The Montreal Cognitive Assessment (MoCA):

Concept and Clinical Review

1. Introduction

The Montreal Cognitive Assessment (MoCA) was developed as a brief screening instrument to detect Mild Cognitive Impairment.[1] It is a paper-and-pencil tool that requires approximately 10 minutes to administer, and is scored out of 30 points. The MoCA assesses multiple cognitive domains including attention, concentration, executive functions, memory, language, visuospatial skills, abstraction, calculation and orientation. It is widely used around the world and is translated to 36 languages and dialects. The test and instructions are freely available on the MoCA official website at www.mocatest.org. No permission is required for clinical or educational use.

This chapter will describe how each MoCA sub-test/domain, assesses various neuro-anatomical areas, and often overlapping cognitive functions. A comprehensive review of studies using the MoCA in multiple clinical settings and populations is provided. An algorithm for using the MoCA in clinical practice is suggested. In conclusion, MoCA limitations, future research and developments are discussed.

2. Cognitive Domains assessed by the MoCA

Visuospatial/Executive

Modified Trail Making Test

Beside visuomotor and visuoperceptual skills, the trail making test–B (TMT-B) requires mental flexibility to shift between numbers and letters which mainly rely on frontal lobe function [2–5]. In functional Magnetic Resonance Imaging (fMRI) studies, shifting ability in the TMT-B revealed greater activation relative to the trail making test A in the left dorsolateral and medial frontal cortices, right inferior and middle frontal cortices, right precentral gyrus, left angular and middle temporal gyri, bilateral intraparietal sulci [6–8]. A study of patients with frontal and non-frontal lobe lesions reported that all patients who had more than one error in the TMT-B had frontal lobe lesions. Specifically, patients with damage in the dorsolateral frontal area were mostly impaired[9]. Left frontal damage tended to cause more impairment than controls and right frontal damage groups, either for execution time or number of errors[10]. Nonetheless, specificity of the TMT-B to frontal lobe lesions is debated as one study reported comparable performance between frontal and non-frontal stroke patients[11].

Copy of the Cube

To copy a cube, subjects have to initially convert a two-dimensional contour to a three-dimensional cube. This ability is enhanced by learning experiences(12,13). After spatial planning, visuomotor coordination also plays a role in copying the cube. Various brain areas are involved; visual perception in the parieto-occipital lobe, planning in the frontal lobe, and integration of visual and fine motor sequences in the fronto-parieto-occipital cortices.

The cognitive mechanisms underlying performance in copying a figure are different according to the underlying disease. Alzheimer's disease (AD) patients with spatial perception/attention impairment had significant atrophy in the right parietal cortex. Complex two-dimensional figure copy were negatively associated with degree of right inferior temporal atrophy and reduction of cerebral blood flow in the right parietal cortex (14,15). Patients with behavioral variant fronto-temporal dementia had spatial planning and working memory dysfunction had significant atrophy in the right dorsolateral prefrontal cortex (16). A correlation between neuro-imaging and cube copying specifically, has not yet been reported.

Even though, a high proportion of either normal subjects (40%) or Alzheimer patients (76%) performed poorly on cube drawing on verbal command, persistent failure to copy a cube from a previously drawn cube is highly discriminative to detect patients with Alzheimer's disease (17). Less educated, older age, female and depressed subjects performed poorly in drawing-to-command and copying conditions.

The Clock Drawing Test

The Clock Drawing Test (CDT) has been widely used and studied for detection of dementia and mild cognitive impairment. Planning, conceptualization, and symbolic representation are involved in drawing a clock's face and in placing all the numbers correctly(18,19). Inhibitory response is required when placing each hand to tell the time of *"ten past eleven"*. Self-initiated-clock-drawing also requires intact visuoconstructive skills which are mainly represented in the parietal lobe.

In volunteers, fMRI demonstrated bilateral activation of the posterior parietal cortex and the dorsal premotor area during task performance suggesting the contribution of the parieto-frontal cortical networks to integrate visuospatial elements and motor control in self-initiated clock drawing(20).

In AD patients, errors in CDT were mainly conceptual and due to semantic memory impairment (21–23). This was supported by various neuroimaging studies that found negative correlation between CDT performance and atrophy of the right/left temporal cortices (24,25), atrophy of the medial temporal lobe (23), reduction in the activation of the left superior parietal lobe (26), and hypo metabolism of the right parietal cortex (27) in patients with cognitive impairment caused by AD pathology.

White matter hyperintensities (WMH) is also related to performance on CDT (23). Patients with severe WMH and patients with Parkinson's disease (PD) performed poorly and similarly on all subscales of CDT(28). Even though

both groups were different in term of neuropathology, they both have disrupted subcortico-frontal pathways. PD affects the subcortical dopaminergic pathway projecting to the prefrontal cortex (28,29).

The scoring criteria for the CDT in the MoCA has been simplified to decrease scoring complexity, scoring time, and minimize inter rater variability.

Despite the simpler scoring instructions, suboptimal inter and intra-rater reliability for MoCA's CDT were recently reported(30). CDT may be influenced by literacy status and education level (21,31,32,33).

<u>Naming</u>

The three animals in the MoCA (Lion, Rhinoceros and Camel) are infrequently seen in Western and even in Asian countries. The failure to name these animals may point to various types of cognitive impairment. If subjects cannot name but can give contextual information about the animal, for example, *"It lives in the desert (Camel)",* this could suggest either word finding difficulty or semantic memory impairment. If subjects cannot tell both the name and the context, they may have impaired visuoperceptual skills with inability to recognize the animal (failure in the cube copy and the CDT can support this possibility). They may also be impaired in both visuoperception and semantic memory such as in moderate to severe AD or advanced PD with dementia. Low education or cultural exposition to such animals can also be responsible.

In AD, impairment tend to reflect a breakdown in semantic processes which is different from visuoperceptual deficits caused by subcortical dementia such as Huntington's disease (HD) (33).Some studies have shown that semantic dysfunction is the primary cause of misnaming in both cortical or subcortical dementia (34,35).

The neuronal network involved in naming is category-dependent (36–39). In healthy subjects, the commonly activated regions were bilateral occipital lobes including the fusiform gyrus, and pars triangularis of the left inferior frontal gyrus (37–39). This activation pattern may be explained by processing of visual features and shape analysis, in the primary visual cortex and fusiform gyrus, and the subsequent retrieval process from semantic and conceptual knowledge of animals mediated by the pars triangularis of the left inferior frontal gyrus (39,40). Interestingly, animal naming was also associated with activation of the frontal regions linked to the limbic emotional system such as the left supplementary motor area and the anterior cingulate gyrus (37,38). It has also been shown that animal naming is more associated to primary visual cortex activation than naming of tools which is associated with the frontal and parietal lobe activation (premotor cortex and postcentral parietal cortex) (37).

<u>Attention</u>

The Digit Span

Digit Span Forward (DSF) measures retention of auditory stimuli and articulatory rehearsal. Digit span backward (DSB) requires transient working memory, and more demanding ability in transforming digits into a reversed order before articulating. This extra-step requires central executive processing (41).

Neuronal networks involved in digit span processing have been shown in many neuroimaging studies. In healthy subjects, using near-infrared spectroscopy (NIRS) a relationship between activation of the right dorsolateral prefrontal cortex and performance on DSB was observed (42). Other studies have shown greater activation of the bilateral dorsolateral prefrontal cortices, prefrontal cortex and left occipital visual regions for DSB compared to DSF (41–44). These findings confirm the need for executive function to complete the DSB task. Activation of the visual cortex during DSB supports the hypothesis that visuospatial processing may be involved during mental reversal imaging of digit sequences (42,43).

Amnestic Mild Cognitive Impairment (MCI) and AD patients performed poorly on both tasks compared with normal controls (45–47). PD patients with amnestic MCI had some impairment in DSB, but not DSF (48). Early impairment of executive function caused by subcortico-frontal dopaminergic dysfunction explains the isolated poor performance on DSB among PD patients. At the cutoff <3 digits, the sensitivity and specificity of DSB in detection of major cognitive disorders (including dementia, delirium and cognitive impairment not otherwise specified) are 77% and 78%, respectively (49). With the same cutoff, DSB can detect 81% of the delirium patients, however, with false positive rate of 37% (49). Moreover, impaired digit span in elderly subjects with subjective memory complaints is a predictor for the conversion from subjective memory complaints to mild cognitive impairment (50).

Concentration and Calculation

Letter A tapping test

Concentration, which is defined as sustained and focused attention, is the primary function required for proper identification of letter A and inhibition of inappropriate non-letter A tapping. It has good sensitivity to cognitive impairment in mild traumatic brain injury and persistent post-concussion syndrome (51,52). Speed of response to externally-paced stimuli accounts for this test's sensitivity (52). This task has not been well studied in neurodegenerative diseases. In the MoCA validation study, MCI subjects and Normal Controls had comparable normal performance, however, AD subjects were significantly more impaired on this task.(1)

Serial 7 subtractions

Calculation is an essential part of everyday social and living activities. In normal subjects, bilateral parietal and prefrontal cortices have been reported to be consistently activated during mental calculation, along with left inferior frontal lobe and angular gyrus activation (53–57). Some studies suggest that the linguistic representation and visuospatial imaginary also play a role in mental calculation (54,58). Specific to serial 7 subtraction, fMRI had

reported similar greater activation in the bilateral premotor, the posterior parietal and the prefrontal cortices when normal participants performed this task compared with the control condition (59). The prefrontal cortex activation is associated with working memory which is required to maintain the previous answer in a loop for further subtractions.

In AD patients, a reduction of fMRI activation or PET glucose metabolism in the inferior parietal cortex was observed during mental calculation (53,60). Some studies also reported a reduction in activation in the bilateral lateral prefrontal cortices (53), and the left inferior temporal gyrus (60). These hypofunctional areas are the same as the ones reported being significantly activated in normal subjects.

<u>Language</u>

Sentence Repetition

Sentence repetition assesses language skills which are supported by left temporo-parietal-frontal circuit. Repeating complex sentences also requires attention and concentration to memorize the words which are supported by working memory systems in the frontal lobes (61). AD patients had lower scores on this task compared with normal subjects(1,61,62). Education also plays a role in sentence repetition, and interpretation of the results should take into consideration subjects' education level (63).

Letter F fluency

Verbal fluency is divided into phonemic (letter) and semantic (category) fluency. Letter F fluency in the MoCA mainly depends on frontal lobe function compared with semantic fluency, which is sustained by both temporal and frontal lobes. Letter F fluency requires coordination of lexicosemantic knowledge, shifting from word to word, working memory, searching strategy and inhibition of irrelevant words which all highly depend on frontal lobe function and to a lesser extent the temporal lobe.

Patients with frontal lesions produced fewer words than healthy controls(64–67). Left frontal lesions play a greater role in letter fluency impairment than right frontal lesions(64,67,68). However, specificity of the frontal lobe dysfunction to letter fluency impairment is still debated as patients with non-frontal left hemisphere lesions also performed worse than patients with right hemisphere frontal and non-frontal lesions(67).

Neuroimaging studies indicate that letter fluency activates a variety of frontal (left dorsolateral prefrontal cortex, left inferior frontal gyrus, supplementary motor area) and non-frontal areas (anterior cingulate cortex, bilateral temporal and parietal lobes (69–71). Both lesional and neuroimaging studies suggest high sensitivity of the test, but low specificity, to detect frontal lobe dysfunction (72). Low specificity may partly depend on education level and literacy status, as this task requires grapheme-phoneme correspondence. Lower educated and illiterate subjects

generate fewer words than subjects with higher education (73–75). Since letters do not exist in certain languages, letter fluency was replaced by semantic fluency (animal naming) for languages such as Chinese, Korean, in the MoCA test (76,77).

As phonemic fluency is highly associated with frontal executive function, pathologies affecting frontal lobe or fronto-subcortical circuits, such as in PD and HD patients, frequently impair this function more than lesions of the temporo-parietal lobe which are associated with storage of lexicosemantic knowledge (48,78–80). In contrast, patients with Alzheimer's pathology will more likely have semantic fluency impairment early in the course of their disease (81). Patients with depression have also impaired phonemic fluency as a result of probable overall global cognitive slowing (82).

Abstraction

Similarity between objects requires semantic knowledge and conceptual thinking. In right-handed subjects, the left perisylvian glucose metabolism was closely associated with performance on the Wechsler Similarities Test (WST) (57). On PET imaging, the metabolic reduction in the left temporal lobe and left angular gyrus of Alzheimer's disease patients correlates with impairment on test for similarities(83). Frontal executive function and the parieto-temporal semantic knowledge may be involved in this task for more difficult and demanding word pairs (83). AD and Huntington's disease patients performed poorly on the WST compared to normal controls. Patients with Fronto-Temporal dementia have more deficits than AD patients in the similarities subtest of the Frontal Assessment Battery when controlled for MMSE level (84). Moreover, performance decline in the WST is predictive of AD conversion in non-demented participants (85).

Delayed recall

More words to recall (5 versus 3), less learning trials (2 versus up to 6), and more time between immediate recall and delayed recall (5 minutes versus 2 minutes) probably explains MoCA's superior sensitivity for amnestic MCI detection compared to the MMSE. In the first MoCA validation study, MCI patients recalled on average 1.17 words out of 5, while normal controls recalled 3.73words (1).

Category and multiple choice cues provide useful information to distinguish encoding memory impairment which does not improve with cueing from retrieval memory impairment that do improve with cueing.

Retrieval memory impairment may be associated with medial parietal and frontal white matter loss (86), posterior cingulate hypometabolism (87), pathologies affecting subcortical structures (88), hippocampal-parieto-frontal network (86). Retrieval memory deficits are seen in pathologies affecting sub-cortical structures such as Vascular Cognitive Impairment (89,90), Parkinson's disease (91), Huntington's disease (92,93). However, the retrieval deficit hypothesis of PD-related memory impairment has been debated, as some studies have shown that even given cues,

PD patients still had impairment in recognition (94,95). Retrieval memory deficits can also be seen in Depression (96,97), Frontotemporal Dementia (98,99), Normal Pressure Hydrocephalus (100), and HIV Cognitive Impairment (101,102).

Encoding memory impairment correlates with hippocampal atrophy and hypometabolism (86,87,103). AD patients typically perform poorly on delayed free recall without improvement after cueing, and also have higher rates of intrusion compared with PD and HD patients (104). Encoding memory deficits are also seen in Wernicke and Korsakoff syndromes, strategically located ischemic or hemorrhagic strokes or tumors that affect the Papez circuit (Hippocampus, fornix, Mamillary bodies, Thalamus, and Cingulate cortex), and post surgical excision of the Medial Temporal lobes for Epilepsy control as first described in H.M. by Milner (105–107).

Orientation

Impairment in orientation has been shown to be the single best independent predictor of daily functions in patients with dementia, and is also associated with caregiver burden and psychological distress (108,109). Temporal orientation yields high sensitivity in detection of dementia and patients with delirium. Errors in identifying the date has the highest sensitivity (95%), but also lowest specificity (38%) (110). Identification of the year or month was suggested to detect cognitively impaired subjects with optimal validity(110). However, orientation is not a good indicator to detect milder stages of cognitive impairment(1). Temporal orientation can also predict overall cognitive decline over time (111). Moreover, patients with temporal disorientation tend to be impaired on verbal memory as well (112). Orientation to place is not discriminative in milder stages of cognitive impairment and dementia, but may be able to detect very severe cognitive impairment which is also obvious without cognitive screening.

3. MoCA Development and Validation

The MoCA (Copyright: Z. Nasreddine MD) was developed based on the clinical intuition of one of the authors of the validation study (ZN) regarding domains of impairment commonly encountered in MCI and best adapted to a screening test (1). An initial version covered 10 cognitive domains using rapid, sensitive, and easy-to-administer cognitive tasks. Iterative modification of the MoCA took place over 5 years of clinical use. An initial test version was administered to 46 consecutive patients (mostly diagnosed with MCI or AD) presenting to the Neuro Rive-Sud (NRS) community memory clinic with cognitive complaints, a MMSE score of 24 or higher, and impaired neuropsychological assessment. They were compared with 46 healthy controls from the same community with normal neuropsychological performance. Five items did not discriminate well and were replaced. Scoring was then adjusted, giving increased weight to the most discriminant items. The final revised version of the MoCA (version 7.1) covers eight cognitive domains and underwent a validation study at the Neuro Rive-Sud (NRS) community memory clinic on the south-shore of Montreal and the Jewish General Hospital memory clinic in Montreal (1). Participants were both English and French speaking subjects divided into three groups based on cognitive status; normal control (n=90), Mild Cognitive Impairment (n=94), and mild Alzheimer's disease (n=93). MoCA was

administered to all groups, and its sensitivity and specificity were compared with those of the MMSE for detection of MCI and mild AD.

Optimal cutoff scores

Sensitivity was calculated separately for the MCI and AD groups. One point was added to the total MoCA score to correct for education effect for subjects with 12 years or less education. The MoCA exhibited excellent sensitivity in identifying MCI and AD (90% and 100%, respectively). In contrast, the sensitivity of the MMSE was poor (18% and 78%, respectively). Specificity was defined as the percentage of NCs that scored at or above the cutoff score of 26. The MMSE had excellent specificity, correctly identifying 100% of the NCs. The MoCA had very good to excellent specificity (87%). When MMSE and MoCA scores were plotted together (**Figure 1**), the large majority of NC participants scored in the normal range, and the large majority of AD patients scored in the abnormal range on both MMSE and MoCA. In contrast, 73% of MCI participants scored in the abnormal range on the MoCA but in the normal range on the MMSE(1).
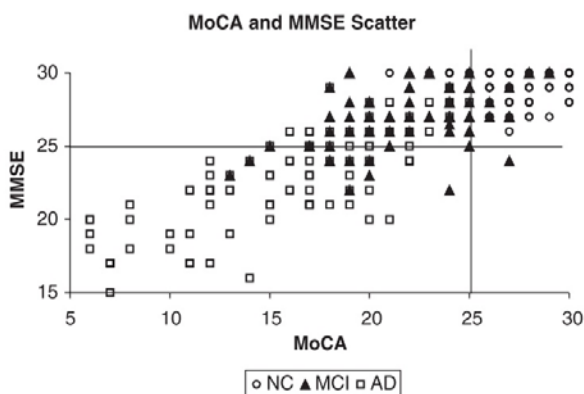

Figure 1: Scatter plot of the Montreal Cognitive Assessment (MoCA) and the Mini-mental State Examination (MMSE) scores for normal controls (NC) and subjects with Mild Cognitive Impairment (MCI) and mild Alzheimer's disease (AD). Reproduced with permission(1)

The test-retest reliability was 0.92. The internal consistency of the MoCA was good with a Cronbach alpha on the standardized items of 0.83(1). In addition, the positive and negative predictive values for the MoCA were excellent for MCI (89% and 91%, respectively) and mild AD (89% and 100%, respectively).

Recommendations

The Third Canadian Consensus Conference on the Diagnosis and Treatment of Dementia (CCCDTD3) recommended administering the MoCA to subjects suspected to be cognitively impaired who perform in the normal range on the MMSE (113). Immediate and Delayed recall, Orientation, and letter F fluency subtest of the MoCA have been proposed by the National Institute for Neurological Disorders and Stroke (NINDS) and the Canadian Stroke Network (CSN) to be a 5-minute Vascular Cognitive Impairment screening test administrable by telephone (114). The MoCA has also been recommended for MCI or Dementia screening in review articles (115–117).

Practical approach

It is important to emphasize that MoCA is a cognitive screening instrument and not a diagnostic tool, hence clinical judgment, based on thorough clinical evaluation, is important in interpreting MoCA test results and correctly diagnosing patients who present with cognitive complaints. **Figure 2** illustrates a practical approach to evaluate patients with cognitive complaints. Patients presenting with cognitive complaints and no functional impairment in their activities of Daily living (ADL) would be better assessed by the MoCA as first cognitive screening test. Subjects presenting with cognitive complaints and ADL impairment would probably better assessed by the MMSE first, then the MoCA if the MMSE is in the normal range.
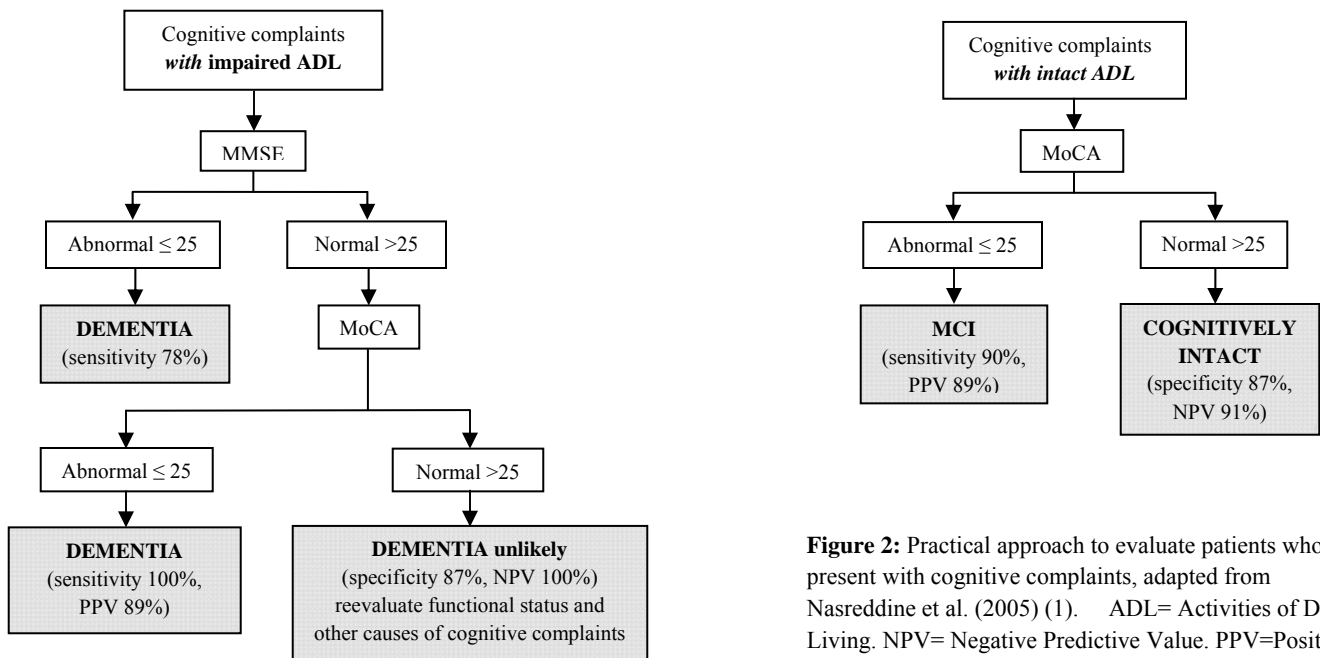
Figure 2: Practical approach to evaluate patients who present with cognitive complaints, adapted from Nasreddine et al. (2005) (1). ADL= Activities of Daily Living. NPV= Negative Predictive Value. PPV=Positive Predictive Value. MCI=Mild Cognitive Impairment

4. Demographic effect on MoCA performance

Education has been consistently reported affecting total MoCA scores (1,76,118–123). Trail making test and digit span of the Japanese version of the MoCA significantly correlate with years of schooling (124). The cube copy, semantic fluency (substitution of letter F fluency), abstraction, serial-7 subtraction and naming in the Korean version of the MoCA positively correlated with education (77). There are many cutoff scores reported according to the level of education of the studied population. In general, studies recruiting a higher proportion of low educated subjects recommend lower cutoff scores for the education correction.

MoCA has been shown to be age (76,119,121,122) and gender independent (76,119,121–123). However, in a large normative study in USA, age negatively correlated with MoCA scores. Upon further analysis, age was a significant

factor in MoCA scores mostly for less educated subjects (123) which could be explained by low cognitive reserve among less educated individuals which may result in lessened ability to recruit neuronal network and compensate age-related cognitive changes. Moreover, lower educated subjects are known to have more vascular risk factors that could also impair their cognition (125).

5.   Mild Cognitive Impairment (MCI) and Alzheimer's Disease (AD)

The MoCA was extensively studied as a screening tool for detection of MCI and Alzheimer Disease. Sensitivity for MCI detection was on average 86% (Range 77%- 96%). Sensitivity to detect AD was on average 97% (Range 88-100%). Specificity defined as correctly identifying Normal Controls, was on average 88% (Range 50% to 98%). **Table 1** summarizes the MoCA validation in MCI and AD in diverse populations and languages. Variability in sensitivity and specificity is explainable by differences in selection criteria for normal controls, diagnostic criteria for MCI and AD, community or memory clinic setting, confirmation with neuropsychological battery, age and education levels, and possibly linguistic and cultural factors.

Table 1: MoCA studies in MCI and AD

Author (year)	Language	Subjects (n)	Education (years)	Condition to be screened	Cutoff point	Sn	Sp	PPV	NPV
Nasreddine et al. (2005)(1)	English & French	277 NC 90, aMCI 94, AD 93	11.86	aMCI vs NC	25/26*	0.90	0.87	0.89	0.91
				AD vs NC	25/26*	1.00		0.89	1.00
Smith et al. (2007) (126)	English	67 MCC 12, MCI 23, Dem (AD 18, VaD 13, PDD 1)	12.1	MCI vs MCC	25/26*	0.83	0.50	-	-
				Dem vs MCC		0.94	0.50		
Ng Hoi Yee (2008) (120)	Cantonese-Hong Kong	158 NC 74, aMCI 54, AD 30	5.37	aMCI vs NC	23/24*	0.79	0.75	0.70	0.83
Lee et al. (2008)(77)	Korean	196 NC 115, MCI 37, AD 44	8.03	MCI vs NC	22/23*	0.89	0.84	0.65	0.96
				AD vs NC	22/23*	0.98	0.84	0.70	0.99
Luis et al. (2009)(119)	English	118 NC 74, aMCI 24, AD 20	14.00	aMCI vs NC	23/24*	0.96	0.95	-	-
Rahman et al. (2009) (127)	Arabic	184 NC 90, MCI 94	High school (49%)	MCI vs NC	25/26*	0.92	0.86	-	-
Tangwongchai et al. (2009)	Thai	120 NC 40, MCI 40, AD 40	10.59	MCI vs NC	24/25 ε	0.80	0.80	0.80	0.80
				AD vs NC	21/22 ε	1.00	0.98	0.98	1.00
Duro et al. (2010)(128)	Portuguese	212 MCI 82, AD 70, ODD 60	≤4 (n = 117)	MCI	25/26*	Correctly identified 84.1% δ			
				Dementia	25/26*	Correctly identified 100% δ			
Fujiwara et al. (2010)(124)	Japanese	96 NC 36, aMCI 30, AD 30	11.98	aMCI vs NC	25/26*	0.93	0.89	0.88	0.94
				AD vs NC	25/26*	1.00	0.89	0.88	1.00
Selekler et al. (2010)(129)	Turkish	205 NC 165, MCI 20, AD 20	11.59	MCI/AD vs NC	21/22*	0.81	0.78	0.46	0.95
Larner (2011)(130)	English	150 NC 85, MCI 29, Dem 36	-	MCI/Dem vs NC	25/26*	0.97	0.60	0.65	0.96
Zhao et al. (2011) (118)	Chinese	300 NC 150, aMCI 150	5-12 yr (97%)	aMCI vs NC	23/24*	0.77	0.90	-	-
Karunaratne et al. (2011) (131)	Sinhala	98 NC 49, AD 49	10.34	AD vs NC	23/24*	0.98	0.80	-	-
Damian et al. (2011) (132)	English	135 Cognitively normal 89, Cognitively impaired 46	15.30	normal vs impaired	23/24*	0.87	0.75	0.38-0.54	0.95-0.97
Freitas et al. (2012) (133)	Portuguese	360 NC 180, MCI 90, AD 90	6.38	MCI vs NC	21/22*	0.81	0.77	-	-
				AD vs NC	16/17*	0.88	0.98	-	-

* (star)1 additional point for subjects who have ≤ 12 years of education
ε (epsilon) 1 additional point for subjects who have ≤ 6 years of education

6. Vascular Cognitive Impairment (VCI)

Multiple studies have addressed the usefulness of the MoCA in Vascular Cognitive Impairment (See **Table 2**).

A. *Asymptomatic cerebrovascular disease patients with vascular risk factors*

The MoCA has been shown to detect cognitive decline in asymptomatic subjects with hypertension alone, or thickening of the carotid artery wall, or multiple vascular risk factors (134,135). Cognitive decline was also detected in subjects with TIA or first ever stroke if they had more than two vascular risk factors or low cerebral perfusion on transcranial Doppler ultrasound (134,135). MoCA also correlated with the Framingham coronary and stoke risk scores (136).

Advanced internal carotid artery stenosis (>70%)/occlusion is also negatively correlated with MoCA but not MMSE scores in asymptomatic subjects (137,138).

Subtle cognitive impairment among subjects from cardiac and diabetic/endocrine outpatient clinics of tertiary-referral hospital were detected using the MoCA with sensitivity of 83-100%, but with lower specificity of 50-52% (139).

B. *Symptomatic cardio-vascular disease*

Cognitive Impairment Post-stroke or TIA

The MoCA has been shown to detect cognitive impairment in 65% of subjects 3 months post-stroke (140). Also 30 to 58% of subjects with TIA or stroke who were considered normal on the MMSE scored below the normal cut-off on the MoCA ranging from 14 days to up to 5 years after the event (146, 148). See Table 2 for a summary of studies of the MoCA for vascular cognitive impairment. Another study, using Neuropsychological assessment as gold standard, found that MoCA had a sensitivity of 67%, and a specificity of 90% for detection of cognitive impairment post acute stroke (156). In this study, the neuropsychological battery was not performed at the same time as the MoCA (24 versus 7 days respectively) which could explain significant differences in sensitivity and specificity compared to other studies, and the MoCA cut-off used was MoCA ≤20 (158).

Heart failure

54% to 70% of non-demented community-dwelling adults with heart failure (HF) (ejection fraction 37-40%) had low cognitive scores on the MoCA (≤26) (149, 150). Reduction in ejection fraction and various associated vascular risk factors such as hypertension, dyslipidemia or diabetes mellitus may contribute to chronic reduction of cerebral blood flow in HF patients (145–147).

Sub-optimal self-care and functional dependency

MoCA identified MCI in patients with heart failure that had suboptimal self-care behaviors (148).
Using the MoCA as a cognitive assessment instrument, the self-rated version of the instrumental activities of daily living (IADL) scale was administered to evaluate functional dependence among 219 non-demented patients with cardiovascular diseases and risk factors (149). MCI was diagnosed when MoCA was less than 23/30. Less dependence was associated with higher MoCA scores, and a person who scored in the MCI range was 7.7 times more likely to report need for assistance with 1 or more activity of daily living. This study indicated that subtle cognitive impairment was an independent predictor of functional status in patient with cardiovascular disease (149).

Cerebral small vessel disease

MoCA was shown to be sensitive to white matter disease and a history of stroke, detecting cognitive impairment with a sensitivity of 73% and specificity of 75% (77, 155).

Subcortical ischemic vascular dementia (SIVD)

Subcortical ischemic vascular injury has been proposed to be associated with cognitive impairment as a result of neuronal circuit disconnection between subcortical regions, frontal cortex and other cerebral regions following repeated silent subcortical injuries (150–153). Vascular dementia was also detected by the MoCA with a sensitivity of 86.8% and specificity of 92.9% (121).

Monitoring of treatment

Cognitive outcomes after undergoing carotid endarterectomy (CEA) in severe unilateral internal carotid artery stenosis were studied using MoCA and MMSE as primary outcome measures. Symptomatic carotid stenosis (SCS) and asymptomatic severe carotid stenosis ≥60% (ACS) patients with the age- and sex-matched control subjects who underwent laparoscopic cholecystectomy (LC) were compared. At baseline, the SCS group, but not the ACS, was significantly more impaired on the MoCA and MMSE total scores compared with the LC group. Postoperatively, only the SCS patients had significant improvement on both tests when comparing pre-operative and 12-month post-operative performance (154).

Table 2: MoCA Studies in Vascular Cognitive Impairment

First Author (year) MoCA language	Objective of study	Subject (N)	Results
		Measurement	
Martinić-Popović (2006,(134); 2007, (135)) Croatian	To assess subtle cognitive decline in patients with first ever cerebrovascular disease (CVD) and in subjects without CVD symptoms but with CVD risk factors (CV-RF)	CVD (N=81 (134) & 110 (135)) CV-RF (N=45)	The MoCA provided superior sensitivity than the MMSE in detection of MCI in CVD and CV-RF patients
		MoCA & MMSE	
Wong et al. (2008) (155) Cantonese-Hong Kong	To assess the validity of the MoCA in detection of white matter lesions (WML)	NC 33, WML 33	At the cutoff 21/22, the sensitivity and specificity to detect of WML are 0.82 and 0.73, respectively. The PPV and NPV are 0.80 and 0.75, respectively.
		MoCA	
Wong et al. (2009) (76) Cantonese-Hong Kong	To assess the validity of the MoCA in detection of small vessel disease (SVD)	NC 40, SVD 40	At the cutoff 21/22 [ε], the sensitivity and specificity to detect of SVD are 0.73 and 0.75, respectively. The PPV and NPV are 0.74 and 0.73, respectively.
		MoCA	
Martinić-Popović (2009 (137); 2011 (138)) Croatian	To assess MCI in patients with asymptomatic advanced internal carotid artery stenosis (ICS)	asymptomatic ICS (N=26 (137) & 70 (138))	The MoCA proved to be a more sensitive tool than the MMSE for assessment of MCI in stroke-free patients with advanced ICS whose decline was most pronounced in the visuospatial/ executive, delayed recall and abstraction subtest of the MoCA.
		MoCA & MMSE	
Dong (2010) (141) English, Chinese, Malay	To assess cognitive impairment in acute post-stroke patients (mean 4.2±2.4 days post-stroke)	Stable post-stroke patients (N=100)	32% of the normal MMSE (>24) patients were defined as cognitively impaired patients by the MoCA (≤21). The visuospatial/executive function, attention and delayed recall subtest of the MoCA provided a good discriminative power.
		MoCA & MMSE	
Pendlebury (2010) (156) English	To assess cognitive impairment in 6-month and 5-year post-stroke patients	Stable TIA/stroke patients (N=413)	57% of patients with normal MMSE (≥27) had abnormal MoCA (<26) which were associated with deficits in the delayed recall, abstraction, visuospatial/ executive function, and sustained attention subtest of the MoCA.
		MoCA & MMSE	
Cumming (2011) (140) English	To assess the feasibility of the MoCA as a global cognitive screening tool in stroke trials.	3-month post-stroke patients (N=294)	Of those surviving to 3 months, the MoCA was completed by 80% of the patients. A majority of patients with stroke (65%) were considered as cognitive impairment according to the MoCA cutoff scores <26.
		MoCA	
Harkness (2011) (144) English	To assess MCI in patients with heart failure (HF) aged 65 years of more	HF (N=44)	More than 70% of patients scored <26 on the MoCA, suggesting MCI, had significant deficits in the delayed recall, visuospatial/executive function, and language compared with the patients who scored ≥26.
		MoCA	
Godefroy et al. (2011) (142) French	To assess the validity of the MoCA in detection of cognitive impairment in post-stroke patients	Infarct (N=88), Hemorrhage (N=7)	Using the adjusted score with cutoff 20/21*, the MoCA provided good sensitivity and specificity of 0.67 and 0.90, respectively, to detect of post-stroke cognitive impairment with the PPV of 0.93 and NPV of 0.57.
		MoCA, MMSE	
McLennan et al. (2011) (139) English	To assess the validity of the MoCA in detection of MCI in patients with CVD & CV-RF	CVD and risk factors (N=110)	At cutoff 23/24[α], the MoCA sensitivity to detect aMCI & mMCI are 1.00 & 0.83, respectively. The specificity is poor at 0.50 and 0.52 in detection of aMCI & mMCI, respectively.
		MoCA	
Athilingam (2011) (143) English	To assess MCI in patients with heart failure (HF) aged 50 years of more	HF (N=90)	54 % of participants scored ≤26 on the MoCA, whereas, only 2.2% scored < 24 on the MMSE. Delayed recall, visuospatial/executive function and language subtest of the MoCA were impaired in more than 60% of patients.
		MoCA & MMSE	
You et al. (2011) (121) Cantonese	To assess the validity of the MoCA in detection of mild to moderate VaD	NC 61, mild VaD 30, moderate VaD 40	At cutoff 21/22, the MoCA provided sensitivity of 0.87 and specificity of 0.93 in detection of mild VaD from normal controls. At cutoff 13/14, the MoCA provided sensitivity of 0.81 and specificity of 0.86 in detection of moderate VaD from mild VaD.
		MoCA	

[ε] (epsilon) 1 additional point for subjects who have ≤ 6 years of education
* (star) The score adjustment method according to age and education is available in the article (142).
[α] (alpha) 1 additional point for subjects who have ≤ 12 years of education
aMCI = amnestic mild cognitive impairment; mMCI=multi-domain mild cognitive impairment; NC = normal control; VaD = vascular dementia
PPV = positive predictive value; NPV = negative predictive value

7. Parkinson's Disease (PD)

The prevalence of dementia in PD is between 20-40% (157). The early cognitive changes are mediated by fronto-striatal disconnection, such as executive function and attention (158). Single domain impairment is found more frequently than multiple domain deficits in early stage (158,159). Progression of PD affects other cognitive domains such as memory (157,160). The association between cognitive impairment and cholinergic denervation & frontostriatal dopaminergic deficits among PD and PD with dementia (PDD) has been demonstrated by neuroimaging studies (161,162). Detection of cognitive impairment in PD is clinically useful as it predicts the conversion to PDD (160), contributes to caregiver's distress (163), and guides timing to initiate cognitive enhancing treatment (164).

The MoCA has an adequate sensitivity as a screening tool for detection of PD-MCI or PDD in a clinical setting, based on diagnostic criteria and neuropsychological test batteries (165,166). Half of PD patients with normal age and education-adjusted MMSE scores were cognitively impaired according to the recommended MoCA cutoff (25/26) (167,168) as it lacks a ceiling (165,169,170). Sensitivity and specificity for PDD were 81-82% and 75-95% respectively. Sensitivity and specificity for PD-MCI are 83-90% and 53-75% respectively (166).

Baseline MoCA scores predicted the rate of cognitive deterioration among PD patients. The group of rapid decliners had lower scores on total MoCA score, clock drawing, attention, verbal fluency and abstraction subtest when compared with slow decliners (171).

Table 3: MoCA in Parkinson's disease (PD)

First Author (year) language	Objective of study	Subject (N)	Results
		Measurement	
Gill (2008) (170) English	To establish the cognitive screening characteristics of the MoCA in PD patients	PD (N=38)	There was no ceiling effect of the MoCA. The test–retest intraclass correlation coefficient was 0.79. The inter-rater intraclass correlation coefficient was 0.81. The correlation coefficient between the MoCA and a neuropsychological battery was 0.72.
		MoCA & MMSE	
Zadikoff (2008) (169) English	To establish the MoCA and MMSE scores characteristics in PD	PD (N=88)	The MoCA showed less prone to ceiling effect and identify more MCI in PD patients than the MMSE.
		MoCA & MMSE	
Nazem (2009) (167) English	To examine the MoCA performance in PD patients with normal global cognition according to the MMSE score	PD (N=100)	52% of subjects with normal MMSE scores had cognitive impairment according to their MoCA scores (<26). The impaired patients scored worse than unimpaired patients on visuospatial/executive, naming, attention, language and delayed recall subtest of the MoCA.
		MoCA & MMSE	

Hoops et al. (2009) (165) English	To assess the validity of the MoCA in detection of MCI and dementia among PD patients	PD-N (N=92), PD-MCI (N=23), PDD (N=17)	● At cutoff 26/27$^\alpha$, the MoCA provided sensitivity of 0.83 and specificity of 0.53 in detection of PD-MCI. ● At cutoff 24/25$^\alpha$, the MoCA provided sensitivity of 0.82 and specificity of 0.75 in detection PDD ● At cutoff 26/27$^\alpha$, the MoCA provided sensitivity of 0.90 and specificity of 0.53 in detection of PD with cognitive impairment (PD-MCI & PDD)
		MoCA	
Dalrymple-Alford et al. (2010) (166) English	To assess the validity of the MoCA in detection of MCI and dementia among PD patients	PD-N (N=72), PD-MCI (N=21), PDD (N=21)	● At cutoff 20/21$^\alpha$, the MoCA provided sensitivity of 0.81 and specificity of 0.95 in detection of PDD from PD-MCI/PD-N. ● At cutoff 25/26$^\alpha$, the MoCA provided sensitivity of 0.90 and specificity of 0.75 in detection PD-MCI
		MoCA	
Luo (2010)(171) Chinese	To define and compare the cognitive profiles and clinical features of PD patients with slow or rapid cognitive deterioration rate (CDR), with normal controls (NC)	PD(N=73) NC (N=41)	The total scores and subscores for visuospatial abilities, verbal fluency and delayed recall of the MoCA were significantly lower in the PD than NC. The rapid CDR group (MoCA decline >1 point/year) was older, later age at onset, faster movement deteriorated and more impaired in CDT, attention, verbal fluency and abstraction subtest than the slow CDR group.
		MoCA	
Robben (2010) (172) Dutch	To pilot a three-step cognitive diagnostic model for patients with PD dementia (PDD)	PDD (N=15) PD no dementia (N=26)	It is efficient and feasible to use the three consecutive diagnostic steps for PDD as the following: Screening questionnaire → if +→ the MoCA or FAB or ACE-R as screening tools → if +→ a detailed NPE as diagnostic tools.
		Screening questionnaire; MoCA/FAB/ACE-R; Detailed NPE	

$^\alpha$(alpha) 1 additional point for subjects who have ≤ 12 years of education
PD-N = cognitively normal Parkinson's disease; PD-MCI = mild cognitive impairment Parkinson's disease; PDD = Parkinson's disease with dementia
ACE-R= Addenbrooke's Cognitive Examination-revised; FAB= Frontal Assessment Battery; NPE=Neuropsychological examination

MoCA was shown to have good reliability in this population. The test–retest correlation coefficient is 0.79, and the inter-rater correlation coefficient is 0.81 (170). The superiority of the MoCA compared to the MMSE is probably explained by its more sensitive testing of executive, visuospatial, and attention domains which are frequently impaired in PD. Some of MoCA's limitations is that there are no studies yet regarding its sensitivity to detect of cognitive change over time or after treatment (173) and MoCA contains items that require fine motor movement in such as trail making test, cube copy and clock drawing (5/30 point) which can impact results when administering the test to patients with severe motor symptoms.

8. Huntington's disease

Subtle cognitive impairment has been shown to precede motor manifestations of Huntington's disease (HD) (174–177). While global cognitive function is relatively preserved in asymptomatic carriers of HD mutation (AC), attention, psychomotor speed, working memory, verbal memory and executive function are often impaired early (175–177). These impaired functions are caused by abnormal fronto-striatal circuitry as shown in morphological and functional studies (178,179). It is interesting to note AC participants who were intact in memory subtest performed similarly to non-carriers on all other domains, and AC subjects with cognitive deficits performed qualitatively similarly to the symptomatic HD patients (177).

Two studies compared the ability of the MoCA and the MMSE in detection of cognitive impairment in HD patients with mild to moderate motor symptom. Compared with the MMSE, the MoCA achieved higher sensitivity (MoCA 97.4%; MMSE 84.6%), however, comparable but not impressive specificity (MoCA 30.1%; MMSE 31.5%). in discriminating the HD from normal subjects (180,181). The superiority of the MoCA compared to the MMSE in this population is explained by more emphasis in the MoCA on cognitive domains frequently impaired in early HD. Clock drawing, trail making, cube copy, abstraction and letter F fluency in the MoCA increase its ability to detect executive and visuo-spatial dysfunction. Five word delayed recall, digit span, letter tapping/vigilance test in the MoCA provide a better assessment of memory and attention. The limitation for interpreting these results is that the available studies did not use standardized neuropsychological evaluation as a gold standard for classifying cognitive function in HD.

9. Brain tumors

MoCA detected cognitive impairment among patients with brain metastases in 70% of patients who performed the MMSE in the normal range (≥26/30). Patients had abnormal delayed recall (90%) or language (90%) followed by deficits in visuospatial/executive function (60%) and the other sub-domains (182).

Detection of MCI among patients with primary and metastatic brain tumors using a standardized neuropsychological assessment as a gold standard has also shown the superiority of the MoCA compared to the MMSE in sensitivity but at the expense of lower specificity. MoCA sensitivities and specificities were 62% and 56% respectively, whereas MMSE sensitivities and specificities were 19% and 94% respectively. Visuospatial/executive function items of the MoCA correlated with patients' perceived quality of life (ability to work, sleep, enjoy life, enjoy regular activities and accept their illness) (183).

The cognitive function is one of the survival prognostic factors and correlates with tumor volume in metastatic brain cancer (215, 216). The survival prognostic value of the MoCA was studied among patients with brain metastases (186). After dichotomizing MoCA scores into two groups based on average scores (≥22 and <22), below-average MoCA scores were predictive of worse median overall survival (OS) compared with above-average group (6.3 versus 50.0 weeks). Stratified MoCA scores were also predictive of median OS, as the median OS of patients who performed the MoCA scores in the range of >26, 22-26, and <22, were 61.7, 30.9 and 6.3 weeks, respectively. MoCA scores were superior to the MMSE scores as a prognostic marker. Although, the MoCA scores correlated with the median OS, it is essential to clarify that cognitive impairment does not directly result in decreased survival. Lower MoCA scores may represent other unmeasured confounders such as the extent of disease, location of tumor or previous treatment (186).

10. Systemic Lupus Erythematosus (SLE)

Cognitive dysfunction is a common symptom of SLE-associated neuropsychiatric manifestation. It can occur independently of clinical overt neuropsychiatric SLE.(187–193) Magnetic resonance spectroscopy reveals the association between metabolic change in white matter of non-neuropsychiatric SLE (non-NSLE) patients and cognitive impairment.(188,194) Early cognitive impairments in non-NSLE patients are verbal fluency, digit symbol substitution and attention (193,195). Some investigators suggested that the pattern of cognitive decline in non-NSLE is mostly classified as subcortical brain disease since the psychomotor and mental tracking impairment are observed early (196) The domains which are subsequently impaired in patients who develop neuropsychiatric SLE (NSLE) symptoms are memory, psychomotor speed, reasoning and complex attention (195,197).

The MoCA was validated among SLE patients in hospital-based recruitment, using the Automated Neuropsychologic Assessment Metrics (ANAM) as a gold standard. At the standard cutoff scores<26/30, the MoCA provided good sensitivity (83%), specificity (73%) and overall accuracy (75%) in detection of cognitive impairment (198).

11. Substance use disorders

The validity of the MoCA to detect of cognitive impairment in subjects with non-nicotine substance dependence disorders according to the DSM-IV criteria was established by using the Neuropsychological Assessment Battery-Screening Module (NAB-SM) as a gold standard to define cognitively impaired participants. The NAB-SM is composed of 5 domains: attention, language, memory, visuospatial, and executive function. The participants were composed of alcohol dependence (65%; n=39), dependence on opioids (32%; n=19), cocaine (17%; n=10), cannabis (12%; n=7), benzodiazepine (10%; n=6), and amphetamine (8%; n=5). At the optimal cutoff point of 25/26, the MoCA provided acceptable sensitivity and specificity of 83% and 73%, respectively, with good patient acceptability (199).

12. Idiopathic Rapid eye movement Sleep Behavior Disorder (Idiopathic RBD)

RBD is characterized by the intermittent loss of REM sleep electromyographic atonia that elaborate motor activity associated with dream mentation. Approximately 60% of cases are idiopathic (200). MCI is found in 50% of idiopathic RBD and most of them are single domain MCI with executive dysfunction and attention impairment (201). Visuospatial construction and visuospatial learning may be impaired in neuropsychologically asymptomatic idiopathic RBD patients who have normal brain MRI (202). Subtle cognitive changes in idiopathic RBD may reflect the early stage of neurodegenerative diseases (202) as some studies reported an association between idiopathic RBD and subsequently development of Parkinson's disease (PD), Lewy body dementia (LBD) and multiple system atrophy (203–205). Moreover, cognitive change in idiopathic RBD are similar (visuoconstructional and visuospatial dysfunction) to LBD (206) and (executive dysfunction) to early PD (158).

The MCI screening property of the MoCA was validated among 38 idiopathic RBD patients, based on neuropsychological assessment as a gold standard. At the original cutoff point of 25/26, the MoCA had a sensitivity for cognitive impairment of 76% and specificity of 85% with an accuracy of 79%. However, for screening purpose, the higher cutoff (26/27) may be applied as it increases sensitivity to 88%, at the expense of reduced specificity (61%). The demanding visuospatial/executive functions subtests of the MoCA makes it sensitive for detection of mild cognitive impairment in idiopathic RBD patients who are impaired early in these domains (207).

13. Obstructive Sleep Apnea (OSA)

The MoCA was administered to the 394 obstructive sleep apnea (OSA) patients categorized into four groups according to severity based on the total number of apnea and hypopnea per hour of sleep (AHI), measured by polysomnography. The groups were composed of primary snoring (AHI< 5 events/h), mild OSA (AHI 5-20 events/h), moderate OSA (AHI 21-40 events/h) and severe OSA (AHI>40 events/h) group. The total MoCA scores progressively decreased as the severity of OSA increased. The scores of moderate-to-severe OSA groups were significantly lower than the scores of the primary snoring and mild OSA groups. Furthermore, defining MCI with a cutoff of 25/26, the moderate-to-severe OSA groups were more classified as MCI than the other groups. Domains that were significantly impaired in severe OSA group, compared to the primary snoring group, were delayed recall, visuospatial/executive function, and attention/concentration. Even though, the mild OSA group performed similarly to the primary snoring group on total MoCA scores, impairment in the visuospatial/executive function and delayed recall were more prominent. Moreover, MoCA scores correlated with oxygen saturation levels (208).

14. Risk of falls

The MoCA was used to classify 158 community-dwelling women as MCI or cognitively intact by the cutoff point of 25/26 (209). The short form of Physiologic Profile Assessment (PPA) was used to assess the fall risk profile. In the PPA, the postural sway, quadriceps femoris muscle strength, hand reaction time, proprioception and edge contrast sensitivity are evaluated. Participants with MCI had higher global physiological risk of falling and greater postural sway compared with the counterpart. However, the other 4 PPA components were not significantly different between the two groups. This study suggested that screening for MCI is valuable in preventing falls in the elderly.

15. Rehabilitation outcome

MoCA was shown to be more sensitive than the MMSE for detection of MCI in inpatient rehabilitation setting (210). The association between cognitive status measured by the MoCA and rehabilitation outcomes was studied among 47 patients admitted to the geriatric rehabilitation inpatient service (211). Patients had an orthopedic injury (62%), neurological condition (19%), medically complex condition (11%) and cardiac diseases(4%). MoCA had

good sensitivity (80%), but poor specificity (30%), at the cutoff scores 25/26 to predict successful rehabilitation outcome. The patients who reached the successful rehabilitation criteria tended to have higher MoCA scores at admission than the patients who did not achieve the rehabilitation goal. Many studies have reported the negative effect of cognitive impairment on the rehabilitation outcomes.(211–214).

Short term rehabilitation program in post-stroke patients (median time post-stroke 8.5 days) who had MCI, MoCA had a significant association with discharge functional status. The discharge functional status was measured by the motor subscale of Functional Independence Measures (mFIM) and motor relative functional efficacy taking the individual's potential for improvement into account (215). The visuospatial/executive domain of the MoCA was the strongest predictor of functional status and improvement. This domain was previously shown as an independent predictor of post-stoke long term functional outcome (216).

16. **MoCA in Epilepsy**

A cross-sectional study examined the Montreal Cognitive Assessment (MoCA) performance in cryptogenic epileptic patients aged more than 15 years with normal global cognition according to the Mini-Mental State Examination (MMSE) score. The mean MoCA score was 22.44 (± 4.32). In spite of a normal MMSE score, which was an inclusion criterion, cognitive impairment was detected in 60% patients based on the MoCA score. The variable that correlated with a higher risk of cognitive impairment was the number of antiepileptic drugs (polytherapy: OR 2.71; CI 1.03-7.15). No neuropsychological batteries were used for comparison (220).

17. **Normative data in multiple languages, cultures, age and education levels**

The Montreal Cognitive Assessment has been translated into 36 languages and dialects and has been used in several populations (see **Table 4** that summarizes published studies and not abstracts). Test and instructions for all languages and dialects are available on MoCA's official website (www.mocatest.org).

Performance on MoCA varied significantly among populations. Differences on MoCA performance in healthy subjects are probably accounted for by cultural, ethnic, age, educational, and linguistic factors. In a community based cognitive survey which included a multiethnic sample of Caucasians, Blacks, and Asians, the majority of subjects (62%) scored below 26 on the MoCA (124). When one considers only the Caucasian group of normal participants in this study, the mean score was 25.6/30 which is only slightly lower than the original cutoff score (25/26). However since standard neuropsychological assessment, neurological examination, and imaging studies, were not performed on the healthy volunteers, subtle cognitive deficits, neurological conditions, or imaging abnormalities may have been missed, which could account for lower performance on the MoCA (126). This is most likely to happen in subjects with lower education and in ethnic communities that are prone to vascular risk factors with consequent subtle vascular cognitive impairment (126).

Table 4: Studies using non-English versions of the MoCA

Language	Number of article	References
French	5	(1)(142) (207)
Japanese	1	(124)
Chinese	7	(120)(121)(76) (141) (171)(118)(208)
Dutch	2	(172)(219)
Portuguese	2	(128)(133)
Turkish	1	(129)
Croatian	4	(134)(135)(137)(138)
Sinhala	1	(131)
Arabic	1	(127)
Malay	1	(141)
Italian	1	(154)
Korean	1	(77)
Thai	1	(220)

18. Future research

To provide reliable and valid intercultural multi-lingual norms on the MoCA, a strict protocol (see MoCA-ACE: Age, Culture and Education Study, not published yet) defining cognitively healthy subjects has been devised with strict criteria excluding subjects with any known risks for cognitive impairment. The MoCA-ACE protocol excludes for example subjects with vascular risk factors, sleep apnea, obesity, or who take sedative medications that may be important confounders in community based surveys (126).

To decrease possible learning effects when administering the MoCA multiple times in a short period of time, two new alternative and equivalent versions of the MoCA have been validated (not published yet, available on www.mocatest.org).

To better address the need for a specific and sensitive cognitive screening tool for illiterate and lower educated populations, a new version of the MoCA, the MoCA-Basic (MoCA-B) is being validated.

To better predict AD conversion among MCI subjects, a new MoCA Memory Index Score (MoCA-MIS) that takes into account delayed recall cueing performance is being validated.

19. Conclusion

The MoCA promises to be a potentially useful, sensitive and specific cognitive screening instrument for detection of Mild Cognitive Impairment in multiple neurological and systemic disease that affect cognition across various cultures and languages.

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