Underestimation of Cognitive Impairment by Mini-Mental State Examination Versus the Montreal Cognitive Assessment in Patients With Transient Ischemic Attack and Stroke

A Population-Based Study

Sarah T. Pendlebury, MRCP, DPhil; Fiona C. Cuthbertson, MCSP; Sarah J.V. Welch, RGN; Ziyah Mehta, DPhil; Peter M. Rothwell, FRCP, FMedSci

Background and Purpose—The Mini-Mental State Examination (MMSE) is insensitive to mild cognitive impairment and executive function. The more recently developed Montreal Cognitive Assessment (MoCA), an alternative, brief 30-point global cognitive screen, might pick up more cognitive abnormalities in patients with cerebrovascular disease.

Methods—In a population-based study (Oxford Vascular Study) of transient ischemic attack and stroke, the MMSE and MoCA were administered to consecutive patients at 6-month or 5-year follow-up. Accepted cutoffs of MMSE <27 and MoCA <26 were taken to indicate cognitive impairment.

Results—Of 493 patients, 413 (84%) were testable. Untestable patients were older (75.5 versus 69.9 years, P<0.001) and often had dysphasia (24%) or dementia (15%). Although MMSE and MoCA scores were highly correlated (r2=0.80, P<0.001), MMSE scores were skewed toward higher values, whereas MoCA scores were normally distributed: median and interquartile range 28 (26 to 29) and 23 (20 to 26), respectively. Two hundred ninety-one of 413 (70%) patients had MoCA <26 of whom 162 had MMSE ≥27, whereas only 5 patients had MoCA ≥26 and MMSE <27 (P<0.0001). In patients with MMSE ≥27, MoCA <26 was associated with higher Rankin scores (P=0.0003) and deficits in delayed recall, abstraction, visuospatial/executive function, and sustained attention.

Conclusion—The MoCA picked up substantially more cognitive abnormalities after transient ischemic attack and stroke than the MMSE, demonstrating deficits in executive function, attention, and delayed recall. (Stroke. 2010;41:1290-1293.)

Key Words: cognitive impairment ■ dementia ■ stroke ■ vascular cognitive impairment

Cognitive impairment after stroke is common and predicts dependency, institutionalization, and early mortality.1 Approximately 10% of patients with first-ever stroke develop new dementia and at least 30% have dementia after recurrent stroke.2 Although there is thus a need for short feasible tests of global cognition in stroke, there is no consensus about which test is most appropriate; the Mini-Mental State Examination (MMSE) is widely used but it is insensitive to mild cognitive impairment and may be suboptimal in assessing cognitive abnormalities associated with cerebrovascular disease.3

The recently developed Montreal Cognitive Assessment (MoCA) is designed to be sensitive to mild cognitive impairment and, unlike the MMSE, includes executive and attentional tasks.4 The MoCA has been evaluated in mild cognitive impairment of the Alzheimer type4 and in Parkinson disease5 but has not been widely used in patients with cerebrovascular disease in whom frontal lobe deficits may be prominent. We compared the MoCA and the MMSE in a population-based study of transient ischemic attack (TIA) and stroke.

Methods

Patients were participants in the Oxford Vascular Study, a prospective population-based cohort study of all acute vascular events occurring within a defined population of approximately 91 000. The Oxford Vascular Study was approved by the Oxfordshire Clinical Research Ethics Committee (CO.043) and informed consent was obtained. The MMSE was administered at the beginning of the study and the MoCA at the end of a 30 minute follow-up appointment together with the modified Rankin score. All patients with TIA or stroke, who were alive and seen for either their 6-month or 5-year follow-up between November 2007 until June 2009, were included in the current study. Reasons for not being tested were recorded.

For comparisons between tested and untested patients, t tests were used for continuous variables and Fisher exact test for categorical variables. A cutoff of ≥27 on the MMSE was chosen to indicate normal cognitive function6 and the accepted cutoff of <26 on the MoCA was taken to indicate cognitive impairment.4

Received January 22, 2010; accepted February 8, 2010.
From the Stroke Prevention Research Unit (S.T.P., F.C.C., S.J.V.W., Z.M., P.M.R.), University Department of Clinical Neurology, John Radcliffe Hospital and the University of Oxford, Oxford, UK; and the National Institute of Health Research Biomedical Research Centre (S.T.P.), John Radcliffe Hospital, Oxford, UK.
Correspondence to Sarah T. Pendlebury, MRCP, DPhil, Stroke Prevention Research Unit, Level 6 West Wing, John Radcliffe Hospital, Oxford OX3 9DU, UK. E-mail sarah.pendlebury@clneuro.ox.ac.uk
© 2010 American Heart Association, Inc.

Stroke is available at http://stroke.ahajournals.org DOI: 10.1161/STROKEAHA.110.579888
For both the MMSE and the MoCA, mean raw and percent (as percent of the maximum possible score) subtest scores were calculated and z scores were derived by converting the mean raw score and SD to the standard normal distribution with mean 0 and SD 1 (lower z scores indicating greater discrimination between subjects).

Results

Four hundred ninety-three consecutive patients were seen at either their 6-month (n = 289) or 5-year (n = 204) follow-up (Table 1). Nontestable patients (16%) were significantly older (P < 0.001) than the tested patients and more likely to have had a previous cerebrovascular event (35% versus 19%, P = 0.009). Untestability was more likely after stroke than TIA (P < 0.01) and at 5-year versus 6-month follow-up (P = 0.009). The most common reasons for being untestable were dysphasia (24%) and dementia (15%) with inability to use the dominant hand affecting 7 of 80 (9%; Table 1).

In 413 tested patients, MMSE scores were skewed toward higher values (median and interquartile range 28 [26 to 29]), whereas MoCA scores were normally distributed (23 [20 to 26]). Two hundred ninety-one of 413 (70%) patients had low MoCA (<26) of whom 162 had normal MMSE (>27), whereas only 5 patients with normal MoCA (>26) had MMSE <27 (P < 0.00001; Figure). Results were similar in older versus younger and better versus poorly educated patients. Rankin scores were significantly lower in patients with MMSE >27 and MoCA >26 than in those with MMSE ≥27 and MoCA <26 or in those with MMSE <27 (P < 0.001).

Individual subtests of the MMSE and MoCA are described in Table 2 together with the results. Z scores were <7 for all MoCA subtests, whereas in 4 MMSE subtests (registration, naming, comprehension, and reading), z scores were >10 indicating poor discrimination between subjects. Comparison

---

Table 1. Demographic Details for Tested and Untested Patients and Reasons for Being Untestable for 6-Month and 5-Year Follow-Up Cohorts and for Both Cohorts Combined

<table>
<thead>
<tr>
<th>Reason Not Testable</th>
<th>TIA</th>
<th>Stroke</th>
<th>TIA</th>
<th>Stroke</th>
<th>TIA</th>
<th>Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIA</td>
<td>1</td>
<td>10</td>
<td>4</td>
<td>4</td>
<td>5</td>
<td>21†</td>
</tr>
<tr>
<td>Dementia</td>
<td>0</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Poor vision</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>3</td>
<td>5</td>
<td>21</td>
</tr>
<tr>
<td>Poor english</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Unwell</td>
<td>1</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Coexistent neurological disorder</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Learning disability</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Illiteracy</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wrist fracture</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Benign tremor</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Arthritis</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Deafness</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>MMSE done, declined MoCA</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>No reason documented</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>21†</td>
</tr>
<tr>
<td>Total no.</td>
<td>7</td>
<td>29</td>
<td>17</td>
<td>27</td>
<td>24</td>
<td>100</td>
</tr>
</tbody>
</table>

*Age at the time of the index event.
†Numbers in parentheses are percentages.

---

Figure. Bubble plot of MMSE versus the MoCA for all patients (6-month and 5-year cohorts combined; Spearman correlation r² = 0.79, P < 0.01). Vertical line shows the cutoff at MMSE <27 and the horizontal line shows the cutoff at MoCA <26. The shaded bubbles indicate those patients who scored ≥27 on the MMSE but <26 on the MoCA.
between those with MMSE ≥27 and MoCA ≥26 (n=117) versus MMSE ≥27 and MoCA <26 (n=162) showed significant differences (P≤0.002, Fisher exact test) between the 2 groups in all MoCA subtest scores except calculation.

**Discussion**

The MoCA picked up substantially more cognitive deficits than the MMSE in patients with TIA and stroke. Fifty-eight percent of patients with normal MMSE (≥27) had abnormal MoCA, and these patients were more dependent by the Rankin scale than those with normal MoCA, highlighting the clinical relevance of our findings.

The MoCA differentiated well between different levels of cognitive ability, whereas the MMSE had a clear ceiling effect; over half the patients with MMSE scores ≥27 were designated as cognitively impaired using the MoCA. This echoes the findings of the original MoCA study in which 75% of patients with mild cognitive impairment on neuropsychological testing had normal MMSE but abnormal MoCA.4 In our study, the MoCA demonstrated deficits in multiple domains that were not detected by the MMSE, including executive function and attention (not tested by the MMSE) and recall and repetition (MMSE items too easy). In Parkinson disease, in which frontal lobe deficits are an early cognitive feature, more than half of those with normal MMSE...
were impaired on the MoCA and the latter correlated well with a neuropsychological battery.5

There are limitations to our study. First, determination of the specificity and sensitivity of the MoCA for cognitive impairment in our population could not be made because formal neuropsychological testing was not performed; whereas a score of <26 on the MoCA has been defined as consistent with cognitive impairment in a memory clinic population,4 isolated cognitive deficits and/or depression or apathy poststroke may result in spuriously low MoCA scores and thus a loss in specificity. Second, we did not formally assess reproducibility of the MoCA, although associations were robust to different observers as evidenced by the strong similarity between the results obtained in the 6-month and 5-year cohorts. Finally, the MoCA was always performed at the end of the appointment and fatigue may have increased the likelihood of error, although appointments were kept short to minimize this effect.

In conclusion, we have shown that the MoCA picks up substantially more cognitive abnormalities than the MMSE in patients with cerebrovascular disease. Further work is required to determine sensitivity and specificity of the MoCA in relation to formal neuropsychological testing and its ability to predict dementia in longitudinal studies.

Sources of Funding
The Oxford Vascular Study is funded by the UK Stroke Association, the Dunhill Medical Trust, the National Institute of Health Research (NIHR), the Medical Research Council, and the NIHR Oxford Biomedical Research Centre. S.T.P. is supported by the NIHR Oxford Biomedical Research Centre, Oxford, UK.

Disclosures
None.

References
Underestimation of Cognitive Impairment by Mini-Mental State Examination Versus the Montreal Cognitive Assessment in Patients With Transient Ischemic Attack and Stroke: A Population-Based Study
Sarah T. Pendlebury, Fiona C. Cuthbertson, Sarah J.V. Welch, Ziyah Mehta and Peter M. Rothwell

Stroke. 2010;41:1290-1293; originally published online April 8, 2010;
doi: 10.1161/STROKEAHA.110.579888
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2010 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/41/6/1290

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/