FOCAL SEIZURES AND BRAIN CT SCANS IN PSYCHIATRIC PATIENTS

To the Editor: In a 1981 report, Sarnat and colleagues1 surveyed 123 psychiatric patients who underwent CT scanning of the brain for the first time. Although this patient population had concurrent electroencephalographic seizures, another 81 patients with "normal" or "isolated" seizures had CT scans. These two groups then were compared to a control group of 100 psychiatric patients, selected for the basis of psychiatric illness in many hospitals. Although it seems reasonable to use psychiatric patients as controls for investigating major psychiatric illnesses, it may be less reasonable to use psychiatric patients as controls for investigating psychiatric symptoms. John J. Bell and colleagues2 have taken this approach, using the psychiatric patient as a control group. It appears that careful measurement of structural abnormalities facilitates the search for more reliable guide to CT scanning than of focal-convulsion signs in this population.

Thomas F. Beversdorf, M.D.
Freeman G. Hill, M.D.
Linda O. Widrow, Ph.D.
Veterans Administration Medical Center
Memphis, TN 38104

Richard G. W. Ellis, M.D.
The Johns Hopkins Hospital
Orlando, FL 32803


ALZHEIMER'S DISEASE: DOES NEURON PLASTICITY PRECEDE TO ANXIOUS NEUROPLASTIC DEGENERATION?

To the Editor: Ghyselinck hypothesizes that distruption of neuroplasticity may be the basis for cellular degenerating diseases (March 14 issue). To explain why neuronal cells in the brain are affected and not others, he suggests that cells with large axonal trees, because of their great demand for axonal transport, are especially vulnerable to axonal disruption. Gephyrin's inability to tolerate these transport abnormalities, he suggests, may be central to Alzheimer's disease. We suggest that cell plasticity as well as the state of the axonal tree may give some clues to the cellular transport. The plasticity of nonneural cells has been related to a variety of degenerative states, some of which cannot be transport-related. A profound neuroplastic defect in the axonal transport system is in the development of presenile dementia, presumably accompanied by a specific defect of new neuron plasticity.

Nuclear staining a high degree of plasticity probably form the substrates of abnormality and hence, lead to abnormalities in Alzheimer's disease. Neuropeptide synthesis has been associated with abnormality in the nervous system, and abnormalities in the basal ganglia are observed in some cases of Alzheimer's disease. Alzheimer's disease also damages the basal ganglia and attributing this to a pathological state in the basal ganglia, so on reconvalescence as the result of potential clinician's plastination. A presynaptic-elicited plasticity of the axonal transport system may occur in the basal ganglia, presumably accompanied by a specific defect of new neuron plasticity.

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SHARED ANTIGENS ON ACETYLCYLCHOLINE RECEPTOR AND BACTERIAL PROTEINS

To the Editor: The report by Sferrazza et al. (Jan. 24 issue) describes the sharing of antigenic determinants between the nitric-oxide synthase (eNOS)-recognizing monoclonal antibodies directed against the nitric-oxide-synthase and the bacterial antigen VirB2. The authors state that their primary evidence for the demonstration of bacterial antigens were the results obtained with the bacterial protein VirB2. The authors further note that these results were obtained with the bacterial protein VirB2 and that the bacterial antigens were shared with the monoclonal antibodies directed against the bacterial protein VirB2.

This may well turn out to be correct, but there is one question that needs more discussion. In the study by Sferrazza et al., the bacterial protein VirB2 was both of the eNOS class. Since only the bacterial antigens were the eNOS class, this indicates a limitation.

However, the issue of shared antigens with bacterial antigens is also of interest. The authors state that their results with the bacterial protein VirB2 were obtained with the bacterial antigen VirB2, and that the shared antigens were with the bacterial protein VirB2. This may be true, but it is important to note that the bacterial antigens were shared with the monoclonal antibodies directed against the bacterial protein VirB2. Therefore, the bacterial antigens were also shared with the monoclonal antibodies directed against the bacterial protein VirB2.

In summary, the data presented by Sferrazza et al. demonstrate an interesting cross-reaction between a bacterial protein and a monoclonal antibody. However, further studies are needed to confirm these findings.