middle cerebral artery: reference values at rest and during hyper-ventilation in healthy volunteers in relation to age and sex.


**Background Factors and Clinical Symptoms of Major Depression With Silent Cerebral Infarction**

To the Editor:

In their recent article, "Background factors and clinical symp- toms of major depression with silent cerebral infarction," Fujikawa et al.1 used magnetic resonance imaging to determine whether depressed patients had or did not have silent cerebral infarction and then compared the two groups in terms of a variety of risk factors for stroke and depression. It is surprising that the authors do not relate their findings to a large body of literature reporting similar results but using different terminology, i.e., leukoencephalopathy, leukoaraiosis, deep-white-matter hyper-intensity, or subcortical hyperintensity. These terms are essentially used to describe hyperintensities on T$_2$-weighted spin-echo magnetic resonance images of the brain.

Small hyperintensities are related to perivascular spaces; larger hyperintensities (maximum linear dimension, >5 mm) are usually seen on pathological examination to consist of areas of myelin pallor, infarcts, or lacunes. These large signal hyperintensities are the basis of patient classification by Fujikawa et al.1 A brief review of the literature that links these hyperintensities to depression may be of interest to your readers. Since the publication of our initial report indicating that these hyperintensities are common in elderly depressed patients,2 numerous researchers have noted the high frequency and severity of these hyperintensities in elderly depressed patients compared with control subjects.3,4 Coffey et al.5 noted a higher incidence of cortical infarctions and leukoencephalopathy in depressed patients, and Figiel et al.6 reported that the frequencies of large deep white-matter hyperintensities and lesions of the basal ganglia were greater in late-onset depressed patients than in patients with early-onset depressed patients of similar age. Basal-ganglia hyperintensities have also been linked to an increased likelihood of delirium induced by antidepressants or electroconvulsive treatment.6,7 This fairly extensive literature and the report by Fujikawa et al.1 suggest that cerebrovascular damage may indeed be important in the pathophysiology of major depression in the elderly and worthy of further study.

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References


12. Normal $^{99m}$Tc-HmPAO Distribution in Large Subcortical Middle Cerebral Artery Infarct

The term “luxury perfusion” is used to describe situations of paradoxical cerebral blood flow (CBF) increase or flow values that are high in comparison with metabolic demand. The idea prevailed until 1993 that the $^{99m}$Tc-hexamethylpropyleneamine oxide ($^{99m}$Tc-HmPAO) hyperfixation observed in the subacute stage after cerebral infarct was due to luxury perfusion. However, recent observations have shown that in these circumstances, hyperfixation with $^{99m}$Tc-HmPAO does not always correspond with CBF increase. In the following case, single-photon computed tomography (SPECT) was clearly abnormal with $^{18}$F and $^{99m}$Tc ethylcysteinate dimer ($^{99m}$Tc-ECD) but paradoxically normal with $^{99m}$Tc-HmPAO in the subacute stage of middle cerebral artery (MCA) infarct.

A 34-year-old man came to our hospital on May 21, 1991, with meningeal hemorrhage consequent to rupture of a left carotid artery aneurysm. He underwent surgery 3 days later without
'Normal' 99mTc-HmPAO distribution in large subacute middle cerebral artery infarct.
M Steinling, D Huglo, H Kolesnikov, R Vergnes and M Rousseaux

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