THE ROD ORIENTATION TEST IN PATIENTS WITH RIGHT-HEMISPHERE INFARCTION

A clinical study of spatial perception in 154 subjects.

PROEFSCHRIFT

TER VERKRIJGING VAN DE GRAAD VAN DOCTOR IN DE
GENEESKUNDE
AAN DE ERASMUS UNIVERSITEIT ROTTERDAM
OP GEZAG VAN DE RECTOR MAGNIFICUS
PROF. DR. J. SPERNA WEILAND
EN VOLGENS BESLUIT VAN HET COLLEGE VAN DEKANEN.
DE OPENBARE VERDEDIGING ZAL PLAATSVINDEN OP
WOENSDAG 8 DECEMBER DES NAMIDDAGS
TE 3.45 UUR

DOOR

JOHANNES DAVID MEERWALDT
geboren te Rotterdam

1982

grafische verzorging:

ROCO DAVIDS
ALBLASSERDAM

PROMOTOR : PROF. DR. A. STAAL

CO-REFERENTEN : PROF. DR. H. VAN CREVEL
PROF. DR. M.W. VAN HOF

Aan mijn ouders
Aan Ien, Eliane en Diekje

From the Department of Neurology Academic Hospital 'Dijkzigt'
Rotterdam, The Netherlands

CONTENTS

CHAPTER 1

INTRODUCTION: HEMISPHERIC ASYMMETRY AND DISORDERS OF SPATIAL ORIENTATION

HEMISPHERIC ASYMMETRY

The concept of hemispheric cerebral dominance arose out of Broca's fundamental discovery (1861) of the association between motor aphasia and disease of the posterior left frontal lobe.

It may seem surprising that it took clinicians so long to discover what is so obvious today. Other principles of cerebral localization had been described much earlier, such as the innervation of the limbs by the contralateral hemisphere (Hippocrates, Pourfour du Petit 1710), or the association between loss of speech and lesions of the frontal lobes (Bouillaud 1825). But to assume a difference in function between the left and the right hemisphere of the brain, was a tremendous and daring step.

For some decades the concept of left-hemisphere dominance was applied only to language functions. However, with the work of Liepmann (1900) on apraxia and that of Gerstmann (1930) on disturbances of the body schema, the concept was extended to cover other aspects of mentation.

The left hemisphere was regarded as the "major" hemisphere by which the cognitive functions were mediated. Consequently, the right hemisphere was called the "minor" hemisphere of which substantial regions were often designated as "silent" areas with no specific function. There were, however, some clinicians who believed that the right hemisphere also possessed distinctive functional properties. Jackson (1876) in particular believed that the posterior area of the right hemisphere played a crucial role in subserving visual recognition and visual memory. He reported a patient with a tumour in the temporo-occipital region of the right hemisphere who had a facial agnosia and dyspraxia for dressing and who was visually disorientated on clinical examination. Jackson's ideas about the distinctive role of the right hemisphere were not generally accepted. Most neurologists and ophthalmologists were convinced that disturbances in visual perception (no recognition in spite of normal visual acuity) and orientation (disordered

appreciation of the spatial relationship between objects) reflected bilateral parieto-occipital disease.

At the beginning of the twentieth century Rieger (1909), however, postulated the existence of two differently lateralized cerebral mechanisms, one subserving verbal-conceptual functions and located in the left hemisphere, the other subserving spatial thought and located in the posterior region of the right hemisphere. His idea was rather similar to that expressed by Jackson. Babinski (1918) gave a description of anosognosia in the form of unawareness of a left hemiplegia. As more patients showed an apparently specific association of this defect of awareness with right-hemisphere disease, the question arose whether there was a centre for the integration of somatosensory information in the right hemisphere. Dide (1938) formulated a syndrome of the right parietal area, which consisted of spatial disorientation, constructional apraxia, anosognosia of Babinski and certain types of sensory and motor impairment.

As the evidence supporting the concept of right-hemisphere specialization consisted of only a few case reports, it was not until more comprehensive studies had been carried out during and after World War II that the right-hemisphere "dominance" for certain types of perception was more generally accepted. Especially important were the studies by Hécaen and co-workers (1951, 1956) and Zangwill and co-workers (Ettlinger, Warrington and Zangwill 1957; McFie, Piercy and Zangwill 1950). These authors examined relatively large samples of patients with localized right-brain damage and underlined the features marking their spatial performance. However, these studies lacked a systematic comparison with the performances of left-brain-damaged patients, and the evidence for right-hemispheric superiority on spatial tasks was not yet decisive.

Hécaen (1962) investigated the occurrence of spatial disorders in a large number of patients selected not because they showed a given symptom but because they had a unilateral post-rolandic lesion. Thus it became possible to estimate the frequency of spatial disorders after injury of either side of the brain. Contralateral neglect, inability to find the way on a map, loss of topographical memory, constructional apraxia and apraxia for dressing were all significantly more frequent in patients with right than with left-brain damage.

Finally, the investigation of patients who underwent a cerebral commissurotomy and also animal experimentation have contributed to our present knowledge of hemispheric asymmetry (Franco and Sperry 1977; Sperry, Gazzaniga and Bogen 1969; Teng and Sperry 1974; Zaidel, Zaidel and Sperry 1981). Comprehensive reviews of the history of cerebral dominance are given by Benton (1977) and De Renzi (1982).

DISORDERS OF SPATIAL ORIENTATION

EARLY HISTORY

In 1888 Badal published a detailed description of a patient with intact visual acuity but with severe impairment of the sense of space among other deficits.

It concerned a 31-year-old woman who was admitted to his hospital because of severe eclampsia. During the first weeks after delivery she hallucinated and appeared to be blind. Some weeks later it was noticed that although her visual acuity was normal, she could somehow not find her way about the hospital and had difficulty in localizing objects. On examination she could not answer simple questions about the interconnections of the main streets of Bordeaux, although she had long been familiar with them. Perimetry revealed loss of the lower half of the visual fields and a constriction of the upper visual fields. She could read letters and figures, but seemed to have lost all sense of direction with respect to printed material so that she was incapable of spelling words and of continuous reading. She showed severe impairment in estimating the size, distance, location and spatial relations of objects. She also showed a pronounced dyspraxia for dressing and an inability to draw even the simplest design from a model or from memory. The spatial disorientation extended to auditory stimuli as well. On the other hand, there were no disturbances in position sense: she had a correct idea of the position in which a limb was placed and, with her eyes closed, could reproduce the position correctly with the other limb.

Badal interpreted his patient's symptoms as reflecting a single basic disability that was separate from the visual system. He reached no definite conclusion on whether the deficits were organic or functional in nature.

Of the unfortunate victims of World War I, some survived bullet wounds in the back of the head, and this provided an opportunity to study disorders of visual orientation in young patients without the diffuse cerebral pathology and general mental impairment that so often complicated the clinical picture in older patients. Important observations were made by a number of authors, among them Holmes (1918) and Kleist (1918). Holmes described a series of carefully studied cases in all of whom the visual acuity was at least moderately good. The patients showed a variety of disturbances in visual behaviour, which Holmes divided into two major groups:

1. disturbances in spatial localization, in orientation and in the ability to estimate distance and size; and
2. disturbances in ocular fixation.

Included in the first group were deficits as inaccuracy in attempting to touch an object within reach, colliding with easily visible objects and unusual

difficulty in trying to circumvent them, inability to learn the way about the hospital and impairment in reading. Disturbances in ocular fixation were apparent when patients were asked to look at something, after which they would stare and then move their eyes in an apparently aimless fashion until they happened to find the object sought. Anatomical studies indicated that in all instances the lesions were bilateral and lay within the temporo-parieto-occipital area.

TYPES OF SPATIAL DISORDERS

Benton (1969) summarized the case reports of neurologists on this topic and drew up a list of seven main types of spatial disorders:

1. *Defective localization of stimuli in external space.*
   Inability to localize objects in space, to estimate their size, and to judge their distance from the observer. There is no simple relation between this disability and the presence or absence of a visual field defect. As for the errors in estimating distance, there is a tendency to underestimate the distance of remote objects and to overestimate the distance of near objects.
2. *Defective short-term memory for spatial location.*
   Many patients who perform adequately on spatial localization or discrimination tasks as long as the stimuli are present before them, will show a defect after removal of the stimuli, i.e. when a short-term memory element is introduced into the task (Alajouanine 1960).
3. *Defective route finding.*
   Inability to trace a path or follow a route from one place to another. This is essentially a disorder in execution and these patients often have a constructional apraxia as well. They may be quite capable of giving a verbal description of familiar routes, although these are likely to be lacking in detail. Brain (1941) explained the defective route finding of his patients (with right-hemisphere lesions) on the basis of a consistent tendency to avoid making turns to the left. He considered this tendency an expression of "neglect" of the left half of space. This explanation may be valid for many cases. There are, however, patients who do not show this preference for right turns in their defective route finding, but who instead show confusion at points where they must make a choice, with apparently random responses. In these cases there appears to be a failure in forming a basic spatial schema of the route.
4. *Reading and counting disability.*
   The dyslexia shown by these patients is of a spatial, not of a symbolic character. Single symbols (letters, words) are readily recognized, but continuous reading is faulty. One form is related to unilateral spatial

neglect. The patient initially fixated on a point which is at some distance to the right of the beginning of the line, he reads to the end of that line and then returns to a point on the next line which is again somewhat to the right of the beginning of the line. The result is that he cannot make any sense of what he reads and soon becomes confused. The other form of reading disability reflects an even more severe disturbance. The patient may start reading at varying points on a line and may also skip lines. Confronted with a text he may select words at random and polysyllabic words may be read backwards if they can be read at all. In milder cases only one or two words in a line may be skipped and sometimes the patient will insert words in an effort to make sense of what he is reading.

Defects in counting may exist together with these types of reading disability. Figures located in one or other part of the visual field may not be noticed, just as words at the beginning or the end of a line are ignored in reading.

5. *Defective topographical memory.*
   This is the inability to recall and describe familiar routes and the loss of former geographical knowledge. It represents a failure to retrieve long-established visual-spatial memories. The presence of this disability is established by having the patient describe familiar geographical constellations. Map tests are often used to assess geographical memory. Most patients with impairment of visual localization do not show loss of long-term topographical memory. Conversely, patients with a generalized memory defect are likely to have impaired topographical memory but no disturbance in visual localization.
6. *Visual-constructive disabilities.*
   Kleist (1918, 1934) considered constructional apraxia an impairment of translating intact visual perceptions into appropriate motor action. However, his strict formulation of the disability as being "executional" in nature was disputed by others. They thought of constructional apraxia as the psychomotor expression of a defect in spatial thinking. Later there were attempts to relate a visual-spatial type of constructional apraxia to lesions of the right hemisphere and an executional type (as formulated by Kleist) to lesions of the left hemisphere (Benton 1965, 1967). There is, however, little doubt that most patients who show impaired spatial orientation as expressed in disturbed localization, route finding, or other deficits, show serious impairment in visual-constructive activities as well. But not all patients with an impairment of visual-constructive activities show a disturbance in spatial orientation. Various types of failure may be shown. In making a block construction the patient may seem to be at a loss as to how to proceed, and finally may succeed in integrating only a few components into a partial simple structure.

With lesions of the right hemisphere, the patient may succeed in making the construction, but either rotates its position as compared with the model or rotates some parts within the construction. This is usually interpreted as an expression of "loss of directional sense". Another error is the omission of one half of a drawing or construction, as is shown by patients with unilateral spatial neglect.

7. *Simultaneous agnosia.*
   This is the inability to relate spatially separated objects or events to each other as in the interpretation of pictures portraying action. The patient suffering from simultaneous agnosia will be able to name some or all of the details but will fail to grasp the interaction among them. On questioning, he may complain that, as he fixates on one detail, the others disappear and that this prevents him from integrating successive experiences.

In older articles about spatial orientation (Badal 1880, Foerster 1890, Meyer 1900) the patients showed gross disturbances of several or all of the above-mentioned disabilities. The lesions were considered to be in both temporo-parieto-occipital areas. Spatial disorientation had also been described in patients with more anteriorly localized lesions, and even in patients with frontal lobe disease. Later studies of more elementary aspects of spatial orientation would show both a regional and a hemispheric specialization of the cerebral cortex.

RIGHT VERSUS LEFT HEMISPHERE IN SPATIAL ORIENTATION

McFie et al. (1950) described the clinical findings and the test results of eight patients with spatial disorientation (neglect of the left side of visual space, visual-constructive disabilities, apraxia for dressing, topographical disorientation, deformation of the visual co-ordinates) due to parieto-occipital lesions of the right hemisphere. They wondered whether the disabilities were correlated in any specific fashion with lesions of the minor hemisphere or whether they might be the result of unilateral parieto-occipital lesions of either hemisphere, as generally held at that time. The first supposition would be in accordance with other experimental observations by Lenz (1944), Paterson and Zangwill (1944) and Bender and Jung (1948) suggesting that the right parieto-occipital area may subserve some special function relating to visual-spatial cognition. Ten years later, McFie and Zangwill (1960) reported eight cases of visual-constructive impairment associated with temporo-parieto-occipital lesions of the left hemisphere. They found that in contrast with the previously described group with right-sided lesions the disability in this group was rarely associated with unilateral neglect, apraxia for dressing, or failure in tests involving spatial analysis, but

that it was frequently associated with right-left disorientation. There were also qualitative differences in the nature of the visual-constructive disturbance between the groups with left and right-sided lesions, the former showing more difficulty in manipulation. They concluded that the visual-constructive impairment in the group of patients with left-sided lesions corresponds to the classical description of constructional apraxia and that it is essentially different from the disability associated with spatial disorientation found with right-sided lesions.

Case histories of single or few cases were the principal source of data on spatial disorientation due to cerebral lesions. Although the value of careful studies of selected individual cases is undeniable, false inferences about the nature as well as the site of disease may be drawn from them. Apart from the pitfalls of determining the site and extent of tissue damage from clinical examination, analysis of the effect of localized dysfunction based upon selected cases is susceptible to several other sources of error:

a. The frequency of cases with a given lesion but without given deficits is not assayed.
b. When lesions are multiple, the deficits may be incorrectly ascribed to some of the lesions (commonly to the largest of them).
c. Abnormal performance in tests is sometimes assumed, without controls, or without sufficient data on the controls for the reader to judge the adequacy of matching.
d. Relative dissociation of deficits may be reported without regard to possible normal variability in these functions.

Carmon and Benton (1969) have investigated the accuracy of perception of the direction or the number of stimuli applied to the palmar surface of the hands in patients with unilateral disease, in either the right or the left hemisphere, and in patients without evidence or history of brain disease. The frequency of mistakes was higher in the contralateral hand than in the ipsilateral hand in both groups of patients in both somatoperceptual tasks. The two groups of patients showed no difference in the pattern of performance with respect to the tactile perception of the number of stimuli; there was approximately an equally severe deficit of the contralateral hand in each group, and the performance of the ipsilateral hand was not significantly different from normal. On the other hand, the two groups of patients showed a significant difference with respect to the tactile perception of direction. Although there was an equally severe deficit of the contralateral hand in the two groups, the performance of the ipsilateral hand was often also defective in the patients with lesions of the right hemisphere and essentially normal in the patients with lesions of the left hemisphere. Carmon and Benton concluded that these findings provided further evidence to support the

concept that the right hemisphere plays a more important role than the left in the appreciation of spatial relations. One year later Fontenot and Benton (1971) repeated the examination on another group of patients. Then, however, they only tested the perception of the direction of tactile stimuli, in order to exclude disparate information. Also, aphasic patients were now included in the patient population. The conclusions, however, were unaltered.

ANTERIOR VERSUS POSTERIOR CEREBRAL REGIONS IN SPATIAL ORIENTATION

Hannay, Varney and Benton (1976) described a study of visual localization in patients with unilateral cerebral disease. They found not only that the rates of patients with a right-hemisphere lesion were significantly worse than those of patients with a lesion of the left hemisphere, but also that within the right-hemisphere group, patients with a posteriorly localized lesion (temporo-parietal, parietal) had the highest error rates. They concluded that the right hemisphere and particularly the posterior area plays an important role in subserving visual localization. De Renzi et al. (1968, 1969) came to the same conclusion with relation to the tactile modality. Nevertheless, some authors only stressed the relevance of the antero-posterior dimension, but not the hemispheric asymmetry (Semmes et al. 1955, Butters and Barton 1970, Benson and Barton 1970). Semmes et al. tested the visually and tactually-guided behaviour in 62 subjects with traumatic injuries to the brain, and in 17 control subjects. The test required the subject to follow (walking) routes represented on maps. Five of the path diagrams were perceived visually; ten other routes, five for each hand, were perceived by touch alone. The tactual maps were rotated or mirrored images of the visual ones. The result was that the mean rates of the groups with a lesion of the parietal lobe, either left or right, were significantly inferior to those of the control group and of the other brain-damaged subgroups. Butters and Barton tested 35 patients (31 patients with damage to the left hemisphere and only 4 with damage to the right hemisphere) in three tasks requiring horizontal and vertical rotations. They found that damage to either the right or the left parietal lobe resulted in impairment in all three tasks, whereas lesions of other cortical areas resulted in slight if any deficits. They suggested that an inability to form different perspectives in imagination underlies many of the impairments associated with parietal lobe damage.

THE ROD ORIENTATION TEST

The analysis of the main types of spatial disorders (Benton 1969) clearly shows that each of these types may result from disruption of different

mechanisms (for instance, defective route finding is produced by unilateral neglect as well as by a failure to build up the basic spatial schema expressed in the route), but it is also evident that derangement of a single function may be reflected in symptoms classified under different categories (defective scanning can underly defective localization of stimuli, reading and counting disabilities as well as visual-constructive apraxia). Although the mechanisms underlying spatial orientation are imperfectly understood, spatial disorders could be subdivided as follows:

1. disorders of space exploration and localization
2. disorders of space perception
3. disorders of spatial memory.

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. (1971) pointed out that when spatial perception is tested at an elementary level, by asking subjects to copy the position of an adjustible rod (rod orientation test), there is an almost complete dominance of the posterior region of the minor hemisphere. I have chosen this test as the main instrument in my studies of spatial perception.

AIMS OF THE PRESENT STUDY

From our present knowledge the selection of patients in the studies discussed so far can be criticized in several respects:

a. The site of the lesion was often deduced from the neurological signs, for instance the presence or absence of a visual field defect, which is imprecise.
b. The cause of the lesion could be described only as vascular, neoplasm, or trauma, without information about oedema or a midline shift.
c. In many studies the age, sex and cause of the disease were different for patients with damage to the left and to the right hemisphere.
d. It was rarely mentioned how many patients with damage to the left hemisphere had aphasia.
e. The neurological signs were not recorded.
f. In several studies the number of patients in some subgroups was too small to allow valid conclusions from the test results.

The advent of computed tomography (CT) has largely eliminated the problem of determining the site, size and nature of cerebral lesions.
This has allowed me to elucidate further the role of different regions of the

right hemisphere in spatial perception by applying the rod orientation test (De Renzi et al. 1971) only to patients with cerebral infarction, with known size and without a midline shift.
Furthermore, the extent and the speed of recovery during the first year after the stroke were studied, the influence of the volume of the lesion on the extent and the speed of recovery was assessed, and the test results of the rod orientation test were compared with the rates of the line orientation test (Benton et al. 1978).
Thirdly the development of spatial perception in children was studied.
Finally, I investigated a patient with agenesis of the corpus callosum, to test the hypothesis that such a patient would show a disconnection syndrome for spatial information.

CHAPTER II

THE ROD ORIENTATION TEST IN PATIENTS WITH RECENT CEREBRAL INFARCTION

PART I
RELATION BETWEEN THE SITE OF THE LESION AND THE TEST PERFORMANCE

Many patients show obvious signs of spatial disorientation on clinical examination as mentioned in Chapter I (inability to localize objects in space, impaired memory for the localization of objects or places, etc.). Slight disturbances of spatial perception are sometimes noticed by the patient, especially in certain occupations such as carpenter or tailor. Others, however, only show a disturbance of spatial perception on formal testing.

Spatial perception can be measured by asking the patient to state when a luminous line, projected on a screen, slowly rotated by the examiner, appears to be straight up. The displacement of the subjective vertical with respect to the true vertical represents the score.

Twenty control subjects and 40 patients were tested in this way by Bender and Jung (1948). Each hemisphere group consisted of 20 patients with a frontal, temporal, parietal or occipital lesion. Patients with a lesion of the frontal or the parietal lobes had the highest error rates. Furthermore, the error rates of the right-brain-damaged patients were higher than those of the left-brain-damaged patients. The findings of Bender and Jung were confirmed in another study by Tzavaras and Hécaen (1971).

In clinical studies dating back to World War II the importance of the posterior region of the right hemisphere in estimating line orientation had already been emphasized. In 1944 Lenz reported that six of 56 patients with war injuries of the brain showed a systematic error when required to hold a stick, held in the middle, in a vertical or horizontal position or to draw a vertical and a horizontal line on the table. The error was particularly apparent when the patient was blindfolded, and consisted in the rightward inclination of the top end of the stick when asked to hold it in the vertical position, and in the upward inclination of the right end of the stick when

asked to hold it in the horizontal position. Two patients had a right parietal lesion, in the other four the lesion involved both parietal lobes. No distortion of spatial co-ordinates was found in three patients with left parietal damage. Lenz's findings were confirmed by McFie and co-workers when the same test was given to ten patients with visual-constructive disability; five had a right parieto-occipital lesion (McFie, Piercy and Zangwill 1950), the other patients had a posterior parietal lesion of the left hemisphere (McFie and Zangwill 1960). Three patients in the first group showed the same distortion of spatial co-ordinates as found by Lenz. The patients with a lesion of the left hemisphere made no error of this type.

In Aubert's phenomenon the patient states when a luminous line is vertical, after his first being tilted to the right or to the left of the midline. In normal subjects the procedure produced either a systematic deviation of the line to the side of the body tilt (A error) or to the opposite side (E error).
This test was done by Teuber and Mishkin (1954) in a group of patients who had suffered penetrating gunshot wounds of the brain 7-10 years before. E errors were found in all patients, but they were significantly higher in those with pre-rolandic lesions than in the patients with post-rolandic lesions. However, when the brain-damaged patients were subdivided according to left, right or bilateral hemispheric damage, only the patients with right-sided lesions did significantly worse than the controls. From these results it could be concluded that the right hemisphere is involved in determining the visual vertical, but that different regions of the hemisphere play a critical role depending on the nature of the task; when it requires only visual abilities, the integrity of the parietal lobe is crucial, when it implies the integration of postural with visual functions the performance is disrupted by frontal lobe damage (De Renzi 1982).

The reproduction of various positions of a rod in space is another way to assess spatial perception, and for this purpose De Renzi et al. (1971) devised the rod orientation test. They found an almost complete dominance of the posterior region of the right hemisphere. However, as pointed out earlier, this study was not ideal with regard to the selection of patients. First, patients with an anterior lesion of the right hemisphere were few, as the authors noted themselves. Second, the site of the lesion was deduced from the presence or absence of a visual field defect, which is imprecise. Finally, their subjects included not only patients with vascular lesions, but also patients with tumours or other brain lesions, without any information about the presence of oedema or of a midline shift.

I have tried to elucidate the role of the different regions of the right hemisphere in spatial perception, while eliminating the problem of site and aetiology by testing only patients with a cerebral infarct, as assessed by CT scanning.

METHODS

Subjects

68 patients with damage to either the left or the right hemisphere were investigated. 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 58.4 years), and 35 patients with a lesion of the right 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 lesion in these 68 patients.

Table 1. Site of the lesion (mapping of cerebral cortex from CT scans after Gado et al. 1979).

		left hemisphere	right hemisphere
exclusively pre-rolandic lesion	Frontal	3	7
	Fronto-temporal	4	2
pre- and post-rolandic lesion	Fronto-parietal	1	
	Fronto-temporo-parietal	2	1
	Temporo-parietal	4	5
	Temporal + basal ganglia	1	2
	Temporal	8	
	Temporo-occipital	2	5
exclusively post-rolandic lesion	Temporo-parieto-occipital	1	3
	Occipital	7	10
		33	35

Three main groups could be distinguished: exclusively pre-rolandic lesions, lesions with a pre- and post-rolandic extension and exclusively post-rolandic lesions. For the left hemisphere this amounted to 7, 18 and 8 patients respectively, and for the right hemisphere to 9, 13 and 13 patients. Of the 33 patients with a lesion of the left hemisphere, 24 had aphasia. The neurological signs (table 2) of the patients were recorded, along with age, sex and dexterity.

The control group consisted of 40 patients (26 males, 14 females) visiting the out-patient department, without a history or signs of brain disease, but suffering from disc lesion, lumbago or mononeuropathy. 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) (Raven 1972).

Table 2. Neurological signs.

		left hemisphere	right hemisphere
slight weakness	arm		3
	leg		1
	both	8	7
moderate weakness	arm		1
	leg		
	both	4	4
severe weakness	arm		
	leg		
	both	5	4
sensory impairment		12	20
isolated visual field defect		5	10
isolated aphasia		5	

Testing procedure

The method used was the rod orientation test as devised by De Renzi et al. (1971). 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 might be important in view of a possible restoration of function, all patients were tested two weeks after the stroke.

Figure 1 shows the apparatus which consists of two pairs of rods, 38 cm apart, fixed on a board of 55 cm x 15 cm. Each pair is made up of a vertical rod which can rotate 360° around its axis, and of a second rod, which is fixed to the first by a hinged joint in such a way that it can pivot up and down in the sagittal plane.

The patient was seated in front of the apparatus and was permitted to move his head and eyes but not his trunk. One pair of the rods represents the model and the patient was asked to set the rods of the other pair in the same position. There were two versions of the test given, by inspection and by palpation. 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 blindfolded and had to estimate the spatial position of the rods of the model by palpation; otherwise the instructions were identical. The time was recorded, but no time limit was set. The patient was permitted to go from the

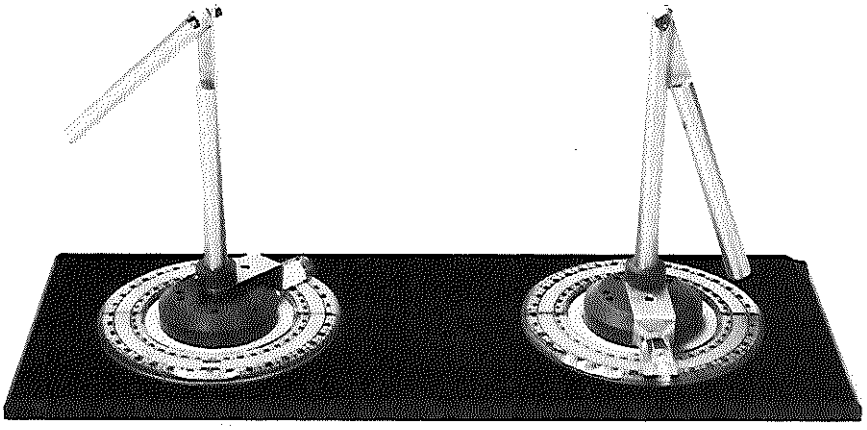

Figure 1. Apparatus used in the rod orientation test.

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 (table 3).

Table 3. Spatial co-ordinates of the model in the five trials.

vertical angle	horizontal angle
90°	180°
45°	90°
130°	320°
60°	160°
150°	230°

Of the subjects in the control group, half were instructed to use the left hand, while the other half used the right. (Three of the seven left-handed patients used their right hand.) After each trial the differences between the copy and the model were measured in the horizontal and the vertical plane. At the beginning of each trial the position of the test rods was always the same, with the second rod pivoted down on the first and facing the patient.

RESULTS

There were four mean error rates for each part of the test: horizontal error, vertical error, model left or right. Figure 2a and 2b show the mean error rates plus or minus twice the standard error of the mean of the control group and the six groups of patients. In the control group, correlation co-efficients 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 statistically significant difference in mean error rates between the patients with aphasia and the other patients with damage of the left hemisphere. Thirdly, within the control group there were no significant 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 significant 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.
Table 4 shows the mean duration of the test in the visual and the tactile modality, both for the control group and the brain-damaged subgroups.
Table 5 shows the mean duration of the test in the control group for the individual patients both for the left and the right hand.

The hypotheses to be tested were whether the error rates differentiated between the performance of:
1. the control groups and the brain-damaged subgroups,
2. the six different subgroups of brain-damaged patients.

For this purpose an analysis of variance was used. From the study of the literature 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 analysis of the first hypothesis, there was a statistically significant difference between the control group and the six brain-damaged subgroups together, in both modalities tested (table 6).

Table 4. Mean duration of the test (in seconds).

	visual	tactile
Controls	25	69
RH exclusively pre-rolandic	28	60
RH pre- and post-rolandic extension	31	69
RH exclusively post-rolandic	47	80
LH exclusively pre-rolandic	26	65
LH pre- and post-rolandic extension	24	58
LH exclusively post-rolandic	40	81

RH: right hemisphere. LH: left hemisphere.

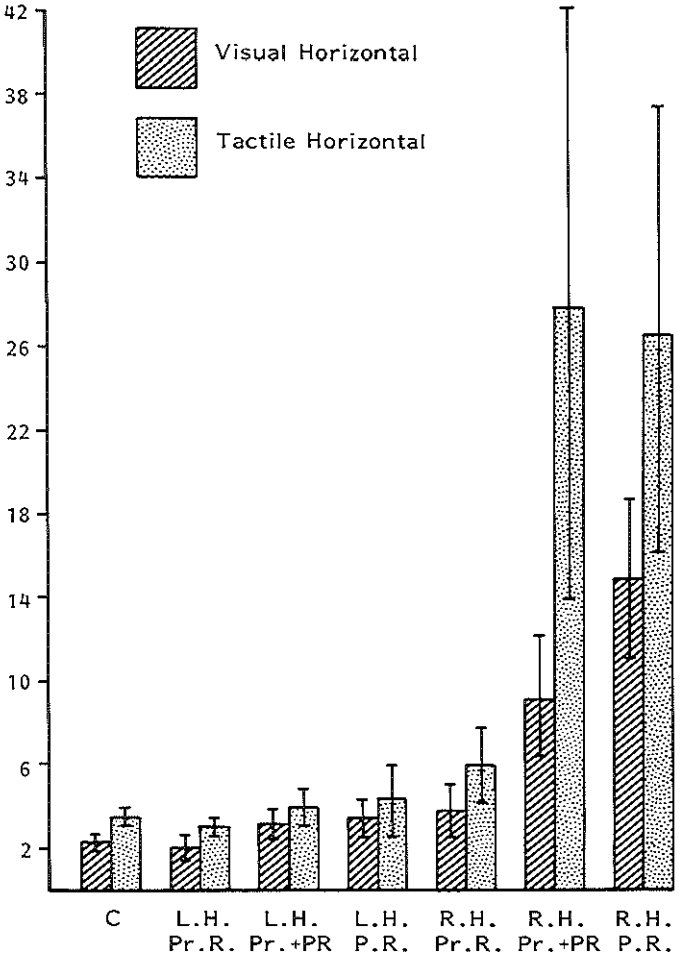

Figure 2a. Mean error rates plus or minus twice the standard error of the mean.
L.H. : left hemisphere
R.H. : right hemisphere
Pr.R. : pre-rolandic
P.R. : post-rolandic
Pr. + PR: pre- and post-rolandic extension

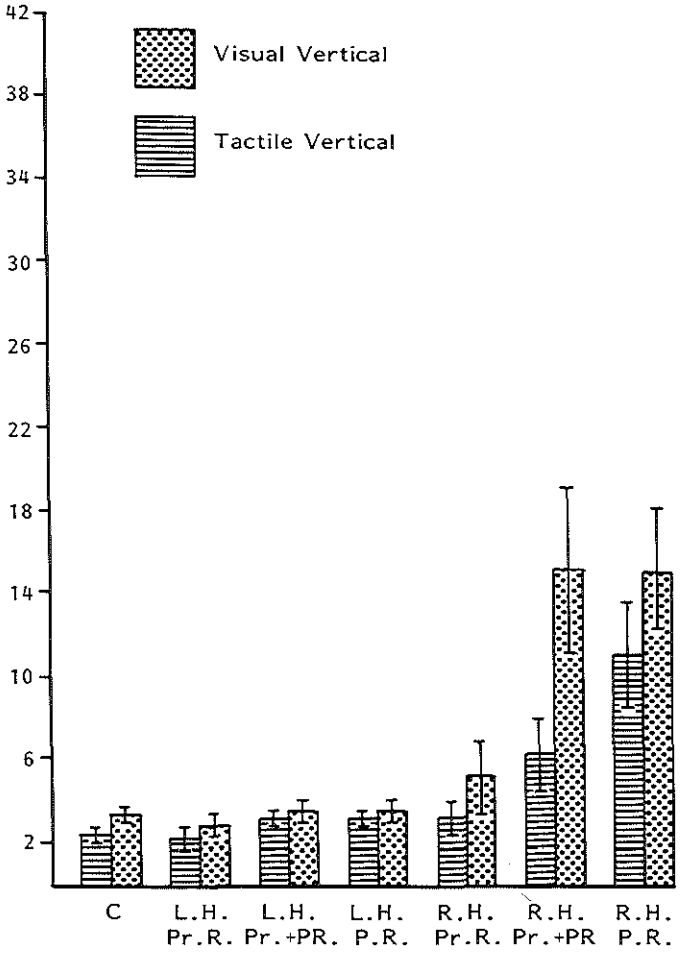

Figure 2b. Mean error rates plus or minus twice the standard error of the mean.
L.H. : left hemisphere
R.H. : right hemisphere
Pr.R. : pre-rolandic
P.R. : post-rolandic
Pr. + PR: pre- and post-rolandic extension

Table 5. Mean duration of the test in the control group both for the left and the right hand.

left		right	
visual	tactile	visual	tactile
23	84	25	65
10	66	23	56
18	73	21	70
20	74	22	79
26	33	24	67
34	56	32	73
36	123	20	72
40	58	20	53
25	86	26	97
14	98	48	84
48	77	41	52
14	36	19	70
36	91	35	104
20	37	17	52
22	88	28	49
23	23	28	70
26	74	14	69
28	63	16	60
17	39	17	84
22	102	14	73
25.1 sec.	69 sec.	24.5 sec.	69.9 sec.

Table 6. Comparison of the brain-damaged subgroups with the control group, and with each other.

Control group	- six brain-damaged	visual	horizontal	$p<0.001$
	subgroups together	visual	vertical	$p<0.001$
$N = 40$	$N = 68$	tactile	horizontal	$p<0.001$
		tactile	vertical	$p<0.001$
The six brain-damaged subgroups		visual	horizontal	$p<0.001$
compared with each other		visual	vertical	$p<0.001$
		tactile	horizontal	$p<0.001$
		tactile	vertical	$p<0.001$

In comparing the performance of the control group and each brain-damaged subgroup separately, I found a highly significant difference between the control group and the right-hemisphere subgroups with a pre- and post-rolandic extension of the lesion, or an exclusively post-rolandic lesion.
The patients with a left-hemisphere lesion or an anteriorly located lesion of the right hemisphere performed worse than the control subjects, but only rarely was there a significant difference, in one of the four mean error rates at the utmost.

Analysis of the second hypothesis showed that there was also a significant difference between the performances of the six brain-damaged subgroups (table 6). When the subgroups of the left hemisphere were compared with those of the right, a significant difference was found between patients with right post-rolandic or right pre- and post-rolandic lesions and any of the left-hemisphere subgroups, for both parts of the test. Intra-hemispheric comparison showed no significant difference for the left hemisphere. For the right hemisphere, however, there was a significant difference for both parts of the test between patients with an exclusively pre-rolandic lesion on the one hand and patients with a pre- and post-rolandic extension of the lesion or an exclusively post-rolandic lesion on the other (table 7).

Table 7. Comparison of brain-damaged subgroups with one another.

				mean error rates		
RH group pre-rolandic N = 9	- RH group pre- and post-rolandic N = 13	visual	horizontal	3.78° - 9.24°	p	≪0.01
		visual	vertical	3.15° - 6.14°	p	<0.05
		tactile	horizontal	5.91° - 27.8 °	p	<0.05
		tactile	vertical	5.14° - 15.3 °	p	<0.001
RH group pre-rolandic N = 9	- RH group post-rolandic N = 13	visual	horizontal	3.78° - 15.09°	p	<0.001
		visual	vertical	3.15° - 10.91°	p	<0.001
		tactile	horizontal	5.91° - 26.89°	p	<0.001
		tactile	vertical	5.14° - 14.89°	p	<0.001
RH group pre- and post-rolandic N = 13	- RH group post-rolandic N = 13	visual	horizontal	9.24° - 15.09°	p	<0.05
		visual	vertical	6.14° - 10.91°	p	≪0.01
		tactile	horizontal	27.8 ° - 26.89°	NS	(p>0.91)
		tactile	vertical	15.3 ° - 14.89°	NS	(p>0.96)

RH: right hemisphere

On comparison of the mean error rates of patients with a pre- and post-rolandic extension of the lesion and those with an exclusively post-rolandic lesion, a statistically significant difference was found only for the visual part of the test (for the p values see table 7). The patients with an exclusively post-rolandic lesion did significantly worse. Accordingly, in the patient group with a pre- and post-rolandic extension of the lesion, the tactile part of the test was performed with significantly larger errors than the visual part.

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.
It turned out that all these lesions had one area in common (figure 3).

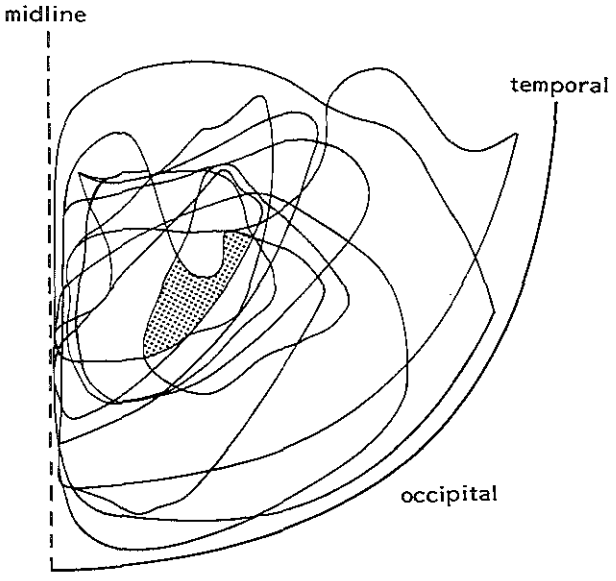

Figure 3. Drawing of a CT-scan section at the level of the cella media. All the infarctions drawn in this figure have the shaded area in common.

In terms of cerebral anatomy (Gado et al. 1979) this area 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
b. on the slice below the previous one: the white matter between the lateral occipital gyrus, middle temporal gyrus and the parieto-occipital sulcus.

DISCUSSION

Patients with an exclusively post-rolandic lesion of the right hemisphere had the highest mean error rates in both the visual and the tactile part of the rod orientation 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 low 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 pre- and post-rolandic extension of the lesion. In this group the mean error rates for the tactile version of the test were almost as high as in the group with an exclusively post-rolandic lesion. In the visual version, however, the patients with a pre- and post-rolandic extension of the lesion performed significantly better than the patients with an exclusively post-rolandic lesion ($p<0.05$), but significantly worse than the patients with an exclusively pre-rolandic lesion ($p<0.001$). Independence of test modality was found only in patients with an exclusively post-rolandic 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 were often desperate during the test and did not know what to do with the rods. True, 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 (sometimes there was even a significant difference), but the differences were much smaller than for patients with a pre- and post-rolandic extension of the lesion or an exclusively post-rolandic lesion of the right hemisphere (figure 2). This mild deterioration of performance in patients with a left-hemisphere lesion, or an exclusively pre-rolandic lesion of the right hemisphere is perhaps a non-specific effect of disease (two weeks after the stroke).

Thus the finding of De Renzi et al. (1971) that the right posterior region of the brain plays a very important role in spatial perception has not only been confirmed, but it was possible to make a further functional subdivision within the right hemisphere. Benton et al. (1978) found a positive association between the presence of a right posterior lesion and an impaired performance in another test that involved judgment of line orientation in one plane, but the test was presented only by visual means.

Computerized tomography made it possible 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.

CONCLUSIONS

1. By testing spatial perception at an elementary level I found an almost complete dominance of the posterior region of the minor hemisphere.
2. Patients who had an exclusively post-rolandic lesion of the right hemisphere, performed poorly in both the tactile and the visual version of the test. In patients who had a pre- and post-rolandic extension of the lesion, the performance on palpation was significantly worse than on inspection.
3. That these subdivisions were found in contrast with earlier investigations is probably the result of a more precise localization and of the exclusion of remote effects by computerized tomography.
4. The cerebral infarctions of patients with an exclusively post-rolandic lesion of the right hemisphere (who all had a visual field defect) had an area in common in the white matter of the occipital lobe.

PART 2
RELATION BETWEEN THE SIZE OF THE LESION AND THE TEST PERFORMANCE

The introduction of computer-assisted tomography (CT scan) has stimulated new interests in the correlation between the behavioral disorders from damage of the brain, and the site and extent of the underlying cerebral lesion (Mazzocchi et al. 1978; Naeser and Hayward 1978). Boller et al. (1970) calculated the volume of the lesions from the brain-scan image and correlated this with a functional deficit in simple reaction time.

I devised a simple procedure for assessing the size of a lesion and used it to investigate a possible correlation between the size of the lesion and (1) the test performance in patients with spatial disorientation from a cerebral infarct, (2) the extent of recovery on a spatial task after one year, and (3) the speed of recovery.

METHODS

Material

The items needed are:

a. Photographs of the CT-scan sections upon which the lesion is visible.
b. A planimeter (the Ott polar planimeter). With this the surface of the lesion on a CT-scan section can be measured.

Procedure

With the planimeter the surfaces of the lesion were measured on all the sections of the CT scan upon which it was visible. The planimeter gives the surface in nonius units. In order to get the actual size of the surface of the lesion in square centimetres, the measurement in nonius units had to be multiplied by a certain figure. The reduction factor of the photograph is included in the calculation of this figure.

I assumed that the shape of an infarct approximates that of a double cone with its axis at right angles to the plane of scanning. By this assumption the volume could be calculated by taking the product of the distance between the sections and the sum of the surfaces of all sections. This is easy to see from an example. If the lesion is visible on three CT-scan sections and the surfaces of the lesion on these sections are A, B and C respectively, and the distance between two sections is h (figure 4), then the volume can be calculated with the following formula

$$V = \tfrac{1}{2}Ah + \tfrac{1}{2}(A+B)h + \tfrac{1}{2}(B+C)h + \tfrac{1}{2}Ch = h(A+B+C).$$

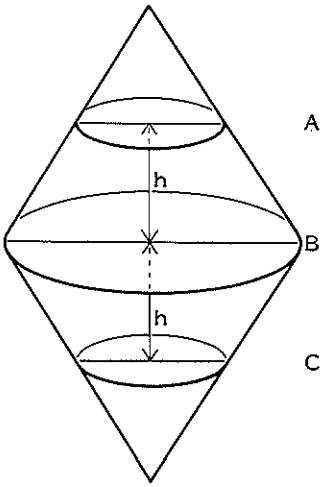

Figure 4.

In fresh infarcts the margins can be blurred. So the margins were chosen to include only marked hypodensity in order to reduce this inaccuracy. Each surface was measured three times and the average was calculated. (The difference between the highest and the lowest sum of the volumes was 2.6 per cent.)

DISCUSSION

The described procedure is simple, and after some training it takes little time to assess the size of the lesion.

A possible limitation in the assessment of the volumes of the lesion by means of the CT scan became evident after the introduction of the positron emission computed tomography (PECT). By this method the local cerebral metabolic activity is measured, and there is a direct correlation between the metabolic rate for glucose and brain function (Kuhl et al. 1979).

Metter et al. (1981) studied five stable aphasic patients by PECT using ^{18}F-2-fluoro-2-deoxy-D-glucose (FDG) and CT to investigate the relationship between the clinical syndrome, CT and the local cerebral metabolic rate for glucose (LCMRglc). They found that the metabolic abnormalities demonstrated by PECT were more extensive than the CT evidence of structural damage in all five patients. The finding of suppression of metabolism in non-infarcted brain is consistent with the observations by Kuhl et al. (1979), who found extensive cortical striatal and thalamic hypometabolism in stroke patients by FDG PECT in the absence of any CT abnormality.

At present there are several practical restrictions in the use and interpretation of FDG PECT. The most important in respect to measuring the volume of the lesion is that the resolution of the positron tomograph is less than that obtained with CT, making delineation less precise. At any rate, the size of infarcts on CT can be used as a relative measure, as in this investigation.

INFLUENCE OF LESION SIZE ON NEUROPSYCHOLOGICAL DISORDERS

The measurement of the size of the infarct was used to investigate a possible correlation with the test performance on the rod orientation test (two weeks after the stroke) in patients with a disturbance of spatial perception from a cerebral infarct. Figure 5 shows the results.

The location of infarcts producing aphasia and the influence of the size of the lesion on the severity of aphasia were studied on radionuclide scans by Kertesz et al. (1977). The size of the lesion was calculated by measuring the area on the lateral view with a planimeter. They found a positive correlation between the size of the lesion and the severity of the aphasia (tested with the Western Aphasia Battery). In 1979 Kertesz et al. investigated the correlation between the type of aphasia in patients with a left-hemisphere stroke and the site and extent of the lesion as seen on CT (patients with both acute and chronic aphasias were included). The volume of the lesions was determined by a digitizer programme on the computer. Again they found a positive correlation between the lesion size and the severity of the aphasia, but this

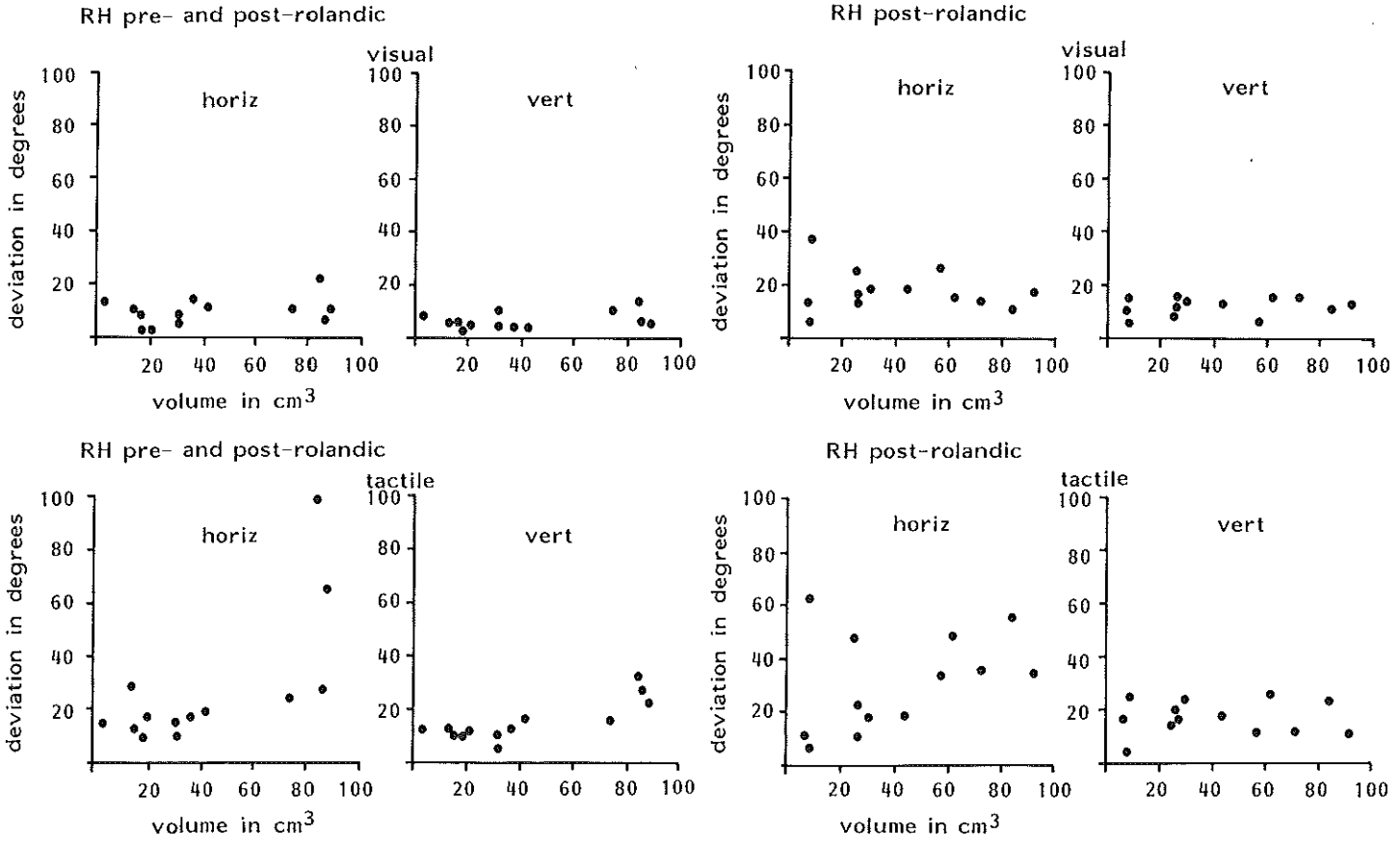

Figure 5. Relation between lesion size and test performance on the rod orientation test.

only reached statistical significance in the chronic group. The relation between clinical deficits from infarcts of the right hemisphere and the extent of the lesion were studied by Kertesz and Dobrowolski (1981). Non-language parameters tested were praxis, block design drawing and Raven's coloured progressive matrices. They found that lesion size did not correlate significantly with any of the parameters.

I did not find a relation between the volume of the lesion and the test performance on the rod orientation test two weeks after the stroke, as shown in figure 5.

CHAPTER III

RECOVERY OF PERFORMANCE ON THE ROD ORIENTATION TEST IN PATIENTS WITH INFARCTION OF THE RIGHT HEMISPHERE

PART I
PREVIOUS STUDIES ON RECOVERY OF BRAIN FUNCTION

The mechanisms underlying recovery of neuropsychological functions are incompletely understood. Some of the functional recovery occurring in the first three weeks after an infarct could be ascribed to the reversal of the early abnormalities. Blood flow may increase to areas not irreversibly damaged (Kohlmeyer 1976), oedema may subside, and pressure associated with haemorrhage or dynamic changes in the flow of cerebrospinal fluid may return to normal. However, functional recovery often continues after that. The rate of recovery is thought to be maximal for two to three months after an infarct and then to slow down (Basso et al. 1975, Kertesz and McCabe 1977). It is generally believed that beyond one year recovery is less likely to occur (Culton 1969, Kertesz and McCabe 1977).

Many theories have been advanced to explain the second phase of recovery. It can be argued that since destruction of a particular area of the brain does not result in permanent loss of function, a particular function cannot be "localized" to the destroyed portion of the brain. One of the earliest opponents of phrenological localization was Flourens (1824), who demonstrated recovery after ablative experiments in pigeons. Lashley (1938) based his well-known theory of *equipotentiality* on similar extensive ablations in rats. Munk (1881) proposed that regions of the brain which were not otherwise occupied could assume functions mediated previously by an injured area (the theory of *substitution*). According to him the area substituting for the damaged one would otherwise never have become involved in mediating the function in question.

According to Jackson (1873) the nervous system is organized hierarchically with higher centres controlling lower ones. Damage at a higher level causes the release of lower centres from inhibition and may lead to compensation.

Von Monakow (1914) postulated that damage to the brain deprives other intact regions of normal stimulation. The sudden loss of input to the otherwise normal areas produces in them a particular type of shock, which he called *diaschisis*. Eventually, according to Von Monakow, the undamaged portions of the brain resume normal functioning and only those symptoms remain for which loss of the damaged area was directly responsible.
Age was emphasized as an important factor by Lashley (1938), who concluded that young animals show better recovery from brain damage than adult ones. Kennard and McCulloch (1943) demonstrated that unilateral precentral lesions in immature animals have minimal effects when compared with similar lesions in adult ones. From the critical review by Johnson and Almli (1978), however, it is clear that the age effect in relation to brain damage is still an unresolved issue.
The influence of age on the recovery of aphasia is controversial. According to Basser (1962) and Hécaen (1976) the recovery from aphasia acquired before the age of ten to twelve is excellent. Others, however, have failed to show any correlation between age and recovery (Culton 1971, Smith et al. 1972). Finally, the type of aphasia and the underlying disease will influence the extent of recovery (Kertesz et al. 1979, Van Dongen and Loonen 1977).

At the cellular level recovery could be due to changes of the properties of the structural elements or to outgrowth of abnormal interneural connections. Von Monakow observed a decrease in sensitivity of neurons receiving fibres from a damaged area (diaschisis), which decrease was supposed to disappear over a period of time. Due to diaschisis secondary changes would lead to a more extensive loss of function than caused by the actual lesion. After diaschisis disappears, the loss of function is restricted to the primary lesion. Several processes could explain diaschisis, such as development of temporary brain oedema, or loss of the trophic influence of the damaged neurons. This loss of trophic influence from destroyed neurons could lead to a depression in the metabolism of postsynaptic cells and in this way to a decreased ability of these cells to interact with other neurons (Smith and Kreutzberg 1976). According to Van Hof (1981) this hypothesis explains the onset of diaschisis but not its disappearance. This is often explained by the occurrence of denervation supersensitivity, which would make the remaining connections more effective. However, Kuffler et al. (1971) studied the parasympathetic ganglion cells in the heart of the frog; they found that after denervation iontophoretic microapplication of acetylcholine enlarges the area sensitive to the transmitter but not the sensitivity at the location of the original nerve endings. This argues against denervation supersensitivity being the main factor in the disappearance of diaschisis.

Liu and Chambers (1958) were among the first to show that regeneration and sprouting of axons, which occurs after a complete or partial lesion of a

peripheral motor nerve, may take place in the central nervous system. However, there is no experimental support for the view that sprouting leads to any functional improvement. On the contrary, it may lead to an even greater deficit (Schneider and Jhaveri 1974).

In contrast to the extensive literature on recovery and treatment of aphasia, very little information is available about recovery of non-verbal function. Campbell and Oxbury (1976) examined the performances of right-hemisphere-damaged patients three to four weeks and six months after a stroke on verbal and non-verbal tasks. Those who demonstrated neglect at the initial examination had remained impaired on visuospatial tasks six months later in spite of the resolution of neglect. Lawson (1962) emphasized that the presence of left unilateral neglect retarded recovery.

PART 2
EXTENT OF RECOVERY

In Chapter II disturbances of spatial perception were found in a group of patients with recent infarction (two weeks before) in the posterior region of the right hemisphere. Patients with an exclusively post-rolandic lesion performed poorly in the visual as well as in the tactile part of the test. In patients with a pre- and post-rolandic extension of the lesion there was a clear difference between the two modalities tested, the performance in the tactile version of the test being worse.

The same patients were re-examined one year after the stroke, as it is known from other neurological deficits that spontaneous recovery rarely continues after one year.

The following questions were the starting-point for this part of the study:

1. Is there recovery of spatial functions?
2. If recovery of spatial functions occurs, is there still a difference between the patients who were originally affected and other brain-damaged subgroups (with right frontal or left-sided lesions)?
3. Do patients with no or only a slight recovery have some characteristic findings in common on clinical examination?
4. Is there still a difference between patients with a pre- and post-rolandic extension of the lesion (performance much worse on palpation than on inspection) and patients with an exclusively post-rolandic lesion of the right hemisphere (equal impairment in both parts of the test)?
5. Does the volume of the lesion influence the extent of recovery?

METHODS

Subjects

The patients examined in the present study were also described in Chapter II. All had a cerebral infarct without a midline shift as assessed by CT scanning. After one year only patients with a pre- and post-rolandic lesion and those with an exclusively post-rolandic lesion of the right hemisphere were re-examined. Both groups originally consisted of thirteen patients, but in each group one patient refused to participate again in this study. Tables 1 and 2, Chapter II, give more detailed information about the site of the lesion and about the clinical signs.

Testing procedure

The method used was the rod orientation test described on page 24.

RESULTS

There were four mean error rates for both the visual and the tactile version of the test: horizontal error, vertical error, model left or right. There were no significant differences in error rates according to the side of presentation of the model, neither for the control group, nor for the brain-damaged subgroups, as stated before. In view of this, further statistical analysis was done on the mean of all individual error rates.

For the statistical analysis an analysis of variance was used. Table 8 illustrates that in the year after the stroke a significant change in performance had occurred 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 pre- and post-rolandic extension of the 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) (table 9).

These were the only patients who showed a tactile extinction phenomenon and who had the highest mean error rates on the initial examination two weeks after the stroke.

Of the twelve patients with an exclusively post-rolandic lesion one still performed poorly in both parts of the test (table 10).

On clinical re-examination this woman had not only a left homonymous hemianopia, but also an impaired memory for the localization of objects, as well as defective route finding.

Table 8. Comparison of the mean error rates of the performance two weeks and one year after the stroke.

			mean error rates	
RH exclusively post-rolandic	visual	horizontal	15.87° - 4.25°	$p<0.01$
	visual	vertical	9.84° - 3.66°	$p<0.01$
2 weeks - 1 year	tactile	horizontal	21.86° - 4.71°	$p<0.01$
N = 13 N = 12	tactile	vertical	11.50° - 4.68°	$p<0.01$
RH pre- and post-rolandic	visual	horizontal	13.32° - 3.63°	$p<0.01$
	visual	vertical	6.96° - 3.53°	$p<0.01$
2 weeks - 1 year	tactile	horizontal	30.71° - 5.75°	$p<0.01$
N = 13 N = 12	tactile	vertical	16.63° - 5.76°	$p<0.01$

RH: right hemisphere

Table 9. Mean error rates of four patients with a partial recovery one year after the stroke.

			mean error rates
RH group with a	visual	horizontal	4.17°
pre- and post-rolandic	visual	vertical	4 °
extension of the lesion	tactile	horizontal	11.8 °
and a partial recovery	tactile	vertical	9.7 °

RH: right hemisphere

Table 10. Mean error rates of one patient with a partial recovery one year after the stroke.

			mean error rates
RH group with an	visual	horizontal	10.2°
exclusively post-	visual	vertical	7.0°
rolandic lesion and	tactile	horizontal	16.8°
a partial recovery	tactile	vertical	8.2°

RH: right hemisphere

Calculation of the volumes from the CT scans showed that these five patients with a partial recovery had the largest lesions, ranging from 74 to 92 cm^3 (mean 85 cm^3) (figure 6). The volumes of the lesions of the other patients with a pre- and post-rolandic extension of the lesion or an exclusively post-rolandic lesion of the right hemisphere were considerably smaller and ranged from 3 to 72 cm^3 (mean 29 cm^3). There was no relation between the volumes of the lesion and the mean error rates at the initial examination two weeks after the stroke.

No other patient had any symptoms after one year which could be attributed to disturbances of spatial perception.

Comparison of the test results of the re-examined patients with those of patients with an infarct at other sites than the posterior right hemisphere, showed no statistically significant difference. But there was still a significant difference between the mean error rates of the control subjects and the re-examined patients (table 11).

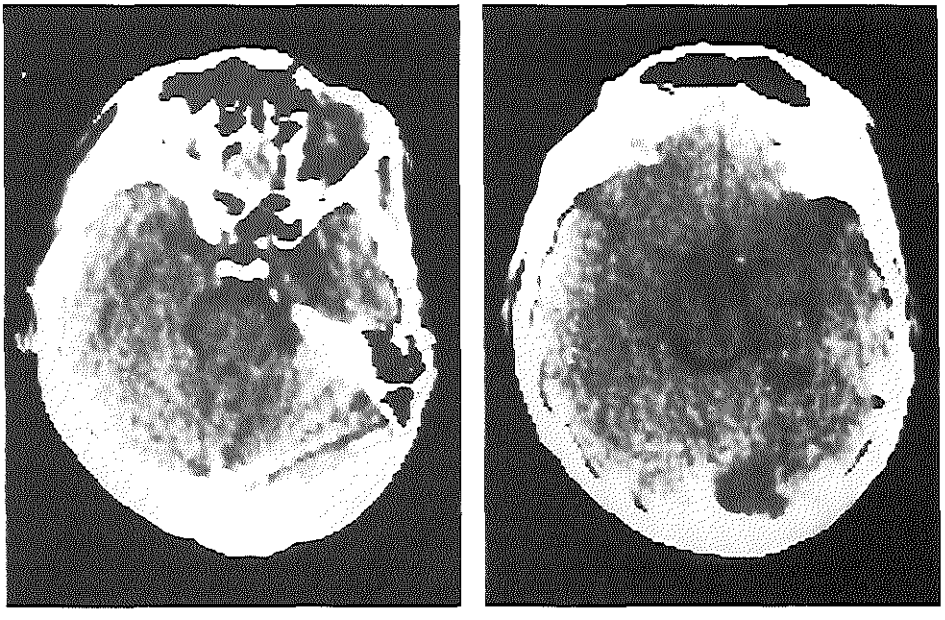

Figure 6. CT scan of a patient with a large lesion.

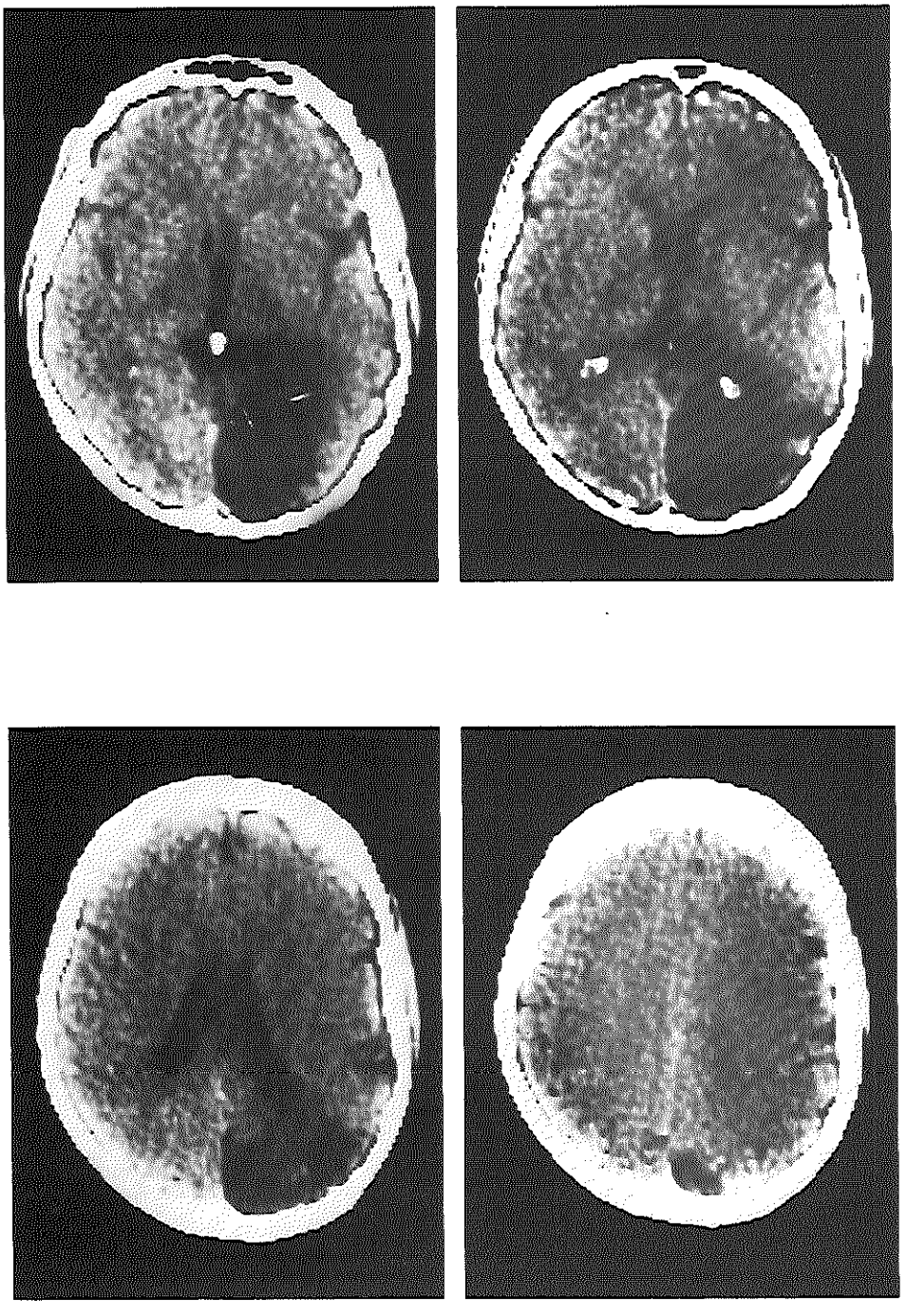

Figure 6. CT scan of a patient with a large lesion.

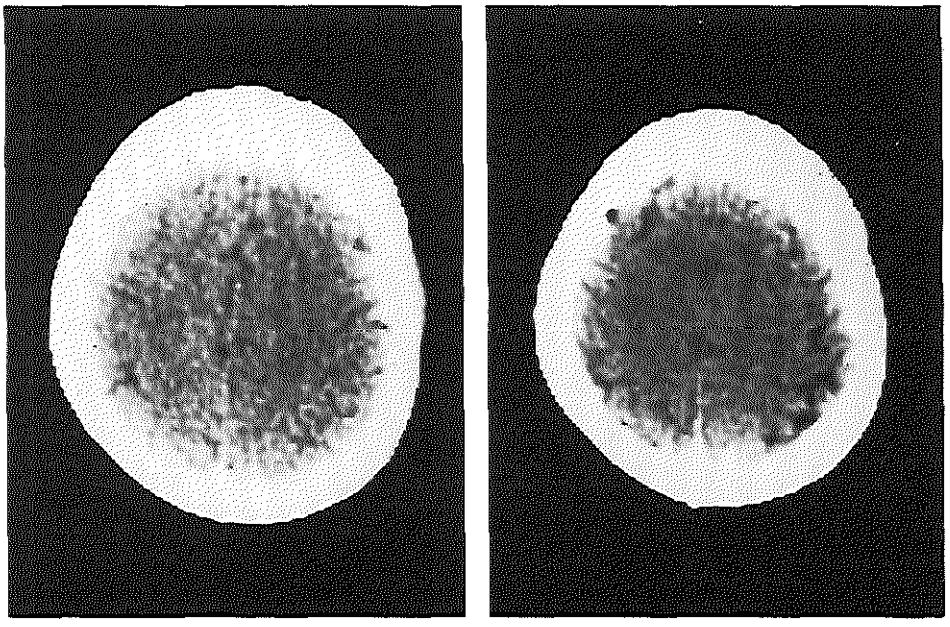

Figure 6. CT scan of a patient with a large lesion.

Table 11. Mean error rates of the control group and the re-examined groups.

				mean error rates	
Control group -	RH group	visual	horizontal	2.24° - 3.63°	$p<0.001$
	pre- and post-	visual	vertical	2.22° - 3.53°	$p<0.001$
	rolandic	tactile	horizontal	3.39° - 5.75°	$p<0.001$
	(after one	tactile	vertical	3.40° - 5.76°	$p<0.001$
	year)				
N = 40	N = 12				
Control group -	RH group	visual	horizontal	2.24° - 4.25°	$p<0.001$
	post-rolandic	visual	vertical	2.22° - 3.66°	$p<0.001$
	(after one				
	year)	tactile	horizontal	3.39° - 4.71°	$p<0.05$
N = 40	N = 12	tactile	vertical	3.40° - 4.68°	$p<0.05$

RH: right hemisphere

DISCUSSION

The results show that a distinct recovery from disturbances of spatial

perception occurred after one year in the majority of patients with an infarct of the posterior region of the right hemisphere.
In contrast, Campbell and Oxbury (1976) did not find a significant improvement on re-examination six months after the stroke. There are several possible explanations for the difference with the results of this study:

1. Patient selection (infarct in the posterior region of the right hemisphere in this study, presence of visuospatial neglect in theirs).
2. Complexity of the test. Only when spatial perception is tested at an elementary level, an almost complete dominance of the right posterior region is found. But when a task requires the elaborate integration of spatial data, the left hemisphere contributes as well (De Renzi et al. 1971).
3. The time when the patients were initially tested (two weeks after the stroke in this study, three to four weeks in theirs). A slight restoration of function could have occurred 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 certainly deserves further investigation.

CONCLUSIONS

1. Recovery of disturbances of spatial perception had occurred one year after the stroke in this group of patients, although incompletely. There was still a significant difference between the mean error rates of the control subjects and the re-examined patients.
2. Of the twelve patients with a pre- and post-rolandic extension of the lesion, four still showed a difference in performance between the tactile and the visual part of the test. These were the only patients who showed a tactile extinction phenomenon and who had the highest mean error rates on the initial clinical examination. Of the twelve patients with an exclusively post-rolandic lesion, one still performed poorly in both parts of the test.
3. These five patients had the largest lesions in their groups, ranging from 74 to 92 cm^3 (mean 85 cm^3).

PART 3
SPEED OF RECOVERY

Noticing that recovery from disturbances of spatial perception did occur in the majority of patients with an infarct in the posterior region of the right

hemisphere one year after the stroke, I tried to determine the time course of this recovery by examining a group of patients at regular intervals. In addition, it seemed worthwhile to compare the serial results of the rod orientation test with those of another test stated to detect disturbances in spatial perception. For this purpose the line orientation test developed by Benton et al. (1978) was selected. Finally, I studied the relationship between the size of the lesion and the speed of recovery.

METHODS

Subjects

Another group of 16 patients with damage of the posterior region of the right hemisphere was examined. They all had a cerebral infarct without a midline shift as assessed by CT scanning. Eight patients had a pre- and post-rolandic extension of the lesion, and eight had an exclusively post-rolandic lesion (table 12).

Table 12. Site of the lesion.

pre- and post-rolandic lesion	fronto-parietal	2
	temporo-parietal	6
exclusively post-rolandic lesion	temporo-parieto-occipital	2
	parieto-occipital	3
	occipital	3

Each group consisted of five males and three females, their ages ranged from 42 to 70 years, mean age 58.2 years. The neurological signs (table 13) of the patients were recorded along with age and sex. All patients were right-handed.

Testing procedure

The methods used were the rod orientation test described on page 24 and the line orientation test. All patients were tested at regular intervals, namely 2 weeks, 6 weeks, 3 months, 6 months and, if necessary, 1 year after the stroke. Figure 7 shows the line orientation test.

Table 13. Neurological signs in 16 patients with a posterior lesion of the right hemisphere, who underwent sequential tests of spatial function.

		right hemisphere
slight weakness	arm leg both	7
moderate weakness	arm leg both	1
severe weakness	arm leg both	4
sensory impairment		12
isolated visual field defect		4

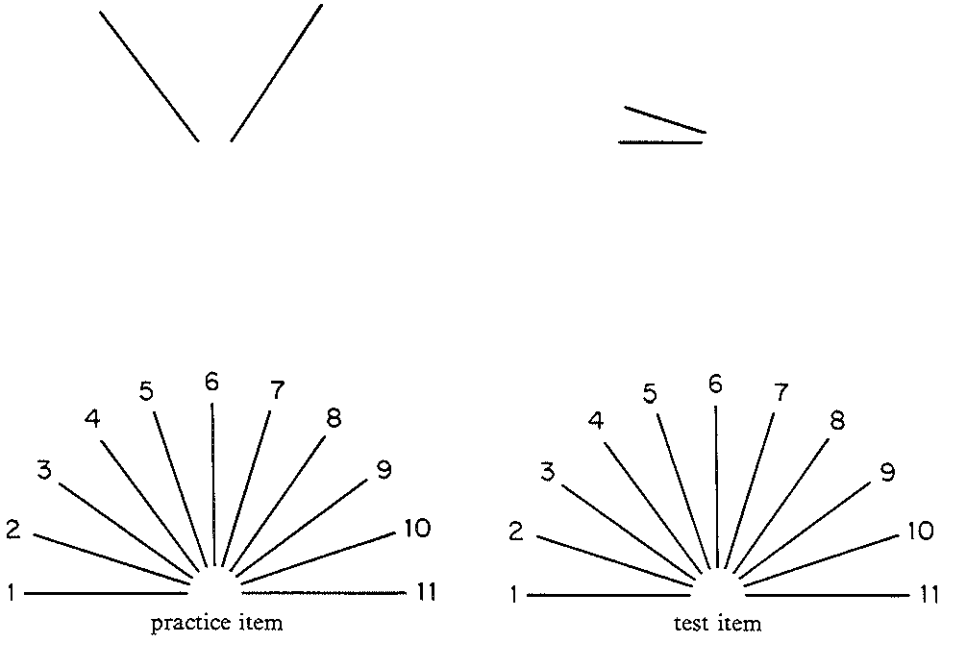

Figure 7. Response-choice display.

The response-choice display of the line orientation test consisted of an array of lines numbered 1 through 11, each separated by an angle of 18°. Each stimulus consisted of two lines that represented either the proximal, middle or distal one half (1.9 cm) of a response-choice line (3.8 cm) (figure 7 right). Five practice items employing the full-length lines preceded the test items (figure 7 left). The subjects were informed whether or not their responses were correct on the practice items but not on the test items. The task was to indicate by pointing or naming the two lines in the response-choice display that had the same angle (pointed in the same direction) and occupied the same location as the two stimulus lines. No time limit was set. The material was assembled in booklet form (30 items) so that, when opened cross-wise the stimulus appeared on the top page and the response-choice display on the bottom page of the booklet. The responses were scored as correct if both stimulus lines were accurately identified.

RESULTS

Figures 8-23 show the course of the scores on the rod orientation test above the abscissa and those on the line orientation test below it. On the abscissa the follow-up period is marked in months. The scores on the line orientation test have been corrected for age and sex (Benton et al. 1978).

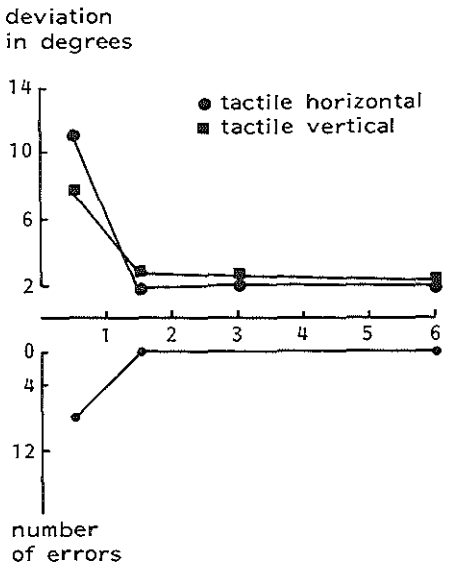

Figure 8.

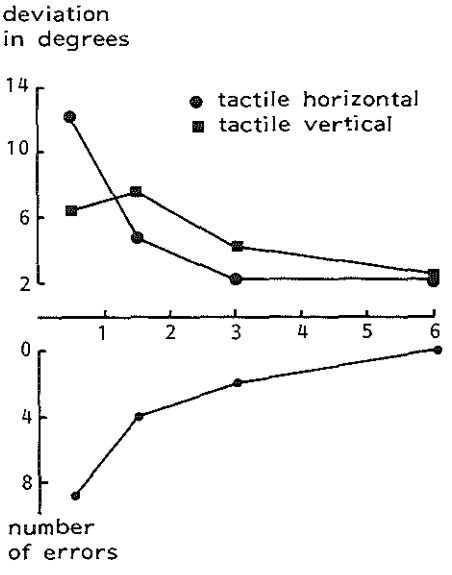

Figure 9.

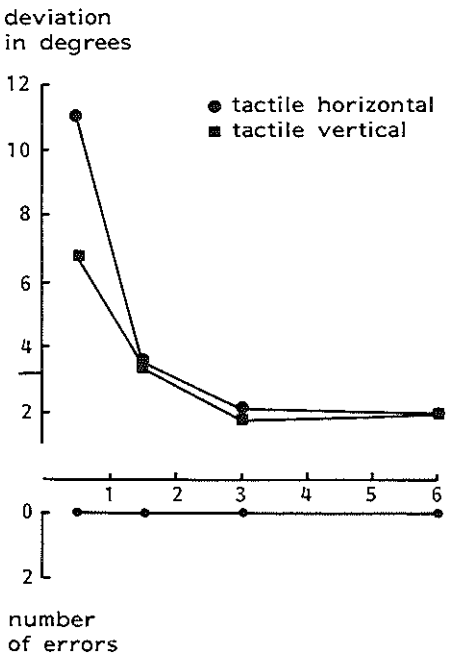

Figure 10.

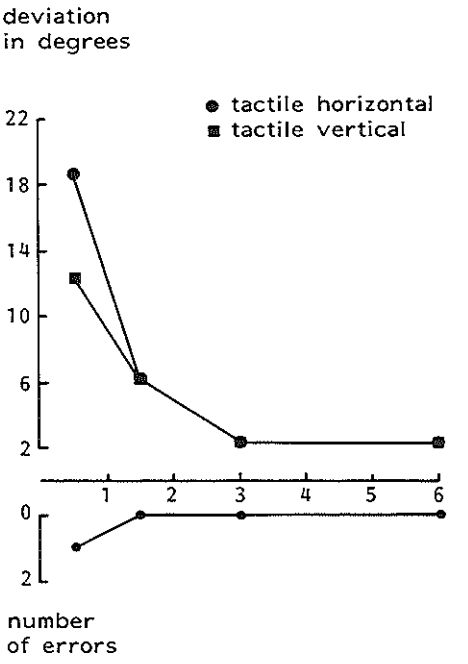

Figure 11.

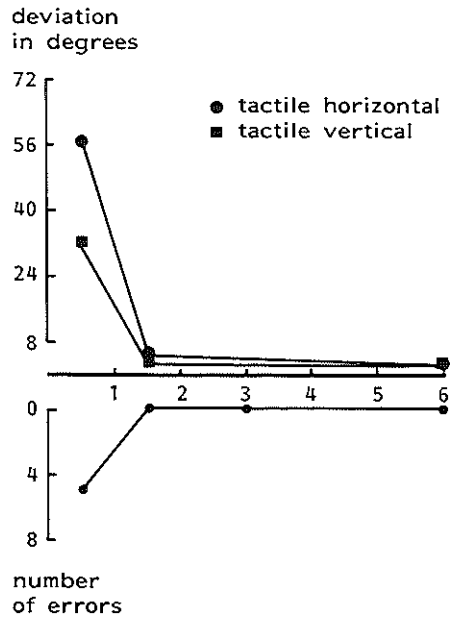

Figure 12.

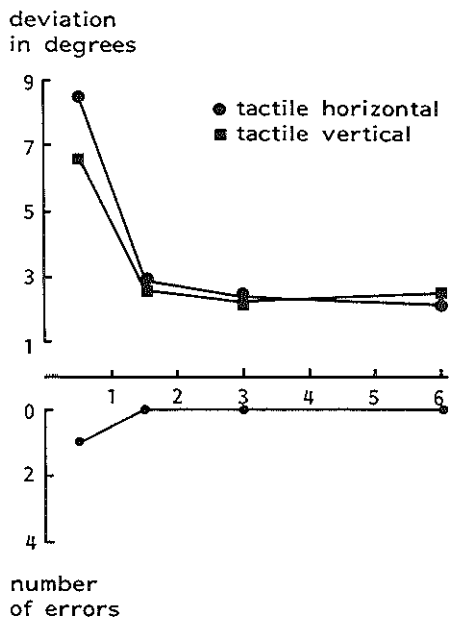

Figure 13.

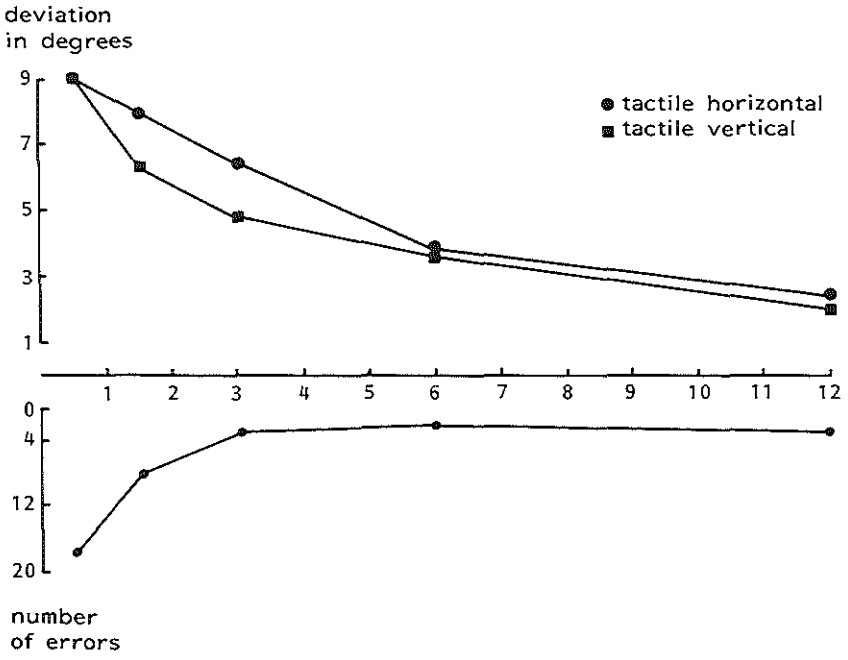

Figure 14.

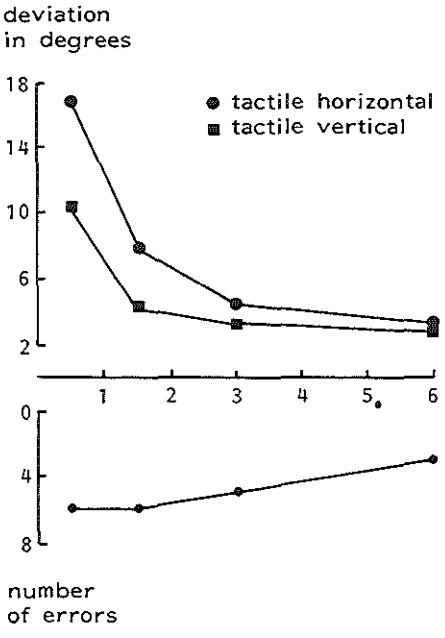

Figure 15.

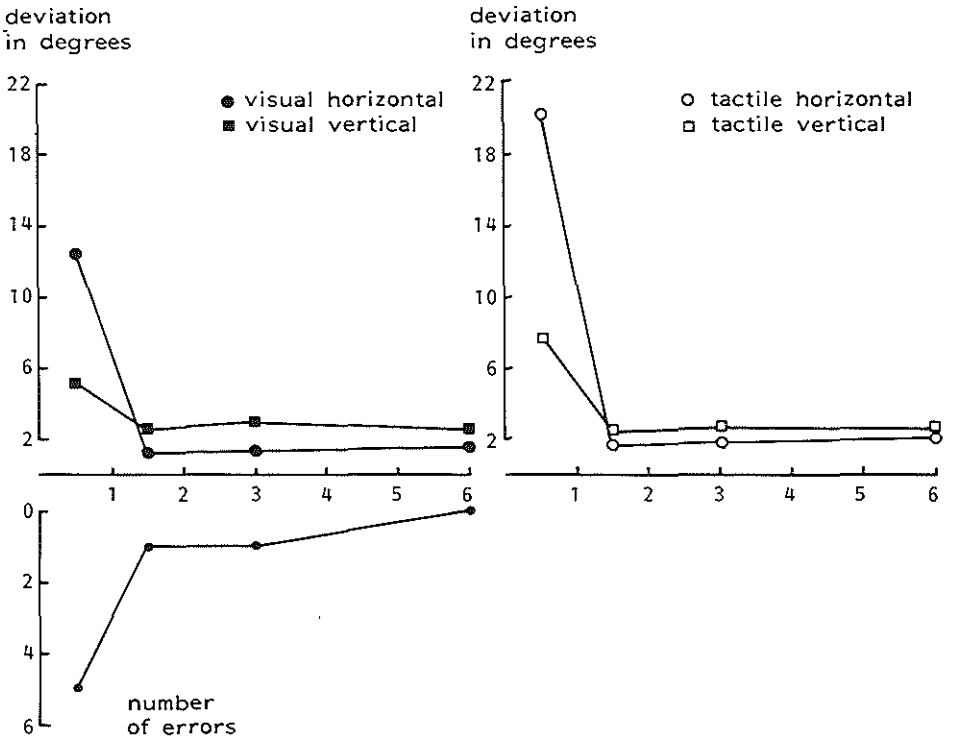

Figure 16.

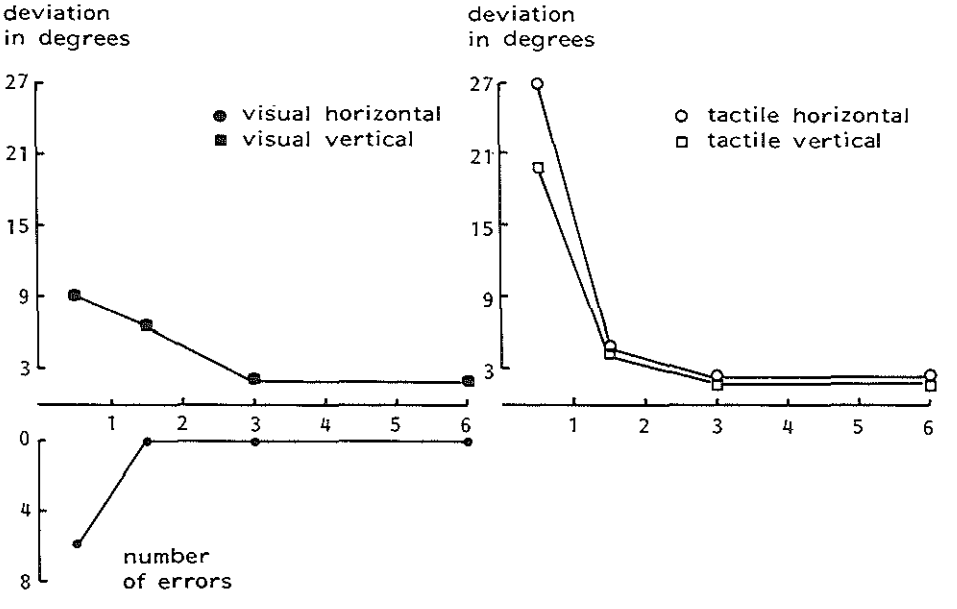

Figure 17.

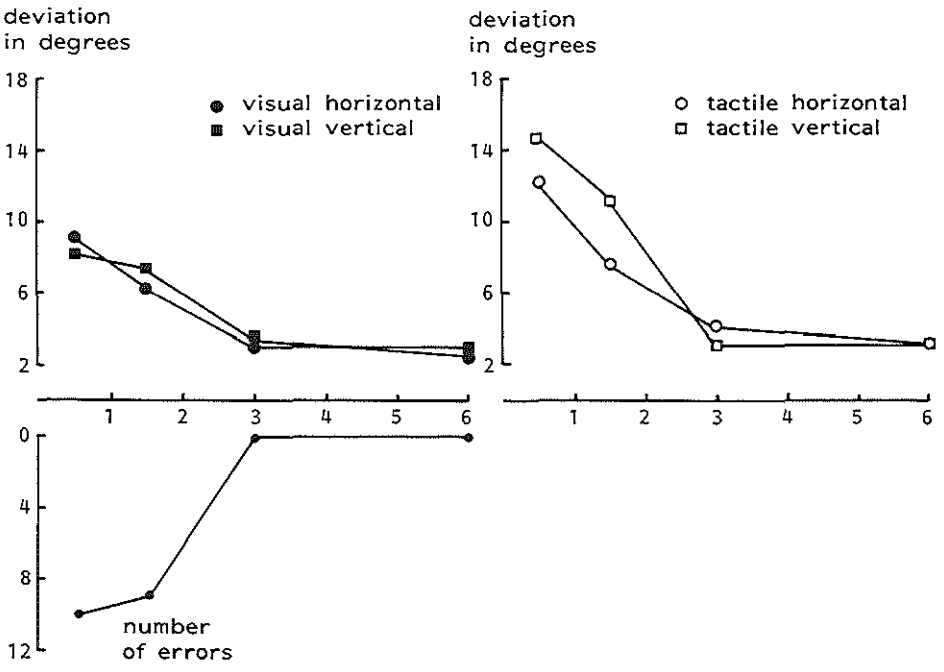

Figure 18.

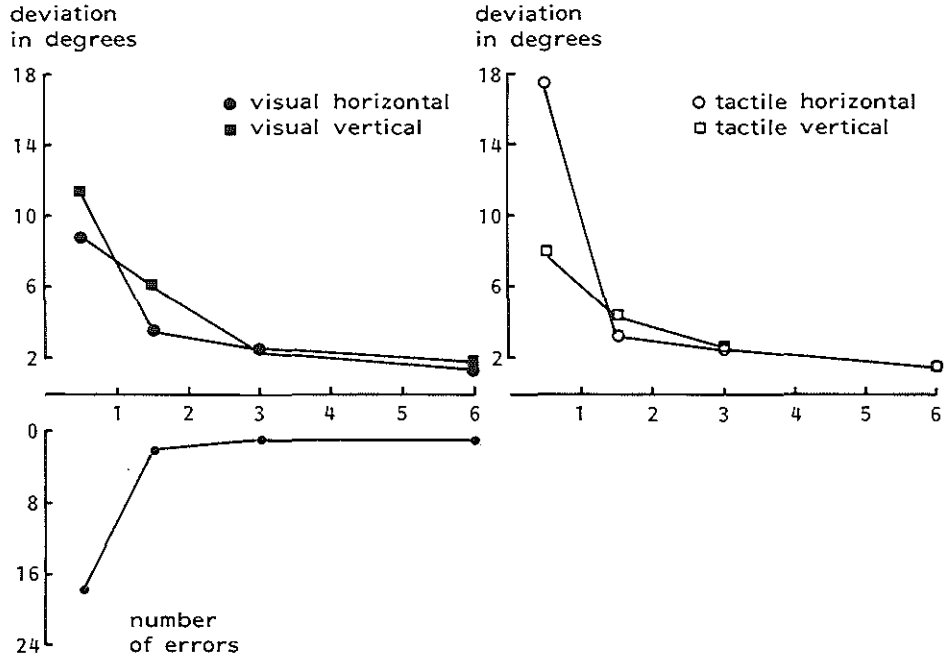

Figure 19.

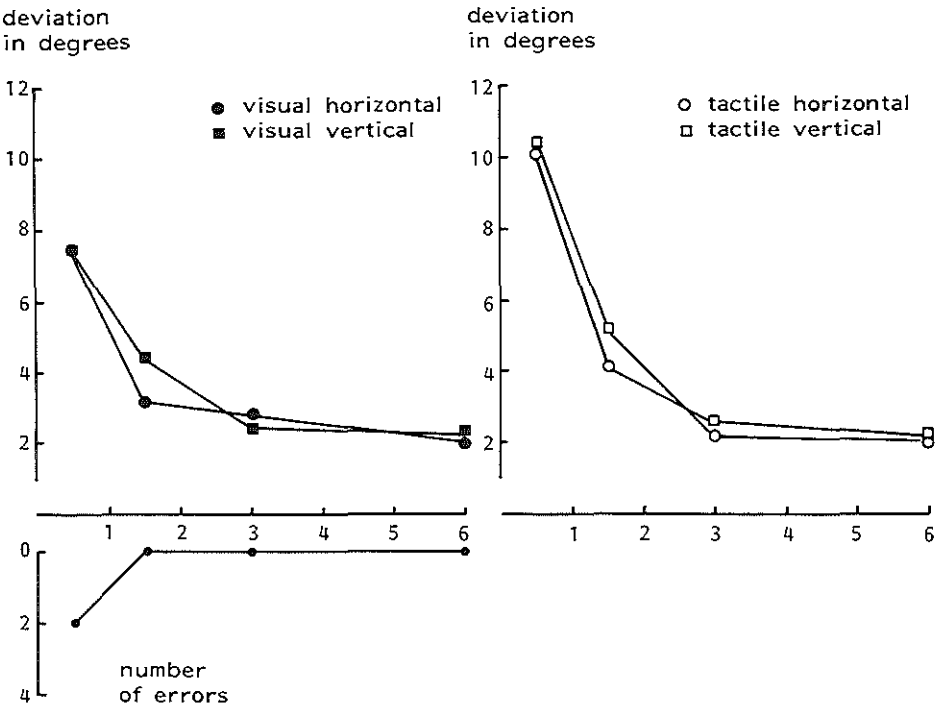

Figure 20.

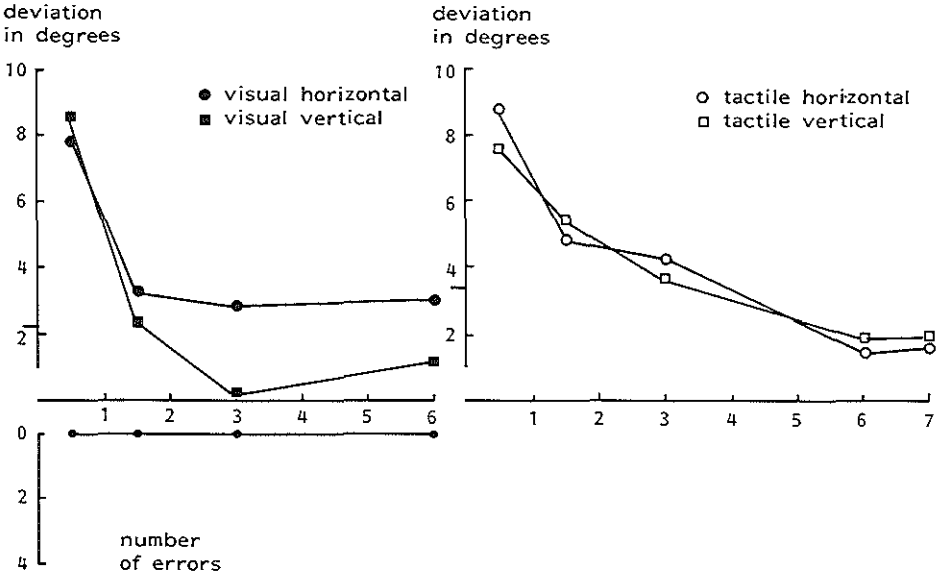

Figure 21.

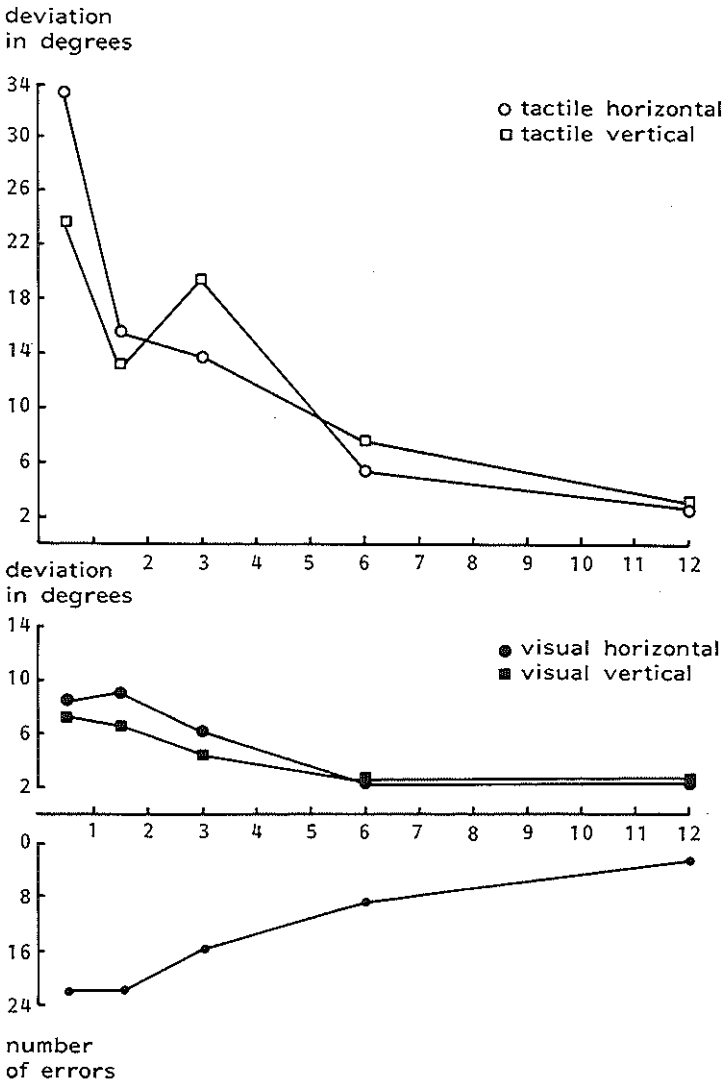
deviation
in degrees
34
30
26
22
18
14
10
6
2
o tactile horizontal
□ tactile vertical
1 2 3 4 5 6 7 8 9 10 11 12
deviation
in degrees
14
10
6
2
● visual horizontal
■ visual vertical
1 2 3 4 5 6 7 8 9 10 11 12
0
8
16
24
number
of errors

Figure 22.

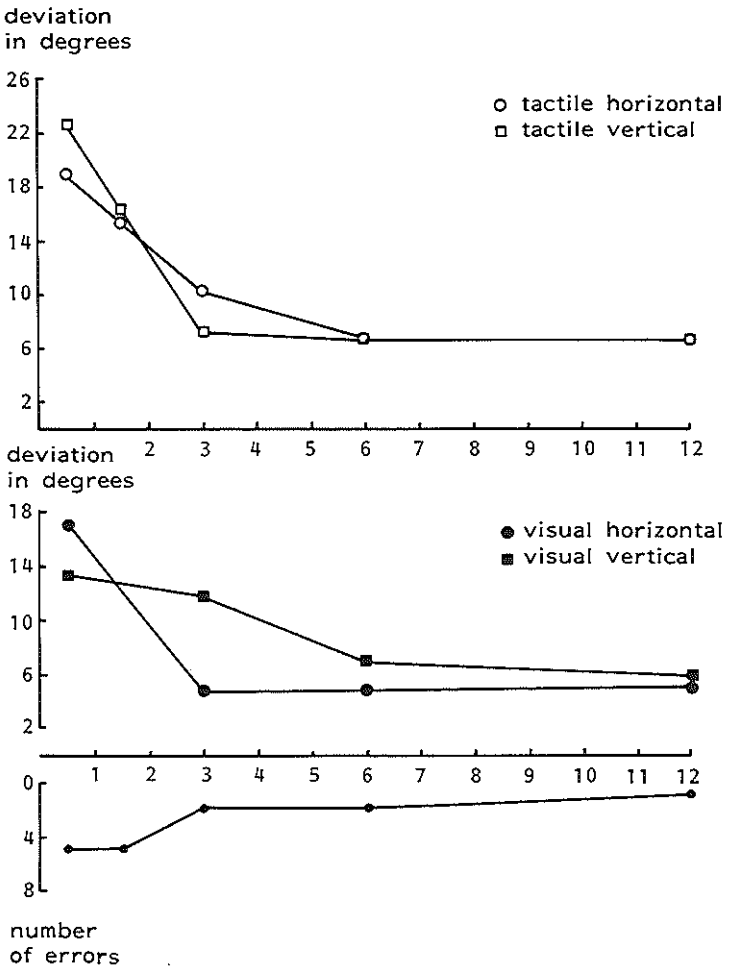

Figure 23.

Table 14 shows the interpretation of the scores on the line orientation test, both for the patient group with a pre- and post-rolandic extension of the lesion and the patient group with an exclusively post-rolandic lesion. There were only three patients with a defective performance on this test.

For the rod orientation test there were four mean error rates for both the visual and the tactile part of the test: horizontal error, vertical error, model left or right. As there were no significant differences in error rates according to the site of presentation of the model in these two brain-damaged subgroups, further statistical analysis was done on the mean of all individual error rates.

Table 14. Interpretation of the scores on the line orientation test.

	corrected score	RH group pre- and post-rolandic	RH group exclusively post-rolandic
superior	29 - 30	3	1
high normal	27 - 28		1
normal	23 - 26	2	3
low normal	21 - 22	2	
borderline	19 - 20		1
mildly defective	17 - 18		
moderately defective	15 - 16		
severely defective	0 - 14	1	2

RH: right hemisphere

For the statistical analysis an analysis of variance was used. Table 15 shows that in the six months after the stroke a significant change in performance had occurred in the two groups as a whole for the visual as well as for the tactile part of the test.

The comparison of the test results of these patients with those of the control group (this group consisted of 40 patients without a history or signs of brain disease; Chapter II) showed no longer a statistically significant difference.

Table 15. Comparison of the mean error rates of the performance two weeks and six months after the stroke.

				mean error rates	
RH exclusively		visual	horizontal	9.97° - 2.70°	$p<0.001$
post-rolandic		visual	vertical	8.85° - 2.75°	$p<0.001$
2 weeks -	6 months	tactile	horizontal	18.5 ° - 2.80°	$p<0.001$
N = 8	N = 8	tactile	vertical	14.3 ° - 2.80°	$p<0.001$
RH pre- and		visual	horizontal	3.06° - 1.87°	$p<0.01$
post-rolandic		visual	vertical	2.77° - 1.92°	$p<0.01$
2 weeks -	6 months	tactile	horizontal	17.95° - 2.35°	$p<0.01$
N = 8	N = 8	tactile	vertical	11.35° - 2.47°	$p<0.01$

RH: right hemisphere

In the patient group with an exclusively post-rolandic lesion one patient still had a defective performance on the visual as well as on the tactile part of the test both six months and one year after the stroke.

A patient was considered to have recovered, for the purpose of this study, when the error rates became equal with the mean error rates of the adult control group plus twice the standard deviation.

The volumes of the lesions were calculated in the way described in Chapter II. Figure 24 shows the relation between the volume of the lesion and the recovery time both for the patient group with a pre- and post-rolandic extension of the lesion (figure 24 left) and for the patients with an exclusively post-rolandic lesion of the right hemisphere (figure 24 right). Although there is no linear relationship, some form of relation does exist.

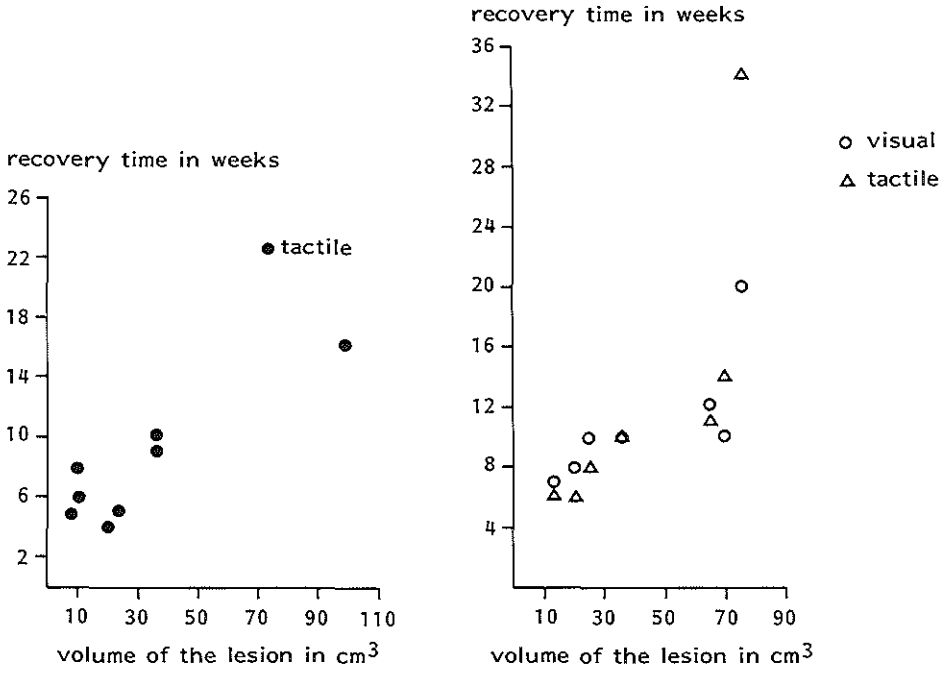

Figure 24. Relation between lesion size and recovery time.

DISCUSSION

Most patients performed again at a normal level between three and six months after the stroke. It is striking that in a previous study patients with a

posterior lesion of the right hemisphere performed significantly worse than the control patients one year after the stroke, which was not found in the present investigation. In terms of individual patients, this difference in findings is reflected by the fact that in the previous study five patients recovered only partly, whereas in the present group only one patient showed a partial recovery.

The difference found cannot be explained on the basis of a difference in lesion size, because the mean volume of the infarct was 29 cm^3 in the first study and 36 cm^3 in the present group. The most probable explanation for the improvement in performance in the present group is training. They were tested five times while the patients in the first group were tested only twice.

A crude relationship between the size of the lesion and the speed of recovery was found (figure 24).

The lesions of the patients with an exclusively post-rolandic lesion had an area in common, which overlapped the common area shown in figure 3.

The results show that the line orientation test was much less sensitive in assessing a disturbance of spatial perception than the rod orientation test in the sixteen patients with a posterior lesion of the right hemisphere: only three patients had a defective performance with the line orientation test. However, all had a disturbance of spatial perception when examined with the rod orientation test, those with a pre- and post-rolandic extension of the lesion principally on the tactile modality, and those with an exclusively post-rolandic lesion on both modalities tested. Since the line orientation test can only be presented by visual means, no failures were expected on this test in the first group of patients. Accordingly, Benton et al (1978) had no failures in their patient group with a perirolandic lesion. Of the three patients with a disturbed performance on the line orientation test, one had a pre- and post-rolandic extension of the lesion, and two an exclusively post-rolandic lesion. All three scored 'severely defective' (table 14). As appears from the recovery profiles, this defective performance was found only two weeks after the stroke in two patients. In one patient the defective performance continued for 22 weeks, which went parallel with the recovery of the disturbance of spatial perception as measured with the rod orientation test.

There are several possible explanations for the difference in results with the line orientation test in this investigation and in the study done by Benton et al. (1978):

1. Aetiology: only infarcts in this study; vascular, neoplasm and other brain lesions in theirs.
2. The presence or absence of oedema or a midline shift could influence the test results. These data were not mentioned in their article.
3. Low sensitivity of the line orientation test (for their results see table 16).

Table 16. Performance level and locus of lesion in patients with right-hemisphere disease.

(data from Benton et al. 1978)

locus		pass	fail
Prefrontal	V.F.D.+	0	0
		4	0
	V.F.D.−	4	0
Perirolandic	V.F.D.+	3	0
		13	0
	V.F.D.−	10	0
Posterior	V.F.D.+	1	7
		2	8
	V.F.D.−	1	1
Indeterminate	V.F.D.+	2	4
		7	9
	V.F.D.−	5	5

V.F.D.: visual field defect

From these results it appears that in the patient group with a right posterior lesion eight patients failed and only two passed. In the patient groups with a prefrontal or perirolandic lesion all patients passed this test. Therefore, many patients who were classified in the indeterminate group (large lesions encompassing two or more of the other categories; of these patients 7 passed the test and 9 failed) probably belong in the right posterior group.

CONCLUSIONS

1. It was possible to draw up recovery profiles of patients with disturbances of spatial perception. Most patients performed at a normal level between three and six months after the stroke.
2. In this patient group there was a recovery of spatial disorientation in all patients except one. Six months after the stroke there was no significant difference between the mean error rates of the brain-damaged patients and the control subjects.

3. The lesions of the patients with an exclusively post-rolandic lesion had an area in common, which overlapped the common area that was found in an earlier investigation.
4. Compared with the rod orientation test, the line orientation test was not very sensitive in assessing disturbances of spatial perception.

CHAPTER IV

DEVELOPMENT OF SPATIAL PERCEPTION IN CHILDREN

After testing the adult control patients, the question arose how children in different age groups without a history or signs of brain disease would perform on the rod orientation test. Therefore, thirty children were tested, ten in each of three age groups, namely a 4-6 year, a 7-9 year and a 10-13 year group. They had all been admitted to the University Children's Hospital; 18 of them had asthma, 2 cystic fibrosis, 3 diabetes, 3 uretral stenosis and 4 uncertain internal disorders.

Testing procedure

The method used was the rod orientation test as described on page 24. In the age group 4-6 years, only the visual version of the test was given as these children were easily frightened when blindfolded. In the other age groups, both the visual and the tactile version of the test were given. 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 children were first given five trials with the model to the right and then five trials with the model placed on the other side, each time with the rods placed in a different position (table 3). Both parts of the test were done with the right hand as well as with the left.

RESULTS

There were four mean error rates for each part of the test, both for the right and the left hand: horizontal error, vertical error, model left or right. For the statistical analysis an analysis of variance was used. In the three different age groups there were no significant differences between the error rates of the left and the right hand, nor according to the side of presentation of the model. Therefore, further statistical analysis was done on the mean of all individual error rates, separated only for horizontal and vertical errors.
Figure 25 shows the mean error rates of the examined children on the ordinate and their rank number on the abscissa. Both in the youngest age

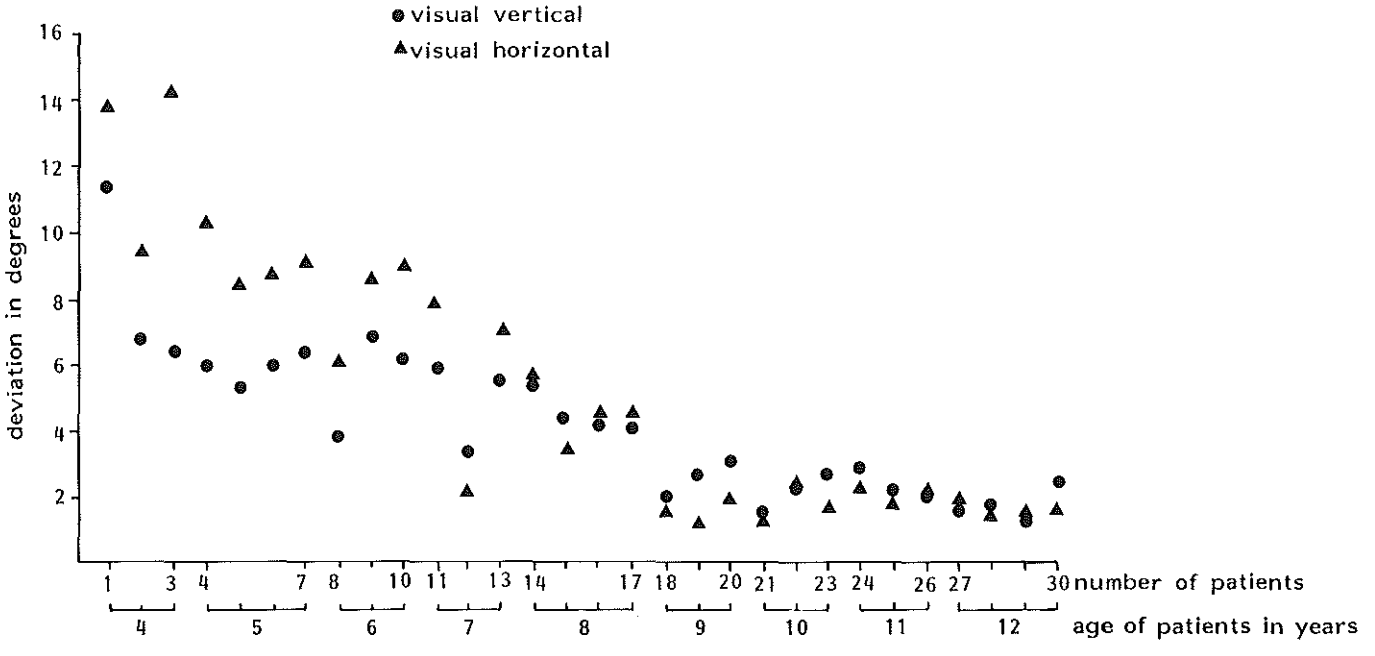

Figure 25. Mean error rates of the examined children in the different age groups.

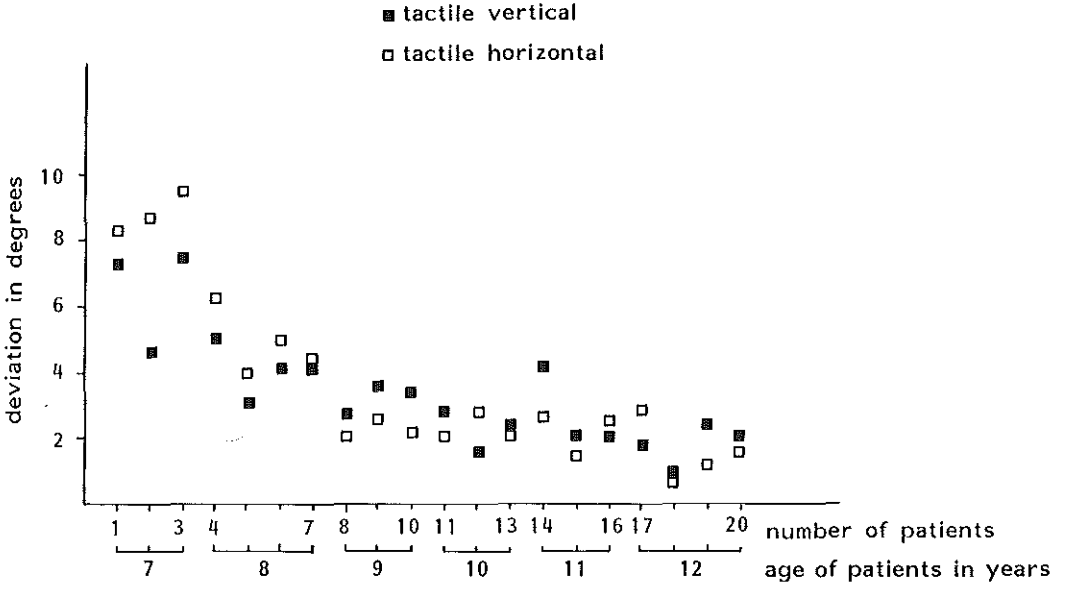

Figure 25. Mean error rates of the examined children in the different age groups.

group (4-6 years) and partly in the older age group (7-9 years) the horizontal error was greater than the vertical error.
In comparing the error rates of the children in the age group 10-13 years with those of 40 adults without a history or signs of brain disease (Chapter II), I found a statistically significant difference only for the tactile part of the test, in which the adults did worse ($p<0.01$).

Table 17 shows the mean duration of each part of the test in the different age groups, both for the left and the right hand.

Table 17. Mean duration of the rod orientation test for the left and the right hand.

	visual		tactile	
	R hand	L hand	R hand	L hand
4- 6 years	36 sec.	33.6 sec.		
7- 9 years	28 sec.	29.5 sec.	55.5 sec.	54.4 sec.
10-13 years	32.2 sec.	31.6 sec.	84.9 sec.	86.5 sec.

DISCUSSION

The children in the youngest age group had the highest mean error rates. With increasing age the error rates decreased (figure 25) and the performance of the children in the age group 10-13 years was about the same as that of the adults for both parts of the test (on the tactile part they were even better). It was surprising to find that there was no significant difference between the error rates made with the left or the right hand, or according to the side of presentation of the model, not even in the youngest age group. It is striking that both in the youngest children (4-6 years) and partly in the intermediate age group (7-9 years), the horizontal error was greater than the vertical error. The same was found in the adult patients with an infarct in the posterior region of the right hemisphere, especially in the tactile part of the test. Directional perception in the horizontal plane is perhaps more difficult than in the vertical plane. This difference disappears when the central nervous system matures, but re-appears after damage to the right posterior region. Chance could be another possible explanation for this finding in children, as the differences between the error rates in the horizontal and the vertical plane were small.

Table 17 shows that there is hardly any difference between the mean duration of the test for the left and the right hand, both in the visual and in the tactile part.

In the visual part of the test the youngest children were just playing with the rods before placing them in the final direction, but without any correction afterwards. That the mean duration in the tactile part of the test was much longer in the age group 10-13 years compared with the age group 7-9 years is probably because the older children were much more intent on an accurate result. The same applies to the performance in the visual part.

Bairstow and Lazlo (1981) have tested kinaesthetic sensitivity to passive movements in children from 5-12 years. They administered two tests of which one, a bimanual test, measured angle discrimination and the other accuracy of perception and memory of complex movement patterns. In both tests the apparatus was placed under a masking box. From the age of five in the first test and from the age of seven in the second, they, too, found an improvement in performance with increasing age. The results of the second test could be compared with those of the tactile part of the rod orientation test, at least in the horizontal plane.

CHAPTER V

DISTURBANCES OF SPATIAL PERCEPTION IN A PATIENT WITH AGENESIA OF THE CORPUS CALLOSUM

A girl, born 9. 8. 1973, was admitted to our hospital in 1974 with retarded development and macrocephaly. Gestation and delivery had been uneventful. Her birth-weight was 3300 grammes. She was the fourth child of healthy parents, the other children were normal. The mother had noticed that she developed more slowly than her other children. The family history revealed no abnormalities.

Neurological examination at the age of 8 months: Alert, inactive baby of normal length and weight. The head circumference was 49 cm, exceeding the 98th percentile; the anterior fontanelle was large but not tense. There was a rotatory clock-wise nystagmus and a convergent strabismus.

Both arms and legs were moved symmetrically. There was a left-hand preference. She grabbed an object with her full hand and brought it to her mouth. She could not sit without support and her head balance was unsteady.

The CT scan revealed a typical picture of agenesia of the corpus callosum (figure 26).

At the age of eight years, she attended primary school (IQ 104, Raven progressive matrices test). On examination she had a latent nystagmus and a convergent strabismus. There was a preference for the left hand, the fine finger movements were clumsy, especially of the right hand. There was a slight intention tremor on both sides. She could not stand on one leg and the tandem gait was difficult.

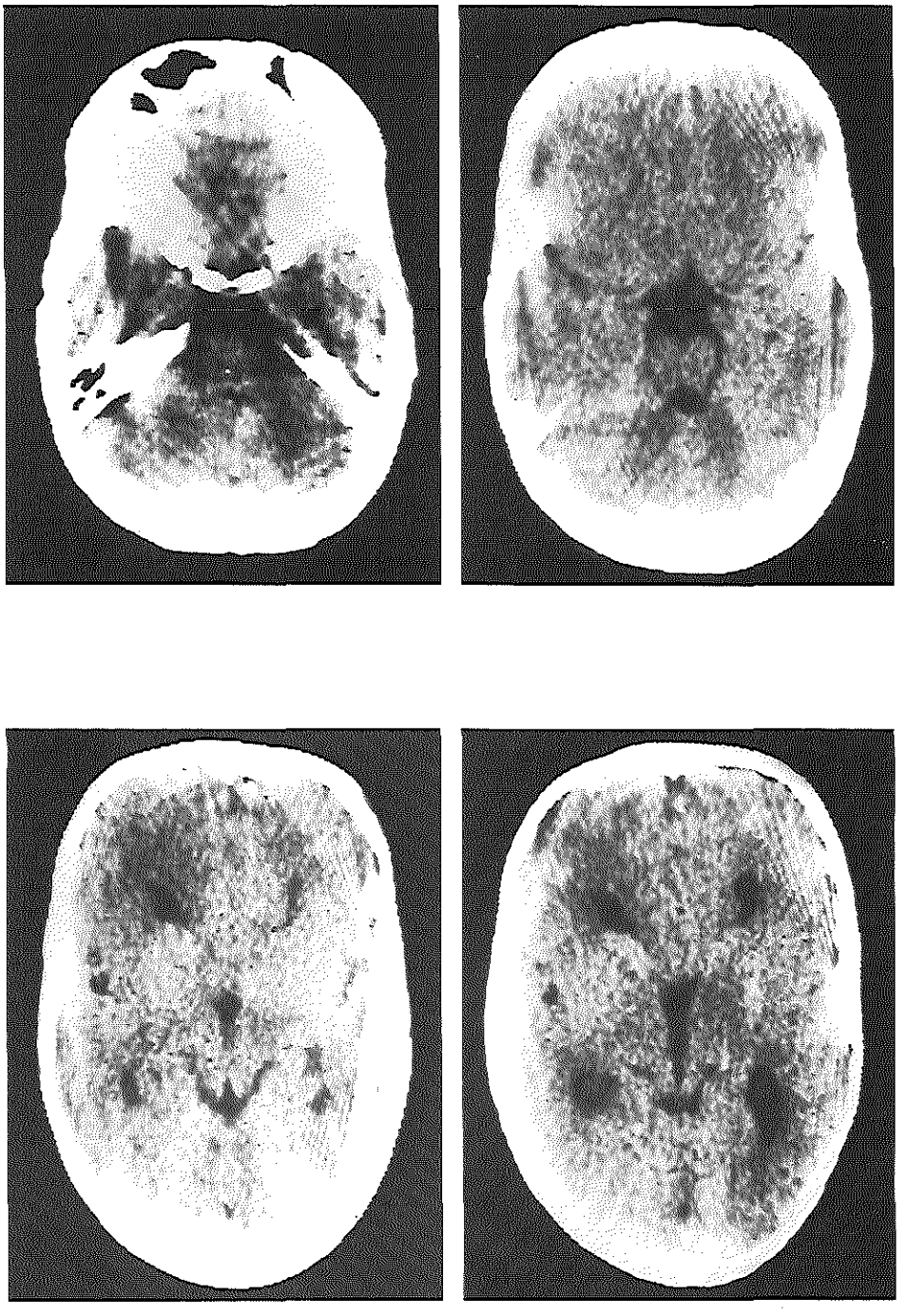

Figure 26. CT scan of a child with agenesia of the corpus callosum.

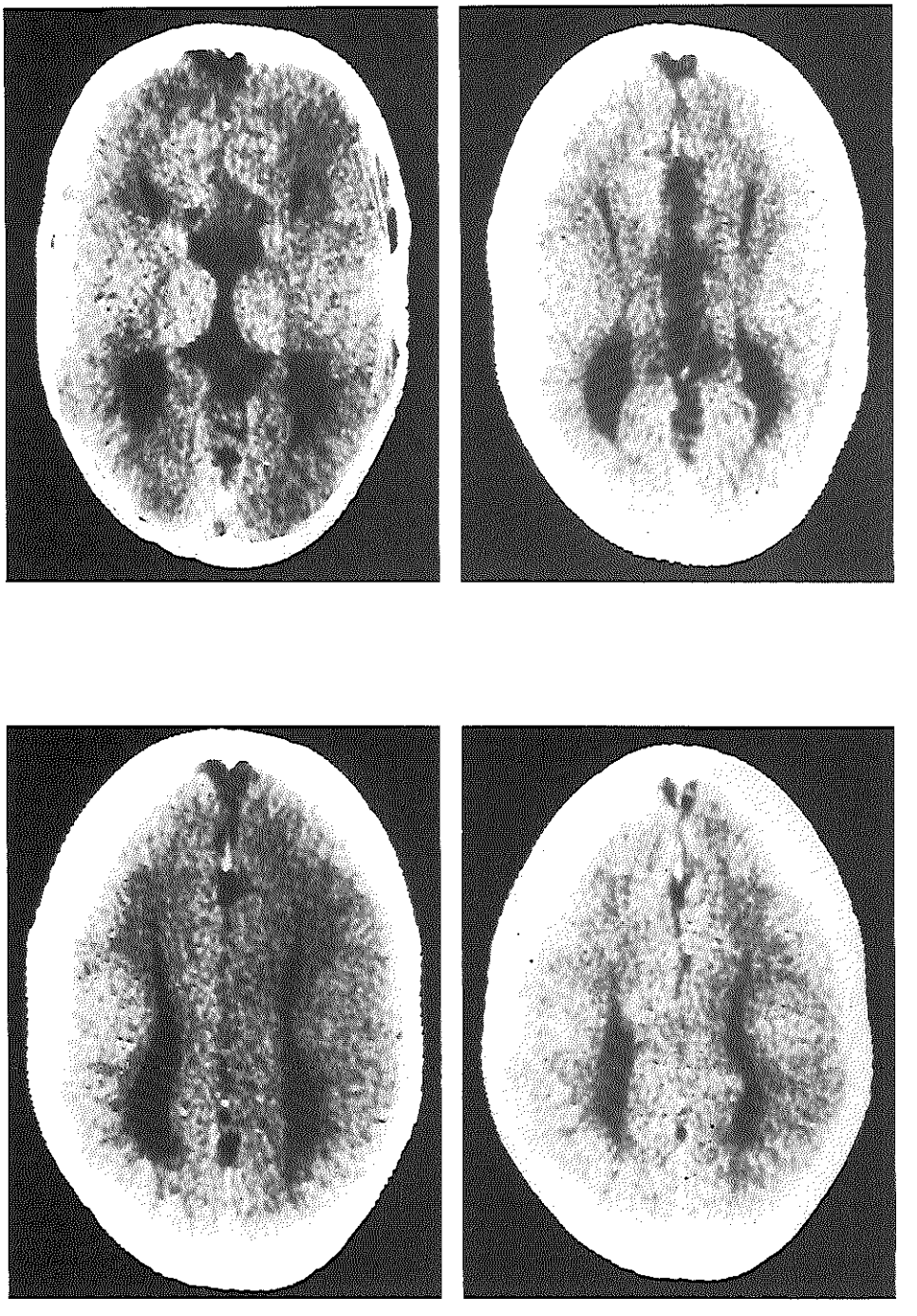

Figure 26. CT scan of a child with agenesia of the corpus callosum.

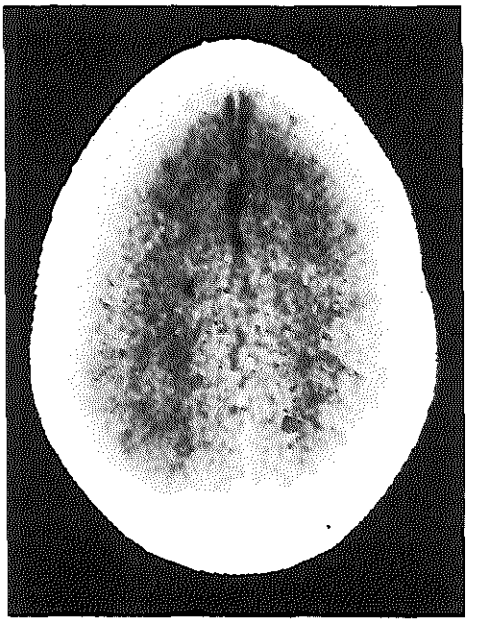

Figure 26. CT scan of a child with agenesia of the corpus callosum.

RESULTS

Table 18 shows the mean error rates and the mean duration (of model left and right) of the rod orientation test both for the left and the right hand.

Table 18. Mean error rates and mean duration (in seconds) of the rod orientation test in a patient with agenesia of the corpus callosum.

		left hand		right hand	
		mean error rates	mean duration	mean error rates	mean duration
visual	horizontal	6°	42	7.8°	45
visual	vertical	4.2°		3°	
tactile	horizontal	7°	83	28°	123
tactile	vertical	5.8°		18°	

From these results it appears that there was a serious disturbance in spatial perception and the longest mean duration for the tactile part of the test only when the patient used her right hand.

DISCUSSION

These results fitted in with a disturbed interhemispheric transfer of tactile information and pointed to a right-hemisphere dominance for spatial perception in this girl at the age of eight years.
Only when she used her left hand, the tactile information directly reached the right hemisphere. When she used her right hand, however, the tactile information reached the left hemisphere only (figure 27).
Apparently, for an accurate motor response of the right hand in the rod orientation test "cross-talk" with the right posterior region is necessary. In this patient this was impossible on palpation with the right hand, but in the visual part of the test information reached both hemispheres, and in consequence the error rates were within the normal range. These results confirm the previous findings that when spatial perception is tested at an elementary level there is an almost complete dominance of the right hemisphere.

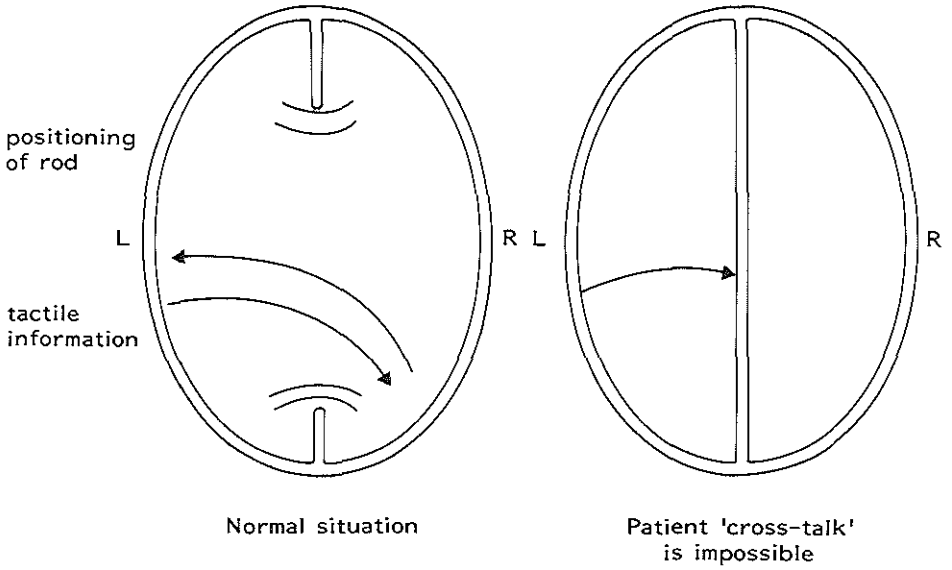

Figure 27.

PATIENT DATA

In the first part of the study 68 patients were tested. They all had a cerebral infarct without a midline shift, as assessed by CT scanning. All patients were right-handed. 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 hemisphere (20 males, 15 females, the ages ranging from 30 to 80 years, mean age 66.4 years). For the site of the lesions see table 1. Three main groups were distinguished: exclusively pre-rolandic lesions, lesions with a pre- and post-rolandic extension, and exclusively post-rolandic lesions, as inferred from the CT scan.
For the left hemisphere this amounted to 7, 18 and 8 patients respectively, and for the right hemisphere to 9, 13 and 13 patients. Patients with a left-hemisphere lesion and those with an exclusively pre-rolandic lesion of the right hemisphere were tested only once: two weeks after the stroke.
Patients with a pre- and post-rolandic extension of the right-hemisphere lesion or an exclusively post-rolandic lesion of the right hemisphere were tested two weeks, and one year after the stroke.
A separate group of 16 patients who participated in the second part of the study were tested at fixed intervals, namely two weeks, six weeks, three months, six months and, if necessary, one year, after the stroke. There were eight patients both in the group with a pre- and post-rolandic extension of the lesion and in the group with an exclusively post-rolandic lesion (table 12).
All patients were examined and tested by the author. At the time of testing all patients were fully conscious. In the examined group there were no patients with a hemispatial neglect. Wherever paresis or weakness is mentioned, a pyramidal weakness is meant, mostly with hyperreflexia and a Babinski sign.
The control group consisted of 40 patients (26 males, 14 females) visiting the out-patient department without a history or signs of brain disease (diagnosis: lumbar disc disease, lumbago, mono-neuropathy). 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).
Thirty children were tested in order to study the development of spatial perception. There were ten children in each of three age groups, namely 4-6 years, 7-9 years and 10-13 years. They had all been admitted to the University Children's Hospital; 18 of them had asthma, 2 cystic fibrosis, 3 diabetes, 3 uretral stenosis and 4 uncertain internal disorders.

Patients with an exclusively pre-rolandic lesion of the right hemisphere

Male, born 18. 8. 1906.
Examination on admission (13. 12. 1978):
slight facial asymmetry and slight paresis of the left leg.
CT: right-sided fronto-temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	1.6
	vertical	3.4
tactile	horizontal	7.4
	vertical	9.1

Female, born 7. 10. 1907.
Examination on admission (24. 5. 1978):
slight facial asymmetry, slight left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	7.4
	vertical	5.3
tactile	horizontal	6.3
	vertical	5.7

Male, born 6. 12. 1909.
Examination on admission (28. 12. 1977):
gaze paralysis to the left, moderate left-sided central facial paresis, severe left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.7
	vertical	2.3
tactile	horizontal	3.1
	vertical	3.9

Male, born 28. 1. 1915.
Examination on admission (16. 9. 1977):
slight left-sided central facial paresis, moderate paresis of the left arm and slight left-sided sensory disturbances.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	5.3
	vertical	1.8
tactile	horizontal	11.9
	vertical	10.2

Female, born 5. 7. 1924.
Examination on admission (15. 9. 1978):
slight facial asymmetry, moderate left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	5
	vertical	2.3
tactile	horizontal	3.2
	vertical	3.1

Male, born 6. 8. 1929.
Examination on admission (7. 12. 1978):
slight left-sided central facial paresis, severe left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	4
	vertical	5.1
tactile	horizontal	5.2
	vertical	4.9

Male, born 8. 1. 1932.
Examination on admission (15. 9. 1977):
slight left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	4.2
	vertical	2.7
tactile	horizontal	6.7
	vertical	2.9

Male, born 17. 2. 1945.
Examination on admission (29. 2. 1980):
slight left-sided hemiplegia.
CT: right-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	1.8
	vertical	2.8
tactile	horizontal	4
	vertical	3

Female, born 13. 1. 1949.
Examination on admission (12. 12. 1978):
slight left-sided central facial paresis, severe left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided fronto-temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2
	vertical	2.7
tactile	horizontal	5.4
	vertical	3.4

Patients with a pre- and post-rolandic extension of the lesion

Male, born 20. 11. 1904.
Examination on admission (12. 1. 1978):
slight left-sided central facial paresis, moderate left-sided hemiplegia, and slight left-sided sensory disturbances.
CT: right-sided centro-temporal infarct.
Volume of the lesion: 3 cm^3.
Re-examination after one year: slight left-sided hemiplegia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	14.3	3.8
	vertical	7.6	3.8
tactile	horizontal	15.9	4.1
	vertical	13.2	4.1

Female, born 25. 2. 1907.
Examination on admission (22. 9. 1977):
moderate left-sided central facial paresis, severe left-sided hemiplegia, with moderate left-sided sensory disturbances and a tactile extinction phenomenon.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 84 cm^3.
Re-examination after one year: moderate facial paresis, moderate left-sided hemiplegia, with slight left-sided sensory disturbances.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	22.2	5.2
	vertical	13.9	4.9
tactile	horizontal	98.2	10.1
	vertical	31.5	10.4

Male, born 7. 10. 1907.
Examination on admission (3. 7. 1977):
left-sided central facial paresis, slight left-sided hemiplegia, with moderate left-sided sensory disturbances.
CT: right-sided temporo-occipital infarct.
Volume of the lesion: 42 cm^3.
Re-examination after one year: slight left-sided sensory deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	5.5	3.2
	vertical	3.6	4.2
tactile	horizontal	18.6	4
	vertical	15.8	2.9

Male, born 1. 5. 1910.
Examination on admission (17. 11. 1978):
moderate left-sided central facial paresis, moderate left-sided hemiplegia, and slight left-sided sensory disturbances.
CT: right-sided temporo-occipital infarct.
Volume of the lesion: 37 cm^3.
Re-examination after one year: slight facial asymmetry, and slight left-sided hemiplegia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	13.7	3.1
	vertical	4.2	2.8
tactile	horizontal	18.2	4.3
	vertical	12	3.9

Male, born 15. 1. 1911.
Examination on admission (16. 2. 1978):
slight left-sided central facial paresis, and clumsiness of the left hand.
CT: right-sided temporo-occipital infarct.
Volume of the lesion: 14 cm^3.
Re-examination after one year: slight facial asymmetry.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	10	3.6
	vertical	5	3.6
tactile	horizontal	29.3	4.3
	vertical	11.8	4.3

Male, born 8. 2. 1911.
Examination on admission (24. 10. 1978):
dressing apraxia, finger agnosia, and a severe spatial disorientation.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 31 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	6.6	3.1
	vertical	9.8	4.2
tactile	horizontal	15.3	3.9
	vertical	10.2	3.9

Male, born 25. 8. 1915.
Examination on admission (19. 5. 1978):
slight left-sided central facial paresis, slight left-sided hemiplegia, with slight left-sided sensory disturbances.
CT: right-sided temporo-occipital infarct.
Volume of the lesion: 31 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	6.3	3.3
	vertical	3.6	2.7
tactile	horizontal	10.3	4.7
	vertical	5	4.2

Male, born 26. 2. 1916.
Examination on admission (26. 10. 1977):
left-sided central facial paresis, slight paresis of the left arm, with slight left-sided sensory disturbances and a sensory extinction phenomenon.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 86 cm^3.
Re-examination after one year: slight facial asymmetry, and pyramidal paresis of the left arm.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	7.1	6
	vertical	6.1	3.2
tactile	horizontal	27.1	11.6
	vertical	26.1	8.5

Male, born 30. 12. 1922.
Examination on admission (17. 10. 1977):
severe spatial disorientation, left-sided central facial paresis, slight weakness of the left arm, and moderate sensory disturbances on the left side and a tactile extinction phenomenon.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 74 cm^3.
Re-examination after one year: slight left-sided sensory disturbances.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	10.8	0.7
	vertical	10	3.3
tactile	horizontal	24.6	10.7
	vertical	14.9	8.3

Female, born 22. 2. 1928.
Examination on admission (30. 7. 1978):
slight left-sided central facial paresis, slight left-sided hemiplegia, with moderate left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 20 cm^3.
No follow-up, the patient refused to participate again in the study.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.6
	vertical	3.5
tactile	horizontal	17.2
	vertical	11.8

Female, born 23. 6. 1932.
Examination on admission (18. 2. 1976):
left-sided homonymous hemianopia, left-sided central facial paresis, severe left-sided sensory disturbances, and a tactile extinction phenomenon.
CT: right-sided fronto-temporo-parietal infarct.
Volume of the lesion: 88 cm^3.
Re-examination after one year: slight left-sided central facial paresis, slight left-sided sensory disturbances and a tactile extinction phenomenon.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	10.1	4.8
	vertical	5.2	4.6
tactile	horizontal	64	14.9
	vertical	22	11.7

Male, born 21. 10. 1928.
Examination on admission (4. 10. 1979):
left-sided homonymous quadrantic anopia, slight left-sided central facial paresis, moderate left-sided hemiplegia, with slight left-sided sensory disturbances.
CT: right-sided temporo-occipital infarct.
Volume of the lesion: 18 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	2.6	2.8
	vertical	2.7	2.2
tactile	horizontal	9.9	2.7
	vertical	10.1	2.4

Female, born 23. 6. 1930.
Examination on admission (24. 10. 1977):
slight left-sided hemiplegia, with moderate left-sided sensory disturbances.
CT: right-sided temporo-central infarct.
Volume of the lesion: 16 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	8.3	4.7
	vertical	4.7	2.5
tactile	horizontal	12.5	2.5
	vertical	11	3.2

Patients with an exclusively post-rolandic lesion

Male, born 24. 12. 1899.
Examination on admission (18. 2. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 26 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	14.4	5.1
	vertical	15	2.6
tactile	horizontal	11.4	5.5
	vertical	15.9	5

Female, born 4. 5. 1900.
Examination on admission (9. 12. 1977):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 30 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	18.7	5
	vertical	13.5	4.3
tactile	horizontal	17.6	5.6
	vertical	24.1	4.5

Female, born 28. 2. 1904.
Examination on admission (15. 4. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 8 cm^3.
Re-examination after one year: left-sided homonymous quadrantic anopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	37.2	3.1
	vertical	15.4	4.7
tactile	horizontal	63	5.5
	vertical	24.4	5.4

Female, born 29. 10. 1909.
Examination on admission (12. 12. 1977):
left-sided homonymous hemianopia, slight left-sided facial paresis.
CT: right-sided temporo-parieto-occipital infarct.
Volume of the lesion: 25 cm^3.
Re-examination after one year: left-sided homonymous quadrantic anopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	25.5	3.4
	vertical	9.2	2.8
tactile	horizontal	48	2.9
	vertical	15	3.7

Male, born 25. 7. 1910.
Examination on admission (12. 1. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 26 cm^3.
Re-examination after one year: left-sided homonymous quadrantic anopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	15.1	4.3
	vertical	13.2	3.5
tactile	horizontal	22.5	4.5
	vertical	18.9	3.3

Male, born 4. 8. 1911.
Examination on admission (21. 8. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 44 cm^3.
Re-examination after one year: left-sided homonymous hemianopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	18.5	3.9
	vertical	12.4	2.9
tactile	horizontal	19	3
	vertical	16.5	4.2

Female, born. 22. 2. 1916.
Examination on admission (20. 10. 1977):
left-sided homonymous hemianopia and moderate left-sided sensory disturbances.
CT: right-sided occipital infarct.
Volume of the lesion: 57 cm^3.
Re-examination after one year: left-sided homonymous hemianopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	26.1	2.6
	vertical	5.6	4.4
tactile	horizontal	33.2	3
	vertical	9.6	5.9

Male, born 19. 8. 1917.
Examination on admission (23. 10. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 8 cm^3.
Re-examination after one year: left-sided homonymous hemianopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	6.2	1.4
	vertical	5.8	1.6
tactile	horizontal	6.8	3.6
	vertical	4.2	2.8

Male, born 17. 2. 1921.
Examination on admission (8. 6. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 7 cm^3.
Re-examination after one year: no neurological deficit.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	13.3	4.6
	vertical	14.1	4.3
tactile	horizontal	10.2	4.9
	vertical	15.9	3.9

Male, born 10. 6. 1924.
Examination on admission (6. 9. 1977):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 84 cm^3.
No follow-up, the patient refused to participate again in the study.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	11.6
	vertical	10.7
tactile	horizontal	55
	vertical	23

Male, born 15. 4. 1925.
Examination on admission (29. 5. 1978):
left-sided homonymous hemianopia, left-sided sensory disturbances and an astereognosia.
CT: right-sided temporo-parieto-occipital infarct.
Volume of the lesion: 72 cm^3.
Re-examination after one year: left-sided homonymous hemianopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	14.5	2.1
	vertical	15.1	3.9
tactile	horizontal	34.9	3.1
	vertical	11.5	4.4

Female, born 15. 12. 1931.
Examination on admission (1. 12. 1977):
left-sided homonymous hemianopia and spatial disorientation.
CT: right-sided occipital infarct.
Volume of the lesion: 92 cm^3.
Re-examination after one year: left-sided homonymous hemianopia and spatial disorientation.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	17.3	10.2
	vertical	13.3	7
tactile	horizontal	34.9	16.8
	vertical	9.8	8.2

Female, born 11. 9. 1941.
Examination on admission (23. 6. 1978):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 62 cm^3.
Re-examination after one year: left-sided homonymous hemianopia.
Mean error rates rod orientation test:

		2 weeks	1 year
visual	horizontal	15.8	4.3
	vertical	15.3	1.8
tactile	horizontal	47.8	3.5
	vertical	25.4	2.9

Patients with an exclusively pre-rolandic lesion of the left hemisphere

Male, born 11. 1. 1904.
Examination on admission (26. 11. 1978):
slight right-sided central facial paresis, slight right-sided hemiplegia, and a global aphasia.
CT: left-sided fronto-temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	1.2
	vertical	2
tactile	horizontal	2.2
	vertical	2.1

Male, born 1. 12. 1940.
Examination on admission (6. 7. 1978):
motor aphasia, slight facial asymmetry, moderate right-sided hemiplegia, and slight right-sided sensory disturbances.
CT: left-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	1.2
	vertical	2.3
tactile	horizontal	3.9
	vertical	4

Male, born 29. 6. 1942.
Examination on admission (3. 3. 1979):
global aphasia, slight facial asymmetry, slight right-sided hemiplegia, and a diminished reaction to pain on the right side.
CT: left-sided fronto-temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.1
	vertical	1.2
tactile	horizontal	3.4
	vertical	3

Female, born 29. 9. 1923.
Examination on admission (22. 12. 1977):
global aphasia, slight facial asymmetry, moderate right-sided hemiplegia, and slight right-sided sensory disturbances.
CT: left-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3.5
	vertical	3
tactile	horizontal	2.8
	vertical	2.7

Female, born 12. 6. 1937.
Examination on admission (23. 7. 1978):
slight right-sided hemiplegia, and a global aphasia.
CT: left-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.7
	vertical	3
tactile	horizontal	3
	vertical	2.8

Female, born 2. 1. 1914.
Examination on admission (24. 8. 1977): motor aphasia.
CT: left-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.1
	vertical	2.5
tactile	horizontal	3
	vertical	2.5

Male, born 22. 5. 1930.
Examination on admission (23. 9. 1977): motor aphasia.
CT: left-sided frontal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.1
	vertical	1.6
tactile	horizontal	2.7
	vertical	2.2

Patients with a pre- and post-rolandic extension of the lesion

Female, born 4. 5. 1906.
Examination on admission (13. 2. 1981):
sensory aphasia and a right-sided homonymous hemianopia.
CT: left-sided temporo-occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.1
	vertical	1.6
tactile	horizontal	3.6
	vertical	3

Female, born 23. 11. 1908.
Examination on admission (27. 7. 1978):
severe right-sided hemiplegia and slight sensory disturbances of the right arm.
CT: left-sided fronto-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.6
	vertical	3.1
tactile	horizontal	1.3
	vertical	2.8

Male, born 20. 4. 1909.
Examination on admission (8. 2. 1979):
global aphasia, right-sided hemianopia, moderate right-sided central facial paresis, severe right-sided hemiplegia and diminished reaction to pain on the right side.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.9
	vertical	2.6
tactile	horizontal	4.1
	vertical	3.3

Male, born 25. 2. 1911.
Examination on admission (1. 12. 1978): global aphasia.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3.2
	vertical	2.7
tactile	horizontal	5.7
	vertical	4.6

Male, born 30. 6. 1911.
Examination on admission (9. 6. 1977):
global aphasia, moderate right-sided central facial paresis, severe right-sided hemiplegia.
CT: left-sided fronto-temporo-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3
	vertical	2
tactile	horizontal	2.2
	vertical	3.6

Male, born 3. 4. 1912.
Examination on admission (23. 7. 1977):
global aphasia and slight facial asymmetry.
CT: left-sided temporo-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	5.5
	vertical	3.5
tactile	horizontal	2
	vertical	2.6

Male, born 21. 2. 1913.
Examination on admission (4. 8. 1977):
sensory aphasia and a right-sided homonymous quadrantic hemianopia.
CT: left-sided temporo-occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	5.7
	vertical	4.6
tactile	horizontal	8.4
	vertical	6

Male, born 5. 5. 1914.
Examination on admission (13. 1. 1978):
slight facial asymmetry, slight right-sided hemiplegia and slight right-sided sensory disturbances.
CT: left-sided temporo-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.6
	vertical	4.9
tactile	horizontal	2.6
	vertical	3.6

Female, born 30. 8. 1914.
Examination on admission (2. 11. 1978): global aphasia.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3
	vertical	3
tactile	horizontal	3.7
	vertical	3.7

Female, born 8. 10. 1916.
Examination on admission (31. 12. 1977):
global aphasia, right-sided central facial paresis, slight right-sided hemiplegia and slight right-sided sensory disturbances.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.4
	vertical	2.5
tactile	horizontal	8.2
	vertical	4.6

Male, born 4. 10. 1921.
Examination on admission (9. 10. 1977):
sensory aphasia, slight right-sided hemiplegia and slight right-sided sensory disturbances.
CT: left-sided centro-temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.7
	vertical	2.6
tactile	horizontal	2.8
	vertical	2.5

Male, born 28. 11. 1922.
Examination on admission (30. 9. 1977):
global aphasia, moderate right-sided central facial paresis, severe right-sided hemiplegia.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.2
	vertical	3.1
tactile	horizontal	3.2
	vertical	2.5

Male, born 25. 1. 1929.
Examination on admission (24. 11. 1978): global aphasia.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	0.8
	vertical	4
tactile	horizontal	3.1
	vertical	3

Female, born 24. 5. 1929.
Examination on admission (28. 2. 1978):
slight facial asymmetry, slight right-sided hemiplegia and slight right-sided sensory disturbances.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.9
	vertical	2.3
tactile	horizontal	4.5
	vertical	2.6

Female, born 12. 11. 1930.
Examination on admission (31. 10. 1977):
global aphasia, moderate right-sided central facial paresis, severe right-sided hemiplegia and slight right-sided sensory disturbances.
CT: left-sided temporo-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.6
	vertical	2.5
tactile	horizontal	3.7
	vertical	3.1

Male, born 5. 5. 1931.
Examination on admission (28. 8. 1978):
global aphasia, slight right-sided hemiplegia and astereognosia.
CT: left-sided temporo-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3.1
	vertical	3.4
tactile	horizontal	2.8
	vertical	2.1

Male, born 2. 11. 1936.
Examination on admission (9. 10. 1977):
global aphasia, moderate right-sided hemiplegia and moderate right-sided sensory disturbances.
CT: left-sided temporal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	7.8
	vertical	2.1
tactile	horizontal	8.4
	vertical	6

Female, born 12. 9. 1947.
Examination on admission (14. 8. 1977):
slight right-sided central facial paresis, moderate right-sided hemiplegia and moderate right-sided sensory disturbances.
CT: left-sided fronto-temporo-parietal infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	4.3
	vertical	4
tactile	horizontal	6.6
	vertical	6

Patients with an exclusively post-rolandic lesion

Male, born 22. 10. 1904.
Examination on admission (2. 1. 1979):
right-sided homonymous hemianopia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3.1
	vertical	3.4
tactile	horizontal	3.7
	vertical	3

Female, born 4. 5. 1906.
Examination on admission (4. 7. 1978):
amnesic aphasia and a right-sided homonymous hemianopia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	4.7
	vertical	4.7
tactile	horizontal	2.2
	vertical	1.7

Male, born 8. 12. 1909.
Examination on admission (26. 10. 1977):
right-sided homonymous hemianopia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2
	vertical	2
tactile	horizontal	5.7
	vertical	5.5

Male, born 1. 1. 1910.
Examination on admission (5. 2. 1978):
right-sided homonymous hemianopia and a sensory aphasia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.9
	vertical	3
tactile	horizontal	1.6
	vertical	2.2

Male, born 13. 12. 1916.
Examination on admission (15. 12. 1978):
right-sided homonymous hemianopia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	2.6
	vertical	4.9
tactile	horizontal	2.6
	vertical	3.6

Male, born 1. 7. 1918.
Examination on admission (21. 5. 1978):
right-sided homonymous hemianopia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3.1
	vertical	1.4
tactile	horizontal	3.3
	vertical	1.9

Female, born 12. 9. 1921.
Examination on admission (29. 11. 1978):
global aphasia, right-sided homonymous hemianopia and a moderate right-sided central facial paresis.
CT: left-sided temporo-parieto-occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	5.4
	vertical	3.3
tactile	horizontal	6
	vertical	3.9

Male, born 8. 1. 1923.
Examination on admission (16. 6. 1977):
right-sided homonymous hemianopia.
CT: left-sided occipital infarct.
Mean error rates rod orientation test:

		2 weeks
visual	horizontal	3.5
	vertical	2.3
tactile	horizontal	8.8
	vertical	6

Patients with a pre- and post-rolandic extension of the lesion

Female, born 8. 6. 1905.
Examination on admission (18. 8. 1980):
slight left-sided central facial paresis, slight left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 36 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	1	1.4	1.2	1.4
	vertical	2.2	1.8	1.8	2
tactile	horizontal	18.6	6.2	2.4	2.2
	vertical	12.6	6	2.2	2.0
line orientation test		1	0	0	0

Male, born 6. 5. 1911.
Examination on admission (12. 6. 1980):
moderate left-sided central facial paresis, severe left-sided hemiplegia and moderate left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 10 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months	1 year
visual	horizontal	2.8	4	2.4	1.6	1.8
	vertical	2.8	3.8	4.2	3.6	3.2
tactile	horizontal	56.8	5	4.4	2.6	2.8
	vertical	31.8	2.8	4.2	3.8	3.5
line orientation test		5	0	0	0	0

Female, born 2. 5. 1916.
Examination on admission (31. 7. 1980):
left-sided central facial paresis, severe left-sided hemiplegia, and slight left-sided sensory disturbances.
CT: right-sided fronto-parietal infarct.
Volume of the lesion: 8 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	3	1.2	1.2	1.2
	vertical	2.2	1.6	1.8	2
tactile	horizontal	11	1.8	2	2
	vertical	7.8	2.8	2.6	2.4
line orientation test		8	0	0	0

Male, born 27. 8. 1927.
Examination on admission (9. 9. 1980):
slight left-sided hemiplegia and severe left-sided sensory disturbances.
CT: right-sided fronto-parietal infarct.
Volume of the lesion: 23 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	3.5	2.2	2	1.8
	vertical	3.2	2	2	2.2
tactile	horizontal	11	3.4	1.8	2
	vertical	6.8	3.6	2.2	2
line orientation test		0	0	0	0

Male, born 7. 10. 1929.
Examination on admission (8. 7. 1980):
moderate left-sided central facial paresis, severe left-sided hemiplegia and moderate left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 99 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months	1 year
visual	horizontal	3.2	3	2.2	2.2	2.2
	vertical	3.6	2.4	2.4	2.2	2
tactile	horizontal	9	8	6.8	3.8	2.4
	vertical	9.2	6.4	4.8	3.6	2
line orientation test		18	8	3	2	3

Male, born 29. 1. 1931.
Examination on admission (6. 11. 1980):
moderate left-sided central facial paresis, severe left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 10 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	3.4	3	1.8	2
	vertical	3.4	3	1.2	1.8
tactile	horizontal	16.8	7.8	4.4	3.2
	vertical	10.2	4.2	3.2	2.8
line orientation test		6	6	5	3

Female, born 15. 5. 1933.
Examination on admission (14. 6. 1981):
slight left-sided hemiplegia and moderate left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 20 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	2 months	6 months
visual	horizontal	2.6	1	1	1.4
	vertical	1.8	0.8	1	1.2
tactile	horizontal	8.4	2.8	2.4	2.2
	vertical	6.6	2.6	2.2	2.4
line orientation test		1	0	0	0

Male, born 22. 7. 1939.
Examination on admission (24. 3. 1981):
slight left-sided central facial paresis, slight left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided temporo-parietal infarct.
Volume of the lesion: 36 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	5	2	2.4	2.6
	vertical	3	1.6	2.4	2.2
tactile	horizontal	12	4.8	2.2	2.2
	vertical	6.4	7.6	4.2	2.4
line orientation test		9	4	2	0

Patients with an exclusively post-rolandic lesion of the right hemisphere

Female, born 16. 11. 1912.
Examination on admission (4. 2. 1981):
left-sided homonymous hemianopia.
CT: right-sided temporo-parieto-occipital infarct.
Volume of the lesion: 25 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	9.2	6.8	2.4	2.6
	vertical	9	6.4	2.4	2
tactile	horizontal	26.8	4.8	2.6	2.4
	vertical	20.4	4.4	2	2.2
line orientation test		6	0	0	0

Male, born 9. 9. 1913.
Examination on admission (1. 5. 1981):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 20 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	8.8	6	2.4	1.6
	vertical	11.4	3.6	2.6	1.8
tactile	horizontal	17.4	3.2	2.4	1.4
	vertical	8	4.2	2.6	1.4
line orientation test		18	2	1	1

Male, born 4. 12. 1917.
Examination on admission (26. 4. 1981):
left-sided homonymous hemianopia.
CT: right-sided occipital infarct.
Volume of the lesion: 13 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	7.4	3.2	2.8	2
	vertical	7.5	4.4	2.4	2.2
tactile	horizontal	10.1	4.2	2.6	2.2
	vertical	10.4	5.2	2	2.2
line orientation test		2	0	0	0

Male, born 12. 4. 1918.
Examination on admission (19. 1. 1981):
left-sided homonymous hemianopia, moderate left-sided central facial paresis, moderate left-sided hemiplegia and moderate left-sided sensory disturbances.
CT: right-sided parieto-occipital infarct.
Volume of the lesion: 65 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	9	6.2	3	3
	vertical	8.2	7.4	3.2	2.8
tactile	horizontal	12.2	7.6	4	3.2
	vertical	14.8	10.6	3	3
line orientation test		10	9	0	0

Female, born 4. 7. 1923.
Examination on admission (17. 8. 1980):
left-sided homonymous hemianopia, slight left-sided hemiplegia and slight left-sided sensory disturbances.
CT: right-sided occipital infarct.
Volume of the lesion: 68 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	7.8	3.2	2.8	3
	vertical	8.6	2.4	0.2	1.2
tactile	horizontal	8.8	4.8	4.2	1.4
	vertical	7.6	5.4	3.6	1.8
line orientation test		0	0	0	0

Female, born 14. 6. 1928.
Examination on admission (30. 1. 1981):
left-sided homonymous hemianopia, slight left-sided hemiplegia and moderate left-sided sensory disturbances.
CT: right-sided parieto-occipital infarct.
Volume of the lesion: 75 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months	1 year
visual	horizontal	17	12.2	4.6	4.8	5
	vertical	13.2	12	11.8	6.8	5.8
tactile	horizontal	18.8	14.8	10	6.4	6.4
	vertical	22.6	16.2	7	6.4	6.6
line orientation test		5	5	2	2	1

Male, born 30. 9. 1929.
Examination on admission (18. 1. 1981):
left-sided homonymous hemianopia, slight left-sided central facial paresis, slight left-sided hemiplegia and a left-sided sensory extinction phenomenon.
CT: right-sided temporo-parieto-occipital infarct.
Volume of the lesion: 76 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months	1 year
visual	horizontal	8.2	9	6	2.2	2.4
	vertical	7.2	6.6	4.4	2.4	2.6
tactile	horizontal	33.6	15.4	13.6	7.4	3
	vertical	23.6	13	19.4	5.4	2.8
line orientation test		22	22	16	9	3

Male, born 11. 12. 1932.
Examination on admission (28. 4. 1981):
left-sided homonymous quadrantic hemianopia.
CT: right-sided parieto-occipital infarct.
Volume of the lesion: 36 cm^3.
Mean error rates rod orientation test and scores of the line orientation test:

		2 weeks	6 weeks	3 months	6 months
visual	horizontal	12.4	1.2	1.4	1.6
	vertical	5.6	2.6	3	2.4
tactile	horizontal	20.2	1.6	2	2.2
	vertical	7.8	2.4	2.8	2.8
line orientation test		5	1	1	0

Control subjects

Mean of the error rates of the rod orientation test of 40 control subjects.

visual	horizontal	2.2
	vertical	2.2
tactile	horizontal	3.4
	vertical	3.4

Children

Mean error rates of the rod orientation test of 30 children.

	visual	
	vertical	horizontal
4 year-olds	11.4	13.9
	6.8	9.4
	6.4	14.2
5 year-olds	6	10.3
	5.3	8.4
	6	8.7
	6.4	9.1
6 year-olds	3.8	6.1
	6.7	8.5
	6.2	8.9

	visual		tactile	
	vertical	horizontal	vertical	horizontal
7 year-olds	5.9	7.8	7.4	8.3
	3.4	2	4.7	8.7
	5.5	7	7.6	9.5
8 year-olds	5.3	5.7	5.1	6.2
	4.4	3.4	3.1	4
	4.2	4.5	4.1	5
	4.1	4.5	4.1	4.5
9 year-olds	2	1.4	2.8	2
	2.6	1.2	3.6	2.6
	3.1	1.9	3.4	2.2

	visual		tactile	
	vertical	horizontal	vertical	horizontal
10 year-olds	1.5	1.2	2.9	2.1
	2.3	2.4	1.6	1.8
	2.7	1.6	2.4	2.1
11 year-olds	2.9	2.3	4.2	2.7
	1.8	1.8	2	1.5
	2.2	2.3	2.1	2.6
12 year-olds	1.7	1.9	1.9	2.9
	1.7	1.4	0.9	0.7
	1.2	1.7	2.4	1.2
	2.4	1.5	2.1	1.6

SUMMARY

This thesis describes a clinical study of disturbances of spatial perception.

Chapter I gives a short review of hemispheric asymmetry. For a long time the right hemisphere was regarded as the minor hemisphere, of which substantial regions were thought to have no specific function. Jackson, however, believed that the posterior area of the right hemisphere played a crucial role in subserving visual recognition and visual memory, but few of his contemporaries agreed with him.

Later Rieger, Babinski and Dide also stressed the importance of the right posterior region in spatial tasks. But is was not until more comprehensive studies had been done in the 1950's (Hécaen and co-workers; McFie and co-workers) that the right-hemisphere dominance for certain types of perception was more generally accepted. The investigation of patients who underwent a commissurotomy has contributed to our present knowledge of hemispheric asymmetry (Sperry and co-workers).

Several types of spatial disorientation can be distinguished clinically: (a) defective localization of stimuli in external space, (b) defective short-term memory for spatial location, (c) defective route finding, (d) reading and counting disabilities, (e) defective topographical memory, (f) visual-constructive disabilities, and (g) simultaneous agnosia.

In older articles these disabilities were attributed to bilateral temporo-parieto-occipital lesions. But spatial disorientation has also been described in patients with unilateral posterior lesions on either side and even in frontal lobe disease. Later more comprehensive studies were done, because analysis of single cases is susceptible to several sources of error. In many of these studies it was concluded that the right posterior region plays an important role in subserving visual and tactile localization (Hannay, Varney and Benton; De Renzi). Yet, some authors only stressed the relevance of the antero-posterior dimension but not the hemispheric asymmetry (Semmes et al.; Butters and Barton). 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.

In other words, spatial perception has usually been studied at a rather complex level, one at which it is difficult to differentiate spatial from other factors. De Renzi et al. developed a relatively simple test in which the position of a rod in space has to be reproduced.
I have chosen this rod orientation test to study (a) the role of different areas of the brain in the perception of spatial information, (b) the extent and speed of recovery of disturbances of spatial perception and (c) the development of spatial perception in children.

In the first part of Chapter II I have tried to establish which parts of the brain are the most important for spatial perception. Two versions of the rod orientation test were given, one with the aid of vision and the other only by touch. The subjects were 40 normal controls, 35 patients with a right-hemisphere lesion and 33 patients with a left-hemisphere lesion. All brain infarcts were not associated with a midline shift as assessed by CT scanning. In the control group it appeared that age and intelligence did not influence the test results. A significant difference was found between the mean error rates of patients with a pre- and post-rolandic extension of the lesion or an exclusively post-rolandic lesion of the right hemisphere and the other brain-damaged subgroups or controls. In patients with a pre- and post-rolandic extension of the lesion there was a difference in performance between the visual and the tactile modality.
The performance on the tactile modality was significantly worse. Patients with an exclusively post-rolandic lesion had high mean error rates in both the visual and the tactile part of the test, so that in this group the performance was independent of the modality tested.

In the patient group with an exclusively post-rolandic lesion of the right hemisphere the CT-scan pictures were drawn at a normal scale by means of a pantograph. It turned out that all these lesions had one area in common, in the white matter of the occipital lobe.

In the second part of this chapter a method for assessing the size of the lesion is described and used to investigate a possible relationship between the size of the lesion and the test performance in patients with disturbances of spatial perception. It turned out that there was no relation between the size of the lesion and the test performance two weeks after a stroke.

Chapter III first gives a review of various explanations for the recovery of function after brain lesions in general. In contrast to the extensive literature on recovery and treatment of aphasia, there is very little information available about recovery of spatial functions.

Twenty-four patients who had shown disturbances of spatial perception two weeks after their stroke, were re-tested after one year to assess the *extent* of recovery. They all had an infarct in the posterior region of the right

hemisphere. Most patients showed a recovery of spatial functions, although incompletely. Four of the twelve patients with a pre- and post-rolandic extension of the lesion showed only a slight recovery and these four patients had the highest mean error rates and a tactile extinction phenomenon at the initial examination. The only patient with an exclusively post-rolandic lesion who had recovered only slightly, still had an impaired memory for the location of objects and defective route finding. These five patients had the largest lesions ranging from 74 to 92 cm^3 (mean 85 cm^3).

To determine the *speed* of recovery on the rod orientation test, 16 other patients with severe spatial defects on testing after two weeks were re-tested 6 weeks, 3 months, 6 months and if necessary 1 year after the stroke. Eight patients had a pre- and post-rolandic extension of the lesion and eight had an exclusively post-rolandic lesion of the right hemisphere.
Furthermore, the test results of the rod orientation test were compared with the results of the line orientation test (Benton et al.). For all patients a recovery profile was drawn up. All except one showed recovery of spatial functions. Six months after the stroke there was no significant difference between the test results of these patients and those of the control group.
Only 3 of the 16 patients showed a disturbed performance on the line orientation test, which result indicates a low sensitivity of the line orientation test.
The volumes of the lesions were calculated and there was a gross relation between the size of the lesion and the duration of the recovery period.
The lesions of the patients with an exclusively post-rolandic lesion had an area in common which overlapped the common area already found (Chapter II).

Chapter IV describes a study about the development of spatial perception in children without evidence or history of brain disease. Thirty children in three different age groups were tested, namely 4-6 years, 7-9 years and 10-13 years, each group consisting of ten children. From the test results it appeared that the children in the youngest age group had the highest mean error rates. The error rates decreased with increasing age and the performance of the children in the age group 10-13 years was similar to that of the adult control group for both parts of the test.

In Chapter V test results of an eight-year-old child with an agenesia of the corpus callosum are discussed. The deficient performance of the right hand in the tactile part of the test is compatible with a disturbed interhemispheric transfer.

SAMENVATTING

Dit proefschrift behelst een klinisch onderzoek over ruimtelijke perceptiestoornissen.

Hoofdstuk I geeft een kort overzicht over "hemispheric asymmetry". Gedurende lange tijd werd gedacht dat grote delen van de rechter hemisfeer geen specifieke functie hadden. Jackson echter meende dat het achterste deel van de rechter hemisfeer een belangrijke rol speelt bij het uitvoeren van visueel-ruimtelijke taken. Slechts enkele van zijn tijdgenoten waren het met hem eens, maar later benadrukten o.a. Rieger, Babinski en Dide ook het belang van dit deel van de rechter hemisfeer bij ruimtelijke taken. De dominantie van de rechter hemisfeer voor bepaalde soorten waarnemingen werd echter pas in bredere kring geaccepteerd nadat uitgebreidere onderzoekingen waren gedaan (Hécaen en medewerkers; McFie en medewerkers). Onderzoek van patiënten met een doorsnijding van het corpus callosum heeft veel bijgedragen tot de kennis over lateralisatie (Sperry en medewerkers).

Klinisch kunnen een aantal typen ruimtelijke oriëntatiestoornissen worden onderscheiden: (a) een onvermogen om voorwerpen in de ruimte te localiseren en om de grootte ervan en de afstand tot de waarnemer te schatten, (b) een verminderd geheugen voor de localisatie van voorwerpen, (c) een onvermogen om een route te volgen van de ene plaats naar de andere, (d) lees- en rekenstoornissen, (e) een gestoord topografisch geheugen, (f) visueel-constructieve stoornissen en (g) simultaan-agnosie.

Deze stoornissen zouden het gevolg zijn van dubbelzijdige temporo-pariëto-occipitale laesies. Er zijn echter ook ruimtelijke oriëntatiestoornissen beschreven bij patiënten met enkelzijdige posterior-laesies, hetzij links hetzij rechts, en zelfs bij patiënten met een frontaal letsel.

In uitgebreidere onderzoekingen die later gedaan zijn, werd geconcludeerd dat het rechter posterior-gebied een belangrijke rol speelt bij de uitvoering van visuele en tactiele taken (Hannay, Varney en Benton; De Renzi). Sommige auteurs benadrukten echter nog steeds het belang van de localisatie van de laesie binnen één hemisfeer, maar niet het verschil tussen beide hemisferen (Semmes en medewerkers; Butters en Barton). Het verschil in

resultaten dat bij deze onderzoekingen werd gevonden kan waarschijnlijk teruggevoerd worden op een verschil in de gebruikte tests. De meeste tests om ruimtelijke vermogens te onderzoeken zijn òf subtests van intelligentietests òf werden ontworpen om goed gedefinieerde klinische syndromen te onderzoeken. Met andere woorden, ruimtelijke perceptie werd op een te complex niveau onderzocht, waardoor het moeilijk was een onderscheid te maken tussen de invloed van ruimtelijke en andere factoren.
De Renzi en medewerkers ontwikkelden een betrekkelijk eenvoudige test, waarbij de stand van een staaf in de ruimte nagebootst moet worden. Deze "rod orientation test" werd gebruikt om na te gaan: (a) het belang van verschillende delen van de hersenen voor de perceptie van ruimtelijke informatie, (b) de mate en snelheid van herstel van ruimtelijke perceptiestoornissen en (c) de ontwikkeling van ruimtelijke perceptie bij kinderen.

In het eerste deel van hoofdstuk II heb ik geprobeerd vast te stellen welke delen van de hersenen het belangrijkst zijn voor de perceptie van ruimtelijke informatie. Er werden twee modaliteiten getest, de visuele en de tactiele. Onderzocht werden: 40 controle-patiënten, 35 patiënten met een letsel in de rechter hemisfeer en 33 patiënten met een letsel in de linker hemisfeer. Alle patiënten met hersenletsel hadden een infarct zonder verdringing van de mediane structuren, zoals werd aangetoond d.m.v. een CT-scan. Leeftijd en intelligentie bleken geen invloed te hebben op de testresultaten in de controlegroep. Er werd een significant verschil gevonden tussen de gemiddelde foutenscore van patiënten met een pre- en post-rolandische uitbreiding van de laesie of een zuiver post-rolandische laesie in de rechter hemisfeer en de andere hersenbeschadigde subgroepen of de controles.
In de groep met een pre- en post-rolandische uitbreiding van de laesie was er een significant verschil tussen de testresultaten voor beide onderzochte modaliteiten ten nadele van de tactiele. Patiënten met een zuiver post-rolandische laesie hadden een hoge gemiddelde foutenscore, zowel voor het visuele als het tactiele deel van de test. In deze groep was het testresultaat dus onafhankelijk van de onderzochte modaliteit.

De laesies op de CT-scan van patiënten met een zuiver post-rolandische laesie werden d.m.v. een pantograaf tot normale grootte teruggebracht. Al deze laesies bleken een occipitaal gelegen subcorticaal gebied gemeenschappelijk te hebben.

In het tweede deel van dit hoofdstuk wordt een methode beschreven om het volume van de laesie te berekenen. Deze methode werd gebruikt om een mogelijk verband na te gaan tussen het volume van de laesie en de testresultaten van patiënten met ruimtelijke perceptiestoornissen. Er bleek geen relatie te bestaan tussen het volume van de laesie en de testresultaten twee weken na een herseninfarct.

In hoofdstuk III worden eerst verschillende verklaringen voor het herstel van functies na een hersenletsel in het algemeen besproken. Verder worden een aantal onderzoekingen aangehaald die gedaan zijn over het herstel van functies.
In tegenstelling tot de uitgebreide literatuur over het herstel en de behandeling van afasieën zijn er weinig gegevens beschikbaar over het herstel van ruimtelijke functies.

Vierentwintig patiënten die twee weken na hun herseninfarct een ruimtelijke perceptiestoornis hadden, werden na één jaar opnieuw getest om de *mate* van herstel na te gaan. Alle patiënten hadden een infarct in het achterste deel van de rechter hemisfeer. Bij de meeste patiënten was er, zij het onvolledig, een herstel van ruimtelijke functies. Vier van de patiënten met een pre- en post-rolandische uitbreiding van de laesie toonden slechts een gering herstel. Deze vier patiënten hadden bij het eerste onderzoek de hoogste gemiddelde foutenscore en een tactiel extinctiefenomeen. Slechts één van de patiënten met een zuiver post-rolandische laesie herstelde nauwelijks. De vijf patiënten met slechts een gering herstel hadden de grootste laesies, variërend van 74 tot 92 cm^3 (gemiddeld 85 cm^3).

Om de *snelheid* van het herstel na te gaan bij onderzoek met de "rod orientation test", werden 16 patiënten die twee weken na een herseninfarct een ruimtelijke perceptiestoornis bleken te hebben, na 6 weken, 3 maanden, 6 maanden en zo nodig 1 jaar opnieuw onderzocht. Acht van hen hadden een pre- en post-rolandische uitbreiding van de laesie en de overige acht een zuiver post-rolandische laesie in de rechter hemisfeer.
Bovendien werden de resultaten van de "rod orientation test" vergeleken met die van de "line orientation test" (Benton en medewerkers). Met behulp van de scores werd er een herstelcurve gemaakt voor alle patiënten. Uitgezonderd één patiënt toonden allen een volledig herstel van ruimtelijke functies. Zes maanden na het herseninfarct was er geen significant verschil meer tussen de testresultaten van de onderzochte patiënten en die van de controlegroep.
Slechts bij drie van de 16 patiënten werden bij onderzoek met de "line orientation test" (Benton en medewerkers) aanwijzingen gevonden voor een ruimtelijke perceptiestoornis, hetgeen duidt op een geringe sensitiviteit van de "line orientation test".
Berekening van de volumina van de laesies en duur van het herstel toonde aan dat tussen beide een relatie bestaat.
De laesies van de patiënten met een zuiver post-rolandische laesie in de rechter hemisfeer hadden een occipitaal gelegen subcorticaal gebied gemeenschappelijk, dat samenviel met het reeds eerder gevonden gebied (hoofdstuk II).

In hoofdstuk IV worden de resultaten besproken van een onderzoek over

de ontwikkeling van ruimtelijke perceptie bij kinderen, bij wie noch anamnestisch noch bij onderzoek aanwijzingen bestonden voor hersenletsel. Er werden 30 kinderen in drie verschillende leeftijdsgroepen onderzocht, te weten 4-6 jaar, 7-9 jaar en 10-13 jaar. Iedere groep bestond uit 10 kinderen. in de jongste leeftijdsgroep hadden de hoogste gemiddelde foutenscore. Naarmate de kinderen ouder werden, verbeterde de score en kinderen van 10-13 jaar behaalden hetzelfde resultaat als de controlegroep.

In hoofdstuk V worden de ziektegeschiedenis en de testresultaten besproken van een meisje met een corpus callosum-agenesie. Haar slechte prestaties bij gebruik van de rechter hand in het tactiele deel van de test passen bij een gestoorde overdracht van informatie tussen de linker en rechter hemisfeer.

REFERENCES

ALAJOUANINE, T. Les grandes activités du lobe occipital. Paris, Masson et Cie., 1960.

BABINSKI, J. Anosognosie. Rev. Neurol. 25, 365-367, 1918.

BADAL, J. Contribution à l'étude des cécités psychiques: alexie, agraphie, hémianopsie inférieure, trouble du sens de l'espace. Arch. Ophthal. (Paris) 8, 97-117, 1888.

BAIRSTOW, P.J. and J.I. LASZLO. Kinaethetic sensitivity to passive movements and its relationship to motor development and motor control. Develop. Med. Child Neurol. 23, 606-616, 1981.

BASSER, L.S. Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. Brain 85, 427-460, 1962.

BASSO, A., P. FAGLIONI and L.A. VIGNOLO. Etude controlée de la rééducation du langage dans l'aphasie: comparaison entre aphasiques traités et non-traités. Rev. Neurol. 131, 607-614, 1975.

BENDER, H. and R. JUNG. Abweichungen der subjectiven optischen Vertikalen und Horizontalen bei Gesunden und Hirnverletzten. Arch. f. Psychiat. 181, 193-212, 1948.

BENSON, D.F. and M.I. BARTON. Disturbances in constructional ability. Cortex 6, 19-46, 1970.

BENTON, A.L. The problem of cerebral dominance. Canad. Psychol. 6, 332-348, 1965.

BENTON, A.L. Constructional apraxia and the minor hemisphere. Confin. Neurol. (Basel) 27, 1-17, 1967.

BENTON, A.L. Historical Notes on Hemispheric Dominance. Arch. Neurol. 34, 127-129, 1977.

BENTON, A.L., N.R. VARNEY and K. de S. HAMSHER. Visuospatial Judgment. Arch. Neurol. 35, 364-367, 1978.

BOLLER, F. A behavioral evaluation of brain scan estimates of lesion size. Neurology 20, 852-859, 1970.

BOUILLAUD, J.B. Traité clinique et physiologique de l'encéphalite. Paris, J.B. Ballière, 1825.

BRAIN, W.R. Visual disorientation with special reference to lesions of the right cerebral hemisphere. Brain 64, 244-272, 1941.

BROCA, P. Remarques sur le siège de la faculté du langage articulé suivies d'une observation d'aphémie. Bull. Soc. Anat. (Paris) 6, 330-357, 1861.

BROCA, P. Localisation des fonctions cérébrales: Siège du langage articulé. Bull. Soc. Anthrop. (Paris) 4, 200-203, 1863.

BUTTERS, N. and M. BARTON. Effect of parietal lobe damage on the performance of reversible operations in space. Neuropsychologia 8, 205-215, 1970.

CAMPBELL, D.C. and J.M. OXBURY. Recovery from unilateral visuo-spatial neglect. Cortex 12, 303-312, 1976.

CARMON, A. and A.L. BENTON. Tactile perception of direction and number in patients with unilateral cerebral disease. Neurology 19, 525-532, 1969.

COLONNA, A. and P. FAGLIONI. The performance of hemisphere damaged patients on spatial intelligence tests. Cortex 2, 293-307, 1966.

CULTON, G.L. Spontaneous recovery from aphasia. J. Speech Hearing Res. 12, 825-832, 1969.

DIDE, M. Les désorientations temporo-spatiales et la prépondérance de l'hémisphère droit dans les agnoso-akinèsies proprioceptives. Encéphale 33, 276-294, 1938.

DONGEN, H.R. van and M.C.B. LOONEN. Factors related to the prognosis of acquired aphasia in children. Cortex 13, 131-136, 1977.

ETTLINGER, G., E. WARRINGTON and O.L. ZANGWILL. A further study of visual-spatial agnosia. Brain 80, 335-361, 1957.

FLOURENS, P. Recherches Expérimentales sur les Propriétés et les Fonctions du Système Nerveux dans les Animaux Vertebrés. Paris, Crevot, 1824.

FOERSTER, R. Über Rindenblindheid: Albrecht v. Graefes. Arch. Ophthal. 36, 94-108, 1890.

FONTENOT, D.J. and A.L. BENTON. Tactile perception of direction in relation to hemisphere locus of lesion. Neuropsychologia 9, 83-88, 1971.

FRANCO, L. and R.W. SPERRY. Hemisphere lateralization for cognitive processing of geometry. Neuropsychologia 15, 107-114, 1977.

GADO, M., J. HANAWAY and R. FRANK. Functional anatomy of the cerebral cortex by computed tomography. J. Comp. Ass. Tom. 3, 1-21, 1979.

GERSTMANN, J. Zur Symptomatologie der Herderkrankungen in der Uebergangsregion unteren parietal- und mittleren Okzipital-windung. Dtsch. Z. Nervenhlk. 116, 46-49, 1930.

HÁNNAY, H.J., N.R. VARNEY and A.L. BENTON. Visual localisation in patients with unilateral brain disease. J. Neurol. Neurosurg. Psychiat. 39, 307-313, 1976.

HÉCAEN, H. Clinical symptomatology in right and left hemisphere lesions. In: V.B. Mountcastle (ed.), Interhemispheric Relations and Cerebral Dominance, Baltimore, Johns Hopkins, 1962.

HÉCAEN, H. Acquired aphasia in children and the autogenesis of hemispheric functional specialization. Brain and Language 3, 114-134, 1976.

HÉCAEN, H., J. AJURIAGUERRA and J. MASSONET. Les troubles visusconstructifs par lésions parieto-occipitales droites. Encéphale 40, 122-179, 1951.

HÉCAEN, H., W. PENFIELD, C. BERTRAND and R. MALMO. The syndrome of apractognosia due to lesions of the minor cerebral hemisphere. Arch. Neurol. Psychiat. 75, 400-434, 1956.

HOF, M.W. van. Development and recovery from brain damage. In: Maturation and Development: Biological and Psychological Perspectives. Clinics in Developmental Medicine No. 77, 1978. K.J. Connolly and H.R. Prechtl (eds.). Spastics International Medical Publications, London, Philadelphia, 1981.

HOLMES, G. Disturbances of visual orientation. Brit. J. Ophthal. 2, 449-468, 506-516, 1918.

JACKSON, J.H. Case of a large cerebral tumour without optic neuritis and with hemiplegia and imperception. R. Ophthalmol. Hosp. Rep. 8, 434-444, 1876.

JACKSON, J.H. On the nature of the duality of the brain. Reprinted Brain 38, 80-103, 1915.

JOHNSON, D. and C.R. ALMLI. Age, brain damage and performance. In S. Finger (ed.), Recovery from brain damage. New York, Plenum Press, 1978.

KENNARD, M.A. and W.S. McCULLOCH. Motor response to stimulation of cerebral cortex in absence of area 4 and 6 (macaca mulatta). J. Neurophysiol. 6, 181-190, 1943.

KERTESZ, A. and P. McCABE. Recovery patterns and prognosis in aphasia. Brain 100, 1-18, 1977.

KERTESZ, A., D. LESK and P. McCABE. Isotope localization of infarcts in aphasia. Arch. Neurol. 34, 590-601, 1977.

KERTESZ, A., W. HARLOCK and R. COATES. Computer tomographic localization, lesion size and prognosis in aphasia and non-verbal impairment. Brain and Language 8, 34-50, 1979.

KERTESZ, A. and S. DOBROWOLSKI. Right hemisphere deficits, lesion size and location. Journal of Clinical Neuropsychology 3, 283-299, 1981.

KLEIST, K. Die Hirnverletzungen in ihrer Bedeutung für die Localisation der Hirnfunctionen. Alg. Z. Psychiat. 74, 542-562, 1918.

KLEIST, K. Kriegsverletzungen des Gehirns in ihrer Bedeutung für die Hirnlocalisation und Hirnpathologie. Handbuch der ärztlichen Erfahrungen im Weltkriege 1914/1918. Vol. 4, 1934.

KOHLMEYER, K. Aphasia due to focal disorders of cerebral circulation: some aspects of localisation and of spontaneous recovery. Neurolinguistics 4. Recovery in Aphasics, Amsterdam, Swets and Zeitlinger BV, 1976.

KUFFLER, S.W., M.J. DENNIS and A.J. HARRIS. The development of chemosensitivity in extrasynaptic areas of the neuronal surface of the frog. Proceedings of the Royal Society of London, Series B 177, 555-563, 1971.

KUHL, D.E., M.E. PHELPS, A.P. KOWELL et al. Effects of stroke on local cerebral metabolism and perfusion: mapping by emission computed tomography of ^{18}FDG and $^{13}NH_3$. Ann. Neurol. 8, 47-60, 1980.

LASHLEY, K.S. Factors limiting recovery after central nervous lesions. J. Nerv. Mental Diseases 88, 733-755, 1938.

LAWSON, I.R. Visual-spatial neglect in lesions of the right cerebral hemisphere: a study in recovery. Neurology 12, 23-33, 1962.

LENZ, H. Raumsinnstörungen bei Hirnverletzungen. Dtsch. Z. Nervenheilk. 157, 22-64, 1944.

LIEPMANN, H. Das Krankheitsbild der Apraxie. Mschr. Psychiat. Neurol. 8, 15-44, 102-132, 182-197, 1900.

LIU, C.N. and W.W. CHAMBERS. Intraspinal sprouting of dorsal root axons. Arch. Neurol. (Chicago) 79, 46-61, 1958.

MAZZOCCHI, F. and L.A. VIGNOLO. Computer assisted tomography in neuropsychological research: A simple procedure for lesion mapping. Cortex 14, 136-144, 1978.

McFIE, J., M.F. PIERCY and O.L. ZANGWILL. Visual spatial agnosia associated with lesions of the right cerebral hemisphere. Brain 73, 167-190, 1950.

McFIE, J. and O.L. ZANGWILL. Visual constructive disabilities associated with lesions of the left cerebral hemisphere. Brain 83, 243-260, 1960.

METTER, C.J., C.G. WASTERLAIN, D.E. KUHL, W.R. HANSON and M.E. PHELPS. ^{18}FDG Positron Emission Computed tomography in a study of aphasia. Ann. Neurol. 10, 173-183, 1981.

MEYER, O. Ein- und doppelseitige homonyme Hemianopsie mit Orientierungsstörungen. Mschr. Psychiat. Neurol. 8, 440-456, 1900.

NAESER, M.A. and R.W. HAYWARD. Lesion localisation in aphasia with cranial computed tomography and the Boston Diagnostic Aphasia Exam. Neurology 28, 545-552, 1978.

PATERSON, A. and O.L. ZANGWILL. Disorders of visual space perception associated with lesions of the right cerebral hemisphere. Brain 67, 331-358, 1944.

POURFOUR DU PETIT, F. (1710). Lettres d'un médecin des hôpitaux; la première lettre, contient un nouveau système du cerveau. Namur, Charles Gerard Albert. Cited by L.C. McHenry, Garrison's history of Neurology, Springfield, Illinois, C.C. Thomas, p. 120, 1969.

RAVEN, J.C. Progressive matrices sets I and II. Crichton Royal Dumfries, 1972.

DE RENZI, E. Disorders of space, exploration and cognition. John Wiley, & Sons, 1982.

DE RENZI, E., P. FAGLIONI and G. SCOTTI. Judgment of spatial orientation in patients with focal brain damage. J. Neurol. Neurosurg. Psychiat. 34, 489-495, 1971.

RIEGER, C. Ueber Apparate in dem Hirn. Arb. Psychiat. Klin. Wuerzburg 5, 176-197, 1909.

SCHNEIDER, G.E. and S.R. JHAVERI. Neuroanatomical correlates of spared or altered function after brain lesions in the newborn hamster. In: D.G. Stein, J.J. Rosen, N. Butters (eds.), Plasticity and Recovery of Function in the C.N.S., New York, Academic Press, 1974.

SEMMES, J., S. WEINSTEIN, L. GHENT and H.L. TEUBER. Spatial orientation in man after cerebral injury. Analyses by locus of lesion. J. Psychol. 39, 227-244, 1955.

SMITH, A., R. CHAMOUX, J. LERI, R. LONDON and A. MURASKI. Diagnosis, intelligence and rehabilitation of chronic aphasics. Finn Arbor: University of Michigan, Department of Physical Medicine and Rehabilitation, 1972.

SMITH, B.H. and G.W. KREUTZBERG. 'Neuron-target cell interactions'. Neurosciences Research Program Bulletin 14, 217-411, 1976.

SPERRY, R.W., M.S. GAZZANIGA and J.E. BOGEN. Interhemispheric relationships: the neocortical commissures; syndromes of hemisphere disconnection. In: Handbook of Clinical Neurology, Vol. 4, R.J. Vinken and G.W. Bruyn (eds.), Amsterdam, North Holland, 1969.

TENG, E.L. and R.W. SPERRY. Interhemispheric rivalry during simultaneous bilateral task presentation in commissurotomized patients. Cortex 10, 111-120, 1974.

TEUBER, H.L. and M. MISHKIN. Judgment of visual and postural vertical after brain injury. J. Psychol. 38, 161-175, 1954.

TZAVARAS, A. and H. HÉCAEN. Etude des coordinées visuelles subjectives au cours des lésions corticales unilatérales. Rev. Neurol. 125, 458-461, 1971.

ZAIDEL, E., D.W. ZAIDEL and R. SPERRY. Left and right intelligence: case studies of Raven's Progressive Matrices following brain bisection and hemidecortication. Cortex, 17, 167-186, 1981.

ACKNOWLEDGEMENTS

I am grateful for the generous help of many who have contributed towards the completion of these studies.

Prof. Dr. A. Staal always showed much interest and he encouraged me to complete these studies. His suggestions have contributed much to the final form of this thesis.

Dr. J. van Gijn gave much valuable advice on the investigation, on the preparation of the manuscript and on the improvement of the English.

Prof. Dr. H. van Crevel and Prof. Dr. M.W. van Hof offered constructive criticism during the final stage of this work.

Drs. P.A.T. Carbaat provided the facilities for the examination of many of the patients described in Chapter III, at the 'Gemeenteziekenhuis' in Dordrecht.

W.C.J. Moolenaar gave advice on statistics.

J.A. Meerwaldt designed the cover.

C. Goedegebuur made the apparatus used in the rod orientation test.

Mrs. J. Doornbosch-Konijn typed the manuscript conscientiously and gave many suggestions for the use of the English.

The illustrations were done by the Audiovisual Department of the Erasmus University Rotterdam.

CURRICULUM VITAE

De schrijver van dit proefschrift werd op 24 december 1946 te Rotterdam geboren. Hij bezocht het Rotterdamsch Lyceum te Rotterdam, waar hij in 1967 het eindexamen HBS B aflegde. Hij studeerde geneeskunde aan de Erasmus Universiteit te Rotterdam en werd in 1974 tot arts bevorderd.

Zijn opleiding tot neuroloog vond plaats in het Academisch Ziekenhuis te Rotterdam (Prof. Dr. A. Staal) en in het Deltaziekenhuis te Poortugaal (Dr. M.H. Cohen Stuart) van begin 1975 tot begin 1979.

Sinds 1 april 1979 is hij als staflid verbonden aan de afdeling neurologie van het Academisch Ziekenhuis te Rotterdam.