Spatial disorientation in right-hemisphere infarction

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SUMMARY Spatial orientation was tested with the rod orientation test. The subjects were 40 normal controls and 68 brain-damaged patients with cerebral infarcts. Patients in whom the lesion included the post-rolandic region of the right hemisphere performed worse than controls or patients with lesions at other sites. Patients with an exclusively postrolandic (usually occipital) lesion showed higher error rates than patients with a combined prerolandic and postrolandic lesion, but only for the visual part of the test. These patients were re-examined one year after the stroke. Most of them showed an incomplete recovery of spatial function.

Clinical case studies and experimental work support the view that for spatial functions there is both a regional and a hemispheric specialisation of the cerebral cortex.¹⁻⁴ Impairment of spatial orientation appears to be predominantly related to damage of the posterior region of the right hemisphere. Some authors have stressed only the relevance of the antero-posterior dimension but not of the hemispheric asymmetry,⁵⁻⁷ while others have confirmed the role of the right hemisphere, but not the relevance of the left posterior region.⁸⁻¹¹ The differences between these studies were probably task-dependent. Most of the tests for assessing spatial abilities were derived from intelligence tests, or were designed to examine other well-known clinical defects such as constructional apraxia or topographical disorientation. As a result, spatial perception has been studied at a rather complex level, one at which it is difficult to differentiate spatial from other factors.

De Renzi *et al*¹² devised a rod orientation test to study the perception of spatial co-ordinates, based on the fact that the appreciation of the direction of a segment in space is one of the factors underlying spatial perception. They found that when spatial perception is tested at this simple level, there is an almost complete dominance of the posterior region of the minor hemisphere. However, the selection of patients for this study was not ideal. Firstly, patients

with an anterior lesion of the right hemisphere were few, as the authors themselves noted. Secondly, the site of the lesion was deduced from the presence or absence of a visual field defect, which is imprecise. Finally, they included not only patients with vascular lesions, but also patients with tumours or other brain lesions, without information about the presence of oedema or of a midline shift.

In the first part of our study we tried to elucidate the role of different regions of the right hemisphere in spatial orientation, we tried to overcome the problem of site and aetiology by testing only patients with a cerebral infarct, as verified by CT scanning. In the second part, we investigated the extent of recovery by re-examining the same patients one year after the stroke, for it is known that with other neurological deficits spontaneous recovery rarely continues after one year. Only Campbell and Oxbury¹³ examined a group of patients 3-4 weeks and 6 months after a stroke. They found no improvement in the performance of visuo-spatial tasks. That so little is known about the recovery of spatial perception is probably owing to the fact that most previous studies included not only patients with vascular lesions, but also patients with tumours and other lesions. The natural course of these diseases made a follow-up study impossible.

Methods

Subjects

We investigated 68 patients with damage to either the left or the right hemisphere. All patients had a cerebral infarct without a midline shift, as assessed by CT scanning. There were 33 patients with a left hemisphere lesion (21 males, 12 females, the ages ranging from 25 to 75 years, mean age 57.4 years) and 35 patients with a lesion of the right

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Table 1 Site of the lesion¹⁵

		Left hemisphere	Right hemisphere
Exclusively prerolandic lesion	Frontal	3	7
	Fronto-temporal	4	2
Prerolandic and postrolandic lesion	Fronto-parietal	1	
	Fronto-temporo-parietal	2	1
	Temporo-parietal	4	5
	Temporal + basal ganglia	1	2
	Temporal	8	
Exclusively postrolandic lesion	Temporo-occipital	2	5
	Temporo-parieto-occipital	1	3
	Occipital	7	10
		33	35

hemisphere (20 males, 15 females, the ages ranging from 30 to 80 years, mean age 66.4 years). Table 1 shows the site of the lesions in these 68 patients. We distinguished three main groups: exclusively prerolandic lesions, lesions with a prerolandic and postrolandic extension, and exclusively postrolandic lesions, as inferred from the CT scan. For the left hemisphere there were 7, 18 and 8 patients in the three groups respectively, and for the right hemisphere 9, 13 and 13 patients. In the group of patients with a lesion of the left hemisphere, 15 had aphasia. The neurological signs of the patients were recorded, along with age, sex, dexterity, level of education, occupation, and medication. In the follow-up study only the patients with a prerolandic and postrolandic lesion, or an exclusively postrolandic lesion of the right hemisphere were re-examined. In each of these two groups one patient refused to participate again in this study. The control group consisted of 40 subjects (26 males, 14 females) without evidence or history of brain disease. Seven persons in this group were left-handed. The ages ranged from 18 to 72 years (mean 47.5 years). All the control subjects underwent an intelligence test (Raven progressive matrices test).¹⁴

Testing procedure

The method used was the rod orientation test as employed previously by de Renzi *et al.*¹² Both the visual and the tactile part of the test were carried out by the brain-damaged patients with the hand on the side of the cerebral lesion. Because the timing of the test could be important in view of a possible restoration of function, all patients were tested two weeks after the stroke.

In the visual part of the test the patient was not allowed to touch the rods of the model and he was requested to put those of the other pair (copy) in the same position. In the tactile part of the test the patient was blindfold and had to estimate the spatial position of the rods of the model by palpation; otherwise the instructions were identical. No time limit was set. The patient was permitted to go from the model to the copy and back again as often as he wished. The order of presentation of the two tests alternated from patient to patient. In both the visual and the tactile part of the test, the patients were first given five trials with the model placed on the side of the lesion and then five trials with the model placed on the other side, each time with the rods in a different position. Half the subjects in the control group were instructed to use the left hand, while the other

half used the right. (Three of the seven left-handed patients used the right hand.) After each trial the differences between the copy and the model were measured in the horizontal and the vertical plane. The measurement of the angles of the rods was done in the same way as described by de Renzi *et al.*¹² The same applied to the test positions of the model in the five trials.

Results

There were four mean error rates for each part of the test: horizontal error, vertical error, model left or right. The figure shows the mean error rates (± 2 SEM) for the control group and for the six groups of patients. In the control group correlation coefficients were calculated to examine the influence of age or intelligence on the test results. These factors appeared to be of no importance. Furthermore, there was no difference in mean error rates between the patients with aphasia and the other patients with damage of the left hemisphere. Within the control group there were no differences between the error rates made with the left or the right hand. In the further statistical analysis these two groups were taken together. Lastly, there were no differences in mean error rates according to the side of presentation of the model, neither for the control group, nor for the six brain-damaged subgroups. In view of this last finding, further statistical analysis was done on the mean of all individual error rates.

The hypotheses to be tested were whether the error rates differentiated between the performance of (1) the control groups and brain-damaged subgroups, and between (2) the six different subgroups of brain-damaged patients. For this purpose an analysis of variance was used. It was expected that patients with a posterior lesion of the right hemisphere would have the highest mean error rates for both parts of the test.

On analysing the first hypothesis, we found a difference between the control group and the six brain-damaged subgroups together on both mod-

Figure Mean error rates plus or minus twice the standard error of the mean.

alities tested ($p < 0.001$). Analysis of the second hypothesis showed that there was a difference within the performances of the six brain-damaged subgroups ($p < 0.001$). Intra-hemispheric comparison of the left-brain-damaged subgroups yielded no difference. Inter-hemispheric comparison yielded a difference for both parts of the test: patients with left-sided lesions at any site performed better than patients with right prerolandic and postrolandic or exclusively right postrolandic lesions, but not better than patients with right prerolandic lesions. Accordingly, in the right-hemisphere subgroups there was a difference for both parts of the test between patients with an exclusively prerolandic lesion on the one hand and patients with a combined prerolandic and postrolandic lesion ($p \leq 0.01$), or those with an exclusively postrolandic lesion ($p < 0.001$) on the other. In comparing the mean error rates of patients with a prerolandic and postrolandic lesion and those with an exclusively postrolandic lesion, there was a difference only for the visual part of the test ($p \leq 0.01$). The patients with an exclusively postrolandic lesion did worse. This is because patients with a combined prerolandic and postrolandic lesion performed better in the visual part of the test than in the tactile part.

In the follow-up study the hypotheses to be tested were whether the error rates differentiated between the performance of (1) the re-examined groups at two weeks and one year, and between (2) the re-examined groups on the one hand and the control groups or other brain-damaged subgroups on the other. For statistical analysis the "Wilcoxon matched pairs signed rank test" and an analysis of variance were used. Table 2 shows that in the year after the stroke there was a change in performance in the two groups as a whole, for the visual as well as for the tactile part of the test. Of the twelve patients with a combined prerolandic and postrolandic lesion, four still showed a difference in performance between the visual and the tactile part of the test (the mean error rates for the tactile part were at least twice the mean error rates of the visual part). These were the only patients who showed a tactile extinction phenomenon and who had the highest mean error rates on the initial clinical examination. In the patient group with an exclusively postrolandic lesion, one patient still performed poorly in both parts of the test (the error rates for both parts of the tests were at least twice the mean error rates of all patients in this group). On re-examination she not only had a left homonymous hemianopia, but also

Table 2 Comparison of the mean error rates of the performance two weeks and one year after the stroke

			Mean error rates		
RH exclusively postrolandic 2 weeks—1 year N = 13 N = 12	visual	horizontal	15.87°	4.25°	p ₂ < 0.01
	visual	vertical	9.84°	3.66°	p ₂ < 0.01
	tactile	horizontal	21.86°	4.71°	p ₂ < 0.01
	tactile	vertical	11.50°	4.68°	p ₂ < 0.01
RH prerolandic and postrolandic 2 weeks—1 year N = 13 N = 12	visual	horizontal	13.32°	3.63°	p ₂ < 0.01
	visual	vertical	6.96°	3.53°	p ₂ < 0.01
	tactile	horizontal	30.71°	5.75°	p ₂ < 0.01
	tactile	vertical	16.63°	5.76°	p ₂ < 0.01

RH: right hemisphere.

an impaired memory for the localisation of objects, and defective route finding.

In calculating the size of the lesions from the CT scans, we found that these five patients had the largest lesions, ranging from 62 to 88 cm³ (mean 78 cm³). The volumes of the lesions of the other patients with a combined prerolandic and postrolandic or an exclusively postrolandic lesion of the right hemisphere ranged from 3 to 56 cm³. No other patient had any complaints after one year that could be attributed to spatial disorientation. In comparing the test results of the re-examined patients with those of patients with an acute lesion at sites other than the posterior right hemisphere, no difference was found. But there was still a difference between the mean error rates of the control subjects and the re-examined patients ($p \leq 0.01$).

The exact site of the lesion was assessed by CT scanning. In all the patients with a right posterior lesion the CT scan pictures were drawn (at normal scale) by means of a pantograph. All these lesions were found to have one area in common which included, (a) on the cut at the level of the cella media: the white matter between the lateral occipital gyrus and the parieto-occipital sulcus, and (b) on the slice below the previous one: the white matter between the lateral occipital gyrus, middle temporal gyrus and the parieto-occipital sulcus.¹⁵

Discussion

Patients with an exclusively postrolandic lesion of the right hemisphere had the highest mean error rates in both the visual and the tactile part of the test, as expected. This group consisted of ten patients with an occipital infarct, and three with a temporo-parieto-occipital infarct. All these patients had a visual field defect. That the relevance of this site does not depend on a defect of visual functions is clear from the mean error rates of patients with a defect in the right visual hemifields from lesions of the left hemisphere. The most striking findings in the patient group with a lesion of the right hemisphere

are those in the patients with a combined prerolandic and postrolandic lesion. In this group the mean error rates were for the tactile version of the test almost as high as those in the group with an exclusively postrolandic lesion. In the visual version, however, the patients with a prerolandic and postrolandic right-hemisphere lesion performed better than the patients with an exclusively postrolandic lesion, but worse than the patients with an exclusively prerolandic lesion. Independence of test modality in spatial disorientation was found only in patients with an exclusively postrolandic lesion of the right hemisphere. The highest error rates for the tactile version of the test were obtained in patients with a predominantly right parietal lesion and a tactile extinction phenomenon on clinical examination. These patients often were incapable during the test and did not know what to do with the rods. Both the patients with a lesion of the left hemisphere and those with an anteriorly located lesion of the right hemisphere performed worse than the control subjects. This mild deterioration of performance in patients with a left-hemisphere lesion, or an exclusively prerolandic lesion of the right hemisphere was, perhaps, a nonspecific effect of disease (two weeks after the stroke).

Thus the finding of de Renzi *et al* that the right posterior region of the brain plays a very important role in spatial perception not only has been confirmed, but also has been extended for we also could make a further subdivision within the right hemisphere. That we found these subdivisions, while earlier investigations did not, may have been the result of a more precise localisation and of the exclusion of remote effects (defined by computed tomography). Benton *et al*¹¹ found a positive association between the presence of a right posterior lesion and an impaired performance in a test that involved judgment of line orientation in one plane, but the test was presented only by visual means.

The results of the follow-up study show that a distinct recovery from spatial disorientation occurred after one year in the majority of patients with

infarction of the posterior region of the right hemisphere. Campbell and Oxbury¹³ did not find an improvement on re-examination six months after the stroke. There are several possible explanations for this difference from our results: (1) patient selection. Infarcts in the posterior region of the right hemisphere in our study, the presence of visuo-spatial neglect in theirs, (2) complexity of the test. Only when spatial perception is tested at a very basic level is there an almost complete dominance of the right posterior region. When a task requires the elaborate integration of spatial data, the left hemisphere contributes as well (De Renzi *et al*²), (3) the time when the patients were tested initially (two weeks after the stroke in our study; three to four weeks in theirs). A slight restoration of function could occur in this period. The anatomical substrate underlying the recovery of spatial perception is not yet known. It is not clear, for instance, whether this function is compensated within the right hemisphere or "transferred" to the posterior region of the left hemisphere. This issue deserves further investigation.

Computed tomography enabled us to assess the site and extent of the lesion fairly accurately. It is possible that the "common area" of the cerebral lesions in patients with the highest mean error rates in both the visual and the tactile part of the test is important in the integration of spatial perception.

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