

Evaluating the Efficacy of the Mini-Mental State Examination (MMSE)

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**ABSTRACT**

In 2003 the Neurology Department at Groote Schuur Hospital (GSH) requested the resident neuropsychologists to design a reliable, theory-driven neurocognitive screening battery, which could replace the Mini-Mental State Examination (MMSE), in which they had lost clinical confidence. This study evaluates the efficacy of the MMSE in the South African context. Primarily a screening tool for dementia, the MMSE is currently the most widely used screening tool of cognitive functioning. Based on a random sample of 10 patients selected from the Neuropsychology Division at GSH, a qualitative analysis, using the outcome of the full clinical assessment conducted by the Neuropsychology Division, was employed to evaluate the patients' MMSE performance. The results from this careful analysis show the MMSE to be inadequate and outdated as an initial screening tool for the following reasons. Tests of executive function, fundamental to a modern account of dementia, are absent from the MMSE. Furthermore, given its lack of theoretical underpinnings and limited diagnostic application, the MMSE is unsuitable for the current clinical context, one which requires a screening tool that assesses other domains of cognitive function beyond dementia. It is thus concluded that the MMSE is being applied beyond the purpose for which it was initially intended, in a context where the demands placed on it are beyond its diagnostic capabilities.

Key Words: MMSE; screening; hypothetico-deductive approach; normative approach; bedside tests; Neuropsychology; Dementia, neurocognitive; executive function

In 2003, the Neurology Department at Groote Schuur Hospital (GSH) approached their Neuropsychology Division to address the persistent inability of the Mini-Mental State Examination (MMSE) to assist them diagnostically. Through their clinical experience in the South African context, they recognised the critical need for a better, more thorough, diagnostically meaningful screening tool, which could also be used independently by non-experts (i.e. in the absence of a neuropsychologist to refer to). Therefore, the motivation for a larger study, by Ph.D. student Ross Balchin, stemmed from the need to develop such a tool to assist in the absence of neuropsychological expertise. The creation of a multi-faceted, theory-driven screening battery was thus undertaken incorporating into it many internationally established ‘bedside’ tests. As a part of this larger project, it was required that a separate evaluation of the efficacy of the MMSE be conducted.

Published in 1975 by Folstein, Folstein and McHugh, the MMSE (Appendix 1) is currently the most widely used, single measure of global cognitive functioning (Rapp, Espeland, Hogan, Jones, & Dugan, 2003). It was intended as an initial screening device for dementia patients (Folstein, Folstein & McHugh, 1975). As a formalised screening tool, it is utilised in evaluating mental state in research and clinical practice, and tests for alterations in brain behavior (Ostrosky-Solis, Lopez-Arango & Ardila, 2000). It offers a quick, practical means of screening for the presence of cognitive impairment (Dubin, 1998). Although it excludes questions concerning mood, abnormal mental experiences or forms of thinking, Folstein, Folstein and McHugh (1975) attest to its thoroughness. The MMSE is reported to give an accurate early diagnosis of dementia (Nathan, Wilkinson, Stammers and Low, 2001). Lezak (1995) delineates it as a test of a restricted set of cognitive functions — namely orientation, language, reading, constructional abilities, recall, attention and calculation — doing so in a concise and simplistic manner. The short and easy 30 items allow a rapid evaluation of various cognitive domains (Ostrosky-Solis *et al.*, 2000). Folstein *et al.* (1975) add that the MMSE is not sufficient to replace a full clinical appraisal in reaching a final diagnosis, as it cannot carry this diagnostic responsibility alone.

### **Demographic factors concerning the MMSE**

Studies done on the MMSE can be broadly divided into those focusing on clinical factors, and those examining demographic factors that influence its administration and performance. A number of issues have been examined regarding demographic factors. The MMSE has

specific sensitivity to level of schooling. A study conducted by Ostrosky-Solis et al. (2002) showed it to be useful only in confirming the presence of severe cognitive alterations in participants with over five years of schooling. The MMSE was given to 430 normal participants of three age ranges. Results showed the educational effect proved notoriously stronger than the age effect. It was concluded that the MMSE is an instrument with little diagnostic utility among participants with a low education level. The Crum, Anthony, Bassett and Folstein (1993) Age and Education Weighted Norms Table (Appendix 3), established the population-based norms for the MMSE by age and education level. This table proves helpful in an analysis of MMSE scores.

Demographic arguments stress the importance of the influence of non-cognitive variables on the interpretation of MMSE performance when assessing subjects other than those for whom the original psychometric criteria were developed. Despite the widespread adoption of the MMSE, the significant effects of age, sex/gender, language, ethnicity, education and social status on MMSE performance are recognised. Those with diverse ethnic backgrounds are at risk when confounders like language, cultural bias and education interact with their true cognitive abilities. This was highlighted in a population-based study, conducted by Black et al. (1999), on community-dwelling Mexican Americans, aged 65 and older. Rates of cognitive impairment varied significantly with age, education, literacy, marital status, language of interview and immigrant status. Further analysis indicated that when screening for cognitive impairment, the MMSE is strongly influenced by non-cognitive factors. Such confounds are widely present within the South African context.

Shuttleworth-Jordan (1996) stresses the importance of considering acculturation processes and demographic variables in the frequent employment of standardised test material. Acculturation refers to the “modification of the culture of a group or an individual as a result of contact with a different culture” (Mkhize, 2004, p. 30). When determining test scores, socio-cultural factors – culture, education and language – are important, as they are in constant flux due to environmental circumstances. Furthermore, acknowledging the influence of acculturation processes is essential. One need merely look at South Africa and the results of black urbanization towards greater literacy, and exposure to Westernised educational opportunities in English. The ideal screening tool would take into account the dynamic nature of socio-cultural influences, or these diverse and constantly shifting positions along a continuum of Westernisation, urbanisation and literacy.

**Clinical factors concerning the MMSE**

When testing whether or not simple tools — one being the MMSE — could be an adjunct to early recognition of dementia in primary care, Nathan et al. (2001) compared the performance of patients with mild dementia, depression and controls on tests of frontal executive function. Although the MMSE proved effective in measuring the severity of a dementia or cognitive decline over a period of one to two years, and furthermore sufficient in its ability to describe patterns of deteriorating mental functions, they found it is less sensitive when mild impairments are present — resulting in potential false negatives. It has been established, however, as more sensitive and specific for moderate to severe cognitive impairment. Nathan et al. (2001) suggest it is possible that tests of executive function included in the MMSE, or used in conjunction with it, might reduce the false negative classification rate of mild dementia. This was supported in a study by Barba, Parlato, Iavarone and Boller (1995) in which tests of executive function were used on subjects with mild to moderate Alzheimer's Disease (AD). Findings showed differences between control groups and AD groups on tests of verbal fluency and cognitive estimates tests.

Despite the assumption that the MMSE can give an accurate diagnosis of dementia, Nathan et al. (2001) claim this is difficult, due to the multiple pathological causes of dementia, which have different clinical presentations, and blurred boundaries between normal mental functioning and dementia in the elderly. Simard (1998) suggests it is impossible to differentiate the *type* of dementia by using a cut-off score. Other weaknesses, described by Rapp et al. (2003), include the minimal number of test items, the narrow range of cognitive functions assessed, ceiling effects, and the limited range of likely scores on individual items.

Simard (1998) affirms that the MMSE lacks tasks designed to assess executive functions, i.e. tasks assessing capacity to abstract, or to judge a social situation. Juby, Tench and Baker (2002) further highlight this problem. Frequently, the early stages of dementia can present with executive cognitive dysfunction, while other cognitive abilities remain intact. People with executive difficulties often show a normal MMSE score, but still have severe cognitive limitations. After reviewing the charts of 68 patients who had undergone the executive interview for the diagnosis of executive dysfunction, as well as an MMSE and clock drawing test, Juby et al (2002) found that among 32 of these patients who had a normal MMSE score, 22 obtained an abnormal executive interview score.

A modified version of the MMSE (3MS), designed by Teng and Chui (1987), has added items for assessing abilities in abstract reasoning and verbal fluency, and an expanded scoring scheme for certain items. It was designed to sample a broader variety of cognitive functions, cover a wider range of difficulty levels, and ultimately to enhance validity and reliability. In a study conducted by Teng, Chui and Gong (1990), the sensitivity and specificity of the 3MS was compared with that of the MMSE in a sample of 228 dementia patients and 150 controls. The 3MS was more reliable in that it showed superior sensitivity and specificity to dementia. Similarly, Tombaugh, Hubly, McDowell and Kristjansson (1996) found superior sensitivity and specificity for the 3MS when comparing scores for AD patients and controls on both measures. Simard (1998) emphasizes the superiority of the 3MS due to its extended scoring system and additional questions. Overall, it has better validity in identifying dementia and all levels of cognitive impairment.

To adequately evaluate the efficacy of the MMSE in the South African context, a proper understanding of the different approaches to neuropsychology — and how these are utilised — is required. In addition to this, an understanding of the classifications of dementia, and the basic functional systems of the brain, is also needed.

### **The Psychometric Approach**

The psychometric approach to practising neuropsychology involves measuring cognitive deficits, relying on standardised test scores derived from normative populations. A test response is represented by a score, which is explained in comparison to a standardised population (Lezak, 1995). The aim of this approach is to demonstrate statistically how a patient's test performance compares to that of "a standardised population (the group of individuals tested for the purpose of obtaining normative data on the test)" (Lezak, 1995, p.154). The psychometric approach underlies the majority of research conducted on the MMSE, and is used by many neuropsychologists in clinical testing. The MMSE relies heavily on the psychometric approach, which quantifies one's degree of deviation from the norm.

### **The Hypothetico-Deductive Approach**

An alternative approach in the practice of neuropsychology is the hypothetico-deductive approach (used in this study). Walsh (1991) highlights that cognitive deficits are defined in terms of impaired test performance; however, there may be different determinants of failure,

therefore test scores should not be taken at face value. Built on this view, the hypothetic-deductive approach seeks to qualitatively test hypotheses made about possible underlying causes of cognitive deficits observed in patients. This involves an assessment of the patient using a flexible range of tests designed to assess specific domains of cognitive function, which can be linked to neuro-anatomical correlates within the brain. Qualitative information is gathered (rather than merely reporting tests scores as with psychometric practice), which informs and directs the clinical assessment, the outcome of which produces a description and explanation of the patient's individual deficits.

### **The Frontal Lobes**

The frontal lobes are subdivided into deep white matter (subcortex), and mesial, orbital/basal, and dorsolateral cortex. The main functions of the deep white matter include spontaneous initiative and curiosity, with damage leading to asponenaity, adynamia, impersistence and even akinetic mutism. Patients with such damage can follow instructions, but show little initiative. Mesial function involves the selective application of voluntary arousal, with damage resulting in ideational perseveration, confabulation, reduplicative, paramnesia or contaminated consciousness (Strub & Black, 1985).

Inhibition and response suppression are principal orbital/basal functions, with damage leading to disinhibition, impulsiveness, utilisation behavior, distractibility and socially inappropriate behavior. The main function of the dorsolateral convexity (pre-motor or pre-frontal) involves the subordination of goal-directed actions to stable verbally-regulated programs. Damage leads to poor problem-solving, concrete thought, inability to shift sets, disorganised and poorly-thought-through behavior, and lack of self-critical awareness (Strub & Black, 1985).

### **Right Hemisphere Function**

The right hemisphere is dominant for tasks requiring spatial and constructional skills, and, according to Devinsky (1992), is specialised in tasks for directed attention. Right hemisphere damage results in a range of disorders: visuospatial and constructional disorders include constructional apraxia, dressing apraxia, topographical disorientation and loss of topographical memory; prosopagnosia; anosognosia; impersistence; unilateral spatial neglect; attentional deficits and delirium; and emotional indifference (Devinsky, 1992).

Damage to the right inferior-parietal lobe can lead to a Right Hemisphere Syndrome (RHS), which has three fundamental components, the first being deficits of spatial cognition and perception. The second component is unilateral neglect — impairment in the ability to orient towards, perceive or act on stimuli from one side, despite preserved primary motor and sensory functioning. Anosognosia is the third, which is the lack of awareness of one's own deficits (Devinsky, 1992).

### **Language Function**

Language function is found in the left hemisphere and includes speech production, language comprehension, naming, repetition, reading and writing, repetition. Language 'production' refers to fluency of speech, and whether paraphasia (literal or semantic) is present. Language comprehension refers to the ability to follow verbal commands. Naming difficulties can be present in both spontaneous speech and with confrontation naming (Solms & Turnbull, 2002).

### **Memory Function**

Memory function includes three primary processes – encoding (hippocampal), retrieval (either executive or diencephalic) and working memory (WM). Amnesia due to a problem with encoding of memory results from damage to the hippocampal formation either bilaterally or unilaterally, depending on what pathology is present. Diencephalic amnesia arises from damage to the diencephalic structures, including the thalamus, hypothalamus, basal forebrain nuclei, or adjacent frontal cortex — such patients have a marked retrieval problem (disturbance of memory search mechanisms), with confabulation. The third type of amnesia, 'frontal amnesia', is not a true amnesia, but a memory retrieval problem as a result of executive impairment, i.e. poor control and supervision of memory retrieval processes. Finally, memory problems of WM refer to the inability to hold information in short-term memory (Solms & Turnbull, 2002).

### **The Dementias**

Philcox et al (2000) define dementia as an acquired state of persistent diffuse impairment of higher cerebral function due to physical disease. Most dementias are of slow onset and gradual evolution, resulting in overall deterioration of the brain in memory, language, spatial orientation, executive function and personality. Dementia can involve the cortex or subcortex (deterioration of grey or white matter). Hart and Semple (1994) identify the subtypes of



cortical dementia. The first is AD, characterised by widespread cortical atrophy and enlargement of ventricles, usually progressing in four stages. Stage one typically involves memory problems of a hippocampal (axial) nature; stage two entails personality changes tending towards vagueness, anomic difficulties, and the progression towards amnesia begins to worsen. Stage three presents recognisable focal cortical syndromes (i.e. aphasia, apraxia, agnosia, etc.). By stage four, an overall deterioration and complete functional incapacity is reached.

A second classification of cortical dementia, termed fronto-temporal dementia (FTD), has two variants. Pick's disease typically starts in the frontal lobes, with personality change and emotional lability. Executive problems typically follow, including: poor judgement and reasoning ability, concrete thought, generativity and planning problems (Strub & Black, 1985). Pick's disease often occurs with Kluver-Bucy Syndrome – sexual inappropriateness and hypersexuality. The second variant is known as Lewy Body Disease (LBD), where patients exhibit a fluctuating dysexecutive cognitive picture, accompanied by hallucinations (Hart & Semple, 1994). Lewy bodies can be found in the cerebral cortex, as well as the substantia nigra and other deep neuron groups. The most striking feature is the marked fluctuations in cognitive impairment, suggestive of acute confusional episodes (Tomlinson, 1992).

Another type of cortical dementia is Semantic Dementia (SD), which preferentially attacks the left perisylvian convexity (Devinsky & D'Esposito, 2004). Patients with SD perform relatively well on most language and cognition tasks, but poorly on tasks requiring intact semantic memory – confrontation naming, word-picture matching, naming/answering questions about presented pictures. In attempting to name pictures, patient may make semantic paraphasic errors. In language testing, normal phonology and syntax are usually present. The hallmark of SD is that patients appear to have intact recall of past autobiographical information, with recent memory unaffected.

Parkinson's Disease (PD) can also result in dementia, with the principal features being a failure to initiate activities spontaneously, an inability to develop a successful approach to problem solving, impaired or slowed memory, bradyphrenia, deficits in visuospatial perception, poor performance on word fluency tests, and difficulties with shifting set. PD is caused by dopamine depletion in the basal ganglia/substantia nigra. Its hallmark is that it is a

movement disorder, characterised by akinesia, bradykinesia, rigidity of limb and trunk muscles and a tremor (Hart & Semple, 1990).

Subcortical dementia, termed vascular or multi-infarct dementia, occurs in a step-wise progression – patients have a series of multiple strokes in their white matter, and have a history of vascular problems. Symptoms include nocturnal confusion, with a subcortical frontal picture (i.e. adynamia, asponitancy and impersistence).

Finally, it is important to consider depression as a differential diagnosis to dementia, as the initial clinical picture can appear similar – it is essential to rule out a history of depression (Hart & Semple, 1994). Most patients diagnosed with Depressive Pseudo Dementia (DPD) suffer from depression-induced cognitive symptoms outside the range of dementia, but complain of memory disturbance and an inability to think or concentrate.

The primary focus of this study is to investigate the perceived inability of the MMSE to perform its diagnostic job in the current South African clinical context. This will be done by examining a series of clinical cases qualitatively, using the hypothetico-deductive approach of clinical reasoning, to analyse how accurate and useful the MMSE really is as an initial screening tool; and to determine, in the event that it proves inaccurate, why exactly this is the case (i.e. in what specific areas it falls short, and why?).

## **METHOD**

### **Design**

This study uses case studies, involving an examination of individual patients by comparing their performance on the MMSE to the outcome of a thorough neurocognitive investigation performed by neuropsychologists as part of their clinical investigations at the hospital. The use of a detailed form of assessment to contrast to a crude form (i.e. screening) is done in order to be able to evaluate and critique the MMSE in detail. The rationale for this type of design stems from the need to provide a detailed qualitative investigation of the thorough neuropsychological assessment carried out on the patient. This detail was required in order to identify and explain which areas of the MMSE, if any, were lacking when it came to eliciting certain neurocognitive deficits. Only by carefully examining the exact line of clinical inquiry (clinical reasoning) followed by the neuropsychologist to reach his/her final diagnosis (including his/her selective use of tests, and the theoretical rationale behind this usage) with each case, could this type of analysis be successfully done.

### **Participants**

The 10 participants were patients with neurocognitive deficits, who were referred to the Neuropsychology Division with queries of cognitive problems/dementia, and had completed an MMSE. The sample size was chosen on practical grounds, as the study required detailed qualitative investigation. The sampling procedure, retrospective in nature, involved examining the Neuropsychology Division's patient archives (from 2003 until present), to find records that contained an MMSE score (not all patients have one or the score breakdown is not recorded). This constitutes a random sampling approach, collecting the first 10 patients found to have complete records (i.e. an MMSE score, along with its breakdown, and the final neuropsychological diagnosis).

### **Data Collection/Materials**

The materials used include the MMSE, the patients' GSH medical records, the tests used by the neuropsychologists, and the neurocognitive reports written on the patients. The tests used can logically be divided into those that test the following domains of cognitive functioning: orientation, language, memory, gnosis, the right hemisphere function, and executive function. There are four tests used in the assessment of memory. The Babcock Story (BS) tests verbal memory. Alternatively, the 4-Hidden objects test represents a simpler alternative to the BS.

Similarly, the Rey Complex Figure (RCF) tests visual memory, whilst a simpler form can be administered if required. Finally, WM is assessed using the digit span task (Lezak, 1995).

Assessment of language function can be subdivided into 6 key areas. The first is the assessment of comprehension. Formal testing of comprehension is done using the Token Test (TT). Speech production is assessed during the interview process, with formal testing done using the Cookie Theft Test to test spontaneous speech (i.e. verbal fluency) and reveal possible paraphasias. Testing of confrontation naming uses the Boston Naming Test (BNT), whilst informal naming ability is observed during spontaneous speech (Lezak, 1995). Repetition is assessed by instructing patients to repeat sentences of increasing length, whilst writing is tested by patients writing to dictation, or jotting down a sentence of their choice. Reading is also assessed.

Testing of the RHS has three components. The first is the assessment of spatial cognition and perception. Spatial constructional difficulties are assessed using the RCF, whilst perceptual difficulties are assessed using the Cube Analysis Test. The second component of RHS is neglect, assessed by physical examination (i.e. the patient has to respond to bilateral stimuli in the visual, tactile and auditory spheres), and also using the Scene Drawing Test, as a paper/pencil assessment. The third component, anosognosia, is assessed by examining the patient's spontaneous reporting of their deficits, and prompting the patient about his/her deficits if required (Lezak, 1995).

The assessment of executive/frontal function is divided into four domains. The assessment of deep white matter is done using the FAS test to test generativity. Mesial function is assessed using the Babcock Story, to test for confabulation and executive (memory) retrieval problems. The third domain, orbital/basal function, is assessed using the FAS, and Red-Green Test (RGT) to test the patient's impulsivity (Lezak, 1995). Finally, the dorsolateral region (pre-motor or pre-frontal cortex) has the function of subordinating goal-directed action to stable verbally-regulated programs and is assessed using the Fist/Side/Palm (F/S/P) Test, or the Tapping Rhythm Test for pre-motor function; whilst pre-frontal function is assessed using the 18-Book Problem, the Colour Word Interference Test (CWIT), the 20 Questions Test, Cognitive Estimation and Proverbs. WM is also assessed here, using the Digit Span Test (Lezak, 1995).

**Analysis/Experimental Design**

With each case study, a qualitative evaluation of the patient's MMSE score was made, using the clinical diagnosis/outcome and testing procedure of the neuropsychologists. This does not mean that these two forms of assessment were deemed to be equal, nor does it presume that they performed the same role in the clinical setting. Rather, this comparison was done in order to use a detailed/comprehensive form of assessment in order to qualitatively investigate potential areas of weakness in what is widely acknowledge to be a less stringent form of assessment.

Data analysis included an investigation of where (if anywhere) the patient lost points on the MMSE. The total MMSE score of each patient was recorded and his/her performance was evaluated in comparison to the clinical diagnosis, on two primary grounds. The first being to see whether the patient achieved a normal score, as denoted by the Age and Education Weighted Norms Table for the MMSE (Appendix C), when in fact neurocognitive deficits were found to be present. The second was to see, in instances where the patients' MMSE score was below average, whether the tests where the patient lost points were consistent with the areas of neurocognitive deficit within the clinical diagnosis. Once these areas were examined, the qualitative investigation of establishing exactly what aspects of the MMSE are lacking, if any, could be done. This analysis process involved a detailed examination of the exact line of clinical inquiry (clinical reasoning) that the neuropsychologist followed in order to reach a final diagnosis (including his/her selective use of tests, and the theoretical rationale behind this usage) with each case. The insights gained from this qualitative analysis could then be applied to the MMSE scores to see exactly why, in instances where there was a disparity, the MMSE failed to do an adequate job as a screening tool.

**Procedure**

Under supervision, a random archival search of patient records from the Neuropsychology Division at GSH was undertaken, bearing in mind ethical responsibilities at all times. This archive contained the full reports of all patients seen. Such reports contained the referral question, a summary of the patient's medical history, the full breakdown of the neuropsychological assessment done (including the tests used), and the neuropsychologist's conclusion and impression of the case. Each patient's hospital folder was also consulted in order to obtain to a full medical history.

## RESULTS

### Case 1: Mr S

*Past medical history:* Mr S was 67 years old with a grade 8 education. His MMSE score was 30/30 (he should have score 26 according to Crum *et al.*, 1993). He had an 11 month history of abnormal behavior, visual and gustatory hallucinations, and signs of amnesia. The first episode consisted of five days of visual hallucinations. The family recalled odd behavior, nonsensical speech, frequent falling, postural dizziness and gustatory hallucinations. He described this episode clearly, including several complex hallucinations. He referred to these as detailed and dreamlike, but odd and bizarre, however, they did not upset him and he appeared unperturbed.

Mr S had four episodes during the first year, realising what he had seen could not really happen. He was unable to recall the next three episodes, but his wife's account entailed a hallucinatory cousin visiting him, constant falls, speaking nonsense and slurred speech. When an episode appeared, Mr S reported strong gustatory hallucinations of bitter taste, a thick sensation in his tongue, and seeing strange paint on the wall. On one occasion his wife noted body jerking and urinary incontinence. Moderate alcohol usage was reported, as was onset of stress when his wife became severely ill.

*Referral question:* Query dementia? Query psychiatric picture?

*Neuropsychological impression (following assessment):* Dementia with Lewy bodies (LBD)

*Analysis:* Neurocognitive testing of Mr S showed he was orientated to person, place and time (PPT) and held an intelligent discussion on the upcoming elections; thus he demonstrated lucidity and coherence, and was not considered delirious. With the BNT he showed no anomia. This test was also used to test his higher visual function to rule out possible visual problems as a reason for hallucinations — it was concluded he had no higher visual problems. Visuospatial construction was assessed using the RCF in which the patient produced an excellent copy. This ruled out the possibility of constructional apraxia. Furthermore, verbal memory was excellent as assessed by the BS, evident in his repeat of the story on a 30 minute delay. Visual memory, assessed with the RCF, was excellent. These two tests ruled out the possibility of an axial amnesia.

Having ruled out potential causes for hallucinations, the patient's executive functioning and abnormal behavior were examined. Problems with executive function became clear when he showed marked difficulties with certain executive tasks. He performed poorly on CWIT. Simple concrete problems were fine, as was the tapping task; however, he took a minute and a half to complete Trails A. He showed poor WM with a digit span of four, and was very slow- only scoring 11 - on the FAS Test. He did better on category fluency. He could not understand the 18 Books Test, and was extremely concrete on Proverbs and 20-Questions.

Mr S's account of his hallucinations is consistent with organic hallucinosis (they are detailed, bizarre and do not upset him unduly), and it was confirmed they are not a result of visual problems. Executive dysfunction, combined with these hallucinations, lead to the conclusion of LBD.

In this case, the MMSE was inadequate in picking up executive dysfunction because it lacked items testing the dorsolateral convexity. Furthermore, the MMSE does not adequately test WM – the 'Registration' task asks for repetition of a mere three items, which is not taxing enough to test the average seven units of a normal WM. There are no time limits on any of the MMSE items; therefore the patient can take as long as he wants to respond, whereas for FAS there is a specification of one minute per letter. Additionally, the MMSE has no tests to elicit abstract thought processes, and, as a result, did not recognise the patient's concrete ideation. Finally, the MMSE did not pick up on the attention problem he evidently displayed. This may be because the MMSE is not detailed enough – the item for attention on the MMSE is no more a test for attention than any of the other tests. Ultimately the MMSE was unsuccessful in picking up any of the central features displayed by this patient with LBD, evident in his achieving a score of 30/30.

## **Case 2: Mrs O**

*Past medical history:* Mrs O was 58 years old and scored 30/30 on the MMSE (normal score 29). She had a year's history of memory loss for words and phrases. Deterioration in vocabulary and comprehension was noted, along with frequent inappropriate responses, repetitions, and an inability to follow logical arguments. She struggled with verbs and verb construction. She increasingly avoided novel situations in which she had previously been adventurous. She got excited over simple things, emitting childlike responses. She also reported significant alcohol use. She admitted getting lost in Rondebosch, attempting to

rationalise this episode saying she did not know Cape Town well. She tried to rationalise her lack of knowledge for current events, and reported feelings of depression over the loss of her linguistic ability.

*Referral question:* Complaints of memory loss for words and phrases — please assess.

*Neuropsychological impression (following assessment):* Semantic Dementia

*Analysis:* Neuropsychological investigation began with an assessment of language in response to the patient's subjective complaints. An assessment of comprehension was done first. The patient's history revealed a number of comprehension difficulties, especially with lengthy instructions and multi-staged questions. In some cases even simpler questions were not responded to appropriately - she did not always answer the question asked of her, but something on a related topic. Often her word substitutions were inappropriate, and there was evident word finding difficulty in spontaneous speech, along with circumlocution and some semantic paraphasias; she often used her arms and hands to fill in the gaps in communication. When comprehension was formally tested using the TT, it improved as the task was not made more complicated by verbal responses — in tasks requiring nonverbal responses she performed significantly better.

Mrs O was orientated to PPT; however, she appeared sketchy on current events. Assessment of naming revealed severe anomia. A striking loss of semantic concepts was seen when she was unable to describe the use of objects (pictures) on the BNT, which she was also unable to name. Her high incorrectness on BNT was alarming considering she was high functioning. The assessment found her repetition and reading intact, however, writing was impaired, although not as badly as her spontaneous speech. After the language assessment, her memory was tested so to determine whether her cognitive impairment was limited to the language sphere. No hippocampal amnesic problems were revealed; however, a problem was found with verbal WM, recorded at five units. Testing showed her memory to be preserved. Visuospatial ability was intact, and she was not agnosic.

The MMSE did not pick up Mrs O's naming problems. The assessment of naming is insufficient (Item 6) as the patient is only required to name a 'pencil' and a 'watch'. These objects are extremely familiar and not sensitive enough, and there are too few. As with the



TT, Item 9 has the patient follow a three-stage command, with no verbal response required — the MMSE has no comprehension task requiring the patient to respond verbally, except for the Serial Sevens Test. Item 10 on the MMSE should have been able to pick up on her writing problem, however the length of the sentence is not specified, and the shorter the sentence, the less chance of noticing a writing impairment. She should have failed in three of the language tests on the MMSE. Almost half the MMSE is dedicated to language (there are five primary language tasks), yet it was still unable to elicit anything regarding her profound language problem.

### **Case 3: Mrs P**

*Past medical history:* Mrs P aged 47, had a grade 12 education. She scored 30/30 on the MMSE (normal score 28). She had a long-standing history of psychiatric problems and alcohol abuse, and a family history of psychiatric problems. She was thought to be psychotic but a CT head scan showed major frontal atrophy. She had a three-year history of deterioration and was diagnosed with major depression, along with a personality disorder. At home she refused to make food or clean, and requested to live in the ward after being admitted three times in three years. Her problems began three years previously, and she became very religious, developing paranoid delusions.

On assessment, she acted appropriately, denying delusions and hallucinations, and presenting no obvious thought disorder. She said her family was supportive. Since the onset, she underwent radical personality changes: reduced personal hygiene, spending up to 16 hours in bed, and not doing her washing. She walked many kilometres until her feet blistered. She had limited speech (only spoke when spoken to). She thought she was depressed as she never felt like doing anything. When her husband went out she suffered from severe anxiety which triggered her walking. She exhibited no psychotic features, was on antidepressants, and complained of severe headaches. At 22, she suffered from an anxiety attack; otherwise, she had a clear history — she previously looked great, cooked, entertained, was talkative and social. This changed over a four-year period.

*Referral question:* Query depression? Query memory problems? Query dementia?

*Neuropsychological impression (following assessment):* Fronto-temporal Dementia (FTD)

*Analysis:* Neurocognitive testing of Mrs P revealed a clinical picture consistent with FTD. She was orientated to PPT. Testing of naming (BNT) showed a mild anomia – her language was reduced and she displayed mild word finding difficulty. Her copy of the RCF was mildly impaired, as she failed to draw the overall shape, however, she did pay careful attention, noting mistakes and repeatedly starting over – a planning problem was evident. Her immediate recall of the RCF was consistent with the quality of the copy, while the delayed recall was adequate. Testing of WM (digit span) revealed a score of 6. Further testing of executive function included the F/S/P, where her performance was fine. With the FAS, her generativity was slightly reduced, scoring 9, 9 and 10 for the three letters respectively (normal being around 25-35). Her performance on the 20-Questions Test was poor – her questions were concrete in nature, masquerading as abstract questions, e.g. ‘can you drive it’? On the trail-making task her performance was slow for both trials. Performance on the Stroop Test was fine. She made a determined effort to follow rules on all tasks.

The overall impression of her neurocognitive performance revealed some deficits in executive function, while memory remained intact. Executive impairments were consistent with a mesial variant rather than an orbital/basal picture. Her history was consistent with FTD, displaying all the core features: problems with generativity, planning, and her striking lack of ability with abstract thought. Her affect was flat and her movement inert.

The MMSE was incapable of picking up Mrs P’s naming problem because the naming task is not sensitive enough. Furthermore, there was no test to reveal whether the patient is concrete in her thinking (i.e. struggles with abstract thought). Regarding her problems with generativity, the MMSE has no executive test/test requiring the patient to self-generate her own concepts/ideas within a finite timeframe dictated to them. Additionally, no task on MMSE has planning as an essential component.

#### **Case 4: Mr M**

*Patient medical history:* Mr M was aged 44, with a grade 10 education. He scored 23/30 on the MMSE (normal score 28). He was often home alone, thinking about his life and getting emotional. He became distracted when thinking about past memories, and forgetful, bringing on painful headaches. His mother died when he was young which he struggled to talk about, saying it was extremely personal. His brother was stabbed, the other died from Meningitis, as

did his sister. He frequently worried, but claimed he was not depressed even though he was sad and lonely most of the time.

He was first admitted to GSH in 2001 complaining of weak feet and knees. His right leg stiffened and got worse. He was unable to walk without crutches. By the end of the first year his assessment revealed weakness, incontinence, gait difficulties, frontal lobe disinhibition and cognitive dysfunction. He often fell and could not make it to the toilet in time. A CT head scan showed hypodensity in his right corona radiata. By 2005 he received a disability grant and began slurring his speech and speaking slowly, often repeating himself. He was boarded from work and complained of headaches and being emotional. Mr M presented with a seven-year history of progressive weakness of spasticity of the lower limb. He showed frontal signs: perseveration, labile mood and pout reflex. His condition got progressively worse over the years.

*Referral question:* Patient was diagnosed with autosomal dominant spastic tetra-paresis. On assessment he was found to have profound cognitive dysfunction. Query a dementia that is predominantly frontal?

*Neuropsychological impression (following assessment):* No evidence that his cognitive difficulties have worsened. Despite showing poor executive function with respect to memory, motor sequencing and performance on the FAS, it was concluded Mr M has a Depressive Pseudodementia (DPD) i.e. his depression can largely explain his cognitive performance.

*Analysis:* On neurocognitive assessment, Mr M was oriented to PPT, with a digit span of 5. On memory assessment using the 4-Hidden Objects Test, his immediate recall was good, but delayed was poor; however, he did benefit from prompting (an indication of executive impairment). On the BS, he performed poorly, again befitting from prompting. He did not confabulate (suggesting mesial functioning was intact). On the RCF, he adopted a poor strategy, so the test was stopped and a simplified version was given, resulting in a good copy; however, both the immediate and delayed recall were poor. He showed no difficulty with the BNT. His F/S/P was poor, although the performance might be explained by spasticity in his hand. He showed no problem with tapping and the RGT was good, suggesting no problem with impulse control or inhibition. His calculation and estimation were good, but his 18-Books answer was concrete.

A number of discrepancies were found between his MMSE performance and performance on the neuropsychological tests. On the MMSE, Mr M lost 2 for 'Orientation', but was found to be orientated by the neuropsychologist; 1 for 'Calculation and Attention', but these functions were also found intact; 3 for 'Recall' (his most significant loss of points) and 1 for design copy. This score of 23/30 classifies him as officially demented according to the MMSE guidelines. Both the MMSE and the neuropsychologists' tests revealed problems with memory, however, the quality of the memory problem was different. The neuropsychologists attributed it to executive problems (i.e. retrieval problems) whereas the MMSE was unable to differentiate between an encoding and a retrieval problem. The MMSE picked up a memory problem but the wrong type, mistaking an executive problem for a memory problem because it did not have adequate tests to differentiate between the two. The final point, lost on design copy, making his score even lower, could be explained by his spasticity; however, the MMSE does not factor primary non-cognitive deficits into test performance.

#### **Case 5: Mr DB**

*Patient medical history:* Mr DB was aged 69, with a grade 12 education. He scored 29/30 on the MMSE (normal score 28/30). He was admitted to GSH with a diagnosis of Dopamine Dysregulation Syndrome (characterised by anxiety, panic attacks, irritability and exaggerated rebound depression and dysphoria). A former smoker, he had been self-administering large doses of Carbidopa for a few months as a result of experiencing too many "off" periods (freezing). He said he 'craved' the drug. He subsequently began suffering from hallucinations (without delusions), increased sexual drive, insomnia, dyskinetic movement and falling. The excessive dosage was gradually reduced and his symptoms improved. He was diagnosed 18 years prior with idiopathic PD.

*Referral question:* Query dementia?

*Neuropsychological impression (following assessment):* Parkinson's Disease. No dementia, but specific executive problems to do with planning, generativity and initiative; memory intact.

*Analysis:* On neurocognitive assessment, Mr. DB was oriented to PPT. Although he was difficult to understand (hypophonic), his LTM was good and his description of his condition

was accurate. His use of language was fitting and he was talkative, cooperative and generally in good spirits. Assessment of memory revealed a digit span of five and therefore a WM problem. He demonstrated a perfect performance on the 4-Hidden Objects Test. Mr DB recalled only four items on the BS on his first recall; improving slightly to 8 items on his second attempt. There were hints of confabulation – he recounted on separate recalls that in saving the boy caught under the bridge, the man cut his hands “and they were bleeding”. He answered accurately on all prompts.

On executive assessment, Mr. DB’s performance was adequate when responding to proverbs and the similarities test. His performance was perfect on the tapping task. In response to the referral question/query of dementia, memory and executive function were tested – looking out for signs of sub-cortical or Alzheimer’s-type dementia. Based on his age, medical condition and educational background, his performance on the tasks was sound with no evidence of a dementing process. He did, however, have an adynamic-type dysexecutive picture.

Mr DB’s performance on the MMSE revealed him to be normal and ‘above average’. The neuropsychologists found no subcortical or Alzheimer’s type dementia, but rather, problems consistent with an adynamic dysexecutive type presentation (consistent with the cognitive picture seen with PD). The MMSE was unable to pick up his demonstrated frontal problems of an adynamic type, and his WM impairment. There are no executive tests on the MMSE that test the cognitive functions of the frontal subcortex (deep white matter), assessing for asponaity, adynamia and impersistence. Tests used to illicit a dementia of this type should involve time restrictions on tasks which focus on the patient’s ability to initiate a response as these are important features of such cognitive impairment.

### **Case 6: Mr H**

*Patient medical history:* Mr H was aged 63, with a grade 9 education. He scored 25/30 on the MMSE (normal score 28). He presented with socially inappropriate behavior (laughter), labile mood, everyday forgetfulness, and was incontinent. He showed no obvious hallucinations, however, he did have vague paranoia especially regarding burglars.

*Referral question:* Query fronto-temporal dementia? Check his complaints of memory problems.

*Neuropsychological impression (following assessment):* Early stages of a FTD

*Analysis:* Mr H's WM was intact and he was orientated to PPT; however he exhibited serious executive problems involving concrete thought (in the similarities and 20-questions tests), impulsivity, a lack of insight (and concern towards his illness), and lack of self-correction on his test performances. This clear executive impairment was consistent with the early stages of a fronto-temporal type dementia.

The MMSE scored him 'below average' according to his age and education, but not demented (above 23), thereby missing his dementia. He lost one point for 'Orientation' (month) – as with the neuropsychologists' testing; three for 'Attention and Calculation' and one for 'Recall'. Therefore his performance does not fit with the executive impairment found by the neuropsychologist. The MMSE falls short in that there are no tests for impulse control or abstract thought (e.g. the similarities test, cognitive estimation, 20-Questions).

### **Case 7: Mr MK**

*Patient medical history:* Mr MK was aged 74, with an economics degree. He scored 29/30 on the MMSE (normal score 28). He was referred from Geriatrics because of memory problems, irritability and acute listlessness, with a family history of AD. A CT head scan showed generalised cortical and cerebellar atrophy, as well as dilatation (atrophy of peri-ventricular tissue). A SPECT scan was indicative of a compromised vascular supply and neural changes, consistent with small infarctions of the cortex or generalised atrophy. He had an extensive medical history including deep vein thrombosis (DVT), peripheral vascular disease (PVD), and a transurethral prostatectomy, however, these did not appear relevant to his cognitive difficulties reported.

Mr MK complained of "old-timer's disease", with trouble remembering things e.g. where he was driving. He worried he had AD like his sister and was concerned over the financial ramifications. When asked about his mood he insisted it was the same as always. He was unable to identify a definite time as to when his memory difficulties began, but suggested three years prior. He had word finding difficulties.

*Referral question:* Query neurocognitive functioning with particular emphasis on possible AD and Vascular Dementia?

*Neuropsychological impression (following assessment):* Severe WM problem and difficulties with executive functioning. No evidence of a mesial-temporal memory loss; however the assessment was consistent with a mild to moderate dysexecutive syndrome of the dorsolateral convexity.

*Analysis:* On neurocognitive assessment, Mr MK was orientated to PPT. His remote memory appeared good: he gave a reasonable history, with relevant dates. His RCF copy was adequate, with excellent recall. His BS performance was not consistent with an axial amnesia, although he did not benefit from prompting. There was no evidence of confabulation. His performance on the FAS was good. There appeared to be a moderate deficit on WM and/or executive function: he achieved five digits forward and five digits backward, despite his high intellectual functioning. His motor sequencing was mildly impaired. Performance on the CWIT was poor and his explanation of proverbs ranged from concrete to excellent; his judgment appeared intact.

With Mr MK, the MMSE missed an executive picture. There is no adequate test of WM – normal capacity is seven units – Item 3 (‘Registration’) only assesses three units which is not taxing enough to test WM adequately. Additionally, ‘Recall’ (Item 5) is a test of LTM, not WM, as Item 4 ‘Attention and Calculation’ acts as a distraction from Item 4, and therefore is not immediate recall. Furthermore, no test for motor sequencing exist (MK’s motor sequencing was impaired), and dorsolateral tests were also missing.

### **Case 8: Mr E**

*Patient medical history:* Mr E was aged 54, with a grade 8 education. He scored 20/30 on the MMSE (normal score 27). He reported a history of hypertension and difficulties with language production, naming and extreme forgetfulness, forgetting where he put things, and often putting on his shirt back to front. Twelve months prior assessment, he reported the onset of a gradual cognitive decline. His wife confirmed he suffered from nocturnal confusion, however, she said his sleeping and eating remained normal. A CT head scan showed severe generalised atrophy, and peri-ventricular white matter changes. He also

presented a history of Transient Ischemic Attacks (TIA's). He remembered an incident where he fell at work. He took Fluoxetine for depression.

*Referral question:* Query expressive and receptive aphasia; multi-infarct dementia or AD?

*Neuropsychological impression (following assessment):* He had a severe dysexecutive syndrome of the frontal-subcortical-type (including adynamia), and dysarthria (disturbance of articulation).

*Analysis:* On assessment Mr E was orientated to PPT. He had severe dysarthria, but no anomia. His comprehension appeared intact, with no evidence suggesting aphasia. He was found to be moderately apraxic. On tests of memory, WM was poor with a digit span of three; however, performance on the 4-Hidden Objects test demonstrated that his immediate and delayed verbal memory was intact, as was his visual memory (using a simplified version of the RCF).

Mr E's performance on executive tests revealed severe adynamia – on the FAS test he only gave one or two words per letter. His severe dysarthria, coupled with this adynamia, greatly affected his speech. With the RGT he displayed obvious disinhibition. His self-verification was poor throughout. He also showed poor complex reasoning on the Problem-Solving Task. His performance on F/S/P demonstrated poor motor sequencing, and an inability to subordinate actions to verbal commands. His marked adynamia and poor WM, ideational perseveration, poor complex reasoning, inability to subordinate actions to verbal commands, poor motor sequencing, disinhibition and poor self-verification all point to severe dysexecutive syndrome of the fronto-subcortical-type. This dysexecutive presentation in the context of intact episodic memory, nocturnal confusion, hypertension and a history of TIA's fitted a multi-infarct picture of cognitive impairment.

Mr E's MMSE score suggested he was demented. He lost one point in orientation (name of ward), one for registration, three for attention and calculation (serial seven's) and two points for recall. His neuropsychological assessment showed his immediate and delayed memory to be intact. In language the patient lost one point for repetition, and one for writing. On assessment he was shown to have no language problems and so should not have lost MMSE points here. He lost one point for copying, and only one on speech (repetition), the MMSE



therefore not picking up his severe speech problem. The MMSE did pick up a problem, as the final score showed he was dementing, but the reason he probably lost so many points was a result of his WM impairment — but, the MMSE allows no room for interpretation. It is not possible to have a screening tool that claims to screen for dementia but does not have tests for executive function and WM. With a frontal dysexecutive picture it is necessary to have timed tests for adynamic presentations, an adequate WM task, and tests of complex reasoning.

### **Case 9: Mrs D**

*Patient medical history:* Mrs D was aged 79, with a grade 7 education. She scored 16/30 on the MMSE (normal score 25). She complained of longstanding forgetfulness. She could not specify the length of time of her impaired memory; however, according to her son, she became increasingly forgetful over the past five years. She forgot the contents of a conversation minutes after talking, to take her medication, and she frequently lost money. It became a real concern for her family, especially when they began noticing topographical disorientation — her getting lost in familiar shopping centres and confused as to her whereabouts even at home. Nocturnal confusion was also reported. Again, her son described one incident when his mother awoke at 3am and started taking the curtains down. Neither Mrs D nor her son reported any difficulties comprehending or producing speech, or with finding words. She has a history of hypertension and diabetes, with evidence of mild right upper limb spasticity. Her executive functioning deteriorated with her memory over two years, and she presented with some visuospatial problems.

*Referral question:* Query Dementia — pattern consistent with AD?

*Neuropsychological impression (following assessment):* Impaired memory on an axial basis, consistent with Dementia of Alzheimer's type. The possibility of a vascular overlay could not be excluded.

*Analysis:* On assessment Mrs D was orientated for time, but not place. She believed she was at the memory clinic she visited the week before. With a digit span of 5, her WM was impaired, but her attention was good, and she was able to give the months of the year backwards without problems. She did, however, do poorly on tests of verbal and visual memory. On the 4-Hidden Objects Test she benefited somewhat from prompting, but there were a lot of false positives and she appeared to be frequently guessing.

On tests of executive functioning Mrs D was able to solve basic complex problems, but her performance broke down as complexity increased. Mild motor sequencing abnormalities were noted, as was ideational perseveration throughout. She was severely impulsive and a decrease in verbal fluency, tested by the FAS test, was also noted, with an average of 6 words per letter, per minute. This picture was consistent with a mild dysexecutive syndrome, although this could not explain her poor performance on memory tests. With language, Mrs D's spontaneous speech was fluent and grammatical, and no word finding difficulties were found. Her language was thus judged to be intact. Her performance on visuo-spatial tasks, however, was poor, and her ability to perceive depth and size was impaired. She showed severe difficulty on constructional tasks, illustrated by a poor performance on the RCF.

She lost five points for orientation on the MMSE, which does not have an item for orientation for person. She lost four points for 'Attention and Calculation', and none for 'Registration', which is consistent. She lost one for 'Recall'; however, the clinical assessment found her memory to be significantly worse. The reason for this seems obvious, as memory tests on the MMSE are not complex enough to pick up memory problems. Mrs D lost a point for naming objects, and two for the 3-step command — she should not have lost points for language as neurocognitive assessment found her language to be intact. She lost one mark for copying. The MMSE did not pick up visuo-spatial difficulties and constructional difficulties, nor did it have tests to specifically elicit perseveration, the patient's concreteness or motor sequencing abnormalities, again this illustrates an absence of executive tests.

### **Case 10: Mrs S**

*Patient medical history:* Mrs S was aged 56, with a grade 9 education. She was Afrikaans speaking, and scored 23/30 on the MMSE (normal score 28). She had a history of steady cognitive decline and psychological symptoms of depression and anxiety, and her attention had decreased. For the year prior to assessment she stayed in ICU (this stay was prolonged), subsequent to this she developed peripheral neuropathy. She had an intention tremor and since her stay in hospital, had shown WM and attention problems, along with frontal release signs. A CT head scan revealed mild cerebral atrophy, considered normal for her age.

*Referral question:* Poor STM and attention, constructional apraxia, frontal lobe release signs, and possible anxiety/depression added on to this problem, compounding it. Query hypoxic damage?

*Neuropsychological impression (following assessment):* Higher cortical functions normal.

*Analysis:* An assessment of neurocognitive function in relation to hypoxic damage showed no language difficulties – no anomia, language production or language comprehension difficulties. Tests of memory revealed no signs of axial amnesia. She was able to lay down continuous memory both visually and verbally. A digit span of six suggested normal WM. Tests of gnosis showed no apperceptive agnosia and she was able to perceive and reproduce line drawings. No constructional apraxia deficits were noted. Difficulties in copying were probably due to the intention tremor rather than problems with spatial cognition. Executive functions were also assessed and found to be normal. She reported no history of any psychological conditions. Furthermore, she claimed her emotions were, and always had been, normal.

On the MMSE, she lost two points on ‘Orientation’, three for ‘Attention and Calculation’, one for language (writing), and one for copying – suggesting she was demented. The fact that she was Afrikaans may have been a shortcoming, as the MMSE is only in English. Because it does not take into account background information about the patient, such inaccuracies can readily occur.

## **DISCUSSION**

A common finding throughout the results is the persistent inability of the MMSE to pick up on executive/frontal problems, evident in all the cases except 4 and 10. Upon investigation, this finding stems from a gross lack of items to test executive function. There are no items to test abstract thought processes (case 9). It is unable to pick up adynamia due to the fact that there are no time limits on any of the items, thereby eliminating the opportunity to demonstrate problems associated with generativity (case 5). Other executive test items missing from the MMSE include ones for inattention (case 1) and planning (case 3). These findings support the claims made by Simard (1998) regarding a lack of tests of executive function. Additionally, the findings from the Barba et al. (1995) study are firmly supported here in that the inclusion of executive tests can result in more reliable diagnoses. It also is

apparent from cases 5, 7, 8 and 9 that the MMSE's testing of WM is inadequate, as the 'registration' task intended to test WM only uses three objects, with 'normal' being seven units for human WM span.

Investigation of the cases reveals that the screening of language function is insufficient in that there is a clear lack of sensitivity in the test for naming (case 3). The items in the naming task are not cognitively taxing, or sensitive enough (there are only two easy items to be named), to elicit a naming problem. The MMSE is also not always capable of picking up writing impairment, due to the fact that the writing task has no specifications as to the length of the sentence — the shorter the sentence, the more chance there is of missing a writing impairment (case 2).

Another problem is the MMSE's lack of sensitive memory tests. Case 4 demonstrates its inability to differentiate between an encoding and a retrieval problem. Multiple-choice questions are required in order to test for retrieval problems. The current retrieval item is not taxing enough - only three items are asked to be recalled. The neuropsychologists deem 4 items to be the minimum for testing simple recall. Finally, there are no tests of delayed recall, crucial for a thorough screening of memory. Case 9 further demonstrates that the items are not complex enough to pick up memory problems. In support of Simard (1998), the measures of memory function included in the MMSE are notably limited. The MMSE also lacks a test of the patient's orientation to 'person', making the assessment of 'orientation' incomplete, and allowing for associated cognitive problems to be potentially missed.

The MMSE was created in 1975 as a screening tool for cognitive function, primarily dementia (Folstein, Folstein & McHugh, 1975). An investigation of the literature reveals how assessment of neurocognitive disorders, especially the dementias, has advanced significantly since the 1970s, when the understanding of the frontal lobes, and dementia, were limited. A modern understanding of frontal lobe function is imperative to having a full appraisal of brain function, because executive functions ultimately govern and mediate all cognitive functioning in a hierarchical way (Feinberg & Farah, 2003).

The degree to which executive impairment is involved in neurocognitive performance sheds critical diagnostic light on the pattern of neurocognitive deficit present. This is because the determination of these specific patterns of cognitive deficit ultimately dictates which clinical

diagnosis is made. Here, ‘pattern’ connotes the specific order in which deficits declare themselves over time. Executive dysfunction can frequently precede other cognitive dysfunction presented in the early stages of certain dementias (Juby *et al.*, 2002). Therefore, although the MMSE might effectively measure the severity of a dementia, it is incapable of detecting the early stages of those dementias which have executive dysfunction as an initial presentation. The lack of executive tests thus presents an obvious problem for the MMSE, which was designed in an era where an understanding of executive function was not included as an integral part of neurocognitive assessment.

Having used the neuropsychologist’s thorough neurocognitive assessments as a framework, a second problem was identified - the MMSE’s design lacks theoretical underpinnings. In other words, the test items were not designed with the theory of how different functions of the brain are organised and operate in mind. This is perhaps best illustrated with the WM example (cases 5,7,8 and 9) where, if such theory was drawn upon, the test item would test up to seven ‘units’, not three. The lack of executive tests further highlights this point. Another issue highlighting the lack of theoretical underpinnings is that the MMSE does not factor in that multiple causes of failure on any particular item can occur — it does not ask questions to exclude other possible causes of failure on its test items. Only a screening tool designed with a ‘decision-tree’ approach to rule out other possible causes of failure, would correct this problem. These findings highlight the need for a theory-driven battery, which can relate cognitive performance to underlying brain mechanisms.

In conclusion, given its lack of theoretical underpinnings and limited diagnostic application, the MMSE is seemingly unsuitable for the current clinical context, as this context requires a screening tool that assesses all domains of cognitive function in a diagnostically meaningful way. The demands placed on the MMSE are beyond its diagnostic capabilities. This context requires a screening tool that can ‘cover’ for the absence of clinical expertise, given the lack of resources available. With an awareness of so many errors, it seems only appropriate to look on the MMSE as an origin of multiple hypotheses about a patient, as the final score appears to be inconclusive. This study has contributed to a doctoral study being conducted by Ross Balchin. This study’s evaluation of the efficacy of the MMSE helps in determining what improvements can be made to screening tools which are much needed in the South African context. This contribution speaks to the real-world problem of the need for a more useful

screening tool, and will ultimately lead to the provision of a better diagnostic screening tool, which will be beneficial in leading to better patient management and care.

No information revealing patient identity was published, and identifying information was excluded, ensuring anonymity and confidentiality. Due to time constraints, only 10 patients were included in this study. This is perhaps not so much a limitation, as a necessity given the nature of the data analysis required. Additionally, given that the patients in this study were primarily neurological patients, findings were limited to such cases.

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## Appendix A

**The Mini-Mental State Examination**

Patient .....

Examiner .....

Date .....,.....

**“MINI-MENTAL STATE”****Maximum**

Score            Score

**ORIENTATION**

5            (    ) What is the (year) (season) (date) (day) (month)?

5            (    ) Where are we: (state) (county) (town) (hospital) (floor).

**REGISTRATION**

3            (    ) Name 3 objects: 1 second to say each. Then ask the patient all 3 after you have said them.

Give 1 point for each correct answer. Then repeat them until he learns all 3. Count trials and record.

Trials

**ATTENTION AND CALCULATION**

5            (    ) Serial 7's. 1 point for each correct. Stop after 5 answers. Alternatively spell “world” backwards.

**RECALL**

3            (    ) Ask for the 3 objects repeated above. Give 1 point for each correct.

**LANGUAGE**

9            (    ) Name a pencil, and watch (2 points)

Repeat the following “No ifs, ands or buts.” (1 point)

Follow a 3-stage command:

“Take a paper in your right hand, fold it in half, and put it on the floor”

(3 points)

Read and obey the following:

CLOSE YOUR EYES (1 point)

Write a sentence (1 point)

Copy design (1 point)

\_\_\_\_\_ Total score

## Appendix B

**Instructions for administration of the Mini-Mental State Examination****Orientation**

- (1) Ask for the date. Then ask specifically for parts omitted, e.g., “Can you also tell me what season it is?” One point for each correct.
- (2) Ask in turn “Can you tell me the name of this hospital?” (town, county, etc.). One point for each correct.

**Registration**

Ask the patient if you may test his memory. Then say the names of 3 unrelated objects, clearly and slowly, about one second for each. After you have said all 3, ask him to repeat them. This first repetition determines his score (0-3) but keep saying them until he can repeat all 3, up to 6 trials. If he does not eventually learn all 3, recall cannot be meaningfully tested.

**Attention and calculation**

Ask the patient to begin with 100 and count backwards by 7. Stop after 5 subtractions (93, 86, 79, 72, 65).

Score the total number of correct answers.

If the patient cannot or will not perform this task, ask him to spell the word “world” backwards. The score is the number of letters in correct order. E.g. dlrow = 5, dlorw = 3.

**Recall**

Ask the patient if he can recall the 3 words you previously asked him to remember. Score 0-3.

**Language**

*Naming:* Show the patient a wrist watch and ask him what it is. Repeat for pencil. Score 0-2.

*Repetition:* Ask the patient to repeat the sentence after you. Allow only one trial. Score 0 or 1.

*3-Stage command:* Give the patient a piece of plain blank paper and repeat the command. Score 1 point for each part correctly executed.

*Reading:* On a blank piece of paper print the sentence “Close your eyes”, in letters large enough for the patient to see clearly. Ask him to read it and do what it says. Score 1 point only if he actually closes his eyes.

*Writing:* Give the patient a blank piece of paper and ask him to write a sentence for you. Do not dictate a sentence; it is to be written spontaneously. It must contain a subject and verb and be sensible. Correct grammar and punctuation are not necessary.

*Copying:* On a clean piece of paper, draw intersecting pentagons, each side about 1 in., and ask him to copy it exactly as it is. All 10 angles must be present and 2 must intersect to score 1 point. Tremor and rotation are ignored.

Estimate the patient’s level of sensorium along a continuum, from alert on the left to coma on the right.

## Appendix C

**AGE and EDUCATION WEIGHTED NORMS TABLE FOR MINI-MENTAL STATE EXAMINATION**

**Ages 18 - 85+ and Education Level 0 - 13+ YEARS**

Source: Crum R, Anthony JC et al. (1993) Population-based norms for the Mini-Mental State Examination by age and educational level.

*JAMA* [May 12, 1993], 269(18), 2386-2391.

Compiled by: Dr. Bill Lynch - BIRU [2B2-PAD]

AGE:	EDUCATIONAL LEVEL [Yrs]:							
	0 - 4		5 - 8		9 - 12		13 or more*	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
18-24	<b>22</b> [N = 17]	2.9	<b>27</b> [N = 94]	2.7	<b>29</b> [N = 1326]	2.2	<b>29</b> [N = 783]	1.3
25-29	<b>25</b> [N = 23]	2.0	<b>27</b> [N = 83]	2.5	<b>29</b> [N = 958]	1.3	<b>29</b> [N = 1012]	0.9
30-34	<b>25</b> [N = 41]	2.4	<b>26</b> [N = 74]	1.8	<b>29</b> [N = 822]	1.3	<b>29</b> [N = 989]	1.0
35-39	<b>23</b> [N = 33]	2.5	<b>26</b> [N = 101]	2.8	<b>28</b> [N = 668]	1.8	<b>29</b> [N = 641]	1.0
40-44	<b>23</b> [N = 36]	2.5	<b>27</b> [N = 100]	1.8	<b>28</b> [N = 489]	1.9	<b>29</b> [N = 354]	1.7
45-49	<b>23</b> [N = 28]	3.7	<b>26</b> [N = 121]	2.5	<b>28</b> [N = 423]	2.4	<b>29</b> [N = 259]	1.6
50-54	<b>23</b> [N = 34]	2.6	<b>27</b> [N = 154]	2.4	<b>28</b> [N = 462]	2.2	<b>29</b> [N = 220]	1.9
55-59	<b>22</b> [N = 49]	2.7	<b>26</b> [N = 208]	2.9	<b>28</b> [N = 525]	2.2	<b>29</b> [N = 231]	1.5
60-64	<b>23</b> [N = 88]	1.9	<b>26</b> [N = 310]	2.3	<b>28</b> [N = 626]	1.7	<b>29</b> [N = 270]	1.3
65-69	<b>22</b> [N = 126]	1.9	<b>26</b> [N = 633]	1.7	<b>28</b> [N = 814]	1.4	<b>29</b> [N = 358]	1.0
70-74	<b>22</b> [N = 139]	1.7	<b>26</b> [N = 533]	1.8	<b>27</b> [N = 550]	1.6	<b>28</b> [N = 255]	1.6
75-79	<b>21</b> [N = 112]	2.0	<b>25</b> [N = 437]	2.1	<b>27</b> [N = 315]	1.5	<b>28</b> [N = 181]	1.6
80-84	<b>20</b> [N = 105]	2.2	<b>25</b> [N = 241]	1.9	<b>25</b> [N = 163]	2.3	<b>27</b> [N = 96]	0.9
85 and up	<b>19</b> [N = 81]	2.9	<b>23</b> [N = 134]	3.3	<b>26</b> [N = 99]	2.0	<b>27</b> [N = 52]	1.3
<b>ALL AGES</b>	<b>22</b> [N = 892]	2.3	<b>26</b> [N = 3223]	2.2	<b>28</b> [N = 8240]	1.9	<b>29</b> [N = 5701]	1.3

\*College experience or higher degree

Appendix D

**Research Ethics Committee Letter of Approval**