Recognition and imitation of pantomimed motor acts after unilateral parietal and premotor lesions: a perspective on apraxia

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Abstract

We compared gesture comprehension and imitation in patients with lesions in the left parietal lobe (LPAR, $n = 5$) and premotor cortex/supplementary motor area (LPMA, $n = 8$) in patients with damage to the right parietal lobe (RPAR, $n = 6$) and right premotor/supplementary motor area (RPMA, $n = 6$) and in 16 non-brain damaged control subjects. Three patients with left parietal lobe damage had aphasia. Subjects were shown 136 meaningful pantomimed motor acts on a videoscreen and were asked to identify the movements and to imitate the motor acts from memory with their ipsilesional and contralesional hand or with both hands simultaneously. Motor tasks included gestures without object use (e.g. to salute, to wave) pantomimed imitation of gestures on one's own body (e.g. to comb one's hair) and pantomimed imitation of motor acts which imply tool use to an object in extrapersonal space (e.g. to hammer a nail). Videotaped test performance was analysed by two independent raters; errors were classified as spatial errors, body part as object, parapraxic performance and non-identifiable movements. In addition, action discrimination was tested by evaluating whether a complex motor sequence was correctly performed. Results indicate that LPAR patients were most severely disturbed when imitation performance was assessed. Interestingly, LPAR patients were worse when imitating gestures on their own bodies than imitating movements with reference to an external object use with most pronounced deficits in the spatial domain. In contrast to imitation, comprehension was not or only slightly disturbed and no clear correlation was found between the severity of imitation deficits and gesture comprehension. Moreover, although the three patients with aphasia imitated the movements more poorly than non-aphasic LPAR patients, the severity of comprehension errors did not differ. Whereas unimanual imitating performance and gesture comprehension of PMA patients did not differ significantly from control subjects, bimanual tasks were severely disturbed, in particular when executing different movements simultaneously with the right and left hands. © 2000 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Since Liepmann's [44,45] first description, one of the pertinent issues in apraxia research is the question whether not only the production but also the recognition and understanding of motor acts is disturbed. According to Liepmann [45,46] model apraxic symptoms can either appear consequent to disturbances of the mental representation of movements, or of the actual carrying out of movements, or of the translation of the mental representation into movement production. It was suggested that apraxic disturbances may be traced back to an impairment of representation of the form or the meaning of the particular action [4]. The interaction between cognitive representation and production has been the focus of critical discussions [25,26,40,42,57,58]. Jeannerod and Decety [42] came to the conclusion that the motor impairments observed in apraxic patients result from a specific alteration in their ability to mentally evoke actions, or to use stored

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motor representations for forming mental images of actions.

In a series of experiments Heilman et al. [32–35] and Rothi et al. [61,62] examined pantomime discrimination and comprehension in patients with ideomotor apraxia. Heilman et al. [32] investigated whether patients with posterior dominant lesions that include the parietal lobe differ from patients with anterior lesions in their ability to discriminate between correctly and incorrectly performed motor acts. Twenty subjects were classified into four groups, according to the locus of the lesion (anterior versus posterior) and whether or not they were suffering from ideomotor apraxia. Results indicate that apraxic patients with posterior lesions have more difficulties discriminating between correctly and incorrectly performed acts than apraxics with anterior lesions. However, the authors were unable to identify the exact localisation of the lesions and some of their patients did not even have a CT scan. Rothi et al. [61] reported that apraxic patients with posterior lesions had difficulties in comprehending the meaning of pantomimes using a non-verbal paradigm. In contrast, Heilman [34] described three patients who could not perform meaningful actions on verbal command, but who were unimpaired in picking out the correct act from several alternatives performed by the experimenter. These findings [34] are consistent with the results by Roy and Square [63], who postulated a dissociation between conceptual and production components. The findings are further supported by Rapcsak et al. [57] who described a patient with bilateral damage to the posterior parietal lobe. The female patient was unable to pantomime simple movements with either the left or the right hand. She also showed deficits in the usage of real tools, albeit with some improvement over her performance on imitation. However, the patient showed no problems in the interpretation and discrimination of presented pantomimes of actions. The authors interpreted the results as empirical proof of a dichotomy between the mental comprehension of an action and its motor realisation.

Here we addressed the question whether motor impairments observed in apraxic patients are associated with difficulties in the recognition and understanding of motor acts. For this purpose, the comprehension and imitation of gestures was thoroughly examined by a video test set comprising 136 representational movements that had to be identified and imitated. It was argued that in the case of a common disturbance of comprehension and generation of motor behaviour, the disorder would affect an integrative sensorimotor transformation process. Alternatively, a selective impairment of movement production would argue for a primary motor disorder. Such a view is supported by the results of kinematic movement analysis, showing 'low level' executional errors of various movement components [9,36,55,56].

It was further examined whether the occurrence of recognition and production deficits depends on the types of movements examined and the site and the side of the lesion. For this purpose, the ability to recognise and to imitate different categories of pantomimed unimanual and bimanual motor acts was examined in patients with parietal damage and in normal control subjects. For comparison, patients with premotor damage or a lesion of the precentral motor strip were tested in order to evaluate the specificity of the parietal disturbances. This question was also of interest because parietal and premotor areas are densely interconnected.

It is important to emphasize that in the present study our apraxic patients with parietal damage presented with relatively dorsally located lesions barely involving the supramarginal gyrus in the inferior parietal lobe and temporal lobe structures. There is evidence of a differential involvement of the anterior and posterior parts of the parietal cortex in motor preparation and execution. Brain lesion studies of apraxic patients [1–3] and imaging studies [12,39] indicate that the inferior parietal cortex near the supramarginal gyrus may be more important than the superior parietal cortex in the correct selection of movements. Using positron emission tomography and measurement of regional cerebral blood flow (rCBF) as an index of cerebral activity, Deiber et al. [12] found that the posterior parietal cortex is more critically involved in the correct selection of movement on the basis of spatial attention, whereas the anterior parietal cortex was more activated in the use of visual information for motor preparation. Thus, we assume that our findings would be different if patients with ventrally located lesions were examined.

2. Subjects

All subjects gave their informed consent to participate in this study. The study was based on 11 patients with lesions in the parietal cortex (mean age 62 years, range: 55–71 years), 14 patients with lesions in dorsolateral premotor cortex or supplementary motor area (mean age 53 years, range: 22–76 years) and 16 healthy control subjects (mean age 57 years, range: 28–73 years). In addition, three patients with lesions of the precentral gyrus (PC) (mean age 51 years, range: 47–54 years) were examined. All patients were investigated during in-patient treatment at the Department of Neurology, Heinrich-Heine University of Düsseldorf. Control subjects were recruited from the neighbours of UH and MW and from the healthy spouses of the patients. All subjects gave informed consent before entering the study.

Five of the parietal lesions were in the left (LPAR) and six in the right hemisphere (RPAR); three patients with left parietal lobe damage had aphasia. There were

eight patients with damage to the left premotor/supplementary motor area (LPMA) and six with damage to the right premotor/supplementary motor area (RPMA); two precentral (PC) lesions were in the left hemisphere (LPC), and one in the right hemisphere (RPC). As in previous work [17,27], dorsolateral premotor lesions were allocated to the region anterior to the precentral sulcus, corresponding to area 6aα of Vogt's cytoarchitectonic map. This historical delineation of the premotor areas in the human was chosen for the following reasons: (i) the homologies of the human PMd and monkey's postarcuate (= precentral) premotor areas are still unclear [2,19,73]; (ii) the distinction of the anterior (area 6a) and posterior (area 4) lesions is virtually impossible; and (iii) so far, activation studies have also not clarified the issue [38].

All subjects were right-handed and naive as to the objectives of the study. Handedness was established by means of the TÜLUC [68] questionnaire. The handedness score was based on hand preference shown in 20 everyday situations, such as writing, teeth-brushing or catching a ball.

The average duration of the examination was 6 h and included a neuropsychological screening procedure (see below). The patients were tested on 2 consecutive days. On the first day, patients underwent a neuropsychological evaluation; on day 2, the experimental sessions on gesture comprehension and imitation were performed. In addition, a third testing day was needed for those patients who scored below average on the Reitan Indiana Aphasia Screening Test. These patients were examined on the full battery of the standardised German aphasia test, Aachener Aphasie Test (AAT); the additional test duration for the aphasic patients was 2–3 h. In order to avoid fatigue, all testing sessions were limited to 1 h periods, followed by 15 min of relaxation.

3. Methods

3.1. Neuropsychological screening procedure

Two formal intelligence tests were administered to all patients: (1) a verbal German intelligence scale (Mehrfach-Wortschatz Test B, MWT-B); and (2) a non-verbal IQ-test, the Raven Test (Standard Progressive Matrices). In addition, Trail Making Parts A and B, two tests for visual neglect (Albert's Test and Line Bisection) and a formal memory and attention test battery (Syndrom-Kurztest zur Erfassung von Aufmerksamkeits- und Gedächtnisstörungen, SKT) were used. Only patients who scored with an IQ > 89 in at least one of the IQ tests and who did not show evidence of neglect disturbances or other apparent deficits in attention, mnestic performance or cog-

nitive flexibility were included in this study [27].

All patients with left-sided brain damage were also subjected to the German version of the Reitan Indiana Aphasia Screening Test (translation by Jim Maxwell and Hendrik Niemann). Language functions were formally assessed with the full battery of the Aachener Aphasie Test (AAT) only in those three patients who scored below average on the Aphasia Screening Test. Thereafter, types of aphasic disturbances were classified according to the results obtained by the AAT [37].

3.2. Neurological disturbances as revealed by clinical examination

All patients underwent a thorough clinical examination. The major clinical findings are summarised in Fig. 1.

Ideomotor apraxia was examined by our extensive test battery. Tactile apraxia was diagnosed on the basis of the misconceived explorative finger and hand movements during object handling, manipulation and active touch [52]. Other movements of the hands and fingers, force and tapping were normal. In addition, disturbances of interlimb coordination frequently observed in patients with premotor lesions were examined, as described by Freund and Hummelsheim [17]. Subjects were asked to perform alternating windmill and cycling movements in both directions.

4. Action production: imitation of pantomimed motor acts

4.1. Classification of meaningful movements

Meaningful movements may be grouped into two broad categories, those directed to manipulate objects, which have been called transitive movements and those gestures meant to express ideas or feelings, which have been called intransitive movements [15]. According to Hecaen [30] and Hecaen and Rondot [31] intransitive movements should be further subdivided into symbolic gestures (e.g. to salute, to make the sign of the cross) and should be kept separate from expressive gestures (e.g. to threaten somebody) as they would reflect different psychological purposes subserved by discrete neuronal mechanisms. However, in our opinion the legitimacy of this distinction appears to be questionable if it applies to gestures produced out of context, as the performance is not sustained by the emotional mechanisms implicated in the real situation. In accordance with De Renzi [15], in the present study the category intransitive gestures thus consists of both symbolic and expressive gestures alike. In contrast, transitive movements were subdivided into two main categories: (i)

pantomimed movements centred around the body (ego-centric space); and (ii) movements made in extrapersonal space. This was done because there is evidence that movements in relation to one's own body-scheme can be more affected than movements made in extrapersonal space [11,23,51].

Initials	Age	Sex	Type of lesion	Location of lesion	Major clinical and neuropsychological deficits
A¹					
Left Parietal Lesions (LPAR)					
I.K.-R.	70 y	female	Ischemic infarct	Circumscribed lesion of the left postcentral gyrus	Severely impaired position sense and hypaesthesia of the right arm, tactile apraxia of the right hand
E.K.	66 y	male	Metastasis	Left paracentral lesion affecting the precentral and postcentral gyri invading the anterior posterior parietal area	Right-sided hemiparesis (mild), astereognosia and impairment of position sense of the right hand; amnesic aphasia
H.W.	71 y	female	Ischemic infarct	Left-sided lesion of the occipital lobe and the parieto-occipital junction	Hemianopia to the right, dyslexia, dyscalculia
L.R.	70 y	female	Ischemic infarct	Left temporo-parieto-occipital region and the inferior posterior parietal lobe	Severe global aphasia and minor brachio-facial hemiparesis of the right side
K.G.	56 y	male	Metastasis	Left precentral-parietal lesion	Minor right-sided hemiparesis
A²			Right Parietal Lesions (RPAR)		
W.B.	69 y	male	Brain abscess operated	Large lesion of the upper and lower right parietal lobe with involvement of the occipital and temporal lobe	Deficits in the perception of the spatial features of visual stimuli without neglect
H.-R.D.	55 y	male	Ischemic infarct	Right posterior parietal lesion affecting the inferior parietal lobe and the area around the postcentral sulcus	Hemihypaesthesia on the left with pronounced disturbance of position sense and two point discrimination; astereognosia and tactile apraxia of the left hand
S.R.	61 y	male	Ischemic infarct	Right superior parietal lesion affecting parts of the intraparietal sulcus	Tactile apraxia, disturbance of position sense and astereognosia of the left hand
W.H.	57 y	male	Intracerebral hematoma	Extensive lesion of the right temporal lobe, the inferior mesial parietal area and the temporo-parieto-occipital region	Psychomotor deficit, spatial incoordination, confabulations
W.S.	55 y	female	Oligodendro-glioma II operated	Extensive right paramedial lesion affecting the precentral gyrus, postcental gyrus and the anterior posterior parietal lobe	None
U.S.	58 y	female	Metastasis	Extensive lesion of the right pre- and postcentral gyrus and the upper and lower posterior parietal lobe	Hemiparesis and left-sided hemihypaesthesia

Fig. 1. Prominent clinical signs and outline of the lesions for the axial plane showing their largest extent. (A) Brain diagrams for each patient, on the top right the major sulci are shown. Arrows point to the central sulcus in each drawing. (B) Shows a summary of the extent and the etiology of the lesion and major clinical deficits for each patient. PCS, precentral sulcus; CS, central sulcus; PoCS, postcentral sulcus.

A₃   **Left Premotor Lesions (LPMA)**

H.H.	40 y	male	Astrocytoma II after laser therapy	Left lateral upper premotor lesion	Severe impairment in conditional learning; deficits in rhythm reproduction
S.F.	66 y	female	Glioblastoma operated	Large left prefrontal lesion near the midline	Deficits in cognitive flexibility
B.B.	40 y	female	Astrocytoma II	Circumscribed lesion of the left precentral area	Right-sided proximal hemiparesis and reciprocal coordination disorder
M.K.	61 y	male	Astrocytoma II	Left mesial precentral lesion affecting the white matter	Deficits in conditional motor learning
D.G.	71 y	female	Convexity meningeoma operated	Left mesial precental lesion invading deep into the adjacent white matter	Impaired bimanual coordination; deficits in rhythm reproduction; impairment in conditional motor learning
K.S	56 y	male	Metastasis	Circumscribed lesion anterior to the left precentral gyrus	Focal seizures of the right arm; disturbance of the fine motor control of the right hand with alteration of handwriting; slight right-sided hemiparesis
N.K.	43 y	male	Astrocytoma II with cystic degeneration	Disseminated extensive lesion of the left prefrontal cortex	Deficits in rhythm reproduction; disturbed motor learning
W.G.	76 y	female	Meningeoma relapse	Extensive lesion of the left frontal pole with a fingerform edema reaching to the precentral gyrus	Reciprocal coordination disorder

A⁴   **Right Premotor Lesions (RPMA)**

A.S.	66 y	female	Oligodendro-glioma	Circumscribed lesion of the right lateral premotor area	Limb-kinetic apraxia, deficits in conditional motor learning, impaired rhythm reproduction
B.S.	43 y	female	Frontal cyst after sinus thrombosis	Right upper prefrontal lesion	Increased tendon reflexes on the left with positive Babinski sign; distinct disturbance of rhythm reproduction and handwriting
M.J.	22 y	female	Astrocytoma II	Small lesion of the mesial upper frontal lobe	Focal seizures with aversion of head and eyes to the left; slight proximal weakness of the left arm
K.R.	57 y	male	Astrocytoma II	Extensive lesion of the right precentral and prefrontal area	Minor left sided hemiparesis with disturbed fine motor control in the left hand; reciprocal coordination disorder
O.W.	38 y	male	Ischemic infarct	Right medial artery infarction affecting the mesial precentral region, supplementary motor area and cingulate	Alien-hand syndrome of the left hand with slight impairment of dexterity; deficits in bimanual coordination and temporal organization of movements; impairment in conditional motor learning
B.H	56 y	male	Traumatic lesion	Small lesion of the right upper mesial frontal lobe	Minor slowing of alternating finger movements and disturbance of the dexterity and the ability to copy finger movements and reciprocal coordination disorder

Fig. 1. (*Continued*)

A5

				Left Precentral Lesions (LPC)	
I.R.-K.	54 y	female	Ischemic infarct	Small lacunar lesions at the basis of the left central, precentral and postcentral sulci	Right-sided hemiparesis, astereognosia, tactile apraxia
G.Z.	47 y	female	Ischemic infarct	Small lacunar lesion at the basis of the left central sulcus affecting the precental gyrus	Right-sided hemiparesis leg>arm, right-sided hypaesthesia and impaired position sense
				Right Precentral Lesions(RPC)	
M.F.	52 y	male	Ischemic infarct	Old lacune in the right centrum semiovale on the basis of the precentral gyrus	Moderate paresis of the right leg

B1

left parietal lesions

right parietal lesions

central lesions
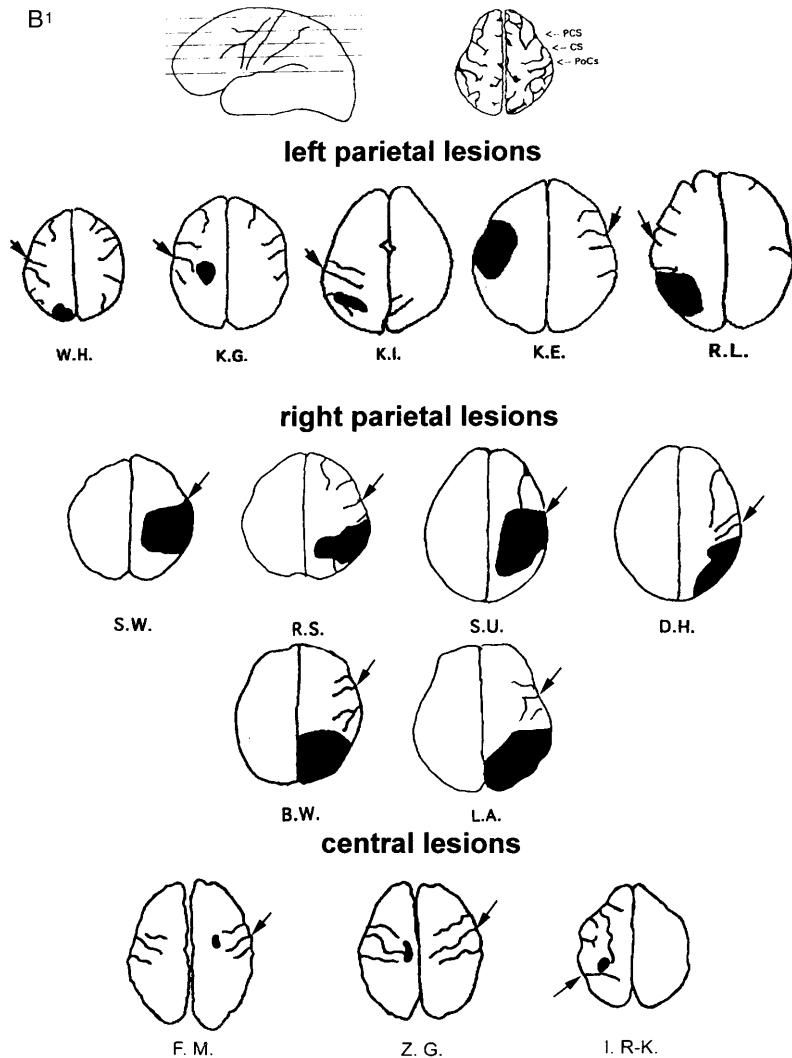
Fig. 1. (*Continued*)

Patients were asked to imitate meaningful actions as shown on a video screen. Actions included unimanual and bimanual motor tasks. In all conditions, performance was separately scored for the lesional and contralesional hand. In the unimanual motor tasks, the order of hand conditions was randomised across trials. Performance on all production tests was videotaped and was later scored independently by two examiners. It was assumed that a high inter-rater reliability provides a valid assessment of the subjects' motor performance.

The two raters were shown the same videotape and asked to make, for each movement imitation, a judge-

ment whether the action was correctly performed or not. If movement production was judged to be inadequate, the raters were asked to classify the types of errors as follows:

1. Spatial errors, including directional errors, were defined as an inconsistency between the movement path and the purpose of the action (e.g. if a pantomimed performance of 'drinking coffee out of a coffee mug' clearly showed that the object was initially moved away from the subject's mouth instead of approaching it) and/or a spatial misplacement at the target position (e.g. instead of toothbrushing pantomimed action appeared at the patient's cheek);
2. Body part as object (e.g. eating soup with a finger and not with a spoon);
3. Parapraxic errors were defined as a correctly executed movement which, however, was not appropriate for the target action (e.g. instead of cutting one's nails the pantomimed act was identified as brushing one's nails);
4. Non-identifiable and/or incomplete movements (e.g. the hand was lifted without aiming at a specific target).

The change-weighted correlation coefficient κ was applied for measuring reliability among raters. The following rating measures were documented: PAR, $\kappa = 0.89$; p(Z) < 0.01; PMA, $\kappa = 0.92$; p(Z) < 0.01; Ml, $\kappa = 0.95$; p(Z) < 0.01; CO, $\kappa = 0.98$; p(Z) < 0.01.

4.2. Unimanual motor tasks

4.2.1. Imitation of symbolic gestures

Ten symbolic and conventional gestures were successively presented on a video-screen. Items included waving, saluting and threatening. Each presentation lasted for 8 s. Immediately after each presentation, a coloured rainbow-pattern appeared on the screen and subjects were asked to imitate the action.

4.2.2. Imitation of pantomimed motor acts in relation to a person's own body

The actor on the videoscreen performed ten pantomimed movements in relation to his own body. Movements included hygienic routines, such as combing one's hair, toothbrushing, nail-clipping and shaving.
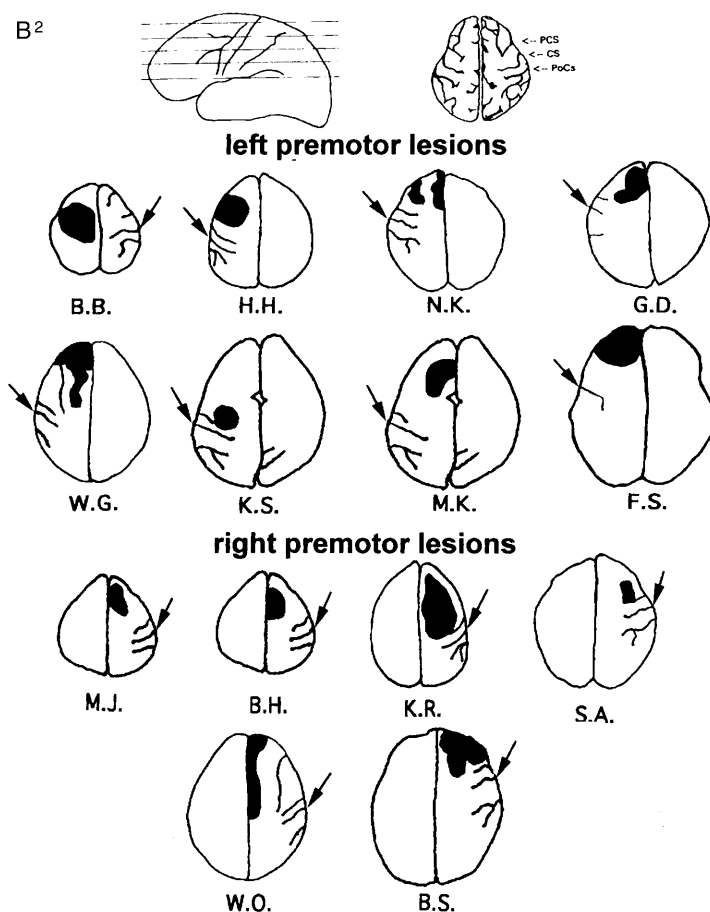

Fig. 1. (Continued)

4.3. Pantomiming the use of an object in extrapersonal space

Subjects were asked to imitate ten pantomimed actions with respect to the location of an imagined recipient of a tool's action in extrapersonal space. Performance included hammering, stamping and pouring fluid into a glass.

4.4. Bimanual motor tasks

Subjects were requested to imitate 20 meaningful movements using both hands simultaneously. Half of the actions presented consisted of two homogeneous components, whereby both hands performed an identical movement to an imagined object, such as wringing or piano-playing with symmetrical finger movements. The other ten actions consisted of heterogeneous movement patterns. In this condition, subjects were requested to use their left and right hand simultaneously and to perform two different pantomimed motor acts to an imagined object. For instance, when subjects pantomimed the action 'slicing bread' they were instructed to use the left hand for advancing the imagined loaf, while simultaneously turning an imagined handle with the other hand. Another example is 'cooking a pudding'. In this task, subjects were asked to pantomime pouring pudding powder into the boiling milk with their left hand, while simultaneously stirring it with the other hand.

4.5. Tool use

Subjects were asked to demonstrate the use of ten common household tools (e.g. screw-driver, pneumatic pump) presented as static pictures on a video-screen. Each item was presented for 8 s and appeared without context-specific cues on an identical neutral background (examiner's desk). As before, a coloured rainbow-pattern appeared on the screen immediately after each presentation. Subjects were asked to pantomime the movements associated with the appropriate tool functioning using his ipsilateral or contralateral hand.

Tool use was further evaluated with the tools provided as real items on the examiner's table. For this purpose, an additional set of ten common tools and the objects of the tools action were provided (e.g. a pair of scissors and paper). Only one tool and the relevant object were presented at a time. Subjects were asked to demonstrate the tool's action.

4.6. Completion of serial motor acts

Subjects were instructed to complete a series of motor acts. For this purpose, a series of ten complex motor sequences was presented on the video-screen.

For instance, it was shown how to pack various items into a suitcase before a journey. A series of items placed on the actor's table was put, one after each other, into the suitcase. The film stopped after the last item was inside the case. The patient was asked to demonstrate which movement(s) would appear next in the sequence. The minimum requirement for an answer to be scored correctly was pantomiming the movement necessary for closing the lid of the case. However, most subjects spontaneously continued the sequence task by adding the movements how to lock the case and to carry it out of the room.

5. Recognition and evaluation of motor acts

5.1. Recognition of meaningful pantomimed motor acts

5.1.1. Symbolic gestures
Subjects were shown ten symbolic gestures on a video-screen; each presentation lasted for 8 s. Subjects were asked to describe the meaning of the gestures and to indicate the context in which they appear (e.g. taking an oath at court, putting one's hands-up at school).

5.1.2. Recognition of pantomimed motor acts in relation to one's own body
Subjects were asked to identify and to describe ten meaningful pantomimed movements with respect to their own body, such as hair brushing, using a lipstick and nail colouring.

5.1.3. Recognition of pantomimed performance of the use of an object in extrapersonal space
Subjects were asked to identify and to describe the meaning of ten pantomimed actions with respect to an imagined recipient of a tool's action in extrapersonal space, such as writing, using a screw-driver or cutting a piece of paper.

5.2. Knowledge relevant to the serial organisation of action

5.2.1. Evaluation of motor sequences
Action evaluation was tested by asking the subjects to judge whether complex motor sequences presented on a video-screen were flawlessly or incorrectly performed. Movement sequences were presented within the usual context and with the aid of the appropriate objects and tools. For instance, the tasks preparing fried eggs or extracting the juice from an orange were videotaped in a kitchen with the use of the appropriate household equipment.

A total number of 20 different sequences was presented. A total of 50% of the serial motor acts were correctly performed; the other tasks were characterised

by either sequence errors ($n = 5$) or conceptually inappropriate tool use (e.g. to hammer a cork-screw into the cork). Subjects were asked to judge whether a given sequence was correctly or incorrectly performed. If they scored a performance as incorrect, they were asked to name and identify the sequence and performance errors.

5.2.2. Completion of serial action tasks

Knowledge of the serial position of motor acts was examined by asking the subjects to describe which movement comes next in an unfinished series of movements. Subjects were shown ten correctly performed but incomplete motor sequences, such as lighting a cigarette or using an electric coffee machine. They were asked to describe which movement is lacking to complete the motor act.

5.2.3. Conceptual knowledge of tool function

Subjects were asked to describe verbally the function of ten common household tools (e.g. hammer, can-opener) presented on a video-screen. Each tool appeared as a static item without context-specific cues on a neutral background and remained visible for 10 s.

Knowledge of tool function was further evaluated by presenting the subjects with an additional set of ten common tools. This time, tools were presented as real items on the examiner's table. Subjects were asked to visually inspect each tool and to describe the conceptual knowledge of actions required for appropriate tool use (e.g. in order to pound a nail one has to put the handle of the hammer in one's palm and to swing the hammer repeatedly through the air).

6. Results

6.1. Mapping of brain lesions

Mapping of brain lesions was carried out by an investigator who was completely unaware of the neuropsychological results. Structural lesions were outlined on CT or T1-weighted MR scans obtained around the time of the examination (CT scanner: General Electric CGR CE 1000; MR scanner: Siemens Magnetom 1.5 T, spin echo MR sequence, 600 ms repetition time, 15 ms echo time, two excitations, 6.0 mm slice thickness). Brain lesions were defined as parenchymal defects with gray scale values clearly different from those of the normal tissue. All CT and MR sections were obtained parallel to the canthomeatal line, thereby allowing anatomical mapping on corresponding templates derived from the atlas of Matsui and Hirano [49]. For this purpose, each CT or MR brain section containing the lesion was proportionally magnified in order to fit with the maximum anteroposterior and transverse dimensions of the brain atlas [69].

Fig. 1 shows the individual main clinical findings along with brain lesion outlines and a summary of the extent and the etiology of the lesion.

6.2. Action production: imitation of pantomimed motor acts

6.2.1. Unimanual motor performance

For each movement category, the performance of the ipsilesional and contralesional hand was evaluated. Fig. 2 gives a comparison of the subjects' ability to imitate symbolic gestures (A), to pantomime actions towards one's own body (B) and motor acts imitating tool use in extrapersonal space (C).

Two factor ANOVA was performed, in which patients were compared with controls on production versus comprehension (see below) tasks. In patients with left or right parietal lobe lesions the impairment on production was significantly higher to any impairment in recognition ($P < 0.01$).

Results indicate that ipsilateral and contralateral hand performance of the patients with left parietal lesions was worst followed by patients with right parietal lobe damage ($P < 0.01$). Among the various types of movements examined, pantomiming performance of meaningful symbolic gestures was the least affected category. Interestingly, patients with damage to the left parietal lobe were worse when imitating gestures aimed towards their own bodies as compared to symbolic gestures ($P < 0.01$) and movements with reference to an external object ($P < 0.01$). The fact that our control subjects as well as our patients with premotor or M1 lesions (see below) had no more difficulties in mastering the pantomimed movements made in extrapersonal space as compared to movements centred around the body makes it unlikely that the second task was more difficult.

Imitating performance of our patients with PMA lesions did not differ significantly from the controls or from patients with PC lesions in any of the movement categories examined (Fig. 2). Furthermore, the performance of our patients with PMA lesions did not differ from patients with supplementary motor (SMA) damage; due to the small sample size of the dorsolateral and medial premotor areas, the two groups were pooled together.

Inadequate movement production was classified according to the type of error made by the patients. The results are based on the total score obtained for unimanual pantomiming performance, whereby all three movement categories were pooled together. Fig. 3 shows the mean error rate for the various lesion groups. It can be seen that the most frequent types of errors that occurred in patients with lesions to the left parietal lobe were spatial errors which expressed themselves as a spatial misplacement at the target position and/or an
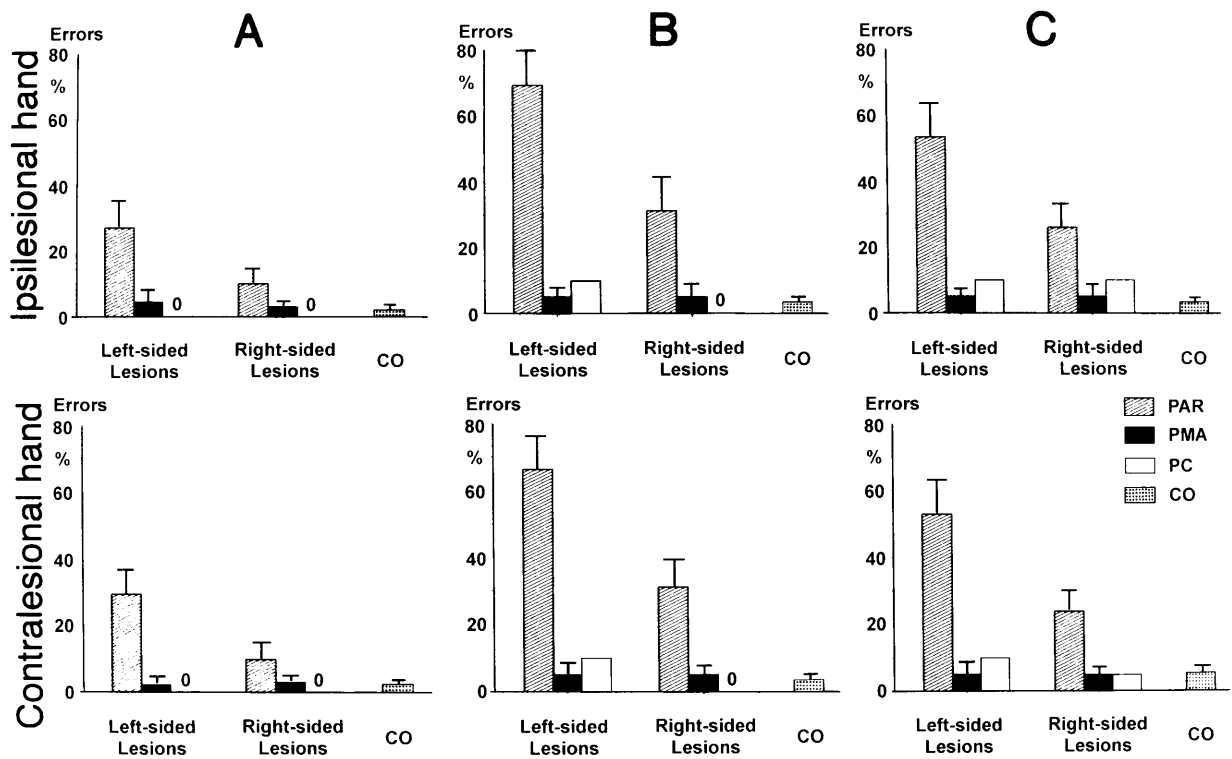
Fig. 2. Distribution of errors (in %) in pantomimed imitations using the ipsilesional (top) or contralesional hand (bottom). For the patient group, the percentage of errors refers to the use of the ipsilesional (top) or contralesional (bottom) hand as compared to the dominant hand of the normal volunteers. (A) Symbolic and meaningful gestures; (B) actions towards one's own body and (C) imagined tool use in extrapersonal space. PAR, parietal lobe; PMA, premotor areas; PC, precentral gyrus; CO, controls. Vertical lines give the S.D.

inconsistency between the movement path and the purpose of the action (Fig. 3).

None of our patients had any difficulties in spontaneously supplying the missing link in an incomplete sequence. Their performance was as follows (% correct): LPAR, 97; RPAR, 90; LPMA, 98; RPMA, 95, PC, 100; CO, 99.

6.2.2. Bimanual motor tasks

Although patients with PMA lesions were not impaired in pantomiming unimanual movements, they showed most pronounced deficits in imitating bimanual movements. Results indicate most pronounced deficits in the bimanual heterogeneous condition when executing different movements simultaneously with the right and left hands in patients with damage to the PMA (Mann–Whitney U-test, $P < 0.01$). For instance, when asked to pantomime the movement 'pouring pudding powder into the boiling milk with one hand while simultaneously stirring it with the other hand', subjects could correctly perform both movements separately, but were unable to carry out both movements simultaneously. Their execution of movement could be characterised as that mostly only one hand at a time was active (stirring with the right hand or pouring with the left). Sometimes stirring was carried out using both hands at the same time, followed by a bimanual pouring movement.

Patients with PMA lesions were further subdivided into subjects with preferential damage to the dorsolateral premotor areas and subjects with damage to the medial wall motor areas (supplementary motor area, cingulate motor areas). Results indicate that patients with lesions to the lateral premotor areas were less severely impaired in performing bimanual heterogeneous movements than patients with preferential damage to the medial wall motor areas (Mann–Whitney U-test for very small samples, $P < 0.01$) (Fig. 4). Results are based on the percentage of errors made by subjects with left or right sided damage to the lateral or medial wall motor areas.

6.2.3. Action recognition and evaluation

Only minor disturbances in comprehending the meaning of a pantomimed action were found among patients with parietal lesions; patients with PMA, SMA or PC damage were not impaired on these tasks. Fig. 5 gives a comparison of the subjects' ability in identifying symbolic gestures (A), pantomimed actions towards one's own body (B) and motor acts of an imagined tool's action in extrapersonal space (C).

A two factor ANOVA was performed to test whether comprehension deficits were significant in patients with unilateral parietal lesions as compared to control subjects. Results indicate a significant impairment for the
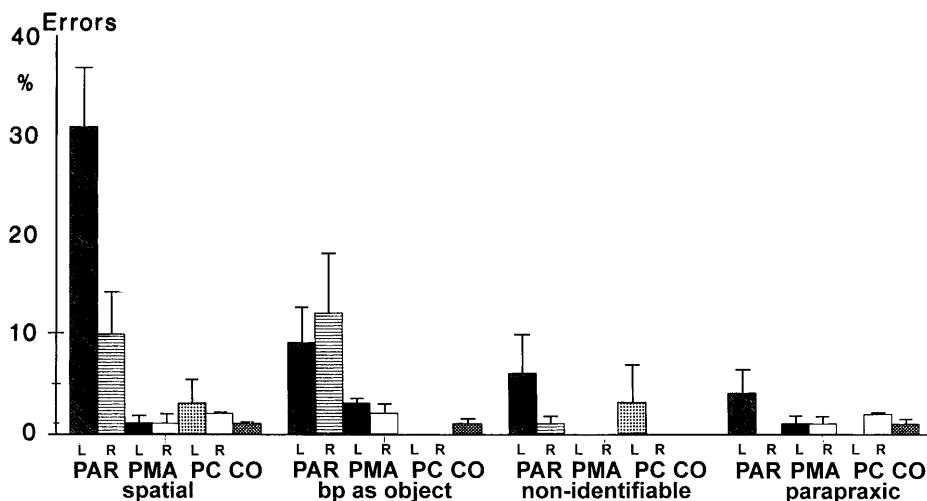
Fig. 3. Classification of errors in motor performance pantomimed on imitation. For this purpose, the three movement categories (see Fig. 2) were pooled together. PAR, parietal lobe; PMA, premotor areas; PC, precentral gyrus; CO controls; bp, body part as objects (see text). Vertical lines, S.D.

patients with left-sided damage ($P < 0.05$), the results of patients with a right-sided parietal lesion did not differ significantly from the control subjects.

Most interestingly, a heterogeneous picture of disturbances of recognition and production was found in the individual patients. It can be seen from Fig. 6 that some of the apraxic patients showed no deficits at all in the discrimination of actions at the mental level, although their production was severely disturbed. In spite of the preponderance of production errors, almost half of the patients also showed some comprehension errors (Fig. 6).

The severity of comprehension errors did not differ between aphasic and non-aphasic patients with parietal lobe damage, although the aphasic subjects performed the movements more poorly (Fig. 7).

None of our patients had difficulties in judging whether a given sequence was correctly or inadequately performed. They had no problems in naming and identifying sequence or performance errors. Their performance was as follows (% correct): LPAR, 97; RPAR, 96; LPMA, 98, RPMA, 97; PC and CO, 100. They also had no difficulties in identifying the missing link in an incomplete sequence. Their performance was as follows (% correct): LPAR, 97; RPAR, 92; PMA, PC and CO, 100.

6.2.4. Knowledge of tool use

None of the patients in this study showed any problems in the comprehension of the use of a tool or in its actual use. None of our subjects made any errors when knowledge of tool function was evaluated by presenting the subjects with a set of real tools. They identified all tools correctly (100%). When asked to use the tools, their performance was as follows (% correct): LPAR,

98; RPAR, 100; LPMA, 97; RPMA, PC and CO, 100.

The percentage of correct responses in identifying the function of common household tools presented as a static picture on a video screen was as follows: LPAR, 100, RPAR 98; PMA, PC and CO, 100. However, when asked to pantomime the use of the tool shown on the videoscreen, thus producing the relevant action without the tool being present, patients with parietal lesions were significantly impaired; the percentage of correct responses was as follows: LPAR, 66 ($P > 0.01$); RPAR, 88 ($P > 00.5$); LPMA, 94; RPMA, 95; PC and CO, 98.
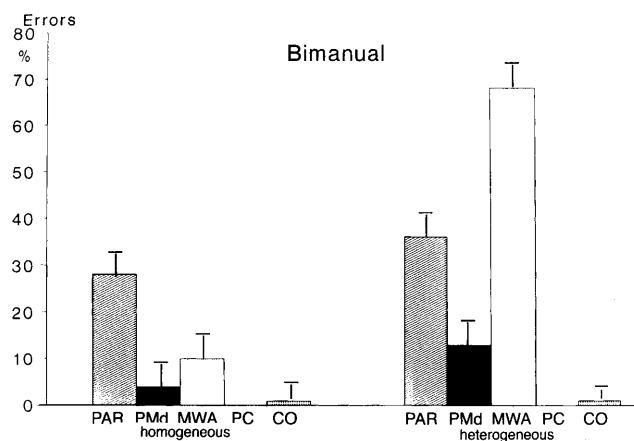

Fig. 4. Percentage of errors in pantomiming bimanual homogeneous and heterogeneous (see text) movements. For this purpose, lesions of the dorsolateral premotor areas (PMd) and the medial wall motor areas (MWA: SMA and cingulate motor areas) were analysed separately. PAR, parietal lobe; PC, precentral gyrus; CO, controls; vertical lines, S.D.
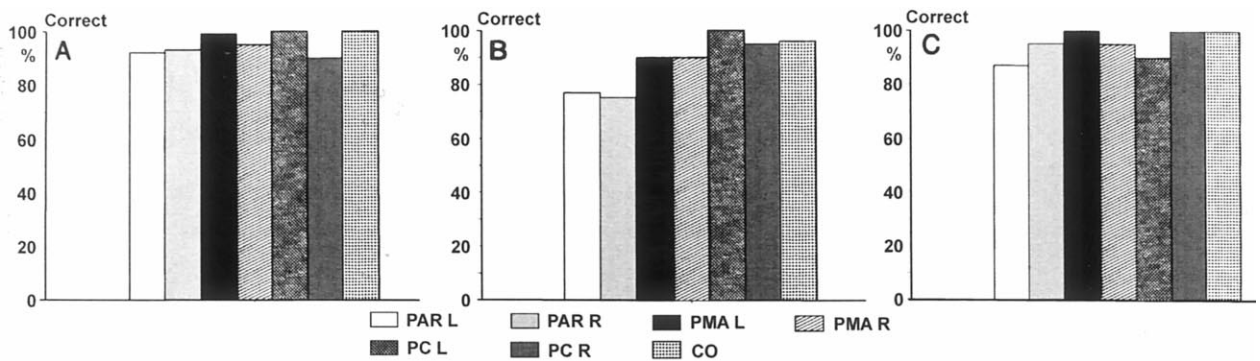
Fig. 5. Distribution of correct responses (in %) in the recognition of (A) symbolic and meaningful gestures; (B) movements towards one's own body and (C) imagined tool use in extrapersonal space. PAR, parietal lobe; PMA, premotor areas; PC, precentral gyrus; CO, controls; L, left-sided lesion; R, right-sided lesion. Vertical lines give the S.D.

7. Discussion

7.1. Parietal cortex

The basic finding of this study was that the comprehension of the symbolic and representational content of motor acts is not or only slightly disturbed in apraxic patients with parietal lobe lesions. The dissociation between the most pronounced disturbances in the production of movements and relatively preserved comprehension of the symbolic meaning is not in support of the hypothesis that apraxic disturbances are rooted in a common disturbance of representation of the form or the meaning of the particular action. Rather, the apraxic disturbance in our parietal patient group appears primarily as a motor production disorder, thus supporting a dissociation between conceptual and production components [57,63].

The results reflected a heterogeneous picture of disturbances of recognition and production in the individual patients. Some of the severely apraxic patients showed no deficit in the discrimination of actions at the mental level, although their production was severely disturbed. There was no correlation between the number of errors in gesture production and gesture comprehension. However, the present findings indicate an overall impairment for the patients with left-sided parietal damage in the recognition and understanding of motor acts.

The lack of a consistent gesture comprehension deficit is possibly due to its sampling bias. Our parietal patients presented with relatively dorsally located lesions barely involving temporal lobe structures. There is evidence from single cell recordings in monkeys [54] and from brain imaging studies in man [47,60] that action recognition may be mediated by temporal lobe structures

It is therefore not surprising that, in the present study, not only the comprehension of language, but also that of expressive motor behaviour was rather well

preserved. We assume that the data would be different when the patients were not selected for parietal or premotor lesions because the ventral stream functions, such as understanding of symbolic content, appear to be relatively intact.

The present study makes a further point. The severity of comprehension errors in our aphasic patients did not differ from our non-aphasic apraxics with parietal lobe damage. These findings make it unlikely that the involvement of aphasia in apraxia could be interpreted as a language-dependent disturbance in the recognition and understanding of motor acts. Corina et al. [10] reported left hemisphere specialisation of American sign and spoken language in deaf and hearing individuals. Their results suggest that left hemisphere specialisation derives from the linguistic nature of the movement: no evidence of hemispheric asymmetry was found for production of either symbolic or arbitrary gestures.

The only consistent focal deficit found in the understanding of pantomimed motor acts was that gestures related to one's own body were differentially affected with regard to severity as compared to production errors. Interestingly, a most pronounced production impairment was observed after left parietal lobe lesions, when subjects were asked to imitate gestures on their own bodies rather than imitating movements with reference to an external object. These findings may suggest that the basic deficit in these subjects concerns the ability to code and comprehend movements in relation to their body-scheme [11,23,51]. According to Goldenberg [23], faulty apprehension of spatial relationships between body parts manifests itself not only in imitation on oneself but also on a wooden mannikin. His findings can be adduced as evidence for an overall disturbance of representation of the human body-scheme independent of whether actions relate to the own body or to an external model. In the present investigation, we cannot distinguish between errors in spatial guidance of movement, errors in missing the target and spatial errors made from memory. It may be

argued that the difference between the relatively preserved extrapersonal space gestures and the body directed space movements might also be accounted for by the fact that posterior inferior parietal and intraparietal sulcus may play a greater role in extrapersonally guided movements.

The types of production errors classified into four categories (spatial, body part as object, parapraxic, non-identifiable) indicate that patients with parietal lobe lesions presented with most pronounced deficits in spatial parameters. This is in agreement with previous findings that movement production impairments in patients with ideomotor apraxia are characterised predominantly by spatiotemporal errors [9,36,55,61,62].

The majority of the apraxic disturbances following parietal lesions appeared in the spatial domain, such as deviating trajectories with correct directions and/or incorrect spatial positioning of the hand or finger on the goal-point of the executable movement. Clark et al. [9] reported errors in spatial exactness and a spatial-temporal disintegration in highly atomised movements in apraxic patients (slicing bread). These disturbances were also seen when movements were not carried out under verbal commands. Apraxic patients showed deficits in the plane of motion, the shape of the trajectory and in the coupling of hand speed and trajectory shape under various contextual cue conditions. In conjunction with the data presented here these results illustrate the wide range from elementary to complex motor dysfunctions.

Taken together, these findings support Heilman's [32] model of apraxia, which suggests that apraxia results from destruction of spatiotemporal representations of learned movements rather than from a disconnection between the receptive language areas in the left hemisphere and the motor cortices [21,22]. In order to perform a skilled learned act, one must place particular body parts in certain spatial positions in a specific order

at specific times. This implies that the nervous system stores knowledge of motor skills. When this knowledge is called into use, it is retrieved from motor memory rather than being constructed de novo [19,26]. According to Heilman's [32] model spatiotemporal representations of learned, skilled movements are stored in the parietal cortex which is thought to instruct the premotor cortex for the necessary movements.

A surprising feature of apraxic deficits is their context dependence so that movements, which cannot be made in accordance with verbal command or imitation, can be flawlessly produced in another context [14,15]. The findings suggest a context-specific interaction between cognitive representation and production. Jeannerod and Decety [42] came to the conclusion that motor impairments observed in apraxic patients may result from a specific alteration in their ability to mentally evoke actions, or to use stored motor representations for forming mental images of actions. Thus, the deficit arises when the patient shifts from a strategy where object-orientated actions are processed automatically, to when the content of these actions has to be explicitly represented.

Most of our parietal patients did show elementary and/or complex somatosensory disturbances, such as a stereognosis or graphanaesthesia. Such disturbances were not in focus of the present article but were analysed in an earlier study [52]. That study showed that damage of parietal sensory association cortex or its underlying white matter does not only produce elementary and perceptual/cognitive somatosensory deficits, but also an impairment of active touch and of the manipulative capacity of the hand required for object exploration. This combined deficit of object recognition and manipulation reveals the intricate interdependence between the sensory and motor processes and represents a unimodal somato-sensory or somato-motor dysfunction (tactile or manipulative apraxia [19,52]). The
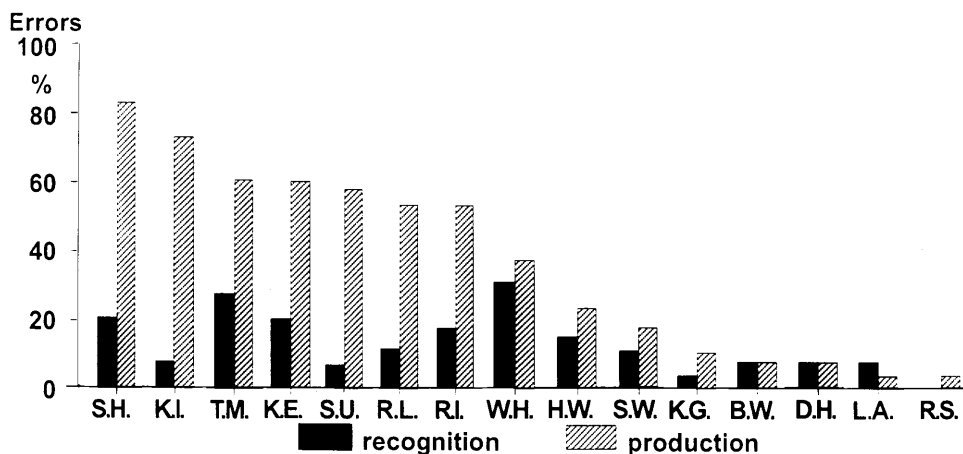

Fig. 6. Distribution of errors (in %) made by individual subjects with parietal lesions in the recognition and production of pantomimed motor acts. Patients are ordered according to production error rate.
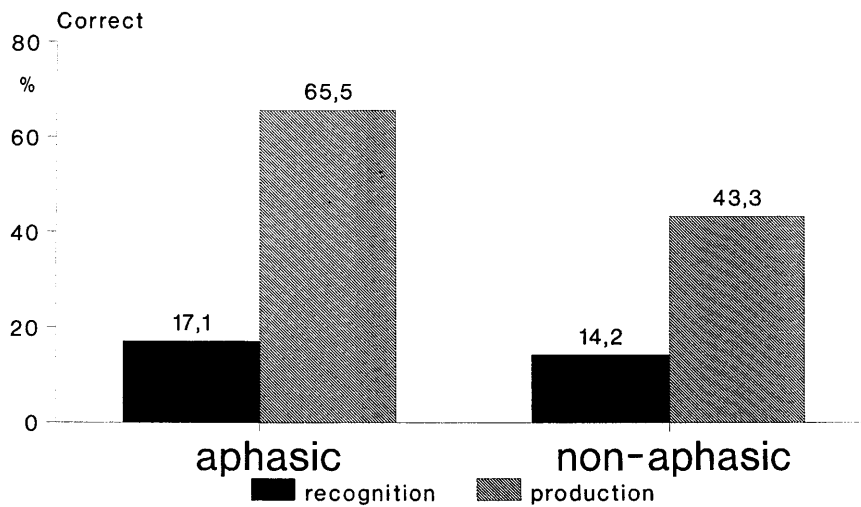
Fig. 7. Distribution of errors (in %) in the recognition and production of pantomimed movements in aphasic and non-aphasic patients.

apraxic visuomotor or speech behaviour seen in patients with damage to the parieto-occipital or temporal areas are other examples of apraxic disturbances bound to a particular modality. The ideational and ideomotor apraxias seen after lesions of the left inferior posterior parietal lobule are supramodal and affect both sides of the body.

7.2. Premotor areas

Our main finding was that patients with PMA damage are unimpaired in unimanual motor performance and in gesture comprehension. Motor impairments after PMA damage were restricted to the bimanual condition when executing different movements simultaneously with the right and left hands (heterogeneous condition). The most pronounced deficits were seen in patients with damage of the medial wall motor areas (supplementary motor area, cingulate motor areas). Their execution of movement could be characterised as that mostly only one hand at a time was active (e.g. stirring with the right hand followed by pouring with the left hand). Sometimes stirring was carried out using both hands at the same time, followed by bilateral pouring movements. In contrast, the performance of homogeneous movements was normal.

Deficits of simultaneous, but independent movements of both hands have been observed in monkey experiments [6,7,43,70–72] and in patients with mesio-frontal lesions [8,26,28,44,72] Brinkman [5] observed in monkeys with SMA lesions that the two hands started to behave in a similar manner instead of sharing the task between them. Following callosotomy this deficit disappeared. Wiesendanger et al. [70,71] trained monkeys on a bimanual pull-and-grasp task, whereby the subjects were required to open with their left hand a drawer with a baited food-well and simultaneously to reach

with their right index finger into the food-well in order to retrieve the reward. Thereafter, a unilateral SMA lesion was placed in three monkeys, and after several weeks of recovery, a second SMA lesion was placed on the other side. In all three monkeys, changes of movement parameters were observed in the limb contralateral to the lesion, such as delays in movement initiation, increased variability in the timing structure and increased movement times. However, the principle of motor equivalence, characteristic for invariant goal achievement, was rather well preserved. The above motor impairments were transient and recovered within a month. The authors concluded that although the SMA participates in bimanual coordination, it appears to be less involved in the co-ordination of well-practised bimanual skills.

The significance of PMA for the integration of bilateral motor behaviour has already been emphasized by Nielsen [50]. In contrast to patients with parietal lobe damage, most of the deficits seen after PMA lesions affect the performance of both arms [17,18,26–28]. Freund and Hummelsheim [17] found that patients with dorsolateral PMA damage showed a reciprocal coordination disorder between the two sides. This deficit became most apparent when the patient was asked to produce alternating windmill movements with their arms or paddling movements with their legs in the backward direction. On the basis of a combined lesion and activation study, we have recently provided evidence that the medial wall areas and here in particular, the cingulate motor areas play a pivotal role for bimanual interaction [67].

Martin et al. [48] reported an activation in the left premotor area when subjects named tools. But the results of this investigation show that lesions of the dorsolateral premotor cortex or the medial wall areas do not cause an impairment in demonstrating object

use to computer presented pictures. Earlier, we have shown that lesions of the premotor cortex in monkeys [29] and man [25–27] cause severe impairments on tasks in which the subject had to learn arbitrary associations between sensory stimuli and a set of motor responses. Patients and monkeys with premotor lesions are impaired when they must recall a movement from memory on the basis of a visual cue. But here, a crucial factor seems to be that in all these experiments it was visual cues from an arbitrary context that must prompt the recall of specific actions. Passingham [52] has shown that monkeys with premotor lesions can recall actions if they are prompted by the identity of the object they must manipulate. This interpretation might also be applied to the present findings: Our patients with premotor damage were unimpaired in demonstrating object use to computer presented pictures. Furthermore, clinical observations of these patients do not reveal any deficits in selecting the appropriate action during their daily ward activities. But the same patients have serious difficulties in test situations where an arbitrary sensory cue is used for directing the movement.

Motor imagery is known to activate medial wall and dorsolateral premotor areas [39,66]. It has recently been shown that the observations of actions also activates the premotor circuitry. Evidence for such an observation/execution matching system in primates [20,59] was first presented by Rizzolatti et al. [59] and Gallese et al. [20] when they registered neurones in the ventral premotor cortex (area F5) that discharged when the monkey performed an action but also when the animal observed an action made by the experimenter or by another monkey. The authors argued that these 'mirror neurones' reflect the representation of the observed behaviour. It was concluded that these neurones play a crucial role in the understanding of motor events. According to Jeannerod [41] and Jeannerod and Decety [42], the neurones responsible for the motor image formation are the same the subject will later activate during planning and preparation of the action thus providing action-schemas. These experimental results were complemented by fMR and magnetic stimulation studies confirming the existence of an observation/execution matching system [13,24,60] in the human. The localization of this system in F5, the likely homologue of Boca's area in the monkey, led to the assumption that it plays a pivotal role for the understanding of gestures and other expressive motor behaviour.

This concept was recently extended by data showing more distributed and somatotopically organised premotor activations during action observation (Buccino et al. [7]). Whenever these actions were object-related parietal cortex was additionally activated. These data emphasize that action observation is processed in dorsal stream areas in order to automatically generate an internal replica of that action. The dissociation between the effects of parietal lesions that interfere with the imitation of action sequences but leave their recognition intact is in accordance with the processing of action-related, pragmatic information along the dorsal and of semantic decoding of observed actions along the ventral stream.

References

[1] Asanuma H. The Motor Cortex. New York: Raven Press, 1989.

[2] Asanuma H, Keller A. Neuronal mechanisms of motor learning in mammals. NeuroReport 1991;2:217–24.

[3] Basso A, Faglioni P, Luzzatti C. Methods in neuroanatomical research and an experimental study of limb apraxia. In: Roy EA, editor. Neuropsychological Studies of Apraxia and Related Disorders. Amsterdam: Elsevier, 1985:179–202.

[4] Bay E. The concepts of agnosia, apraxia and aphasia after history of a hundred years. Journal of the Mount Sinai Hospital 1965;32:637–50.

[5] Brinkman J. Lesions in supplementary motor area interfere with a monkey's performance of a bimanual coordination task. Neuroscience Letters 1981;27:276–80.

[6] Brinkman C. Supplementary motor area of the monkey's cerebral cortex: short- and long-term deficits after unilateral ablation and the effects of subsequent callosal section. Journal of Neurosciences 1984;4:918–29.

[7] Buccino, G, Binkofski F, Fink GR, Fadiga L, Fogassi L, Gallese V, Seitz RJ, Zilles K, Rizzolatti G, Freund H-J. Action observation activates premotor and parietal areas in somatotopic manner: A mRI study (submitted for publication).

[8] Chan J-L, Ross ED. Left-handed mirror writing following right anterior cerebral artery infarction: evidence for nonmirror transformation of motor programs by right supplementary motor area. Neurology 1988;38:59–63.

[9] Clark MA, Merians AS, Kothari A, Poizner H, Macauley B, et al. Spatial planning deficits in limb apraxia. Brain 1994;117:1093–106.

[10] Corina DP, Vaid J, Bellugi U. The linguistic basis of left hemisphere specialization. Science 1992;255:1258–60.

[11] De Ajuriaguerra J, Hecaen H, Angelergues R. Les apraxies. Varietes cliniques et lateralisation lesionelle. Reviews of Neurology 1960;102:566–91.

[12] Deiber M-P, Ibanez V, Sadato N, Hallett M. The cerebral structures participating in motor preparation in humans: a positron emission tomography study. Journal of Neurophysiology 1996;75:233–7.

[13] Decety J, Grezes J, Costes N, Perani D, Jeannerod M, Procyk E, Grassi F, Fazio F. Brain activity during observation of actions. Brain 1997;120:1763–77.

[14] De Renzi E, Motti F, Nichelli P. Imitating gestures: a quantitative approach to ideomotor apraxia. Archives of Neurology 1980;37:6–10.

[15] De Renzi E. Methods of limb apraxia examination and their bearing on the interpretation of the disorder. In: Roy EA, editor. Neuropsychological Studies of Apraxia and Related Disorders. Amsterdam: Elsevier, 1985.

[16] Fadiga L, Fogassi L, Pavesi G, Rizzilatti G. Motor facilitation during action observation: a magnetic stimulation study. Journal of Neurophysiology 1995;73:2608–11.

[17] Freund H-J, Hummelsheim H. Lesions of premotor cortex in man. Brain 1985;108:697–733.

[18] Freund H-J. Motor dysfunction in Parkinson's disease and premotor lesions. European Journal of Neurology 1989;29:33–7.

[19] Freund H-J. The apraxias. In: Asbury AK, McKhann GM, McDonald WI, editors. Diseases of the Nervous System. Clinical Neurobiology II. Chichester: Wiley, 1992:751–67.

[20] Gallese V, Fadiga L, Fogassi L, Rizzolatti G. Action recognition in the premotor cortex. Brain 1996;119:593–609.

[21] Geschwind N. Disconnection syndromes in animals and man. Brain 1965;88:237–94 585–644.

[22] Geschwind N. The apraxias: neural mechanisms of disorders of learned movements. The American Scientist 1975;63:188–95.

[23] Goldenberg G. Imitating gestures and manipulating a mannikin — the representation of the human body in ideomotor apraxia. Neuropsychologia 1995;33:63–72.

[24] Grafton ST, Fadiga L, Arbib MA, Rizzolatti G. Premotor cortex activation during observation and naming of familiar tools. Neuroimage 1997;6:231–6.

[25] Halsband U. Neuropsychologische und Neurophysiologische Studien zum Motorischen Lernen. Lengerich: Pabst Science, 1999.

[26] Halsband U. Brain mechanisms of apraxia. In: Milner AD, editor. Comparative Neuropsychology. Oxford: Oxford University Press, 1998:184–212.

[27] Halsband U, Freund H-J. Premotor cortex and conditional motor learning in man. Brain 1990;113:207–22.

[28] Halsband U, Ito N, Tanji J, Freund H-J. The role of premotor movement in man. Brain 1993;116:243–66.

[29] Halsband U, Passingham RE. Premotor cortex and the conditions for movement in monkeys (*Macaca fascicularis*). Behavioural Brain Research 1985;18:269–77.

[30] Hecaen H, Albert ML. Human Neuropsychology. New York: Wiley, 1978.

[31] Hecaen H, Rondot P. Apraxia as a disorder of signs. In: Roy EA, editor. Neuropsychological Studies of Apraxia and Related Disorders. Amsterdam: Elsevier, 1985:86–110.

[32] Heilman KM. Ideational apraxia — a redefinition. Brain 1973;96:861–4.

[33] Heilman KM. Apraxia. In: Heilman KM, Valenstein E, editors. Clinical Neuropsychology. New York: Oxford University Press, 1979.

[34] Heilman KM, Rothi LJ, Valenstein E. Two forms of ideomotor apraxia. Neurology 1982;32:342–6.

[35] Heilman KM, Rothi LJG. Apraxia. In: Heilman KM, Valenstein E, editors. Clinical Neuropsychology. New York: Oxford University Press, 1993:141–60.

[36] Hermsdörfer J, Mai N, Spatt J, Marquardt C, Veltkamp R, Goldenberg G, et al. Kinematic analysis of movement imitation in apraxia. Brain 1996;119:1575–6.

[37] Huber W, Poeck K, Weniger D, Willmes K. Der Aachener Aphasie-Test. Göttingen: Hogrefe-Verlag, 1993.

[38] Humphrey DR, Tanji J. What features of voluntary motor control are encoded in the neuronal discharge of different cortical motor areas? In: Humphrey DR, Freund H-J, editors. Motor Control: Concepts and Issues. Chichester: Wiley, 1991:413–43.

[39] Ingvar DH, Philipson L. Distribution of cerebral blood flow in the dominant hemisphere during motor ideation and motor performance. Annals of Neurology 1977;2:230–7.

[40] Iacoboni M, Woods RP, Brass M, Bekkering H, Mazziotta JC, Rizzolatti G. Cortical mechanisms of human imitation. Science 1999;24(286):2526–8.

[41] Jeannerod M. Mental imagery in the motor context. Neuropsychologia 1995;33:1419–32.

[42] Jeannerod M, Decety J. Mental motor imagery: a window into the representational stages of action. Current Opinion in Neurobiology 1995;5:727–32.

[43] Kazennikov O, Wicki U, Corboz M, Hyland B, Palmeri A, Roulier EM, et al. Temporal structure of a bimanual goal-directed movement sequence in monkeys. European Journal of Neurosciences 1994;6:203–10.

[44] Laplane D, Talairach J, Meininger V, Bancaud J, Orgogozo JM. Clinical consequences of corticectomies involving the supplementary motor area in man. Journal of Neurological Sciences 1977;34:301–14.

[45] Liepmann H. Das krankheitsbild der apraxie (Motorischen Asymbolie). Mitschriften der Psychiatrie 1900;8:15–44 102–132, 182–197.

[46] Liepmann H. Drei Aufsätze aus dem Apraxiegebiet. Berlin: Karger, 1908.

[47] Martin A, Haxby JV, Lalonde FM, Wiggs CL, Ungerleider LG. Discrete cortical regions associated with knowledge of color and knowledge of action. Science 1995;270:102–5.

[48] Martin A, Wiggs CL, Ungerleider LG, Haxby JV. Neural correlates of category-specific knowledge. Nature 1996;379:649–52.

[49] Matsui T, Hirano A. An Atlas of the Human Brain for Computed Tomography. Stuttgart: Gustav Fischer Verlag, 1978.

[50] Nielsen JM. Agnosia, Apraxia, Aphasia — Their Value in Cerebral Localization. New York: Hoeber, 1946.

[51] Ochipa C, Rothi LJ, Heilman KM. Ideational apraxia: a deficit in tool selection and use. Annals of Neurology 1989;25:190–3.

[52] Passingham RE. Premotor cortex: sensory cues and movement. Behavioural Brain Research 1985;18:175–86.

[53] Pause M, Kunesch E, Binkofski F, Freund H-J. Sensorimotor disturbances in patients with lesions of the parietal cortex. Brain 1989;112:1599–625.

[54] Perret DI, Harries MH, Bevan R, Thomas S, Benson PJ, Mistlin AJ, Chitty AJ, Hietanen JK, Ortega JE. Frameworks of analysis for the neural representation of animate objects and actions. Experimental Biology 1989;146:87–113.

[55] Platz T, Mauritz K-H. Human motor planning, motor programming, and use of new task-relevant information with different apraxic syndromes. European Journal of Neuroscience 1995;7:1536–47.

[56] Poizner H, Mack L, Verfaellie M, Rothi LJG, Heilman KM. Three-dimensional computergraphic analysis of apraxia. Brain 1990;113:85–101.

[57] Rapcsak SZ, Ochipa C, Anderson KC, Poizner H. Progressive ideomotor apraxia: evidence for a selective impairment of the action production system. Brain and Cognition 1995;27:213–36.

[58] Raven JC, Court J, Raven J, Jr. Standard Progressive Matrices. Weinheim: Beltz Test Gesellschaft, 1987.

[59] Rizzolatti G, Fadiga L, Gallese V, Fogassi L. Premotor cortex and the recognition of motor actions. Cognitive Brain Research 1996;3:131–41.

[60] Rizzolatti G, Fadiga L, Matelli M, Paulesu E, Perani D, Fazio F. Localization of grasp representations in humans by PET: 1. Observation versus execution. Experimental Brain Research 1996;111:246–52.

[61] Rothi LJ, Heilman KM, Watson RT. Pantomime comprehension and ideomotor apraxia. Journal of Neurology, Neurosurgery and Psychiatry 1985;48:207–10.

[62] Rothi LJ, Mack L, Verfaellie M, Brown P, Heilman KM. Ideomotor apraxia: error pattern analysis. Aphasiology 1988;2:381–7.

[63] Roy EA, Square PA. Common considerations in the study of limb, verbal and oral apraxia. In: Roy EA, editor. Neuropsychological Studies of Apraxia and Related Disorders. Amsterdam: North Holland, 1985:111–59.

[64] Ryding E, Decety J, Sjoholm H, Stenberg G, Ingvar H. Motor imagery activates the cerebellum regionally. A SPECT rCBF

study with 99mTc-HMPAO. Cognitive Brain Research 1993;1:94–9.

[65] Steinmetz H, Rademacher J, Huang YX, Hefter H, Zilles K, Thron A, Freund H-J. Cerebral asymmetry: MR planimetry of the human planum temporale. Journal of Computed Assisted Tomography 1989;13:996–1005.

[66] Stephan KM, Fink GR, Passingham RE, Silbersweig D, Ceballos-Baumann AO, Frith CD, et al. Functional anatomy of the mental representation of upper extremity movements in healthy subjects. Journal of Neurophysiology 1995;73:373–86.

[67] Stephan KM, Binkofski F, Halsband U, Schnitzler A, Wunderlich G, Tass P, et al. The role of the ventral medial wall motor areas in bimanual coordination: a combined lesion and activation study. Brain 1999;122:351–68.

[68] Tülüc. Tübinger-Luria-Christensen Neuropsychologische Untersuchungsreihe. Weinheim: Beltz Test Gesellschaft, 1983.

[69] Tailarach J, Tournoux P. Co-planar Stereotactic Atlas of the Human Brain. New York: Thieme, 1988.

[70] Wiesendanger M. Recent development in studies of the supplementary area of primates. Reviews of Physiology, Biochemistry and Pharmacology 1986;103:1–59.

[71] Wiesendanger M. The riddle of supplementary motor area function. In: Mano N, Hamada I, DeLong MR, editors. Role of the Cerebellum and Basal Ganglia in Voluntary Movement. Amsterdam: Elsevier, 1993:253–66.

[72] Wiesendanger M, Rouillier EM, Kazennikov O, Perrig S. Is the supplementary motor area a bilaterally organized system? In: Lüders HO, editor. Advances in Neurology: Supplementary Sensorimotor Area, vol. 70. New York: Lippincott-Raven, 1996:85–94.

[73] Zilles K. Cortex. In: Paxinos GT, editor. The Human Nervous System. San Diego, CA: Academic Press, 1990.