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 dystonio-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 hemisphere89 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 practic functions, such as the gyrus supramarginalinus,237 gyrus superior and gyrus inferior of the parietal lobe,237 the premotor cortex,2411 and corpus callosum with its radiations.46112

Despite the well-known involvement of the basal ganglia and related structures in motor performances,1314 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 practic 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 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 Vignolo1819 and of Arrigoni and De Renzi,20 which permit a quantitative assessment of practic 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.
Patients had various lesions, and it was observed that ideomotor apraxia was always mild. In the four patients (cases 2, 4, 5, and 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...

Table 1  Clinical and EEG findings

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

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

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Table 3  Score in apraxia tests

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

*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         | 2         | 2         |
| Case 5           | 6         | 2         | 2         |
| Case 6           | 7         | 1         | 2         |
| Case 7           | 6         | 1         | 3         |
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type of study. It offers the advantage of an assess-
ment 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 involve-
ment of the cerebral cortex. Finally atrophy of the
cerebral cortex, in so far as it can be adequately evalu-
ated using CT scans, was never so marked (and was
often absent) as to have a role in the apractic dys-
functions 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 tested 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 hemi-
sphere damaged patients 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 consider-
ations apply to the other types of apraxia present in
our patients. On the other hand, the absence of util-
isation 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 where 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 per-
forming (see table 4), as one could expect in
bradykinesia.

Constructional apraxia and specific tests for it
have been differently evaluated. Some authors have used copying drawings to study visuo-spatial
capacities, but others used it to evaluate practic
functions. The failure to execute adequately the test
has also been differently interpreted in right and
left cerebral cortex damaged patients: as a true prac-
tic 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, sim-
lification 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. One exception, which however cor-
rorobates the idea that in most of our patients con-
structional apraxia was due to a true defect in practic
function, is patient 1. She presented inter alia with
left lateral hemianopias and she often showed,
besides simplification of the entire model, fragment-
tary 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 com-
pletely different mistakes. We are therefore inclined
to consider the defects observed in our patients as
due to a practic dysfunction, although, in construc-
tional 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 diachisis) or extensive oedema, or to
any other secondary, transitory change, not detect-
able 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 pre-
sent 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 controlat-
eral), and it is widely interconnected also with vari-
ous parts of the thalamus, with the globus pallidus,
subthalamus and especially with the substantia
nigra. All these structures are links in complex
feed-back circuits and it is therefore not surprising
that apraxia can occur whatever the damaged struc-
ture is. In experimental animals there is evidence
that the basal ganglia, as well as the associative
parietal cortex, are involved in planning and control-
ling motor performance, as in experimental animals
there is evidence that the basal ganglia, as well as
the associative parietal cortex, are involved in planning and controll-
ling motor performance.
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.

References

1. De Jong RN. The Neurological Examination 3rd Ed. New York: Harper and Row, 1967.
2. Liepmann H. Ueber die Funktion de Balkens beim Handeln und die Beziehungen von aphasie und apraxie zur Intelligenz I, II, Med Klin 1907;725-9, 765-9.
3. De Ajuriaguer J, Hecaen H, Angelergues E. Les apraxies. Variétés cliniques et latéralisation lésionelle. Rev Neurol (Paris) 1960;102:499-566.
4. Geschwind N, Kaplan E. A Human Cerebral Deconnection Syndrome. Neurology (Minneap) 1962;10:675-85.
5. Geschwind N. Disconnection Syndromes in Animal and Man II. Brain 1965;88:585-644.
6. Geschwind N. The Apraxias: Neural Mechanisms of Disorders of Learned Movements. Am Sci 1975;63:188-95.
7. Pieczuro A, Vignolo LA. Studio sperimentale sull'aprassia ideomotora. Sistema Nervoso 1967;19:131-43.
8. Dee HL, Benton AL, Van Allen MW. Apraxia in relation to hemispheric locus of lesion and aphasia. Trans Am Neurol Assoc 1970;95:147-50.
9. De Renzi E, Motti F, Nichelli P. Imitating gestures: a Quantitative Approach to ideomotor apraxia. Arch Neurol 1980;37:6-10.
10. Basso A, Luzzatti C, Spinnler H. Is ideomotor apraxia the outcome of damage to well-defined regions of the left hemisphere? Neuropsychological study of CT corre-
11. Morlås J. Contribution à l'étude de l'apraxie. Thèse de Paris 1928.
12. Liepmann H. Die Linke Hemisphäre und das Handeln. Münch Med Wochenschr 1905;52:2322-5, 2375-8.
13. Marsden CD. The enigma of the basal ganglia and movement. Trends in Neurosciences 1980;3:284-7.
14. McGeer PL, McGeer EG. The control of movement by the brain. Trends in Neurosciences 1980;3:III-IV.
15. Kimura D. Neuromotor Mechanisms in the Evolution of Human Communication. University of Western Ontario, Department of Psychology, Research Bulletin 1978:454.
16. De Renzi E, Vignolo LA. The Token Test: a sensitive test to detect receptive disturbances in aphasia. Brain 1962;85:665-78.
17. Oldfield RC. The assessment and analysis of handedness: The Edinburgh inventory. Neuropsychologia 1971;9:97-113.
18. De Renzi E, Pieczuro A, Vignolo LA. Oral apraxia and aphasia. Cortex 1966;2:50-73.
19. De Renzi E, Pieczuro A, Vignolo LA. Ideational apraxia: a quantitative study. Neuropsychologia 1968;6:41-52.
20. Arrigoni G, De Renzi E. Constructional apraxia and hemispheric locus of the lesion. Cortex 1964;1:170-97.
21. Tredici G, Pizzini G, Bogliun G, Tagliafue M. The site of motor cortico-spinal fibers in human internal capsule. A computerized tomographic study of restricted lesions. J Anat (Lond) 1982;134:199-208.
22. Kimura D, Archibald Y. Motor functions of the left hemisphere. Brain 1974;97:337-50.
23. McFie J, Zangwill OL. Visual-constructive disabilities associated with lesions of the left cerebral hemisphere. Brain 1960;83:243-60.
24. Warrington EK, James M, Kinsbourne M. Drawing disability in relation to laterality of cerebral lesion. Brain 1966;89:53-82.
25. De Renzi E. L'apprassia costruttiva. In: Bisioch E, Denes F, De Renzi E, Foglioli P, Gainotti G, Pizzamiglio L, Spinnler HR, Vignolo LA. eds, Neuropsychologia Clinica. Milan: Franco Angeli Editore, 1977.
26. Piecza M, Hecaen H, Ajuriaguerra J. Constructional apraxia associated with unilateral cerebral lesions. Brain 1960;83:225-42.
27. Brodal A. Neurological Anatomy in Relation to Clinical Medicine. 3rd ed. New York: Oxford University Press, 1981.