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IMITATING GESTURES AND MANIPULATING A MANNIKIN—THE REPRESENTATION OF THE HUMAN BODY IN IDEOMOTOR APRAXIA

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Abstract—Imitation of meaningless gestures was examined in patients with left brain damage (LBD), right brain damage (RBD) and controls. In addition to imitation on the own body, patients were asked to replicate the gestures on a life-sized mannikin. Manual dexterity was assessed by manipulation of beads, and general visuospatial abilities by block-design. LBD patients who displayed apraxia when imitating gestures on their own bodies scored dramatically worse than any other group when imitation was assessed on the mannikin. By contrast, on block-design and manipulation of beads patients with RBD were inferior not only to LBD patients without apraxia but also to apractic patients. Analysis of CT scans revealed that apraxia occurred with frontal, parietal and deep lesions, and that the impairment on the manipulation of the mannikin was present regardless of lesion site. The results support the contention that the basic deficit underlying impaired imitation of meaningless gestures in apraxia is to be sought at a conceptual level. Possibly, patients with apraxia are not able to evoke and represent conceptual knowledge about the human body which is necessary for performing the apparently simple task of imitating gestures.

INTRODUCTION

Around the turn of the century Liepmann conducted the first systematic group study of limb apraxia [21]. He examined performance of symbolic gestures in normal controls, patients with left brain damage and patients with right brain damage. Patients with brain damage demonstrated the gestures with the hand ipsilateral to the lesion. Only patients with left hemispheric lesions committed apractic errors. Most patients with apraxia had aphasia too, but performance of gestures was faulty even when the correct gestures were demonstrated and the patients were only required to imitate them. To Liepmann, impaired imitation of gestures proved that apraxia cannot be referred to being a sequel of aphasia or "asymbolia" [9]. He stated that errors at the imitation of gestures testify "that there is not only a weakness of memory in its common sense, that is, an inexactness of the spatial–temporal image (of the movement), but a difficulty or inability to direct the members of the left hand according to certain spatial conceptions" [21, p. 26]. Liepmann introduced the term "ideomotor apraxia" to denote the inability to translate a correct idea of the intended movement into an appropriate motor act. The view that the basic deficit in ideomotor apraxia concerns the execution rather than the evocation of gestures received support later studies which demonstrated that in patients with ideomotor apraxia imitation of meaningless and novel movements is as faulty as that of symbolic gestures [7, 20, 23, 28].

The straightforward conclusion that the left hemisphere is dominant for the control of any

skilled movements of either hand is, however, contradicted by the clinical experience that there is no general clumsiness of the left hand in patients with apraxia. On the contrary, a striking dissociation between grossly impaired performance in apraxia testing and apparently normal skill in many other situations is a hallmark of the clinical presentation of ideomotor apraxia [10, 13, 14, 17, 21, 24, 25]. The widely accepted definition of apraxia as a disorder of skilled movement not caused by weakness, akinesia, deafferentation, abnormal tone or posture, and movement disorders such as tremors or chorea [11, 14] implies that there are situations in which the apractic hand is moved with completely normal skill. Otherwise, the exclusion of other motor disorders which is crucial to the diagnosis of apraxia could never be verified.

An idea which could account for deficient imitation of meaningless gestures without postulating a general motor dominance of the left hemisphere is, that the basic deficit in ideomotor apraxia concerns the ability to code and comprehend movements in relation to own body [4, 23, 29]. Both symbolic and meaningless gestures are aimed at producing explicitly defined positions or movements of body parts. By contrast, most movements outside the testing situation are aimed at and determined by external targets and are therefore spared from ideomotor apraxia, although they may be vulnerable to ideational apraxia. The terms "*dyssomatognosie spatiale*" [4, 23] and "reflexive apraxia" [29] have been proposed to characterize this conception of ideomotor apraxia.

The purpose of the present study was to test the hypothesis that faulty apprehension of the movements and configuration of body parts underlies impaired imitation of meaningless movements in ideomotor apraxia. Implicit in the experimental design is the assumption that the basic disorder affects a general concept of the human body whose validity does not depend on whether a person's own body is concerned or not. It was hypothesized that such a conceptual disturbance would show up when postures of the hand are to be replicated with the hand of a mannikin rather than with one's own hand, although the motor demands of manipulating a mannikin are fundamentally different from those of performing the postures on oneself. To rule out the possibility that a general deficiency of motor execution affects any skilled motor performance, a test of motor skill which required the manipulation of beads was included in the experimental design. It was hypothesized that a disorder of motor execution, which manifests itself in tasks posing motor demands as differing in imitation of gestures and manipulation of a mannikin, would also show up in another test of manual dexterity. To explore whether a faulty apprehension of spatial relationships between body parts might be an expression of a more general disturbance of visuospatial processing, the comprehension and manipulation of spatial relationships outside the human body was assessed by block design. As block design demands motor manipulation of blocks, this test served as a further control for a general disturbance of motor execution.

METHOD

Imitation of meaningless gestures

(a) *On oneself:* The patients were asked to imitate 10 meaningless gestures of the hand (see Fig. 1) The examiner sat in front of the patient and demonstrated the gesture "like a mirror", that is, if the patients used their left hand, the examiner demonstrated with the right hand and vice versa. Imitation started immediately after each presentation. For each posture, 2 points were credited when imitation was correct after the first presentation. Otherwise, the demonstration was repeated and 1 point was given for a correct imitation on second trial.

(b) *On a mannikin:* The patients were seated opposite a life-sized wooden mannikin whose arms and hands could be moved like that of a human being. They were asked to replicate with one hand of the mannikin gestures demonstrated by the examiner. The examiner sat besides the mannikin and demonstrated the gestures "like the

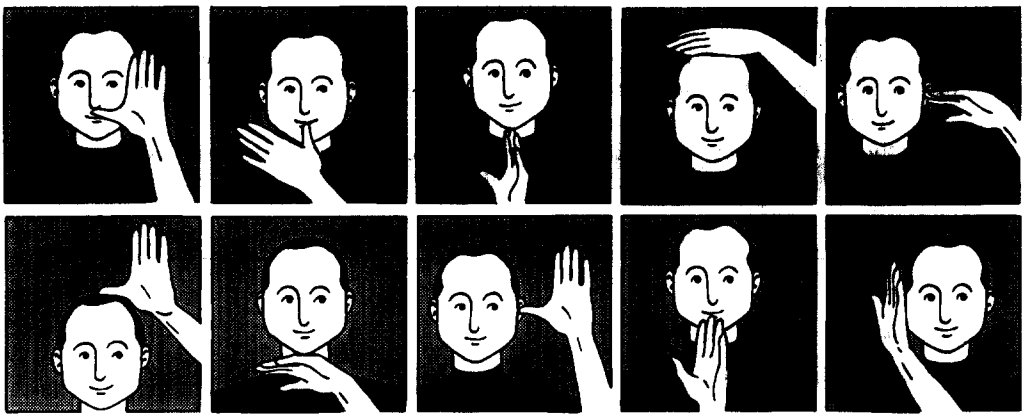

Fig. 1. Ten meaningless gestures of the hand for imitation.

mannikin", that is, if the patient manipulated the mannikin's left hand, the examiner demonstrated with the left hand and vice versa. The gestures used, the course of examination and the criteria for scoring were the same as with the imitation on one's own body. To ensure understanding of the instruction, imitation of a gesture not used in the examination was demonstrated in the instruction, and if the first item of the examination was failed on both trials, correct performance of this gesture was demonstrated too.

Manipulation of beads

Patients were asked to take with one hand three beads from three vertical rods and to stick them on three other rods. They were not allowed to collect and transpose the beads one after another, but had to collect all three beads in the hand before beginning to stick them on the target rods. The rods were aligned in a frontal plane. Patients who used their left hand moved the beads from the left set of three sticks to the right one, and vice versa. Between trials, the hand rested on a mark in front of the middle of the device. After two successful practice trials, 10 trials were run, and the time from leaving the starting point until the delivery of the last bead was measured with a stopwatch. If a bead fell out of the hand, the trial was repeated. The mean time of the 10 trials was taken for the statistical evaluation.

In this task, manual dexterity is challenged when one bead has to be transferred between hand and rod while at the same time one or two beads are carried within the hand. The hand has to be divided into two functional compartments, one for carrying beads and one for moving beads from and to the rod and the beads have to be moved between these two compartments. As a further difficulty, the boring of the beads has to be aligned exactly with the position and the direction of the rod when the bead is stuck on the rod.

Block design

The WAIS—R subtest block design was administered in the usual way. As a modification, testing was not interrupted when the prescribed time limits had expired. If a patient arrived at the correct solution after that time, one point was subtracted from the minumum score. Raw scores were used for statistical evaluation.

Token-test

All patients with left hemisphere damage were administered the German version of the Token Test [16]. The raw number of correct responses was scored.

Procedure of testing

Patients with brain damage used the hand ipsilateral to the lesion for imitation of gestures, pantomime of object use and manipulation of beads. They moved the same hand of the mannikin, but were allowed to use both of their hands for the manipulation of the mannikin if this was not prevented by hemiplegia. Controls used either the right or the left hand for all tests including the manipulation of the mannikin.

Subjects

Eighty-five right-handed subjects were examined. There were 35 patients with left brain damage (LBD), 20 patients with right brain damage (RBD), and 30 controls without any evidence for brain damage. All patients with brain damage had suffered a single, unilateral cerebrovascular accident (CVA), and all LBD patients had aphasia.

RESULTS

No significant differences on any measure were found between controls who had used their left hand and those who had used their right hand. These two groups were therefore brought together to make one control group.

Figure 2 shows the scores on imitiation of meaningless gestures. In controls, there was a

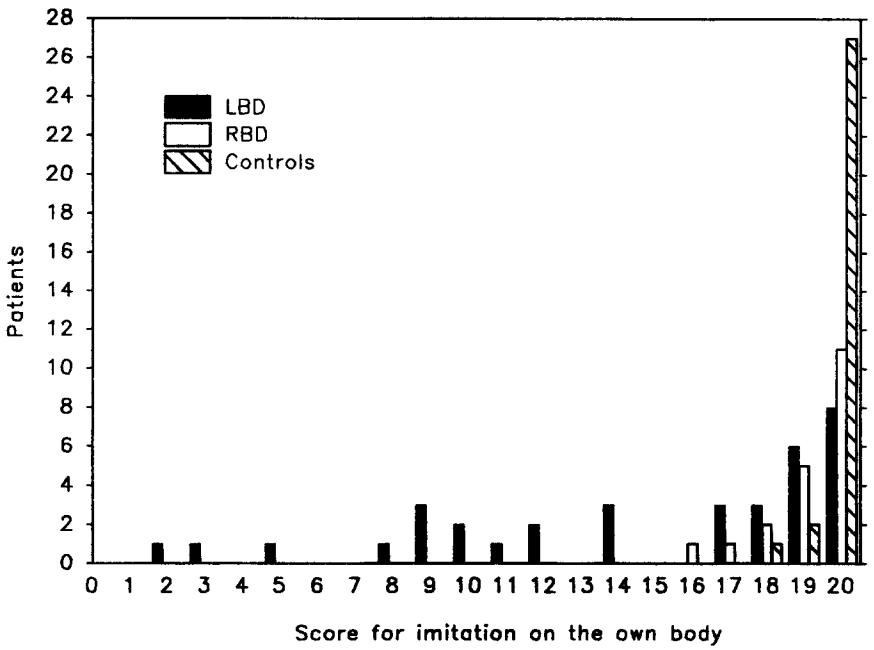

Fig. 2. Results of apraxia testing.

clear ceiling effect. Ninety per cent of the control patients obtained the maximum score of 20 and only one control scored as low as 2 points below the maximum. The distribution of scores of RBD patients had a similar but somewhat less steep bias and the worst score was 16. Patients with LBD can be divided into two groups: 20 of them scored within a range of 17–20, that is, as well as RBD patients and very similar to controls, while 15 scored between 2 and 14. The latter group was considered as being apractic, and further comparisons were conducted between apractic patients, LBD patients without apraxia. RBD patients, and controls. By this division, apraxia was distinguished from the mild impairment of imitation which can also be found in some patients with RBD [7] and was investigated as an exclusive symptom of LBD damage.

Table 1 shows the clinical and demographic data of the four groups. While the mean age of the whole LBD group was very similar to that of the other groups (57.8 ± 14.7), the division of LBD patients in those with and without apraxia resulted in a significant difference of age across groups, as patients with apraxia were on average older than those without. Analyses of covariance with group as main factor and age as covariate showed a significant effect of age only on the speed of manipulation of beads [$F(1, 68) = 15.0$, $P < 0.0005$].

Table 1. Demographic and clinical data of patients

	Apractic	LBD without apraxia	RBD	Controls		*P*	Pairwise differences
Mean age	65.1 (12.7)	52.3 (13.8)	58.0 (12.0)	54.6 (14.3)	$F(3, 81) = 3.0$*	0.034	A > L
Women/men	4/11	9/11	10/10	12/18	$\chi^2 = 2.1$	> 0.2	
Median months since CVA	2 (1-12)	2 (1-36)	1.5 (1-24)		$\chi^2 = 0.8$†	> 0.2	
Ischaemic infarction/bleeding	13/2	12/8	12/8		$\chi^2 = 3.2$	> 0.1	
Arm plegic/not plegic	10/5	11/9	14/6		$\chi^2 = 1.1$	> 0.2	

*ANOVA with *post-hoc* Tukey tests.
†Kruskall–Wallis non-parametric analysis of variance.
Values in parentheses are standard deviations for age, and ranges for duration.

Table 2. Results of experimental tests

	Apractic Mean (S.D.)	LBD without apraxia Mean (S.D.)	RBD Mean (S.D.)	Controls Mean (S.D.)		*P*	Pairwise differences
Imitation on manniquin	8.0 (3.9)	16.5 (2.3)	16.2 (1.3)	17.9 (1.6)	$F(3, 81) = 66.7$	< 0.00005	A < L, A < R, A < C*
Manipulation of beads†	8.9 (1.7)	8.6 (1.1)	9.6 (1.6)	7.7 (1.3)	$F(3, 67) = 7.2$‡	0.0003	R > C*
Block Design	12.8 (7.9)	20.2 (9.4)	7.3 (5.0)	—	$F(2, 51) = 14.2$	< 0.00005	R < L, R < A*
Token test	4.1 (4.9)	21.0 (13.1)			$t(24.1) = 5.2$	< 0.0005	

*Tukey test.
†Age adjusted time in seconds.
‡Group effect in ANCOVA with age as covariate.

Table 2 presents the results of the analysis of covariance for manipulation of beads and of analyses of variance or *t*-tests, respectively, for the remainder of the experimental tests.

Apractic patients scored dramatically worse than any other group when imitation of postures was assessed on the mannikin. Compared to LBD patients without apraxia, they also did less well on block design and on the Token Test and were slower in the manipulation of beads. However, with block design and the manipulation of beads, RBD patients had the greatest difficulty. On block design, RBD patients scored significantly lower than LBD patients with or without apraxia. On manipulation of beads RBD patients were slower than any of the other groups, although on *post-hoc* testing they differed only from controls. From observation of their performance it appeared that the RBD patients had particular problems with the exact alignment of the bead's boring to the direction of the rods. When imitation on the mannikin was compared among LBD patients with and without apraxia in an analysis of covariance with results of the Token Test, there was a significant effect of the Token Test [$F(1, 31)=4.29$, $P=0.047$] but the difference between both groups remained highly significant [$F(1, 31)=24.77$, $P<0.0005$].

CT scans were available from all but one brain damaged patients. Lesions were classified as to whether they encroached upon the frontal, the insular, the temporal or the parietal cortex, the basal ganglia, the thalamus, and the subcortical white matter. As an index of lesion size, the number of affected regions was summed up (Table 3).

The number of affected regions was larger in patients with apraxia than in both other groups. Furthermore, there were significant differences in the frequency of frontal and parietal lesions. The frequency of frontal lesions was lower in LBD patients without apraxia but did not markedly differ between apractic patients and patients with RBD. Parietal lesions were more common in apractic patients than in both other groups. There were only two apractic patients in whom neither the parietal nor the frontal cortex was damaged. Both had deep lesions which affected the basal ganglia and white matter in one, and white matter only in the other. These patients scored 4 and 7 which is in the severely defective range, with the mannikin. There were three apractic patients in whom the lesion affected the frontal cortex and spared the parietal cortex, and seven patients with the reverse dissociation. The mean score on the mannikin was 10.7 (S.D. 5.9) for the patients with frontal lesions and 7.3 (S.D. 4.0) for those with parietal lesions. This difference is statistically insignificant ($t=1.1$, d.f.$=8$, $P>0.2$). A similar slight difference in favour of the patients with frontal lesions was present for imitation on own body (patients with frontal lesions: mean 11.7, S.D. 2.5; patients with parietal lesions: mean 9.6, S.D. 3.5).

DISCUSSION

The hypothesis that motivated that study predicted that faulty apprehension of spatial relationships between body parts would manifest itself not only in imitation on oneself but also in imitation on a mannikin. The results are in accordance with this prediction. However, the associated occurrence of two deficits does not prove that they result from a common functional disturbance. Alternatively, the association may stem from anatomical contiguity of brain regions responsible for each of the functions and from the effect of lesion size. A larger lesion within an area devoted to one of the functions will not only cause a more severe deficit of this function but also have a greater chance to encroach upon neighbouring regions subserving basically different functions. In the present study, this caveat certainly applies to the comparison between LBD patients with and without apraxia. Patients with apraxia had

Table 3. Distribution of lesions

	Apractic $n=15$	LBD without apraxia $n=20$	RBD $n=19$		P	Pairwise difference
Frontal cortex	40	5	32	$\chi^2=6.6$	0.03	A > L, R > L*
Insular cortex	60	30	26	$\chi^2=4.8$	0.09	
Temporal cortex	67	30	37	$\chi^2=5.1$	0.08	
Parietal cortex	67	15	11	$\chi^2=15.8$	0.0004	A > L, A > R*
Subcortical white matter	47	45	35	$\chi^2=0.4$	>0.2	
Basal ganglia	40	15	11	$\chi^2=5.1$	0.08	
Thalamus	0	5	20	$\chi^2=5.1$	0.08	
Number of regions affected	3.2 (1.4)	1.5 (0.8)	1.7 (0.9)	$F(2, 51)=4.8$	<0.00005	A > L, A > R†

CT was missing for one patient with RBD. For ease of comparison, the numbers of patients in whom the lesion affected the given region are expressed in per cent of the group size. For the number of lesions affected, mean and S.D. (in parentheses) are given.

*χ^2 test.

†Tukey test.

larger lesions, and they did worse than patients without apraxia not only on imitation on the mannikin, but also on the Token Test, block design and manipulation of beads, even if for the latter two measures the differences were not statistically significant.

The analysis of the apractic patients' lesions did, however, not disclose any evidence that extension of the lesion into a critical region of the left hemisphere caused impaired imitation on the mannikin. In accord with previous studies we found apraxia to be present in patients with parietal lesions, frontal lesions and deep lesions [2, 19]. Imitation on the mannikin was equally defective in all three groups of patients. To sustain the assumption that the association between impaired imitation on the body and on the mannikin was caused by anatomical contiguity of the cerebral systems supporting both tasks, one would have to assume that both systems are distributed in a very similar fashion throughout the left hemisphere. A common basic functional disturbance is a more convincing explanation for the associated occurrence of impaired imitation on the mannikin and on the patient's own body.

If we accept a common basic disturbance as the source of errors in imitation on the patient's own body and on the mannikin, this does not necessarily prove that the disturbance concerns the apprehension of spatial relationships between body parts. Alternatively, it may stem from a disorder of motor execution which affects the execution of any skilled movement or from a pervasive visuospatial impairment which affects the apprehension of any spatial relationships. Both of these possibilities are made unlikely by a comparison between apractic patients and patients with RBD.

Although the lesions of RBD patients happened to be on average smaller than those of apractic patients, RBD patients did worse than apractic patients on block design and were slower in the manipulation of beads. The latter difference failed to reach statistical significance, but is nonetheless in unequivocal contrast to the superior performance of RBD patients on both imitation tasks. Imitation of the mannikin, block design and manipulation of beads require the manipulation of external objects under visual feedback control. It would appear that the demands on motor execution are more similar among them than between imitation on the mannikin and imitation on own body, where no external object is involved, and where the target positions are largely hidden from view (see Fig. 1). At the same time, the opposite group differences of block design and imitation on the mannikin speak against a general weakness of visuospatial processing as being the cause of impaired imitation on the mannikin.

In sum, it does appear that impaired imitation of movements on a mannikin by apractic patients can neither be dismissed as being due to a spurious association of deficits caused by anatomical contiguity, nor to a general disturbance of the execution of any skilled motor action, nor to a pervasive impairment of visuospatial processing. It does seem worthwhile to consider in more detail the hypothesis that defective apprehension of the configuration of body parts underlies impaired imitation of movements in apraxia.

Defective imitation of gestures on oneself and on a mannikin cannot be ascribed to the disturbance of a personal "body schema" which integrates information about the extension and position of one's own body [5, 29]. The need to evaluate the position and configuration of one's own body is implicit in the planning of any goal directed motor action [3, 18]. Body parts are moved relative to other body parts, and external targets could not be reached if the initial position of the body and its parts were not taken into account. A disturbance of the implicit calculation of the position and configuration of one's own body would manifest itself in misreaching and insecurity of all movements to external objects, and would have affected not only manipulation of the mannikin but also manipulation of the beads and the blocks.

The common disturbance of imitation on oneself and on the mannikin can neither be explained by a lack of explicit awareness of the position and configuration of one's body. Explicit awareness of the position and configuration of oneself is rarely called on when movements are directed to external objects. Consequently, if awareness is defective of only one's own body's configuration, this would lead to errors when gestures are demonstrated on oneself but not when they are demonstrated on a mannikin. If there is a common source to errors in imitation on oneself and on the mannikin, it must reside in the evocation and application of a general concept of the human body which applies regardless of whether one's own body is concerned or not.

The need to conceive one's own body as just one instance of a general concept of the human body is inherent to the task of imitating gestures, even if probed on oneself. Imitation of gestures has been said to test the integrity of a direct route from visual perception to motor control [26, 27], but on closer scrutiny it involves intermediate steps of considerable complexity. If the examiner sits opposite to the patient, the required movement is a mirror image of the perceived one. If, for another example, the examiner is taller than the patient, exact reproduction of their movement would overshoot the target on the patient's body. Studies in the physiology of motor control [3, 18] have demonstrated that planning of limb movements specifies target positions rather than movement paths. When gestures are to be imitated the target positions are not indicated directly but have to be inferred from a mental transposition of the demonstrated movement to oneself, and this transposition has to abstract from accidental determinants of the perceived movements as are, for example, the size or position of the demonstrating person. A feasible way to achieve this translation would be to conceptualize the perceived movements in their relationship to a general concept of the human body which applies irrespectively of the size and position of the body and irrespectively of whether the body belongs to the examiner, the patient, or a mannikin. In a way, the patient's body is then used as a mannikin on which the generally valid features of the movement are demonstrated. The apparently meaningless movements do have meaning insofar as they represent conceptual knowledge about the human body.

This interpretation does not necessarily imply that conceptual knowledge about the human body is a distinct category of knowledge which is affected selectively in ideomotor apraxia. It may be that the human body is just one instance of a multi-part mechanical object, and that the basic disorder concerns the apprehension of relationships between the functionally significant parts of such objects. Possibly, the crucial similarity between the wooden mannikin and the patient's body does not reside in their human shape but in the fact that both are multi-part mechanical objects. The present study did not test the manipulation of other objects with a mechanical complexity comparable to that of the human body. It may be that apractic patients would have had as many difficulties with them as they had with the mannikin.

Regardless of whether the basic disturbance concerns the spatial relationships between body parts or, more generally, the relationships between significant parts of multi-part objects, it resides at a conceptual level. The inability to evoke conceptual knowledge necessary for the planning of certain motor tasks has already been recognized as underlying other manifestations of limb apraxia. Faulty use of tools and simple objects has been related to the unavailability of knowledge about their appropriate use [6, 22, 23, 26], and faulty demonstration of symbolic gestures to the inability to evoke the appropriate shape of gestures when given their meaning [1, 8, 9, 12, 15]. If faulty imitation of movements is accepted as a conceptual disturbance too, none of the manifestations of limb apraxia is left to

correspond to a deficit of motor execution, that is, to the inability to translate a correct concept of the intended movement into an appropriate motor act.

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