

## Clinical practice guidelines for subarachnoid haemorrhage. Diagnosis and treatment<sup>☆</sup>



### Guía de actuación clínica en la hemorragia subaracnoidea. Sistemática diagnóstica y tratamiento

Dear Editor:

I would like to thank the Spanish Society of Neurology for their opportune and long-awaited article 'Clinical management guidelines for subarachnoid haemorrhage. Diagnosis and treatment'.<sup>1</sup> With their appearance online in 2012, these guidelines filled a void in our medical literature, following the publication of the European guidelines,<sup>2</sup> the guidelines of the Spanish Society of Neurosurgery,<sup>3</sup> and the American Heart Association's reviewed guidelines.<sup>4</sup> I would also like to take advantage of this opportunity to add some comments.

Stroke units are unquestionably necessary; however, not all vascular neurologists are equally able to admit patients with aneurysmal subarachnoid haemorrhage (aSAH) to their stroke units. In reality, there are marked differences between hospitals. Paradoxically, cerebral ischaemia, an entity with similar morbidity and mortality, is treated by specialists and in a different unit. In a study published in 2005, Alberts et al.<sup>5</sup> recommended using trauma centre principles to treat patients with neurovascular diseases. In a previous article, we called attention to the lack of effectiveness of current treatment approaches in neurovascular emergencies.<sup>6</sup> Regarding endovascular treatment, Arias<sup>7</sup> stated that 'radiologists, neurologists, and neurosurgeons may soon work together in this field'.

Time to treatment and treatment type may have legal consequences, and these guidelines do not specify the time frame for early treatment. Due to this lack of precision, some clinicians define early treatment as that delivered within 24 hours of aneurysm rupture whereas others use the 72-hour time mark. Treatment within 3 days of subarachnoid haemorrhage has traditionally been regarded as early treatment.<sup>2,3</sup> According to the literature, maximum incidence of rebleeding takes place during the first few hours; early treatment should be provided to avoid this severe complication, which worsens clinical outcomes. However, any treatment approach to aneurysm repair not fulfilling certain conditions is worse than delaying the treatment of aSAH.

According to Vivancos et al.,<sup>1</sup> mortality from this event has decreased considerably in the past years. We analysed 2 series of patients with aSAH separated by a period of 10 years (Table 1). During that period, endovascular treatment began to be used in our hospital. From a register of patients with neurovascular diseases, we selected those with aSAH

**Table 1** Comparison of 2 series (1993-1997 and 2008-2012) of patients with aneurysmal subarachnoid haemorrhage.

| No. of patients | 169        | 292        | P-value |
|-----------------|------------|------------|---------|
| Older than 65   | 28 (16.5)  | 69 (23.6)  | <.05    |
| Deaths          | 39 (23.1)  | 62 (21.2)  | ns      |
| Clipping        | 131 (77.5) | 120 (41.2) | <.05    |
| Coiling         | 6 (3)      | 147 (50.3) | <.05    |
| Rebleeding      | 29 (17.2)  | 28 (9.6)   | <.05    |
| WFNS > 4        | 20 (11.8)  | 83 (28.4)  | <.05    |
| Late diagnosis  | 34 (20.1)  | 42 (14.3)  | ns      |

WFNS, World Federation of Neurological Surgeons scale.

belonging to one of 2 periods: 1993-1997, when endovascular treatment was not available at our hospital; and 2008-2012, after full implementation (endovascular treatment was first introduced in 1998). We gathered the following data: age (>65 years), clipping, endovascular treatment, rebleeding, WFNS scale scores (>4), late diagnosis, and in-hospital mortality. Statistical analysis was performed using OpenEpi version 3.01. The total number of patients was considerably higher in the second period for unexplained reasons. No statistically significant differences were found between the 2 periods in terms of mortality and late diagnosis. The percentage of patients undergoing surgery was higher in 1993-1997; on the other hand, endovascular treatment increased significantly between 2008 and 2012. The incidence of rebleeding was considerably lower in the second period. The percentage of patients with poor neurological outcomes was significantly higher in the second period. According to our data, mortality associated with aSAH seemed stable, although we do not know whether this was due to the higher number of patients who survived but were incapacitated. The second period saw a rise in the number of older patients and those having a poorer neurological state. The decreased incidence of rebleeding in the second period was due to early treatment, which was rarely administered during the first period. Late diagnosis of aSAH is still a major concern in our setting. The increased use of endovascular techniques has not had a significant impact on outcomes since the most crucial factor remains SAH severity.<sup>8</sup>

Lastly, for those interested in delayed ischaemic neurological deficits (vasospasm), these guidelines offer a thorough review of drugs for vasospasm which have been proved ineffective.

## References

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## Validation of the Spanish-language version of Mini-Addenbrooke's Cognitive Examination as a dementia screening tool<sup>☆</sup>



### Validación de la versión española del Mini-Addenbrooke's Cognitive Examination para el cribado de demencias

*Dear Editor:*

The number of people who seek medical care due to cognitive symptoms has increased significantly in recent years, mainly due to the increase in the population's life expectancy. Cognitive assessments are essential in the differential diagnosis of dementia,<sup>1</sup> since they contribute to treatment decision-making, which will affect the quality of life of patients and their families.

Quick cognitive screening tests are especially useful in our setting due to long waiting lists and limited resources that do not allow specialists to administer more thorough neuropsychological tests.

Addenbrooke's Cognitive Examination III (ACE-III)<sup>2</sup> has recently been validated in Spanish.<sup>3</sup> Both the current and previous versions of this test are widely used in memory units and dementia research centres around the world.<sup>4</sup> The ACE-III is known for its ability to detect dementia and differentiate between dementia subtypes.<sup>5</sup> However, its use is not as widespread as one might like since it takes 15–20 minutes to administer.

Hsieh et al.<sup>6</sup> have developed and validated the Mini-Addenbrooke's Cognitive Examination (M-ACE), a brief version of the ACE-III. These authors reduced the original version using Mokken scaling<sup>7</sup> and administered the new version to patients with Alzheimer-type and frontotemporal dementia and to healthy controls. The Mini-Mental State Examination (MMSE) was used as the gold standard.<sup>8</sup>

The M-ACE includes 5 items (orientation to time, semantic fluency, clock face drawing, immediate recall, and delayed recall) with a maximum score of 30. Maximum administration time is approximately 5 minutes. In the original validation study, scores  $\leq 25/30$  were identified as the cut-off point for dementia with both high sensitivity (85%) and specificity (87%). The M-ACE was found to be more sensitive than the MMSE and showed a less pronounced ceiling effect.

We have studied the psychometric properties of this new version of the ACE-III in our population using the same methodology applied by its authors and the complete sample recently gathered for the ACE-III validation study.

We selected items from the original questionnaire that are included in the M-ACE and created a new score. Of the 175 subjects comprising the sample, 92 were cognitively healthy controls (age:  $77.0 \pm 6.4$  years; education:  $8.4 \pm 5.8$  years) and 83 were patients (age:  $78.4 \pm 6.8$  years; education:  $7.4 \pm 4.7$  years) diagnosed with different types of dementia in mild stages: Alzheimer disease (46 patients, 55.4%), vascular dementia (4, 4.8%), mixed dementia (9, 10.8%), dementia associated with Parkinson's disease (11, 13.3%), Lewy body dementia (6, 7.2%); frontotemporal dementia (5, 6%), alcoholic dementia (1, 1.2%), and atypical parkinsonism with dementia (1, 1.2%). All participants were at least 65 and they were recruited from neurology departments at Hospital Clínico San Carlos in Madrid and Hospital de la Santa Creu i Sant Pau in Barcelona.

Our sample was significantly older and had a lower educational level than the sample in the study by Hsieh et al.<sup>6</sup> In the reliability analysis, the scale showed high internal consistency (Cronbach  $\alpha = 0.828$ ).

Results on the M-ACE were compared to those on the ACE-III and MMSE; clinical diagnosis of dementia was used as the factor for determining cut-off points (Fig. 1). With an area under the curve (AUC) = 0.94, M-ACE scores  $\leq 16/30$  were identified as the cut-off point for dementia with high levels of sensitivity (86.7%) and specificity (87.0%). This means that the M-ACE achieves better discrimination indices than the MMSE (AUC = 0.91; score  $\leq 24/30$ ; sensitivity = 88.0; specificity = 78.3) and the ACE-III (AUC = 0.92; score  $\leq 65/100$ ; sensitivity = 83.1; specificity = 80.4) (Table 1).

The M-ACE demonstrated a high diagnostic ability, with values above 85% for discrimination between healthy controls and subjects with mild dementia. The optimal cut-off point was 16.5, although a slightly higher point (17.5)

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