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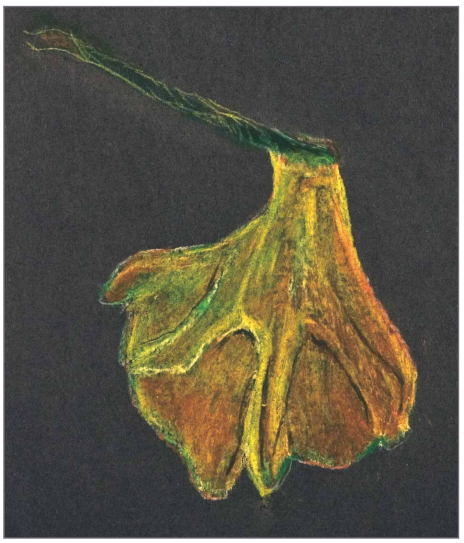
Alzheimer's Disease and other dementias



Art by Arne Pedersen

FOCUS ON VASCULAR DEMENTIA

The Canadian Review of
Alzheimer's Disease and other dementias

Photograph provided by Dalia Gottlieb-Tanaka, PhD
Chair of The Society for the Arts in Dementia Care

On the Cover…

The pumpkin on the cover was painted in 2007 by Arne Pedersen. Arne was a senior with Alzheimer's who lived and attended art sessions at Adanac Park Lodge in Vancouver. Arne pursued perfection and drew close to realism with elegantly balanced colours and composition. He worked with Keisei Anzai, a registered Art Therapist, who was born in Hong Kong but studied in the UK and the US. She is an acclaimed potter, who works with children, people with addiction, and seniors with dementia. In Keisei's words: Art is a quiet, unconscious way that acknowledges the self. It nourishes, brings comfort, improves self-esteem and creates meaningful moments of the "now". When Dr. Dalia Gottlieb-Tanaka attended an exhibit at the Lodge, she selected the artwork for further exhibitions. Arne painted this 9"x12" image on black construction paper using oil pastels. He passed away in Vancouver on May 2, 2009 at the age of 88.

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EDITORIAL

Towards a Better Understanding of Vascular Dementia

By Peter J. Lin, MD, CCFP

Large cortical strokes and vascular dementia have been well established and studied, but the subcortical variety is less well known. In this issue, Dr. McCracken reviews subcortical ischemic vascular dementia (SIVD). There are two basic pathological processes behind SIVD. The first is a generalized narrowing of the small blood vessels around the ventricles, which leads to hypoperfusion of the myelinated axons. This produces the classic periventricular white matter changes on MRI scans, which are hallmark for Binswanger's disease. The second pathological process is the occlusion of small arterioles leading to a lacunar infarct. Both of these processes are accelerated by hypertension and diabetes.

The symptoms of a lacunar infarct depends on its location. Like a meteor hitting a desert vs. hitting a major city, the outcome is quite different. On MRI scans, there are often lacunar infarcts or white-matter lesions that are asymptomatic because they have not hit a critical area. They may be silent, but they are proof that the disease process is active. Hence, aggressive treatment of vascular risk factors, such as blood pressure, diabetes, lipids and anti-platelets, is prudent even though it has not yet been formally proven to reduce the risk of dementia.

In his piece, Dr. Dalziel writes about how targeted screening is very cost effective. In other words, go fishing where there are fish. The Memory Impairment Risk Calculator uses age, number of vascular risk factors and family history of dementia to estimate the patient's risk. The Mini-Cog, which combines three-word recall and clock drawing test, is a quick screening test that separates those likely to have dementia vs. those that do not. The detection rate can be further improved by adding animal naming in one minute to the test. If a patient screens positive, then the ABC checklist can be used to assess cognition, function and behavior.

Dr. Coolican gives us his take on VAS-COG 2009, which took place in Singapore in January this year. The puzzle of dementia continues but, as seen at the meeting, progress has been made. New imaging techniques show promise in detecting the burden of disease and may help predict who will progress to dementia.

Presenters at the meeting discussed clinical tools including the MOCA, which may be more useful than the MMSE, as well as the concept that the burden of disease is a combination of three things: plaques and tangles, microvascular insults, and brain atrophy in critical areas.

In Dr. Thorpe's article, she reviews how symtpoms of dementia and depression can often overlap and that these conditions may mimic each other, yet their treatments are quite different (anti-depressants vs. cholinesterase inhibitors or NMDA receptor blockers). This emphasizes the importance of a proper diagnosis. Dr. Thorpe also discusses that depression can lead to a greater risk of dementia later in life by damaging the hippocampus. On the other hand, as dementia progresses, behavior and mood issues that emerge (apathy, agitation and insomnia) could easily be mistaken for depression. There are numerous scales for diagnosis, but nothing replaces a good history, especially from a caregiver. Dr. Thorpe also reminds us to check for anemia, vitamin B12, thyroid, electrolyte imbalance and drug effects (interactions, anticholinergic effects).

This issue is full of great articles and, hopefully, they will stimulate our minds, and provide us with pearls to take back to our patients.

Diagnosis and Treatment of Subcortical Ischemic Vascular Dementia

The small-vessel variant of ischemic vascular disease appears to have been neglected in the vascular dementia (VAD) model and the overall cerebrovascular pathologic picture. Subcortical ischemic vascular dementia (SIVD) is quite difficult to identify and diagnose due to the difficulty of establishing a causal relationship between the changes and cognitive deficits seen by a primary-care physician, as well as identified through various brain-mapping techniques.

By Peter N. McCracken, MD, FRCPC

Apoplectic dementia was the term utilized in the past when patients presented with sudden cerebral bleeding, softening or tumors that resulted in an abrupt change in cognition, which could be progressive and incurable. In the early 1900s, it was widely accepted that atherosclerosis caused gradual stenosis of the brain vessels, causing parenchymal lesions that lead to dementia. However, the differentiation between other dementias and VAD were not clear, and the association between clinical strokes and cognitive changes were not firmly established. Hardening of the arteries was a term applied to a number of different dementia syndromes, including at times, and erroneously, Alzheimer's disease (AD).

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Vascular Dementia

Modern concepts of VAD began to evolve in the 1970s when studies from Newcastle revealed that subjects with dementia and little, if any, AD pathology demonstrated a relationship between lost tissue volume from infarctions and the degree of global cognitive decline. Hachinski[1] subsequently introduced the term multi-infarct dementia (MID) in 1974, which includes a history of clinical strokes with focal neurologic signs and symptoms, and stepwise cognitive decline.

The medical community has perhaps over-focused on this model of VAD, neglecting the sizeable contribution from small-vessel disease to the overall cerebrovascular pathologic picture. Hence, this article pertains to the small-vessel variant of ischemic vascular disease: SIVD.

Subcortical Ischemic Vascular Dementia

The greatest challenge in diagnosing SIVD lies in establishing a causal relationship between the magnetic resonance imaging (MRI) changes and the cognitive deficit(s) from the viewpoint of a primary-care physician. One can argue that causality is irrelevant, but if a patient has SIVD, management of the vascular risk factors should be optimized, regardless of their exact role in causing the dementia syndrome. What is clear is that the etiology of SIVD involves hypertension and diabetes mellitus (DM), particularly if poorly controlled.

Although there are minor differences in wording, most dementia definitions share the following elements:

1) acquired intellectual decline despite a clear sensorium;
2) effects on multiple cognitive domains which usually include memory; and
3) sufficient severity to interfere with customary everyday activities.[2]

During the past five years, emphasis has been placed on identifying early stages of vascular cognitive impairment in order to administer treatments earlier in the disease. Three historic syndromes fall under the current rubric of SIVD:[3]

1) lacunar state, first described by Marie[4] and Perraud;[5]
2) thalamic or strategic infarction dementia; and
3) subcortical arteriosclerotic encephalopathy, or Binswanger's syndrome.

These syndromes are associated with frontal-type behavioral symptoms. A unifying hypothesis based on disruption of cortical and subcortical circuits has been proposed.[6] Three such circuits are relevant to non-motor behavior:

1) a dorsomedial prefrontal circuit related to executive function;
2) a medial prefrontal circuit related to initiation and drive; and
3) an orbital prefrontal circuit related to social behavior.

An alternate hypothesis is that deep white-matter lesions disrupt the white-matter tracts necessary for cognition and emotion. These include association and commissural, striatal and subcortical fibres that interconnect with distributed neural circuits. Widespread lesions of the white matter have major effects on initiation and frontal executive function because of preferential disruption of long association fibres. However, it should be remembered that such changes at times remain clinically silent, gradually exhausting the clinical reserve of affected individuals.

Clinical SIVD features include sudden hemiparesis, dementia, dysarthria, pseudobulbar palsy, and changes in affect including inappropriate laughing or crying, small-stepped gait, and urinary incontinence. Aphasia and hemianopsia are usually absent. The distribution of lacunes in the subcortical gray matter and diffuse softening of the white matter, particularly of the frontal lobes, have been noted. Behavioral symptoms include lack of volition and akinetic mutism, which were thought to be characteristic of prefrontal lobe lesions.[3]

Strategic infarct dementia (*e.g.,* thalamic dementia) typically involves distribution of the paramedian thalamic artery. This usually includes the dorsomedial nuclei, closely connected with the prefrontal lobes. Dementias associated with such strategic syndromes are featured by marked apathy, impaired attention and mental control, with anterograde and retrograde amnesia, as well as striking executive dysfunction.[3]

Binswanger's syndrome was first described by Otto Binswanger.[7] He outlined eight cases of slowly progressive mental deterioration and pronounced white-matter changes with secondary dilatation of the ventricles. Alzheimer subsequently reported the microscopic features, including severe gliosis of the white matter and hyalination, intimal fibrosis, and onion-skinning of the long medullary arteries. Chronic hypoperfusion of the periventricular and deep white-matter zones is postulated as the mechanism of injury.

The clinical features of Binswanger's include an insidiously progressive dementia, persistent hypertension or systemic vascular disease, lengthy clinical course with long plateaus, and the accumulation of focal neurologic signs including asymmetric weakness, pyramidal signs, pseudobulbar

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Table 1

Diagnosis of Subcortical Ischemic Vascular Disease (SIVD)[3]

Findings that increase the likelihood of SIVD

1. History of sudden onset or stepwise decline in neurologic or cognitive function.
2. History of chronic hypertension or DM, especially if inadequately treated.
3. History of small stroke (pure motor or sensory).
4. Focal neurologic findings such as reflex asymmetry, pronator drift, Babinski sign, small-stepped or shuffling gait.
5. Evidence of hypertensive or diabetic end-organ disease (*e.g.,* retinopathy, LV hypertrophy, nephropathy).
6. Evidence of SIVD on structural neuroimaging study.

palsy, and gait disturbances. The neurobehavioral symptoms include apathy, lack of drive, depression, and alterations of mood. Of course, the periods of slowly progressive deterioration can mimic AD. Sensitivity for detecting vascular brain injury widened significantly with the advent of modern imaging: computed tomography (CT) in the 1970s, and MRI in the 1980s and 1990s. The previous threshold occurred at the level of symptomatic stroke. More recently, neuroimaging has revealed evidence of silent brain injury without a history of a corresponding clinical event.

However, we should be clear that evidence-based standards that address sensitivity and specificity of clinical criteria against a reference standard are limited. In contrast to AD, there is no gold standard agreed upon for the diagnosis of VAD. Several findings in the clinical history and examination increase the likelihood of SIVD (Table 1).[3] The versatility and power of MRI do offer exciting clinical and research opportunities. High-yield MRI at three or more tesla offer unprecedented anatomic resolution; functional MRI and perfusion MRI give excellent temporal resolution; and diffusion tensor imaging provides information about architectural integrity.

Epidemiology

After AD, cerebrovascular disease is the second most common type of dementia. The incidence and prevalence of SIVD, however, is unknown.[8]

Epidemiologic studies tend to focus on a broad category of VAD, often adapting different classifications, and being frequently unable to reliably identify mixed AD/VAD.[9] Furthermore, few epidemiologic studies employ neuroimaging, leading to underestimates or precluding SIVD. Among patients hospitalized for first stroke, the proportion of events attributed to SIVD is 10% to 30%.[8] In another study, the proportion of cases attributed to SIVD varies from 36% to 67%[10] among patients diagnosed with VAD.

Pathophysiology

Small arteries refer to arterioles within the brain parenchyma. These vessels range in size from 100 to 600 microns in diameter, and do not have an internal elastic lamina. Arteriolosclerosis refers to the progressive deposition of hyaline in the smooth-muscle wall of the small arteriole. Hypertension and DM accelerate this degenerative process. As the media of the small artery undergoes progressive lipohyalinosis, the lumen narrows and the vessel becomes more tortuous and coiled. Ultimately, the process leads to fibrinoid necrosis and increased risk of thrombosis or hemorrhage.

Two pathophysiologic pathways cause ischemic brain injury associated with small-artery disease. The first pathway involves an occlusion of an arterial lumen and leads to lacunar infarction. The second is characteristic of a generalized narrowing of small arterioles and hypoperfusion, which leads to incomplete infarction of the deep white matter. While both pathways share roots in common risk factors and small-artery disease, they differ in the anatomic location and pathologic extent of their injury, as well as the accompanying clinical symptomatology. The end stages of these two pathways are known as lacunar state and Binswanger's syndrome. At later stages, the two pathways often converge.

When the lumen of an artery is occluded, acute ischemic injury is centered in its primary irrigation territory. For small penetrating arteries, the resulting so-called

lacunar infarct is usually < 1.5 cm in diameter. Sites of predilection include the white matter, especially in the frontal lobe, followed by the putamen, thalamus, pons, and caudate. Arterial occlusion is associated with complete infarction of all tissue elements, eventually leading to cystic necrosis. Small, or non-strategically located infarcts may be completely asymptomatic or silent. Symptomatic lesions are recognized by the sudden appearance of neurologic dysfunction such as pure motor weakness, pure sensory loss, or behavior or cognitive change.

Clinical Treatment

The primary treatment and prevention of SIVD is to manage the vascular risk factors to prevent further strokes. Close attention should be paid to the lowering of blood pressure and the control of DM. Antiplatelet agents should be utilized depending on the exact details of the clinical situation. Several longitudinal community-based studies, including the Framingham Heart Study,[11] Rotterdam Scan Study,[12] and the Honolulu Asian Study,[13] provide evidence that identification and control of risk factors in midlife may reduce the risk for cognitive impairment late in life. Of note, there are very few long-term trials measuring treatment outcomes in SIVD.

In the follow-up of the Systolic Hypertension in Europe Study, long-term antihypertensive treatment over 3.9 years reduced the risk of dementia by 55%, from 7.4 to 3.3 cases per 1,000 patients.[14] Even though this study included more than simply SIVD, energetic treatment of those vascular risk factors can been seen to alter long-term outcomes favorably. On the other hand, previous cross-sectional studies have suggested that MRI-defined SIVD is associated with marked cognitive impairment and, specifically, deficits of mental speed, attention, and executive skills. The LADIS Study[15] revealed that SIVD subjects seemed to be vulnerable to a rapid period of cognitive decline in these domains.[15] Further work will be required to hone in much further on this particular subset.

The primary treatment and prevention of SIVD is to manage the vascular risk factors to prevent further strokes. Close attention should be paid to the lowering of blood pressure and the control of DM.

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Targeted Screening for Dementia

Dementia may be the most important health problem for the baby-boomer generation and healthcare system. As new disease-modifying drugs become available in the next decade, it will be critical to have strategies and mechanisms in place to diagnose dementia at its earliest, even preclinical, stage. These strategies include public and professional awareness, reorganization of primary care to facilitate enhanced roles of nurses and nurse practitioners, and targeted screening of high-risk seniors.

By William B. Dalziel, MD, FRCPC

The Alzheimer Society of Canada recently released *Rising Tide: The Impact of Dementia on Canadian Society,* which states:

- there are currently 500,000 Canadians with dementia;
- one in 11 Canadians older than 65 years has dementia;
- 50,000 Canadians younger than 60 years have dementia;
- one in four Canadians has a family member with dementia;
- one in two Canadians knows someone with dementia;
- and there will be 250,000 new cases of dementia in Canada in the next five years.

Dementia Presentation

Based on practice size and percentage of elderly patients, the average family physician has approximately 40 to 50 patients with dementia, and can expect eight to 10 patients to develop the condition each year. Yet more than half of all cases may go undetected in the primary-care setting[1] where dementia presents in four main scenarios:

1. A family member brings the patient to see their family physician with concerns about the patient's memory (95% specific for dementia), but the physician rules out delirium, depression, drug side effects and other reversible causes. Unfortunately, the average delay from first symptom to dementia presentation is more than two years.
2. Delirium either uncovers premorbid mild dementia or has incomplete resolution, which occurs in approximately 1/3 of all cases leading to dementia.
3. A health professional notices early warning signs or red flags (Table 1).
4. A targeted screening of high-risk but asymptomatic persons results in dementia presentation.

Would you Screen this Patient for Cognitive Impairment or Dementia?

Mr. AD is an 80-year-old male who has been your patient for 23 years. He has recently been diagnosed as hypertensive and is in for a BP check, which is 165/80 mmHg despite treatment with diuretics. He has no memory complaints or family history of dementia. Would you, as the family physician, screen the patient for cognitive impairment or dementia? How? In many cases, the patient would not be screened.

Principles to Justify Screening

Screening for cognitive impairment is justified based on common principles:

1. Dementia is common, present in 8% of people older than 65 years, and found in 35% of people older than 85 years.
2. It is the third most expensive disease, costing $10 billion annually, and is the number-one cause of long-term-care institutionalization.
3. The screening test should have reasonable sensitivity/specificity.

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Suggested screenings include the memory impairment screen (MIS); a Mini-Cog test which is a three-item recall and clock drawing; and the Montreal Cognitive Assessment Test (MOCA) or the General Practitioner Assessment of Cognition (GPCOG).

4. The screening test should be short and take no more than five minutes. To stay within this time frame, it is suggested to conduct the MIS, the Mini-cog or the GPCOG.
5. There are treatments available to improve clinical outcomes:
   a. treatment of vascular risk factors;
   b. cholinesterase inhibitors (donepezil, rivastigmine, galantamine), gamma aminobutyric acid antagonists (memantine); and
   c. caregiver education and support.

The biggest barriers to screening are the time restrictions and the belief that early detection has no benefits. As healthcare moves from solo primary-care practitioners to groups or teams, often other professionals (*i.e.,* nurse practitioners and nurses) can be much more involved in screening and cognitive assessment, sparing the physician's time. Table 2 presents benefits of early recognition of cognitive impairment and dementia.

Table 1

Early Warning Signs of Dementia

- Frequent hospitalizations or visits to emergency room
- Confusion, delirium, sickness, surgery
- Poor historian, vague, repetitive questions and stories
- Changes in mood, personality or behavior
- Decreased social interaction
- Subacute change in function, dwindles
- Poor understanding or compliance with instructions
- Poor medication compliance leading to poor disease control such as chronic heart failure (CHF) and chronic obstructive pulmonary disease (COPD)
- Driving: accident, problems, tickets, family concerns
- Neglect (appearance, home, nutrition)

Table 2

Benefits of Early Recognition of Cognitive Impairment/Dementia

Social

- Right/need to know
- Social/financial planning
- Safety: compliance, driving, cooking
- Advance directives, planning
- Improving caregivers well-being with education & support delays the AD patient's nursing home placement by 1.5 years[2]

Medical

- Treat reversible cause/components
- Risk factor treatment (BP, lipids, DM, etc.)
- Compliance strategies for cooking and medications instructions (improve the care of other chronic diseases: BP, DM, CHF, COPD, etc.)
- Cholinesterase inhibitor (CI) or memantine treatment
- Crisis avoidance and contingency

Mild Cognitive Impairment vs. Dementia

Early cognitive impairment (if beyond normal aging changes) is generally mild cognitive impairment (MCI), prevalent in 10% to 15% people older than 65 years, or dementia, prevalent in eight percent of people older than 65 years.

MCI is a construct of preclinical dementia, and often represents cognitive impairment which does not cause functional impairment like dementia. Ten to 15% of patients with MCI progress to dementia per year, but over a 10-year follow-up, approximately 30% do not. In the next five to 10 years when, hopefully, we have disease-modifying agents for dementia and MCI, it will be important to have primary-care screening mechanisms in place to better identify MCI and early dementia. The benefits of the early detection of MCI include regular monitoring to rule out progression to dementia, treatment of vascular risk factors, and reassurance to the patient that this is not yet Alzheimer's disease (AD).

The MOCA, available at *www.mocatest.org*, is significantly superior to the Folstein Mini-Mental

Table 3

Memory Impairment Risk Calculator

	Number of Vascular Risk Factors			
Age(years)	**0**	**1**	**2**	**3 or more**
65-69	2%	4%	8%	16%
70-74	4%	8%	16%	32%
75-79	8%	16%	32%	50%+
80-84	16%	32%	50%+	50%+
85 and over	32%	50%+	50%+	50%+

How to Use the Risk Calculator

1. Circle your age range on the left side of the Memory Impairment Risk Calculator above.
2. Check all your vascular risk factors below. On the risk calculator above, circle the number of vascular risk factors. Your RISK SCORE for memory impairment appears where the Age row and Risk Factors column intersect.

Vascular Risk Factors (Check Off All that Apply)

- ☐ High blood pressure
- ☐ Diabetes
- ☐ High cholesterol
- ☐ Obesity
- ☐ Sedentary (no exercise)
- ☐ Stroke or TIA (transient ischemic attack)
- ☐ Angina or heart attack
- ☐ Peripheral vascular disease
- ☐ Atrial fibrillation
- ☐ Currently smoking

# of Risk Factors ☐

What Should you do with your Risk Score

This table calculates the potential risk for memory impairment. Scoring higher risk (16% or more) means it is worth doing a "Memory Quick Screen" (it does not mean dementia or AD is present). Scoring low risk (less than 16%) means low likelihood of important memory problems, but if you have noticed significant changes in memory or functional ability to do things, the Memory Quick Screen should be done.

Status Examination (MMSE) in detection of MCI and early dementia, and takes no more than 10 minutes.

Should the Elderly be Screened for Cognitive Impairment?

There appear to be clinical benefits if elderly patients are screened for cognitive impairment and dementia. Yet screening is still disputed. The Canadian Consensus Guidelines (1998) state there is no evidence for or against screening. The American Academy of Neurology Guidelines suggest screening is justified only in a high-risk subpopulation. Personally, I support the concept of targeted screening only for patients who have a high-risk of dementia. As prevalence increases, true positives increase and false positives decrease. To establish a high-risk subgroup, physicians should use the "Rule of Two."

The Rule of Two

The greatest risk factor for dementia is age. People older than 65 years have a 2% chance of being affected by dementia, their risk doubling every five years.[3] Furthermore, each first-degree relative with a history of dementia doubles the risk to the patient. Each vascular risk factor also doubles the risk.[4-6]

The Memory Impairment Risk Calculator, as seen in Table 3, was developed for a pharmacist screening project in Ottawa. The elderly person calculates their own risk and, if their test indicates high risk

Table 4
Dalziel's Non-validated Two-minute Targeted Screening Test

Test	Domain(s) Assessed	Failure	Odds Ratio[8,9]
1. Three-item recall	Registration, short-term memory	0 or 1/3	3.1
2. Four-legged animals in 1 minute	Executive function, language	< 15	20.2
3. Clock drawing	Visualspatial and memory (numbers), executive function (hands)	Abnormal*	24.0

* Mild irregularities in number placement do not count as a failure.

(> 15%), they can hand the test to the pharmacist who will then conduct a two-minute screening test. Results with suggestions for further assessment are sent to the family physician. Other scenarios with high prevalence of dementia in which to consider targeted screening include post-stroke, post-delirium, post-first onset depression after 65 years, and entry into a retirement home.

Tests Used for Targeted Screening of High-risk Individuals

Brodaty's clinical review[7] concluded: "It is recommended that general practitioners consider using the GPCOG, Mini-Cog, or MIS when screening for cognitive impairment or case detection." These tests are short (< 5 minutes), easy to administer, have been validated in general practice samples, and have reasonable classification rates compared to the Folstein MMSE.

In my clinical practice and teaching, I have added animal fluency (four-legged animal naming in one minute) to the Mini-Cog. This has not been validated, but animal fluency, a good test of executive function, has been separately validated with an odds ratio of 20.2 if less than 15 animals are named in one minute. Table 4 presents the two-minute targeted screening test.

There are always concerns about the consequences of a false positive screen. If a high-risk elderly person screens positive, the consequences are a collateral history from a family member or friend for ABC symptoms. The ABC checklist (Table 5) with a Memory Quick Screen for cognitive problems include activities of daily living (A), behavior changes (B) and cognitive changes (C). If there are no ABC changes or symptoms, it is a false positive, and the general practitioner should follow up in one year. If there have been changes, comprehensive cognitive assessment is indicated.

Mr. AD's Assessment and Follow-up

Let us now return to the hypothetical 80-year-old man in your practice previously mentioned. Due to advanced age and hypertension, Mr. AD's overall risk of cognitive impairment or dementia is 32%. His Memory Quick Screen was 1/3 recall, he named nine animals, and his clock drawing showed the most common early abnormality (hands on the clock drawn to 10 & 11). His wife was invited to the next office visit to review ABC changes. She said: "Now that you mention it, he has been getting more forgetful, a little irritable and apathetic. He's making mistakes with finances and is having trouble using the computer." Of note, family may often dismiss such changes as normal aging or unimportant.

Based on practice size and percentage of elderly patients, the average family physician has approximately 40 to 50 patients with dementia, and can expect eight to 10 patients to develop the condition each year.

Table 5

ABC Checklist for Cognitive Problems (if Memory Quick Screen is Positive)

	OK	Problem(s) with:	
1. ADLs	☐	☐ Shopping ☐ Finances ☐ Hygiene/grooming ☐ Hobbies/leisure ☐ ↓ or problems in dressing/bathing	☐ Housekeeping ☐ Cooking ☐ Tools/appliances ☐ Transportation ☐ Needs more help/guidance
2. Behavior	☐	☐ Apathy/↓ initiative ☐ Anxiety ☐ ↓ alertness/"tuned out" ☐ Poor judgement/self control ☐ Aggression	☐ Depression/moody ☐ Hallucinations ☐ Hiding/hoarding ☐ Emotions labile/inappropriate ☐ Agitation/anger
3. Cognition	☐	☐ Repetition (stories, questions) ☐ Word finding ☐ Medication compliance (dosette) ☐ Misplacing things ☐ Confused in unfamiliar circumstances	☐ Forgetfulness ☐ Orientation/gets lost ☐ Focus/following ☐ Reading/TV ☐ Fails to recognize family/friends

Other observations (including duration/progression of problems):

Form should be filled out by patient and family/caregiver.

Conclusions

Targeted screening for seniors who are at high risk for cognitive impairment can facilitate earlier recognition of dementia. Treatment options used earlier can lead to improved outcomes. These include the use of specific antidementia drugs, treatment of vascular risk factors, strategies to improve adherence in other chronic diseases and enhanced support and education for caregivers.

With the aging demographics and society of specialists in dementia, the predicted 250,000 new cases in the next five years indicate that the primary-care system will need to take a larger role of the early identification, diagnosis and management in the more straightforward cases of persons with dementia.

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Clinical Research and the Family Practice: VAS-COG 2009

The 4th Congress of the International Society for Vascular, Behavioral and Cognitive Disorders (VAS-COG) was held in Singapore in January this year. Dr. Coolican attended to inform himself about the vascular causes of various brain disorders including dementia. The conference held much information, including new brain-imaging techniques, genomics and DNA mapping, neuropathology, obesity and more. Here, Dr. Coolican gives us his take on Alzheimer's disease (AD), mixed dementia and the relevance of the clinical research presented at the conference for primary caregivers.

By Paul J. Coolican, MD, CCFP, FCFP

From a family doctor's perspective, dementia is a common progressive debilitating condition with a variable course which results in complete incapacity and premature death.

From a clinical researcher's perspective, dementia is a huge challenge which is now beginning to be understood in ways that could lead to effective strategies for prevention and treatment. Researchers see enormous opportunities as new technologies (*e.g.,* specialized neuroimaging and genetic mapping) allow increased access and better understanding of the function of the brain aging normally or diseased.

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VAS-COG 2009

The large gap between these two visions prompted me as a family physician to attend VAS-COG being held in Singapore in January of this year. I wanted to understand where researchers were going and what, if any, impact their work would have on my work as a family physician in the near and long term.

The conference housed a diverse group who, at first, appeared to be firing off madly in all directions. Papers and plenaries jumped from highly technical reviews of new magnetic resonance imaging (MRI) techniques for brain imaging, to brain metabolism, to genetics, to neurohistopathology, to genomics and DNA mapping, to obesity, to neuropathology, to diabetes and all points in between.

I first believed that I would get nothing out of this broad morass of technical information, but soon began to find my way over the next three days at the conference. I will continue by sharing some general impressions, as well as a few specific findings which should be of interest to primary-care providers.

History of Alzheimer's Disease

In 1906, Alzheimer first identified pathological findings of neuritic plaques and neurofibrillary tangles as characteristics of senile dementia. In 1975, Folstein developed a clinical tool which gave a standardized score to diagnose and stratify AD.

It is believed that Alzheimer's type dementia is caused by the aforementioned plaques and tangles, the disease progressing as they increase. However, several longitudinal studies have shown that a large percentage of non-demented individuals were found on autopsy to have many plaques and tangles, and that a number of demented individuals classified clinically as having AD only had a few of either.

In addition, when an antibody vaccine to amyloid, the main component in these plaques, was developed and tested, researchers found that,

while the plaques disappeared, there was no predictable response in the clinical course of the disease.

So, What Gives?

Most clinicians believe that most dementias are a mixed disorder, and that these insults may be both vascular and neurodegenerative. Many attempt to answer this question in a wide variety of ways, such as with new developments in neuroimaging techniques, particularly with MRI. Not only do these studies demonstrate the importance of vascular insults to the brain, but they also indicate the importance of the location of these insults, which include lacunar infarctions, micro-hemorrhages and white-matter hyperintensities reflecting vascular damage to white-matter tracts in the subcortical and periventricular areas of the brain.

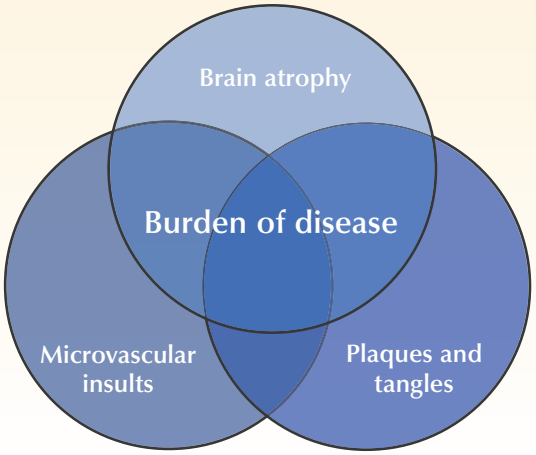
Many of these insults are silent and do not cause overt clinical symptoms such as stroke or TIA. They reinforce the concept that most dementia is a mixed pattern of AD and vascular dementia (VAD). In addition, they show the importance of the location of white-matter changes, particularly in specific areas of the corpus callosum, and the cingulate body in the severity and progression of dementia. They reinforce loss in the hippocampal area and the medial temporal lobe in clinical syndromes which are predominantly AD-type presentations (memory and executive function).

Burden of Disease

There are some clinicians who believe clinical dementia is a reflection of the burden of disease. In comparing neuropathologic and imaging studies, two individuals might have no clinical evidence of dementia on testing or may have severe dementia with similar burden in terms of the amount of plaques or tangles. However, these two individuals would have large differences in brain atrophy or vascular damage.[1]

Figure 1

The Concept of Contributors to Burden of Disease in Clinical Dementia[2]



One presentation at the conference used a Venn diagram (Figure 1)[2] to suggest there are three predominant components which, in the opinion of the presenter, contribute to the burden of disease: neurofibrillary plaques and tangles, microvascular insults and general brain atrophy.[2] This hypothesis was not uncontested. Consensus appeared to be that the concept of burden of disease was sound, but that a factor such as global brain atrophy was not specific enough. In the presentation, the MRI studies showed more specific areas of atrophy, and the presenter argued that they were more useful indicators and more predictive of the burden of disease.

Relevance to Clinicians

Does any of this have any clinical relevance to a 75-year-old man in your office whose daughter thinks he is starting to become withdrawn or forgetful? The short answer is: yes. If the patient in question has a vascular disease or other risk factors, research suggests that either may be a major contributor to his risk of cognitive decline.[3]

While there were no studies presented at the conference which showed reduced progression in dementia where vascular risks were treated, several studies did show that untreated or inadequately treated hypertension, atherosclerosis, obesity and diabetes were associated with earlier and more severe cognitive decline. The epidemiologic studies and the longitudinal studies, presented by Drs. Whitmer and Arvanitakis respectively, showed the importance

of diabetes, central obesity and other risk factors in the later development of dementia as well. While this should come as no surprise to family physicians, it should guide us in our early treatment of these risk factors, and in screening of these individuals for mild cognitive impairment (MCI) and dementia.

Even more exciting is the research which has shown that neuroimaging techniques may help predict those with MCI without functional dementia, who will progress to dementia.[4] This now may allow us to stratify risk in a manner similar to the risk engines used for cardiac disease in our clinical guidelines. In addition, we know that statins may lower stroke risk in individuals at high risk for cardiovascular disease.[5] The message appears to be that we should identify and treat risk factors in individuals with MCI, a strong family history of dementia, and those with documented cerebrovascular disease on neuroimaging (by computed tomography scan or MRI).

Canadian Honored

At the Congress, Dr. Vladimir Hachinski, a Canadian best known for his work in vascular dementia, was honored by the Singapore Chapter of Neurologists. He addressed the need to rethink the current classifications used for dementia. He urged members present to "think outside the box." Other noted Canadian researchers and clinicians who participated or presented included Dr. Sandra Black from Toronto and Dr. Ken Rockwood from Halifax.

Conclusions

The wide variety of presentations was overwhelming at a congress such as this. I found myself needing a primer in physics, neuroanatomy, biochemistry and genetics. Listed below are several brief take-away messages from the plenaries and poster presentations at the conference:

- Time for one test in your busy office? MOCA is a better screen for mixed dementias than the MMSE, and can be done in 10 minutes. (Available at *www.mocatest.org*).[6]
- Screen your diabetics early and regularly for dementia.[7] Screen all your older patients with vascular risk factors (hypertension, hyperlipidemia, smokers).
- Late-onset depression and/or depression in individuals with cognitive impairment is associated with a poor response to antidepressants. Vascular depression may respond to ECT.[8,9]
- If a patient has MCI, consider requesting an MRI to examine white-matter hyperintensities, lacunes, brain atrophy (global and specific areas).[10] These may help to identify burden of disease, and the patients who are more likely to progress to dementia.

In addition, patients with MCI should have their vascular risk factors identified and treated aggressively to protect the brain from silent vascular insult. While we cannot provide an effective disease treatment for these individuals at present, we can help patients and families plan for future health needs.

The brain is a complex and still poorly understood organ. As its secrets are slowly unraveled, the hope is that we can maintain its function over time in the face of degenerative disorders.

As a family physician looking in on this conference, I was impressed by the progress made, and yet struck by how much further we still have to go to make a significant impact on this difficult disease.

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Depression vs. Dementia: How Do We Assess?

Depressive disorder and dementia are common in older people, and may occur separately or together. Diagnosis is often challenging because of the frequency of symptoms which are common to both disorders. Unfortunately, underdiagnosis of depression results in missed opportunities to improve functioning, decreased quality of life and possibly even increased mortality. Yet, overdiagnosis of depression may result in unnecessary adverse effects of psychotropic medications. This article suggests approaches to differential diagnosis.

By Lilian Thorpe MD, PhD, FRCP

Dementia increases with age, with an overall prevalence in Canada of 8% in those 65 years and older, 2.4% in those aged 65 to 74 years, 11.1% in those aged 75 to 84 years, and 34.5% in those aged 85 years and older.[1] Alzheimer's disease (AD) is thought to be the most common type of dementia in all age groups. However, younger age groups are more likely than older age groups to be diagnosed with other dementias, such as frontotemporal dementia and vascular dementia.[2]

Major depressive disorder is also common, but is only one of a number of disorders listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)[3] with prominent depressive symptoms (Table 1).[3] These include dysthymic disorder, depressive episodes of a bipolar disorder, mood disorders secondary to a medical disorder (such as hypothyroidism), mood disorders secondary to a substance, adjustment disorders and bereavement. Depressive disorder is commonly seen in all stages of adult life and, while its prevalence is slightly lower in the elderly,[4] its sequelae are probably greater in more frail people, exerting a more deleterious effect on functional abilities and even increasing the length of stay for hospitalizations related to primary medical conditions.[5] Depressive disorder in seniors can occur as part of a lifelong recurrent disorder, or it can present for the first time in old age. It is frequently concurrent with other medical and mental disorders, including various dementias.

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Relationships Between Dementia and Depression

Dementia and depression have a complicated relationship, with at least two contradictory directions of potentially causal influence. One hypothesis suggests that depression leads to dementia, and another that suggests that dementia itself leads to depression.

The depression-to-dementia direction is supported by evidence that depressive disorder is a risk factor for developing dementia in later life[6] and, consistent with this, the best-studied people with a biological predisposition to develop AD (those with Down Syndrome) are thought to have a high risk of suffering from depression.[7] The association between depression and later development of dementia is still not completely understood. One possibility is that depression is an early, prodromal phase of dementia,[8] and is caused by the same pathophysiologic initiators that result in dementia. There is also evidence that depression is associated with damage to brain locations integral to cognitive processes, such as the hippocampus, possibly by decreasing neurogenesis.[9] This

Table 1

DSM-IV Mental Disorders with Prominent Depressive Symptoms[3]

- Major depressive disorder
- Dysthymic disorder
- Bipolar disorder (depressive episode)
- Mood disorders secondary to a general medical condition
- Mood disorders secondary to a substance (such as a medication)
- Adjustment disorder with depressed mood
- Bereavement

process may lower the threshold for later observable cognitive loss, eventually increasing age-adjusted dementia rates. Behaviors associated with depression, such as heavy alcohol uses and vascular risk factors like cigarette smoking,[10] may also independently increase later cognitive loss, while medications prescribed to treat depression, especially those with strong anticholinergic effects, could conceivably have adverse cognitive effects, although this effect is likely more transient.

The dementia-to-depression direction in the potentially causal relationship between the two disorders is supported by findings that people with dementia appear to have a higher prevalence of depression.[11] However, prevalence rates vary widely depending on the study population (psychiatric outpatients, Alzheimer registries, old-age homes), instruments used, and diagnostic definitions. Most problematically, the term depression is used to denote different clinical concepts, which are not always equivalent to a diagnosis of DSM-IV major depressive disorder.

Muller-Thomsen et al[12] illustrated large variability in the diagnosis of depression in dementia by using four different scales in the same population, and found that between 27.5% and 53.4% of people with mild AD and between 36.3% and 68.4% with moderate to severe AD were found to rate positive for depression. Studies comparing differences in the prevalence of carefully diagnosed depressive disorders between matched demented and non-demented populations are not frequent, but suggest that motivational deficits in dementia may be the greatest difference between these groups, rather than typical DSM-IV major depressive disorder.[13] However, regardless of the exact prevalence of formally diagnosed depressive disorder in dementia, it does seem that depressive syndromes are very common in those with dementia, and that this comorbidity causes increased deficits in functioning, increased problematic behavior,[11] increased nursing-home placement,[14] increased caregiver stress,[15] and increased mortality.[16]

Under- and Overdiagnosis of Depression in Dementia

Depressive disorder has long been thought to be underdiagnosed in those with dementia,[17,18] although a recent Danish study suggests that this may now have changed, at least in Denmark.[19] Underdiagnosis of depression in demented seniors is clearly undesirable, as depressive disorders in the demented elderly have been associated with additional burden, as described above. Undertreatment with antidepressants may also result in over treatment of depression-associated behaviors with benzodiazepines and possibly neuroleptics. Adverse effects of benzodiazepines and neuroleptics are well recognized and include increased falls, decreased alertness, extrapyramidal side effects, decreased mobility, decreased functioning and increased mortality. Efforts have been made to increase the recognition of depression in those with dementia, and widely used instruments such as the Minimum Data Set[20] include quality indicators to alert administrators of patients with likely depression who are not being treated with antidepressants. Review of these quality indicators may precipitate discussion with attending physicians, who have the opportunity to institute appropriate treatment.

Unfortunately, this process may also result in an overdiagnosis of depressive disorder due to the high prevalence of behavioral symptoms in dementia such as apathy and reactive mood symptoms, which overlap with those seen in major depressive disorder. Treatment with antidepressants is increasingly also known to be associated with adverse effects, most problematically in older, frail populations. Anticholinergic effects of the tricyclic antidepressants may cause confusion, constipation, urinary reten-

tion, and visual-accommodation problems. Postural hypotension may cause falls, and cardiac effects are particularly dangerous in overdose. Newer medications, such as selective serotonin reuptake inhibitors (SSRIs) and venlafaxine, were initially felt to be much safer, but have been increasingly associated with different, rather than fewer, adverse effects.

Gastrointestinal side effects and sleep disturbances appear to be more common with this group of medications. Recent research has suggested that SSRIs are no less likely than tricyclic antidepressants to cause falls.[21] They are also associated with a higher prevalence of hyponatremia,[22] and most recently research has suggested they increase fragility fractures.[23] Finally, SSRIs have been associated with increased apathy,[24] even in those who have been appropriately diagnosed with depression and have responded to this medication.

Challenges in the Diagnosis of Depression in Dementia

Diagnosing depressive disorder in the context of dementia is often difficult due to overlapping symptoms between depression and dementia, communication problems and lack of insight. Behavioral and psychological symptoms of dementia (BPSD) are integral parts of the clinical presentation of dementia, although this is often thought of as a disorder of progressive cognitive decline. BPSD includes many of those symptoms also seen characteristically in DSM-IV depressive disorders. Of the core symptoms of depressive disorder, listed in Table 2, sleep disturbances, changes in eating behavior, decreased initiative and interest (apathy), psychomotor agitation, and poor concentration (in advanced dementia) are common in dementia without depression. Reactive symptoms such as anxiety and tearfulness are also seen frequently in dementia without depression, and may be related to retained awareness of deficits in the early stages of dementia, poor coping skills and disorientation in the later stages, or to the mood lability accompanying vascular brain disease (which commonly overlaps with AD).

Table 2

DSM-IV Symptoms of a Major Depressive Episode

- Depressed mood
- Markedly diminished interest or pleasure
- Significant weight change
- Changes in sleep patterns
- Psychomotor agitation or retardation
- Fatigue or loss of energy
- Feelings of worthlessness, excessive or inappropriate guilt
- Diminished ability to think or concentrate
- Recurrent thoughts of death, suicidal ideation or suicidal actions

Much less common in dementia without depression are consistent sadness, marked morning mood worsening, feelings of worthlessness or excessive or inappropriate guilt, recurrent thoughts of death, suicidal ideation or suicidal actions.

Approach to Clinical Diagnosis

A variety of instruments have been developed to screen for depression in the cognitively intact population. These include the Beck Depression Inventory,[25] the Centre for Epidemiological Studies-Depression Scale,[26] the Geriatric Depression Scale,[27] the Hamilton Depression Rating Scale,[28] the Montgomery and Asberg Depression Rating Scale[29] and the Zung scale.[30] Although these scales vary considerably in how much they are affected by impairments in language, awareness and comprehension, none is useful in the later stages of dementia. Of greater usefulness in patients with advanced dementia are the Dementia Mood Assessment Scale[31] and the Cornell Scale for Depression in Dementia.[32]

However, the gold-standard differential diagnosis of depression in dementia is a careful clinical assessment, which includes obtaining information directly from the patient and from collateral sources, ideally those with good knowledge of the person. This assessment should include:

- Careful symptom history including:
  - detailed description;
  - time course and progression of symptoms; as well as
  - association with other confounding factors such as environmental stressors which include:

Table 3

Typical Presentations of Mood Symptoms in Dementia and Depression

Symptom	Dementia	Depression
General response to cognitive and functional decline	Frequent lack of concern or denial about symptoms.	Amplification of and excessive preoccupation with deficits.
Mood	Normal most of the time. Unhappiness is reactive to circumstances and fluctuates. Labile, especially with vascular dementia. Mood often brightens with stimulation and support.	Subacute (weeks) onset of pervasively sad mood, most of the day and nearly every day. Doesn't brighten much with stimulation.
Interest, initiative	Gradual loss of interest and initiative (apathy) over a longer period of time (years rather than weeks). Not accompanied by statements of sadness, tearfulness, or other distress. Still enjoys activities in a structured environment.	Subacute loss of interest and pleasure over a few weeks, frequently accompanied by sad mood and affect, and occasionally statements of guilt, hopelessness and self-harm.
Eating behavior and weight	Gradual loss of weight (over months to years) which is common in dementia. Large increases in weight may be secondary to decreased activity, medications, and hyperorality in patients with frontal behavioral presentations (more common in frontotemporal dementia like Pick's Disease).	Subacute changes (weeks) in appetite leading to increase or decrease in weight.
Sleep	Gradual disruption of the sleep-wake cycle (over months to years) due to brain changes of dementia, resulting in frequent night-time wakening and daytime sleeping.	Subacute changes in sleep over a few weeks (increase or decrease).
Psychomotor agitation	Gradual (months to years) increase in agitation, generally worse during the latter part of the day (sundowning). Patient much worse in unfamiliar settings (catastrophic reaction), and often seeking people or places from earlier life experiences.	Subacute (weeks) onset, often worse in the morning, may be present persistently throughout the day. Generally accompanied by other depressive symptoms such as nihilistic statements or excessive guilt.
Psychomotor retardation	Seen infrequently in mild to moderate dementia, but occasionally in very advanced dementia, and may be mimicked by Parkinson's dementia (facial masking, slow motor functioning) or advanced Pick's Disease.	Subacute onset of psychomotor retardation (over weeks) in severe depression.
Energy	Generally a normal energy level, but reduced activity due to poor initiation related to decreased executive functioning.	Subacute decrease in energy and increased complaints of fatigue.
Guilt or worthlessness	Uncommon, although transient statements of worthlessness might be seen in times of stress in those with preserved awareness of their own decline.	Common in severe depression, usually accompanied with low mood as well as changes in appetite and sleep.
Concentration and thinking	Concentration is normal in early dementia, but impaired in late dementia. Thinking ability declines throughout the course of dementia.	Subacute loss of concentration and sustained focus. Often indecisive and concerned about making mistakes.
Suicidal thoughts and actions	Uncommon.	Common.

  - pain;
  - poor nutritional status;
  - other medical conditions; and
  - recent changes in medications.
- Particular attention should be paid to depressive symptoms which are less common in dementia alone such as:
  - hopelessness;
  - expressions of guilt;
  - feelings of worthlessness; and
  - thoughts of self-harm.
- Frontal symptoms, such as disinhibition, perseveration and decreased initiative, suggest dementias with a strong frontal component rather than depression.
- Information about family history of mood disorders, previous personal history of depression and previous response to therapy for depression.
- Direct interview of the person, paying particular attention to: consistently low mood and affect that does not respond to stimulation; hopelessness, expressions of guilt; feelings of worthlessness; and thoughts of self-harm.
- Laboratory investigations, such as hematology, thyroid function, electrolytes, vitamin B12, and drug levels of medications, known to have a propensity to cause mood symptoms.

In addition to the above, neuroimaging might be performed to explore the potential contribution of vascular pathology to mood lability and apathy, and to rule out other neurologic problems such as normal-pressure hydrocephalus.

After this assessment, the clinician has to weigh the information obtained, taking into account the likelihood that the accumulated information represents depression rather than dementia alone. For example, isolated symptoms of apathy without associated sadness, crying, or changes in sleep or appetite are unlikely to represent a depressive disorder, whereas consistently sad mood or affect, not brightening during interpersonal contact and associated with subacute changes in sleep and appetite are much more likely to represent depressive disorder that requires medical treatment. Table 3 summarizes mood symptoms seen in depression and dementia, with a brief discussion about their more typical presentation in each disorder.

Conclusion and Treatment Issues

Sometimes it is very difficult to make a firm diagnosis of depression in the context of dementia, especially when the dementia is very advanced. The clinician will occasionally choose to instigate treatment regardless of diagnostic certainty, weighing the possible benefits versus potential adverse outcomes of treatment.

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Alzheimer Society
CANADA

News from the Alzheimer Society of Canada

Early Diagnosis Key to Critical Planning, Treatment and Support

Less than 25% of cases of Alzheimer's disease (AD) in Canada are diagnosed and treated. Yet, cognitive impairment and dementia are present in about 20% of the elderly population, and are rated among the top three concerns of elderly people.[1]

The Alzheimer Society is a strong proponent of early diagnosis of AD and related dementias. Early diagnosis is key to ensuring that people with this condition have the power to plan their futures, including their need for increasing healthcare support, and ensuring the individual receives the best help and care possible. Access to treatment, advice, information and support from community support services, including support groups at the Society, is particularly important during the process of diagnosis and throughout the disease's progression.

"There remains a misconception among many Canadian doctors that since you can't cure or stop the progression of dementia, a clinical diagnosis is less important," says Dr. Jack Diamond, the Society's Scientific Director. "However, in reality, early diagnosis is an essential first step towards receiving appropriate help and support. Delay in diagnosis means that people with dementia and their caregivers suffer unnecessarily from uncertainty about what is happening, and are deprived of the treatment and care they need."

"Moreover," continues Dr. Diamond, "new treatments undergoing clinical trials are fast approaching the time when they'll be available for doctors to prescribe, some quite possibly within three to five years. The earlier the diagnosis, the sooner the treatment can begin, and the more effective it will be."

For caregivers and supporting family members, early diagnosis is also critical. By accessing information, community services and support early, caregivers are better able to understand the disease, the changes that occur in the brain and the effect these changes have on the person's abilities and behavior. This understanding can lead to less stress and better health of the caregivers themselves.

"Without a diagnosis and a full understanding of what that diagnosis means, caregivers can become frustrated, irritated and angry because the person they love is changing in a way they do not understand," says Mary Schulz, the Society's Director of Information, Support Services and Education. "When there is a diagnosis, they can seek appropriate support and learn about strategies that can help maintain as high a quality of life as possible for everyone involved."

Part of the early diagnosis process involves getting an accurate diagnosis as soon as possible. Over the years, research on the use of different diagnostic methods has led to better diagnostic tools, but there is still confusion over which methods are sufficiently valid. Diagnosis of dementia is traditionally made by a physician, and remains a sensitive process involving a number of methods to accurately draw a conclusion.

As concern about AD increases, some have argued that population-based screening (also referred to as

broad screening) should be offered. Individuals who have no symptoms or other indication of a specific disease are tested to determine whether they might have signs of that disease. Screening should be undertaken only when there is clear evidence that early treatment of the condition results in more good than harm. Examples of effective screening programs include testing for high blood pressure and certain cancers.

Based on direction from Canada's leading AD clinicians and researchers, and the conclusions of evidence-based task forces in Canada, the US and the UK, the Society does not support population-based screening for memory or cognitive problems, whether these screenings take place in a primary care setting, a pharmacy or other non-clinical public location.[2]

World Alzheimer's Day 2009

The importance of early diagnosis is reaching global heights this fall as Alzheimer's Disease International (ADI) and its 77 member countries recognize World Alzheimer's Day September 21 with the message *Diagnosing Dementia: Seeing it Sooner.* The Society, a founding member of ADI, will be part of the global effort to help increase public awareness about both the disease and the importance of early diagnosis.

"If everyone in the population were aware that memory loss, confusion and difficulty with day-to-day tasks are not a normal part of aging, then they would be much more likely to seek professional advice when these problems occur," says Schulz. "Of course, it is particularly important that health professionals, especially primary care professionals, are aware of the importance of early diagnosis of dementia – for the person with the disease, their family members and for the healthcare professional themselves."

For more information on World Alzheimer's Day events, the Society's position on population-based memory screening, or to become an Alzheimer advocate, please contact 1-800-616-8816 or visit the Society's website at *www.alzheimer.ca*.

References:
1. Feldman HH, Jacova C, Robillard A, et al. Diagnosis and treatment of dementia: 2. Diagnosis. CMAJ 2008; 178(7):825-36.
2. Population Screening for Dementia, Position Statement. Alzheimer Society of Canada. Available at: *www.alzheimer.ca/english/society/policies-popscreening.htm.* Accessed July 2009.

The Alzheimer Society is the leading, nationwide health organization for people affected by dementia in Canada. The Society is a principal funder of Alzheimer research and training, provides enhanced care and support to people with the disease, their families and their caregivers, while acting as a prominent voice in the call for policy change within all levels of government. Active in more than 140 communities across Canada, the Alzheimer Society is also at the forefront of worldwide efforts to fight dementia as a founding member and affiliate of Alzheimer's Disease International.