MATHEMATICAL MODELS OF CLASSICAL CONDITIONING: A CRITICAL REVIEW AND EXTENSIONS

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  - General 0679
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  - Linguistics 0290
  - Modern 0291
- Literature
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  - Classical 0294
  - Comparative 0295
  - Medieval 0297
  - Modern 0298
  - African 0316
  - American 0591
  - Asian 0305
  - Canadian (English) 0352
  - Canadian (French) 0355
  - English 0593
  - Germanic 0311
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  - Middle Eastern 0315
  - Romance 0313
  - Slavic and East European 0314

PHILOSOPHY, RELIGION AND THEOLOGY

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- Religion
  - General 0318
  - Biblical Studies 0321
  - Clergy 0319
  - History of 0320
  - Philosophy of 0322
- Theology 0469

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- American Studies 0323
- Anthropology
  - Archaeology 0324
  - Cultural 0326
  - Physical 0327
- Business Administration
  - General 0310
  - Accounting 0272
  - Banking 0770
  - Management 0454
  - Marketing 0338
- Canadian Studies 0385
- Economics
  - General 0501
  - Agricultural 0503
  - Commerce Business 0505
  - Finance 0508
  - History 0509
  - Labor 0510
  - Theory 0511
- Folklore 0358
- Geography 0366
- Gerontology 0351
- History
  - General 0578
  - Ancient 0579
  - Medieval 0581
  - Modern 0582
  - Black 0328
  - African 0331
  - Asia, Australia and Oceania 0332
  - Canadian 0334
  - European 0335
  - Latin American 0336
  - Middle Eastern 0333
  - United States 0337
- History of Science 0585
- Law 0398
- Political Science
  - General 0615
  - International Law and Relations 0616
  - Public Administration 0617
- Recreation 0814
- Social Work 0452
- Sociology
  - General 0626
  - Criminology and Penology 0627
  - Demography 0938
  - Ethnic and Racial Studies 0631
  - Individual and Family Studies 0628
  - Industrial and Labor Relations 0629
  - Public and Social Welfare 0630
  - Social Structure and Development 0700
  - Theory and Methods 0344
- Transportation 0709
- Urban and Regional Planning 0999
- Women's Studies 0453

THE SCIENCES AND ENGINEERING

BIOLOGICAL SCIENCES

- Agriculture
  - General 0473
  - Agronomy 0285
  - Animal Culture and Nutrition 0475
  - Animal Pathology 0476
  - Food Science and Technology 0359
  - Forestry and Wildlife 0478
  - Plant Culture 0479
  - Plant Pathology 0480
  - Plant Physiology 0817
  - Range Management 0777
  - Wood Technology 0746
- Biology
  - General 0306
  - Anatomy 0287
  - Biostatistics 0308
  - Botany 0309
  - Cell 0379
  - Ecology 0329
  - Entomology 0353
  - Genetics 0369
  - Limnology 0793
  - Microbiology 0410
  - Molecular 0307
  - Neuroscience 0317
  - Oceanography 0416
  - Physiology 0433
  - Radiation 0821
  - Veterinary Science 0778
  - Zoology 0472
- Biophysics
  - General 0786
  - Medical 0760

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- Biogeochemistry 0425
- Geochemistry 0996
- Geodesy 0370
- Geology 0372
- Geophysics 0373
- Hydrology 0388
- Mineralogy 0411
- Paleobotany 0345
- Paleoecology 0426
- Paleontology 0418
- Paleozoology 0985
- Palynology 0427
- Physical Geography 0368
- Physical Oceanography 0415

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- Environmental Sciences 0768
- Health Sciences
  - General 0566
  - Audiology 0300
  - Chemotherapy 0992
  - Dentistry 0567
  - Education 0350
  - Hospital Management 0769
  - Human Development 0758
  - Immunology 0982
  - Medicine and Surgery 0564
  - Mental Health 0347
  - Nursing 0569
  - Nutrition 0570
  - Obstetrics and Gynecology 0380
  - Occupational Health and Therapy 0354
  - Ophthalmology 0381
  - Pathology 0571
  - Pharmacology 0419
  - Pharmacy 0572
  - Physical Therapy 0382
  - Public Health 0573
  - Radiology 0574
  - Recreation 0575
  - Speech Pathology 0460
  - Toxicology 0383
- Home Economics 0386

PHYSICAL SCIENCES

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- Chemistry
  - General 0485
  - Agricultural 0749
  - Analytical 0486
  - Biochemistry 0487
  - Inorganic 0488
  - Nuclear 0738
  - Organic 0490
  - Pharmaceutical 0491
  - Physical 0494
  - Polymer 0495
  - Radiation 0754
- Mathematics 0405
- Physics
  - General 0605
  - Acoustics 0986
  - Astronomy and Astrophysics 0606
  - Atmospheric Science 0608
  - Atomic 0748
  - Electronics and Electricity 0607
  - Elementary Particles and High Energy 0798
  - Fluid and Plasma 0759
  - Molecular 0609
  - Nuclear 0610
  - Optics 0752
  - Radiation 0756
  - Solid State 0611
- Statistics 0463

Applied Sciences

- Applied Mechanics 0346
- Computer Science 0984
- Engineering
  - General 0537
  - Aerospace 0538
  - Agricultural 0539
  - Automotive 0540
  - Biomedical 0541
  - Chemical 0542
  - Civil 0543
  - Electronics and Electrical 0544
  - Heat and Thermodynamics 0348
  - Hydraulic 0545
  - Industrial 0546
  - Marine 0547
  - Materials Science 0794
  - Mechanical 0548
  - Metallurgy 0743
  - Mining 0551
  - Nuclear 0552
  - Packaging 0549
  - Petroleum 0765
  - Sanitary and Municipal 0554
  - System Science 0790
- Geotechnology 0428
- Operations Research 0796
- Plastics Technology 0795
- Textile Technology 0994

PSYCHOLOGY

- General 0621
- Behavioral 0384
- Clinical 0622
- Developmental 0620
- Experimental 0623
- Industrial 0624
- Personality 0625
- Physiological 0989
- Psychobiology 0349
- Psychometrics 0632
- Social 0451

Shortened version of the thesis title (to put on the side of the thesis):

Mathematical Models of Classical Conditioning

TABLE OF CONTENTS:

ABSTRACT

The history of classical conditioning is summarized The contributions and weaknesses of several earlier models of classical conditioning are studied Two new neuronal models are proposed The first, called the *delay-producing connections* (or DPC) model, is an extension of the Klopf (1988) and Sutton & Barto (1981) models The DPC model makes two contributions (1) it represents the trace of each conditioned stimuli (CS) by differential equations, and (2) it replaces each CS in the activation rule with a trace of the relevant CS A method is suggested to measure the trace of a CS The second model, called the *adaptive delays* (or AD) model, is proposed as an extension of the DPC model to account for the phenomenon of inhibition of delay Both models reproduce the shape of a CR, the curve of efficacy of conditioning as a function of the interstimulus interval (ISI), the dependence of the optimal ISI on CS duration, the extinction of a CR (even for long lasting CSs as opposed to Klopf's (1988) model), and several other properties of classical conditioning

RÉSUMÉ

L'historique des découvertes du conditionnement classique est résumé. Les forces et les faiblesses de divers modèles existants de conditionnement classique sont étudiées. Deux nouveaux modèles neuronaux sont proposés. Le premier, le modèle à *connexions produisant des délais* (ou CPD), est une extension des modèles de Klopf (1988) et de Sutton et Barto (1981). Le modèle CPD apporte deux contributions: (1) il représente la trace d'un stimulus conditionnel (SC) par une équation différentielle, et (2) il remplace les SCs, dans la règle d'activation, par leur trace. Une méthode est proposée pour mesurer la trace d'un SC. Le second modèle, le modèle à *délais adaptatifs* (ou DA), est développé à partir du modèle CPD, afin de rendre compte du phénomène de croissance de la latence de la réponse conditionnelle (RC) dans la technique d'anticipation. Les deux modèles reproduisent la forme de la RC, la courbe de l'efficacité du conditionnement en fonction de l'intervalle entre le SC et le stimulus inconditionnel (SI), la dépendance de l'intervalle SC—SI optimal envers la durée du SC, l'extinction (même pour un long SC, contrairement au modèle de Klopf (1988)) et de nombreuses autres propriétés du conditionnement classique.

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1. INTRODUCTION

Classical conditioning is at the interface of three main fields of psychology physiological psychology, animal learning and the neural network paradigm in cognitive psychology New findings have expanded the scope of classical conditioning processes Traditional views considered classical conditioning to merely involve secretory, reflexive, or emotional processes However, evidence now shows the involvement of classical conditioning in drug tolerance effects, relapses to drug abuse, the placebo effect, the operation of the immune system, vicarious learning and even in rule-governed behavior "Classical conditioning [ ] has been found to occur in simpler and simpler organisms and recently even demonstrated in brain slices and in utero " (Turkkan, 1989) New discoveries in classical conditioning have shed some light on its neurobiological mechanism Classical conditioning has also been studied at the level of the neuron (Byrne, 1987, Carew, Hawkins, and Kandel, 1983, Hawkins, Carew, & Kandel, 1986, Thompson et al , 1984)

1.1 Definition of Classical Conditioning:

A classical conditioning (or Pavlovian conditioning) process is a learning procedure involving four basic components

(1) the *Unconditioned Stimulus* (US),

(2) the *Unconditioned Response* (UR),

(3) the *Conditioned Stimulus* (CS),

and (4) the *Conditioned Response* (CR)

Prior to conditioning, the US innately elicits the UR, but the CS does not elicit the CR. During conditioning, the CS is paired with the US Following conditioning, the CS elicits the CR The strength (or intensity) of the CR increases steadily during acquisition until a maximum or asymptotic level is reached

The classic example of this procedure is the historic case of Pavlov's dog When food (US) is presented to a dog, the dog starts to salivate (UR) When a bell is presented to a dog, without food, it produces nothing but an *orienting response* (OR) If the bell (CS) is repeatedly associated with the presentation of food, the dog will learn to salivate (CR) when the bell rings, even if the bell is now not followed with food (no US or extinction) Classical conditioning is a form of associative learning However, the animal being conditioned not only makes an association between two stimuli, but also learns a temporal relationship between a CS and a US Because this association is non-commutative, it represents the learning of a fundamental causal relationship A subject learns that the CS predicts the US, that is when a subject detects a CS, a US is expected

With classical conditioning an organism is able to discriminate cause and effect relationships in the environment

1.2 Aims and Method:

After a presentation of the physiological computational context and after a brief review of properties of classical conditioning and a review of some models of Pavlovian conditioning, I will present two new models of Pavlovian conditioning The first, presented in Chapter 4, and called the *delay-producing connections* (or DPC) model, is an extension of the Klopf (1988) and Sutton & Barto (1981) models The DPC model includes two contributions It proposes (a) representing the trace of each CS by a third-order differential equation, and (b) replacing each CS in the activation rule by the trace of the CS The second model, presented in Chapter 5, and called *adaptive delays* (or AD) model, is an extension of the DPC model which accounts for the phenomenon of inhibition of delay and for CS preexposure effects The AD model assumes that the amplitude of the trace of a CS as well as the position of its peak can change during conditioning Both models have interesting features each reproduces the shape of a CR, the curve of efficacy of conditioning as a function of the interstimulus interval (ISI), the dependence of the optimal ISI on CS duration, the extinction of a CR (even for long lasting CSs as opposed to Klopf's (1988) model), and several other properties of classical conditioning However, the AD model does not give a reliable account of some properties that are explained by the DPC model I also present a new method of measuring different parameters of classical conditioning (Section 5 5 of Chapter 5) My work is theoretical and the "experiments" accomplished are computer simulations

Many models of Pavlovian conditioning are discrete as a function of intensity of stimuli (e g , Hall & Pearce, 1980, Rescorla & Wagner, 1972, Wagner, 1975). This means that a stimulus is assumed to be either present or absent One of my aims is to incorporate the intensities of the stimuli as real numbers Another way to divide models is according to their real-time properties Some model are non-real-time models (e g , Hall & Pearce, 1980, Rescorla & Wagner, 1972, Wagner, 1975) and others are real-time models (e g , Gluck & Thompson, 1987, Klopf, 1988, Sutton & Barto, 1981) "Real-time" in this context does not mean continuous time, it means "the temporal association of signals each critical event in the sequence leading to learning has a time of occurrence associated with it, and this time plays a fundamental role [ ]" (Klopf, 1988), i e the time sequence of events is important Non-real-time models usually have the trial as the time unit Real-time models, however, make it possible to explore time contingencies and to produce more sophisticated predictions This is important because close to 30% of the

major properties of classical conditioning (see Table D 1 in Appendix D) are real-time properties With a real-time model it is possible to explain the influence of stimulus duration and delays between stimuli on the resulting CR intensity This influence is critical For example, if the CS is presented after the US (backward conditioning) a very weak CR develops, while a strong CR develops when the CS is presented just before the onset of the US (as in trace conditioning) Also, with a model that is continuous in regard to intensity, it is possible to explain the influence of the US intensity and the CS intensity on the CR strength

Models of Pavlovian conditioning can be divided into two categories in another manner, according to their claim to universality The first category claims to explain all types of classical conditioning Rescorla & Wagner's (1972) model and the Wagner (1981) SOP model would be in this category The second category seeks to explain a specific reflex of a given animal An example of this latter category would be Gluck-Thompson's (1987) model of the siphon retraction reflex of the Aplysia, and the Schmajuck-Moore (1989) model of hippocampal manipulations on the classically conditioned *nictitating membrane response* (NMR) The approach that I take falls into the first category However, in achieving greater generality, there is a risk of appearing more arbitrary To minimize that risk, I apply my models to a particular set of data, namely NMR in rabbits

1.3 Developments in Classical Conditioning:

Since the seminal works of Bechterev (1913), Watson (1920) and Pavlov (Pavlov, 1927), classical conditioning has traditionally been thought to apply to unconditioned reflexes of secretion, motor response or emotional response Conditioning of secretion was shown with saliva, gastric juices (Pavlov, 1927), pancreatic enzymes, and insulin (Mayer, 1953) Conditioning of motor response has been studied, for example, with withdrawal of the leg (Bechterev, 1913), and the nictitating membrane reflex (Baldwin & Baldwin, 1981, Hughes & Schlosberg, 1938) Conditioning of emotional response was observed with the conditioning of fear (Watson, 1916), galvanic skin response (Perruchet, 1979), cardiac response (Bouchard, 1974, Morin et al , 1987), and food aversion (Garcia et al , 1966) Recently, the set of URs to which classical conditioning applies has been extended, although some of the results are still a matter for debate

Drug Tolerance

Siegel and his associates (Siegel et al , 1976, 1977) used morphine as the US producing an UR of analgesia They suggested that the environment where the drug is taken can act as a CS and that the CR is opposed to the UR Morphine produces analgesia

and hypoactivity while the opposed CR, they propose, produces hyperalgesia and hyperactivity It has also been suggested that conditioning is responsible, at least in part, for the phenomenon of drug tolerance (MacRae & Siegel, 1988, Schnur & Martinez, 1989, Siegel, 1976, 1987) Tolerance to a drug develops when, with repeated use, the effectiveness of the drug declines and thus larger doses are necessary to achieve the same pharmacological effects Siegel (1977) found that exposure to the environment (CS) without the drug (US), once the association has been "conditioned", results in the extinction of the opponent CR, the elimination of the CR produces a stronger reaction to the drug itself Siegel and his associates (Siegel et al , 1982) observed that drug overdose typically occurs when an addict takes his or her usual drug dose, but in an unfamiliar environment This opposite conditioned reaction (CR) to drugs has been shown with drugs other than morphine. Siegel (1972, 1975) showed that animals to whom insulin was administered displayed a CR of hyperglycemia; Lang (1966) observed that animals with a history of atropine administration demonstrate hypersalivation as a CR; Greeley (1984) and Mansfield and Cunningham (1980) discovered opposed CR to the hypothermic effect of alcohol; Hinson, Poulos & Cappell (1982) showed opposed CR to the sedative effect of pentobarbital, Poulos (1981) showed opposed CR to the anorexic effect of amphetamine. Mathematical models of addiction assume an exponential decay over time of recidivism pressure and relapse rate (Fan & Elketroussi, 1989) This is consistent with the view that the environment acts as a CS and that the CR is to take drugs This CR is extinguished in an exponential fashion as a usual classically conditioned CR would be

Placebo Effect

Wickramasekera (1985) proposed that the *placebo effect* could be modeled using classical conditioning, and furthermore he made 17 specific predictions from the model. One of these predictions is a description of the *nocebo* effect as the antithesis to placebo effect. CSs can acquire harmful effects through association with illness.

Immune System

Ader and Cohen (1982, 1985) accidentally discovered that environmental events could suppress the functioning of the immune system Following the pairing of saccharin-flavored water (CS) with cyclophosphamide (US), a drug that produces nausea and suppresses the immune system, some of the test animals died as a result of the presentation of the CS without the US Later they confirmed those preliminary results by studying the development of the immune disease lupus erythematosus in New Zealand mice Other studies have successfully classically conditioned other immune responses such as histamine release in guinea pigs (Russell et al , 1984), natural killer cell activity

in mice (Ghanta et al , 1985), adjuvant arthritis in rats (Klosterhalfen & Klosterhalfen, 1983) and delayed-type hypersensitivity in normal humans (Smith & McDaniels, 1983)

Vicarious Learning:

A subject can learn to respond to a particular stimulus as a result of observing the experience of others The development of a CR following such an observation is called *vicarious conditioning* Bandura (1977) demonstrated that CS-CR associations can be acquired by humans through vicarious conditioning experiences Mineka et al (1984) found that monkeys learned to fear snakes after exposure to another monkey reacting fearfully to a snake Without such exposure, the monkeys did not fear snakes

The foregoing studies indicate that Pavlovian conditioning is such a widespread phenomenon that it deserves detailed study Furthermore a model capable of predicting the properties of classical conditioning is likely to have applications in a wide range of domains

1.4 The Neural Network Paradigm:

Models containing networks of neuron-like units have become dominant in the study of both cognitive psychology and artificial intelligence (e g , Anderson & Rosenfeld, 1988, Feldman, 1985, Lacouture & Marley, 1991, Rumelhart & McClelland, 1986; Shultz & Schmidt, 1991) Artificial neural networks are frequently referred to as connectionist models, parallel distributed processing (PDP) models, or adaptive/self-organizing networks. These networks typically have a large number of units and are applied to phenomena such as visual pattern perception, decision making, word recognition, and coordinated motor actions

Main Characteristics of Neural Network Models:

Artificial neural networks are assumed to possess two basic features

a) Each neuron-like unit is governed by two equations, an activation rule and a learning rule The *activation rule* involves the combination of inputs entering the unit and determines what the output should be. *Connection weights* are associated to each connection. The influence of an input on the output under the activation rule is proportional to its connection weight The *learning rule* specifies how the connection weights should be modified.

b) Interactions between units imply the transmission of activation levels from the output of one unit to the input of another. Most often outputs are postulated to be an all or none firing (0 or 1 usually) The input level of the receiving unit is often the product of the current activation level by the current connection weight at the receiving unit

Brief History

McCulloch and Pitts (1943) were the inventors of our modern concept of the activation rule They contended that a neuron will fire in an all-or-none fashion if the sum of its inputs exceeds a certain threshold This is mathematically represented by Equations 1.1 and 1 2

$$Y(t) = f\Big(\sum_{i=1}^{n} [\, V_i(t)\, X_i(t) \,] \Big), \tag{1.1}$$

$$\text{where } f(Z) = \begin{cases} 1 & \text{if } Z > T, \\ 0 & \text{otherwise,} \end{cases} \tag{1 2}$$

and where·

$Y(t)$ is the output at time t,

$V_i(t)$ is the connection weight of input i at time t;

$X_i(t)$ is the activation level of input i at time t,

$f(Z)$ is the activation function (or threshold function),

and T is the threshold

Donald O Hebb (1949) is the originator of the principle on which many modern learning rules are based He wrote·

> "When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency as one of the cells firing B is increased "

In other words, a connection weight increases if presynaptic activity is contiguous in time to postsynaptic activity This is called the Hebbian learning rule, and is mathematically represented by Equations (1 3) and (1.4)

$$\Delta V_i(t) = c\, X_i(t)\, Y(t), \tag{1.3}$$

where· $\Delta V_i(t)$ is the change in connection weight of input i at the time step t,

and c is a rate parameter

The same Equation in a differential form (Sutton & Barto, 1981) reads:

$$dV_i = c\, X_i(t)\, Y(t), \tag{1.4}$$

where dV_i is an infinitesimal variation of V_i at time t.

In effect, Hebb applied the ancient law of temporal contiguity at the neural level. In mathematical terms, the weight value V_i is the correlation of the activation of unit i with the output

Amazingly, the review of the main learning rules for neural networks is at the same time a review of some major models of classical conditioning Furthermore, the Hebbian learning rule has recently interested physiological psychologists because of the phenomenon of long-term potentiation (LTP) Bliss and Lomo (1973) discovered that intense electrical stimulation of the presynaptic perforant path of the hippocampus caused a long-term increase in the magnitude of excitatory postsynaptic potentials in the dentate gyrus, this increase is called LTP The results suggest that neurons are Hebbian in character with respect to their learning mechanism Researchers are trying to shed some light on the relationship between this physiological phenomenon and the behavior of whole animals (Shapiro & Caramanos, 1990)

Widrow-Hoff Learning Rule

Sutton and Barto (1981) realized that what has been called the Widrow-Hoff learning rule (Widrow & Hoff, 1960) was equivalent to the Rescorla & Wagner (1972) model of classical conditioning (Rescorla & Wagner, 1972) This rule is also known as the delta rule, or the adaline rule, or the LMS (least mean square) rule (Gluck & Bower, 1988; Rumelhart, McClelland et al , 1986) The Widrow-Hoff rule is an iterative procedure for solving a set of linear inequalities An exact solution to such a set of linear inequalities exists if the desired response is a linearly separable function of the stimulus patterns. This rule is expressed by the following equation:

$$\Delta V_i(t) = c_i [Z(t) - Y(t)] X_i(t), \quad (15)$$

where Z(t) is a special input signal

The Rescorla & Wagner rule has the form:

$$\Delta V_i(t) = \alpha_i \beta [\lambda(t) - \sum_{j \in S} V_j(t)], \quad (16)$$

where: α_i is the salience of the CS_i,

β is related to the associability between the US and the CS_i

(Rescorla & Wagner assume that there are two possible values for β

β_0 which is the value of β when there is no reinforcement and

β_1 which is the value of β when there is a reinforcement),

$\lambda(t)$ is the intensity of the US at time t,

and S is the set of (indexes of) all stimuli present on the current trial

α_i and β are constant valued learning rate parameters $\lambda(t)$ has a constant positive value when the US is present and equals zero when the US is absent

We can show that Equation 1 5 is a special case of Equation 1 6 when $X_i(t)$ is equal to 1 when the CS_i is present and is equal to 0 when the CS_i is absent By putting $Y(t)=\lambda(t)$, and $c_i = \alpha_i \beta$ in Equation 1 5, we get

$$\Delta V_i(t) \qquad \alpha_i \beta \left[\lambda(t) - \sum_{j=1}^{n} [X_j(t) V_j(t)] \right] \qquad (1\ 7)$$

Notice that $\sum_{i \in S} V_j(t)$ and $\sum_{j=1}^{n} [X_j(t) V_j(t)]$ are two equivalent notations The first term is the sum of all the weights of the CSs present in a trial. The second term is the sum of all the weigths multiplied by $X_i(t)$, and $X_i(t)$ is equal to 1 when the CS_i is present and is equal to 0 when the CS_i is absent Thus, Equations 1.7 and 1.5 are equivalent under the given conditions to Equation 1 6.

Klopf (1972, 1982)

Klopf introduced the notions of synaptic eligibility and reinforcement into real-time learning mechanisms, taking into account sequential rather than simultaneous events His learning rule is.

$$\Delta V_i(t) = c\, X_i(t-k)\, Y(t-k)\, S(t), \qquad (1.8)$$

where. $S(t)$ is the neuronal membrane potential ($S(t)$ is approximately equal to $Y(t)$);

and k is the nominal interval of time required for a neuronal output to feed back and influence the neuronal input

In this model, presynaptic activity $X_i(t-k)$ and postsynaptic activity $Y(t-k)$, when they occur in conjunction, render a synapse eligible for modification However, the efficacy of an eligible synapse does not change unless the subsequent membrane potential, $S(t)$, is nonzero This $S(t)$ functions as a reinforcer This learning rule correlates events of the past [$X_i(t-k)$ and $Y(t-k)$] with an event of the present [$S(t)$]

Sutton & Barto (1981)

Sutton and Barto invented a learning rule for their model of classical conditioning which is also used for other applications of neural nets (Simpson, 1990) Their rule is.

$$\Delta V_i(t) = c\, \bar{X}_i(t) [\, Y(t) - \bar{Y}(t) \,], \qquad (1.9)$$

where. $$\bar{X}_i(t) = \alpha\, \bar{X}_i(t-1) + X_i(t-1), \qquad (1.10a)$$

$$f(Z) = \begin{cases} 1 & \text{if } Z > T, \\ 0 & \text{otherwise,} \end{cases} \qquad (1.2)$$

$$Y(t) = f\Big(Z(t) + \sum_{i=1}^{n} [V_i(t) X_i(t)] \Big), \qquad (1.10b) \text{ from } (1.1)$$

and $$\bar{Y}_j(t) = \beta\, \bar{Y}_j(t-1) + (1-\beta)\, Y_j(t-1). \qquad (1.10c)$$

The variables and parameters are the following

$\overline{X}_i(t)$ is the trace of $X_i(t)$ ($\overline{X}_i(0) = 0$).

$X_i(t)$ signals the presence of the CS_i when the CS_i is present $X_i(t) = 1$, otherwise $X_i(t)=0$.

$Y(t)$ is the output response at time t $Y(t)$ is the UR when $X(t) = 0$ and $Y(t)$ is the CR when $Z(t)=0$.

$\overline{Y}(t)$ is the trace of $Y(t)$ at time t ($\overline{Y}(0) = 0$).

$Z(t)$ is the intensity of the US at time t,

α is a positive constant,

and β is a positive constant with value less than 1.

It is important to note that in the preceding Equations 1.10a and 1.10b, the notation (t-1) does not represent current real time t minus one unit of real time, but rather it represents the time step number minus one time step. This notation is used throughout the thesis. It is a standard in this area (see Klopf, 1986, 1988, Sutton & Barto, 1981).

The curve of $\overline{X}_i(t)$ looks like an average of $X_i(t)$ in the last Δt multiplied by a constant. The curve of $\overline{Y}(t)$ looks like an average of $Y(t)$ in the last Δt. If we interpret $\overline{X}_i(t)$ and $\overline{Y}(t)$ as means, the rule in Equation 1.9 computes the correlation between the mean value of $X_i(t)$ and the deviation from the mean of $Y(t)$.

Klopf (1986) & Kosko (1986)

Klopf (1986) has proposed a correlation equation that correlates the changes in $X_i(t)$ with the change in $Y(t)$. This same rule was independently discovered by Kosko (1986). It is called differential Hebbian learning. The relation is

$$\Delta V_i(t) = c\,\Delta X_i(t-k)\,\Delta Y(t) \qquad (1.11)$$

Klopf (1988)

Klopf (1988) modified his differential Hebbian learning rule in order to simulate classical conditioning. This rule is called drive-reinforcement learning (D-R) and is as follows:

$$\Delta V_i(t) = \Delta Y(t) \sum_{k=1}^{r} C_k \left| V_i(t-k) \right| \Delta X_i(t-k), \qquad (1.12)$$

where: $\Delta V_i(t) = V_i(t+1) - V_i(t)$,

$\Delta Y(t) = Y(t) - Y(t-1)$,

$$\Delta X_i(t-k) = \begin{cases} X_i(t-k) - X_i(t-k-1) & \text{if } [X_i(t-k) - X_i(t-k-1)] > 0, \\ 0 & \text{otherwise.} \end{cases}$$

Here: $X_i(t)$ is the frequency of action potentials at the ith synapse at time t,

$\Delta X_i(t-k)$ represents a positive change in presynaptic level at time t-k,

$Y(t)$ is the postsynaptic frequency of firing at discrete time t,

$\Delta Y(t)$ represents a change in postsynaptic level at time t,

τ is the longest interstimulus interval, measured in discrete time steps, over which delay conditioning is effective;

and C_k is an empirically established learning-rate constant

Note that $X_i(t)$ and $Y(t)$ are not limited to the binary values 0 and 1 They represent frequencies of firing, so they can take any real value between 0 and, say, 300 As E D. Adrian (1946) found, the frequency of firing (or frequency of action potentials) in a nerve cell is related to the intensity of the stimulus (Kuffler, Nicholls, & Martin, 1984) Action potentials are brief electrical pulses, about 0 1 V in amplitude, that last for about 1 msec and move along nerves Thus this model can simulate CS intensity variation effects Since Y(t) can take other values than just 0 or 1, it is defined with a different activation function $f(Z)$ than Equation 1 2. The activation function is now the following:

$$f(Z) = \begin{cases} 0 & \text{if } Z < T, \\ Z & \text{if } T \leq Z \leq 1; \\ 1 & \text{if } Z > 1 \end{cases} \qquad (1.13)$$

Drive-reinforcement learning theory has been widely used since 1988 (Baird & Klopf, 1992; Baird and Klopf, 1993, Gluck, Parker & Reifsnider, 1988; Klopf, Morgan & Weaver, 1993, Morgan, Patterson & Klopf, 1990)

Classical conditioning, by definition, is a form of associative learning. Weights in neural networks represent association strengths between units It therefore makes sense to posit that an effective model of classical conditioning will make an interesting learning rule for neural networks In this perspective, the search for an effective learning rule is the search for an effective model of classical conditioning The converse also seems to be true Neural networks can be used to simulate classical conditioning and make it possible to reproduce behaviors that other models of Pavlovian conditioning cannot adequately account for (Kehoe, 1989; Klopf, 1988, Sutton & Barto, 1981) Examples of those behaviors are (see Chapter 2 for definitions) compound conditioning, negative patterning (XOR), and positive patterning

Neural networks using learning rules analogous to classical conditioning models can then be applied to more cognitive problems such as categorization learning (Shanks, 1991), belief persistence (Vallée-Tourangeau, 1993), evaluation of causality, and so on. In this way, there is a bridge between behavioral and cognitive psychology

2. THE MAJOR PROPERTIES OF CLASSICAL CONDITIONING

Classical conditioning is a very complex phenomenon that exhibits numerous properties The logical procedure is to study those characteristics before building a model reproducing Pavlovian conditioning Thus the properties of Pavlovian conditioning are presented in detail, since they are what the models attempt to fit The reader can refer to Table D 1 in Appendix D for a summary listing of the following properties

2.1 Types of Classical Conditioning

We have seen that classical conditioning can be shown with different USs, for example with food, loud noise shock (Hall & Pearce, 1980), morphine injection (Siegel, 1982), immune system depression (Adler & Cohen, 1980, 1982, 1985), puff of air in the eye (Gormezano et al , 1983), and so on. The CSs can also be in different modalities, for example visual, auditory, tactile, or gustative (Klein, 1991)

Conditioning can be either *excitatory* or *inhibitory* In excitatory conditioning the CS acquires the ability to signal the presence of a US, whereas in inhibitory conditioning the CS signals the absence of a US (Doré, 1988) The strength of an excitatory CS can only be measured by the strength of the CR, but the strength of an inhibitory CS can be evaluated by indirect measures The presence of an inhibitory CS_1 can make the acquisition of a new CR (CS_2 - US) slower This is called the *retardation test* (Marchand & Moore, 1974, cited by Gormezano, Kehoe, & Marshall, 1983) Also if a second CS (CS_2) has acquired the ability to produce a CR (CR_2) then the simultaneous presentation of the inhibitory CS_1 with the excitatory CS_2 produces a smaller CR_2. This is called the *summation test* (Rescorla, 1969, cited by Doré, 1988) Excitatory and inhibitory conditioning can be referred to as the learning of a positive and a negative correlation respectively (Kamin & Kremer, 1971)

At one time, there was a concern regarding the nature of the conditioned response. Is the CR just the UR elicited by the CS? Or is the CR a behavior distinctively different from the UR? Pavlov (1927) defended a stimulus substitution (S-S) theory suggesting that as a result of conditioning, the CS becomes able to elicit the UR More recently, the consensus is that even when the CR is analogous to the UR, they are never perfectly identical (Mackintosh, 1974) In fact, the CR seems to mimic the UR in some cases and to be opposed to it in other cases When a CR mimics a UR it is a *conditioned facilitation* When a CR is opposed to a UR it is a conditioned diminution and the CR is called a *compensatory CR* (Wagner, 1981)

The following are examples of a CR that mimics the UR conditioning of the salivation reflex with dogs (Pavlov, 1927), conditioning of the nictitating membrane response with rabbits (Gormezano et al , 1983), human eyelid conditioning (Kimble and Ost, 1961, cited by Wagner, 1981), siphon retraction reflex with Aplysia (Kandel & Schartz, 1985) The following are examples of compensatory CR CR to the sedative effect of pentobarbital (Hinson et al , 1982), CR to the hypothermia effect of alcohol (Greeley, 1984, Mansfield & Cunninghan, 1980) and all the other examples mentioned earlier for drug tolerance Cardiac deceleration is often the CR to shock-paired CSs, whereas the UR to shock is cardiac acceleration (Obrist, Sutterer, & Howard, 1972)

Note that both an inhibitory CS and a compensatory CS have the ability to decrease the UR However, the compensatory CS generates a measurable CR while an inhibitory CS produces a "silence of behavior" Table 2 1 summarizes the different types of CR with their motivational properties Often the behavior is complicated by a mixture of facilitation and diminution depending on what is measured. For example Wagner (1981) reports that, in limb flexion conditioning in the dog with a cortical US, the probability of a detectable UR to a threshold US was increased by a prior CS, while the vigor of the UR to a training-level US was decreased.

	Aversive Conditioning		Apetitive Conditioning	
	Facilitating CR	Compensatory CR	Facilitating CR	Compensatory CR
Conditioned Excitation	Aversive	Apetitive	Apetitive	Aversive
Conditioned Inhibition	Neutral	Neutral	Neutral	Neutral

Types of Pavlovian conditioning with their motivational properties.

Table 2.1

Conditioning situations can vary according to the rate of reinforcement. In *continuous reinforcement* a CS is followed by a US 100% of the time, while a *partial reinforcement* procedure is one in which only a portion of the CSs that are presented are followed by the US.

2.2 Properties of Pavlovian Conditioning

The types of conditioning that have just been described possess many characteristics Those characteristics are summarized in the rest of this chapter

Acquisition, Extinction and Spontaneous Recovery

The typical *acquisition curve* of the CR, i e the strength of the CR as a function of trials, is usually taken to be sigmoid in shape (S-shaped) (Mackintosh, 1974, Pavlov, 1927) This means that the first trials show a positively accelerating curve for the CR while the subsequent trials show a CR amplitude that accelerates negatively and reaches a constant value, the asymptotic value of the CR Acquisition has a slower initial rate for partial reinforcement but the asymptotic levels of the CR are high (Gormezano et al , 1983) Spaced CS-US presentations produce faster acquisition of the CR than does massed presentation (Klein, 1991) Caffeine and benzedrine (two stimulants) increase the rate of acquisition Sodium bromide (a depressant) slows the rate of acquisition (Hilgart & Marquis, 1940 cited by Klein, 1991)

The conditioned response decays when the CS is presented without the US This phenomenon, called *extinction*, is observed to be negatively accelerating and normally reaches zero as an asymptote (Klein, 1991) The extinction of an excitatory CR is usually described as being slower than its acquisition (Bush & Mosteller, 1955, Rescorla & Wagner, 1972, Wagner, 1981) However, conditioned inhibitors extinguish much more slowly (if at all) than conditioned excitators Extinction is slower in partial reinforcement procedures than in continual reinforcement procedures This is called the *partial reinforcement extinction effect (PREE)* (Gormezano et al , 1983) Extinction is faster with massed extinction than with spaced extinction (Hilgard & Marquis, 1940 cited by Klein, 1991) Empirical investigation reveals that the longer the CS-alone exposure lasts, the greater is the reduction in CR strength (Monti & Smith, 1976, Shipley, 1974, cited by Klein, 1991) The two stimulants caffeine and benzedrine slow extinction but the depressant sodium bromide enhances extinction (Hilgard & Marquis, 1940 cited by Klein, 1991).

If one waits a certain period of time after an extinction process and then presents the CS again, the CR often reappears The return of the CR following extinction is called *spontaneous recovery* (Doré, 1988) Continued presentation of the CS without the US eventually leads to the long term suppression of the CR (Klein, 1991) I interpret this last observation in the following terms the more CS-alone presentations there are, the smaller the spontaneous recovery Bouton (1991) found that *reacquisition* of an extinguished CS is dependent on the context In a context similar to the extinction context, the reacquisition would be slower than the original acquisition

Time Relationships Between the US and the CS

Delay conditioning is a conditioning procedure in which CS onset precedes US onset and CS offset occurs at the same time or after US onset Delay conditioning produces conditioned excitation (Klein, 1991)

Trace conditioning is a conditioning procedure in which the CS is presented and terminated prior to US onset Trace conditioning produces conditioned excitation (Klein, 1991) As the delay between CS onset and US onset (i e., the *interstimulus interval* or ISI) increases, the rate of acquisition increases, reaches a peak, then decreases, and tends toward zero for long ISI The asymptote of the CR is more a function of the ISI than of the CS duration (Klein, 1991)

In the *simultaneous conditioning* paradigm, the CS and US onsets occur at the same time, as do CS and US offsets Simultaneous conditioning produces small excitatory conditioning or no conditioning at all (Flaherty, 1985 cited by Klopf, 1988). Some conditioning has been reported for simultaneous conditioning in the case of fear conditioning (Burkhardt & Ayres, 1978, cited by Klopf, 1988)

In *backward conditioning*, the US is presented and terminated prior to the CS onset Currently, the consensus appears to be that backward conditioning can lead to a weak conditioned excitation initially but that extended backward conditioning usually yields conditioned inhibition (Wagner, 1981)

Delay conditioning produces stronger conditioning than trace conditioning Trace conditioning produces stronger conditioning than simultaneous conditioning Finally, simultaneous conditioning produces stronger conditioning than backward conditioning (Klein, 1991) "Longer optimal ISIs have been observed for long fixed delay CSs than for short trace CSs" (Schneiderman, 1966, cited by Sutton and Barto, 1981)

In a *temporal conditioning* experiment, there is no distinctive CS. Instead, the US is presented at regular intervals, and over time the CR will appear just prior to the onset of the US (Klein, 1991).This leads to a weak and unstable CR (Doré, 1988). Another temporal phenomenon was revealed by Pavlov's classic research (1927) with dogs He demonstrated the development of the ability to suppress the CR until the end of the CS-US interval, a phenomenon he labeled *inhibition of delay*. The CR peak tends to be located around the time of US-UR onset (Gormezano et al , 1983) Kimmel reported that the latency of the galvanic skin response (GSR) increases with training (Kimmel, 1965 cited by Klein, 1991). The variance of the CR seems to increase with the CS-US interval (Gormezano et al , 1983) Millenson and his associates (Millenson et al , 1977), studying the nictitating membrane reflex in rabbits, gave the US sometimes at 700 msec after CS onset and sometimes at 200 msec after CS onset The CR exhibited 2 peaks, one at 200

msec and one at 700 msec The peak of the CR at 700 msec appears only when the US is not presented at 200 msec The peak height ratio is proportional to the number of reinforcement ratio for both delays CS preexposure has been shown to slow the acquisition of inhibition of delay (Schachtman, Channel, & Hall, 1987)

US and CS Effects

US duration effects designate the fact that the longer the US lasts, the faster the acquisition and the higher the asymptote of the CR (Gormezano et al , 1983) In the same way as US duration, an increased US intensity produces faster acquisition and a higher asymptote of the CR This is called *US intensity effects* (Prokasy, Grant, & Myers, 1958 cited by Klein, 1991) Prior exposure to a US reduces its ability to reinforce excitatory conditioning, a phenomenon called *US preexposure effect* (Randich & LoLordo, 1979) A subset of the US preexposure effect is the *proximal US effect* In rabbit eyelid conditioning (Terry, 1976) and in taste aversion conditioning (Domjan & Best, 1977), it has been shown that presentation of a "proximal US" shortly before a CS is paired with a "conditioning US" attenuates the latter US's potency

CS intensity can affect the strength of the conditioned response A greater CR strength is produced by a more intense CS (Barnes, 1956, Frey, 1969, cited by Klein, 1991), a phenomenon called *CS intensity effects* However, an intense CS does not produce an appreciably greater CR than a weak CS Nonetheless, if both the intense and the weak CS are experienced, the intense CS will produce a significantly greater CR than the weak CS (Grice and Hunter, 1964 cited by Klein, 1991)

Nonreinforced presentations of a CS prior to reinforced presentations retards subsequent acquisition of the CR and lowers the asymptotic strength of the CR These are the *CS preexposure effects* or *latent inhibition* (Baker & Mackintosh, 1977, Lubow & Moore, 1959) Latent inhibition affects both excitatory and inhibitory conditioning (Klein, 1991). In *sensory preconditioning*, two neutral stimuli CS_1 and CS_2, are paired prior to reinforcement In a second phase, CS_1 is paired with a US The CS_1-US pairing results in the ability of the CS_2, as well as the CS_1, to elicit the CR The best procedure to produce a strong effect requires that (1) the CS_2 precedes the CS_1 by several seconds and (2) use of only a few CS_2-CS_1 pairings in order to prevent the development of learned irrelevance (Rizley & Rescorla, 1972 cited by Klein, 1991) Extinction of the CR_1 also extinguishes the CR_2 (Rizley & Rescorla, 1972)

Configural Learning

Configural learning designates the classical conditioning procedures involving more than one CS The following describes properties in different configural learning situations

The acquisition of a CR is impaired when the CS-US interval is too long However, several studies (reported by Klein, 1991) have observed that the attenuation of conditioning produced by a CS-US interval can be reduced if a second stimulus is presented between the CS and US Pavlov (1927) found that a more intense tone overshadowed the association of a less intense tone with the US The rate of acquisition is greater and the asymptote of the CR is higher for the more salient CS when two CSs of different salience are presented together with the US (Klein, 1991) This is called *overshadowing* Overshadowing does not always occur when two cues of different salience are paired with a US, in fact there are some circumstances in which the presence of a salient cue leads to a stronger CR than would have occurred if the less salient cue had been presented alone (Garcia & Rusiniak, 1980, cited by Klein, 1991) When this last situation occurs it is called the *potentiation of a less salient cue*

Kamin (1968) demonstrated that the presence of a previously associated cue (CS_1) will prevent, or block, the development of a new association between a second cue (CS_2) and the US We say that there is *blocking* of CS_2 by CS_1 Mackintosh and his associates (Dickinson, Hall, & Mackintosh, 1976, Mackintosh, Bygrave, & Picton, 1977, Pearce & Hall, 1980) showed that a surprising[1] event (surprising US or CS) can prevent the CS_1 from blocking the CS_2-US association This is sometimes called *unblocking*

Pavlov (1927) observed that following CS-US pairing, presenting the CS with another neutral stimulus (CS_2) enabled the CS_2 to elicit the CR Note that the CS_2 is presented a certain length of time before the CS_1 as in a CS-US pairing The usual CS-US pairing is a first-order conditioning, while learning the CR as just described, by association of CS_2 with CS_1, is a *second-order conditioning* According to Pavlov any conditioning process of an order higher than one is *higher-order conditioning*. The strength of a CR acquired through higher-order conditioning is weaker than that developed through first order conditioning. The CR to the CS_2 rises and then extinguishes after repeated pairing with the CS_1 The CS_1 also extinguishes (Klein, 1991)

The phenomenon called *conditioned inhibition* can be produced in different ways One way is to establish an excitatory conditioning by pairing a first CS (CS_1) with a US, in a first phase In a second phase, the CS (CS_1) is presented at the same time as a new CS (CS_2) but the US is absent The CS_2 becomes a conditioned inhibitor. In relation to conditioned inhibition, it is opportune to introduce a notation that is useful to describe experiments with several conditioned stimuli The different CSs are represented by

[1] A surprising stimulus in this context is a stimulus that is presented for the first time or that is presented at a new intensity level

different letters The minus (-) sign as a superscript indicates that the configuration is not reinforced, while a plus (+) sign indicates that the configuration is reinforced Conditioned inhibition would be therefore represented by A^{+} AB^{-} As opposed to second order conditioning, successive pairing of CSs A and B does not lead to extinction of both CRs They both reach an asymptote Note that the difference between conditioned inhibition and second order conditioning is that in conditioned inhibition the onset and offset of the stimuli are synchronized, while in second order conditioning stimulus B precedes stimulus A (Klein, 1991)

In an *overexpectation* task, each of two CSs (A and B) are paired separately with a US and both acquire the maximal CR Both CSs are then combined, in a second phase, and the compound AB is reinforced with the same US as each received in the first phase The first trials of the second phase exhibit a CR that is greater than each individual response, after which the CR to the compound decreases If we test each individual CS in a third phase, each of their CRs are smaller than in phase I (Rescorla & Wagner, 1972) This sequence can be represented by A^{+} B^{+}, AB^{+}, test A test B In a *superconditioning* procedure, a first CS (A) is treated in such a way that it becomes a conditioned inhibitor (e g , X^{+} AX^{-}), then stimulus A is paired with a second stimulus B and reinforced B will have a faster rate of acquisition and a higher asymptote in presence of the inhibitor A than it would have without the inhibitor (Rescorla & Wagner, 1972) This sequence can be depicted by X^{+} AX^{-}, AB^{+}

In a *discrimination learning* experiment, presentations of the compound AX are reinforced and mixed with presentation of the compound BX that is not reinforced (AX^{+} BX^{-}) The conditioned response to AX tends toward an asymptote, the conditioned response to BX increases and then decreases The CR to A is greater than the CR to X The CR to X is greater than the CR to B The CR to B tends toward zero (Rescorla & Wagner, 1972) The *psedodiscrimination* procedure is a treatment in which the compounds AX and BX are both partially reinforced on a 50% schedule (i e , reinforced, on average, once every two presentations of the compound) This can be represented by AX^{-} 0 5, AX^{+} 0.5, BX^{-} 0 5, BX^{+} 0 5 The conditioned response to X after training is greater than the CR to A or to B The CR to A is equal to the CR to B (if they have equal salience) Even when we control for the number of US presentations, the CR to X is much greater in a pseudodiscrimination task than in a discrimination task (Rescorla & Wagner, 1972, Wagner, Logan, Haberlandt, and Price, 1968)

Compound conditioning uses configuration A^{+} AX^{+} that is, A is reinforced when presented alone and the compound AX is also reinforced This procedure produces a greater CR for A than for X Another compound conditioning experiment uses the

configuration A^- AX^+, where A is not reinforced when presented alone and the compound AX is reinforced This procedure produces the reverse result, namely a greater CR for X than for A (Rescorla & Wagner, 1972)

Positive patterning involves a more complex discrimination than the previous ones Positive patterning involves reinforced presentations of the compound (AB^+) intermixed with an equal number of unreinforced presentations of the separate components (A^- B^-) (Kehoe, 1989) The CR to the compound AB is greater than the CR to the separate elements (A or B) The CR to the separate elements (A or B) increases at first and then decreases during training *Negative patterning* involves the nonreinforced presentation of the compound AB (AB^-) intermixed with an equal number of reinforced presentations of the separate components (A^+ B^+) (Kehoe, 1989) The CR to AB increases faster than the CR to A or B and then decreases, tending toward zero Negative patterning takes more trials to learn than positive patterning. Kehoe (1989) has shown that the learning of conditioned inhibition (A^+ AB^-) in a first phase accelerates learning of negative patterning in a following phase

There are other types of configural learning tasks, such as the *feature positive* task (AB^+, A^-) and *stimulus compounding* (A^+, B^+, test AB) Recently Pearce (1993) used the following configuration A^+, B^+, C^+, ABC^- The compound of three stimuli was not reinforced while the individual stimuli were reinforced. He observed that the CR to the pairs of stimuli (AB, AC, or BC) decreased faster than the CR to the individual cues (A, B, or C)

This impressive list of properties is only a brief summary of the vast literature on classical conditioning. The list also constitutes the data domain that mathematical models of Pavlovian conditioning currently attempt to explain.

3. MODELS OF CLASSICAL CONDITIONING: A CRITICAL REVIEW

In the previous chapter some models of classical conditioning were described in the perspective of their use as learning rules for neural networks Now a few models of classical conditioning are going to be described and evaluated by comparing their predictions with the experimental properties of classical conditioning For each model, I give a description of their main assumptions, their major contributions and their major problems. The reader can refer to the Tables D 1 and D 2 in Appendix D for a summary of the properties of classical conditioning accounted for by the different models

3.1 Historical Perspective

Pavlov (1927) suggested that as a result of conditioning, the CS becomes able to elicit the same response as the US. According to Pavlov, the presentation of the US activates one area of the brain Stimulation of the neural area responsible for processing the US leads to the activation of a brain center responsible for generating the UR There is an innate, direct connection between the brain center stimulated by the US and the brain center controlling the UR When the CS is presented, it excites a distinct brain area When the US follows the CS, the brain centers responsible for processing the CS and the US are active at the same time In Pavlov's view, the simultaneous activity in two neural centers leads to a new functional neural pathway between the active neural centers The establishment of this connection causes the CS to activate the neural center processing the CS, which then arouses the US neural center, and finally the US center activates the response center for the CR In short, Pavlov asserts that the CS becomes a substitute for the US. Since Pavlov's (1927) work, it has been shown that in many circumstances of classical conditioning the CR does not mimic the UR Compensatory CRs are in contradiction with Pavlov's (1927) view (Schnur & Martinez, 1989, Siegel, 1987, 1988)

Konorski (1967) posited that the representation produced by any stimulus involves the activation of a series of "units" in order, in the same way as in a neuronal projection system, beginning with receptive units, followed by transit units, and eventually terminating with so-called gnostic units The units at several levels of the representational series are capable, when activated, of producing behavioral effects A CS will come only to activate a portion of the sequence appropriate to the US Konorski assumed that only the highest level, gnostic units, of the US representation can be activated by a CS and only the activity of such units will be reflected in the CR His view can account for the difference between the CR and the UR Wagner and his associates

drew inspiration from Konorski's (1967) theoretical view to build their SOP (Wagner, 1981), and AESOP models (Wagner & Brandon, 1989).

Hull (1943, 1952) introduced a mathematical formalism for learning and motivation that has been very influential. The ability of verbal theories to predict behavior is limited by their imprecise nature Hull's formalism was an attempt to correct this imprecision Hull (1943) developed a formula that expressed how the strength of a behavior is related to learning and motivation The formula was

$$ {}_sE_r = {}_sH_r \times D, \qquad (3.1) $$

where ${}_sE_r$ is the strength of the behavior (note. s r stands for Stimulus-Response),

${}_sH_r$ is the strength of the learned response (the habit),

and D is the strength of the drive.

For example, the strength of the behavior ${}_sE_r$ could be measured by the number of trials necessary to obtain extinction after an acquisition phase, the habit ${}_sH_r$ could be measured by the number of training trials presented during the acquisition phase; and the drive D could be measured by the number of hours of food deprivation in a case where food is used as a reinforcer By 1952, Hull had modified his initial formulation to add the construct of incentive motivation, symbolized as K K represents the quantity and quality of the reinforcement. Thus the formula becomes:

$$ {}_sE_r = {}_sH_r \times D \times K. \qquad (3.2) $$

This relation implies that the increment (or decrement) in learning is dependent upon the amount of habit already conditioned and upon the strength of the reinforcement.

In 1955, Bush and Mosteller presented a stochastic model of conditioning that was closely related to Hullian theory This model specifies the changes in probability of a response (Δp_n) as a result of a learning trial by the following equation:

$$ \Delta p_n = \beta (\lambda - p_n), \qquad (3.3) $$

where β is the learning rate parameter,

p_n is the probability of a response on trial n,

and λ is the asymptote of learning

β and λ are determined by the nature of the US and CS involved on a trial. This model reproduces the negative acceleration and the asymptote of the acquisition curve.

3.2 The Rescorla & Wagner (1972) Model

Main Assumptions of the Rescorla-Wagner Model

The reader can refer to Equation 1 6 for a formal description of this model. The four main premises of the Rescorla-Wagner (1972) model are the following :

1- There is a maximum associative strength that can develop between a CS and a

US The asymptotic level of conditioning is determined by the strength of the US

2- The amount of associative strength gained on a particular training trial is affected by the level of prior training

3- The rate of conditioning varies depending on the CS and the US used The rate is high with some stimuli, but low with others

4- The level of conditioning on a particular trial is influenced not only by the amount of prior conditioning to the stimulus but also by the level of previous conditioning to other stimuli associated with the US

The Major Contributions of the Rescorla-Wagner (1972) Model

The Rescorla-Wagner (1972) model gives a good account of the phenomena of overshadowing and blocking The model also provides an explanation for the US preexposure effect (Baker & Mackintosh, 1979), and for the acquisition of a conditioned inhibition Predictions are in agreement with experimental data for procedures of configural learning such as overexpectation, superconditioning, discrimination learning, pseudodiscrimination, and compound conditioning

The Problems of the Rescorla-Wagner (1972) Model

Behavioral data show that conditioned inhibition does not extinguish, while the Rescorla-Wagner (1972) model predicts its extinction. For example, a conditioned inhibitor is represented by a negative value of $V_i(t)$ and during extinction the US is absent (i.e. $\lambda(t) = 0$). Therefore Equation 1.6 becomes $\Delta V_i(t) = \alpha_i \beta [-V_i(t)]$ This term is positive since $V_i(t)$ is negative, so $|V_i(t)|$ decreases and therefore inhibition is extinguishing. The results of a simulation[1] of their model, shown in Figure 3 1, illustrate this problem. Notice that in the following figures CS 1, CS 2, V 1, and V 2 stand for CS_1, CS_2, V_1, and V_2

In order to predict the acquisition of a conditioned excitation in a second-order conditioning procedure with the Rescorla-Wagner (1972) model, one has to make a variable substitution V_1 becoming λ The model does not predict either the CS preexposure effect or sensory preconditioning Since the Rescorla-Wagner (1972) model is not a real-time model, none of the intratrial effects are explained by the model This means that delay conditioning, trace conditioning, backward conditioning, simultaneous conditioning, inhibition of delay, the shape of the curve of the CR, the US duration effects, and the CS duration effects are left unexplained

[1] In addition to my own new models, I simulated the Rescorla & Wagner (1972) model, the Pearce & Hall (1980) model, and the Klopf (1988) model The results are summarized in Appendix D, Table D 1 All figures, including Figure 3 1, result from these simulations

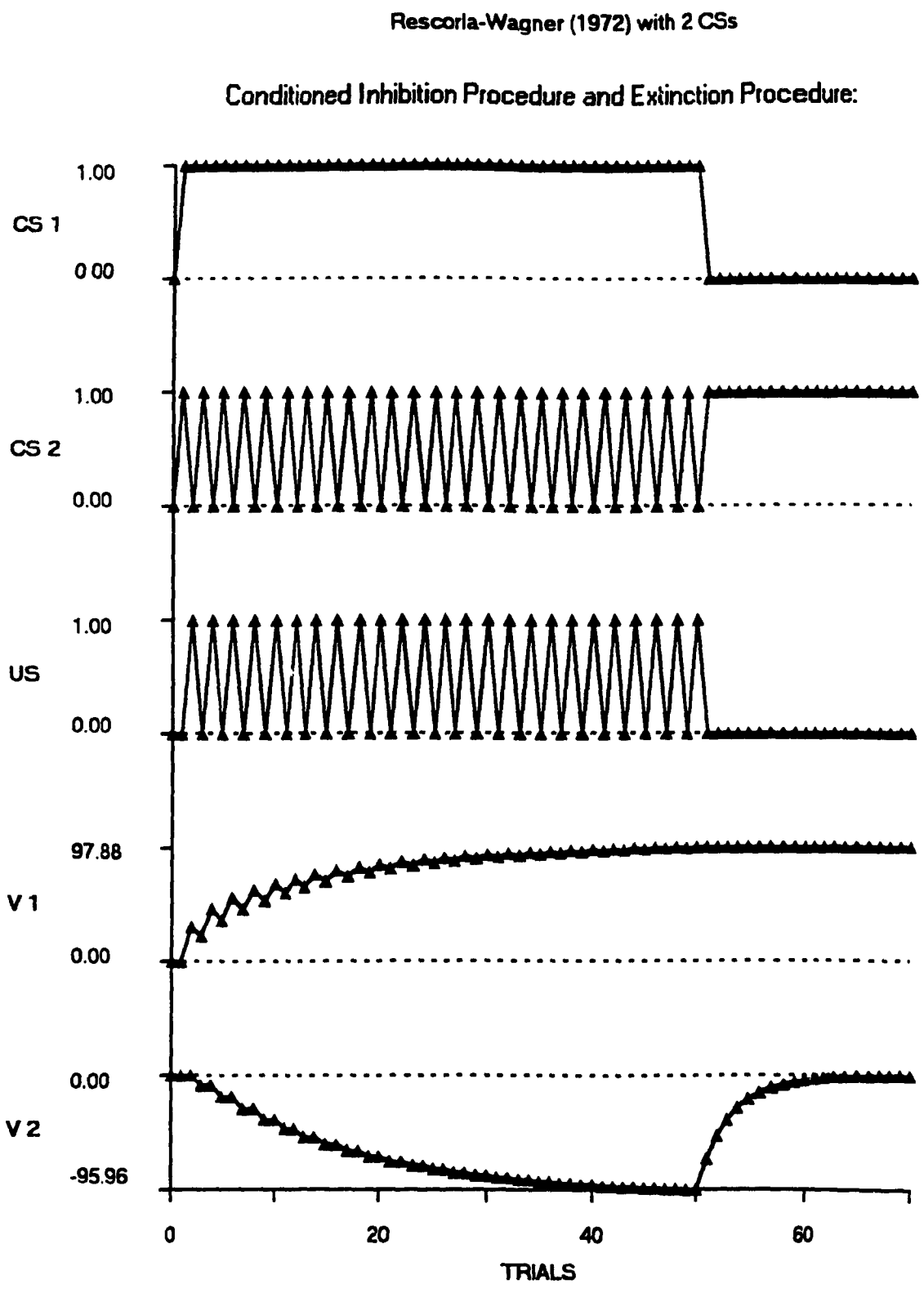

Figure 3.1 The CS2 in an acquisition phase becomes a conditioned inhibitor ($V2 < 0$) and in the extinction phase V2 tends toward zero.

3.3 The Sutton & Barto (1981) Model

Assumptions of the Sutton & Barto (1981) Model

The reader can refer to Chapter 1 (Equations 1.9, 1.10a, 1.10b, 1.2, 1.1) for a formal description of the model. The five core assumptions of the Sutton & Barto (1981) model are the following

1- There is a trace for each CS_i, that follows the amplitude of the CS_i, but increases more slowly and decays more slowly than the CS_i itself.

2- The CS_i comes to elicit the same response as the US, corresponding to Pavlov's (1927) stimulus substitution theory The CR and the UR are added together in what we can call the output response.

3- There is a trace of the output response which follows the amplitude of the output response but which increases more slowly and decays more slowly than the output response itself.

4- The trace of the CS_i causes the corresponding connection weight $V_i(t)$ to become "tagged" as being eligible for modification for a certain period of time (the duration of the trace of the CS_i). This is Klopf's (1972) idea

5- The effectiveness of a reinforcement is proportional to the difference between the output response and the trace of the output response.

The Major Contributions of the Sutton & Barto (1981) Model

Sutton and Barto's (1981) model is a real-time model which makes it possible to address the question of intratrial effects Their model reproduces a curve somewhat analogous to the asymptotic CR versus ISI (inter-stimulus interval) The model can simulate the CS and US duration effects. The behavioral observation that trace conditioning is stronger than simultaneous conditioning is reproduced. The inhibitory aspect of the backward conditioning procedure is predicted by the model The model also accounts for second-order conditioning Several other properties are explained the CS intensity effects, conditioned inhibition, blocking, overshadowing, US intensity effects, and several configural learning procedures

Problems with the Sutton & Barto (1981) Model

In the formulation of their model, Sutton and Barto posit that $X_i(t)$ is equal to 0 or 1 and that the constants c and α are the same for each CS This does not make it possible to have CSs of different salience In my modeling, I assume that $X_i(t)$ varies between 0 and 1, as do Z(t), Y(t) and $\overline{X}_i(t)$ According to Klopf (1988), the Sutton & Barto (1981) model does not reproduce experimental data for the delay conditioning procedure. The Sutton & Barto (1981) model predicts acquisition of a conditioned inhibition for delay conditioning instead of a conditioned excitation. This last problem occurs because in a

delay conditioning procedure the CS is present during the complete duration of the US. $\overline{X}_i(t)$ is necessarily greater at the end of the US than at the beginning of the US If we call t_1 the time step of US onset and t_2 the time step of US offset, then $\overline{X}_i(t_2) > \overline{X}_i(t_1)$ Let $\overline{Y}(t)$ - Y(t-1) and Z(t) = 1 when the US is present We have $\Delta V_i(t) \cong 0$ for $t_1 < t < t_2$, because $|Y(t)-Y(t-1)| \cong 0$ for $t_1 < t < t_2$ Then, after the first trial, the total change of connection weight ΔV_i is

$$\begin{aligned} \Delta V_i &\cong \Delta V_i(t_1) + \Delta V_i(t_2), \\ &= c\,\overline{X}_i(t_1)\,[\,Y(t_1) - Y(t_1-1)\,] + c\,\overline{X}_i(t_2)\,[\,Y(t_2) - Y(t_2-1)\,]. \end{aligned} \quad (3.5)$$

Because we are studying the first trial, $V_i(t) = 0$ for $t \leq t_1$ Also, since we postulate that learning is slow, i e $|\Delta V_i(t_2)| = \varepsilon << 1$, we obtain that

$$\begin{aligned} Y(t_1) &= f\Big(Z(t_1) + \sum_{i=1}^{n} [\,V_i(t_1)\,X_i(t_1)\,]\Big), \\ &= f(1+0), \\ &= 1, \end{aligned} \quad (3.6)$$

$$\begin{aligned} Y(t_1-1) &= f\Big(Z(t_1-1) + \sum_{i=1}^{n} [\,V_i(t_1-1)\,X_i(t_1-1)\,]\Big), \\ &= f(0+0), \\ &= 0, \end{aligned} \quad (3.7)$$

$$\begin{aligned} Y(t_2) &= f\Big(Z(t_2) + \sum_{i=1}^{n} [\,V_i(t_2)\,X_i(t_2)\,]\Big), \\ &= f(0+\varepsilon 0), \\ &= 0, \end{aligned} \quad (3.8)$$

$$\begin{aligned} Y(t_2-1) &= f\Big(Z(t_2-1) + \sum_{i=1}^{n} [\,V_i(t_2-1)\,X_i(t_2-1)\,]\Big), \\ &= f(1+\varepsilon.1), \\ &\cong 1. \end{aligned} \quad (3.9)$$

By substituting Equations 3.6-3.9 in Equation 3.5, we obtain:

$$\begin{aligned} \Delta V_i &\cong c\,\overline{X}_i(t_1)\,[\,1-0\,] + c\,\overline{X}_i(t_2)\,[\,0-1\,], \\ &= c\,[\overline{X}_i(t_1) - \overline{X}_i(t_2)] \end{aligned}$$

Because we have $\overline{X}_i(t_2) > \overline{X}_i(t_1)$, this means that ΔV_i is negative and delay conditioning produces conditioned inhibition.

Another problem is that the Sutton & Barto (1981) model simulates only facilitating CR, i.e. a CR that mimics the UR. Furthermore, experimental data show that the optimal ISI can be longer than the CS duration (Gormezano et al., 1983). The Sutton & Barto (1981) model cannot reproduce such a phenomenon because $\overline{X}_i(t)$ decreases as

soon as CS_t offset occurs Their model also does not explain the following phenomena spontaneous recovery, CS preexposure effects, sensory preconditioning, the shape of the curve of the CR, and inhibition of delay

3.4 The Klopf (1988) Model

Assumptions of Klopf's (1988) Model

The reader can refer to Chapter 1 (Equation 1.12 and the following) for a formal description of the model Klopf's (1988) model is based on the following eight major assumptions:

1- The CS_t comes to elicit the same response as the US This is the stimulus substitution theory of Pavlov (1927) The CR and the UR are added together in what we can call the response.

2- The change in connection weight of the CS_t at a given time is proportional to the product of the change of the CS amplitude by the change of the output response at that time

3- Only positive changes in input CS levels are used in the correlation

4- Instead of correlating approximately simultaneous CS level changes and output response changes, earlier CS level changes should be correlated with later output response level changes

5- The interval between correlated changes in input CS level and changes in output response level ranges from one time step to the maximum effective ISI in delay conditioning

6- The magnitude of an earlier CS level change (at a time t-k) correlated with a later output response level change (at a time t) should be weighted by a factor C_k C_k is an empirically established learning-rate constant that is proportional to the efficacy of conditioning when the ISI is k

7- The change in a connection weight is proportional to the absolute value of the current connection weight.

8- Connection weights are either inhibitory or excitatory, i e connection weights are either negative (Vi_t) or positive (Ve_t)

Major Contributions of the Klopf (1988) Model

Klopf's (1988) model reproduces experimental data for the delay conditioning procedure, as opposed to the Sutton & Barto (1981) model which did not His model also accounts for the initial positive acceleration in the S-shaped acquisition curves in animal learning Klopf's (1988) model reproduces the correct order of magnitude for the different time contingency procedures, i e. delay conditioning is stronger than trace

conditioning, trace conditioning is stronger than simultaneous conditioning, and simultaneous conditioning is stronger than backward conditioning The curve of the asymptotic strength of the CR as a function of ISI is reproduced by the Klopf (1988) model The optimal ISI in his model can be longer than the CS duration, in agreement with experimental data (Gormezano et al , 1983) and as opposed to the Sutton & Barto (1981) model

The model can simulate the CS and US duration effects. The inhibitory aspect of the backward conditioning procedure is predicted by the model. The model also accounts for second-order conditioning. Several other properties are explained. the CS intensity effects, conditioned inhibition, blocking, overshadowing, US intensity effects, and several configural learning procedures.

Problems with Klopf's (1988) Model

I realized that Klopf's (1988) model predicts an extinction rate that varies as a function of the CS duration. This is presented in Figure 3.2. This is very different from the results of my DPC model presented in Chapter 4 (see Figure 4.10). In fact the extinction rate in Klopf's (1988) model decreases steadily as a function of the CS duration and eventually reaches zero This means that the CR of a long CS, i.e. a CS with a duration that is longer than the maximum effective ISI in delay conditioning (i.e. τ), does not extinguish at all An example of such a case is presented in Figure 3.3

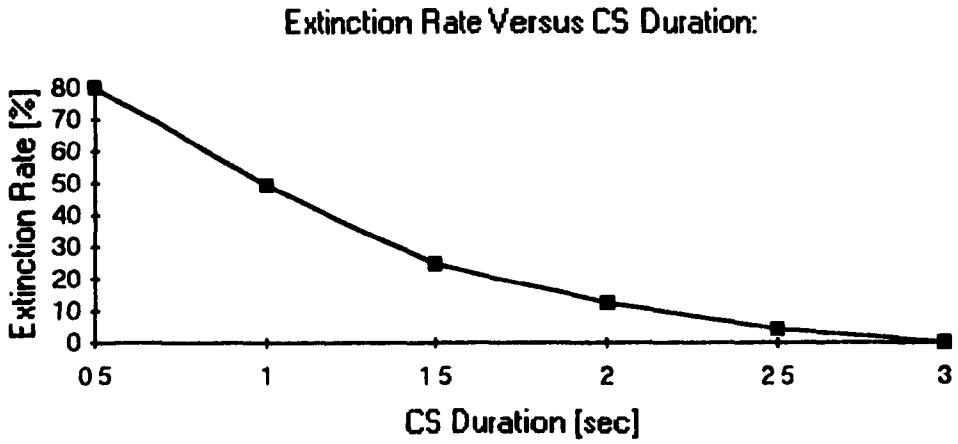

Figure 3.2 The extinction rate predicted by Klopf's (1988) model decreases as a function of the CS duration, the ISI and the US being constant. Extinction rate in this figure is obtained after an acquisition phase and 2 extinction trials. The extinction rate is equal to the ratio of the strength of the CR on the second test trial over the strength of the CR on the first test trial The strength of the CR was the same for the first trial of each CS duration (i.e. 0.6) The acquisition phase had 10 trials for the CS duration of 0.5 sec and 5 trials for the other CS durations.

For example, if we have a CS duration of t_1 and an ISI of t_2, with $t_1 > \tau > t_2$, then there would be acquisition of a CR (a conditioned excitation), because ISI is t_2 which is less than τ. However, during the extinction process, when no US is presented, the decrease of $\Delta V_i(t)$ occurs when $\Delta Y(t) < 0$ which is at the CS offset. If we suppose that the CS onset occured at a time t_0, then at the CS offset t is equal to t_0+t_1 and we have

$$\Delta V_i(t_0+t_1) = \Delta Y(t_0+t_1) \sum_{k=1}^{\tau} C_k \left| V_i(t_0+t_1-k) \right| \Delta X_i(t_0+t_1-k) \qquad (3.10)$$

The positive part of $\Delta X_i(t_0+t_1-k)$ is equal to zero for the whole duration of the CS, except at the CS onset when $t = t_0$, i.e. $\Delta X_i(t_0) > 0$ which is for $k = t_1$. However, C_k is equal to zero for $k = t_1$, because $t_1 > \tau$. Thus $\Delta V_i(t_0+t_1) = 0$, which means that $V_i(t)$ does not change and therefore there is no extinction.

Another problem with Klopf's (1988) model is that "C_j is an empirically established learning-rate constant that is proportional to the efficacy of conditioning when the ISI is j" (Klopf, 1988). However, Klopf (1988) did not specify exactly how the efficacy of conditioning is measured, i.e. he did not specify the CS and US durations used to measure the efficacy of conditioning. The CS and US duration are important because they change the shape of the curve of efficacy of conditioning as a function of the ISI. In fact, "longer optimal ISIs have been observed for long fixed delay CSs than for short trace CSs" (Schneiderman, 1966, cited by Sutton and Barto, 1981). If the maximum value of C_k is at $k = j$, then because each C_k is constant, the optimal ISI is always at $t = j$ time steps. In other words, because each C_k is constant, Klopf's (1988) model cannot reproduce the fact that the optimal ISI varies with CS duration.

Klopf's (1988) model simulates facilitating CRs, i.e. CRs that mimic the UR, but not compensatory CRs. His model also does not explain the following phenomena: spontaneous recovery, CS preexposure effects, sensory preconditioning, the shape of the curve of the CR, and inhibition of delay.

To summarize the above discussion, the Rescorla and Wagner (1972) model is highly efficient; with few equations it simulates many properties of classical conditioning. However, it is not a real-time model and consequently does not reproduce a whole set of properties of classical conditioning. Klopf's (1988) model reproduces the properties accounted for by the Rescorla and Wagner (1972) model plus several real time properties, but it has many equations. The Sutton and Barto (1981) model is intermediate between Rescorla and Wagner's (1972) model and Klopf's (1988) model: it is a real-time model and is efficient but does not account for as many properties as Klopf's (1988) model. Overall, Klopf's (1988) model seems superior to the others.

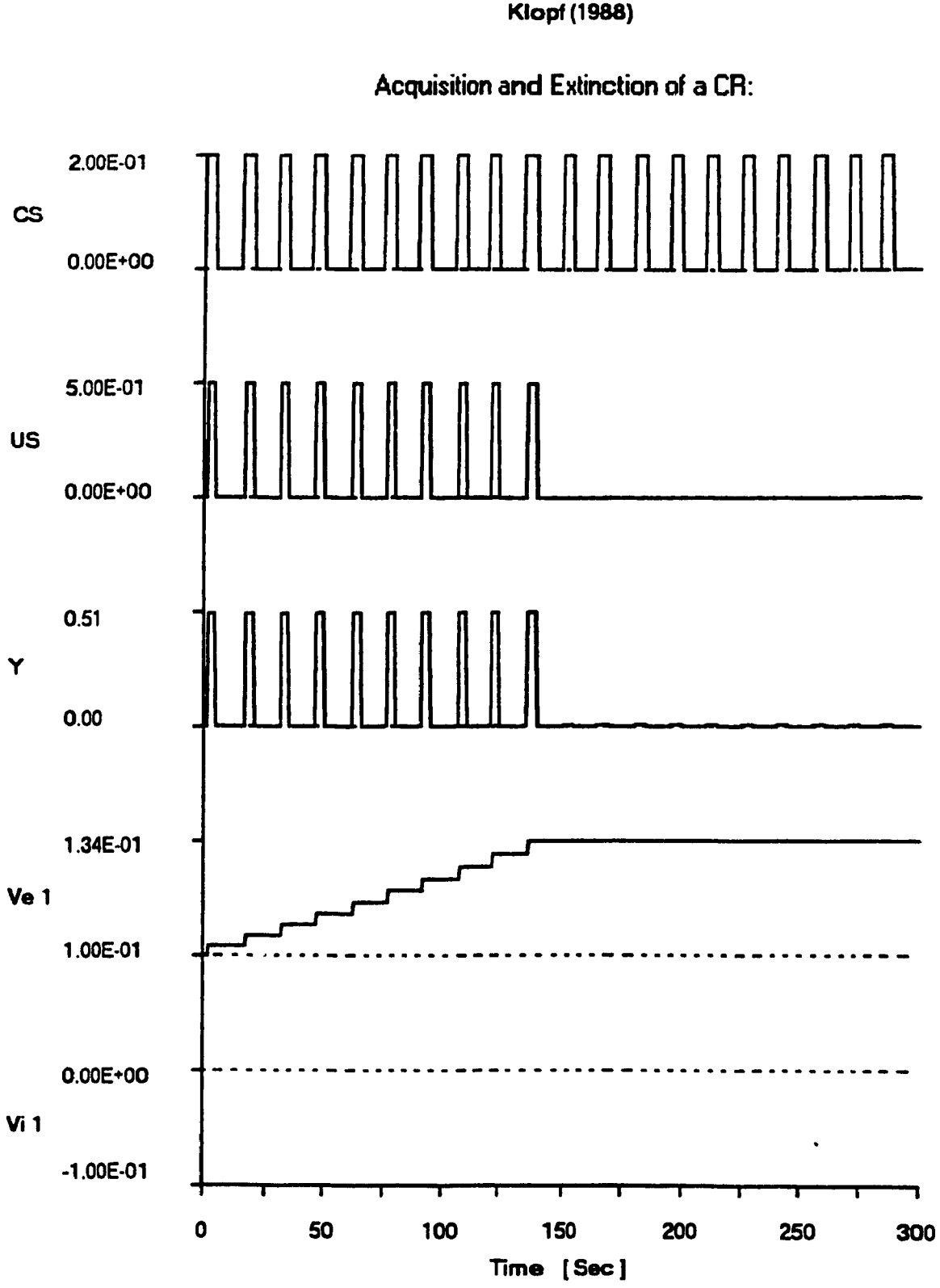

Figure 3.3 A long CS acquires the ability to elicit a CR, but the CR doesn't extinguish. Connection weights (Vi1, Ve1) stay constant when the CS is presented alone.

4. THE DELAY PRODUCING CONNECTIONS MODEL

In this chapter, I develop a real-time model of classical conditioning This model is motivated by the previously described properties and builds upon previous models of classical conditioning There are two major differences between the model presented here and previously mentioned models (Hebb, 1943, Klopf, 1972, 1986, 1988, Rescorla-Wagner, 1972, Sutton-Barto, 1981). The first feature is a change in the activation rule that enables Y(t) to mimic a real CR The second feature is that the connections have the ability to produce delays. After conditioning, this model produces fixed delays between a CS_i onset and the peak of the corresponding CR This delay is fixed as opposed to variable in my later adaptive delays model The model is consequently called "delay-producing connections (or DPC) model" Section 4 2 presents arguments for why these features were introduced

4.1 Formal Description of the Delay-Producing Connections Model

The model can be summarized by the following equations with the notation defined in Table 4.1. The rationale underlying these assumptions is set out in detail after they have been presented.

$$V_i(t) = V_i(t-1) + \Delta V_i(t), \tag{4 1a}$$

$$\Delta V_i(t) = \alpha_i(t-2)\,\beta\,(Y(t)-\overline{Y}(t-1)), \tag{4 1b}$$

$$\alpha_i(t) = \begin{cases} 0 & \text{if } \overline{X}_i(t) < 0, \\ \overline{X}_i(t) & \text{otherwise,} \end{cases} \tag{4 2}$$

$$\frac{d^3\overline{X}_i(t)}{dt} + 3\theta\,\frac{d^2\overline{X}_i(t)}{dt} + 3\theta^2\,\frac{d\overline{X}_i(t)}{dt} + \theta^3\,\overline{X}_i(t) = 2\Lambda\frac{dX_i(t)}{dt}, \tag{4 3}$$

$$Y(t) = f\Big(\lambda(t) + \sum_{i=1}^{n}\,[\,\alpha_i(t)\,V_i(t)]\Big), \tag{4 4}$$

$$f(Z) = \begin{cases} 0 & \text{if } Z < T, \\ Z & \text{if } T \le Z \le M, \\ M & \text{if } Z > M, \end{cases} \tag{4 5}$$

$$\overline{Y}(t) = \beta_B\,\overline{Y}(t-1) + (1-\beta_B)\,Y(t-1), \qquad \text{from (1 10c)}$$

and $\alpha_i(0) = V_i(0) = \overline{X}_i(0) = \overline{Y}(0) = 0 \qquad \forall\, i \in \{1, .., n\}$

For the definitions of the symbols, the reader can refer to Table 4 1

Symbol	Definition
A	is a constant that controls the amplitude of the peak of $\alpha_i(t)$
$\alpha_i(t)$	is the trace of the CS_i at time t
β	is a learning rate parameter related to the US
β_{IB}	is an extinction rate for the trace of Y(t)
$f(Z)$	is the activation function
$\lambda(t)$	is the intensity of the US at time t
M	is the maximum value of Y(t)
T	is a threshold for Y(t) (as Klopf (1988) did, I will take T=0)
T_p	is a delay between the onset of CS_i and the peak of $\alpha_i(t)$ when the CS_i is a step-like input
θ	is a constant fixing the delay between the onset of CS_i and the peak of $\alpha_i(t)$ Normally, $\theta = 2 / T_p$
$V_i(t)$	is the connection weight between the CS_i and the output at time t.
$X_i(t)$	is the amplitude of the CS_i at time t
$\overline{X}_i(t)$	is the virtual trace of the CS_i at time t. When $\overline{X}_i(t)$ is positive it is equivalent to $\alpha_i(t)$, but when $\overline{X}_i(t)$ is negative, it represents an inertia (or a reluctance, or a delay) to produce a trace of the CS_i.
Y(t)	is the response at time t
$\overline{Y}(t)$	is the trace of the response Y(t) at time t.

Definitions of the Variables and Parameters of the DPC Model.

Table 4.1

4.2 Assumptions of the Delay Producing Connections Model

Initial Hypothesis

In accordance with a number of authors (Hebb, 1943, Klopf, 1972,1982,1986; Rescorla-Wagner, 1972, Sutton & Barto, 1981), the initial hypothesis is that the rate of change in connection weight is proportional to the product of a function of the CS_i and a function of the CS_i and the US. Formally, this hypothesis can be expressed as

$$\Delta V_i(t) = f[X_i(t)]\, F[\lambda(t), Y(t), \overline{Y}(t)], \qquad (4.6)$$

where $Y(t) = \lambda(t) + \sum_{i=1}^{n} X_i(t)V_i(t)$, from (1.1)

$\lambda(t)$ is the intensity of the US at time t,

$Y(t)$ is the neuronal output at the time t,

and $\overline{Y}(t)$ is a form of average of the values of $Y(t)$ over the last few time steps $\overline{Y}(t)$ is a function of $Y(t-k)$

In the models we study we have either $\overline{Y}(t) = 0$, $\overline{Y}(t) = \beta_B \overline{Y}(t-1) + (1-\beta_B) Y(t-1)$, or $\overline{Y}(t) = Y(t-k)$ In Hebb's (1943) learning rule, the corresponding functions are $f[X_i(t)] = cX_i(t)$ and $F[\lambda(t), Y(t), \overline{Y}(t)] = Y(t)$ (see Equation 1 3) For Rescorla and Wagner (1972), the functions are $f[X_i(t)]=\alpha_i$ and $F[\lambda(t), Y(t), \overline{Y}(t)] = \beta[2\lambda(t)-Y(t)] = \beta[\lambda(t)-\sum_{i=1}^{n} X_i(t)V_i(t)]$, where α_i is the salience of the CS_i and $\lambda(t)$ is the strength of the US (see Equation 1 7) Klopf's (1972, 1982) model used $f[X_i(t)] = cX_i(t-k)$ and $F[\lambda(t), Y(t), \overline{Y}(t)] = Y(t-k)Y(t)$ (see Equation 1 8), while in Klopf (1986) $f[X_i(t)] = cAX_i(t-k)$ and $F[\lambda(t), Y(t), \overline{Y}(t)] = Y(t)-Y(t-k)$ (see Equation 1 11), taking $\overline{Y}(t) = Y(t-k)$ In his more recent model Klopf (1988) adopts a more complex formulation that cannot be reduced to the form $f[X_i(t)]F[\lambda(t), Y(t), \overline{Y}(t)]$ (see Equation 1 12) Sutton and Barto (1981) have used $f[X_i(t)] = c\,\overline{X}_i(t)$ and $F[\lambda(t), Y(t), \overline{Y}(t)] = [Y(t) - \overline{Y}(t)]$ (see Equation 1 9) taking $\overline{Y}(t) = \beta_B\,\overline{Y}(t-1) + (1-\beta_B)\,Y(t-1)$ (see Equation 1 10b)

In the present context the function $f[X_i(t)]$ can be interpreted as the trace of the CS_i and the function $F[\lambda(t), Y(t), \overline{Y}(t)]$ as the "associability" of the US Associability represents the amount of reinforcement a US can provide at a given moment in time The absolute value of $F[\lambda(t), Y(t), \overline{Y}(t)]$ is linked to the "surprise" caused by the US onset or offset

To have the rate of change of V_i proportional to a function of the amplitude of the CS_i makes sense because: (1) the strength of the CR during acquisition increases with the number of trials (Pavlov, 1927), i e the number of CS_i presentations, (2) more intense CSs have a faster acquisition rate.

In the same way, to have ΔV_i proportional to a function of the US and the CS is logical because: (1) acquisition is faster and the asymptote higher for a stronger US, (2) the level of conditioning on a particular trial is influenced not only by the amount of prior conditioning to the stimulus but also by the level of previous conditioning to other stimuli associated with the US (Kamin, 1969, Rescorla-Wagner, 1972)

The shape of the curve of the CS_i trace

The first question is: what is the shape of the curve of the CS_i trace (i e , $f[X_i(t)]$)? From the properties of classical conditioning, which were reviewed in Chapter 2, several characteristics of the curve of $f[X_i(t)]$ can be deduced In the following, I let $\alpha_i(t) = f[X_i(t)]$. Consequently, the change of connection weight becomes

$$\Delta V_i(t) = \alpha_i(t)\, F[\lambda(t), Y(t), \overline{Y}(t)]. \qquad (4\ 7a)$$

Trace conditioning and backward conditioning are symmetrical situations in terms of the timing relationships between the CS and the US but produce very different results with backward conditioning being weaker than trace conditioning. This leads to the assumption that the trace of a CS (i e , $\alpha_i(t)$) and the associability of a US (i.e., $F[\lambda(t), Y(t), \overline{Y}(t)]$) must have different shapes as a function of time

From the fact that trace conditioning is stronger than backward conditioning, one can deduce two other things. First, the decay of $\alpha_i(t)$ must be slower than the increase of $F[\lambda(t), Y(t), \overline{Y}(t)]$, otherwise $\alpha_i(t)$ and $F[\lambda(t), Y(t), \overline{Y}(t)]$ would not overlap and there would be no trace conditioning Second, the increase of $\alpha_i(t)$ must be slower than the decay of $F[\lambda(t), Y(t), \overline{Y}(t)]$, otherwise $\alpha_i(t)$ and $F[\lambda(t), Y(t), \overline{Y}(t)]$ would overlap more and backward conditioning would be stronger

The position of the peak of $\alpha_i(t)$ must change with the CS duration, because "longer optimal ISIs have been observed for long fixed delay CSs than for short trace CSs" (Schneiderman, 1966, cited by Sutton and Barto, 1981) This could be explained if we suppose that $\alpha_i(t)$ increases slowly and that this increase is diminished by CS offset However, $\alpha_i(t)$ must continue to increase even after CS offset because often the optimal ISI in a trace conditioning procedure is longer than CS duration For example, with the nictitating membrane reflex (NMR) of rabbits, a CS of 50 msec with a US of 50 msec has an optimal ISI of aproximately 250 msec (Gormezano et al , 1983).

$\alpha_i(t)$ must decay slowly even when the CS is still present because "a long CS is ignored shortly after it begins, whereas even an instantaneous overt CS causes an internal representation of some significant duration" (Sutton & Barto, 1981).

The shape of the curve of $\alpha_i(t)$ might be analogous to the shape of the curve of the asymptotic strength of the CR as a function of the ISI. This means that $\alpha_i(t)$ would be zero for a certain delay after CS onset, then $\alpha_i(t)$ would slowly increase and reach a peak (in 250 msec for NMR) and then would have an even slower decay. This latter hypothesis has been proposed by Gluck and Thompson (1987) and in a modified form by Klopf (1988) However, we have to be cautious with this hypothesis because acquisition is not only the result of $\alpha_i(t)$, but rather the result of the product of $\alpha_i(t)$ with $F[\lambda(t), Y(t), \overline{Y}(t)]$

On the basis of the foregoing reasoning, the predicted shape of the trace of a CS (i.e , $\alpha(t)$) for a short CS and a long CS is portrayed in Figures 4 1 and 4.2 respectively

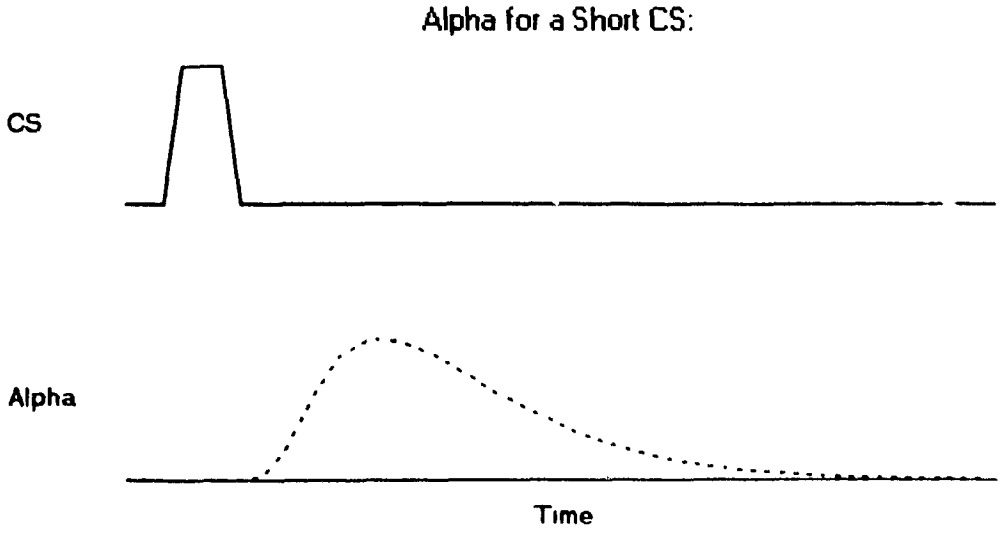

Figure 4.1 The theoretical curve of $\alpha_i(t)$ for a short CS_i $\alpha_i(t)$ continues to increase even after CS_i offset $\alpha_i(t)$ shows a slow increase and an even slower decay

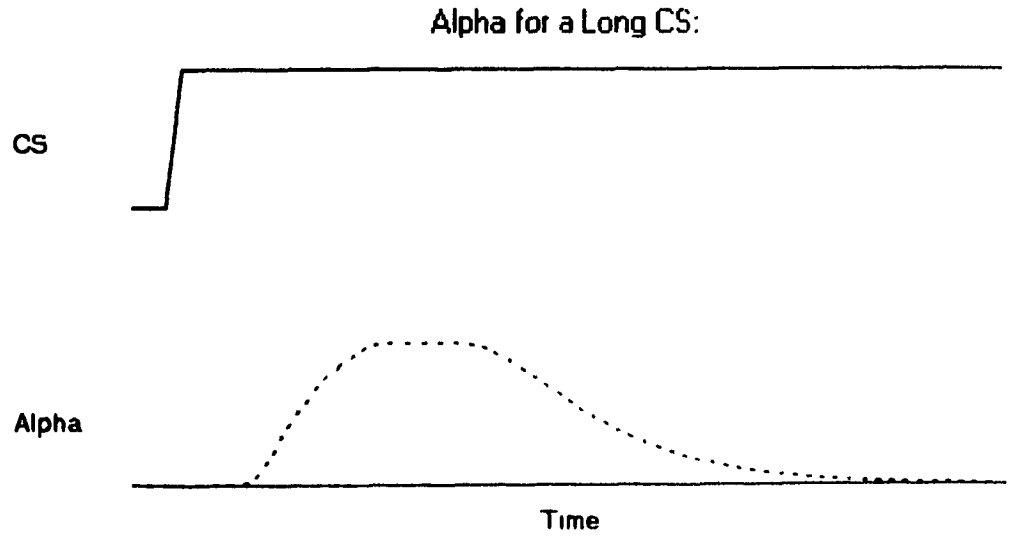

Figure 4.2 The theoretical curve of $\alpha_i(t)$ for a long CS_i The peak of $\alpha_i(t)$ occurs later for a long CS than it does for a short CS_i $\alpha_i(t)$ decreases even when the CS_i is still present.

The Shape of the Associability of a US

Logically, the next question is: what is the shape of the associability of a US (i.e., $F[\lambda(t), Y(t), \overline{Y}(t)]$)? Hebb's (1943) suggestion leads to $F[\lambda(t), Y(t), \overline{Y}(t)] = Y(t)$, with $Y(t) = \lambda(t) + \sum_{i=1}^{n} [X_i(t)\ V_i(t)]$ (see Equation 1.3), Rescorla and Wagner (1972) chose $F[\lambda(t), Y(t), \overline{Y}(t)] = \beta[2\lambda(t)-Y(t)] = [\ \lambda(t) - \sum_{i=1}^{n} X_i(t)\ V_i(t)\ ]$ (see Equation 1.7), Sutton and Barto (1981) set $F[\lambda(t), Y(t), \overline{Y}(t)] = [Y(t)-\overline{Y}(t)]$ where $\overline{Y}(t) = \beta\overline{Y}(t-1) + (1-\beta)Y(t-1)$ (see Equation 1.9), and Klopf (1986, 1988) chose $F[\lambda(t), Y(t), \overline{Y}(t)] = Y(t) - Y(t-1)$ (see Equations 1.11 and 1.12).

There is an advantage in not having the associability of a US ($F[\lambda(t), Y(t), \overline{Y}(t)]$) depend on $\lambda(t)$ explicitly, as opposed to the formulation of Rescorla-Wagner (1972). There is no way for a connection to be "informed" of $\lambda(t)$ other than through the postsynaptic membrane. Thus, it makes more sense that $F[\lambda(t), Y(t), \overline{Y}(t)]$ be solely a function of $Y(t)$ (the sum of all inputs to a neuron) and of $\overline{Y}(t)$, rather than depending on $\lambda(t)$ explicitly as well. It is a coherent way to define a neuron-like unit. Furthermore, an equation of $F[\lambda(t), Y(t), \overline{Y}(t)]$ as a function of a difference between $Y(t)$ and a trace of $Y(t)$ ($Y(t-1)$ or $\overline{Y}(t)$) can take into account the fast increase and fast decay hypothesized earlier for $F[\lambda(t), Y(t), \overline{Y}(t)]$. Therefore, $F[\lambda(t), Y(t), \overline{Y}(t)] = F[0, Y(t), \overline{Y}(t)] \equiv F(\Delta Y(t))$ seems a reasonable choice.

Taking $Y(t)$ as the response is equivalent to adopt the stimulus substitution theory of Pavlov (1927), i.e. the CR mimics the UR. However, there is a problem with the definition of $Y(t)$. $Y(t)$ is the response that follows the stimulus inputs $\lambda(t)$ and $X_i(t)$. When $\lambda(t)=0$ and $X_i(t)\neq 0$, $Y(t)$ is the CR. When $\lambda(t)\neq 0$ and $X_i(t)=0$, then $Y(t)$ is the UR. $Y(t) = f\left(\sum_{i=1}^{n} [X_i(t)\ V_i(t)]\right)$ is the CR, but it does not have the shape of a real experimental CR. Since $X_i(t)$ is generally a square wave, a CR which is equal to $Y(t)$ will also be a square wave, while the typical curve of a CR as a function of time has a skewed right bell shape (Gormezano et al., 1983). In fact, the curve of the CR looks like the hypothesized curve of $\alpha_i(t)$ (see Figure 4.3). This leads me to assume that $Y(t)$ should be modulated by $\alpha_i(t)$. The activation rule that I have been discussing thus far is given by Equation 1.10b of Chapter 1, taking $z(t) = \lambda(t)$, i.e.

$$Y(t) = f\left(\lambda(t) + \sum_{i=1}^{n} [\ X_i(t)\ V_i(t)]\right). \qquad \text{from (1.10b)}$$

Replacing $X_i(t)$ by $\alpha_i(t)$ in Equation 1.1, results in

$$Y(t) = f\left(\lambda(t) + \sum_{i=1}^{n} [\ \alpha_i(t)\ V_i(t)]\right). \qquad (4.4)$$

$F[\lambda(t), Y(t), \overline{Y}(t)]$ keeps the same notation, only the definition of $Y(t)$ changes The CR becomes equal to $f\left(\sum_{i=1}^{n} \alpha_i(t)\, V_i(t)\right)$ ($\lambda(t) = 0$ because $\lambda(t)$ corresponds to the US which is not usually present when testing for the CR) and has a skewed bell shape as long as $\alpha_i(t)$ retains this shape and as long as $f(Z)$ is not operating at saturation $f(Z)$ is not operating at saturation if $0 < f(Z) < M$, where M is the maximum value of $f(Z)$ This would be coherent with Klopf's (1988) definitions of the variables $Y(t)$ and $X_i(t)$ For Klopf (1988), $Y(t)$ and $X_i(t)$ represent firing frequencies. This means that they can take on a wide range of values (between 0 and about 300 Hz), not only the values 0 or 1 as in many other models (e.g., Sutton-Barto, 1981) A definition of $f(Z)$ that would be consistent with my previous assumptions would be the form given in Equation 4 5, namely:

$$f(Z) = \begin{cases} 0 & \text{if } Z < T, \\ Z & \text{if } T \le Z \le M, \\ M & \text{if } Z > M \end{cases}$$

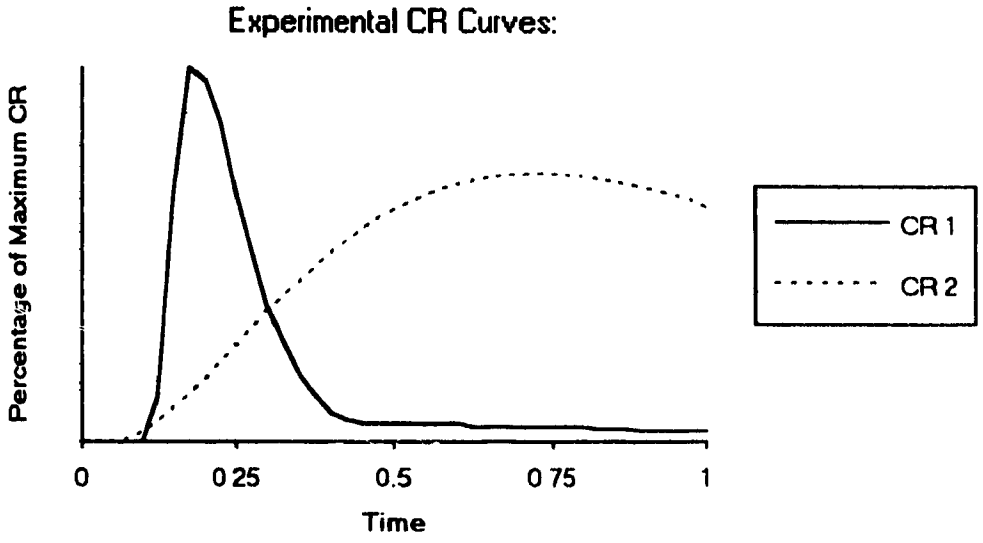

Figure 4.3 Approximative curves of experimental CRs These CRs are of the nictitating membrane reflex in rabbits. CR_1 illustrates an ISI of 200 msec while CR_2 corresponds to an ISI of 750 msec These curves are based on Gormezano *et al.* (1983)

Now that the curve of $Y(t)$ is more clearly determined, it is possible to give a more precise definition of the associability of a US: $F[\lambda(t), Y(t), \overline{Y}(t)] \equiv F(\Delta Y(t))$ What definition of $\Delta Y(t)$ should we adopt. $\Delta Y(t) = Y(t) - \overline{Y}(t)$ (Sutton-Barto, 1981) or $\Delta Y(t) = Y(t) - Y(t-1)$ (Klopf, 1988)? At this point the equation for $\Delta V_i(t)$ is $\Delta V_i(t) = \alpha_i(t) F(\Delta Y(t))$ In a backward conditioning procedure the US offset occurs before the CS onset $\Delta Y(t)$

becomes negative after the US offset (because $Y(t)<Y(t-1)$) and $\Delta Y(t)$ will subsequently tend toward zero (as $Y(t)$ tends toward zero). After the CS onset $\alpha_i(t)$ slowly increases. If $\Delta Y(t)$ stays negative for too short a period of time, $\alpha_i(t)$ would be very small, the product $\alpha_i(t)F(\Delta Y(t))$ would also be very small, and $\Delta V_i(t)$ would not change much. If $\Delta Y(t)$ is equal to $(Y(t)-Y(t-1))$, $\Delta Y(t)$ would be negative for only one time step after US offset, which is too short for $\alpha_i(t)$ to be noticably greater than zero. By choosing $\Delta Y(t)$ equal to $(Y(t)-\overline{Y}(t))$, the amount of time that $\Delta Y(t)$ stays negative can be controlled through $\overline{Y}(t)$. Thus, we will take $\Delta Y(t) = Y(t)-\overline{Y}(t)$ and $F(\Delta Y(t)) = \beta\, \Delta Y(t)$. Using also Equation 4.7a, the equation for $\Delta V_i(t)$ then becomes

$$\Delta V_i(t) = \alpha_i(t)\, \beta\, (Y(t)-\overline{Y}(t)), \qquad (4.7b)$$

where β is a learning rate parameter related to the associability of the US (as in the Rescorla-Wagner (1972) model).

Classical conditioning can be seen as a form of causality learning, the CS being the cause and the US being the effect. It makes sense for the change of associative strength to correlate a prior value of the CS with a current value of the US, as did Klopf (1972, 1982, see Equation 1.8). Thus, it would be coherent to take $\alpha_i(t-k)$ instead of $\alpha_i(t)$ in Equation 4.7b. This change implies an even slower increase of the CS_i trace, i.e. $f[X_i(t)] = \alpha_i(t-k)$, which reflects the fact that conditioning is weaker for backward than for simultaneous conditioning, and weaker for simultaneous than for delay and trace conditioning. Thus, we obtain: $\Delta V_i(t) = \alpha_i(t-k)\, \beta\, (Y(t)-\overline{Y}(t))$. However, after a few simulations using $(Y(t)-\overline{Y}(t))$, it appeared that this term did not vary quickly enough, not producing enough inhibition, i.e. extinction was too slow. $(Y(t)-\overline{Y}(t-1))$ gave better results, thus we arrive at

$$\Delta V_i(t) = \alpha_i(t-k)\, \beta\, (Y(t)-\overline{Y}(t-1)) \qquad (4.8)$$

What value of k should be used? The process of extinction of a CR with the delay producing model is different from other real-time models (e.g., Klopf, 1988; Sutton & Barto, 1980). In fact, in the first part of the CR, when $\Delta\alpha_i(t)$ is positive, $\Delta Y(t)$ is positive and $V_i(t)$ increases. However, in the second part of the CR, when $\Delta\alpha_i(t)$ is negative, $\Delta Y(t)$ is negative and $V_i(t)$ decreases. The mechanism that insures that $V_i(t)$ decreases more in the second part than it has increased in the first part is the presence of the -k in Equation 4.8. During extinction the US is absent, so $\lambda(t) = 0$ and $Y(t)$ depends only on $\sum_{i=1}^{n} \alpha_i(t)V_i(t)$. The number of time steps where $\Delta V_i(t)$ is greater than zero (i.e. the number of time steps where $(Y(t)-\overline{Y}(t-1))$ is greater than zero) is reduced by k time steps (because $\alpha_i(t-k) = 0$ for k more time steps than with $\alpha_i(t)$, when $(Y(t)-\overline{Y}(t-1)) > 0$). However, the number of time steps where $\Delta V_i(t)$ is smaller than zero stays the same with

$\alpha_i(t-k)$ or $\alpha_i(t)$ (because $\alpha_i(t-k) > 0$ for the whole period when $(Y(t)-\bar{Y}(t-1)) > 0$). This insures more decrease than increase, unless $\alpha_i(t)$ or β is too large. I tried the value k = 1 and the model was satisfactory but β had to be small (around 0.2) in order to produce extinction and conditioned inhibition. Such a small value of β produces a slow acquisition. Then I tried k=2 and this produced a more stable extinction for an higher value of β (around 0.6). Therefore, I adopt k = 2.

An Equation for the Trace of a CS

At this point the trace of the CS_i (i.e., $f[X_i(t)]$) and the associability of a US (i.e., $F[\lambda(t), Y(t), \bar{Y}(t)]$) depend on $\alpha_i(t)$. The problem is: what is the equation of $\alpha_i(t)$? Gluck and Thompson (1987) have, in their stochastic model, a plasticity function $\Phi(t)$ that is very much like our deterministic $\alpha_i(t)$. They used the equation $\Phi(t) = T(t)(1-T(t))$, where T(t) is a function that is zero at t = 0, but which jumps to 1 when a CS action potential is generated, and then exponentially decays. Thus, after an action potential is generated $\Phi(t)$ is

$$\Phi(t) = e^{-\theta(t-t_0)}(1-e^{-\theta(t-t_0)}) \qquad \text{for} \quad t \geq t_0, \tag{4.9}$$

where t_0 is the time of the last action potential. This equation is not ideal for $\alpha_i(t)$, aside from the fact that the connection has to "memorize" the time t_0 by some mysterious mechanism. The curve of an experimental CR has another property that is not included in $\Phi(t)$: close to the CS onset, the CR is positively accelerating. The slope of the CR is zero and then slowly increases (see Figure 4.3) (Gormezano et al., 1983). However, Equation 4.9 does not have a positively accelerating curve in the neighborhood of $t = t_0$, as can be seen in Figure 4.4.

Another curve that is analogous in shape to Equation 4.9, is $\alpha_i(t) = t\,e^{-\theta t}$, but as can be seen in Figure 4.5, it is also not positively accelerating in the neighborhood of t = 0.

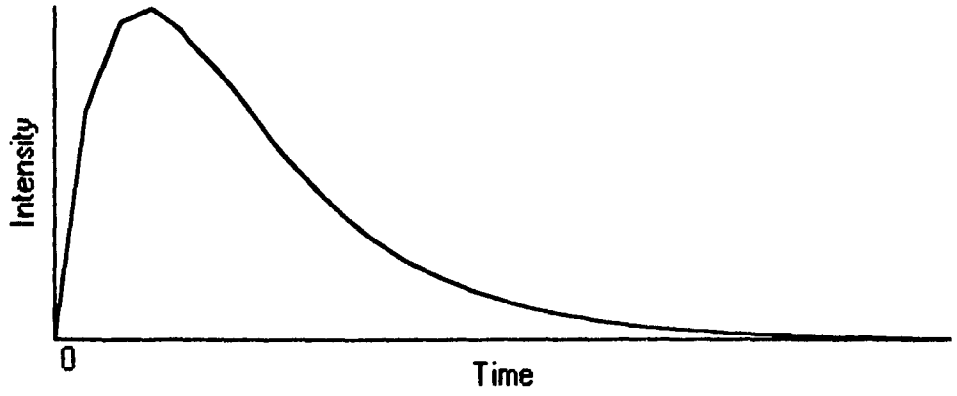

Figure 4.4 The graph of $\alpha_i(t) = e^{-0t}(1 - e^{-0t})$

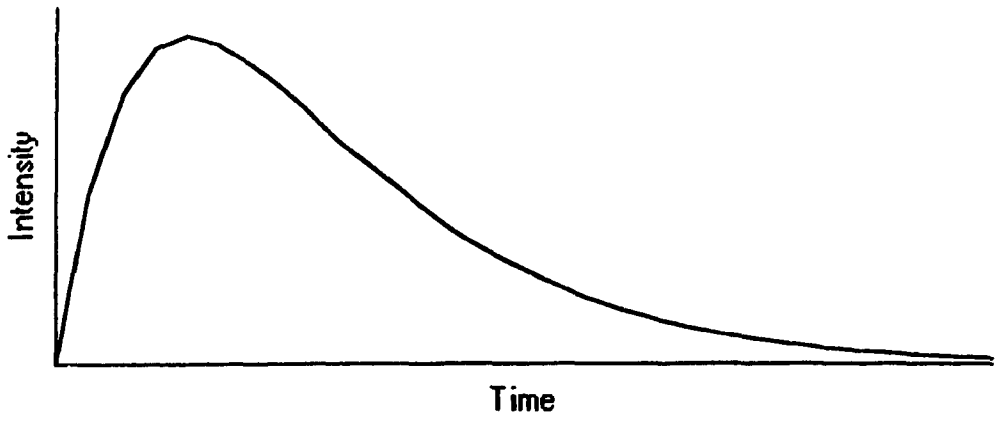

Figure 4.5 The graph of $\alpha_i(t) = t\,e^{-\theta t}$

The simplest equation that fulfills the specifications for $\alpha_i(t)$ and is positively accelerating just after CS_i onset, is:

$$\alpha_i(t) = t^2 e^{-\theta t} \qquad (4.10)$$

(See Appendix A for other mathematical reasons to use Equation 4.10.) Figure 4.6 illustrates Equation 4.10. T_p is defined as the delay between CS_i onset and the peak of $\alpha_i(t)$. Note that $T_p = 2/\theta$.

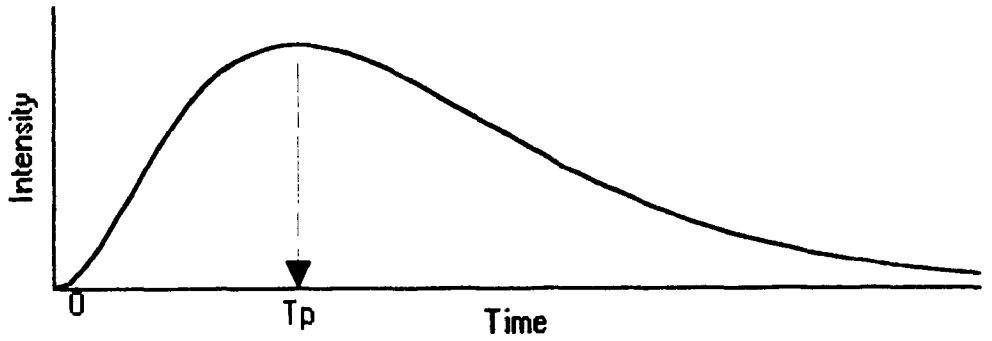

Figure 4.6 The equation $\alpha_i(t) = t^2 e^{-\theta t}$ is positively accelerating in the neighborhood of $t = 0$. There is a delay of $Tp = 2/\theta$ between the onset of the CS_i and the peak of $\alpha_i(t)$, for a step-like CS_i.

The next step is to represent $\alpha_i(t)$, the trace of the CS_i, by a differential equation. The use of a differential equation makes the system more physically realistic because a connection only has to "memorize" information regarding the last few time steps (e.g., $\alpha_i(t-1)$, $\dot{\alpha}_i(t-1)$, and $\ddot{\alpha}_i(t-1)$) instead of "memorizing" t_0 for an indefinite period

of time as with Equation 4 9 Appendix A contains the demonstration of a third-order differential equation, whose output is equal to $At^2e^{-\theta t}$ for a step-like input $X_i(t)$ This equation is the following.

$$\frac{d^3\overline{X}_i(t)}{dt} + 3\theta \frac{d^2\overline{X}_i(t)}{dt} + 3\theta^2 \frac{d\overline{X}_i(t)}{dt} + \theta^3 \overline{X}_i(t) = 2A \frac{dX_i(t)}{dt} \qquad (4\ 3)$$

In Equation 4 3, A is a constant that controls the amplitude of the peak of $\alpha_i(t)$ and $\overline{X}_i(t)$ is the trace of the CS_i When $\overline{X}_i(t)$ is positive it is equivalent to $\alpha_i(t)$, but when $\overline{X}_i(t)$ is negative, it represents an inertia (or a reluctance, or a delay) to produce a trace of the CS_i By taking only the positive part of $\overline{X}_i(t)$, we get the following equation for $\alpha_i(t)$

$$\alpha_i(t) = \begin{cases} 0 & \text{if } \overline{X}_i(t) < 0, \\ \overline{X}_i(t) & \text{otherwise} \end{cases} \qquad (4\ 2)$$

The motivation of each component of the model is now completed

4.3 Results of Simulations Using the DPC Model

The DPC Model was applied to various situations in classical conditioning Simulations in this chapter and the next one were executed with the Microsoft Excel 4 0 spreadsheet on an IBM PC compatible computer [1]

Choice of Parameters

The constants in the model were chosen to approximate the nictitating membrane response (NMR) in rabbits The parameters used in the simulations are presented in Appendix C. The NMR was chosen because more data were available on its real-time properties θ was chosen around the optimal ISI and then increased until the simulation reproduced the experimental curve of the asymptotic strength of the CR as a function of the ISI Note that this method is as valid as Klopf's (1988) decision to take values of C_k that fitted the same curve and as valid as Gluck and Thompson's (1987) choice of θ to fit the same curve. Using this process, θ was found to be equal to 9 09 Hz Now that θ is fixed, A can be found by using the following equation from Appendix A

$$A = \frac{\alpha_{max}\theta^2 e^2}{4}. \qquad (A.10)$$

If we want the maximum value of $\alpha_i(t)$ (i.e., α_{max}) to be equal to one, then A is equal to 153. β was chosen with higher values than experimental data would suggest, in order to

[1] I thank Richard S Sutton for the suggestion that I use a spreadsheet to develop the simulations This idea saved me some time

accelerate execution, because of time and computer memory limitations [2] Therefore β was chosen equal to 0 6 β_B controls the damping of the trace of Y(t) ($0 < \beta_B < 1$), $\beta_B = 0$ implies a very small damping ($\bar{Y}(t) = Y(t-1)$), while $\beta_B = 0\ 9$ produces a slow change in $\bar{Y}(t)$ At first, β_B was fixed at zero and then slowly increased to obtain a plausible amount of backward conditioning The final value used for β_B was 0 1 M, the maximum value for Y(t), was chosen high enough to avoid Y(t)=M M is equal to 3. The amplitudes of the CSs are set equal to 1 0 and the intensity of the US is set equal to 1 0 This combination of values for M, the CS amplitudes and US intensity is equivalent to Klopf's (1988) choices for M, he used M = 1, but set the US intensity around 0.5 and the CS amplitudes around 0 2 In this way M is greater than Y(t).

Results

The DPC model predicts a wide range of classical conditioning phenomena, including backward conditioning, delay conditioning and trace conditioning, conditioned and unconditioned stimulus duration and intensity effects, partial reinforcement effects, interstimulus interval effects, second-order conditioning, conditioned inhibition, extinction, blocking, overshadowing, compound conditioning, discrimination learning, overexpectation and superconditioning effects. However, those results are not presented in detail for this model, since they are presented in detail for the adaptive delays model in Chapter 5 which is an extension of the present model. Only the most interesting results are presented in the current chapter The reader can refer to Appendix C for more details on the parameter values used and to Table D 1 in Appendix D for a summary of the properties predicted by the model.

The most interesting characteristics of the model are the following:

1- Its output Y(t) mimics the CR, as can be seen from the result of a simulation in Figure 4.7 The predicted CR is positively accelerating immediately after the CS onset and skewed to the right.

2- The curve of $\alpha_i(t)$, the trace of the CS_i, reproduces the behavior that has been deduced for short and long CSs. (See Figure 4 8.) The peak of $\alpha_i(t)$ is reached more quickly after the onset of CS_i for a short CS_i than for a long CS_i, while $\alpha_i(t)$ decreases for a long CS_i even if the CS_i is still present. This means that the optimal ISI will be smaller for shorter CSs than for longer CSs. This prediction differs from Klopf's model where the constants C_k are independent of the CS duration (see Equation 1.12).

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[2] This is a rather common practice. The examples of simulations provided by Gluck-Thompson (1987) reach an asymptote in only 3 or 4 trials for the acquisition of a CR .

3- With the model presented here, extinction occurs for CSs of any duration Figure 4.9 shows an example of extinction for a CS of 1000 msec, and Figure 4 10 gives the curve of the extinction rate as a function of the CS duration Figure 4 10 is very different from the equivalent curve for Klopf's (1988) model as discussed in Chapter 3 (see Figure 3.2) Klopf's (1988) model predicts no extinction for long CSs, i e for CSs that are longer than τ (the longest ISI over which delay conditioning is effective)

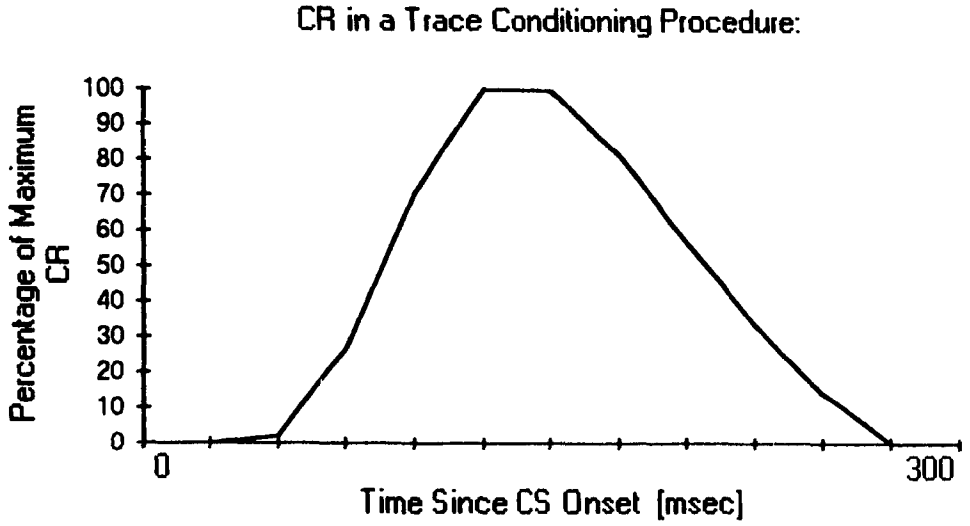

Figure 4.7 The CR obtained with a simulation of the delay-producing connections model for a trace conditioning procedure

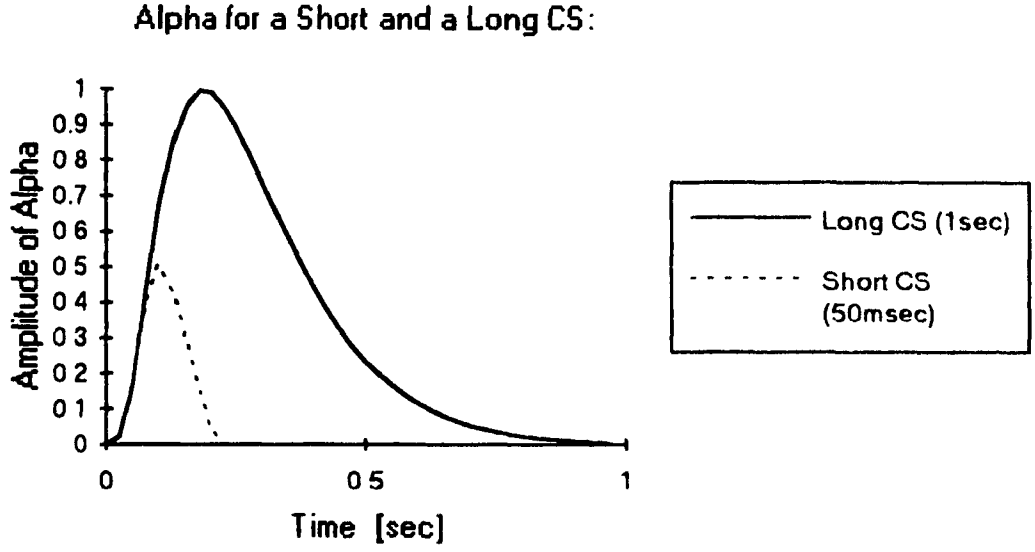

Figure 4.8 The curves of $\alpha_i(t)$, the trace of the CS_i, computed using Equation 4.11 and 4.12 for two different CS durations. The short CS duration is 50 msec. The long CS duration is 1000 msec The peak of $\alpha_i(t)$ for a short CS occurs before the peak of $\alpha_i(t)$ for a long CS. $\alpha_i(t)$ is decreasing for the long CS even when the long CS is still present.

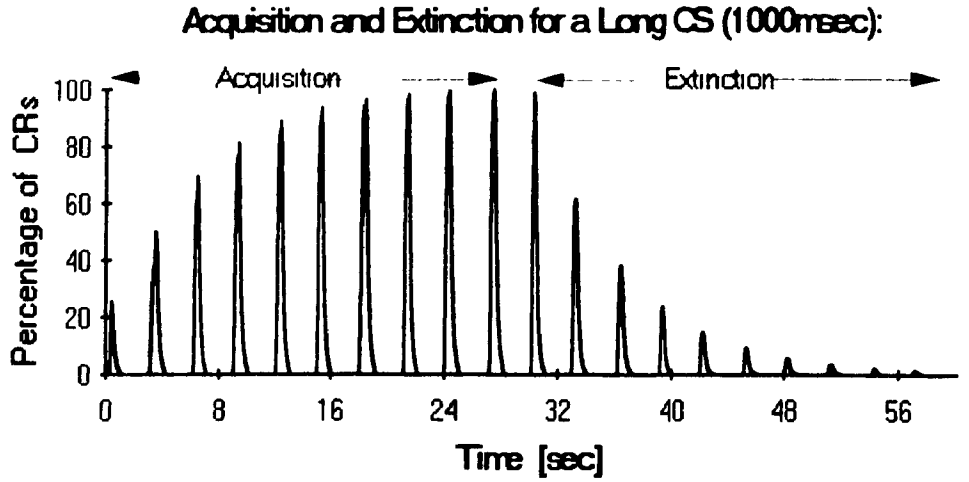

Figure 4.9 Acquisition and extinction with a CS that lasts for 1 sec. Extinction is rapid even if CS duration is long. This simulation was done in a delay conditioning procedure The ISI was 250 msec, the intertrial interval (ITI) was 3 sec.

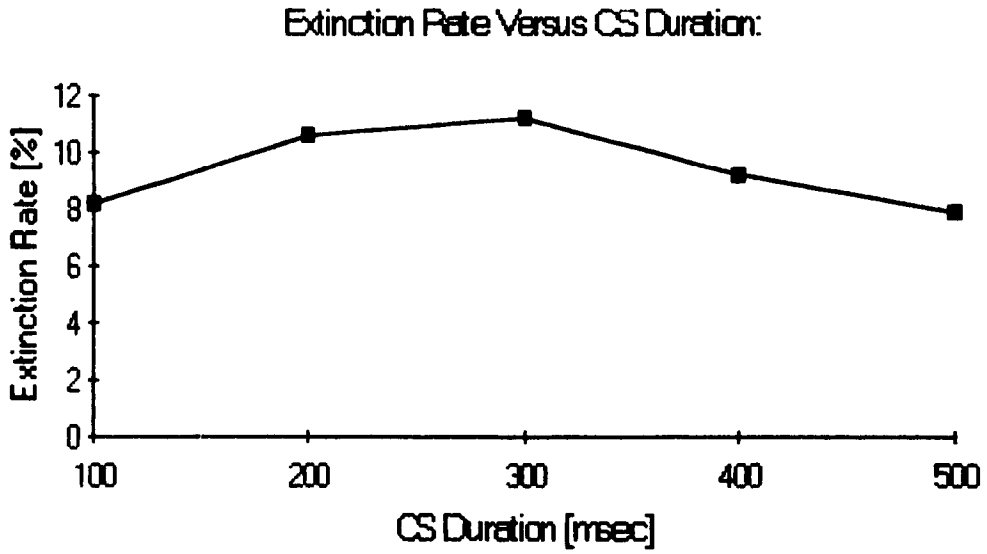

Figure 4.10 Extinction rate as a function of CS duration, the ISI and the US being constant The extinction rate in this figure is obtained after an acquisition phase and 2 extinction trials The extinction rate is equal to the ratio of the strength of the CR on the second test trial over the strength of the CR on the first test trial. The acquisition phase had 10 trials for the CS duration of 100 msec, 3 trials for the CS duration of 500 msec, and 5 trials for the other CS durations CRs were of equivalent amplitude.

4- Finally, by choosing θ appropriately, the model reproduces the relationship between the asymptotic strength of the CR and the ISI, as depicted in Figure 4.11

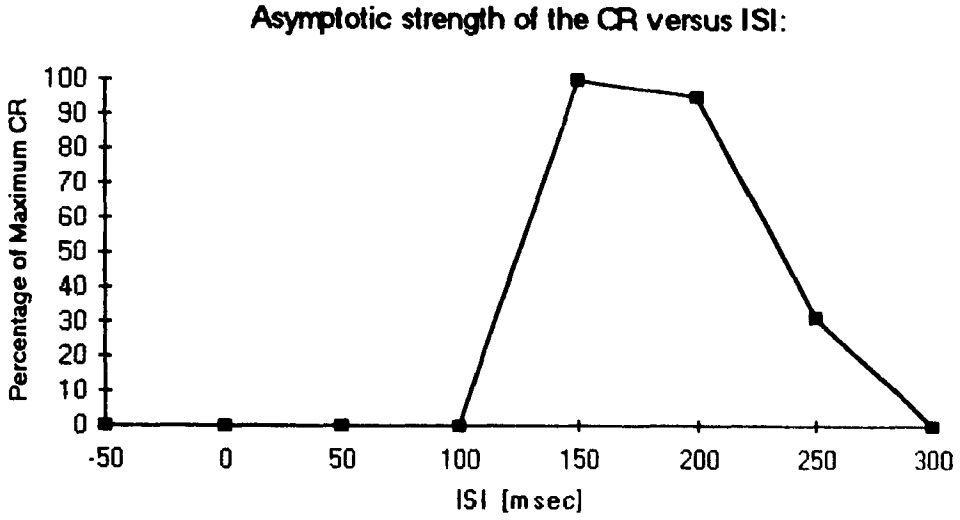

Figure 4.11 The strength of the CR as a function of the ISI in a trace conditioning procedure, as obtained with the delay-producing connections model

Inhibition of Delay

Although these results are interesting, there is a problem. With experimental CRs, the shape of the CR changes during training (Gormezano et al , 1983, Pavlov, 1927) With a long ISI the peak of the CR moves during training toward the US onset At the end of the training period, the peak of the CR is at the US onset This phenomenon is known as inhibition of delay Inhibition of delay could be the result of an "emergent property" of a network of several units, where each unit behaves according to some learning rule. In fact, the most prevalent explanations for inhibition of delay, among theorists, involve real-time neural models These models use delay lines (Desmond, 1990, Desmond & Moore, 1988, Desmond & Moore, 1991, Grossberg & Schmajuk, 1989; Sutton & Barto, 1990) and assume that each CS generates a cascade of stimulus elements $X_i(t)$, each with its own time course The associative strength $V_i(t)$ of each element is proportional to its intensity $X_i(t)$ at US onset. The CR is the result of the sum across the products for each element ($\sum_{j=1}^{n}$ [$X_j(t)$ $V_j(t)$]) at a time t Therefore, the result of that sum is a CR with a peak at US onset

To explain how delay lines operate in real brains, we could invoke transmission delays through the nerve cells Action potentials travel along a nerve at a speed up to 120 meters/sec (Kuffler, Nicholls, & Martin, 1984) To create a delay of one second, we would need 120 meters of nerves However, the chemical transmission at the level of the synapse is slower than the electrical transmission A transmission delay of 0 5 to 1.0 msec is associated with a chemical synaptic transmission (Kuffler, Nicholls, & Martin, 1984) It would therefore take 1000 to 2000 synaptic transmissions to generate a delay of one second These numbers are not impossible, but they suggest that the explanation of delays is not likely to be related to transmission delays

Instead, delays could be due to activation delays. By activation delay, I mean the amount of time a cell takes to reach its maximum firing frequency This amount of time could be of the order of several msec and is therefore a plausible source of delays in the brain The delay-producing connections model can be extended to include a mechanism analogous to delay lines If we have a random distribution of θ (a different θ_{ij} for each connection), each connection would have its own time course. A set of such connections would reproduce the effects of a delay line. This last hypothesis deserves to be investigated, but for now I will investigate in another direction, which is to assume that θ is modified during training

5. THE ADAPTIVE DELAYS MODEL

The following chapter describes an extension of the DPC model of Chapter 4 This extension was developed to provide an explanation of inhibition of delay and CS preexposure effects Inhibition of delay can be seen as an emergent property of a network that would use the DPC model, but the adaptive delays model is an attempt to explain inhibition of delay at the level of connections To do so, delays of variable lengths between the CS onset and the US onset were introduced In other words, the output delay at a connection i adapts to the input delay between the CS_i and the US These characteristics led me to choose the name "adaptive delays (or AD) model" A decrease of the amplitude of $\alpha_i(t)$, as a result of nonreinforced CS presentations, was introduced to explain the CS preexposure effects Section 5 2 explains the rationale for these changes

5.1 Formal Description of the Adaptive Delays Model

The model can be summarized by the following equations

$$V_i(t) = V_i(t\text{-}1) + \Delta V_i(t), \tag{4 1a}$$

$$\Delta V_i(t) = \alpha_i(t\text{-}2)\, \beta\, (Y(t)\text{-}\overline{Y}(t\text{-}1)), \tag{4 1b}$$

$$\alpha_i(t) = \lfloor \overline{X}_i(t) \rfloor, \qquad \text{from (4 12)}^1$$

$$\lfloor Z \rfloor = \begin{cases} z & \text{if } z > 0, \\ 0 & \text{otherwise,} \end{cases} \tag{5 1}$$

$$\frac{d^3 \overline{X}_i(t)}{dt} + 3\theta_i \frac{d^2 \overline{X}_i(t)}{dt} + 3\theta_i^2 \frac{d\overline{X}_i(t)}{dt} + \theta_i^3 \overline{X}_i(t) = 2A\theta_i^{\prime\prime} \frac{dX_i(t)}{dt}, \tag{5 2}$$

$$\Delta\theta_i(t) = -h\theta_i(t)\alpha_i(t) + (s/\Delta t)\, \theta_i(t)\, [\alpha_i(t\text{-}2) - \alpha_i(t\text{-}3)]\, \lfloor \Delta Y(t) \rfloor^2, \tag{5 3}$$

$$Y(t) = f\Big(\lambda(t) + \sum_{i=1}^{n} [\alpha_i(t)\, V_i(t)]\Big), \tag{4 4}$$

$$f(Z) = \begin{cases} 0 & \text{if } Z < T, \\ Z & \text{if } T \le Z \le M, \\ M & \text{if } Z > M, \end{cases} \tag{4 5}$$

$$\overline{Y}(t) = \beta_B\, \overline{Y}(t\text{-}1) + (1\text{-}\beta_B)\, Y(t\text{-}1), \tag{1 10}$$

and $\alpha_i(0) = V_i(0) = \overline{X}_i(0) = \overline{Y}(0) = 0 \qquad \forall\, i \in \{1, \ldots, n\}$

[1] This notation is borrowed from Donegan, Gluck and Thompson (1989)

Symbol	Definition
A	is a constant that controls, with D, the amplitude of the peak of $\alpha_i(t)$
$\alpha_i(t)$	is the trace of the CS_i at time t.
β	is a learning rate parameter related to the US
β_B	is an extinction rate for the trace of Y(t).
D	is a constant that controls the decrease of the amplitude of the peak of $\alpha_i(t)$ as a function of habituation.
$f(Z)$	is the activation function
h	is the habituation rate of $\theta_i(t)$
$\lambda(t)$	is the intensity of the US at time t.
M	is the maximum value of Y(t)
s	is the sensitization rate of $\theta_i(t)$
T	is a threshold for Y(t) (as Klopf (1988) did, I will take T=0).
T_p	is a delay between the onset of the CS_i and the peak of $\alpha_i(t)$ when the CS_i is a step-like input
$\theta_i(t)$	is a constant fixing the delay between the onset of CS_i and the peak of $\alpha_i(t)$. Normally $\theta_i(t) = 2 / T_p$.
$V_i(t)$	is the connection weight between the CS_i and the output at time t.
$X_i(t)$	is the amplitude of the CS_i at time t.
$\overline{X}_i(t)$	is the virtual trace of the CS_i at time t. When $\overline{X}_i(t)$ is positive it is equivalent to $\alpha_i(t)$, but when $\overline{X}_i(t)$ is negative, it represents an inertia (or a reluctance, or a delay) to produce a trace of the CS_i
Y(t)	is the response at time t.
$\overline{Y}(t)$	is the trace of the response Y(t) at time t

Definitions of the Variables and Parameters of the Adaptive Delays Model.

Table 5.1

Note that if h = 0 and s = 0, then $\Delta\theta_i(t) = 0$, $\theta_i(t)$ is constant and the AD model is equivalent to the delay producing connections model. In other words, the DPC model is a special case of the AD model.

5.2 Assumptions of the Adaptive Delays Model

An evolving $\alpha_i(t)$

Wagner (1976), Mackintosh (1975), Pearce and Hall (1980) have suggested that the associativity of a CS_i (the equivalent of the magnitude of $\alpha_i(t)$) changes during conditioning. If we admit that the trace of the CS_i, i.e. $\alpha_i(t)$, changes with training, then the initial hypothesis that $\alpha_i(t)$ is a function solely of the CS_i no longer holds. However, that hypothesis was a first approximation. In this section, I continue to maintain that $\alpha_i(t)$ depends predominantly on the CS_i and hypothesize that the change of $\alpha_i(t)$ is slow. I make that hypothesis because the movement of the peak of the CR, during the process of inhibition of delay, is slow. This new premise does not affect my reasoning concerning the shape of $\alpha_i(t)$, because $\alpha_i(t)$ changes slowly and the premises were primarily based on the hypothesis that the change of connection weight was explained by a product of the two functions $f[X_i(t)]$ (the trace of the CS_i) and $F[\lambda(t), Y(t), \bar{Y}(t)]$ (the associability of a US), rather than resting on the invariability of $f[X_i(t)]$.

The new hypothesis is as follows. When $\lambda(t)=0$, $Y(t)$ is equal to the CR. According to Equation 4.4, $Y(t)$ is modulated by $\alpha_i(t)$ in the following way: $Y(t) = f\left(\lambda(t) + \sum_{i=1}^{n} [\alpha_i(t)\, V_i(t)]\right)$. Since the peak of the CR moves temporally during conditioning, one can hypothesize that it is a change in the position of the peak of $\alpha_i(t)$ which causes this movement. T_p, the position in time of the peak of $\alpha_i(t)$ ($T_p = 2 / \theta$), could change as a result of the habituation and the sensitization processes.

Habituation is a decrease in the strength of a behavioral response that occurs when an initially novel eliciting stimulus is repeatedly presented. The most prevalent physiological explanation of habituation involves a synaptic mechanism (Kandel & Schwartz, 1985), or more precisely a presynaptic mechanism. Work on Aplysia has shown that reduced responsiveness occurs as a result of a long-lasting depression of transmitter release from the terminals of the sensory neurons to the motor neurons. The presynaptic action potential becomes shortened in duration. This gives rise to a weaker inward calcium current and therefore to a smaller amount of transmitter release. Sensitization is the enhancement of an animal's reflex responses as a result of the presentation of a strong stimulus.

Since the CS is by itself a neutral stimulus, it is logical to think that it should be governed by the laws of habituation. This hypothesis is confirmed by CS preexposure effects. CS preexposure effects correspond to the following phenomenon: nonreinforced presentations of a CS prior to reinforced presentations retard subsequent acquisition of the CR. These effects can be explained, in a first approximation, if we assume that $\alpha_i(t)$

habituates to the nonreinforced presentations of the CS_i (i.e. to $X_i(t)$). In other words, the amplitude of $\alpha_i(t)$ would decrease with nonreinforced presentations of the CS_i and this would slow a subsequent acquisition of the CR with reinforced presentations of the CS_i.

When there is a long CS and the US onset is at the end of the CS, since there is no reinforcement at first, $\alpha_i(t)$ would habituate to the CS_i (that is $X_i(t)$). The amplitude of $\alpha_i(t)$ would decrease and the position of its peak would move forward in time in the direction of the US onset (T_p would increase) This hypothesis is summarized in Figure 5.1 Conversely, a sensitization, provoked by the US onset, would change the amplitude of $\alpha_i(t)$ and move the position of its peak toward the US onset. Sensitization would have the effect of increasing or decreasing T_p as well as increasing or decreasing the maximum amplitude of $\alpha_i(t)$.

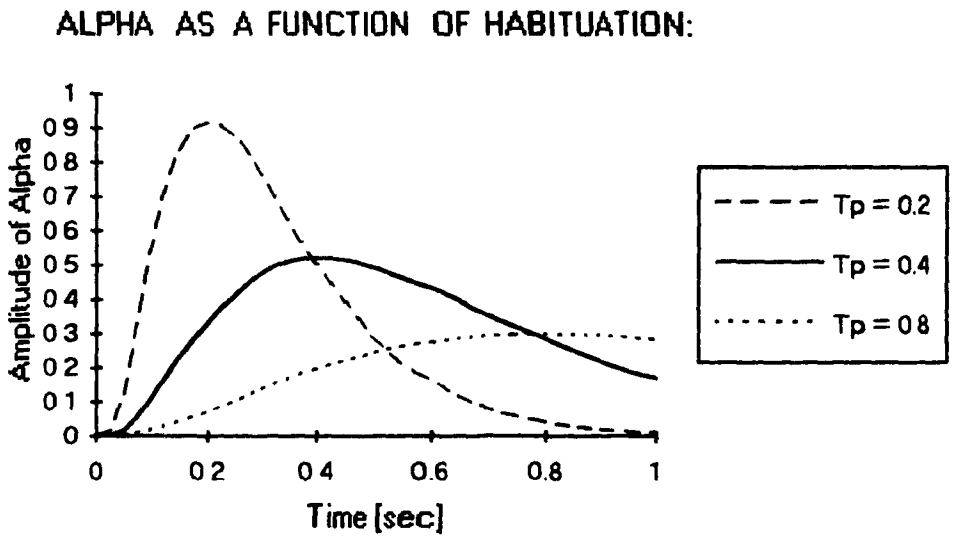

Figure 5.1 The assumed behavior of $\alpha_i(t)$, the trace of the CS_i, as a function of habituation In this example, when the amplitude of $\alpha_i(t)$ goes from around 0.9 to around 0.3, its peak moves from $t = 0.2$ to $t = 0.8$ sec.

The mechanisms of habituation and sensitization that are proposed here may or may not exist at the level of the synapse of real neurons. That is not the point at issue here Even if these mechanisms of modification of T_p do not exist physiologically, this learning rule could offer an interesting way to approximate the behavior of a population of neurons by a single connection. If only for this reason, it is worthwhile pursuing this line of thought. Furthermore, to model the CS preexposure effects and inhibition of delay in a single process is an attractive possibility.

It therefore makes sense to implement habituation and sensitization at the level of the connection weights In fact, habituation and sensitization have been introduced in learning rules by several authors (Gluck & Thompson, 1987, Grossberg & Schmajuk, 1987, Hawkins & Kandel, 1984). Gluck and Thompson in their stochastic model of Aplysia used the following equation for habituation:

(in our notation) $\Delta V_i(t) = -\beta_2 V_i(t) X_i(t)$,

where β_2 is a constant that governs the rate of habituation Grossberg and Schmajuk (1987) use, to simulate habituation, the differential equation $\frac{dy}{dt} = B(1-y) - C S y$ where B, C and S are constants Here again the variation of a variable (y) is proportional to this same variable multiplied by a negative constant In my DPC model, the variable that would show the phenomenon of habituation is θ_i The habituation term will then be $-h\theta_i(t)$, where h is the habituation rate However, habituation should occur only when there is a CS_i This can be obtained if we multiply $-h\theta_i(t)$ by $X_i(t)$ or $\alpha_i(t)$ Should we multiply by $X_i(t)$ or by $\alpha_i(t)$? If we have a very long CS_i, $\alpha_i(t)$ will increase, reach a peak, and then decrease toward zero, while $X_i(t)$ will stay at a constant value during the full duration of CS_i. This means that if we use $X_i(t)$ in the habituation term, a very long CS could provoke a significant decrease of $\theta_i(t)$ in just one trial. This effect would be minimized by using $\alpha_i(t)$; the habituation term then becomes $-h\theta_i(t)\alpha_i(t)$ The detailed explanation for the choice of sensitization term is provided in Appendix B The final expression for the variation of $\theta_i(t)$ is:

$$\Delta\theta_i(t) = -h\theta_i(t)\alpha_i(t) + (s/\Delta t)\,\theta_i(t)\,[\alpha_i(t-2) - \alpha_i(t-3)]\,\lfloor \Delta Y(t) \rfloor^2, \tag{5 3}$$

$$\text{where } \lfloor Z \rfloor = \begin{cases} z & \text{if } z > 0, \\ 0 & \text{otherwise,} \end{cases} \tag{5 1}$$

$$\theta_i(t+1) = \theta_i(t) + \Delta\theta_i(t), \tag{5.4}$$

and s is a sensitization rate constant.

In order to have the amplitude of $\alpha_i(t)$ decrease when T_p increases, i.e. when θ decreases, we replace A by $A\theta^D$ in Equation 4 3. This yields

$$\frac{d^3\overline{X}_i(t)}{dt} + 3\theta_i \frac{d^2\overline{X}_i(t)}{dt} + 3\theta_i^2 \frac{d\overline{X}_i(t)}{dt} + \theta_i^3 \overline{X}_i(t) = 2A\theta_i^D \frac{dX_i(t)}{dt}, \tag{5 2}$$

The maximum amplitude of $\alpha_i(t)$, α_{max}, is determined by Equation A10 (see Appendix A): $\alpha_{max} = A(2/\theta)^2 e^{-2}$ Substituting $A\theta^D$ for A in Equation A10 results in

$$\alpha_{max} = 4A\theta^{D-2} e^{-2}, \tag{5.5}$$

where D is a constant that governs the decrease of the amplitude of $\alpha_i(t)$, the amplitude of $\alpha_i(t)$ being proportional to θ to the power D-2 If D=2, then $\alpha_{max} = 4Ae^{-2}$, and the amplitude of $\alpha_i(t)$ is independent of θ That means that there is no habituation for D=2 If D < 2, then α_{max} increases with increasing T_p Therefore to arrive at an habituation of the amplitude of $\alpha_i(t)$, one must take D > 2 In the following I will assume that D > 2. If Equation 5 5 is expressed as a function of T_p, instead of θ, things are clearer. Substituting $\theta = 2 / T_p$ in Equation 5 5, we obtain

$$\alpha_{max} = \frac{4(2^{D-2})A\ e^{-2}}{T_p^{D-2}} \qquad (5.6)$$

From Equation 5.6 it is easy to see that if T_p increases, α_{max} decreases (provided that D>2).

Because T_p adapts itself to the delay between the onset of the CS_i and the onset of the US, the model is called *adaptive delays model.*

5.3 Results of Simulations Using the Adaptive Delays Models

The AD model was applied to a series of situations in classical conditioning. The results of these simulations are presented below. The final parameter values used are displayed in Appendix C

Choice of Parameters

As in the DPC model, the constants in the model were chosen to approximate the nictitating membrane response (NMR) in rabbits. We can define T_{pmin} as the shortest delay biologically possible between the CS onset and the peak of the CR. T_{pmin} is fixed by the shortest ISI that generates a positive associative strength in trace conditioning In rabbit NMR, the shortest ISI in a trace conditioning procedure that generates conditioning is equal to 100 msec, with a CS of 50 msec and a US of 50 msec. By simulation T_{pmin} was found for this case to be equal to 175 msec. T_{p0}, the initial value for the delay between the CS onset and the peak of $\alpha_i(t)$, was chosen arbitrarily because no data were available to suggest its value It was chosen close to T_{pmin}, but a bit higher in order to accelerate learning in trace conditioning for long ISI (i.e. greater than 250 msec). Δt, the time interval between two time steps, has to be smaller than approximately one quarter of T_{pmin}, otherwise the equation of $\alpha_i(t)$ is unstable. Taking a certain safety margin, Δt was fixed at 25 msec.

The amplitude of $\alpha_i(t)$ is controlled by the parameters A and D (see Equation 5.5). D was selected by approximation, starting at D = 2.0 and increasing until the simulation reproduced the experimental curve of the asymptotic strength of the CR as a

function of the ISI If we choose the maximum amplitude of $\alpha_i(t)$ equal to one, then A can be determined by Equation 5 6 and by using $T_p = T_{pmin}$ in the following way

$$\alpha_{max} = \frac{4(2^{D-2})A\ e^{-2}}{T_{pmin}^{D-2}}. \tag{5 7}$$

$$\text{and so } A = \frac{T_{pmin}^{D-2}\ e^{2}\alpha_{max}}{4(2^{D-2})}, \tag{5 8}$$

With $\alpha_{max} = 1$, $D = 2\ 8$ and $T_{pmin} = 0.175$ sec, this gives $A = 0.26$

The values of h, s and β were chosen by trial and error If one increases the habituation rate h, one must increase the sensitization rate s, otherwise habituation will dominate over sensitization and consequently the amplitude of the CR will decrease even if there is a reinforcement. The sensitization rate s tends to synchronize the peak of $\alpha_i(t)$ with the onset of the US s affects the learning rate because, for a short US, when the peak of $\alpha_i(t)$ arrives at the same time as the onset of the US, $\Delta\alpha_i(t)$ is very small Also the rate of change of $V_i(t)$ is proportional to $\Delta\alpha_i(t)$ for a short US and $\Delta\alpha_i(t)$ is small around the peak of $\alpha_i(t)$ (see Appendix B, Equation B3) Consequently, the faster $\alpha_i(t)$ synchronizes itself with the onset of the US, the faster learning stops The habituation parameter h also affects the learning rate, because it decreases the amplitude of $\alpha_i(t)$ h, s and β were chosen with higher values than experimental data would suggest, in order to accelerate execution, because of time and computer memory limitations [2] I then assume that one trial of simulation corresponds to N experimental trials Unless otherwise specified, in the following simulations h is equal to 0 05, s is equal to 0 03, and β is equal to 0.6. If β is greater than approximately 0.7 the increase of $V_i(t)$ is too fast, and there is no extinction and no conditioned inhibition

β_B controls the damping of the trace of Y(t) ($0 < \beta_B < 1$) $\beta_B = 0$ implies a very small damping ($\bar{Y}(t) = Y(t-1)$) while $\beta_B = 0\ 9$ produces a slow change in $\bar{Y}(t)$ At first, β_B was fixed at zero and then slowly increased to obtain a plausible amount of backward conditioning The final value used for β_B was 0 1 M, the maximum value for Y(t), was chosen high enough to avoid Y(t)=M. M is equal to 3 The amplitudes of the CSs are set to 1 0 and the intensity of the US is also set to 1.0.

[2]As mentioned in Chapter 4, this is a rather common practice. The examples of simulations provided by Gluck-Thompson (1987) reach an asymptote in only 3 or 4 trials for the acquisition of a CR .

Remarks About the Figures

Figures 5 2 to 5 31, for the most part, have the same format. On each figure there are several curves and each curve has its own scale Two curves that are the same size on paper might have different scales The scale of a curve is placed at its left As an example, the notation 9 99E-02 means 9.99×10^{-2}, i.e. 0 099 Alpha and Theta are equivalent notations for $\alpha(t)$ and $\theta(t)$ V1 and V2 are equivalent notations for $V_1(t)$ and $V_2(t)$ When a US and a CS overlap, the CR often resembles two superimposed peaks. Usually, a low, broad peak merges into a high, narrow peak The narrow, high peak should be ignored, it is only a transitory effect. The Sutton-Barto (1980) model and the Klopf (1988) model produce the same transitory effect. This effect could be minimized with a smaller β, but this would slow the acquisition of a CR

Types of Conditioning

The AD model makes it possible to simulate conditioned excitation as well as conditioned inhibition, as can be seen in Figures 5 2 and 5 3 respectively. Figure 5.2 shows the acquisition of a conditioned excitation, while Figure 5.3 shows the acquisiton of a conditioned inhibition A conditioned excitator CS_i has a positive value of associative strength $V_i(t)$, while a conditioned inhibitor has a negative value of $V_i(t)$. In a simple case, when a US starts at a time t_1 and ends at a time t_2, if we have $\alpha_i(t_1\text{-}2) > \alpha_i(t_2\text{-}2)$, this produces excitation and V_i increases If we have $\alpha_i(t_1\text{-}2) < \alpha_i(t_2\text{-}2)$, this produces inhibition and V_i decreases (see a demonstration in Appendix B) The model however only allows simulation of facilitating CRs, i.e. CRs that mimic the UR. This is also the case with the Sutton and Barto (1981) model or Klopf (1986, 1988) models

Acquisition and Extinction

The model exhibits the main properties of the acquistion of a CR. The curve of acquisition is sometimes S-shaped (see Figure 5 2) or is simply negatively accelerating at other times (see Figure 5 4) If the delay between the peak of $\alpha_i(t)$ and the onset of the US is long, then the peak of $\alpha_i(t)$ moves toward the onset of the US (by the hypothesized mechanism of habituation and sensitization). This leads to a positively accelerating acquisition curve However, when the onset of the US is close to the peak of $\alpha_i(t)$, acquisition is already high and there is only a negatively accelerating acquisition curve for the CR All the curves of acquisition of a CR are negatively accelerating at some point and reach an asymptote Partial reinforcement leads to a slower acquisition curve This effect can be seen by comparing Figure 5.4 with Figure 5.5. Both figures represent equivalent delay conditioning procedures, except that Figure 5.4 is a continual reinforcement and Figure 5 5 is a partial reinforcement (the CS is reinforced once out of two presentations). After the same number of US presentations, the conditioned response

in continual reinforcement reaches 0 2, while it reaches 0 08 in the partial reinforcement situation

Extinction is represented in Figure 5 3, 5 4 and 5 5. These figures display a negatively accelerating extinction, where the amplitude of the CRs tends toward zero For smaller CRs, extinction is slower than acquisition For trace conditioning, with a very short CS, $\alpha_i(t)$ is very small and extinction, being proportional to $\alpha_i(t)$, will be very slow Conditioned inhibitors don't extinguish, i e the negative value of $V_i(t)$ stays the same during extinction, as shown in Figure 5 3 This concords with experimental data. Extinction is slow for partial reinforcement when the CR acquired is small

Time Contingencies

The strength of the CR produced by the AD model for the different time contingency paradigms generally follows experimental findings Delay conditioning produces stronger CRs than trace conditioning, as can be seen by comparing Figure 5 6 with Figure 5.7 After 20 trials of delay conditioning the CR reaches 0 28 (see Figure 5 5), while after 20 trials of trace conditioning the CR reaches 0 09 (see Figure 5 7) Simultaneous conditioning, as illustrated by Figure 5 8, leads to inhibition, so trace conditioning is stronger. Simultaneous conditioning is stronger than backward conditioning in the sense that it produces stronger inhibition. Backward conditioning is displayed in Figure 5.9.

The efficacy of delay conditioning is a function of the ISI, as represented in Figure 5 10. The CR is absent for small ISIs, increases rapidly for ISIs between 150 msec and 200 msec, then decreases progressively (the curve doesn't show ISIs greater than 350 msec because the intertrial interval was 1 sec).

The asymptotic strength of the CR as a function of the ISI in a trace conditioning procedure is pictured in Figure 5 11 The magnitude of the CR depends on the ISI There is no CR for a ISI smaller or equal to 50 msec The strength of CRs increases with ISIs between 50 and 150 msec, then the CR decreases with increasing ISIs This is in conformity with experimental results (Gormezano et al., 1983) CS duration influences the asymptotic value of the CR For a CS duration shorter than the optimal ISI, a longer CS produces a stronger CR. If we compare Figure 5 12 and Figure 5 7, both depict the same procedure except that the simulation of Figure 5.7 uses a CS of 50 msec, while the CS of Figure 5.12 lasts for 100 msec. The result is an asymptotic strength of 0 09 for the CS of 50 msec and of 0.40 for the CS of 100 msec.

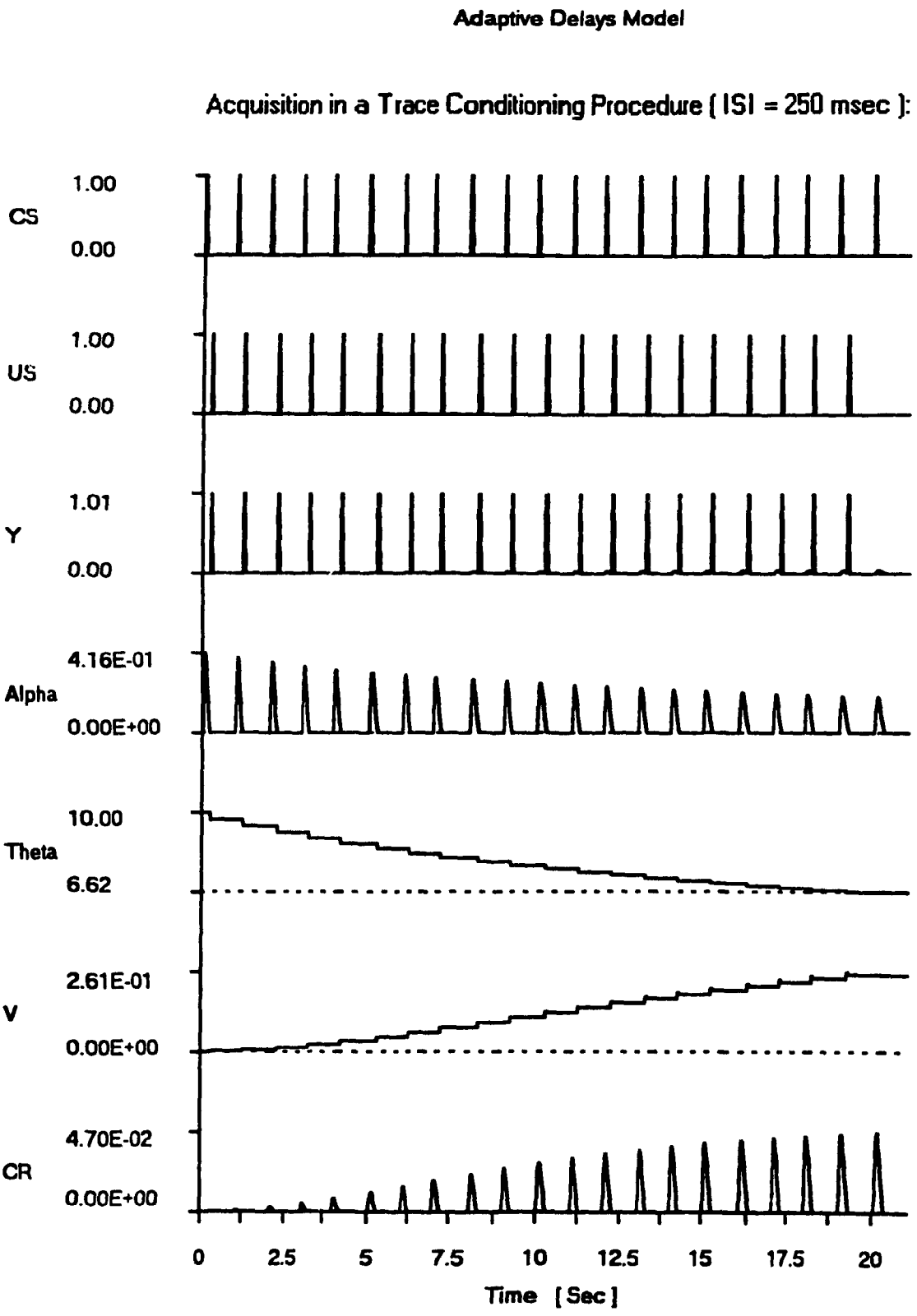

Figure 5.2 The acquisition of conditioned excitation in trace conditioning. The acquisition curve of V and the CR is a sigmoid. Both CS and US last for 50 msec.

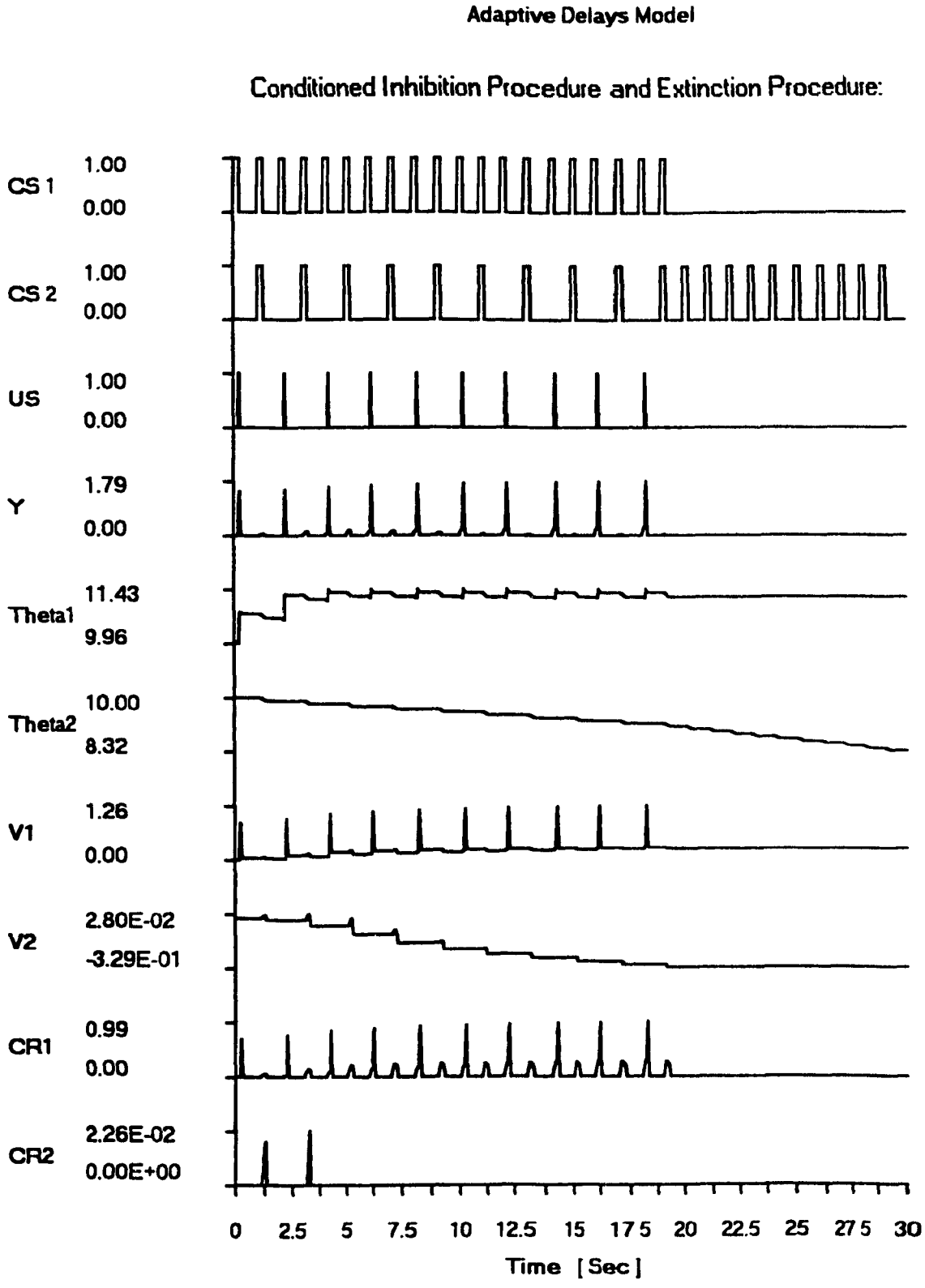

Figure 5.3 The acquisiton of a conditioned inhibition in a delay conditioning procedure. The conditioned inhibition does not extinguish.

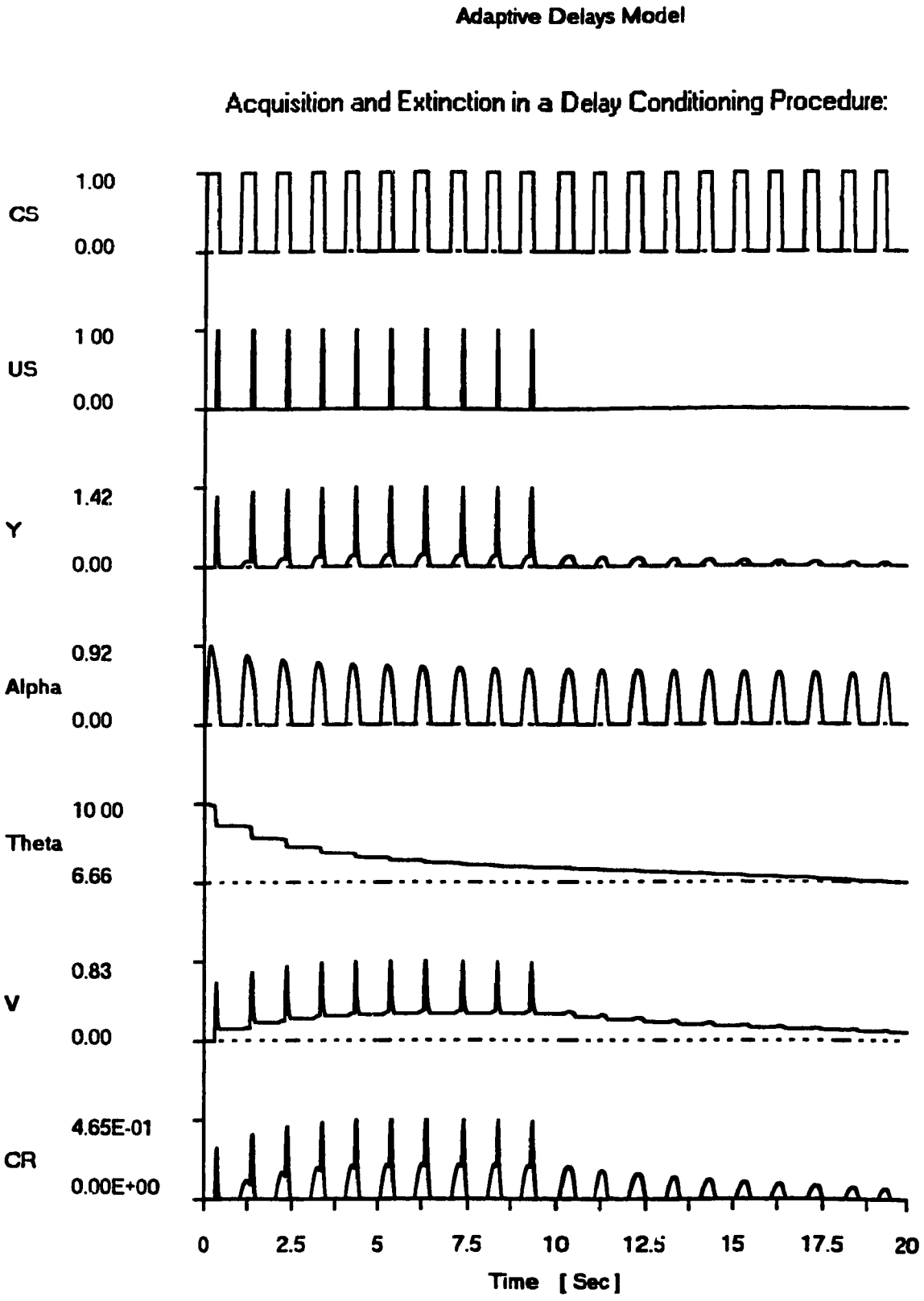

Figure 5.4 The acquisition of a CR is negatively accelerating in this case of delay conditioning. Extinction is also negatively accelerating.

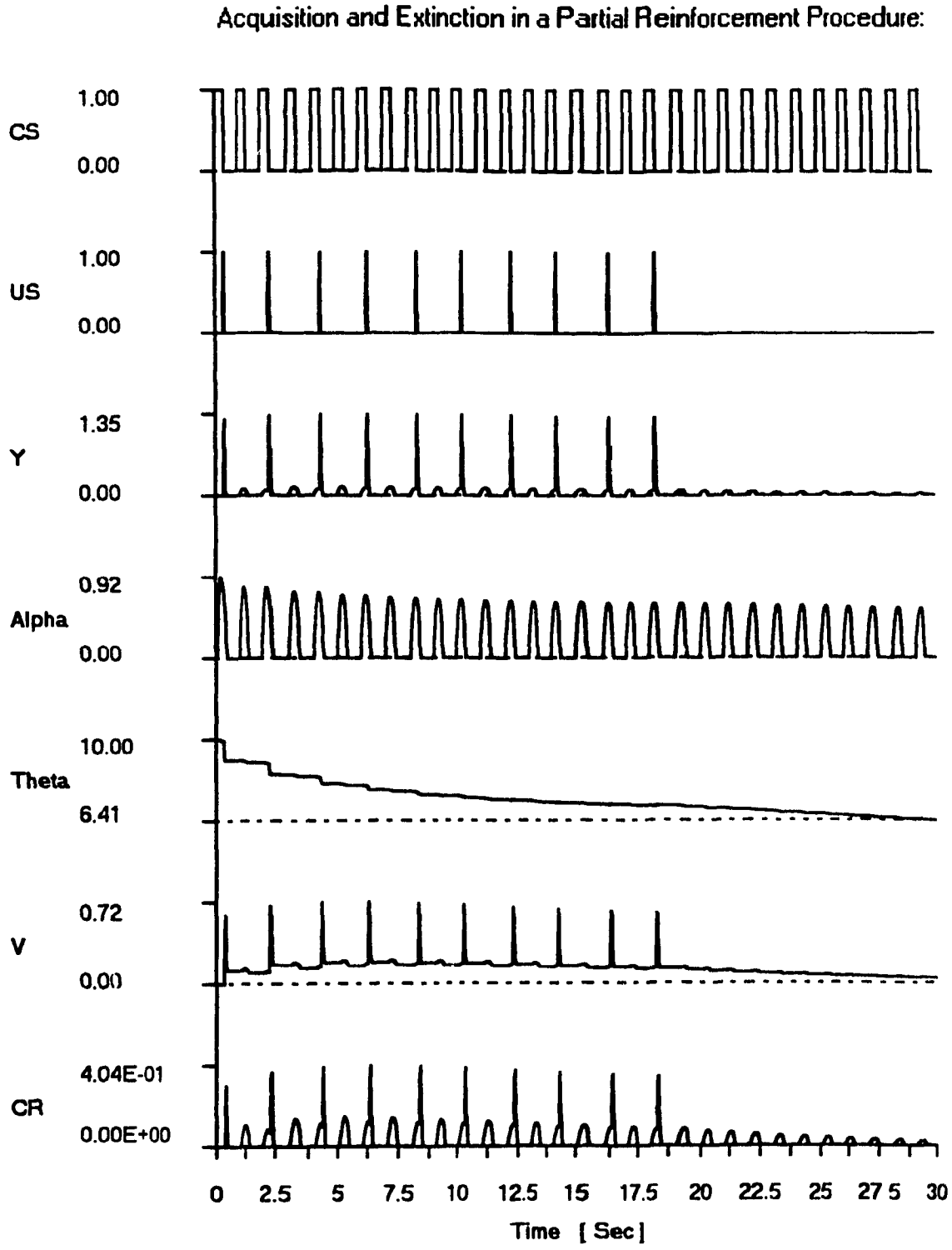

Figure 5.5 In this partial reinforcement, the CS is reinforced once out of two presentations. The CR reaches 0.08 instead of 0.2 in the continual reinforcement situation of Figure 5.4, for the same number of reinforcements.

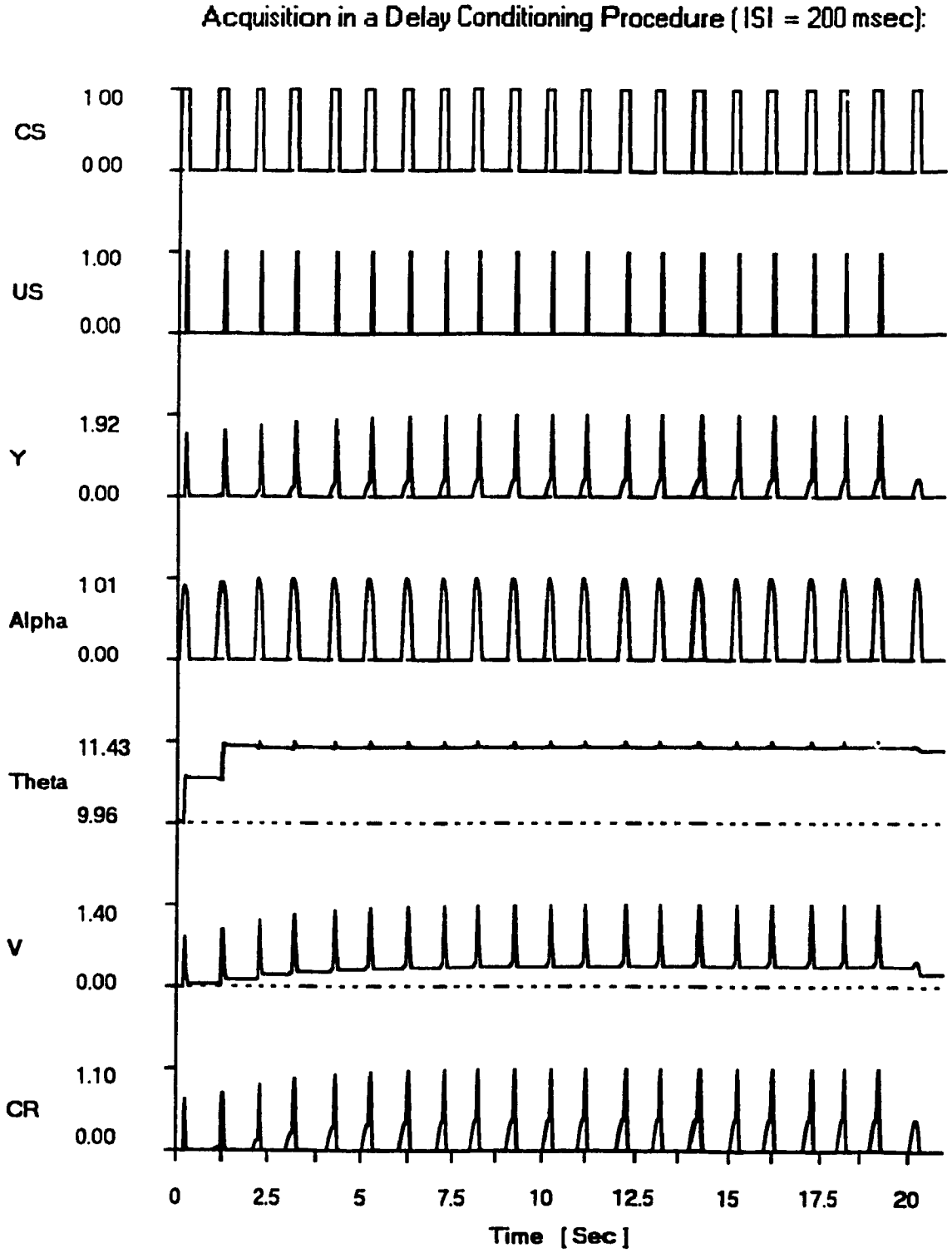

Figure 5.6 The curve of acquisition of a CR in this delay conditioning procedure is negatively accelerating. A test trial indicates that the amplitude of the CR is 0.28.

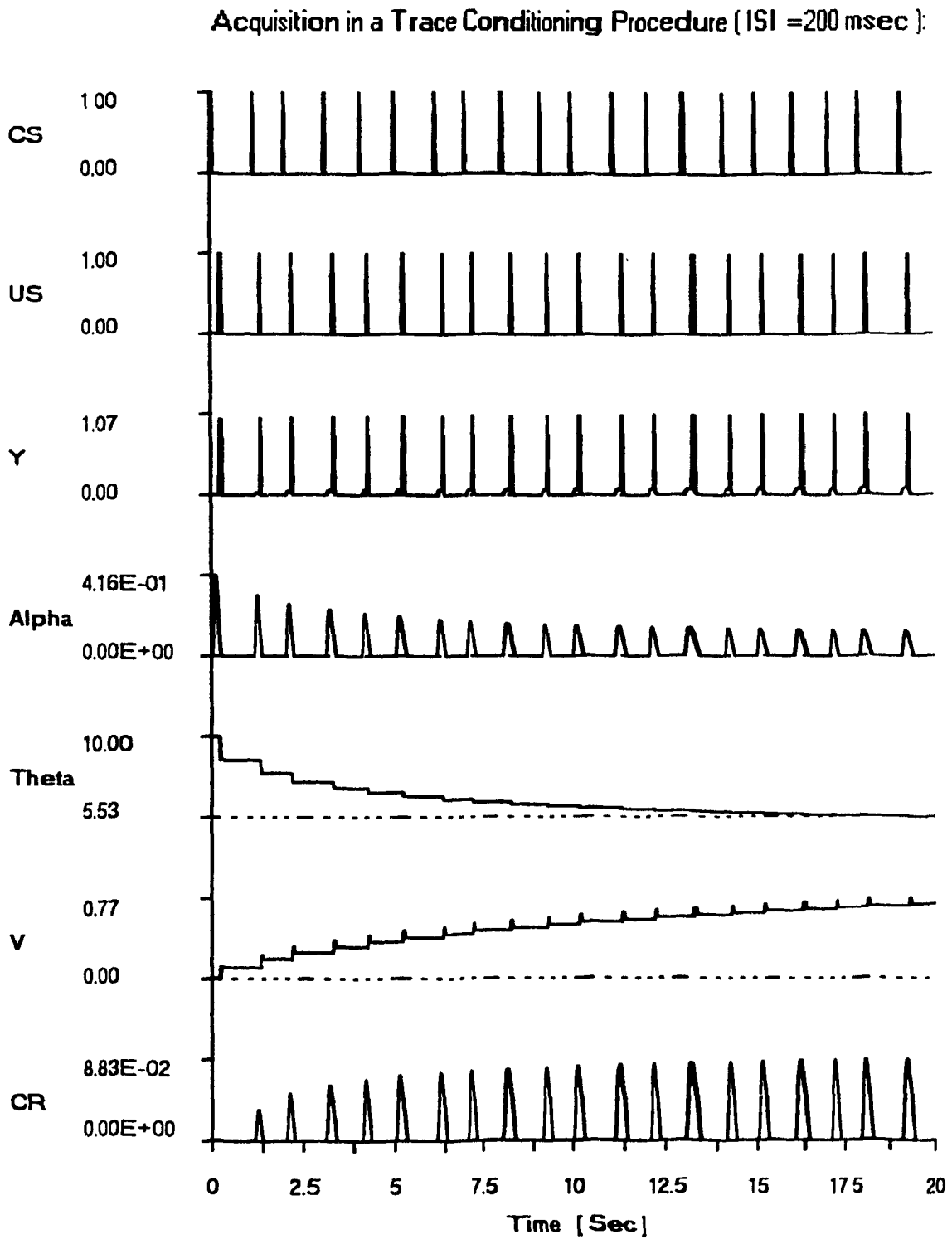

Figure 5.7 The acquisition of a CR in trace conditioning procedure reaches an asymptote at 0.09. The asymptote of the CR in delay conditioning (Fig. 5.6) is lower.

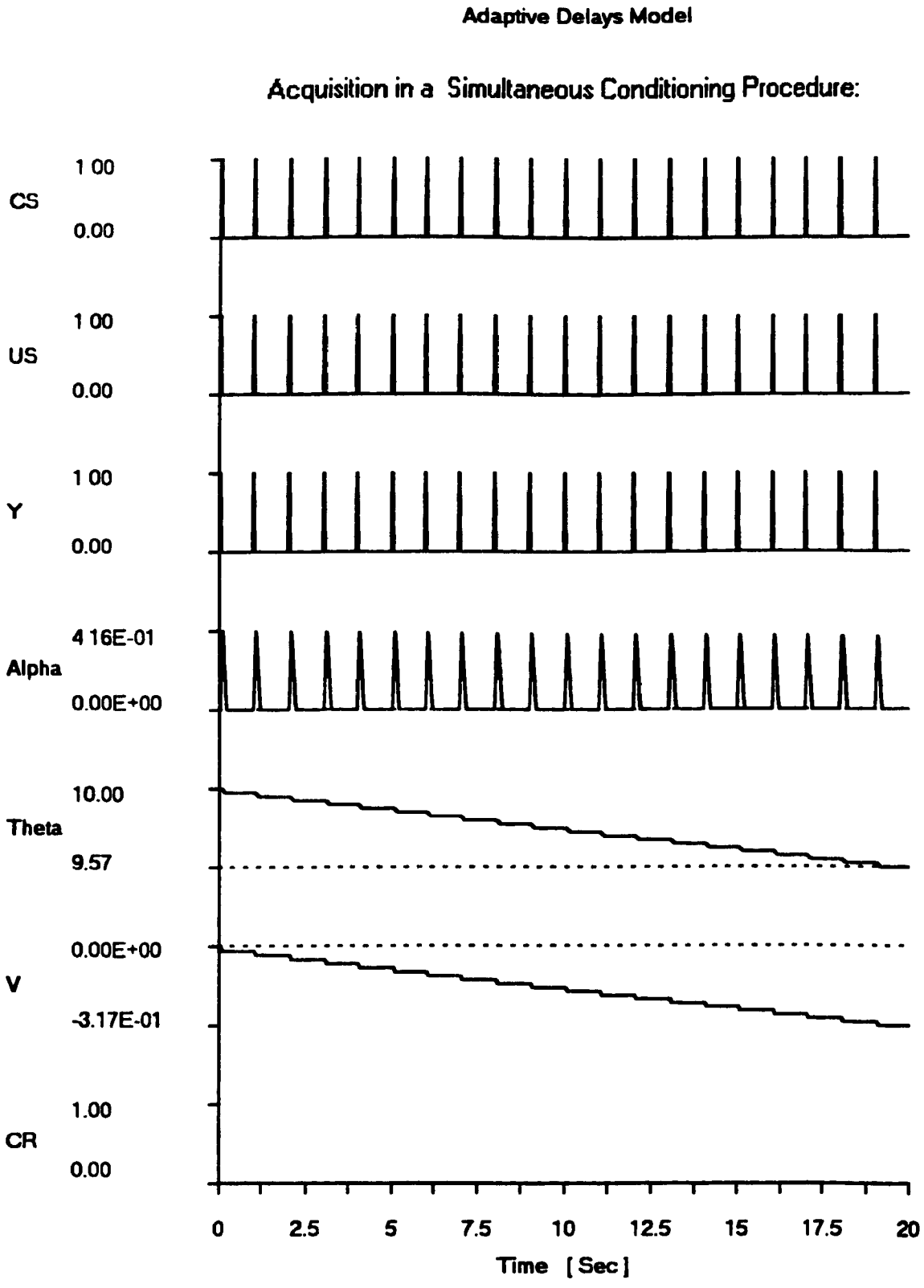

Figure 5.8 Simultaneous conditioning produces no CR. The CS becomes a conditioned inhibitor, V decreases steadily. Y is not affected, since the peak of $\alpha(t)$ arrives after the offset of the US.

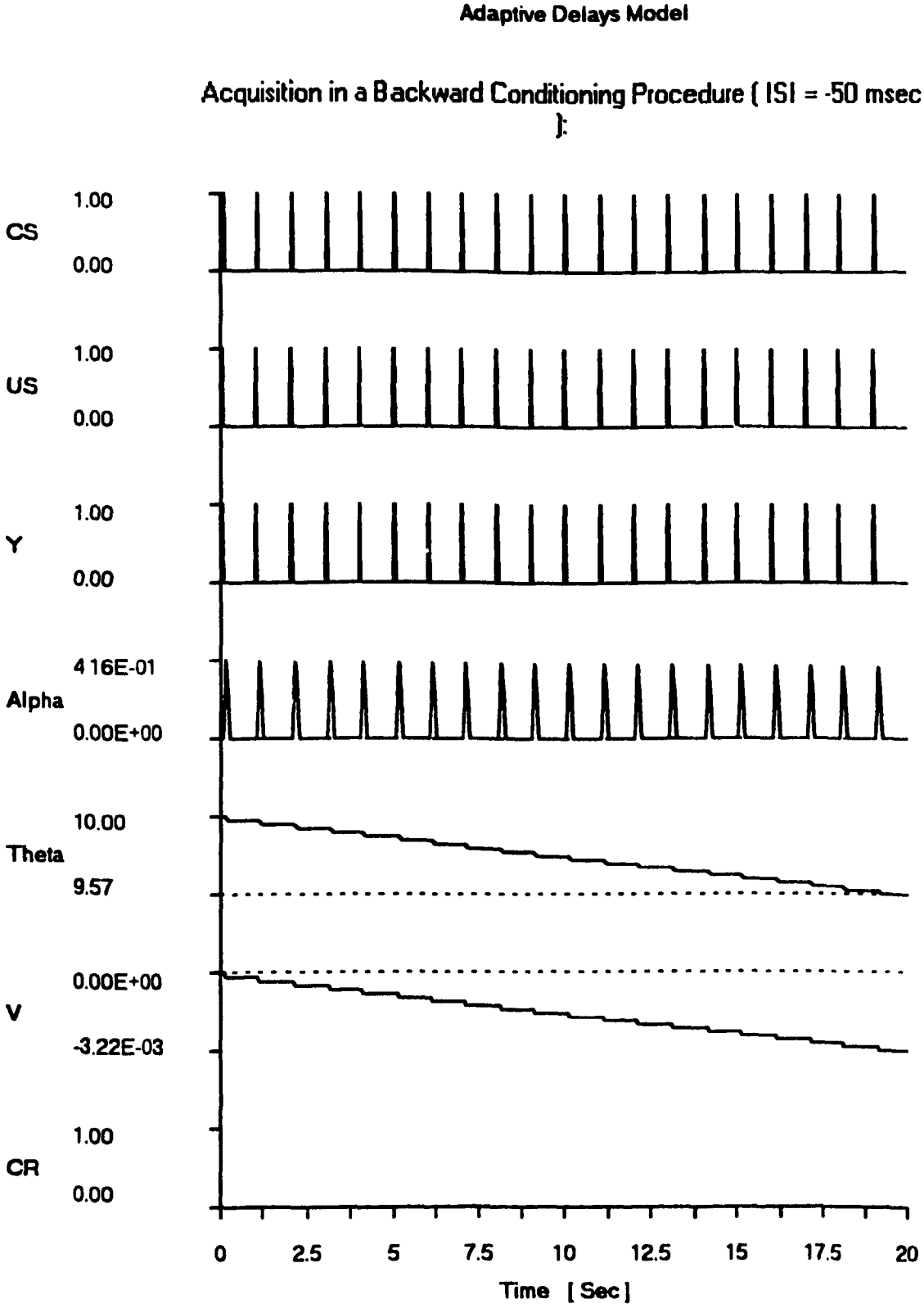

Figure 5.9 Backward conditioning produces no CR. The CS becomes a small conditioned inhibitor, since V is negative. This effect is weaker than for Figure 5.8.

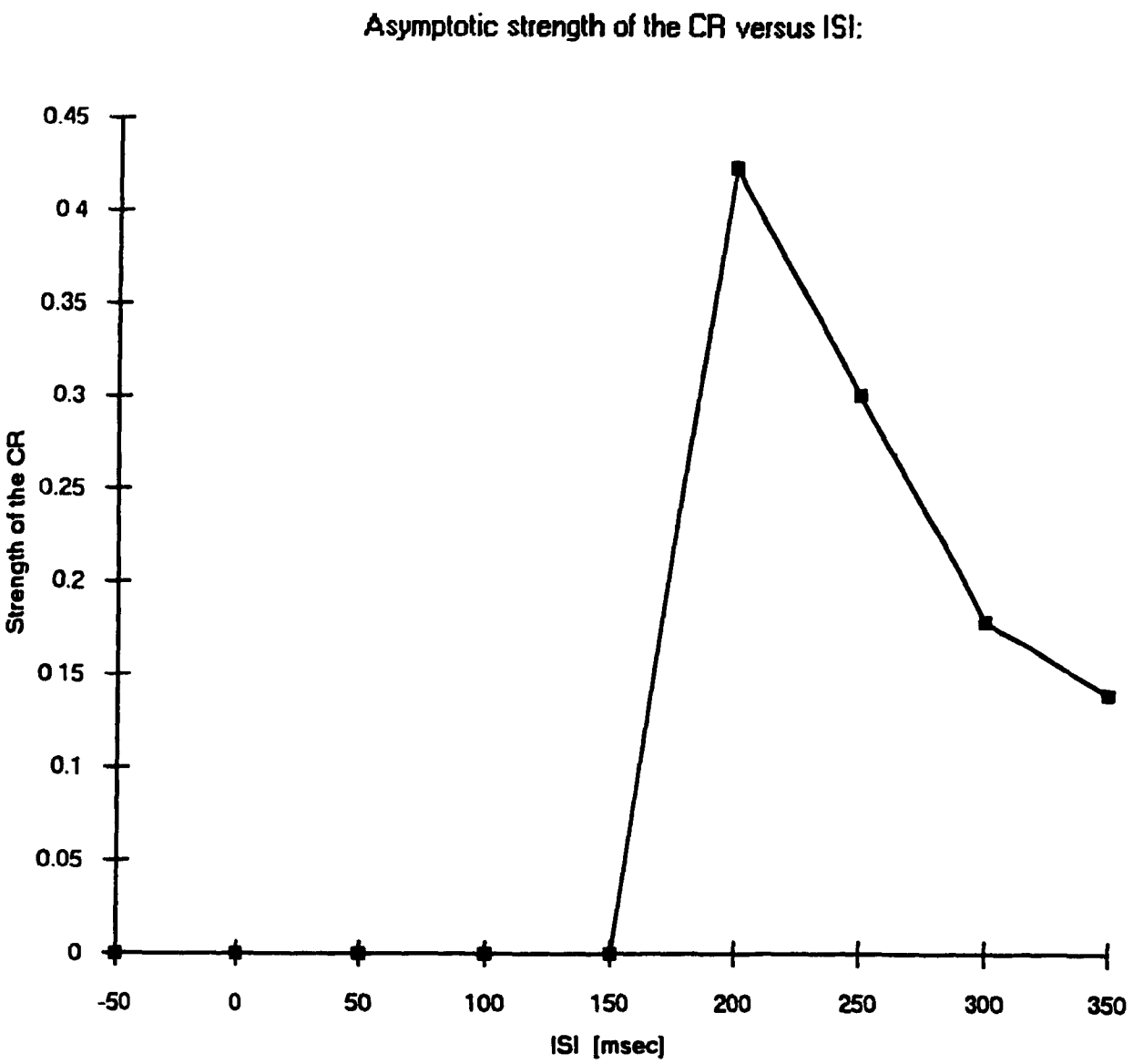

Figure 5.10 The asymptotic strength of the CR as a function of the ISI. The CS lasts for a variable period of time in this graph. The CS lasts for 50 msec with ISI = -50 and 0 msec, it lasts for 100, 150, 200, 250, 300, 350 and 400 msec with ISI = 50, 100, 150, 200, 250, 300 and 350 msec respectively. The US lasts for 50 msec. The efficacy of conditioning depends on the ISI and CS duration. When the ISI equals -50 msec, it is a backward conditioning situation. When the ISI equals 0, it is a simultaneous conditioning situation All the other values of the ISI represent delay conditioning procedures.

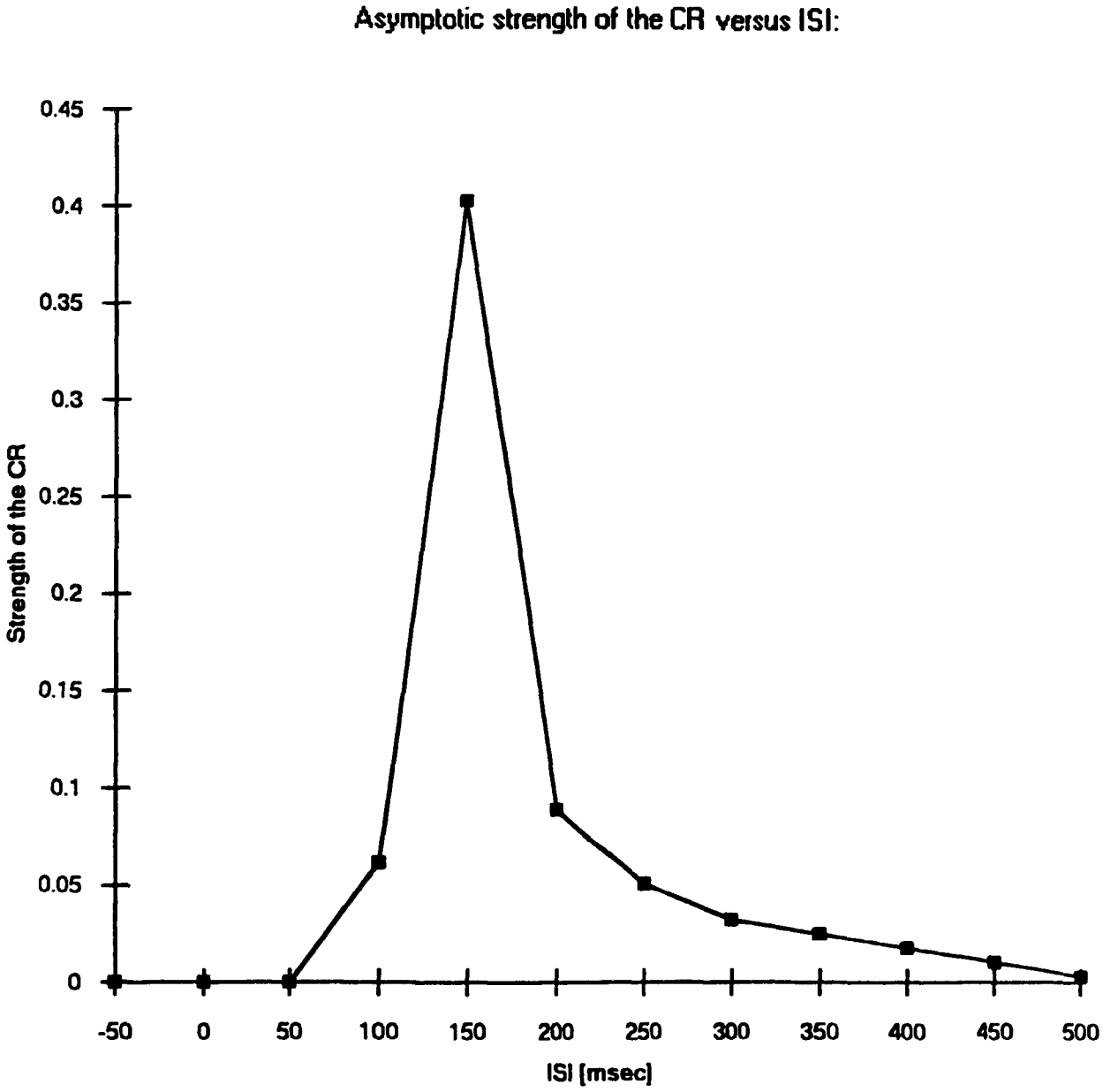

Figure 5.11 The asymptotic strength of the CR as a function of the ISI. The CS and the US last for 50 msec. When the ISI equals -50, it is a backward conditioning situation When the ISI equals 0 it is a simultaneous conditioning situation. When the ISI equal 50, it is a delay conditioning situation. All the other values of the ISI represent a trace conditioning procedure. The efficacy of conditioning is a function of the ISI. There is no CR until ISI is greater than 50 msec, then the strength reaches a peak at 150 msec and progressively decreases. The peak corresponds to a shorter ISI than in Figure 5 9, because the CSs are shorter here.

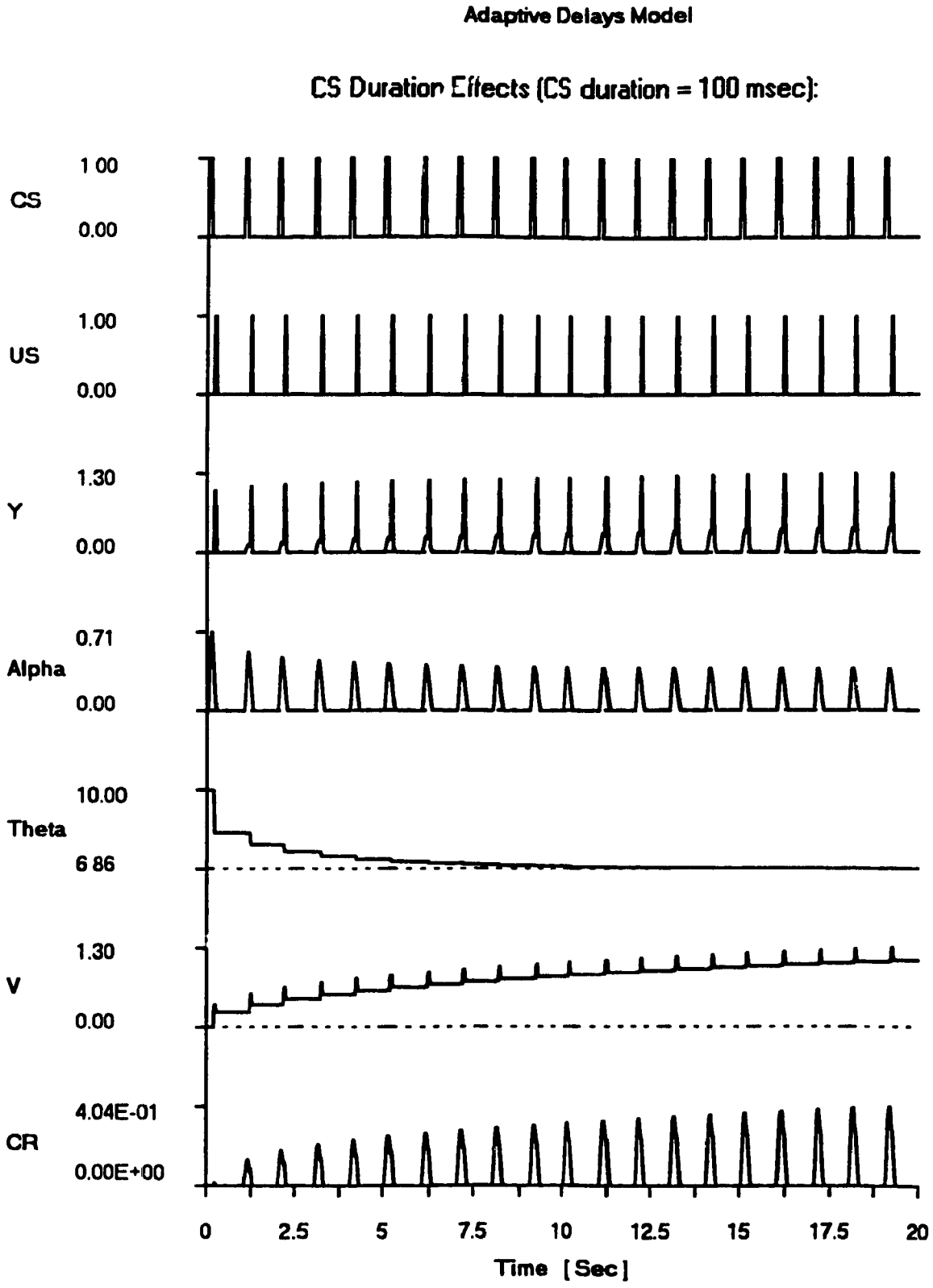

Figure 5.12 A longer CS produces a stronger CR. The CR reaches 0.4 here with a CS of 100 msec, while it reaches only 0.09 in Figure 5.7 for a CS of 50 msec in duration.

Simultaneous conditioning is supposed to produce little excitatory conditioning or no conditioning at all (see Chapter 2) The AD model predicts inhibitory conditioning for short CSs and USs (see Figure 5 8) This is because the onset of the US occurs when $\alpha_i(t-2)$ is zero and does not contribute to a change in V_i, while the offset of the US occurs when $\alpha_i(t)$ is increasing, and gives at that point a negative ΔV_i If we look at Figure 5 8, we can see that the response Y(t) is not inhibited by the acquisition of the conditioned inhibition. This is because the peak of $\alpha_i(t)$ occurs after the offset of the US With post-asymptotic conditioning, $\alpha_i(t)$ would not interfere because T_p keeps increasing by habituation. However, CSs and USs longer than the optimal ISI produce much less inhibition because at that point in time $\alpha_i(t)$ is small

Backward conditioning procedures produce weak inhibitory conditioning For instance, Figure 5.9 shows that after 20 trials, V_i reaches -0 03 The model does not produce the effect of the first trials being excitatory, each trial is inhibitory in this situation

CR Properties

The model was designed especially to simulate CR properties and so it does The curve of a CR is different from the square wave curve of a CS (see Figure 5 13) The variance of the CR increases with increasing ISI (see Figure 5 13) The CR is also positively accelerating at first as an experimental CR (see Figure 5 13) We see in Figure 5.13 that inhibition of delay is well reproduced The peak of the CR moves during training and is positioned at US onset at the end of training

CS and US Effects

An example of the US duration effects is obtained by comparing Figure 5 7 with Figure 5 14 Both situations of trace conditioning are identical except that Figure 5 7 is produced with a US of 50 msec, while Figure 5 14 is produced with a US of 100 msec The US of 100 msec produced a CR of 0 16, while the US of 50 msec produced a CR of 0.08. A longer US produces a higher asymptote for the CR

Figures 5 2 and 5.15 provide an example of US intensity effects in a trace conditioning procedure The figures differ in US intensity Figure 5 2 is the output of a simulation with a US intensity of 1 and Figure 5 15 is the output of a simulation with a US intensity of 0 5 The US intensity of 1 produces a CR of 0 047, while the US intensity of 0.5 produces a CR of 0.021 Although this result is in accordance with experimental data, other simulations of the same effects were inconclusive As was mentioned in the introduction of the present chapter, the parameters h, s and β were given unrealistically high values in order to accelerate simulations It has also been explained that s, the sensitization parameter, affects the learning rate the faster the sensitization, the faster the

CR reaches an asymptote The intensity of the US affects the sensitization process, with a stronger US producing a stronger sensitization (see Equation 5 3) With a stronger sensitization, the CR reaches an asymptote faster and the asymptotic value of the CR is smaller than it would have been with a smaller US This chain of events can be grasped by comparing Figure 5 7 with Figure 5 16 The model is sensitive in regard to s.

CS intensity effects are explained by the model This is seen by comparing Figure 5 17 with Figure 5 7 Figure 5 7 represents a simulation using a CS intensity of 1, and Figure 5 16 a simulation with a CS intensity of 0 5 These graphs show that acquisition is faster with the stronger CS The CR obtained with a CS intensity of 1 is 0 09, while the CR obtained with the CS intensity of 0.5 is 0.04 However, CS durations greater than the optimal ISI do not show such an increase of the CR for an increase of CS duration.

In a CS preexposure procedure, the CS is presented several times before the pairing with the US. In this case, we can expect the habituation process to cause a decrease of $\alpha_1(t)$, which should slow the acquisition process. This is what the simulation reveals as represented by Figures 5 6 and 5 18. With a preexposure to the CS, the CR needs more trials to reach the asymptotic value of the CR (in accordance with experimental data), but the asymptotic value is the same with or without preexposure as opposed to experimental data where the asymptote is smaller. The CS preexposure is therefore only partially simulated by the model.

Configural Learning

An overshadowing effect is presented in Figure 5.19. A CS with a salience equal to 1 is presented in conjunction with a CS that has a salience equal to 0.2. The first CS produces a CR of 0 31, while the second produces a CR of 0 025 at asymptote. The CS with the smaller salience has a smaller CR at asymptote, as expected.

The blocking procedure is successfully simulated by the model only if we take s equal to zero. Figure 5.20 represents a blocking procedure obtained in a delay conditioning paradigm, by taking s=0 The CS_1 is presented in a first phase and the corresponding CR reaches 0 23, while the CR to the CS_2 presented paired with the CS_1 in a second phase only reaches the value 0 02 When s is greater than zero, the peak of the CR_1 acquired in the first phase moves during conditioning. In the second phase the CR_1 acts as a reinforcer for the second CS because $\alpha_2(t)$ and CR_1 are not simultaneous, and there is therefore no blocking. When s = 0, as with the DPC model, the delay before the peak of the conditioned response is fixed and the blocking procedure is well reproduced

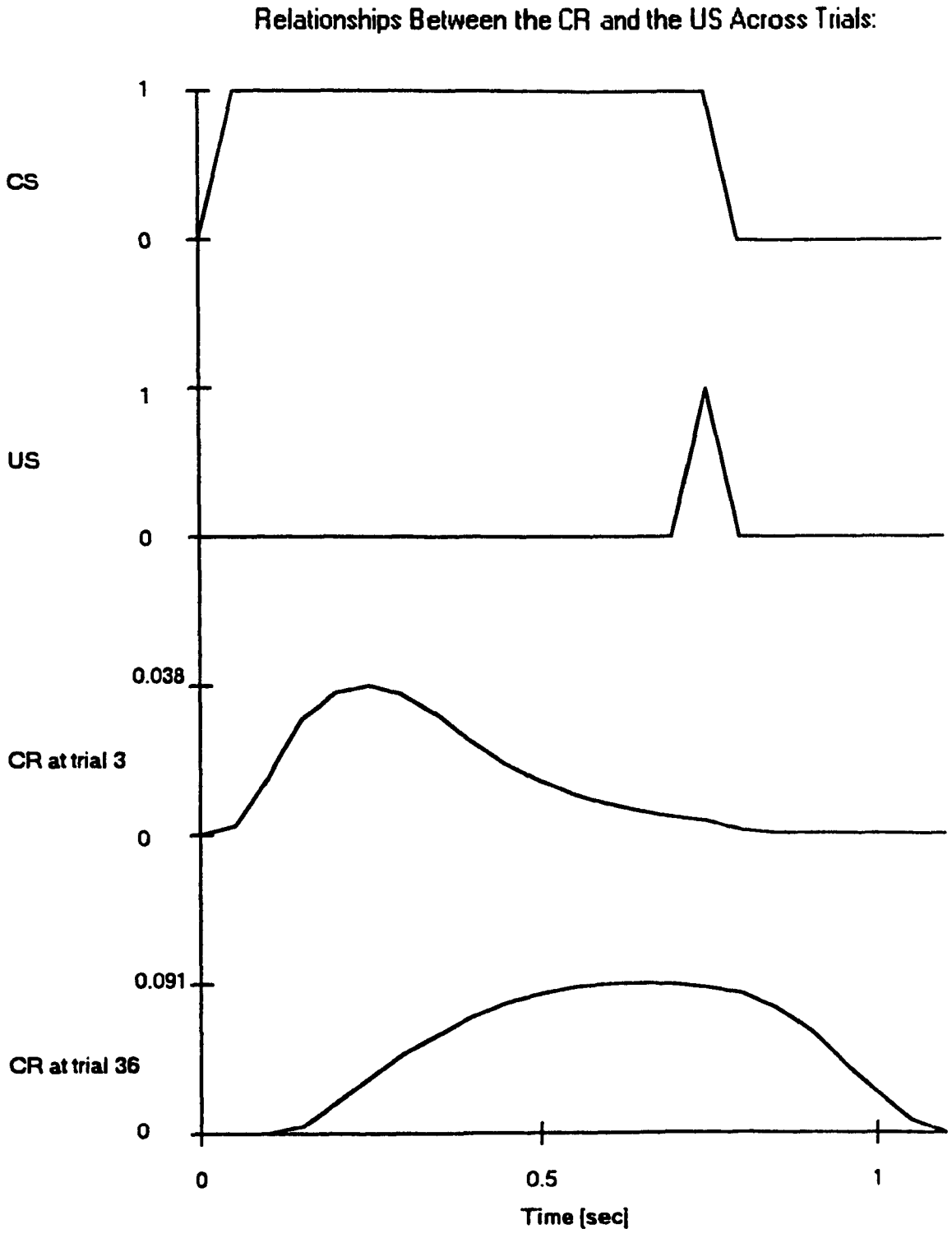

Figure 5.13 During conditioning, the peak of the CR moves toward the onset of the US and its variance increases.

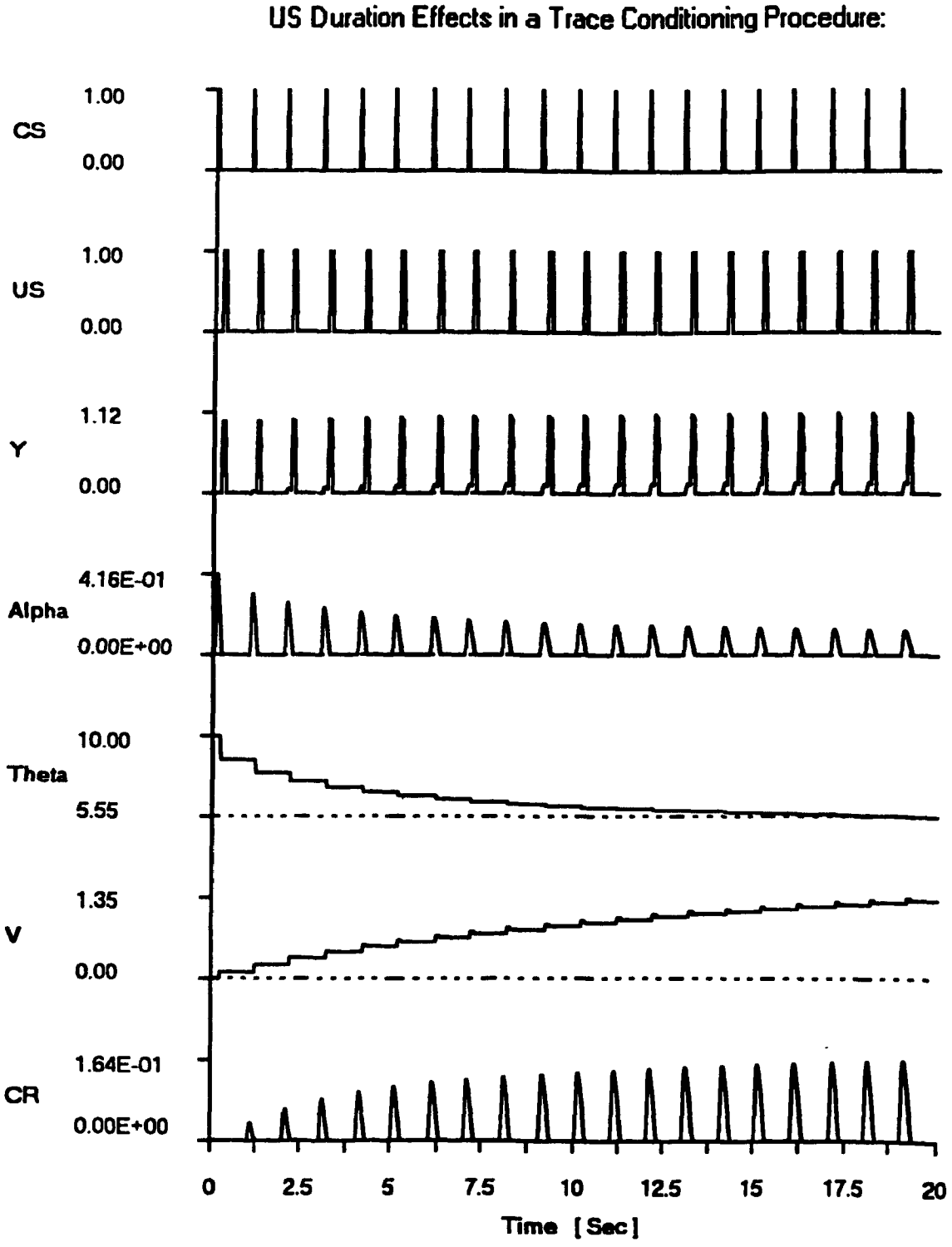

Figure 5.14 A longer US produces a higher asymptote for the CR. The US of 100 msec produced a CR of 0.16, while the US of 50 msec (see Figure 5.7) produced a CR of 0.09.

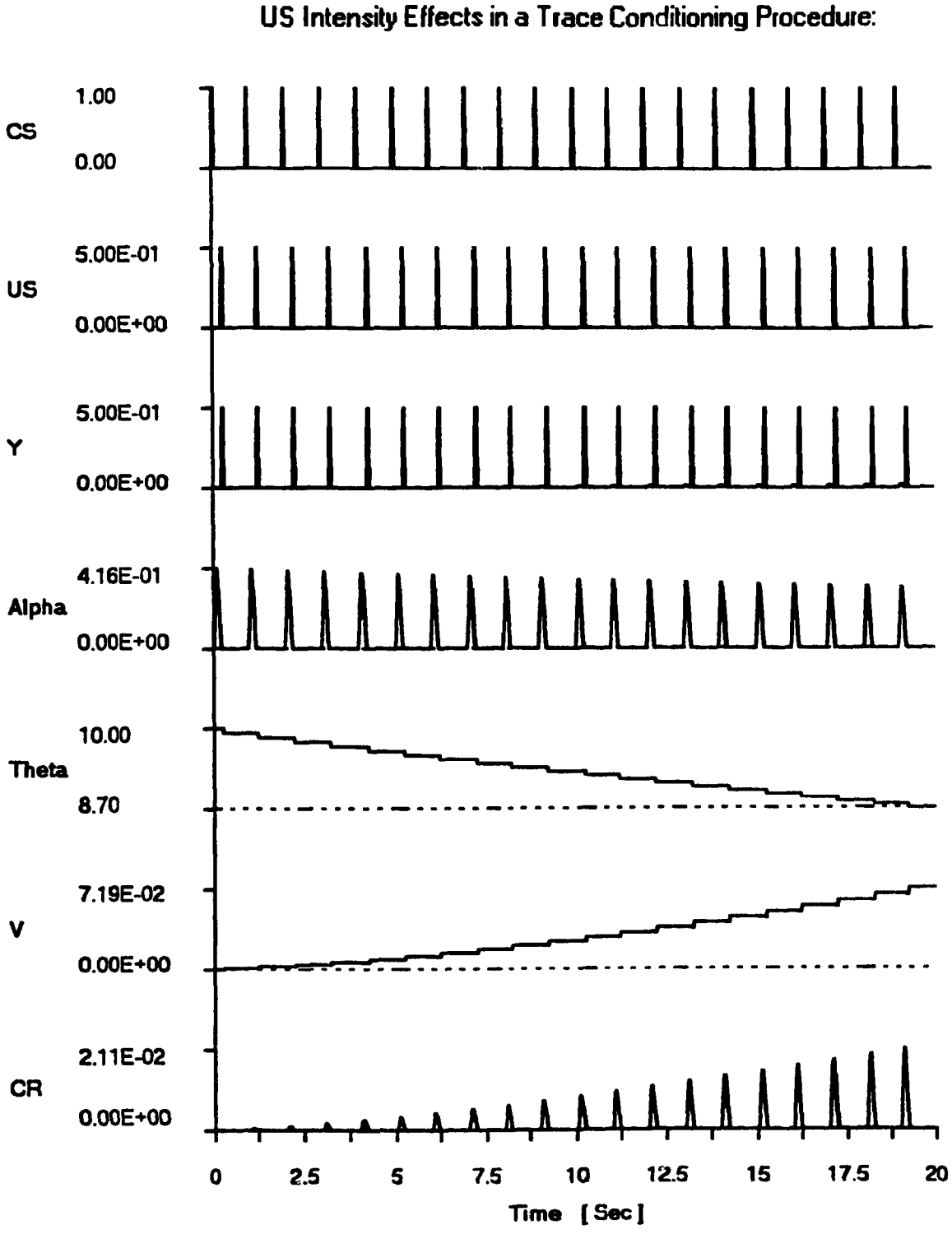

Figure 5.15 A less intense US produces a smaller CR. A US intensity of 0.5 produces a CR of 0.02, while a US intensity of 1 in Figure 5.2 produces a CR of 0.047.

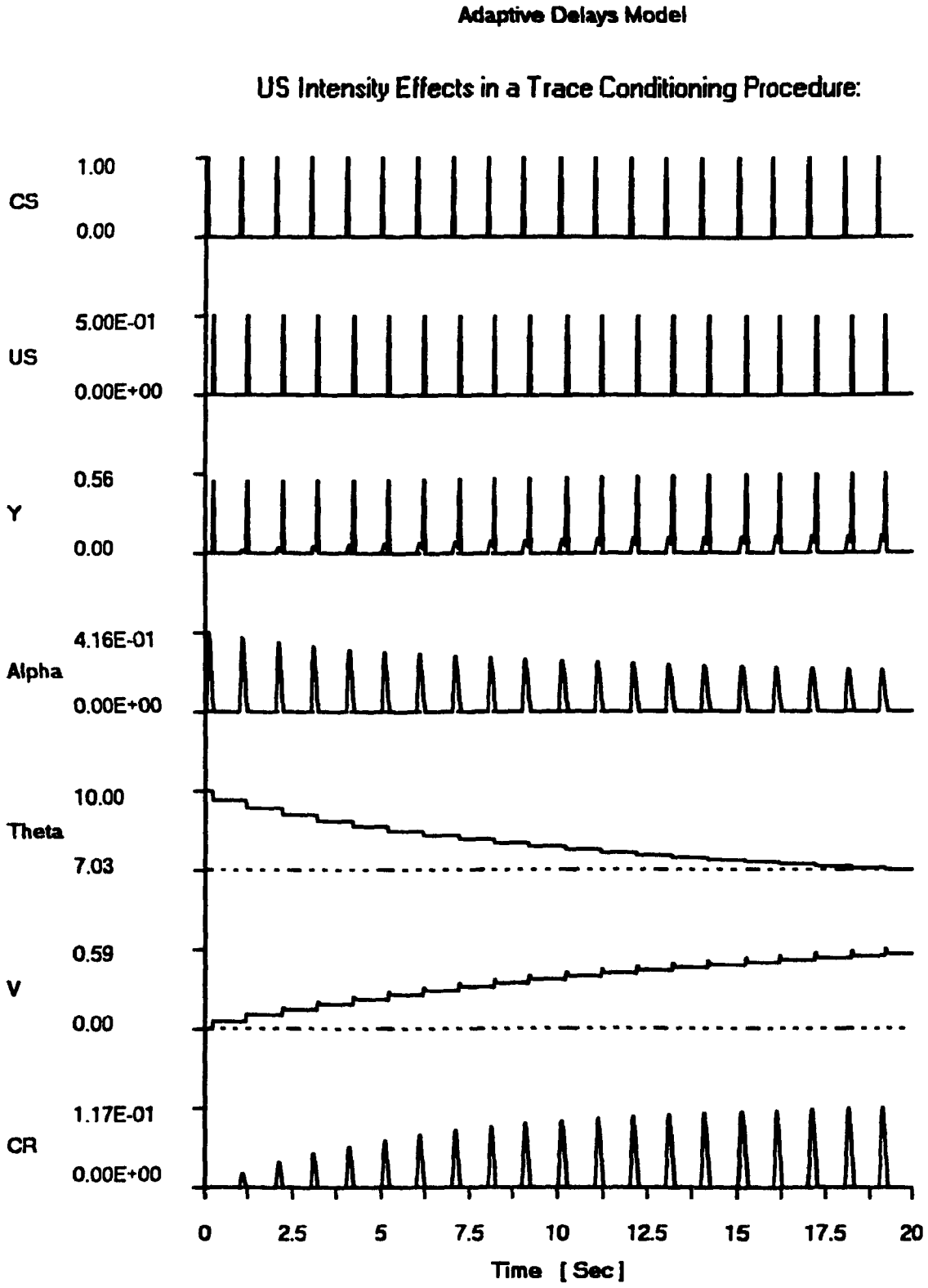

Figure 5.16 A problem arises with higher values of s (here s=0.03). The CR is stronger with a US intensity of 0.5, than it is in Figure 5.7 with a US intensity of 1.

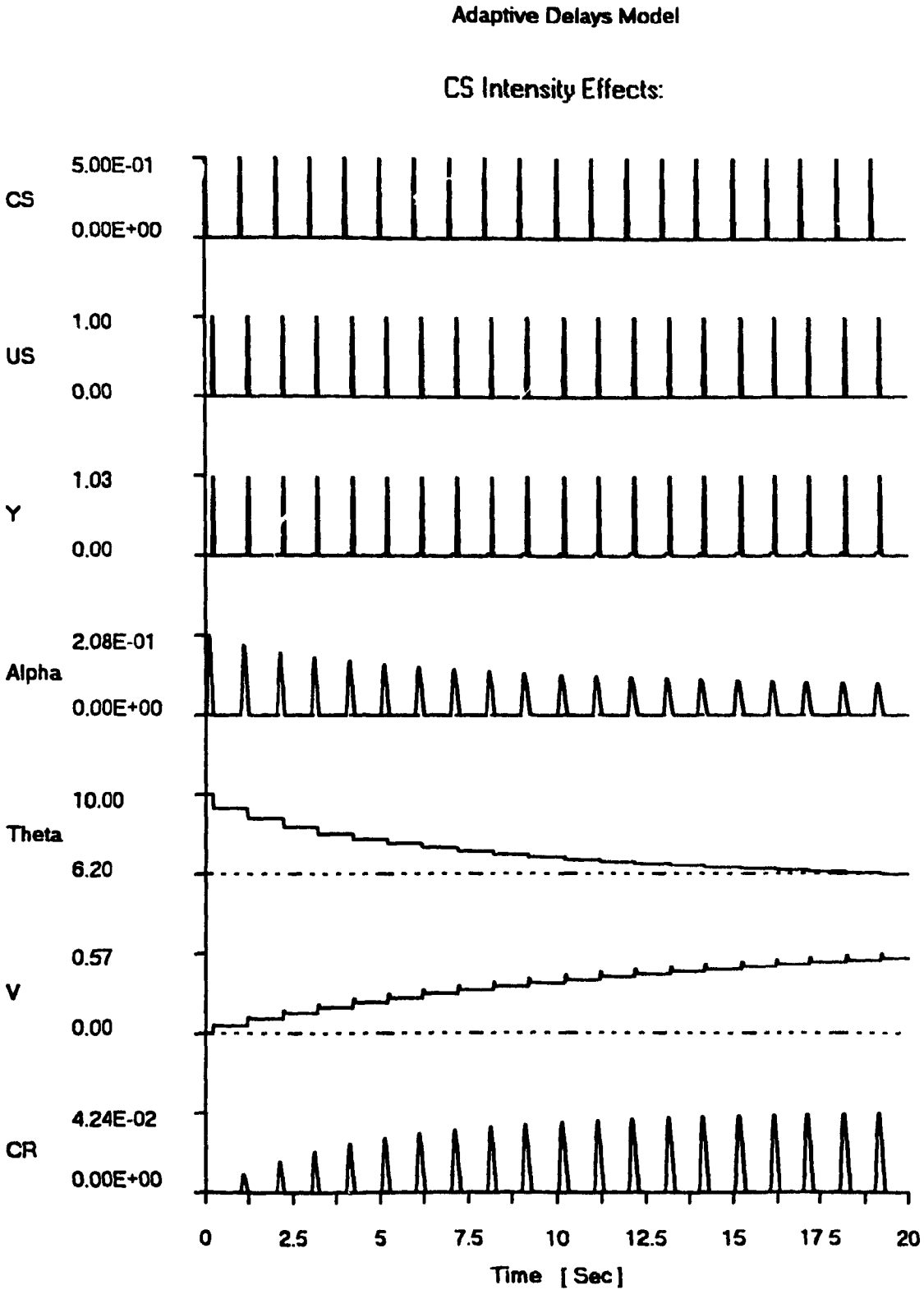

Figure 5.17 The CR obtained with the CS intensity of 0.5 is 0.04, while the CR obtained in Fig. 5.7 with a CS intensity of 1 is 0.09. Acquisition is also slower here.

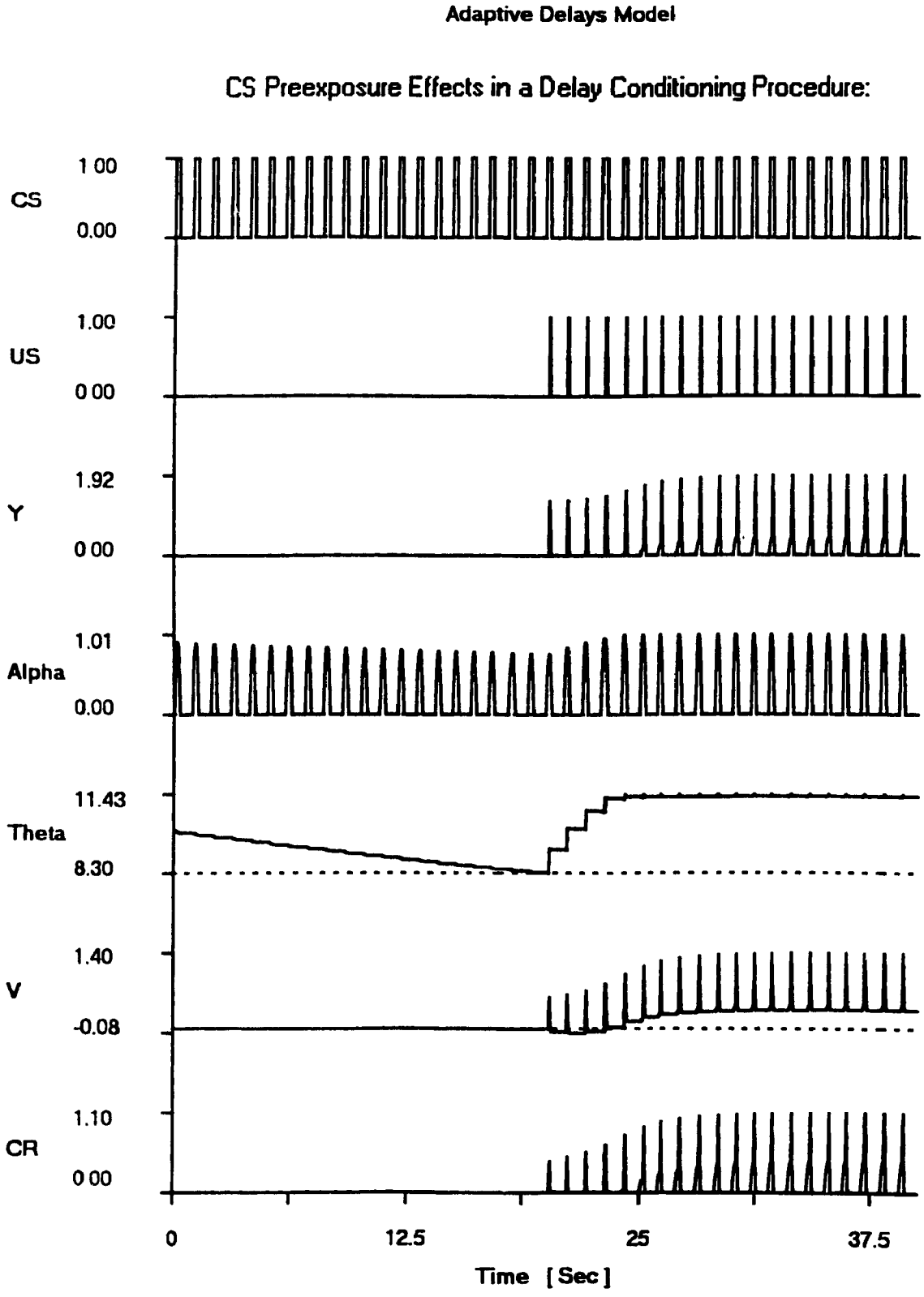

Figure 5.18 Preexposure to the CS in a first phase slows the acquisition of the CR in a second phase.

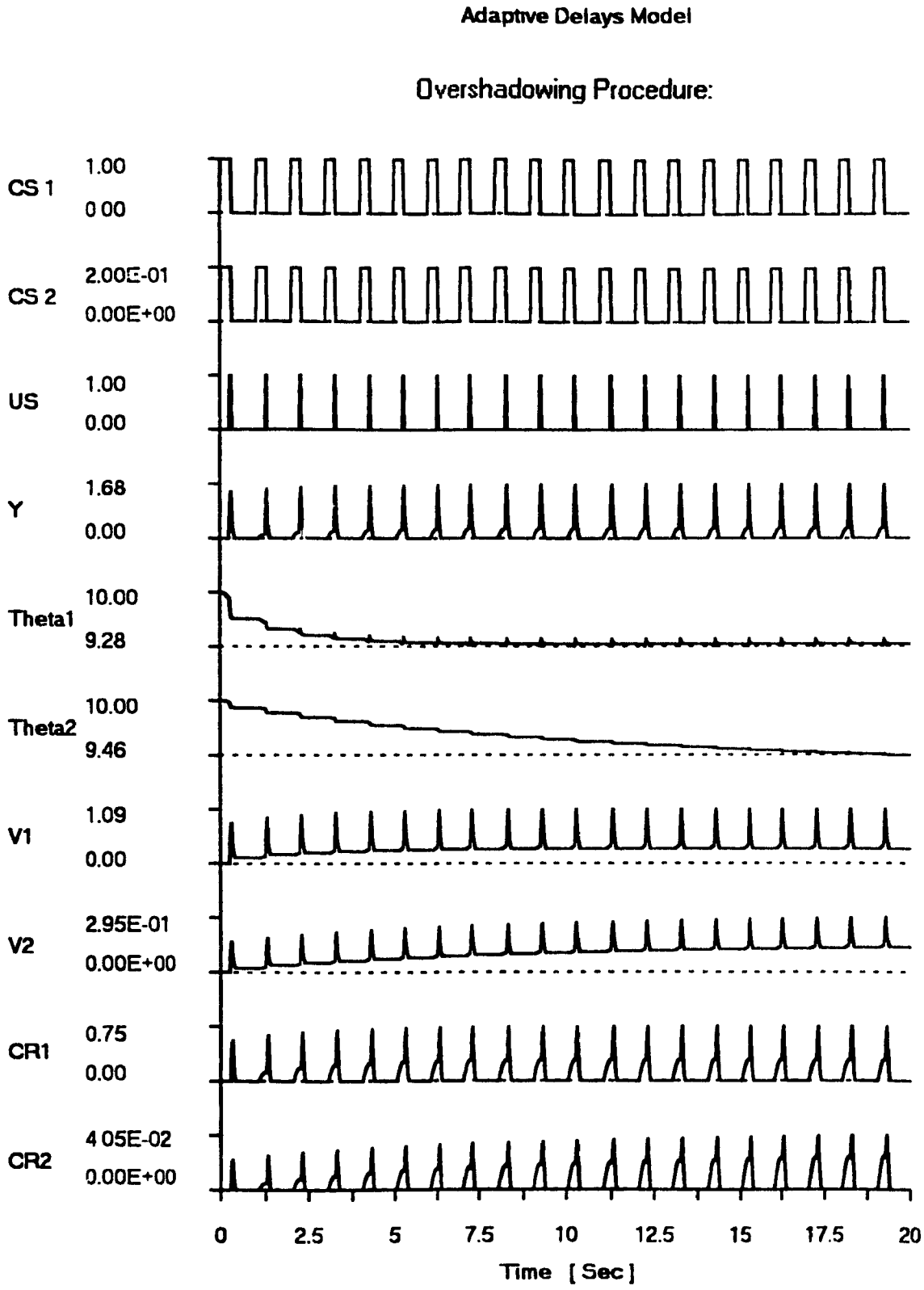

Figure 5.19 The less salient CS (CS_2) acquire a smaller CR than the more salient CS (CS_1). CS_1 produces a CR of 0.31, while the CS_2 produces a CR of 0.025 at asymptote

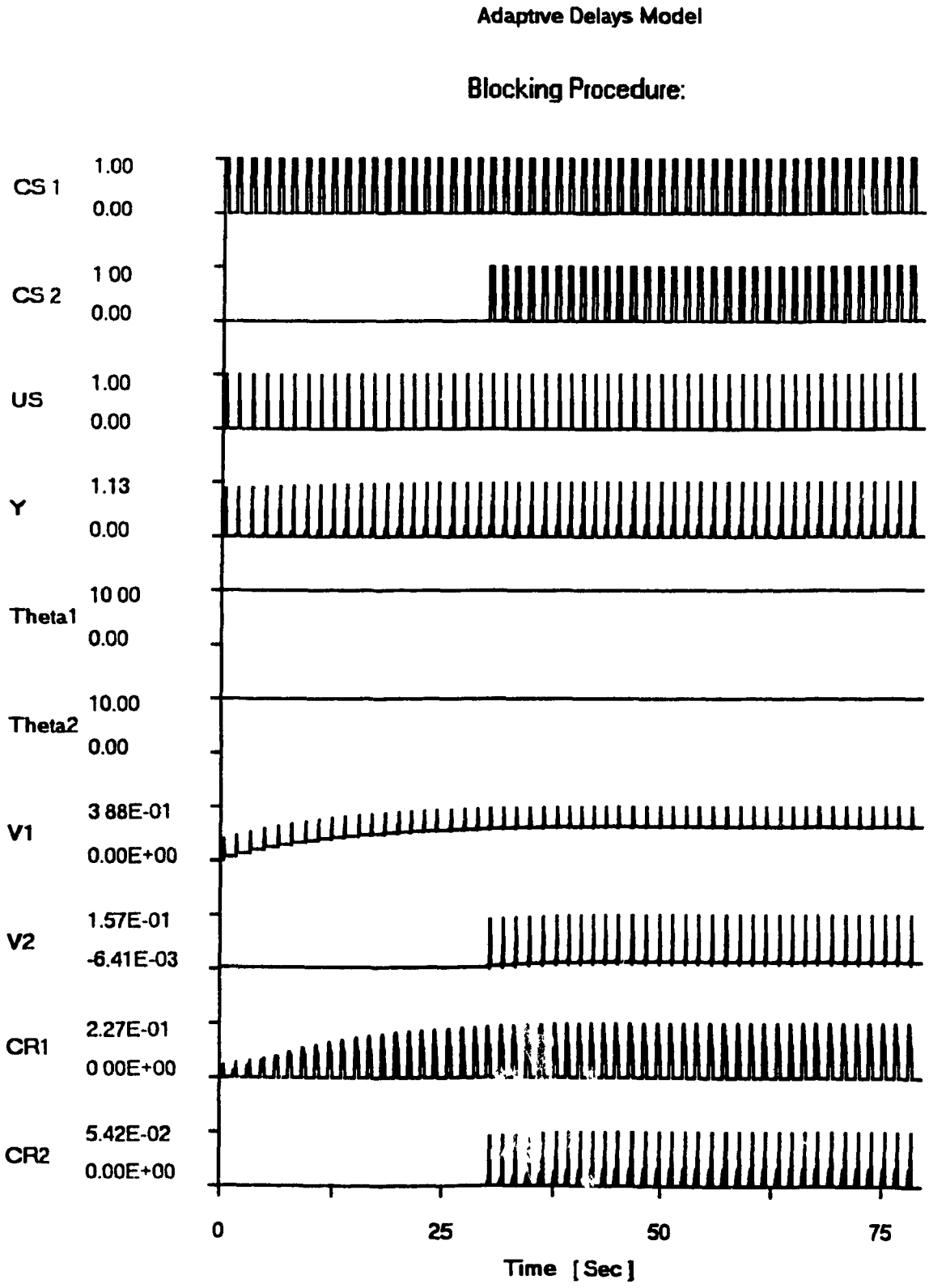

Figure 5.20 The CS_1 is presented alone and its CR reaches 0.23. In a second phase the CR to the CS_2, presented at the same time as CS_1, reaches only 0 01. The acquisition of a CR to CS_2 is blocked by the presence of the previously acquired CR_1.

An example of second-order conditioning is shown in Figure 5 21 The CR acquired in a first phase is used as a reinforcer for another CS (CS_2) presented in the second phase The CS_2 acquires a CR_2 and then this CR_2 extinguishes itself while the CR_1 extinguishes also In an overexpectation procedure two CSs are separately paired with a US and then they are presented together at the same time as the US Figure 5 22 pictures what is happening in this situation At first, the response Y(t) is higher when the CSs are presented together than it is when the CSs are presented individually Subsequently, the response to the compound stimulus decreases The CR to the individual CSs also decreases in this final phase (Klein, 1991)

Figure 5 23 represents the superconditioning procedure A stimulus CS_3 acquires a stronger conditioned response in presence of the conditioned inhibitor CS_2 If a CS is presented alone with the US (as in Figure 5 6) it elicits a CR of 0 41, but presented with the inhibitor it elicits a CR of 0 99

A discrimination learning procedure is represented by the sequence AX+ BX- The compound formed by the stimuli A and X is reinforced, while the compound formed by the stimuli B and X is not reinforced Such an experiment produces a stronger CR for A than for X; a stronger CR for X than for B; the CR to B tends toward zero, the CR to X increases and then decreases, the CR to BX increases and then decreases afterward, the CR to AB is greater than the CR to BX All these properties are evident in the simulation results presented in Figures 5 24 and 5 25 In these figures A is CS_1, B is CS_2, and X is CS_3 Figure 5.26 gives an example of a pseudodiscrimination procedure This procedure is summarized by the following AX+ 0.5/ AX- 0 5/ BX+ 0 5/ BX- 0 5 The compound AX is reinforced half of the time and the compound BX is reinforced half of the time The essential features of this phenomenon are presented in Figure 5 26 (CS_1 = A, CS_2 = B, and CS_3 = X) The CR to X is equal to 0 7 and is greater than the CR to A and the CR to B The CR to A is approximately equal to the CR to B The CR to X (0 7) is greater than the CR to X at the end of the discrimination procedure ($\cong$ 0 03) (Figure 5 23) However, to obtain a pseudodiscrimination simulation that corresponds to experimental data, it was again necessary to set $h = 0$, $s = 0$ and $\beta = 0\ 2$.

The compound conditioning procedure where a stimulus A is reinforced when it is presented alone and is also reinforced when it is presented in conjunction with X (this is summarized by A+ AX+), should produce a stronger CR to A than to X Figure 5 27 shows this effect, the CR to A (CR_1) reaches 0 44, but the CR to X (CR_2) is zero at the end of training In another compound conditioning procedure, a stimulus A is not reinforced when it is presented alone, but is reinforced when presented in conjunction with X (this is summarized by A AX+) This should produce a stronger CR to X than to

A Figure 5.28 shows this effect, the CR to X (CR_2) reaches 0.34 while the CR to A (CR_1) tends toward zero at the end of training.

The AD model, being a single unit model, does not have the structure necessary to account for more complex forms of configural learning For example positive patterning, negative patterning, and feature positive procedures are not explained by the model An account of such phenomena can be made by a network model, but not by a single unit model (Kehoe, 1989) However, the DPC model or the AD model could be implemented at the network level to test for these phenomena

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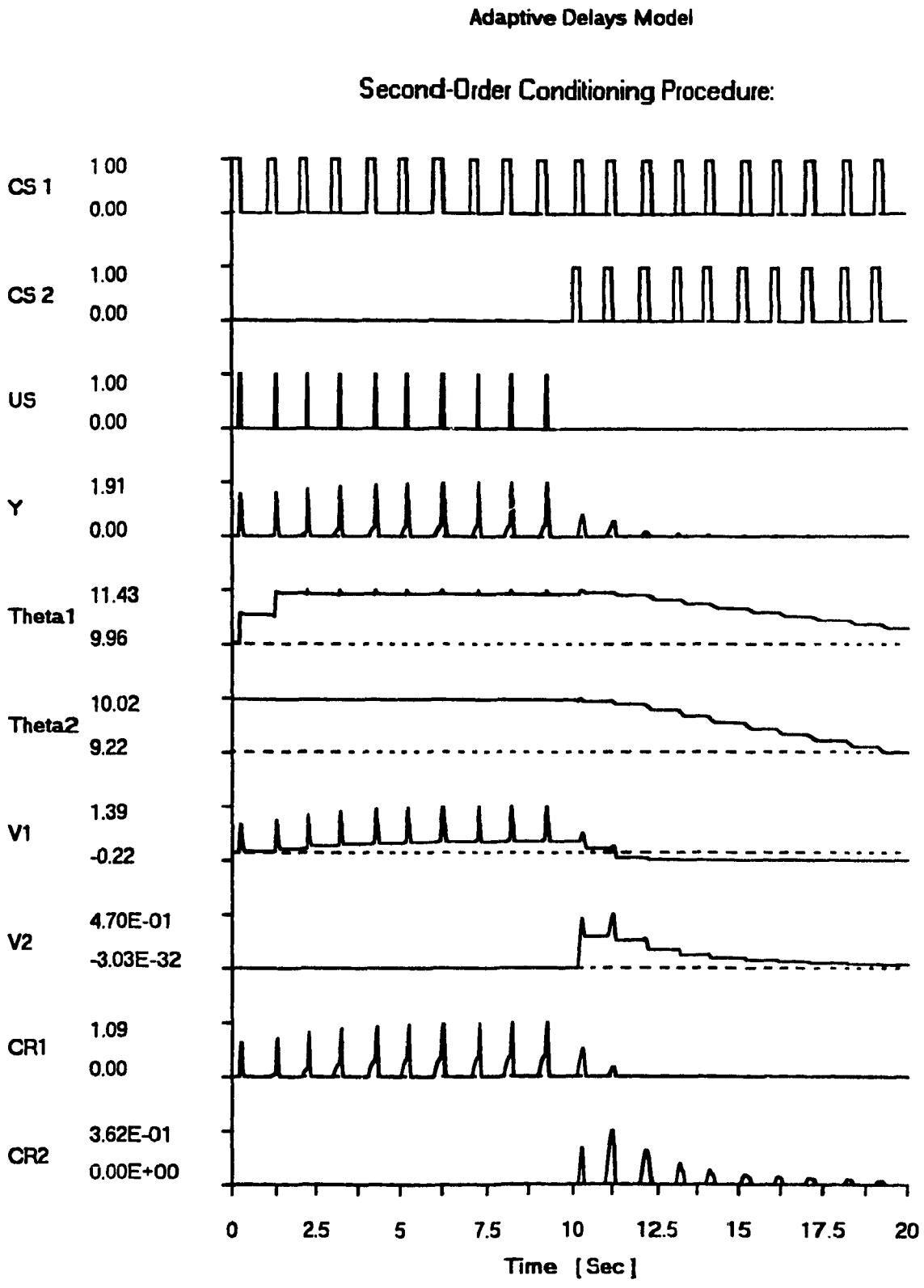

Figure 5.21 The CR_2 acquired in a first phase is used as a reinforcer for the CS_2 in a second phase. The CS_2 acquires a CR_2 and subsequently both CRs are extinguished.

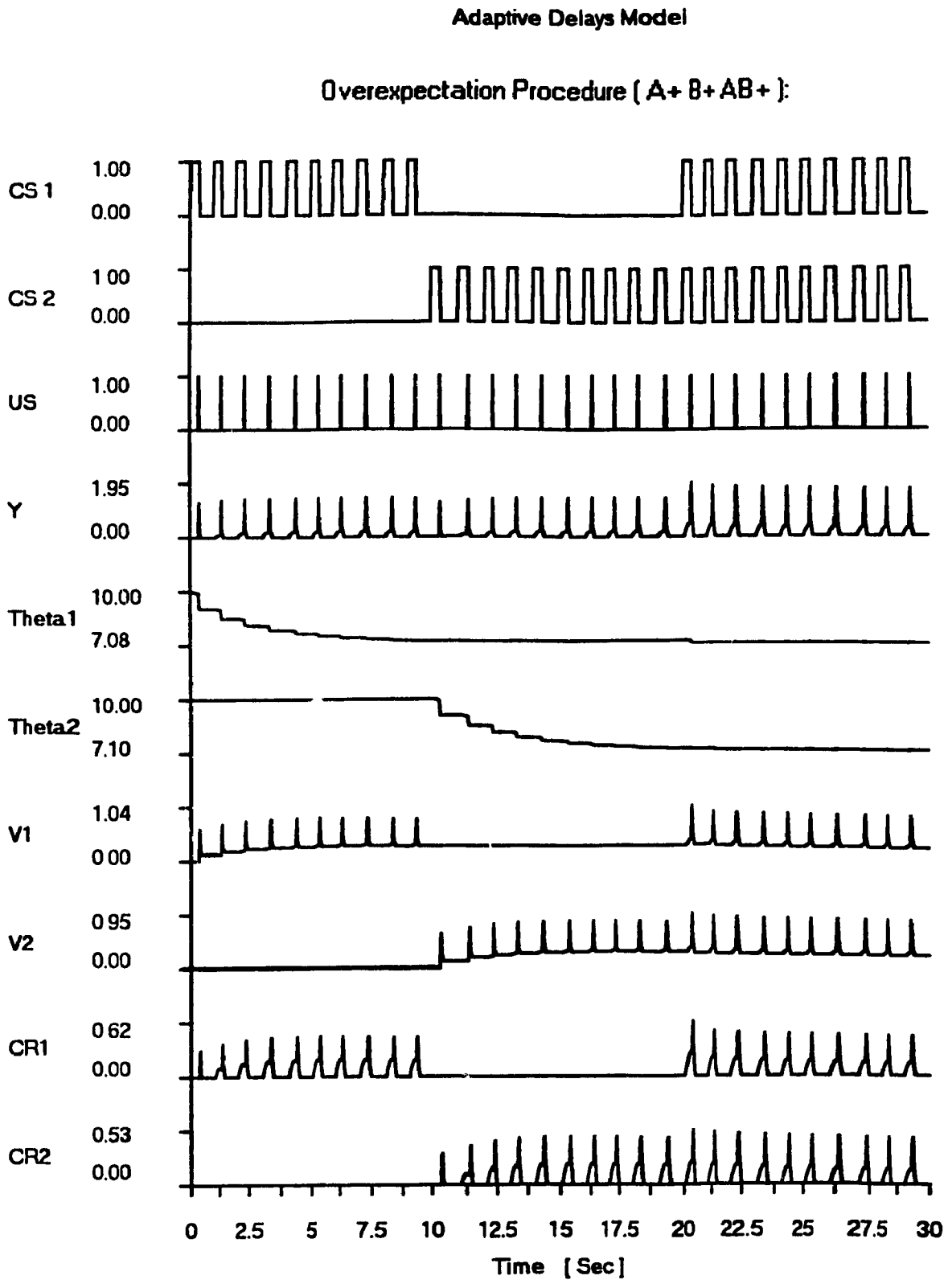

Figure 5.22 CS_1 and CS_2 separately acquire a CR, then they are paired together with the US. At first Y is higher than it is with individual CSs, then Y and both CRs decrease.

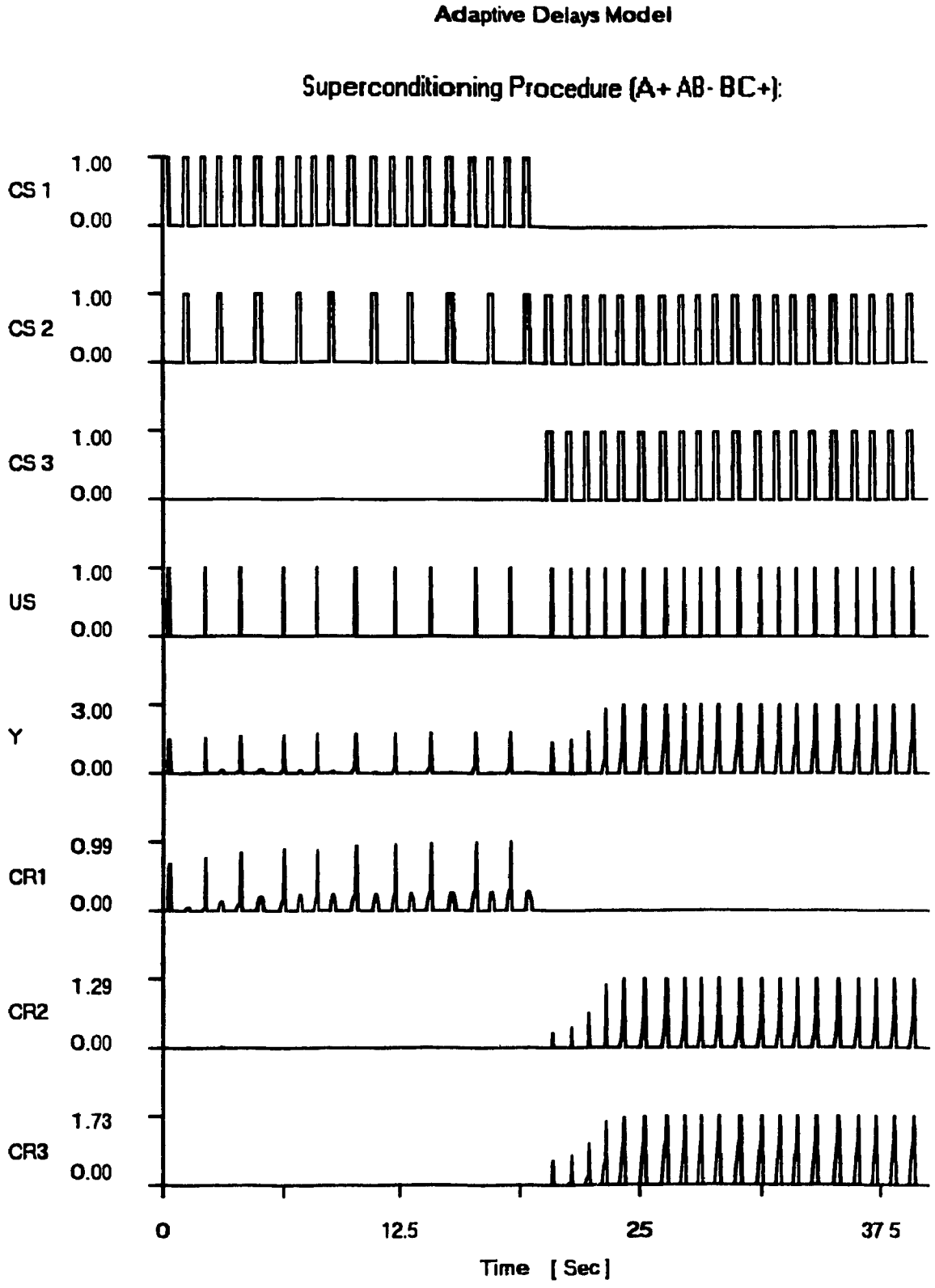

Figure 5.23 In phase 1, the CS_2 becomes a conditioned inhibitor. The CS_3 acquires a stronger CR (0.91) paired with the CS_2, than it does (0.4) when not paired, as in Fig. 5.6

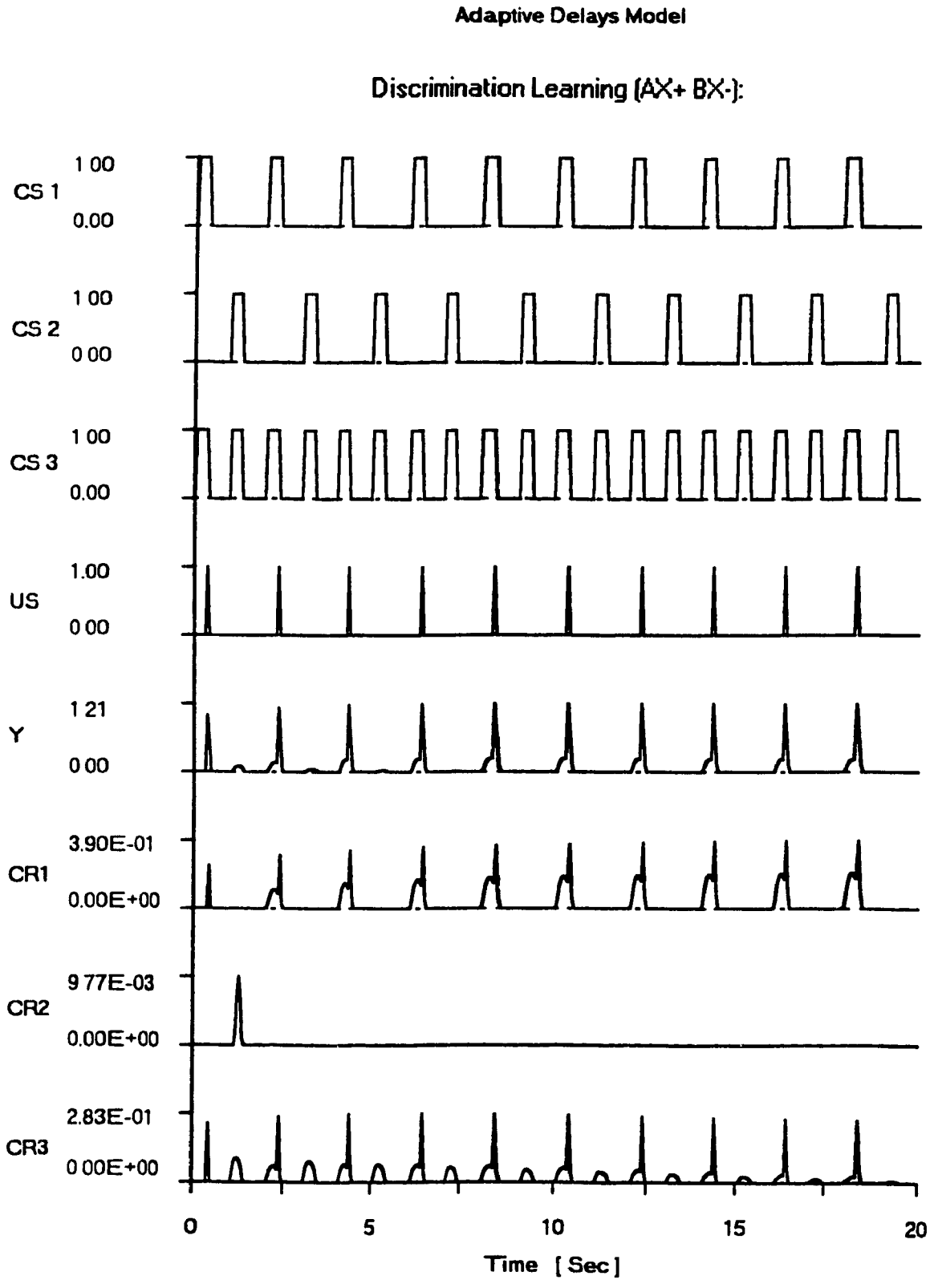

Figure 5.24 Here we have A=CS_1, B=CS_2, and X=CS_3. The compound AX is reinforced, while BX is not reinforced. V_2 increases and then decreases. See Fig. 5.25.

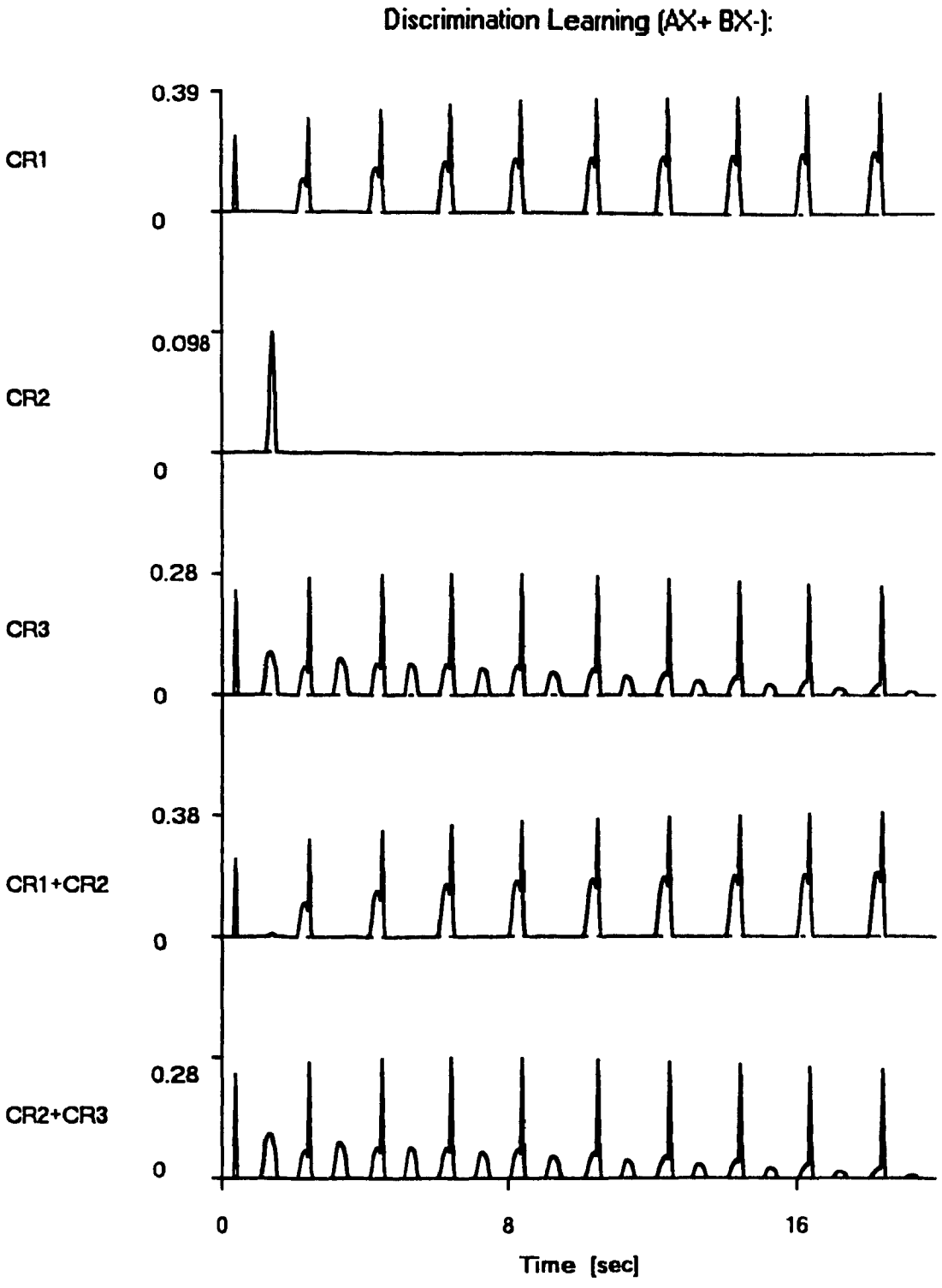

Figure 5.25 The CR to A is greater than the CR to X, which in turn is greater than the CR to B. The CR to AX is greater than the CR to BX. See also Figure 5.24.

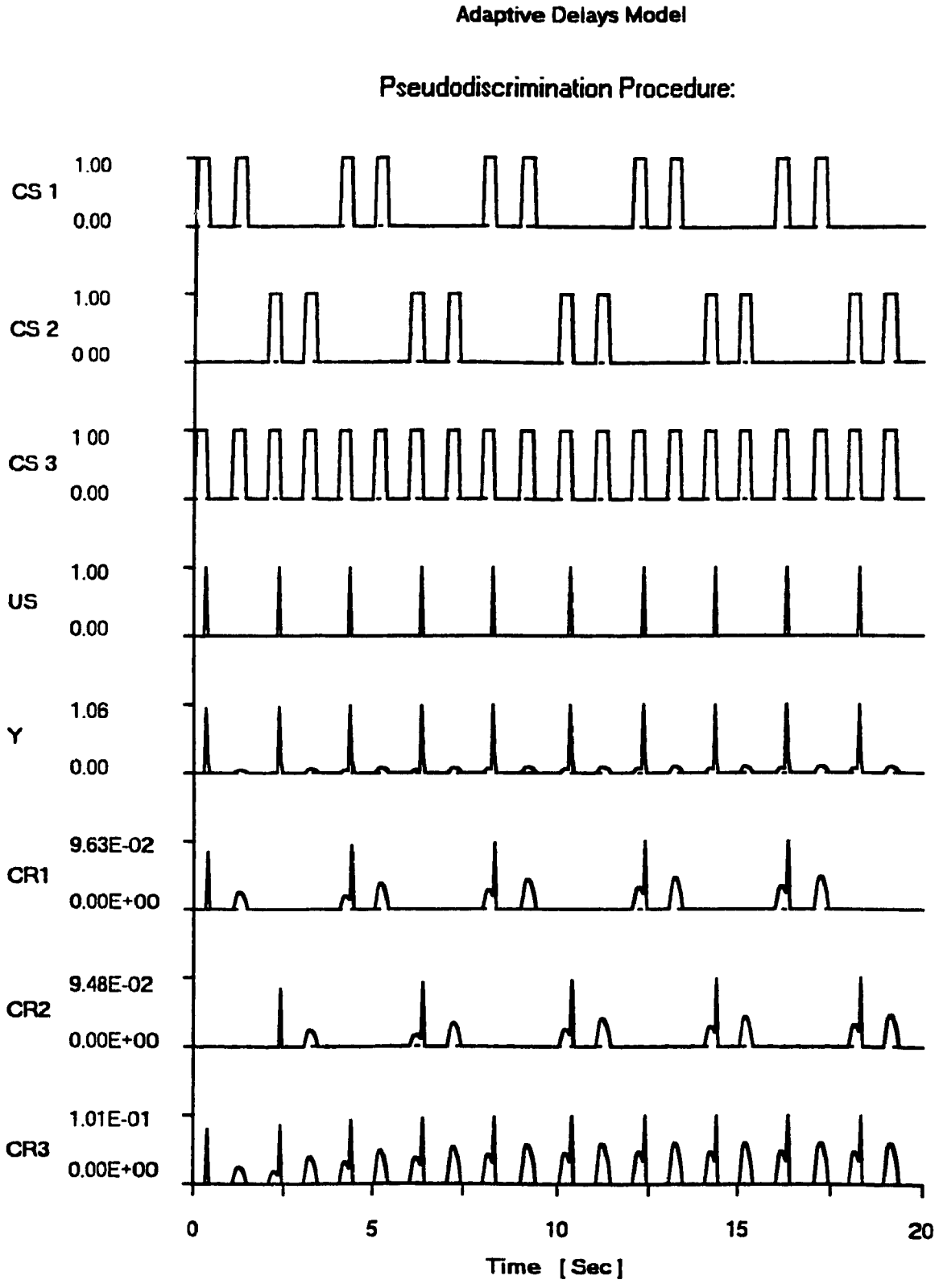

Figure 5.26 (CS_1 = A, CS_2 = B, and CS_3 = X). The CR to X is equal to 0.7 and is greater than the CR to A and the CR to B. The CR to X (0.7) is greater than the CR to X at the end of the discrimination procedure ($\cong$ 0.03) (see Figure 5.25).

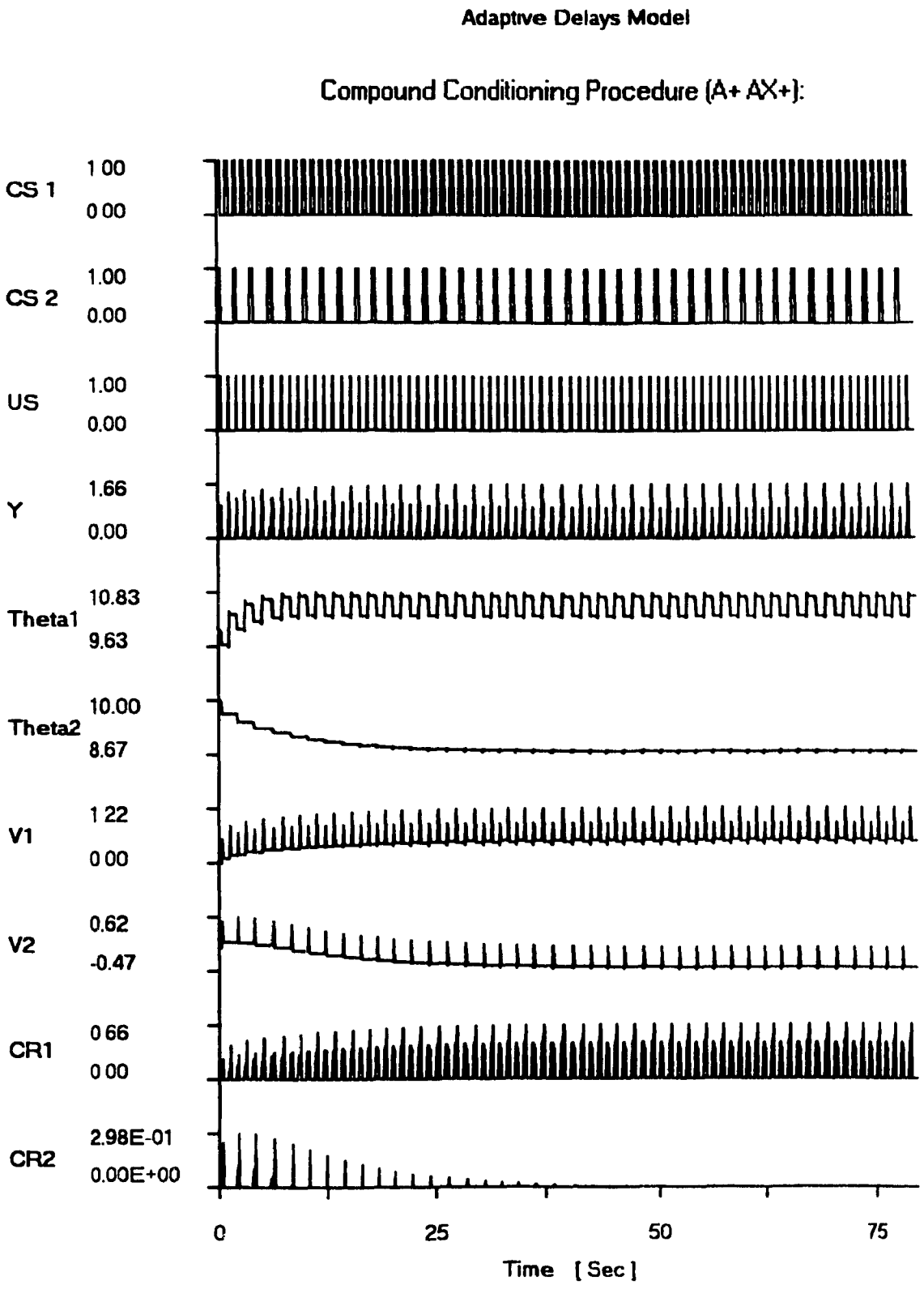

Figure 5.27 This compound conditioning procedure produces a stronger CR to A than to X. The CR to A (CR_1) reaches 0 44, but the CR to X (CR_2) is 0 at the end of training

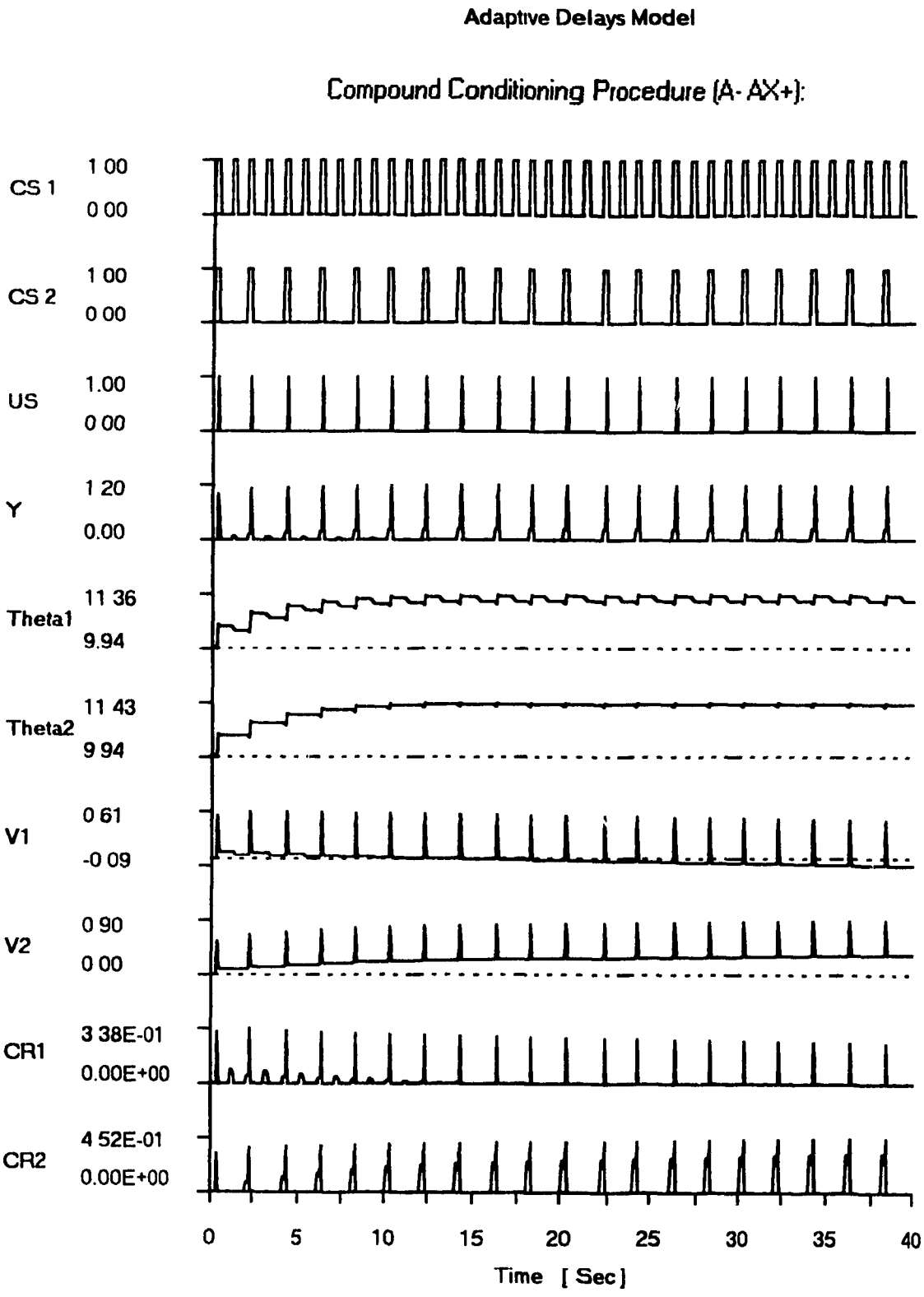

Figure 5.28 This compound conditioning procedure produces a stronger CR to X than to A. The CR to X (CR_2) reaches 0.34, but the CR to A (CR_1) is 0 at the end of training.

Temporal Conditioning:

In a temporal conditioning procedure, there is no distinctive CS Instead, the US is presented at regular intervals The two-neuron network of Figure 5 29 is an attempt to simulate this phenomenon The idea is that the first neuron produces a UR when a US is presented. The UR of the first unit becomes the CS of the second unit The second unit also receives the US as an input At a given time, the most recent US produces a UR that becomes a CS for the second unit, and that CS should produce a CR whose peak hopefully will be synchronized with the next US onset This would model temporal conditioning. Figure 5.30 presents the results of a simulation using such a two-neuron network The most recent CR can be considered as a prediction of the next US The CR is weak, in accordance with experimental data Unfortunately the peak of the CR does not coincide with the next US, but rather occurs in the middle of the delay between the two USs

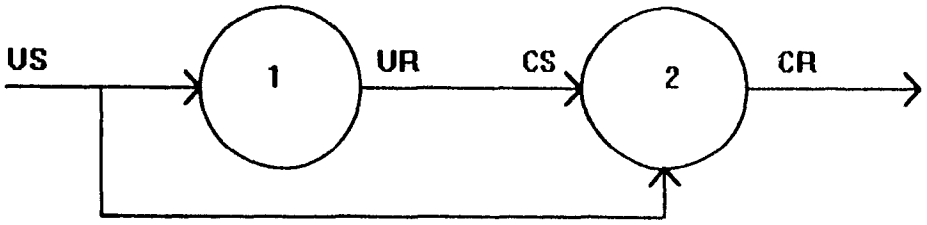

Figure 5.29 A two-neuron network to model the temporal conditioning paradigm There is no distinctive CS The first unit produces a UR when a US is presented This UR becomes the CS of the second unit

5.4 Predictions of Both Models

The DPC model and the AD model generate some original and testable predictions (for which I have no data) The following predictions are common to both models because they do not depend on adaptation of 0 as a function of time

1- Longer CSs have a faster extinction of their corresponding CR than shorter CSs This prediction is very different from Klopf's (1988) model, which predicts slower extinction for long CSs (long in comparision with τ in Equation I 12)

2- Simultaneous conditioning produces inhibition instead of a small excitatory conditioning or no conditioning at all However, the inhibitory effect ($\alpha_i(t)V_i(t)$) occurs after the US (for short USs with the DPC model)

3- The optimal ISI for short CSs is smaller than the optimal ISI for long CSs This is because the peak of $\alpha_i(t)$ occurs sooner after the onset of the CS for short CSs than for long CSs.

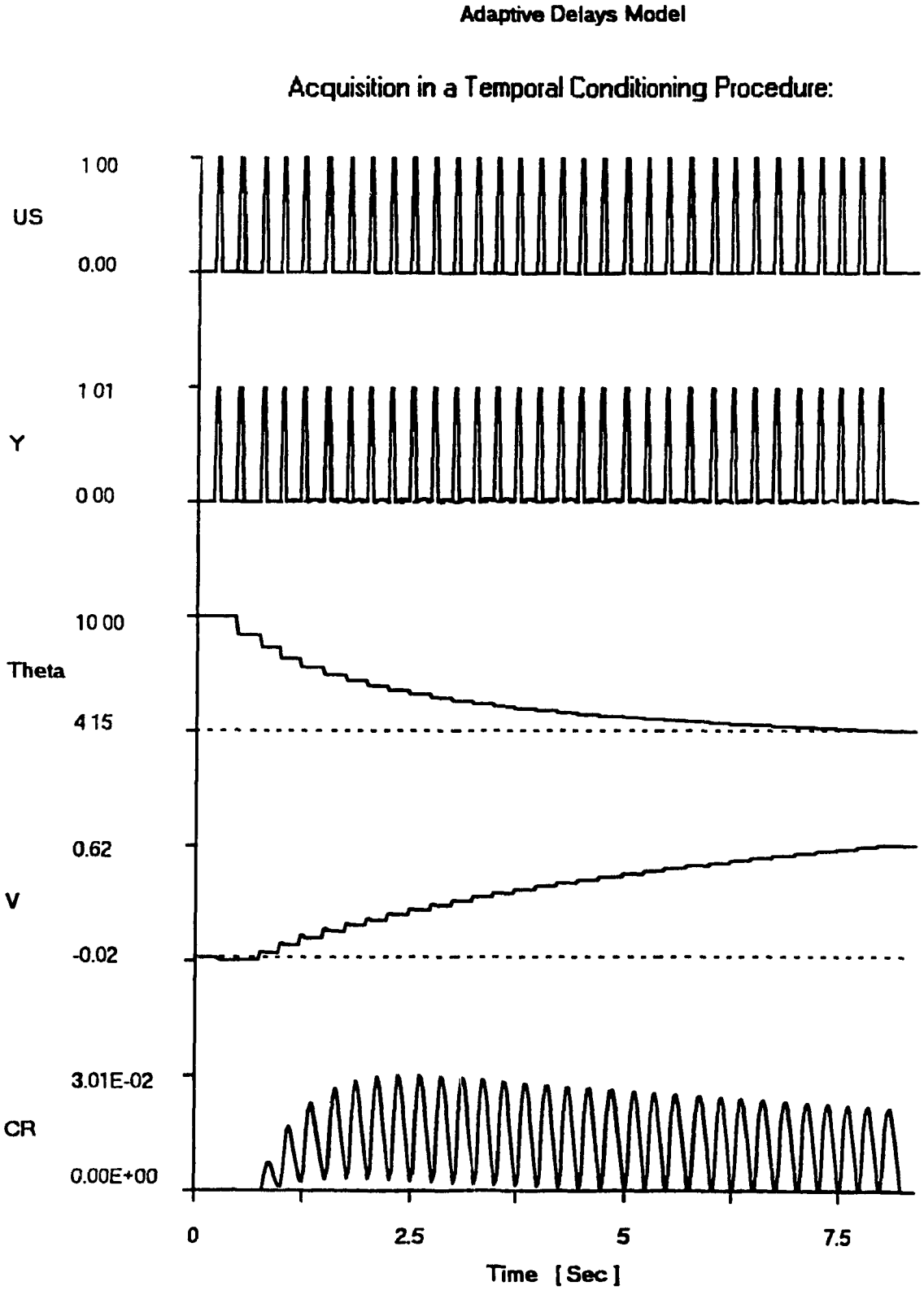

Figure 5.30 In a temporal conditioning procedure, there is no distinctive CS. Instead, the US is presented at regular intervals. The most recent CR can be interpreted as a prediction of the next US. However, the peak of each CR is in the midle of two USs.

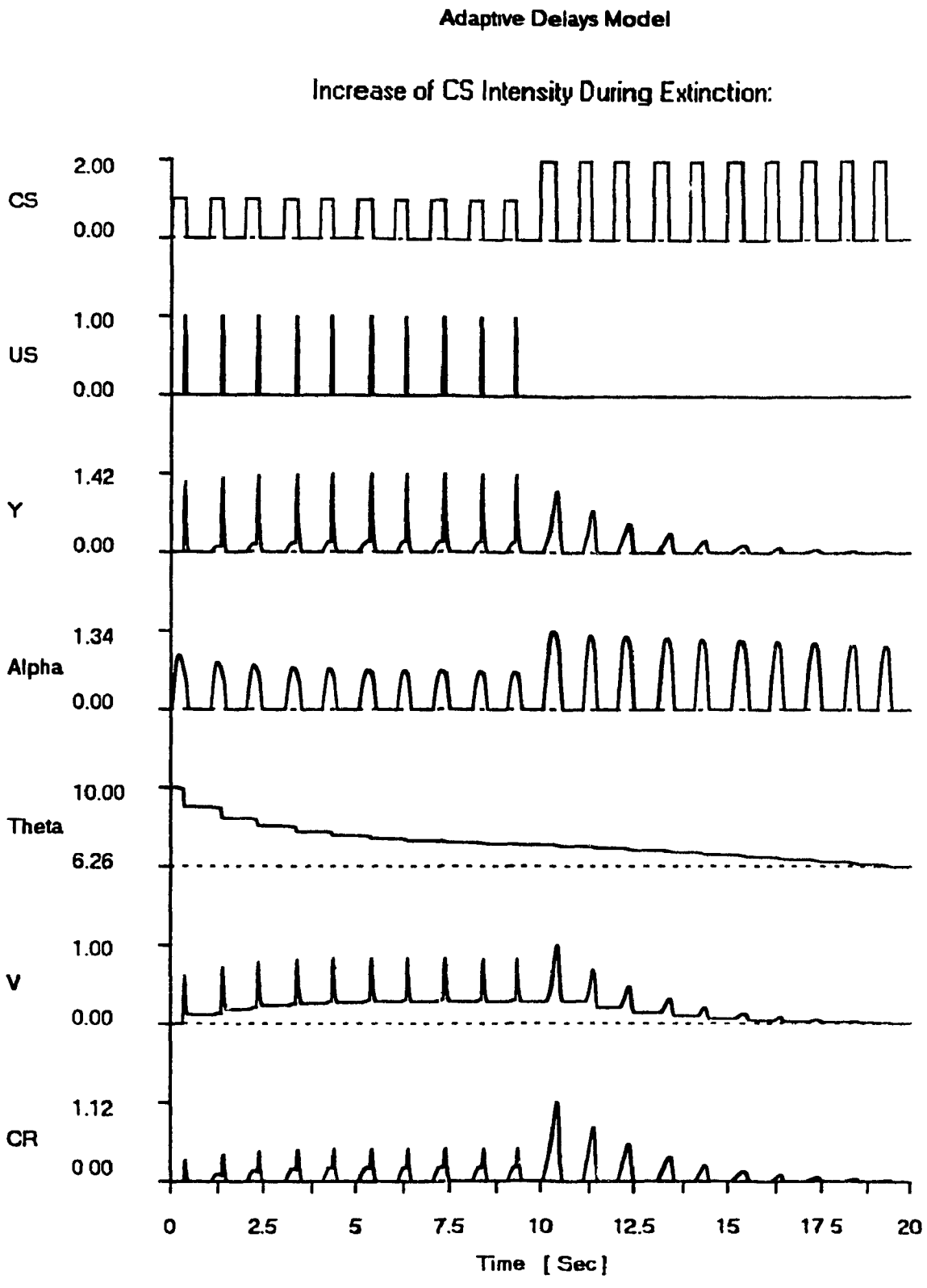

Figure 5.31 After acquisition, doubling the intensity of the CS should produce a stronger CR (1.12 instead of 0.2) and a faster extinction (see Figure 5.4 as comparison).

4- If in a first phase a CS is paired with a US and acquires the ability to elicit a CR, then an increase in the intensity of the CS in a second phase should increase the amplitude of the CR This phenomenon is represented in Figure 5 31 The model does not take into account the generalization decrement that should occur when one modifies the CS Several other models predict the same effect (Gluck-Thompson, 1987, Klopf, 1988, Wagner, 1981, Sutton-Barto, 1981) Generalization decrement will be opposed to that effect and should tend to decrease the CR If the generalization decrement is stronger than the increase of the CR due to intensity, at least this intensity effect should produce a skewness in the curve of the CR as a function of the intensity of the CS The skew should be in the direction of higher CS intensity.

The following predictions are specific to the AD model (i e for cases where $h \neq 0$ or $s \neq 0$) because they depend on the adaptation of θ as a function of time

1- The extinction process should modify the shape of the CR by increasing the delay between the onset of the CS and the peak of the CR This is caused by the habituation term in Equation 5 3

2- In some cases CS preexposure could in fact accelerate the acquisition of a CR This would happen if the initial peak of $\alpha_i(t)$ is much prior to the onset of the US If the right number of CS presentations are made before conditioning begins, the peak of $\alpha_i(t)$ would arrive closer to the onset of the US and learning would be faster

5.5 A Method for the Measure of the CS Trace

The theoretical shape of the curve of $\alpha_i(t)$ is derived from a number of experimental results. I have used the curve of the experimental CR, the curve of the strength of the CR as a function of the ISI, and have theorized about the differences between backward, simultaneous, trace and delay conditioning However, it would be preferable to find a way to measure $\alpha_i(t)$ directly This method can be deduced from Equation 4 8, which is

$$\Delta V_i(t) = \alpha_i(t-k)\, \beta\, (Y(t)-\bar{Y}(t-1)) \qquad (4\ 8)$$

I propose to use a long lasting CS, much longer than the optimal ISI The CS should last as long as an ISI that is too long to produce noticeable conditioning Klopf (1988) lets τ denote the longest ISI over which delay conditioning is effective If the CS duration is equal to t_1, then I require $t_1 > \tau$ The US duration should be even longer than the CS duration. If US duration is equal to t_1, then $t_2 > t_1$ A long CS makes it possible to capture the shape of $\alpha_i(t)$, without a possible perturbation that is hypothesized to occur at the CS offset A long US is required to eliminate the influence of the US offset Only a few trials of conditioning should be made in case the shape of $\alpha_i(t)$ is modified during

conditioning and also because the contribution of $\alpha_1(t-2)V_1(t)$ to $Y(t)$ should be negligeable compared with $\lambda(t)$. If all those conditions are met, then the curve of the strength of the CR, after only a few trials as a function of ISI, should be proportional to the curve of $\alpha_1(t-2)$ as a function of time This is shown in Figure 5.32, the curve of the strength of the CRs, obtained with the previously described procedure, is proportional to the curve of $\alpha_1(t-2)$

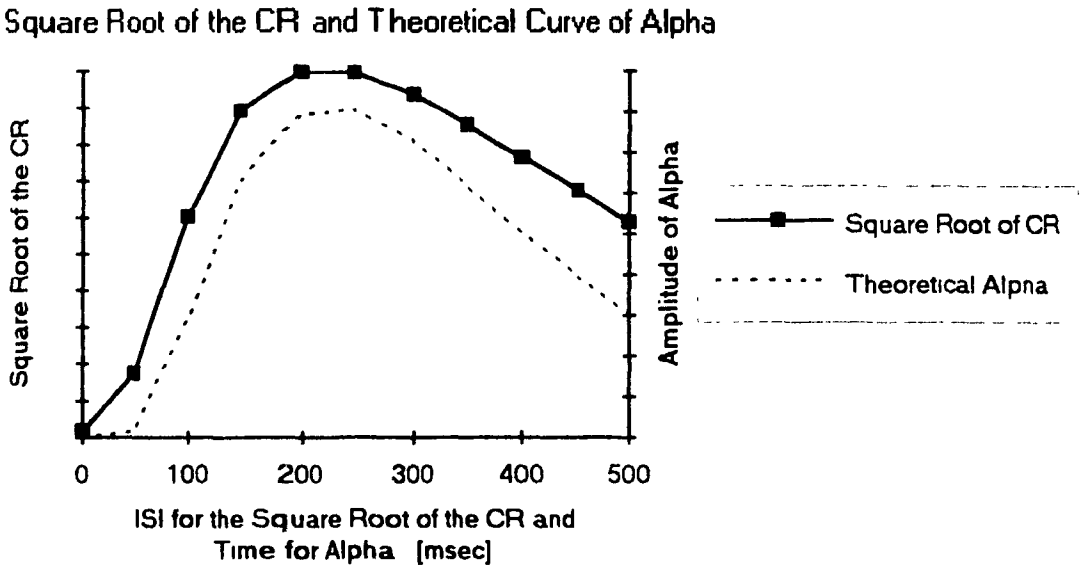

Figure 5.32 The curve of the square root of the strength of the CRs (obtained in a test trial after two CS-US pairing) as a function of ISIs is compared to the theoretical curve of $\alpha_1(t-2)$ as a function of time The CS had a duration of 1500 msec and the US had a duration of 3000 msec. The CR is in fact the result of a simulation using $\alpha_1(t-2)$ Both curves are not equal, they are proportional. This is why there are two Y scales.

This phenomenon can also be mathematically proven. We call ΔV_1 the total change of connection weight after one trial; λ_0 the US intensity; t_{cs} the time of CS onset; t_1 the CS duration; t_2 the US duration; and t_0 the time of the US onset. Thus, we have:

$$\Delta V_1 = \sum_{t=t_{cs}}^{t_0+t_2} \alpha_1(t-2)\,\beta\,(Y(t)-\overline{Y}(t-1))$$

However, $(Y(t)-\overline{Y}(t-1)) = 0$ between $t_{cs} < t < t_0$. Therefore, we obtain:

$$\Delta V_1 = \sum_{t=t_0}^{t_0+t_2} \alpha_1(t-2)\,\beta\,(Y(t)-\overline{Y}(t-1)).$$

We have hypothesized that $\alpha_1(t-2)V_1(t) << Y(t)$ This implies that $(Y(t)-\overline{Y}(t-1)) \cong 0$, when the US is present, i.e. for $(t_0+1) < t < (t_0 + t_2)$ At $t = t_0 + t_2$ we have $\alpha_1(t-2) \cong 0$. Thus:

$$\Delta V_i = \alpha_i(t_0\text{-}2)\,\beta\,(Y(t_0)\text{-}\overline{Y}(t_0\text{-}1))$$

Since US intensity is λ_0, then $Y(t_0)$ is equal to λ_0 and $\overline{Y}(t_0\text{-}1)\cong Y(t_0\text{-}1)\cong 0$ We finally get

$$\Delta V_i = \alpha_i(t_0\text{-}2)\,\beta\,\lambda_0$$

The strength of the CR after N trials is (for a small N)

$$\begin{aligned} CR &\cong Y(t_0\text{-}2) \qquad \text{when } \lambda(t)=0, \\ &\cong \alpha_i(t_0)\,N\,\Delta V_i\,, \\ &= N\,\alpha_i(t_0)\alpha_i(t_0\text{-}2)\,\beta\,\lambda_0\,, \\ &\cong N\,\beta\,\lambda_0\,[\alpha_i(t_0\text{-}2)]^2 \end{aligned}$$

This implies that the strength of the CR after one trial is proportional to the square of $\alpha_i(t_0\text{-}2)$, where $\alpha_i(t_0\ 2)$ is the value of $\alpha_i(t\text{-}2)$ after a delay equal to the ISI following CS onset. This method does not give the exact value of $\alpha_i(t\text{-}2)$, but it gives its shape

6. DISCUSSION

The AD model is in agreement with experimental data in a number of areas Thus, it accurately reproduces the phenomenon of inhibition of delay, the shape of the CR is a skewed right bell shape positively accelerating in the neighborhood of the onset of the CS, the CR of long CSs extinguishes (as opposed to Klopf's (1988) model), the optimal ISI for short trace CSs is shorter than the optimal ISI for long delay CSs; and several other properties (see Table D 1 in Appendix D for a summary) The AD model is in partial agreement with behavioral data concerning the CS preexposure effect, the sigmoid curve of acquisition of a CR and temporal conditioning However, the AD model has encountered a few problems in regard to other procedures of classical conditioning Low values had to be attributed to s, h, and β in order to reproduce the behavioral data regarding blocking, pseudoconditioning, and the US intensity effects, in contrast to higher values used for all other properties The gain obtained by introducing delays of variable duration at the level of the connections is offset by the loss of other properties of classical conditioning Furthermore, to build the AD model certain assumptions were introduced, some of them post hoc such as for the sensitization term for $\theta_i(t)$ I also had to include new constants and variables. In other words, the ratio of benefit over cost is too low in the case of the AD model (see Table D 2 in Appendix D)

Chapter 5 may be considered as an extended proof by contradiction I posited that the amplitude and the peak of the trace of the CS_i $[\alpha_i(t)]$ vary in parallel during conditioning The results are ambigous. We are left with the following set of possibilities. it is possible that neither the amplitude nor the peak of $\alpha_i(t)$ vary, or that only the amplitude or only the peak of $\alpha_i(t)$ vary, or finally that they both vary independently Certain results of the simulations provide clues that might help solve this problem The phenomenon of blocking was not explained by the AD model, but it was explained by the DPC model Let us call CS_1 the CS that is reinforced in the first phase of a blocking procedure, and CS_2 the CS that is reinforced in conjunction with the CS_1 in the second phase The reason why blocking is not predicted by the AD model is that the CS_1 increased the delay (T_p) between the onset of the CS_1 and the peak of the CR_1, in conformity with inhibition of delay However, in doing so, $\alpha_1(t)$ and $\alpha_2(t)$ were no longer simultaneous in the second phase, the peak of $\alpha_1(t)$ occurring after the peak of $\alpha_2(t)$. Thus, the CR_1 was acting as a second-order reinforcement in regard to the CS_2. Instead of blocking, there was potentiation of conditioning It therefore seems difficult to implement the presence of variable delays at the level of the connection and still account

for the phenomenon of blocking Thus, two possibilities remain either the amplitude of $\alpha_i(t)$ varies or it does not This question is examined a little further on

The delay-producing connections model, on the other hand, is in accordance with experimental data for the following phenomena the shape of the CR is a skewed right bell shape positively accelerating in the neighborhood of the onset of the CS, the CR of long CSs extinguishes, the optimal ISI for short trace CSs is shorter than the optimal ISI for long delay CSs, and several other properties of classical conditioning (see Table D 1 of Appendix D for details) Furthermore, when I encountered problems with the AD model in regard to the phenomena of blocking, pseudodiscrimination, and the US intensity effects, I had to set s and h at zero in order to simulate these properties When s and h are equal to zero, the AD model becomes equivalent to the DPC model The DPC model is more robust in accounting for the characteristics of classical conditioning The DPC model makes use of fewer assumptions than the AD model In other words, the ratio of benefit over cost for the DPC model is higher than for the AD model

How can we improve the DPC model? Several avenues could be taken for future research. One avenue would be, as discussed earlier, to add an habituation of $\alpha_i(t)$ to the CS and a sensitization of $\alpha_i(t)$ to the US, i e a variation of the amplitude of $\alpha_i(t)$, without a change in the position of its peak This assumption could account for the CS preexposure effects If a CS is presented repeatedly without reinforcement, the amplitude of $\alpha_i(t)$ would decrease and the acquisition of a CR should be slower afterward If we add a spontaneous recovery of the habituated response, i.e if we suppose that $\alpha_i(t)$ returns to its original amplitude after an habituation because of the mere passage of time, this could account for the phenomenon of spontaneous recovery of the CR The spontaneous recovery of an habituated response is a common property of habituation (Doré, 1988, Kandel & Schwartz, 1985, Thompson & Spencer, 1966) These two assumptions would also explain why massed extinction is faster than spaced extinction In a spaced extinction, there would be almost no habituation, i e no decrease of $\alpha_i(t)$, only $V_i(t)$ would decrease, given that when the delay between two CS presentations is long, $\alpha_i(t)$ returns to its original value by spontaneous recovery A massed extinction, however, would produce a strong habituation, i e a strong diminution of $\alpha_i(t)$ Since the amplitude of the CR_i is the result of the product of $\alpha_i(t)$ by $V_i(t)$, the decrease of the CR would be faster with massed extinction than it would with spaced extinction These assumptions would also explain why massed acquisition is slower than spaced acquisition Because of habituation, $\alpha_i(t)$ would be smaller in a massed acquisition than in a spaced acquisition and acquisition should be slower It remains to be seen whether

the simulation of these latter hypotheses has any undesirable effects on the predictions of the model for other properties of classical conditioning

To explain inhibition of delay with the DPC model is more straightforward Using the principle of delay lines, we can assume that for one CS a neuron receives several connections, and each connection has its own time course, i e its own value of $(T_p)_k$ (thus of θ_k) The connections reaching their peak closer to the US onset will overshadow the other connections whose peak is farther from the US onset The peak of the CR being equal to the sum of $\alpha_i(t)V_i(t)$ for each connection, its peak will be close to the US onset The DPC model seems to offer an interesting method of implementing a delay line

The acquisition curve of the DPC model is negatively accelerating and is not a sigmoid In order to account for the initial positive acceleration in the S-shape acquisition curves observed in animal learning, several authors use the same method (Frey & Sears, 1978, Gluck & Thompson, 1987, Klopf, 1988) They simply multiply their learning rule by the connection weight ($V_i(t)$) For example, Klopf (1988) used the following equation:

$$\Delta V_i(t) = \Delta Y(t) \sum_{k=1}^{\tau} C_k |V_i(t-k)| \Delta X_i(t-k) \qquad (1\ 12)$$

If $\Delta V_i(t)$ is proportional to $|V_i(t-k)|$, then for low values of $|V_i(t-k)|$ learning is slow When $|V_i(t-k)|$ increases, learning is faster, this corresponds to the positively accelerating section of the acquisition curve If $|V_i(t-k)|$ increases even more, then $\Delta Y(t)$ decreases and compensates for the increase of $|V_i(t-k)|$ and this is the negatively accelerating section of the acquisition curve However, $|V_i(t-k)|$ cannot be equal to zero, otherwise no learning would occur This means that $|V_i(t-k)|$ must have an initial and a minimum value different from zero This implies that one must use inhibitory connections and excitatory connections, i e connection weights that are either negative or positive When a connection weight is negative it stays negative, and when a connection weight is positive it stays positive This restriction doubles the number of connections and consequently the number of equations necessary to account for the properties of classical conditioning This is why this possibility was avoided in the DPC model However, having inhibitory connections that are permanently inhibitory and excitatory connections that are permanently excitatory, is more physiologically realistic (Klopf, 1988, Kuffler, Nicholls, & Martin, 1984) Thus, to multiply the learning rule by $|V_i(t-k)|$, makes reproduction of interesting properties possible, but has a high cost Another possibility can be investigated. It would be simpler to multiply the learning rule by $(v + |V_i(t-k)|)$, where v is a positive constant As this term will never be equal to zero, it would not be necessary to use connections that are exclusively excitatory or

exclusively inhibitory This would not double the number of equations, it would simply introduce another constant (i e v)

Another problem encountered by the DPC model is that the curve of the asymptotic strength of the CR as a function of the ISI (see Figure 4 11) decreases more slowly than the experimental curve This is due to the fact that the curve of $\alpha_i(t)$ for a CS decreases too rapidly This problem can be solved in two ways One way is to use the suggestion made to explain inhibition of delay, which is to have several connections for each CS, each connection having its own value of $(T_p)_k$ In this way, some connections will have higher values for $(T_p)_k$, the CR will decay more slowly and the curve of the asymptotic strength of the CR as a function of the ISI will decay more slowly Another way would be to find another equation for $\alpha_i(t)$, that would decay more slowly than the CS This equation would replace Equation 4 3

The New and the AD models account for facilitating CRs, i e CRs that mimic the US Compensatory CRs are left unexplained by the models There are two domains in which compensatory CRs are more frequent CRs to drugs (Greeley, 1984, Hinson et al , 1982, Siegel & Cunninghan, 1980, Obrist, Suterer, & Howard, 1972, Siegel, 1982) and affective CRs (Solomon & Corbit, 1980, 1974, Wagner, 1981, Wagner & Brandon, 1989) In order to take into account the fact that a compensatory CR is opposed to the UR, we could replace the activation rule

$$Y(t) = f\Big(\lambda(t) + \sum_{i=1}^{n} [\alpha_i(t)\, V_i(t)]\Big), \tag{4 6}$$

with the following rule

$$Y(t) = f\Big(\lambda(t) + r \sum_{i=1}^{n} [\alpha_i(t)\, V_i(t)]\Big), \tag{6 1}$$

where r is a constant equal to 1 for facilitating CRs and equal to -1 for compensatory CRs Wagner and Brandon (1989) suggested that there are two distinct UR sequences — a sensory sequence and an emotive one The sensory and emotive attributes of a US activate separate sequences of activity Further, the latency of the sensory and emotive activity sequences can differ in that some timing properties of the emotive sequence are slower than the corresponding timing properties of the sensory sequence Consequently, the shape of the curve of a compensatory CR differs from that of a facilitating CR The typical UR of an emotional US (Solomon & Corbit, 1980, 1974) looks like Figure 6 1, i.e the UR increases rapidly, reaches a peak, and then decreases even when the US is still present This is the case for example with the heart rate of a dog as a function of time, when shocks are used as US (Solomon & Corbit, 1980, 1974) When $\sum_{i=1}^{n} [\alpha_i(t) V_i(t)] = 0$,

Y(t) is supposed to be equal to the UR, i e $Y(t) = f(\lambda(t))$ Typically $\lambda(t)$ is a square wave, so Y(t) is a square wave too However, the UR in Figure 6.1 is not a square wave This means that we should replace $\lambda(t)$ by a function of $\lambda(t)$, a function that would have the shape of a typical UR This is the same kind of rationale that made me replace $X_i(t)$ by $\alpha_i(t)$ For example, we could replace $\lambda(t)$ by $(\lambda_a(t)-\lambda_b(t))$, where $\lambda_a(t)$ is the excitatory process of $\lambda(t)$ and $\lambda_b(t)$ is the opponent process of $\lambda(t)$ The activation rule becomes.

$$Y(t) \quad f\left(\lambda_a(t)-\lambda_b(t) + r \sum_{i=1}^{n} [\, \alpha_i(t)\, V_i(t)] \right) \tag{6 1}$$

Some possible equations for $\lambda_a(t)$ and $\lambda_b(t)$ are

$$\frac{d}{dt}\lambda_a(t) = K_a(\lambda(t) - \lambda_a(t)), \tag{6.2}$$

and $$\frac{d}{dt}\lambda_b(t) = K_b(\lambda(t) - \lambda_b(t)), \tag{6 3}$$

where K_a is a time constant that governs the rate of variation of the excitatory process;

K_b is a time constant that governs the rate of variation of the opponent process,

and $K_b < K_a$

With a facilitating CR, K_a would be very high and K_b would be zero Thus, we obtain: $(\lambda_a(t)-\lambda_b(t)) \cong (\lambda(t)-0) = \lambda(t)$ However, Equations 6 1, 6 2 and 6 3 have to be tested.

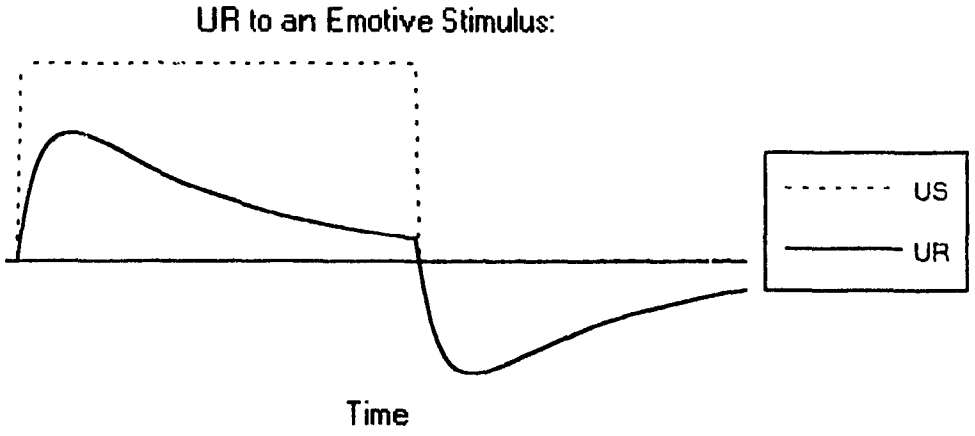

Figure 6.1 General shape of a UR to a square wave emotive US as a function of time For example, the US could be a shock and the UR could be a heart rate

The DPC model does not account for more complex forms of configural learning, for example. positive patterning, negative patterning, and feature positive procedure. Such phenomena can be accounted for by a network model, not by a single unit model

(Kehoe, 1989) However, the delay-producing connections model or the AD model could be implemented at the network level to simulate these phenomena

Some phenomena lead one to think that the rules of classical conditioning might apply to cases of learning where there is no reinforcer The phenomenon of sensory preconditioning and the McCullough effect in vision are two examples of such phenomena In sensory preconditioning, two neutral stimuli CS_1 and CS_2 are paired prior to reinforcement In a second phase, the CS_1 is paired with a US The CS_1-US pairing results in the ability of the CS_2, as well as the CS_1, to elicit the CR Extinction of the CR_1 also extinguishes the CR_2 (Rizley & Rescorla, 1972) This can be explained if we suppose that an association has been created between a sensory representation of the CS_1 and a sensory representation of the CS_2, with this association occurring before the presentation of the reinforcer In the visual system, opponent color responses can come to be elicited by achromatic stimuli which have been paired with chromatic stimuli (McCullogh, 1965, cited by Schull, 1979), this is called the McCullogh effect Schull (1979) provides the following example of this effect "If [ ] one spends a few minutes viewing black vertical stripes [ ] superimposed upon a bright red background [ ], the stripes acquire a long-lasting ability to elicit an opponent response [ ] hours or days later, long after simple afterimages have dissipated, when one views vertical black and white stripes they appear black and light green " Again no reinforcer is present, yet both cases are a form of associative learning as is classical conditioning Therefore, it is possible that the learning rules of classical conditioning might apply to a vast domain of phenomena These examples of associative learning without a reinforcer suggest that it could make sense to use real-time learning rules of classical conditioning (like the DPC learning rule) in neural networks and to apply these networks to perceptual or cognitive problems

7. REFERENCES

Ader, R & Cohen, N (1982) Behaviorally conditioned immunosuppression and murine systemic lupus erythematosus *Science*, **215**, 1534-1536

Ader, R & Cohen, N (1985) CNS-immune system interactions Conditioning phenomena *Behavioral and Brain Sciences*, **8**, 379-426

Adrian E D (1946) *The Physical Background of Perception* Oxford, UK: Clarendon Press

Anderson, J A & Rosenfeld, E (Eds) (1988) *Neurocomputing: Foundations of research. Cambridge*, MA MIT Press

Baird III, L C & Klopf, A H (1992) Extensions of the Associative Control Process (ACP) Network Hierarchies and Provable Optimality *Proceedings of the Second International Conference on Simulation of Adaptive Behavior*, Honolulu, Hawaii, December

Baird III, L C & Klopf, A H (1993) A Hierarchical Network of Provably Optimal Learning Control Systems Extensions of the Associative Control Process (ACP) Network *Adaptive Behavior*, **1**, 321-352

Baker, A G & Mackintosh, N J. (1977) Excitatory and inhibitory conditioning following uncorrelated presentations of the CS and US. *Animal learning and behavior*, **5**, 315-319

Baker, A G & Mercier, P (1989) Attention, retrospective processing and cognitive representations In S B Klein & R R Mowrer (Eds) , *Contemporary learning theories: Pavlovian conditioning and the status of traditional learning theory* (pp 85-101) Hillsdale, N J. Erlbaum

Baldwin, J D & Baldwin, J I (1981) *Beyond Sociobiology* New York, N Y.. Elsevier

Bandura, A. (1977) Self-efficacy Toward a unifying theory of behavior change *Psychological Review*, **84**, 191-215

Barnes, G W (1956) Conditioned stimulus intensity and temporal factors in spaced-trial classical conditioning *Journal of Experimental Psychology*, **51**, 192-198.

Bechterev, V M (1913) Objektive Psychologie oder Psychoreflexologie. *Die Lehre von der Assoziationsreflexen Teubner.*

Bouchard, C (1974) *Effets de la rétroaction binaire positive et négative sur la régulation du rythme cardiaque* Ph D. Thesis, Department of Psychology, McGill University, Montreal

Bouton, M E (1991) Context Time and Memory Retrieval in the Interference Paradigm of Pavlovian Learning *Psychological Bulletin*, **114**. 80-99

Burkhardt, P E & Ayres, J J B (1978) CS and US duration effects in one-trial simultaneous fear conditioning as assessed by conditioned suppression of licking in rats *Animal Learning & Behavior*, **6**, 225-230

Bush, R R & Mosteller, F (1955) *Stochastic Models for Learning* New York, N Y Wiley

Byrne, J H (1987) Cellular analysis of associative learning *Physiological Reviews*, **67**, 329-439

Carew, T J , Hawkins, R D , & Kandel, E R (1983) Differential classical conditioning of a defensive withdrawal reflex in Aplysia californica *Science*, **219**, 397-400

Desmond, J E (1990) Temporal adaptive responses in neural models the stimulus trace In M. Gabriel & J W Moore (Eds), *Learning and Computational Neuroscience* (pp 421-456) Cambridge, MA MIT Press

Desmond, J E , & Moore, J W (1988) Adaptive timing in neural networks the conditioned response *Biological Cybernetics*, **58**, 405-415

Desmond, J E & Moore, J W (1991) Altering the synchrony of stimulus trace processes tests of neural-network model *Biological Cybernetics*, **65**, 161-169

Dickinson, A., Hall, G & Mackintosh, N J (1976) Surprise and the attenuation of blocking *Journal of Experimental Psychology, Animal Behavior Processes*, **2**, 313-322

Domjan, M. & Best, M R (1977) Paradoxical effects of proximal unconditioned stimulus preexposure Interference with and conditioning of a taste aversion *Journal of Experimental Psychology, Animal Behavior Processes*, **3**, 310-321

Donegan, N H , Gluck, M A & Thompson, R F (1989) Integrating Behavioral and Biological Models of Classical Conditioning *The Psychology of Learning and Motivation*, **23**, 109-156

Doré, F Y. (1988) *L'apprentissage une approche psycho-éthologique* St-Hyacinthe Edisem

Fan, D P & Elketroussi, M (1989) Mathematical Model for Addiction Application to Multiple Risk Factor Intervention Trial Data for Smoking *Journal of Consulting and Clinical Psychology*, **57**, 456-459

Feldman, J. A (Ed) (1985) Special issue on connectionist models and their applications , *Cognitive Science*, **9**, 1-169

Flaherty, C F (1985) *Animal learning and cognition* New York, N Y Knopf

Frey, P. W (1969) Within- and between-session CS intensity performance effects in rabbit eyelid conditioning *Psychonomic Science*, **17**, 1-2

Frey, P W & Sears, R J (1978) Model of Conditioning Incorporating the Rescorla-Wagner Associative Axiom, a Dynamic Attention Process, and a Catastrophe Rule *Psychological Review*, **85**, 321-340

Garcia, J, Ervin, F R & Koelling, R A (1966) Learning with prolonged delay of reinforcement *Psychonomic Science*, **7**, 121-122

Garcia, J, & Rusiniak, K W (1980) What the nose learn from the mouth In D Muller-Schwarze & R M Silverskin (eds), *Chemical Senses* New York, N Y Plenum

Ghanta, V K , Hiramoto, R N , Solvason, H B. & Spector, N H (1985) Neural and environmental influences on neoplasia and conditioning of NK activity *Journal of Immunology*, **135**, 848-852

Gluck, M A & Bower, G (1988) Evaluating an adaptive network model of human learning *Journal of Memory and Language*, **27**, 166-195

Gluck, M A & Granger, R (1993) Computational models of the neural bases of learning and memory *Annual Review of Neuroscience* **16**, 667-706

Gluck, M A , Parker, D B & Reifsnider, E (1988) Some biological implications of a differential-Hebbian learning rule *Psychobiology*, **16**, 298-302

Gluck, M A & Thompson, R F (1987) Modeling the Neural Substrates of Associative Learning and Memory A Computational Approach *Psychological Review* **94**, 176-191

Gormezano, I , Kehow, E J & Marshall, B S (1983) Twenty Years of Classical Conditioning Research with the Rabbit *Progress in Psychobiology and Physiological Psychology*, **10**, 197-275

Greeley, J , Lê, D A , Poulos, C X & Cappell, H (1984) Alcohol is an effective cue in the conditional control of tolerance to alcohol *Psychopharmacology*, **83**, 159-162

Grice, G R & Hunter, J J (1964) Stimulus intensity effects depend upon the type of experimental design *Psychological Review*, **71**, 247-256

Grossberg, S & Schmajuk, N A (1987) Neural dynamics of attentionally modulated Pavlovian conditioning Conditioned reinforcement, inhibition, and opponent processing *Psychobiology*, **5**, 195-240

Hawkins, R D , Carew, T J , & Kandel, E R (1986) Effects of interstimulus interval and contingency on classical conditioning of the Aplysia siphon withdrawal reflex *Journal of Neuroscience*, **6**, 1695-1701

Hawkins, R D & Kandel, E R Is There a Cell-Biological Alphabet for Simple Forms of Learning? *Psychological Review*, **91**, 375-391

Hebb, D O (1949) *Organization of Behavior*, New York, N Y Wiley.

Hilgard, E R & Marquis, D G (1940) *Conditioning and Learning* New York, N Y Appleton- Century-Crofts

Hughes, B & Schlosberg, H (1938) Conditioning in the white rat IV The conditioned lid reflex. *Journal of Experimental Psychology*, **23**, 641-650

Hull, C L (1943) *Principles of Behavior*, Englewood Cliffs, N J Prentice-Hall

Hull, C L (1952) *A behavior system. An introduction to behavior theory concerning the individual organism* New Haven Conn Yale University Press

Hinson, R E, Poulos, C X & Cappell, H (1982) Effects of pentobarbital and cocaine in rats expecting pentobarbital *Pharmacology Biochemistry & Behavior*, **16**, 661-666

Kamin, L J (1968) "Attention-like" processes in classical conditioning In Jones, M R (Ed), *Miami symposium on the prediction of behavior aversive stimulation*, Miami, Fla University of Miami Press

Kandel, E R & Schwartz, J H (1985) *Principles of Neural Science* New York, N Y Elsevier

Kehoe, E J (1989) Connectionist Models of Conditioning A Tutorial *Journal of the Experimental Analysis of Behavior* **52**, 427-440

Kehoe, E J (1992) More Than Meets the Eye Animal Learning and Knowledge Induction *13th Machine Intelligence Conference*, Ross Priory, Scotland 1-31

Kimble, G A & Ost, J W P (1961) A conditioned inhibitory process in eyelid conditioning *Journal of Experimental Psychology*, **61**, 150-156

Kimmel, H D (1965) Instrumental inhibitory factors in classical conditioning In W F Prokasy (Ed), *Classical conditioning A symposium* New York, N Y Appleton-Century-Crofts, (pp 148-171)

Klein, S B (1991) *Learning* Second Edition New York, N Y McGraw-Hill

Klopf, A H (1972) Brain function and adaptive systems A heterostatic theory *Air Force Research Laboratories Research Report*, AFCRL-72-0164

Klopf, A H (1982) *The Hedonistic Neuron A theory of memory, learning, and intelligence* Washington, D C Hemisphere Publishing Corporation

Klopf, A. H (1986) Drive-reinforcement model of single neuron function An alternative to the Hebbian neuronal model In J Denker (Ed), *AIP Conference Proceedings 151: Neural Networks for Computing* New York, N Y American Institute of Physics, (pp 265-270)

Klopf, A H (1988) A neuronal model of classical conditioning *Psychobiology* **16**, 85-125

Klopf, A H (1992) Modeling Nervous System Function with a Hierarchical Network of Control Systems that Learn *Proceedings of the Second International Conference on Simulation of Adaptive Behavior*. Honolulu, Hawaii

Klopf, A H, Morgan, J S & Weaver, S E (1993) A Hierarchical Network of Control Systems that Learn Modeling Nervous System Function During Classical and Instrumental Conditioning *Adaptive Behavior*, **1**, 263-319

Klosterhalfen, W & Klosterhalfen, S (1983) Pavlovian conditioning of immmunosuppression modifies adjuvant arthritis in rats *Behavioral Neuroscience*, **97**, 663-666

Konorski, J (1967) *Integrative activity of the brain* Chicago, Ill University of Chicago Press

Kosko, B (1986) Differential Hebbian Learning, In J. Denker (Ed), *AIP Conference Proceedings 151: Neural Networks for Computing*, (pp 277-282) New York, N Y American Institute of Physics

Kuffler, S W, Nicholls, J G & Martin, A R (1984) *From Neuron to Brain.* Second Edition Sunderland, MA Sinauer Associates Inc Publishers

Lacouture, Y & Marley, A A J. (1991) A Connectionist Model of Choice and Reaction Time in Absolute Identification *Connection Science*, **3**, 401-433

Lang, W J, Brown, M L, Gershon, S & Korol, B (1966) Classical and physiologic adaptive conditioned responses to anticholinergic drugs in conscious dogs. *International Journal of Neuropharmacology*, **5**, 311-315

Leemon C B III, & Klopf, A H (1992) Extensions of the Associative Control Process (ACP) Network Hierarchies and Provable Optimality *Proceedings of the Second International Conference on Simulation of Adaptive Behavior*, Honolulu, Hawaii

Lubow, R E & Moore, A V (1959) Latent inhibition the effect of nonreinforced preexposure of the conditioned stimulus *Journal of Comparative and Physiological Psychology*, **52**, 415-419

Mackintosh, N J (1974) *The Psychology of Animal Learning.* New York, N Y Academic Press

Mackintosh, N J (1975) A theory of attention: variations in the association of stimuli with reinforcement *Psychological Review*, **82**, 276-298

Mackintosh, N J, Bygrave, D J & Picton, D M B (1977) Locus of the effect of a surprising reinforcer in the attenuation of blocking *Quarterly Journal of Experimental Psychology*, **29**, 327-336

MacRae, J R & Siegel, S (1987) Extinction of tolerance to the analgesic effect of morphine Intracerebroventricular administration and effects of stress *Behavioral Neuroscience*, **101**, 790-796

Mansfield, J G & Cunningham, C C (1980) Conditioning and extinction of tolerance to the hypothermic effect of ethanol in rats *Journal of Comparative and Physiological Psychology*, **94**, 962-969

Marchant, H G , III, & Moore, J W (1974) Below-zero conditioned inhibition of the rabbit's nictitating membrane response *Journal of Experimental Psychology*, **102**, 350-352

Mayer, J (1953) Genetic, traumatic, and environmental factors in the etiology of obesity *Psychological Review*, **33**, 472-508

McCullough, C Color adaptation of edge-detectors in the human visual system *Science*, **149**, 1115-1116

McCulloch, W & Pitts, W (1943) A logical calculus of the ideas immanent in nervous activity, *Bulletin of Mathematical Biophysics*, **7**, 115-133

Mineka, S , Cook, M & Miller, S (1984) Fear conditioned with escapable and inescapable shock Effects of a feedback stimulus *Journal of Experimental Psychology: Animal Behavior Processes*, **10**, 307-323

Monti, P M. & Smith, N F (1976) Residual fear of the conditioned stimulus as a function response prevention after avoidance or classical defensive conditioning in the rat *Journal of Experimental Psychology: General*, **105**, 148-162

Morgan, J S , Patterson, E C & Klopf, A H (1990) Drive-reinforcement learning a self-supervised model for adaptive control *Network*, **1**, 439-448

Morin, P. P , Verrette, J L., Dodson, J J & Doré, F Y (1987) Computer automated method to study cardiac conditioning to a chemical cue in young salmon *Physiology & Behavior*, **39** 657-666

Obrist, P A , Sutterer, J. R & Howard, J L (1972) Preparatory cardiac changes A psychobiological approach In A H. Black & W F Prokasy (Eds), *Classical Conditioning II* New York, N Y Appleton

Pavlov, I. P (1927) *Conditioned Reflexes* Oxford Oxford University Press (Dover Edition, 1960)

Pearce, J M & Hall, G (1980) A Model for Pavlovian Learning Variations in the Effectiveness of Conditioned But Not of Unconditioned Stimuli *Psychological Review*, **87**, 532-552

Perruchet, P (1979) Conditionnement classique chez l'homme et facteurs cognitifs I Le conditionnement végétatif. *L'année psychologique*, **79** 527-557

Poulos, C X , Wilkinson, D A & Cappell, H (1981) Homeostatic regulation and Pavlovian conditioning in tolerance to amphetamine-induced anorexia *Journal of Comparative and Physiological Psychology*, **95**, 735-746

Prokasy, W F , Jr , Grant, D A & Meyers, N A (1958) Eyelid conditioning as a function of unconditioned stimulus intensity and intertrial interval *Journal of Experimental Psychology*, **55**, 242-246

Randich, A & LoLordo, V M (1979) Associative and nonassociative theories of the UCS preexposure phenomenon implications for Pavlovian conditioning, *Psychological Bulletin*, **86**, 523-548

Rescorla, R A (1969) Pavlovian conditioned inhibition *Psychological Bulletin*, **72**, 77-94

Rescorla, R A , & Wagner, A R (1972) A theory of Pavlovian conditioning Variations in the effectiveness of reinforcement and nonreinforcement In A. H Black & W F Prokasy (Eds), *Classical conditioning II: Current research and theory* New York, N.Y Appleton-Century-Crofts

Rizley, R C & Rescorla, R A (1972) Associations in second-order conditioning and sensory preconditioning *Journal of Comparative and Physiological Psychology*, **81**, 1-11

Rumelhart, D E , McClelland, J L & the PDP Research Group (1986) *Parallel distributed processing: Explorations in the microstructure of cognition. Vol 1: Foundations* Cambridge, MA MIT Press/Bradford Books

Russell, M., Dark, K , Cummins, R , Ellman, G Callaway, E & Peeke, H. V S (1984) Learned histamine release *Science*, **225**, 733-734

Schmajuk, N A & Moore, J. W (1989) Effects of hippocampal manipulations on the classically conditioned nictitating membrane response simulations by an attentional-associative model *Behavioural Brain Research*, **32**, 173-189

Schneiderman, N Interstimulus interval function of the nictitating membrane response of the rabbit under delay versus trace conditioning. *Journal of Comparative and Physiological Psychology*, **62**, 397-402.

Schull, J (1979) A Conditioned Opponent Theory of Pavlovian Conditioning and Habituation *The Psychology of Learning and Motivation*, **13**, 57-90.

Schnur, P & Martinez, R A (1989) Environmental control of morphine tolerance in the hamster *Animal Learning and Behavior*, **17**, 322-327.

Shanks, D R. (1991) Categorization by a Connectionist Network. *Journal of Experimental Psychology: Learning, Memory, and Cognition* **17**, 433-443

Shapiro, M L. & Caramanos, Z (1990) NMDA antagonist MK-801 impairs acquisition but not performance of spatial working and reference memory *Psychobiology* **18**, 231-243

Shipley, R H (1974) Extinction of conditioned fear in rats as a function of several parameters of CS exposure *Journal of Comparative and Physiological Psychology*, **87**, 699-707

Shultz, T R & Schmidt, W C (1991) A Cascade-Correlation Model of Balance Scale Phenomena In *Proceedings of the Thirteenth Annual Conference of the Cognitive Science Society* (pp 635-640) Hilldale, N J. Erlbaum

Siegel, S. (1972) Conditioning of insulin-induced glycemia *Journal of Comparative and Physiological Psychology*, **78**, 233-241

Siegel, S. (1975) Conditioning insulin effects. *Journal of Comparative and Physiological Psychology*, **89**, 189-199

Siegel, S (1977) Morphine tolerance acquisition as an associative process *Journal of Experimental Psychology: Animal Behavior Processes*, **3**, 1-13

Siegel, S. (1988) Drug anticipation and the treatment of dependence *National Institute on Drug Abuse Research Monograph Series*, **84**, 1-24

Simpson, P K (1990) *Artificial Neural Systems: Foundations, Paradigms, Applications, and Implementations* New York, N Y Pergamon Press

Smith, G R. & McDaniels, S. M (1983) Psychologically mediated effect on the delayed hypersensitivity reaction to tuberculin in humans *Psychosomatic Medicine*, **45**, 65-69.

Solomon, R L & Corbit, J D (1974) An Opponent-Process Theory of Motivation I Temporal Dynamics of Affect. *Psychological Review*, **81**, 119-145

Solomon, R L & Corbit, J D (1980) The Opponent-Process Theory of Acquired Motivation. *American Psychologist*, **35**, 691-712

Sutton, R S & Barto, A. G (1981) Toward a Modern Theory of Adaptive Networks: Expectation and Prediction *Psychological Review* **88**, 135-170

Sutton, R S. & Barto, A. G. (1990) Time-Derivative Models of Pavlovian Reinforcement In M. Gabriel & J W Moore (Eds), *Learning and Computational Neuroscience: Foundations of Adaptive Networks* (pp 497-537) Cambridge, MA MIT Press

Terry, W. S (1976) The effects of priming US representation in short-term memory on Pavlovian conditioning. *Journal of Experimental Psychology: Animal Behavior Processes*, **2**, 354-370.

Thompson, R F, Clark, G A, Donegan, N H, Lavond, D G, Madden, J IV, Mamounas, L A, Mauk, M D, & McCormick, D A (1984) Neuronal substrates of basic associative learning In L Squire & N Butters (eds), *Neuropsychology of Memory* (pp 424-442) New York, N Y Guilford Press

Thompson, R F & Spencer, W A (1966) Habituation A model phenomenon for the study of neuronal substrates of behavior *Psychological Review*, **73**, 16-43

Turkkan, J S (1989) Classical conditioning: The new hegemony *Behavioral and Brain Sciences*, **12**, 121-179

Vallée-Tourangeau, F (1993) *Adjustment to Disconfirming Evidence in a Covariation Judgment Task* Ph D Thesis, Department of Psychology, McGill University, Montreal

Wagner, A R. (1976) Expectancies and the priming of STM In S H. Hulse, H. Fowler, & W K Honig (Eds), *Cognitive Processes in Animal Behavior* Hillsdale N J.: Erlbaum

Wagner, A R (1981) SOP A model of automatic memory processing in animal behavior In N Spear & G Miller (Eds.), *Information processing in animals: Memory mechanisms* (pp 5-47) Hillsdale, N.J.: Erlbaum.

Wagner, A. R. & Brandon, S. E. (1989) Evolution of a structured connectionist model of Pavlovian conditioning (AESOP). In S. B Klein & R R. Mowrer (Eds), Contemporary Learning Theories. *Pavlovian Conditioning and the Status of Traditional Learning Theory* (pp 149-189) Hillsdale, N.J. Erlbaum.

Wagner, A R, Logan, F A., Haberlandt, K & Price, T. (1968) Stimulus selection in animal discrimination learning. *Journal of Experimental Psychology*, **76**, 171-180

Watson, J B (1920) Conditioned emotional reactions *Journal of Experimental Psychology*, **3**, 1-14.

Watson, J B. (1916) The place of the conditioned reflex in psychology *Psychological Review*, **23**, 89-116.

Wickramasekera, I (1977) A conditioned response model of the placebo effect: Predictions from the model In L White, B. Tursky & G E. Schwartz (Eds.), *Placebo: Theory, Research and Mechanisms*. New York, N.Y Guilford Press.

Widrow, B & Hoff, M. (1960) Adaptive switching circuits, *1960 WESCON Convention Record: Part 4*, 96-104.

Appendix A
Demonstration of a Differential Equation for $\alpha_i(t)$

Differential Equation for $\alpha_i(t)$

One important property of $\alpha_i(t)$ (the CS_i trace), deduced in Chapter 4, is that $\alpha_i(t)$ continues to increase after CS_i offset with a short CS_i (i.e. a CS duration shorter than T_p $2 / \theta$). If $\alpha_i(t)$ was represented by a first-order differential equation involving CS_i, then $\alpha_i(t)$ would start to decrease at the time of CS_i offset. If $\alpha_i(t)$ was represented by a second-order differential equation involving CS_i, then the acceleration of $\alpha_i(t)$ would be negative with a high magnitude at CS_i offset, and the peak of $\alpha_i(t)$ would still be in the neighborhood of CS_i offset. This was confirmed by simulation for the second-order differential equation obtained by taking $\overline{X}_i(t) = Ate^{-\theta t}$ instead of $At^2e^{-\theta t}$. The use of a third-order differential equation therefore seems necessary. With a third-order differential equation, at CS offset, the slope of acceleration of $\alpha_i(t)$ becomes negative, but the acceleration is still positive, thus creating a delay before $\alpha_i(t)$ reaches its peak.

We want to obtain an output following the equation $At^2e^{-\theta t}$ for a step-like input. The output variable will be called $\overline{X}_i(t)$ and the input variable is $X_i(t)$ (the salience of the CS_i at time t). The step-like input for $X_i(t)$ is represented by the Heaviside function $\mathcal{U}(t)$. $\mathcal{U}(t)$ is defined in the following way:

$$\mathcal{U}(t) = \begin{cases} 0 & \text{for } t \leq 0^-, \\ 1 & \text{for } t \geq 0^+ \end{cases} \qquad (A1)$$

Thus, we have $X_i(t) = \mathcal{U}(t)$. By taking the Laplace transform of $X_i(t)$ we get:

$$X_i(s) = \mathcal{L}(X_i(t)) = \mathcal{L}(\mathcal{U}(t)) = 1/s \qquad (A2)$$

The desired output is $\overline{X}_i(t) = At^2e^{-\theta t}$. Taking the Laplace transform of $\overline{X}_i(t)$ we obtain:

$$\overline{X}_i(s) = \mathcal{L}(\overline{X}_i(t)) = \mathcal{L}(At^2e^{-\theta t}) = \frac{2A}{(s + \theta)^3} \qquad (A3)$$

The transfer function H(s) of a linear differential system is defined as the ratio of the Laplace transform of the output function over the Laplace transform of the input function, i.e.:

$$H(s) = \overline{X}_i(s) / X_i(s) \qquad (A4)$$

Substituting Equations A2 and A3 in Equation A4, the value of H(s) is:

$$H(s) = \frac{\left(\frac{2A}{(s+\theta)^3}\right)}{(1/s)},$$

$$= \frac{2A\,s}{(s+\theta)^3},$$

$$= \frac{2A\,s}{(s^3 + 3\theta\,s^2 + 3\theta^2\,s + \theta^3)} \qquad (A5)$$

From A4 and A5 we obtain

$$H(s) = \frac{\overline{X}_1(s)}{X_i(s)} = \frac{2A\,s}{(s^3 + 3\theta\,s^2 + 3\theta^2\,s + \theta^3)},$$

and so assuming that

$$\left.\frac{d^2\overline{X}_1(t)}{dt}\right|_{t=0} = 0, \quad \left.\frac{d\overline{X}_1(t)}{dt}\right|_{t=0} = 0, \quad \overline{X}_i(0) = 0, \text{ and } \left.\frac{dX_i(t)}{dt}\right|_{t=0} = 0,$$

we obtain

$$(s^3 + 3\theta\,s^2 + 3\theta^2\,s + \theta^3)\,\overline{X}_1(s) = 2A\,s\,X_i(s) \qquad (A6)$$

By taking the inverse Laplace transform of each side of the Equation A6 we obtain:

$$\frac{d^3\overline{X}_1(t)}{dt} + 3\theta\,\frac{d^2\overline{X}_i(t)}{dt} + 3\theta^2\,\frac{d\overline{X}_1(t)}{dt} + \theta^3\,\overline{X}_1(t) = 2A\,\frac{dX_i(t)}{dt}, \qquad (A7)$$

For a step-like input $X_i(t) = \mathcal{U}(t)$, $\overline{X}_1(t)$ will be positive or equal to zero (for $t \le 0$). However, for other kinds of inputs (e.g., $X_i(t) = 1 - \mathcal{U}(t)$) $\overline{X}_1(t)$ will take negative values In Chapter 4, no meaning has been attributed to negative values of $\alpha_i(t)$ $\alpha_i(t)$ will therefore be defined as the positive part of $\overline{X}_1(t)$ Thus we have

$$\alpha_i(t) = \begin{cases} 0 & \text{if } \overline{X}_i(t) < 0, \\ \overline{X}_i(t) & \text{otherwise.} \end{cases} \qquad (A8)$$

Properties of $\alpha_i(t)$

By construction, $\alpha_i(t)$ produced by a step-like input $\mathcal{U}(t)$ is equal to $At^2e^{-\theta t}$ for $t \ge 0$. The peak of $\alpha_i(t)$ is at $t = T_p$ and at this point $\dot{\alpha}_i(T_p) = 0$. This point is found by taking the derivative of $\alpha_i(t)$, i.e. $\dot{\alpha}_i(t) = 2At\,e^{-\theta t} - A\theta\,t^2e^{-\theta t}$ which is equal to zero. Dividing by $At\,e^{-\theta t}$, we get $2 - \theta\,t = 0$ This value of t is by definition equal to T_p, thus

$$T_p = 2/\theta \qquad (A9)$$

If we fix the maximum value of $\alpha_i(t)$ as being equal to a constant α_{max}, then the constant A is determined. Since the maximum value of $\alpha_i(t)$ occurs at $t = T_p = 2 / \theta$, we can find A by putting $t = 2 / \theta$ in $At^2e^{-\theta t}$. This leads to

$\alpha_i(2 / \theta) = \alpha_{max} = A(2 / \theta)^2e^{-(2 / \theta)\theta}$, and then to

$$\alpha_{max} = A(2 / \theta)^2e^{-2}. \qquad (A10)$$

From that we get

$$A = \frac{\alpha_{max}\theta^2e^2}{4} \qquad (A11)$$

In short, (1) the peak of $\alpha_i(t)$ occurs at $t = T_p = 2 / \theta$ after the onset of the CS_i, for a step-like CS_i and (2) A has to be smaller or equal to $\frac{\theta^2e^2}{4}$ in order for the maximum value of $\alpha_i(t)$ to be smaller or equal to one

Appendix B

Development of the Sensitization Term for $\theta_i(t)$

It has been hypothesized in Chapter 4 that a sensitization, provoked by the US onset, would change the amplitude of $\alpha_i(t)$ (the CS_i trace) and move the position of its peak (i.e. T_p) toward the US onset. In this assumption, sensitization increases or decreases T_p as well as increases or decreases the maximum amplitude of $\alpha_i(t)$.

Since $T_p = 2 / \theta$, increasing T_p means decreasing θ and decreasing T_p means increasing θ. When US onset is before T_p, T_p should decrease and when US onset is after T_p, T_p should increase. When $\Delta\alpha_i(t) = \alpha_i(t) - \alpha_i(t-1)$, if US onset is before T_p, then $\Delta\alpha_i(t)$ is positive at US onset and if US onset is after T_p, then $\Delta\alpha_i(t)$ is negative at US onset.

In summary, we want

θ to increase, i.e. T_p to decrease, if $\Delta\alpha_i(t) > 0$ at US onset,

and θ to decrease, i.e. T_p to increase, if $\Delta\alpha_i(t) < 0$ at US onset.

Then a reasonable assumption is that the change in θ is proportional to $\Delta\alpha_i(t)$, i.e. formally

$$\Delta\theta(t) \sim \Delta\alpha_i(t),$$

at the time of US onset $\Delta Y(t)$ is positive at US onset, therefore the positive part of $\Delta Y(t)$ can be taken as a measure of US onset. The positive part of $\Delta Y(t)$ can be expressed by the notation $\lceil \Delta Y(t) \rceil$. If we make $\Delta\theta(t)$ proportional to $\lceil \Delta Y(t) \rceil$, the sensitization will be stronger for strong US and weaker for small USs, which makes sense. So we assume:

$$\Delta\theta(t) \sim \lceil \Delta Y(t) \rceil$$

When a variable is proportional to two quantities, it is also proportional to the product of those two quantities. Thus, we have:

$$\Delta\theta(t) \sim \Delta\alpha_i(t) \lceil \Delta Y(t) \rceil$$

If we introduce a sensitization constant s, we can replace the proportionality symbol ~ by an equality sign. Then $\Delta\theta(t)$ becomes

$$\Delta\theta(t) = s\, \Delta\alpha_i(t) \lceil \Delta Y(t) \rceil \qquad \text{(B1)}$$

A simulation using this term in a delay conditioning procedure, where the initial value of T_p is smaller than the ISI, showed a strange behavior. The delay between CS onset and the peak of the CR increased, reached US onset and then decreased again. This was due to the fact that the CR tends to synchronize its peak with high values of $\Delta Y(t)$, but the CR increases and influences $Y(t)$. T_p tends to synchronize itself with the peak of the derivative of the CR. This is why T_p decreases in a second phase. To correct that

behavior, it is possible to emphasize the difference in magnitude between the CR and the US by taking $\lceil \Delta Y(t) \rceil^2$ instead of $\lceil \Delta Y(t) \rceil$ This power of two transformation will give more weight to the biggest values of $\Delta Y(t)$ and should fix T_p at US onset The equation of sensitization then becomes

$$\Delta\theta(t) = s\, \Delta\alpha_i(t) \lceil \Delta Y(t) \rceil^2 \qquad (B2)$$

Simulations using this new equation showed another problem T_p tends to synchronize itself with the onset of the US and stays there, as intended However, when T_p reaches the onset of the US, the connection weight $\Delta V_i(t)$ decreases slowly toward zero Equation B2 modifies $\theta(t)$ so that the peak of the CR arrives at the same time as the onset of the US The learning rule is, according to Equation 4 1b

$$\Delta V_i(t) = \alpha_i(t\text{-}2)\, \beta\, (Y(t)\text{-}\overline{Y}(t))$$

For example, if a US lasts for 2 time steps, the onset of the US occurs at T_p, and the offset of the US occurs at T_p+2 Taking a US intensity of λ_0, $(Y(t)\text{-}\overline{Y}(t))$ will be approximately equal to λ_0 at US onset and approximately equal to $-\lambda_0$ at US offset The total change of connection weight from T_p to T_p+2, is therefore approximately equal to

$$\Delta V_i \cong \alpha_i(T_p\text{-}2)\, \beta\, (\lambda_0) + \alpha_i(T_p)\, \beta\, (-\lambda_0),$$

i.e $$\Delta V_i \cong \beta\, \lambda_0 (\, \alpha_i(T_p\text{-}2) - \alpha_i(T_p)\,)$$

Since $\alpha_i(T_p)$ is greater than $\alpha_i(T_p\text{-}2)$, given the definition of T_p, then ΔV_i is negative and $V_i(t)$ decreases The solution seems simple — the onset of the US should occur at T_p+2 instead of at T_p In this way, the total change in connection weight between T_p+2 and T_p+4, will be approximately equal to

$$\Delta V_i \cong \beta\, \lambda_0 (\, \alpha_i(T_p) - \alpha_i(T_p\text{+}2)\,) \qquad (B3)$$

Since $\alpha_i(T_p\text{+}2)$ is smaller than $\alpha_i(T_p)$, $V_i(t)$ will not decrease This can be accomplished by taking $\Delta\alpha_i(t\text{-}2)$ instead of $\Delta\alpha_i(t)$, in the above equations, leading to

$$\Delta\theta(t) = s\, \Delta\alpha_i(t\text{-}2) \lceil \Delta Y(t) \rceil^2 \qquad (B4)$$

In order to make the value of $\Delta\theta(t)$ invariant as a function of a change in Δt, we can divide the sensitization term B4 by Δt With this division, it is not necessary to change s if one changes the time step Δt The sensitization term becomes

$$\Delta\theta(t) = (s/\Delta t)\, \Delta\alpha_i(t\text{-}2) \lceil \Delta Y(t) \rceil^2 \qquad (B5)$$

Appendix C
Parameter Specifications for the Computer Simulations of the Neural Models

Rescorla-Wagner's (1972) Model

The simulation of Figure 3 1 used the following parameter and variable values

Learning rate parameter in presence of a reinforcement $\beta_1 = 1\ 0$

Learning rate parameter in absence of a reinforcement: $\beta_0 = 0.9.$

Salience of the first stimulus. $\alpha_1 = 0\ 3$

Salience of the second stimulus $\alpha_2 = 0\ 3.$

Maximum associative strength $\lambda = 100.$

Initial conditions $V_1(0) = V_2(0) = 0.$

Klopf's (1988) Model

The simulations of Figures 3 2 and 3.3 used the following parameter and variable values

Learning rate constants C_1=5.0, C_2=3.0, C_3=1.5, C_4=0 75, C_5=0 25.

Maximum number of time steps over which delay conditioning is effective $\tau = 5.$

The maximum value of Y(t) $M = 1.$

Neuronal threshold: $T = 0.$

Initial conditions $V_{E1}(0) = +0.1,\ V_{I1}(0) = -0\ 1.$

The time step $\Delta t = 0\ 5$ sec, US intensity = 0 5 and CS amplitude = 0 2.

The simulation of Figure 3.2 used ISI = 1 sec, ITI = 15 sec, US duration = 2.5 sec.

Timing of the CS-US configuration of Figure 3.3 is described in Table C.1.

Delay-Producing Connections Model

The Figures 4 7 - 4 11 used the following parameter and variable values:

Learning rate parameter $\beta = 0.6$

Damping factor for the trace of Y(t). $\beta_B = 0\ 1.$

The maximum value of Y(t) $M = 3$

Neuronal threshold $T = 0.$

Constant governing the amplitude of the peak of $\alpha_i(t)$ $A = 1.84.$

Position of the peak of $\alpha_i(t)$: $T_p = 220$ msec.

Initial conditions	$\alpha_i(0) = V_i(0) = 0$
Amplitude of the CSs	1.0
Intensity of the US	1 0.

Figure 4 7 used Δt = 25 msec, ISI = 50 msec, ITI = 1 sec, CS duration = 50 msec, and US duration = 50 msec.

Figure 4 8 used: Δt = 50 msec, CS duration = 50 msec and 1000 msec, and no US

Figure 4 9 used Δt = 50 msec, ISI = 250 msec, ITI = 3 sec, CS duration 1sec, US duration = 50 msec and US intensity = 0 1

Figure 4 10 used: Δt = 25 msec, ISI = 200 msec, ITI=1 5 sec, and US duration 300 msec

Figure 4 11 used Δt = 50 msec, ITI = 1 sec, CS duration = 50 msec, and US duration 50 msec

Adaptive Delays Model

The simulations of Figures 5 2, 5.4 and 5.6 - 5.13 used the following parameter and variable values

Learning rate parameter:	$\beta = 0.6$
Damping factor for the trace of Y(t)	$\beta_B = 0$ 1.
The maximum value of Y(t)	M = 3.
Neuronal threshold	T = 0.
Constant governing the amplitude of the peak of $\alpha_i(t)$	A = 1.84
Minimum delay for the peak of $\alpha_i(t)$:	T_{pmin} = 175 msec
Habituation of $\alpha_i(t)$	D = 2 8.
Habituation rate	h = 0.05
Sensitization rate:	s = 0.03
Initial conditions.	$\theta_i(0)$ = 10 Hz, $\alpha_i(0) = V_i(0)$ = 0
Amplitude of the CSs:	1 0
Intensity of the US:	1 0.

The other figures in Chapter 5 used the same parameter values except as now noted The simulations of Figures 5 2 and 5.15 used the same parameter values except for h = 0, S 0.01 and β = 0.2 The simulation of Figure 5 20 used h =0, s = 0 and β = 0 2 The simulation of Figure 5.16 used a US intensity of 0 5, and the simulation of Figures 5 16 and 5.17 used a CS amplitude of 0 5 The simulation of Figure 5 19 used an amplitude of 0.2 for the CS_2. Figure 5.32 used h =0, s = 0 and β = 0 2 with a CS amplitude of 0 1

Table C.1 summarizes the timing of the CS-US configurations of single CS figures, while Table C.2 does the same for figures with more than one CS.

Timing of the CS-US Configurations of Single CS Figures:

Figure #	Time Step [msec]	CS Duration/ Trials # * [msec]	US Duration/Trials # [msec]	ISI [msec]	ITI [msec]
3 3	500	5000/1-20	3500/1-10	1500	15000
4 9	50	1000/1-20	750/1-10	250	3000
5 2	25	50/1-21	50/1-20	250	1000
5 4	25	375/1-20	50/1-10	325	1000
5 5	25	375/1-30	50/1,3,5,7, ,19	325	1000
5 6	25	250/1-21	50/1-20	200	1000
5 7	25	50/1-20	50/1-20	200	1000
5 8	25	50/1-20	50/1-20	0	1000
5 9	25	50/1-20	50/1-20	-50	1000
5 12	25	100/1-20	50/1-20	200	1000
5 13	50	750/1-36	50/1-2,4-35	700	3000
5 14	25	50/1-20	100/1-20	200	1000
5 15	25	50/1-20	50/1-20	200	1000
5 16	25	50/1-20	50/1-20	200	1000
5 17	25	50/1-20	50/1-20	200	1000
5 18	25	250/1-40	50/21-40	200	1000
5 31	25	375/1-20	50/1-10	325	1000
5 32	25	1500	3000	0,50, ,500	6000

* Trials # represents the trials during which the stimulus was present

Table C.1

Timing of the CS-US Configurations in Figures of Configural Learning:

Figure #	Time Step [msec]	CS 1	CS 2	CS 3	US
5 3	25	0/250/1-20	0/250/2,4, ,18,20-31	none	200/250/1,3, ,19
5 19	25	0/300/1-20	0/300/1-20	none	250/300/1-20
5 20	50	0/400/1-80	0/400/21-80	none	350/400/1-80
5 21	25	0/250/1-10 100/350/11-20	0/250/11-20	none	200/250/1-10
5 22	25	0/375/1-10,21-30	0/375/11-30	none	325/375/1-30
5 23	25	0/250/1-20	0/250/2,4, ,20,21-40	0/250/21-40	200/250/1,3, ,19, 20-40
5 24	50	0/350/1, 3, ,19	0/350/2,4, ,20	0/350/1-20	300/350/1,3, ,19
5 25	50	0/350/1,3 ,19	0/350/2,4, ,20	0/350/1-20	300/350/1,3, ,19
5 26	50	0/350/1,2,5,6,9,10, 13,14,17,18	0/350/3,4,7,8,11,12, 15,16	0/350/1-20	300/350/1,3, ,19
5 27	50	0/350/1-80	0/350/1,3, ,79	none	300/350/1-80
5 28	50	0/300/1-40	0/300/1,3, ,39	none	250/300/1 3, ,39

* Time of onset/ time of offset/ trials during which stimulus was present.

Table C.2

Appendix D

Properties and Models of Classical Conditioning

Properties	Models								
Author(s)	Hebb	Rescorla Wagner	Pearce Hall	Sutton Barto	Sutton Barto 2	Gluck Thompson	Klopf	Mignault DPC	Mignault AD
Year	1943	1972	1980	1981	1981	1987	1988	1993	1993
Types of Conditioning									
Conditioned Excitation	Y	Y	Y	Y	Y	Y	Y	Y	Y
Conditioned Inhibition	N	Y	Y	Y	Y	Y	Y	Y	Y
Facilitating CR	Y	Y	Y	Y	Y	Y	Y	Y	Y
Compensatory CR	N	*	*	N	N	N	N	N	N
Acquisition									
Positive acceleration at first	Y	N	*	N	N	Y	Y	N	*
Negative acceleration later	N	Y	Y	Y	Y	Y	Y	Y	Y
Reaches an asymptote	N	Y	Y	Y	Y	Y	Y	Y	Y
Slower with partial reinforcement	Y	Y	*	Y	Y	Y	Y	Y	Y
Spaced faster than massed	N	N	N	N	N	N	N	N	N
Stimulants increase the rate	N	N	N	N	N	N	N	N	N
Depressants decrease the rate	N	N	N	N	N	N	N	N	N
Extinction									
Negative acceleration	N	Y	Y	Y	Y	Y	*	Y	Y
Asymptote is zero	N	Y	Y	Y	Y	Y	*	Y	Y
Slower than acquisition	N	Y	N	N	N	Y	Y	Y	Y
Slower for inhibitor than excitator	N	N	Y	N	N	Y	Y	Y	Y
Slower for partial reinforcement	Y	Y	*	Y	Y	Y	Y	Y	Y
Massed faster than spaced	N	N	N	N	N	N	N	N	N
Stimulants decrease the rate	N	N	N	N	N	N	N	N	N
Depressants increase the rate	N	N	N	N	N	N	N	N	N
Spontaneous Recovery									
CR reappears after extinction	N	N	N	N	N	N	N	N	N
Smaller CR after more CSs-alone	Y	Y	Y	Y	Y	Y	Y	Y	Y
Reacquisition.									
Faster than acquisition	N	N	N	N	N	N	N	N	*
Time Contingencies									
Delay stronger than trace	Y	N	N	N	N	Y	Y	Y	Y
Trace stronger than simultaneous	N	N	N	Y	Y	N	Y	Y	Y
Simultaneous stronger than backward	Y	N	N	Y	Y	Y	Y	Y	Y
Optimal ISI shorter for short trace CSs than for long delay CSs	N	N	N	N	N	N	N	Y	Y

Properties	Models:								
Author(s)	Hebb	Rescorla-Wagner	Pearce Hall	Sutton Barto	Sutton-Barto 2	Gluck-Thompson	Klopf	Mingault DPC	Mingault AD
Delay Conditioning									
Produces excitator	Y	N	N	N	N	Y	Y	Y	Y
Efficacy is a function of CS onset	N	N	N	N	N	*	Y	Y	Y
Trace Conditioning.									
Produces excitator	N	N	N	Y	Y	Y	Y	Y	Y
Rate of acquisition as a function of ISI reaches a peak and decline	N	N	N	N	N	N	Y	Y	Y
Independent of CS duration	N	N	N	Y	Y	N	*	*	*
Optimal ISI can be longer than CS duration	N	N	N	N	N	Y	Y	Y	Y
Simultaneous Conditioning									
Little or no conditioning	N	N	N	*	*	N	Y	*	*
Backward Conditioning:									
The first trials are excitatory	N	N	N	N	N	N	N	N	N
Subsequent trials are inhibitory	N	N	N	*	Y	N	Y	Y	Y
Temporal Conditioning.									
The CR predicts US onset	N	N	N	N	N	N	N	N	*
CR Properties:									
Shape of the CR different than CS	N	N	N	N	N	N	N	Y	Y
Variance of CR increases with ISI	N	N	N	N	N	N	N	N	Y
Peak of CR at the onset of US	N	N	N	N	N	N	N	N	Y
Inhibition of Delay									
CR moves forward during training	N	N	N	N	N	N	N	N	Y
2 peaks in the CR if 2 USs at 2 ISIs	N	N	N	N	N	N	N	N	N
With 2 ISIs, second peak appears only if first US is absent	N	N	N	N	N	N	N	N	N
US Duration Effects									
Faster acquisition for longer US	*	N	N	N	Y	Y	Y	Y	Y
Higher asymptote for longer US	N	N	N	N	Y	Y	Y	Y	Y
US Intensity Effects									
Faster acquisition for stronger US	Y	N	Y	Y	Y	Y	Y	Y	*
Higher asymptote for stronger US	N	Y	Y	Y	Y	Y	Y	Y	*
US Preexposure Effect									
Decreased asymptote	N	Y	Y	Y	Y	Y	Y	Y	*
CS Intensity Effects									
Faster for stronger CS	Y	Y	Y	Y	Y	Y	Y	Y	Y
Higher asymptote for the strong CS if acquisition is done with both	N	Y	Y	Y	Y	Y	Y	Y	Y
CR ampl independent of CS ampl.	N	Y	Y	Y	Y	N	Y	Y	Y

Table D 1

Properties	Models								
Author(s)	Hebb	Rescorla-Wagner	Pearce-Hall	Sutton Barto	Sutton-Barto 2	Gluck Thompson	Klopf	Mignault DPC	Mignault AD
CS Preexposure Effect:									
Slower acquisition	N	N	Y	N	N	N	N	N	*
Affects excitation and inhibition	N	N	Y	N	N	N	N	N	Y
Sensory Preconditioning:									
CR for a non-reinforced CS	N	N	N	N	N	N	N	N	N
Optimal if CS2 before CS1	N	N	N	N	N	N	N	N	N
Optimal if only a few trials (CS1-CS2)	N	N	N	N	N	N	N	N	N
Bridge between CS and US									
Attenuation of cond. produced by a delay is reduced by a second CS	N	N	N	N	N	N	N	N	N
Overshadowing									
More salient CS -> higher asymptote	N	Y	Y	Y	Y	Y	Y	Y	Y
More salient CS -> faster acquisition	N	Y	Y	Y	Y	Y	Y	Y	Y
Potentiation of a less salient cue									
A strong CS facilitates a weak CS	N	N	N	N	N	N	N	N	N
Blocking									
Past training blocks new learning	N	Y	Y	Y	Y	Y	Y	Y	*
Unblocking									
A surprising US removes blocking	N	N	Y	N	N	N	N	N	N
Second Order Conditioning									
Produces Conditioned Excitation	Y	*	N	Y	Y	Y	Y	Y	Y
Not as strong as first-order	N	Y	Y	Y	Y	*	Y	Y	Y
Will be extinguished	N	N	N	Y	Y	Y	Y	Y	Y
Conditioned Inhibition (A+ AB-)									
If in series CR to B not as strong as CR to A	N	Y	Y	Y	Y	Y	Y	Y	Y
Do not extinguish	N	N	Y	Y	Y	Y	Y	Y	Y
Overexpectation (A+ B+ AB+)									
CR to AB > CR to A or B	Y	Y	Y	Y	Y	Y	Y	Y	Y
CR to AB decreases	N	Y	Y	Y	Y	Y	Y	Y	Y
CR to A or B have decreased	N	Y	Y	Y	Y	Y	Y	Y	Y
Superconditioning (X+AX-,AB+):									
CR to B increases faster than just B+	N	Y	Y	Y	Y	Y	Y	Y	Y
Higher asymptote than just B+	N	Y	Y	Y	Y	Y	Y	Y	Y
BX+ 0 5/ BX- 0 5)									
CR to X > A = B	N	Y	?	Y	Y	?	Y	Y	*
CR to X greater than CR in discrimination	N	Y	?	Y	Y	?	Y	Y	*

Table D 1

Properties	Models:								
Author(s)	Hebb	Rescorla Wagner	Pearce-Hall	Sutton-Barto	Sutton-Barto 2	Gluck-Thompson	Klopf	Mingault DPC	Mingault AD
Discrimination Learning (AX+ BX-)									
CR A > X > B	N	Y	Y	Y	Y	Y	Y	Y	Y
CR to B=0	N	Y	?	Y	Y	Y	Y	Y	Y
CR to X goes up and then down	N	Y	?	Y	Y	?	Y	Y	Y
CR to BX goes up and then down	N	Y	?	Y	Y	?	Y	Y	Y
CR to AB > CR to BX	N	Y	?	Y	Y	?	Y	Y	Y
Compound Conditioning (A+ AX+)									
CR to A >> CR to X	N	Y	Y	Y	Y	Y	Y	Y	Y
Compound Conditioning (A- AX+)									
CR to X >> CR to A	N	Y	Y	Y	Y	Y	Y	Y	Y
AB+ A- B-									
CR to A and CR to B = 0	N	N	N	N	N	N	N	N	N
CR to AB is > zero	N	N	N	N	N	N	N	N	N
Positive Patterning (A- B- AB+)									
CR to A and CR to B = 0	N	N	N	N	N	N	N	N	N
CR to AB > zero	Y	Y	Y	Y	Y	Y	Y	Y	Y
CR to A and CR to B goes up and then down	N	N	N	N	N	N	N	N	N
Negative Patterning (A+ B+ AB-)									
Longer to learn than positive pat	N	N	N	N	N	N	N	N	N
Initially AB stronger CR than A and B	Y	Y	Y	Y	Y	Y	Y	Y	Y
At the end CR to AB = 0	N	N	N	N	N	N	N	N	N
Feature Positive (AB+ A-).									
Can learn it	N	Y	Y	Y	Y	Y	Y	Y	Y
Pearce (1993) (A+B+C+ ABC-)									
AB, BC AC decreases faster than A, B, C	N	N	N	N	N	N	N	N	N
CR of ABC stronger than A B C at first	Y	Y	Y	Y	Y	Y	Y	Y	Y
CR to ABC = 0 at the end	N	N	N	N	N	N	N	N	N

Legend

Y means that the property is accounted for by the model;

* means that the property is partially accounted for by the model;

N means that the property is not accounted for by the model.

Table D.1

Mathematical Properties of Models of Classical Conditioning:									
Properties.	Models:								
Author(s)	Hebb	Rescorla Wagner	Pearce Hall	Sutton Barto	Sutton-Barto 2	Gluck Thompson	Klopf	Mignault DPC	Mignault AD
Year.	1943	1972	1980	1981	1981	1987	1988	1993	1993
Number of Properties Explained	16 2	39 4	36 8	46 4	49 2	45.4	55 6	57 4	57 4
Number of Equations for n CSs:	n+1	n	2n+1	2n+1	2n+2	3n+4	2n+1	2n+2	3n+2
Number of Variables for n CSs	2n+1	2n+1	2n+2	2n+2	2n+3	7n+8	3n+1	4n+1	5n+1
Number of Parameters for n CSs.	n+1	n+3	n+2	2	3	6	7	4	8
Number of Equations for 3 CSs.	4	3	7	7	8	12	7	8	11
Number of Variables for 3 CSs	7	7	8	8	9	29	10	13	16
Number of Parameters for 3 CSs	4	6	2	2	3	6	7	4	8
Benefit / Cost Ratio	1 08	2 46	2 16	2 73	2 46	0 97	2 32	2 3	1 64

Table D.2

Legend

The number of properties was computed from the Table D 1 A "Y" counts for 1 point, a "*" counts for 0.2 points and a "N" counts for none.

The benefit is the number of properties while the cost is the sum of the number of equations, the number of variables and the number of constants.