Isolation of Speech Area from Focal Brain Ischemia

JULIEN BOGOUSSLAVSKY, M.D., FRANCO REGLI, M.D., AND GIL ASSAL, M.D.

SUMMARY A patient with atrial fibrillation and internal carotid artery occlusion developed mixed transcortical aphasia. The CT scan showed two recent distinct infarcts in the dominant hemisphere, one in the precentral artery area (pial artery infarct) and one in the borderzone area between the posterior and middle cerebral arteries territories (watershed infarct). The perisylvian speech areas were spared, but probably disconnected from other areas by the infarcts. The syndrome of isolation of speech area may be caused by vascular conditions which are able to produce simultaneous pial artery and watershed infarcts, and is not necessarily related to more extensive processes of the brain.

Neurological examination

This 68-year-old right-handed man had been taking oral digoxin (0.25 mg/day 5 days in the week) for three years because of chronic non-valvular atrial fibrillation with moderate heart failure. He was admitted one morning after he suddenly lost consciousness for a few minutes, with simultaneous development of speech disturbances and right-sided brachio-facial weakness.

On admission, atrial fibrillation was present (ventricular rhythm 100/min), and blood pressure was 140/90 mm Hg.

Neurological examination

In neurological examination, no cervical bruit could be heard. Optic fundi, ocular mobility and pupils were normal. No hemianopia was present (assessed on unilateral visual stimulation), but the patient showed marked visual hemineglect on the right side: he did not pay attention to the activities of other patients, nurses and doctors in the part of the room situated on his right, and on double simultaneous visual stimulation his responses were limited to the left side. Optokinetisch nystagmus was slowed in the horizontal and vertical directions. A right-sided inferior facial paresis with a slight tactile and algesic facial hypoesthesia (decreased cor-

Skeletal muscle was slow in the horizontal and vertical directions. A right-sided inferior facial paresis with a slight touch and algesic facial hypoesthesia (decreased cor-

From the Neurology Department and Center of Neuropsychology, Center Hospitalier Universitaire Vaudois, Lausanne, Switzerland.

Address correspondence to: Julien Bogousslavsky, M.D., Neurology Department and Center for Neuropsychology, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.

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When trying to reproduce movements carried out by the examiner, the patient showed perseverations. Drawing a cube (copy) was normal. Oral and written calculation was impossible. Other neuropsychological functions could not be examined, because of the comprehension defects.

The standard laboratory data were normal. The ECG showed the known atrial fibrillation but did not show evidence for a myocardial infarct. A CT scan performed the day of admission showed no abnormality, but 7 days later two areas of decreased density suggesting infarction were observed in the left hemisphere, one in the superior and posterior part of the frontal lobe, and one at the parieto-temporo-occipital junction (fig. 1.). Between these two hypodense areas, the perisylvian zone was not involved. After contrast enhancement, the border of the lesions became slightly hypodense. Continuous wave Doppler ultrasounds showed absence of detection of the left internal carotid with decreased flow in the ophthalmic artery, which increased after compression of the superficial temporal artery. Echocardiography showed sclerosis of the aortic and mitral valves without stenosis, with moderate left atrial dilatation. Holter examination showed atrial fibrillation with phases of tachy-arythmia and ventricular extrasystoles (type 1Vα of Lown).

Evolution
The patient was put on intravenous heparin therapy (30,000 U/day), which was changed after one week for oral acenocoumarol (Prothrombin time: 23–30%). The right-sided hemiparesis nearly recovered within two weeks, with disappearance of the hemineglect. Echolalia disappeared and the comprehension disturbances improved (24 correct answers36, token test), but object naming difficulties persisted, with semantic paraphasias, and the verbal fluency remained considerably impaired (1 name of animal produced within 1 minute). The repetition remained unaltered. Verbal and visual learning showed marked impairment (2–3/15 items), but the Stroop test and the Wisconsin card sorting test were within normal range.

Discussion
Our patient with atrial fibrillation and left internal carotid artery occlusion suffered a right-sided moderate facio-brachial hemiparesis with slight superficial hypoesthesia, and spatial hemineglect, associated with severe language disturbances. Markedly reduced verbal fluency and spontaneous speech with object naming impairment, semantic paraphasias, echolalia, and severe disturbances of comprehension contrasted with the absence of phonetic alterations, phonemic paraphasias and agrammatism, with complete sparing of repetition. These language disturbances correspond to the classical mixed (motor + sensory) transcortical aphasia, in which paucity of speech is associated with severe comprehension deficits, but with remarkable preservation of repetition.1-5

The CT scan pictures showed two infarcts in the left hemisphere.
cerebral hemisphere. The first involved the superior and posterior part of the frontal lobe, with extension towards the lateral ventricular horn, and typically corresponded to the upper area supplied by the precentral branch of the middle cerebral artery. This pial artery infarct is highly suggestive of embolic infarction. Interestingly enough, the location of this infarct perfectly corresponds to the most common lesion site involved in motor transcortical aphasia, which is in the white matter anterolateral to the left frontal horn and disrupts the connections between the supplementary motor area and the frontal perisylvian speech zone. In this situation, the frontal motor center of speech (Broca's area) is disconnected from the "supramotor" center initiating the process of speech (supplementary motor area).

The second infarct was located at the junction between the temporal, parietal and occipital lobes, which is also the limit between the middle and posterior cerebral arteries superficial areas. This watershed infarction is typically due to hemodynamic hypoperfusion, although micro-embolization has been suggested in some cases, and may occur in this location as rarely as in less than 3% of internal carotid artery occlusions. The location of this infarct typically corresponds to one of the two cortico-subcortical lesion sites giving rise to sensory transcortical aphasia (poor comprehension with excellent repetition). The second site is more medial, inferior and posterior, and is located in the posterior cerebral artery area.

In our patient, it may be assumed from the CT pictures that the perisylvian speech areas (Heschl's gyrus, Broca's area, Wernicke's area, arcuate fasciculus) were spared by both infarcts, which in fact disconnected them from the rest of the hemisphere, giving rise to the classical isolation of speech area syndrome. A few cases with "isolation aphasia" had anatomic studies, which showed isolation of perisylvian language areas related to extensive damage of other parts of the hemisphere. To our knowledge, only one other case with two discrete lesions (one posterior, one anterior) has been reported. Global aphasia without hemiparesis has already been reported as the result of two pial artery infarcts in the dominant hemisphere, involving the anterior and the posterior part of the perisylvian speech area, respectively. In our case the frontal infarct was more anterior and the posterior infarct more posterior than in these cases of global aphasia, and spared the perisylvian speech areas. Global aphasia without hemiparesis has been considered as a sign of embolic ischemia, which could occur in heart disease and in ICA stenosis. In ICA occlusion, simultaneous occurrence of two distinct infarcts has not been mentioned in the published series with CT studies, and is probably very uncommon. It may be suggested that in our patient, association of atrial fibrillation with occlusion of internal carotid artery was responsible for acute hypoperfusion in the posterior vascular borderzone territory, producing watershed infarction. The anterior pial artery infarct was probably due to embolization in the precentral artery at the moment when the internal carotid artery became occluded.

Isolation of speech area from focal brain ischemia appears to be due to conditions which are able to produce pial artery and watershed infarcts simultaneously.

References
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