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Letter to the Editor

Longitudinal positron emission tomography study in a patient with presumed extracranial internal carotid dissection

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The overall prognosis after internal carotid artery dissection is related to the severity of the stroke signs and to the ability of developing collateral circulation through the circulus of Willis (Guillon *et al*., 1998). Complete recanalization occurs in 62% after occlusion and cannot be predicted from the initial symptoms (Steinke *et al*., 1994). Without recanalization after 6 months, subsequent improvement is rarely shown (Sturzenegger *et al*., 1995). The overall long-term prognosis is considered, on one hand, to be good with a low recurrence rate (Leys *et al*., 1995), but on the other hand, seizures occur more frequently than expected after acute stroke in general (Engelter *et al*., 2000). Blood flow and metabolic studies have rarely been performed in this condition. Single photon emission computed tomography has shown hyperemia in the acute stage, followed later on by severe reduction in flow in patients with persistent neurological deficits and infarction (Rommer *et al*., 1994, Bremerick *et al*., 1997).

We present the case history of a 48 year-old man, who initially complained of right hemicranial headache and developed a moderate left-sided hemiparesis a few days later. The prior history was unremarkable except for alcohol abuse, heavy cigarette smoking and a fall without loss of consciousness one month before admission. Because the patient did not improve, he was transferred to our neurological department one month after his admission to the peripheral hospital. The neurological examination at that time revealed weakness affecting predominantly left arm and face with hyperreflexia and Babinski's sign. The patient was alert with moderate dysarthria, but without sensory disturbances or visual field defects. CT scan revealed a peri-insular and deep brain infarct in the right middle cerebral artery territory (Fig. 1A). Conventional angiography suggested a dissection of the right internal carotid artery above the bifurcation with total occlusion (Fig. 1B). Extensive cardiac and blood examinations were all normal. The patient was treated with 160 mg aspirin daily. The neurological status remained unchanged

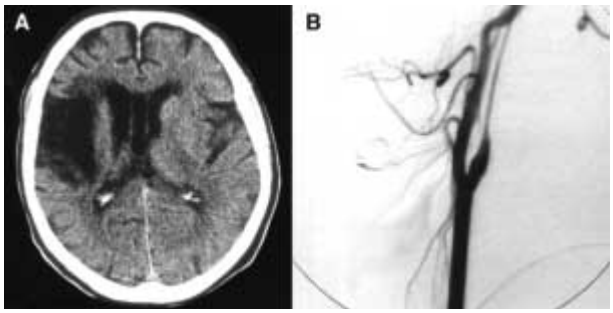

Fig. 1. — A. CT scan of the brain showing the right peri-insular and deep infarct in the middle cerebral artery territory. B. Conventional right carotid angiography showing the severe luminal narrowing of the internal carotid artery beginning approximately 2 cm postbifurcation and involving the entire cervical segment (string sign) with occlusion before the entry to the petrous bone.

during the following 3 years and repeated duplex sonography performed at the time of the repeated positron emission tomography (PET) examinations, still revealed the persistence of the right internal carotid occlusion. After an epileptic seizure, there was a temporary increase of his pre-existing neurological deficit.

PET using the ^{15}O steady state inhalation technique, was performed 2, 14, 26, and 38 months after the initial stroke. At first, PET showed low regional blood flow (rCBF) and metabolic rate for oxygen ($rCMRO_2$) mainly in the infarcted area, but also in the remainder of the right cerebral hemisphere and, to a lesser degree, in the contralateral hemisphere. On follow-up, the rCBF and $rCMRO_2$ remained low in the infarcted area and even further decreased after the epileptic insult. In the other regions, there was a progressive increase of rCBF and $rCMRO_2$, not influenced by the seizure episode (Fig. 2).

The findings of this longitudinal PET study indicate the late and progressive development of collateral circulation in the affected hemisphere, while the improvement of rCBF and $rCMRO_2$ in the contralateral cerebral hemisphere can be due to dissolution of transcortical diaschisis (De Reuck *et al*.,

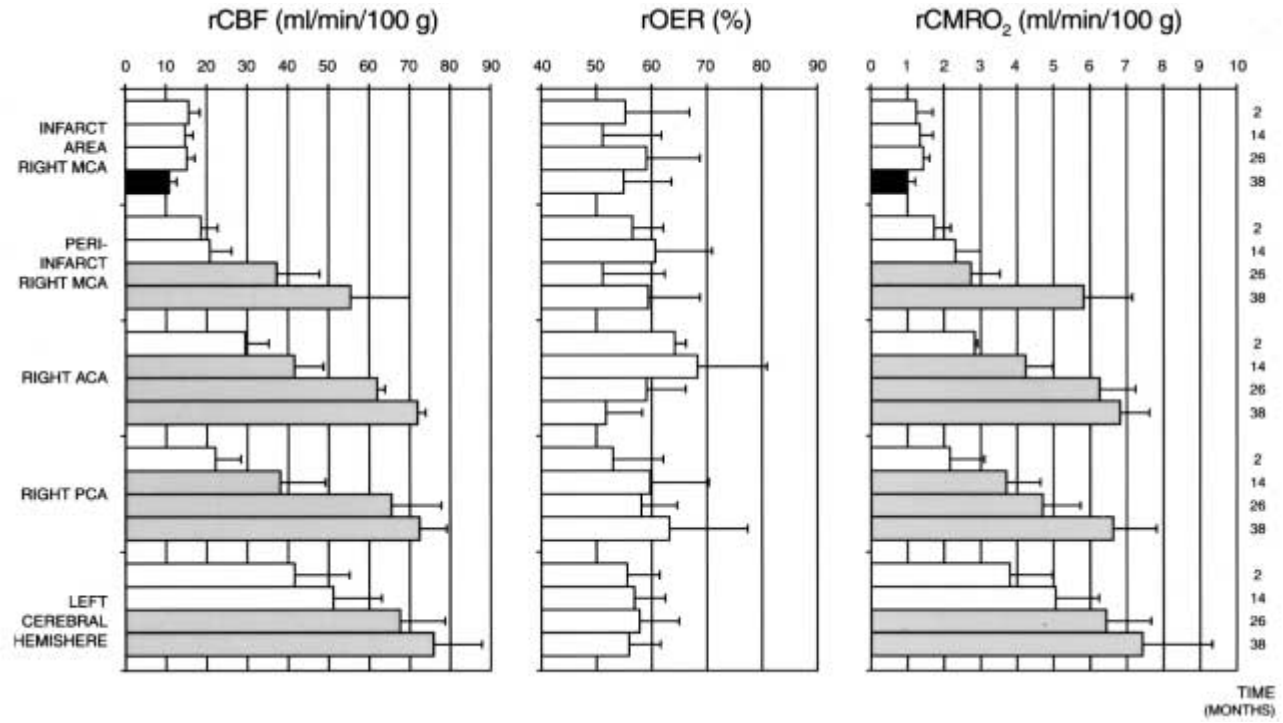

FIG. 2. — Regional cerebral bloodflow (rCBF), oxygen extraction rate (rOER) and metabolic rate for oxygen ($rCMRO_2$) in the infarcted area, peri-infarct region in the middle cerebral artery (MCA), anterior (ACA) and posterior cerebral artery (ACP) territories and in the contralateral hemisphere, 2, 14, 26 and 38 months after the initial stroke. The black and gray bars represent statistically significants differences compared to the results of the first PET study.

1997). The further focal decrease of rCBF and $rCMRO_2$ after the seizure episode can be explained by a new infarct, probably of thrombo-embolic origin (De Reuck *et al*., 2000) and supports the hypothesis that most cerebral infarctions in internal carotid artery dissection are of embolic rather than of hemodynamic origin (Lucas *et al*., 1998).

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