

Imitation and utilisation behaviour

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Abstract

Objective—To investigate the incidence, anatomical correlates, and clinical features of imitation and utilisation behaviour, which are thought by Lhermitte and coworkers to represent a reliable and frequent index of frontal lobe disease.

Methods—78 patients with hemispheric focal lesions were tested in two separate sessions, in which their reactions to a series of gestures performed by the examiner and to the presentation of a set of objects were recorded. The patients were stratified into a frontal (n = 52) and a non-frontal group (n = 26) on the basis of their CT data.

Results and conclusions—Imitation behaviour was present in 39% of the frontal patients and was mainly associated with medial and lateral lesions, at odds with the claim of Lhermitte *et al* that it is a constant accompaniment of lower, mediobasal lesions. In the non-frontal group it was found in three patients, all with damage to the deep nuclei region. Utilisation behaviour was a much rarer phenomenon, present in only two patients, both of whom had frontal damage. Neither imitation behaviour nor utilisation behaviour were found in patients with retrorolandic cortical lesions.

(*J Neurol Neurosurg Psychiatry* 1996;61:396-400)

Keywords: frontal lobe, imitation behaviour, utilisation behaviour

The search for signs or test performances pointing to frontal damage has long been pursued by neurologists and neuropsychologists, but they remain elusive both in terms of sensitivity and specificity. The failure to find a consistent marker of frontal lesion, irrespective of its location, is hardly surprising, given the size of the lobe and its different functional specifications. More disappointing is the unsuccessful attempt to identify signs that are specific to frontal damage and not present in other brain locations. For instance, recent studies have challenged the claim that perseverative errors on the Wisconsin card sorting test are a reliable index of frontal lobe disorders, as their number was also increased in patients with temporal epilepsy¹⁻³ and failed to discriminate frontal from non-frontal patients with stabilised lesions.^{4,5} This lack of specificity is likely contingent on the complexity of the test,

which involves the participation of different abilities and makes its performance sensitive to damage to different brain areas.

An alternative way of overcoming these drawbacks is to focus on more elementary behaviours, such as those represented by the release of primitive, automatic responses to a stimulation. For instance, the grasp reflex, which is present in normal infants and is subsequently inhibited, has been found to have a remarkable degree of sensitivity to lesions encroaching upon the medial frontal and cingulate areas and to be absent in patients with retrorolandic damage.⁶

Lhermitte and coworkers⁷⁻⁹ have drawn attention to a peculiar syndrome exhibited by frontal patients, which manifests itself with a gamut of symptoms: imitation behaviour, utilisation behaviour, and environmental dependency syndrome. Imitation behaviour consists of the reproduction of gestures and utterances that the examiner makes in front of the patient without giving preliminary instructions. Admittedly, the fact that the examiner starts gesturing, without giving any explanation, makes the situation somewhat ambiguous and may lead the patient to the deduction that the examiner's peculiar behaviour was intended to elicit imitation. Indeed, this is the way patients tend to justify their behaviour when questioned. Lhermitte *et al*⁹ point out, however, that no normal subject imitates in this situation and that patients who imitate keep on doing so even when they are explicitly told to stop. Utilisation behaviour is elicited, firstly by provoking the grasp reflex and then by placing an object in front of the patients, without any comment. They react by using it and go on showing the same response to any new object that is presented—for example, water is poured from a bottle into a glass and drunk, an apple is peeled and eaten, spectacles are put on even if the patient is already wearing a pair, etc. The environmental dependency syndrome is a more elaborate manifestation of utilisation behaviour, in which the patient behaves in unfamiliar surroundings as if he or she were in charge of the situation and performs complex actions that are dictated not by his or her role, but by the cues provided by the environment.

Lhermitte *et al*⁷⁻⁹ claimed that these abnormal behaviours are a reliable index of frontal lobe damage. They tested imitation behaviour and utilisation behaviour in a series of 125 patients with various morbid conditions. Imitation behaviour was found in 28 out of 29 patients with frontal lesions, in three out of six

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Received 9 February 1996
and in revised form
10 May 1996

Accepted 16 May 1996

with deep structure lesions, and in only one of the 21 with rolandic-retorolandic lesions (moreover, the patient stopped imitating when told to do so). In the same sample, utilisation behaviour was present in 13 patients with frontal lesions, in two with deep structure lesions and in none with rolandic-retorolandic lesions. All patients with imitation behaviour also showed utilisation behaviour and the locus of the lesion shared by all the positive patients was the lower half of the frontal lobe.

These findings are exciting, in view of the contradictory picture provided by frontal markers tapping cognitive skills, but are in need of replication, because such a close correlation between a pathological sign and damage to a given cerebral area is unusual and would be amazing even with reference to more elementary neurological signs. So far only single case reports confirming the occurrence of utilisation behaviour in frontal and thalamic patients have been published¹⁰⁻¹⁵ and no systematic study has been carried out to verify the incidence of both behaviours in a new sample. The present investigation aims to fill this gap.

Materials and methods

PATIENTS

We tested 78 brain damaged patients admitted to the wards of the Neurological Department of the University of Modena, who met the following criteria: (a) ability to cooperate—namely, to pay attention to the examiner's behaviour and to understand elementary questions. It must be emphasised that the testing procedures (see below) required only minimal verbal instructions. (b) One or more focal hemispheric lesions, documented by CT. On the basis of imaging evidence, patients were divided into two groups, a *frontal* group, comprising patients whose damage encroached on the frontal lobe and a *non-frontal* group, in whom the frontal lobe was spared. If there were multiple lesions, but at least one of them was located in the frontal lobe, or if the lesion involved both frontal and non-frontal areas, the patient was classified as frontal. There were 52 frontal patients; CT was missing in one of them, who was, nevertheless, included in the frontal group as he had a classic anterior cerebral artery infarct. There were 26 non-frontal patients.

PROCEDURE

Testing was carried out in two sessions. The first took place at the bedside and aimed at screening patients who showed either imitation or utilisation behaviour. On the same or the next day, positive patients underwent the second testing session, which was carried out in the laboratory.

First session

After the neurological examination, the examiner, who stood in front of the patient and looked directly at him or her, performed the following gestures: (a) stroking his hair, (b) scratching his forehead, (c) yawning, (d) clap-

ping three times. If the patient imitated, at the end of the four items the examiner asked "why did you imitate me?" and if he or she replied "I thought that was what you wanted", the examiner said resolutely "I didn't tell you to". Then he went on with the following items: (e) humming a tune, (f) sighing, (g) saying "That's enough", and (h) "I'm tired today". After a brief pause, the examiner laid the following objects on the patient's bed one by one: (a) a comb, (b) a ring, (c) a padlock with its key, (d) a box of matches, and recorded the patient's behaviour. Patients who persisted in imitating notwithstanding the examiner's admonition, and/or handled the objects, underwent the second session of the test.

Second session

Utilisation behaviour was tested first. The patient sat in front of a table, at the two ends of which there were two trays, each holding five objects, while a book was in the centre. His or her behaviour was watched unobtrusively for a while, then the examiner sat down opposite the patient and repeatedly stimulated his or her palm to elicit the grasp reflex. Whatever the result of the manoeuvre, one by one the following objects were placed in front of the patient and remained in full view, without comments, for a few seconds: (a) a bottle of water with a glass, (b) a bell, (c) a sheet of paper with a pencil, (d) a pack of cards. If none of them was used, utilisation was no longer tested. If the patient handled and used them, at the end of the series the examiner said "Why did you use them? I didn't tell you to". He then gave the patient the first 12 items of the Raven coloured matrices. When the task was completed, the remaining six objects were removed from the tray and presented.

The subsequent part of the session concerned imitation behaviour. The examiner performed three gestures and then said the following two sentences "Winter is warmer than summer", "Today is . . . (day of the week, month, and year)". If the patient imitated, the first admonition was given "Why did you imitate me? I didn't tell you to" and followed by the administration of items 13-24 of the Raven test. A new series of four gestures and one sentence was given and, if they were imitated, there was a second admonition "I told you not to imitate me". Items 25-36 of the Raven test were given and followed by the last series of four gestures and one sentence.

Results

IMITATION BEHAVIOUR

When present, imitation was a massive and persistent behaviour that affected almost all of the items of the test and recurred in the second session in every patient who had shown it in the first session. An explicit command not to imitate could stop it, but the mere introduction of a pause, during which the patient's attention was drawn to a different subject, was sufficient to see imitation reappearing.

When questioned about their behaviour, patients who imitated (IM+) either seemed

puzzled and said nothing, or justified their actions by claiming that they thought this was the implicit request made by the examiner, sometimes going as far as to add "you told me to do it". Yet a mere misunderstanding can be excluded, as no patient (with one exception) stopped imitating when the examiner denied ever having made such a request or when he explicitly told them not to imitate. Sometimes their comments showed that they had not forgotten what they had been told (for example, "I remember that I made you angry yesterday when I repeated your movements") and were aware of the inappropriateness of their behaviour, which, nevertheless, resurfaced after a while. In a few patients there was evidence from the item where an implausible sentence was pronounced that imitation was not a passive echo of the examiner's performance, but involved at least a certain degree of decoding. To the sentence "winter is warmer than summer", they reacted by saying "No! It is summer that is warmer than winter".

There were 23 patients who showed imitation behaviour (IM+). Twenty belonged to the frontal group (39%) and three to the non-frontal group (11%). Table 1 reports the demographic and clinical features of the patients. The only variable that differentiated them was aetiology, due to the prevalence of vascular over neoplastic pathology in the IM+ group. This likely reflects the fact that an acute disease causes greater dysfunction than a more chronic disease. There was not a higher incidence of imitation behaviour in patients who had one or more lesions involving other lobes in addition to the frontal lobe, in comparison with patients who had a lesion confined to the frontal lobe. The first group represented 37% of IM+ patients and the second 35% of IM- patients. Grasping, a well known marker of frontal damage, was present in some cases, but the correlation with imitation was not close. In the frontal group it was found in 13 out of 20 IM+ patients and in 13 out of 31 IM- patients; in the non-frontal

group in four out of 26 patients (two of them also showed imitation).

To assess the relation between imitation and the locus of lesion, frontal patients were subdivided into three groups, depending on whether the disease was predominantly centred around the mesial, the lateral, or the orbital region. Following Damasio and Damasio,¹⁶ the *mesial region* comprised the cingulate gyrus (areas 23, 24, 31), the supplementary motor area (6), and the mesial parts of the prefrontal (8, 9, 10) and rolandic (4, 3, 1, 2) areas. The *lateral region* comprised the frontal operculum (44, 45), the prefrontal areas (8, 9, 46), the lateral parts of the premotor (6) and rolandic (4, 3, 1, 2) areas, and the paraventricular and supraventricular areas. The *orbital region* comprised the anterior (10), posterior (11, 12, 13, 47), paraventricular, and subventricular areas. A separate group comprised patients whose damage encroached largely on the *deep structures*, either with marginal or with substantial extension to the frontal lobe. Table 2 reports the outcome of this classification.

If we focus on the first four columns of table 2, where patients with damage mainly involving the three sectors of the frontal lobe are reported, no significant group difference emerges. It is, however, worth remarking that whereas no IM+ patient showed a predominant involvement of the orbital region, there were three such patients in the IM- group. The relation between orbital lesions and IM+ behaviours is not significant, even if we take into account patients in whom these areas were only marginally involved: there were six such patients in the IM+ group and two in the IM- group (to whom the three patients with massive orbital lesions must be added). This finding is at variance with the claim of Lhermitte *et al*⁹ that imitation behaviour is associated with damage to the inferior frontal lobe. This preferential location is also disproved by the inspection of the CT of patients with predominant medial and lateral lesions: in almost all of the first group and in most of those of the second the disease was confined to the upper part of the lobe.

The deep nuclei also seem to be related to imitation. In the frontal group they bore the brunt of the damage in 3 IM+ patients, plus a fourth patient, who also had extensive medial frontal damage. The structures involved by the lesion were the lenticular nucleus in all four patients, the head of the caudate nucleus in two patients, and the anterior limb of the internal capsule in one patient. Also the three non-frontal patients, who showed imitation, had damage confined to the deep structures. One had a left thalamic infarct, one a left sided haematoma of the lenticular nucleus and the posterior limb of the internal capsule, and one a left infarct of the head of the caudate and the lenticular nucleus. Their behaviour on the imitation test did not differ from that of frontal patients.

UTILISATION BEHAVIOUR

Utilisation behaviour was much rarer than

Table 1 Demographic and clinical features of the patients

Patients	Age (y) Mean (SD)	Sex		Aetiology			Side of lesion*		
		M	F	V	N	T	L	R	B
Frontal IM+ (n = 20)	64.6 (13.8)	9	11	11	8	1	11	6	3
Frontal IM- (n = 32)	59.6 (14.3)	14	18	9	23	—	15	9	8
Non-frontal (n = 26)	64.7 (14.5)	14	12	21	5	—	12	12	2

M = males; F = females; V = vascular; N = neoplastic; T = traumatic; L = left; R = right; B = bilateral.

*In patients with frontal lesions only the side of the frontal lesion is referred to.

Table 2 Distribution of the frontal patients in subgroups defined by the area mainly involved by the lesion

	MF	LF	MF+ LF	OF	DS	DS+ ML	DS+ LF	DS+ MF+LF
IM+ (n = 20)	7	3	6	—	3	1	—	—
IM- (n = 31*)	9	11	5	3	1	—	1	1

IM+ = presence of imitation; IM- = absence of imitation; MF = medial frontal; LF = lateral frontal; OF = orbital frontal; DS = deep structures with marginal involvement of the frontal lobe.

*CT was missing in one patient.

Table 3 Record of the utilisation behaviour shown by two patients with frontal lesion

Items and examiner's warnings	Patient 12	Patient 17
1 Two trays and one book	He reads the title aloud, turns over the pages, and reads each of the 10 chapter titles	No reaction
2 A glass and a bottle	He pours the water into the glass	No reaction
3 A bell	He handles and rings it	He handles, sniffs, and rings it
4 A pen and a sheet of paper	He handles the pen, leaves it, and handles it again	He takes the top off and asks: "What do I have to write?"
5 A pack of cards	He turns it upside down	He takes it and asks: "Do you want to play rummy?"
First warning	"You gave them to me, it was logical to think that I have to use them!"	"No?"
6 A packet of sweets	He handles and opens it, then looks at the examiner	No reaction
7 A parcel tied with strings	He handles and shakes it	He handles and looks at it
8 A candle and a matchbox	He lights the candle with a match	He takes the matchbox, looks at it, and scratches his thumb nail on the rough side, as if to light it
9 Aftershave	He handles it	He opens and smells it
10 A nut and a nutcracker	He touches both, then puts the nut in the nutcracker	He cracks the nut, complaining that he cannot eat it because he doesn't have his false teeth
11 A pencil and a pencil-sharpener	He touches both	He sharpens the pencil
Second warning	"It was logical to think I had to do it. I wanted to light the candle for a party!"	"I used them because they are here. You already told me not to use them!"

imitation behaviour. It was found in two patients only, both belonging to the IM+ frontal group. Table 3 reports their responses, which ranged from the mere purposeless manipulation of objects to their appropriate use. The second, however, only exceptionally resulted in a coherent activity that thoroughly exploited the functional properties of the object—for example, they poured water into the glass, but did not drink it, took the top off the pen, but did not write. Similarly to imitation behaviour, utilisation behaviour was not modified by the examiner's admonitions.

One patient had a small anterior cerebral artery infarct, encroaching on the cingulate gyrus and the other a haematoma, involving both the mesial and the lateral aspects of the frontal lobe and impinging on the subventricular area.

Discussion

The present study provides a substantial body of data pertaining to imitation behaviour. By comparison, the findings related to utilisation behaviour are too scant to permit reliable inferences.

We confirm the claim of Lhermitte *et al*⁹ that imitation behaviour points to damage of the frontal lobe, either bilateral or unilateral, or of the deep nuclei, but is never found after retrorolandic cortical lesions. The phenomenon is easy to detect, is consistent from session to session, and resists attempts to modify it, in agreement with the remarks made by Lhermitte *et al*.⁹ It would be incorrect to say that it completely escapes the control of volition, as it can be transiently inhibited by the explicit request not to imitate. However, the

fact that it re-emerges whenever attention is diverted and the examiner's admonition is no longer in the focus of consciousness, attests to its compulsory nature. Admittedly, the peculiar behaviour of the examiner, who performs gestures or utters sentences that are out of context and have no apparent motivation, is such as to puzzle patients and to raise queries as to what their reaction should be. It is not rare to perceive a questioning expression on the faces of patients who do not imitate and, very occasionally, they may even ask "what have I got to do?". It is likely that this equivocal situation plays a part in triggering imitation, but, once it has been released, it exerts a sort of magnetic attraction, resists any corrective cue, and dominates behaviour.

The present findings differ, however, from those of Lhermitte *et al*⁹ in a few, important respects. Firstly, the incidence of imitation in frontal patients was lower (39%) than that reported by the French authors, who found it in 97% of their sample, a percentage that, if truly representative of its incidence in the frontal population, would rank this behaviour higher than any other neurological sign in the detection of a focal brain lesion. There was no apparent difference in procedure that might account for this discrepancy and the most likely explanation is that the two samples differ in the selection criteria. We submit that a bias affected the sampling of patients by Lhermitte *et al*.⁹

Secondly, Lhermitte *et al*⁹ stressed that imitation behaviour was crucially associated with damage to the lower part of the frontal lobe, in particular to its mediobasal areas. This conclusion was based on a rather crude and loosely defined classification of frontal patients into those with damage to the upper and lower part of the lobe, which resulted in the finding that the second region was injured in all of the 26 IM+ frontal patients and the first in only 15 of them. Our data are at variance with these findings. We found that imitation behaviour was predominantly associated with damage to the upper medial and lateral frontal cortex, whereas it was absent in the three patients, in whom the brunt of the damage was borne by the orbital cortex. Fukui *et al*¹³ have also reported a patient with imitation behaviour and utilisation behaviour, who had upper damage (bilateral cingulate lesion).

In this respect imitation resembles the grasping reflex, which also shows a close relation to damage to the upper medial and lateral frontal cortex,⁶ although the association between the two signs is far from perfect.

As already mentioned by Lhermitte *et al*,⁹ there is a second region, covering the striate structures, the thalamus, and the adjacent white matter, that when injured can cause imitation with the same features found in patients with frontal lesions. Damage was confined to this region in the three non-frontal IM+ patients and predominantly affected it in three IM+ patients with frontal damage. Deep nuclei damage has been repeatedly reported to cause symptoms traditionally deemed to be within the province of cortical lesions (apha-

sia, apraxia, neglect, etc) and this association has been attributed to the deactivation of the cortical areas to which subcortical structures project. The question that remains unanswered is why only a few patients with deep disease manifest these abnormal behaviours.

Utilisation behaviour was found in two frontal patients (4%) and in none of the non-frontal patients and it took the form of purposeless manipulation (toying, according to the classification proposed by Shallice *et al*¹¹) more often than that of a coherent activity, congruent with the function of the object. Again its incidence was much lower in our sample than in that of Lhermitte *et al*,⁹ in which it was found in 13 out of 29 frontal patients and in two out of six patients with lesion of the deep structures. We do not know to what extent this difference may be contingent on variations in the methodology used to elicit the behaviour, but we could not detect any major variation, except for the fact that Lhermitte⁷ did not simply put the object on the table within the patient's reach, but moved it near to him or her. It seems likely that the crucial factor was the sampling criterion. Incidentally, the very fact that, after the paper of Lhermitte *et al*, utilisation behaviour has been reported in single cases of frontal damage¹⁰⁻¹⁵ seems to suggest that, in the authors' opinion, it was a behaviour so exceptional as to warrant publication and not a sign with an incidence affecting about 50% of frontal patients.

Utilisation behaviour has the same compulsive features that characterise imitation and likely reflects, as suggested by Lhermitte^{7,8} the patient's impaired control over his or her responsiveness to the environment, such that the mere presence of a stimulus, is sufficient to trigger the response independently of internal motivation or plans. The fact that utilisation behaviour is always associated with imitation behaviour, but not vice versa, suggests that utilisation reflects a more severe degree of impairment than imitation.

In this study no systematic assessment of the patients' cognitive skills has been carried out and, consequently, we are not in a position to say what neuropsychological variables differentiate patients with and without imitation behaviour and utilisation behaviour and to speculate on the underlying mechanisms. It is worth remembering that both imitation and

the tendency to grasp objects are behaviours universally found in babies and that they are instrumental in fostering the development of their social skills and their knowledge of the world. These primitive and undifferentiated reactions to the environment recede as the maturation of the frontal lobes provides the anatomical basis for inhibiting actions that would disrupt planned and goal directed behaviours. They re-emerge when the frontal control mechanism is lesioned.

In summary, of many aspects that the syndrome of environmental dependency may take, the one that deserves particular attention by the clinician is imitation behaviour. Its theoretical interest apart, it represents a valuable addition to the range of signs that must be sought when a frontal disease is suspected.

This research was supported by a grant from MURST funds to E De R.

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