Validity and Applications of the Montreal Cognitive Assessment for the Assessment of Vascular Cognitive Impairment

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Key Words
Review · Cognitive screening · Stroke · Dementia · Mild cognitive impairment

Abstract
Cognitive impairment is common among patients with stroke or other cerebrovascular disease and influences long-term outcome, including occupational functioning. Recognition and monitoring of mild cognitive impairment is thus essential to good patient care. The Montreal Cognitive Assessment (MoCA) has been suggested as a brief screening test of vascular cognitive impairment. This paper presents a critical review of the research literature evaluating the validity and utility of this test with the aim of informing future clinical and research practice. A total of 30 papers employing the MoCA in the context of cerebrovascular disease were identified. Reporting of the methods and results of such studies tended to fall short of the established reporting guidelines. Under-specification of the exclusion criteria applied and their impact make it difficult to assess the potential impact of sampling bias and loss to follow-up. Nevertheless, content validity evidence suggests that the MoCA covers most of the domains that represent cognitive impairment in cerebrovascular disease, with mixed evidence for its preferential sensitivity to the type of cognitive impairment encountered in the context of vascular disease. Evidence clearly supports the need to establish norms and cut-offs for the MoCA that are culturally appropriate and that are matched to the range of cognitive impairment that is present in the population being assessed. Recent modifications of the MoCA have been developed for assessing patients with visual impairment or restricted mobility, which may reduce the impact of ‘untestability’ on cognitive screening in the clinic or research context. The MoCA correlates well with other measures of cognitive and functional abilities in patients with cerebrovascular disease, and may also predict future response to rehabilitation and long-term occupational outcome. Further research is needed to provide evidence for the validity of the MoCA in longitudinal studies. However, it compares favourably to the Mini Mental State Examination as a screening test that is sensitive to the milder forms of cognitive impairment that often accompany cerebrovascular disease.

Background
Cognitive impairment is a common consequence of stroke with a significant impact on response to rehabilitation, ability to return to work and ability to resume participation in society. Traditionally, vascular cognitive impairment was understood uniquely as a condition result-
Heart failure and diabetes specifically by cerebrovascular disease were excluded (e.g. or leukoaraiosis. Studies in samples that were not defined internal carotid artery (ICA), silent cerebral infarct and/ ischaemic attack (TIA), occlusion or stenosis of the various cerebrovascular conditions, including stroke, tran- MoCA was administered to patients diagnosed with var-

ty of other tests in stroke patients; since they reported no evidence relevant to the validity of the MoCA for assess-
ment of cognitive impairment, these papers were exclud-
ed. Reference lists of included papers were examined to iden-
tify additional papers for inclusion. The resulting set of 29 studies was examined in detail to extract key features of their methodology and results. Characteristics of these studies are shown in table 1, including population sam-
pwed, sample size, eligibility criteria, proportion of popu-
lation for which data were analysed, mean age and stan-
d deviation, presence/absence of a control group, use of education-correction on the MoCA score, timing and language of assessment.

Quality of Reporting

The population most commonly sampled was patients presenting with stroke or TIA, with approximately half of the studies indicating that the event was neuroradiologi-
cally confirmed. Time elapsed since stroke ranged from 24 h to 5 years. A handful of studies examined data from patients with radiologically confirmed ischaemic lesions, where the time elapsed since the ischaemic event was not a factor, e.g. clinically silent small vessel disease [33]. Car-
tid artery disease was the focus of 4 studies, while aneu-
rysma subarachnoid haemorrhage (SAH) was studied in 2 others. The setting was routinely reported and the pe-
riod of recruitment was reported in 57% of studies.

Exclusion criteria were reported with varying degrees of specificity. A total of 63% of studies reported excluding patients deemed untestable by reason of severe sensory, motor or speech impairment, while 40% reported excluding those with dementia or premorbid cognitive decline. Many papers reported on secondary analyses of data col-
clected from preselected samples of patients who were par-
ticipants in a larger cohort study or trial. Only 27% of papers indicated whether poor fluency in the test lan-
guage was an exclusion criterion and, surprisingly for a test with over 40 translations, only 67% explicitly report-
ed the language in which MoCA was administered. Flow charts of subject recruitment and exclusions as recom-
med by STARD and CONSORT guidelines were rare-
ly provided. Only 40% of studies provided sufficient data to calculate recruitment rates, and this generously in-
cludes papers that reported simply that all patients pro-
vided informed consent. Very few papers provided data on the number of patients who were eligible for the study by reason of having the index condition (e.g. stroke) but were subsequently excluded for inability to complete a valid MoCA [16, 21, 23, 28]. Information on the number of exclusions was provided in 43% of studies, including secondary or follow-up analyses of data sets that reported only on drop-out rates, resulting in missing data. A scant 17% made some effort to assess sampling bias by compar-

MoCA for Vascular Cognitive Impairment

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Table 1. Studies using the MoCA in cerebrovascular disease, by population sampled

<table>
<thead>
<tr>
<th>Year</th>
<th>Ref</th>
<th>First author</th>
<th>Population</th>
<th>Setting/recruitment period</th>
<th>Exclusion criteria</th>
<th>Report rate</th>
<th>Report excl.</th>
<th>Analysed n for sample bias</th>
<th>Age (mean ± SD)</th>
<th>Education correction</th>
<th>Time</th>
<th>Control group</th>
<th>MoCA language</th>
</tr>
</thead>
<tbody>
<tr>
<td>2007</td>
<td>[12]</td>
<td>Popovic</td>
<td>CT-confirmed first ischaemic stroke or TIA</td>
<td>UH neurology admissions</td>
<td>≤45 yo; Ne, Psy, Aph, MMSE ≤20, memory complaints</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>110</td>
<td>56±7</td>
<td>&lt;2 d</td>
<td>CV risk factors</td>
<td>Croatian</td>
</tr>
<tr>
<td>2009</td>
<td>[13]</td>
<td>Wong A</td>
<td>MRI-confirmed small vessel ischaemic stroke</td>
<td>UH acute stroke unit or neurology clinic</td>
<td>Ne, Psy, Sys, Mo, CDR &gt;0.5, cortical infarct</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>40</td>
<td>70±8.5</td>
<td>3 mo</td>
<td>healthy</td>
<td>Chinese (Hong-kong/ Cantonese)</td>
</tr>
<tr>
<td>2010</td>
<td>[14]</td>
<td>Dong</td>
<td>acute (&lt;14 days) CT/MRI-confirmed ischaemic stroke or TIA</td>
<td>Admission to UH for stroke/11 months</td>
<td>&lt;21, Psy, Mo, Aph, dementia, premorbid cognitive decline per IQCODE, severe disability per Rankin scale</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>100</td>
<td>61.2±11.3</td>
<td>&lt;2 wk</td>
<td>memory clinic</td>
<td>SEng/ Chinese/ Malay</td>
</tr>
<tr>
<td>2010</td>
<td>[15]</td>
<td>Pendlebury</td>
<td>first stroke or TIA with 6 months or 5 years of follow-up</td>
<td>Participants in PBC study of vascular events/18 months</td>
<td>Ne, Sys, Mo, Aph, He, Vis, dementia, learning disability, poor in test language</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>413</td>
<td>69.9±12.4</td>
<td>6 mo or 5 yr</td>
<td>none</td>
<td>English</td>
</tr>
<tr>
<td>2011</td>
<td>[16]</td>
<td>Cumming</td>
<td>stroke</td>
<td>Participants in multi-centre acute stroke (&lt;24 h) rehab trial</td>
<td>&lt;18, Sys, Aph, He, Vis, Mo, mild disability per Rankin scale, poor in test language, unstable/abnormal physiological levels e.g. blood pressure, heart</td>
<td>partial</td>
<td>yes</td>
<td>no</td>
<td>294</td>
<td>70.6±11.9</td>
<td>3 mo</td>
<td>none</td>
<td>English</td>
</tr>
<tr>
<td>2011</td>
<td>[17]</td>
<td>Fan</td>
<td>MRI-confirmed acute stroke (&lt;2 weeks)</td>
<td>Admission to hospital neurology service for stroke/5 months</td>
<td>&lt;45, Aph, Mo, decreased consciousness, premorbid cognitive decline per IQCODE, cannot complete testing</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>53</td>
<td>55–75</td>
<td>&lt;2, 6 and 12 wk</td>
<td>none</td>
<td>n.s.</td>
</tr>
<tr>
<td>2011</td>
<td>[18]</td>
<td>Godefroy</td>
<td>acute (&lt;3 weeks), mild-moderate stroke</td>
<td>Hospital acute stroke unit/5 months</td>
<td>Psy, cannot complete neuropsychological testing, mental retardation, severe TBI, illiteracy, poor in test language, severe stroke per NIHSS</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>95</td>
<td>68.2±13.7</td>
<td>&lt;21 d</td>
<td>none</td>
<td>French</td>
</tr>
<tr>
<td>2011</td>
<td>[19]</td>
<td>Kandiah</td>
<td>acute MRI-confirmed lacunar stroke</td>
<td>All stroke patients at neurosciences institute/7 months</td>
<td>Ne, Psy, Sys, Mo, Aph, He, Vis, premorbid cognitive impairment, &lt;6 years education, no informant, no MRI, acute cerebral haemorrhage, large cortical stroke</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>145</td>
<td>55.8±11.9</td>
<td>mean: 109 d</td>
<td>stroke without cognitive impairment</td>
<td>n.s.</td>
</tr>
<tr>
<td>2011</td>
<td>[20]</td>
<td>MacKenzie</td>
<td>stroke or TIA patients with hypertension at risk of medication non-adherence</td>
<td>Stroke prevention clinic participants in intervention feasibility study/7 months</td>
<td>&lt;18, unable to report admission history, poor in test language</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>20</td>
<td>67.5±16</td>
<td>n.s.</td>
<td>none</td>
<td>n.s.</td>
</tr>
<tr>
<td>Year</td>
<td>Ref First author</td>
<td>Population</td>
<td>Setting/recruitment period</td>
<td>Exclusion criteria</td>
<td>Report recruit rate</td>
<td>Report excl.</td>
<td>Analysed n</td>
<td>Age (mean ± SD)</td>
<td>Education correction</td>
<td>Time</td>
<td>Control group</td>
<td>MoCA language</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>2011</td>
<td>[21] Pendlebury</td>
<td>acute (1–14 days) minor stroke or TIA</td>
<td>PBC study of vascular events</td>
<td>Sys, Mo, Aph, He, Vis, dementia, poor in test language, recurrent stroke by follow-up</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>253</td>
<td>73.5±11.8</td>
<td>n.s.</td>
<td>≥1 yr</td>
<td>non-CV English</td>
<td></td>
</tr>
<tr>
<td>2011</td>
<td>[22] Toglia</td>
<td>adult subacute stroke</td>
<td>Stroke cases admitted to rehab unit at UH/18 months</td>
<td>poor language comprehension, incomplete assessments</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>72</td>
<td>70±17</td>
<td>+1 for &lt;12 yr</td>
<td>median 8.5 d</td>
<td>n.s. English?</td>
<td></td>
</tr>
<tr>
<td>2012</td>
<td>[23] Dong</td>
<td>acute (&lt;14 days) CT or MRI-confirmed ischaemic stroke or TIA</td>
<td>Admission to UH for stroke/20 months</td>
<td>&lt;21, Psy, Sys, Mo, Aph, Vis, He, dementia, delirium, premorbid cognitive decline per IQCODE, poor in test language, severe disability per Rankin scale, loss to follow-up</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>239</td>
<td>60.2±11.8</td>
<td>method 1: +1 for ≤6 yr, method 2: regression-adjusted to local norms</td>
<td>&lt;2 wk &gt;&gt;3–6 mo</td>
<td>none</td>
<td>SEng/Chinese/Malay</td>
</tr>
<tr>
<td>2012</td>
<td>[24] Hwang</td>
<td>first hemiparetic stroke, chronic (&lt;6 months)</td>
<td>Recruited by brochure in a community health centre</td>
<td>Ne, Se, Mo, Vis, cognitive complaints, orthopaedic, non-ambulatory</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>47</td>
<td>55±9</td>
<td>n.s.</td>
<td>&gt;6 mo</td>
<td>None</td>
<td>n.s. Korean?</td>
</tr>
<tr>
<td>2012</td>
<td>[25] Ihara</td>
<td>MRI-confirmed small subcortical stroke</td>
<td>Neurology clinic/34 months</td>
<td>mild-to-moderate leukoaraisis</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>12</td>
<td>76±8.7</td>
<td>n.s.</td>
<td>intake</td>
<td>none</td>
<td>Japanese</td>
</tr>
<tr>
<td>2012</td>
<td>[26] Kasai</td>
<td>Ivery mild MRI-confirmed small vessel disease</td>
<td>Kurihama community-dwelling elderly cohort</td>
<td>&lt;75 yr, Ne, &lt;5 lacunar infarcts, large cortical lesions, absence of neurological signs, CDR memory ≠ 0.5, Trails A or B score &lt;1 SD above mean &lt;75, infarcts &lt;4 mm</td>
<td>yes</td>
<td>no¹</td>
<td>no</td>
<td>37</td>
<td>~80</td>
<td>+1 for ≤12 yr</td>
<td>intake</td>
<td>healthy</td>
<td>Japanese</td>
</tr>
<tr>
<td>2012</td>
<td>[27] Marzolini</td>
<td>ambulatory, hemiparetic subacute (≥10 weeks) stroke</td>
<td>participants in a risk factor modification and exercise program</td>
<td>Ne, contraindications to maximal exercise testing, non-completers of intervention</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>41</td>
<td>63.5±13.5</td>
<td>+1 for ≤12 yr</td>
<td>&gt;10 wk post-onset, 6 mo post rehab start</td>
<td>none</td>
<td>n.s. English?</td>
</tr>
<tr>
<td>2012</td>
<td>[28] Pendlebury, Mariz</td>
<td>stroke or TIA with 1 or 5 years follow-up data</td>
<td>participants in PBC study of vascular events/15 months</td>
<td>Sys, Aph, Mo, He, Vis, poor in test language, nursing home residents, missing data</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>91</td>
<td>73.4±11.6</td>
<td>n.s.</td>
<td>1 or 5 yr</td>
<td>none</td>
<td>English</td>
</tr>
<tr>
<td>2012</td>
<td>[29] Pendlebury, Markwick</td>
<td>stroke or TIA with ≥26 months follow-up data</td>
<td>participants in PBC study of vascular events/19 months</td>
<td>Sys, Mo, Aph, MMSE &lt;24, poor in test language</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>86</td>
<td>TIA: 71.4±11.9, &lt;12 yr stroke: 71.4±12.2</td>
<td>&gt;6 mo memory</td>
<td>English study</td>
<td></td>
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<tr>
<td>Year</td>
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<td>Report recruit rate</td>
<td>Report excl.</td>
<td>Analysed n for sample bias</td>
<td>Age (mean ± SD)</td>
<td>Education correction</td>
<td>Time</td>
<td>Control group</td>
<td>Carotid Artery Disease</td>
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</tr>
<tr>
<td>2013</td>
<td>[30]</td>
<td>Pendlebury, Welch</td>
<td>minor stroke or TIA with 1 or 5 years follow-up data</td>
<td>participants in PBC study of vascular events</td>
<td>n.s.</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>91</td>
<td>73.4±7</td>
<td>+1 for ≤12 yr</td>
<td>&gt;1 yr</td>
<td>none</td>
</tr>
<tr>
<td>2012</td>
<td>[31]</td>
<td>Yuan</td>
<td>MRI-confirmed small vessel disease</td>
<td>admitted to UH neurology department, referred for MRI/6 months</td>
<td>&lt;60, Ne, Sys, Psy, severe cognitive impairment, contraindication for MRI, disability in instrumental activities of daily living</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>46</td>
<td>72±6</td>
<td>+1 for ≤12 yr</td>
<td>intake</td>
<td>healthy Chinese (Beijing)</td>
</tr>
<tr>
<td>2012</td>
<td>[32]</td>
<td>Zhang</td>
<td>stroke</td>
<td>cerebral infarct patients treated in hospital/25 months</td>
<td>&lt;60, &gt;75 yo</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>80</td>
<td>66.5±5.6</td>
<td>n.s.</td>
<td>intake</td>
<td>pre-treat, none 3 mo</td>
</tr>
<tr>
<td>2012</td>
<td>[33]</td>
<td>Zhao</td>
<td>MRI-confirmed small vessel disease</td>
<td>3 city hospitals/21 months</td>
<td>Ne, large cortical lesions</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>112</td>
<td>60.2±6.9</td>
<td>+1 for 12 yr</td>
<td>intake</td>
<td>normal MRI</td>
</tr>
<tr>
<td>2013</td>
<td>[34]</td>
<td>Blackburn</td>
<td>acute (&lt;14 days) stroke or high-risk TIA</td>
<td>admission to hyperacute stroke unit</td>
<td>&lt;18, Psy, Mo, Aph, major physical disability</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>50</td>
<td>66.2±5.5</td>
<td>n.s.</td>
<td>&lt;15 d</td>
<td>none</td>
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<tr>
<td>2009</td>
<td>[35]</td>
<td>Martincic-Popovic</td>
<td>asymptomatic severe carotid artery stenosis/occlusion</td>
<td>admissions to UH neurology department referred for ultrasound of carotid arteries</td>
<td>ischaemic stroke, left-handed</td>
<td>no1</td>
<td>partial no</td>
<td>26</td>
<td>66.3±8.7</td>
<td>n.s.</td>
<td>intake</td>
<td>none</td>
<td>Croatian</td>
</tr>
<tr>
<td>2011</td>
<td>[36]</td>
<td>Popovic</td>
<td>asymptomatic severe carotid artery stenosis/occlusion</td>
<td>admissions to UH neurology department referred for ultrasound of carotid arteries/13 months</td>
<td>ischaemic stroke, Psy, Sys, He, Vis, dementia, cognitive complaints, left-handed</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>70</td>
<td>67.5 (45–85)</td>
<td>n.s.</td>
<td>intake</td>
<td>other neuro-logic</td>
</tr>
<tr>
<td>2012</td>
<td>[37]</td>
<td>Baracchini</td>
<td>severe unilateral carotid disease, booked for endarterectomy</td>
<td>surgical service/29 months</td>
<td>&lt;65, major stroke, contralateral severe carotid disease, Psy, dementia, MMSE &lt;24, mental retardation</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>138</td>
<td>symptomatic: 74.9±6.7, asymptomatic: 74.4±6.1</td>
<td>n.s.</td>
<td>pre-surgery, 3 mo post, 12 mo post</td>
<td>n.s.</td>
</tr>
<tr>
<td>2012</td>
<td>[38]</td>
<td>Fu</td>
<td>mild-to-moderate carotid artery disease</td>
<td>participants in intervention study for elderly patients with carotid artery disease</td>
<td>ischaemic stroke, severe carotid artery disease, Ne, Sys, endarterectomy</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>407</td>
<td>high ability: 71.6±4.6, low ability: 72.7±5.1</td>
<td>n.s.</td>
<td>intake</td>
<td>none</td>
</tr>
</tbody>
</table>
MoCA for Vascular Cognitive Impairment

Content Evidence

This source of validity information examines the relationship between the MoCA test and the construct it is intended to measure, i.e., vascular cognitive impairment. The content of the MoCA was selected based on the clinical expertise of its primary author regarding the eight domains of cognitive impairment commonly encountered in patients with clinically diagnosed MCI and healthy controls [42]. The MoCA includes items that place a demand on visuospatial and executive functions, suggesting that it might also be sensitive to the type of cognitive impairment that is most characteristic of patients with vascular disease. Consistent with this hypothesis, patients with stroke and stroke, cognitive impairment performed worse than patients participating in memory and aging study on the visuospatial/executive, fluency, attention and executive functions [14, 19]. The MoCA includes items that place a demand on visuospatial and executive functions, suggesting that it might also be sensitive to the type of cognitive impairment that is most characteristic of patients with vascular disease. Consistent with this hypothesis, patients with stroke and stroke, cognitive impairment performed worse than patients participating in memory and aging study on the visuospatial/executive, fluency, attention and executive functions [14, 19].

Table 1. (continued)

<table>
<thead>
<tr>
<th>Year</th>
<th>Ref</th>
<th>First author</th>
<th>Population</th>
<th>Setting/recruitment period</th>
<th>Exclusion criteria</th>
<th>Report rate</th>
<th>Report excl.</th>
<th>Analysed n for sample bias</th>
<th>Age (mean ± SD)</th>
<th>Education correction</th>
<th>Time</th>
<th>Control group</th>
<th>MoCA language</th>
</tr>
</thead>
<tbody>
<tr>
<td>2012</td>
<td>[39]</td>
<td>Schweizer</td>
<td>aneurysmal SAH with good outcome</td>
<td>research database at large city hospital</td>
<td>n.s.</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>32</td>
<td>55.2±7.8</td>
<td>n.s.</td>
<td>&gt;6 mo post-onset</td>
<td>none</td>
</tr>
<tr>
<td>2012</td>
<td>[40]</td>
<td>Wong G</td>
<td>acute (&lt;96 h) spontaneous aneurysmal SAH</td>
<td>multicentre study/2 years</td>
<td>&lt;25, &gt;75, Ne, Aph, non-responsive to commands, dementia, cognitive impairment, poor in test language, cerebrovascular disease, lost to follow-up</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>90</td>
<td>54±11</td>
<td>+1 for &lt;12 h</td>
<td>&gt;3 mo post-onset</td>
<td>none</td>
</tr>
</tbody>
</table>

Ref = Reference number; n = number patients included; n.s. = not specified; d = day; wk = week; mo = month; yr = year; onset = date of stroke onset; intake = at study enrollment; >> = time to outcome predicted by baseline assessment; CV = cerebrovascular; UH = university hospital; PBC = population-based cohort; SEng = Singaporean English; NIHSS = national institutes of health stroke scale.

Comorbid exclusions abbreviated as follows: Ne = Other neurologic disorder; Psy = psychiatric disorder; Sys = other systemic disorder (e.g., heart failure).

Commonly specified exclusion criteria for patients deemed ‘untestable’ by reason of impairments are Mo = Motor; Se = any sensory; Vis = visual; He = hearing; Aph = aphasia.

1 List of eligibility criteria is incomprehensible.
subtests [29]. Subtest differences could be seen even among those whose Mini Mental Status Examination (MMSE) scores were within normal limits. Second, performance on the visuospatial/executive subtest is worse among patients with occlusion/stenosis of the ICA than among patients with vascular risk factors whose ICA is normal, and worse still for ICA patients with more than 2 vascular risk factors [35, 36]. In contrast, 1 study found no group differences on any subtest scores when comparing patients with mild-to-moderate vascular dementia with a group of patients with mild-to-moderate Alzheimer’s disease who were carefully matched for age, gender, education and total MMSE score [44]. The failure to see any group differences in the latter study could be explained by the inclusion of patients with greater cognitive impairment than those included in other studies. The breadth of cognitive domains affected increases with the progression of dementia, with a resulting decline in the ability of cognitive tests to discriminate between different neuropathologies.

Response Process

This source of validity evidence examines the match between the construct (cognition) and the processes that a respondent actually engages in when encountering a question. For example, it is assumed that a person who is asked to name a picture of a lion on the naming subtest of the MoCA is engaged in the process of activating their semantic representations of this animal and subsequently retrieving a verbal label for it from long-term memory. This assumption may be unwarranted if, for example, the patient is visually impaired and thus unable to perceive the animal accurately. Functional limitations that are common to stroke populations may alter the response process and thus preclude assessment of cognitive ability using the MoCA. Pendlebury et al. [15] found that 12.4% of a population-based stroke/TIA cohort were unable to complete the MoCA 6 months after stroke, and 21.6% were untestable 5 years after the initial event. Reasons included dysphasia, dementia, poor fluency in test language and poor vision, among others. Similar results were obtained in a study examining the feasibility of administering the MoCA as a measure of cognitive outcome in a large trial examining the impact of early rehabilitation after stroke [16]. Despite the fact that these patients had already pre-screened as eligible to participate in a clinical trial, the authors found that of 294 patients assessed at 3 months after stroke, only 75% had complete MoCA data. In 18% of the sample, MoCA data were missing either entirely or in part due to telephone assessment, aphasia or inability to hold a pencil.

In an effort to reduce missing data for patients who cannot attend follow-up assessment, Pendlebury et al. [30] developed a telephone version of the MoCA. The telephone MoCA was compared with the in-person MoCA as a predictor of MCI among patients assessed 1 or more years after a stroke or TIA. Performance on complex language items was worse during telephone administration than during conventional administration of the MoCA, even after excluding patients with hearing impairment. This evidence suggests that the cognitive processes engaged in by individuals assessed by telephone are not identical to those performed by patients assessed in person, thus the two versions may assess slightly different constructs. Interestingly, this study demonstrated good accuracy for detecting MCI among stroke/TIA patients when the 22 items of the telephone version were extracted from the results of face-to-face testing with the MoCA, suggesting that this subset of items is valid for the detection of impairment in visually impaired patients.

Internal Structure

This source of evidence covers aspects of the dimensionality and reliability of a test. Internal consistency of the MoCA, i.e. the extent to which all items measure the same construct, tends to be in the moderate-to-high range, with a Cronbach’s alpha of 0.72 in patients with small vessel disease [13], 0.78 in mild subacute stroke patients undergoing rehabilitation [22], 0.86 in patients studied 3 months after a stroke [16], and 0.83 in patients with vascular dementia [44]. This is comparable to the level of internal consistency found in geriatric community or memory clinic samples using various language versions of the MoCA [42, 44–46]. Freitas et al. [44] observed significant positive correlations between scores on individual MoCA subtests and the total MoCA score in patients with vascular dementia, with coefficients ranging from 0.49 for memory to 0.78 for attention, concentration and working memory.

The factor structure of the Portuguese MoCA was examined in a mixed clinical sample of 212 patients with various dementia types including 12% vascular dementia, although it has not been examined in a group selected for vascular cognitive impairment. Confirmatory factor analysis showed good fit to a 2-factor model composed of memory and attention/executive functions [47]. Analyses
in a larger population of patients with MCI or Alzheimer’s disease and demographically matched normal controls suggested good fit to 1-factor (unidimensional ‘cognition’), 2-factor (memory and attention/executive functions) and 6-factor models (based on the subtest structure proposed by Nasreddine et al. [42]), with the 6-factor model yielding the best fit [48]. Dimensionality in an exclusively cerebrovascular sample has yet to be examined.

Only 1 study in cerebrovascular disease examined test-retest reliability, obtaining a high correlation (0.96) in ischaemic stroke patients with small vessel disease and matched controls tested 2 weeks apart [13]. Studies in older persons from community or memory clinic-based populations have yielded slightly lower reliabilities (0.75–0.92); however, these tended to involve longer test-retest intervals in the range of 4–8 weeks or longer [45–47, 49–51]. The reliability of newly available alternate forms of the MoCA has been established for memory clinic samples comprised of MCI and Alzheimer’s disease [52], but not for patients with cerebrovascular disease. Inter-rater reliability was 0.87 for the Hong Kong version of the MoCA in patients with small vessel disease and 0.95 for the Chinese version of the MoCA in patients with MCI or Alzheimer’s disease [45].

**Consequences**

Another type of validity evidence relates to the consequences of obtaining a given score on the MoCA, and includes assessments of classification accuracy (impaired vs. unimpaired) and the process of establishing cut-offs. A multitude of studies comparing the MoCA to the MMSE as a cognitive screening tool were unanimous in concluding the following: (1) MoCA scores are normally distributed, whereas MMSE scores show ceiling effects, (2) the prevalence of cognitive impairment among patients with cerebrovascular disease using the originally published cut-off scores is greater when assessed using the MoCA than when using the MMSE and (3) while a substantial number of those who pass the MMSE fail the MoCA, the converse is rarely true. This has been demonstrated in clinical samples composed of patients with any stroke [18, 22], any stroke or TIA [12, 14, 15, 20, 21, 29, 34], small-vessel disease or silent cerebral infarct [25, 33], SAH [39, 40] and stenosis or occlusion of the ICA [35, 36].

These results must be seen as weak forms of evidence for the relative sensitivities of the MoCA and the MMSE since the majority of the easiest questions are found on the MMSE rather than on the MoCA, at least in memory clinic patients [53]. Studies using receiver operating characteristic (ROC) curves to establish the optimal diagnostic cut-off point illustrate clearly that the MoCA’s greater sensitivity to impairment is a function of the choice of a conventional cut-off point favouring sensitivity over specificity. Kasai et al. [26] examined the ability of the MoCA to predict a Clinical Dementia Rating (CDR) Scale score of 0.5 or higher among a sample of 392 older community-dwelling participants. The optimal MoCA cut-off based on ROC curves yielded sensitivity of 0.72 and specificity of 0.67 for any impairment versus no impairment, although sensitivity declined to 0.63 (specificity 0.67) when discriminating between MCI (CDR 0.5) and no impairment (CDR 0), and the total scores for these two groups showed considerable overlap. The MoCA did discriminate 37 individuals with very mild small vessel disease as confirmed by MRI and mild cognitive deficits from 164 cognitively normal individuals (area under the curve 0.83, sensitivity 0.78 and specificity 0.74). Notably, however, the MMSE and a combination formed of the MMSE plus MoCA subtests including visuospatial/executive items performed as well as the MoCA at discriminating among those with and without cognitive impairment of any type.

Two studies in patients examined the ability of the MoCA to detect cognitive impairment as defined by neuropsychological assessment. In 90 patients with mild-to-moderate stroke severity, a cut-off of <24 on the MoCA resulted in sensitivity of 0.88 and specificity of 0.71 for detection of cognitive impairment (including dementia), whereas lowering the cut-off to <23 yielded a specificity of 0.77 while lowering sensitivity to 0.78 [18]. In that study, the sensitivities and specificities obtained with optimal MMSE cut-offs were comparable to those obtained for the MoCA. It may be noted that this study has been criticized for the lengthy delay (1–6 months) between acute post-stroke screening with the MoCA and subsequent subacute neuropsychological assessment [54]. A second study assessed the validity of the MoCA in 91 non-demented stroke or TIA patients studied at least 1 year after stroke or TIA [28]. A cut-off of <26 yielded sensitivity of 0.87 and a specificity of only 0.63, whereas a cut-off of <25 increased specificity to 0.82 while reducing sensitivity to 0.77. Sensitivity for the MMSE with a cut-off of <29 was 0.77, with a specificity of 0.78. Similar results were obtained in a third study in 239 patients with stroke or TIA that examined the ability to discriminate between individuals with normal cognition or minimal cognitive impairment and those with moderate-to-se-
vere cognitive impairment [23]. Discriminant validity estimates for the MoCA and the MMSE were comparable, with good sensitivity (approx. 0.88) and moderate specificity (approx. 0.65). Caution is indicated when attempting to select a cut-off based on the results of these studies; the optimal cut-off for detection of cognitive impairment will tend to be lower when the MoCA is used in patient populations that include patients with greater cognitive impairment.

Cognitive test performance may be affected by differences in age, educational level and culture. A cut-off of 26, with 1 point added for <13 years of education, was established for the original Canadian French and English versions of the MoCA [42]. Here, 6 of the studies reviewed explicitly reported using the published education correction, whereas most did not indicate whether education correction was applied for the MoCA, and none of the studies that used established norms explicitly mentioned education-correction for the MMSE scores. Lower cut-offs are required to optimize detection of MCI in memory clinic populations using the Korean [49] and Portuguese [55] language versions of the MoCA, and even in a US community-based sample tested in English [56]. To date, only 2 of the studies conducted in cerebrovascular disease collected normative data in their own cultural milieu to establish appropriate cut-offs and evaluate the impact on diagnostic accuracy of adjusting MoCA scores based on demographic variables. In a French study, correcting the scores of stroke patients for age and education resulted in similar rates of sensitivity (approx. 0.70) and specificity (approx. 0.90) for both tests and yielded no improvement in sensitivity over uncorrected scores [18]. In a Singaporean study of patients with stroke or TIA, significant predictors of screening test scores included age, education, ethnicity, history of smoking and ischaemic heart disease, history of stroke or TIA, and stroke severity. Optimal and comparable accuracies for detecting moderate-to-severe cognitive impairment were obtained for both screening tests when the scores were corrected for these variables [23].

**Relations to Other Variables**

The validity of the MoCA is supported by evidence of associations between total scores and clinical variables relevant to cerebrovascular disease. The MoCA is sensitive to differences in severity of vascular disease, as demonstrated by studies showing lower scores among patients with stroke than in those with TIA only [12, 28]. In patients with ischaemic leuкоariosis, 2 studies showed a correlation between total MoCA score and the number [25] or severity [31] of lacunar/small vessel infarcts, although another study found no association between MoCA performance and the presence versus absence of lacunar or small vessel infarcts [26]. In patients with SAH, worse MoCA performance was found in those patients who suffered delayed cerebral ischaemia [40]. In patients with stenosis/occlusion of the ICA, diabetes, hypertension, hyperlipidaemia and elevated body mass index, but not laterality or bilaterality of occlusion, lower MoCA test scores were predicted [35–37]. Among patients with mild-to-moderate carotid artery disease, lower common carotid artery flow velocity was observed among those who obtained <26/30 on the MoCA [38].

A handful of studies have examined the validity of the MoCA as a cognitive assessment tool in terms of its convergence with other clinically relevant outcomes after stroke. At 3 months after ischaemic stroke with small vessel disease, MoCA scores correlated with composite scores derived from neuropsychological tests of executive and non-executive cognitive functions [13]. At 3 months after aneurysmal SAH, MoCA scores correlated with activity limitations as measured by performance on instrumental activities of daily living and on the modified Rankin scale, as well as with measures of depressive symptoms [40]. Notably, in the latter study, the MoCA did not outperform the MMSE at predicting concurrent functional outcomes, provided the cut-offs were selected based on ROC curves. At 6 months or more after aneurysmal SAH onset, scores on the MoCA subtests of naming and abstraction correlated significantly with return to work [39]. In the chronic stages after hemiparetic stroke (6+ months), MoCA performance was worse among patients who had fallen in the previous 6 months, and correlated with measures of gait quality and speed, as well as postural control [24]. A mixed group of stroke and TIA patients continued to show correlations between impaired MoCA scores and activity limitations at 6 months or 5 years after onset [15]. Finally, in a study of patients surviving to 5 years after a mild stroke or TIA, lower MoCA test scores were observed in those who had demonstrated transient cognitive impairment after stroke, as indexed by recovery of 2+ MMSE points by 1 month after stroke [21].

In stroke patients who were admitted to a rehabilitation unit, poorer MoCA scores at admission correlated with greater functional dependency in motor-specific activities at discharge and lower rates of functional im-
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Sensitivity to Change

To be valid for use in clinical trials, scores on the MoCA must be sensitive to change over time, as a function of natural recovery from or progression of a disorder or in response to an intervention targeting cognitive ability. Large-scale longitudinal studies of cognitive impairment report improvements in cognitive status between the early acute stages of stroke and 3-month follow-up, with relatively stable performance and rates of cognitive impairment up to 5 months after the index event [57]. To date, 2 studies have administered the MoCA at repeated time points to assess cognitive improvement after a vascular event. In the first ever study to measure vascular cognitive impairment using the MoCA [12], 110 patients with first stroke or TIA were assessed with the MoCA and the MMSE at intake and 3 and 6 months later, and compared with a group of 45 patients with vascular risk factors but no evidence of cerebrovascular disease. Group medians for both tests tended to decline over the 6 months of follow-up in both patient groups, but this was particularly noticeable in the MoCA scores, which had already declined into the abnormal range by the 3-month follow-up. Patients with stroke showed a greater decline in MoCA scores over 6 months than did patients with TIA or the control group with vascular risk factors but no cerebrovascular disease. The implications of this study for the MoCA’s sensitivity to change must be viewed with caution due to the anomalous finding of cognitive decline, rather than cognitive recovery, following cerebrovascular accident. It is not entirely clear from the paper whether all patients initially assessed were retained for follow-up or whether those who completed all assessments represented a subgroup of the original sample who were at greater risk of decline. More typical results were reported in a study in 98 Chinese patients who were administered the MoCA and the MMSE within 2 weeks and at 6 and 12 weeks after stroke onset [17]. Scores on both tests improved by 12 weeks, while the incidence of cognitive impairment as identified using cut-off scores decreased. All subtests of the MoCA improved significantly in the course of recovery. Caution is also warranted in interpreting this evidence of sensitivity to change, as the number of patients assessed increased from 53 to 71 across the three time points.

The MoCA served as the primary outcome measure in several recent clinical trials of interventions aimed at improving cognitive functioning in patients with cerebrovascular disease. Non-demented symptomatic and asymptomatic patients with severe carotid disease were assessed with the MoCA before undergoing carotid endarterectomy, and at 3- and 12-month post-operative follow-up [37]. The symptomatic endarterectomy group tended to improve 2+ points from baseline to 12-month follow-up and showed significantly greater change scores than a gall bladder surgery control group, while the asymptomatic endarterectomy group fell between. No changes were observed in the scores from the MMSE, suggesting that the MoCA may be more sensitive to change than the MMSE. At 3 months after a stroke, patients randomized to treatment with combination therapy (aspirin + gingko biloba) were observed to show improvements in total MoCA scores relative to baseline and relative to patients treated with aspirin alone [32]. Finally, patients with motor impairment >10 weeks after stroke participated in a 6-month aerobic and resistance training program [27]. MoCA performance improved over time, particularly on visuospatial/executive and attention subtests, and the improvement in cognition correlated with improvements in fat-free mass of the unaffected limb.

Discussion and Conclusions

The literature to date provides some evidence that the MoCA covers the range of content that is required for the assessment of cognitive impairment in cerebrovascular disease, with the exception of mental processing speed. No studies have yet examined the match between MoCA content and the domains assessed in a full or minimal neuropsychological test battery. There is consensus that MoCA covers domains not assessed by the MMSE. However, evidence that the visuospatial/executive subtest of the MoCA makes it preferentially sensitive to cognitive impairment in the context of vascular disease is mixed.

A 6-factor (subtest) model of the internal structure of the MoCA is supported by clinical data from mixed groups of patients that included those with vascular cognitive impairment. However, evidence suggests that the MoCA score also yields a reasonable measure of unidimensional cognitive ability, and shows internal consis-
tency in the moderately high range across various cerebrovascular disorders.

Stroke studies that included an age- and education-matched control group demonstrate the need to establish normative data appropriate to the socioeconomic, cultural and linguistic context in which stroke research is conducted and to revise the MoCA cut-offs as required. The jury is out on the value of education correction. Since low educational attainment is a known risk factor for developing dementia, the usefulness of correcting for this factor and the best method for doing so is unclear. The findings to date serve to illustrate that an over-reliance on published cut-off scores may weaken the discriminative validity of the MoCA.

Recent studies support the clinical experience that stroke can lead to the kinds of disability (e.g. aphasia and hemiplegia) that preclude the use of the MoCA to assess global cognitive impairment. In an aging stroke population, hearing loss and visual impairment are also problematic for administering a valid MoCA. This limits the generalizability of conclusions that can be drawn from epidemiological studies of cognitive impairment and its evolution after stroke, although it is recognized that other performance-based cognitive assessment tools suffer from the same limitation. In a clinical context, decisions about which patients cannot complete a valid MoCA will likely be left to the clinician’s best judgment. Experience with this and other tools in a clinical setting indicates that some clinicians choose to omit problematic items, interpreting the resulting score qualitatively and with caution. Recent efforts to develop adaptive versions of cognitive screening tests may help to overcome this limitation. For example, the telephone version or a 22-item short version of the MoCA show some utility for assessing post-stroke cognitive impairment in those who are visually impaired and/or unable to attend an in-person assessment.

Evidence is strong for associations between the MoCA and other measures of cognitive and functional abilities measured concurrently. The MoCA compares equally or favourably to the MMSE in terms of sensitivity to cognitive impairment and sensitivity to change over time. There is preliminary evidence that in a stroke rehabilitation context, MoCA performance predicts future response to rehabilitation and long-term occupational outcome, validating it as a tool with clinical relevance. Further evidence of the MoCA’s validity for the longitudinal assessment of vascular cognitive impairment is required, particularly estimates of test-retest reliability, inter-rater reliability, sensitivity to change, and clinically important change in stroke samples or other groups of cerebrovascular patients eligible for interventions that might be expected to target cognitive impairment.

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