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doi:10.1136/jnnp.68.4.511

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Illusory limb movements in anosognosia for hemiplegia

Todd E Feinberg, David M Roane, Jeffrey Ali

Abstract
To clarify the relation between anosognosia for hemiplegia and confabulation, 11 patients with acute right cerebral infarctions and left upper limb hemiparesis were assessed for anosognosia for hemiplegia, illusory limb movements (ILMs), hemispatial neglect, asomatognosia, and cognitive impairment. Five of 11 patients had unequivocal confabulation as evidenced by ILMs. The presence of ILMs was associated with the degree of anosognosia (p=0.002), with hemispatial neglect (p<0.05), and with asomatognosia (p<0.01). The results confirm that a strong relation exists between anosognosia for hemiplegia and confabulations concerning the movement of the plegic limb. There is also a strong relation between ILMs and asomatognosia.

Keywords: anosognosia; confabulation; asomatognosia.

It is often noticed that patients with anosognosia for hemiplegia may claim movement of the paralysed limb. Babinski,1–2 and Babinski and Joltrain,3 who coined the term anosognosia, noted that one patient, when requested to move a plegic arm, responded “Voila, c’est fait.” Many subsequent examiners have confirmed this observation.4–9 We have suggested the term “illusory limb movement” (ILM)10 to describe the phenomenon. We consider it to be a form of confabulation11 which differs from phantom limb phenomena in various ways.10

Some studies have shown an association between anosognosia for hemiplegia and confabulation. For instance, Feinberg et al12 found that patients with anosognosia for hemiplegia and hemianopia tended to confabulate about visual stimuli in the neglected hemispace. However, Lu et al13 found that patients with epilepsy undergoing anaesthesia of either hemisphere during Wada testing produced confabulations regarding tactile stimuli regardless of the presence or absence of anosognosia for hemiplegia. However, as Liu et al14 pointed out, neither of the above studies assessed for the presence of confabulations directly concerned with the patient’s motor weakness. In fact, confabulations directly involving the movement of the limb have never been systematically studied.

We studied a group of patients with right hemispheric lesions, with and without ILMs to examine the relation between ILMs and anosognosia for hemiplegia and related phenomena.

Methods
All patients referred to the neurobehaviour service over a 4 year period were included in the study if they met the following criteria: (1) right cerebral infarction; (2) near total left upper limb paralysis; (3) sufficiently alert, cooperative, and communicative to answer a series of questions. A total of 11 patients participated. All patients were examined within 1 week of onset of hemiplegia. The study was approved by the investigational review board of the medical centre.

Clinical data are summarised in table 1. Left arm strength was rated on a scale of 0 to 5 utilising the criteria of the Medical Research Council of Great Britain14 with 1 representing minimal contraction and 0 total paralysis. Sensory impairment was assessed for pin, touch, and position. Cognitive impairment was tested via the mini mental state examination.15 Hemispatial neglect was measured with a 40 item cancellation task. Anosognosia for hemiplegia was assessed with a 10 item scale developed to measure patients’ awareness of left arm disability (table 2). The items test for knowledge of deficit before and during demonstration of deficit. Responses for each item were scored as 0 if the patient showed awareness of deficit; 0.5 for partial awareness; and 1.0 for complete unawareness or denial. The scores were summed to produce a total score on the scale ranging from 0 to 10. Patients were examined for asomatognosia (denial of ownership of the limb) via a method previously described wherein the left arm is brought over by the examiner into the right hemispace.16

The presence or absence of ILMs was assessed as follows. Patients were first instructed to raise their unaffected right arm and were asked “Is your right arm on the bed or in the air?” Then they were told to raise their plegic left limb and asked if the left arm was on the bed or in the air. Lastly they were told to raise both arms and were asked about the location of each arm.
Table 1  Summary of clinical dates of subjects

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patients with CLMs</th>
<th>Without ILMs</th>
<th>± ILMs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 2 3 4 5</td>
<td>1 2 3 4 5</td>
<td>1 2</td>
</tr>
<tr>
<td>Age</td>
<td>64 81 78 82 84</td>
<td>70 79 78 44 44</td>
<td>56 82</td>
</tr>
<tr>
<td>Sex</td>
<td>M F M F F</td>
<td>M F F F M</td>
<td>F</td>
</tr>
<tr>
<td>Motor strength*</td>
<td>1 0 1 0 0</td>
<td>0 0 0 0 0</td>
<td>0 0</td>
</tr>
<tr>
<td>Sensory impairment</td>
<td>P I P I I</td>
<td>P I I I I</td>
<td>I I</td>
</tr>
<tr>
<td>PIN</td>
<td>+ + + + +</td>
<td>0 + + + +</td>
<td>+ +</td>
</tr>
<tr>
<td>Touch</td>
<td>+ + + + +</td>
<td>0 + + + +</td>
<td>+ +</td>
</tr>
<tr>
<td>Positions</td>
<td>+ + + + +</td>
<td>0 + + + +</td>
<td>+ +</td>
</tr>
<tr>
<td>Visual fields</td>
<td>LHH LHE LHE LHE LHE</td>
<td>NL LHH LHH LHH LHH</td>
<td>LHE LHE</td>
</tr>
<tr>
<td>MMSE score</td>
<td>16 10 21 23 27</td>
<td>24 24 26 18 20</td>
<td>28 28</td>
</tr>
<tr>
<td>Neglect score†</td>
<td>29 20 37 28 20</td>
<td>0 29 0 7 31</td>
<td>28 31</td>
</tr>
<tr>
<td>Anosognosia score‡</td>
<td>10.0 7.5 6.0 6 9</td>
<td>0 3 2 4 1</td>
<td>4 4</td>
</tr>
<tr>
<td>Lesion location (right hemisphere)</td>
<td>TP FTP FTP Th FTP CR BG BG</td>
<td>F FTP FTP FTP FTP TPO CR</td>
<td></td>
</tr>
<tr>
<td>Asomatognosia</td>
<td>+ + + + +</td>
<td>- - - - -</td>
<td>- -</td>
</tr>
</tbody>
</table>

*Based on Medical Research Council of Great Britain Criteria.†,‡ Anosognosia score ranges from 0 (no anosognosia) to 10 (maximum anosognosia). ILM=illuminated lesion; CLMs=clinical lesions; CLMs+=present; CLMs−=absent; ILM+/-=ambiguous; LHH=left homonymous hemianopia; LHE=left hemiextinction; NL=normal; MMSE=mini mental state examination (score represents total of possible 30); F=frontal; T=temporal; P=parietal; O=occipital; Th=thalamus; BG=basal ganglia; CR=corona radiata.

Results

Of the 11 patients examined for ILMs, we found the following: five patients reported movement of the plegic left limb when asked to raise the left arm alone and when asked to raise both arms and were considered to have ILMs; four denied movement of the limb in either circumstance and were considered not to have ILMs. Two patients reported movement of the left arm only when asked to raise both arms and were considered to have a partial form of ILMs (see discussion). Thus we compared the first group, ILM (+) (five cases) with the second group, ILM (−) (four cases, table 1).

ILM (+) patients had higher mean total scores on the anosognosia for hemiplegia scale than did ILM (−) patients (mean (SD) for ILM (+)=7.7 (1.8); ILM (−)=2.2 (1.7), and this difference was significant, (t=4.63, df=7, p=0.002). The ILM (+) patients also had a significantly greater degree of neglect on the cancellation task (mean (SD) for ILM (+)=26.8 (7.1) and for ILM (−)=9.0 (13.7) (t=2.53, df=7, p<0.05). By contrast there was no significant difference between the ILM (+) and the ILM (−) groups in cognitive impairment as measured by the MMSE (t=−1.05, df=7, p=0.33). A somewhat unexpected finding was that all patients with unequivocal ILMs had asomatognosia (5/5), and all patients without ILMs lacked asomatognosia (4/4), and these two phenomena were highly associated (Fisher's exact test, two tailed, p<0.01).

Discussion

This study confirms a relation between anosognosia for hemiplegia and confabulation. The most severe cases of anosognosia for hemiplegia were associated with both ILMs and asomatognosia. Patients with milder anosognosia for hemiplegia did not demonstrate either asomatognosia or definite ILMs. Conversely, patients who lacked ILMs had lower levels of anosognosia for hemiplegia and did not show asomatognosia. Whereas the degree of hemispatial neglect was statistically associated with the presence of ILMs, marked neglect was seen in one patient who lacked ILMs and in the two patients with partial ILMs.

We found a strong correlation between ILMs and asomatognosia. It stands to reason that a patient who imagines the existence of a normal limb contralateral to a brain lesion might disavow the actual, plegic limb. Our finding of an association between ILMs, asomatognosia, and anosognosia might also explain why, in some severe cases of anosognosia, demonstration of the patient’s weakness does not produce increased awareness of defect. Therefore, all patients with anosognosia for hemiplegia should be assessed for the ability to identify the plegic limb as their own.

Our findings have some bearing on the feed forward hypothesis which posits that patients with anosognosia for hemiplegia lack the intention to move a plegic limb and thus attribute immobility not to weakness but to lack of effort. This schema would predict that our patients with anosognosia for hemiplegia would deny movement of the arm. In fact, in our study, the degree of anosognosia for hemiplegia was strongly associated with the tendency to claim movement.

Conclusions drawn from our results are limited by the small sample. Also the operational definition of an ILM proved to be ambiguous as two patients confabulated movement of the plegic limb when asked to raise both limbs but did not confabulate when asked to raise the weak limb alone. Whereas neither of these patients presented with asomatognosia, one had mild
anosognosia, and both had substantial hemispatial neglect. It is possible that these partial cases may represent a mild form of the anosognosia-confabulation cluster or a stage of recovery from more severe forms of anosognosia, asomatognosia, and confabulation. Further study of this sub-group, possibly with longitudinal analysis, will be required to resolve this issue.

We thank Dr David P Bernstein, for assisting with the data analysis and Norma Kamen Bonilla for manuscript preparation.
