Neuropsychological changes related to unilateral lenticulostriate infarcts

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Abstract

According to previous studies, focal capsulostriatal lesions may produce aphasia, hemineglect, gestural apraxia, frontal lobe dysfunction, and memory impairment. A few reports of capsulostriate infarcts secondary to involvement of lenticulostriate arteries have confirmed that aphasia and hemineglect may occur whereas gestural apraxia, anosognosia and frontal-lobe symptoms are rare. Most studies used CT scan assessment and did not exclude possible associated lesions. Neuropsychological changes in 11 patients with lenticulostriate infarcts diagnosed by CT scan were prospectively investigated. MRI in five of the 11 patients showed an associated cortical lesion not seen on CT scan. Patients with pure lenticulostriate infarcts on MRI may exhibit aphasia of mild severity whereas Broca's aphasia, hemineglect, gestural apraxia, and anosognosia were only seen in the subgroup with associated cortical lesions. Aphasia in patients with pure lenticulostriate infarcts was characterised by prominent expressive and lexicosemantic task impairments. The results strongly suggest that cortical involvement is critical to the extent and severity of neuropsychological changes in patients with lenticulostriate infarcts.

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The development of new cerebral imaging techniques during the past 15 years has established the role of the basal ganglia in cognitive functions: focal capsulostriatal lesions may produce aphasia,[1–15] hemineglect,[4 8 11–19] gestural and constructional apraxia,[8 14 17 20–22] frontal lobe dysfunction,[1 12 14 23] and memory impairment.[14 23] In these studies, such cognitive deficits have been reported with different prevalence and severity. This makes difficult any attempt to establish clinicoanatomical correlations in patients with focal striatal lesions and prevents firm statements about the contribution of the basal ganglia to neuropsychological function. This could be for three main reasons[24]: (1) there have only been a few prospective studies; (2) previous studies have included patients with various kinds of capsulostriatal lesions, although the nature of the lesion may contribute to the pattern of neuropsychological changes[25]; (3) previous studies used neuropsychological and radiological evaluations of different types. Evaluation of more homogeneous groups of patients would avoid this lack of consistency, and this could be achieved in studies focusing on patients with similar lesions. Among the various cerebral lesions, infarcts provide most advantages for study of the consequences of focal cerebral lesions because they abruptly disrupt previously undamaged neuronal networks and the territories of vascular supply are well defined. The blood supply of the basal ganglia mainly depends on the lenticulostriate arteries (deep branches of the middle cerebral artery), whereas the anterior choroidal artery supplies the posterior arm of the internal capsule, the globus pallidum, and the posterior part of the putamen.[26] As both kinds of infarcts are generally studied without distinction under the common name of capsulostriate infarcts, they involve different neuronal structures: the lenticulostriate infarcts involve the putamen, the anterodorsal part of the head and the body of the caudate nucleus, the anterior part of the internal capsule, and the centrum ovale. The variability of the neuronal structures affected by both kinds of infarct could also account for the large range of neuropsychological deficits. This possibility supports the need for a precise inventory of the anatomical lesions in each patient. This requirement could be especially true in lenticulostriate infarcts as these are mainly due to a proximal occlusion of the middle cerebral artery[5 27] and are often associated with a cortical infarct.[24]

Previous studies focusing on capsulostriate infarcts within the territory of lenticulostriate arteries confirm the possible presence of aphasia[5 11 13 14] and hemineglect,[1,13,14] whereas constructional apraxia, gestural apraxia, anosognosia and frontal-like symptoms are rarely encountered.[14] Except in the study of Weiller *et al*,[13] the radiological assessment mainly depended on CT scans and the structures involved were not precisely reported.

The aim of our study was to prospectively investigate neuropsychological changes in patients with lenticulostriate infarcts determined on MRI scans. The assessment of frontal lobe dysfunctions has been reported previously.[24]

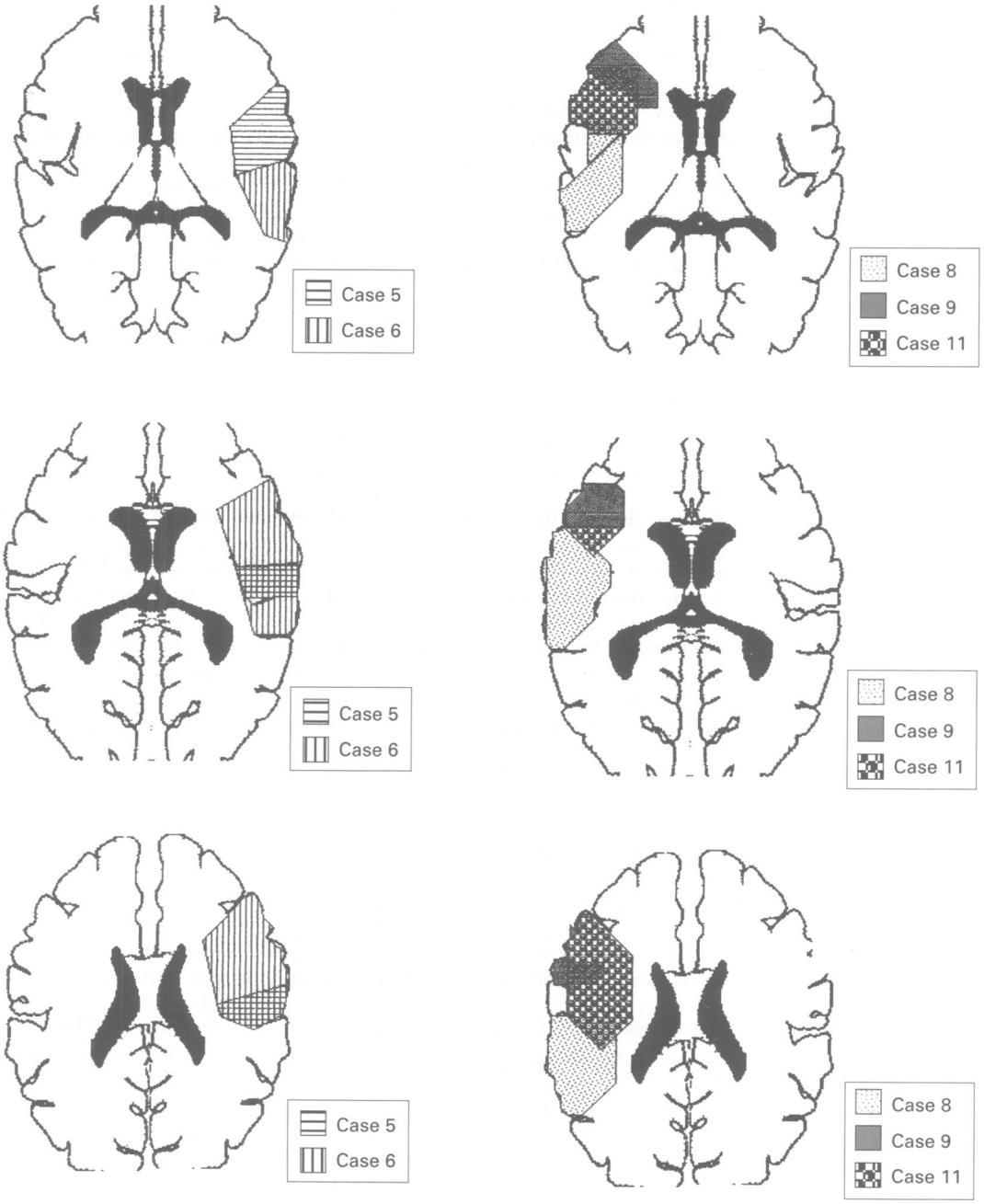
Patients and methods

From the 927 patients admitted to the Lille University Hospital for an acute stroke over a nine month period (October 1989—June 1990), 21 had an isolated unilateral

lenticulostriate infarct on CT scan. Seven of them were excluded for at least one of the following reasons: previous or associated neurological or psychiatric disorders, history of head trauma with loss of consciousness, current neuroleptic or antiepileptic treatment, severe cardiac, respiratory, or renal failure, diabetes mellitus with hypoglycaemic episodes, alcoholism, or illiteracy.

MRI was performed eight weeks after the stroke onset in the remaining 14 patients with the methodology previously reported.[24] Three patients were excluded after MRI because of lesions that were not seen on CT scans: two had another infarct located outside the territory of the ipsilateral middle cerebral artery, and one had white matter and periventricular hyperintensity scores higher than unity according to the criteria of Fazekas *et al.*[28] Thus 11 patients were actually included in the study. Six patients had a pure lenticulostriate infarct on MRI (pure group). In the remaining five patients, MRI showed another infarct not seen on CT scan (cortical associated lesion group) and located in the cortical territory of the ipsilateral middle cerebral artery (frontoinsulotemporal (two cases), insulofrontal (one case), frontoparietal (one case), temporoparietal (one case) (figure)). Nine patients were right handed and two were left handed[9]: one in the pure group with left-brain damage (case 3) and one in the cortical associated lesion group with right-brain damage (case 11).[29] Controls were 11 right handed healthy volunteers from the community. All subjects were native French speakers. There was no significant difference in age and education level between patients and controls. Table 1 gives the demographic characteristics of the subjects and location of lesions.

Neuropsychological assessment was performed six to eight weeks after onset, but



Location of the two left (left) and the three right (right) cortical associated lesions revealed by MRI.

Table 1 Demographic characteristics of patients and controls and location of infarcts on MRI

	Side of the infarct				
	Pure LSI group		*CAL group*		
	Left	*Right*	*Left*	*Right*	*Controls*
n	4	2	2	3	11
Age (y)*	61 (26–78)	48·5 (18–79)	59·5 (48–71)	54 (43–78)	55 (21–80)
Sex (M/F).	2/2	0/2	1/1	1/2	6/5
Education level†*	1 (1–2)	2 (2–2)	1·5 (1–2)	1 (1–2)	2 (1–3)
Right handedness‡*	8·5 (7–10)	9·5 (9–10)	10 (10–10)	8 (3–10)	10 (9–10)
Caudate lesion	3	0	2	2	0
Putaminal lesion	3	2	2	3	0
Pallidal lesion	1	0	0	1	0
AAIC lesion	3	0	1	2	0
Centrum ovale lesion	3	1	2	3	0

*Medians (range).
†Score 1, less than nine years of school; score 2, from nine to 11 years of school; score 3, ⩾12 years of school.
‡Determined from Oldfield's scale.[29] LSI lenticulostriate infarcts; CAL = cortical associated lesion; AAIC = anterior arm of the internal capsule; M/F = males/females.

shortened evaluations of hemineglect and Anton Babinski syndrome were done during the acute stage.

Assessment of general intellectual abilities used the mini mental state examination (MMSE)[30] with backward spelling of the word "rouge" (French translation of "red") and the French version of the Wechsler Adult Intelligence Scale (WAIS).[31]

Attentional state was assessed from visual and auditory reaction times.[32]

Assessment of language used the Boston diagnostic aphasia examination (BDAE)[33] in patients with left-brain damage and in patients with right-brain damage demonstrating language disturbances on clinical evaluation. Phonemic paraphasia were rated separately. Individual performances were evaluated with cutoff scores suggested by BDAE and aphasia was diagnosed with BDAE criteria.[33] In all patients, the following language investigations were carried out: oral comprehension with the shortened token test,[34] buccofacial apraxia with Lehmkuhl's battery,[35] lexicosemantic tasks, and syntactic abilities. For all these tests, individual performances of patients with aphasia were used and scores lower than 2 SDs from control means were considered as impaired. The lexicosemantic tasks included verbal fluency assessment (animals and fruits during one minute, and words starting with letter C during one minute), the similarities subtest of the WAIS,[31] a test of categorisation of name and picture,[36] semantic similarity judgments between 33 couples of words, and picture and colour naming. The oral picture naming test used 35 pictures (10 × 10 cm) from the list of Snodgrass and Vanderwart[37]; the colour naming test was divided into two parts: naming 11 coloured dots and naming usual colour(s) of seven items given orally (banana, flag of communist countries, wedding dress, grapes, grass, tobacco, and melon). Syntactic abilities were investigated by the construction of 36 sentences divided into four parts (10 sentences-completion, 20 sentences-generation: 10 from a phrase and 10 from a word, seven sentences-construction) as carried out by De Lacy Costello and Warrington.[38]

Visuospatial hemineglect was clinically evaluated at the acute stage. Six weeks later, we added a cancellation test[39] rated with the number of left minor right omissions, a line bisection test,[40] and four simple drawing tasks rated on a 0–13 point rating scale. One drawing test was performed by copying using Ogden's criteria[18] and three on a verbal command condition: these were a clock, a daisy, and a human face (appendix). Hemineglect was diagnosed when more than one omission was observed on drawing tests, the bisection test, or the cancellation test.

Anosognosia for motor impairment and hemiasomatognosia intensity were rated during the acute stage and six weeks later with the two 0–3 point Bisiach scales[15] where 0 means no deficit and 3 means a severe deficit. The assessment of autotopognosia required the patient to name and point under verbal and imitation conditions to the six following items on the half of the body ipsilateral to the cerebral lesion: eyebrow, cheek, nostril, thumb, index, and ear.

Ideomotor apraxia was assessed with the battery of Lehmkuhl *et al*[35] requiring realisation of 10 meaningless and 10 symbolic arm and manual movements under verbal and imitation conditions. Ideational apraxia was assessed with the battery of De Renzi *et al*.[41]

Short term memory was assessed with forward digit spans and the spatial spans subtest of the battery 144.[42] The span scores of patients were compared with those of controls.

Three-group comparisons (pure, cortical associated lesion, and control groups) were made by means of the Kruskall-Wallis H test. Subsequently paired group comparisons were made with the Mann-Whitney U test. All tests were two sided; p values <0·05 were regarded as statistically significant.

Results

No patient had Diagnostic and Statistical Manual of Mental Disorders III R criteria of dementia.[43] MMSE scores for both groups of patients were significantly lower than controls and the two subgroups of patients did not differ significantly. Auditory and visual reaction times were not significantly different (table 2).

Four patients showed aphasia and all had left cerebral damage (two of the four patients

Table 2 Median scores and ranges of MMSE, IQ, and median visual and auditory reaction times of controls and patients of the pure lenticulostriate infarct (LSI) and cortical associated lesion (CAL) groups

	Pure LSI group	*CAL group*	*Controls*	*p value*
n	6	5	11	
MMSE	27·5 (29–26)	26 (10–30)	29·5 (29–30)	<0·02
IQ	93 (83–113)	90 (79–99)	NP	—
Mean visual and auditory reaction times (ms)	266 (252–338)	296 (258–843)	264 (201–402)	NS

NP = Not performed.

Table 3 Language assessment in aphasic patients of the pure lenticulostriate infarct (LSI) and cortical associated lesion (CAL) groups

	Aphasic pure LSI group	*Aphasic CAL group*	*Controls*
Severity	3*/4*	2*/2*	5 (5–5)
Token test	29/34	24*/26*	≥29
Fluidity	23*/24*	2*/16*	27 (26–28)
Articulation	6*/7	1*/4*	7 (7–7)
Buccolingual apraxia (/20)	18/20	13·5*/13·5*	19·5(18–20)
Phonemic paraphasia	3*/6*	1/2*	≤1
Verbal paraphasia	1/4*	2*/12*	≤1
Repetition	17*/26	7*/11*	23·7 (20–26)
Reading	32/40	10*/23	38·5 (23–40)
Writing	84/96	17*/40*	83·2 (79–91)

Individual scores are given for aphasic patients; Control scores are means (range), the lowest corresponding to the suggested cutoff score.[33]
*Patient's scores lower than cutoff scores.

Table 4 Performances of aphasic patients of the pure lenticulostriate infarct (LSI) and cortical associated lesion (CAL) groups and controls on lexicosemantic tasks and syntactic abilities

	Aphasic pure LSI group (n = 2)	*Aphasic CAL group (n =2)*	*Controls*
Fluency: Animals	12*/17	3*/6*	20·6 (4·76)
Fruit	10*/15	2*/5*	17 (1·89)
"C"	8*/8*	1*/1*	14 (2·45)
Naming (/33)	26*/27	1*/20*	30·4 (2·06)
Colour naming (/34)	29*/31	1*/27*	32·7 (1·56)
Semantic decision (/20)	16*/17*	0*/16*	20 (0)
Semantic categorisation	40/40	23*/36*	40 (0)
Similarities subtest	3*/13	0*/2*	17·2 (3)
Syntax BDAE	5*/6*	1*/4*	7 (0)
Sentences building (/20)	12*/19	1*/2*	19·3 (0·54)

Individual scores are given for aphasic patients; Control scores are means (SD).
*Patients' scores lower than cutoff scores.

Table 5 Median scores and ranges on gestural apraxia evaluation and on span tests of patients of the pure lenticulostriate infarct (LSI) and cortical associated lesion (CAL) groups and controls

	Pure LSI group	*CAL group*	*Controls*	*p value**
Ideomotor apraxia (/40)	40 (38–40)	35·5 (14–40)†‡	40 (38–40)	<0·01
Ideational apraxia (/14)	14 (14–14)	12 (10–13·5)‡	14 (14–14)	<0·05
Digit span	6 (4–9)	4 (4–6)	5 (4–8)	NS
Spatial span	4 (2–6)	4 (4–6)	5·5 (4–6)	NS

*Kruskall-Wallis test.
†Significant difference between LSI and CAL groups (Mann-Whitney test).
‡Significant difference between CAL group and controls.

Table 6 Number of patients with aphasia, gestural apraxia, hemineglect, and Anton-Babinski syndrome in the pure lenticulostriate (LSI) and cortical associated lesion (CAL) groups according to the side of lesion

	Pure LSI group		*CAL group*	
	Left BD	*Right BD*	*Left BD*	*Right BD*
n	4	2	2	3
Aphasia	2	0	2	0
Gestural apraxia	0	0	2	1
Hemineglect				
Acute stage	0	0	0	3
6–8 weeks	0	0	0	1
Anton-Babinski syndrome				
Acute stage	0	0	0	3
6–8 weeks	0	0	0	0

Results from neuropsychological evaluation 6–8 weeks after stroke onset. The presence or absence of hemineglect and Anton-Babinski syndrome at the acute stage is also given. Left BD = patients with left-brain damage; right BD = patients with right-brain damage.

with left-brain damage and pure lenticulostriate infarcts and both patients with left-brain damage of the cortical associated lesion group). Both aphasic patients with pure lenticulostriate infarcts had capsulostriate lesions that included the caudate nucleus and extended into the periventricular white matter. In the first patient, the overall profile of aphasia was close to that of a conduction aphasia except for the presence of hypophonia. The second patient did not fit any of the classic aphasic syndromes; he exhibited a mild speech reduction with verbal and phonemic paraphasias and arthric deformations. Both cases improved within three months with persistence of a mild word-finding difficulty and slight spontaneous speech reduction. Conversely, the two patients from the cortical associated lesion group exhibited a severe Broca's type aphasia, with initial mutism, evolving to agrammatism in one patient and to Broca's aphasia with anomia in the other.

In patients with aphasia in the pure lenticulostriate infarct group, data analysis showed mild severity of aphasia, sparing of oral comprehension, hypophonia, mild fluency loss, paraphasic errors mainly of phonemic type, and in one patient, an impairment of repetition (table 3). Lexicosemantic tasks were mildly disturbed, especially in one patient (table 4). At a syntactic level, sentence construction and BDAE syntax evaluation were slightly disturbed. All these performances were clearly less impaired than those of the two aphasic patients of the cortical associated lesion group.

Among the five patients with right-brain damage, three showed loss of exploration of the left hemispace with head and eye deviation during the acute stage. These three patients had a cortical associated lesion. Six weeks after onset, head and eye deviation had disappeared and one patient of the cortical associated lesion group showed hemineglect.

No autotopoagnosia was found; the Anton-Babinski syndrome was present in the acute stage in three of the five patients with right-brain damage: all of them had a cortical associated lesion. Six weeks after onset, the Anton-Babinski syndrome had resolved.

Gestural apraxia scores were significantly lower in the cortical associated lesion group (table 5).

Scores on span tests were not significantly different among patient subgroups (table 5).

Table 6 summarises the main results.

Discussion

Our study showed that: (1) pure left lenticulostriate infarcts on MRI may produce aphasia, (2) pure left lenticulostriate infarcts producing aphasia extended into the caudate nucleus and centrum semiovale; (3) gestural apraxia, hemineglect, and the Anton-Babinski syndrome were only found in patients with a cortical associated lesion.

Although the aim of our study was not to compare the sensitivity of CT and MRI evaluations, it confirms the superiority of MRI.[44]

Moreover, it strongly suggests that routine CT evaluation is not accurate enough to study relations between lenticulostriate infarcts and cognitive deficits. Disagreements between previous results[11 13 14] and ours might be explained by the use of MRI in our study. This led us to exclude three patients with a second infarct not seen on CT scans and located outside the ipsilateral middle cerebral artery territory, and to divide lenticulostriate infarcts into two subgroups based on the presence of an associated cortical lesion. Neuropsychological impairments were more severe in the five patients with a cortical associated lesion. Using CT criteria only, as in most previous studies,[11 14] we would have included 14 patients, whereas only six had a pure lenticulostriate infarct and this would have led to an overestimation of neuropsychological disturbances in patients with pure lenticulostriate infarcts.

Aphasia of various types (Broca, Wernicke, conduction, amnesic, or thalamic[2 3 13 14]) has been described in 30% to 80% of patients with left lenticulostriate infarcts.[11 13] In our study, the patients with Broca's aphasia had combined cortical lesions whereas the aphasia in patients with pure lenticulostriate infarct was less severe, close to conduction aphasia in one patient and unclassifiable in the other. Because MRI was required to reveal cortical involvement, it suggests that insufficient CT resolution could lead to erroneous correlations between the aphasia profile and the location of cerebral lesions.

Our two aphasic patients with pure lenticulostriate infarcts had capsulostriate lesions extending into the periventricular white matter, supporting the critical role of associated white matter damage in the pattern and severity of aphasia related to lesions of the striatal area.[10] The role of capsulostriate structures and periventricular white matter in language processing remains uncertain. Disturbances in elaborate language tasks and slowing of speech initiation are often found in subcortical aphasia, and are often attributed to frontal lobe dysfunction.[1 3] The absence of frontal lobe dysfunction and attentional deficit in the group with pure lenticulostriate infarcts[24] argues against this hypothesis. Tasks requiring lexicosemantic processing were mildly disturbed suggesting a defective lexicosemantic access, as previously suggested.[1 6 44-46] Lexicosemantic access needs multiple processing that could require the integrity of the caudate nucleus, which receives multiple afferents from cortical associative areas.[47-48] Lesions of the anterior arm of the internal capsule may interrupt thalamofrontal connexions and frontostriatothalamo frontal loops,[49] the role of the last being suggested by a SPECT study of aphasia related to lesions of the left thalamus.[45]

Visuospatial hemineglect has been reported in 30% of patients with right subcortical strokes[19] and in 20% of patients with lenticulostriate infarcts.[13] In our study, patients with hemineglect or Anton-Babinski syndrome had a combined cortical infarct on MRI. The absence of hemineglect in patients with pure lenticulostriate infarcts might be explained by sparing of thalamoparietal connections.[8]

Only patients of the cortical associated lesion group had significantly lower scores on assessment of gestural apraxia. This result disagrees with those of CT documented subcortical lesions,[20-22] which suggest that gestural apraxia is found in capsulostriatal lesions with a lower frequency and with a milder severity than in cortical lesions. This low frequency and the small size of our sample could explain our negative result. It suggests a critical role for the combined cortical lesion, however.

In the group of patients with cortical involvement, neuropsychological deficits were clearly more severe. This could be due to the greater size of the cerebral lesion or to the critical role of the cortical structures in those neuropsychological functions. Our study cannot discriminate between these two hypotheses as both factors were confounded.

Previous studies with functional imaging emphasised the role of cortical hypoperfusion in patients with aphasia or hemineglect due to capsulostriatal lesions documented by CT scan.[4 9 50] These studies suggested a cortical deafferentation but our results suggest that a cortical infarct not seen on CT might also account for the cortical hypoperfusion. Cortical metabolism has been evaluated in the present patients with pure capsulostriate infarcts using single photon emission CT and these results will be reported later.

Our results suggest that pure lenticulostriate infarcts usually produce mild neuropsychological changes and that more severe aphasia, especially of the Broca's type and other neuropsychological deficits like hemineglect and gestural apraxia are mainly due to associated cortical lesions. These results have to be confirmed by further studies assessing a larger number of patients. Our study supports the need for a precise radiological analysis by MRI, as it shows associated lesions that could account for the variability of clinicoanatomical correlations usually reported.

Appendix: scoring of drawings tests on verbal command condition

Score	*Clock*	*Daisy*	*Human face*
0	No omission	No omission	No omission
1	One quarter vacant	One contralesional missing petal	Contralesional hair missing
2	Half a clock vacant	Partially completed on the contralesional side	Contralesional ear missing
3		Contralesional half completely missing	Missing contralesional eye
4			Half of face completely missing

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