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Tactile apraxia
Unimodal apractic disorder of tactile object exploration associated with parietal lobe lesions

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Summary
Tactile apraxia is characterized by an isolated disturbance of hand movements for use of and interaction with an object (transitive movements) in the presence of preserved intransitive movements (movements without use of an object, for example repetitive movements or gestures). It is, however, still unclear whether motor and sensory abnormalities represent causal or associated features of tactile apraxia. To address this question, quantitative kinematic recordings of exploratory finger movements (transitive movements) and rapid alternating finger movements (intransitive movements) were studied in 20 healthy volunteers and 22 patients with focal lesions of the parietal, anterofrontal and motor cortex. The most severe deficits of manual object exploration were found in patients with parietal lesions, using the hand contralateral to the lesion. Patients with lesions of the anterior parietal lobe who exhibited prominent sensory deficits and astereognosia showed a decrease in frequency and regularity of exploratory finger movements and a marked increase in exploration space. Patients with posterior parietal lesions exhibiting severe astereognosia, apraxia and deficits in dexterity had a greater decrease in frequency and regularity of manipulative movements, but a less pronounced increase of exploration space than the patients with anterior parietal lesions. Although the patients with parietal lobe lesions could generate rapid alternating finger movements, the regularity of these movements was also impaired. In comparison, patients with frontal lobe lesions exhibited impaired contralesional manipulatory and rapid alternating finger movements but no sensory abnormalities or astereognosia. We conclude that tactile apraxia represents a deficit in the programming of exploratory finger movements mediated by the parietal lobe. The comparison with lesions of other regions participating in the cortical network for tactile exploration reveals that apraxia of exploratory movements in parietal lobe lesions represents a disturbance distinct from elementary motor or sensory abnormalities, but closely related to stereognostic functioning.

Keywords: functional magnetic resonance imaging; hand function; kinematic movement analysis; parietal cortex; premotor cortex; sensorimotor integration

Introduction
The unity between perception and action in the form of a 'Gestaltkreis' was originally proposed by Viktor von Weizsäcker in1940 (von Weizsäcker, 1940). Effective grasping and object manipulation are based on three fundamental properties of the motor system: the capacity to generate independent finger movements, the ability to transform sensory information concerning the object to be grasped into an appropriate hand configuration, and a sophisticated somatosensory control of finger movements (see Jeannerod *et al*., 1995). Exploratory manipulation is a hand–object interaction where such a tight interplay between tactile perception and fine finger movements is of crucial importance. It has been shown in psychophysical studies that not only do perceptual goals constrain exploratory action, but conversely, exploration may constrain what is perceived (for a review, see Lederman and Klatzky, 1987, 1997). Thus, higher level knowledge of objects produce patterns of manual exploration movements that lead to haptically driven object representations. Such knowledge-derived use of pre-programmed hand movements was proposed previously by Katz in 1925 and Hippius in 1934 (Katz, 1925; Hippius, 1934), who differentiated between touching with gliding movements, sweeping touch and grasping touch. Most important here, Hippius defined a kinematic touch as an

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analytic and integrative touch permitting the recognition of the material and formal qualities of an object. On the contrary, Dejerine coined the term 'virgin hand' for patients with infantile hemiplegia in whom astereognosis by inexperience was found in the paralysed hand without detectable sensory disturbances (Dejerine, 1907). This condition is similar to the anaesthesia through akinesia of Chrétien (Chrétien, 1902).

Impairments of tactile object recognition have been the object of extensive research since the advent of neurological brain research (Head and Holmes, 1911). Impaired tactile recognition of objects in patients with sensory deficits that could not be accounted for by the severity of disorder was first described by Puchelt (Puchelt, 1844). Hoffmann (1885) first used the term 'astereognosia' for the deficit in recognition of spatial features of objects (Hoffmann, 1885). Wernicke (1876) introduced the term 'Tastlähmung' or tactile paralysis for the general deficit of tactile recognition (Wernicke, 1876). He also differentiated between primary and secondary tactile object recognition, with primary recognition concerned with basal object features and secondary recognition concerned with the object itself (Wernicke, 1895). Correlative deficit-lesion studies suggested that these different stages of object recognition could involve different structures of the parietal cortex (Roland, 1987).

For patients with disturbed transitive exploratory hand movements and preserved intransitive expressive and symbolic movements, Klein (1931) introduced the term 'tactile apraxia' (Klein, 1931). Delay (1935) postulated that the disturbed stereognosia can have an influence on tactile apraxia (Delay, 1935). He cited Lhermitte (Lhermitte, 1933), who stated that the gnostic functions are generally disturbed in apraxia. This notion has been confirmed recently for recognition of one's own hand movements in patients with apraxia (Sirigu *et al.*, 1999). Although cases with tactile agnosia without associated basic sensory deficits, deficits in manual exploration (Critchley, 1953; Caselli, 1991, 1993) or even with preserved spatial abilities (Reed *et al.*, 1996) were described previously, cases with tactile apraxia without concomitant gnostic deficits have not been reported in the literature (Yamadori, 1982; Endo *et al.*, 1996).

Here we describe patients, characterized by specific deficits in tactile object manipulation. The most prominent deficits after lesions of the parietal lobe could not be attributed primarily to one or more motor or sensory disturbances such as paresis, ataxia, dysmetria, ideomotor or ideational apraxia, or motor neglect. The condition we observed in our patients was characterized by the clinical finding of a useless hand in spite of slightly or moderately disturbed sensation and nearly normal force production. In contrast to ideomotor apraxia (Liepmann, 1920), these patients were capable of producing complex intransitive hand movements as well as rapid tapping or power grip. The disorder in our patients was restricted to fine dexterous movements as required for active touch (Gordon 1978), object manipulation and was associated with astereognosis. From these observations the existence of a special form of apraxia, tactile apraxia, was established, thus substantiating early clinical descriptions (Pötzl, 1937; Klein, 1931; Delay, 1935).

Methods

Patients and normal subjects

Twenty right-handed normal subjects (age 24–35 years, mean 28 years; six female, 14 male) were selected. They were all without history of a neurological disease and had normal status on neurological examination and normative kinematic data on tactile object exploration and repetitive finger movements.

Twenty-two patients participated in our study. The inclusion criteria were: (i) one focal cerebral lesion that affected the cerebral cortex at a parietal, anterior frontal or precentral location (Figs 1 and 2); (ii) the ability to use the hand for manipulation of objects and a cognitive state that enabled the patients to understand the experimental conditions; and (iii) absence of hypokinesia, dementia, aphasia, tremor and unwillingness to cooperate.

Ten patients (age 15–73 years, mean 49 years) had ischaemic lesions of the parietal lobe (five on the right and five on the left side; five of the anterior and five of the posterior parietal lobe). Eight patients (age 35–75 years, mean 56 years) had lesions of the anterior frontal cortex. Seven of these patients had ischaemic lesions and one had an astrocytoma. Six lesions were located in the left and two in the right anterior frontal lobe. Four patients had ischaemic lesions of precentral gyrus (age 38–66 years, mean 53 years). Of these lesions, two were located on the left side and two on the right side.

Patients were tested at the chronic stage (35 $\pm$ 20 days after infarction) of the illness. All patients and all normal subjects were right handed as assessed with the Oldfield Inventory (Oldfield *et al.*, 1971).

All patients and subjects gave informed consent for their participation in this study. This study was approved by the Ethics Committee of the Heinrich-Heine University, Düsseldorf.

Clinical examination

Sensorimotor hand function was assessed in both hands by a clinical score (modified by Kunesch *et al.*, 1995). The examination comprised an assessment of arm force, grip force, hand dexterity, limb coordinative functions, the presence of ideomotor and ideatory apraxia, motor attention, as well as proprioception, surface sensibility of the skin and stereognosia. Five degrees of impairment were defined: 0, loss of function; 1, severely affected; 2, moderately affected; 3, slightly affected; 4, normal function. Stereognosia was quantified separately as follows: 0, none; 1, one object recognized; 2, two objects; 3, three objects; 4, four objects recognized. The data from each group of patients were pooled

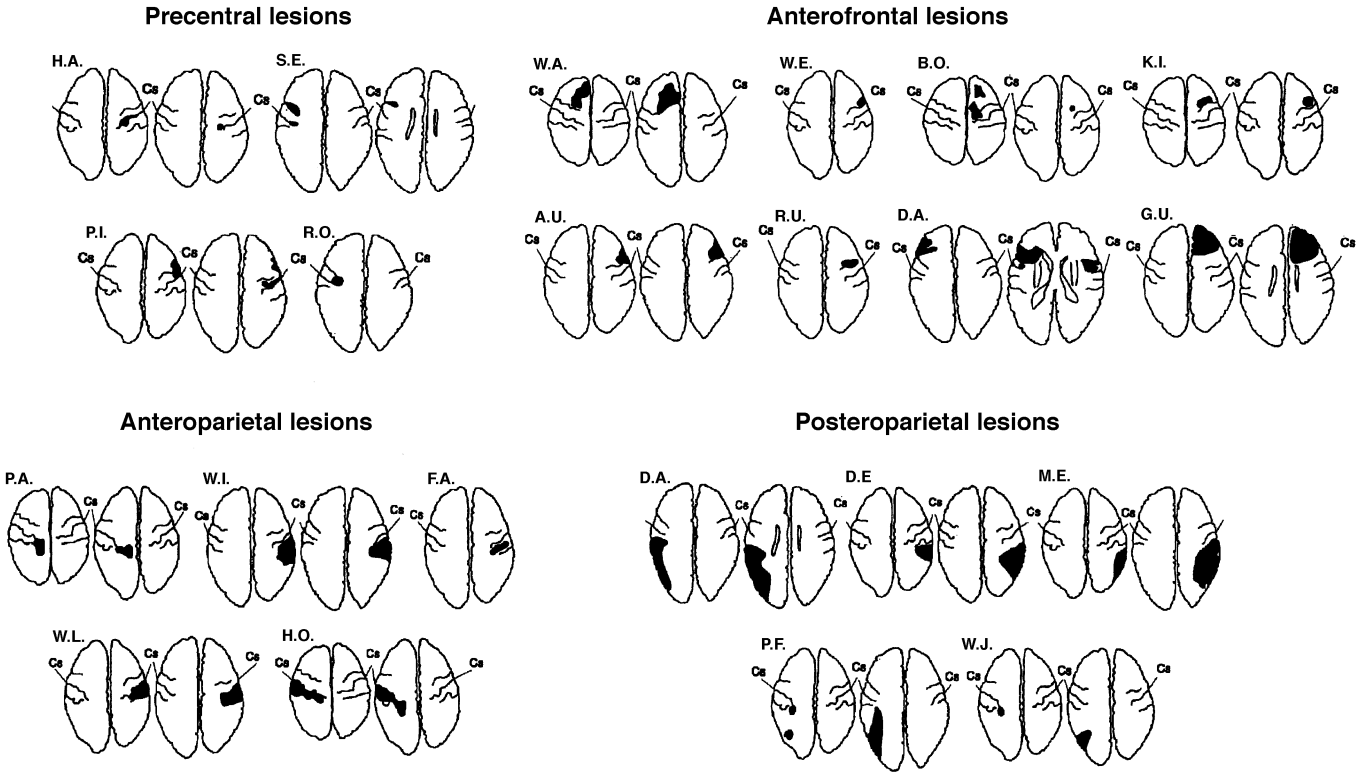

Fig. 1 Lesions of all patients transposed on the templates from the Matsui and Hirano atlas (1979). Representative templates showing the greatest extent of each lesion are presented. Cs = central sulcus.

and mean values and standard deviations were calculated for each item (Fig. 3).

Mapping of structural lesions

As described earlier (Binkofski *et al*., 1996), structural lesions were outlined on computed tomography or T_1-weighted MRI scans obtained in the chronic stage of the lesion (CT scanner: General Electric CGR CE 1000, Milwaukee, Wis., USA; MRI scanner: Siemens Magnetom 1.5 teslas, Erlangen, Germany; spin echo MR sequence, 600 ms repetition time, 15 ms echo time, two excitations, 5 mm slice thickness). On these images, lesions were defined as parenchymal defects isodense/isointense to cerebrospinal fluid. All CT and MRI sections were oriented parallel to the canthomeatal line, thereby allowing anatomical mapping on corresponding templates derived from the atlas of Matsui and Hirano (Matsui and Hirano, 1978). For this purpose, each CT or MRI brain section containing the lesion was proportionally re-scaled in order to fit with the maximal anteroposterior and transverse dimensions of the brain atlas (Matsui and Hirano, 1979).

Kinematic recordings

Subjects were seated in a comfortable chair with their elbows resting on a table, the hand to be tested held in a semipronated position. The lower arm was fixed by a plastic strap at the wrist so that the fingers could be moved freely. Infrared light-emitting diodes were fixed to the ulnar side of the thumb and to the radial side of the forefinger. Subjects were required to perform two tasks: (i) exploratory finger movements and (ii) rapid alternating forefinger–thumb opposition movements. Both hands were tested beginning with the affected hand. The thumb and index finger have been shown previously to be the most important fingers for tactile exploration (Kunesch *et al*., 1989; Seitz *et al*., 1991). Movements were monitored at a frequency of 100 Hz, with a two-camera optoelectronic recording system (Selspot II, Partille, Sweden). In the beginning of the experiment a movement space was calibrated by means of a position reference structure with defined dimensions. Details have been described in a previous article (Kunesch *et al*., 1989).

In some patients additional video recordings of the motor performance were performed in order to document the tactile exploration disorder.

Movement tasks

Exploratory finger movements. The subjects and patients were blindfolded and instructed to explore, at their natural preferred speed and by active touch, small geometrically simple objects (wooden cube with a length of side of 1 cm; wooden ball with a diameter of 1.3 cm; plastic plate with a diameter of 2 cm; metal plate with a sandpaper surface and a diameter of 2 cm). These objects were used in order to allow comparability of the manipulation kinematics between

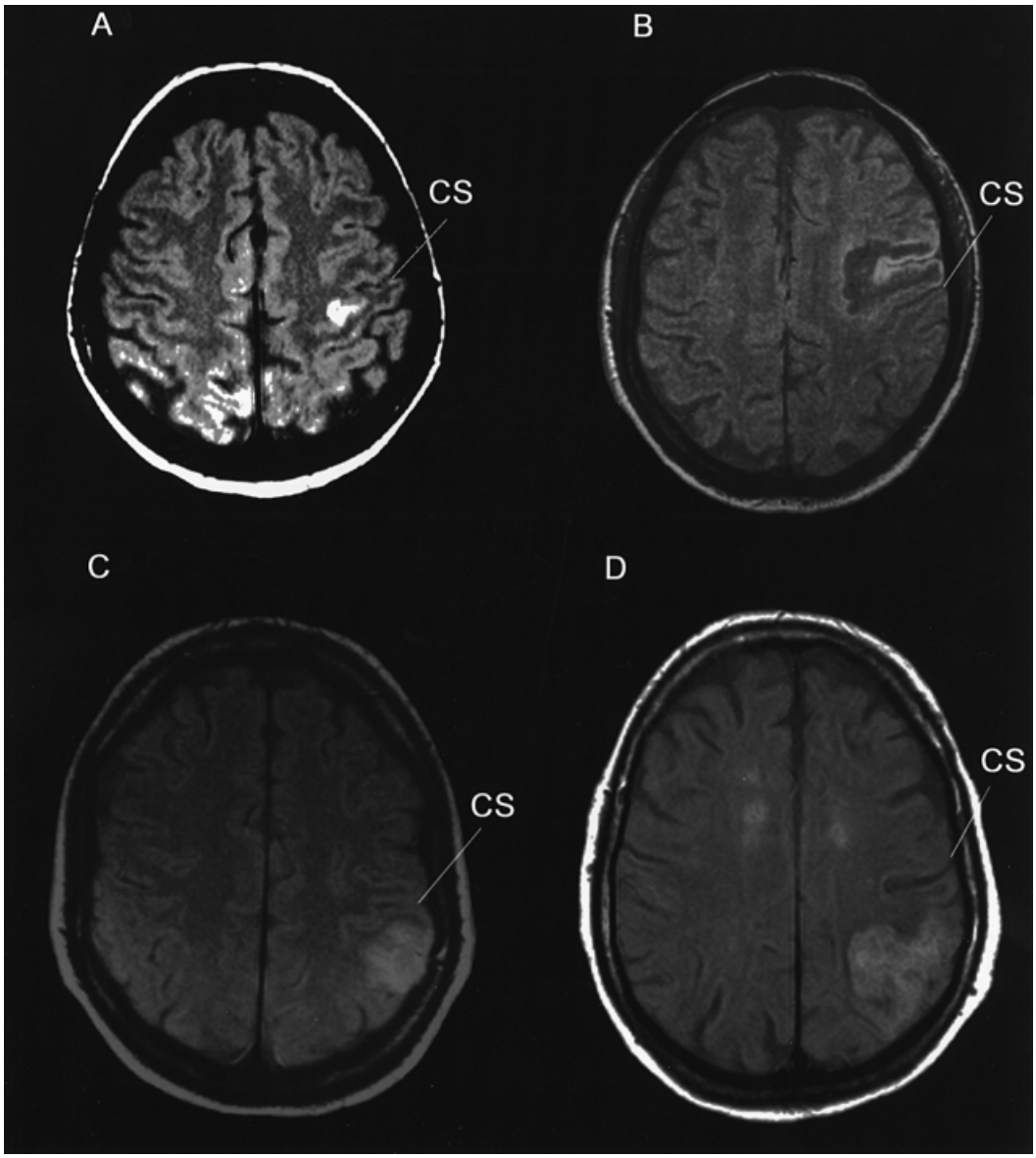

Fig. 2 Examples of original MRI scans of one representative patient from each group: (**A**) precentral lesion; (**B**) anterior frontal lesion; (**C**) anterior parietal lesion; (**D**) posterior parietal lesion. CS = central sulcus.

the patients and normal subjects. Subjects and patients were instructed to identify as many features of the objects as possible (e.g. after recognizing the object as a ball, to detect also a small hole in it) and to continue the scanning movements throughout the data acquisition period of 6–10 s. Both hands were tested separately.

After the tactile exploration, patients were asked to report verbally whether they had recognized the object and to name the recognized features of the scanned objects.

Object recognition. The recognition capacity was classified into three categories: (i) recognition of the explored object; (ii) recognition of some features of the object but not of the object itself; and (iii) no tactile recognition.

Rapid alternating movements. Subjects were instructed to perform rapid alternating movements of the thumb and the index finger at maximal speed by extending both fingers and flexing them until they touched each other. Both hands were tested separately. The recording time was 10 s.

Data analysis

Position data, obtained by the standard Selspot acquisition software, were converted to ASCII-format and analysed by means of Dadisp (DSP Development Corporation, Cambridge, Mass., USA) and Mathematica (Wolfram Research Ltd., Champaign, Ill., USA) software packages. Position and velocity data were filtered by a dualpass Butterworth filter with a cut-off frequency of 8 Hz.

In order to quantify the exploratory finger movements, the following parameters were determined (see Fig. 4): (i) the

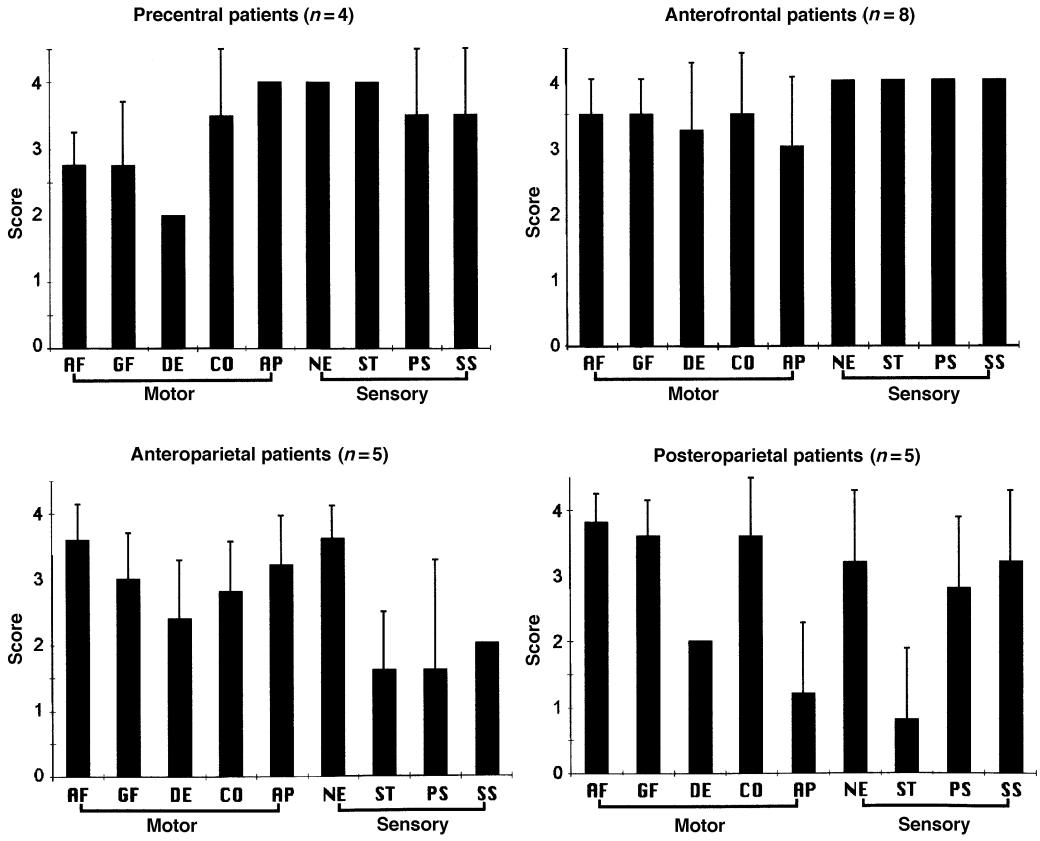

Fig. 3 The spectrum of clinical deficits of each patient's group as assessed by the clinical score. Mean values and standard deviations are presented. Abbreviations of the score items: AF = force of the arm, GF = grip force, DE = dexterity, CO = coordination, AP = ideomotor apraxia, NE = neglect, ST = stereognosia, PS = position sense, SS = surface sensibility.

movement space of the thumb was assessed as the smallest 3D (three-dimensional) cube covering all movement trajectories and calculated as multiplication of the maximal extension of the movements in x, y and z directions. Thumb movement space was chosen since during tactile exploration the thumb is opposed to the other fingers, thus including the movement space also of the index finger. Thumb and index finger are in steady contact with the object during the tactile exploration (Fig. 4). (ii) An *index of regularity* of movements was defined as the area under the dominant frequency peak $\pm$ 0.5 Hz (or if movement frequency was $<$0.5 Hz as the area from 0 to 1.0 Hz) divided by the area under the entire spectrum as shown in Fig. 4. (iii) The *dominant movement frequency* was assessed by means of spectral frequency analysis of the movements and defined as the mean frequency peak in the spectrum.

For the quantification of the rapid repetitive forefinger–thumb opposition movements, the parameters dominant movement frequency and index of regularity were used.

Statistical analysis

Statistical analysis included the assessment of mean values and standard deviations of group kinematic and clinical data. The differences among groups and conditions were assessed by ANOVA (analysis of variance) with Bonferroni correction for multiple comparisons. Additional testing was performed using linear and Spearman's rank correlations.

Results

Clinical data

Lesion location

Precentral lesions. In two patients (P.I. and H.A.), the cortex lining the left central sulcus was affected by an ischaemic lesion (Fig. 2). In Patient P.I. the lesion extended into the lateral premotor cortex. In two other patients (S.E. and R.O.) the right precentral gyrus was affected (Fig. 1). In Patient S.E., an area around the precentral sulcus was additionally affected (Fig. 1).

Anterofrontal lesions. Five patients (A.U., K.I., R.U., W.E. and B.O.) had small lesions rostral to the anterior bank of the precentral gyrus. In Patient B.O., an additional small lesion was localized in the upper prefrontal cortex (Fig. 2). Patient G.U. had a large ischaemic lesion in the left frontal lobe reaching into the lower premotor cortex. The ischaemic lesion of Patient W.A. affected the right lateral premotor

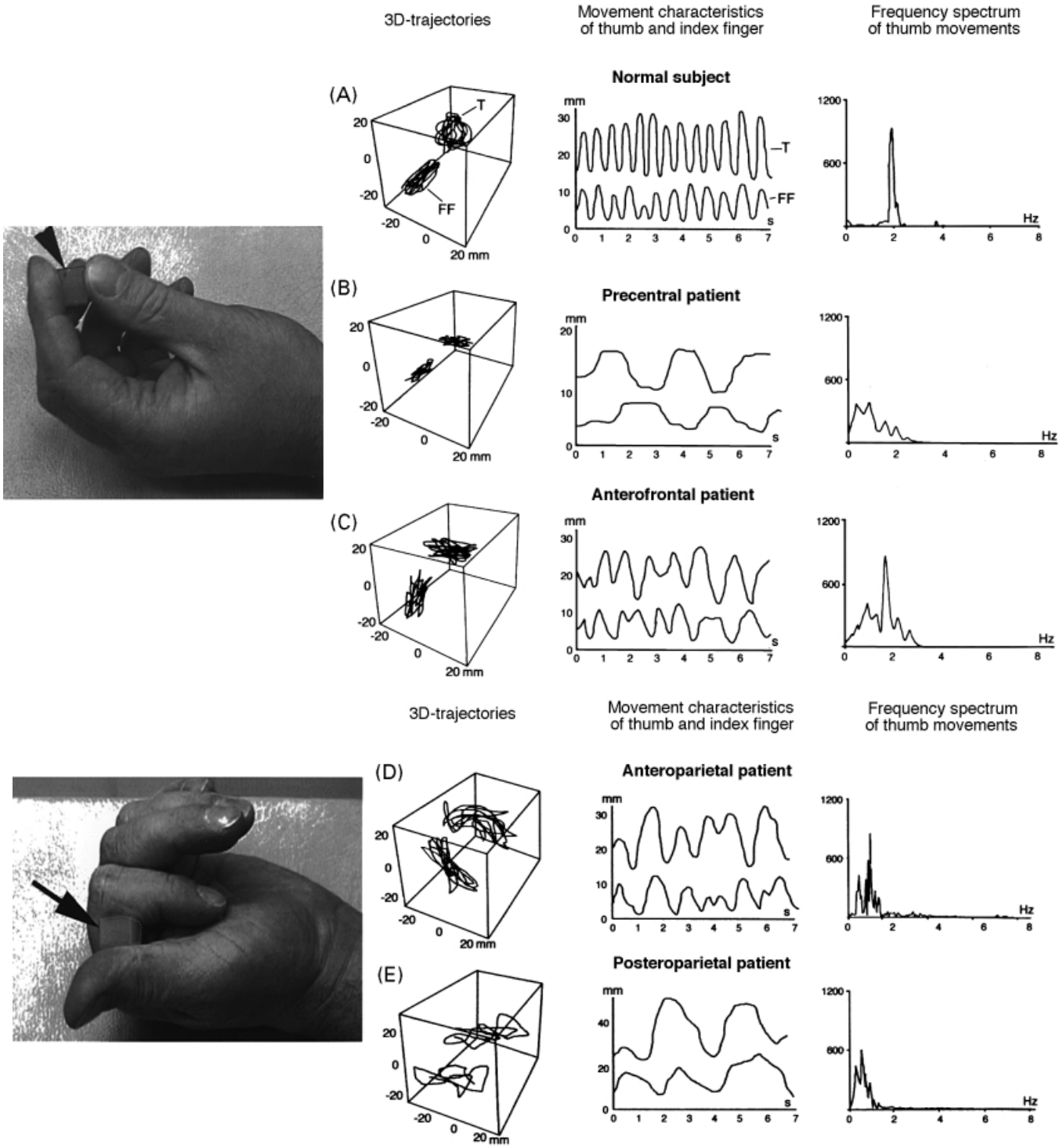

Fig. 4 Exploratory finger movements. The top left-most panel shows a typical position of the thumb, the index finger and the ring finger of a healthy volunteer during tactile exploration of a small object. (**A**) Kinematic analysis of exploratory finger movements in a normal subject. (**B**) A patient with a precentral lesion. (**C**) A patient with an anterofrontal lesion. The analysis includes a 3D reconstruction of thumb (T) and index finger (FF) movement trajectories (*left panels*), the scan paths of the thumb and index finger (*middle panels*) and the frequency distribution of the thumb movements (*right panels*). Note, in the bottom left-most panel, the inadequate position of the fingers in relation to the object in a patient with a posterior parietal lesion. (**D** and **E**) Kinematic analysis of exploratory finger movements in a patient with an anteroparietal lesion (**D**) and a patient with a posteroparietal lesion (**E**).

cortex, but extended into the anterior mesial region. In Patient D.A., the lesion was localized in the left lateral premotor cortex and extended to the lower premotor cortex (Fig. 1). An additional small lesion was localized in the right lower premotor cortex (Fig. 1).

Anteroparietal lesions. In Patients P.A. and H.O. the lesions

were localized in the right postcentral gyrus rostral to the intraparietal sulcus, while the left postcentral gyrus also rostral to the intraparietal sulcus was affected in Patients F.A. and W.L. (Fig. 1). The lesion of Patient W.I. affected the posterior postcentral gyrus and extended to the anterior part of the superior parietal lobule and the area around the anterior intraparietal sulcus (Fig. 2).

Posteroparietal lesions. In Patients D.A. and P.F. extensive lesions affected the right lower lateral posterior parietal lobe and the bottom of the intraparietal sulcus (Fig. 1), whereas in Patients D.E. and M.E. the left lower lateral posterior parietal lobe and the bottom and anterior part of the intraparietal sulcus was affected (Fig. 2). Patient W.J. had a lesion in the right lower posterior parietal lobe and a second small lesion of the right postcentral gyrus (Fig. 2).

Patterns of clinical deficits

Precentral lesions (n = 4). The most prominent deficit in this group of patients was a marked disturbance of dexterity followed by moderate deficits in arm force and in maximal grip force. Coordinative functions and basic sensory functions were marginally affected (Fig. 3).

Anterofrontal lesions (n = 8). This group of patients was characterized by a mild limb kinetic apraxia and mild deficits in dexterity, arm force, grip force and in coordinative functions. No hemineglect or deficits of basal and complex sensory functions were found (Fig. 3).

Anteroparietal lesions (n = 5). In this group of patients, the basic sensory functions and the stereognosia were markedly disturbed followed by moderate disturbance of hand dexterity. Less prominent deficits were found in coordinative functions, in grip force and in praxis. A slight disturbance of arm force and a slight motor hemineglect were also found (Fig. 3).

Posteroparietal patients (n = 5). Marked astereognosia (more accentuated than in the former group) and ideomotor apraxia characterized this patient group, followed by a prominent deficit in dexterity. Motor hemineglect and deficits in basic sensory functions were less pronounced, while arm force, grip force and coordinative functions were almost normal (Fig. 3).

Kinematics of exploratory and repetitive finger movements

Exploratory finger movements

Normal subjects. In normal subjects, the instruction to recognize the features of the objects under exploration evoked, in most cases, vivid exploratory finger movements. A characteristic example of normal scanning movements of the thumb and the index finger is presented in the 3D reconstruction of the movement traces in Fig. 4A (left). It is evident that the scan paths varied continuously and were never repeated. However, some regularities in the patterns of the movements of the thumb and the index finger could be observed. They were, at least in part, induced by the biomechanic constraints of the metacarpophalangeal joints: the index finger performed mainly flexion and extension movements in a regular manner, with only slight sideward movements, while the thumb, having more degrees of freedom, performed more rotatory movements around the carpometacarpal joint. The manipulatory movements of the thumb performed more extensive movements and covered greater movement space than the index finger (Fig. 4A). The index finger and the middle finger, instead, served mostly as opponents. Sometimes the index finger took over the leadership or the thumb and the index finger moved together. But, as is evident from Fig. 4, the fingertips always touched the object. The temporal profiles of the movement along the *x*-axis show regular quasi-sinusoidal movements of the thumb and of the index finger (Fig. 4A, middle panel). As a rule, the spectral analysis of the movement trace of the thumb showed a narrow mean frequency peak at <2 Hz (Fig. 4A, right panel).

Group analysis showed that the mean frequency of the exploratory finger movements in the normal subjects was 1.30 Hz (SD $\pm$ 0.19) for the right hand and 1.27 Hz (SD $\pm$ 0.21) for the left hand (Fig. 4). The mean index of regularity of the thumb movements was 0.82 (SD $\pm$ 0.5) for the right hand and 0.79 (SD $\pm$ 0.48) for the left hand. The mean space of exploration was 8.29 mm^3 (SD $\pm$ 1.86) for the right thumb and 7.98 mm^3 (SD $\pm$ 2.3) for the left thumb (Fig. 5).

All subjects successfully recognized all objects during the scanning procedure. The mean time for recognition of the objects was ~2–3 s; however, according to instruction, further manipulatory movements were performed in order to find out additional features of the objects.

Patients with precentral lesions. The exploratory finger movements in patients with precentral lesions were characterized by decreased movement space ($P < 0.01$; Fig. 5A), decreased regularity of movements ($P < 0.01$; Fig. 5B) and decreased movement frequency ($P < 0.01$; Fig. 5C) on the contralesional side compared with normal subjects. A characteristic example is presented in Fig. 4B, where from the 3D reconstruction of the movement traces it became evident that movements were scarce and used a narrow space of exploration. The temporal profiles of the movements show that the fingers moved in an isolated manner with a low frequency and low amplitude (Fig. 4B).

With only one exception all objects were recognized correctly by patients of this group.

Patients with anterofrontal lesions. The exploratory finger movements in patients with anterofrontal lesions differed from those of normal subjects only in their regularity. As evident from Fig. 5, the only significant difference in the quantitative kinematic analysis was a decrease in the regularity index ($P < 0.001$) on the contralesional side. In contrast, the movement space and the frequency of manipulatory movements on the contralesional side were comparable to normal subjects. A characteristic example of exploratory finger movements in a premotor patient is

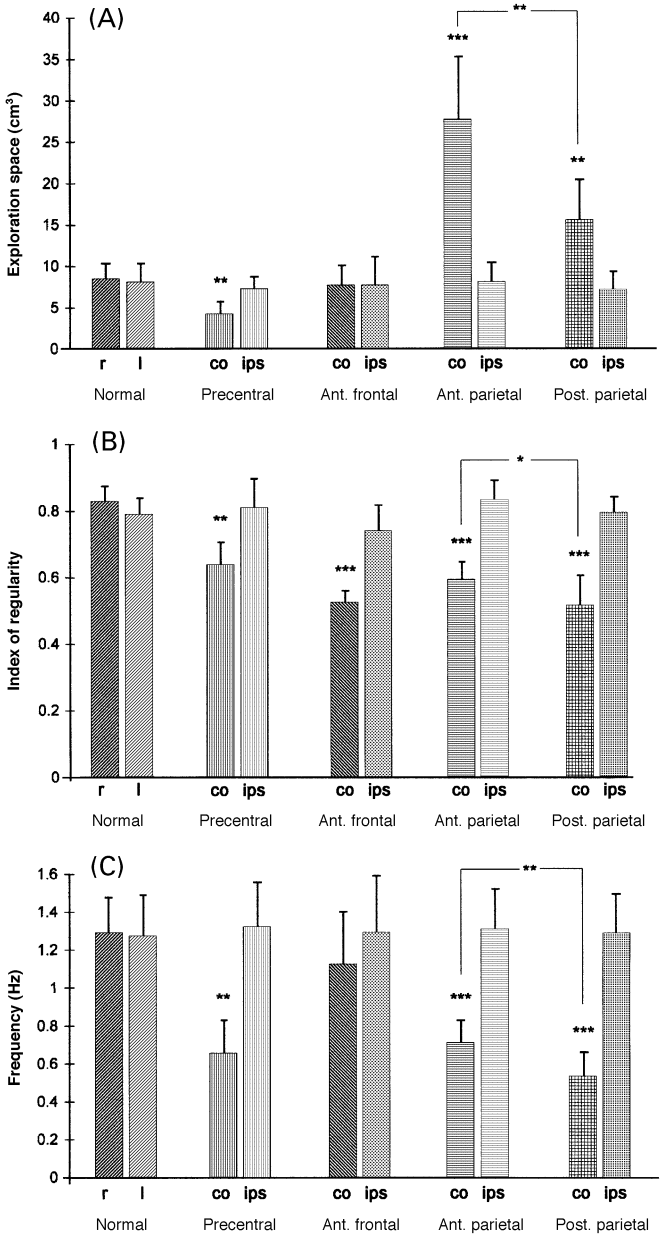

Fig. 5 Quantitative kinematic group data of object exploration in normal subjects and all patient groups (ant. frontal = anterofrontal; ant. parietal = anteroparietal; post. parietal = posteroparietal): (**A**) space of exploration; (**B**) index of regularity; (**C**) mean frequency. Mean values and standard deviations are shown. **Significant difference to the normal group ($P < 0.01$); ***significant difference to the normal group ($P < 0.001$). r = right; l = left; co = contralesional; ips = ipsilesional.

presented in Fig. 4C (left panel). As in normal subjects, the fingertips touched the object. The 3D reconstruction of the movement trajectories of the thumb and the index finger showed more irregular movements compared with the same movement of a normal subject (Fig. 4C, left panel). Temporal profiles of the thumb and index finger movements along the main axis of the movements show, in detail, greater irregularity of the movements with greater amplitude

Table 1 *Results of tactile recognition in patients with parietal lesions*

Patients	Objects			
	Sphere	Cube	PISp	PIGu
Anterior parietal				
P.A.	++	#	#	+
W.I.	+	++	+	#
F.A.	+	+	+	++
W.L.	#	++	#	#
H.O.	#	#	+	#
Posterior parietal				
D.A.	#	+	+	++
D.E.	+	#	+	+
M.E.	+	#	+	++
P.F.	++	+	+	#
W.J.	+	#	+	++

++ = object recognized; + = some features of the object recognized, but no object recognition; # = no features of the objects recognized and no object recognition; PlSp = plate with a sandpaper surface; PlGu = plate with a plastic surface.

modulation than the normal subject (Fig. 4C, middle panel). The spectral analysis of the thumb movements showed a more irregular frequency distribution, with a peak frequency lying, however, within a normal range (Fig. 4C, right panel).

All patients with premotor lesions correctly recognized all the objects.

Patients with anteroparietal lesions. The patients with anteroparietal lesions were able to perform quite vivid manipulatory movements. These movements were characterized by a vast increase in movement space ($P < 0.001$; Fig. 5A), a decrease in regularity ($P < 0.001$; Fig. 5B) and a marked decrease in frequency ($P < 0.001$; Fig. 5C) compared with normal subjects. An example of the abnormal manipulatory movements is shown in Fig. 4D. One characteristic feature, namely the inadequate position of the fingers in relation to the object, was recognized from the video recording. The 3D reconstruction of the movement traces showed great uncoordinated movements of the thumb and the index finger with reduced thumb rotation, but preserved ab- and adduction (Fig. 4D, left panel). The temporal profiles showed markedly increased movement amplitudes and lower frequency of movements (Fig. 4D, middle and right panels).

In general, patients from this group made many errors of tactile recognition of objects using their affected contralesional hand (Table 1). Furthermore, only 20% of objects were recognized completely. In 35% of objects, only some features were recognized and in 45% of objects no features were recognized.

Patients with posteroparietal lesions. The profile of deficits in the manipulative finger movements in this patient group was similar to that in patients with anteroparietal lesions, but there were some quantitative differences (Fig. 5). The most prominent difference was the lower increase in the explora-

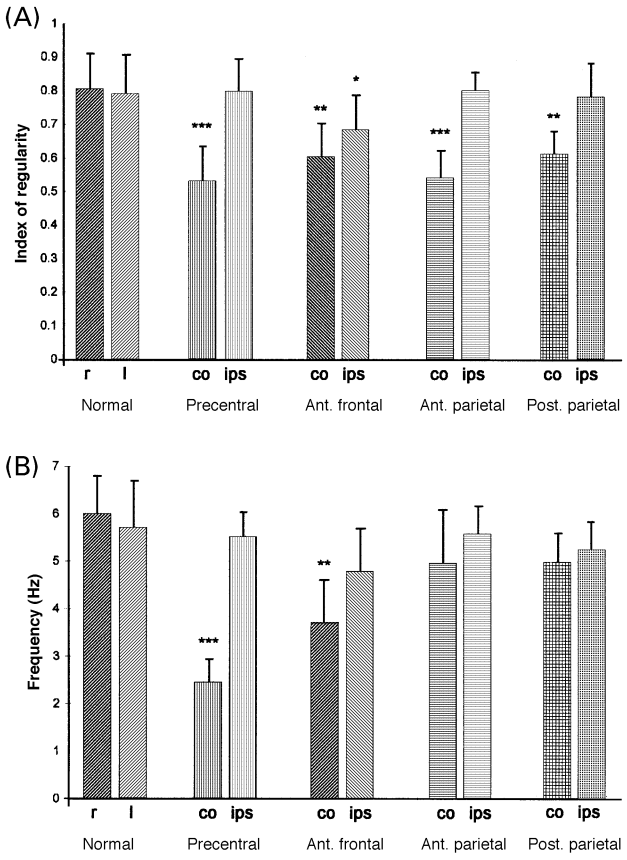

Fig. 6 Quantitative kinematic group data of rapid repetitive forefinger–thumb opposition movements in normal subjects and all patient groups (ant. frontal = anterofrontal; ant. parietal = anteroparietal; post. parietal = posteroparietal): (**A**) index of regularity; (**B**) mean frequency. Mean values and standard deviations are shown. *Low significant difference to the normal group ($P < 0.05$); **significant difference to the normal group ($P < 0.01$); ***significant difference to the normal group ($P < 0.001$). r = right; l = left; co = contralesional; ips = ipsilesional.

tion space as compared with the anterior parietal group ($P < 0.01$; Fig. 5A), which was still significantly greater than in the normal group ($P < 0.01$). There was also decrease in mean regularity index ($P < 0.001$; Fig. 5B) and in the mean frequency of movements ($P < 0.001$, Fig. 5C), which were even more pronounced than in the anteroparietal patients (Fig. 5).

The 3D reconstruction of movement traces in Fig. 4E (left) showed also ill-coordinated movements of the forefinger and thumb with an increased amplitude of movements. The temporal profiles of the movements demonstrated inadequate, large and irregular movements. The spectral analysis showed a low mean peak frequency with several additional frequency peaks (Fig. 4E, right panel).

In this group, when the contralesional hand was used, only 20% of objects were completely recognized, in 50% some features of the object were recognized and 30% of the explored objects were not recognized at all (Table 1).

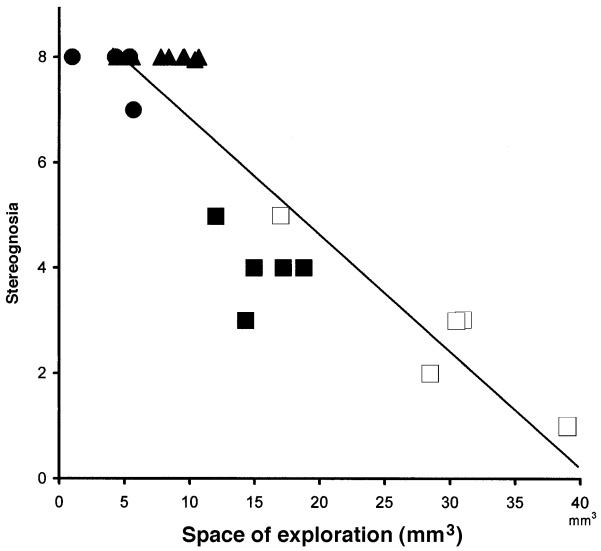

Fig. 7 Negative correlation ($r = -0.92$, $P < 0.0001$) between the space of exploration and stereognosis. Note that lower score values indicate impaired stereognosia. Closed circles = precentral patients; closed triangles = anterofrontal patients; open squares = anteroparietal patients; closed squares = posteroparietal patients.

Repetitive forefinger–thumb opposition movements

Normal subjects. In normal subjects the repetitive forefinger–thumb opposition movements were performed at significantly higher frequency than the exploratory movements and in a very regular manner. The mean dominant movement frequency for the right hands was 6.0 Hz (SD ± 0.81) and for the left hands 5.7 Hz (SD ± 0.98). The regularity index was 0.81 (SD ± 0.11) for the right hands and 0.79 (SD ± 0.12) for the left hands (Fig. 6).

Patients with precentral lesions. The characteristic feature of this group was a marked decrease in the mean frequency of the opposition movements ($P < 0.001$) and in the regularity index ($P < 0.001$) on the contralesional side as compared with the normal group (Fig. 6). In the ipsilesional hand, the mean movement frequency and regularity was not significantly different from the normal group.

Patients with anterofrontal lesions. These patients had a significantly decreased mean frequency of the forefinger–thumb opposition movements ($P < 0.01$) and a decreased index of regularity of these movements ($P < 0.01$) on the contralesional side compared with the normal group. In addition, there was also a slight reduction of the index of regularity on the ipsilesional side (Fig. 6).

Patients with anteroparietal lesions. In this patient group only the index of regularity of the forefinger–thumb opposition movements was markedly reduced on the contralesional side in comparison with the normal group ($P < 0.001$). No significant reduction of the mean frequency of these movements was found (Fig. 6).

Patients with posteroparietal lesions. Again, there was a pronounced reduction of the index of regularity of the forefinger–thumb opposition movements on the contralesional

side. However, the reduction was less severe than in the anteroparietal group ($P < 0.05$) (Fig. 6).

Additional statistical analysis. No correlation was found between the frequencies and indices of regularity in exploratory finger movements versus rapid alternating finger movements. Since the alternating finger movements were exceedingly faster than the exploratory finger movements, these results suggested that the finger movements in exploration were determined by sensory constraints that were not relevant for finger movement production. Correlation analysis showed a negative correlation of stereognosia as assessed by the clinical score and the space of exploration as obtained by the kinematic measurements [$r = –0.9$, $r^2 = 0.81$, $P < 0.0001$ (two tailed), see Fig. 7]. This observation indicated that astereognosia was related to exaggerated exploration movements. However, there was no correlation between space of exploration and dexterity. Thus, the exaggerated exploration movements reflected apractic movement activity not related to a disturbance of fractionated finger movements *per se*.

Discussion

The main finding of this study was the presence of severe abnormalities of exploratory finger movements, despite normal frequencies of repetitive finger movements and almost normal force production, in patients with chronic lesions of the parietal cortex. We attribute this kinematic deficit to a higher level motor disturbance, hitherto called tactile apraxia. In our patients, this condition was associated with the impairment of tactile object recognition (astereognosis). In accordance with an earlier study (Pause *et al*., 1989), we found that, in patients with anterior parietal lesions, other basic somatosensory functions were as severely affected as stereognosis, while patients with posterior parietal lesions showed a preferential impairment of stereognosis. Our new findings extend those of this earlier study, showing that the exaggerated exploration movements associated with astereognosia represent a specific deficit of patients with parietal lobe lesions that was not observed in frontal lobe patients. Thus, our findings are in accord with the classical qualitative descriptions of astereognosia by Puchelt (1844), Hoffmann (1885), Wernicke (1895) and Critchley (1953) and with the systematic analysis of abnormal tactile exploration in patients with brain lesions of different location as described by Roland (1987).

The quantitatively assessed manual exploration of space in patients with lesions of the parietal cortex was markedly enhanced, but was inadequate for the tactile finger–object interaction. As demonstrated by kinematic recordings (Figs 4 and 5), this abnormality was specific for the parietal cortical lesions compared with frontal lesions that affected finger movements in a different manner. Further, we found that the severity of astereognosia was significantly related to the enlargement of the exploration space (Fig. 7). As patients with anterior parietal lesions also showed prominent impairments in simple somatosensory functions (Fig. 3), it is possible that these patients compensated for this somatosensory deficit by exaggerated explorative movements. However, in patients with posterior parietal lesions the exploration space was also enlarged, although their simple somatosensory functions were far less impaired. Thus, the enhanced movement space in conjunction with the slow and irregular exploratory finger movements suggested an impairment of movement programming in patients with parietal lobe lesions. Consequently, parietal lobe lesions interfere with the control of complex finger movements as required for tactile object exploration resulting in movement abnormalities that appear apractic.

For comparison, in patients with lesions of frontal cortex there was a significant reduction of dexterity and maximal grip force when motor cortex was damaged. Recently, we showed that reductions of dexterity and maximal grip force were related to the degree of damage of motor cortex or the pyramidal tract (Binkofski *et al*., 1996). Here we show by kinematic recordings that both the regularity and frequency of exploratory as well as of fast repetitive forefinger–thumb opposition movements were reduced in these patients with frontal lobe lesions (Figs 5 and 6). Of these measures, most prominent were the irregularity of the exploration movements in the patients with lesions of the anterofrontal cortex and the reduction of frequency and regularity of fast repetitive forefinger–thumb opposition movements in the patients with motor cortex lesions. In spite of these deficits, none of these patients suffered from astereognosia or showed an enhanced exploration space.

Since there was no relationship between velocity of exploratory and that of alternating finger movements in our patients with parietal lesions (Figs 5 and 6), the slowing of exploratory finger movements in these patients was not due to an impaired capacity to perform fast finger movements. This, however, was the case in the patients with motor cortex lesions who showed a greater slowing of fast alternating than of exploratory finger movements (Figs 5 and 6). Interestingly, the degree of astereognosia did not correlate with the impairment of dexterity. Motor cortex lesions induce severe impairments in dexterity, while stereognosis is spared. In the parietal patients, the situation is reversed as astereognosia is prominent in spite of preservation of hand and finger movements not involved in active touch. There is not a global affection of dextrous finger movements as such, but a specific deficit of the finely tuned scanning procedure of the fingers during the exploration of objects. We have shown recently (Kunesch *et al*., 1989; Seitz *et al*., 1991) that these exploratory digital palpation movements are normally performed at slow rates (0.5–2 Hz) in order to match the temporal requirements for the collection of sensory information. These type-I movements were distinctly different from the rapid alternating movements employing the hand as a whole, often with the fingers in a steady hold posture. These type-II movements are used for manual skills such as writing, typing, hammering or tapping and are performed at

rates around 4 Hz (Kunesch *et al*., 1989). Our data presented here show their differential impairment so that type-II movements are more severely affected by motor and premotor damage, whereas parietal lesions interfered with type-I movements.

The sensory information obtained in the hand comprises two elements: exteroceptive and proprioceptive. The combination of these two inputs is an important variable specifying the difference between touching and being touched (Gibson, 1962). Active touch can elaborate the unity, stability, plasticity and shape of phenomenal objects. When a single object is grasped with several fingers the subject perceives one object only although several cutaneous receptor sheets are engaged. The complex feature extraction relating spatial information about corners, edges, straight planes can be distinguished with respect to their interrelationship even when subjects cannot identify the patterns formed by the various cutaneous pressures. The percept is the object form and not skin form because the movements of the fingers are not perceived. Features such as curvatures, flat surfaces, slant of surface with respect to gravity, parallel surfaces, plane angles and lengths, must all underlie the object identification. In tactile apraxia, this ability to engage the hand in the motor performance required to collect the sensory information is disturbed. This is even more remarkable in patients with a paretic hand, who can still accomplish that function, as a good exploratory pattern is preserved. It is the shaping of input necessary to gain the information about external objects that cannot be properly accomplished. This is a specific unimodal sensory-motor disturbance confined to the somatosensory modality. In contrast, visuomotor performances or the employment of the hand for the task unrelated to active touch and object manipulation are preserved.

The concomitant impairment of stereognosis and manual praxis as regards object-related hand function on the basis of somatosensory information has implications for our present view on parietal lobe function. First, it shows the unimodal nature of this disturbance, leaving visuomotor tasks such as pointing or reaching towards targets normal as well as intransitive movements and the performance of rapid alternating movements or force production. Secondly, it shows that both the pragmatic and the cognitive aspects of somatosensation are governed by the parietal lobe. This stands in contrast to the visual processing dichotomy with a pragmatic route through the parietal lobe and a recognitive route through the temporal lobe. This dual role of the parietal lobe is in accord with electrophysiological recordings in non-human primates and recent neuroimaging studies showing that the superior posterior parietal cortex plays an important role in hand motor control (Binkofski *et al*., 1999*a*, *b*). In monkeys, the superior parietal lobule is essentially related to the elaboration of proprioceptive information. Neurones from area PE (regis parientalis superior; von Economo and Koskinas, 1925), the area forming most of the superior parietal lobule cortical convexity, are active with passive joint rotation, deep tissue pressure, as well as during active arm movements (Sakata *et al.*, 1973; Mountcastle *et al*., 1975; Kalaska *et al.*, 1990; Lacquaniti *et al.*, 1995). Some of them combine proprioceptive information from different joints, possibly playing a role in a more global representation of body parts (Mountcastle *et al.*, 1975); others put together tactile and joint information (Sakata *et al.*, 1973).

Activation studies support the prominent role of the superior parietal lobule for active touch also in man. Seitz and colleagues asked subjects to discriminate among a series of oblongs differing only in their length (Seitz *et al.*, 1991). The results showed an increase of cerebral blood flow in the primary sensory and motor areas, in premotor cortex, in the supplementary motor area and, most importantly for the present discussion, in the superior parietal lobule. In a recently performed fMRI study we found, accordingly, activations in the primary sensory and motor areas, in premotor cortex, in the supplementary motor area, the secondary somatosensory area and, specifically, in the superior parietal lobule (Binkofski *et al.*, 1999*a*, *b*).

Another important area for the fine tuning of hand and finger movements is the anterior intraparietal area. In the anterior intraparietal area, many neurones discharge during finger and hand movements, while others respond to specific visual 3D stimuli or discharge both during active finger movements and in response to 3D stimuli congruent in size and shape with the coded grasping movement (Taira *et al.*, 1990; Sakata *et al.*, 1992). It is important to stress that anterior intraparietal area neurones discharge not only during object presentation and visually guided hand shaping, but also during object holding and manipulation (Sakata *et al.*, 1992, 1995; Jeannerod *et al.*, 1995). Corresponding findings were reported recently also for the human anterior intraparietal area as there is a good correspondence of activation and lesion studies highlighting the role of the parietal cortex for the control of prehension movements (Binkofski *et al.*, 1998).

The difference in the quantitative parameters of tactile exploration in patients with lesions of different parts of the posterior parietal cortex is in accordance with such a modular organization of the posterior parietal cortex function. A double dissociation of parietal lobe function has recently been found for mirror agnosia and mirror ataxia (Binkofski *et al.*, 1999*c*).

The apractic disturbance of hand use in the tactile domain described here for the patients with posterior parietal lesions closely resembles the classical descriptions that were designated by the term tactile apraxia, first coined by Klein (1931).

The unimodal nature was beautifully illustrated by Klein in 1931, when he described the case of a patient with well-preserved intransitive expressive movements, who could not use objects placed in his hand (Klein, 1931). However, when he was set in front of the object a correct and purposive

reaching and grasping movement was initiated. There was no apraxia when the patient saw the object, but there was apraxia when he started actively to touch it.

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