

## **UC Merced**

### **Proceedings of the Annual Meeting of the Cognitive Science Society**

#### **Title**

A Real-Time Attentional-Associative Network For Classical Conditioning of the Rabbit's NMR

#### **Permalink**

<https://escholarship.org/uc/item/7n04c75c>

#### **Journal**

Proceedings of the Annual Meeting of the Cognitive Science Society, 8(0)

#### **Authors**

Schmajuk, Nestor A.

Moore, John W.

#### **Publication Date**

1986

Peer reviewed

**A REAL-TIME ATTENTIONAL-ASSOCIATIVE NETWORK  
FOR CLASSICAL CONDITIONING OF THE RABBIT'S NMR**

Nestor A. Schmajuk  
Center for Adaptive Systems  
Department of Mathematics  
Boston University  
and  
John W. Moore  
Department of Psychology  
University of Massachusetts

**INTRODUCTION**

Schmajuk and Moore (1986) described two real-time attentional models of classical conditioning. In their present form, both models are capable of real-time descriptions of many classical conditioning paradigms. However, the models do not encompass higher-order conditioning paradigms or sensory preconditioning. They also lack performance rules that permit realistic descriptions of conditioned responding in real time. These considerations prompted the development of a new class of models that incorporate higher-order conditioning, sensory preconditioning, and performance rules.

Because of the large amount of data on classical conditioning of the rabbit's nictitating membrane (NM), this preparation is particularly attractive for a formal treatment. Therefore, the present paper contrasts experimental results regarding classical conditioning using the NM preparation, with computer simulations.

## SECOND-ORDER ATTENTIONAL-ASSOCIATIVE NETWORKS

This section describes a class of attentional-associative network that can be applied to CS-CS as well as CS-US paradigms.

Consider the case of one CS,  $CS_i$ , that predicts event  $k$ . Net associative value,  $\dot{V}_i^k$ , represents the first-order prediction of event  $k$  by  $CS_i$ . Consider now the case of two CSs,  $CS_i$  and  $CS_r$ , that predict event  $k$ . It is assumed that  $CS_i$  predicts  $k$  directly by  $\dot{V}_i^k$  and indirectly by predicting  $CS_r$ , by  $\dot{V}_i^r$ . In turn  $CS_r$  predicts  $k$  by  $\dot{V}_r^k$ . The second order prediction of  $k$  by  $CS_i$ , is expressed as the product  $\dot{V}_i^r \dot{V}_r^k$ .

$B_i^k$ , the first- and second-order prediction of event  $k$  by  $CS_i$ , is

$$B_i^k = ( \dot{V}_i^k + \sum_r w_i^r \dot{V}_i^r \dot{V}_r^k ) \tau_i . \quad [ 1 ]$$

$\dot{V}_i^k$  is the net associative value of  $CS_i$  with event  $k$ . The sum over the index  $r$  involves all CSs with index  $r \neq k$ .  $\dot{V}_i^r$  is the net associative value of  $CS_i$  with all CSs with index  $r \neq k$ .  $\dot{V}_i^r$  is the net associative value of all CS with event  $k$ .  $\tau_i$  is the trace of  $CS_i$ . The mathematical expression for  $\tau_i$  is given below. Coefficient  $w_i^r$  serves to adjust the relative weights of first- and second- order predictions in paradigms such as conditioned inhibition. In order to avoid redundant  $CS_i$ -US and  $CS_i$ - $CS_i$ -US associations,  $w_i^r = 0$  when  $i = r$ , and  $w_i^r > 0$  when  $i \neq r$ .  $B^k$ , the aggregate prediction of event  $k$  made upon all CSs (including the context) with  $\tau > 0$  at a given moment, is

$$B^k = \sum_i B_i^k . \quad [ 2 ]$$

As described below, variable  $B^k$  participates in the computation of  $\dot{V}_i^k$ . In addition, through adequate performance rules,  $B^k$  determines the topography of the NM response.

### THE M-S-S NETWORK

This section describes an attentional-associative network that incorporates variable  $B^k$ . Net associative values,  $\dot{V}_i^k$ , are computed with the rules proposed by Moore and Stickney (1980, 1982, 1985; see also Schmajuk and Moore, 1986).

#### Changes in associative values.

When the  $CS_i$  is accompanied or followed by event  $k$ , the associative value between  $CS_i$  and event  $k$ ,  $V_i^k$ , increases by

$$\Delta V_i^k = \theta \alpha_i \tau_i (1 - V_i^k), \quad [ 3 ]$$

and the antiassociative value,  $N_i^k$ , decreases by

$$\Delta N_i^k = \theta' \alpha_i \tau_i (0 - N_i^k), \quad [ 3' ]$$

When event  $k$  does not occur,  $V_i^k$  decreases by

$$\Delta V_i^k = \theta' \alpha_i \tau_i (0 - V_i^k) B^k, \quad [ 4 ]$$

and  $N_i^k$  increases by

$$\Delta N_i^k = \theta' \alpha_i \tau_i (1 - N_i^k) B^k, \quad [ 4' ]$$

where  $\alpha_i$  is  $CS_i$ 's associability,  $\theta$  ( $0 < \theta \leq 1$ ) is the rate of change in  $V_i^k$ ,  $\theta'$  ( $0 < \theta' < \theta$ ) is the rate of change in  $N_i^k$ ,  $\tau_i$  is the trace of  $CS_i$ , and  $B^k$  is defined by Equation

2.

The net associative value of  $CS_i$  and event  $k$  is

$$\dot{V}_{i^k} = V_{i^k} - N_{i^k}. \quad [ 5 ]$$

### Changes in associability

The associability of  $CS_i$ ,  $\alpha_i$ , may increase, decrease, or remain unchanged depending on the associative value of  $CS_i$  with event  $k$  and the associative value of another CS,  $CS_j$ , with the same event  $k$ .

When  $CS_i$ ,  $CS_j$ , and event  $k$  are presented together, and provided that  $\dot{V}_{i^k} > \dot{V}_{j^k}$

$$\Delta \alpha_{i^k} = c ( 1 - \alpha_i ) ( \dot{V}_{i^k} - \dot{V}_{j^k} ), \quad [ 6 ]$$

where  $\dot{V}_{j^k}$  is the second highest net associative value with respect to event  $k$  of all the CSs present with the  $CS_i$ , including the context.

When  $\dot{V}_{i^k} \geq \dot{V}_{j^k}$

$$\Delta \alpha_{i^k} = c ( 0 - \alpha_i ) ( \dot{V}_{j^k} - \dot{V}_{i^k} ), \quad [ 7 ]$$

where  $\dot{V}_{j^k}$  is the highest net associative value with respect to  $k$  of all the CSs present with the  $CS_i$ . Parameter  $c$  in Equations 6 and 7 is a constant set  $0 < c < 1$ .

When all the components of  $\Delta \alpha_{i^k}$  related to a given  $CS_i$  or the US have been computed, they are combined in the expression

$$\Delta \alpha_i = \sum_k \Phi_k \alpha_{i^k} / \sum_h \Phi_h. \quad [ 8 ]$$

The sum over the index  $k$  in the numerator involves all the

events present with the  $CS_i$ . The sum over the index  $h$  in the denominator involves all the events the subject has encountered in previous experiences in the same context, even though they may not be present at the time  $\Delta \alpha_i$  is computed. The weighting factors,  $\Phi_k$ , are selected such that  $\Phi_{us} > \Phi_{cs} > \Phi_x$ , because the US is presumed to be biologically more significant than the CSs and the context (X).

### NM RESPONSE CONDITIONING

During acquisition of the NM conditioned response (CR), percentage of CRs generated in each session increases, CR latency decreases, and CR amplitude increases. (see Gormezano, Kehoe, & Marshall, 1983)

#### The trace hypothesis

Conditioning is typically more efficacious when the CS precedes the US than when the two are presented together. Hull (1943) proposed that stimuli give rise to traces in the central nervous system that somehow impinge simultaneously on critical loci of learning, despite the non-simultaneous arrangement as observed in the periphery.

It is assumed that a  $CS_i$  generates a trace,  $\tau_i$ , that increases over time to a maximum, stays at this level for a period of time independent of the CS duration, and then gradually decays back to zero.

Formally, trace  $\tau$  is defined for  $t \leq 200$  msec by

$$\tau(t) = CS_{max} ( 1 - e^{-(k_1 t)} ), \quad [ 9 ]$$

where  $CS_{max}$  is the maximum intensity of the CS and  $k_1$  is a constant,  $0 < k_1 < 1$ . Parameter  $k_1$  is selected so that the ISI for optimal conditioning is 200 msec.

$\tau(t)$  remains equal to  $CS_{max}$  as long as the CS does not

decay. If the CS = 0 and  $t > 200$  msec,  $\tau(t)$  decays by

$$\tau(t) = CS_{\max} ( \exp -( k_1 t ) ), \quad [ 10 ]$$

If  $CS_i$  is not present 200 msec after its onset, the trace decays to zero by Equation 10.

#### Performance Rules

Performance rules were selected to relate variable  $B^{US}$  to the topography of NM responses.

Time of CR onset is the earliest time  $t$  such that

$$\sum_{t'=t_i} \sum_j B_j^{US}(t') \geq L_1, \quad [ 11 ]$$

where  $t_i$  denotes the time step at which  $CS_i$  onset occurs. The sum over the index  $j$  involves  $B_j^{US}$  of all CSs with  $\tau_j > 0$ , excluding the context. Sum over index  $t$  involves all time steps for which  $\tau_j > 0$ , starting at the time step when the amplitude of the NM response as defined by Equation 11 equals zero.  $L_1$  is a threshold greater than zero. Equation 11 implies that as  $B_j^{US}$  increases over trials, CR onset moves progressively to an asymptote determined by  $L_1$ .

During the CS period, for time steps  $t > t_i$ , the amplitude of the NM response,  $NMR(t)$ , is changed by

$$\Delta NMR(t) = k_2 ( B^{US}(t) - NMR(t) ), \quad [ 12 ]$$

where  $k_2$  is a constant ( $0 < k_2 < 1$ ).

During the US period, while  $B^{US}(t) < \lambda^{US}(t)$ , is given by Equation 12. However, when  $B^{US}(t) > \lambda^{US}(t)$ ,  $NMR(t)$  increases by

$$\Delta NMR(t) = k_2 ( \lambda^{US}(t) - NMR(t) ), \quad [ 13 ]$$

## SCHMAJUK AND MOORE

When  $B^{US}(t)$  and  $\lambda^{US}(t)$  equal zero,  $NMR(t)$  decays to baseline by

$$\Delta NMR(t) = -k_2 NMR(t). \quad [ 14 ]$$

### COMPUTER SIMULATIONS

In the simulations, continuous time was converted to discrete time steps or bins of 10 msec in duration. Each trial consisted of 60 bins. Otherwise specified, the simulations assumed 200 msec CSs, the last 50 msec of which overlaps the US.

Parameters values for variations of associative values were:  $\theta = 0.1$ , and  $\theta' = 0.001$ . For antiassociative value were:  $\theta = 0.005$  and  $\theta' = 0.1$ , with exception of the inhibitory conditioning cases for which  $\theta = 0.05$ . For variations in associability:  $\Phi_{US} = 1$ ,  $\Phi_A = 0.16$ ,  $\Phi_B = 0.16$ ,  $\Phi_X = 0.01$ , and  $c = 0.6$ . Initial values of  $V_s$  and  $N_s$  were zero for all  $i$ 's. Initial values of associability were always selected  $\alpha_X = 0.1$  and  $\alpha_A = \alpha_B = 0.5$ . For computations of  $B_i^k$ :  $w_i^k = 0.4$  when  $i \neq r$ ; and  $w_i^k = 0$  when  $i = r$ . For computations of the NM CR:  $L_1 = 2$ . For computation of the trace:  $k_1 = 0.1$ , and for the NM response topography:  $k_2 = 0.5$ .

### Simulation results.

Acquisition. Figure 1 shows simulations of a delay conditioning paradigm. As CR acquisition proceeds, CR onset latency decreases, and CR amplitude increases. Maximal response amplitude (CR peak) is located at the time of the US occurrence. Context associability decreases and CS associability increases over trials. Simulation results agree with data on delay conditioning (see Gormezano, et al.,



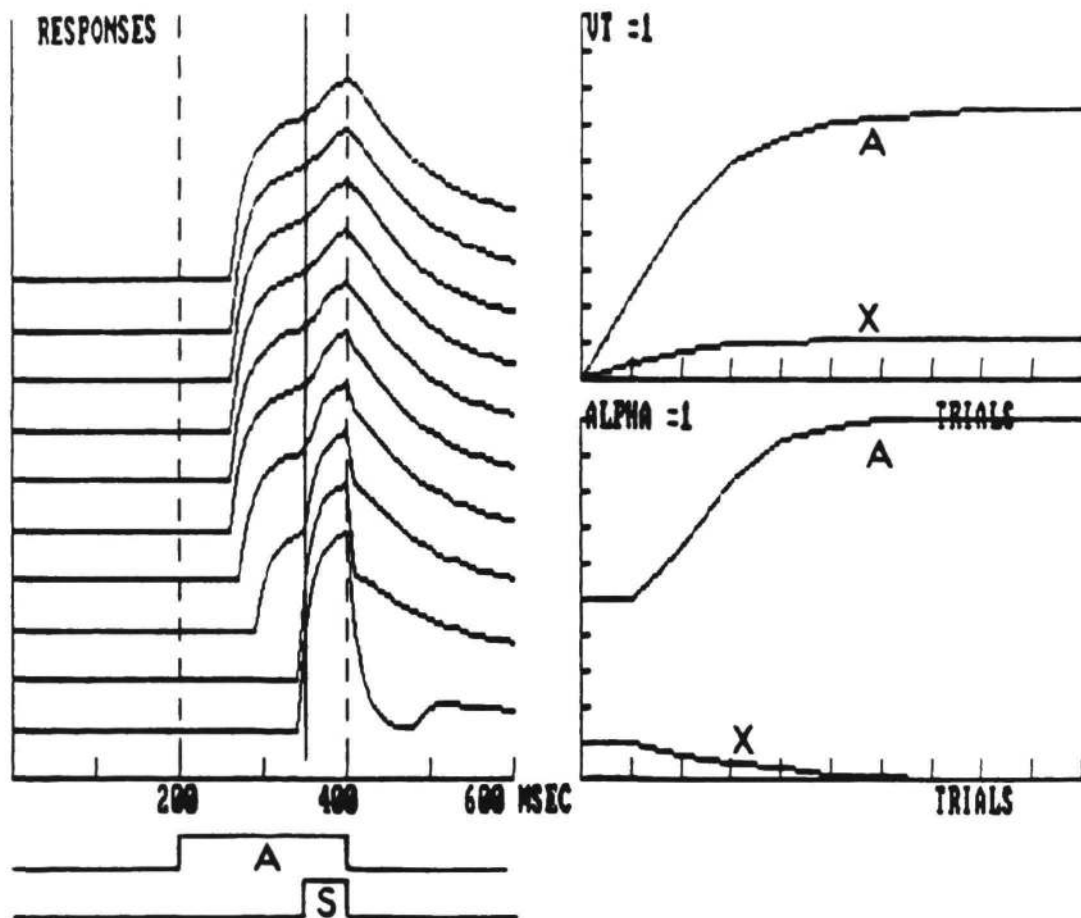


Figure 1. Delay conditioning. A : CS(A). X : Context.  
 Left Panels: NM response topography in 10 reinforced trials.  
 Upper-Right Panels: Net associative values (VT) at the end  
 of each trial, as a function of trials. Lower-Right Panels:  
 Associability (ALPHA) at 350 msec, as a function of trials.

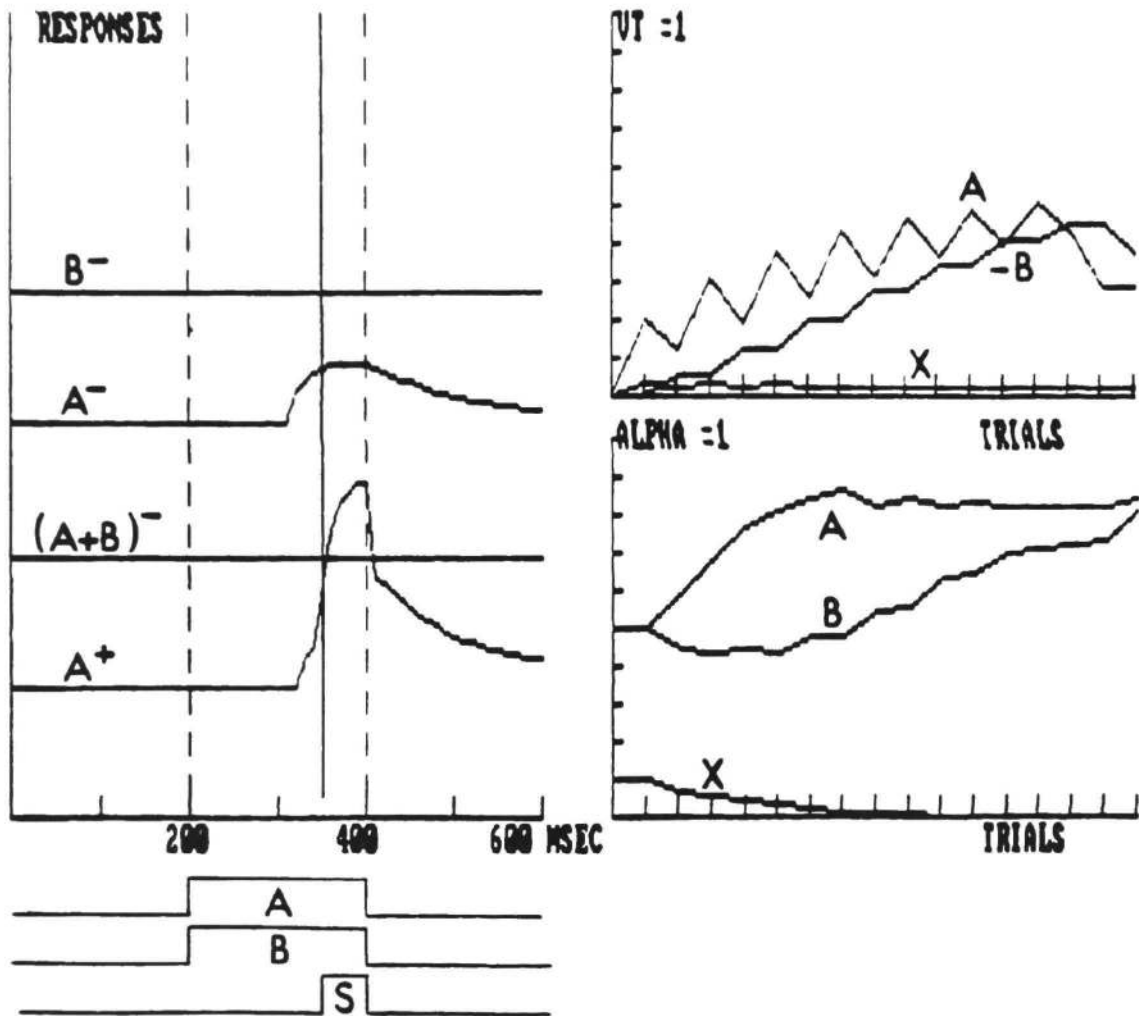


Figure 2. Conditioned Inhibition. A : CS(A). B : CS(B). X : Context. Left Panels: NM response topography in A<sup>+</sup>, (A+B)<sup>-</sup>, A<sup>-</sup>, and B<sup>-</sup> trials, after 10 alternated A<sup>+</sup> and (A+B)<sup>-</sup> trials. Upper-Right Panels: Net associative values (VT) at the end of each trial, as a function of trials. Lower-Right Panels: Associability (ALPHA) at 350 msec, as a function of trials.

1983)

Conditioned Inhibition. Figure 2 shows simulations of a conditioned inhibition paradigm. During conditioned inhibition two types of trials were alternated: reinforced trials consisted of a single reinforced CS (A), and nonreinforced trials consisted of a compound CS (A and B). Stimulus B was the conditioned inhibitor. After 10 simulated trials, the CR elicited by A and B together was smaller than that elicited when A was presented alone because B has acquired inhibitory associative value. Associabilities of both CSs increased and context associability decreased over trials. Simulation results agree with data on conditioned inhibition reported by Marchant, Miss, and Moore (1972).

Blocking. Figure 3 shows simulations of a blocking paradigm. Experimentals received 5 trials with one CS (blocker) paired with the US followed by 5 trials with the same CS and a second (blocked CS) paired with the US. Controls received 5 two-CS trials in which both CSs were presented together and paired with the US. Controls were subject to mutual overshadowing between the two component CSs. The network showed simulated blocking because CR for the designated blocked CS was smaller than CR for the blocker CS, both after 5 training trials. The results agree with blocking data in the rabbit NM response preparation as reported by Marchant and Moore (1973).

Sensory preconditioning. Figure 4 shows simulations of a sensory preconditioning paradigm. In the first phase, 5 nonreinforced trials with a compound CS(A and B). During the second phase, one of the nonreinforced CSs (A) was reinforced for 5 trials. A test trial assessed the CR to CS(B) never paired with the US. Simulations showed that context associability decreases during preconditioning. In the

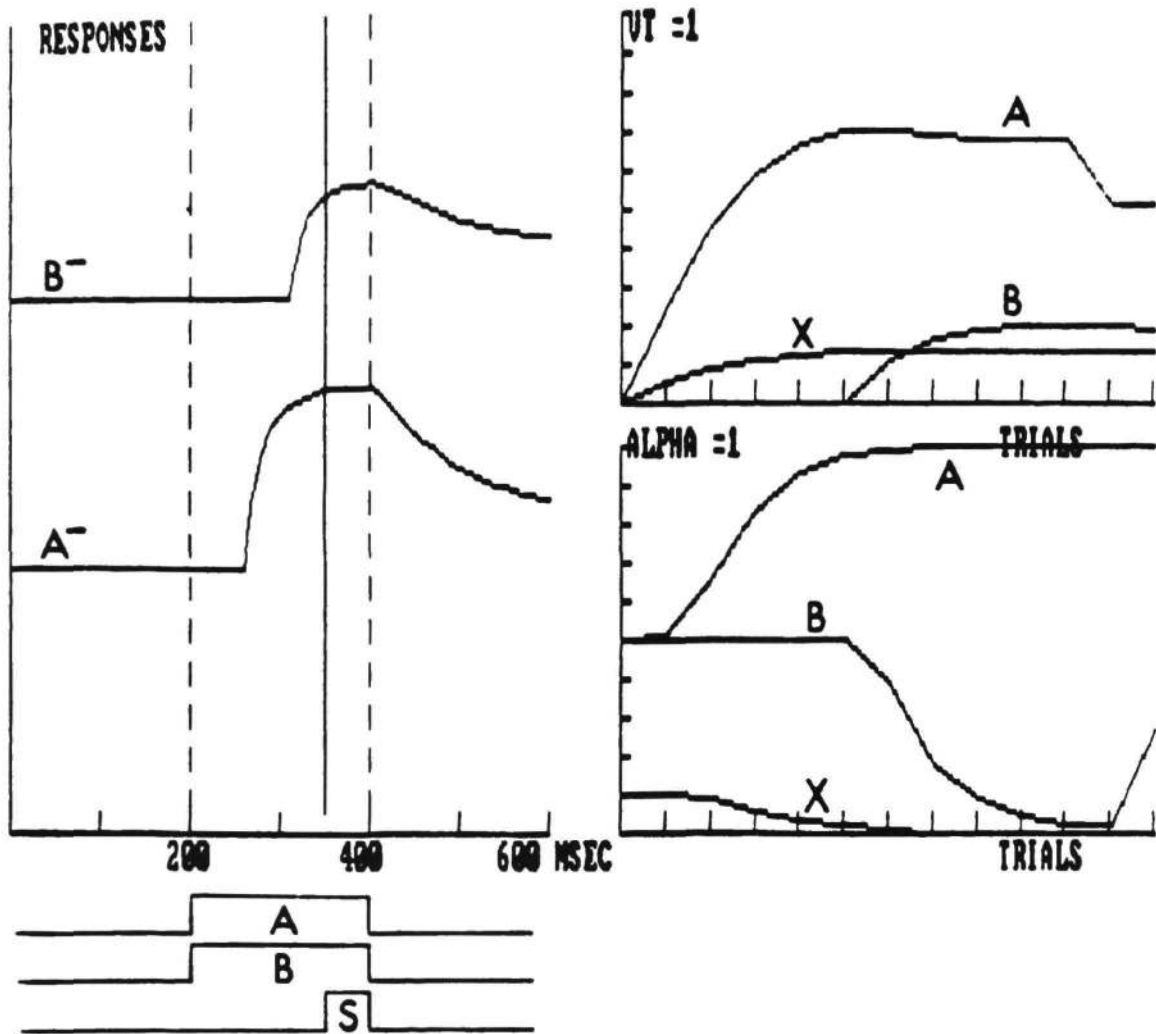


Figure 3. Blocking. A : CS(A). B : CS(B). X : Context. Left Panels: NM response topography in A- and B- test trials, after 5 CS(A) reinforced trials and 5 CS(A) and CS(B) reinforced trials. Upper-Right Panels: Net associative values (VT) at the end of each trial, as a function of trials. Lower-Right Panels: Associability (ALPHA) at 350 msec, as a function of trials.

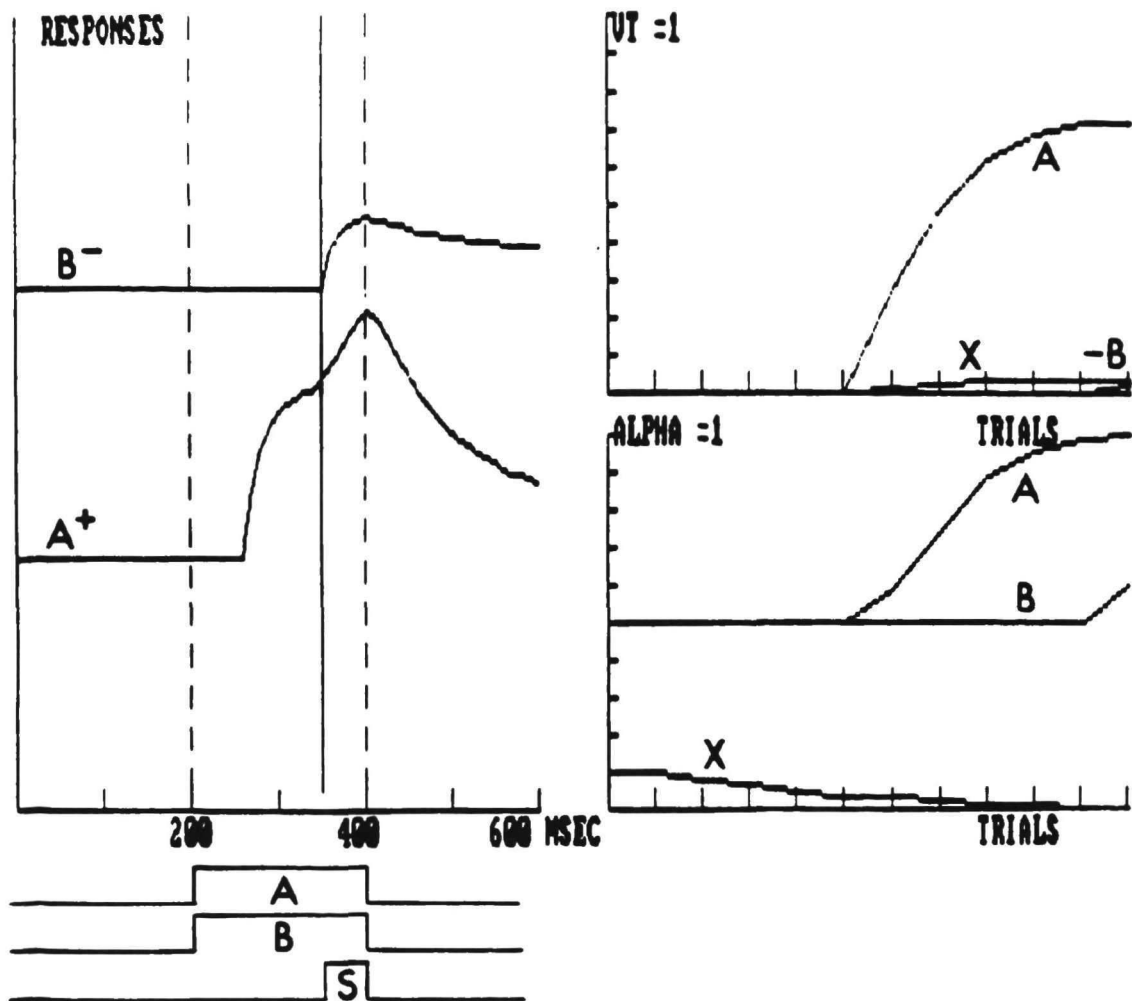


Figure 4. Sensory preconditioning. A : CS(A). B : CS(B). X : Context. Left Panels: NM response topography in A+ and B-trials, after 5 CS(A) and CS(B) nonreinforced trials and 5 CS(A) reinforced trials. Upper-Right Panels: Net associative values (VT) at the end of each trial, as a function of trials. Lower-Right Panels: Associability (ALPHA) at 350 msec, as a function of trials.

nonreinforced test trial CS(B) acquired inhibitory associative value because it was presented in a context with excitatory associative value. CS(B) generated a CR. Simulation results are in agreement with data reported by Port and Patterson (1984).

#### DISCUSSION

The present paper illustrates how the M-S-S network, a second-order attentional-associative architecture, describes several classical conditioning paradigms in real-time. The network incorporates performance rules that convert net associative values into strength and timing of the rabbit's NM response.

#### REFERENCES

- Gormezano, I., Kehoe, E.J., & Marshall, B.S. (1983). Twenty years of classical conditioning research with the rabbit. *Progress in Psychobiology and Physiological Psychology*, 10, 197- 275.
- Hull, C.L. (1943). *Principles of Behavior*. New York: Appleton-Century-Crofts.
- Marchant, H.G., Miss, F.W., & Moore, J.W. (1972). Conditioned inhibition of the rabbit's nictitating membrane response. *Journal of Experimental Psychology*, 95, 408-411.
- Marchant, H.G., & Moore, J.W. (1973). Blocking of the rabbit's conditioned nictitating membrane response in Kamin's two-stage paradigm. *Journal of Experimental Psychology*, 101, 155-158.
- Moore, J.W., & Stickney, K.J. (1980). Formation of attentional-associative networks in real time: Role of the hippocampus and implications for conditioning. *Physiological Psychology*, 8, 207-217,

SCHMAJUK AND MOORE

Moore, J.W., & Stickney, K.J. (1982). Goal tracking in attentional-associative networks: Spatial learning and the hippocampus. **Physiological Psychology**, 10, 202-208.

Moore, J.W., & Stickney, K.J. (1985). Antiassociations: Conditioned inhibition in attentional-associative networks. In R.R. Miller and N.E. Spear (Eds.), **Information processing in animals: Conditioned Inhibition**. Hillsdale: Lawrence Erlbaum.

Port, R.L., & Patterson, M.M. (1984). Fimbrial lesions and sensory preconditioning. **Behavioral Neuroscience**, 98, 584-589.

Schmajuk, N.A., & Moore, J.W. (1986). Real-time attentional models for classical conditioning and the hippocampus. **Physiological Psychology**, 13, 278-290.