prominent episodic memory loss early in the course of the disease. Visual hallucinations are relatively rare, and when they occur, they tend to be later in the course of the disease.

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PERSPECTIVES

Personal Revelations, Experiences and Reflections of an AD Caregiver

Roberta Bedard is a caregiver for her husband who has Alzheimer's disease (AD). She has written many humorous and touching vignettes about her personal experiences in dealing with the development of the disease, and has graciously agreed to feature these vignettes as a series in the *Canadian Alzheimer Disease Review*. Roberta's writings enable readers to share in her journey with AD caregiving, provide valuable insight on the human aspect of disease and stimulate contemplation on the deeper meanings of life and love.

In this feature...

In "You and Me and Spook," Roberta explains how her husband's psychologic and functional status is being affected by AD and how she creatively manages the situations which present to her. We have also included "Laughing"—an uplifting account of how important it is to cherish life and our loved ones, one day at a time.

CHAPTER 4

by Roberta Bedard

You and Me and Spook

For a while now, we have been dealing with "sundowning" which, combined with Ray's REM sleep-wake cycle disorder, leads to some pretty exciting night-time adventures. For instance: Ray imagines that there are enemies hiding in the walk-in closet, ready to attack. So he removes the books from the bookshelf, barricades the closet door with it and piles the books around the floor to trip the bad guys. Or he becomes extremely concerned over whether the puppies have been bathed (we don't have puppies). Only when I reassure him that the puppies are all fluffed up and asleep in their kennel does he go back to sleep. Another time, it is the "circus folk" who have surrounded the house, installing large searchlights. Then they break one of the lights, which necessitates going out to check for broken glass so I won't cut my feet if I should go outside.

But I'm used to this and, other than not getting enough sleep, it is not upsetting, except for the fact that winter is coming, and we need to find a way to get Ray to sleep through the night so he won't go outside in -40°C weather. Our psychogeriatrician has found a medication that seems to work and doesn't over-sedate Ray. Night-times are better now.

Spook is something new. Ray tells me he senses a third person in the house. This explains why he keeps setting the table for three—now I understand. I also understand why he asks me now and again whether we are alone in the house. He is convinced that he will find this person but every time he thinks he has found him or her, it turns out to be me. I reassure him that there is no one here but us, but he is not entirely convinced.

Then I remember. Children have imaginary friends. How would I deal with a child who has an imaginary friend? I would accept the friend and go along with it. So it is with Spook. We have given it a name and a gender (male), and include him in our conversations. Spook is our buddy. We invite him to join us at lunch, but he never shows up. We don't mind; more for us. And we laugh at Spook for missing a meal. Funnily enough, since we started doing that, Ray is no longer concerned with finding Spook and hardly even mentions him. I think that when I denied Ray's reality (of having a third person around somewhere) it made him uncomfortable. How would I feel if the person I loved

most consistently denied what I felt to be true?

According to the Functional Assessment Staging (FAST) scale, Ray is functioning at around stage 5, which corresponds to a child between the ages of 5 and 7 years. Just knowing that makes it so easy to deal with the changes which are beginning to appear. It helps me to understand when he needs help putting on or taking off a sweater. He has trouble getting sweaters over his head, so I'll have to buy a cardigan or two, with big buttons, anticipating the time when small buttons are difficult.

What is interesting is that these changes are sporadic. In between episodes, Ray is himself, he continues to understand concepts and his insights are well worth considering. He sees facets to situations and people that I miss. He continues to be the loving, gentle caregiver, and his wacky sense of humour is still operating. He takes over the crossword puzzle when I quit, and finds esoteric terms that I have never even heard. He is going to an Alzheimer Day Program twice a week now and wows everybody with his personality.

He has told me that, though he may act bashful, he likes it when I kiss him goodbye in front of everybody. I only know this because I asked him if he minded. It is important for me not to make assumptions, but to ask him how he feels and to use all my senses to understand fully his answers.

None of this is hard to do.

Laughing

I've just finished laughing. Big belly laughs that left me gasping. Something that doesn't happen very often. Though I am, in general, satisfied with my life at this time, I have to admit that I have periods of weeping, in the midst of mainly benign periods of quiet enjoyment. But I don't really laugh very often.

Let me explain. My husband and I watch television in the back bedroom, snuggled under the quilt. He more snuggled than I, since I like the room cool-to-cold and he likes it much warmer (he's bald and his head gets cold). My philosophy is if you're cold, put on a sweater—I'll even help. Today he turned up to watch one of our favourite programs in a getup that brought back memories of the sort of thing my children used to do. He was wearing his warm dressing gown over his clothes, ski socks on his feet and, to top it all off, his postman's hat. You know the ones. They have furry earflaps. He looked so pleased with himself. He was making a statement.

I took one look at him and choked. And laughed. And ran out of breath. Tears of laughter came. My sides ached. I gasped and choked, finding myself helplessly banging my heels on the bed. I kept trying to sneak looks at him without laughing and I couldn't pull it off. I would slide a sidelong look at him and be off into gales of uncontrolled laughter.

Ray laughed with me. "I'm a practical Albertan," he said. We had hysterics together.

Then I realized. There are some things about AD that I actually like. It is disinhibiting. When we think of AD patients losing inhibitions, we (or at least I) have tended to think of it negatively. We believe the result is socially unacceptable behaviour, of lashing out.

But if the AD patient is treated with love, with acceptance, with an honesty that recognizes that accomodations can be made with objective truth, then the greater truth of the AD patient's acceptability as a human being is maintained. My husband's self-esteem is intact. We both accept that he has AD, we both talk about it. We both laugh at Spook. We laugh when he forgets that dinner is formal—that is, he has to wear pants. There are times that he tells me that AD is hell, that he forgets what his hand was aiming for when he stretched it out. But these moments are rare. On the whole, he knows that he is still perfect in my eyes.

So he is liberated from inhibitions. This allows his truly wacky sense of humour to flourish. The social unacceptability of wearing a postman's hat to watch TV is pretty benign. When it is done as a humourous statement, it is unalloyed joy. Before Ray developed AD, I had no idea that he could be so funny! Which reinforces for me the benefit of understanding and incorporating the Reisberg Theory into my daily life as a caregiver. We delight in our small children. We appreciate their humour. We look with fondness on their eccentricities. There is no reason to take a different approach with a loved one who has AD.

I am not a fool. I know that, as AD progresses, things will get more difficult. I know that, unless Ray dies of something else first, AD will kill him. We both agree that our goal is to keep him happy and functioning as long as possible, with the sad hope that something else will kill him first. We have said our goodbyes more than once.

Having said that, there is no reason to live under a cloud every day. For now, I take the joy and fun that each day offers. September 11th, 2001 taught a whole continent that our daily lives with our loved ones are what count most when the chips are down.

AD has taught me the same lesson. And I am glad to learn it. Were there an easier way to become fully aware of this, I would take it. But the lesson is still a valuable one and a great gift.

And I can still feel the ache in my ribs from laughing.

Please look for *Chapter 5: New Stage* in the next issue of the *Canadian Alzheimer Disease Review*.

Alzheimer Society
CANADA
News from the Alzheimer Society of Canada

Online Support for People Living with Alzheimer Disease

Canadian Trends for Online Health Information

The Internet is now the second most common source of health information for Canadians, ranking ahead of radio, television and newspapers. It is surpassed only by face-to-face contact with a health professional.

Findings of a 2001 Canadian Medical Association survey revealed that almost half of Canadian physicians "at least occasionally" referred patients to a medical website. Of these physicians, seven out of 10 recommended disease-specific websites.

The Alzheimer Society of Canada's website (*www.alzheimer.ca)* first came online in 1997. This site provides bilingual information on care, services, research and treatment of Alzheimer Disease (AD) in Canada. On average, visitors view 7,000 pages each day.

Who Visits the Website?

A recent survey of visitors to the Alzheimer Society website revealed that 84% of people searching for information knew someone with AD—a spouse, parent, relative or friend. It also revealed that 3% of visitors were people with AD.

Although most people prefer to use the website for research and information-gathering purposes, many use the Alzheimer Society's Care Exchange and Caregiver Forum, and Ask the Expert services.

Online Support

The Forum. The Care Exchange and Caregiver Forum is a discussion board for people to share experiences and exchange information with others who understand. For some it has become a virtual support group connecting people dealing with similar issues.

"Unless you are faced with this disease, you can't begin to understand how it feels to be dealing with a person with AD... It helps me to read this message board and see that others are facing the same problems. It helps me to carry on..."

—Louise, 11:33 AM

It is also a place where those in the early stage of AD can share their experiences of living with the disease.

"I'm now 54 and the changes are not so 'little' anymore... Yup, my brain is dying. Literally. And I get to watch it happen, day by day, week by week... To all you wonderful, so often unrecognized caregivers—I truly can't imagine the sheer hell it must so often be for you...and while those with this disease who carry on 'right to the bitter end' [and lose] awareness of 'dying,' the caretaker's hell continues right on. You are truly the silent victims."

—Marilyne, 2:16 PM

And because the Internet is available 24 hours a day, seven days a week, people in crisis can reach out when they need to.

"It seems that when I am in crisis, this is the place I turn to, automatically. It's almost 4 in the morning and I've not [been] to bed yet... It's going

to be 4 years since I stopped working to care for my mother. There's nobody else to do it. No family living in Canada. I am it. 7 days/24 [hours]… Thanks for your ears and shoulders. I just needed to get it out of my chest for a little while."

—Elisabeth, 4:12 AM

Forum visitors are not required to actively participate. Many benefit simply from reading the messages, knowing they are not alone. The Care Exchange and Caregiver Forum is the most visited part of the website.

Ask the Expert (expert@alzheimer.ca). The Ask the Expert service is intended to help people with questions who have not found the answers on the website. On average, 75 questions per month are submitted from across Canada and around the world. Users of Ask the Expert benefit from:

- a 24-hour response time
- answers tailored to their situation
- direction to a specific area of our website for more information
- referrals to their healthcare professional or local Alzheimer Society for information and resources
- the opportunity for future dialogue

"People who use Ask the Expert differ from people who may attend a support group," explains Ilona Horgen, Director of Support Services and Education at the Alzheimer Society of Canada. "These people want information quickly and they want to pose their question privately, anonymously and at a time that is convenient to them. They are also looking for a personal response."

Ask the Expert is a confidential service of the Support Services and Education department of the Alzheimer Society of Canada. Staff are knowledgeable about programs and services provided by the Society across Canada, and have access to healthcare professionals who provide information on a variety of topics related to AD.

The most frequently asked questions are from caregivers seeking support services close to where they live and disease-management information. Visitors to the website are reminded that the information presented is not a substitute for medical advice and are advised to see their doctor or other qualified healthcare provider for personal assessment.

Health on the Net Code of Conduct (HONcode©). The Health on the Net Foundation, located in Geneva Switzerland, was formed in 1995 to establish a code of conduct for medical and health websites.

The HONcode consists of eight principles which aim to standardize the reliability of medical and health information available on the Internet. They address the authority of the information provided, data confidentiality and privacy, proper attribution of sources, transparency of financial sponsorship and importance of clearly separating advertising from editorial content.

Adopted by over 3000 websites worldwide, the HONcode seal of approval on subscribing sites helps users identify sources of reliable information. The Alzheimer Society of Canada website has been a HONcode member since 1998.

The Alzheimer Society of Canada is a not-for-profit health organization dedicated to helping people affected by Alzheimer Disease. The Society provides support and educational programs for people with Alzheimer Disease and their caregivers. The Society also funds research into finding the causes and cure of the disease, and into improved methods of caregiving. The Society consists of a national office, 10 provincial organizations and more than 140 local groups across the country.

For more information on Alzheimer Disease and related dementias, Alzheimer Society programs and services, and how you can help, contact your local Alzheimer Society or visit the Society's website at www.alzheimer.ca or call 1-800-616-8816.