

stimulate an important debate and to ensure that these questions are not the exclusive domain of clinicians and academics but involve people with dementia and their families.

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### **Addenbrooke's Cognitive Examination as a better discriminator of cognitive impairment than the Mini-mental State Examination in patients with dementia**

We recently completed an audit on cognitive testing used in routine practice within our Older People Community Mental Health Team in East Dorset, U.K. We measured the frequency of use of the Addenbrooke's Cognitive Examination (ACE) following its introduction as the baseline test for cognition, comparing it to the Mini-mental State Examination (MMSE) (Folstein *et al.*, 1975).

Published research has shown that the ACE is a better discriminator of dementia than the MMSE, offering greater sensitivity (82%) and equal specificity (96%) for dementia at a cut-off of 83, compared to the MMSE at a cut-off of 24 which has a sensitivity of 52% and specificity of 96% at this score (Mathuranath *et al.* (2000).

One of the most interesting and important findings on completion of the audit was that there was an increase in the percentage of cases that showed a disparity of scores (i.e. of significant cognitive impairment when the ACE was compared to the MMSE) between 2005 (17%) and 2006 (36%) when the audit was repeated. That is to say, these cases would have had a "normal" MMSE (a score of 24 or greater), but an abnormal ACE score (a score of 83 or less).

More importantly, these cases could have slipped through the screening assessment for dementia on the basis of their MMSE score alone if no other deficit of functioning was found.

Although the ACE does take a little longer than the MMSE to administer, which may affect compliance rates with this tool, we feel that this is offset by obtaining greater detail in a variety of cognitive domains, such as executive functioning, anterograde memory, episodic memory, speech and language deficits, which the MMSE does not do. We feel justified in using the ACE as a primary tool for cognitive assessments.

### **References**

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### **Amnesia due to left hippocampal hemorrhage**

We recently cared for a patient with hypertension who presented with severe amnesia. Magnetic resonance imaging of the brain revealed localized intracerebral hemorrhage in the left medial temporal lobe. There have been only a few case reports of severe amnesia caused by a restricted hemorrhagic lesion in the unilateral medial temporal lobe (Arai *et al.*, 2006). Here, we present an elderly woman who developed severe amnesia due to left hippocampal hemorrhage. The authors obtained informed consent to the publication of this letter from the patient's son, who was her legal guardian.

A 71-year-old right-handed Asian woman with a 20-year history of hypertension came to the hospital with her family because she could not remember what she had said and heard just a few minutes before. Her blood pressure was 190 mmHg systolic and 120 mmHg diastolic. She had no abnormal neurological findings and laboratory evaluations were within normal range. Her score on the revised version of Hasegawa's dementia scale (a Japanese screening test for dementia, almost identical to the Mini-mental State Examination, with a score range 0–30; Katoh *et al.*, 1991) was 15 points. Neuropsychological tests showed recent memory impairment and disorientation with regard to time, although she retained immediate and remote memory. T1-weighted magnetic resonance imaging of the brain revealed enlargement of the lateral ventricles and abnormal hyperintensity of a localized region adjacent to the hippocampus in the left medial temporal lobe (see Figure S1 available as supplementary material attached to the electronic version of this letter at [www.journals.cambridge.org/jid\\_IPG](http://www.journals.cambridge.org/jid_IPG)). She was admitted with hypertensive intracerebral hemorrhage and treated for the management of her blood pressure. One month later, she was discharged from hospital and went home, but her recent memory impairment and temporal disorientation did not ameliorate, and anterograde amnesia persisted.

Amnesia has been known to be caused by several medical conditions and etiologies such as traumatic head injury, cerebrovascular disease, brain tumor,