Methods: The clinical states of all subjects were determined prospectively. The brains were fixed in 10% formalin. The entire hippocampus was sectioned coronally at 50\,\mu m and stained by the Nissl method. The optical fractionator method [Anat. Rec. 231 (1991) 482] was used to estimate the total number of neurons of the granule cell layer, dentate hilus, CA3-2, CA1, and subiculum. Results: We again observed a significant loss of neurons (51\%) in the CA1 of AD cases compared to controls\, ($P<0.05$). In hilus and subiculum of the AD group, we found 28 and 29\%, non significant losses of neurons, respectively. No differences were observed in either CA2-3 or granule cells. In the hippocampi of the possible AD (early stage) group, the distribution of the neuronal estimates in CA1 and subiculum overlapped with those of the control and AD groups. Conclusion: Our new study confirms the presence of a striking disease-specific degeneration of CA1 in AD. Our observations also suggest that when amyloid deposits and neuritic changes are well established in the neocortex of subjects with possible AD, the loss of neurons in hippocampus has not yet occurred or is incipient. Awareness of this difference in the relative vulnerability of neocortex and hippocampus to AD may be relevant for the diagnosis and therapy of early AD.

**NEURONAL LOSS IN THE HIPPOCAMPUS OF INDIVIDUALS IN EARLY AND ADVANCED STAGES OF ALZHEIMER’S DISEASE**

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**Background and Objectives:** Our previous stereological study of the hippocampus in Alzheimer’s disease (AD) showed a marked neuronal loss in CA1 and smaller, but significant losses in hilus and subiculum. This pattern of loss was significantly different from that in normal aging. In this study of subjects from the Baltimore Longitudinal Study of Aging and our Alzheimer’s Research Center, we estimated the total number of neurons in subregions of the hippocampi of (1) subjects with normal cognition, but abundant neuritic amyloid plaques in the neocortex (possible AD by CERAD pathological diagnostic criteria) ($n = 10$); (2) demented subjects with pathologically confirmed AD ($n = 14$) and (3) age-matched controls with normal cogni-