

Although it could be argued that this reduction may simply be the result of reduced life expectancy in MS patients, this is unlikely, as an age-specific Cox survival model also showed a significant reduction in the risk of cancer.² Similarly, it is unlikely that this would represent under-reporting of cancer because patients are typically in closer contact with health practitioners than the normal population.² Explanations could include lifestyle alterations following diagnosis, genetic factors or immunological changes due to MS. Further study of mechanisms is therefore warranted, but more immediately, the results of this meta-analysis will be of use for MS patients and their care givers.

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J Pretnar-Oblak, M Zaletel, T M Hajnšek, *et al.* Isolated bulbar paralysis in a patient with medullar tau pathology: a case report (*J Neurol Neurosurg Psychiatry* 2010;**81**:847–849). The authors misplaced the label number (9) in Figure 1 of this paper and therefore this area indicated does not represent the nucleus ambiguus. The reprinted version of Figure 1 represents the correct area for nucleus ambiguus.

The authors would also like to explain further the labels in figure 2C and 2D. 2C: Extensive tau pathology in DNVN composed of numerous neuropil threads and tau-positive neurons (arrows). 2D: Tau pathology of few neuropil threads in the SN (arrows).

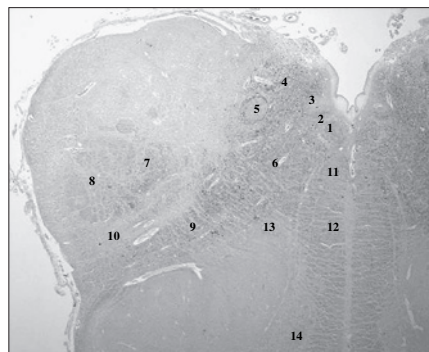


Figure 1 The corrected version with previously misplaced number 9.

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E J Thompson. Lock and key approach to “hidden” encephalitis (*J Neurol Neurosurg Psychiatry* 2006;**77**:901). This paper was published in the journal with an incorrect doi of 10.1136/jnnp.2006.095547. The correct doi is 10.1136/jnnp.2005.095547.

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Alice Nieuwboer, Gert Kwakkel, Lynn Rochester, *et al.* Cueing training in the home improves gait-related mobility in Parkinson's disease: The RESCUE-trial (*J Neurol Neurosurg Psychiatry* 2007;**78**:134–140). This paper was published in the journal with an incorrect doi of doi:10.1136/jnnp.200X.097923. The correct doi is 10.1136/jnnp.2006.097923.

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BOOK REVIEW

Vascular cognitive impairment in clinical practice

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In 2006, the NIH convened a conference, which attempted to establish a new concept, vascular cognitive impairment (VCI). The resulting document has been published¹ and now, 3 years later, several of the key participants, and others, have contributed to a new book on that topic. Edited by leading authorities and joined by several eminent experts, the book addresses clinicians dealing with demented individuals in order to distribute the concept of VCI which has not been accepted unopposed.

The term VCI is supposed to cover the whole spectrum of cognitive decline which occur as a result of vascular changes to the brain. Because these changes result from a multitude of factors (haemorrhage, ischaemia, emboli, small vessel disease, etc), VCI cannot be considered a disease, but neither is it a syndrome since there is very little in common between cognitive changes resulting from different processes (eg, leucoarosis and those due to a single thalamic stroke).

In addition, the vascular lesions occurring mostly in elderly subjects rarely affect an otherwise normal brain. Alzheimer pathology is very common in the elderly, and even if changes fail to satisfy arbitrary pathological criteria of Alzheimer's disease (AD), they cannot and should not be ignored. In fact, a

growing body of evidence forces us to accept that most cases of dementia in the elderly are the result of combined lesions,² and therefore the concept of (pure) VCI may be misleading. The authors of several chapters in the book acknowledge these issues, albeit in a soft voice. It seems that they try to simplify the question of the pathogenesis and manifestations of dementia in old age by resorting to a dualistic approach. Unfortunately, this entrenched position does not benefit scientific progress.

The authors do not even agree on the definition of VCI. Whereas the editors limit it to “from the earliest deficit” (p4), others ask whether VCI can be a prodrome to vascular dementia (p11). If vascular dementia is included in the spectrum of VCI, how can VCI be a prodrome to it? Still others claim that the “brain at risk stage” (ie, prior to cognitive changes) should be included (p54).

Particularly disappointing are the chapters on treatment where data from AD are used freely without mentioning that they do not necessarily apply to other dementia types, particularly vascular dementia. The chapter on “control of vascular risk factors” contains a well structured review of the role of dementia. However, if VCI has so many underlying pathologies, preventative treatments are hard to identify to any of its subtypes. A discussion as to why none of the anti-AD drugs has been accepted by authorities for treatment of vascular dementia is missing. Is rigid control of the risk factors useful in preventing, or at least slowing, the cognitive deterioration? This important clinical question is neglected. While there is some discussion of symptomatic therapies, this is directed at manifestations of dementia in general, perhaps because the authors believe that the clinical behavioural manifestations are similar, both in underlying mechanisms and in presentation, in VCI and in other dementia syndromes. Among atypical antipsychotic agent, the most atypical, clozapine, is not mentioned.

There are a few errors, for example, the claim that “the diagnosis of dementia can be done at the bedside using global cognitive measures (eg, Mini-Mental State Examination)”, p32. Sometimes statements are made without reference (eg, “The levels of A β may be reduced in the users of statin and Ginkgo biloba”, p85).

While the authors of the different chapters have generally completed well the missions assigned to them, the editors and publisher could have done more. Each chapter has its own structure. Summaries, which are so important in such a comprehensive work, are missing in some chapters and replaced by “significance”, “conclusions”, “concluding remarks”, etc, in others. The reference list after each chapter is not always arranged correctly alphabetically, and a unified reference list at the end of the book could avoid unnecessary repetitions. Sometimes unclear references are included (eg, “BNF 2006”, p216).

A good index is a very important tool in a book such as this but, unfortunately, it is rather incomplete. For example, “Binswanger's disease” is mentioned in the text more frequently than four times, as suggested by the index. Abbreviations are used without spelling them out first. Table 14.1, among others, has not been proofread.

Surely, a distinguished publisher like Cambridge University Press could have done a better job.

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