These examples illustrate the kinds of conditioned alterations exhibited by single units in response to the unreinforced stimulus. With respect to the CS^+, our findings of conditioned modification of firing pattern in single elements of parastriate cortex are similar to those previously reported (*10, 11*). The unique feature of the present study was the demonstration of conditioned responses to the unreinforced stimulus. Cells exhibited modification which was selective for reinforcement contingency; that is, they changed either to the CS^+ or to the CS^-. Thus far, we have not observed any single cell that has exhibited conditioned alteration to both CS^+ and CS^-. The number of cells that showed selective change to CS^- (13 out of 86) was approximately equal to the number exhibiting modification to CS^+ (10 out of 86) (*12*).

Differences in the conditions necessary for producing conditioning were also apparent between cells which exhibited modification in response to either the CS^+ or CS^-. In general, a modification to CS^+ did not take place unless there was a distinct cellular response to shock. Such was not the case for cells selectively responsive to CS^-. The latter often did not respond to the UCS directly, although clear-cut changes in the local field potentials indicated that the UCS did influence other elements of the same population (Fig. 1, panels 3, 8, 12, and 14).

We think that these results demonstrate a possible neural substrate of conditioned inhibition (*13*). The existence of single units exhibiting selective conditioned modification to the CS^- suggests that the circuitry mediating conditioned inhibition contains elements not involved in conditional excitatory processes, at least at the level of parastriate cortex. A more definitive demonstration of such functional distinctiveness would require that in a test where reinforcement contingencies are reversed, a cell showing modification in response to the CS^- originally would again be modified in response to the new CS^-. A cell showing conditioned alteration to CS^+ would be expected to exhibit similar faithfulness to its original reinforcement contingency. Yet, even without this most persuasive test, such qualitative specificity as we have described provides a further example of cell-specific behavior in learning comparable to the precise tuning to other aspects of environment and experience which appears to be characteristic of the sensory systems in general (*14*).

The occurrence of anatomically distinct elements subserving conditioned excitation and inhibition would not necessarily be predicted from behavioral research. Yet the latter deals only with outcomes, that is, with the final motor end product. At an earlier stage in the encoding of experience, it appears that conditioned inhibitory and excitatory processes involve separate neuronal systems. Further analysis of the cellular constituents of these separate systems may provide direct information on the time-course and kinetics of these processes at the neuronal level.

LEYLA DE TOLEDO-MORRELL*
Department of Psychology, University of Toronto, Toronto, Ontario, Canada M5S 1A1
THOMAS J. HOEPPNER
Department of Neurological Sciences, Rush Medical College, Chicago, Illinois 60612
FRANK MORRELL
Department of Neurological Sciences, Rush Medical College, and *Marine Biological Laboratory, Woods Hole, Massachusetts 02543*

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9. This "extinction procedure" did not ordinarily result in actual extinction since the latter requires more trials than we employed [see (*10*)], especially in view of the relatively large number of reinforced trials previously delivered. Therefore, the extinction procedure served as a test of conditioning.
10. F. Morrell, in *The Neurosciences: A Study Program*, G. C. Quarton, T. Melnechuk, F. O. Schmitt, Eds. (Rockefeller Univ. Press, New York, 1967), p. 452; in *The Brain and Human Behavior*, A. G. Karczmar and J. C. Eccles, Eds. (Springer-Verlag, New York, 1972), p. 259.
11. Further details on CS^+ responses will be described elsewhere (F. Morrell, T. J. Hoeppner, L. de Toledo-Morrell, in preparation).
12. The other cases (8 out of 86) where modifiability in cell activity was observed consisted either of populations with two or three undifferentiable cells or of units which showed the same response pattern to the two stimuli prior to conditioning. In these examples, a modification occurred in response to both CS^+ and CS^-. In the case of populations, it may very well be that separate elements were responsible for each type of change. Elements not distinguishing CS^+ from CS^- initially showed the same change to both stimuli after conditioning.
13. Specific tests such as summation or retardation which allow one to designate a CS^- as a conditioned inhibitor could not be used since the same elements did not show conditioning to both CS's. However, since measurable alterations in the pattern of neural activity could be induced by either stimulus, these tests were not regarded as critical.
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* Present address: Departments of Neurological Sciences and of Psychology, Rush Medical College, Chicago, Ill. 60612.

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Commissural Transmission in Humans

An important and intriguing question of human neurobiology concerns the relationship between the two cerebral hemispheres. Salamy has observed that "latency differences between ipsilateral and contralateral somatosensory evoked potentials show maturational trends in keeping with the myelogenic timetable and development of the corpus callosum" (*1*, p. 1409). If these differences reflect a maturation of the corpus callosum, this work is of great interest. We believe, however, that the activity of only the faster callosal axons is likely to be measured by this technique. If we assume that only one synapse is involved, and thereby subtract 0.5 msec from the adult values given by Salamy, the estimated interhemispheric conduction time of the P_2, P_1, and N_1 components of the evoked potential may be considered to be 3.0, 6.5, and 7.5 msec, respectively.

Given an interhemispheric conduction distance of approximately 100 mm, the axonal conduction velocity of impulses mediating such information is approximately 13 to 33 m/sec. Such axon conduction velocities might be expected to be mediated by myelinated axons 2.4 to 6.0 μm in diameter (*2*). Yet Tomasch reports fewer than 10 percent of human callosal axons to be more than 2.5 μm in diameter (*3*). Most myelinated axons are less than 1.5 μm in diameter, and fully 40 percent of callosal axons were found to be unmyelinated. In the macaque, electron microscopy (*4*) reveals such unmyelinated axons to be 0.08 to 0.5 μm in diameter. Such axons would

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be expected to have conduction velocities of 0.6 to 1.7 m/sec or less.

We concur with Salamy that his finding "may prove useful in assessing demyelinating disease and cases in which maturational delay is suspected" (*1*, p. 1410), particularly in those cases in which fibers of large diameter are affected. This technique, however, would probably not be useful when diseases or maturational delay selectively affected that great majority of callosal axons that conduct slowly.

HARVEY A. SWADLOW
Department of Psychology, University of Connecticut, Storrs 06268, and *Department of Neurology, Harvard Medical School, Beth Israel Hospital, Boston, Massachusetts 02215*
NORMAN GESCHWIND
Department of Neurology, Harvard Medical School
STEPHEN G. WAXMAN
Department of Neurology, Stanford University Medical School, and *Veterans Administration Hospital (127), Palo Alto, California 94304*

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Salamy stated, "In the visual modality, a stimulus falling on the left hemiretinae projects exclusively to the right occipital lobe and one on the right hemiretinae projects to the left lobe" (*1*, p. 1409). The fact is that the projections of the human visual system are just the opposite. This error, which unfortunately is not too uncommon, probably results from confusing the visual hemifields with the hemiretinae. Salamy then reversed the findings of Andreassi *et al.* (*2*) to correspond to this conceptual error when stating that "significantly longer latencies and lower amplitudes have been observed over the homolateral cortex after hemifield stimulation" (*1*, p. 1409).

The actual relationships in the human visual system are as follows: stimuli in the left visual half-field fall on the right hemiretinae, which project to the right occipital lobe; conversely, stimuli in the right visual half-field fall on the left hemiretinae, which project to the left occipital lobe (*3*). The longer latencies and lower amplitudes of visual evoked potentials observed over the *contralateral* cortex presumably result from transmission of information from the homolateral hemisphere via the corpus callosum (*2*).

JAY E. GOULD
Department of Psychology, University of West Florida, Pensacola 32504

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It would be nice to be able to specify the fiber population involved in the interhemispheric transfer of sensory information from surface recordings. The averaged evoked potential, however, most likely represents a summation of fast and slow activity within a pathway or pathways. Our inability to precisely identify the generator source for each component of the evoked potential makes untenable the assumptions of a single synapse and known conduction distance. Nevertheless, since the observed maturational effect (ipsilateral-contralateral latency difference) corresponds to myelination of the corpus callosum, it is presumably the excitation of these fibers that contribute most prominently to the elaboration of the evoked potential. Therefore, it may be primarily these large-diameter myelinated fibers which are being measured with my technique.

Gould is quite correct in pointing out the discrepancy regarding hemifield-hemiretinal projection. This was brought to my attention by my colleague J. Robinson, but only after the galleys had been returned. Gould, however, is incorrect in his interpretation of the Andreassi report (*1*). Longer latencies and lower amplitudes were, in fact, observed over the ipsilateral hemisphere (with respect the stimulated field) as I originally stated. More importantly, it should be mentioned that this confusion pertains solely to the Andreassi reference and has no bearing whatsoever on the data presented in my report or on the interpretation of the results.

A. SALAMY
Brain-Behavior Research Center, University of California at Sonoma State Hospital, Eldridge 95431

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