# Screening for Cognitive Impairment and Dementia in the Elderly

Christopher J.S. Patterson, David A. Gass

**ABSTRACT:** *Objective:* To review the evidence available to support or refute the recommendation to screen for cognitive impairment (cognitive deficits which do not affect daily function) and dementia in primary care. *Data Sources:* Medline search using terms listed at the end of this article; consultation with experts in the field; review of other published recommendations. *Study Selection:* There were no articles which described a randomized controlled trial of screening versus no screening. Studies were therefore chosen which aided in the definition; natural history; interventions and outcomes including possible negative effects. *Data Synthesis:* No systematic synthesis was performed. Background papers were circulated to a panel of experts prior to the Canadian Consensus Conference on Dementia and conclusions endorsed by consensus. *Conclusions:* 1. There is insufficient evidence to recommend for or against screening for cognitive impairment or dementia. (C); 2. Memory complaints should be evaluated and the individual followed to assess progression. (B); 3. When caregivers or informants describe cognitive decline in an individual, these observations should be taken very seriously; cognitive assessment and careful follow-up are indicated. (A) (See Appendix).

RÉSUMÉ: Dépistage de la dysfonction cognitive et de la démence chez les gens âgés. Objectif: Revoir les données disponibles supportant ou réfutant la recommandation de faire le dépistage de la dysfonction cognitive (dysfonction cognitive qui n'altèrent pas le fonctionnement quotidien) et de la démence dans la pratique de première ligne. Données sources: Une recherche dans la banque de données Medline au moyen des termes dont la liste est incluse à la fin de cet article; la consultation d'experts dans ce domaine; une revue des recommandations publiées. Sélection des études: Il n'y avait pas d'article qui décrivait une étude randomisée, contrôlée, de dépistage comparé à l'absence de dépistage. On a donc choisi les études qui traitaient de la définition; de l'histoire naturelle; des interventions et des résultats incluant les effets négatifs possibles. Synthèse des données: Aucune synthèse systématique n'a été faite. La documentation a été remise à un groupe d'experts avant la Conférence canadienne de consensus sur la démence et les conclusions ont été ratifiées par consensus. Conclusions: 1. Il n'y a pas suffisamment de données pour recommander ou ne pas recommander de faire le dépistage de la dysfonction cognitive ou de la démence. (cote C); 2. Les plaintes concernant la mémoire devraient être évaluées et l'individu devrait être suivi pour en évaluer la progression. (cote B); 3. Quand les aidants ou les informants décrivent un déclin cognitif chez un individu, ces observations devraient être prises très au sérieux; une évaluation cognitive et un suivi attentif sont indiqués. (cote A)

Can. J. Neurol. Sci. 2001; 28: Suppl. 1 – S42-S51

Screening is a technique by which a manoeuvre to detect a specific condition is applied to an entire population at risk.<sup>1</sup> Case-finding identifies individuals who are at risk for, or who have, a specific condition, when the patient is being seen for another reason. For example, the physician may explore whether memory deficits may be the cause of nonadherence to a medication prescription. Diagnosis is the application of one or more tests to determine the pathological cause of a symptom or sign. For example, if an individual complains of memory loss the physician will investigate the cause. This article addresses screening in the primary care setting, consistent with the goals of the Canadian Consensus Conference on Dementia.<sup>2</sup>

Cognition is the process by which information is organized and reorganized.<sup>3</sup> Dementia is a syndrome of acquired cognitive loss which is sufficient to interfere with functioning in

occupational or social activities and which does not occur exclusively in the setting of clouded consciousness.<sup>2</sup> With the exception of pure vascular dementias, which may have a sudden onset, neurodegenerative dementias (most commonly Alzheimer's disease) are preceded by a preclinical state where cognitive deficits are present and early diagnosis is possible. Cognitive impairment is broadly defined by a state in which

From the Department of Medicine, McMaster University, Hamilton, ON (CJSP), Department of Family Medicine, Dalhousie University, Halifax, NS (DAG) Canada Reprint requests to: C. Patterson, Division of Geriatric Medicine, Hamilton Health Sciences Corporation, Chedoke Hospital Campus, Sanatorium Rd., Hamilton, Ontario L8N 3Z5 Canada.

deficits are detectable by testing in one or more domains of cognitive function but where these deficits do not impact on daily function of the individual. The individual may or may not be aware of these deficits. This general definition includes changes which normally accompany aging, those which are more extensive than occur during normal aging but progress slowly or not at all, and also those changes which represent the very earliest stages of dementing illnesses. If early detection is to be valuable, it is necessary to distinguish which of these three conditions exist.

Screening is justified when the following conditions are met.<sup>4</sup>

- 1. The condition is an important health problem (high prevalence and burden of illness).
- 2. The natural history of the condition is understood.
- 3. There is a recognizable presymptomatic phase.
- 4. An effective treatment is available, which is more beneficial when applied in the presymptomatic phase, than when treatment is delayed until symptoms appear.
- 5. A suitable test is available to detect the condition (high sensitivity, high specificity).
- 6. The test is acceptable to the population screened.
- The health care system has the capacity to apply the test and deal with the consequences.

#### AN IMPORTANT HEALTH PROBLEM

For all dementing conditions, both prevalence and incidence rise exponentially with age. For the Canadian population, the most appropriate data are derived from the Canadian Study of Health and Aging (CSHA).<sup>5</sup> For screening in primary care, the prevalence of dementia in institutions (about 50%) is of less importance than prevalence in the community. The community prevalence of dementia at different ages is shown in Table 1.5 The overall incidence of dementia among elderly Canadians is about 19 per 1,000 total population over age 65 per year.<sup>6</sup> The lowest incidence is 3.7 (95% CI 0.7-7.3) among men age 65-69 and the highest is 70.7 (95% CI 52.8-88.5) for men over aged 85. Incidence rates are higher among men age 70-79 than among women of the same age.6 While prevalence and incidence rates are similar to those reported from other countries, the CSHA is the most comprehensive population based study available to date.

A diagnosis of dementia implies difficulty in the performance of instrumental daily activities (eg. management of finances, driving, use of telephone, cooking) and when severe, interference with basic activities of daily living (eg. washing,

**Table 1:** Age-standardized prevalence of dementia in Canada in 1991, using the criteria of the Diagnostic and Statistical Manual (DSM)-III-R, by age-group and residence<sup>5</sup>

Community
1.6%
6.9%
17.8%
4.2%

Table 2: Prevalence of Cognitive Impairment Without Dementia

Age	Prevalence	Population	Criteria	
(years)	(%)			
50-64	15.8	Community, UK <sup>90</sup>	AAMI	
65-74	11.0	CSHA: Random Canada <sup>7</sup>	CIND	
75-84	24.0	CSHA: Random Canada <sup>7</sup>	CIND	
Over 85	30.3	CSHA: Random Canada 7	CIND	
Total over age 65	16.8 - 98	Various <sup>7,87</sup>	Various	

AAMI: Age Associated Memory Impairment (See Appendix) CIND: Cognitive Impairment Not Demented (See Appendix)

bathing, continence, mobility). The presence of dementia increases the risk of accidental injury (eg. falls, motor vehicle collisions). It is more difficult to estimate the burden of suffering from cognitive impairment as there are many different definitions for similar and overlapping conditions which fall under the rubric of cognitive impairment. (see Appendix) Depending upon definition, the prevalence of cognitive impairment in the absence of dementia has been estimated between 11% (Cognitive Impairment No Dementia - CIND: ages 65-74)<sup>7</sup> and 98% (memory impairment on at least one objective test)8 (Table 2). It is even more difficult to estimate the degree of suffering of individuals with cognitive impairment, as many do not recognize their impairment. While some authorities believe that memory complaints represent the earliest stages of dementia.9 others believe that complaints about cognitive problems, especially memory, are more prevalent in individuals who are not demented but who are anxious or depressed.<sup>10</sup> Twenty-two percent of a Dutch community sample of nondepressed, nondemented individuals reported memory complaints; the mean score of these individual on psychometric tests was lower than in noncomplainant individuals.11 In an English community study, 25% of individuals between 65 and 98 years had subjective memory impairment. Compared with those without subjective memory complaints, twice as many were depressed (26% versus 12%), more had dementia (7% versus 2%) or both depression and dementia (4% versus .6%). 12 In a case control study from Finland, those with memory complaints were more likely to display anxiety and negative feelings than those without. 13 Many individuals with dementing illnesses are not aware of, or deny, problems with memory or other aspects of cognitive dysfunction. Others are frustrated by their deficits and may complain of vague symptoms such as fatigue, dizziness and unsteadiness. Thus it is difficult to estimate burden of suffering in those with cognitive impairment without dementia.

# NATURAL HISTORY OF COGNITIVE IMPAIRMENT

While virtually every case of dementia is relentlessly progressive, the natural history of cognitive impairment is less clear, as the rate of progression depends upon the criteria applied to the inception cohort of longitudinal studies. Table 3 lists

Table 3: Progression of Cognitive Impairment to Dementia

Reference	Country	Population	N	Criteria I	Length of Follow-Up (Years)	Annual Rate of Progression (Percent)
Christensen <sup>14</sup> 1997	Australia	Community	N = 897;	36 with MCD	3.6	0.83
Hanninen <sup>91</sup> 1995	Finland	Random Population	N = 229;	AAMI	3.6	2.53 (3.5 ages 75-81)
O'Brien <sup>92</sup> 1992	UK	Community		Benign Senescent	3	3.0
				Forgetfulness		
Hogan <sup>26</sup> 2000	Canada	Community	N = 2914	CIND	5	8
Ritchie <sup>17</sup> 1996	France	Community	N = 283		2	7.5
Bowen <sup>93</sup> 1997	USA	НМО	N = 21	Isolated Memory L	oss 4	12.0
Petersen <sup>94</sup> 1997	USA	Memory Clinic	N = 76	MCI	4	12.0
Braekhus <sup>22</sup> 1995	Norway	Random Population	N = 215	27 MCI, MMSE 24	/25 3	13.3
Devenand <sup>15</sup> 1997	USA	Memory Clinic	N = 127,	Questionable Deme	entia 2.5	16.4

HMO = Health Maintenance Organisation; MCD = Mild Cognitive Disorder

representative longitudinal studies with diagnostic categories. Depending upon the category, the rate of progression from cognitive impairment to dementia is variously estimated at less than 1% <sup>14</sup> to greater than 16% per annum. <sup>15</sup> In each of these series a significant percentage, ranging from 10% <sup>13</sup> to 80% <sup>14</sup> of cognitively impaired individuals, appear to revert to normal cognition.

While there is no doubt that dementing disorders can be identified in a presymptomatic phase, there remains considerable debate in the literature concerning this "boundary between normal aging and very early Alzheimer's". 16,17 This subject has become an intense focus for research. While there is presently no consensus on the most appropriate definition of cognitive impairment, a relatively homogeneous form of amnestic mild cognitive impairment (MCI), defined by clinical characteristics, has a predictable 12% annual rate of conversion to dementia over the four years of longitudinal follow-up. 18 The characteristics of this type of MCI include 1) self-reported memory complaint, preferably corroborated by a family member; 2) a detectable memory deficit abnormal for age (about 1.5 SDs below the norm); 3) normal general cognitive functioning aside from memory; 4) ability to carry out such activities of daily living as driving a car and balancing a checkbook; and 5) absence of dementia. This type of MCI approximates to the condition of "circumscribed memory loss". In comparison, about 1% of a "normal" control population progress to dementia each year. These observations were obtained in a memory clinic at the Mayo clinic.

Identifying individuals with cognitive impairment who will progress to dementia is a major priority in cognitive research. Various neuropsychological measures, including tests of memory and verbal fluency, can identify individuals with an increased risk of subsequent dementia. In a series from Toronto, two tests, the Wechsler Memory Scale (Mental Control Subtest) and the Rey Auditory Verbal Learning Test for delayed recall predicted the development of Alzheimer's disease within two years with an accuracy of 90% (sensitivity 75%; specificity 94.9%). 19

Another combination of psychological tests which identifies

cognitively impaired individuals with an 85% risk of developing dementia in four years includes the delayed recall item from Buschke Selective Reminding Task, recall from the Fuld Objective Memory Evaluation, the Digit Symbol Test from the WAIS and a Verbal Fluency Scale. Neuropsychological testing requires special training and interpretation. Access to such testing is limited and generally costly to the individual unless insurance coverage is available, as psychological services are not generally a benefit of provincial health schemes.

Lower scores on simpler tests such as the Mini Mental State Examination (MMSE)<sup>21</sup> do increase the likelihood of subsequent decline.<sup>22</sup> The addition of the Clock Drawing Test also increases the ability to predict the decline.<sup>23</sup> While some series demonstrate an association between subjective memory complaints and increased risk of progression<sup>12,24,25</sup> other series do not.<sup>10</sup> When caregivers observe cognitive decline within the past year, the risk of progression is also increased.<sup>26</sup>

Several studies have indicated that individuals who possess the E4 allele of the ApoE gene are more likely to progress to dementia of the Alzheimer's type. <sup>27,28</sup> The accuracy of prognosis is greatly enhanced when delayed memory performance is included in the predictive model. <sup>29</sup> Numerous studies have established a relationship between the risk of dementia and educational achievement. Low reading ability, <sup>24</sup> low educational attainment, <sup>30,31</sup> and a smaller number of years of education, <sup>32</sup> were all associated with a higher risk of dementia. A mean of 5.3 years of education versus 13.0 years increased the relative risk of dementia by 2.02 (95% CI; 1.33-3.06). Low lifetime occupational achievement increased the relative risk of dementia by 2.87 (95% CI; 1.32-3.84) when compared with higher level occupations. <sup>32</sup>

In summary, many risk factors for progression of cognitive impairment to dementia have been identified. It is possible to determine which individuals have a higher risk of decline, by further testing with various neuropsychological instruments.

## EFFECT OF EARLY INTERVENTION

The detection of dementia will usually prompt some or all of the following responses from physicians:

## a) Investigation of possible cause and potential for reversal

- Review of history for risk factors such as excessive alcohol consumption, head injury, exposure to heavy metals, drugs or other toxins.
- Physical and neurological examination, which may disclose systemic disease, or focal neurological signs leading to a specific diagnosis.
- iii) Laboratory tests for the detection of metabolic disturbances that may contribute to the cognitive deficits such as hypercalcemia, hypothyroidism, pernicious anemia, renal failure, hepatic failure and other conditions which may be remediable. A minimum of complete blood count, blood glucose, serum calcium, electrolytes and thyroid function are recommended.<sup>2</sup>
- iv) Neuroimaging procedures such as CT or MRI scanning to detect structural abnormalities where indicated.<sup>2</sup>
- v) Detailed neuropsychometric evaluation to detect areas of deficit and preserved function.
- vi)Other investigations as indicated.

While it was once believed that about 1/3 of cases of dementia had "reversible" causes, it is now evident that many conditions may aggravate cognitive deficits but true "reversibility" is very uncommon.<sup>33</sup>

While some or all of the above measures may be considered for individuals identified with dementia, their utility in evaluating the individual with cognitive impairment is unknown.

### b) Social and educational manoeuvres

For individuals identified with dementia, education concerning the nature of cognitive deficits, prognosis and implications may be beneficial. For example, it may spur an individual to formulate advance directives, choose a Power of Attorney for financial and personal care decision making and contemplate issues such as motor vehicle driving and relocation for the future. It could be argued that knowledge of the condition and its prognosis could be of value to the caregiver. Educational interventions for caregivers of demented individuals improve quality of life and delay the necessity for institutional care. 34 Advocacy groups such as the Alzheimer's Society provide valuable support for the caregivers of those with dementia. While these interventions are of proven benefit in individuals with established dementia, their role for individuals with cognitive impairment or their caregivers is unknown. In a British study, relatives did not show any curiosity and seemed puzzled by the attention paid to cognitive deficits discovered in individuals.<sup>35</sup>

# c) Potential pharmacological interventions

Agents with proven efficacy in delaying the progression of cognitive impairment or dementia would provide a rationale for early detection. Large scale randomized controlled trials are currently underway to evaluate the effects of antioxidants, Cox-II inhibitors, cholinesterase inhibitors and other agents. As yet there are no published results from these studies.

Although there is circumstantial evidence from case control studies that estrogenic hormones may protect individuals from dementia, the only published randomized controlled trial of estrogens in early dementia failed to show any benefit in terms of delayed progression.<sup>36</sup> Arandomized controlled trial of estrogens for individuals with cognitive impairment and a high risk of developing Alzheimer's disease is currently underway. Three

older studies of pharmacological interventions in age-associated memory impairment (AAMI) examined the effects of phosphatidylserine and guanfacine; these were short term and did not examine progression to dementia as an outcome measure.<sup>37-39</sup>

There are two cholinesterase inhibitors approved for use in Canada for mild to moderate Alzheimer's disease. Donepezil was the first to be approved<sup>40,41</sup> and, more recently, rivastigimine.<sup>42,43</sup> Both of these drugs produce cognitive improvements that are generally modest, although occasionally substantial clinical improvement occurs. At the conclusion of a six month placebo controlled randomized trial of donepezil, the mean difference in score on the MMSE, (a secondary outcome measure) was 1.36 points in favour of donepezil. The relevance of this observation is that a recently published study of the costs of caring for individuals with Alzheimer's disease (using data from the CSHA) revealed that a one point decline in the MMSE is associated with an estimated cost increase of \$1,343 per person per year.<sup>44</sup> This raises the possibility that therapy with cholinesterase inhibitors may be cost effective.

There are theoretical reasons to believe that antioxidants may be beneficial for retarding the progression of neurodegenerative diseases. A randomized controlled trial of vitamin E or selegiline appeared to delay the progression of established Alzheimer's disease, although there were several methodological flaws in the study.<sup>45</sup>

In summary, there are effective strategies for managing individuals with established dementia with both supportive and pharmacological interventions. However, the value of these interventions in individuals with cognitive impairment who are not demented, or those with dementia discovered by screening, remains to be established.

## **DETECTION MANOEUVRES**

There are four candidate detection manoeuvres. The first is to inquire of the individual whether memory complaints or cognitive problems are present. Surveys have revealed a high prevalence (22-76%) of subjective memory complaints in older individuals. Whether these complaints predict further deterioration is not clear. While some studies have indicated that memory complaints in the presence of cognitive impairment increased the likelihood of subsequent dementia, 9.25 others do not. 10,19,46 Inquiring about memory complaints is of uncertain value in screening for cognitive impairment. Memory complaints were an essential part of the criteria for MCI in the Mayo Clinic longitudinal study. 18

The second approach is to use informant description. This may be formalized in an instrument such as the Informant Questionnaire on Cognitive Decline in the Elderly.<sup>47</sup> This 24 item questionnaire has the advantage of using the informant's knowledge of the individual to detect change and takes into account social and educational background. <sup>48,49</sup> In a recent meta analysis of several studies of an informant questionnaire, Jorm and colleagues calculated a mean sensitivity of 86% and a mean specificity of 80% for the instrument to detect cognitive decline in the elderly. The likelihood ratio for a positive test was 4.3.<sup>50</sup> Informant description of decline (in response to a single question) also contributed to future risk of cognitive decline in the CSHApopulation.<sup>26</sup>

The inability of an individual to perform instrumental and

basic activities of life is strongly correlated with the presence of dementia. This may be valuable for detection of individuals with dementia. In a large community study in France, it was discovered that the constellation of inability to use the telephone, use transportation, handle medications and manage finances had a sensitivity of 94% and a specificity of 71% for the diagnosis of dementia. The likelihood ratio of this constellation was 3.2.<sup>51</sup> However, by definition, individuals with cognitive impairment alone do not have difficulty with these activities.

Another approach is a mental state examination designed to detect cognitive impairment or dementia. In order to be acceptable to primary care physicians, tests need to be brief and easily applied. The best studied instrument is the 30 item MMSE.<sup>21</sup> This has an average sensitivity of 83% and average specificity of 82% against the standard of clinical diagnosis of dementia.<sup>52</sup> The sensitivity rises with the severity of dementia and falls with less severe degrees of dementia or cognitive impairment. Age, formal education, level of intelligence and, in some series, ethnic background, influence the score and "corrected" norms have been suggested. 53-55 Most of the test characteristics of the MMSE have been calculated using data from memory clinics and referred patients. When the MMSE has been used for screening in community surveys and populations similar to those found in primary care, the sensitivity for detection of dementia is lower. At a cut point of 23 the sensitivity was only 69% in primary care practices in Rochester, USA.<sup>56</sup> Various attempts have been made to reduce the number of items on the MMSE to limit administration time without sacrificing sensitivity.<sup>57-59</sup> A survey from Holland revealed that MMSE was no more useful in distinguishing mild from severe dementia than knowledge of the date, current address and present Prime Minister.<sup>57</sup> Because of the influence of education and level of intelligence, short mental status questionnaires will tend to mislabel those without dementia who are of low intelligence or low socioeconomic background but are relatively insensitive for dementia in those of high intelligence or socioeconomic status. Given the most optimistic test characteristics of an instrument such as the MMSE, (ie. sensitivity 83%, specificity 82%) and a community prevalence of dementia of 1.6% age 65-74, 6.9% age 75-84 and 17.8% over age 85, the false positive rates (ie. risk of falsely labeling an individual with dementia) are 93%, 75% and 50% respectively. Application of "corrected" cut off points on the MMSE, taking into account educational level, did not improve sensitivity of the instrument when applied to a community population in Italy. In fact, at a cut point of 23 the sensitivity fell from 85.7% to 71.4% with a corresponding increase in specificity from 90 to 96.3%.60 For detection of cognitive impairment without dementia, the performance of short mental status questionnaires is potentially even more misleading. A meta analysis of other cognitive screening tests (MMSE, Mattis Dementia Rating Scale, Short Portable Mental State Questionnaire and Blessed Dementia Scale) revealed similar characteristics for discriminating between people with or without dementia.<sup>61</sup> Interestingly, brief screening tests performed as well as more specialized psychometric tests for detecting dementia. 62 Despite these reservations, the MMSE is considered the best of the short mental status instruments for screening cognitive disorders and is recommended by the American Neuropsychiatric Association.<sup>63</sup>

#### RESULTS OF SCREENING STUDIES

Screening of all 222 patients over the age of 70 years in a rural practice in Newfoundland, using the Canadian Mental Status Questionnaire<sup>64</sup> revealed that all of the five community dwelling patients with severe dementia were known to the physicians. However, none of the 15 patients with moderate impairment, as identified by this questionnaire, had been recognized by the physicians as impaired. The prevalence of severe cognitive dysfunction was 2.5% and of moderate cognitive dysfunction was 6.8%. <sup>65</sup>

A similar 10 item questionnaire, the Short Portable Mental Status Questionnaire<sup>66</sup> was used to screen all patients over age 60 in a general medical practice in Indianapolis. Of 3954 patients, 5.2% had moderate to severe cognitive impairment and 10.5% had mild impairment. Dementia had been recorded as a diagnosis for less than 25% of patients with moderate to severe cognitive impairment, although they were more likely to have been evaluated for reversible causes. Although the mortality and health service utilization was increased in cognitively impaired individuals, it is not clear whether their outcome could have been changed by earlier recognition of their impairments.<sup>67</sup>

In Boston, USA, 472 individuals were discovered to have cognitive impairment in a population survey of 3,624 people over age 65. Diagnostic evaluation did not lead to the detection of any "reversible" dementing disorders. Of the 472 individuals, 83.5% were given a diagnosis of probable Alzheimer's disease.<sup>68</sup>

In a three phase study in Eastern Baltimore, USA, 78% of 3,481 subjects completed the National Institute of Mental Health Interview Survey Questionnaire together with a version of the MMSE. Eighty percent of a random sample of these subjects (N=1806) were examined by psychiatrists. Thirty-six of the 44 diagnosed by a psychiatrist as having definite or probable dementia were subjected to full neurological investigations. The prevalence of dementia was 6.1% in this population and no cases of "reversible" dementia were found. <sup>69</sup>

The CSHArecruited a representative sample of people age 65 or over, drawn from 36 urban and surrounding rural areas in the 10 Canadian provinces.<sup>5</sup> The study involved 9,008 people from the community and 1,255 from institutions. As part of the screening battery, the Modified Mini Mental State Examination (3MS)<sup>70</sup> was used to identify individuals with cognitive impairment or dementia. The cut point was less than 78, equivalent to a score of 24 on the MMSE. Using this instrument, 24.8% (8.0% with dementia; 16.8% with CIND) scored below the cut point.<sup>7</sup> Of the CIND group, 15.2% fulfilled criteria for mild cognitive impairment ICD-10 type II; 5.2% MCI-ICD-10 type III; 1.2% MCI-ICD-10 type I; 13% fulfilled criteria for age associated cognitive decline, 1.2% age associated memory impairment, 0.9% age consistent memory impairment, 0.3% late life forgetfulness; 6.4% DSM-III-R type II and 0.06% DSM-III-R type I.71 Common etiologies for subcategories of CIND included cerebrovascular disease (9.85%); depression (8.0%); "general vascular" (7.5%); psychiatric (6.6%); alcohol abuse (5.1%); mental retardation (2.3%) and others.<sup>71</sup> Thus, screening a population with instruments such as the MMSE is likely to identify about one quarter of the older population as demented or cognitively impaired. The five year mortality rate was significantly higher in all those with CIND than in the normal cognitive group, 48.4% versus 30.4% (p< 0.0001). Of those initially categorized with CIND, 42.1% developed dementia after five years, compared with 14.7% of those categorized as cognitively normal, p<0.0001.<sup>26</sup> As would be expected, those with any of the MCI diagnoses, late life forgetfulness or age associated cognitive decline had the worst prognosis. Nearly 50% of those with MCI (ICD-10-type III; DSM-III-R- type II and late life forgetfulness) developed dementia after five years. About 20% of those with age associated memory impairment or age consistent memory impairment developed dementia.

Screening populations such as the CSHA with short mental status questionnaires identifies a group which has worse outcomes in terms of mortality and progression to dementia. These findings are confirmed by other studies: In a 20 year follow-up study from the UK, lower scores on mental tests were associated with an increase in all cause mortality.<sup>72</sup> In a general practice in the USA lower scores on the Short Portable Mental Status Questionnaire were associated with an increased number of visits to the emergency room, increased risk of hospitalization and a higher all cause mortality (8.2% in the cognitively impaired/demented compared with 2.8% of those scoring in the normal range) in the year following testing.<sup>67</sup>

Simply identifying an at risk population is not sufficient. Further investigation (including neuropsychological testing) is necessary to identify those with CIND who have conditions such as depression, mental retardation, and distinguish those who have mild cognitive impairment, early dementia or who are cognitively "normal".

Using CSHAdata, a formula has been derived which predicts progression to dementia with data that are readily available to the family physician. The equation is  $(100\text{-}(MMSE/30 \times 100) + .25 \times age) + 10$ , (if memory problems were reported by an informant). This formula has a sensitivity of 72.9% and a specificity of 67.7% for predicting cognitive decline. <sup>26</sup>

Thus, of all the potential screening methods, a short mental status questionnaire appears most promising. The MMSE has been best studied and has a sensitivity of 69-82%, depending upon severity of dementia, level of education, socioeconomic status and population studied.

It should be noted that, while there is significant overlap between the different categories of cognitive impairment, even the diagnosis of dementia is highly dependent upon diagnostic criteria. For example, applying different criteria to the same population (CSHA) the prevalence of dementia was as low as 3.1% (using criteria of ICD-10) to 29.1% (with DSM-III criteria).<sup>73</sup> Even the "gold standard" is clearly less than ideal. The earlier that diagnosis is attempted, the less likely it is to be accurate.

## ACCEPTABILITY TO POPULATION

In general, while mental status questionnaires are readily performed by older individuals providing their purposes are explained, some individuals resent the intrusion. There may be risks of harm to the individual. Labeling an individual with cognitive impairment or dementia may cause distress and illness behaviour.<sup>74</sup> Diagnosing a condition which has the potential to progress to a disease for which there is no cure, (although some symptomatic treatments are now available) is not likely to alleviate anxiety. Attitudes of health professionals, business professionals and lay people have been documented to have

substantial negative impact on the older individual with dementia.<sup>75</sup> Whether the same pertains to individuals labeled with cognitive impairment without dementia is not known, however the potential exists for such harm.

#### CAPACITY OF SYSTEM TO ACCOMMODATE SCREENING

The first issue is whether physicians will utilize screening manoeuvres. It is well-known that the uptake by physicians of screening recommendations provided by the Canadian Task Force on Preventive Health Care is poor. 76 A survey of primary care physicians in the Ottawa-Carleton region of Canada revealed that 82% of physicians believed that cognitive screening was needed but only 24% routinely screened their patients.<sup>77</sup> Physicians at the Mayo Clinic considered the MMSE of little value for routine screening but was useful as a clinical test.<sup>78</sup> While not strictly comparable, when the results of screening for functional deficits were revealed or concealed to physicians by random allocation, there was no change in physician behaviour or patient outcome.<sup>79</sup> If one quarter of the population are discovered to have cognitive deficits by routine screening, the added consumption of health care resources would include additional investigations, consultations, and numerous referrals for neuropsychological testing (which would impose a financial burden on individuals identified). Potential benefits would include the ability of individuals and their caregivers to plan ahead, and the possibility of therapeutic interventions. Balanced against this is the risk of mislabeling significant numbers of individuals, thereby incurring significant costs in additional and sometimes unnecessary investigations. Labeling of individuals with an unpleasant diagnosis is another potential harm.

## DISCUSSION

While cognitive impairment is common in older individuals, it probably does not result in a great burden of suffering. The natural history is becoming more clear and relatively homogeneous categories, such as MCI, have a predictable rate of conversion to dementia. A screening test, such as MMSE will identify 25% of the older population with CIND or dementia, a group which has a higher mortality and morbidity. Whether earlier identification offers any advantage over waiting until a dementing illness becomes clinically detectable, remains a high priority for research. The strongest argument against screening is that the available detection manoeuvres are inaccurate and run the risk of falsely labeling many individuals who do not have a problem, while failing to detect those (especially better educated or highly intelligent individuals) who may be having cognitive difficulties. Instruments to detect subtle degrees of cognitive impairment are available but are not practical for widespread use in primary care screening.

Despite the introduction of agents which have some value in treating symptomatic Alzheimer's disease, potential preventive strategies (such as estrogenic hormone replacement; treatment of vascular risk factors) have not been demonstrated to change clinical outcome. Decisions to address vascular risk factors or estrogen replacement are unlikely to be influenced by the presence or absence of cognitive impairment. The prudent physician will remain alert for clues which suggest cognitive

decline and be particularly attentive to observations by relatives and caregivers which suggest cognitive decline in their patients.

A stronger, but as yet unproven, argument can be made for screening older individuals for dementia. The presence of dementia, regardless of cause, signifies increased morbidity and mortality. Risks to the individual (eg. accidental injuries) and stress to the caregiver increase significantly as the disease progresses. Furthermore, the financial burden (including medications, formal and informal caregiving and, most importantly, costs of institutional care) rises with the severity of the condition. Earlier detection of dementia leading to a specific diagnosis of Alzheimer's could result in timely prescription of symptomatic medications such as donepezil or rivastigmine. For those individuals with dementia responsive to medications, one could anticipate potentially reduced caregiver stress and possibly delayed admission for institutional care. This could result in improved quality of life for the affected individual and reduced financial burden of care to society. On the other hand, delayed admission to institutional care imposes a greater burden on caregivers. The case for screening becomes more persuasive in older individuals, as the prevalence of dementia rises.

The relatively low sensitivity of short cognitive tests, such as the MMSE, limit the use of this instrument for dementia screening in primary care. Some characteristics of informant description make this an appealing alternative approach to screening. Inquiry about the ability of the individual to perform instrumental activities of daily living is also an attractive screening manoeuvre, as, in addition to its value for detecting dementing disorders, it has utility in detecting physical problems as well. However, lack of prospective trials describing the sequence of detection, investigation, intervention and outcome prevents a firm recommendation for any type of screening. Rather, a high index of suspicion in older individuals, especially those over the age of 85 where community prevalence of dementia approaches 20%, and careful attention to caregivers' descriptions are supported by a fair level of evidence.

Prospective studies to determine the outcome and opportunities for intervention of individuals screened for dementia, are a high research priority.

## RECOMMENDATIONS

- 1. There is insufficient evidence to recommend for, or against, screening for cognitive impairment in the absence of dementia (*C: level II-ii evidence*).
- 2. Memory complaints should be evaluated and the individual followed to assess progression (*B: level II-ii evidence*).
- When caregivers or informants describe cognitive decline in an individual, these observations should be taken very seriously: cognitive assessment and careful follow-up are indicated (A: level II-ii evidence).

#### Validation

The US Preventive Services Task Force offered a similar recommendation in 1996.<sup>80</sup> The Canadian Task Force on the Periodic Health Examination made a similar recommendation in 1994.<sup>81</sup>

The Task Force on Health Assessment of the Society of General Internal Medicine recommended inclusion of Screening for Cognitive Impairment in 1989. 82

An earlier draft of this manuscript, together with the recommendations was approved by the Canadian Task Force on Preventive Health Care in 1999.

#### APPENDIX: Types of Cognitive Impairment

## Mild cognitive impairment (Derived from DSM-III-R)<sup>83</sup>

- *Type 1.* Short- and long-term memory impairment only, with no functional disabilities.
- Type 2. Short- and long-term memory impairment, no functional disabilities, and at least one of the following: impairment in abstract thinking, impaired judgment, disturbance of higher cortical function (eg. aphasia, apraxia, agnosia), or personality change.

## Mild cognitive impairment (Derived from ICD-10)84

- *Type 1.* Short- or long-term memory impairment only, with no functional disabilities.
- Type 2. Short- or long-term memory impairment and a decline in intellectual abilities with no functional disabilities.
- Type 3. Short- or long-term memory impairment with a decline in intellectual abilities, a personality change, and with no functional disabilities.

## Age-associated memory impairment (AAMI)85-87

Subjective memory complaints with gradual onset in individuals who were at least 50 years of age with adequate intellectual function and who scored 24 or higher on the 3MS and at least 1 SD below the mean established for young adults on a standardized test for recent memory.

# Modifications of AAMI<sup>88</sup>

Perceived decreases in day-to-day memory functioning in 50-to 79-year-old individuals were verified by a standardized self-report memory questionnaire. Individuals required verbal and performance IQ scores between 90 and 130. The following three subcategories are defined according to performance on memory tests relative to established norms:

## (a) Age-associated memory impairment

Performance at least 1 SD below the mean established for young adults on one or more tests. This category includes individuals whose performance on a memory test was above average relative to age norms.

# (b) Age-consistent memory impairment (ACMI)

Performance within + 1 SD of the mean established for age on 75% or more of the tests that were administered.

### (c) Late-life forgetfulness (LLF)

Performance between 1 and 2 SDs below the mean established for age on 50% or more of the tests that were administered.

## (d) Age-associated cognitive decline (AACD)

Gradual decline in any one cognitive area that was present for at least six months and performance at least 1 SD below norms for age on relevant neuropsychological tests.

## Exclusion criteria

All criteria required exclusion of subjects with depression,

delirium, mental retardation, or other psychiatric disorders. The proposed versions of AAMI excluded individuals with functional impairment.

Other descriptions which relate to, or overlap with AAMI include Age-Related Cognitive Decline (ARCD), defined in DSM-IV as "an objectively identified decline in cognitive functioning consequent to the aging process that is within normal limits given the person's age". Mild Cognitive Disorder (MCD) is a term used by the World Health Organization and requires: objective evidence or history of cerebral dysfunction; cognitive dysfunction reported by self or reliable informant; and abnormality on psychological tests. This disorder is not specific to aging. The term Benign Senescent Forgetfulness (BSF) was used to describe a nonprogressive state, characterized by mild cognitive deficits, although this term has generally fallen out of favour. Sequences of the complex of the control of the complex of the complex

Cognitive Impairment, Not Demented (CIND) score less than 78 on 3MS; dementia excluded; includes various categories of cognitive impairment, depression, mental retardation, etc.<sup>7</sup>

#### SEARCH STRATEGY

#### 1987-1997

Memory disorders (mh) [Diagnosis: Prevention & Control]

### 1993 - Dec. 1997

("cognition disorders" [MESH] AND "geriatric assessment" [MESH] AND "mass screening" [MESH])

(("geriatric assessment" [MESH] AND "mass screening" [MESH] AND (cognition [TEXT] OR cognitive [TEXT])) AND (("randomized controlled trials" [MESH] OR "comparative studies" [MESH]) OR "cohort studies: [MESH]) OR "case-control studies" [MESH]))

(("cognition disorders" [MESH] AND "geriatric assessment" [MESH]) AND ((("randomized controlled trials" [MESH] OR "comparative studies" [MESH]) OR "cohort studies" [MESH]) OR "case-control studies" [MESH]))

(("cognition disorders" [MESH] AND "aged" [MESH] AND "mass screening" [MESH]) AND "clinical trial" [PTYP] OR "meta-analysis" [PTYP] OR "randomized controlled trials" [MESH] OR "comparative studies" [MESH] OR "cohort studies" [MESH] OR "case-control studies" [MESH]))

"Related Articles" search on 1991 CTFPHE CMAJ update on screening for cognitive impairment, sorted by visual scan.

#### Dec. 1997 - May 2000

("Cognition disorders" [MESH] and "Aged" [MESH]) Handsorted for last 2 months

# 1966 - Sept. 2000

("mini mental state examination" [TEXT] AND "sensitivity and specificity" [TEXT])

#### REFERENCES

- Whitby L. Screening for disease definitions and criteria. Lancet 1974; ii: 819-821.
- Patterson C, Gauthier S, Bergman H, et al. The recognition, assessment and management of dementing disorders: conclusions from the Canadian Consensus Conference on Dementia. Can

- Med Assoc J 1999; 160 (Suppl 12): S1-S16.
- Lezak MD. Neuropsychological Assessment. New York. Oxford University Press. 1976;14.
- Wilson JMĆ, Jungner G. Principles and Practice of Screening for Disease. Public Health Paper. No. 34. WHO. Geneva. 1968.
- Canadian Study of Health & Aging Working Group. Canadian study of health & aging: study methods and prevalence of dementia. Can Med Assoc J 1994; 150(6): 899-913.
- Canadian Study of Health & Aging Working Group. The incidence of dementia in Canada. Neurology 2000; 55: 66-73.
- Graham JE, Rockwood K, Beattie B, et al. Prevalence and severity of cognitive impairment with and without dementia in an elderly population. Lancet 1997; 349: 1793-1796.
- Small G, Ivnik RJ, Petersen RC, et al. Age-associated memory impairment diagnoses: problems of reliability and concerns for terminology. Psychol Aging 1991; 6: 551-558.
- Reisberg B, Ferris S, DeLeon M, Crook T. The Global Deterioration Scale for assessment of primary degenerative dementia. Am J Psychiatry 1982; 139: 1136-1139.
- Jorm AF, Christensen H, Korten A, et al. Do cognitive complaints either predict future cognitive decline or reflect past cognitive decline? A longitudinal study of an elderly community sample. Psychol Med 1997; 27: 91-98.
- Jonker C, Launer LJ, Hooijer C, Lindeboom J. Memory complaints and memory impairment in older individuals. J Am Geriatr Soc 1996; 44: 44-49.
- Tobiansky R, Blizard R, Livingston G, Mann A. The Gospel Oak Study Stage IV: the clinical relevance of subjective memory impairment in older people. Psychol Med 1995; 25: 779-786.
- Hänninen T, Reinikainen KJ, Helkala E-L, et al. Subjective memory complaints and personality traits in normal elderly subjects. J Am Geriatr Soc 1994; 42: 1-4.
- Christensen H, Henderson AS, Korten AE, et al. ICD-10 Mild cognitive disorder: its outcome three years later. Int J Geriatr Psychiatr 1997; 12: 581-586.
- Devenand DP, Folz M, Gorlyn M, Moeller JR, Stern Y. Questionable dementia: clinical course and predictors of outcome. J Am Geriatr Soc 1997; 45: 321-328.
- Petersen RC. Mild cognitive impairment or questionable dementia? Arch Neurol 2000; 57: 643-644.
- Ritchie K, Leibovici D, Ledésert B, Touchon J. A typology of subclinical senescent cognitive disorder. Br J Psychiatr 1996; 168: 470-476
- Petersen RC, Smith GE, Waring SC, et al. Mild cognitive impairment: clinical characterization and outcome. Arch Neurol 1999; 56: 303-308.
- Tierney MC, Szalai JP, Snow WG, Fisher RH. The prediction of Alzheimer's disease. Arch Neurol 1996; 53: 423-427.
- Masur DM, Sliwinski M, Lipton RB, Blau AD, Crystal HA. Neuropsychological prediction of dementia and the absence of dementia in healthy elderly persons. Neurology 1994; 44: 1427-1432.
- Folstein MF, Folstein SE, McHugh PR. Mini-Mental State: a practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975; 12: 189-198.
- Braekus A, Laake K, Engedal K. A low, "normal" score on the Mini-Mental State Examination predicts development of dementia after three years. J Am Geriatr Soc 1995; 43: 656-661.
- 23. Ferrucci L, Cecchi F, Guralnik JM, et al for the FINE Study Group. Does the clock drawing test predict cognitive decline in older persons independent of the Mini-Mental State Examination? J Am Geriatr Soc 1996; 44: 1326-1331.
- Schmand B, Jonker C, Hooijer C, Lindeboom J. Subjective memory complaints may announce dementia. Neurology 1996; 46: 121-125
- Schofield PW, Marder K, Dooneief G, et al. Association of subjective memory complaints with subsequent cognitive decline in community-dwelling elderly individuals with baseline cognitive impairment. Am J Psychiatry 1997; 154: 609-615.
- Hogan DB, Ebly EM. Predicting who will develop dementia in a cohort of Canadian seniors. Can J Neurol Sci 2000; 27: 18-24.
- 27. Petersen RC, Smith GE, Ivnik RJ, et al. Apolipoprotein E status as

- a predictor of the development of Alzheimer's disease in memory-impaired individuals. JAMA1995; 273: 1274-1278.
- Tierney MC, Szalai JP, Snow WG, et al. A prospective study of the clinical utility of ApoE genotype in the prediction of outcome in patients with memory impairment. Neurology 1996; 46: 149-154.
- Tierney MC, Szalai JP, Snow WG, et al. Prediction of probable Alzheimer's disease in memory-impaired patients: a prospective longitudinal study. Neurology 1996; 46: 661-665.
- Small GW, La Rue A, Komo S, Kaplan A, Mandelkern MA. Predictors of cognitive change in middle-aged and older adults with memory loss. Am J Psychiatry 1995; 152: 1757-1764.
- Jorm AF, Christensen H, Henderson AS, et al. Cognitive complaints of cognitive decline in the elderly: a comparison of reports by subjects and informants in a community survey. Psychol Med 1994; 24: 365-374.
- Stern Y, Gurland B, Tatemichi TK, et al. Influence of education and occupation on the incidence of Alzheimer's disease. JAMA1994; 271: 1004-1010.
- Clarfield AM. The reversible dementias: do they reverse? Ann Intern Med 1988; 109: 476-486.
- Mittelman MS, Ferris SH, Shulman E, Steinberg G, Levin B. A family intervention to delay nursing home placement of patients with Alzheimer 'sdisease: a randomized controlled trial. JAMA 1996; 276: 1725-1731.
- O'Connor DW, Pollitt PA, Brook CPB, Reiss BB, Roth M. Does early intervention reduce the number of elderly people with dementia admitted to institutions for long term care? BMJ 1991; 302: 871-875.
- Mulnard RA, Cotman CW, Kawas C, et al. Estrogen replacement therapy for treatment of mild to moderate Alzheimer 'sdisease: a randomized controlled trial. Alzheimer's Disease Cooperative Study. JAMA2000; 283(8): 1007-1015.
- Crook TH, Tinklenberg J, Yesavage J, et al. Effects of phosphatidyl serine in age-associated memory impairment. Neurology 1991; 41: 644-649
- Israel L, Melac M, Milinkevitch D, et al. Drug therapy and memory training programs: a double-blind randomized trial of general geriatric patients with age-associated memory impairment. Int Psychogeriatr 1994; 6: 155-170.
- McEntee WJ, Crook TH, Jenkyn LR, et al. Treatment of ageassociated memory impairment with guanfacine. Psychopharm Bull 1991; 27: 41-46.
- Rogers SL, Friedhoff LT and the Donepezil Study Group. The efficacy and safety of donepezil in patients with Alzheimer's disease: results of a US multicenter, randomized double-blind, placebo-controlled trial. Dementia 1996; 7: 293-303.
- Rogers SL, Farlow MR, Doody RS, Mohs R, Friedhoff LT and the Donepezil Study Group. A 24-week, double-blind, placebocontrolled trial of donepezil in patients with Alzheimer's Disease. Neurology 1998; 50: 136-145.
- 42. Rösler M, Anand R, Cicin-Sain A, et al on behalf of the B303 Exelon Study Group. Efficacy and safety of rivastigmine in patients with Alzheimer's disease: international randomized controlled trial. Br Med J 1999; 318: 633-640.
- Nordberg A, Svensson A-L. Cholinesterase inhibitors in the treatment of Alzheimer's disease. A comparison of tolerability and pharmacology. Drug Safety 1998; 19(6): 465-480.
- Hux MJ, O'Brien BJ, Iskedjian M, et al. Relation between severity of Alzheimer's disease and costs of caring. Can Med Assoc J 1998; 159: 457-465.
- 45. Sano M, Ernesto C, Thomas RG, et al. A controlled trial of selegiline, alpha tocopherol or both as treatment for Alzheimer's disease. The Alzheimer's Disease Cooperative Study. N Eng J Med 1997; 336: 1216-1222.
- Flicker L, Logiudice D, Carlin JB, Ames D. The predictive value of dementia screening instruments in clinical populations. Int J Geriat Psychiatry 1997; 12: 203-209.
- Jorm AF, Jacomb PA. The informant questionnaire on cognitive decline in the elderly. (IQCODE). Socio-demographic correlates, reliability, validity, and some norms. Psychol Med 1989; 19: 1015-1022.
- 48. Jorm AF. Assessment of cognitive impairment and dementia using

- informant reports. Clin Psychol Rev 1996; 16.
- Jorm AF, Christensen H, Henderson AS, et al. Informant ratings of cognitive decline of elderly people: relationship to longitudinal change on cognitive tests. Age Ageing 1996; 25: 125-129.
- Jorm AF. Methods of screening for dementia: a meta-analysis of studies comparing an informant questionnaire with a brief cognitive test. Alzheimer Dis Assoc Disord 1997; 11: 158-182.
- Barberger-Gateau P, Commenges D, Gagnon M, et al. Instrumental activities of daily living as a screening tool for cognitive impairment and dementia in elderly community dwellers. J Am Geriatr Soc 1992; 40: 1129-1134.
- 52. Tombaugh TN, McIntyre NJ. The Mini-Mental State Examination: a comprehensive review. J Am Geriatr Soc 1992; 40: 922-935.
- O'Connor DW, Pollitt PA, Treasure FP, Brook CPB, Reiss BB. The influence of education, social class, and sex on Mini-Mental State Scores. Psychol Med 1989; 19: 771-776.
- Kittner SJ, White LR, Farmer ME, et al. Methodological issues in screening for dementia: the problem of education adjustment. J Chronic Dis 1986; 39: 163-170.
- Bleeker ML, Bolla-Wilson K, Kawas C, Agnew J. Age-specific norms for the Mini-Mental State Examination. Neurology 1988; 38: 1565-1568.
- Tangalos EG, Smith GE, Ivnik RJ, et al. The Mini-Mental State Examination in general medical practice: clinical utility and acceptance. Mayo Clin Proc 1996; 71: 829-837.
- 57. Wind AW, Schellevis FG, Van Staveren G, et al. Limitations of the Mini-Mental State Examination in diagnosing dementia in general practice. Int J Geriat Psychiatry 1997; 12: 101-108.
- Wells JC, Keyl PM, Chase GA, et al. Discriminant validity of a reduced set of Mini-Mental State Examination items for dementia and Alzheimer's disease. Acta Psychiatr Scand 1992; 86: 23-31.
- Feher EP, Mahurin RK, Doody RS, et al. Establishing the limits of the Mini-Mental State. Examination of "Subtests". Arch Neurol 1992; 49: 87-92.
- Cossa FM, Sala SD, Musicco M, Spinnler H, Ubezio MC. Comparison of two scoring systems of the Mini-Mental State Examination as a screening test for dementia. J Clin Epidemiol 1997; 50(8): 961-965.
- Christensen H, Hadzi-Pavlovic D, Jacomb P. The psychometric differentiation of dementia from normal aging. Psychol Assess 1991; 3: 147-155.
- 62. Stuss DT, Meiran N, Guzman DA, Lafleche G, Willmer J. Do long tests yield a more accurate diagnosis of dementia than short tests? A comparison of 5 neuropsychological tests. Arch Neurol 1996 Oct; 53(10): 1033-1039.
- Malloy PF, Cummings JL, Coffey CE, et al. Cognitive screening instruments in neuropsychiatry: a report of the committee on research of the American Neuropsychiatry Association. J Neuropsychiatry Clin Neurosci 1997; 9(2): 189-197.
- Robertson D, Rockwood K, Stolee P. A short mental status questionnaire. Can J Aging 1982; 1: 16-20.
- Worral G, Moulton N. Cognitive function: survey of elderly persons living at home in rural Newfoundland. Can Fam Physician 1993; 39: 772-777.
- Pfeiffer E. A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. J Am Geriatr Soc 1975; 23: 433-441.
- Callahan CM, Hendrie HC, Tierney WM. Documentation and evaluation of cognitive impairment in elderly primary care patients. Ann Intern Med 1995; 122: 422-429.
- Evans DA, Funkenstein HH, Albert MS, et al. Prevalence of Alzheimer's disease in a community population of older persons. JAMA1989; 262: 2551-2556.
- Folstein MF, Anthony JC, Parhad I, et al. The meaning of cognitive impairment in the elderly. J Am Geriatr Soc 1985; 33: 228-235.
- Teng EL, Chui HC. The Modified Mini-Mental State (3 MS) Examination. J Clin Psychiatry 1987; 48: 314-318.
- Ebly EM, Hogan DB, Parhad IM. Cognitive impairment in the nondemented elderly. Arch Neurol 1995; 52: 612-619.
- Gale CR, Martyn CN, Cooper C. Cognitive impairment and mortality in a cohort of elderly people. Br Med J 1996; 312: 608-611

- Erkinjuntti T, Østbye T, Steenhuis R, Hachinski V. The effect of different diagnostic criteria on the prevalence of dementia. N Engl J Med 1997; 337: 1667-1674.
- Marshall KG. Prevention. How much harm? How much benefit?
  Physical, psychological and social harm. Can Med Assoc J 1996; 155(2): 169-176.
- Lasoski MC, Thelen MH. Attitudes of older and middle aged persons towards mental health intervention. Gerontologist 1987; 27: 288-292.
- Hutchinson B, Woodward CA, Norman GR, Abelson J, Brown JA. Provision of preventive care to unannounced standardized patients. Can Med Assoc J 1998; 158: 185-193.
- 77. Busch C, Kozak J, Elmslie T. Screening for cognitive impairment in the elderly. Can Fam Physician 1997; 43: 1763-1768.
- Tangalos EĞ, Smith GE, İvnik RJ, et al. The Mini-Mental State Examination in general medical practice: clinical utility and acceptance. Mayo Clin Proc 1996; 71: 829-837.
- Rubenstein LV, Calkins DR, Young RT, et al. Improving patient function: a randomized trial of functional disability screening. Ann Intern Med 1989; 111: 836-842.
- US Preventive Services Task Force. Screening for dementia. In: Guide to Clinical Preventive Services 2nd Ed. Alexandria, Virginia: International Medical Publishing. 1996; 531-540.
- Canadian Task Force on the Periodic Health Examination. The Canadian Guide to Clinical Preventive Health Care. Goldbloom R. ed. Ottawa. Canada Communication Group. 1994; 902-911.
- Rubenstein LV, Calkins DR, Greenfield S, et al. Health Status Assessment for Elderly Patients: Report of the Society of General Internal Medicine Task Force on Health Assessment. J Am Geriatr Soc 1989; 37: 562-569.
- American Psychiatric Association Committee on Nomenclature and Statistics. Diagnostic and Statistical Manual of Mental Disorders, 4th Ed. Washington, D.C.: American Psychiatric Association, 1994.
- World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders. Diagnostic Criteria for Research. Geneva: World Health Organization. 1993.
- 85. Morris JC, McManus DQ. The neurology of aging: normal vs. pathological change. Geriatrics 1991; 46: 47-48.
- Forstl H, Hentschel F, Sattel H, et al. Age-associated memory impairment and early Alzheimer's disease. Only time will tell the difference. Arzneimittelforschung 1995; 45: 394-397.
- Larrabee GJ, Crook TH. Estimated prevalence of age-associated memory impairment derived from standardized tests of memory function. Int Psychogeriatr 1994; 6: 95-104.
- 88. Crook T, Bartus RT, Ferris SH, et al. Age-associated memory impairment: proposed diagnostic criteria and measures of clinical change-report of a National Institute of Mental Health Work Group. Devel Neuropsychol 1986; 2: 261-276.
- Kral VA. Senescent forgetfulness: benign and malignant. Can Med Assoc J 1962; 86: 257-260.
- Barker A, Jones R, Jennison C. A prevalence study of age-associated memory impairment. Br J Psychiatr 1995; 167: 642-648.
- Hänninen T, Hallikainen M, Koivisto K, et al. A follow-up study of age-associated memory impairment neuropsychological predictors of dementia. J Am Geriatr Soc 1995; 43: 1007-1015.
- O'Brien JT, Beats B, Hill K, et al. Do subjective memory complaints precede dementia? A three-year follow-up of patients with supposed "benign senescent forgetfulness". Int J Geriatr Psychiatry 1992; 7: 481-486.
- Bowen J, Teri L, Lukull W, et al. Progression to dementia in patients with isolated memory loss. Lancet 1997; 349(9054): 763-765.
- Petersen RC, Smith GE, Waring SC, et al. Aging memory and mild cognitive impairment. Int Psychogeriatr 1997; 9: Suppl 1: 65-69.

#### INTERNET REFERENCES

- 1. CMACPG Infobase: www.cma.ca/cpgs
- National Library of Medicine for Clinical Practice Guidelines: http://text.nlm.nih.gov/

## Appendix

In preparing background papers, authors were instructed to use the rules of evidence developed by the Canadian Task Force on the Periodic Health Examination.<sup>6</sup> The following categories were utilized to grade the levels of evidence.

- Evidence obtained from at least one properly randomized controlled trial (RCT).
- II) i) Evidence obtained from well-designed controlled trials without randomization.
  - Evidence obtained from well-designed cohort or case control analytic studies preferably from more than one centre or research group.
  - iii) Evidence obtained from comparisons between times or places with or without the intervention. Dramatic results in uncontrolled experiments are included in this category.
- III) Opinions of respected authorities, based on clinical experience, descriptive studies or reports of expert committees.

Each background paper concluded with recommendations which were graded as follows:

- A. There is good evidence to support this manoeuvre.
- B. There is fair evidence to support this manoeuvre.
- C. There is insufficient evidence to recommend for or against this manoeuvre but recommendations may be made on other grounds.
- D. There is fair evidence to recommend against this procedure.
- E. There is good evidence to recommend against this procedure.

Ideally, A or E recommendations are supported by level I evidence. The paucity of level I evidence in the field of dementia resulted in recommendations frequently being based upon less rigorous evidence. A "C" recommendation does not imply that the manoeuvre is useless or harmful: there is simply insufficient evidence to make a stronger recommendation. For each recommendation the grading and strength of supporting evidence is given.