Memory for context but not for content: a neuropsychological follow up

In 1990 Berti et al.1 described a patient who became amnesic after a transcallosal excision of a subependymoma of the septum pellucidum. The authors suggest a possible role for septal nuclei in memory function. Recently another patient who became amnesic after transcallosal excision of an intraventricular oligodendroglioma has been reported.2 Nevertheless, we have no information about the long term clinical evolution of the amnesia in this type of patient. By chance I had the opportunity to retest the patient of Berti et al in June 1994, six years after operation. The patient is now 49 years old. She is only mildly disoriented in time and does not show everyday coping problems, except that she writes down every single task she has to carry out (dates, things to buy, phone calls she has to make) and she does not dare to go out alone, because she is not sure that she will be able to find her way back. She can always remember that “she has something to do or say, but not what”. When she was told that she was going to meet an examiner who tested her in 1988, she remembered having gone through many testing sessions after surgery with two female doctors. During her conversation with the examiner, if somebody interrupted her, she always forgot the conversation; moreover, she kept on repeating the same subjects, as for example, her husband’s heart attack or her daughter working as a nurse.

The patient was submitted to a neuropsychological battery that included the same tests she had performed six years earlier and some new ones. The patient immediately recognised the “old” tests, but her performance was still below the cut off in memory tasks (table). Moreover, her comments were exactly the same as before—for example, when she heard the beginning of a short story (the story starts with the name of a lady called Pesenti from Bergamo) that she had to recall later, she remarked that her father was working for a man called Pesenti in Bergamo, which was the same comment she made in 1988. Then she con babulated and was unable to recall most of the story; she remembered having told a story, but she could not remember its content.

As far as I know, this is the only follow up of an amnesic syndrome after lesion of septal nuclei, and a more definite role for this structure in memory is suggested. This patient’s ability to carry on an almost normal life is mostly due to her ability to remember the “context”! what she cannot remember is the “content”! She “knows” that she has something to remember, but she does not know what; this is at variance with global amnesic patients, who do not remember even that there is something to remember. This feature may occasionally be experienced by normal subjects in everyday life, but for this patient it is the rule. Lesions of septal nuclei are therefore not sufficient to produce a permanent global amnesia, but can cause an impairment in grasping the content of the recollection, even if recognition is still possible.

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Neuropsychological assessment of the patient

<table>
<thead>
<tr>
<th>Test</th>
<th>June 1988</th>
<th>June 1994</th>
<th>Normal score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digit span</td>
<td>5-50</td>
<td>5-75</td>
<td>(&gt;= 3-75)</td>
</tr>
<tr>
<td>Corsi span</td>
<td>4-75</td>
<td>(&gt;= 3-50)</td>
<td></td>
</tr>
<tr>
<td>Paired associate</td>
<td>7</td>
<td>6*</td>
<td>(&gt;= 6-50)</td>
</tr>
<tr>
<td>Short story</td>
<td>6*</td>
<td>6*</td>
<td>(&gt;= 8)</td>
</tr>
<tr>
<td>Spatial learning</td>
<td>0*</td>
<td>6-70*</td>
<td>(&gt;= 6-75)</td>
</tr>
<tr>
<td>Phonological fluency</td>
<td>15*</td>
<td>25</td>
<td>(&gt;= 17)</td>
</tr>
<tr>
<td>Semantic fluency</td>
<td>33</td>
<td>33</td>
<td>(&gt;= 25)</td>
</tr>
<tr>
<td>Raven coloured</td>
<td>26-5</td>
<td>35</td>
<td>(&gt;= 18)</td>
</tr>
<tr>
<td>Progressive matrices:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ideomotor apraxia</td>
<td>72 (r), 72 (l)</td>
<td>(&gt;= 53)</td>
<td></td>
</tr>
<tr>
<td>Constructional apraxia</td>
<td>20 (r), 20 (l)</td>
<td>(&gt;= 15)</td>
<td></td>
</tr>
<tr>
<td>Affective matrices</td>
<td>56-50</td>
<td>(&gt;= 31)</td>
<td></td>
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</tbody>
</table>

* Pathological score; r = right hand; l = left hand.

Validity of the London handicap scale

I read with interest the article by Harwood et al.,1 which studied the acceptability, validity, and reliability of a new handicap scale. A number of methodological issues need to be considered before we can accept this new scale as “a useful addition to the range of available outcome measures.” The authors state that the scale was acceptable to patients, with a “good” response rate. The overall response rate was 67%. It would be of interest to know the expected response rates to mailed questionnaires, particularly in the population studied. Although mean scores for various characteristics between responders and non-responders (Table 1), the results of formal significance testing of these differences are not clearly stated in the text. Non-responders were more likely to be non-white; is this a similar finding to other types of mailed questionnaires in that population? “Handicap” in this study was measured according to six underlying dimensions (mobility, physical independence, occupation, social integration, orientation, and economic self sufficiency). The rationale of reducing “handicap” to these dimensions seems to be based on the World Health Organisation (WHO) classification; however, the authors do not state any evidence to support this rationale. Although the face validity appears reasonable, has this dimension ever been confirmed by a factor analysis? If not, it would be of interest to see the results of such a procedure on the authors’ dataset.

The assessment of construct validity for the handicap scale was based on a comparison of outcome measures using existing scales, and whether the handicap score behaved as expected (for example, “more disabled subjects will, on average, be more handicapped”). A significant correlation between handicap and outcome scores was said to be sufficient affirmation of the construct validity of the handicap score described. Some points arise that need further clarification. Firstly, the construction of “disability” is not provided. Secondly, what evidence is there that “disability” and “handicap” are indeed different constructs? The validity of this distinction seems to be crucial if we are to accept scales that are specifically designed to measure handicap, as opposed to using currently available activities of daily living/disability scales, and (6) accept some of the statements made in the article (for instance, the strong correlation between the handicap score and the Nottingham extended activities of daily living score reflecting the importance of the items in this scale for overall handicap).

For practical purposes, construct validity of a new scale can be appraised by different strategies.1 “Distinctive dissociations” can be used to show that the new scale measures things that are different from existing scales. An attempt to show distinctive dissociation was made by calculating correlation coefficients between the London handicap score and outcome scores of other scales. A second strategy, of more practical importance, is “validation by application”—this assesses the application of a new scale in identifying, discriminating, predicting, or instructing the user of different patient groups. For practical purposes, if a new scale cannot be shown to have an advantage over existing scales in this regard, then the need for it must be questioned.

All too often in the past scales have been described with no real considerations for methodology. I therefore welcome the attempts of Harwood et al.1 in trying to validate this scale. I suggest, however, that before we accept yet another neurological scale, “handicap” must first be shown to be a distinctly different construct from “disability”. Secondly, a clear operational advantage for its use over existing scales must be demonstrated.

1 Harwood RH, Gompertz P, Ebrahim S. An occupation one scale? or the construction of a new scale. J Neurol Neurosurg Psychiatry 1994;57:825–9

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