The field of aging and dementia is focusing on the characterization of the earliest stages of cognitive impairment. Recent research has identified a transitional state between the cognitive changes of normal aging and Alzheimer’s disease (AD), known as mild cognitive impairment (MCI). Mild cognitive impairment refers to the clinical condition between normal aging and AD in which persons experience memory loss to a greater extent than one would expect for age, yet they do not meet currently accepted criteria for clinically probable AD. When these persons are observed longitudinally, they progress to clinically probable AD at a considerably accelerated rate compared with healthy age-matched individuals. Consequently, this condition has been recognized as suitable for possible therapeutic intervention, and several multicenter international treatment trials are under way. Because this is a topic of intense interest, a group of experts on aging and MCI from around the world in the fields of neurology, psychiatry, geriatrics, neuropsychology, neuroimaging, neuropathology, clinical trials, and ethics was convened to summarize the current state of the field of MCI. Participants reviewed the world scientific literature on aging and MCI and summarized the various topics with respect to available evidence on MCI. Diagnostic criteria and clinical outcomes of these subjects are available in the literature. Mild cognitive impairment is believed to be a high-risk condition for the development of clinically probable AD. Heterogeneity in the use of the term was recognized, and subclassifications were suggested. While no treatments are recommended for MCI currently, clinical trials regarding potential therapies are under way. Recommendations concerning ethical issues in the diagnosis and the management of subjects with MCI were made.

The boundary between normal aging and early or mild Alzheimer’s disease (AD) is an area of intense interest for theoretical and practical reasons. Basic research, such as the identification of secretase inhibitors and the development of an immunization model for the prevention of amyloid deposition, underscores the importance of developing techniques for early detection.1,2 Parallel with these endeavors, clinical research aimed at identifying the earliest signs of cognitive impairment has progressed.3-5

A slight impairment in cognitive function, typically memory, with otherwise normal performance has been designated mild cognitive impairment (MCI) and has become a topic of considerable research.3,6 From June 24 through 26, 1999, investigators from around the world gathered in Chicago, Ill, at the Current Concepts in Mild Cognitive Impairment conference to review the world’s literature regarding the concept of MCI and to suggest directions for future research.

MILD COGNITIVE IMPAIRMENT

Mild cognitive impairment refers to a transitional state between the cognition of normal aging and mild dementia.7 As shown in Figure 1, the criteria for definitive AD,
while still controversial, have been reasonably well estab-
lished on a neuropathological basis.8 Similarly, in re-
cent years, the criteria for clinically probable AD have
also been well characterized,9 and the correlation be-
tween clinical and pathological findings in suspected cases
of AD has been good.10 However, as Figure 1 depicts, most,
if not all, patients with AD experience a subtle cognitive
decline before reaching the clinical threshold for the di-
agnosis of clinically probable AD. This transitional pe-
riod has become known as MCI and is now the subject
of several multicenter treatment trials involving more than
4000 subjects. The current clinical trials use an adapta-
tion of the MCI criteria shown in the Table.

Because other presentations of MCI exist, the type
just described has been termed amnestic MCI because its
definition emphasizes memory loss. Most of these sub-
jects will progress to AD at a rate of 10% to 15% per year,
compared with healthy control subjects who convert to
a rate of 1% to 2% per year.3,4 Data from the Mayo Alz-
heimer’s Disease Research Center, Rochester, Minn, which
has been observing a group of these subjects for more
than 10 years, have demonstrated a conversion to AD of
up to 80% during approximately 6 years (Figure 2).

Criteria for Amnestic Mild Cognitive Impairment

- Memory complaint, preferably corroborated by an informant
- Impaired memory function for age and education
- Preserved general cognitive function
- Intact activities of daily living
- Not demented

The differentiation of MCI from normality is an impor-
tant area of study. Whereas clear diagnostic and re-
search criteria for dementia are now available in inter-

RATING SCALES

There are several useful rating scales available for char-
acterizing subjects along a continuum from normal ag-
ing through various stages of dementia.11,12 Although these
scales are useful for describing subjects at various levels
of involvement, they do not necessarily coincide with the
clinically relevant conditions of normal aging, MCI, and
mild AD. For example, the Clinical Dementia Rating
(CDR) is a popular scale used to classify subjects along
a continuum from normal (CDR 0) through questionable dementia (CDR 0.5) to mild (CDR 1), mod-
erate (CDR 2), and severe (CDR 3) dementia. Although
some investigators believe that CDR 0.5 is equivalent to
MCI, others contend that CDR 0.5 actually describes a
broader population that includes subjects with MCI and
mild AD.13 Another instrument, the Global Deteriora-
tion Scale (GDS) stages subjects from GDS 1 (normal)
to GDS 2 (normal with subjective memory impair-
ment), GDS 3 (mild dementia), and GDS 4 through 7
(more severe stages of dementia).12 Within this rating
scale, subjects with MCI could correspond to a GDS of
either 2 or 3. These potential relationships are depicted in
Figure 3. The amnestic form of MCI is an identifi-
able entity that does not necessarily map to a specific stage
on these rating scales, thereby justifying a separate ter-

NORMAL AGING

The differentiation of MCI from normality is an impor-
tant area of study. Whereas clear diagnostic and re-
search criteria for dementia are now available in inter-
national disease classification systems, the notion of normal aging is less well understood. As disabilities associated with aging become treatable, they may be considered pathologic conditions rather than inevitable features of the physiological aging process. Because of evolving ideas of what constitutes normal aging, cross-sectional comparisons of older and younger populations are likely to be misleading because of these age-cohort effects.

Within population studies, the concept of successful aging is sometimes proposed as an alternative to normality. Successful aging refers to a state of health in which there are measurable positive features across a spectrum of health measures. It extends beyond cognitive and functional definitions by considering the value of self-related psychological well-being. The Canadian Study of Health and Aging concluded that self-rated psychological well-being is a potential outcome measure for longitudinal investigation of individuals with either no cognitive impairment or minimal cognitive impairment.

Previous attempts at characterizing cognitive changes intrinsic to normal aging have produced several terms, such as benign senescent forgetfulness, age-associated memory impairment, and age-associated cognitive decline. These terms are generally meant to reflect the extremes of normal aging rather than to describe a precursor of pathologic aging. While some investigations of these concepts demonstrate dementia conversion rates that are not different from those of healthy subjects, others have indicated an increased conversion rate. Subjects with MCI have a condition that is different from normal aging, and longitudinal outcome results indicate that they are likely to progress to AD at an accelerated rate.

**EPIDEMIOLOGY**

There has been little work on the incidence or prevalence of MCI as defined herein. This is partially because of the recent characterization of this condition. Population-based studies, especially those that ascertain cases based on psychometric test results rather than symptoms, may identify a population of individuals with mild cognitive dysfunction that differs from the amnestic MCI described clinically. Prevalence rates for age-associated memory impairment in these studies range from 17% to 34%, while age-associated cognitive decline has been estimated as having a prevalence of 26%. Other investigators have indicated that the prevalence rate for conditions such as age-associated memory impairment can range up to 85%, depending on the specific memory tests used to fulfill the criteria. In the Canadian Study of Health and Aging, the classification of cognitive impairment with no dementia had a prevalence rate of 17%; this is likely a more inclusive term than MCI alone.

An important factor in considering the frequency of MCI concerns the source of subjects for a given study. It is likely that a small number of subjects with MCI will present to memory disorders or dementia clinics. Most individuals who present to a dementia clinic probably already have dementia. However, if one surveys a community population, it is probable that more cases of MCI will be found.

All individuals who present clinically with mild cognitive symptoms may not share the same fate ultimately. Some may go on to develop AD, while others may progress to another dementia. It is possible that some of the subjects will never progress to any significant extent. This broad group of individuals with mild cognitive complaints could be considered as having MCI. Recognizing that there are multiple sources of heterogeneity in such a classification, it is desirable to further specify criteria for subsets of MCI.

Thus far, we have focused herein on the most common subset of subjects with MCI, those with amnestic MCI. These are individuals who present with a subjective memory complaint, preferably corroborated by an informant, and have an objective memory impairment compared with age and education norms, but who are performing reasonably well on indexes of general cognitive function and have generally preserved activities of daily living. As discussed earlier, these subjects do not meet standard criteria for AD. While subjects with amnestic MCI usually progress to AD at a high rate, not all will progress (Figure 2). On the other hand, some subjects who present with amnestic MCI may have other pathologic processes involving the medial temporal lobes, eg, hippocampal sclerosis. It is as yet uncertain whether clinical variations of AD or disorders with concomitant AD, such as dementia with Lewy bodies, can present as amnestic MCI. There is evidence that subjects with dementia associated with Lewy bodies have relative preservation of the hippocampi, making this amnestic MCI presentation less likely for subjects diagnosed as having dementia with Lewy bodies.

It theoretically should be possible to select a different subset of subjects with MCI by altering the core definition and criteria. Figure 4 depicts other hypothetical presentations of MCI in addition to amnestic MCI. Although none of these other prodromal conditions have been validated, MCI could be defined as mild impairment in multiple cognitive domains, without requiring memory deficit. These subjects may have multiple areas of cognitive impairment that fall outside of predicted norms, but none are sufficiently severe to constitute dementia. Subjects with MCI defined in this fashion may also progress to AD, but they could also progress to other disorders.

Finally, MCI could conceivably present as impairment in a single cognitive domain other than memory. For example, a pronounced language disturbance might progress to primary progressive aphasia, or an alteration...
in attentional abilities or comportment and a dysexecutive syndrome might progress to frontotemporal dementia. Although no such MCI cases have been reported, it is reasonable to posit a prodromal period for frontotemporal dementia, analogous to amnestic MCI as a precursor of AD.

It has become apparent that heterogeneity of MCI is derived from differences in causes, clinical symptoms, and research methods. For clarity, it is recommended that the term MCI be qualified with an appropriate modifier, such as amnestic MCI, to inform the reader as to the specific criteria being used to characterize the condition and its most likely outcome.

NEUROPSYCHOLOGICAL ASSESSMENT

Screening instruments are routinely used to quantify the degree of cognitive impairment in patients with dementia and are likely to be particularly helpful early in a dementia illness, when functional and behavioral disturbances are absent. The value of neuropsychological measures in helping to identify early cases of dementia has been documented by cross-sectional and longitudinal studies. Cross-sectional comparisons of patients clinically diagnosed as having mild AD compared with older healthy persons have shown that patients with mild AD consistently perform worse than their comparison subjects. Patients with early AD are impaired on tests of memory, including tests of delayed recall, and often on tests of new learning. Deficiencies in language, executive function, and attention are also present in many patients with mild AD, and deficits in more than one cognitive domain are a better indicator of mild AD than memory impairment alone. These cross-sectional data indicate that sensitive neuropsychological measures can help identify cases of AD at early stages.

Longitudinal studies of older healthy persons at high risk for developing dementia because of advanced age have shown that persons who later develop AD perform more poorly on cognitive tests at baseline compared with those who remain free of dementia. These longitudinal data indicate that neuropsychological tests may help to identify persons likely to convert to MCI or AD before they meet conventional diagnostic criteria.

Alzheimer’s disease and MCI cannot be diagnosed by neuropsychological tests alone, and clinical judgment is always required. First, performance on neuropsychological tests is affected by many factors, including education, age, cultural background, and illnesses other than AD. Second, neuropsychological measures cannot fully distinguish among different types of dementia, because there is substantial overlap in neuropsychological profiles. The usefulness of any battery for identifying cases of MCI will depend on its composition, size, and supporting data. A brief battery, including measures of new learning, delayed recall, attention, and executive function, could provide valuable information for screening and diagnosis if interpreted properly.

NEUROIMAGING

Neuroimaging potentially is a powerful tool for the differential diagnosis of cognitive impairment and for monitoring change. Cross-sectional and longitudinal studies have used structural (computed tomography and magnetic resonance imaging) and functional (single-photon emission computed tomography, positron emission tomography, and magnetic resonance spectroscopy) modalities in the evaluation of MCI. There is agreement on the occurrence of hippocampal atrophy in amnestic MCI compared with cognitively intact controls, and on the finding that hippocampal atrophy can predict the rate of conversion from MCI to AD. Hippocampal atrophy seen in MCI can be correlated with autopsy evidence of atrophy and neuronal loss.

In addition to hippocampal atrophy, changes in the entorhinal cortex may enhance the sensitivity of an early diagnosis of MCI or AD. However, a recent study comparing entorhinal measures with hippocampal volumes indicates that the entorhinal measurement may not always be superior. Neuroimaging measures of change are more informative than a measurement made at one point in time. Rates of hippocampal atrophy for individuals who convert from control subjects to MCI or from MCI to AD are comparable, suggesting that the MCI group may be transitional to early AD.

Functional neuroimaging may also aid in the detection of subjects with MCI. In group studies using positron emission tomography, early metabolic deficits in the temporoparietal region have been demonstrated in those at risk for familial AD based on family history and those with an apolipoprotein E4 genotype. Similarly, patients with a genetic risk for AD also show metabolic reduction in the posterior cingulate cortex. Magnetic resonance spectroscopy may eventually provide further evidence of functional changes in MCI, although investigations have been limited thus far.

In summary, neuroimaging studies support the view that MCI, especially amnestic MCI, shares features with AD, such as hippocampal atrophy, so that the presence or development of atrophy will therefore predict conversion to clinical AD. Hippocampal atrophy, however, may not be specific, and careful selection and follow-up of subjects are important. Future directions are likely to concentrate on measuring rates of change on neuroimaging studies and on finding the shortest intervals necessary to demonstrate significant changes.

NEUROPATHOLOGY

Little is known about the neuropathology of MCI and related disorders, because there have been few longitudinal studies that include neuropathological confirmation. Subjects with mild impairment are unlikely to die of MCI, so neuropathological information is obtained only when someone who happens to have the clinical designation of MCI dies of other causes. In a neuropathological series of 16 patients diagnosed clinically as having MCI or AD and a CDR of 0.5, all demonstrated neurofibrillary tangles in the hippocampus and entorhinal cortex, while 7 also had many senile plaques in the neocortex. Other indexes of neuronal degeneration, such as changes in synaptic markers and neuronal loss, were seen in the CA1 region of the hippocampus as assessed by unbiased stereology.
In a clinical pathological series of 62 patients (39 without dementia, 15 with a CDR of 0.5, and 8 with established AD), all of those with CDR 0.5 ratings had the neuropathological features of AD. These investigators postulated that senile plaques in the neocortex may represent the initial event in AD and may escalate age-related formation of neurofibrillary tangles in the medial temporal lobe beyond the usual level. Neocortical senile plaques and entorhinal tangles were also seen in some subjects with no detectable cognitive decline, suggesting that there may be a neuropathological picture of AD that precedes an MCI state.

In another report of 10 autopsy cases with clinical diagnoses of amnestic MCI, 6 met neuropathological criteria for AD, while an additional 2 had argyrophilic grain disease. Two cases had neurofibrillary tangle–only disease. Data from the Religious Orders Study have supported clinical observations that the causes of MCI may be heterogeneous, because they have shown that non-AD disorders that involve the medial temporal lobe may account for the memory impairment. When these rare non-AD disorders are excluded, most pathologic conditions of MCI are likely to be early-stage AD.

In summary, most patients with amnestic MCI demonstrate neurofibrillary pathologic conditions in the medial temporal lobes. It is possible that these neurofibrillary changes contribute to the memory impairment. Some evidence indicates that patients with CDR 0 ratings (no memory impairment) also have neurofibrillary tangles in the medial temporal lobe; hence, this finding does not always correlate with a memory impairment. Most patients with MCI appear to have the neuropathological changes of AD, and a few clinically similar subjects have non-AD pathologic conditions.

BIOMARKERS

Because MCI may be clinically and pathologically heterogeneous, biomarkers may be particularly useful for identifying subtypes. However, there are no definitive data on the usefulness of biomarkers in classifying MCI.

Biomarkers in the cerebrospinal fluid (CSF) have been used as indicators of the degenerative process of AD. Several laboratories have shown that CSF tau concentrations are significantly elevated in the CSF and that Aβ-42 levels are significantly decreased in patients with AD, compared with age-matched cognitively healthy control subjects. Tau levels are also significantly increased in mildly demented subjects with AD (Mini-Mental State Examination score of 25), yielding sensitivity and specificity values approaching 0.90. More recently, elevated CSF tau has been confirmed in autopsy-proven AD cases.

Longitudinal studies have demonstrated that nearly all subjects with MCI who convert to AD have high CSF tau values, whereas in nonprogressive MCI, tau levels remain low. Furthermore, CSF tau levels do not seem to increase during the course of AD. This suggests that the measurement of CSF tau might be used effectively for identifying incipient AD among patients diagnosed clinically as having MCI. In summary, biomarkers could be useful in a multistep approach to the diagnosis of MCI or dementia, and disease-specific biomarkers could be useful in treatment intervention follow-up.

PROGRESSION

Several longitudinal studies of aging and dementia provide information regarding outcomes of patients with MCI. In addition, some studies of aging shed light on cognitive progression in individuals who are functioning normally at one point in time, but who subsequently develop MCI or AD. Both types of studies provide information regarding the ultimate usefulness of the designation of MCI.

Using the criteria outlined in the table for amnestic MCI, investigators at the Mayo Alzheimer’s Disease Research Center longitudinally observed 155 individuals with a clinical diagnosis of MCI. These individuals evolved to dementia at a rate of approximately 12% per year. Variables that tended to predict a more rapid decline included apolipoprotein E4 carrier status, poor performance on a cued recall test, and atrophic hippocampi on MRI. As indicated in Figure 2, up to 80% of this group had evolved to dementia in approximately 6 years.

In Toronto, Ontario, investigators observed a group of subjects with memory complaints who had been referred to primary care practitioners. These subjects were followed longitudinally for 2 years, and approximately 20% progressed to dementia. These investigators believed that performance on a memory test and possibly apolipoprotein E4 status were predictors of decline.

Investigators from the University of Washington Alzheimer’s Disease Patient Registry, Seattle, also followed up a group of memory-impaired individuals who did not meet criteria for dementia. These subjects were observed for up to 48 months, and approximately 50% of the group progressed to dementia during that time. These investigators did not find any specific psychometric tests that predicted who was more likely to become impaired.

In various studies using different MCI criteria, subjects with memory problems that fall short of dementia progress to dementia or AD at a rate of between 10% to 15% per year, making this condition an identifiable risk factor for the development of AD. These studies, taken together, indicate that different criteria in multiple studies may be identifying the common phenomenology of amnestic MCI.

TREATMENT

There is no evidence that MCI, once diagnosed, can be successfully treated. Inclusion criteria for MCI trials will likely continue to focus on subjects with amnestic MCI who do not meet diagnostic criteria for AD. The end points for MCI trials will depend on the presumed benefits of the intervention. Some interventions may result in improvement of symptoms. End points to demonstrate this type of benefit will likely be limited to sensitive tests of delayed memory (verbal and visual) and clinicians’ global ratings. In other cases, slowing the rate of progression may be the goal, so delaying a clinical milestone (eg, de-
development of a second domain of difficulty) or conversion to clinically diagnosable AD will be the appropriate end points. Several clinical trials have already been initiated to test the use of cholinesterase inhibitors, antioxidants, and anti-inflammatory drugs in patients with MCI. Most of these trials are based on the recently initiated trial being conducted by the Alzheimer's Disease Cooperative Study.

Safety and tolerability will be required for drugs used in MCI trials, because many subjects will be treated at the early stage of the disease when symptoms are mild. Determination of optimal drug dosage should be worked out in individuals with AD, when possible, because dosage-finding studies expose individuals to a greater risk of adverse effects, and the risk-benefit ratio may be different for subjects with MCI vs AD.

Many classes of agents can be considered for use in subjects with amnestic MCI. Cholinesterase inhibitors may be considered for use for their symptomatic effects and circumspect laboratory and clinical evidence that they may have a biologically modifying effect on the processing of amyloid or other mechanisms in the progression of the disease. Hormones, particularly estrogens, have been epidemiologically demonstrated to be associated with delayed onset of the appearance of AD.

Histological and epidemiological studies suggest the presence of inflammation early in AD. Clinical trials of the effect of cyclooxygenase-2 (COX-2) inhibitors in patients with AD and MCI are under way. Antioxidants such as vitamin E have been demonstrated to delay the time to clinical end points in patients with moderate AD, and vitamin E is being studied in patients with MCI. Interruption or modification of protein metabolism is believed by many AD researchers to represent the best approach to disease-modifying therapy. Agents that interfere with Aβ production and aggregation or that reduce Aβ burden may confer protection against Aβ-mediated neurodegeneration in AD. Another molecular approach focuses on the development of agents that interfere with tau phosphorylation or aggregation. Finally, *Gingko biloba* may have antioxidant properties and will undergo large-scale testing in primary prevention trials. Treatments should not be prescribed based on current speculations, but must await confirmation from well-designed clinical trials.

**ETHICAL ISSUES**

The concept of MCI raises several ethical issues. The primary ethical questions concern the strength of the evidence that MCI is a valid pathologic condition, and whether the benefits of establishing this new category outweigh the risks to patients. Making an accurate diagnosis is a key element in the practice of medicine and has several benefits. It helps clinicians identify specific conditions and aids in the choice of appropriate treatment. In addition, the establishment of a diagnosis enhances communication among clinicians, helps provide information to patients, identifies a need for understanding and support, and stimulates research. On the other hand, labeling patients with a disease has psychological and psychosocial consequences. Therefore, investigators and clinicians who are proposing MCI as a new syndrome must develop the data needed to convince skeptics that it is a valid entity.

Patients diagnosed as having amnestic MCI often complain about memory loss. This suggests that individuals with MCI have insight and should be told about this diagnostic label. On the other hand, some individuals with MCI, especially if ascertained by population-based testing, may be unaware of their memory impairment or may believe that their symptoms are related to normal aging or lifelong problems. Patients who present clinically should probably be managed differently from patients discovered by applying research criteria to large populations.

Because amnestic MCI progresses to AD at a high rate, it is associated with significant morbidity, potential economic loss to individuals and society, and frustration and distress in caregivers. Mild cognitive impairment itself is therefore an appropriate target for treatments to improve symptoms and to reverse or prevent the condition.

**RECENT LITERATURE**

Since the Current Concepts in Mild Cognitive Impairment conference, several relevant studies have appeared in the literature. These will be mentioned without an evaluative comment because they were not reviewed by the conference attendees.

Investigators at Massachusetts General Hospital, Boston, followed up a group of subjects with a diagnosis of questionable dementia (CDR 0.5) for 3 years and found that this group progressed to AD at a rate of 6% per year. This study documented the importance of the clinical interview in the characterization of these subjects. An editorial comment on this article called attention to the distinction between a clinical diagnosis of MCI and the use of rating scales. Another study on these subjects demonstrated the usefulness of magnetic resonance imaging–based volumetric measurements of the entorhinal cortex in predicting progression in individuals with questionable dementia. A study from France applied a modified version of MCI criteria to a population of subjects being observed longitudinally and found that certain neuropsychological criteria for MCI, when retrospectively applied, were unreliable in characterizing the progression of subjects over time. A Washington University research group performed an autopsy-based study of their subjects with a CDR 0.5 diagnosis and found that all had a dementing illness; 88% had neuropathological features of AD. A study from Sweden demonstrated the reliability of impaired glucose metabolism and a cognitive measure of visuospatial performance in predicting progression from MCI to AD. Finally, a recent evidence-based medicine practice review of early detection and MCI by the American Academy of Neurology recommended that subjects with MCI be identified and followed up because of their increased risk for developing AD.

**SUMMARY**

Mild cognitive impairment is becoming an increasingly recognized clinical entity. Most clinicians are aware of...
patients who appear to have a mild deficit in memory or some other aspect of cognition but who are not demented. There are important differences between clinical practice settings and research settings. For example, researchers use comprehensive neuropsychological testing to document that memory loss is clinically significant and to make certain there are no other domains of significant cognitive impairment that might mimic criteria for mild AD. Clinicians should be wary about designating a patient as having MCI in the absence of specific criteria. Although therapies are being developed that might prevent progression of amnestic MCI to AD, there is no evidence to support prescribing therapeutic agents to patients with MCI at this time. Our conference discussion highlighted the current thinking on MCI from a recent international conference. Mild cognitive impairment deserves recognition and further study because, as preventive treatments for AD become available, it will become incumbent on clinicians to identify persons at risk for AD and those with the earliest signs of clinical impairment. It may be that at least some of what is now designated as MCI will be recognized in the future as the earliest symptomatic stage of AD. Such an evolution will be stimulated by evidence that pharmacological therapy is beneficial at this stage and by the clinician’s ability to recognize earlier forms of AD.

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