Pathogenesis of reduplicative paramnesia

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SUMMARY The incidence of reduplicative paramnesia was sampled with a structured interview in 50 consecutive alcoholic inpatients. Four had reduplicative paramnesia (RP group) and 46 did not (non-RP group). Three of four patients in RP group had acute right hemispheric lesions and none had a left hemispheric lesion; 19 non-RP patients had left hemispheric lesions, 2 had right, and 25 had none. These data are in keeping with the previous suggestions that the neuroanatomical basis for reduplicative paramnesia is an acute right hemispheric lesion superimposed on chronic diffuse or bifrontal deficit.

Reduplicative paramnesia1 is a specific disturbance in memory characterised by subjective certainty that a familiar place, person, object or body part has been duplicated. It is most commonly seen in postruntrumatic encephalopathy,2 although it has been described in other conditions as well, such as tumours of the third ventricle and upper brainstem, rupture of aneurysms of the circle of Willis and arteriovenous malformations, prefrontal lobotomy, after electroconvulsive therapy and other metabolic and toxic encephalopathies.3,4 It has been suggested that it is a result of a combination of right hemispheric and frontal-lobe pathology.4–11

These suggestions, however, emanate from analyses of single case studies or selected cases. To provide a better estimate of neuropathological correlates of reduplicative paramnesia, we administered a structured interview designed to elicit paramnesic phenomena to 50 consecutive patients who met the study’s criteria. Based on past experience in this centre, the following criteria for patients’ selection were adopted to increase the probability of discovering patients with reduplicative paramnesia. The patients were (1) at risk for diffuse cortical or bifrontal dysfunction secondary to alcoholism and (2) had an acute neurological event necessitating the admission.

Subjects and methods

Fifty consecutive alcoholic inpatients admitted for an acute event (stroke, trauma, intoxication, withdrawal state, etc.) were studied. All were males (age 30–76 years). None had an acute confusional state when studied. The structured questionnaire given to elicit reduplicative paramnesia is given in table 1. This questionnaire covered the possibilities of reduplicative paramnesia for place, person and body parts. It was initially administered by a neurology resident (HH) and the presence of reduplicative paramnesia was later verified independently by a board-certified neurologist (NPV) and a licensed neuropsychologist (MFG). The reduplicative paramnesia was rated as present only if the belief persisted for a week or more despite counter-argument. The clinical characteristics of the two groups were compiled in detail using both direct patient interviews and their clinical records. Three of the patients with reduplicative paramnesia were followed up to a period of one year jointly by the neurology resident and the staff neurologist.

The relative frequency of presence or absence of right hemispheric lesions in the two groups was tested with chi-square test for independence with Yate’s correction.

Results

Four patients had reduplicative paramnesia. Two of these had reduplicative paramnesia for place alone; one, for both place and person and one, for place and body parts. Examples of reduplicative paramnesia are shown in table 2. The clinical, radiological and neuropsychological characteristics of these four patients are shown in table 3. Forty-six patients did not show any evidence of reduplicative paramnesia. Three of four patients in the reduplicative paramnesia group had acute hemispheric lesions (all right sided).

One patient (with reduplicative paramnesia for place, No 1, table 3) had no clinical or computed tomographic evidence of a hemispheric lesion. Complete neuropsychological batteries were available on three patients with reduplicative paramnesia. Two of
the patients showed adequate retention of new information as indicated by Wechsler Memory Quotients in the normal range (Cases 1 and 4). All three patients showed (1) a superiority of verbal intellectual skills over spatial reasoning skills in varying degrees (WAIS-R verbal IQ superior to performance IQ); (2) deficits on frontal lobe mediated measures of self-regulation [proportionately slower speeds on Trails B (alternating between numbers and letters) than Trails A (connecting numbers only)], and motor programming (impaired Luria 3-Step Test); and (3) an intact language ability, as suggested by adequate naming ability (Boston Naming Test) and intact WAIS-R vocabulary.

Forty-six patients did not show evidence of reduplicative paramnesia. Of these, 21 had acute hemispheric lesions, mostly left sided (19/21). The relative frequency of right hemispheric lesions in the reduplicative paramnesia group compared with that in non-reduplicative paramnesia group using 2 x 2 contingency table chi-square test with Yates’s correction was significant ($\chi^2 = 5.28, df = 1, p < 0.05$).

Two patients improved rapidly over several weeks.

One patient showed persistent reduplicative paramnesia even after one year. One patient (case 2) was lost to follow up.

### Discussion

Pick\(^1\) used the term reduplicative paramnesia for the first time to describe reduplicative beliefs in a patient presumed to have degenerative brain disease. The pathogenesis was felt to be diffusely cortical. Weinsten and Kahn\(^3\) postulated reduplicative paramnesia to be a form of denial of illness expressed in metaphorical language. The neuroanatomical basis was nonspecific, although unlike Pick’s original case, many patients had focal brain lesions. Benson, Gardner and Meadows\(^4\) described three patients with bilateral and right hemispheric damage secondary to head injury. They postulated reduplicative paramnesia to be a compound symptom: The right hemispheric dysfunction alters spatial coding of the environment and severe frontal dysfunction prevents recognition of this alteration. Similar cases had been described earlier by Paterson and Zangwill.\(^6\)

### Table 1

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>64</td>
<td>63</td>
<td>23</td>
<td>56</td>
<td>—</td>
</tr>
<tr>
<td>Type of RP</td>
<td>Place, person</td>
<td>Place</td>
<td>Place, body parts</td>
<td>Place</td>
<td>—</td>
</tr>
<tr>
<td>CT scan</td>
<td>No focal lesion</td>
<td>Place</td>
<td>R Brain infarction</td>
<td>R Brain contusion</td>
<td>R Brain infarction</td>
</tr>
<tr>
<td>Verbal IQ</td>
<td>100</td>
<td>—</td>
<td>78</td>
<td>85</td>
<td>85–115</td>
</tr>
<tr>
<td>Performance IQ</td>
<td>85</td>
<td>—</td>
<td>72</td>
<td>70</td>
<td>85–115</td>
</tr>
<tr>
<td>Wechsler MQ</td>
<td>94</td>
<td>—</td>
<td>62</td>
<td>105</td>
<td>85–115</td>
</tr>
<tr>
<td>Trails A</td>
<td>43 s</td>
<td>—</td>
<td>85 s</td>
<td>55 s</td>
<td>20–40</td>
</tr>
<tr>
<td>Trails B</td>
<td>145 s</td>
<td>—</td>
<td>231 s</td>
<td>365 s</td>
<td>40–112</td>
</tr>
<tr>
<td>Trails B/A</td>
<td>3/4/1</td>
<td>—</td>
<td>2/7/1</td>
<td>6/6/1</td>
<td>2/1</td>
</tr>
<tr>
<td>Luria 3-Step†</td>
<td>3</td>
<td>—</td>
<td>5</td>
<td>13</td>
<td>20–36</td>
</tr>
<tr>
<td>Boston Naming</td>
<td>58</td>
<td>—</td>
<td>53</td>
<td>51</td>
<td>30–58</td>
</tr>
</tbody>
</table>

*Had only brief bedside neuropsychological evaluation.
†Number of 3-step sequences completed in 20 seconds, right and left hand combined.

### Table 2

<table>
<thead>
<tr>
<th>Type</th>
<th>Example</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Place</td>
<td>“I am at Allen Park VA in California.”</td>
<td>Patient was at Allen Park VA in Michigan.</td>
</tr>
<tr>
<td></td>
<td>“I stayed at Henry Ford hospital ship.”</td>
<td>Patient was admitted to Henry Ford Hospital following an injury sustained on a ship.</td>
</tr>
<tr>
<td>Person</td>
<td>“My wife is dead.”</td>
<td>Patient’s wife was deceased, patient had not remarried, was not living with anyone.</td>
</tr>
<tr>
<td></td>
<td>“I’ll go home and talk to my wife about whether I should be admitted.”</td>
<td></td>
</tr>
<tr>
<td>Body-parts</td>
<td>“I cannot use my elbow. It’s an animal’s arm.”</td>
<td>Patient had torn ligaments and fractures in right arm.</td>
</tr>
<tr>
<td></td>
<td>“My real arm is in California.”</td>
<td></td>
</tr>
</tbody>
</table>
Pathogenesis of reduplicative paramnesia

Our data, based on a consecutive series rather than selected cases, are in keeping with these observations. The hemispheric lesions in patients with reduplicative paramnesia were all right-sided. Those in patients without reduplicative paramnesia were mostly left-sided. In addition, all reduplicative paramnesia patients showed selective deficits in spatial reasoning and relative sparing of verbal reasoning.

Evidence of diffuse cortical or bifrontal dysfunction in our study is inferred rather than visualised. It is supported by the study design (only alcoholic patients were studied; alcoholism has been shown to be a frequent cause of selective bifrontal dysfunction). In addition, impaired self-regulation and motor programming in our reduplicative paramnesia patients provided support for such dysfunction.

Thus, it appears that acute right hemispheric damage superimposed on chronic frontal deficit is the basic substrate for reduplicative paramnesia. The former, besides impairing visuospatial analysis also results in impaired facial recognition and memory, and emotional disorder (flat mood or euphoria). The frontal lobe lesion may contribute to the pathogenesis either by causing amnesia so that a location or person is recalled from remote memories or by disconnecting from temporolimbic structures leading to a distorted sense of familiarity, but not identity, about a place or person. The fact that two of our reduplicative paramnesia patients had adequate anterograde memory indicates that reduplicative paramnesia is not inconsistent with intact memory. In such cases, the bifrontal deficit may contribute by reducing the self critical analysis of one’s altered beliefs and memories.

References
Pathogenesis of reduplicative paramnesia.

H Hakim, N P Verma and M F Greiffenstein

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