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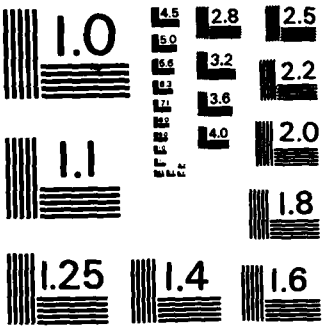

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Neurological investigations of adaptive neural networks were conducted using the classically conditioned nicitating membrane response of rabbit. Experimental approaches involved recording from single brain neurons from awake, behaving animals for the purpose of determining the loci and characteristics of neurons with activity correlated with the NM CR or its inhibition; the use of discrete brain lesions that selectively eliminate the NM CR while at the same time sparing the basic reflex pathway; fiber-tracing anatomical techniques designed to clarify the inter-connectivity among brain regions essentials for the NM CR. These regions include discrete portions of the cerebellum and brain stem. Information from physiological studies has been incorporated into mathematical modes of learning used by adaptive network researchers, and anatomical findings have guided the development of related neuronal models.

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Annual Technical Report No. 2 May 20, 1985

Air Force Office of Scientific Research

RE: AFOSR 83-0215 (Adaptive Networks)

Dr. W.O. Berry, Program Manager

Biological Investigations of Adaptive Networks:

Neuronal Control of Conditioned Responding

Principal Investigator: Dr. John W. Moore

University of Massachusetts, Amherst

I Summary

Neurobiological investigations of adaptive neural networks were conducted using the classically conditioned nictitating membrane response (NM CR) of rabbit, a widely used model system for studies of learning. One experimental approach involved recording from single brain neurons from awake, behaving animals for the purpose of determining the loci and characteristics of neurons with activity correlated with the NM CR or its inhibition. A second approach involved the use of discrete brain lesions that selectively eliminate the NM CR while at the same time sparing the basic reflex pathway. A third approach employed fiber-tracing anatomical techniques designed to clarify the inter-connectivity among brain regions essential for the NM CR. These regions include discrete portions of the cerebellum and brain stem. Information from physiological studies has been incorporated into mathematical models of learning used by adaptive network researchers, and anatomical findings have guided the development of related neuronal models.

II Research Objectives

A. Overview The primary objective for the reporting period was to conduct neurobiological studies relevant to adaptive networks using the classically conditioned nictitating membrane (NM) response as a model system. The classically conditioned NM response (NM CR) in rabbit has been widely adopted as a model system for behavioral, neurobiological, and theoretical studies of mammalian associative learning. Last year's report might be consulted in the interest of continuity. Also, my Research Progress and Forecast Report dated 11/27/84 contains a good deal of information regarding the past year's activities, especially in detailing the rationale for our theoretical and experimetal work during the reporting period. That report describes some relevant scientific interactions that are only briefly noted here.

It is generally held that many brain regions contribute to learning the NM CR and that these regions interact with each other via pathways that parallel the basic reflex pathway subserving the unconditioned response (UR). Neural circuits responsible for the CR converge, on the response side, among motoneuron pools that retract the eyeball, principally the accessory abducens nucleus (AAN). On the stimulus side, rostral elements of the sensory trigeminal complex subserve the unconditioned stimulus (US). A comparatively simple disynaptic reflex arc completes the circuit between stimulation of the eye and defensive retraction of the globe. NM extension is a passive consequence of eyeball retraction. Circuits mediating the NM CR include discrete portions of the cerebellum and reticular information. These systems appear to interact with other brain regions that modulate

the rate of CR acquisition, CR topography, and inhibition; they include the septo-hippocampal complex and poorly differentiated regions of the midbrain.

Behavioral/biological studies of learning using the rabbit NM preparation have implications for adaptive network research because they can guide and validate assumptions underlying mathematical models learning. Promising models suggest algorithms that are potentially applicable to problems of artificial or machine learning. In addition, by successfully modelling the fine-grain topographical features of the learned response in real time, our efforts would appear relevant to problems of sensory motor control in two domains: physiology and robotics.

We have been particularly interested in the model described in 1981 by Drs. R.S. Sutton and A.G. Barto of the Department of Computer and Information Sciences (COINS) in connection with their studies of adaptive networks (R.S. Sutton & A.G. Barto, Toward a modern theory of adaptive networks: Expectation and prediction. Psychological Review, 1981, 88, 135-170). Our group, primarily John E.Desmond, Dr. N.E. Berthier, Diana Blazis, and myself, have developed real-time variants of this model capable of predicting patterns of neuronal firing and CR topography (latency, amplitude, and form) typically observed in the rabbit NM preparation. The model has validity for a variety paradigms and training protocols. This effort has profited from interactions with Barto and Sutton and other experts in relevant behavioral domains: E. James Kehoe and I. Gormezano. In a related line of work, Nestor Schmajuk and myself are rendering attentional models of associative learning from psychology into computational adaptive networks capable, not only describing neural correlates of learned behavior and CR

topography in real time, but also of describing the contribution of the hippocampal formation to the NM CR.

B. Purpose of Specific Studies Specific studies during the reporting period fall into four main categories: electrophysiological, anatomical, lesions, and theoretical.

1. Electrophysiological Briefly, our physiological studies involve single or multiple-unit extracellular recording from brains of awake, behaving animals. Their purpose is to determine the precise loci and firing pattern of neurons with activity related to the CR or its inhibition. This information is crucial for understanding, not only which brain regions and structures are involved in a particular aspect of the CR, but how these regions and structures interact to perform their designated tasks. Neuronal representations of these processes in the form of connectionistic schemas can be viewed as models of parallel cooperative computation. During the initial year, we designed and assemble a computer-assisted laboratory for single-unit neuronal recording concurrent with behavioral testing for the purpose of investigating the relationship between neuronal activity and the NM CR. During the past year we have largely completed two studies (doctoral dissertations) and a preliminary study of cerebellar cortex.

2. Anatomical Over the past year, our anatomical studies have addressed the interconnectivity of brain structures essential for the NM CR, especially the anatomical relationships among the accessory abducen nucleus (AAN), the principal source of motor control the NM CR, and magnocellular red nucleus (RN), an essential premotor link in the circuit. We use implanted WGA-HRP for fiber tracing studies designed to verify our previously reported findings with HRP that RN projects to AAN.

3. Lesion Studies These studies address the question of brain regions/structures essential for the NM CR and its inhibition. One completed study shows that RN lesions made prior to any training prevents acquisition of the NM CR by the eye contralateral to the lesion but not the ipsilateral eye. Furthermore, training of the disrupted eye, although futile with respect to that eye, facilitates acquisition by the unaffected eye, thereby demonstrating positive transfer. Another lesion study is a continuing investigation study into circuits involved in conditioned inhibition (CI). It is exploring the role of lateral septum and the habenula in this aspect of the behavior.

4. Theoretical As mentioned above, this aspect of our activity uses data on the NM CR from behavioral and physiological domains to guide formulation of computational models of learning and assess their validity. One class of models under scrutiny are constrained variants of the original Sutton-Barto (S-B) model consisting of a single adaptive element. The other class of models are real-time networks of elements derived from attentional theories of learning. The latter class of models are not necessarily incompatible with the S-B model; they simply provide an alternative approach.

III. Status of Research

A. Electrophysiological Studies

1. Analysis of learning related single neurons of the brain stem This is John Desmond's dissertation project. Extracellular recording from single neurons were obtained during behavioral training. Most sampled units were located in pontine reticular formation. The

relationship between unit firing patterns and topographical features of the CR was investigated with an impressive array of quantitive tools developed by Desmond, including multiple regression, peristimulus and perireponse histograms, and time-correlograms. Such analyses form a crucial step in efforts to develop computational models models of learning related neuronal activity.

Two main types of CR-related neuronal activity were observed. On-cells increase their activity so as to mirror aspects of CR topography such as latency and amplitude. These neurons form a template of the CR as much as 100 msec before the CR begins. Such neurons are either causally related to the CR or else they reflect processing and representation within a parallel system, as portrayed in previously published neuronal models. A subset of neurons with CR-related activity decrease their firing rates in relation to the NM CR. Figures 1 and 2 appended to this report summarize and illustrate the findings of this study. Desmond's dissertation will be issued as a technical report this summer.

2. Multiple-unit correlates of conditioned inhibition This study is Michael Vigorito's dissertation project. Its methodology resembles that employed in the Desmond study, except that multiple-unit recordings were the rule and single units an exception. Certain theoretical models assume that CI develops in a distinct system that parallels those involved in CR acquisition. Inhibition of a CR presumably arises because of interactions between these two systems at some as yet unknown premotor stage. In this connection it is interesting that a neuronal interpretation of the S-B model does not require a separate system in order to generate CI. Thus, the S-B model cannot be construed as an accurate representation of how CI emerges in

real brains. Nevertheless, a good deal of our simulation work with S-B model concerns scenarios that lead to the development of CI (or negative "synaptic weights" in the model's argot). The model makes interesting and testable predictions about CI in scenarios for which there is as yet no relevant data.

Recording from brain regions now thought to be important for conditioned inhibition, but not for acquisition or generation of the NM CR, Vigorito observed neurons that increased their firing rate as the CR was actively suppressed in the presence of a conditioned inhibitor. Units of this type were observed in lateral septal nuclei, lateral habenula, and ventromedial thalamus (see Figures 3-5). By contrast, units that increase their firing in the absence of CRs were not observed in Desmond's study.

3. Single-unit recording from cerebellar Purkinje cells Neil Berthier has conducted pilot studies showing that hemispheric lobule VI of cerebellar cortex contains Purkinje cells with activity related to the NM CR. Lesion studies have shown that hemispheric lobule VI is essential for the NM CR. Techniques were developed for separate analyses of simple and complex spikes (see Figure 6). These techniques are fundamental to physiological investigations of cerebellum and motor learning, especially from the perspective of well known models of Marr and Albus. The possible role of cerebellar cortex to the NM CR is the subject of a recent lead article by Gelleman and Miles in Trends in NeuroScience (May 1985: vol. 8, pp. 181-183).

B. Anatomical Studies

Studies of the interconnectivity among brain regions implicated in the NM CR were continued using WGA-HRP (TMB) as a marker. Because unilateral lesions of magnocellular red nucleus (RN) and its

projections eliminate the NM CR on the side opposite to the lesion, we used WGA-HRP to determine the trajectory of two of the primary input/output fiber systems to RN: the brachium conjunctivum, which conveys information from anterior nucleus interpositus of the contralateral cerebellar hemisphere; the rubrobulbar tact, which carries information from red nucleus to the region of AAN on contralateral side of the brain stem. A preliminary description of this work has been published in Physiology & Behavior.

Continuing investigation of the crucial connection from RN to motoneurons with WGA-HRP suggests that the principal contribution to the CR from RN to AAN is by way of interneurons in the spinal trigeminal complex. Thus, it now appears that RN may exert the greatest part of its control over the NM CR by exciting a secondary sensory component of the reflex pathway rather than by exciting AAN directly. Nevertheless, WGA-HRP confirms the existence of a sparse direct projection from large RN cells of the dorso-medial portion of RN to AAN, as described in a previously published HRP study from this laboratory.

In order to facilitate data reduction and scientific communication in connection with our anatomical studies, our histogy technician, Anne Dovydaitis, devoted a considerable portion of her time during the past year to producing a representative series of parasagittal sections of the rabbit brain stem and cerebellum. This series complements the series of transverse sections produced Dovydaitis during the initial year of the grant.

C. Lesion Studies

1. Lesions of brachium conjunctivum and rubrobulbar tract eliminate the NM CR As noted, unilateral lesions of these two fibers

systems eliminate the NM CR but not the UR. Other lesions of the dorsolateral pons caudal to the abducens nerve have no effect on behavior. This study, which was conducted by technician Marcy Rosenfield, reinforced our earlier conclusion that the supratrigeminal reticular formation, like the cerebellum, is essential for the NM CR. A description of this wortk has been published in Physiology & Behavior.

2. Bilateral lesions of hypothalamus do not impair conditioned inhibition Diana Blazis, a graduate student in my laboratory since 9/84, conducted this study as an undergraduate honors thesis. A variety of theoretical considerations, together with earlier lesion studies by Moore and his collaborators here and in London, suggested the possibility of hypothalamic involvement in CI. Blazis' study suggests otherwise, and subsequent inspection of the histological material suggested that the habenula may be involved in CI. This hypothesis is consistent with Vigorito's recording study and earlier lesion studies involving the midbrain from this laboratory. Blazis and Rosenfield have initiated lesion studies based on these leads. They are currently investigating effects of septal area lesions on CI, and they plan to extend their studies to implicated portions of the thalamus, and habenula.

3. Lesions that disrupt the NM CR do not affect the UR From a variety of theoretical perspectives (see original proposal leading to the current award), the extent to which lesions that disrupt the CRs might also influence the UR is an important issue. For example, certain neuronal models suggest that CR-disrupting lesions should produce a slight, but nevertheless significant, attentuation of the UR over a range of intensities of the eliciting stimulus. This does not

appear to be the case, based on data presented at the 1984 Society for Neuroscience meetings and in a paper by Rosenfield and myself to appear in Behavioral Brain Research.

D. Theoretical Studies

1. Physiologically constrained Sutton-Barto learning models Literally hundreds of simulations experiments have been conducted in efforts to discover variants of the S-B model that are most consistent with behavioral data on the NM CR. These simulation results have been shared with interested experts at other institutions, and a preliminary report will be presented at the 1985 meetings of the Cognitive Science Society. Figures 7 illustrates a simulation of CI using the latest and, to date, most satisfactory variant of the model.

2. Physiologically constrained attentional models This is basically Nestor Schmajuk's dissertation project. He and I performed hundreds of simulation studies and prepared two papers for publication. These are currently being revised, and a third paper is in preparation. These will be submitted as technical reports this summer. Figure 8 illustrates simulations of response topographies and underlying neuronal firing from the two attentional models under development: (a) the latest variant of the Moore-Stickney model (e.g., see IV #2 below), which successfully predicts many of the effects of hippocampectomy of the NM CR, and (b) Schmajuk's real-time network rendering of the Pearce-Hall model (J.M. Pearce & G. Hall, Variations in the effectiveness of conditioned but not unconditioned stimuli. Psychological Review, 1980, 87, 532-552).

IV Technical Reports

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5. Rosenfield, M.E. & Moore, J.W. Red nucleus lesions impair acquisition of the classically conditioned nictitating membrane response but not eye-to-eye savings or UR amplitude. Behavioral Brain Research, In press.

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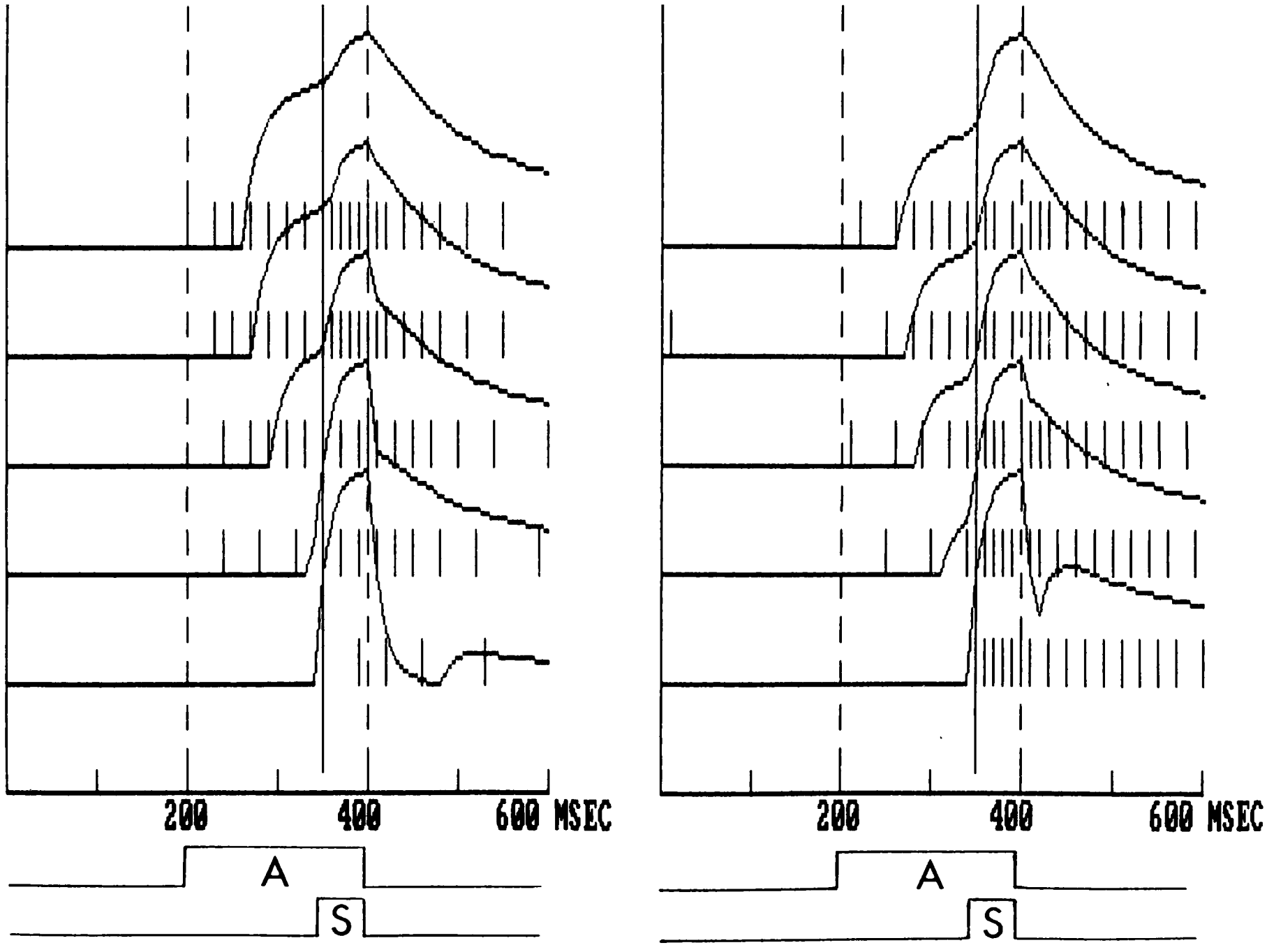
7. Moore, J.W., Desmond, J.E., Berthier, N.E., Blazis, D.E.J., Sutton, R.S., & Barto, A.G. Connectionistic learning in real time: Sutton-Barto adaptive element and classical condition of the nictitating membrane response. To appear in Proceedings of the Seventh Annual Meeting of the Cognitive Science Society.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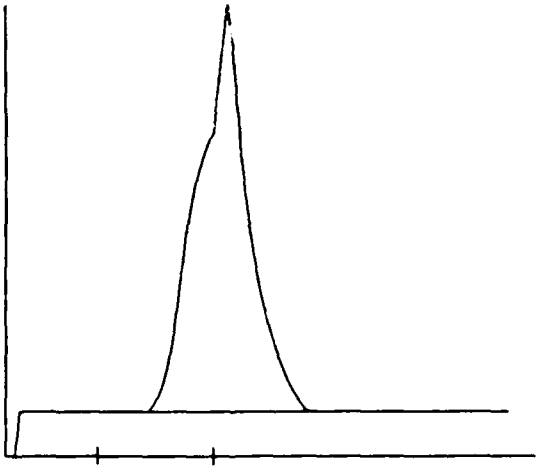
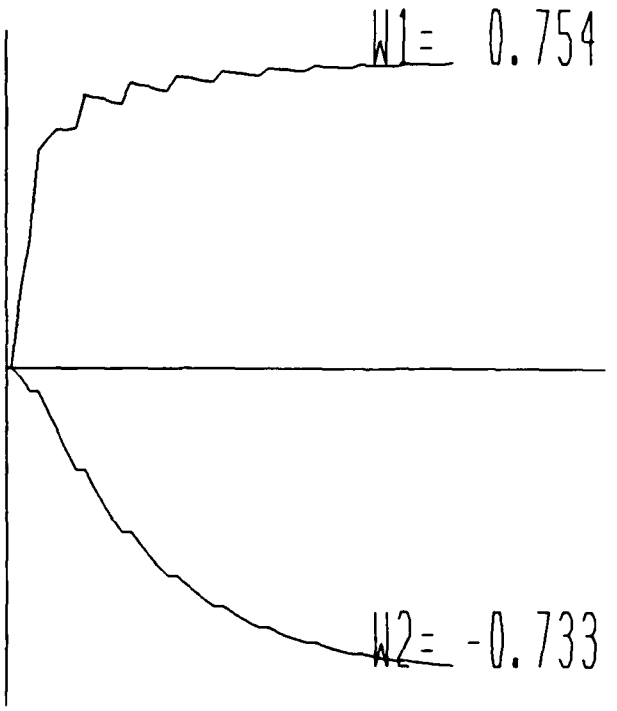
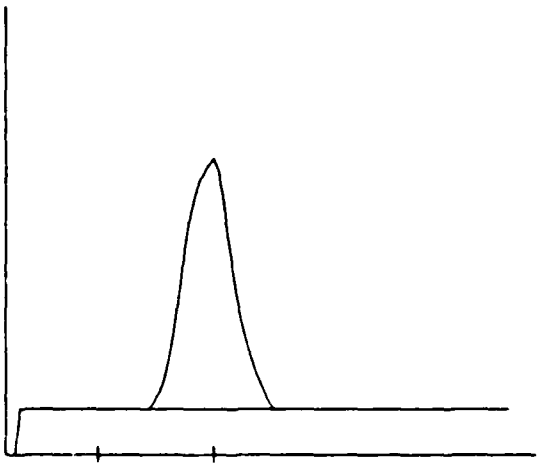
Conditioned Inhibition. Two 250 ms CSs,trial types A+, AB- ; 50 trials



Y VS TIME FOR EACH TRIAL

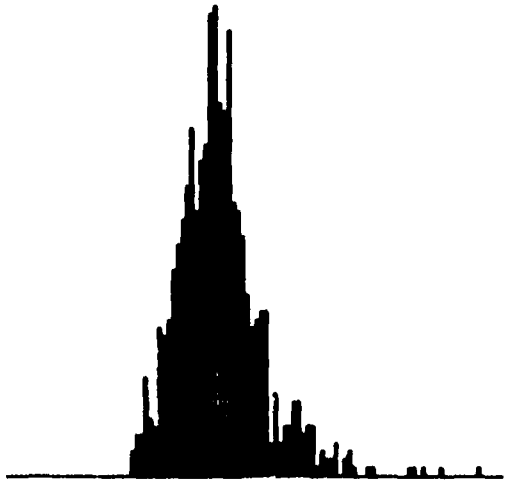


SYNAPTIC WEIGHT BY TRIALS



Y VS TIME FOR STIMULUS

FIGURE 7



A+

AB-

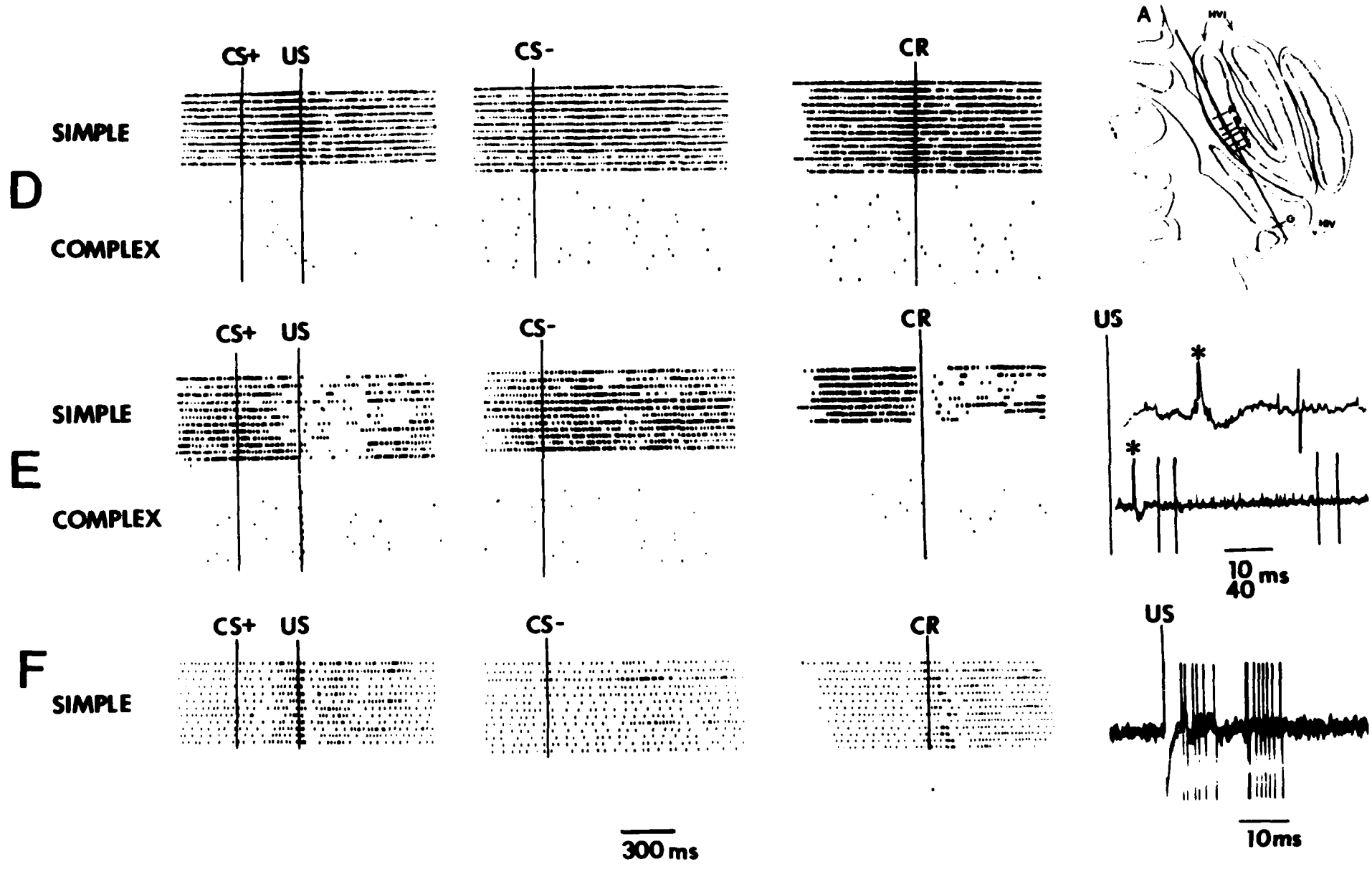

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FIGURE 6

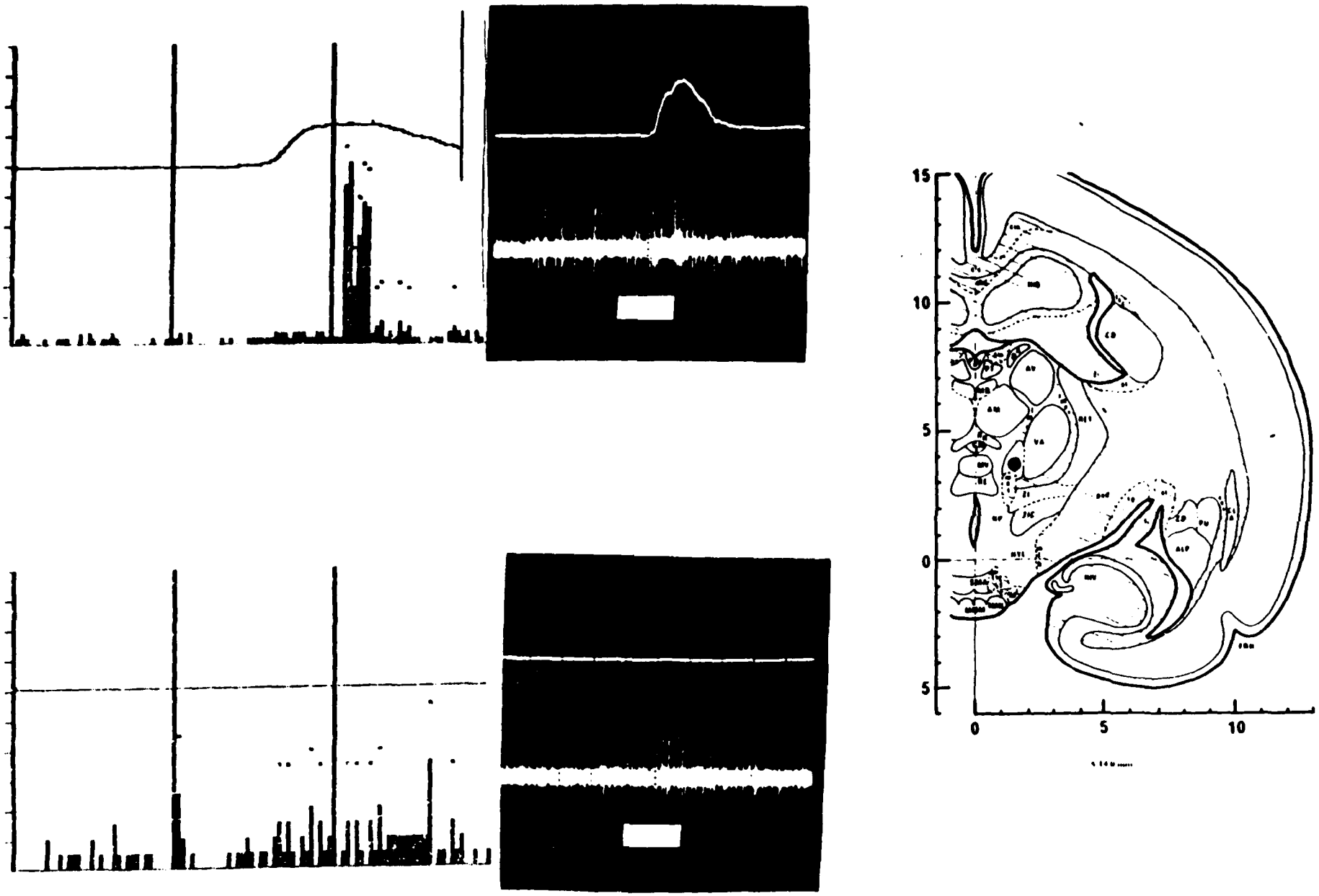


FIGURE 5

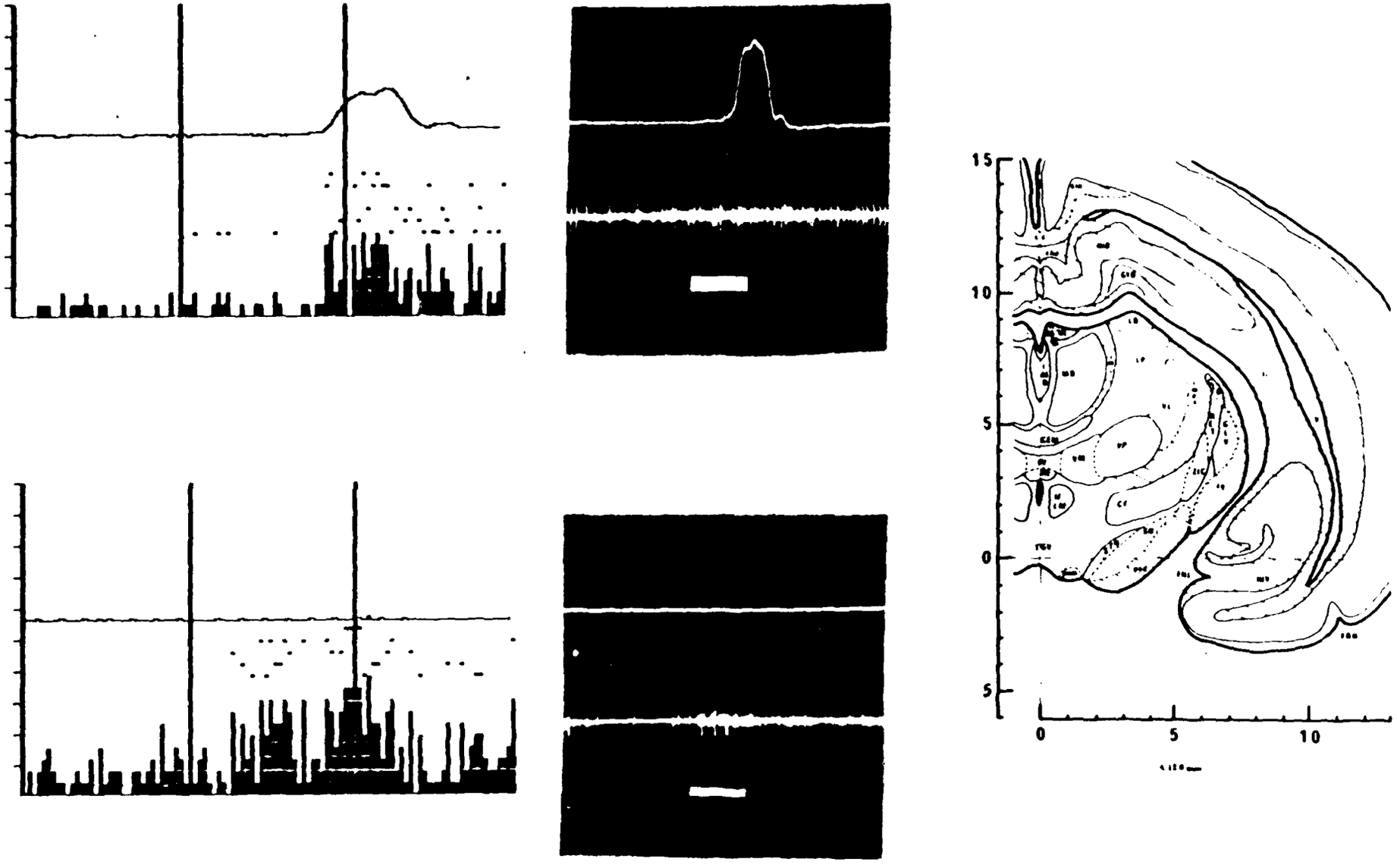
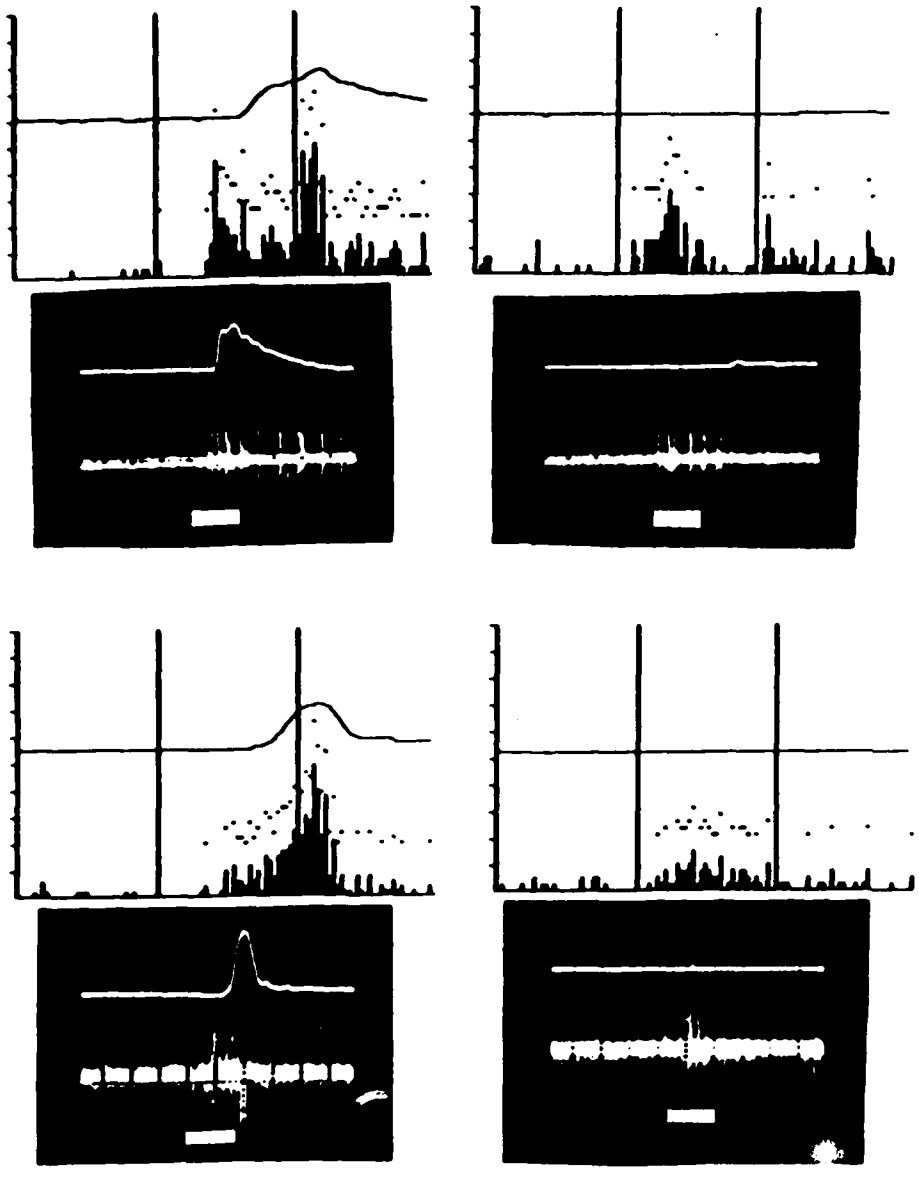


FIGURE 4



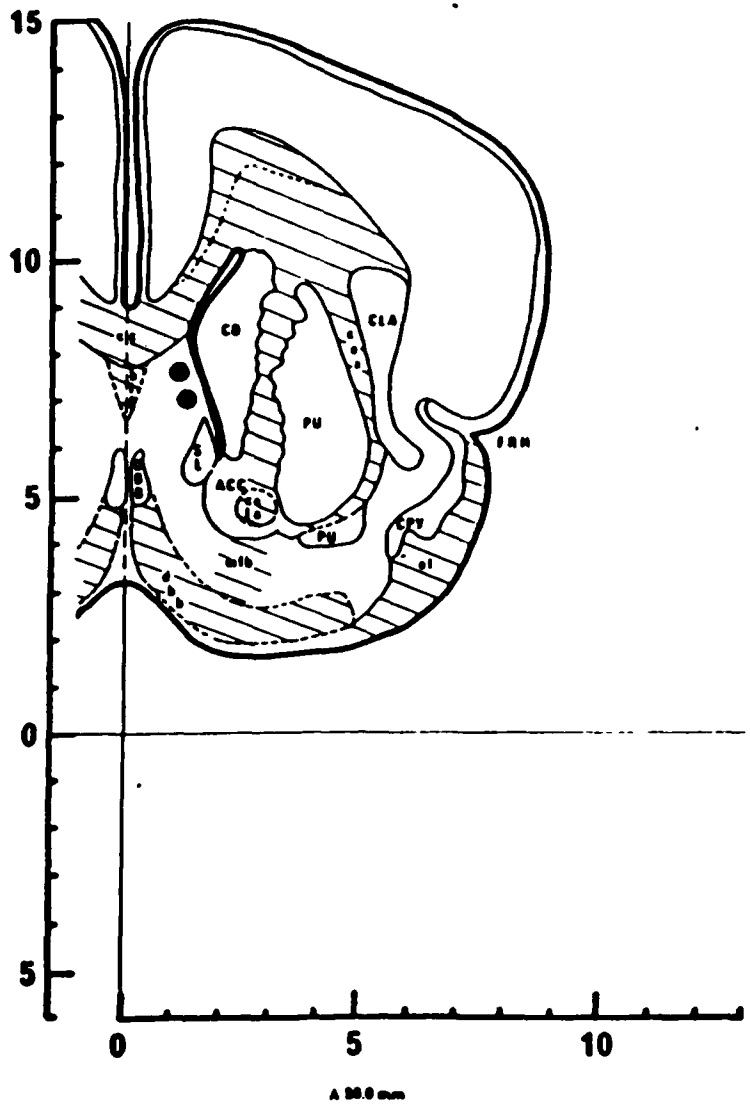


FIGURE 3

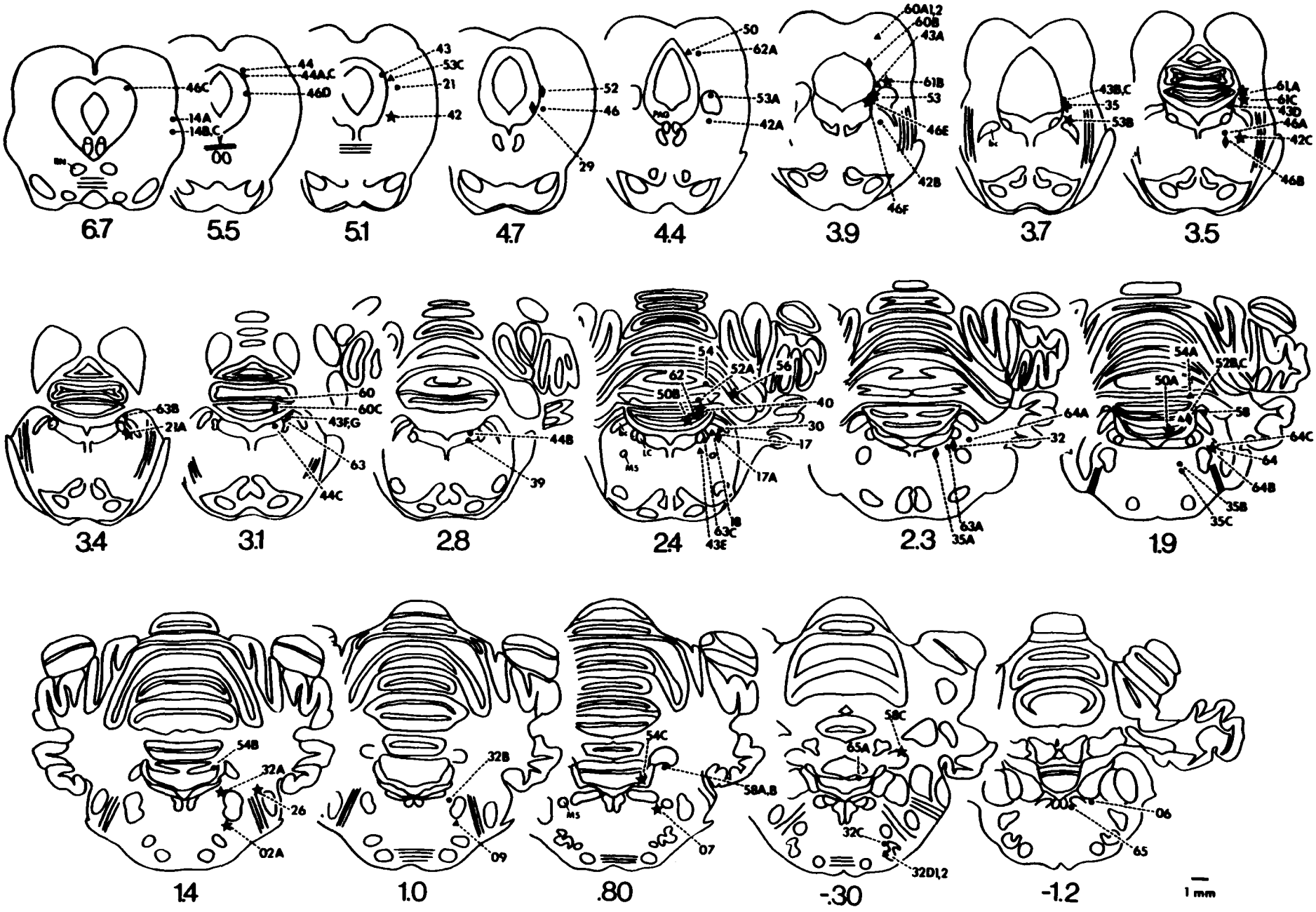
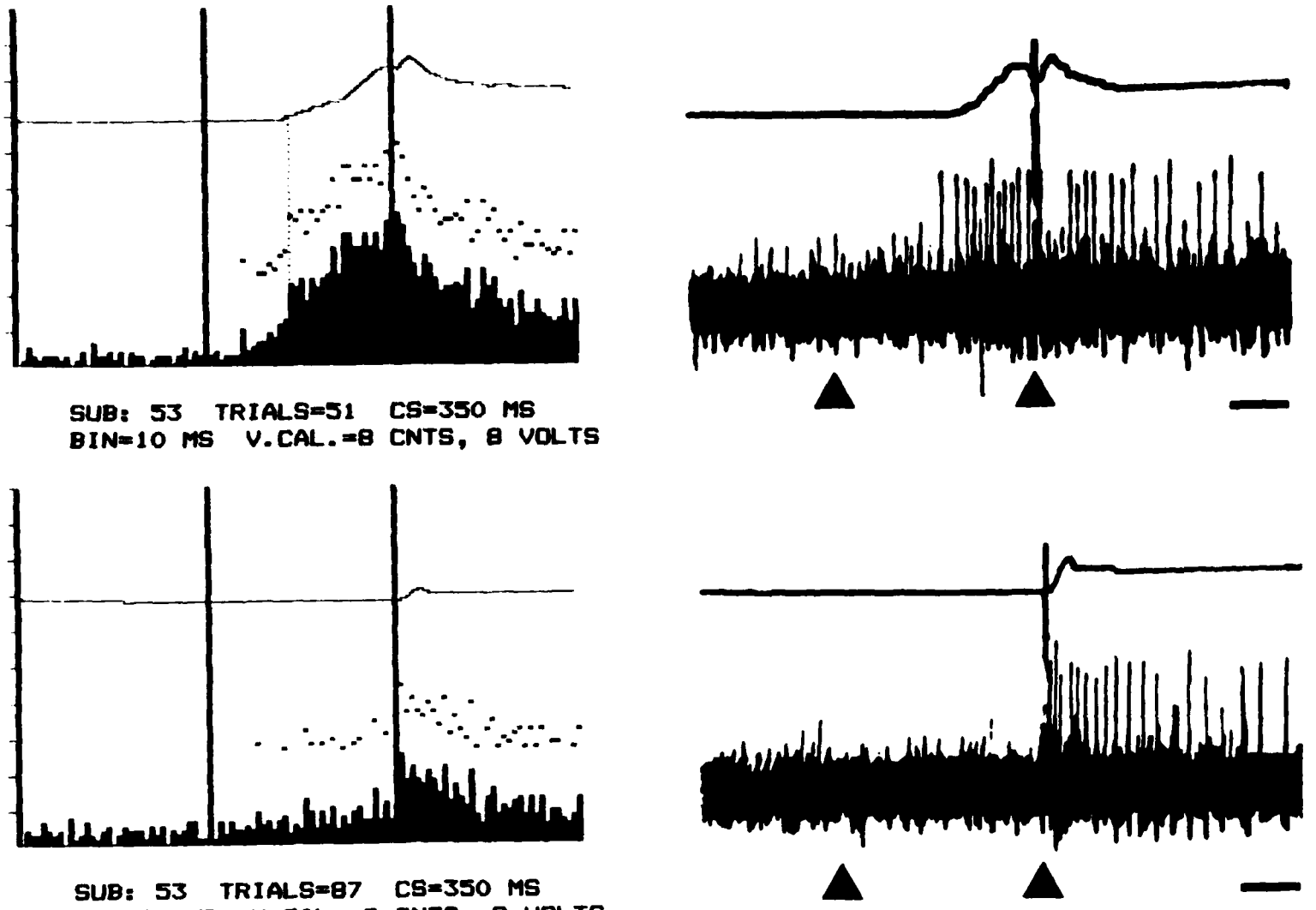


FIGURE 2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BIN=10 MS V.CAL.=8 CNTS, 8 VOLTS
SUB: 53 TRIALS=87 CS=350 MS
BIN=10 MS V.CAL.=8 CNTS, 8 VOLTS

FIGURE 1

time, the US elicited a similar pattern of activity, as illustrated in the far right panel of Row F which shows 3 superimposed sweeps.

Figure 7. Illustrative simulations with current version of the constrained Sutton-Barto (S-B) model. Fifty (50) trials of conditioned inhibition training were simulated. Half the trials consisted of a single CS (A) paired with the US in a forward-delay paradigm and with a CS-US interval of 250 msec. The remaining trials consisted of CS A compounded with another CS (B). These AB- trials were not reinforced, and they alternated with A+ trials.

The upper-left graph shows the simulated response of the S-B adaptive element on single trials at the end of training. The vertical bars on the horizontal axis mark CS and US onsets. The curve rising to a peak just after the US refers to an A+ trial. The horizontal line above the axis refers to AB-trials. By the end of training, the positive synaptic weight of A is cancelled by the negative contribution of B. The upper-right figure shows the acquisition of positive weight by A and negative weight by B over training. The middle-left figure shows simulated trials for A and B presented separately and without the US. The lower figures show simulated peristimulus-time histograms of neuronal firing based on the model's output. These histograms were acummulated over all A+ (left) and AB- (right) training trials.

Figure 8. Simulated CR-UR complexes with superimposed neuronal spikes generated by Moore-Stickney (left) Schmajuk-Pearce-Hall (right) attentional models for simple forward-delay paradigms (lower row). Acquisition of CRs proceeds from bottom to top.

the recording electrode tip.

Figure 6. Activity of Purkinje cells (PCs) from hemispheric lobule VI of cerebellar cortex. Panel A (upper right) shows a reconstruction of an electrcde track through HVI and HIV. Individual PCs are labeled A-G. Cells D-F showed CR-related activity. Cells D and E showed both simple and complex spikes; Cell F showed only simple spikes. The first column are rasters showing each the cell's activity on CS+ trials a two-tone differential conditioning produce was used; the second column shows activity CS-trials. The third column of rasters are trials. The third column of rasters are trials on which a CR occurred The vertical line through each column of rasters indicates the onset of stimuli (1-2) or the CR (3).

The row labeled D shows that Cell D increased its rate of simple spike activity in anticipation of the CR.

Row E shows that Cell E reliably decreased its firing of simple spikes in anticipation of the CR. Furthermore, the US tended to trigger complex spikes and a subsequent deactivation of simple spike activity. Cell E behaved in a manner anticipated in Albus' classic theoretical paper on cerebellar learning. (Albus, J.A. A theory of cerebellar function. Mathematical Bioscience, 1971,10, 25-61). The far right panel of Row E shows Cell E's response to the US more clearly: two different trials are shown at sweep speeds of 10 and 40 msec/cm; asterisks (*) mark complex spikes.

Row F shows that Cells F's CR-related simple-spike activity resembled that elicited by the US. Early in the 350 msec CS-US interval, spiking increased; it then decreased midway through th interval and increased once more before a CR. Although compressed in

electrode tips for animals SM1 (upper circle) and SM3 (lower circle)

Figure 4. Neuronal recordings and MNR tracings from rabbit midbrain near the habenular complex. The CS-US interval was 350 msec, and the US was a 50 msec train of dc pulses to the eye. The top tracings of Column 1 represent the average movement of the NM and the PSTH for L+ trials. The bottom tracings show the NMR and PSTH for LN-trials. Column 2 shows NMR and neuronal activity from a single L+ trial (top)and LN- trial (bottom). On LN- trials a large increase in neuronal activity was observed that continued after the offset of the CS. A much smaller increase in neuronal activity correlated with the CR was observed on L+ trials as well as a large response to the US. At the right side of the figure is a representative section through the midbrain showing the location of the recording electrode tip.

Figure 5. Neuronal recordings and NMR tracings from rabbit midbrain immediately dorsal to the mammillotahalamic tract in the nucleus ventralis posterior thalami. The CS-US interval was 350 msec, and the US was a 50 msec train of dc pulses to the eye. The top tracings of Column 1 represent the averaged movement of the NM and the PSTH for L+ trials. The bottom tracings show the NMR and PSTH for LN-trials. Column 2 shows NMR and neuronal activity from a single L+ trial (top) and LN- trial (bottom). On LN- trials after an initial increase at CS onset a larger increase in neuronal activity was observed that continued after the offset of the CS. A large increase in neuronal activity in response to the US was observed on L+ trials with little change during the CS. At the right side of the figure is a representative section through the midbrain showing the location of

FIGURE CAPTIONS

Figure 1. CR and non-CR PSTHs and unit activity for Cell 53. Top left: CR PSTH. Dotted line indicates CR onset at 1% maximum deflection. Bottom left: Non-CR PSTH. Top right: Representative unit activity and CR on a single CS+ (1200Hz) trial. In top and bottom right panels, triangles below unit activity depict CS onset andoffset. US onset (top right only) is contiguous with CS offset. Calibration: 100 ms. Bottom right: A single CS+ trial with no CR. A UR is observed along with post-US spike activity.

Figure 2. Reconstruction of recording electrode positions. Selected transverse sections are depicted, and numbers at the base of each section represent the distance (mm) rostral to the midpoint of the accessory abducens nucleus (the 0.0 mm section, not depicted). Other numbers refer to individual cells. Three types of neurons with CR related activity are shown: Stars indicate excitatory cells; diamonds are inhibitory cells; triangles are temporally correlated cells. Circles indicate cells withg no significant correlations.

Figure 3. Neuronal recordings and NMR tracings for animals SM1 (upper tracings) and SM3 (lower tracings). The CS-US interval was 350 msec. and the US was a 50 msec train of dc pulses to the eye. Column 1 shows L+ trials and Column 2 shows LN-trials. An increase in neuronal activity that leads the CR can be seen on L+ trials. An increase in neuronal activity also occurs on LN-trials but this activity occurs earlier in the CS than that seen on L+ trials. At the right side of the figure is a representative section through the septum showing the

1984, and I. Gormezano, of the University of Iowa, regarding behavioral data and simulation experiments with the S-B model. In addition, I remain in close contact with Dr. C. H. Yeo and his London collaborators concerning neural substrates of the NM CR. Dr. Yeo visited this laboratory for four days during the spring.

Our interactions with Dr. A. G. Barto and with group, including Richard Sutton, now at GTE in Waltham, Mass., continue as before. During the spring semester, John Desmond, Neil Berthier, and I attended Barto's course on connectionistic learning in AI. The course considered gradient descent algorithms, e.g., the Widrow-Hoff rule and Perceptrons, learning automata theory, Boltzmann machines, matrix memory models, and recent work from Barto's group.

VII New Discoveries

The primary new discovery during the reporting period concern the complex nature of CR related single unit activity in brain stem and cerebellar cortex. These findings will be reported at forthcoming scientific meetings to be held this fall in Cambridge (UK), Boston, and Dallas.

VIII Additional Information

Two highly qualified individuals with training in intracellular neurophysiology plan to join our group as post-doctoral associates. One is William Richards who is completing a Ph. D. under Joseph Farley at Princeton; the other is Clive Prince, who is completing a Ph. D. in Physiology under Lynn Bindman at University College London.

V Professional Personnel

1. John W.Moore, Ph.D., Professor of Psychology (Neuroscience Behavior) and Associated Professor of Computer and Information Sciences.

2. Neil E.Berthier, Ph.D., Research Associate.

VI Interactions

A. Formal Presentations by Moore

1. Slide presentation, Society for Neuroscience, Anaheim, California, 10/84.

2. Invited lecture to Cognitive Neuroscience Group, Cornell University Medical Center, New York, New York, 11/28/84.

3. Discussant, Session on Model Systems, Winter Conference of Neurobiology of Learning and Memory, Park City, Utah, 1/85.

4. Principal participant, Session on Time, Winter Conference of Animal Learning and Behavior, Winter Park, Colorado, 1/85.

B. Formal collaboration Douglas Coulter, working in Daniel Alkon's laboratory at Marine Biological Laboratory, Woods Hole, Mass., and John Disterhoft of Northwestern University are investigating biophysical properties of hippocampal pyramidal cells in slices from rabbits that had NM CR training. In 2/85, I provided the apparatus they are now using for the training phases of these experiments. The collaboration has resulted thus far in a coauthored paper to be presented at the 1985 meetings of the Society for Neuroscience.

C. Informal collaborations These include contacts and sharing with Drs. E. James Kehoe, who was on campus for several weeks in the fall of

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