

# Human Autonomy and the Frontal Lobes. Part I: Imitation and Utilization Behavior: A Neuropsychological Study of 75 Patients

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A type of pathological behavior, imitation behavior (IB), is newly described. In this behavior patients imitate the examiner's gestures, although not instructed to do so. Patients explain that they thought they had to imitate the examiner. IB is the first stage of utilization behavior (UB). Neuropsychological examination of 40 patients with IB, of 35 with UB, and of 50 disease controls demonstrates the existence of a frontal syndrome and two determining features of such behavior: dependence on (1) the social and (2) the physical environments. Loss of intellectual control was also required for the occurrence of such behavior. UB and/or IB were present in 96% of the 29 patients with focal lesions of the frontal lobes. Computed tomographic scans in 26 of these patients showed involvement of the inferior half of the anterior part of one or both frontal lobes. IB and UB are interpreted as release of parietal lobe activities, resulting from impairment of frontal lobe inhibition.

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The term *utilization behavior* (UB) has been used previously to describe a disturbance in responses to external stimuli [17, 18], which indicated an exaggerated dependency on the environment for behavioral cues. UB was correlated with lesions of one or both frontal lobes. In this report, a new type of UB-related behavior is described, which we propose calling *imitation behavior* (IB) because patients imitate the gestures and behavior of the examiner despite the fact that they have not been asked to do so, and continue imitating after being asked to stop. One hundred twenty-five patients with cerebral lesions (56 with focal lesions) were tested for IB and UB and formed the basis for a neuropsychological study, the aim of which was to pinpoint the pertinent features of these behaviors. Localization of lesions was attempted using computed tomographic (CT) scan images in 26 patients with frontal lobe lesions.

## Subjects and Patients

### Normal Subjects

More than 200 normal subjects (male and female) were examined. Their ages ranged from 25 to over 70 years for adults, and from 2 to 6 and 10 to 16 years for children.

### Patients

One hundred twenty-five patients with a definite diagnosis of cerebral lesions were tested for IB between November 1982 and June 1983. All patients underwent a complete neurolog-

ical examination and extensive neuropsychological and behavioral testing. Fifty-six patients exhibited focal lesions due to vascular disease, tumor, or trauma, and 69 patients had other disorders (Table 1). Patients with degenerative dementia were only mildly affected. All 125 patients were divided into three groups. Group I consisted of 40 patients with IB but without UB; Group II, 35 patients with UB and IB; and Group III, a control group of 50 patients in whom IB and UB were never observed. To limit the extent of this paper, we only report in detail the findings of focal lesion patients and summarize those of other patients. The general characteristics of focal lesion patients in Groups I, II, and III, respectively, were as follows: age:  $54.2 \pm 3.4$ ,  $55.1 \pm 3.6$ , and  $50 \pm 3.8$ ; sex: 6 men and 11 women; 9 men and 6 women; and 16 men and 8 women; and handedness: 15 right-handed and 2 left-handed; 14 right-handed and 1 left-handed; and 20 right-handed and 4 left-handed. The educational level of the patients with focal lesions in the three groups was as follows: elementary school: 11, 9, and 10; high school: 5, 5, and 10; and university level: 1, 1, and 4. The general characteristics of the other patients did not differ significantly from the three groups of patients with focal lesions.

## Methods

### Investigation of IB

The patient was seated opposite the examiner with or without a table in between. As in the investigation of UB [18], the examiner remained completely neutral and indifferent to the patient. He answered no questions and did not react to remarks made by the subject.

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Table 1. Cause and Localization of Lesions

Brain Area	Group I	Group II	Group III
FOCAL LESIONS (56 cases)			
Frontal	15 R: 6; L: 7; B: 2 T: 7; I: 3; H: 4; tr: 1	13 R: 4; L: 5; B: 4 T: 5; I: 5; H: 2; tr: 1	1 R: 1 T: 1
Deep structures	1	2	3
Retrorolandic	1	0	16
Pre- and retrorolandic	0	0	4
Total	17	15	24
OTHER LESIONS (69 cases)			
Alzheimer's disease <sup>a</sup>	4	10	5
Parkinson's disease <sup>a</sup>	5	3	7
Progressive supranuclear palsy	4	1	4
Chorea	0	0	0
Normal pressure hydrocephalus	2	1	0
Multiple vascular accidents	3	0	6
Others	5	5	4
Total	23	20	26

<sup>a</sup>See details in the text.

R = right; L = left; B = bilateral; T = tumor; I = ischemic cerebrovascular accident in the territory of anterior communicating artery or anterior cerebral artery; H = hematoma; tr = traumatism.

**EXAMINER'S GESTURES.** Only a few examples are mentioned. *Body gestures:* bending the head and resting the chin on the hand, tapping the leg with the hand in time to various rhythms, whimpering, kicking something or just making the movement, crossing the legs; *symbolic gestures:* thumbing one's nose, military salute; *gymnastic gestures; gestures involving objects* (all objects may be used): folding a sheet of paper and putting it in an envelope, eating various kinds of food, chewing paper, combing the hair; *language and sounds:* uttering short sentences even if untrue, singing well-known tunes; *writing and drawing.*

**INTERVIEW.** The interview started immediately if the patient was not imitating. If he was, it was preferable for the examiner to make some series of gestures before questioning the patient. The subject was told to use his memory and list all the gestures that the examiner had made. He was then asked why the examiner had made these gestures and why he had copied them. If IB was demonstrated, the examiner said he would repeat the gestures but, whatever he did, the subject was not to imitate him, unless verbally asked to do so. It was preferable to ask the patient to repeat the request. The examiner then diverted the patient's attention before starting the test again. If the patient continued to imitate the examiner's gestures, the examiner asked him the same questions and pointed out that the subject had been told not to imitate.

#### Neuropsychological Examination

Neuropsychological examination was designed to assess three points. (1) Mental deterioration was assessed using Raven's P.M. 47 (progressive matrices) and Wechsler's memory tests and by having the patient draw (by reproduction and from memory) the complex figure from Rey. (2) Frontal

syndrome was investigated using the following six tests: conceptual classification through similarities from the Wechsler Adult Intelligence Scale and Wisconsin card sorting [21] in its abridged form [22]; composition of a first story based on eight pictures and a second based on eight sentences, presented in random order [27]; verbal fluency tests; Jones-Gotman and Milner's adapted design fluency [15]; tests of the repetition of alternate sentences designed to bring out verbal perseverations [19]; and tests of the repetition of Luria's drawing series [19]. (3) Milder psychological disorders were distinguished by a special behavior scale, designed to assess the following features: apathy, restlessness, impulsiveness, indifference, euphoria, disinterestedness, cheerfulness, stereotypy, indifference to moral or social rules, dependence on the social environment, lack of attention, dependence on stimuli from the physical environment, programming disorders, personality disorders, and disorders of mental and emotional control. The importance of each of these sixteen features was assessed by the number of "true/false" answers to five items in each feature (eighty items in all). The answers were recorded taking into account medical observations, data collected by the psychologist, and information from the patient and his or her family. All results were analyzed statistically by Student's *t* test.

#### Anatomical Study of Frontal Lobe Lesions

In 26 patients with IB and UB who had a focal frontal lobe lesion, an analysis of the CT scan was undertaken to identify the critical areas in the appearance of IB and UB (1 patient from each of Groups I and II had an aneurysm of the anterior communicating artery and a normal CT scan). Three sections of the CT scan, inclined at 10 degrees to the orbitomeatal line (CML), were used at 6.5 cm (A), 3.6 cm (B),

and 3.0 cm (C) from the caudal plane. Three drawings were prepared for each patient. The drawings of the 26 patients were then superimposed according to each section.

## Results

### *Imitation Behavior*

**NORMAL SUBJECTS.** Normal subjects never imitated the examiner. They were unconcerned but surprised, without otherwise making the slightest remark. When the examiner asked them to list the gestures, they looked perplexed and answered correctly, often getting the order of the gestures wrong. When asked why the examiner had performed these gestures, they hesitated and replied: "To test me"; "I don't know—perhaps to see my reactions"; and so on. The answers varied little with personality or age. When the examiner asked them if it had crossed their mind to imitate him, their answer was: "No, not at all." Boys and girls between the ages of 12 and 16 reacted by laughing and calling the examiner a clown. Children between the ages of 5 and 6, from different ethnic communities and social surroundings (white, black, and Arab), were examined at their nursery school. All of them later told their teacher "The doctor was very nice, but it's funny how bad-mannered he is; he thumbed his nose at all of us." Children 2 to 4 years old sometimes took an object—a ball, for instance—and threw like the examiner, but they were merely playing with him.

**PATIENTS.** Seventy-five patients demonstrated IB (35 with and 40 without UB). Almost all patients imitated the examiner starting with the first gesture (Fig 1). For the others, a more abrupt gesture, or one not usually made during a medical interview (leg slapping), was enough to start the IB. All gesture sequences were imitated without surprise: the patients tried to follow as best they could the order they thought they had to obey. No patient ever forgot a detail of gestural sequence (e.g., when lighting a candle, he would always blow the match out). If the gestures were not easy to perform, the patient adapted himself perfectly to overcome the difficulties. Male patients even imitated such socially unacceptable gestures as using a urinal, or urinating against a wall, in front of 20 or 30 people. Some of them smiled when imitating unusual gestures, (kneeling as if to receive a blessing or putting on eyeglasses when already wearing some).

Several patients refused to imitate. They indicated that they considered the gesture ridiculous, or did not want to perform it (e.g., a patient who wore a wig refused to comb his hair). During the test involving the repeating of sentences, some patients expressed their disagreement but, afterwards, repeated the stimulus statement. For example, in answer to the stimulus "I prefer winter to summer," a patient said: "Oh, no! I like summer very much" (with a personal inflection)

and then "I prefer winter to summer" (without inflection). When interviewed after an examination, all patients could remember the examiner's gestures and, when questioned as to the reason for their imitative behavior, replied that because the examiner had made the gesture, they felt they had to imitate him. On being told that they had not been told to imitate the gestures, their answer was that obviously since the gestures had been made, they must be imitated. After being told not to imitate, most patients displayed the same IB. The others complied or adopted an attitude somewhere in between, with the attitude that they were supposed to imitate. Sometimes they asked if they should imitate and then imitated the gestures.

### *Neuropsychological Results*

**FOCAL LESIONS.** The occurrence of IB and UB was not influenced by sex, education, or handedness. Group I and II patients were older, though not significantly so. However, this factor was taken into account for the purpose of accuracy in the statistical calculations. Test scores in Groups I and II showed moderate deterioration of intelligence and memory compared with Group III (Table 2), but were not significantly different between Group I and II patients. It is noteworthy that all three groups contained patients with normal scores. Specific tests showed a frontal syndrome in Groups I and II: in the Wisconsin card-sorting test, the number of criteria was smaller and perseveration on a distinct criterion greater in Groups I and II than in Group III; likewise, in Luria's sequence test, the graphic perseverations were greater in Groups I and II than in Group III. The scores of the tests showed a moderate frontal syndrome in Groups I and II according to the clinical data.

In the behavioral scale, both Groups I and II had a significantly higher score for stereotypy, indifference, disinterestedness, indifference to social rules, apathy, programming disorders, loss of intellectual control, and dependence on the social environment (Table 3). Dependence on the physical environment was noteworthy: answers in Group I were the same as in the control group (Group III), and the scores of Group II were significantly higher.

**OTHER LESIONS.** The overall results of the 69 patients (Table 1) categorized as having other lesions were similar to those of the focal lesion groups. Some differences were observed. (1) Disturbances in the Wisconsin card-sorting test were more significant in Group III patients with other lesions than in Group III patients with focal lesions. (2) The graphic perseverations in Luria's sequence test and the reduction of verbal fluency were statistically more severe in Group II than in Group III patients with other lesions. (3) In the behavioral scale, Group I and II patients with other



A



B



C



D

*Fig 1. Imitation behavior. (A) Threatening gesture. (B) Putting on spectacles. (C) Combing hair. (D) Smelling a flower. (E) Kneeling in prayer.*

lesions had a statistically significant increased scoring for stereotypy, apathy, programming disorders, loss of intellectual control, and dependence on the social environment compared with Group I and II patients with focal lesions; likewise, dependence on the physical environment was statistically significant in Group II when compared with Group I and III patients with other lesions; the only differences were the severity of the neuropsychological disturbances in Group III (versus the focal lesion patients in Group III) and the more severe loss of attention in Groups I and II than in Group III, which was not observed in the three groups with focal lesions.

In all 43 patients with IB or UB, a frontal syndrome was observed by clinical examination and neuropsychological tests, but no temporoparietal clinical disturbances were noted. In the 5 patients with Alzheimer's disease with aphasia, apraxia, or Balint's syndrome (Group III), neither IB nor UB was present. In the 8 patients with Parkinson's disease with IB or UB in Groups I and II, mental deterioration was noted; this was not so in the 7 patients in Group III. The patients with supranuclear progressive palsy displayed mental deterioration.



E

#### *Pathological Results*

**FOCAL LESIONS.** IB with or without UB was present in 28 of the 29 patients with frontal lobe lesions (96%) (see Table 1). The only patient without IB or UB exhibited a right frontal glioma with headache as the only symptom; IB appeared 3 weeks later. Topograph-

Table 2. Estimation of Intellectual and Memory Efficiency and of the Frontal Syndrome in Patients with IB, UB, and Controls

	Group I (IB)	Group II (UB)	Group III (controls)
Intellectual and memory efficiency			
PM 47	23 ± 2.5	19 ± 2.8	25.6 ± 1.9
Complex figure of Rey	24.5 ± 3.1	22 ± 3.5	30 ± 2.3
Wechsler memory test	78.2 ± 4.7	80.7 ± 6.4	84.9 ± 4.7
Frontal syndrome			
Wisconsin card sorting			
Number of criteria	1.7 ± 0.4	1.1 ± 0.4	2.5 ± 0.4
Number of patients with perseveration	8 <sup>a</sup>	8 <sup>a</sup>	2
Graphic perseverations	1.2 ± 0.2 <sup>a</sup>	1 ± 0.2 <sup>a</sup>	0.6 ± 0.1
Verbal fluency	15.1 ± 2.4	12.8 ± 2.5	17.4 ± 2.2
Similarities	6.5 ± 1.3	5.2 ± 1.1	6.9 ± 1.1
Verbal perseverations	1.4 ± 0.2	1.2 ± 0.2	1 ± 0.1
Composition of stories	1.4 ± 0.3	0.9 ± 0.4	1.8 ± 0.3
Graphic fluency	1 ± 0.5	0.7 ± 0.5	1.5 ± 0.5

<sup>a</sup>*p* < 0.5 for Group I and Group II compared with Group III.

Results are expressed as means ± SEM.

IB = imitation behavior; UB = utilization behavior.

Table 3. Behavioral Scale of the Three Groups

Behavioral Scale	Group I	Group II	Group III
Restlessness	0.9 ± 0.3	0.9 ± 0.3	1 ± 0.3
Impulsiveness	0.9 ± 0.3	1.1 ± 0.3	1.1 ± 0.3
Euphoria	1.4 ± 0.3	1.1 ± 0.3	1.1 ± 0.2
Cheerfulness	1.1 ± 0.3	1.3 ± 0.3	1.3 ± 0.4
Decrease in attention	3.8 ± 0.3	4.1 ± 0.3	3.3 ± 0.4
Apathy	3.3 ± 0.4	3.3 ± 0.5	1.4 ± 0.4
Indifference	2.5 ± 0.3	3.2 ± 0.4	1.3 ± 0.4
Disinterestedness	2.2 ± 0.4	2.6 ± 0.5	1.1 ± 0.2
Stereotypy	3.2 ± 0.3	3.2 ± 0.4	2 ± 0.3
Indifference to social rules	1.4 ± 0.3	1.9 ± 0.3	0.4 ± 0.2
Dependence on the social environment	3 ± 0.4	3.5 ± 0.5	1.6 ± 0.3
Dependence on the physical environment	0.9 ± 0.3	2.1 ± 0.4	0.8 ± 0.2
Programming disorders	3.2 ± 0.4	3.6 ± 0.4	1.9 ± 0.4
Loss of intellectual control	3.5 ± 0.4	3.9 ± 0.3	1.9 ± 0.4
Loss of emotional control	1.4 ± 0.3	1.6 ± 0.3	2 ± 0.4
Personality disorders	1.6 ± 0.4	1.9 ± 0.4	2.1 ± 0.3

Results are expressed as means ± SEM. ---→ *p* < 0.05; —→ *p* < 0.01.

ical analysis by CT scan in 26 patients (Figs 2, 3) indicated that the lower half of the frontal lobe was affected in all patients, while the upper region was affected in only 9 of 14 patients in Group I and 6 of 12 patients in Group II.

IB with or without UB was present in 3 patients with deep lesions: a right capsulothalamic hematoma in Group I; a bilateral infarction in the caudate nucleus and anterior arm of the internal capsule, and a left capsulothalamic hematoma in Group II. In Group III, the lesions were a left posterior thalamic hematoma, a posterior thalamic glioma, and a pedunculothalamic infarction.

Neither IB nor UB was present in 20 of 21 patients with rolando-retrorolantic focal lesions. The patient with IB was doubtful: She exhibited a Wernicke's aphasia and a state of excitement, and wanted to show the examiner that she could understand and do anything. She stopped imitating immediately after being told to do so.

## Discussion

These observations confirm and extend the previous studies on UB [17, 18]. IB and UB are both manifestations of a basic disorder and differ only in severity. They reflect an imbalance in the patients between dependence on and independence from external stimuli, which leads them to become dependent on these stimuli. The sight of a movement is perceived in the patient's mind as an order to imitate (Fig 1); the sight of an object implies the order to use it. Intellectual deterioration was moderate and not significantly different in the three groups and could therefore not explain

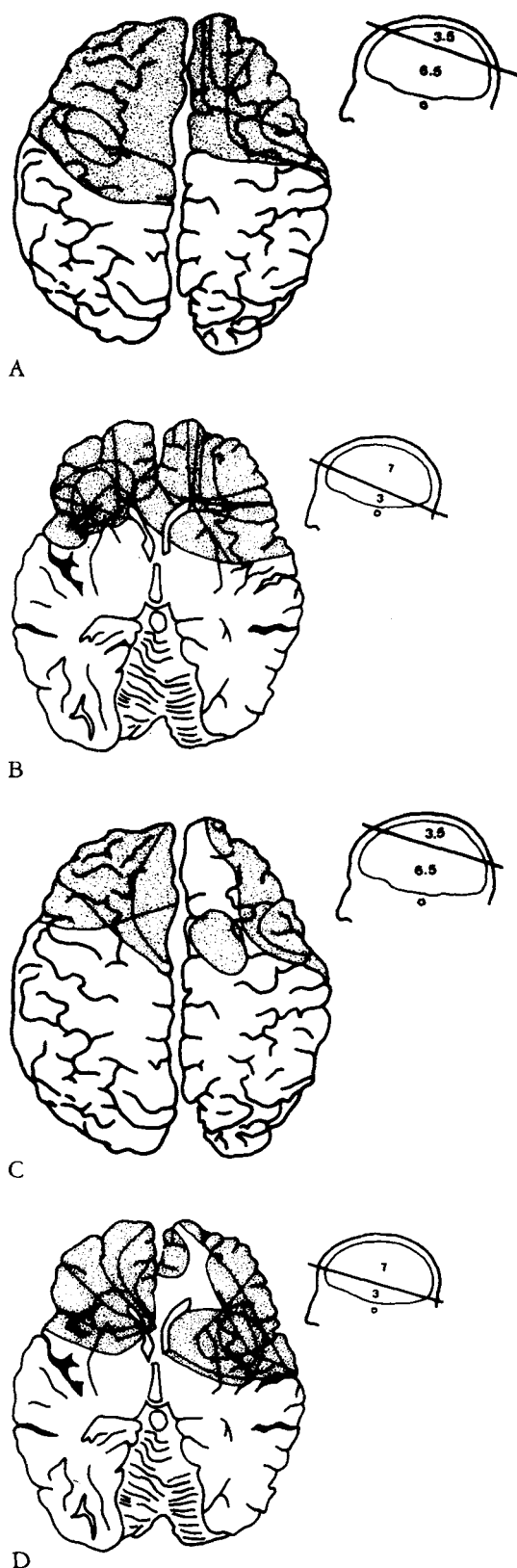
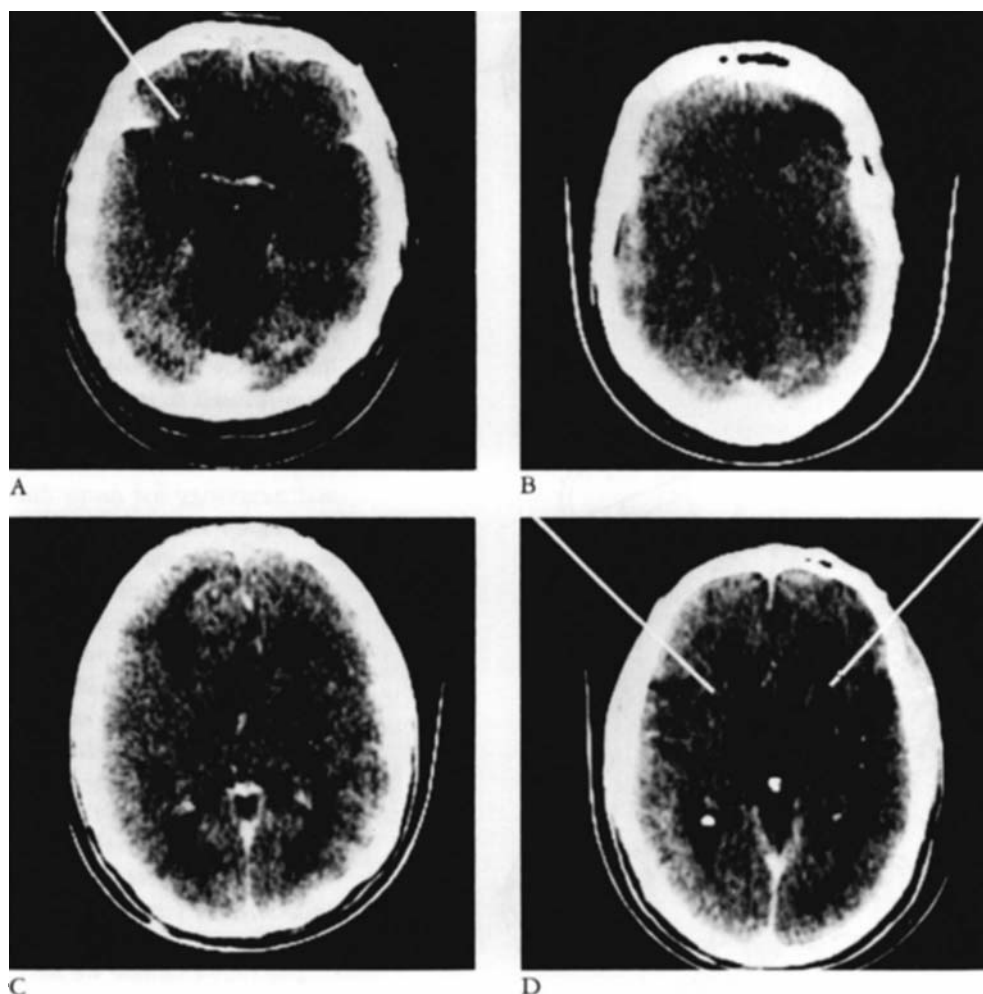


Fig 2. Superimposed drawings of the pure frontal lesions from the computed tomographic scan. (A and B) Cases of imitation behavior; (C and D) cases of utilization behavior.

IB or UB. Several patients continued to lead an almost normal life, although they rarely continued their jobs. One patient with an astrocytoma of the right frontal lobe carried on working in the post office, even though IB had been observed for more than 2 years. Furthermore, several women suffering from an astrocytoma (right or left frontal lobe) continued doing domestic chores. IB is an integral part of the conscious awareness of the patients; it is a voluntary act, not an automatic or reflex response. The patients thought they had to imitate the examiner, but were critical of the inadequacy of some of their gestures and in some cases refused to make them or even to try. This is why IB is quite different from the classic *echolalia* and *echopraxia* that have been common terms in psychiatry and neurology for more than a century. According to Dromard's study in 1905 [6], echopraxia is "an impulsive or automatic imitation of other people's gestures, an imitation which is performed immediately with abruptness and speed of a reflex action. . . . No intellectual or voluntary process is involved in its objective representation and fulfilment. Irrespective of whether the gesture is natural or bizarre, helpful or dangerous, it is invariably reproduced." It has been observed in extreme cases of dementia [9], and in cases of dementia praecox [16]. In the pathology of tics [12], echopraxia is a completely automatic reaction and occurs without the patient even being in an examiner-patient situation; the patient criticizes his own actions, but cannot refrain from acting.

The only example we have found of IB was a case of a female patient with palilalia reported by Dupré and Le Savoureux [8]. The authors describe her behavior as "an almost continual repetition of gestures and postures, which were performed *in front of the patient*. . . . When we drew the subject's attention to her passive obedience, she was aware of her gestures and, as is usually the case, she explained them by saying that she thought she was being ordered to perform such and such gesture or to assume such and such a posture." This case was considered to be pseudobulbar palsy, but today it would probably be diagnosed as progressive supranuclear palsy.

Normal subjects, including children, made no attempt to imitate; nor did they think they had to. It was only by resorting to suggestion that pseudo-IB and pseudo-UB were provoked. One normal subject was examined with the aid of an accomplice, who imitated what the examiner did but she still neither imitated nor used anything. Another normal subject was examined with two such accomplices. She watched with amazement as the two imitated the examiner's gestures, and after about a dozen gestures, began to imitate the gestures and grip and use objects. When questioned later, she said that she had done the same as "everyone else" because she was ashamed of just sit-



*Fig 3. Focal lesions of the frontal lobe. (A) Right frontal metastasis in a 57-year-old man. (B) Left frontal astrocytoma surgically removed from a 26-year-old woman. (C) Right frontal astrocytoma in a 24-year-old man. (D) Infarction of the head of the caudate nucleus and the anterior limb of the internal capsule in a 55-year-old man.*

ting there, being made to feel “guilty” about showing that she did not understand what was going on. This shows to what extent the behavior of patients with IB and UB differs from that of normal subjects.

In IB and UB, patients are abnormally dependent on the environment. Two features of the behavioral scale are directly implicated (see Table 3): dependence on the social environment, which was significantly aggravated in both UB and IB patients, and dependence on the physical environment, which was not apparent in IB patients but was severe in UB patients. The first feature should be understood as necessarily solicited or stimulated in undertaking an action. The second represents a tendency for the patient to be attracted by any stimuli from the outside world that would drive him or her to act without being asked. A third feature, the “loss of intellectual control,” produced neither IB nor UB but reflected the patients’ lack of self-criticism in restraining purposeless gestures and imitating ridiculous or socially unacceptable acts. The other features, which were increased in IB or UB, were characteristic of a frontal syndrome but were probably not determinant in these behaviors (Tables 2 and 3). The reactions

of patients totally cured of their frontal lesion can be summarized as one of surprise. They were perplexed when recalling their IB and UB, and the fact that they had no controlling thoughts of their own. This last feature is undoubtedly due to apathy, disinterest, and indifference, as measured by the behavioral scale.

IB appears first in that the patient becomes dependent on the examiner’s gestures and the social environment while still remaining independent of objects and the physical environment. Physical dependence (UB) appears later, while social dependence (IB) persists. During recovery, as the patient improves from the frontal syndrome, UB disappears before IB. In cases of worsening of the frontal lobe lesion, IB may disappear before UB because (as a result of apathy) patients

lose interest in the examiner's gestures but are still stimulated by objects.

#### *Anatomical Data and Physiopathological Considerations*

Frontal lesions appear to be of fundamental importance in all cases of IB and UB. These behaviors were observed in 28 of 29 cases of focal frontal lesions (96%) and in only a single questionable instance in 21 cases not affecting the prefrontal areas (4 cases of extensive pre- and retrorolandic lesions and 17 cases of focal retrorolandic lesions). The topographical analysis of the frontal lesions (26 cases) indicated that the inferior half and mediobasal area of the frontal lobe were always affected (see Figs 2, 3). This is in agreement with the only case that has been studied anatomically (bilateral infarction of the Heubner artery territory) [18] and with the frequency of ischemic accidents in the territory of the anterior communicating and anterior cerebral arteries (8 cases). UB and IB appeared as secondary dysfunctions of the frontal lobe in patients exhibiting lesions of the deep structures (see Table 1). The dorsomedial nucleus of thalamus, which was affected in 2 of 3 patients, has projections from its pars magnocellularis (inferior thalamic peduncle) to the orbital areas and from its pars parvocellularis (anterior thalamic radiations) to the dorsolateral areas [1, 2]. In the third patient, the inferior thalamic radiations were bilaterally affected in the anterior limb of the internal capsule; the head of the caudate nucleus was also destroyed by the infarction, but this lesion can probably be excluded from the genesis of IB and UB, as the lesions seen in the 2 previous patients involved neither the head of the caudate nucleus nor its afferent or efferent fibers. In the 3 patients without IB or UB (Group III), the thalamic lesions were probably posterior and spared the dorsomedial nucleus.

It has been suggested [18] that UB is caused by impairment of the inhibitory action of the frontal lobe on the parietal lobe, thereby releasing parietal lobe activity. The same hypothesis may be applied to IB. The presence in human beings of a parietofrontal connection via the superior longitudinal fasciculus has been known for some time [23]. It links the associative parietal areas with a large part of the prefrontal areas (including the orbitofrontal areas). Thus, it is unrelated to the multiple interconnections between the anterior part of the parietal lobe and the rolando-prerolandic area which constitutes the physiological basis for the motor system. Studies in monkeys using the Nauta technique show that there are numerous projections to the prefrontal areas from the parietal lobe [14]. These projections are situated in areas 8, 9, 10, 44, and 45 [24]. Physiologically, all these connections should respect the rule of reciprocity [1]. Lesions of the orbital cortex did not result in major changes in the parietal lobe [24]. These findings have been confirmed and

extended by studies using horseradish peroxidase, which indicated that lesions of the associative parietal lobe resulted in changes in areas 8, 45, and 46; in the nucleus basalis; and in the reticular system [13, 20]. These findings may be extrapolated to the human brain, bearing in mind the special features and developments found there, such as the great development of the parietal and frontal lobes, the anatomophysiological reorganization, and the presence of new structures. The inhibitory influence of the frontal lobes has been shown physiologically in monkeys [3, 5, 11, 25] and in human beings [4, 10, 19]. These influences include inhibition of inappropriate motor activities by acting on effector mechanisms, inhibition of internal behavior and impulses that tend to produce the motor activities, and inhibition of responses to disturbing or irrelevant stimuli [11]. In humans, various disorders caused by prefrontal lesions, especially stereotypes, may be explained by a defect in the inhibitory activity of the frontal lobe on cortical patterns elaborated at a distance from this lobe. According to the reported anatomical case [18] and to the anatomical data obtained from the CT scan images, frontal lesions related to IB and UB are located mainly in the inferior part of the frontal lobe. In contrast, we can suppose that the frontoparietal connections may be scattered over a large portion of the frontal lobe, with their density increasing from the superior (or middle) to the inferior part of the lobe.

The neuropsychological interpretation of IB and UB can be summarized as follows. All information coming from the body and outside world is collected in areas of the sensory cortex, and systems developed in the parietal cortex are responsible for integrating the unending sequences of stimuli. As a result, normal activity of the parietal lobe tends to create links of dependence between the subject and stimuli from the outside world, while some of the functions of the frontal lobe have an inhibitory effect on the parietal lobe. In normal subjects, the equilibrium between these two activities is dynamic, so that the subject's behavioral dependence on or independence of the outside world is a function of the quality of the external stimuli and of the subject's internal mental activity. Frontal lobe damage results in liberation of the parietal lobe activity, leaving the patient subject to all external stimuli. Actually, IB and UB were never observed in the cases of frontal lobe lesions with disseminated cerebral lesions that involved also the parietal lobes (metastases).

The mental disorders that result from unilateral frontal lobe lesions are quite different from disorders of higher functions (speech and visuospatial activity) that are brought on by lesions of specialized areas of the cerebral cortex that exhibit hemispheric dominance. Although there are disturbances of ipsilateral activities (dynamic aphasia and visuospatial disorders)



that may result from lesions of one frontal lobe, the major effect of these lesions is a disturbance in cognitive or emotional behaviors, the expression of which depends on the whole brain. In cases of IB and UB, the most frequent frontal lobe lesions were unilateral (22/28), without relation to the hemispheric dominance (right, 10; left, 12). There are two possible ways in which loss of autonomy as seen in IB or UB may result from a unilateral frontal lesion. First, the modifications of frontoparietal activities induced by the ipsilateral frontal lesion may cause functional disturbances in the frontal and parietal lobes of the normal hemisphere via the corpus callosum. Second, the lesion of one frontal lobe may induce changes in the reticular system as a result of the multiple connections that exist between the frontal cortex and the reticular system. The efferent fibers of the latter may then modify the activity of the normal frontal lobe. These two explanations are not mutually exclusive. Both are concerned with dysfunction that occurs some distance from the structural lesion, and may make it possible to understand the improving of IB and UB when the lesion is unilateral and nonprogressive. In bilateral frontal lesions, behavior is reorganized at a lower level, and we do not know if IB and UB persist or disappear with time.

It is difficult to interpret the lesions in degenerative diseases without pathological study. However, neurological examination and psychological testing always disclosed a pure frontal syndrome in cases of Alzheimer's (or Pick's) disease with IB or UB, and only a temporoparietal syndrome in those cases that did not show IB or UB. The consistent appearance of IB and UB in both progressive supranuclear palsy and Parkinson's disease with mild dementia is particularly striking, because a common biochemical disorder and a lesion of the nucleus basalis [7] are considered to underlie the dementia in both diseases. The frontal projections of the nucleus basalis can probably explain the frontal syndrome, IB and UB; however, this nucleus also projects to other areas of the cortex. It is noteworthy, in this context, that neither IB nor UB was observed in patients having dementia associated with Huntington's chorea. From a clinical point of view, it is of interest that IB and UB were observed in all cases of normal pressure hydrocephalus and were one of the earliest disturbances.

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