

Apraxia in deep cerebral lesions

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SUMMARY In a series of 50 patients with cerebrovascular lesions (demonstrated with CT scan), seven patients had lesions located in the basal ganglia and/or thalamus. All these seven patients were apractic. Ideomotor apraxia was present in all patients; five also had constructional apraxia, and one had bucco-facial apraxia. None of the patients had utilisation apraxia. These observations indicated that apraxia is not only a "high cerebral (cortical) function", but may depend also on the integrity of subcortical circuits and structures.

Apraxia, a defect in motor performances and behaviour without concomitant relevant paresis, ataxia, incoordination or dystonic-dyskinesias,¹ is generally ascribed to lesions in the cerebral cortex or to damage of the white matter just beneath the cortex.²⁻⁷ Both left and right hemisphere^{8,9} lesions may cause apraxia and, although it has been claimed that the different types of apractic disturbances are associated with lesions in particular areas of the cerebral cortex, the importance of apraxia in the topographic diagnosis has been recently challenged.¹⁰ Nevertheless some parts of the cerebral cortex appear to be crucial in praxic functions, such as the gyrus supramarginatus,^{2,3,7} gyrus superior and gyrus inferior of the parietal lobe,^{2,3,7} the premotor cortex,^{2-4,11} and corpus callosum with its radiations.^{4,6,11,12}

Despite the well-known involvement of the basal ganglia and related structures in motor performances,^{13,14} there are no extensive reports of apraxia in cases of lesions located in these structures. However, the possibility has been suggested.¹⁵ We report seven patients, part of a wider study on practice disturbances in cerebrovascular disorders, in whom the lesions involved different parts of the basal ganglia and neighbouring structures. In all these patients apraxia occurred, indicating that praxic functions are not solely a task of cerebral cortex.

Subjects and methods

In our series of nearly 50 patients with cerebrovascular lesions (either ischaemic or haemorrhagic) subjected to

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specific tests for apraxia, seven had lesions located in the basal ganglia and/or thalamus, without concomitant involvement of the cerebral cortex. All these seven patients had some type of apraxia. The site of the lesion (fig 1) was demonstrated by computed tomography (CT). The CT scan was performed 10 days after the stroke, to allow stabilisation of the clinical and tomographic findings. Table 1 summarises the clinical findings of the seven patients.

Verbal comprehension was assessed with the "Token Test"¹⁶ (table 2). All the patients were right handed (determined by Oldfield test).¹⁷ In evaluating the various type of apraxia we used the tests of De Renzi, Pieczuro and Vignolo^{18,19} and of Arrigoni and De Renzi,²⁰ which permit a quantitative assessment of praxic functions.

The test for ideomotor apraxia consisted in the execution of ten symbolic, common gestures (for example, cross oneself, salute, threaten someone etc.). The test, when possible, was performed with both hands. For every item, two points were scored for a correct ready performance; one point when the correct performance was preceded by hesitation or repeated trials; zero point when the requested gesture was not at all or only partially executed. This rating score was also used in utilisation and bucco-facial apraxia (see below).

In utilization apraxia or ideational apraxia test,¹⁹ the patients were asked to use appropriately objects (hammer, scissors, match etc.) under visual control. Any failure to reach the maximum possible score (14 points) indicates the presence of utilisation apraxia. The bucco-facial apraxia was evaluated by requesting the patient to perform ten different expressive movements (for example to puff, to blow a kiss, to yawn, to whistle etc.).

In the constructional apraxia test the patients had to copy ten geometrical drawings, with the preferred hand, if possible. Two points were scored when the drawing was correct in shape, size, orientation, number of lines; one point when the copy was defective, but still recognisable; zero point for a drawing markedly defective or/and unrecognisable. All these tests were performed 20-25 days after the stroke.

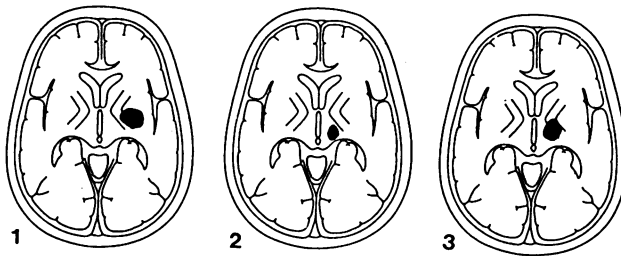


Fig 1 Schematic representation of the lesion in CT scans.

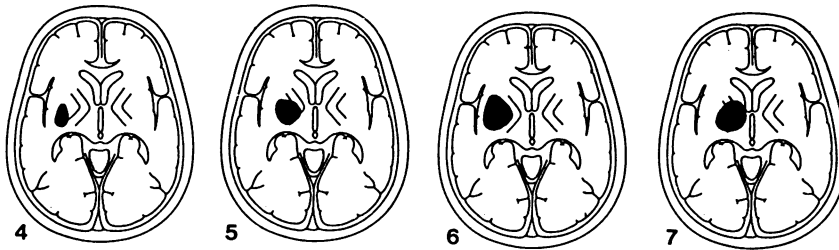


Table 1 Clinical and EEG findings

	Age (yr)	Sex	Type of lesion	Clinical findings	EEG
Case 1	59	F	Ischemic	Left hypotonic hemiplegia and hypoaesthesia, left facial paralysis, left lateral homonymous hemianopsia	Bilateral, low amplitude theta and delta waves
Case 2	44	M	Hemorrhagic	Mild left limb weakness; paralysis of upward gaze	Normal,
Case 3	75	F	Hemorrhagic	Left spastic hemiplegia and hypoaesthesia; left facial paralysis	Bilateral, diffuse, rapid activity
Case 4	59	M	Ischemic	Mild walking bradykinesia and legs rigidity	Bilateral, diffuse theta activity
Case 5	53	M	Ischemic	Right hemiparesis and hypoaesthesia	Normal
Case 6	65	F	Hemorrhagic	Mild bilateral rigidity	Diffuse theta and delta waves prevailing in left temporal lobe
Case 7	60	M	Hemorrhagic	Right spastic hemiplegia and hypoaesthesia; right paralysis of the VII and XII cranial nerves	Normal

Table 2 Score in Token Test

	Education (yr)	Token Test*
Case 1	5	29
Case 2	2	29
Case 3	5	26
Case 4	5	28
Case 5	10	33
Case 6	4	22
Case 7	8	23

*Maximum possible score 36. Cut off¹⁶ for normal subjects 27; for right brain damaged patients 22

Results

Ideomotor apraxia was present in all the seven patients. None had utilisation apraxia; one (case 2) had bucco-facial apraxia and five of the seven constructional apraxia (table 3). The ideomotor apraxia was always mild. In the four patients (case 2, 4, 5, 6), for whom testing with both hands was possible,

performance with the two hands was identical. The score obtained by the patients in the items of the test is reported in table 4. It can be seen that the low score of our patients was mainly due to failure to execute some gestures (score = 0) and not to hesitation or delay (score = 1). No correlation between the score in the Token Test and the score in the ideomotor apraxia test was found. The follow-up of patients 6 and 7 showed a further deterioration of ideomotor apraxia in subsequent testings.

In patients 1, 2, 3, 4 and 6 (table 3) there was also a constructional apraxia, which was extremely marked in patients 1 and 6. The main feature of the defect observed in patients with mild constructional apraxia (cases 2, 3 and 4) and in the severe case 6, was reduction of size and simplification of the drawings (fig 2). By contrast, in patient 1 the defect mainly consisted in a fragmentary drawing, wrongly oriented and often lacking of particulars in the left half of the drawing (fig 3). This patient was also

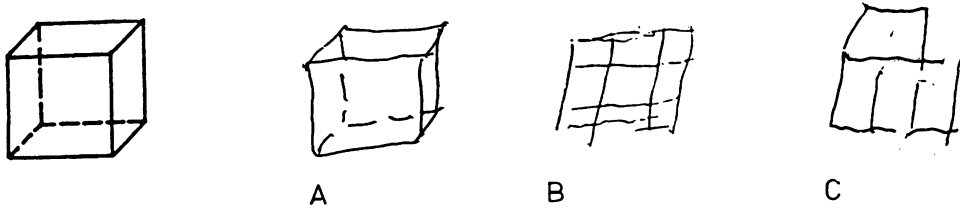


Fig 2 Examples of constructional apraxia in patient 2 (B) and 6 (C). The drawings are sketchy and simplified. Patient 5 (A) did not show constructional apraxia.

Table 3 Score in apraxia tests

	Ideomotor apraxia*		Utilisation apraxia†		Bucco-facial apraxia‡	Constructional apraxia§
	Right	Left	Right	Left		
Case 1	15	plegia	14	plegia	18	2
Case 2	15	15	14	14	12	10
Case 3	15	plegia	14	plegia	20	12
Case 4	15	15	14	14	18	14
Case 5	14	14	14	14	20	20
Case 6	15	15	14	14	18	1
Case 7	plegia	13	plegia	14	17	16

*The maximum possible score for ideomotor apraxia is 20, cut-off 17

†The maximum possible score is 14 and any failure to reach this score indicates utilization apraxia

‡Maximum possible score 20, cut-off 16

§Maximum possible score 20, cut-off 15

Table 4 Score in the items of ideomotor apraxia test

	Score = 2	Score = 1	Score = 0
Case 1	7	1	2
Case 2	8	0	2
Case 3	7	1	2
Case 4	7	1	2
Case 5	6	2	2
Case 6	7	1	2
Case 7	6	1	3

affected by a left hemianopia. There was no correlation between the severity of the ideomotor apraxia and of the constructional apraxia.

Cases 1, 4 and 5 had ischaemic lesions, while the other four patients had spontaneous intracerebral haemorrhages. Cortical cerebral atrophy, mild to moderate, was present in the CT scans of patients 4, 5 and 7. It is worth noting that these patients had similar scores in ideomotor apraxia to those of the patients without cortical atrophy and were even the "best" in constructional apraxia.

The EEG was normal in cases 2, 5 and 7 and showed only mild, diffuse slowing in the other patients, in keeping with the location of the lesions in deep structures. There was no evidence of a correlation between the EEG findings and the performance in any of the apraxia tests. In all patients clinical signs of cortical involvement were completely absent.

Discussion

The presence of apraxia in patients with lesions restricted to the basal ganglia and thalamus, without clinical or other signs of cortical dysfunctions, poses some interesting questions on the functions of the basal ganglia.

This study was based on clinico-pathological correlations, the site of lesion being determined *in vivo* by computed tomography. Despite some limitations, computed tomography is an adequate means for this

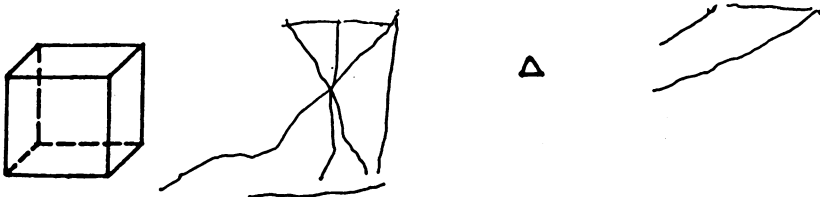


Fig 3 Examples of the defects in copying drawing in patient 1, who was also affected by left hemianopsia. There are errors and lack of particulars in the left half of the drawing.

type of study.²¹ It offers the advantage of an assessment of the clinical and pathological findings at the same time. However CT scan is not *per se* sufficient to exclude the presence of other cerebral cortical lesions (for example isodense lesions). Nevertheless the past histories of the patients, clinical signs, EEG findings, all agreed with CT scan in excluding the presence of cortical lesions. Moreover it seems extremely improbable that all these seven unselected patients have undetectable involvement of the cerebral cortex. Finally atrophy of the cerebral cortex, in so far as it can be adequately evaluated using CT scans, was never so marked (and was often absent) as to have a role in the apractic dysfunctions noted in our patients. Furthermore, there was no correlation between the severity of cortical atrophy and the score in apraxia tests.

Apraxia may adequately be demonstrated and classified only by use of specific tests. The ones we used in our study appear to be appropriate, on account of their wide usage and possibility of quantification. Furthermore, it must be stressed that they were originally tested¹⁸⁻²⁰ on a population comparable with that of our study. Comprehension was reasonable or good in all patients, although some patients (cases 3, 6 and 7) scored below the cut-off on the Token Test. Their score on the Token Test was however better than that of right hemisphere damaged patients¹⁶ and their performance on the apraxia tests did not correlate with Token Test scores.

Clinically apraxia is often not apparent and the patients complain of a disturbance only when apraxia interferes in particular motor performance. Ideomotor apraxia is a pure intentional symbolic activity (a laboratory activity) and affected people may retain their capacity to perform everyday motor activities. It is therefore not surprising that ideomotor apraxia is not reported in deeply placed lesions, unless it is searched for. The same considerations apply to the other types of apraxia present in our patients. On the other hand, the absence of utilisation apraxia, a disorder which may interfere with everyday tasks, further supports the idea that the lack of reports of apraxia in basal ganglia lesions depends on a lack of specific investigations.

Our patients presented with a mild ideomotor apraxia. Although clinically none of them had arm bradykinesia, one could argue that the ideomotor disturbances were due, not to practic defects, but to subclinical bradykinesia, revealed by the test. The failure of our patients in the ideomotor apraxia test was however mainly due to an incapacity to execute some specific gestures, not to hesitation or slow performing (see table 4), as one could expect in bradykinesia.

Constructional apraxia and specific tests for it have been differently evaluated. Some authors²² have used copying drawings to study visuo-spatial capacities, but others²³ used it to evaluate practic functions. The failure to execute adequately the test has also been differently interpreted²⁴⁻²⁶ in right and left cerebral cortex damaged patients: as a true practic defect in left cerebral damaged patients, as a visuo-spatial defect in right damaged patients. No differences have been found between our patients with right and left hemisphere damage. The features of the defect in our patients (for example, simplification of the model) are more in keeping with an apractic rather than with a visuo-spatial disturbance, being similar to those reported in left damaged patients.^{23, 26} One exception, which however corroborates the idea that in most of our patients constructional apraxia was due to a true defect in practic function, is patient 1. She presented *inter alia* with left lateral hemianopia and she often showed, besides simplification of the entire model, fragmentary drawing and a lack of details in the left half of the model (fig 3). On the other hand patient 6, who also suffered from constructional apraxia of similar degree, but did not have a hemianopia, made completely different mistakes. We are therefore inclined to consider the defects observed in our patients as due to a practic dysfunction, although, in constructional apraxia, visual "driving" is, of course, very important.

Having tested out patients several days after the stroke excludes the possibility that apraxia may be due to acute phenomena (for example Von Monakow's diaschisis) or extensive oedema, or to any other secondary, transitory change, not detectable in CT scans. The deterioration of the apraxia in repeated determinations in patients 6 and 7, further supports this view. It is therefore justifiable to say that the ideomotor and constructional apraxia present in our patients, were due to the lesions located in the basal ganglia and/or the thalamus.

The striatum has wide connections with the associative parietal cortex (ipsilateral and contralateral), and it is widely interconnected also with various parts of the thalamus, with the globus pallidus, subthalamus and especially with the substantia nigra.²⁷ All these structures are links in complex feed-back circuits and it is therefore not surprising that apraxia can occur whatever the damaged structure is. In experimental animals there is evidence that the basal ganglia, as well as the associative parietal cortex, are involved in planning and controlling motor performance.^{13, 14}

Our observations of apraxia in human patients with lesions located in the basal ganglia and/or thalamus (or even in more caudal structures) indi-

cate that the integrity of subcortical circuits and structures which intervene in motor behaviour, may be important in practic functions, so far considered a task purely of the cerebral cortex.

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