

ACUTE TRANSCORTICAL MIXED APHASIA

A CAROTID OCCLUSION SYNDROME WITH PIAL AND WATERSHED INFARCTS

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SUMMARY

Four of 1 200 consecutive patients with their first stroke showed acute transcortical mixed aphasia (TMA) characterized by nonfluent speech with impaired naming, semantic paraphasias, echolalia, impaired comprehension, good repetition, reading, and writing on dictation. All 4 had left internal carotid artery (ICA) occlusion with ipsilateral anterior pial territory infarction (precentral-central sulcus artery territory) and watershed infarction between the middle and posterior cerebral artery territories, which spared and 'isolated' the perisylvian speech areas. Although rare, acute TMA is highly suggestive of infarction due to ICA occlusion, in that it is probably related to simultaneous embolism (anterior pial infarction) and haemodynamic insufficiency (posterior watershed infarction).

INTRODUCTION

Although the concept of transcortical aphasia goes back to Wernicke (1874), it was Kurt Goldstein (1917) who first used this terminology to describe speech disturbances which do not involve the elementary components of language. Spontaneous speech and verbal outflow may be decreased (transcortical motor aphasia) or comprehension may be impaired (transcortical sensory aphasia), but typically there is no agrammatism or syntax dysfunction, and no phonemic paraphasia, with a remarkable sparing of the abilities of verbal reproduction, such as repetition and dictation. The preservation of the elementary components of speech corresponds anatomically to the absence of involvement of the perisylvian speech areas, including Broca's area, Wernicke's area, the arcuate fasciculus and Heschl's gyrus. In transcortical motor aphasia, there is an interruption of the connections between the supplementary motor area and Broca's area, usually from a lesion anterolateral to the left frontal horn (Mazzocchi and Vignolo, 1979; Freedman *et al.*, 1984). In transcortical sensory aphasia, the lesion may be parieto-occipital (posterior cerebral artery territory) or posterior parietal (watershed area between the posterior and middle cerebral artery territories) (Kertesz *et al.*, 1982; Bogousslavsky and Regli, 1986a). In the mixed form (TMA), the motor and

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sensory components combine and yield a characteristic speech disorder, with nonfluent spontaneous speech, poor naming and comprehension, but good repetition. In this aphasia, in which there is no reciprocal access between the words processed phonemically and the semantic processing (Heilman *et al.*, 1976, 1981), the speech areas may be considered to be 'isolated' from the rest of the brain. It has been termed the syndrome of isolation (Geschwind *et al.*, 1968), or deactivation (Cambier *et al.*, 1980) of the speech areas. This rare syndrome has been studied mainly in its chronic form related to diffuse brain processes, in global aphasia evolving towards TMA, or in patients with recurrent strokes (Geschwind *et al.*, 1968; Brown, 1975; Whitaker, 1976; Cambier *et al.*, 1980; Assal *et al.*, 1983; Speedie *et al.*, 1984). We have studied 4 patients who demonstrate that TMA may occur acutely, in the context of an acute ischaemic stroke. The typical finding of two independent infarcts in the left hemisphere, which had a similar topography in all 4 cases, together with the presence of an occlusion of the ipsilateral internal

TABLE. CHARACTERISTICS OF APHASIA

	Case 1	Case 2	Case 3	Case 4
Spontaneous speech	Reduced (sentences: not more than 3 words)	Isolated words	Reduced (sentences: not more than 4 words)	Absent
Semantic paraphasia	+	+	+	+ (often 'aberrant')
Phonemic paraphasia	—	—	—	—
Dysarthria	—	—	—	—
Voice volume	Normal	Normal	Normal	Normal
Naming ¹	1/76	0/76	Absent or semantic paraphasias (Boston Test not done)	0/76
Repetition ³				
Series of 3-8 phonemes	Normal	Normal for series of 5 items, tendency to repeat only end of series of > 5 items	Normal	Normal for series ≤ 4-5 items, tendency to repeat end of series
Series of 3-5 words	Normal		Normal	
Series of 3-10 words	Normal		Normal	
Echolalia	+	+	+	+
Auditory comprehension ²	0/36	0/36	Only simple orders (Token Test not done)	Only simple orders, 4/36
Spontaneous writing	Only his name	Absent	Absent	Absent
Dictation	Few omissions or perseveration of letters	Normal	Normal	Normal
Reading Letters	Normal	Normal	Normal	Normal
Text	Normal except slight hemineglect, but no understanding	Normal, but no understanding	Normal, but no understanding	Normal, but no understanding

¹ Boston Naming Test (Kaplan *et al.*, 1976). ² Token Test (De Renzi and Vignolo, 1962). ³ Batterie des examens neuropsychologiques du CHUV (Assal, 1985).

carotid artery (ICA), suggest that acute TMA may be specific of stroke due to ICA occlusion with simultaneous embolization and haemodynamic failure.

PATIENTS

The 4 patients were French-speaking. They were selected from among 1 200 patients with a first stroke admitted consecutively to our service over a 6-yr period. They corresponded to 2.4% of the 170 stroke patients with ipsilateral ICA occlusion. This proportion is accurate as all stroke patients undergo a standard protocol of investigations, including Doppler ultrasound with frequency analysis. Cerebral CT, ECG, standard blood tests, ECG monitoring and, when indicated, echocardiography are also performed systematically. Every patient with a supratentorial stroke undergoes neuropsychological testing, using a standard battery of tests (Assal, 1985). TMA was defined as a nonfluent aphasia with impaired naming, semantic but no phonemic paraphasia, and no dysarthria, together with impaired comprehension with or without an echolalic tendency, and good repetition, reading and writing on dictation (though without understanding). The topography of pial territory infarcts on CT was evaluated following the guidelines of Damasio (1983). Our method for diagnosing borderzone infarction between the territory of the middle, anterior and posterior cerebral arteries (watershed infarcts) has been detailed elsewhere (Bogousslavsky and Regli, 1986b). One patient (Case 1) has been published previously with a detailed neuropsychological study (Bogousslavsky *et al.*, 1985).

Case reports

Case 1. A 68-yr-old right-handed man with atrial fibrillation for 3 yrs suddenly lost consciousness. When he regained consciousness a few minutes later, he had right-sided weakness and speech disturbances. On admission, blood pressure was 140/90 mmHg and he had an irregular pulse (100/min) due to atrial fibrillation. He had a moderate right hemiparesis involving the face and

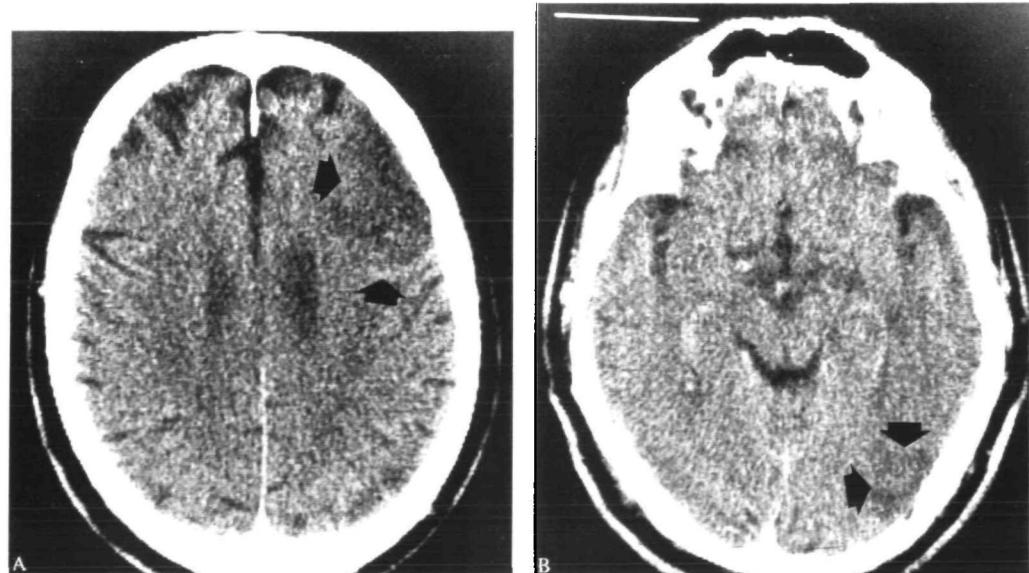


FIG. 1. CT scan. *Case 1.* A, infarct in the territory of the left precentral artery. B, watershed infarct between the territory of the left middle and posterior cerebral arteries (arrows).

upper limb, with hyperactive tendon reflexes. The plantar response was flexor on the left, and indifferent on the right. Tactile and pain sensation were mildly decreased in the right side of the face. On unilateral visual stimulation, no hemianopia was present, but he had right visual extinction on double simultaneous visual stimulation, and he tended to neglect visual information in the right hemispace. Spontaneous speech was markedly reduced and the patient did not produce sentences of more than 3 words ('je veux manger', i.e., 'I want to eat'), with several semantic paraphasias. Object naming was much impaired (1/76, Boston Naming Test), with semantic paraphasias ('stylo' (pen) instead of 'maison' (house), 'chaise' (chair) instead of 'table' (table)). This contrasted with full preservation of the repetition of phonemes and short and long (10 words: 'il fait beau aujourd'hui et j'aimerais aller me promener dehors') sentences; moreover, there was a tendency to repeat every word or sentence pronounced by the examiner. Comprehension was severely disturbed (0/36, Token Test) and echolalia usually replaced the execution of orders. Reading was spared, except for some omissions due to hemineglect. Writing with the left hand on dictation showed only rare letter omissions or perseverations, but spontaneously the patient wrote only his name. A CT scan on admission was normal, but 1 wk later showed 2 superficial infarcts in the left hemisphere, 1 in the superior-posterior part of the frontal lobe (precentral artery territory), and 1 in the posterior parietal region (watershed territory) (fig. 1). On Doppler ultrasound with frequency analysis, the left ICA was not detected and the flow was decreased in the ipsilateral ophthalmic artery, with increase after compression of the ipsilateral superficial temporal artery. Echocardiography showed left atrial enlargement without thrombus. Standard blood tests were unremarkable, except for a haematocrit at 0.47. Within 2 wks, the patient improved markedly and on discharge he had only a slight pronator drift on the right, with decreased spontaneous speech, object-naming difficulties with semantic paraphasias and moderate comprehension impairment (24/36, Token Test).

Case 2. A 72-yr-old right-handed man, who was a smoker and had been hypertensive for 10 yrs, had an episode of right facial weakness with dysarthria for 2 h. This was followed 4 days later by shaking of the right hand for 10 min and inability to speak. On admission, blood pressure was

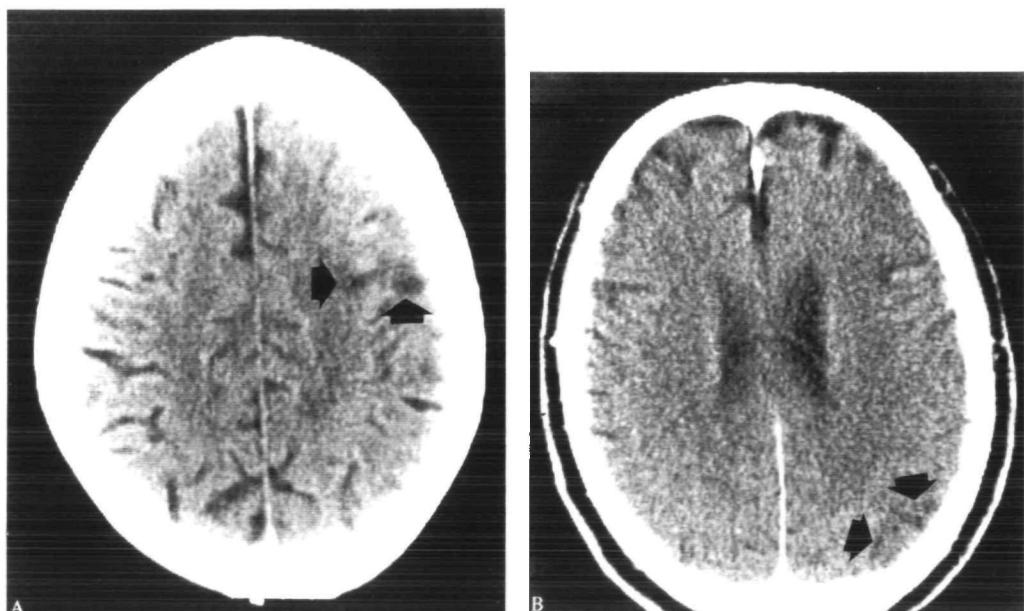


FIG. 2. CT scan with contrast, *Case 2*. A, infarct in the territory of the precentral-central artery branches on the left. B, watershed infarct between the territory of the left middle and posterior cerebral arteries (arrows).

160/80 mmHg, with a regular pulse (80/min). A mild faciobrachioradial hemiparesis was present, with an extensor plantar response on the right. The visual fields could not be examined because of lack of comprehension. No hemisensory defect was noted. There was a left Horner's syndrome with miosis and narrowing of the palpebral fissure. Only isolated words were produced spontaneously. Object naming was absent or yielded semantic paraphasias ('bibelot' (curio) instead of 'aquarium', 'car' (bus) instead of 'train'). The repetition of phonemes, words and sentences was normal for short items, but with words of more than 5 phonemes and sentences of more than 5 words, the patient tended to repeat only the last part of the word or of the sentence. Comprehension was nil (0/36, Token Test), and was often replaced by echolalia. Reading was slowed but otherwise normal, but the patient had no understanding of the text. He did not write spontaneously, but on dictation (sentences up to 5 words) the only abnormalities were related to the right-sided weakness. A CT scan performed 1 day after admission showed 2 superficial infarcts in the left hemisphere, 1 cortical-subcortical in the superior and posterior frontal region (precentral artery-central sulcus artery territory), and 1 in the posterior parietal region (watershed territory) (fig. 2). Doppler ultrasound and digital intra-arterial angiography showed occlusion of the left internal carotid artery without reversed ophthalmic flow. Because of poor resolution, the collateral circulation to the left hemisphere could not be adequately assessed. ECG showed flat or inverted T waves in I, II, III, aVL, aVF. The fasting blood cholesterol concentration was 7.3 mmol/l and the haematocrit 0.48. On discharge 3 wks later, there was only a mild pronator drift and the aphasia had improved (more fluent spontaneous speech, Token Test 14/36, object naming showing only episodic semantic paraphasias). As comprehension improved and the visual fields could be assessed, a lower right quadrantanopia was demonstrable.

Case 3. A 59-yr-old right-handed man without known risk factors except for cigarette smoking was admitted 2 days after he experienced moderate left frontotemporal headaches with speech disturbances and tingling in the right index finger. On admission, his blood pressure was 180/100 mmHg, with a regular pulse (86/min). He had bilateral carotid bruits. There was a mild right lower

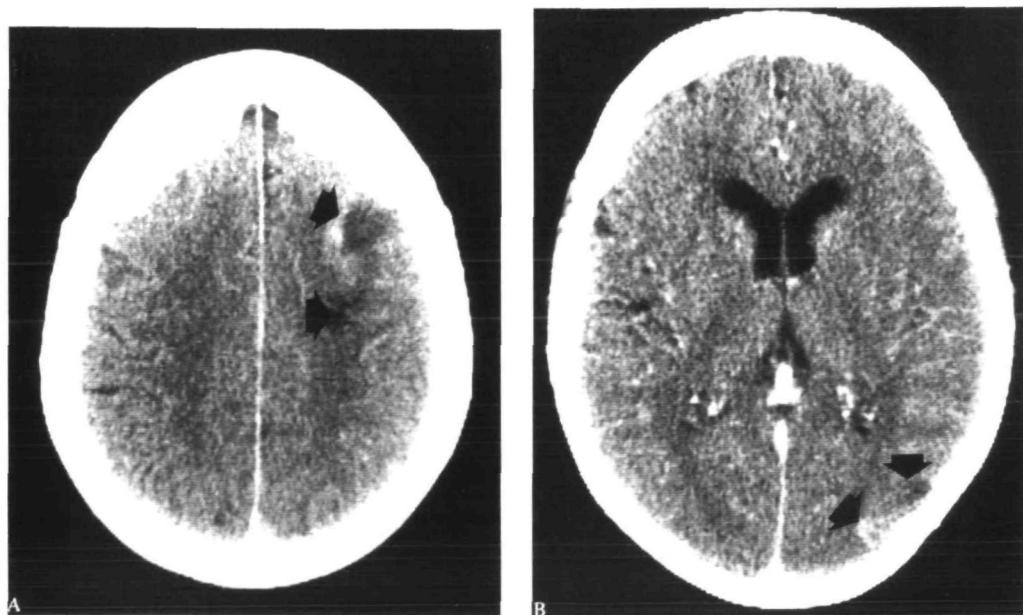


FIG. 3. CT scan with contrast, *Case 3*. A, infarct in the territory of the precentral-central artery branches on the left. B, watershed infarct between the territory of the left middle and posterior cerebral arteries (arrows).

facial weakness and a right pronator drift, with normal tendon reflexes and flexor plantar responses. Tactile, pain, temperature, vibration and position sensation was slightly decreased on the right side of the body with decreased abdominal reflexes on that side. A lower right quadrantanopia was present. There was no dysarthria, but the patient did not produce sentences of more than 4 words and had word-finding difficulties ('maisonnée' (house) instead of 'bibliothèque' (library), 'piscine' (swimming pool) instead of 'aquarium'). Naming was replaced by semantic paraphasias or was absent. There was no agrammatism, but the sentences were meaningless. He could comprehend only very simple orders, such as 'close your eyes' or 'open your mouth'; more complex orders were not executed, and the patient tended to repeat the order. Repetition of isolated phonemes and words and of sequences (3-8 items) was normal. Reading of a text was normal, but retrieval of its meaning was impossible. The patient did not write spontaneously, but there was no impairment on dictation. A CT scan on the day of admission showed 2 superficial infarcts in the left hemisphere, 1 in the posterior prefrontal region (precentral artery territory), and 1 in the posterior parietal region (watershed territory) (fig. 3). Doppler ultrasound with frequency analysis showed no diastolic flow in the left common carotid artery, no detection of the left ICA, with reversed ipsilateral ophthalmic flow ipsilaterally, and increased systolic peak frequency (8-9 kHz) in the right ICA suggesting 75% stenosis; on transcranial Doppler, the flow velocity was decreased on the left as compared to the right (32 cm/s vs 50 cm/s). The retinal artery pressure was decreased on the left (60/30 mmHg vs 80/60 on the right). An ECG showed diffuse ischaemic changes. The haematocrit was 0.47, but other routine blood tests were normal. Over the following few days, the patient improved remarkably and, on discharge, one month later, there was only a slight pronator drift, with isolated word-finding difficulties and rare semantic paraphasias; verbal fluency and comprehension had returned to normal.

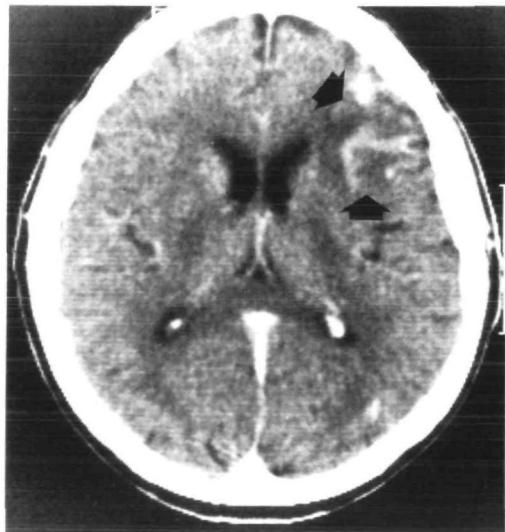


FIG. 4. CT scan with contrast, *Case 4*, showing enhancing infarcts (arrows) in the precentral artery territory and in the posterior watershed area on the left.

Case 4. This 66-yr-old right-handed man was a smoker and had known hypercholesterolaemia. During a walk in the mountains, he suddenly experienced right-sided weakness and was unable to speak for 15 min. This initial muteness was followed by the development of abnormal speech, which seemed 'incoherent' or 'confused' to his wife, without deformed words. On admission 18 h later, his blood pressure was 140/90 mmHg, with a regular pulse (76/min). There was a mild right lower facial weakness with downward drift of the outstretched right arm. The tendon reflexes were

increased in the right upper limb. The plantar responses were flexor. Within the limits of the comprehension disturbances, sensation seemed unimpaired. The patient did not speak spontaneously. Object naming was nil: either the patient could not find any word or he made a semantic paraphasia which was often 'aberrant' (e.g., 'elephant' instead of 'watch'). There was no dysarthria and no phonemic paraphasia. Only simple orders were executed ('open your mouth', 'close your eyes'). More complex orders were repeated but not executed. The repetition of series of phonemes or words of up to 5 items was normal, but with longer series, the patient tended to repeat only the 4 or 5 last items. Reading was performed only on vigorous verbal and visual stimulation by the examiner, but the meaning of the text was not understood. The patient did not write spontaneously but on dictation or on copying a text; he made no errors. A CT scan on the day of admission showed 2 superficial infarcts in the left hemisphere, 1 in the middle posterior region of the frontal lobe (precentral artery-central sulcus artery territory), and a smaller one involving the cortex of the posterior parietal region (watershed territory). On Doppler ultrasound with frequency analysis, there was a decreased systolic peak and no diastolic flow in the left common carotid artery, and no detection of the left ICA, with reversed ipsilateral ophthalmic flow. An ECG and routine blood tests were normal; haematocrit was 0.45. Within 3 wks, the speech disturbances improved, with recovery of some spontaneous speech, object naming (Boston Naming Test 25/76), and comprehension (Token Test 15/36). Echolalia had disappeared. The neurological examination was normal.

DISCUSSION

In our 4 patients, the most striking disturbance was acute TMA, which was associated with some degree of hemiparesis, hemisensory impairment or hemianopia. TMA seems to be a very uncommon manifestation of stroke, as it was not reported in any of 411 patients with a vascular lesion in the dominant hemisphere on CT who had neuropsychological testing (Barat *et al.*, 1978; Mazzocchi and Vignolo, 1979; Basso *et al.*, 1985). The first report of a patient with speech disturbances suggestive of TMA seems to be that by Hübner (1889), who studied a 64-yr-old man who had a transient loss of speech which recurred 1 month later; on examination, spontaneous speech was reduced to 'ja, jawohl, nunja', and there was loss of comprehension of spoken and written language contrasting with good repetition, writing spontaneously and on dictation, and reading. At autopsy, some cortical atrophy was noted, and the patient had 3 infarcts in the left hemisphere, 2 superficial (1 in the posterior-inferior part of the parietal lobe, 1 at the sylvian edge of F_3 , 1 in the lenticulocapsular region); the state of the cerebral arteries was not mentioned. Cambier *et al.* (1980) reported a 60-yr-old man who suffered a left anterior capsular infarct followed 2 months later by a second stroke with extension of the first lesion; a posterior watershed infarct was also visible on the CT scan. TMA was observed after the second stroke. Ross (1980) reported a 52-yr-old man with a subarachnoid haemorrhage due to a ruptured anterior communicating artery aneurysm, whose CT scan showed extensive infarction in the left anterior cerebral artery territory; he suggested that the motor component of TMA was due to involvement of the left supplementary motor area and that the sensory component was due to involvement of the left supplementary sensory area. However, the extent of brain damage was difficult to assess in this patient with severe subarachnoid bleeding, and it must

be emphasized that TMA has not been reported in any other patient with an infarct limited to the left anterior cerebral artery territory, even when both motor and sensory supplementary areas are involved (Környey, 1975; Bogousslavsky *et al.*, 1987b). Ross's patient was initially unconscious, then had global aphasia, and TMA was observed only 4 months later (it was not acute). In all other reported cases of TMA in cerebral ischaemia, large, diffuse, often bilateral lesions were present (though the perisylvian speech areas were completely or partially spared), and TMA usually developed after a phase of global aphasia (Geschwind *et al.*, 1968; Brown, 1975; Whitaker, 1976; Assal *et al.*, 1983; Speedie *et al.*, 1984). In one patient, the cause was carbon monoxide intoxication (Geschwind *et al.*, 1968), but it was unclear in the other patients, in whom no information on the state of the cerebral arteries was given. Transcortical features have been reported in aphasia due to subcortical stroke with lenticulocapsular or thalamic involvement (Mohr *et al.*, 1975; Cappa and Vignolo, 1979; McFarling *et al.*, 1982; Puel *et al.*, 1984; Bogousslavsky *et al.*, 1986c). However, in this setting, the aphasia differs from TMA by a decrease in voice volume, only slight comprehension impairment, and frequent disturbance of articulation and prosody, so that it has been called 'atypical aphasia' or 'dissident aphasia' (Damasio *et al.*, 1982; Hungerbühler *et al.*, 1984; Puel *et al.*, 1984).

Our findings suggest that acute TMA may be specific of stroke due to ICA occlusion. All 4 patients had a stereotyped topography of the 2 infarcts, which occurred simultaneously in the left hemisphere: one was anterior to Broca's area and was of pial territory type (precentral-central sulcus arteries territory), and the other was posterior to Wernicke's area and was watershed between the middle and posterior cerebral artery territory. Pial territory infarcts are usually embolic in ICA occlusion, occurring usually at the time of occlusion (Castaigne *et al.*, 1970; Pessin *et al.*, 1979). On the other hand, although embolism may account for rare cases of infarction in arterial borderzones (Torvik, 1984; Bogousslavsky and Regli, 1986a), most watershed infarcts are related to haemodynamic failure (Zülch, 1961; Adams *et al.*, 1966; Ross Russell and Bharucha, 1978). When unilateral, watershed infarcts are usually associated with severe stenosis or occlusion of the ipsilateral ICA, in up to 90% of the cases (Wodarz, 1980; Bogousslavsky and Regli, 1986a). The occurrence of simultaneous infarcts has usually not been emphasized in the series of patients with ICA occlusion examined by CT (Radü and Moseley, 1978; Takagi and Shinohara, 1981; Goto *et al.*, 1982; Ringelstein *et al.*, 1983; Bogousslavsky and Regli, 1984). But conditions other than ICA occlusion have never been reported to yield the simultaneous anterior pial and posterior watershed infarcts which may produce acute TMA, probably because any other condition is unlikely to cause embolism simultaneous to haemodynamic insufficiency in one hemisphere.

A syndrome other than acute TMA, global aphasia without hemiparesis, may be due to two infarcts occurring simultaneously in the dominant hemisphere (Van Horn and Hawes, 1982). It is usually due to two discrete pial territory infarcts

involving Broca's and Wernicke's areas, but sparing the motor strip and cortico-spinal pathways (Damasio, 1981; Van Horn and Hawes, 1982; Tranel *et al.*, 1986). However, this syndrome may be due to causes of embolism other than ICA occlusion (Van Horn and Hawes, 1982; Legatt *et al.*, 1987). It may also occur from a single infarct in the dominant hemisphere (Ferro, 1983), and is even not specific of cerebral infarction (Legatt *et al.*, 1987).

On the other hand, another two-infarct syndrome, the optocerebral syndrome, which is due to simultaneous watershed infarction in the brain and ipsilateral optic nerve head, is highly suggestive of ipsilateral ICA occlusion (Radovici and Lasco, 1948; Waybright *et al.*, 1982; Bogousslavsky *et al.*, 1987a). In stroke patients with ICA occlusions, acute TMA has the same prevalence as the optocerebral syndrome (2.4%) (Bogousslavsky *et al.*, 1987a), and may be as specific for ICA occlusion. Although rare, acute TMA yields a characteristic aphasic picture. Its occurrence in acute stroke should point towards combined pial and watershed territory infarction in the setting of ICA occlusion, probably from simultaneous embolic and haemodynamic phenomena.

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