R. G. Schneider, *Science* **129**, 719 (1959); D. L. Rucknagel, *J. Lab. Clin. Med.* **49**, 896 (1957); W. S. Pollitzer, A. I. Chernoff, L. L. Horton, M. Froehlich, *Science* **129**, 216 (1959); C. V. Tondo and F. M. Salzano, *Science* **132**, 1893 (1960); T. Arends, *Nature* **190**, 93 (1961); H. E. Sutton, G. A. Matson, A. R. Robinson, R. W. Koucky, *Am. J. Human Genet.* **12**, 338 (1960).
6. This study was supported in part by contract Nonr-1228 (14) between the Office of Naval Research and Northwestern University and in part by funding under Public Law 480, Section 104(c).

17 December 1962

"Caudate-Induced" Cortical Potentials: Comparison between Monkey and Cat

Abstract. *The segment of the internal capsule which carries axons relating to the sensorimotor cortex does not closely adjoin the caudate in the monkey as it does in the cat. Therefore, in seeking evidence for caudate-induced cortical responses, activation of the adjoining internal capsule by stimulus spread can be avoided. In the monkey, caudate stimulation never produced cortical responses, and only capsule stimulation evoked the potential complex which has been attributed to caudate stimulation in the cat.*

Much electrophysiological evidence suggests a direct as well as an indirect route between the caudate nucleus and cerebral cortex, especially the motor and somatosensory areas (*1, 2*). However, caudate-cortical connections have been disputed by others (*3*) with evidence that it is the spread of the stimulus to the internal capsule, and not the primary effect of caudate excitation, which yields the cortical responses. The controversy stems mainly from the fact that electrophysiological investigation relating to caudate-cortical projection has been carried out in the cat. There the sensorimotor cortex lies anterior to the caudate head, and capsular fibers coursing toward the sensorimotor area hug the caudate closely (Fig. 1, top). To resolve the problem we have studied the situation in the monkey, in which the sensorimotor cortex lies behind the caudate head, and fibers passing between the thalamus and sensorimotor cortex are not contiguous to that nucleus (Fig. 1, bottom).

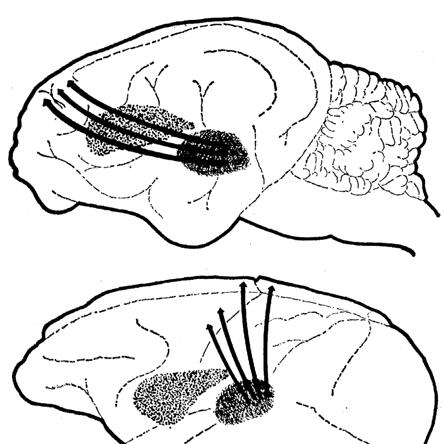

Fig. 1. Diagrammatic relationship of the caudate nucleus to the internal capsule (those fibers radiating from the thalamus to the sensorimotor cortex) in cat (top) and monkey (bottom). Stippled area, caudate; shaded area, thalamus; heavy lines, capsular fibers radiating from the thalamus to the sensorimotor cortex.

Twelve monkeys (macaque) were prepared under Surital anesthesia. Wound surfaces were heavily infiltrated with procaine, and the animals were immobilized with Flaxedil (gallamine triethiodide) and were carried on artificial respiration. Stimulating electrode tips were directed stereotaxically into either the caudate or the internal capsule, stimulus sites being verified histologically. The caudate head was the usual stimulus site, although more posterior areas of the nucleus were also stimulated. The internal capsule was activated at levels adjacent to thalamic nuclei VL and VPL. Recording was from the pre- and post-rolandic cortex, electrodes being arranged either transcortically or with one placed on the cortical surface and the other on the frontal periosteum. Responses were displayed on an oscilloscope after amplification with a resistance-capacitance coupled amplifier, and photographed. Comparative observations were made upon the anterior and posterior sigmoid gyri of the cat.

In the cat a single caudate stimulus evoked a *short latency* diphasic potential followed by a 250-msec *positive deflection.* After the positivity a series of 8- to 12-per-second rhythmic waves ["caudate spindle" (*2*)] appeared (Fig. 2, cat, *A* and *B*). Upon slowly repetitive (5- to 8-per-second) stimulation, *recruiting type potentials* could be elicited (Fig. 2, line *C*). Identical responses could be obtained with weaker stimuli applied to the adjacent internal capsule. In the monkey, *only* stimulation of the internal capsule evoked cortical responses like those activated from caudate stimulus positions in the cat (Fig. 2, column 3). Caudate stimulation never produced cortical responses in the monkey, this being the case even when stimuli 50 times the intensity of those delivered to the capsule were used (Fig. 2, column 2).

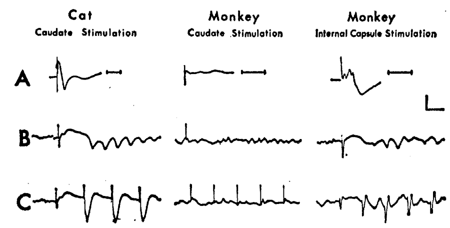

Fig. 2. Evoked responses from motor cortex in cat and monkey. *A*, single responses on fast time line; time marker adjacent to each trace = 16 msec. *B*, single responses on slow time line. *C*, repetitively evoked responses (six per second). Time marker (horizontal line of right angle in upper right) for *B* and *C* = 100 msec. Vertical line of right angle is 1 mv cal for all responses. Only early latency diphasic potential is evident in the expanded time line in *A* (left and right columns). In the same columns in *B*, the ensuing positivity and "caudate spindle" are evident as well. No responses appear in the center column. Straight vertical lines are shock artifacts. Positive is up, and polarity refers to the electrode on the cortical surface.

We conclude that all components of the cortical response to caudate stimulation (early latency potential, caudate spindle, and recruiting type potentials) thought to be caudate-induced and presented as evidence for caudate-cortical projections (*1, 2*) are activated from the internal capsule and not from the caudate directly (*4*).

SIDNEY GOLDRING
LYNDON U. ANTHONY
PAUL E. STOHR
JAMES L. O'LEARY
Beaumont-May Institute of Neurology and *Divisions of Neurosurgery* and *Neurology, Washington University School of Medicine, St. Louis, Missouri*

References and Notes

1. R. Jung and J. F. Tonnies, *Arch. Psychiat. Nervenkr.* **185**, 701 (1950); F. A. Mettler, C. A. Hovde, H. Grundfest, *Federation Proc.* **11**, 107 (1952); C. Ajmone-Marsan and M. Dilworth, *Electroencephalog. Clin. Neurophysiol.* Suppl. 3, 85 (1953); T. Shimamoto and M. Verzeano, *J. Neurophysiol.* **17**, 278 (1954); D. P. Purpura, E. M. Houspian, H. Grundfest, *Arch. Ital. Biol.* **96**, 145 (1958).
2. N. A. Buchwald, E. J. Wyers, T. Okuma, G. Heuser, *Electroencephalog. Clin. Neurophysiol.* **13**, 509 (1961).
3. C. Terzuolo and N. Stoupel, *Bruxelles Méd.* **33**, 411 (1953); A. M. Laursen, *Acta Physiol. Scand.* **53**, 318 (1961).
4. Aided by grants from the U.S. Public Health Service (NB O1517) and the Allen P. and Josephine B. Green Foundation.

7 December 1962