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Commentary

Where's the Chicken?

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The paper by Sasaki et al (1) in this issue reminds me of a phenomenon that occurred in the early days of network television in North America. Arthur Godfrey, a variety show host, was in the habit of gently chiding the sponsors of his show. During a commercial for Lipton instant chicken soup, he remarked, "The chicken is there. You just can't see it."

And so it is with the nucleus basalis of Meynert. Like the chicken in Lipton soup, we know it lies in the substantia innominata, but so far, we can't see it. Yet this paper is remarkable when one reviews the history of the neuroradiology of Alzheimer disease. Early investigators noted that global atrophy on the pneumoencephalogram was a frequent accompaniment of both Alzheimer disease and normal aging. It was only natural that, in the early days of computed tomography (CT), attempts were made to relate the presence and degree of dementia qualitatively and quantitatively to the finding of large ventricles and sulci (2, 3). This gave rise to objective and subjective, manual and automated, area and volume measures of the ventricles and sulci (4, 5). When all was said and done, it was determined that, although average ventricular and sulcal dimensions were larger in *groups* of persons with dementia when compared with *groups* of age-matched control subjects, the CT scan by itself was of little use in distinguishing a person with Alzheimer disease from a normally aging person or from persons of all ages with a variety of other dementing or nondementing degenerative diseases.

In the late 1970s and early 1980s, neuroscientists began to define the "biochemical anatomy" of dementia. Specifically, this involved microanatomic study of the location of cholinergic neurons and the concentrations of choline acetyl transferase, acetylcholine, and acetylcholinesterase in various parts of the brain. It became apparent that there were important "association areas," particularly in the medial temporal lobes, that harbored cholinergic neurons and were integral to the maintenance of normal cognitive function, particularly memory. Because pathologic studies showed that these areas often contained markers of Alzheimer disease such as neurofibrillary tangles, neuritic plaques, and granulovacuolar degeneration, and chemical and anatomic evidence of neuronal dropout, CT studies of persons with dementia concentrated on imaging the medial temporal lobes. The work of George and deLeon and their colleagues (6, 7) showed that sequential CT studies of the amount of "hippocampal cerebrospinal fluid" bore a direct relationship to the degree of cognitive decline that could be measured in groups of persons with mild and no dementia. However, the consensus was that the major role of CT in Alzheimer disease was to rule out potentially reversible causes of dementia.

With the introduction of magnetic resonance imaging, several groups, convinced that the degree of atrophy in the medial temporal lobes was significant, measured the "interuncal distance" (8, 9). This subsequently was shown to correlate with the degree of overall cerebral atrophy, but was of little value in predicting cognitive function.

However, with this concentration on magnetic resonance study of the temporal lobes, it became apparent that the ability of magnetic resonance to differentiate exquisitely between gray and white matter enabled detailed study of the anatomy and volume of the hippocampal formation and amygdala, association areas where there were known to be pathologic and chemical changes of Alzheimer disease. By concentrating on magnetic resonance changes in the hippocampal formation and amygdala, these studies (10, 11) provided a way to study this disease and eliminate the variable of global atrophy in trying to separate healthy elderly patients from those with Alzheimer disease.

Index terms: Brain, anatomy; Brain, magnetic resonance; Dementia; Prosencephalon; Commentaries

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Although the nucleus basalis of Meynert has been recognized anatomically for many years, it was only in the 1980s that it came to be recognized as an important association center that demonstrated neuronal dropout in Alzheimer disease (12, 13) and, later, in a number of other dementing illnesses. The resolution limits of CT prevented attempts to see this structure, let alone the larger substantia innominata in which it lies embedded. Sasaki et al (1) have shown that it is indeed feasible to measure the dimensions of the substantia innominata in vivo, and that these measures relate to the presence or absence of Alzheimer disease. We are not yet quite there, but we have come a long way from the imaging of "atrophy" to a more detailed study of the critical association areas of the brain that relate to cognitive function. Sasaki and colleagues (1) have laid down one more challenge, to identify and characterize in vivo the size, signal, and spectral properties of the nucleus of Meynert—the "chicken" that lies hidden in the "chicken soup" of the substantia innominata.

References

1. Sasaki M, Ehara S, Tamakawa Y, et al. MR anatomy of the substantia innominata and findings in Alzheimer disease: a preliminary report. *AJNR Am J Neuroradiol* 1995;16:2001–2007
2. Huckman MS, Fox J, Topel J. The validity of criteria for the evaluation of cerebral atrophy by computed tomography. *Radiology* 1975;116:85–92
3. Hahn FJY, Rim K. Frontal ventricular dimensions on normal computed tomography. *AJR Am J Roentgenol* 1976;126:593–596
4. Gado M, Hughes CP, Danziger W, et al. Volumetric measurements of cerebrospinal fluid spaces in demented subjects and controls. *Radiology* 1982;144:535–538
5. George AE, deLeon MJ, Rosenbloom S, et al. Ventricular volume and cognitive deficit: a computed tomographic study. *Radiology* 1983;149:493–498
6. George AE, deLeon MJ, Stylopoulos LA, et al. CT diagnostic features of Alzheimer disease: importance of the choroidal/hippocampal fissure complex. *AJNR Am J Neuroradiol* 1990;11: 101–107
7. deLeon MJ, Golomb J, George AE, et al. The radiologic prediction of Alzheimer disease: the atrophic hippocampal formation. *AJNR Am J Neuroradiol* 1993;14:897–906
8. Dahlbeck SW, McCluney KW, Yeakley JW, et al. The interuncal distance: a new MR measurement for the hippocampal atrophy of Alzheimer disease. *AJNR Am J Neuroradiol* 1991;12:931–932
9. Howieson J, Kaye JA, Holm L, Howieson D. Interuncal distance: marker of aging and Alzheimer disease. *AJNR Am J Neuroradiol* 1993;14:647–650
10. Jack CR, Petersen RC, O'Brien PC, Tangalos EG. MR-based hippocampal volumetry in the diagnosis of Alzheimer's disease. *Neurology* 1992;42:183–188
11. Lehericy S, Baulac M, Chiras J, et al. Amygdalohippocampal volume measurements in the early stages of Alzheimer disease. *AJNR Am J Neuroradiol* 1994;15:929–937
12. Whitehouse PJ, Price DL, Clark AW, et al. Alzheimer disease: evidence for selective loss of cholinergic neurons in the nucleus basalis. *Ann Neurol* 1981;10:122–126
13. Whitehouse PJ, Price DL, Struble RG, et al. Alzheimer's disease and senile dementia: loss of neurons in the basal forebrain. *Science* 1982;215:1237–1239