Memory Care Monthly

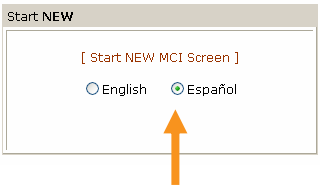
Supporting Healthcare Professionals in Caring for the Aging June 2007

MCI Screen Product Improvements

Next time you login to your account at www.mccare.com, you will notice the following improvements:

Spanish version of the MCI Screen

Spanish language wordlist and instructions are now available for the MCI Screen. When starting a new MCI Screen, the English language version is the default. However, before beginning, you have the option to select Spanish as the language of choice. Please note that the report at the completion of the MCI Screen will still be in English. The Spanish version includes the original wordlist from the Spanish version of the CERAD battery.



Select “Español” before starting a new MCI Screen.

Memory Performance Index

In addition to the Overall Impression of the MCI Screen (Impaired vs. Normal), which we currently report, we now provide a quantitative measure of cognitive function called the Memory Performance Index (MPI). The MPI will enable you to track improvement or decline across successive tests and better understand the extent to which the memory of any given patient is either normal or impaired for their age group.

The MPI score and scale will appear on any new MCI Screens you complete. The MPI score is not available for previously completed MCI Screens.

The MPI scale ranges from 0 to 100. Approximately 1% of individuals tested will be classified as “Borderline” (scores of 49.3 < MPI < 50.7). For these individuals, their score cannot be clearly classified into a Normal or Impaired result.

For the Normal range of scores (50.7 ≤ MPI ≤100), larger values indicate better performance when compared to an individual's peers after accounting for effects of age, gender and education. For example, a score of 93 indicates better cognitive performance than a score of 85.

For the Impaired range of scores (0 ≤ MPI ≤ 49.3), smaller values indicate worse performance when compared to an individual's peers after accounting for effects of age, gender and education. For example, a score of 10 indicates worse cognitive performance than a score of 15.

Streamlined Report

Based on customer feedback, we have shortened the MCI Screen report to only show each patient’s results. The additional information currently offered on the MCI Screen Report, such as the ADRD diagnostic guidelines and the references, will be available in an optional expanded view of the report. As an additional part of the streamlining process, we have eliminated the “patient report” option. The physician report will continue to be the standard format for report printing.

Alzheimer's Neuropathology and Beta Amyloid: A Unifying Hypothesis

Understanding beta amyloid's role in Alzheimer's disease (AD) has greatly advanced understanding about treating AD. Autopsy and transgenic mouse model research since the 1980s have led to a general consensus that beta amyloid plays a significant role in the neuropathology and clinical course of AD[1]. Beta amyloid accumulates early on in the asymptomatic stage, years before AD manifests clinical symptoms, and continues to accumulate into the severe stages[2]. The beta amyloid 1-42 species accumulates in neuritic plaques of AD and associates with cognitive and functional impairment in animal models. Furthermore, transgenic AD animal models have shown that intraneuronal beta amyloid 1-42 production significantly contributes to the development of fibrillar beta amyloid, neuritic plaques, synapse loss and neurofibrillary tangles, all of which comprise the neuropathologic hallmarks of AD[3]. Treatments that reduce the levels of soluble beta amyloid and tau protein improve behavioral deficits in AD mouse models[4], and in humans who received Elan's first monoclonal antibody against beta amyloid 1-42 but did not experience adverse effects of encephalitis[5].

This convergence of agreement on the significance of beta amyloid in AD has led to a focus of treatments that lower soluble, oligomeric beta amyloid production and/or increase its clearance. Beta amyloid lowering agents have been identified, and include the cholinesterase inhibitors[6-8], human blood donor-derived immunoglobulins[9], possibly the statins[10], non-steroidal anti-inflammatory drugs[11], curcumin[12], and possibly others.

References

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Decline in Executive Function May Precede Memory Loss in Patients with Mild Cognitive Impairment

A recent study funded by National Institutes of Health found that executive function associated with non-amnestic mild cognitive impairment (MCI-EF) may precede memory loss in the earliest stages of preclinical dementia. Rhonda Au, PhD and associates from Boston University School of Medicine analyzed data on 1850 healthy subjects from Framingham Offspring Study. All subjects underwent brain MRI and neuropsychological testing. After neuropsychological screening, 1602 subjects did not have memory or executive function deficits while 115 had amnestic MCI and 133 had MCI-EF.

Brain MRI measures used were: total cerebral brain volume (TCBV), hippocampal brain volume (HBV) and white-matter hyperintensities (WMH). Compared to those without MCI, those with both subtypes of MCI had smaller TCBV. Those with memory impairment were almost twice as likely to have smaller HBV. Those with executive function deficits were almost 3 times more likely to have large WMH. Those with extensive WMH volume had a 5-fold increased risk for MCI-EF vs. amnestic MCI. Reference: Presented at American Academy of Neurology 59th Annual Meeting.

Easily Administered Neurocognitive Tests Can Measure Progress to Alzheimer's

539 participants from Alzheimer's Disease Cooperative Study (ADCS) MCI trial were included in a study that aimed to determine which cognitive tests helped predict progression from mild cognitive impairment (MCI) to Alzheimer's disease (AD). The subjects were followed for 36 months and the overall rate of progression from AD to MCI was 16%. Researchers recommended a combination of tests which included the APOE allele status, the Symbol Digit Modalities Test, delayed 10-Word Recall Test, NYU Delayed Paragraph Test, and the ADAS-cog total score. Combined, these tests had a predictive accuracy of 81% for determining progression to AD. Without the APOE test, the predictive accuracy was 80%. This study was led by Dr. Adam S. Fleisher from University of California, San Diego, and associates. Reference: Fleisher A.S. et al., Neurology, 68(19):1588-1595, 2007.

Neuropsychiatric Symptoms May Signal Progression to Alzheimer's

Dr. Katie Palmer and colleagues at the Karolinska Institute in Stockholm, Sweden studied whether symptoms of depression and anxiety in patients with mild cognitive impairment (MCI) were related to future development of Alzheimer's disease (AD). The study included 185 individuals with MCI and researchers found that depressive symptoms were not associated with an increased risk of progressing from MCI to AD. However, with each increasing number of symptoms of anxiety, the risk of progressing to AD nearly doubled. Those with increased risk of progressing to AD also included individuals reporting problems with decision making and also those with persistent worrying. In elderly subjects without cognitive impairment, the risk of developing AD nearly doubles in those with mood-related depressive symptoms. Reference: Palmer K. et al., Neurology, 68(19):1596-602, 2007.

Early Hormone Replacement Therapy May Reduce Risk of Dementia

Research from Women's Health Initiative Memory Study links hormone replacement therapy before age 65 to reduced risk of dementia by 46% and Alzheimer's disease by 64%. Victor Henderson, MD, Stanford University in Palo Alto led the study. References: Presented at American Academy of Neurology 59th Annual Meeting.

Two Studies Provide Data on Stroke Rates and Timeliness of Treatment

In the May 18 issue of *Morbidity and Mortality Weekly Report* two studies were published about stroke. The first study was conducted by researchers from the Centers for Disease Control and Prevention. Data from the 2005 Behavioral Risk Factor Surveillance System Survey was analyzed. Overall, 2.6% of non-institutionalized adults had history of stroke and the rate was the same for both men and women. Prevalence of stroke increased with age and was .8% for people between age 18 and 44; 8.1% for people 65 years of age and older. American Indians, multiracial persons, and blacks had stroke rates of 4% or more. Whites had a rate of 2.3%. Additionally, the rate of stroke was higher in persons with less than 12 years of education compared with college graduates: 4.4% vs. 1.8% Connecticut had the lowest rate of 1.5%; Mississippi had the highest rate of 4.3%.

The second study was based on analysis of data from Georgia, Illinois, Massachusetts, and North Carolina. According to the data, only 48% of stroke patients arrived in the emergency room within 2 hours making it difficult to provide the appropriate care. Ambulance transportation was used in 53.4% of the cases and made a difference in the arrival time. 36.2% of non-ambulance patients arrived in 2 hours compared to 56.8% of those transported by ambulance. Reference: Morbidity and Mortality Weekly Report, 56(19), 2007.

Centrally Active ACEIs May Slow Cognitive Decline

According to a study led by Kaycee M. Sink, MD, MS from Wake Forest University School of Medicine in Winston-Salem, North Carolina, centrally active angiotensin-converting enzyme inhibitors (ACEIs) are associated with slower rates of cognitive decline. Data from 5888 people older than 65 enrolled in the Cardiovascular Health Study (CHS), a long-term study of cardiovascular risk factors, was analyzed. Of the participants, 1142 were treated for hypertension and had no dementia at baseline. Of this number, 68 had heart failure and were excluded, 20 were lost to follow-up and excluded. Among the remaining 1054 participants, 640 were not taking ACEIs and 414 were taking ACEIs during study. Of those taking ACEIs, 278 were taking centrally active ACEIs. The mean age in cohort was 74.8±4.9 years and the median follow-up period for this cohort were 6 years. Two outcome measures were used in the study: incident all cause dementia was determined based on review of magnetic resonance imaging and change in cognitive function was determined using the Modified Mental State Examination. During follow-up period, 159 cases of incident dementia were identified. When use of all ACEIs was compared to other antihypertensive drugs, there was no increased risk of dementia or change in cognitive function. However, in those taking centrally active ACEIs, the decline in cognitive function was 50% less than that associated. Reference: May 5,2007 Annual Meeting of American Geriatrics Society in Seattle, WA.

Treatments for CNS Conditions: Recent FDA Approvals

Exelon (rivastigmine tartrate) has been approved for the treatment of mild to moderate Parkinson's disease (PD) dementia. PD dementia can effect up to 40% of patients with PD. Exelon is sold by Novartis and is also approved for treatment of mild to moderate Alzheimer's.

Cymbalta (duloxetine HCl) has been approved for treatment of generalized anxiety disorder. Currently used as an antidepressant, the drug is produced by Lilly.

Vyvanse (lisdexamfetamine dimesylate) has been approved for the treatment of attention-deficit/hyperactivity disorder. The drug is marketed by Shire in collaboration with New River Pharmaceuticals Inc.

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19782 MacArthur Blvd. #310, Irvine, CA 92612
www.mccare.com • (888) 565-5535