Letters To The Editor

Letters to the Editor will be published, if suitable, as space permits. They should not exceed 1,000 words (typed double space) in length, and may be subject to editing or abridgement.

Regional Cerebral Blood Flow in Patients With Transient Ischemic Attacks Studied by Xenon-133 Inhalation and Emission Tomography

To the Editor:
The recent article, "Regional Cerebral Blood Flow in Patients with Transient Ischemic Attacks Studied by Xenon-133 Inhalation and Emission Tomography," by Vorstrup, Hemmingsen et al1 from the Bispebjerg Hospital, report detailed elegant CBF data, but the data was interpreted in an unorthodox way with speculations to clinical relevance, pathology, and pathophysiologic. In this article, the authors chose to center the discussion upon three patients who had transient symptoms, no CT lesion, caroid artery stenosis, and persistent focally diminished rCBF despite successful vascular reconstructive surgery. The authors postulate that these patients had "a state of elective ischemic necrosis of the parenchyma (especially neurons) without emolism."

Scholz2 is cited as the only reference for this condition which is usually called selective neuronal necrosis. Neuronal necrosis is a known pathologic finding but usually follows a diffuse hypoxic or toxic insult. Scholz in his lengthy chapter did not correlate the pathology with clinical conditions and never used the word, Schlaganfall (stroke). There is no hint that any of the patients with selective neuronal damage he studied had focal vascular disease. We are unaware of any description of focal neuronal necrosis due to a corresponding focal vascular occlusive lesion and would be indebted to the authors if they could supply such a citation.

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References

To the Editor:
Drs. Burke and Caplan have commented on our study "rCBF in patients with transient ischemic attacks studied by 133Xenon inhalation and emission tomography," in particular our interpretation of the focal low flow areas seen in several cases having a normal CT scan. As these focal changes persisted 3 months after reconstructive neck vessel surgery, in one case after an extracranial-intracranial bypass shunt, we considered that a hemodynamic impediment could not account for the reduced rCBF. We therefore speculated on possible neuropathologic lesions that might underly these pathophysiologic findings. We quite agree, that the study by Scholz1 only described diffuse selective neuronal cell damage in patients with generalized hypoxia, as after cardiac arrest. However, Lassen et al2 have earlier reported on 2 stroke cases in whom a selective neuronal necrosis was seen surrounding the areas of complete infarction. Also recent experimental studies have provided supportive evidence of selective neuronal necrosis both following brief (15 minutes to 3 hours) ischemia,3 and following a permanent occlusion of the middle cerebral artery.4 We are presently counting the neuronal density in close and remote cortical regions in 6 patients with large chronic cerebral infarcts. In one such patient, CBF studies had been obtained 3 months prior to the patient’s death by a brain-stem apoplexy. The CBF studies had shown large flow areas in CT negative regions. In this case as well as in the other 5 cases having large infarcts, there was no evidence of a selective neuronal cell damage adjacent to the infarct (unpublished observations).

Functional inactivation caused by undercutting of afferent neurons might explain the low flow areas seen in our TIA patients with CT verified infarction. However, in the patients without proven infarction, this explanation is not very satisfactory.

Postmortem histopathologic studies do not permit a decision about the functional state of a neuron. It may yet be a possibility, that transient cerebral ischemia can cause direct (hypoxic) cell damage and hence, a lowered metabolic demand visualized in vivo as a low flow area.

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References

Idiopathic Hypertrophic Subaortic Stenosis and Cerebral Ischemia

To the Editor:
We would like to comment on the interesting paper by Furlan et al, "Cerebrovascular complications associated with idiopathic hypertrophic subaortic stenosis," published in Stroke.1 This is a follow-up study of a large number of patients with idiopathic hypertrophic subaortic stenosis (IHSS): out of these, 11 developed cerebrovascular complications (5 had a stroke and 6 had a TIA) in an average follow-up of 5.5 years, but focal cerebral ischemia was never the presenting manifestation of IHSS.

Idiopathic Hypertrophic Subaortic Stenosis and Cerebral Ischemia

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