hereby especially, the hypometabolism in this region did not correlate with the cortex area in the MR scan.

Another point raised by Dr. Gorman is the question whether whiplash injury could "trigger" Alzheimer's disease-like pathology. Up to now this was only speculation. Nevertheless, there is increasing evidence of a link between head injury and the subsequent onset of Alzheimer's disease. Deposits of amyloid beta-proteins are found not only in cases of dementia pugilistica but in some patients dying after a single episode of severe head injury (*4*). A tenfold increase in the risk of Alzheimer's disease was associated with both apolipoprotein E epsilon 4 and a history of traumatic head injury, compared to a twofold increase in risk with apolipoprotein E epsilon 4 alone, whereas head injury in the absence of an apolipoprotein E epsilon 4 allele did not increase risk (*5*).

Because of head restraints, whiplash trauma today also produces a head impact that can lead to direct brain damage. However, the question whether senile dementia could be the long-term outcome of an earlier whiplash injury remains open.

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Technetium-99m-HMPAO Brain SPECT in Systemic Lupus Erythematosus with Central Nervous System Involvement

TO THE EDITOR: We read with great interest the article by Lin et al. (*1*) about brain SPECT in systemic lupus erythematosus. After working for the past 2 years on a similar group of patients, we formed conclusions similar in some aspects, but different in others, which may be the result of a different methodological approach. We previously reported a part of our observations (*2,3*); the rest of this material is in preparation to be published.

We performed brain SPECT using a triple-head gamma camera, MRI and anticardiolipin antibody (ACA) assays in 66 systemic lupus erythematosus, 14 Sjögren's syndrome, 12 diffuse scleroderma and 4 mixed collagenosis patients. In most, an acetazolamide stress test was performed 3 days later. SPECT results were compared with a group of 25 healthy volunteers. Brain SPECT was abnormal in 91% of systemic lupus erythematosus patients, with MRI abnormal in only 26%. Until that point, our observations are in agreement with those of Lin et al. (*1*). In our opinion, however, although visual interpretation may sometimes be superior to semiquantitative, some subtle changes may be overlooked in visual analysis, especially regarding intrahemispherical asymmetries. We found diffuse hypoperfusion of both frontal lobes when compared to HMPAO cerebellar uptake and healthy control subject indices in 30% of systemic lupus erythematosus, 50% of Sjögren's syndrome and none of diffuse scleroderma patients. This correlated well with cognitive impairment assessed by psychometric tests.

Also, analysis of interhemispherical asymmetries revealed significant problems, which are underlined by the authors. We believe that the subtle changes seen on images of systemic lupus erythematosus parients require comparison to physiological asymmetries of a healthy volunteer database and visual interpretation is not sufficient. As in systemic lupus erythematosus, at least in our patient group, diffuse HMPAO uptake defects are more frequent than focal ones and comparison with healthy control subject results seem to be particularly important, especially in analyzing basal ganglia HMPAO uptake, where, even in healthy subjects standard deviation of interhemispherical HMPAO may be high (s.d. = 8.7% in our material). Therefore, basal ganglia SPECT results should be interpreted with caution. Another problem is ACA assay results. The authors did not find correlation with clinical findings. How did the correlation with SPECT images look? This is important because thrombo-embolic brain infarcts are more frequent in ACA patients (*4*). This was consistent with our SPECT findings, where 9 of 12 patients with ACA had multiple focal HMPAO uptake defects (more than five per patient) resembling multi-infarct dementia.

Last, but not least, there is the problem of future guidelines in systemic lupus erythematosus brain research. What is the appreciable in Lin et al.'s (*1*) article is the size of the patient groups, subdivision to different pathological groups and insight into basal ganglia. The other interesting points seem to be cerebrovascular reactivity in systemic lupus erythematosus, reversibility of cerebral blood flow (CBF) changes and comparison with other connective tissue diseases. Lin et al. (*1*) describe improvement in brain perfusion in a patient after methylprednisolone therapy. During control scanning after steroid therapy, we found brain perfusion improvement in 12 of 20 patients, no change in 6 and new hypoperfused areas in 2. Of 6 ACA-positive patients, none showed improvement. Another crucial point might be reactivity to acetazolamide-induced hypercapnia, as carbon dioxide is the most important physiological regulator of regional CBF redistribution. We found it altered (no change or paradoxical HMPAO uptake improvement in 48% of patients). In comparing systemic lupus erythematosus with the other connective tissue diseases, we found a pattern similar to systemic lupus erythematosus in mixed collagenosis, multifocal-defect pattern but not hypofrontality in Sjögren's syndrome and little or no CBF changes in diffuse scleroderma. This may be related to different pathological mechanisms.

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REPLY: We thank Dr. Lass et al. for their comments and agree that comparison with a normal database of ^{99m}Tc-HMPAO brain SPECT may be the best way to diagnose central nervous system (CNS) involvement in systemic lupus erythematosus patients. Establishment of a normal database, however, is an enormous task. Dr. Lass collected 25 healthy volunteers, but this may not be enough. Dividing the normal control subjects into different age groups is necessary since age may affect the regional blood flow in the brain tissue (*1,2*). In addition, the use of steroids