'UTILIZATION BEHAVIOUR' AND ITS RELATION TO LESIONS OF THE FRONTAL LOBES

by F. LHERMITTE

(From the Clinique de Neurologie et de Neuropsychologie, Hôpital de la Salpêtrière, 47, Boulevard de l'Hôpital, 75013 Paris, France)

SUMMARY

A new type of behaviour, termed 'utilization behaviour', was observed among patients affected with left or right unilateral, or bilateral, frontal lesions. It is an extension of bilateral manual grasping behaviour (magnetic apraxia). The tactile, visuotactile and visual presentation of objects compels the patients to grasp and use them. This behaviour was obtained with miscellaneous utilitarian objects. For the patients, the presentation of objects implies the order to grasp and use them. It is proposed that the balance between the subject's dependence on and independence from the outside world is disturbed. With frontal lesions, the inhibitory function of the frontal lobes on the parietal lobes is suppressed. The result is a release of the activities of the parietal lobes so that the subject becomes dependent on visual and tactile stimulation from the outside world. Five cases are reported as examples: one anatomoclinical case with bilateral lesions of the frontal lobes, one case with lesions in the left frontal lobe and three cases with lesions in the right frontal lobe. The role of lesions affecting different parts of the frontal lobes is discussed. The neuropathological observations lead to the suggestion that lesions of the orbital surface of the frontal lobe, and perhaps of the head of the caudate nucleus, are responsible for this behaviour.

INTRODUCTION

Wilson and Walshe (1914) had already observed the grasping reflex before Adie and Critchley (1927) described 'forced grasping' and 'groping' in relation to lesions of the contralateral frontal lobe. Denny-Brown (1956, 1958) used the term 'magnetic apraxia' for more elaborate manual activity in which the patient tries to grasp objects presented by the examiner as tactile and visual stimuli. In most instances, this activity is unilateral. Denny-Brown (1958) interpreted this as a release of parietal lobe activity following suppression of inhibition by the frontal lobe. Other authors have also observed it after unilateral lesions (Castaigne et al., 1961) or bilateral frontal and diffuse lesions of the cerebral cortex (Tissot et al., 1975).

In this paper, the proposal is made that bilateral magnetic apraxia corresponds to the release of parietal psychomotor behaviour, but at a higher level than suggested by Denny-Brown (1958). The expression 'manual grasping behaviour' seems more
appropriate. Our purpose is chiefly to describe and interpret a new aspect of the
description resulting from lesions of one or both frontal lobes: the presentation of
everyday objects within reach of the hands or in the field of vision impels the patient
to grasp and use them. We suggest calling this ‘utilization behaviour’.

PATIENTS

Initially, in 1968, whenever utilization behaviour was observed, the same behaviour was examined in
a normal subject and in another patient suffering from a cerebral lesion that did not affect the frontal
lobes. This study has since been performed in more than 100 normal subjects and in a considerably
greater number of patients. The evaluation became routine during clinical examination of all patients
with cerebral lesions. The details of the examination procedure are discussed later under Methods and
Results.

We at first began classifying cases of utilization behaviour due to bilateral frontal lesions. As the
years elapsed, we observed this behaviour so frequently in cases when (but only when) there was
bilateral manual grasping behaviour (magnetic apraxia) that documentation of bilateral frontal lesions
was discontinued. From 1968 until March 1982, more than 40 cases of utilization behaviour with
bilateral lesions of the frontal lobes were observed (Alzheimer’s disease, frontal glioma considered to
show bilateral involvement, surgically treated aneurysms of the anterior communicating artery). In
Alzheimer’s disease, we have only taken into account patients in whom neuropsychological
examination revealed the existence of frontal signs (apathy, nonfluent aphasia, disturbances in the
sequencing of gestures, pseudoamnesia and sphincter disorder), with no signs of right or left
parietotemporal affection (such as ideomotor and ideational apraxia, amnestic or conduction aphasia,
left-sided spatial neglect). In this article we describe the case of a female patient with bilateral frontal
lesions, chosen because a neuropathological study was undertaken, and 4 cases of unilateral frontal
lobe lesion (one left-sided, 3 right-sided).

CASE HISTORIES

Case 1 (No. 36.675)

A right-handed social welfare inspector, was aged 62 years when she developed, on January 4, 1975,
a subarachnoid haemorrhage with sudden headache and loss of consciousness for about 10 min,
followed by disorientation and incontinence. The CSF was haemorrhagic and angiography revealed a
large aneurysm connected by a small neck to the second segment of the right anterior cerebral artery or
the anterior communicating artery. The only previous history was of a sudden episode of loss of
consciousness for 20 min, without convulsions but with urinary incontinence nine years before. The
patient was operated upon on January 29, 1975 (Professor Pertuiset). Since the rupture made ablation
of the aneurysm impossible, a Yasargil clip was applied to the neck. The right frontal lobe was
obviously damaged over an area of roughly 2 cm². Post-operative recovery was uneventful, apart from
a severe frontal syndrome which continued to worsen (somnolence and incoherent speech, together
with urinary and faecal incontinence), because of which a ventricular CSF shunt was inserted on
March 14, 1975.

One month later, neurological examination revealed difficulty in maintaining upright posture
because of retropulsion. Muscular power was normal. The tendon reflexes of the upper limbs were
brisk. The plantar responses were flexor. Vision and sensation were normal. The sphincter difficulties
had subsided. The remainder of the examination was normal. Language was not aphasic and gestures
were normally performed. Conversation was unimpaired and psycholinguistic achievement was
normal. Reproduction of a story was normal; narration of ‘Little Red Riding Hood’ was also normal, apart from some perseveration. Luria gestures and drawings were correctly executed, with few perseverations. Memory for past and recent events was normal; the patient gave precise information as to her surgical operations. She remembered correctly stories that she had been told several minutes before. She knew she was in the Salpêtrière Hospital. However, she was unable to remember the dates of the days when she was examined. She made mistakes in mental calculations.

Bilateral manual grasping behaviour was obvious. Utilization behaviour was obtained for a glass and a jug of water, an apple and a knife, a banana, bread, a nail, a plane and a hammer. Moreover, when she took hold of a plate, a knife and a fork, she imitated the gestures of cutting the food and bringing it to her mouth. This behaviour rapidly declined and then ceased. One month later, the frontal syndrome had completely disappeared. Memory was considered as ‘excellent’, but mentally she was somewhat slow. Later, the patient wished to return to work. She had two epileptic attacks, but these have not recurred since treatment with phenobarbitone.

An EEG (May 5, 1975) revealed prominent slow waves in bilateral anterior corticosubcortical areas, predominantly on the right side. An isotope gamma scan showed the existence of a right corticosubcortical anterior frontal lesion, 3 cm long and 2 cm deep. General examination was normal, with the exception of a variable elevation of blood pressure.

Fig. 1. Case 1. Inferior aspect of the frontal lobes with the neurosurgical scar involving lateral cortex of the right frontal lobe.
This patient was not observed in the course of the ensuing months. We were informed that in the Spring of 1976, she had several ischaemic vascular episodes involving the right and left hemispheres. She was admitted to the neurosurgery department (Professor Pertuiset) where she died because of a deterioration in her general state and bronchopneumonia. The postmortem examination was performed by Dr F. Gray (Charles Foix Laboratory).

Macroscopic examination of the brain (1100 g). A cavity related to the surgical operation was found in the right orbital lobe (fig. 1). Old ischaemic lesions had destroyed the head of the caudate nucleus on both sides and extended on the left to the entire territory of Heubner's artery. The corpus callosum showed a haemorrhagic lesion in its midportion and was seriously atrophic anteriorly. Several recent infarcts were observed in the territories of the right and left middle cerebral arteries and in the left posterior cerebral artery. A clip was found on the neck of the aneurysm in the anterior communicating artery. No aneurysm remained. The initial segments of the anterior cerebral arteries were shrunken but unobstructed; the right pericallosal artery was atrophic and occluded.

Microscopic examination of the brain. The operation site appeared as a necrotic cavity affecting both orbital lobes, but larger on the right (fig. 2a). Numerous old ischaemic lesions, certainly present at the time of operation, were observed (figs. 2b and c) in the territory of Heubner's artery on both sides and in the deep and anterior territory of the left middle cerebral artery. The corpus callosum showed numerous necrotic foci, in particular in its anterior part which was atrophic. The white matter of the anterior part of the centrum ovale of the two frontal lobes showed diffuse demyelination, sparing the U fibres (fig. 2a). The latter was related to the necrotic lesion of the corpus callosum, hydrocephalus and numerous foci of lacunar necrosis. Recent ischaemic lesions were observed in the territories of both middle and the left posterior cerebral arteries. The mamillary bodies, both Ammon horns, the thalamus and the posterior part of the cingulum were normal. There were diffuse vascular lesions, with hyaline arterial degeneration and dilatation of the perivascular spaces together with macrophages that often contained haemosiderin.

Case 2 (No. 30.785)

A right-handed male Post Office worker, aged 35 years, had a generalized epileptic seizure on October 11, 1973 without premonitory phenomena and without any postictal deficits. The patient had no previous history of neurological disease and was asymptomatic. Neurological examination was normal. An EEG (October 12, 1973) revealed intermittent paroxysmal slow activity, particularly in the temporal region, obviously greater on the left. An isotope gamma scan showed a focus of increased uptake in the left hemisphere. Left carotid angiography (fig. 3) revealed a superficial left frontal arteriovenous vascular malformation, anterior to the ascending frontal gyrus, on F2. The dilated afferent arteries were anterior branches of the left middle cerebral artery; the malformation was drained by two dilated veins, running into the superior longitudinal sinus. Further selective angiography was performed through the femoral artery under general anaesthesia. The two anterior cerebral arteries and their branches, the meningeal branches of the left external carotid and the left posterior cerebral artery were normal and were not involved in supplying the malformation. The other
Fig. 2. Case 1. A, coronal section of frontal lobes ventral to the foot of F₃; bilateral infarction of the territory of Heubner's artery; old haematoma of the medial part of the corpus callosum; bilateral demyelination of the white matter. B, coronal section of the left frontal lobe, through the foot of F₃ and the optic chiasma. C, coronal section of the right frontal lobe at the same level as above.
cervical and intracranial arteries were normal. General clinical examination, haematology and CSF were normal.

The patient underwent a surgical procedure (Professor Pertuiset) on November 21, 1973. A superficial arterialized vein leading to the superior longitudinal sinus was ligated. The malformation was located at the bottom of a sulcus within the cerebral parenchyma, but did not reach the frontal horn of the ventricle. The draining veins, including a deep one, were clamped; the superficial arteries of medium diameter afferent to the malformation were ligated; the malformation was extracted, during which it was necessary for the surgeon to press strongly with the retractor on Broca's gyrus. Neuropathological examination of a fragment of the malformation revealed a delicate arteriovenous rete including haemorrhagic foci.

Surgical intervention was followed by aphasia involving serious difficulties in verbal articulation. Speech utterance was reduced to certain phonemes: *pe, é, té, tí, péi*. Oral comprehension and the execution of simple commands were clearly perturbed. Reading comprehension and writing to dictation were defective. A frontal syndrome also existed, with serious and bilateral disturbances of the Luria sequences of gestures, and grasping and utilization behaviour with both hands. The patient used a jug and a glass of water, a plate, a fork and an orange; he lit a candle with a match drawn from a box; he started writing with a fountain pen on a sheet of paper which he had grasped by himself. No sensory or motor abnormalities were apparent on the right side of the body except for a right central facial paralysis. The remainder of the neurological examination was normal. There was no sucking reflex.

The patient remained in hospital until March, 1974 for speech therapy. His difficulties in understanding rapidly disappeared; naming became possible but involved some verbal paraphasias, repetition was mildly impaired. Aphasia and manual grasping behaviour disappeared in the ensuing months. The patient was regularly examined by Dr J. L. Signoret until June, 1978. No neurological disorder was observed. The patient resumed a normal socioprofessional life, although he was given other employment in the Post Office, his history of seizures rendering him unsuitable for driving.

Case 3 (No. 48.245)

A right-handed skilled metallurgist presented for the first time on February 1, 1979, at the age of 48 years. For about six weeks he had suffered from left frontal headaches which had become bilateral; they usually occurred in the afternoon and were sometimes pulsatile. Neurological and general examination was normal, as was an EEG. Over the following months, the headaches became more persistent and the patient often vomited. At the same time, he noticed some difficulty with micturition (urgency sometimes leading to slight incontinence), and from time to time, weakness in both lower limbs. Neurological examination on March 18, 1981 revealed brisk knee jerks and bilaterally extensor plantar responses. There were no signs of frontal damage. Two CT scans (fig. 4A) demonstrated a tumour of the anterior part of the third ventricle, where the contrast appeared heterogeneous, and dilatation of the lateral ventricles due to bilateral occlusion of the foramina of Monro.

An operation was performed on March 26, 1981 (Professor Philippon). Access to the brain was by a wide right frontal approach, a 1 cm corticectomy on F₁-F₂ at 1 cm from the midline, opening of the lateral right ventricle and introduction of an operating microscope. An encapsulated grey-tinged tumour was present in the choroid plexus compressing the foramen of Monro. The tumour was removed in pieces, except for a small superior posterior fragment adhering to the choroid tela. Neuropathological examination (Dr Foncin) revealed the presence of old haemorrhagic foci containing crystals of cholesterol, part of the choroid plexus more or less modified, and granulomatous tissue containing haemosiderin and fibrovascular bundles. Conclusion: old haematoma with fibrous granuloma, probably related to a choroid plexus vascular malformation.

The patient’s condition clearly worsened after surgery. He was again transferred to the Neurology and Neuropsychology Clinic on April 7, 1981, with an obvious right frontal syndrome. He showed considerable inertia and severe apathy, a left central facial paralysis, a slight left-sided motor deficit, and manual grasping
behaviour with utilization behaviour (see fig. 7) when presented with items such as a glass and a jug of water, a plate, a fork and a slice of sausage and pineapple, and a pack of cigarettes and a lighter. Neuropsychological examination on two occasions based on standard tests showed deficient spatial orientation; perturbed ‘mental control’ (third subtest of Wechsler’s clinical memory scale) a memory span limited to 4 items; severely disturbed ability to calculate; great difficulties in visual-construction tests, chiefly concerning the left side; some left-sided spatial neglect; inability to reproduce Luria sequences of gestures and drawings with marked perseveration; Wisconsin perseverations; a WAIS IQ of 70; and memory difficulties related to the learning of verbal and visual phenomena, as well as to the recall of recent events. Language was difficult to study since the patient was Spanish and had only attended school until the age of 9 years (verbal IQ 66). A CT scan (fig. 4b) showed considerably reduced density in the anterior part of the right frontal lobe. The patient was examined on August 18, 1981 by Dr. Serdaru who noted that he could drive his car, that he was working at home and took care of his garden. Neuropsychological examination six days later with the same battery of tests as in the previous examinations showed the same abnormalities but to a lesser degree; the left-sided spatial neglect had also decreased. Inertia was, however, considerable and ‘mental control’ deficient. The patient tended to minimize his troubles. This patient never had a sucking reflex.

**Fig. 4. Case 3.** A, preoperative CT scan showing a tumour of the anterior part of the third ventricle. B, postoperative CT scan showing considerably reduced density of the anterior two-thirds of the right frontal lobe and of the right caudate nucleus.
Case 4 (No. 58.654).

A man aged 63 years with no previous history of cardiovascular disease suddenly developed a left hemiplegia during the night of October 1, 1981. He was admitted to the Neurology and Neuropsychology Clinic on the following morning. He was somnolent; his head and his eyes were permanently deviated towards the right; there was a complete left hemiplegia with sensory loss, the tendon reflexes were brisk on the left and the left plantar response was extensor. On being awakened, the patient showed anosognosia and left-sided neglect; his visual fields were full. The heart and neck vessels were normal, the pulse regular at 60/min and the blood pressure 170/120 mm Hg. The ECG was normal, as were haematological studies. Two Doppler examinations showed occlusion of the intracranial segment of the left internal carotid artery; right carotid and ophthalmic flow was not seriously diminished. A CT scan showed significantly reduced density in the right frontoparieto-temporal region.

Over the course of some hours, consciousness improved. During the next few days, the patient became fully alert; he no longer suffered from anosognosia, his left-sided neglect improved, as did the paralysis of the left lower limb. On October 5, 1981, he could walk normally, whereas the left central facial paralysis remained severe and that of the upper limb total. His condition remained static in the following weeks. A further CT scan eleven days later (fig. 5) showed reduced density in the territories of the middle and anterior cerebral arteries with contrast enhancement.

At the beginning of November, only limited voluntary movement of the left shoulder and thumb was possible. Although not yet expected, it was easy to recognize manual grasping behaviour of the right upper limb, together with utilization behaviour (the patient had to be helped since he could only use his right upper limb). While seated, the patient took a glass, gave it to the examiner and then

**FIG. 5. Case 4. CT scan after contrast.** A, at 71 mm showing enhancement of the medial face of the frontal cortex and second frontal convolution. B, at 62 mm same as above.
picked up a jug. He poured water into the glass and, having put down the jug, took the glass from the hand of the examiner and drank the water. Taking a pack of cigarettes, he hesitated a moment, then opened it and drew out a cigarette. He looked puzzled at it, being a nonsmoker. A few seconds later, he held it to the mouth of the examiner who accepted it and taking the lighter which was in the examiner's hand, near his knees, the patient lit the cigarette. Questioned on this behaviour, he simply said 'You held out objects to me; I thought I had to use them'. The patient left the Neurology Department on November 3, 1981 to continue his rehabilitation.

This patient has left-sided dominance for the lower limbs; he is right-handed, using the right upper limb for writing and for using everyday objects, drinking and eating, handling a rifle, shaving, brushing his teeth and dressing. All members of his family are right-handed.

Case 5 (No. 60.171)

A right-handed man aged 77 years, with no previous history of neurological disorder, began to have behavioural problems in February 1982. On February 17, 1982, when returning home in Paris, he got out at the wrong Metro station. On the following day, he left home for no reason at 5 p.m. and did not return that night. The next day his wife was notified that he was in a suburban café and was confused, mistaking a waitress for her, asking why he had not remained in a 'clinic' where he could see his daughter and grandchildren. During the next few days he had trouble walking, fell several times, and had episodes of urinary incontinence.

He was admitted to the Neurology and Neuropsychology Clinic on March 11, 1982. He was alert, was unstable walking, had a slight left hemiparesis and a left

---

**Fig. 6.** Case 5. CT scan after contrast medium. A, at 80 mm showing the right frontal glioma. B, at 53 mm, same as above.
extensor plantar response. He was disorientated in time and space, neglected the left when drawing or writing, made errors when copying a cube and when locating cities on the map of France. Language was normal. In conversation his judgement seemed normal. Bilateral manual prehension was so intense that when the observer had him take hold of his left forearm with the other hand, it was almost forced grasping. With objects, bilateral utilization behaviour was evident (see fig. 8) and was observed with a glass and carafe of water, with bread, with three pairs of glasses that he placed on his nose, one on top of the other, with a sheet of paper and an envelope, and with a urinal which he placed between his thighs while trying with the other hand to insert his penis. There was no sucking reflex when his lips were stimulated with a finger.

A CT scan performed on March 11, 1982 (Professor Bories) revealed a glioblastoma which extended throughout the whole right frontal lobe (fig. 6). The patient was discharged six days later and returned home.

**METHODS AND RESULTS**

**Normal Subjects**

The subject and the examiner are seated face to face. The subject has received no information about the test. The test requires absolute silence on the part of the examiner and those around him (whatever their number) so as to give no encouragement which might be understood as an order. At no time does the examiner speak, even if the subject asks him a question. His eyes are fixed on the subject’s hands and on the objects presented to him. The test begins with the solicitation of manual grasping behaviour: the examiner places his hands on the subject’s palms and stimulates them with repeated slow and/or brisk movements. The subject (always) lays his hands out more or less flat with the fingers extended. The stimulation may be continued for 30 s or more without the subject ever manifesting grasping behaviour. The examiner stimulates in the same manner, but using an object, one of the patient’s hands; then, with another object, both hands. The test continues with objects the subject might be expected to take and use (such as a pack of cigarettes, lighter, carafe, dish, fork, knife, fruit, bread). The examiner repeats the stimulation on the palms, the fingers and near the hands. He can hold out the objects and withdraw them abruptly as if he wanted to incite the patient to seize them. The hands of the subject remain constantly supine and immobile and never grasp the object. If the examiner releases the object which is stimulating the palm, it is very rare that the subject will grasp it to keep it from falling. In our experience, the normal subject makes no comments; rarely he asks ‘What do you want me to do?’ In this case the examiner does not respond and continues the test as if he heard nothing without modifying his gaze which remains fixed on the subject’s hands. In no case does grasping behaviour appear. The same test can be performed with a table placed between the examiner and the subject. The result is the same. Questioned about these stimulations, the responses hardly vary ‘You touched my hands with your
hands, with a glass and a carafe, then with a pack of cigarettes and a lighter.’ The examiner continues to question ‘Yes, but why?’ The subject answers with hesitation ‘I thought you wanted to know if I could feel the objects’; ‘You examined the sensitivity of my skin’; ‘You were probably looking for something, but I don’t know what’; or ‘I don’t know.’ A last important point: at any moment in logical or absurd situations (suddenly in the middle of a sentence) if the examiner holds out his right upper limb and his hand as if to shake hands, at least all French subjects respond to this gesture by shaking hands with the observer. In the same way, if in the course of a conversation, the examiner rises at the end of a sentence, in almost all cases, normal subjects will rise understanding that the conversation is over.

When the initial study was first presented orally at the Société Française de Neurologie in 1981, we began to examine a member of the audience selected at random. This person behaved like any normal subject.

Patients with Utilization Behaviour

The test begins with manual grasping behaviour. The examiner places his hands in the hands of the patient to get him to rise, to take a few steps, to sit down again. Short visual-manual stimulations are repeated with the objects by withdrawing the object quickly as in play. The objects are then shifted within the field of vision, far away from the patient’s hands, which incites the patient to make a large gesture with one of his upper limbs in order to grasp them.

The hands of the patient being free, the examiner then shows a utilitarian object—a glass, for instance—within the field of vision of the patient, which he then brings within reach of one of the patient’s hands. The patient grasps the object. The examiner then holds a bottle of water towards the other hand under the same conditions (fig. 7A, B). The patient naturally grasps it. Usually, the patient remains more or less puzzled; often, his eyes seem to question the examiner. This phase lasts up to 15 s. The patient then pours the water from the bottle into the glass which he raises to his mouth and drinks (fig. 7A, B). The examiner presents a new object—an orange or an apple, for instance—in the field of vision, more or less in the vicinity or within reach of a hand which grasps it; he then presents a knife and a plate. The patient puts the plate on a table, peels the fruit and eats it normally. After a few tests, the visual stimulation is sufficient and often the patient grasps and uses the objects without any hesitation. The test can be performed with a multitude of objects provided that one of them is necessary in order to use the other (figs. 7 and 8) (hammer and nail; butter, knife and bread; cheese, knife and bread; plate, fork, knife and piece of cake; envelope and sheet of paper). With a fountain pen and a sheet of paper, the patient puts the latter on a table, removes the cap of the pen and starts writing his name or that of the hospital or the beginning of a sentence. The patient puts on three pairs of glasses and at the end of this test wears all three. If the patient is a smoker, he draws a cigarette from the pack and lights it with a lighter (fig. 7E, F). If he is a nonsmoker, he uses neither the pack of cigarettes nor the lighter, but if the examiner brings a cigarette to his own lips, the patient kindly lights it with the lighter...
Fig. 7. Examples of utilization behaviour in Case 3 (extracts from a film). A, B, drinking behaviour. C, D, eating behaviour. E, F, smoking behaviour.
Fig. 8. Examples of utilization behaviour in Case 5 (extracts from a film). A, putting glasses on his nose. B, combing his hair. C, D, folding paper and putting it in an envelope. E, F, using the urinal.
that he holds in one hand. Sometimes the patient draws a cigarette from the pack and offers it to the examiner, then he lights it with the lighter. The only object that patients never use, except for one instance (fig. 8E, F) is the urinal. It must be admitted that this object plays a special part in human behaviour as it involves at once a physiological need as well as a social inhibition.

If the examiner asks the patient why he grasped the objects and used them, the answer is always the same ‘You held them out to me, I thought I had to use them.’ The examiner can try to object to this behaviour ‘I didn’t tell you anything nor ask you for anything.’ Or he gives repeatedly and even brutally this order ‘You are mistaken; from now on, don’t grasp any of the objects I will show you; and in no case must you use them.’ After about 20 to 30 s. during which the patient’s attention has to be diverted, the examiner begins the test again with the same objects or with others. The behaviour remains unchanged. If then the examiner pretends to be surprised that the patient should have forgotten his order, the latter replies most of the time ‘It’s true, I remember.’ ‘Then why?’ ‘Because you held out the objects to me and I thought I had to grasp and to use them.’ These exercises can be repeated, the same behaviour recurs regularly. Only a few patients respected the order not to grasp or use the objects. In some cases, the behaviour is more variable. Numerous patients grasp 2 or 3 utilitarian objects then ask the examiner ‘Must I use them?’ The examiner does not answer. The patient, after a short time, repeats his question which receives no reply. The patient keeps the objects in his hand without any limit of time until the examiner holds out his own hand, which incites the patient to hand the objects over to him.

No patient suffering from cerebral lesions that did not damage the frontal lobes exhibited this utilization behaviour.

DISCUSSION

Utilization behaviour of this type has not so far been reported. Somewhat similar behaviour was observed by Bancaud et al. (1976) in their study on electrical stimulation of the anterior part of the cingulate area (area 24) (83 epileptic patients; 521 stimulations). Only one patient drew a cigarette from the pack, brought it to his mouth and lit it. These authors did not emphasize this fact and have shown that electrical stimulation of this region induced the reappearance of primitive manual and buccal behaviour. Such behaviour has nothing in common with the behaviour described here. A case of complex behavioural disturbance was reported by Laplane et al. (1981) related to bilateral necrosis of the rostral part of the corpus callosum and of territory of Heubner’s artery. A ‘compulsive’ psychomotor behaviour was observed in the patient, together with memory defects and difficulties with attention. ‘The patient seized the object and used it properly as if it was a normal thing to do.’ This behaviour was considered to result from regression to primitive behaviour. Our patients had no compulsive behaviour; they simply took and used the objects
presented to them, often with hesitation, always with a slow or normal temporal rhythm.

There has been renewed interest in unilateral forced manual grasping since Denny-Brown suggested (1956, 1958) that this motor activity reflected a modification of space exploratory behaviour. In the monkey, Denny-Brown and Chambers (1958) showed that this behaviour is organized within the parietal cortex and that it is released by lesions of the frontal cortex; inversely, the functions organized within the frontal cortex control and inhibit parietal activity, hence the appearance of repellent behaviour after parietal lesions. Later, Hyvärinen and Poranen (1974) showed that in monkeys, the movement is 'initiated' in the parietal area before being 'performed' in the motor zone. The frontal lobe is presumed to inhibit the parietal lobe. Denny-Brown (1956, 1958) applied this hypothesis in human behaviour after frontal lesions. 'We believe that the two types of responses represent two areas of normal organisation, positive and negative, of the tropism to the environment managed by the cerebral cortex. Damage to the mechanism of either one releases abnormal activity of the other' (Denny-Brown, 1958). Moreover, he pointed out that this activity concerned only the side of the body contralateral to the cerebral lesion, with the exception of cases in which the frontal lesion was very severe.

Denny-Brown (1958), in equating human brain function with that of the monkey, reduced this behaviour to an elementary sensorimotor level. This interpretation does not fit with the behaviour described in this study. (1) Manual grasping behaviour is bilateral; it is observed with bilateral and unilateral lesions of frontal lobes. (2) Utilization behaviour is also bilateral and is observed in the same kinds of lesions; it is a more severe form of manual grasping behaviour and never appears by itself. In these cases, tactile, visual-tactile, or visual stimuli imply to the patient an order to grasp the objects presented and to use them. Observation of the behaviour of these patients is instructive in this respect: the stage when they hesitate and do not use the grasped objects; the stage when, after a moment of hesitation, they use them; the stage when, without hesitation, they use them at once. The verbal comments of the patients when they are questioned confirmed this categorically. We never heard a normal subject or one in which the frontal lobes were intact asking whether he had to use these objects (only, rarely, 'What do you want me to do?'). The contrast is striking when considering another stimulus, offering one's hand to shake. This stimulus is so powerful that all subjects, normal and pathological, respond to it in all situations. Another gesture which has identical value is rising at the end of a sentence in a conversation taking place between several seated persons and, in the past, a third gesture had similar power, greeting a person met in the street by removing one's hat.

A last point, no suction behaviour was observed in the four cases with unilateral lesions of the frontal lobe. Denny-Brown (1958) observed it but he used a spoon as a stimulus (1958, fig. 3A, B). In this case, the behaviour was adapted to the stimulus and was not related to the release of a reflex of automatic suction which can be provoked by nonsignificant stimuli (such as the finger or a tissue).
We believe that the interpretation of Denny-Brown (1956, 1958) of an imbalance between the activities of the frontal and parietal lobes must be interpreted at a higher than purely sensorimotor level which involves the entire activity of the brain. It can be expressed as follows. All the information coming from the body and from the outside world is received in areas of the sensory cortex which surround the parietal lobe; systems develop in the parietal area which unite these unending sequences of stimuli. These systems activate other unknown patterns, material counterparts of their meaning, and prepare the response of the patient. The result is that the normal activity of the parietal lobe tends to create links of dependence between the subject and stimuli from the environment, while some of the functions of the frontal lobe allow the subject to remain aloof from the outside world and to ensure his independence by modulating and inhibiting the activities of the parietal cortex. With normal subjects the balance between these two activities is fluid so that the subject’s behaviour is more or less dependent or independent of the outside world as a function of the quality of the external stimuli and internal mental activity. Frontal damage suppresses to varying degrees this function and thus releases the activity of the parietal lobe, that is, it tends to subject the patient to all external stimuli.

It must be stressed that the programme for the gesture is performed without any internal motivation. Patients eat even after their lunch, drink without any need, smoke, write or use the urinal (Case 5) in the same conditions. Without any food, the patient (Case 1) replicated the gestures of cutting with a plate, a knife and a fork.

This interpretation incites us to reject the terminology of magnetic apraxia suggested by Denny-Brown (1958). The meaning of ‘magnetic’ is strictly limited to physics, but the metaphor has its charm. The word ‘apraxia’ is incorrect. The so-called kinetic, ideomotor, and ideational apraxias belong to the organization of gestures, starting from the most elementary sensorimotor levels, up to the most elaborate levels, namely, symbolic and semantic. This field is related to, but different from, that which in the subject’s brain makes him more or less dependent on external stimuli. It is better to refer to it as ‘manual grasping behaviour’.

In consequence, we questioned whether lesions of the parietal lobes were not able to induce ‘indifference’ or even ‘escape’ behaviour, in response to tactile, manual, visual-tactile or visual stimuli which are more elaborate than the repellent response. However, we have never observed this except for the usually left-sided body and space neglect, which can exceptionally be seen on the right. This disturbance is quite different from utilization behaviour.

Like manual grasping, utilization behaviour is frequent in presenile degenerative diseases, in particular Alzheimer’s disease, but it can be seen in other types of bilateral frontal lesions. It can also be observed with both hands after lesions unequivocally limited to the left or the right frontal lobe. But it is certainly far less frequent. We cannot give an objective estimate of its frequency in unilateral frontal lesions.

Denny-Brown (1958) has studied anatomical cases of unilateral ‘magnetic apraxia’ with lesions of the medial part of the frontal lobe (tumour or occlusion of
anterior cerebral artery), 'but the lesion was too extensive for exact anatomic analysis'. On the basis of the five observations presented in this paper, it also seems difficult to define precisely the frontal structures responsible for this behaviour. In Case 2 (fig. 3) an arteriovenous malformation was situated unquestionably in the corticosubcortical premotor area of the external face of the frontal lobe; but the frontal syndrome and utilization behaviour were only observed subsequent to surgical intervention. It is thus certain that the intervention caused other frontal lesions. According to the CT scanning images for Cases 3, 4 and 5 (figs. 4B, 5 and 6), the lesions affected the medial surface, the orbital surface and the anterior pole of the frontal lobe (in Case 3, the lateral face of the frontal lobe was quite normal). More precise data are provided by the neuropathological study in Case 1 (figs. 1 and 2). The lateral and medial cortex of the frontal lobes were normal; only the white matter of the centrum ovale was demyelinated (secondary degeneration to the haemorrhagic and necrotic foci in the anterior part of the corpus callosum). In these conditions, it is probable that the cortex of the medial and lateral surfaces of the frontal lobes do not play a role in the liberation of the manual grasping and utilization behaviour. Lesions of the anterior part of the corpus callosum were not involved (the central and posterior parts of the corpus callosum were normal). The lower frontal pole, the orbital surface of the frontal lobes and the region of the Heubner's artery (in particular, the caudate nucleus) were necrotic. The role of the caudate nucleus is problematic, since it is connected by large fascicles to the orbital and medial areas. The pole and the orbital surface of the frontal lobe have a strong influence on motoricity (ascending and premotor frontal gyrus; deep grey nuclei), but also on many other corticosubcortical areas of the brain. These lesions may therefore be responsible for the manual grasping and utilization behaviour. It might thus be admitted that utilization behaviour, together with the other frontal symptoms, decreased three-and-a-half months after the operation and were not present at the time of the neuropathological examination. It is not possible to define more accurately the exact sites of the lesions in the frontal lobe responsible for the release of this pathological behaviour. The question arises as to whether such behaviour emerges when disturbances of frontal lobe function are induced by deep lesions of the brain (especially of the thalamus). We have not so far observed such a case, but this possibly cannot be excluded.

ACKNOWLEDGEMENTS

We wish to thank Drs J. L. Signoret and Y. Agid for their highly appreciated collaboration, Drs O. Lyon-Caen and M. Serdaru for their technical help, Dr F. Gray who carried out the neuropathological study, and Professor Strauss and Dr M. Ruberg for translation into English.
REFERENCES


(Received January 5, 1982. Revised September 16, 1982)