A NEW ASSOCIATIVE CONDITIONING ELEMENT FOR CONSUMMATORY CLASSICAL CONDITIONING

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SUMMARY
This thesis documents the development, operation, and performance of a new nonlinear analog system designed to exhibit a wide range of (mainly) consummatory classical conditioning behavior. This new system, referred to hereafter as the Associative Conditioning Element (ACE), may be categorised as a functioning Artificial Neural Network (ANN) element of unusually large mechanistic and behavioral complexity. However, depending upon the perspective of the reader, ACE may also be considered as a neuronal model of classical conditioning, or as a psychological theory of animal learning and memory.

The performance specifications for ACE consist entirely of empirical results from behavioral animal experiments, primarily focussing upon the Nictitating Membrane (third eyelid) Response (NMR) of the rabbit. However, the widespread applicability of classical conditioning means that ACE’s behavior also generalises to many other response systems and animal species, and where appropriate NMR results are not available, those from other response systems are utilised. ACE is assessed by comparing the results of computer simulations of its operation with corresponding empirical results.

ACE produces behavior attributable to a single biological neuron, or small group of neurons. Despite the widespread ANN view that neural systems consist of densely interconnected networks of very simple elements, the demonstration of basic classical conditioning behavior by presynaptic facilitation in the marine mollusc Aplysia, and the sparsely interconnected organisation of relatively humble creatures in general, suggests that individual neurons may indeed have considerable information processing capability. ACE begins to bridge the gap between the excessive simplicity of standard ANN elements, and the considerable complexity of biological neurons which is only beginning to be understood and appreciated.

An exploration of the functional relationship between memory and learning has yielded a new nonlinear subsystem of interacting CS-specific (or synaptic) memory types, collectively referred to here as the Neural Multiprocess Memory
Model (NMMM). The NMMM is progressively developed from the standard ANN adaptive synaptic weighting, initially producing spontaneous regression and recovery behavior, then U-shaped memory retention, and finally comprehensive adaptive associability behavior. The adaptive associability mechanism supports both negatively accelerated and sigmoidal acquisition curves, latent inhibition, learned irrelevance, the Partial Reinforcement Effect (PRE), and accelerated learning following alternating acquisition/extinction training sessions.

A new nonlinear Conditioned Stimulus Trace Circuit (CSTC) has been developed with sufficient complexity to accurately model the shape of the mean NMR topography, and the way in which it changes subject to variations in CS duration and amplitude. This CS "trace" is available for generation of an appropriately timed Conditioned Response (CR), and to selectively gate the effect of experience subsequent to CS presentation upon the associative strength of the CS. The latter role enables the acquisition of a CR which is timed to peak approximately at the time reinforcement is expected, the production of anticipatory CRs, and the implementation of trace conditioning.

An associative Short Term Memory (STM) is made available by the NMMM which supports a modified intratrial version of the Rescorla–Wagner model, in which expectation of reinforcement and actual reinforcement experienced are able to be compared to determine change in associative strength, despite the asynchronous nature of CS and US presentation, and their different temporal profiles. This allows ACE to exhibit behavior such as stimulus amplitude effects, acquisition of conditioned excitation and inhibition, extinction, overshadowing, compound conditioning effects (e.g., blocking and unblocking), and discriminative stimulus effects.

And finally, the learning rules controlling changes in associative STM also support the nonassociative phenomena of secondary extinction, and reinstatement. As a result, ACE is able to capture a sense of the prevailing temporal context, and so, for example, support the acquisition of a conditioned inhibitor by successive discrimination, without relying upon conditioned excitation to background stimuli.
ABBREVIATIONS

ACE: Associative Conditioning Element
ALTM: Associability Long Term Memory
CR: Conditioned Response
CS: Conditioned Stimulus
CS+: Excitatory Conditioned Stimulus
CS−: Inhibitory Conditioned Stimulus
CCST: Compressed Conditioned Stimulus Trace
CST: Conditioned Stimulus Trace
CSTC: Conditioned Stimulus Trace Circuit
DEC: Decremental feedback signal
EAC: Enable Associability Change
EXC: Excitatory CS channel (synaptic) output
INC: Incremental feedback signal
INH: Inhibitory CS channel (synaptic) output
ISI: Inter-Stimulus Interval
ITI: Inter-Trial Interval
LTM: Long Term Memory
MGSM: Medium Gating Short Term Memory
MTM: Medium Term Memory
N: Nonreinforced
NMMM: Neural Multiprocess Memory Model
NMR: Nictitating Membrane Response
OUT: Output signal from ACE
PR: Partial Reinforcement
PRE: Partial Reinforcement Effect
R: Reinforced
RGM: Reinforcement Gating Short Term Memory
ROC: Rate Of Change preset input to the CSTC
RSTM: Reinforcement Short Term Memory
STM: Short Term Memory
UR: Unconditioned Response
US: Unconditioned Stimulus
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INTRODUCTION

STUDIES OF NATURAL INTELLIGENCE
CLASSICAL AND OPERANT CONDITIONING
ARTIFICIAL NEURAL NETWORKS
SYNAPTIC LEARNING RULES
TYPES OF FEEDBACK
  No Feedback
  Full Highly Specific Feedback
  Reinforcement Feedback
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INTRODUCTION

It was only a few decades ago when it appeared that the development of digital computers, which reliably do exactly what they are instructed at very high speed, might solve most information processing problems. However, digital computers are now so powerful, compact, and affordable that the task of working out and expressing exactly what they should do is becoming an overwhelming burden, in all but the simplest or most modular of problems. In other words, the point is being approached where the task of programming a computer to solve each type of difficult problem is becoming impractical. What is needed are new types of computers which learn how to solve new and complex problems - "intelligent" computers.

With the increasingly liberal use of the term "intelligence" in recent times, one could be forgiven for thinking that it is well understood. However this is not the case. Any attempt to produce a specific definition of intelligence is fraught with peril, and usually serves well as the basis of vigorous debate. Nonetheless, the formulation of at least some minimum criteria for intelligence is required to clarify the objectives of man-made systems designed to produce intelligent behavior. This can also assist the process of understanding intelligence itself. As a relatively non-controversial starting point, consider the following general definition: Intelligence is the capacity to acquire and apply knowledge.

This very general definition has several more specific implications. A memory capability is required in which to store acquired knowledge so that it may later be applied. The capacity to acquire knowledge implies some degree of sensory (or receptive) capability and a definite capacity to learn or adapt. The capacity to apply knowledge implies some ability to respond (or react) to circumstances appropriately by utilising relevant knowledge. Furthermore it is impractical, if not impossible, for any form of intelligence with finite computational resources to acquire or apply all possible knowledge from a complex environment. Therefore, intelligence will usually require some means of selectively acquiring that knowledge which, if applied, may be of assistance in the pursuit of its goals. This implies that an intelligent system also needs to
be receptive to some expression of its goals, and be capable of utilising this information to help it selectively acquire and apply useful knowledge. Such goal information may be provided in the form of an explicit input, or may be implicitly expressed in the functional design of the system. Thus, from the general definition above it can be inferred that: Intelligence requires memory, learning, sensory and goal input, goal directed behavior, and response output.

Naturally occurring intelligence, as supported in its various biological forms, exhibits all of the above basic characteristics. The uninitiated might therefore deduce that "Artificial Intelligence" (AI) refers to a field of study with the general aim of developing man-made systems which exhibit all of the same basic characteristics. However, even a cursory survey of the behavior of systems developed under the AI guise will indicate that this is not so. Indeed, any system with characteristics which vaguely resemble any of those of natural intelligence seems to qualify as an AI system - though this observation, in itself, need not necessarily be taken as a criticism of AI work in general, as:

(i) AI research need only produce useful cost-effective systems to be intellectually and commercially justifiable. In other words, man-made systems need not actually be intelligent to be useful, even if they are classified as AI. However, an increasing awareness of the limitations of such AI systems is motivating a resurgence of interest in natural intelligence, in the hope that more generally applicable and more powerful mechanisms will be developed.

(ii) AI research which now only deals with fragments of intelligence, may at a later date conceivably contribute to a synthesis of more complete intelligent behavior. Indeed, it is usually a deliberate decision to divide the difficult problem of understanding and reproducing intelligence into many smaller (and ideally separate) problems in the expectation that these will prove less intractable. This approach is currently typified by largely isolated studies into the nature of pattern recognition, knowledge representation and application, learning, memory, goal seeking behavior, motor control, and sensorimotor control.

However, despite considerable effort, progress in AI aimed at producing intelligent behavior has been less than impressive, suggesting that an
alternative type of approach to reducing the complexity of the intelligent behavior to be produced may be more appropriate. This is the avenue explored in the research described herein.

The scale of the problem is reduced here by decreasing the level of intelligent behavior to be modelled, while still maintaining all of the basic characteristics of intelligence. With this approach, intelligent systems are then seen as comprising (usually less) intelligent elements. The general aim of the research described herein is to develop one such type of intelligent element.

The specific nature of the intelligent element to be developed may be characterised by determining the following:

(i) Inter-element specifications. These define the number of inputs and output(s) of the element, and the type of signal they convey.

(ii) Behavioral performance. This defines how the element output(s) respond to various important sequences and/or combinations of input conditions. In this case, the behavioral performance will be a detailed specification of the intelligent behavior required of the element.

(iii) Intra-element mechanisms. Theae refer to the type of internal signals used, and the types of ways in which they may interact.

In seeking to develop a new AI element capable of capturing some of the behavioral advantages of natural intelligence, an obvious source of data for all three of the types of specifications outlined directly above are the natural systems themselves. All of these types of design constraints for the intelligent element under development here have been extracted from aspects of natural systems. This is not because natural systems necessarily represent the best type of solution, but because they still represent the only available comprehensive working solution.

Regarding inter-element specifications, the architectural constraints of biological neural networks are invoked. A complete system is therefore composed of multiple processing elements, interacting extensively via many simple scalar signals. These inter-element signals convey only amplitude information, which varies over time. They are therefore "non-symbolic" in an
explicit sense. A neural network architecture was chosen primarily because it is demonstrably capable of supporting sophisticated intelligent behavior (e.g., biological brains). As for the number of inputs and outputs, the minimum required to exhibit all of the basic characteristics of intelligent behavior are used. One output for response, one goal input, and several sensory inputs are sufficient for this purpose.

The behavioral criterion corresponds to basic associative conditioning, of a type which appears to be fundamental to most adaptive biological nervous systems. More specifically, it addresses the surprisingly complicated field of classical conditioning, which despite extensive study is still only partly understood. The general field of classical associative conditioning was chosen as the source from which to select a large number of behavioral constraints, mainly because it incorporates all of the basic characteristics of intelligence discussed above. These behavioral constraints have been carefully chosen to correspond to that which might conceivably be produced by a single neuron, or small group of neurons. That a single neuron could conceivably exhibit such complex behavior is suggested by the demonstration of basic classical associative conditioning behavior by presynaptic facilitation in the marine mollusk Aplysia (e.g., Carew, Abrams, Hawkins, and Kandel, 1984).

And finally, the intra-element mechanisms invoked to realise the functions of these units are restricted to relatively simple types of interactions, such as summation, multiplication, and accumulation of internal scalar quantities, which can be achieved by the chemical and electro-chemical processes underlying neural function, (or analog electronic hardware). Note, however, that this is combined with a preparedness to invoke, where required, relatively complicated combinations of such simple types of interaction.

Thus, in summary, the research described herein lies at the intersection of three areas of consideration depicted in Figure 1-1. It may at first appear that the voluntary imposition of such constraints upon an AI system's behavior and design could only make the task of its development all the more difficult. However, in the case of the research described herein, the reverse was found to be true. This is essentially because the bodies of knowledge
related to all three types of constraints (behavioral, inter-element interaction, and intra-element mechanisms) are very much incomplete, and the expertise required to develop such a system is not yet established. It is not really possible to determine, in advance, a complete behavioral specification for the operation of a specific system component that is known to exist, or which is known to be able to be implemented. Nor is it possible to fully specify the characteristics of the mechanisms capable of producing the desired behavior. Therefore, all of the constraints together help produce a more complete specification of the initial route to take through a vast terrain of possible system development paths.

The object of the research described is a new artificial neural network element referred to as the Associative Conditioning Element (ACE). As will be demonstrated using both computer simulation results and theoretical argument, ACE differs from other neuronal models of classical conditioning (e.g., Barto and Sutton, 1985; Klogf, 1987) in its robustness, its mechanistic and behavioral complexity, and the degree to which its behavior corresponds with empirical results.
This document, after some preliminary material of an introductory nature, progressively develops the conceptual and then the mechanistic detail of ACE. Not until the final chapters does a complete concept of ACE emerge. To help provide some specific indication of where the research documented is headed, a complete schematic diagram of ACE is provided below in Figure 1-2. It is evident that although ACE is only a single type of element designed to operate within a highly distributed neural network system architecture, it is in itself a system of considerable complexity.

ACE comprises multiple CS (Conditioned Stimulus) input channels, and a common output stage upon which all of them converge. All CS input channels are mechanistically identical, and so only a single representative CS input channel is illustrated interfacing with the output stage in Figure 1-2. ACE's single output signal (OUT) indicates when, and to what extent, US (Unconditioned Stimulus) INPUT activation has learned to be expected by each activated CS INPUT signal. OUT therefore has the amplitude and temporal characteristics required to generate a CR (Conditioned Response), though subsequent (and as yet undeveloped) elements would learn the qualitative nature of the CR. Each CS input channel contains the CS Trace Circuit illustrated in the lower section of Figure 1-2, which generates a sustained "trace" of prior CS INPUT activity suitable for acquisition and performance of an appropriately timed CR, and the Neural Multiprocess Memory Model illustrated in the upper section of the CS input channel in Figure 1-2, which utilises past experience to appropriately regulate the impact of current experience upon the expectation of the US by the CS. A decision was made to sequentially document and develop the performance and operation of each of the main subsystems in relative isolation, before later integrating them to complete ACE and reveal those aspects of ACE's behavior requiring their cooperative interaction. This approach clearly reveals the independent capabilities of each subsystem, and therefore facilitates their possibly separate utilisation or further development in future systems. Although devoid of detailed explanation at this point, Figure 1-2 provides a useful reference point for future chapters, as it indicates where each separately developed subsystem fits into ACE as a whole.
FIGURE 1-2. Complete schematic diagram of the Associative Conditioning Element (ACE). A single representative CS input channel is shown, comprising the Neural Multiprocess Memory Model (NMMM) and the CS Trace Circuit (CSTC). Also shown is the common output stage upon which multiple CS input channels converge.
STUDIES OF NATURAL INTELLIGENCE

The first half of the twentieth century saw the emergence of an enthusiastic attempt by (mainly) psychologists to unravel all natural intelligence by studying animals in contrived experimental paradigms. At the heart of this effort was classical reflex theory, also known as Stimulus–Response (or "S–R") theory, in which behavior is thought to consist of sets of responses to specific stimuli. Unfortunately, the success of this theory induced an unrealistic expectation that it would account for most aspects of behavior. When, in the latter half of this century it became apparent that the grand expectations would not be met, the entire field of study was largely discredited. However, while the wildly optimistic hopes for reflexology are now considered naive, its ability to conceptualise animal behavior in controlled experimental settings at a general level of explanation remains largely accepted. Reflexology is now seen in a more balanced perspective, as an important component of animal behavior, and as a possible doorway opening onto the field of neural processing mechanisms. Theoretical models which include concepts of reflexology are now much more sophisticated, and integrate these concepts with other aspects such as drive level, expectation, selective attention, and higher level processing.

Researchers of animal learning have historically sought to reveal either specialised adaptive capabilities in a comparative study of different species, or to reveal functional relations between behavioral changes and environmental changes using a more analytic approach. Romanes (1882) and Morgan (1894) are cited by Mackintosh (1974) as pioneers of the former approach, seeking to produce evidence of mind in animals other than humans. Morgan went further, arguing that the apparent complexity of overt behavior may result from the operation of simpler underlying associative processes. Although initially continuing on from Morgan, Thorndike's (1898, 1911) main achievement was to appreciate the necessity for, and then to develop, the controlled "operant conditioning" experimental paradigm, to help reveal the nature of underlying associative processes. The operant conditioning experimental paradigm, and analysis of the results it produced, were further advanced by the productive work of Skinner (1938, 1966). The other main pioneer of controlled behavioral
experiments was Pavlov (1927), who found it necessary to investigate associative conditioning in order to further his physiological analysis of bodily functions and behavior, and in so doing developed the "classical conditioning" paradigm.

The analytic, controlled experimental study of learning now overshadows its predecessor, and has done so for some sixty years. While the varied environmental pressures upon different animal species may have resulted in the emergence of differences in learning processes, the apparent similarities are now far more compelling.

CLASSICAL AND OPERANT CONDITIONING

Both classical conditioning and operant (or instrumental) conditioning are most clearly defined in terms of experimental procedure, as their underlying mechanisms are both less easily separated, and poorly understood. In a classical conditioning experiment, the experimenter arranges a contingency between presentation of a stimulus to the subject, and the subsequent delivery of reinforcement. In an operant experiment, a contingency is arranged between the subject's behavior (i.e. a response) and subsequently delivered reinforcement. Those who first appreciated this clear operational distinction between stimulus-contingent and response-contingent reinforcement suggested that differences in the underlying mechanisms may exist (Konorski and Miller, 1937; Schlosberg, 1934, 1936, 1937; Skinner, 1935, 1937, 1938). Eater, the dominant trend was to assume that the operational distinction signified essentially no underlying mechanistic difference (Rescorla and Solomon, 1967). However, it now seems that the earlier view was probably more correct.

The issue is further complicated, because the scheduling of stimulus-contingent or response-contingent reinforcement does not necessarily ensure that the subject experiences only this contingency. In fact, it is very difficult to design either classical or operant procedures in which some mixture of the two types of contingencies is not experienced by the subject. However, there is a firm basis for rejecting the notion of a single mechanism which completely accounts for both types of contingency. Instances of classical conditioning
exist, such as omission schedules, in which an operant contingency cannot account for learning, suggesting that a classical conditioning mechanism is required (Mackintosh, 1983, p. 32). Conversely, instances of operant conditioning exist for which a classical conditioning mechanism cannot account, suggesting that an operant conditioning mechanism is required. Mackintosh (1983, p. 98) argues that both types of conditioning utilise similar types of mechanisms in the formation of their respective associations, but that these are translated into performance using different mechanisms.

The research described herein focusses upon classical conditioning, primarily in order to limit its scope to manageable proportions. The development of a robust mechanism supporting classical conditioning should also facilitate subsequent attempts to account for operant conditioning. In addition, most aspects of classical conditioning can be demonstrated using a single neural network element, whereas multiple elements may be required to demonstrate most aspects of operant conditioning.

Much of the technical vocabulary currently used in association with classical conditioning was established by Ivan Pavlov (1927). Pavlov's prior Nobel prize winning research (1904) into the physiology of the digestive system led him to investigate the processes underlying the anticipatory salivation exhibited by dogs. When meat powder (the unconditioned stimulus, or US) was placed directly onto a dog's tongue, it reliably salivated (the Unconditioned Response, or UR) without prior training. In contrast, the sound of a ringing bell initially caused an orienting reaction, but no salivation. However, if the ringing bell was then closely followed (or "reinforced") by presentation of the US on several successive trials, it acquired the salivation response. Subsequent presentation of the sound of the ringing bell (the Conditioned Stimulus, or CS) alone then resulted in salivation (the Conditioned Response, or CR). This nomenclature is used frequently throughout this text. Note, however, that while this simplified account of Pavlov's early work with dogs still serves well as an introductory example of classical conditioning, it does not reveal the many complexities which have since been clearly demonstrated. These will be referred to at the appropriate junctures in later chapters.
ARTIFICIAL NEURAL NETWORKS

Artificial Neural Networks (ANNs), sometimes referred to simply as neural networks, are a broad class of information processing systems with an internal architecture resembling the highly distributed and interconnected organisation of biological networks. However, that may often be about as far as the similarity to biological systems goes. The nature of the ANN processing elements is usually very different to that of biological neural hardware, typically being very simple analogues of neural and synaptic function. The type of behavior ANNs are designed to produce also bears varying degrees of correspondence to the behavior of biological networks. ANNs are thus not, in general, models of biological neural networks.

The highly distributed architecture common to both artificial and biological neural networks is becoming increasingly attractive to computational theorists, partly because it implicitly subdivides a large computationally intensive problem into many smaller (and often similar) components. Such subdivision facilitates implementation of a system on distributed parallel hardware, which in turn facilitates the real-time solution of highly complex problems. The distributed architecture of neural networks also encourages the development of new distributed methods of solving problems.

SYNAPTIC LEARNING RULES

Perhaps, the single most enticing capability of biological neural systems is their general ability to learn, or adapt, in such a way as to improve their performance. Even before the synaptic doctrine appeared, Herbert Spencer postulated in 1862 that the ability of one cell to excite another could change as a function of prior activity, and that this was the basis of memory (Levy and Desmond, 1985, p. 105). Later, the famous Hebb synapse model (Hebb, 1949) emerged, in which synaptic efficacy changed in proportion to the product of presynaptic input and postsynaptic neural output signals. However, since both signals are non-negative, the efficacy of the Hebb synapse can only increase, which is now usually regarded as inadequate. Recognition of this apparent deficiency has spawned numerous varieties of Hebbian, pseudo-
Hebbian and anti-Mebbian learning rules, all of which are still relatively simple. The former, more Hebbian-like learning rules, supplement Hebbian increases in synaptic efficacy with some type of rule enabling decreases, while the latter also deviate in their conditions for increasing synaptic efficacy. The debate regarding whether Hebbian learning occurs within biological neural networks lingers on even today, with some claiming supporting empirical evidence (Singer, 1985), and others claiming an absence of such evidence (Camardo, Siegelbaum, and Kandel, 1984).

The current resurgence of activity in ANN research, which is largely dominated by Hebbian-derived learning rules, prompted the following comments from Allen Selverston (1988):

"For biologists, the hyperbole surrounding the promise of artificial networks being able to duplicate mental functions produces a certain feeling of deja vu. Such computational schemes have come and gone with a period of about fifteen years and it is premature to depict the present upswelling of activity as anything more or less than previous attempts to explain mentation." (Selverston, 1988, p. 109).

"While the use of the mammalian brain as a model for computer engineers may sound plausible and even somewhat glamorous, the fact is that we really know very little about the brains detailed microcircuitry. In addition, neurons with grossly oversimplified physiological properties, and synapses whose main function seems to be only blind obedience to Hebbian learning rules, have come to be the foundation of this new approach." (Selverston, 1988, p. 109).

Selverston (1988) goes on to list numerous cellular and synaptic properties which are generally ignored by ANN researchers. Since they are primarily concerned with overall behavioral performance, ANN researchers seek to introduce only the minimum component complexity required, as additional complexity would only unnecessarily complicate an already difficult task, and consume additional processing resources. The main problem, is that no one really yet knows precisely which cellular and synaptic properties are functionally critical, and which are superfluous. Furthermore, the still
rudimentary **physiological** understanding of neural function (let alone neural network function) precludes the use of a biologically correct and complete neural element in ANN systems.

The abundance of behavioral data available from studies of associative conditioning, while still far from fully understood, seems to be a more **reliable** and direct source of data to aid in the design of ANN elements than does the limited detailed physiological **data** of neuron function currently **available**. In any case, it is an animal's behavior which is directly shaped by reinforcement (discussed further below), and not the internal means by which it produces its behavior. Many differing mechanistic solutions are probably capable of **generating** the required behavior. Indeed, different and sometimes overlapping hardware solutions to common behavioral problems exist both within an individual's nervous system, and among different species.

It is therefore **not** necessarily a **mistake** for ANN neuronal models to generally ignore most specific **cellular** and synaptic properties of biological neurons. However, the already apparent complexity of real neurons should at least suggest to ANN researchers that elements of greater complexity than are currently being utilised may be required to produce the desired adaptive behavior.

**TYPES OF FEEDBACK**

ANNs may be subdivided into three main categories, on the **basis** of the type of feedback required to guide learning. The three categories of feedback are none, full, and reinforcement feedback. Each is discussed in turn below.

**No Feedback**

Those ANNs which require **absolutely no feedback** include Hopfield networks (Hopfield, 1982), Boltzman machines (Ackley, Hinton and Sejnowski, 1985), and the **Cognitron** and Neocognitron (Fukushima, 1975, 1988). The **first** two **utilise** adaptive equilibrium processes in which symmetrically interconnected elements interact to enable the total network to reach some minimal energy state, or
"cost function". However, these types of systems have not been shown to be relevant to the learning mechanisms underlying biological neural network function (Klopf, 1987, p. 99). Fukushima’s network models automatically form "clusters" of similar spatial pattern inputs, according to an inherent (built-in) measure of similarity. These clusters can sometimes provide sufficient behavioral specificity, because "similar" input patterns usually require similar behavioral responses. However, the environment is ultimately the best determinant of what degree of discrimination is required in various circumstances, and should therefore play some role in guiding the formation of such clusters.

Barto comments that "Unsupervised learning is more accurately regarded as supervised learning with a fixed, built-in teacher", and that "A supervised system is in fact more adaptive than is an unsupervised system because it forms clusters in order to solve problems posed to it by environmental contingencies rather than to solve a problem of its own." (Barto, 1985, p. 239). As such, the "unsupervised" learning exhibited by systems which utilise no environmental feedback, is really less powerful than that of systems which utilise such feedback.

Full Highly Specific Feedback

ANNs of this type require detailed specification of the desired output on every learning trial. The most popular of these types of systems in recent times are those utilising "back propagation" (Werbos, 1974, 1988; Rumelhart, Hinton, and Williams, 1986). In brief, these systems provide a means of propagating the specific difference between desired and actual output response of the output layer, back through the intermediate layers so that they can adjust their performance in a manner which reduces the output error signal. These types of networks tend to be algorithmic in nature, usually reaching an optimum solution quite reliably, for particular types of prespecified problems.

While an external teacher can play an important role in natural intelligence at higher cognitive levels, it does not usually do so at fundamental cognitive
levels. Furthermore, the overall gradual refinement of response by explicit comparison with desired response can only allow a system to attain a prespecified response to particular inputs. There is no scope here for the system to discover new improved responses on its own, or to adjust to novel situations for which a prespecified response cannot be provided.

Reinforcement Feedback

This type of system requires some feedback from the environment, but does not require an external teacher. Explicit desired-response feedback is replaced by a less specific reinforcement feedback signal. In the context of operant conditioning the reinforcement feedback is a simple scalar signal which indicates the degree of success of the system's response, without providing any specific information regarding how behavior should be altered to improve performance. It is the responsibility of the system to determine such details. These systems tend to be heuristic in nature, providing reasonably good adaptive performance in new and changing circumstances, where algorithmic optimal solutions are unable to be predetermined.

In the case of classical conditioning, a reinforcement feedback system is phylogenetically predisposed to associate particular stimuli (USs) with particular and generally appropriate responses (URs), and then to learn to produce (normally similar) responses (CRs) following perception of previously neutral antecedent stimuli (CSs). Hence, classical conditioning more directly extracts information from the reinforcement feedback signal concerning the type of response to be produced, than operant conditioning, but less fully exploits the potential to produce successful new types of response. In the context of classical conditioning, the reinforcement feedback signal may appear to act like an external teacher, though no explicit active external teacher actually exists - only the contingency-rich external environment.

Reinforcement feedback systems operate in a manner most analogous to natural intelligent systems. In fact, it is the very same reinforcement feedback described here that is used to guide learning in the associative conditioning experiments which provide the behavioral specifications for the research
described herein. As a consequence, theories of classical associative conditioning (e.g., Konorski, 1948, 1967; Rescorla and Wagner, 1972) are also theories of reinforcement feedback systems. Similarly, neuronal models of classical conditioning (e.g., Barto and Sutton, 1985; Klopf, 1987), including ACE, are also AI models of reinforcement learning at the single element level. A small scale network of reinforcement feedback elements has also been shown to produce reasonable results using reinforcement feedback (Barto, Sutton, and Anderson, 1983).

**INTRA-ELEMENT MECHANISMS**

Neural functions are mediated by chemical, and electrochemical processes. These differ considerably in nature from the electronic processes underlying digital computer component operation, and can appear to be simpler, at least to the casual observer. However, this simplistic view is at odds with even the current partial knowledge of intracellular processes, which is revealing an increasingly complicated network of chemical interactions (e.g., Carew, Abrams, Hawkins, and Kandel, 1984). The spatial interconnection pathways which form the substrate of most neural interaction, and which already make good use of all three spatial dimensions, are augmented by an impressively diverse set of chemically discrete interconnections which share physical pathways. This enables a very complex system to occupy a tiny volume, by reducing the number of physical structures required to implement discrete spatial pathways which tend to consume more space. In addition, the relatively slow rates of change of accumulated substances, due to both slow production, transport, and consumption mechanisms, can become a critically important aspect of intraneural function. Hence the mechanistic complexity of a biological neuron is even greater than its already intricate physical structure alone might suggest.

The basic building blocks from which complex neural mechanisms are formed tend to be dominated by operations approximating analogue multiplication and summation, which affect rates of production, accumulation, consumption, or transport of chemical quantities. Also, the use of cumulative quantities which cannot physically take on negative values introduces non-linearities similar to rectification, in which only positive values are allowed. However, as alluded to
above, such *simple* types of interaction may be *combined* in relatively sophisticated *local* circuits to produce much more sophisticated behavior.

In summary, mathematical models of intsa–neural mechanism would be dominated by operations, which although *simple* in themselves, *may* be combined to form very sophisticated systems. The same approach is *adopted* for the development of the intra–element mechanisms documented herein, in the expectation that it *may* help guide the research along a type of path which is demonstrably capable of supporting *classical* conditioning behavior.
2

MODELS OF CLASSICAL CONDITIONING

INTRODUCTION 2-2

THE RESCORLA–WAGNER MODEL 2-3

THE BARTO–SUTTON NEURONAL MODEL 2-6

THE KLOPF NEURONAL MODEL 2-7
INTRODUCTION

Models of classical conditioning were preceded by more general explanatory ideas, which became progressively less vague. The early ideas of merit emerged from those researchers pioneering experimental procedures for studying conditioning phenomena in animals.

There is a tendency for the uninitiated to initially visualise associative conditioning as an entirely excitatory process, with increasing strength of association producing greater excitation. However, the apparent influence of an opposing inhibitory process assumed a role of major importance for many early researchers. The importance of inhibition in associative conditioning was recognised by Pavlov (1927) in his pioneering work, and indeed was responsible for most of his ideas. Konorski (1948) further formulated more specific concepts of inhibition, which have since become the basis of most recent models. Konorski regarded inhibition as an associative phenomenon which complemented excitatory associative phenomena, and proposed some possible models.

Konorski considered that both excitatory and inhibitory conditioning were mediated by the learning of connections between CS and US centres, having an excitatory or inhibitory (respectively) influence upon the activation of the US centres. In effect, the inhibitory connections determined the threshold level which excitatory inputs must exceed in order to activate the US centre. In other words, the net excitatory and inhibitory influences are summed. This is why experimental summation procedures use observed reductions in the efficacy of conditioned excitors in order to infer the strength of conditioned inhibitors, which on their own elicit no specific observable response. Just as the excitatory connections strengthen if the US centre reinforcement increases unexpectedly following CS presentation, inhibitory connections strengthen if US reinforcement is less than expected following CS presentation.

Konoraki (1964) later abandoned this view, introducing "no-US" and "US" gnostic units which inhibited one another, and postulated only excitatory conditionable pathways between the CSs and these gnostic units. However,
Rescorla (1979) considered that this change was probably motivated more by Konorski's desire to integrate conditioned inhibition into his new framework, rather than to provide a better fit with experimental data. In any case, the newer model was behaviorally essentially identical to the first model which incorporated only US gnostic units. Konorski's original model remains the most influential for modern-day researchers developing neuronal models of classical conditioning.

While most of the basic concepts were shaped by the work of the early experimentalists, there has been an increasing trend in recent times for mathematicians and engineers to apply their problem solving skills to the basic issue of classical conditioning, and for experimental psychologists to rigorously define their models mathematically. Those researchers producing the most noteworthy models for classical conditioning have been prepared to address its considerable behavioral complexity (Rescorla and Wagner, 1972; Barto and Sutton, 1985; Klopf, 1987; Grossberg and Schmajuk, 1987, 1989; Gluck and Thompson, 1987; Kehoe, 1988). Most of this work has also utilised computer simulation to test model performance, and facilitate comparison with results from animal experiments. That work which is most relevant to the research to be described will now be summarised critically.

THE RESCORLA-WAGNER MODEL

The symmetry between excitatory and inhibitory rules of associative learning desired by Konoreki was exemplified by a later model which was, in this respect, completely symmetrical. Two researchers who had been developing separate theories of Pavlovian conditioning decided to collaborate when they realised that their differently expressed theories were functionally very similar. The result was the Rescorla-Wagner model (Rescorla and Wagner, 1972). This model was significant because it provided a more integrated theoretical account of associative conditioning phenomena than other preceding theories, and was clearly expressed in mathematical form. Rescorla and Wagner found that a formal mathematical specification of their verbally expressed formulations greatly assisted comparison between the model's performance and
experimental results, and also facilitated development of the model itself. Their model is described by the following equation:

$$\Delta V = a \cdot b \cdot (L - V) \quad [2-1]$$

Where:
- $V = \text{Compound associative strength (} + \text{ve=excitatory, } - \text{ve=inhibitory)}$
- $\Delta V = \text{Change in the associative strength of a CS after each trial}$
- $a = \text{Learning rate associated with a CS } (0 \leq a \leq 1)$
- $b = \text{Learning rate associated with the US } (0 \leq b \leq 1)$
- $L = \text{Asymptotic strength of association supported by the US}$

The term $(L - V)$ in Equation [2-1] expresses the difference between the associative strength supported by the US, and the associative strength of the preceding CS(s). In other words, the difference between the effect of actual reinforcement delivered and that expected. The associative strength of each CS is thus changed when a discrepancy between expectation and result is detected, and changed in such a way as to diminish this discrepancy. Terms $a$ and $b$ control the rate of learning, subject to variations in the salience of the CS, and the effectiveness of the US, respectively. Note that $V$ can be either positive or negative, corresponding to an excitatory or inhibitory compound CS respectively, and that the same simple learning rule governs changes in the associative strength of both types of CS.

The main power of the Rescorla-Wagner model comes into play when compound CSs are employed. Terms $\Delta V$, $a$, and $b$ remain specific to individual CSs, but $(L - V)$ relates to the discrepancy between compound expectation and compound reinforcement delivered. This means that the change in associative strength of each CS component of the compound CS is dependent upon the total associative strength of the compound, which integrates a number of important empirical results. These include stimulus amplitude effects, acquisition of conditioned excitation and inhibition, extinction, overshadowing (except perhaps 1 trial overshadowing), compound conditioning effects (e.g., blocking and unblocking), and discriminative stimulus effects - all of which are defined in Rescorla and Wagner (1972).
Despite its success, the Rescorla–Wagner model suffers many limitations, the most serious being that it is a "trial-level" model. In other words, it only seeks to approximate the state of affairs at the end of each acquisition or extinction trial, and does not address the intratrial processes which produce the end of trial results. Nor does it address aspects of timing within each trial, such as the effect of ISI, or the temporal characteristics of the CS, US, CR, or UR. As such, it cannot reveal many of the underlying processes capable of producing associative learning, and can only approximate the gross results of several general types of experimental procedure.

As impressive as it still is, the breadth of behavior accounted for by this relatively simple model is by no means complete. Furthermore, some characteristics of the behavior that it does produce do not accord well with empirical results. For example, the Rescorla–Wagner model predicts that a conditioned inhibitor (CS–) will extinguish if presented alone. Experimental results indicate that while a conditioned excitor (CS+) will extinguish if presented without subsequent reinforcement, a CS– presented alone (without a simultaneously presented CS+ which expects more reinforcement than actually arrives) is not significantly affected (Zimmer-Hart and Rescorla, 1974). This vivid asymmetry in the behavior of conditioned excitors and inhibitors contradicts the complete symmetry of the Rescorla–Wagner model.

Also, none of the terms in Equation [2-1] relate specifically to direct CS or US characteristics, but are instead abstractly defined quantities somehow derived from them. For example, it is not specified mathematically how the learning rates a and b, associated with the CS and US respectively, are calculated. Similarly, L is not mathematically defined in terms of the US. It is only specified that all three variables increase with increases in the intensity of their respective stimuli.

Finally, the Rescorla–Wagner model predicts a strictly negatively accelerating acquisition curve, despite the common occurrence of sigmoidal acquisition curves (Spence, 1956; Mackintosh, 1974).
THE BARTO–SUTTON NEURONAL MODEL

Barto and Sutton (1985) sought to tackle *intratrial* phenomena not addressed by the Rescorla–Wagner model (1972). They were particularly interested in supporting the anticipatory nature of the CR, and ISI dependency. Their model also responded to direct neuronal CS and US inputs, without using abstractly derived quantities like the Rescorla–Wagner model.

The *Barto–Sutton* model uses averaged previous CS input activation to gate (or enable) changes in the associative strength of the CS. The direction and extent of change is then determined by subsequent changes in output activity. Associative learning between a CS input and the US input is possible because the US also activates the model's output. Using changes in output activity to determine changes in associative strength means that a simple form of both higher order conditioning and sensory preconditioning are also automatically supported.

Conditioned inhibitors are prevented from extinguishing when presented alone, by only permitting non-negative output signals. This type of solution was also suggested by Rescorla (1979) to improve the Rescorla–Wagner model.

However, the *Barto–Sutton* model is excessively sensitive to the timing of CS offset, producing conditioned inhibitors in delayed conditioning paradigms when excitors have been shown to result, in the case where CS and US overlap significantly (Klopf, 1987). Also, like the Rescorla–Wagner model, it still produces only a negatively accelerated acquisition curve.

In general, the main achievement of the *Barto–Sutton* model was to demonstrate that fine grained *intratrial* phenomena could be tackled using a mathematically explicit model. However, its deficiencies invite further attempts to produce more robust and comprehensive models.
THE KLOPF NEURONAL MODEL

Klopf’s (1987) recent drive–reinforcement neuronal model represents an attempt to further improve the modelling of the intra-trial processes underlying classical conditioning. It can be considered as an extension to the Barto-Sutton model, which in turn represents an attempt to temporally refine the Rescorla–Wagner model. Increments in, rather than previous activation of, CS input activity are correlated with subsequent changes in nodal output activity, occurring some time later.

In the abstract of his interim report, Klopf (1987) claims that "It is shown that the proposed neuronal model predicts the basic categories of classical conditioning phenomena including delay and trace conditioning, conditioned and unconditioned stimulus duration and amplitude effects, partial reinforcement effects, interstimulus interval effects including simultaneous conditioning, second-order conditioning, conditioned inhibition, extinction, reacquisition effects, backward conditioning, blocking, overshadowing, compound conditioning, and discriminative stimulus effects." However, examination of the text of his report reveals that the degree of fit between the models' behavior and empirical results is not quite as impressive as the abstract might suggest. Furthermore, being an extension of the Rescorla-Wagner model automatically means that stimulus amplitude effects, conditioned inhibition, extinction, overshadowing, compound conditioning and discriminative stimulus effects are already able to be accounted for.

The "partial reinforcement effects" referred to above do not include the partial reinforcement effect (PRE), in which the rate of extinction following partially reinforced acquisition is retarded, by comparison with that following fully reinforced acquisition. Instead, it refers to the reduced rate, and sometimes reduced asymptote, of conditioning resulting from partially reinforced acquisition (Klopf, 1987, p. 42). This phenomenon is far more simply accounted for than the PRE, and in fact is to be expected from most simple neuronal models of conditioning, since the presence of nonreinforced trials during acquisition Bowers the mean effect of reinforcement.
The "interstimulus interval effects" referred to above do in fact refer to the inverted U shaped curve expressing learned performance as a function of interstimulus interval. However, the shape is not attained by interacting quantities which can produce a smoothly changing relationship, but instead by an arbitrarily determined set of coefficients controlling the rate of learning over coarse 500ms intervals (Klopf, 1987, p. 45). This type of "solution" does not really provide any insight into the type of mechanism which might be responsible for the behavior.

Finally, backward conditioning produces conditioned inhibition with Klopf's model (p. 57), which is by no means unanimously established empirically.

Mechanistically, Klopf's neuronal model also suffers two further deficiencies: (i) If the CS and US terminate simultaneously, and the duration of each is progressively increased, the extent of negative feedback provided by offset of output signal which occurs at CS and US offset, quickly diminishes to zero. In these circumstances, the associative strength of the CS would keep on growing with each acquisition trial, never levelling off.

(ii) Klopf (1987) uses CS excitatory weight to gate changes in it, to produce both S shaped acquisition (p. 4) and faster reacquisition (p. 55). However, the faster reextinction also observed following the alternate acquisition-extinction sessions which produce more rapid reacquisition is not supported by this mechanism, or any other aspect of the model. Using CS excitatory weight to gate its own learning also necessitates some means of avoiding a zero excitatory CS value. Furthermore, this technique invariably produces sigmoidal acquisition curves when acquisition begins with a weakly excitatory CS, which while consistent with most subject-averaged empirical results, is not consistent with the results from all individual subjects (Spence, 1956, p. 65).
OVERVIEW

In this chapter, the type of association to be supported by ACE, the nature of its inputs, and the nature of its output are established. The basic characteristics of ACE differ markedly from those of previous neuronal models of classical conditioning. Issues concerning the internal organisation of ACE are covered in later chapters.

Unlike most other neuronal models of classical conditioning, ACE forms an association between the CS and a representation of the US, rather than between the CS and the CR. Consequently, the output from ACE is not equivalent to the CR, but instead is best regarded as an indicator of the expectation of the US by the CS. This output signal is suitable for subsequent generation of the CR, and as a source of conditioned reinforcement for both higher-order conditioning and instrumental conditioning. Considerable empirical evidence in support of this concept is presented.
HIGHER-ORDER CONDITIONING

It has become routine for neuronal models of classical conditioning to directly support both first-order and higher-order conditioning. This is usually achieved by implementing a literal interpretation of stimulus equivalence in which a CS and a US are indistinguishable as sources of reinforcement and response production. Both Klopf (1987) and Barto and Sutton (1985) attribute the US input with the same effect upon the output of their models as an excitatory CS, and the consequent changes in output signal as the sole source of reinforcement.

Aside from minimising system complexity, the main advantage of this approach is that a simple form of higher-order conditioning is automatically supported by whatever learning rules are developed to support primary conditioning. However, significant differences exist between the empirical characteristics of higher-order and first-order conditioning, which suggest that such total stimulus and reinforcer equivalence is ill conceived.

Gormezano (1984) notes that a US is considerably more effective than an excitatory CS as a source of reinforcement at longer ISIs. Holland and Rescorla (1975a, b) found that the performance of a higher-order CS can be essentially independent of drive level, while the performance of a first-order CS appears to be strongly modulated by drive. In addition, the type of CR able to be easily conditioned to a CS is often quite restricted for first-order conditioning, but is much less restricted in higher-order conditioning (Rescorla, 1984). Also, "the nature of the CR elicited by a CS paired with a US does not seem to have any effect on the nature of the CR it is able to establish to a second CS as a result of higher-order conditioning." (Mackintosh, 1983, p. 16).

These observations suggest that the locus of higher-order association is functionally distinct from that of first-order conditioning, effectively precluding its inclusion within a single neuronal model of first-order classical conditioning.
Mackintosh (1983) argues that "A theory of [classical] conditioning must first of all be a theory of how associations are established between stimuli ... and reinforcers, and only then a theory of how associations are translated into performance. The two questions are distinct, and attempts to ignore the distinction have provided bad answers to both questions." (p. 19). The reasoning used to reach this conclusion can be summarised as follows:

Formation of simple S-R (Stimulus–Response) associations alone should be rejected because they cannot account for the ability of a CS to reinforce new conditioning (either higher-order or instrumental) in terms of its corresponding CR. This is primarily because the nature of the higher-order or instrumental response subsequently conditioned can be very different to the CR elicited by the CS. Mackintosh cites several experimental results in support of this (Holland, 1977; Leyland, 1977; Nairne and Rescorla, 1981). The implication is that the reinforcing ability of a CS cannot be dependent upon its CR, but instead may depend upon an association between the CS and the original US. This CS-US association would then be followed by a subsequent association with a particular CR, in order to translate the CS-US association into performance of the CR. Even though a single ACE does not directly support either higher-order or instrumental conditioning, it does need to support classical conditioning in a manner which facilitates this behavior when interacting with other elements within a network. For example, "A first-order CS paired with food can be used not only to establish second-order classical conditioning to a second CS, but also to serve as a conditioned reinforcer for instrumental responses on whose occurrence it is made contingent" (Mackintosh, 1974, p. 89).

Further support for a CS-US rather than a CS-CR association comes from the results of specially devised blocking experiments (Blanchard and Honig, 1976; Leyland and Mackintosh, 1978; Holland, 1977). Mackintosh (1983, p. 18) notes how these indicate that the ability of one CS to block conditioning to a second CS depends upon the use of a common reinforcer (US) for each, and not on the similarity of their CRs, or their similarity to CRs potentially conditionable
to the second CS. Since ACE is to directly support blocking phenomena, this also strongly suggests that ACE should form CS-US associations.

It would therefore be a mistake to make the output of a conditioning element such as ACE correspond to either the UR, or the CR. Indeed, there is considerable evidence against the formation of direct CS-UR associations (Mackintosh, 1983, pp. 51-56). Instead, ACE's output signal should be regarded as a "CS expects US" output, which is subsequently converted into a CR by further ANN elements.

Note that this conclusion is very different to that implicitly assumed in the neuronal models of both Klopf (1987) and Barto and Sutton (1985), which regard the output from their elements as corresponding to both the UR and the CR. This seems a most unlikely solution, since Soltysik (1971) found that increasing satiation may have a drastic effect upon a salivary CR, but little affect upon the UR - which is difficult to achieve when both are united at the point at which the CR is first adaptively derived. Furthermore, as discussed above, they seem unaware of the mounting indirect evidence which suggests that reinforcer equivalence is better accounted for by inter-element interactions, rather than intra-element processes. Perhaps the lure of a potentially simple solution which directly produces both responses, and which also facilitates the ability of each to act as a common source of reinforcement, diverted the attention of Klopf (1987) and Barto and Sutton (1985) away from these complexities.

If ACE's output does not correspond with the UR, then additional ANN elements are required to produce the UR in response to the US. It follows that, unlike the neuronal models of both Klopf (1987) and Barto and Sutton (1985), the US input need not produce an output response from ACE. Indeed, a more flexible relationship between the CR and the UR can be facilitated by not allowing the US to directly activate ACE.

If the US input does not activate ACE's output, then changes in output activation alone no longer contain sufficient information to support a
mechanism of reinforcement of CS–US associations. Thus, a new reinforcement mechanism also needs to be developed.

In summary, ACE is to support the formation and maintenance of associations between conditioned stimuli and reinforcement, with its output providing a measure of the positive expectation of reinforcement. Subsequent ANN elements, to be developed by research other than that described herein, will be responsible for converting this expectation of reinforcement into the performance of a specific CR. The US input to ACE will function only as a source of reinforcement, and will not be able to directly energise ACE's output. All of these fundamental characteristics differentiate ACE from the neuronal models of Klopf (1987) and Barto and Sutton (1985).

CS INPUT CHARACTERISTICS

From the experimenter's point of view, both the CS and the US are conceptually discrete sensory events. However, the subject is presented with numerous component stimuli, from both the explicitly scheduled CS and US events, and the experimental context. From this jumble of stimuli, the subject needs to selectively associate particular stimulus components with some aspect of the US (discussed further below) based upon the contingencies it experiences, its prior experience, and any phylogenetically determined preferences for associating particular types of events.

An ANN element, operating within a distributed system, is effectively precluded from being presented with a neatly predefined CS input by the potential diversity of a CS. Instead, an ANN element is more likely to be presented with an array of different component CS inputs, from which it may selectively associate with the US representation of the element, those which most reliably predict an unexpected subsequent change in US availability. A unified CS representation will usually then be formed during acquisition of the CS–US association. A subject given sufficient opportunity to discriminate a CS from other stimuli, will associate those stimulus aspects which distinguish the CS from other stimuli, with the subsequent unexpected changes of reinforcement.
The above considerations suggest that many potential CS stimulus features should converge upon a single ACE, so that some of those features which differentiate the CS from other stimuli may come to be associated with the US. However, a single ACE need not become dedicated to a single CS, for it would then be unable to support the blocking phenomenon observed amongst multiple CSs which share a common reinforcer. Hence, the multiple CS stimulus features will activate a mixture of different, possibly incomplete and overlapping, learnt CS definitions, each consisting of at least one CS input. Each CS stimulus feature corresponds to a "CS input" to ACE. For the purposes of testing, a single CS input is often sufficient to demonstrate many types of conditioning behavior, and so can be considered to correspond to a complete external CS event. However, in practice, and for the demonstration of some specific types of behavior (such as overshadowing and blocking), multiple CS inputs will be simultaneously activated.

US INPUT CHARACTERISTICS

Unlike a CS, a US requires no explicit experimental conditioning in order to produce its UR. Indeed, this is why Pavlov referred to it as the Unconditioned Stimulus, and its response as the Unconditioned Response (Pavlov, 1927, p. 25). It is therefore reasonable to assume that unlike a CS, an internal classification of the US has already been formed. It remains to consider how specific this classification is to a particular US. Two main possibilities exist, corresponding to two types of classical conditioning, which are distinguished by the type of the UR they produce (Konorski, 1967). The first, termed "preparatory conditioning" produces URs which are diffuse expressions of a general emotional state, such as approaching an appetitive reinforcer, withdrawing from an aversive one, excitement, or suppression of ongoing activity. Preparatory URs are relatively insensitive to the particular stimulus attributes of the US. For example, a shock administered in many different ways will produce a similar preparatory response.
The second type of classical conditioning is "consummatory conditioning", which produces well defined discrete reflexive responses, such as salivation, blinking and pecking. In contrast to preparatory conditioning, consummatory conditioning produces responses which are very dependent upon the precise nature of the US. For example, a shock delivered to the cheek will produce an eye-blink, while one delivered to the paw will produce a leg flexion UR.

Konorski (1967) assumed that in most cases both preparatory and consummatory conditioning occur. Reasonably consistent empirical evidence suggests that this distinction is indeed valid, that preparatory conditioning is a necessary precursor to consummatory conditioning, and that preparatory conditioning involves an association between a CS and a centralised emotional representation of the type of US, while consummatory conditioning involves association between a CS and precise sensory attributes of the US (Mackintosh, 1983, pp. 56–62).

It was necessary to commit ACE to one type of conditioning, to enable a closer match with specific empirical results, and to limit the scope of research. In the case of classical conditioning, the nature of the CR will usually be similar to that of the UR, and so will also be similar specificity. Consummatory conditioning was selected, because its very nature produces more specific results which facilitate system design and evaluation. The single US input to ACE can therefore be considered to be a highly specific preformed classification of a US, and it is this with which newly formed CS classifications become associated.
DRIVE, PERFORMANCE, AND LEARNING

There is an apparent tendency for drive level to modulate the performance of classically conditioned responses. For example, a satiated subject produces a weaker salivary CR than an unsatiated subject (Soltysik, 1971). However, it is unclear if drive directly modulates CR performance, or if instead it exerts some indirect influence.

The effect of drive level upon learning (as opposed to performance) is even less clear. It appears that drive level exercises an indirect influence upon learning via its direct modulation of performance (Kimble, 1961, p. 413). In the absence of any strong contradictory evidence, it will be assumed that drive level does not directly modulate learning. Intuitively, this has the effect of enabling the acquisition of associations when drive level is low, which will then be available for use when drive level is high.

It remains to consider if in modulating performance of CRs, whether drive level should modulate the "CS expects US" output from ACE. Clearly, it is not necessary that drive act directly upon ACE's output, as many alternative sites downstream of ACE will be available. Furthermore, if ACE's output was directly modulated by drive level, then so also would be the conditioned reinforcement it supports for both higher-order and instrumental conditioning. Since the author is unaware of any evidence in support of this, it is assumed that drive level does not act directly upon ACE, and therefore that ACE does not require a drive input.
SUMMARY

ACE is to directly support first-order consummatory classical conditioning. The basic configuration of ACE, in terms of its inputs and its output, is illustrated below.

![Diagram of ACE configuration](image)

**FIGURE 3-1. Basic input/output configuration of ACE.**

Multiple CS inputs converge upon ACE where they may become associated with subsequent unexpected changes in US input activity. Each individual CS input corresponds to a stimulus feature which may potentially form part, or all, of a learned CS definition, as a result of conditioning. The US input is highly specific to an individual US event, which is considered to produce a similarly specific UR. Both the US and the UR are assumed to be predefined.

ACE's output does not correspond directly to either the UR or the CR, but instead is best thought of as indicating the strength of the US which is expected by previously presented CSs. This output signal is suitable for subsequent generation of the CR, and as a source of conditioned reinforcement for both higher-order conditioning and instrumental conditioning.
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SUMMARY
OVERVIEW

To the Artificial Neural Network (ANN) researcher, associative memory corresponds to the adaptive synaptic weighting which modulates the efficacy of each ANN element input. This weighting is usually retained in a single memory which exhibits simple (normally perfect) retention characteristics, and is modified by various types of simple "learning rules". In the case of the more biologically relevant ANN work, the learning rules normally utilise some combination of pre- and post-synaptic activity to modulate the rate and asymptote of learning. Achieving desirable experience-induced changes in synaptic weighting then consists mainly of devising complex network interactions to produce postsynaptic activity which makes the simple learning rules function more appropriately. This fundamental view is now so firmly entrenched that it requires considerable mental effort to accept that the associative memory itself, if developed further, might be able to contribute substantially to the production of more appropriate learning behavior.

An exploration of the functional relationship between memory and learning is documented herein which has yielded a new nonlinear system of interacting CS-specific (or synaptic) memory types, collectively referred to here as the Neural Multiprocess Memory Model (NMMM). The NMMM is progressively developed from the standard ANN adaptive synaptic weighting, initially producing spontaneous regression and recovery behavior, then U-shaped memory retention, and finally comprehensive adaptive associability behavior. The adaptive associability mechanism supports both negatively accelerated and sigmoidal acquisition curves, latent inhibition, learned irrelevance, the partial Reinforcement Effect (FRE), and accelerated learning following alternating acquisition/extinction training sessions. The NMMM is designed to function as part of the Associative Conditioning Element (ACE), and depends upon interaction with the rest of ACE for its complete operation. Consequently, computer simulation results illustrating the NMMM's full capabilities will be provided when all of ACE is simulated (Chapter 8). This Chapter consists mainly of a qualitative explanation of the NMMM's operation, although computer simulation results are provided herein for those parts of the NMMM that are able to be demonstrated in isolation.
INTEGRATING LEARNING AND MEMORY

Learning and memory tend to be regarded as conceptually discrete processes, with learning pertaining to the acquisition of knowledge, and memory responsible for the retention of knowledge. However, since memory is a necessary component of learning, it cannot avoid contributing to the nature of learning. More specifically, the retention and transfer characteristics of the memory employed have a large impact upon the characteristics of learning, and the temporal characteristics of transfer from acquisition to deployment. Therefore, as the nature and rules of learning increasingly become the focus of ANN research, perhaps the nature and role of memory should also be receiving more attention.

The idealised characteristics of digital computer memory seem to have encouraged a view within the ANN community that the issue of memory is essentially resolved, and that attention should now be focussed upon the development of learning rules and network interaction. Computer memory is typically characterised by extremely fast rates of storage and recall, perfect transfer of data, and perfect retention for practically unlimited intervals of time. With memory like this at our disposal, it is easy to understand the lack of interest in biological memory systems, which are inferior in all of the above respects. However, this type of functional reductionism may not be appropriate.

Having separately identified and categorised the phenomenon of memory, criteria have evolved for its operation which are absolutely separate from its potential applications. No information processing other than storage and retrieval of data is permitted, so that each "memory module" is a general purpose unit which is application-nonspecific. As powerful and useful as this methodology has been, it may not be particularly compatible with the highly distributed and integrated nature of biological neural network systems. In these systems, memory appears to be distributed among processing units in a fine-grained manner, such that mechanisms of acquisition, retention, and deployment are tightly integrated.
In such a case it may not be appropriate to separately idealise the characteristics of memory. In fact, the "non-ideal" information processing and integration characteristics of biological memory can, and most probably do, complement and enhance the learning and deployment processes. The highly integrated functional organisation of natural systems most probably represents an efficient solution to the general problems posed by adaptive systems, about which we still have much to learn.

CONCEPTS OF BIOLOGICAL MEMORY

The Greek philosopher Plato (428–347 BC) likened the nature of human memory to impressions made in soft wax, in which successive traces progressively obliterate preceding ones. Aristotle, a pupil of Plato, later extended this concept to include associations among these traces in order to facilitate recall. As neurological knowledge grew, neural pathways were considered to correspond to associations, and the plastic efficacy of these pathways with memory.

Muller and Pilzecker (1900) suggested that neural pathways must temporarily "reverberate" or remain active after associations are initially formed in order for them to consolidate and become permanent. Their "consolidation theory" provided a possible explanation for the phenomenon of retrograde amnesia, in which a traumatic event can prevent memory of immediately preceding events.

As research progressed further, it became apparent that animal memory included a wide variety of phenomena, for which a single simple memory model was unlikely to account. Researchers abandoned unified models of memory in favor of more specialised models of specific aspects and types of memory. This led to the emergence of multiprocess theories of human memory. Atkinson and Shiffrin (1977) attempted to integrate very short term sensory memory, a general purpose working short term memory, and a long term memory into which information was transferred from short term memory (Figure 4-1).
In the Atkinson and Shiffrin (1977) multiprocess memory model, sensory memory is a characteristic of sensory transducers or other neural hardware associated with sensory analysis. All sensory activation is temporarily retained before it passively decays away. A simple example of sensory memory is visual persistence. The Short Term Memory (STM) depicted in Figure 4-1 also passively decays, but at a slower rate. Also its capacity is very much more limited, and control structures are posited which enable both selective transfer of information into STM, and extended retention of this information by rehearsal. A considerable degree of processing is required to support STM operation, most of which is poorly understood and only crudely specified. STM contents are regarded as abstracted "chunks" of information, such as words or numbers. Although STM capacity is somewhat disputed, early empirical tests with humans indicated an SIM capacity of seven chunks, plus or minus two chunks (Miller, 1956). STM contents are automatically transferred into Long Term Memory (LTM), with effectiveness of transfer increasing with strength and duration of STM activity. LTM was assumed to be permanent, and so free of passive decay.

While such a generalised multiprocess memory model addresses virtually none of the detailed processing issues of interest to ANN researchers, it does highlight the functional role of specialised memory systems.
ARTIFICIAL NEURAL NETWORK MEMORY

In the context of ANNs, memory is often restricted to the adaptive weighting of connections (Figure 4-2). The connection weighting memory is usually long term in nature, exhibiting indefinite perfect retention. This LTM reacts immediately to local conditions, usually at a limited rate of change to reduce the impact of recent events, and facilitate the integration of experience over many trials. When applied to classical conditioning, the CS output leads to generation of the CR, and LTM determines the strength of the CR.

![Diagram of synaptic memory](image)

Figure 4-2. The standard configuration of synaptic memory for ANNs. The operator immediately below LTM is analog multiplication. Individual CS output signals are typically summed at each ANN element. The CS input and CS output signals may also be considered to correspond to synaptic input and synaptic output signals respectively.

When STM is taken into consideration, it is usually achieved by temporarily sustaining the activation of the element so that it slowly passively decays when not driven by active inputs. This is sometimes supplemented by reentrant positive feedback connections (Grossberg, 1988). Such element-based CS-nonspecific STM can help support cooperative-competitive inter-element interactions to process parallel streams of input activation. However, this type of memory only stores nonassociative information. Furthermore, since the element activity itself forms the essence of this type of STM, it can become a complex problem for the network to differentiate between stored memory contents, and retrieved memory contents in the process of being applied. In other words, it becomes a nontrivial attentional problem to set aside most STM contents briefly while selectively attending to particular STM contents.
It is shown below how this problem can be avoided by incorporating a new form of associative SIM which modulates the extent to which an element responds to input activation, and which is distinctly separate to element activation. If applied at each of the element's CS inputs, this would be akin to a STM version of synaptic LTM (Figure 4-2).

A combined SIM and EIM synaptic memory system is not only appealing for its potential to separate real time performance from STM contents, it can also provide a simple account for the experimental phenomena of spontaneous regression and spontaneous recovery. This type of synaptic memory system does not appear to have been seriously investigated by other ANN researchers.

SPONTANEOUS REGRESSION AND SPONTANEOUS RECOVERY

Spontaneous regression describes a post-acquisition training partial decline in learned performance (Figure 4-3). This is differentiated from simple passive decay by a relatively rapid decline to an intermediate level of responding, rather than a typically slower decline towards zero responding. Spontaneous recovery describes a post-extinction training partial restoration of learned performance.

Spontaneous recovery has been observed within both classical (e.g., Pavlov, 1927, p. 58) and operant (e.g., Ellson, 1938) conditioning experiments. Although given less attention, and tending to be much less pronounced than spontaneous recovery, spontaneous regression has also been observed in classical (e.g., Konorski, 1948, p. 83) and operant (e.g., Mote and Finger, 1943; Spear, Hill, and O'Sullivan, 1965) conditioning experiments.
Both spontaneous regression and spontaneous recovery seem to have been completely overlooked by ANN researchers, perhaps because they may seem to be of little behavioral utility. However, when viewed in the context of a combined synaptic STM and LTM system, a nontrivial behavioral advantage emerges. Consider the following problem: When only synaptic LTM is used, as is usually the case in ANNs, at what rate should LTM learning proceed? A fast rate of learning will enable an organism to rapidly adjust its behavior to suit new or changing environmentally imposed contingencies. This has obvious advantages in life threatening situations. However, if the experienced contingencies are of a statistical nature, as would be commonly experienced by individual elements of a highly distributed neural network system operating in a complex environment, then a slower learning rate is required to capture the mean long term contingencies from many individual trials.

Spontaneous regression and recovery may provide a clever mixture of the behavioral advantages of both slow and fast learning rates. On the time scale
of individual trials, learning is able to proceed at a rate faster than that which is optimal for integration of long term experience, so that temporarily contiguous performance can more rapidly adjust to recent contingencies. But as time proceeds, long term experience is permitted to partially reassert itself, with the net steady-state result (being reached sometime after the last trial) representing a compromise between short and long term experience.

ASSOCIATIVE STM AND LTM

Since Pavlov (1927, p. 58) first observed spontaneous recovery behavior, the pursuit of a theoretical explanation for it has occupied many notable researchers (Pavlov, 1927, p. 60; Hull, 1943, pp. 285–286; Skinner, 1938, 1950; Estes, 1955). A new Neural Multiprocess Memory Model (NMMM) integrating associative STM and LTM will now be proposed, which is intended to account for not only spontaneous recovery, but also spontaneous regression. That both spontaneous regression and recovery may result from the operation of a common mechanism was suggested some time ago by Estes (1955). However, the NMMM provides a new and relatively simple account for these spontaneous phenomena, and does so in the form of a functioning model rather than just a theoretical construct. It will be shown later that this new memory system is also easily extended to support other important memory related phenomena of substantial behavioral utility.

Figure 4-4 illustrates how the proposed NMMM is organised. A synaptic (CS-specific) form of STM replaces the synaptic LTM normally used to modulate the efficacy of interelement pathways (Figure 4-21, while LTM is relegated to a background role, in which it is not directly accessible. The impact of LTM upon performance is thus only indirectly apparent via its influence upon STM. In turn, LTM is not directly affected by experience. The effect of experience upon STM, through the system learning rules (detailed in Chapter 7), is simulated in this chapter by altering the initial value of STM - because it is the memory retention and transfer characteristics of the NMMM that are of primary interest here, and experience-induced changes in STM occur at much faster rates. LTM value is then indirectly affected by experience via STM.
FIGURE 4-4. The NMMM, integrating associative STM and LTM. All signals and quantities may attain positive or negative values, except for the synaptic input, which is restricted to non-negative values. +/- indicates that the effect of the signal matches its polarity, while -/+ indicates a converse relationship. The operator immediately below STM is analog multiplication, while that immediately above STM is analog summation. (While the system could easily be reorganised and expanded to utilise only non-negative signals and quantities, this would only unnecessarily complicate the diagram.)

The above NMMM is defined by the following difference equations:

$$\text{STM}[T+1] = \text{STM}[T] + \text{STMchg}.(\text{LTM}[T] - \text{STM}[T])$$  \hspace{1cm} [4-1]

$$\text{LTM}[T+1] = \text{LTM}[T] + \text{LTMacC}.\text{pos}(\text{STM}[T] - \text{LTM}[T]) - \text{LTMdep}.\text{pos}(\text{LTM}[T] - \text{STM}[T])$$  \hspace{1cm} [4-2]

Where:
- SIM = synaptic Short Term Memory.
- STMchg = rate of change of STM.
- LTM = synaptic Long Term Memory.
- LTMacC = LTM accumulation rate, and LTMdep = LTM depletion rate.
- pos(x) = x, if x >= 0, and pos(x) = 0, if x < 0.
- T = current time state, and T+1 = next time state.

Note: The time interval between successive time states is standardised within all ACE equations to 10ms, this being approximately the largest value capable of supporting with sufficient temporal resolution the most rapid rates of change of cumulative quantities, which occur in the CSTC (Chapter 5).
$STM[T]$ is a variable which refers to the level of synaptic Short Term Memory at the current time state T, and $STM[T+1]$ to the level at the next time state $T+1$. Similarly, $LTM[T]$ and $LTM[T+1]$ are variables which refer to the levels of synaptic Long Term Memory at the current and next time state respectively. Equations 14–11 and [4–21] therefore describe how the levels of synaptic $STM$ and $LTM$ for the next time state (10ms later) are derived from their current levels. Synaptic $STM$ and $LTM$ may be regarded as cumulative quantities which take a finite time to change value.

Equations 14–11 and [4–21] relate directly to the NMMM schematic diagram (Figure 4–4), with the addition that a constant is associated with every signal which affects the value of a cumulative quantity. $STM_{chg}$ is a constant which determines the rate at which $STM$ changes, controlling rates of both accumulation (when $LTM[T] > STM[T]$) and depletion (when $LTM[T] < STM[T]$). $LTM_{acc}$ determines the rate at which $LTM$ accumulates, and $LTM_{dep}$ the rate at which it depletes.

This type of nomenclature is standard throughout this document, with 3 or 4 upper case characters mnemonically denoting system variables. Symbols for constants are formed by appending three lower case characters to the end of the upper case mnemonic symbol for the variable with which the constant is associated. "$acc$", "$dep$", and "$chg$" are standard suffixes denoting that the constant affects the accumulation, depletion, and change (both accumulation and depletion) rates of the associated variable respectively.

The NMMM illustrated in Figure 4–4 behaves as follows: after $STM$ is disturbed by a recent experience, $STM$ and $LTM$ interact in a manner which gradually makes them attain the same value, being somewhere in between each of their initial values. The relative rate of change of each memory determines the extent to which each influences the final value. In addition, if different rates of accumulation and depletion are employed (as in Equation [4–2] for $LTM$), an increase in $STM$ can lead to a greater or lesser change in final value than a decrease. This is clearly illustrated in Figure 4–5, in which the accumulation rate of $LTM$ is twice that of its depletion rate, resulting in spontaneous regression of $33\%$, but a more complete spontaneous recovery of $50\%$. 
FIGURE 4-5. Memory retention behavior exhibited by the NMMM, incorporating STM and LTM only. (a) Spontaneous regression behavior. Initially STM = 1.0 and LTM = 0.0, to simulate a relatively rapid increase in STM from 0.0. (b) Spontaneous recovery behavior. Initially STM = 0.0 and LTM = 1.0, to simulate a rapid decline in STM from 1.0. STMchg = 0.0001, LTMacc = 0.0002, and LTMdep = 0.0001, from Equations C4-11 and 14-21.
By supporting spontaneous regression and recovery behavior, the NMMM achieves the apparent mixture of both slow and fast learning rates discussed previously above. This can be visualised as follows:

Figure 4-6. Apparent change in time of the learning rate supported by the NMMM. The fixed rapid instantaneous STM learning rate is depicted at the far right (bold vertical axis). However, as the instant at which STM alteration occurred recedes in time, the effective learning rate diminishes towards a lower asymptote.

At any instant in time, the effective learning rate is relatively fast because STM can be altered rapidly by recent experience via the learning rules (Figure 4-4). However, as the STM-altering experience recedes into the recent past, LTM exerts its stabilising influence upon STM, and the apparent affect of the experience diminishes. This diminished impact of experience upon subsequent performance is normally the result of a decreased learning rate in a conventional ANN synaptic memory model (Figure 4-2). Hence, in the long term, the effective learning rate is substantially slower than that in the short term. This enables the NMMM to support both rapid adjustment to changing short term contingencies, and long term averaging of experience dating back to the distant past.
The way in which STM and LTM are organised within the NMMM is superficially similar to that within the multiprocess memory model of Atkinson and Shiffrin (1977) (Figure 4-1). In particular, the background sole of LTM and the exclusive use of STM in actual performance is common to both. However, further similarities are difficult to find, as the NMMM is relevant to very distributed and Pine-grained operation, the mechanism of interaction between LTM and SIM is specifically defined and different in nature, and the STM is as widespread as LTM, with each having the same capacity. In other words, the 2 memory models operate at distinctly different levels, with the Atkinson and Shiffrin (1977) multiprocess model being distinctly "high level", and the NMMM being very much "low level".

Also, a substantial interval of time (in the order of minutes) is required to enable transfer of information from STM to LTM, or in other words for "consolidation" to occur. This relates well to Muller and Pilzecker's (1900) consolidation theory, and its attempt to explain the phenomenon of retrograde amnesia.

ASSOCIATIVE MTM

The NMMM may be considerably enhanced by incorporating a new adaptive associability mechanism, which is described later in this chapter. This new mechanism requires appropriate controlling signals from a form of associative Medium Term Memory (MTM), and it is primarily for this reason that MTM will now be considered. The addition of MTM to the NMMM also improves the extent to which its memory retention behavior correlates with empirical results. Figure 4-7 below illustrates how MTM is integrated within the NMMM.
FIGURE 4-7. MTM-enhanced NMMM, in which changes in LTM are now mediated by changes in MTM.

MTM-enhanced NMMM difference equations:

\[
\begin{align*}
MTM[T+1] &= MTM[T] + MTM_{acc.\,pos}(STM[T]-LTM[T]-MTM[T]) \quad - MTM_{dep.\,pos}(MTM[T]+LTM[T]-STM[T]) \quad [4-4] \\
LTM[T+1] &= LTM[T] + LTM_{acc.\,pos}(MTM[T]) - LTM_{dep.\,pos}(-MTM[T]) \quad [4-5]
\end{align*}
\]

As shown above, the (STM-LTM) signal which used to directly modify LTM, now modifies MTM instead, which in turn drives changes in LTM. While the basic role of SIM and LTM remains unaltered, MIM now requires time to accumulate towards (STM-LTM) before LTM can begin to appreciably change. The rate of change of MIM is comparable to that of LTM, so that the effect of relatively rapid learning-rule induced changes in SIM upon LTM is delayed.

MIM only plays an active role during the transfer of information between SIM and LTM. Unlike both SIM and LTM, MTM has a steady-state value of zero. Figure 4-8 illustrates how an experience-induced change in SIM value produces a smoothly changing inverted U-shaped deviation in the magnitude of MTM, while SIM and LTM attempt to attain the same intermediate value. Since MTM is capable of neither the indefinite retention of LTM, nor the rapid learning-rule induced changes in STM, referring to it as "Medium Term
"Memory" seems appropriate - even though all three memories have comparable rates of spontaneous change.

Addition of MTM to the NMMM can also significantly alter the course and extent of STM spontaneous regression and recovery, and provide a further opportunity to accentuate the asymmetrical response of the NMMM to changes in STM value by making MTMacc > MTMdep. The existence of such an asymmetry is suggested by the tendency for spontaneous recovery to be much more pronounced than spontaneous regression (Mackintosh, 1974, p. 471).

Figure 4-8a illustrates how spontaneous regression of STM contents now exhibits a pronounced U-shaped response, and ultimately regresses only 10% (compared to 33% without MTM in Figure 4-5a). In contrast, the spontaneous recovery illustrated in Figure 4-8b exhibits only a slight inverted U-shaped response more closely resembling the shape of its counterpart without MTM (Figure 4-5b), and recovers 62% (compared to 50%).

The U-shaped SIM retention of Figure 4-8a results from the following sequence of events: Immediately after the induced increase in STM from 0.0 to 1.0, LTM is still at its original level of 0.0. Similarly, MIM has not had sufficient time to react to the difference between STM and LTM, and therefore remains close to its steady state value of 0.0. STM starts to gradually decline towards the value of LTM, creating the left half of the U-shaped STM response. However, MTM and LTM have also been accumulating in this period, so that after approximately 2 minutes, LTM and SIM values cross. This corresponds to the point in time at the bottom of the U, and with LTM = SIM, would constitute the new steady state value for STM, were it not for the fact that MIM has not had time to deplete back to 0.0. This causes LTM to overshoot STM, which then also increases, forming the start of the right half of the U. The LTM and STM values cross again as a result of the slow rate of change of MTM, producing the damped oscillatory STM and LTM behavior depicted in Figure 4-8a.
FIGURE 4-8. Memory retention behavior exhibited by the MTM-enhanced NMM, incorporating STM, MTM, and LTM. (a) U-shaped STM spontaneous regression. Initially STM = 1.0 and LTM = 0.0, to simulate a rapid increase in STM. (b) STM spontaneous recovery. Initially STM = 0.0 and LTM = 1.0, to simulate a rapid decline in STM. STMchg = 0.0001, MTMacc = 0.0001, MTMdep = 0.00005, LTMacc = 0.0002, and LTMdep = 0.0001, from Equations [4-31, 4-41, and C4-51.
This U-shaped STM behavior compares very favourably with empirical results (Figure 4-9), depicting the retention in honeybees of excitatory conditioning of color and odor using food reinforcement. As previously described, STM level in the NMMM directly determines the current associative strength of a CS, and therefore current response performance. The STM behavior depicted in Figure 4-8a therefore correlates well with Menzel's data (Figure 4-9) in the following respects: The position of the bottom of the U, both in time and magnitude; the maximum amplitude attained in the right half of the U; and the apparent presence of damped oscillations in the right half of the U. The very gradual loss of retention in honeybees, which is almost total after 10 days, may reflect a separate active extinction process, since free-flying bees produced this result.

**FIGURE 4-9. Retention of excitatory conditioning** of free-flying bees trained to a color (long dashes and closed circles) and fixed bees conditioned to an odor (short dashes and open circles, from Mercer and Menzel, 1982). **Note the log scale the axes.** The outer ordinate is the percentage of **correct** choices for free-flying bees, the inner ordinate the responses (proboscis extension) of fixed bees after one conditioning **trial.** Reprinted from Menzel (1984).
Another specific empirical example of U-shaped retention can be found in the avoidance responding of rats (Kamin, 1957). Kamin trained rats over 2 sessions of 25 trials, and found that as the interval between sessions was increased from 0 minutes to 19 days, the number of avoidance responses made in the second session exhibited a pronounced deficit at an interval of 1 hour - a result known as the Kamin effect. Although the time scale of this result is approximately 30 times greater, its general shape also closely resembles that obtained from the MTM-enhanced NMMM (Figure 4–8a). The difference in time scale for this behavior may merely result from the different types of response systems being measured.

Addition of MTM into the NMMM also produces new characteristics which accord with empirical results that were not initially intentionally designed for. The minor STM overshoot depicted in Figure 4–8a for a single excitatory acquisition trial can be substantially accentuated by massing many excitatory acquisition trials (with short ITIs), to produce a very marked overshoot. This type of overshoot has been observed when massed trials are used and rapid acquisition occurs (e.g., Lubow, Markman, and Allan, 1968). Furthermore, when MTM is used to drive changes in associability (as described below), this overshoot may also be responsible for the overtraining reversal effect (Mackintosh, 1974, pp. 602–607).

Menzel (1984, p. 265) also notes how such U-shaped retention resembles the "so-called primacy and recency effects in human verbal learning, where it is found that items encountered first and last are better recalled than middle items (Weiskrantz 1970)." It would seem likely at this stage that U-shaped retention is more than just a curiosity, and that it may, in itself, contribute to advantageous adaptive behavior.
ASSOCIABILITY

"Associability" is a standard concept proposed to account for empirically observed experience-dependent variations in the ease with which the effect of a CS may be altered by training. The basic idea is that an organism not only learns how to respond to a CS, it also learns how fast it should learn how to respond. It is shown below how the behavior of the NMMM can be further extended to include useful adaptive associability behavior that also correlates well with a wide body of empirical data.

Changes in the associability of a CS have been inferred from the results of latent inhibition and learned irrelevance procedures (Mackintosh, 1983, pp. 222–236). It is also claimed here that the Partial Reinforcement Effect (PRE), and the increased effectiveness with which subjects adjust to alternate sessions of fully reinforced and then fully nonreinforced massed trials (Mackintosh, 1974, p. 441), are also able to be mediated by a single adaptive associability mechanism. Furthermore, this same mechanism can produce both sigmoidal (or "S" shaped) and negatively accelerating acquisition curves.

Computer simulation results to support the above claims will be provided in Chapter 8, when the NMMM is functioning as an integral part of ACE. The emphasis here will be upon the development and basic operation of an adaptive associability mechanism capable of producing behavior of directly apparent utility.

In order to actually implement an adaptive associability memory mechanism, several detailed aspects of its functional characteristics need to be determined. These include the temporal characteristics of retention of the level of associability, the precise manner in which associability regulates learning, and the conditions controlling its modification. Furthermore, if necessary, the characteristics of any additional types of memory required to support adaptive associability need to be determined. These issues will now be considered in turn.
Temporal Characteristics of Associability Retention

The long retention capability of altered associability levels exhibited in all of the above mentioned empirical behavior is indicative of LTM storage. In the absence of relevant data, it would seem that dedicated associability STM buffering or interaction is not required. Therefore a single Associability Long Term Memory (ALTM) should suffice to retain the current level of associability between a CS and a US. Note that like synaptic STM, this type of associability is also associative in nature.

Effecting Changing Associability

A point of control capable of supporting adaptive associability within the NMNN is the link which enables learning rules to modify STM contents (Figure 4–10). If ALTM were to modulate modification of STM, this would produce a literal implementation of the apparent nature of associability whereby the ease with which associations are modified is directly controlled.

![Diagram](image_url)

**FIGURE 4-10.** The NMNN, now incorporating adaptive associability levels, which are retained in a dedicated Associability Long Term Memory (ALTM). ALTM controls the associability of the CS input by modulating the effect of STM learning rules upon STM contents.

This type of adaptive associability may be thought of as an adaptive supplement to the combined fast and slow learning behavior already supported
by the NMMM. In terms of the apparently changing learning rate supported by the NMMM (Figure 4–6), this adaptive associability can be visualised as follows (Figure 4-11): The single apparent learning rate curve, supported by the interaction between STM and LTM, is now able to be varied over a range by changing the value of ALTM. Since ALTM is to be altered by experience, the potential exists for ongoing automatic selection of the most appropriate apparent learning rate curve for the particular environmental contingencies recently experienced. Without such an adaptive associability mechanism, only one particular apparent learning rate curve could be preset.

**FIGURE 4-11.** Range of apparent learning rate curves supported by the NMMM incorporating adaptive associability. Increasing associability displaces the entire apparent learning rate curve vertically upwards (on a logarithmic vertical scale). The limits are determined by the range of associability values.
Associative MGM

It remains to develop an **associability learning** mechanism which produces changes in associability that are both behaviorally appropriate, and consistent with empirical results. The inverted U-shaped behavior of MTM (Figure 4–8) provides a potentially suitable controlling signal for an adaptive associability mechanism that meets the above requirements.

As will be discussed below, what is desired is a quantity which indicates the type (reinforced or nonreinforced) of trial(s) recently experienced, excluding the trial currently being experienced. The slow rate of change of MTM ensures that it is not substantially affected by the currently experienced trial, thus satisfying the latter requirement. The intermediary role MIM already plays in transferring STM changes to LIM make it almost, but not quite, suitable for retaining a history of recently experienced trials.

As indicated in Figure 4–8a, a record of a previous increment in STM is only retained in MTM for a few minutes, during which MTM actually goes temporarily negative. What is required is some means of sustaining and stabilising MTM contents, but without substantially affecting LIM or SIM behavior. This can be achieved by gating changes in MTM value with a new quantity which is increased by CS input activity, and then passively decays so that MTM cannot rapidly deplete back towards 0.0. This new quantity is called Memory–Gating Memory, which is hereafter abbreviated to MGM. Figure 4–12 illustrates how MGM is integrated into the NMMM.

Note that in order to counter the effect upon LTM of sustaining MIM contents, it is also necessary to gate changes in LIM using MGM. This prevents sustained non-zero MTM values from producing excessive changes in LTM value. Also, to prevent MIM from remaining permanently at a non-zero value as a result of the previous trial, a simple passive decay mechanism is also implemented (Equation 14–71) to enable residual MTM levels to gradually dissipate. The rate of MTM decay is determined by the constant MTMc {eq}^{ch}g{eq}.
The NMMM, now also incorporating Memory-Gating Short Term Memory (MGM) to modify MTM behavior in preparation for its use to produce appropriate changes in ALTM.

The difference equations for the NMMM with MTM and LTM change now gated by MGM are provided below. Note that Equation [4-9] is incomplete in that it does not specify how the CS input increases MGM contents. This is fully specified later in Chapter 7. It is sufficient for current purposes to assume that CS input activation initially increases MGM contents from 0.0 to 1.0, so that modification of MTM and LTM is enabled (or gated) following CS-input activity.

\[
MTM[T+1] = MTM[T] + MTMacc.pos(STM[T]-LTM[T]-MTM[T]).MGM[T] \\
\quad - MTMdep.pos(MTM[T]+LTM[T]-STM[T]).MGM[T] \\
\quad - MTMchg.MTM[T] \\
LTM[T+1] = LTM[T] + LTMacc.pos(MTM[T]).MGM[T] \\
\quad - LTMdep.pos(-MTM[T]).MGM[T] \\
MGM[T+1] = MGM[T] - MGMdep.MGM[T]
\]

As shown below (Figure 4-13a), this new memory configuration is still able to produce memory retention behavior which compares favourably with that of honeybees (Figure 4-9), when the constants MTMacc and MTMdep are doubled.
FIGURE 4-13. Memory retention behavior exhibited by the MTM-enhanced NMMM, with MTM and LTM change now gated by MGM. (a) Spontaneous regression behavior. (b) Spontaneous recovery behavior. $\text{STM}_{\text{chg}} = 0.0001$, $\text{MTM}_{\text{acc}} = 0.0002$, $\text{MTM}_{\text{dep}} = 0.0001$, $\text{LTM}_{\text{acc}} = 0.0002$, $\text{LTM}_{\text{dep}} = 0.8001$, and $\text{MGM}_{\text{dep}} = .00005$, from Equations 14-61, 14-71, [4-8], and 14-91.
However, MTM behavior has now been modified so that it consistently maintains a trace of prior reinforcement (Figure 4-13a) or nonreinforcement (Figure 4-13b) which is available for hours after the last trial. Also shown in Figure 4-13 are the MGM traces, which after initial energisation by the CS input, rapidly decay leaving MTM at a significant nonzero value of correct polarity.

Although MGM was introduced to gate changes in MTM and LTM primarily in preparation for the use of MTM as a suitable source to drive changes in adaptive associability, it also has the effect of isolating STM from both MTM and LTM, so that STM is available for use as a temporally sensitive register of ongoing US availability. The advantageous behavior able to be supported as a result of this STM isolation is discussed in Chapter 7.

Implementing an Adaptive Associability Mechanism

A new adaptive associability mechanism will now be implemented by utilising the long term memory used to retain associability levels (ALTM), the means by which ALTM modulates the rate of learning, and the new stabilised behavior of MTM resulting from the introduction of MGM.

The NMMM, with the basic elements of this adaptive associability mechanism now integrated within it, is depicted in Figure 4-14. Although computer simulation results demonstrating its operation are only able to be provided when all of ACE is functioning together (Chapter 8), and some mechanistic enhancements are later made (Chapter 7), the qualitative operation of this adaptive associability mechanism in isolation can be fruitfully discussed here. Its basic operation and behavioral characteristics will be revealed by considering in turn 2 types of reinforcement schedules that require the acquisition of extreme associability values for optimum learning behavior.
FIGURE 4-14. The NMMM now utilizing MTM to produce behaviorally appropriate adaptation of associability. The bipolar STM learning rule signal is now separated into 2 non-negative signals, as provided by the mechanism that produces them (Chapter 7). Only that which increases STM (corresponding to reinforcement) can gate changes in ALTM. The 2 new operators towards the upper right corner split the bipolar MTM signal into 2 separate non-negative signals. Icons in the form of miniature input/output graphs indicate that the lower left-most operator passes only non-negative input values, while the upper right one passes only non-positive values which are then converted into non-negative output values of the same magnitude.

Consider first the special case of a schedule in which reinforcement is consistently provided on a variable number of successive trials, and then consistently omitted on a variable number of successive trials, with the succession of reinforced and nonreinforced trials occasionally alternating. An example of such a schedule is provided below, where R denotes a Reinforced trial and N denotes a Nonreinforced trial.

R R R R R R R R N N N N N N N N N N R R R R R R R R R R R R R R R R R R R R R R R R N N N N . . .

Under these circumstances, the current trial very reliably indicates that the next trial will be of the same type (N or R), and the most appropriate learning rate would be a very fast one. A subject would then rapidly adjust to each change in trial type, quickly producing a strong CR when R trials are
experienced, and quickly extinguishing to produce no CR when N trials are experienced.

During the acquisition phase produced by each succession of R trials, MTM contents are made to increase to large positive values, by the same mechanism that increases MTM following a single R trial (the effects of which are simulated in Figure 4-13a). Furthermore, as indicated in Figure 4-14, the reinforcing effect of each R trial is required to enable changes in ALTM to occur. The large positive MTM values then ensure that the ALTM level is increased by each successive R trial, producing the desired high level of associability.

If the sequence of R trials is very long, conditioning will eventually asymptote out to a fixed maximum level, limiting the maximum attainable level of ALTM. Subsequent sequences of R trials will then have little effect upon ALTM level. If short sequences of R trials are employed, ALTM will be progressively increased by each successive sequence of R trials, but its maximum attainable value will still be limited by the asymptotic limit of the strength of associative conditioning.

During the extinction phase produced by each succession of N trials, MTM contents are made to decrease and attain large negative values, by the same mechanism which results in negative MTM values from a single N trial (the effects of which are simulated in Figure 4-13b). In contrast to the acquisition phase, the lack of reinforcement in the extinction phase ensures that the negative MTM values are not able to reduce the value of ALTM. An exception does exist at the commencement of each acquisition phase which is preceded by an extinction phase, where negative MTM values are permitted to reduce ALTM level. However, since MTM has a steady state value of zero, and its magnitude follows an essentially inverted U-shaped profile after STM contents are disturbed by experience (Figure 4-13), this effect can be minimised by inserting an additional delay between N=R transitions. In any case, even if no such delay is employed, this negative effect upon ALTM level will be insufficient to defeat the positive effect of the acquisition phases because of
the number of consecutive R trials. Therefore, a large net increase in associability results from this alternating acquisition-extinction series.

If subjects are given training which consists of alternating fully reinforced acquisition sessions and extinction sessions of massed trials, it is well established that the rate of both reacquisition and reextinction does indeed progressively increase (Mackintosh, 1974; pp. 441-442). It is suggested here that such training produces an increase in associability, which as explained above, is responsible for the increased efficiency of adaptation that has been observed in empirical results (e.g., Bullock and Smith, 1953; Gonzalez, Holmes, and Bitterman, 1967; Davenport, 1969).

Furthermore, it has been shown that the learning rate (as measured by the rate of extinction during extinction sessions) increases with increasing difference between the ITI within each session, and the interval between extinction and acquisition sessions (Capaldi, Leonard, and Keir, 1968). This is also consistent with the above mentioned ALTM negation effect produced when an acquisition session commences shortly after an extinction session - since both this effect, and that of the acquisition session (which increases ALTM) are more effective at shorter ITIs because of the medium term retention characteristics of MTM.

Now consider a randomly sequenced schedule of partial reinforcement, in which the probability of any trial being reinforced is, for example, 50%, and the type of the current trial in no way predicts the type of the next trial. A sequence of such trials might look something like this:

R N R N R N R R R R N N R R R R N R R N R R R R R N R R ...  

The most appropriate learning rate for this type of random schedule would be a very slow one, because it would enable the subject to integrate the outcomes of many trials and produce a consistent CR, with a strength proportional to probability of reinforcement.
With changes in ALTM enabled by the reinforcing effects of every R trial, and the direction and extent of ALTM change determined by those trials preceding each R trial (and in particular the last trial), the adaptive associability mechanism depicted in Figure 4-14 will produce a much lower ALTM value for this type of random schedule than for the systematic one previously discussed. The extent of the difference in ALTM values resulting from this random, and the previous systematic, reinforcement schedule is dependent upon MTM levels being essentially independent of each currently experienced R trial, so that an N–R sequence tends to result in a strong decrease in ALTM, while an R–R sequence tends to produce a strong increase.

The random reinforcement schedule is a type of what is commonly referred to as a Partial Reinforcement (PR) schedule, because only some of the acquisition trials are reinforced. It is well established that a PR schedule will reduce the rate of subsequent extinction training to a rate substantially slower than that subsequent to continuously reinforced acquisition. This effect is known as the Partial Reinforcement Effect (PRE), and will also result from operation of the above adaptive associability mechanism because the reduced associability level affects both extinction and acquisition learning rates.

Furthermore, the following specific characteristics derived from empirical PRE results (Mackintosh, 1974, pp. 443–447) are also consistent with this adaptive associability mechanism:

(i) N trials only increase resistance to extinction when they are followed by an R trial, and become less effective as the interval between them is increased beyond several minutes.

(ii) Early in training, resistance to extinction is primarily determined by the number of N–R transitions.

(iii) Later in training, and at short ITIs (less than 20 min), the number of successive N trials (or "N-length") preceding each R trial in a given session is primarily responsible for determining the extent of the PRE. However, at long ITIs (greater than 20 min), the proportion of N trials in acquisition is a more important factor than N-length.
Characteristics (i) and (ii) result directly from the operation of the basic mechanisms discussed above. The third characteristic results from the asymptotic level of associative conditioning, which also determines the maximum change in ALTM, being determined (reduced) by the (increased) proportion of N trials. Also, the capacity for the negative effect of successive N trials to more effectively temporally sum within MTM increases at shorter ITIs, because of MTM's medium term retention characteristics. This allows N-length to become a more important determinant of the PRE than the proportion of N trials at short ITIs.

SUMMARY

A new network of CS-specific (i.e. synaptic) memory types, referred to as the NMMM, has been progressively developed which produces a wide range of behavioral characteristics that correlate well with many empirical results associated with advantageous adaptive behavior.

The NMMM incorporates synaptic SIM and LTM, which interact in a complementary manner to provide rapid adjustment to new environmental contingencies in the short term, and appropriate integration of recent events with all previous experience in the long term. This is claimed to be the utility of spontaneous regression and recovery behavior, which is generated primarily by these 2 interacting memory types.

The separation between short and long term experience offered by SIM and LTM is further enhanced by the addition of MTM, which mediates change in LTM. These three memories interact to make STM exhibit U and inverted U shaped retention curves, and provide a mechanistic basis for a consolidation theory of memory.

When the inverted U-shaped behavior of MTM is slightly modified by MGM, which is specially designed for this purpose, MTM becomes a highly suitable controlling signal for the modification of adaptive associability, which is retained in ALTM,
If a CS is consistently correlated with reinforcement during massed excitatory acquisition, then its associability is increased. If the CS is reliably associated with reinforcement sometimes, and reliably associated with nonreinforcement at others, as in alternate massed fully reinforced and nonreinforced acquisition sessions, then its associability may increase even further. This enables it to rapidly adapt to the prevailing contingency, and quickly produce the most appropriate response.

If a CS is inconsistently reinforced then its associability becomes (or remains) relatively low (producing the PRE) so that long term averages can be integrated, and responding can continue through intervals of nonreinforcement. This is appropriate because previous experience indicates that some reinforcement may still be obtained, despite its recent absence.

Such alterations of associability can be, and often are, regarded as high level attentional phenomena, in which it is learnt to attend to, or to disregard, a particular CS. Thus, the implementation of this behavior at such a low, fine grained level, considerably relieves the demands upon network level interactions. It also provides a particularly selective form of attentional processing that is specific to both the CS and the US.
Early theories of associative conditioning, and most artificial neural network learning rules, advocate temporal contiguity as the basis for associative conditioning. However, strict simultaneity between a CS and a US normally yields little or no responding. Conditioning is usually most effective when CS onset precedes US onset, and can normally be reliably produced even when a CS terminates before US onset. Furthermore, the temporal profile of the CR is not the same as that of the CS, and can routinely be made to peak after the CS has terminated. This suggests that a trace of the CS is used, rather than the CS itself, both to produce the CR and to modulate associative learning. The CS Trace Circuit (CSTC) produces a CS trace signal suitable for both of these roles. This chapter describes the development of the CSTC, presents the results of computer simulations of its operation, and compares them with empirical results from experiments upon animals, and computer simulation results from the Spectral Timing Model (Grossberg and Schmajuk, 1989).
INTRODUCTION

The Conditioned Stimulus Trace Circuit (CSTC) is a new nonlinear system designed to model the temporal profile of the rabbits' Nictitating Membrane (third eyelid) Response (NMR). The NMR preparation, initially developed by Gormezano and associates in the early sixties, has been utilized to study classical conditioning. The Unconditioned Stimulus (US) is typically a brief puff of air to the cornea, the Unconditioned Response (UR) is the resultant extension of the nictitating membrane (i.e. the NMR), and the Conditioned Stimulus (CS) typically an audible tone. The initially ineffective CS will acquire a NMR of its own, the Conditioned Response (CR), as a result of repeated presentations of the CS followed shortly afterwards by the US. The main features of the NMR preparation that make it attractive for studying classical conditioning are that acquisition produces an orderly change in conditioned response frequency, latency, and amplitude, with very little contribution to responding from other sources (Gormezano, Kehoe and Marshall, 1983).

The overall temporal profile of the largely discrete NMR is represented by summing over subjects and averaging the momentary CR amplitudes following onset of CS presentation, creating a smoothly changing mean topography of response. When activated by a CS input, the CSTC produces an output signal closely matching the shape of the empirically obtained mean topography of NMR, and the way in which its amplitude and shape vary with changes in CS duration, CS amplitude, and the Inter-Stimulus Interval (ISI) between CS and US onset.

Without a CSTC (or similar), the shape and timing of the CS output signal is identical to that of the CS input, and thus, in these respects, can only convey information about the CS input. It is far more appropriate to utilise timing of the CS output signal to convey when a US is expected by a CS (in addition to how strongly it is expected, which is encoded in the amplitude of the output signal).
The CSTC has been designed as a module to be used within the Associative Conditioning Element (ACE), which models a comprehensive set of consummatory classical conditioning behavior. ACE is responsible for mediating the learning process in which the overall amplitude of the CR (i.e. the associative strength of the CS) is altered as a result of acquisition and extinction training. Consequently, the CSTC is primarily concerned with the temporal profile of the response.

Grossberg and Schmajuk (1989) have developed a "Spectral Timing Model" which also models the mean NMR topography. It proposes that a spectrum of individual response topography curves are triggered by CS input activity, with each peaking at a different time and having a shape similar to the empirical NMR topographies. Two sets of equations define the operation of the Spectral Timing Model. The first set (Equations [A2-1], [A2-2], and [A2-3], in Appendix 2) are responsible for the generation of the individual response topographies. The second set (Equation [A2-4]) model the appropriate selection of response profiles during acquisition, which is dependent upon the CS, the US, and the ISI (i.e. LTM learning). The CSTC performs a similar function to the first set of equations, but more closely matches a greater range of empirical results by using entirely new equations ([5-1] to [5-5] below). The larger system in which the CSTC is to be integrated (ACE) performs the remaining LTM modification associated with classical conditioning, a subset of which is modelled by the second set of Spectral Timing Model equations. The spectral aspect of the Spectral Timing Model has been retained however.

Figure 5-1 indicates how the CSTC output is used both to produce an appropriately shaped CR topography, and to gate the effect of the learning rules upon the associative strength of the CS. As Grossberg and Schmajuk (1989) point out, the latter enables that CR which coincides beat with US presentation to acquire associative strength most rapidly, and thus dominate the timing of the total CR produced. This results in the maximal CR amplitude approximately coinciding with US onset, as observed by Smith (1968). Furthermore, temporally generalised responses occurring about the time reinforcement is expected are produced by the smoothly changing shape of the CSTC output signal.
FIGURE 5-1. The relationship between CS input, CS Trace Circuit (CSTC) output, the associative strength of the CS, and the CR produced by the CS. The 2 operators marked with an asterisk are analog multiplication.

It was Pavlov (1927, p. 39) who first suggested that the central nervous system maintained a sustained trace of stimulus activation in order to support the well established phenomenon of trace conditioning (Mackintosh, 1974, p. 57), in which the CS terminates before US onset. Hull (1943, 1952) later extended Pavlov's simple trace concept with his "molar stimulus trace", which as well as slowly passively decaying after CS offset, also first exhibited a latency of 100-200ms, followed by a sapid accumulation to reach its peak some 450ms after CS onset. With evidence accumulating at the time of optimum classical conditioning in many response systems at an ISI of approximately 500ms, Hull proposed that his molar stimulus trace would have a form resembling the dependence of acquisition upon ISI. Furthermore, Hull suggested that the CR became associated with the specific trace amplitude occurring at the time of US onset, and that the anticipatory nature of the CR resulted from generalisation along the amplitude dimension of the trace. Hence, Hull attempted to simultaneously account for trace conditioning, inverted U-shaped acquisition as a function of ISI, and anticipatory CRs with his molar stimulus trace construct.

In relation to the means by which temporal discrimination and generalisation behavior is achieved, Hull's molar stimulus trace construct, a "macromolar trace hypothesis" because it employs only one CS trace, appears unnecessarily sophisticated. By comparison, the CSTC and the Spectral Timing Model, both "micromolar trace hypotheses", produce this behavior in a much more straight
forward manner. Note, however, that the Spectral Timing Model does not in itself actually maintain a sustained CS trace, but instead relies upon preprocessing for such storage of previous CS activation. In contrast, the CSTC does actually produce an output signal which forms a sustained trace of briefly activated CS inputs. Since the CSTC output also contributes to modulation of the learning rules, this characteristic enables it to directly support trace conditioning. Regarding inverted U-shaped acquisition versus ISI, the CSTC, being a micromolar trace hypothesis, cannot in itself fully account for this (Millenson, Kehoe, and Gormezano, 1977). However, in combination with the learning rules developed in Chapter 7, this behavior is also easily produced.

FUNCTIONAL DESCRIPTION

While the general role of the CSTC has its origins in Hull's molar stimulus trace concept, and the Spectral Timing Model, the extent to which it was intended that CSTC performance match a wide range of empirical results (Smith, 1968; Gormezano, Kehoe, and Marshall, 1983; Millenson, Kehoe, and Gormezano, 1977; Wilkie, 1987) necessitated a fresh approach to its functional development. A "black box" development approach was used in which empirical results provide the required performance specifications, but the system's constituents and means of operation was initially unknown, unspecified, and unconstrained. The result is a new nonlinear system that incorporates new mechanisms which have not been derived from any previous CS trace circuit or model, and which from the very start has been developed to meet purely behavioral criteria. Consequently, no aspect of the CSTC's design, other than the use of simple analogue quantities and types of interaction, has been based upon neurophysiological data. It is therefore not claimed that the CSTC necessarily models the actual biological mechanisms underlying the mean NMR topography. The CSTC is depicted schematically in Figure 5-2. It is defined by differential equations which function continuously and in real time, using signals, variables, and interactions of an analogue mature. These are then simulated using difference Equations [5-1] to [5-5] (below) on a digital computer. The interval between successive system states corresponds to 10ms in real time, which was found to produce sufficient temporal resolution.
The CSTC incorporates four cumulative quantities, variables A, B, C, and D. Quantity A is used to progressively reduce the effect of a sustained CS, so that the CSTC is primarily sensitive to the onset of CS input activation, and insensitive to increases in CS duration in excess of the ISI. Quantity B retains a memory of previous CS input activity, which is available to drive a subsequent response, that may peak some time after CS termination (or "offset"). Unlike the Spectral Timing Model, the CSTC does not require that the CS input "...stays on for a fixed (unspecified) time after CS offset because it is internally stored in short term memory..." by preprocessing circuitry (Grossberg and Schmajuk, 1989, p. 82). Quantities C and D are then primarily responsible for producing the desired mean response topography shapes when driven by B. The overall timing of the response is under the control of the Rate Of Change (ROC) input, which determines the rates at which A, C, and D change. The ROC input value remains static during each test. In practice, ROC
would be a preset constant, and each CSTC would have a different ROC input value in order to produce a spectrum of different temporal response profiles.

**CSTC Equations:**

\[
\begin{align*}
A(T+1) &= A(T) + Aacc \cdot \text{pos}(CS(T) - A(T)) \cdot \text{ROC} \cdot CS(T) \\
&\quad - Adep \cdot \text{pos}(A(T) - CS(T)) \cdot \text{ROC} \\
B(T+1) &= B(T) + Bacc \cdot \text{pos}(CS(T) - A(T)) - B(T) \\
&\quad - Bdep \cdot \text{pos}(B(T) - \text{pos}(CS(T) - A(T))) \\
C(T+1) &= C(T) + Cchg \cdot (B(T) - D(T)) + \text{pos}(D(T) - C(T)) \cdot \text{ROC} \\
D(T+1) &= D(T) + Dchg \cdot (C(T) - D(T)) \cdot \text{ROC} \\
\text{CST}[T] &= C(T) \cdot D(T) \cdot X
\end{align*}
\]

where: \( \text{pos}(x) = x, \) if \( x \geq 0 \).
\( \text{pos}(x) = 0, \) if \( x < 0 \).

And:

- \( Aacc = 0.5 \)
- \( Bacc = 0.020 \)
- \( Cchg = 2.0 \)
- \( Adep = 0.05 \)
- \( Bdep = 0.005 \)
- \( Dchg = 0.4 \)

\( 0.02 \leq \text{ROC} \leq 0.20 \) typically, \( 1 \leq X \leq 50 \) typically.

Difference Equations [5-1] to [5-4] describe how the cumulative quantity values at the next time state \( T+1 \) are derived from the values at the current time state \( T \). Each Equation relates directly to the CSTC schematic diagram (Figure 5-2), with the addition that a constant is associated with every signal which affects the value of a cumulative quantity. For example, \( Aacc \) determines the rate at which \( A \) accumulates, and \( Adep \) the rate at which \( A \) depletes. In general, symbols for constants are formed by appending three lower case charactera to the end of the upper case mnemonic symbol for the variable with which the constant is associated. \"acc\", \"dep\", and \"chg\" are standard suffixes denoting that the constant affects the accumulation, depletion, and change (both accumulation and depletion) rates respectively of the associated variable.
The CSTC produces a CS Trace (CST) output signal (Equation [5-5]), which is formed by the product of \( C, D, \) and a scale factor \( X, \) which is used to scale CST values for graphical presentation. In practice, \( X \) would be replaced by the associative strength of the CS.

The CSTC's operation is most easily explained by way of example. Figure 5-3 shows how quantities \( A, B, C, \) and \( D \) change following a 50ms CS of unity amplitude when generating a peak response at approximately 500ms after CS onset.

![Figure 5-3](image)

**FIGURE 5-3. Typical response of all cumulative quantities (A to D) and the CST output, to a 50ms trace CS of unity amplitude. ROC = 0.05 and \( X = 10.\)**

Initially \( A=B=C=D=0. \) Quantity \( A \) accumulates towards the amplitude of the CS input, at a rate directly dependent upon the amplitude of the CS. The [CS-A] difference signal, which has an exponentially decreasing magnitude, acts to make \( A \) eventually attain the same value as the CS input signal. \( B \) accumulates towards the level of this difference signal, but only partially gets there before CS offset occurs 50ms later. \( B \) then slowly decays away. The increased value of \( B \) makes \( C \) increase, which in turn drives an increase in \( D. \) A simple negative feedback loop makes \( D \) accumulate towards \( C. \) While \( D < C, \) another
feedback loop makes $C$ increase until $D \geq B$. However, by this time, the level of $C$ is more than twice that of $B$, and still driving $D$ upwards. When the level of $D$ increases beyond that of $B$, $C$ is quickly depleted, slowing the rate of increase in $D$. A final feedback loop (located above $C$ in Figure 5-2) effectively results in the level of $C$ (instead of $D$) being compared with $B$ in order to adjust $C$, after $C < D$. This prevents an undershoot of $C$ from occurring. $D$ then decays away, and $C$ tracks $B$ as it continues to passively decay. The output signal is formed by the product of $C$ and $D$, because $C$ provides the required characteristic overshoot, and $D$ the required faster than linear increase before $C$ peaks.

RESULTS

Shape of Mean Topography of Response

Figure 5-4 shows the empirical mean NMR topographies obtained by Smith (1968), using a 50ms trace CS. (It is referred to as a "trace" CS because the CS terminates before US onset, which defines a trace conditioning procedure.) Smith's empirical results indicate that peak time, shape and relative size of topography varies with ISI, and is relatively independent of US strength. Figure 5-5a shows the results of computer simulations using a unity amplitude 50ms CS, with the ROC input adjusted to four discrete settings which make the peaks correspond approximately in time with the empirical results.

It can be seen that the peak amplitudes of each response in Figure 5-4 follow an inverted U function of time. This type of behavior may be utilised to help produce the commonly observed inverted U relationship between rate (and usually extent) of acquisition and ISI (Gormezano, Kehoe, and Marshall, 1983) — and is achieved in the CSTC (Figure 5-5a) primarily by not modulating the rate of increase of substance $B$ by the ROC input. Thus, at short ISIs (which corresponds to large ROC input values), $B$ attains diminishing maximum levels, resulting in smaller amplitude CSTC output responses. When the results of Figure 5-5a are then adjusted in amplitude, as could occur as a result of LIM learning, it becomes particularly clear how close the shape of the simulated results matches that of the empirical results (Figure 5-5b).
FIGURE 5-4. Mean topography of NMR based on five test trials during day 10, using a 50 ms CS with ISIs of 125, 250, 500, and 1000 ms, and 1, 2 and 4 mA intensity shocks as the US. Reprinted from Smith (1968).
FIGURE 5-5. Four CST signal responses to a 50ms trace CS of unity amplitude, with ROC settings of 0.2, 0.1, 0.05, and 0.02, for responses peaking from left to right. (a) $X = 40$ for all responses. (b) $X = 32, 37, 30, and 42$ for responses peaking from left to right. These values were chosen to make the relative size of each response, and their overall aspect ratio, approximately match that of Smith's (1968) empirical results (Figure 5-4) in order to facilitate comparison.
The response curves of the Spectral Timing Model are illustrated in Figure 5-6. A comparison of Figures 5-4, 5-5, and 5-6 reveals that the CSTC more closely reproduces the shape of the experimental topographies, particularly at the shorter and longer ISI's. The main difference relates to the way the response topography decays after peaking, with the CSTC exhibiting the characteristic rapid fall-off that merges into a distinctly slower exponential-like decay. The CSTC also better approximates the almost symmetrical shape of the upper half of the response, about its peak amplitude.

\[
\sum_i f(x_i)y_i z_i
\]

FIGURE 5-6. Computer simulation results for the Spectral Timing Model output response to CS alone input, after 10 learning trials at ISIs of 5, 125, 250, 500, and 1000 ms (left to right), with a 50 ms duration US of 10 unit amplitude. Reprinted from Grossberg and Schmajuk (1989).

While the shape of the CSTC output response compares more favourably with empirical mean topography of NMR results than the Spectral Timing Model, the CSTC's main advantage is its ability to appropriately respond to variations in CS duration and amplitude. This behavior will now be discussed.
CS Duration Effects

A delayed conditioning procedure, in which the CS remains active until US presentation, produces curves similar in shape to those of the trace CS procedure (Figure 5–7). These empirical results primarily reveal the presence of multiple peaks, and by so doing provide support for the existence of a spectrum of differently timed mean response topographies, rather than a single tunable one. However, these results also illustrate the effect that CS duration has on the response topography. CS duration effects are not supported by the Spectral Timing Model, because it assumes that the CS amplitude is retained perfectly in SIM for some unspecified time, presumably far in excess of the time at which the response peaks.

Millenson, Kehoe, and Gormezano (1977) used 5 groups of 6 rabbits to generate the results depicted in Figure 5–7. Groups 200F and 700F received trials at an ISI of only 200ms and 700ms respectively. Groups P 7/8, P 1/2, and P 1/23 received 200ms ISI trials on 7/8, 1/2, and 1/8 of all trials respectively, and were trained at an ISI of 700ms on all remaining trials, with the sequence of trials randomly determined.

Perhaps the most interesting observation relates to the P 1/8 group, in which a 200ms CS produces a 700ms peaking response topography of only half the amplitude of that produced by a 700ms CS. In other words, it would appear that a CS has an on-going influence upon the strength of response. However, this influence is not constant in time, since a 2/7 duration CS produces a 1/2 height response, indicating that the CS exerts most of its influence soon after onset. Furthermore, from the P 7/8 group, it would appear that a 700ms CS produces a 200ms peaking response topography of Little (if any) additional amplitude to that produced by a 200ms CS, indicating that the CS has little effect after the peak amplitude of response has been reached. And finally, since a 200ms CS produces less of a 700ms peaking response than a 700ms CS, but a 700ms CS does not appreciably alter the tail-end of a 200ms peaking response, the on-going influence of the CS must be dependent upon the timing of the response. In terms of the CSTC, this justifies why the ROC input modulates the rate of increase in A, which in turn limits the influence of CS
duration to an exponentially decreasing function of time over the interval from CS onset to US onset.

FIGURE 5-7. Mean topography of NMR, after 3 and 10 days of acquisition to intermixed 200ms and 700ms ISI delay conditioning trials, using a 50ms duration US. CR strength was measured in response to a 200ms duration test CS (left column), and a 700ms test CS (right column). Reprinted from Millenson, Kehoe and Gormezano (1977).
The output response of the CSTC, with the ROC input adjusted to two levels so that the output peaks at 200ms and 700ms when driven by 200ms and 700ms long CS inputs respectively, is shown in Figure 5-8. Four response curves are shown, one for each combination of input duration and the ROC input setting. These confirm that the CSTC response amplitude is affected by input duration as described above, in accord with the empirical results (Figure 5-7).

Note that, because the spectral aspect of the Spectral Timing Model is retained, multiple peaked responses like those illustrated in Figure 5-6 can be obtained by summing individual CSTC output responses. For example, the pronounced 200 and 700ms double peaking response can be obtained by summing the two individual responses to a 700ms duration CS illustrated in Figure 5-8.
CS Intensity Effects

Reducing the CS input amplitude to the CSTC reduces the rate at which quantity A accumulates, and its ultimate asymptotic amplitude. If the CS is of relatively short duration compared to the ISI in acquisition, then reducing CS amplitudes will primarily reduce the amplitude attained by A, B, C, D, and the output response. However, the timing of the CSTC output response is essentially unaltered.

If, on the other hand, the CS is sustained beyond the ISI, then reducing its amplitude will lengthen the period over which A is still accumulating towards its asymptotic level. Normally a unit amplitude CS results in A accumulating to approximately 90% of the CS amplitude at the end of the ISI. However, a smaller CS will result in A continuing to accumulate at a more significant rate after the normal peak response has been produced, which will decrease the subsequent rate of decay of B, or even possibly maintain B's accumulation until some time following the normal response peak time.

In contrast, the Spectral Timing Model (Grossberg and Schmajuk, 1989) responds to reduced amplitude CS inputs by slowing down its overall response timing in proportion to the reduction in input amplitude. Grossberg and Schmajuk cite the experimental data of Wilkie (1987) using pigeons, and his suggested explanation, in support of this behavior. However, the behavior of the CSTC provides an alternate explanation to the experimental results.

While it is somewhat tenuous to relate data from different response systems, species, and experimental procedures, the generality of classical and operant conditioning behavior provides some degree of support for this practice - particularly when more closely related data is unavailable.

Wilkie (1987) trained 5 pigeons to discriminate between 2s and 10s light presentations, such that they pecked 1 of 2 colored keys when illuminated at the end of each 2s light presentation, and the other key at the end of each 10s light presentation. In pretraining, the intensity of the light was alternated between 2 levels (being different for each bird) at the end of each of 30
sessions of 80 trials. Averaged data recorded over the last 3 of 30 sessions of training with each light intensity, for all 5 birds, indicated that "the pigeons discriminated short and long presentations of dim and bright light equally well" (Wilkie, 1987, p. 36). In the experiment proper, the light intensity was randomly set either bright or dim (with 50% probability), and the duration randomly varied between 2s (25%), 4s (16.7%), 6s (16.7%), 8s (16.7%) and 10s (25%) over a further 35 sessions of 80 trials. Correct responses to 2s and 10s light presentations (bright or dim) were immediately reinforced by 5s access to grain, while all other responses went unreinforced.

The number of times the 'short' key was chosen for each of the 10 duration and intensity combinations was separately recorded, indicating the probability of choosing a 'short' response rather than a 'long' one. The empirical results obtained from each of the 5 pigeons, and the overall average, are reproduced in Figure 5-9. This indicates a curious interaction between light duration and intensity, in which intensity has little effect at the short duration of 2s, but exerts progressively more influence as the light duration increases. At the 10s duration, the dim light generally produces a significant increase in relative preference for the short response.

In an attempt to explain these results, both Wilkie (1987) and Grossberg and Schmajuk (1989) suggest that the bright and dim light presentations affect the rate of some form of internal clock, which is used to discriminate short and long durations. However, they both provide only speculative, though conceivably possible, explanations as to why this discrimination is not affected by light intensity at short durations. The results of computer simulations of the CSTC are presented below which directly produce this behavior (Figure 5-10).
FIGURE 5-9. Percentage of trials on which the pigeons chose the "short" response alternative following termination of dim (solid circle) and bright (hollow circle) lights of 2, 4, 6, 8, and 10s duration. The dim/bright light intensities for pigeons 1 to 5 were 0.04/18.5, 0.36/20.5, 0.45/33, 0.90/34, and 0.90/14 cd/(m.m) respectively, measured at 3-5cm. Reprinted from Wilkie (1987).
While pigeons condition well at ISIs from 2s to 10s, the rabbits' NMR conditions most effectively at ISIs of less than 1s. Consequently the timing of the short and long responses needs to be scaled down by a factor of ten, such that 200ms and 1000ms for the CSTC responses correspond (for the purposes of this comparison) to 2s and 10s for the pigeons' responses.

Figure 5–10 shows the output responses produced by the CSTC, with the ROC input set to two levels to produce peak outputs at approximately 200ms and 1000ms for sustained unity amplitude CS inputs. The CS inputs are sustained so that all intermediate duration response amplitudes are indicated by the amplitude of a single response curve at the intermediate durations. While the pigeon CSs were terminated at intermediate periods, their response amplitudes were measured directly after CS offset, which will be close to the response amplitudes produced if the CS had remained active, because of the smoothly changing nature of the mean response topography.

To more clearly illustrate the relative shapes and amplitudes of response for each CS input amplitude, the graph scale factor X for each response pair has been adjusted to produce a maximal response amplitude of 0.9 for each graph. (The CSTC response amplitudes actually decline markedly with decreasing CS input amplitude, as would be expected.) The ratio of X values for the short and long responses in Figure 5–10a is set so that both responses have comparable peak amplitudes for unity CS input. This ratio remains unaltered for subsequent results in which the CS input amplitude is reduced (Figures 5–10b and 5–10c).

Figures 5–10a, 5–10b, and 5–10c indicate how the shape and relative amplitude of the short and long CSTC responses change as CS input amplitude is reduced. The first point to note is that a reducing CS input amplitude has little effect upon the position of the (first) peak in both short and long responses, and it reduces the overall response amplitudes dramatically (note the changed scale factors). The second and most important point to note is that the course of the responses after they have first peaked is substantially enhanced by reduced CS input amplitude.
FIGURE 5-10. The effect of CS intensity on CSTC output responses, at the two ROC input settings of 0.2 and 0.03 for the "short" and "long" responses peaking at approximately 200 ms and 1000 ms respectively. CS duration is 2000 ms for all plots. (a) CS = 1.0, X = 17.0 and 1.76 for the short and long response respectively. (b) CS = 0.3, X = 70.1 and 7.26. Figure 5-10c follows.
The effect of CS intensity on CSTC output responses, at the two ROC input settings of 0.2 and 0.03 for the "short" and "long" responses peaking at approximately 200 ms and 1000 ms respectively. CS duration is 2000 ms for all plots. (c) CS = 0.1, X = 316 and 32.7 for the short and long response respectively. Figures 5-10a and 5-10b are on the previous page.

The latter above mentioned effect selectively enhances the probability of the short response in two ways. First, it reduces the rate of decline of the short response more effectively than the long one. So much so, in fact, that a second (and higher) response peak can be produced for the short response (Figure 5-10c). And second, since both responses are most enhanced after they have first peaked, the short response experiences its boost before the long one.

Consequently, at short durations, before either response has peaked, reduced CS input intensity only marginally enhances selection of the short response. Then, in the interval between the (first) peaks of the short and long responses, smaller amplitude CS inputs selectively enhance the probability of producing the short response; and the extent of this effect increases gradually after the short response peaks.
This is most clearly illustrated by converting the individual response curves of Figure 5-10 into a single 'probability of short response' curve (Figure 5-11). Note how favorably this compares with the experimental results obtained by Wilkie (Figure 5-9), particularly with the individual responses of birds one and two. These birds experience the lowest absolute intensities of light corresponding to the dim light. Note also that the second peak in the short response for small amplitude input (Figure 5-10c) produces small oscillations in Figure 5-11 resembling those of birds one and two.

**FIGURE 5-11.** The effect of CS input amplitude on probability of producing a short, rather than a long response. Note that the time scale has been adjusted to correspond with that of Wilkie's empirical results (Figure 5-9). The three curves were derived from the results depicted in Figures 5-10a, 5-10b, and 5-10c as follows: \( \Pr\{\text{Short Response}[T]\} = \frac{\text{Short}[T]}{\text{Short}[T]+\text{Long}[T]} \).
The CSTC 5-23

SUMMARY

A new nonlinear analog Conditioned Stimulus Trace Circuit (CSTC) has been developed with sufficient complexity to accurately model the shape of the mean NMR topography, and the way in which it changes subject to variations in CS duration and amplitude.

Digital computer simulation results have been presented, along with relevant empirical results from animal behavior experiments, which indicate that the behavior of the CSTC corresponds well with animal behavior in the above respects. Furthermore, computer simulation results from the Spectral Timing Model (Grossberg and Schmajuk, 1989) which also addresses the mean NMR topography have been presented, which illustrate that the CSTC more accurately reproduces the response topography shape produced by a 50ms trace CS. Unlike the CSTC, the Spectral Timing Model does not address the CS duration effects at all, nor does it adequately address CS intensity effects.

The CSTC thus provides a very compact and self-contained solution to the problem of generating a suitable "trace" of CS activity, by making more effective use of the CS duration and amplitude information also made available to it by the CS input. This CS "trace" is available for generation of an appropriately timed CR, and to selectively gate the effect of experience subsequent to CS presentation upon the associative strength of the CS, which in turn determines CR strength.
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OVERVIEW

ACE is organised into multiple CS input channels and a common output stage, upon which all of the CS input channels converge. The Neural Multiprocess Memory Model (NMMM) and the CS Trace Circuit (CSTC), which were previously developed separately in Chapters 4 and 5 respectively, may be combined to produce an almost complete CS input channel. This Chapter describes how the individual expectations of reinforcement from each CS input channel are combined to produce the compound 'CS expects US' output of ACE, and the way in which the US input is processed in order to determine the compound asymptotic strength of association able to be supported by a US. These are all CS-nonspecific mechanisms, and collectively form the common output stage of ACE. The two signals generated by this output stage are fed back to each input channel to drive the SIM learning rules described in Chapter 7.
EXPECTATION OF REINFORCEMENT

Each individual CS input channel is primarily responsible for acquiring the predictive relationship between its CS input, and subsequent delivery (or nondelivery when otherwise expected) of reinforcement. More specifically, each input channel captures the classical conditioning contingency between CS and subsequent US presentation, in addition to the temporal relationship between the CS and the US.

If the US input is more likely to occur shortly following CS input activation than at other times, then a positive contingency exists, and the CS input channel produces an excitatory output signal (EXC) in response to CS presentation. Conversely, a negative contingency leads to the production of an inhibitory output signal (INH) from the CS input channel. As illustrated in Figure 6-1, the "CS expects US" output (OUT) is obtained by subtracting the sum of all inhibitory CS channel output signals (INH) from the sum of all excitatory CS channel output signals (EXC), and then only passing non-negative values.

FIGURE 6-1. That past of the output stage which determines the compound expectation of reinforcement, and the way in which it interfaces with a single typical CS input channel. The operators immediately right of both INH and EXC represent convergent summation from each of the CS input channels to the single output stage.
In many respects OUT corresponds well with term V in Equation [2-1] of the Rescorla-Wagner model (Rescorla and Wagner, 1972), which represents the compound associative strength of all presented CSs. In ACE, OUT also doubles as the decremental feedback signal DEC. Rejection of negative values is an improvement suggested by Rescorla (1979) that prevents further decreases in associative strength when compound inhibitors are presented, which otherwise occurs in the Rescorla-Wagner model.

Note, however, that ACE is a real-time model dealing with intra-trial processes and interactions, while the Rescorla-Wagner model is a trial-level model that aims to account for the result of each trial only. Hence, OUT represents a phasic, timed expression of the expectation of reinforcement that is capable of driving a correctly timed CR, whereas V is a continuous expression of the strength of association between a CS and a US.

Difference Equations 16-11 and [6-2] mathematically define how the OUT and DEC signals are determined from the individual excitatory and inhibitory output signals from each CS input channel.

\[
\text{OUT}[T] = \text{pos}(\text{sum}(N=1 \text{ to } n: \text{EXC}[T, N]) - \text{sum}(N=1 \text{ to } n: \text{INH}[T, N])) \quad [6-1]
\]
\[
\text{DEC}[T] = \text{OUT}[T] \quad [6-2]
\]

Where:
- \( \text{pos}(x) = x \), if \( x \geq 0 \).
- \( \text{pos}(x) = 0 \), if \( x < 0 \).
- \( N \) = Number of CS input channels.
- \( T \) = time state number.

The interval between successive time states is 10ms.

It transpires that this part of the output stage is the most conventional mechanism deployed within ACE. In terms of the generation of the output signal, it corresponds to what has for some time been the basic output stage configuration for ANN elements. However, the range of behavior that it is to help support when functioning as an integral part of ACE considerably extends its conceptual role. This will become apparent in Chapters 7 and 8.
REINFORCING EFFECT OF THE US

Each CS input channel also responds to actual (as opposed to expected) US input activation via the incremental feedback signal (INC), which is distributed to each CS input channel from the output stage. As is the case with DEC, INC has its intratrial counterpart in the Rescorla–Wagner model.

The reinforcing signal INC corresponds to an intratrial phasic version of the compound asymptotic strength of association supported by the US, which is represented by term L in Equation [2-1]. Each CS input channel may therefore be regarded as the site at which compound associative strength (V) is subtracted from compound asymptotic strength of association supported by the CS (L), in order to determine how individual associative strengths need to be altered to reduce the difference between L and V. The short term retention capability of associative STM within each CS input channel (Chapter 4) enables this comparison to occur, despite the asynchronous nature of CS and US presentation, and the different response shapes of the CSTC output signal (CST, Chapter 5) and the reinforcement feedback signal INC.

Incorporating the essence of the Rescorla–Wagner model within ACE automatically means that stimulus amplitude effects, conditioned inhibition, extinction, overshadowing, compound conditioning and discriminative stimulus effects are able to be supported (in addition to the behavior produced by the NMMM and the CSTC).

US Duration Effects

Experiments studying consummatory classical conditioning of the rabbits' NMR have shown that increasing US duration provides more effective reinforcement, but that progressive increases in US duration provide progressively less additional reinforcement (Ashton, Bitgood, and Moore, 1969; Tait, Kehoe, and Gormezano, 1981).

If the US input were to correspond directly to the INC feedback signal which reinforces CS-US associations in the STM of each CS input channel, then the
above qualitative relationship between US duration and extent of reinforcement would already be essentially supported by the temporal modulation of the reinforcing effect of INC upon STM by RGM. A detailed explanation of the RGM's role and behavior is provided in Chapter 7. Figure 6-3 illustrates, among other things, a typical temporal profile of RGM values that would dominate modulation of acquisition at an ISI of 630 ms. The important point to note is that after peaking shortly following US onset (as is consistent with the results of Smith, 1968), RGM gradually declines in amplitude. Thus the integrated effect of INC over time is increased at a diminishing rate by progressive increases in US duration (for US durations in excess of 70 ms), producing the desired qualitative behavior. However, quantitatively, this mechanism alone appears to be insufficient.

Ashton, Bitgood, and Moore (1969) studied the effects of US duration and intensity upon delayed conditioning of the rabbits' NMR using a 630 ms ISI, and US durations of 50 ms and 350 ms. Their results show that the extent of acquisition on day 2 to a 350 ms US was 1.25, 1.63, and 1.94 times greater than that to a 50 ms US (for US intensities of 4, 2, and 1 mA respectively). While the ratio diminished to approximately 11 later in conditioning, this may have been due to a ceiling effect. These results therefore indicate that a 350 ms US appeared to provide somewhere between 25% and 100% more reinforcement than a 50 ms US.

It is clear from Figure 6-3 that if the US input corresponded to INC, which is in turn modulated by RGM, then a 350 ms US would be almost 7 times more effective than a 50 ms US, as determined by the ratio of the areas under the CST curve over the intervals 630-680 ms, and 630-980 ms.

In order to provide a more pronounced attenuation of the INC signal within the first 100 ms of the US duration, and so provide a better fit with empirical results, a new cumulative quantity will now be introduced which is referred to hereafter as Reinforcement STM (RSTM). As shown in Figure 6-2, RSTM accumulates at a rate proportional to the extent by which the US input amplitude exceeds the RSTM amplitude. This same signal forms the reinforcing feedback signal INC. Thus, as RSTM accumulates, INC diminishes.
FIGURE 6–2. The complete output stages of ACE, shown interfacing with a single typical CS input channel. The output stage now incorporates Reinforcement STM to appropriately modulate the effect of increasing US duration upon the compound strength of association able to be acquired by the CS input channels.

The difference Equations defining how RSTM and INC are generated are as follows:

\[
\text{INC}[T] = \text{pos}(\text{US}[T] - \text{RSTM}[T]) \tag{6-3}
\]
\[
\text{RSTM}[T+1] = \text{RSTM}[T] + \text{RSTMacc}.\text{INC}[T] - \text{RSTMdep}.\text{RSTM}[T] \tag{6-4}
\]

Where:
- \( \text{pos}(x) = x, \) if \( x \geq 0. \)
- \( \text{pos}(x) = 0, \) if \( x < 0. \)
- RSTMacc = accumulation rate of RSTM.
- RSTMdep = depletion rate of RSTM.
- \( T = \) current time state.
- \( T+1 = \) next time state.
- The interval between successive time states is 10ms.
Figure 6-3 illustrates the INC signals and RSTM levels produced by 50\text{ms} and 350\text{ms} US durations. It is apparent from Figure 6-3 that the area under the 350\text{ms} INC curve is approximately one and a half times greater than that under the 50\text{ms} INC curve. This ratio is marginally reduced when the effect of RGM modulation of PNC is taken into account. This result, with RSTM used as described above, accords far more favourably with the results of Ashton, Bitgood, and Moore (1969) than when RSTM is omitted.

FIGURE 6-3. Reinforcement Short Term Memory (RSTM) and reinforcing feedback signal (INC) levels following US inputs of 50\text{ms} and 350\text{ms} duration, commencing 630\text{ms} after CS onset. Also shown are the CS Trace (CST) and Reinforcement Gating Short Term Memory (RGM) levels that peak shortly after US onset, which are produced by a 50\text{ms} duration CS. Equations 16-31 and [6-43] were used to generate the RSTM and INC curves, with RSTMacc = 0.30 and RSTMdep = 0.01.

The simple passive decay of RSTM (illustrated below RSIM in Figure 6-2, and defined by the last term in Equation [6-43]) ensures that INC asymptotes towards a positive nonzero minimum value, so that increases in US duration in excess of 350\text{ms} continue to contribute to the reinforcing effect of the US. This is consistent with the empirical results of Tait, Kehoe, and Gormezano.
(1981), in which the percentage of CRs observed continued to increase significantly when the US duration was increased from 1500ms to 6000ms.

The asymptotic minimum level attained by INC when driven by a sustained unity amplitude US is determined by $\frac{RSTM_{dep}}{RSTM_{acc}+RSTM_{dep}}$. In order to produce the substantial reduction in INC described above, this asymptotic minimum needs to be very small (much less than 1). This means that $RSTM_{dep} \ll RSTM_{acc}$. $RSTM_{acc}$ is primarily responsible for determining the rate of decrease of INC. $RSTM_{dep}$ is then set to provide the appropriate asymptotic level.

**US Intensity Effects**

Increasing US intensity has the unsurprising effect of increasing both the rate and the strength of conditioning in many classical conditioning experiments studying different types of response systems and species (Mackintosh, 1974, pp. 70–71). More specifically, empirical results obtained using the NMR preparation are also entirely consistent with these US intensity effects (Gormezano, Keboe, and Marshall, 1983).

It is apparent from Figure 6–2 that the amplitude of the INC feedback signal will be proportional to the amplitude of the US input. Since INC determines the maximum compound strength of association able to be supported by the US, the desired qualitative relationship between CR amplitude and US intensity will be supported. While this relationship is usually more logarithmic than linear (e.g., Smith, 1968), it is assumed that this nonlinearity results from the operation of sensory transducers and sensory preprocessing, and so is not included within ACE.
**SUMMARY**

The common output stage of ACE supports all interaction between the multiple CS input channels, and is responsible for producing the compound "CS expects US" output of ACE. It also accepts the US input, and after some processing, distributes the reinforcing effect of the US to each of the CS input channels.

Although ACE is a real-time intra-trial neuronal model of classical conditioning, the output stage combines the expectation of reinforcement acquired by each CS input channel, and the actual reinforcement subsequently delivered, in a manner consistent with an improved version of the Rescorla-Wagner model. As a consequence, the considerable behavioral capabilities of the Rescorla-Wagner model are also exhibited by ACE, in addition to the behavior produced by the new NMMM and CSTC systems developed in previous Chapters.

The output stage also includes a specially developed Reinforcement Short Term Memory (WSTM). Computer simulation results are presented which indicate how RSTM is utilised to produce a quantitative relationship between US duration, and its reinforcing effect, that is consistent with empirical results from animal experiments using the NMR preparation. The desired qualitative relationship between US intensity, and its reinforcing effect, is also directly implemented.
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OVERVIEW

This chapter develops the remaining mechanisms required to complete each CS input channel of ACE. Since all other aspects of ACE have been developed in the previous chapters, this represents the final stage in the development of ACE. The emphasis here is upon the qualitative development of the mechanisms which bind together the separately developed subsystems of ACE so that they may functionally complement one another. Most of these mechanisms determine how the associative STM in each CS input channel is directly affected by experience, and so are collectively referred to as the STM learning rules. Note however, that because of the highly interactive internal nature of ACE, these learning rules by no means fully encapsulate the effect that experience has upon the strength of associative STM.
INTRODUCTION

The schematic diagram of ACE provided below (Figure 7–1) shows, with the exception of the internal detail of the CSTC, all mechanistic aspects of ACE. In an attempt to clearly explain how ACE functions, an effort was made to modularise its internal mechanisms so that they could be developed and discussed in relative isolation. Three main subsystems were identified as the Neural Multiprocess Memory Model (NMMM), the Conditioned Stimulus Trace Circuit (CSTC), and the output stage, which were discussed separately in Chapters 4, 5, and 6 respectively. All of the mechanisms previously developed in these chapters are shown shaded in Figure 7–1 in order to highlight the remaining (unshaded) mechanisms which complete ACE. Each of these remaining mechanisms is addressed in this chapter, in a mechanistically logical sequence.

Starting at the upper left portion of the unshaded region of Figure 7–1, and traversing around in an essentially anticlockwise direction, these new mechanisms may be outlined as follows. First, a negative feedback loop is used to make Memory Gating Short Term Memory (MGM) value increase towards that of the newly introduced cumulative quantity referred to as Reinforcement Gating Short Term Memory (RGM). An increase in MGM is required so that the results of recent experience temporarily retained in STM may be allowed to transfer via MTM to LTM so that consolidation may occur.

RGM attempts to follow a Compressed Conditioned Stimulus Trace (CCST) of prior CS input activation by another simple negative feedback loop, but has a rate of increase limited to reduce the effectiveness of reinforcement when the Inter-Stimulus Interval (ISI) between CS and US onset is very short. RGM modulates the reinforcing effect of subsequent US presentation to produce the Reinforcing signal that increases SIM, which is labelled "R" in Figure 7–1.

Extinction of conditioned excitors is supported by an Extinction signal, labelled "E" in the Figure 7–1. This extinction signal acts to decrease STM in response to the compound expectation of reinforcement, which is determined by the output stage.
FIGURE 7-1. Schematic diagram of ACE, showing a complete typical CS input channel, and the single output stage. Those mechanisms shown shaded have already been developed and documented in previous chapters, and include the NMMS, the CSTC, and the output stage. Each unshaded mechanism is progressively developed in this chapter.

A special reinstatement signal, labelled "r" in Figure 7-1, is also included to restore STM level back up towards its previous long term value when, following extinction, unpaired US presentations are experienced. Finally, a special mechanism located in the upper right portion of the unshaded region in Figure 7-1 utilises the uncompressed CST signal and the US driven reinforcing feedback signal INC to adaptively regulate the maximum allowable Associability Long Term Memory (ALTM) level.
Acquisition of a classical CR is usually achieved over many successive trials in which a CS and then a US is presented, producing a gradual increase in the strength and/or probability of the CR. If after acquisition, the CS is presented alone on each trial, without any subsequent presentation of the US, then the strength of the CR will gradually decline. This procedure, and the result it produces, are both referred to as "extinction". In other words, an extinction procedure leads to the extinction of the CR.

The signal responsible for producing reductions in synaptic STM, and hence extinction of the CR, is identified with an "E" in Figure 7-1. A positive STM value, previously established by acquisition training, enables a CR to be generated when the CS input is activated, by producing nonzero EXC and OUT signals, leading to generation of the CR. The OUT signal is also relabelled as DEC and fed back to the CS input channel, where after being modulated by the current level of associability for that CS input channel (which is retained in ALTM), it acts to decrease STM value. Modulation of DEC by the current ALTM level enables previous experience to determine the most appropriate rate of extinction, via the adaptive associability mechanism introduced in Chapter 4. This sequence of events ensures that the strength of a CR diminishes towards zero with each nonreinforced CS presentation, thereby supporting basic extinction behavior.

Note that this extinction mechanism is activated whenever a nonzero OUT signal is generated by activation of CS inputs, in whatever context, be it extinction or acquisition training. In other words, this extinction mechanism does not identify when extinction training is being experienced, and then come into action. Instead, it operates whenever positive compound expectation of reinforcement is conveyed by DEC to the CS input channels, and relies upon the operation of a separate reinforcement mechanism (discussed below) to counter this induced decrease in STM value when effective reinforcement is experienced. This basic type of nondiscriminatory operation of an extinction mechanism is not new, being conceptually similar to the reactive inhibition
theory usually associated with Hull's name (Mull, 1943, Chapter 16), and the operation of the Rescorla–Wagner model (Rescorla and Wagner, 1972).

However, the extinction mechanism depicted in Figure 7-1 differs radically from all others in the following very basic way - the extinguishing effect of a positive compound expectation of the US is not gated by prior CS input activity. This means that presentation of a CS previously trained to expect a US will reduce the STM level of not only its own CS input channel, but will reduce the STM levels in all of the CS input channels within ACE. The extent to which the CRs produced by each CS input channel are reduced will depend upon the initial STM level associated with the presented CS, the extent to which it is extinguished, the initial STM levels of each CS input channel relative to that of the presented CS, and their ALTM levels.

While such behavior seems to have received very little attention in recent times, Pavlov (1927, pp. 54-56) noted behavior consistent with this, referring to extinction of the presented CS as "primary extinction", and that of other CSs generating a similar (or "homogeneous") CR as "secondary extinction". Pavlov found that:

(i) The extent of secondary extinction increases with the extent of primary extinction.

(ii) The weaker the CR generated by the primarily extinguished CS compared to that of the secondarily extinguished CSs, the less complete is the secondary extinction. Conversely, "if the stronger CR is subjected to primary experimental extinction the weaker conditioned reflex undergoes complete secondary extinction" (Pavlov, 1927, pp. 55).

(iii) The same relationship between extent of secondary extinction and relative CR strength described in (ii) also applies to components of compound CSs, when only a component CS is subject to primary extinction, and the remainder undergo secondary extinction.

The above empirically observed relationship between primary and secondary extinction is in accord with that expected from the extinction mechanism implemented within ACE, which it should be noted, is entirely different to that proposed by Pavlov (1927, p. 60). However, an effort needs to be made to
explain how secondary extinction can be considered as appropriate behavior. On the surface, it hardly seems appropriate to extinguish all of the CS-US associations leading to the production of a single CR, because one of those CSs which used to predict reinforcement no longer does. In fact, when considered in isolation, this behavior would appear to result in the inappropriate loss of many CS-US associations which would most probably remain useful, and which were acquired after a great deal of experience. However, a case will now be made that, when viewed in a specific wider context, secondary extinction behavior may no longer appear inappropriate, and in fact that it may considerably enhance behavior.

Pavlov (1927, p. 58) somewhat boldly stated that "all those conditioned reflexes which have been fully established invariably and spontaneously return sooner or later to their full strength" following experimental extinction. Although it is unclear, Pavlov seems to be referring to the spontaneous recovery of primarily extinguished CRs, though he may also be including secondarily extinguished CRs. More recent results indicate that such complete spontaneous recovery from primary extinction seems to be the exception, with something like 50% being more typical (Kimble, 1961, p. 284). Empirical results specifically addressing the extent of spontaneous recovery from secondary extinction are apparently unavailable.

The partial spontaneous recovery of a primarily extinguished CS is already supported by the NMMM (Chapter 4). The suggestion here, which is claimed to be new and unique, is that unlike primary extinction, secondary extinction routinely undergoes complete spontaneous recovery. This eliminates the inappropriate long term secondary extinction of CS-US associations that are not actively extinguished. Complete spontaneous recovery of secondarily extinguished CSs occurs because changes in STM are only able to be transferred to MTM and LTM when MGM value is increased by CS presentation, and STM value spontaneously changes towards the value of LTM (Figure 7-1). Although MGM was introduced to gate changes in MM and LM primarily in preparation for the use of MTM as a suitable source to drive changes in adaptive associability, MGM also has the effect of isolating STM from both MTM and ETM.
Potential behavioral advantages of secondary extinction emerge because STM is now available for use as a temporally sensitive register of ongoing US availability. When the immediate effect of the unexpected omission of an otherwise expected US presentation is not specific to a particular CS, then all of the previously reinforced CS components presented in this temporal context will assist one another to promptly extinguish. This capability takes on a whole new dimension if a new CS (not previously reinforced) is presented only in such a temporal context of unexpected US omission. The STM level of its corresponding CS input channel will not only be driven to attain negative values - its presentation will also ensure that negative LTM values are attained. In other words, the development of conditioned inhibitors is now also supported by ACE in successive discrimination procedures. This represents a substantial extension to the simultaneous discrimination capability that most models and theories of associative conditioning are content to achieve.

REINSTATEMENT

When Pavlov (1927, p. 59) noted that the response to an extinguished CS could be largely restored by simply presenting the original US on its own, he described an effect now referred to as reinstatement. Later experiments confirmed the effect (Konorski, 1948, p. 185; Rescorla, 1979), and showed that the US is similarly effective whether signalled by another CS or unsignalled, and that its reinstatement effect remains substantial 24 hours after US presentation (Rescorla & Heth, 1975). Rescorla and Heth (1975) also argued that reinstatement is not specific to CS-US associations, but instead appears to act upon a US representation. This view of reinstatement received further support from Rescorla and Cunningham (1977), who showed that extinction of a second CS is apparently capable of "erasing" the prior reinstatement effect of the US presentation upon the previously extinguished CS, when both CSs are associated with the same US.

A reinstatement effect with the above characteristics can be achieved by enhancing the NMMM with a simple mechanism that generates a reinstatement signal, labelled "r" in Figure 7-1. This reinstatement signal is formed by the
product of the INC feedback signal from the output stage, which is activated primarily by onset of a US presentation, and a rectified version of the amount by which LIM value exceeds STM value. This means that if, as discussed above, recent extinction of a CS has depressed STM value below that of LIM, then an unpaired US presentation will act to rapidly increase SIM value towards that of LIM. Thus, an unpaired US presentation will reinstate previously masked excitatory strength which is still retained in the LTM of each CS input channel.

The extent to which such reinstatement is complete will depend upon the degree to which LTM value has been reduced by the extinguished STM values before the US is presented. This in turn depends upon the elapsed time since the beginning of extinction training, and the extent of SIM extinction. This is consistent with Pavlov's observation that "If the extinction has not been carried very far, a single application of the unconditioned stimulus is often sufficient to restore the reflex to full strength; but if the extinction has been made profound, repeated reinforcements are necessary" (Pavlov, 1927, p. 59).

Even though the associative memory in each CS input channel which mediates this reinstatement effect is specific to both a CS and a US, the resulting reinstatement effect is behaviorally nonassociative. This is because neither the decreases in STM resulting from extinction produced by CS alone presentations, nor the restorative increases in SIM resulting from US alone presentation, require contiguous CS and US presentation.

There are, however, good reasons for utilising the associative NMMM in such a way as to produce an apparently nonassociative reinstatement effect. To begin with, the mechanism mediating nonassociative reductions in STM, which has already been implemented to support extinction, provides at least half of the mechanism required to support reinstatement behavior. The combined short and long term retention characteristics of the NMMM also become an integral part of reinstatement behavior, providing a ready explanation for the dependence upon prior extinction, and the longevity of the reinstatement effect (Rescorla and Heth, 1975). The apparent interaction between reinstatement and spontaneous recovery (Rescorla, 1979), and the observation
that extinction after reinstatement makes subsequent retraining of the CS more difficult (Rescorla and Cunningham, 1977), further implicates the NMMM because of its production of spontaneous recovery behavior, and its pivotal role in the mediation of learning (Chapter 4).

The above reinstatement mechanism also plays an important role when a Partial Reinforcement (PR) schedule is experienced. The effect of Nonreinforced (N) trials to reduce STM level may be largely negated when subsequent Reinforced (R) trials occur, because STM is rapidly elevated back up towards the level of LTM by the US presentation in each R trial. This can substantially reduce the overall impact of the N trials upon the asymptotic strength of association able to be achieved in acquisition, resulting in a higher SIM asymptote than would be expected from the scheduled percentage of R trials. This specific type of behavior is entirely consistent with empirical results (e.g., Gibbs, Latham, and Gormezano, 1978).

REINFORCEMENT

In its most general sense, reinforcement refers to the experimental operation of arranging outcomes contingent upon events to increase the strength and/or probability of a type of behavior. In the context of classical conditioning, the arranged outcome is usually the presentation of a US that is made contingent upon the prior presentation of a CS, and which has the effect of increasing the probability that a CR will be generated. More specifically, reinforcement leads to acquisition of an associative relationship, which in the case of ACE is between a CS input and the US input, that is subsequently available to generate the CR when the CS is presented. Expressed yet another way, reinforcement results in ACE acquiring a predictive relationship between a CS input and the US input.

Acquisition of this CS-US association requires some type of associative mechanism that is capable of selectively producing a persistent increase in the STM level of those CS input channels that were previously activated by the CS, when the US is subsequently presented. The associative effect of acquisition due to reinforcement is mediated by the Reinforcement signal,
labelled "R" in Figure 7-1. The enduring effect of the increase in STM produced by this R signal is achieved because prior CS input activation also enables transfer of changes in STM to MIM and then LTM, via the gating effect of MGM (Chapter 4). The R signal produces increases in STM only when both of the following conditions are met:

(i) The corresponding CS input has been activated prior to US presentation.
(ii) The US is not already fully predicted by the CS input channels.

The mechanism supporting condition (i) utilises a processed version of the CS Trace (CST) signal generated by the CSTC (Chapter 5) to enable increases in STM when the CS is reinforced by subsequent US presentation. It might be possible, depending upon the specific application, to directly use the CST to gate the reinforcing effect of paired CS-US presentations upon STM. However, since the reinforcement mechanism also determines precisely how acquisition is affected by ISI, and the use of delayed or trace conditioning procedures, it was considered prudent to provide the facility to separately tailor the effects of both of these operational variables upon acquisition. The mechanism provided for this purpose is detailed below.

Condition (ii) above is supported by the implementation of a modified intratrial version of the Rescorla-Wagner model (Rescorla and Wagner, 1972). The output stage determines the compound expectation of reinforcement, which via the "extinction" mechanism discussed above, produces decreases in STM. The output stage also determines the asymptotic strength of association able to be supported by the US, which via this reinforcement mechanism, selectively produces increases in STM. STM therefore becomes the site at which compound expectation of reinforcement is compared with actual reinforcement delivered, in order to determine how the individual CS-US associations maintained in the SIM and LTM of each CS input channel should change. When expectation matches the actual reinforcement (or nonreinforcement) subsequently experienced, the effect of the extinction and reinforcement mechanisms upon STM is equal but opposite, resulting in no net change to STM value.

Note that the effect of this reinforcement mechanism upon SIM is also gated by the current ALTM level, Since ALTM also gates the effect of the extinction
mechanism upon STM, ALTM is able to modulate the state of learning without affecting the asymptotic levels resulting from acquisition or extinction training.

**DELAYED AND TRACE CONDITIONING**

Delayed and trace conditioning procedures define the two main types of **temporal** relationship between CS and US presentation in classical conditioning. Figure 7-2 illustrates how in delayed conditioning, the CS is sustained until the US is presented. Normally both the CS and the US terminate together, though in some delayed conditioning procedures the CS may terminate at US onset, or alternatively some time after US offset. In contrast, trace conditioning defines procedures in which the CS terminates before US onset, leaving a distinct interval of time between CS and US presentation. This interval is sometimes referred to as the "trace interval".

**FIGURE 7-2. Temporal relationships between a CS and a US in delayed conditioning and trace conditioning procedures.**
In the case of both delayed and trace conditioning, the ISI refers to the interval between CS onset and US onset. Trace conditioning is nearly always less effective than delayed conditioning, and becomes increasingly less effective as the trace interval is increased (Mackintosh, 1983, pp. 86–87).

The CSTC developed in Chapter 5 was specifically designed to produce a CST output signal with an overall amplitude that decreases as the CS terminates further in advance of the time at which the CST peaks. Although the specific issue then was how performance is modulated by CS duration, when this same CST signal is used in a micromolar (differential correlated reinforcement) mode, the rate and asymptote of acquisition are similarly affected by CS duration. This automatically leads to the common empirically observed result that delayed conditioning is more effective than trace conditioning.

However, the extent of the difference in acquisition between delayed and trace conditioning may not be as dramatic as the extent of the immediate performance differential produced by different CS durations. Furthermore, since in the case of ACE a derivative of the CST is used to modulate both increases in SIM due to reinforcement, and increases in MGM in order to gate STM changes through to MTM and LTM, and since both jointly determine the rate of acquisition, some additional processing is also required to prevent the acquisition rate from being excessively modulated by CS duration. A function is therefore used to compress the range of possible CST signals before it is used to modulate both of the above mechanisms. The relationship between the Compressed CST (CCST) signal (Figure 7-1) and the CST signal is defined as follows:

\[
CCST[T, N] = F(CST[T, N])
\]

\[
F(x) = x/(x + CCSTcon)
\]

Where:
- \( CCST[T, N] \) = Compressed CS Trace of channel N at time state T.
- \( CST[T, N] \) = CS Trace for CS input channel N at time state T.
- F(x) = Compression function.
- \( CCSTcon \) = Compression function constant, with value of 0.2.
It is apparent that the compression function $F(x)$ defined by Equation [7-2] equals 0.0 when $x \approx 0.0$, and approaches unity as $x$ approaches infinity. When $x = \text{CCSTcon} \approx 0.2$, then $F(x) \approx 0.5$. The most important aspect of $F(x)$ is that it is a monotonically increasing function of $x$, and so maintains the relative relationship between the amplitudes of the spectrum of CST signals hypothetically generated by many CS input channels. This ensures that the CST signal which most precisely coincides with US presentation still produces the most rapid rate of SIM acquisition, attains the greatest single share of associative strength, and tends to dominate the overall CR topography—which will therefore tend to peak shortly after US onset.

A final cumulative quantity, hereafter referred to as Reinforcement Gating Short Term Memory (RGM), is introduced to provide a CSTC-independent means of tailoring how the extent of acquisition varies with ISI. Figure 7-1 indicates that RGM attempts to follow the CCST signal via the operation of a simple negative feedback loop. However, its rate of increase is intentionally limited so that it is unable to track those short-peakings signals which rapidly rise and then rapidly decay. Consequently, RGM further reduces the rate and extent of acquisition at short ISIs, but has progressively less effect as the ISI increases. The rate of RGM depletion is set to unity, enabling RGM to accurately track the falling edge of each CCST signal. This minimises disruption to the differential correlated reinforcement mechanism, which supports acquisition of that CR topography which coincides approximately with US onset, at all but the shortest ISIs.

RGM is also used to increase MGM, because it is suitably compressed in terms of its variation in amplitude, but is still appropriately responsive to CS duration and amplitude. However, the choice as to which signal drives increases in MGM is one of the less critical aspects of ACE's design.
ADAPTIVELY REGULATED MAXIMUM ALTM LEVELS

Having established above how STM contents are affected by experience, a consideration of the different asymptotic STM limits produced by delayed and trace conditioning procedures leads to the apparent necessity for some means to appropriately regulate maximum ALTM levels. Although the mechanism responsible for this is more directly associated with the adaptive associability mechanism (Chapter 4) than the STM learning rules described above, it is included here because of its dependence upon: the CST output signal from the CSTC (Chapter 5), the potentially reinforcing effect of US presentation that is mediated by the INC feedback signal (Chapter 6), and the interaction between the effect experience has upon STM and the maximum appropriate ALTM level (discussed above).

A given US of fixed amplitude and duration, presented at a fixed ISI, will support a fixed maximum compound expectation of reinforcement within ACE. When the duration and amplitude of the CS is also fixed, the fixed expectation of reinforcement translates into a fixed asymptotic STM limit. However, if the CS duration is set to less than the ISI to produce a trace conditioning procedure, then the asymptotic STM limit will be different to that when the CS is extended to produce a delayed conditioning procedure.

More specifically, a trace CS produces a small CST, and leads to a large SIM asymptotic limit in order to produce the total expectation of reinforcement supported by the US presentation. Conversely, a delayed CS produces a large CST, and leads to a small STM asymptotic limit. If ALTM can attain only a single fixed maximum level, then at this maximum ALTM level the absolute change in expectation of reinforcement due to each reinforced and nonreinforced trial for trace and delayed conditioning will be identical, but the relative change will be dramatically different. This is because the extinction and reinforcement signals, labelled "E" and "R" respectively in Figure 7-1, will have a much greater impact upon the smaller STM levels achieved in delayed conditioning than upon the larger SIM levels resulting from trace conditioning. The specific settings used in the CSTC may, for
example, lead to differences in CST amplitude, and consequently STM asymptotic limit, of approximately one order of magnitude (a factor of 10).

While it is normal for delayed conditioning to proceed more rapidly than trace conditioning, the difference in rate tends to be much less than an order of magnitude. Furthermore, most of this difference may be accounted for by assuming that naive subjects usually begin with similarly low initial ALTM levels. This automatically produces a more rapid initial learning rate for subjects on a delayed conditioning procedure. Furthermore, this effect is compounded by a more rapid and complete increase in ALTM level.

A simple mechanism which adaptively regulates the maximum ALTM level to maintain a similar maximum learning rate for both trace and delayed conditioning is illustrated in Figure 7-1. The Enable Associability Change (EAC) signal, as its name suggests, enables changes in ALTM to occur when reinforcement is experienced. EAC is formed by the product of the US feedback signal INC, the current ALTM level, and the uncompressed CST signal.

Direct use of the CST signal means that EAC is affected by trace and delayed CS inputs in the same way as the "CS expects US" output. A special new negative feedback loop, illustrated above EAC in Figure 7-1, reduces the extent of the increase in ALTM as EAC becomes increasingly larger, when MTM value is positive. When EAC = 1, no additional increase in ALTM is possible. If EAC exceeds unity amplitude, a negative increase (i.e., a decrease) in ALTM actually results, because the signal which normally acts to increase AETM now attains a negative value. That this condition is permitted is indicated in Figure 7-1 by the use of a "+/-" label on the signal as it impacts upon ALTM. This additional capacity to decrease ALTM level back down to a new lower maximum asymptotic level permits the maintenance of appropriate maximum ALTM levels, despite the possibility of arbitrary transitions between trace and delayed conditioning procedures. Furthermore, the fact that this can occur when only positive MTM values are present, which for example is the case during acquisition, ensures that appropriate adjustment of the maximum ALTM level will occur even during conditions which normally increase ALTM level.
A mechanism which sets a maximum, albeit adaptively regulated, ALTM level can also ensure that unstable operation during acquisition does not occur. This undesirable condition may otherwise arise as increases in AETM enable larger increases in STM, which produce larger MTM values, and which in turn enable progressively larger increases in ALTM. This represents one of the few instances in which a special effort needs to be made to avoid unstable operation within ACE. The need arises because of the positive feedback relationship that exists between increases in ALTM, and increases in STM. Stable operation within the other internal mechanisms of ACE is assisted by the extensive, indeed almost exclusive, use of simple negative feedback loops.

SUMMARY

A qualitative description of the mechanisms that constitute ACE has now been completed by focussing upon those mechanisms which bind together all of the subsystems previously developed in Chapters 4, 5, and 6. These remaining mechanisms are collectively referred here as the STM learning rules because they describe primarily how experience induces relatively rapid changes in associative STM. They thus bear a superficial resemblance to the "learning rules" of ANN terminology. However, because they are embedded within so many other mechanisms which contribute to their proper and complete operation, the mechanisms described in this chapter really only form part of what might be regarded as the set of learning rules for ACE.

Most of the mechanisms were identified in terms of their primary functional role. A new extinction mechanism was introduced which, because of the selective consolidation behavior already supported by the NMMM, was able to be mechanistically exceptionally simple. This extinction mechanism supports not only primary extinction of an excitatory CS when presented alone, but also secondary extinction of all other CS-US associations to the same US. However, while primary extinction is subject to partial spontaneous recovery, the new type of secondary extinction supported here undergoes full spontaneous recovery, thus preventing the inappropriate long term loss of such a widespread set of associations. However, in the short term, such secondary
extinction considerably enhances adaptive behavior by facilitating rapid extinction of many CSs when presented in a context of unexpected omission of the US, and by supporting a successive discrimination capability.

A new reinstatement mechanism was also introduced to directly support the nonassociative restoration of conditioned excitors when, shortly following extinction, the US is presented alone. Like the secondary extinction phenomena, reinstatement is an empirically observed phenomenon of considerable, though perhaps not readily apparent, behavioral utility. Aside from helping to prevent the possibly inappropriate loss of excitation during temporary periods of unexpected US omission, the reinstatement mechanism also helps maintain a stronger CS-US association during partial reinforcement schedules.

A relatively standard micromolar reinforcement mechanism was then introduced, with some enhancements made to reduce the difference between the reinforcing effect of trace and delayed conditioning procedures, and to facilitate isolated tailoring of the reinforcing effect of US presentations at short ISIs. A micromolar, or differential correlated reinforcement, mechanism was used to directly support acquisition of appropriately timed and generalised CRs.

Finally, a new mechanism to adaptively regulate maximum ALTM levels was developed to ensure stable and appropriate operation of the adaptive associability mechanism, even when arbitrary transitions between trace and delayed conditioning procedures are experienced.
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OVERVIEW

After a brief introductory summary of the overall organisation and structure of ACE, computes simulation results for its complete integrated operation are presented and compared with corresponding empirical results from animal experiments. In this chapter an emphasis is placed upon those aspects of behavior not already supported by other existing models or theories, or by isolated subsystems within ACE for which results have already been provided in previous chapters. The integrated behavior discussed herein requires the complete operation of ACE, and highlights the highly integrated nature of its internal operation.
INTRODUCTION

ACE may be classified as a neuronal model of classical conditioning which produces behavior attributable to a single biological neuron, or small group of neurons. It consists of multiple input channels, and a common output stage, as depicted in Figure 8-1. The CS input channels contain all CS-specific (or "synaptic") mechanisms which might be associated with synaptic and post-synaptic membrane function, and interact with only the output stage, via several specialised signals. However, the output stage mediates a great deal of indirect interchannel interaction via its connections with the input channels. While ACE would in practice have many CS input channels, only 4 are required to test and demonstrate most of its behavioral capabilities.

![Block diagram of ACE](image)

**FIGURE 8-1.** Block diagram of ACE. Multiple CS inputs each drive a dedicated CS input channel, which communicates with the common output stage via several signals. The output stage receives direct input from the reinforcing US input, and is also responsible for generating the "CS expects US" output of ACE. All inputs, and the output, convey non-negative signals only.

Multiple CS inputs converge upon ACE where they may become associated with subsequent changes in US input activity that are not already adequately predicted by previously activated CS inputs. The type of behavior supported by the association between the CS inputs and the US input is best characterised as consummatory classical conditioning. Each individual CS input
corresponds to a stimulus feature which may potentially form part, or all, of a learned CS definition, as a result of conditioning. The US input is highly specific to an individual US event, which is considered to produce a similarly specific UR. Both the US and the UR are assumed to be predefined. ACE’s output does not correspond directly to either the UR or the CR, but instead is best thought of as indicating the strength and timing of US presentation which is expected by previously presented CSs. Consequently, unlike the CS inputs, the US input does not activate ACE’s output, but instead acts merely to reinforce conditioning of the CS inputs. Note, however, that while ACE’s output does not explicitly define the type of CR, it does determine the amplitude, probability, and timing of the CR. It is also available as a source of conditioned reinforcement for both higher-order and instrumental conditioning.

The complete mechanistic detail of ACE is illustrated below in Figure 8–2 (identical to Figure 1–2) as a schematic diagram, which shows a single representative CS input channel and the common output stage. The bulk of ACE’s mechanisms are distributed among its multiple input channels, each of which incorporates the newly developed Neural Multiprocess Memory Model (NMMM) and CS Trace Circuit (CSTC), previously described in chapters 4 and 5 respectively. The NMMM incorporates synaptic forms of Short Term Memory (STM), Medium Term Memory (MTM), Long Term Memory (LTM), and Associability Long Term Memory (ALTM), which interact to provide experience-dependent modulation of both rapid short term adaptation and long term integration of experience. The NMMM also incorporates Memory Gating Short Term Memory (MGM) to selectively enable the effect of experience upon STM to be transferred through MIM to LIM following CS input activation, and Reinforcement Gating Short Term Memory (RGM) to selectively enable subsequent US presentations to have a reinforcing effect upon STM.

The CSTC essentially acts as a pulse shaping circuit, converting the CS input signal into a smoothly changing and sustained CS Trace (CST) output signal. The CST signal has a shape characteristic of CR strength and probability, and an overall amplitude resembling the extent of acquisition as a function of ISI. The shape of the CST output signal, and the manner in which it responds to variations in CS input amplitude and duration, are designed to match that of
FIGURE 8-2. Complete schematic diagram of the Associative Conditioning Element (ACE). A single representative CS input channel is shown, comprising the Neural Multiprocess Memory Model (NMMM) and the CS Trace Circuit (CSTC). Also shown is the common output stage upon which multiple CS input channels converge.
the Nictitating Membrane Response (NMR) of the rabbit.

The output stage combines the excitatory (EXC) and inhibitory (INH) output signals from each of the CS input channels to produce the "CS expects US" output signal (OUT), which also forms the decremental feedback signal (DEC). The output stage also receives direct activation from the US input, which after processing using Reinforcement Short Term Memory (RSTM) to emphasise US onset, is distributed among the CS input channels as the incremental feedback signal (INC). Each CS input channel uses INC and DEC to determine how the immediate associative strength in STM should be altered, according to what might be characterised as a highly modified and enhanced intratrial version of the Rescorla–Wagner model (1972).

GENERATION OF RESULTS

The mechanistic detail of ACE depicted in Figure 8–2 is fully specified by the set of difference equations and associated constants provided in Appendix 1. All computer simulation results presented in this chapter were obtained using only this single set of equations, with all associated constants remaining unchanged. Furthermore, the equations and constants developed in those preceding chapters dealing with the subsystems of ACE were directly incorporated into the set of equations provided in Appendix 1. Consequently, all of the computer simulation results presented in Chapters 4, 5, and 6 remain valid for the complete ACE, with the exception of those results pertaining to incomplete portions of the NMMM presented in the first half of Chapter 4.

All of the results presented here were generated using CS and US inputs of unity amplitude and 50ms duration, primarily in order to facilitate comparison between results. While the absolute amplitude of these neuronal inputs cannot readily be equated with particular empirical stimulus intensities, the 50ms duration of the inputs directly equates with that frequently employed in many empirical procedures using the NMR preparation (e.g., Smith, 1968; Smith, Coleman, and Gormezano, 1969; Gormezano, 1984).
Since the ISI for all results generated here is greater than the 50ms CS duration employed, all conditioning is of the trace type, rather than the other main alternative, delayed conditioning. Trace conditioning is commonly employed in experiments using the NMR preparation, and it highlights the critical role played by the CSTC within ACE, since it is actually the CST which is associated directly with the US, and not the CS input.

The ITI used throughout this chapter was fixed at 10s, which is substantially less than the nominal 60s ITI usually employed in NMR preparations. Using a short ITI reduces the time required to generate results for each session of simulated trials. All system equations are updated at what corresponds to every 10ms (100 times per second) to provide sufficient temporal resolution, and each training session consists of 108 or more 10s long trials. The modest hardware used to generate the computer results (outlined below) was approximately an order of magnitude slower than that which would be required to achieve real-time operation, all other things being equal. Consequently, a 200 trial acquisition session with an ITI of 10s required several hours of computation. More sophisticated hardware was available, and would have been utilised if the time consumed generating results with the modest hardware had significantly retarded the development process, which was dominated by theoretical design and literature research.

The 10s ITI is well beyond the maximum ISI capable of producing substantial conditioning, and is also long enough for the short term quantities within ACE to approach their steady state values. The important exceptions to this are associative STM, MTM, LTM, and MGM which require several minutes to approach equilibrium values. Using a relatively short ITI of 10s has the additional advantage of emphasising the medium term effects produced by these cumulative quantities, which are a major feature of ACE's behavior.

Furthermore, the apparent lack of relevant NMR retention data led to the decision to use data from honeybees to calibrate the rates of change of these associative memories in Chapter 4. Hence, adherence to a, 60s ITI would not necessarily produce a close correspondence between simulated and empirical NMR results.
Within the procedures for animal experiments it is normal to introduce a randomly sequenced variation in \( \text{ITI} \) about its mean value, and to randomly sequence Reinforced (R) and Nonreinforced (N) trials in Partial Reinforcement (PR) schedules. However, since ACE is being tested in isolation, and it alone has no capability to acquire or apply any expectation of the timing or outcome between the current and the next trial when the \( \text{ITI} \) is much greater than several seconds, it was not considered necessary to introduce either type of random variation in procedure for the production of the computer simulation results.

Those results presented herein that were conducted over many successive training trials are sampled 1 time state (10ms) before CS onset at the beginning of each trial, in order to more clearly display overall trends. In some cases a significant variation in the amplitude of a cumulative quantity occurs within the early stages of each trial, even when the overall trend is flat and smooth. However, the sampled values still provide a valid indication of the cumulative quantity values as they will affect performance in the next trial.

A 10MHz 80286 AT personal computer with 2Mbyte RAM and a 45Mbyte hard disk drive was employed to generate all computer simulation results. A special development environment was written in Microsoft C language to facilitate sequencing of the 5 inputs, the selective display and logging of the 56 system variables, and management of the 25 system constants. The system equations themselves were also implemented in C using floating point arithmetic, with floating point variables used for all system signals, variables, and constants.

ACQUISITION CURVE SHAPES

Psychologists interested in the construction of formal models of conditioning have often turned to an examination of the course of acquisition in the expectation that its shape will provide some clue as to its underlying mechanisms. Although different response systems and response measurement techniques produce marked variations in rate, and to a lesser extent course of
acquisition (Mackintosh, 1974, p. 10), researchers usually attempt to reduce most results to a single generalised acquisition curve shape. A sigmoidal curve, with an initial acceleration phase followed by deceleration to an asymptotic maximum level of performance, is usually selected as the single shape most characteristic of the course of acquisition in general.

However, many acquisition curves exhibit little or no initial acceleration phase. Furthermore, it is not uncommon for some individual subjects within the same experimental procedure to exhibit sigmoidal acquisition while other individuals do not. Figure 8–3 shows the averaged acquisition curves for 3 groups of human subjects in eye-blink experiments with similar general performance levels, referred to by Spence (1956, p. 60) as homogeneous groups.

![Graph showing averaged acquisition curves for 3 groups of homogeneous human subjects.](image)

**FIGURE 8-3.** Frequency curves of classical eyelid conditioning for 3 groups of homogeneous human subjects. The uppermost curve is that for subjects exhibiting high conditioning performance both early and late in training, the middle curve for subjects exhibiting average performance, and the lowest curve for subjects exhibiting poor performance. Reprinted from Spence (1956, p. 66), which was based on data from Spence and Taylor (1951).

Spence (1956, p. 64) states that "Examination of a fairly large number of such "homogeneous" frequency curves of eyelid conditioning obtained in our
laboratory has shown that when the performance level of the subjects at the beginning of conditioning is relatively low and/or the rate of conditioning is slow a curve with an initial period of positive acceleration is invariably obtained. On the other hand, if the performance level is relatively high at the start of conditioning and/or the rate of conditioning is high the curve is typically negatively accelerated throughout, especially if the grouping of trials is coarse.”

It would therefore seem that those formal models which postulate a simple negatively accelerated rate of acquisition (e.g., Rescorla and Wagner, 1972; Barto and Sutton, 1985), or those which postulate sigmoidal acquisition (e.g., Klopf, 1987), have failed to adequately address the reality that both types of acquisition may occur.

While Figure 8-3 indicates that different individuals may or may not exhibit an initial phase of accelerated acquisition, it does not reveal whether this difference in learning performance is somehow inherent in each individual, or whether it is dependent upon an individual's previous experience. Kremer (1971) found that while a nonpreexposed control group exhibited a negatively accelerated acquisition curve, a CS-alone preexposed (i.e., latent inhibition) group exhibited an initial phase of positive acceleration. Kremer's results are reproduced in Figure 8-4. These suggest that previous experience is capable of determining the nature of subsequent acquisition, and not just its extent or rate of change.

In Chapter 4, a new adaptive associability mechanism within the NMMM was described. This mechanism was intended primarily to support the Partial Reinforcement Effect (PRE), and the increased effectiveness with which subjects adjust to alternate sessions of fully reinforced and then fully nonreinforced massed trials. It will now be shown that this same adaptive associability mechanism is also primarily responsible for the appropriate production of either sigmoidal or negatively accelerated acquisition curves, depending upon the initial associability value. Furthermore, it is later described how latent inhibition and learned irrelevance may also be supported.
FIGURE 8-4. The effect of latent inhibition (CS only) and learned irrelevance (CS/UCS) upon the subsequent acquisition of conditioned suppression in rats. The suppression ratio is obtained by dividing the unsuppressed response rate by the sum of the unsuppressed and the suppressed response rates, producing a ratio of 0.5 when the CS has no suppressive effect, and 0.0 when the CS completely suppresses responding. Reprinted from Mackintosh (1974, p. 40), which in turn was derived from Kremer (1971).

Figure 8-5a illustrates the progressive change in STM during massed excitatory acquisition over 100 training trials, for 3 different initial ALTM values. The acquisition curve shape for ACE is strictly negatively accelerated, mildly sigmoidal, and strongly sigmoidal when the initial ALTM value is high, medium, and low, respectively. These 3 curves compare very favourably with those reproduced in Figure 8-3 from Spence (1956, p. 66).

Furthermore, since as will be discussed below the initial ALTM level is dependent upon previous experience in a manner consistent with latent inhibition and learned irrelevance procedures, the results depicted in Figure 8-5a are also consistent with the results obtained by Kremer (Figure 8-4) when allowance is made for the different dependent variables used in each case. Hence the new adaptive associability mechanism within ACE provides a unifying explanation for all of these empirical results.
FIGURE 8-5. Massed excitatory acquisition of 1 CS input channel over 100 training trials at an ISI of 250ms, when the initial ALTM level = 0.72 (heavy shading), 0.10 (medium shading), and 0.03 (light shading). (a) STM levels, all initially at 0.0 value. (b) Corresponding ALTM levels, starting at the 3 different initial values.
The means by which the different STM acquisition curve shapes illustrated in Figure 8-5a are generated may be explained as follows: Each CS presentation activates the CS input to the single CS input channel used here, producing a CS Trace (CST) signal which, with an ROC setting of 0.1, gradually increases before peaking approximately 300ms after CS onset and then gradually declining. Chapter 5 provides a complete explanation of CSTC operation. After some compression and rate-of-increase limiting by RGM, this trace signal enables the subsequent US presentation 250ms after CS onset to produce a significant increase in STM. The trace signal also increases MGM, which enables the gradual transfer of the induced increase in STM to LTM via MTM. Because of its slow rate of increase, MTM gradually attains larger positive values with each of the early training trials. These progressively larger MTM values are also used to increase ALTM towards its adaptively regulated maximum value, with each reinforcing US presentation.

If ALTM is already at its maximum attainable level at the beginning of acquisition (e.g., Figure 8-5b, heavy shading), then acquisition proceeds at a fast rate from the very first trial, but ALTM cannot be increased further as acquisition proceeds. Since ACE implements a modified intratrial version of the Rescorla–Wagner model (1972), and since ALTM level remains essentially fixed, a strictly negatively accelerated acquisition curve is produced as the difference between expected and delivered reinforcement diminishes. Inspection of Figure 8-5a reveals that the negatively accelerating acquisition curve is not quite the exponential shape produced by the Rescorla–Wagner model (1972). This deviation results from the influence that LTM has upon STM, which is also responsible for producing spontaneous regression and recovery behavior (Chapter 4). The relatively slow rates of change of both MTM and LTM mean that LTM level lags behind that of STM during most of the acquisition session, only catching up as STM comes close to approaching its asymptotic limit. The extent to which LTM influences STM is proportional to the difference between their levels. Consequently, early in acquisition STM is able to increase with little opposition from LTM, but as the difference between them grows, further STM rates of increase are significantly diminished. This produces the gentle shoulder in the negatively accelerating STM curve of Figure 8-5a early in acquisition.
If, in contrast, ALTM is initially low (e.g., Figure 8-5b, light shading), then there exists considerable scope for subsequent increases in ALTM, but massed acquisition of an excitatory CS will initially be very slow. As MTM value gradually increases during the early stages of acquisition, it is able to produce progressively larger increases in ALTM. The very frequent and effective delivery of reinforcement repeatedly gates the increasingly large positive MTM values through to increase ALTM. This enables the strictly negatively accelerated acquisition curve exhibited by the Rescorla–Wagner model (1972) to be supplemented by an initial slow learning phase – resulting in the classic sigmoidal shaped acquisition curve, as is commonly empirically obtained (e.g., Spence, 1956, Chapter 3). Note, however, that the ITI in acquisition needs to be short enough to produce an increase in ALTM of sufficient amplitude and speed to produce the initial phase of accelerating rate of acquisition. As the initial ALTM value is set at progressively larger values, the SIM acquisition curve gradually becomes less sigmoidal, exhibiting less and less initial positive acceleration.

When the associative strength of the CS attains the level supported by the reinforcement, further sustained SIM and LTM increase is not possible. MTM value then approaches zero, preventing further increases in ALTM. In other words, the extent to which the associability of a CS can be increased during its massed acquisition may be sufficiently small to prevent ALTM approaching its adaptively regulated maximum value, particularly when long ITIs are employed. The ALTM curves of Figure 8-5b have no such trouble attaining their full maximum value because of the relatively short 10s ITI employed.

If the initial associability of all CSs is set to a low level, this helps to ensure that other CSs known to correlate well with reinforcement have a strong head start in acquisition, and that any coincidental pairings of reinforcement with a naive CS lead only to minimal conditioning. Only when a firm correlation between CS and US has been established by previous experience is the acquisition of associative strength allowed to proceed more rapidly.
TEMPORAL DISCRIMINATION AND GENERALISATION

The CSTC within each CS input channel produces a CST output signal with a shape closely matching that of the mean topography of the rabbits' NMR (Chapter 5). The ROC preset input to the CSTC (Figure 8–2) determines when the CST peaks. Figure 8–6 illustrates several CST signals generated by CSTCs with the ROC settings indicated. Each CS is assumed to activate many CS input channels with different ROC settings, and thus to generate such a spectrum of CST signals. When the CS is followed shortly afterwards by presentation of the US, the US differentially reinforces each CS input channel according to their relative CST amplitudes at the time the US is presented. Those CS input channels with CST signals which correlate best with (primarily) US onset have their STM values increased most effectively. Since the temporal profile of ACE's "CS expects US" output signal, and consequently that of the CR, is determined by summing the product of each CS input channel's instantaneous CST and STM value, the differential correlated reinforcement (or "micromolar") mechanism is able to tailor the temporal profile of the CR to best coincide with expected US presentation.

FIGURE 8–6. A spectrum of CST output signals generated by the CSTC at ROC settings of 0.50, 0.20, 0.10, 0.05, 0.02, and 0.01, for responses peaking from left to right, respectively.
This is not merely a process of selecting the single most correlated CST signal, and rejecting all of the others. The temporal profile of the CR may be generalised to suit US presentation at variable ISIs by reinforcing, to varying degrees, a mixture of many CS input channels with differently timed CST signals. An extreme example of this is the mixed ISI procedure (e.g., Millenson, Kehoe, and Gormezano, 1977), in which the US is presented at one ISI on some trials, and at another ISI on the remaining trials. A more normal example is US presentation about a single mean ISI, with substantial random variation.

The ability of ACE to also support conditioned inhibition, discussed later, considerably enhances its temporal discrimination capability. The temporal profile of the CR can be trained to be considerably sharper than that of the single CST signal which best correlates with US presentation, by establishing inhibitory (negative) STM levels in those CS input channels with CST signals peaking at adjacent ISIs. The possibility also exists to support strongly asymmetrically (on a log-time scale) shaped CR temporal profiles.

Figure 8-7 shows how the STM levels in 4 simultaneously activated CS input channels change during massed excitatory acquisition. Parts (a), (b), (c), and (d) of Figure 8-7 illustrate the results obtained when ISIs of 130ms, 250ms, 500ms, and 1000ms are used, respectively. The graphed STM levels of each CS input channel are visually differentiated by the line type used to plot each curve. Table 8-1 relates each line type for each part of Figure 8-7 to a specific ROC setting, which in turn can be associated with a specific CST signal using Figure 8-6. It is apparent from Table 8-1 that 4 consecutive ROC settings are used for each training session at a particular ISI, but that different sets of ROC values are employed to ensure that at least one CST signal peaks before and after that CST signal which coincides best with US presentation. This reduces end-effects which otherwise provide those CS input channels with CST signals peaking at the edge of the spectrum with a distinct competitive advantage. In a complete system, more than 4 CS input channels would be employed for the spectrum associated with each CS, and those CST signals at the edge of the spectrum would be relatively small because of the normal operation of the CSTC (Chapter 5). Hence, the use of the ROC groups indicated in Table 8-1 help to generate results which better approximate those
that would be obtained from an ACE with a more complete spectrum of CS input channels which are activated by the CS.

**TABLE 8-1**

<table>
<thead>
<tr>
<th>ROC PRESET</th>
<th>(a)</th>
<th>(b)</th>
<th>(c)</th>
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<td>0.10</td>
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<td>0.05</td>
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<td>0.02</td>
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<td>0.01</td>
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Figure 8-7 indicates how, initially in training at all 4 ISIs, the STM levels in each of the 4 CS input channels increase at a rate dependent upon the amplitude of their corresponding CST signals when the US is presented. Because of the relatively coarse temporal spacing of the CST signals employed here (Figure 8-61, that CS input channel whose CST coincides best with US presentation experiences the fastest rate of STM increase by a large margin. As acquisition proceeds, and the total integrated response strength from all CS input channels becomes a substantial fraction of the maximum level supported by US presentation, a significant negative influence upon all STM levels develops, which is mediated by the DEC feedback signal. For those CS input channels with CST signals which peak too early or too late, this negative influence upon STM soon defeats and then exceeds the relatively small positive influence they experience from reinforcement. This makes their STM levels attain only a low maximum level and then decline. However, the STM level of the CS input channel with a CST that peaks coincident with US presentation continues to grow strongly, albeit at a reduced rate, and attain the bulk of the available associative strength supported by the US.
FIGURE 8-7. STM levels during massed excitatory acquisition over 200 training trials at 4 different ISIs, when 4 CS input channels with the ROC settings indicated in Table 8-1 are simultaneously activated by the same CS. (a) ISI = 130ms. (b) ISI = 250ms. Figures 8-7c and 8-7d are on the next page.
FIGURE 8-7. STM levels during massed excitatory acquisition over 200 training trials at 4 different ISIs, when 4 CS input channels with the ROC settings indicated in Table 8-1 are simultaneously activated by the same CS. (c) PSI = 500ms. (d) ISI = 1000ms. Figures 8-7a and 8-7b are on the previous page.
The ALTM levels of each CS input channel during the same massed excitatory
acquisition at each of the 4 ISIs are depicted in Figure 8-8. The same line
type code used in Figure 8-7 is employed for Figure 8-8, and is detailed in
Table 8-1. Figure 8-8 illustrates that the ability of massed excitatory
acquisition to increase ALTM level is just as selective as its ability to increase
SIM level. In other words, that CS input channel with an ROC setting which
makes its CST signal peak at US onset, experiences a rapid and extensive
increase in associability, while the others remain relatively unaffected. This
means that after 200 trials of massed excitatory acquisition training at a
single specific ISI, ACE not only learns when to expect delivery of the US,
and how intense this expectation should be, but it also learns to more rapidly
associate the CS with the US at this ISI.

If thorough extinction training were to be subsequently experienced, the
learned CS-US association stored in SIM and LTM would essentially be lost.
However, ACE would rapidly and preferentially reacquire the CS-US association
at the ISI originally experienced in acquisition. Such reacquisition would have
a negatively accelerating curve resembling that illustrated in Figure 8-5a
(heavy shading), and would be less impeded by the initial growth of STM in
the other CS input channels because of its more rapid accumulation.

The different asymptotic limits for the ALTM levels for the CS input channels
conditioned at each ISI result from the operation of the adaptively
regulated maximum ALTM level mechanism described in Chapter 7. Even though the
duration and amplitude of both the CS and the US remain fixed for the
acquisition training at different ISIs, the different CST amplitudes produced
by each ROC setting (Figure 8-6), and the different maximum STM levels
supported by training at different ISIs (discussed below), lead to different
maximum ALTM levels.
FIGURE 8-8. ALTM levels during massed excitatory acquisition over 200 training trials at 4 different ISIs, when 4 CS input channels with the ROC settings indicated in Table 8-1 are simultaneously activated by the same CS. (a) ISI = 130ms. (b) ISI = 250ms. Figures 8-8c and 8-8d are on the next page.
FIGURE 8-8. ALTM levels during massed excitatory acquisition over 200 training trials at 4 different ISIs, when 4 CS input channels with the ROC settings indicated in Table 8-1 are simultaneously activated by the same CS. (c) ISI = 500ms. (d) ISI = 1000ms. Figures 8-8a and 8-8b are on the previous page.
While the above examination of the individual STM and ALTM levels within each CS input channel provides some insight into the temporal discrimination process within ACE, the overall behavioral result is most easily appreciated by examining ACE's "CS expects US" output signal, OUT. Figure 8–9a shows the 4 OUT signals produced after the 200 acquisition trials at each of the 4 ISIs. Each OUT signal peaks shortly following US onset, and has a shape and relative overall amplitude closely resembling the empirical results obtained by Smith (1968), which are reproduced in Figure 8–10. As would be expected from examination of Figure 8–7, the OUT signal generated after 200 acquisition trials at a particular ISI is dominated by the CST signal which peaks coincidently with US onset. However, it is also apparent that in Figure 8–9a the temporally adjacent CST signals, and in particular that which precedes the CST signal which coincides with US presentation, also make a significant contribution to the OUT signal. This is revealed by the presence of small bumps or shoulders to the left of the peak in each of the OUT signals. Similar artifacts appear to be present in the empirical results reproduced in Figure 8–10, though these may result from averaging across many subjects and trials.

A further 200 acquisition trials were performed at each of the 4 ISIs to reveal the extent to which the temporal profile of each of the OUT signals is further refined. The 4 separate OUT curves produced after all 400 acquisition trials at each of the 4 separate ISIs are shown together in Figure 8–9b. By comparison with those obtained after 200 acquisition trials (Figure 8–9a), those after 400 trials have substantially higher peaks coinciding approximately with US onset, and less lateral spread. However, the total area under each curve after 200 and 400 trials is essentially conserved. In other words, the total integrated expectation of reinforcement able to be supported by the US at each ISI has already been approached after 200 trials, and subsequent training serves mainly to refine the temporal discrimination at each ISI. It is apparent from Figure 8–9b that this improvement in temporal discrimination is also being supplemented by the establishment of inhibitory outputs from those CS input channels with CST signals which peak well after US onset. This is particularly evident in each of the OUT curves at the 2 shorter ISIs, which because of the inhibitory effect of later CST signals, actually reach a local minima and then increase before later decreasing.
FIGURE 8-9. ACE output signals (OUT) generated after massed excitatory acquisition at ISIs of 130ms (heaviest shading), 250ms (2nd heaviest shading), 500ms (2nd lightest shading), and 1000ms (lightest shading), when 4 CS input channels with the ROC settings used in Figures 8-7 and 8-8 are activated by the CS. (a) Outputs produced after 200 training trials. (b) Outputs produced after 400 training trials.
FIGURE 8-10. Mean topography of NMR based on five test trials during day 10, using a 50ms CS with ISIs of 125ms, 250ms, 500ms, and 1000ms, and US shock intensities of 1mA, 2mA and 4mA. Reprinted from Smith (1968).
The micromolar operation discussed above reveals how multichannel operation considerably extends the behavioral capability of ACE, even when only a single CS is being conditioned to the US in the absence of background stimuli and other CSs. When, as in Figure 8-5, only a single CS input channel is used, the CS simply becomes a conditioned excitor with the same temporal profile as its CST signal. However, when after substantial training at a fixed ISI the same CS drives multiple CS input channels, with different ROC settings to produce a spectrum of CST signals, some CS input channels develop conditioned excitation (positive SIM values), while others develop conditioned inhibition (negative SIM values). In other words, a "conditioned excitor" is actually composed of conditioned excitors which are temporally grouped close about (and particularly before) US onset, and conditioned inhibitors which temporally delineate the extent of their excitatory spread (particularly following US onset). If the ISI is relatively long, then strong conditioned inhibition will also be produced in those CS input channels with CST signals which peak at short ISIs. Indeed, in a separate test when ROC settings of 0.20, 0.10, 0.05, and 0.02 were used, 400 acquisition trials at an ISI of 1000ms (coinciding with the CST generated with an ROC setting of 0.02) produced sufficiently strong early conditioned inhibition to make the early part of the OUT signal actually inhibitory (results not illustrated). In other words, a conditioned excitor was developed which not only contained inhibitory temporal elements, but which actually acted as a net conditioned inhibitor shortly after CS onset, before later acting as a net conditioned excitor at about the time US presentation is expected. This specific type of behavior has been observed in the empirical results of Rescorla (1967b).

Earlier in this chapter a set of results obtained using a single CS input channel starting with 3 different initial ALTM values during massed excitatory acquisition were presented in Figure 8-5, and compared with empirical acquisition curves. Figure 8-11 illustrates a single set of these same computer simulation results, along with the corresponding results generated by a CS input channel, with the same ROC setting and initial ALTM value, from the multichannel acquisition performed at an ISI of 250ms as in Figures 8-7b and 8-8b.
FIGURE 8-11. Comparison of SIM and ALTM levels in 1 CS input channel with ROC = 0.1 during massed excitatory acquisition over 200 training trials at an ISI of 250ms, when this CS input channel alone is activated by the CS (heavy shading), and when the CS also activates 3 other CS input channels with ROC settings of 0.2, 0.05, and 0.02 (light shading). (a) STM levels. (b) ALTM levels.
The difference between the single channel and the multichannel STM acquisition curves illustrated in Figure 8-11a, and between the ALTM acquisition curves shown in Figure 8-11b, are shown heavily shaded. The STM curves show a substantial difference in acquisition rate, with that from the multichannel acquisition being markedly slower due to competition from the other CS input channels with temporally adjacent CST curves. The ALTM curves are qualitatively similar, differing only marginally in their rate of increase, which results directly from the different STM acquisition rates.

Note, however, that another important qualitative difference exists between the STM curves depicted in Figure 8-11a. The single channel acquisition STM curve approaches a fixed maximum asymptotic limit, whereas the multichannel acquisition STM curve approaches a fixed rate of increase. The 2 STM curves actually intersect after approximately 300 training trials, with the multichannel acquisition curve maintaining the same linear rate of increase as that illustrated in Figure 8-11a. Although not illustrated here, this is evident from extrapolation of each STM curve illustrated in Figure 8-11a, and was verified by direct examination of results obtained over a subsequent 200 training trials.

This qualitative difference between the single and multichannel acquisition STM curves provides another insight into the temporal discrimination refinement process illustrated in Figure 8-9. The net excitatory strength produced by all CS input channels remains essentially constant after a given number of acquisition trials (approximately 200), as would be expected since ACE implements an intratrial version of the Rescorla–Wagner model (1972). However, the STM level of the CS input channel shown in Figure 8-11a is able to steadily increase indefinitely because the CS input channels with adjacently peaking CST signals are becoming progressively less excitatory and more inhibitory. Hence, the single channel acquisition STM curve still conveys what is happening to the net excitatory strength during multichannel acquisition. However, comparison between this single channel acquisition SIM curve and that of the corresponding channel in a multichannel acquisition environment further illustrates the ongoing temporal refinement process.
EXTENT OF ACQUISITION AS A FUNCTION OF ISI

The acquired strength of a classically conditioned excitor is critically affected by the period of time between CS onset and US onset, referred to as the Inter-Stimulus Interval (ISI). The precise relationship between extent of acquisition and ISI is dependent upon the animal species studied, the type of response system, the response amplitude measurement technique, the types of CS and US employed, and their intensities and durations.

However, despite the variability, most empirical data exhibit the following general characteristics (e.g., Black and Black, 1967; Boice and Denny, 1965; Gormezano, 1984; Gormezano, 1972; Kehoe, 1979; Kimble, 1961, p. 155; McAdam, Knott, and Chiorini, 1965; Mackintosh, 1974, p. 62; Meredith and Schneiderman, 1967; Schneiderman, 1972; Schneiderman, 1966; Schneiderman and Gormezano, 1964; Smith, 1968; Smith, Coleman, and Gormezano, 1969; Trapold, Homzie, and Rutledge, 1964; Vandercar and Schneiderman, 1967):

(i) No excitatory conditioning is acquired if the CS begins simultaneously with, or following the US onset (i.e., if the ISI is zero or negative).

(ii) The magnitude of acquired excitatory conditioning is an inverted U function of ISI, with the ISI value corresponding to maximal acquisition known as the optimum ISI.

(iii) Little if any excitatory conditioning is acquired when the CS precedes the US by more than some finite period of time, being greater than the optimal ISI, which is response system dependent.

The optimal ISI is typically between 200ms and 750ms for discrete, skeletal CRs such as eyelid/nictitating membrane and leg flexion responses, and typically between 5s and 10s for slower preparatory responses such as salivation, and heart rate conditioning. As would be expected, those systems with short optimal ISIs (less than 1s) also tend to have short maximum ISIs in the order of seconds, beyond which conditioning rapidly becomes apparently ineffective. The slower response systems have less easily defined maximum ISIs which may extend into minutes.
ACE has been specifically designed to model consummatory response systems in general, and the NMR of the rabbit in particular, which has been relatively well studied with respect to the effect of ISI upon acquisition. (Gormezano, 1984; Gormezano, 1972; Kehoe, 1979; Meredith and Schneiderman, 1967; Schneiderman, 1932; Schneiderman, 1966; Schneiderman and Gormezano, 1964; Smith, 1968; Smith, Coleman, and Gormezano, 1969; Vandercar and Schneiderman, 1967). Gormezano (1984, p. 14) notes that "Over a broad range of parameters, our NMR preparation has consistently revealed a tightly bound range of CS-UCS intervals for conditioning with maxima between 200 and 400 msec and levels of responding declining to negligible levels as the ISI approaches 3-4 sec...". Although the precise relationship between response strength and/or probability and the ISI employed in acquisition is dependent upon all of the above mentioned factors, the overall output response results for ACE illustrated in Figure 8-9 are consistent with these empirical results.

**CONDITIONED INHIBITION**

A conditioned inhibitor (CS-) may be defined as a stimulus which, when presented in compound with a conditioned excitor (CS+), significantly reduces the strength or probability of occurrence of the CS+'s CR. The most popular and effective procedure for establishing a CS- is still Pavlov's conditioned inhibition paradigm (Pavlov, 1927, p. 68). The subject is first repeatedly exposed to CS1 consistently followed by reinforcement, firmly establishing it as a conditioned excitor (CS1+) which elicits its own CR. Learning trials, in which CS1+ and CS2 are presented simultaneously in compound without subsequent reinforcement, are then interspersed with ongoing reinforcement of CS1+ done. As training proceeds, the CR initially generated by compound presentation of CS1+ and CS2 becomes progressively weaker, but the CR generated by CS1+ alone retains its strength. In other words, CS2 becomes a conditioned inhibitor (CS2-).

The resulting CS2- is not just specific to CS1+ however, its inhibitory capacity generalizes to other CS+s associated with the same reinforcer used during training (but not to other USs), and is therefore effective in novel contexts (Rescorla, 1984, p. 39-40). This behavior tends to indicate that a
conditioned inhibitor develops an association with a US, rather than a CS+. ACE implements this type of conditioned inhibition by allowing each CS input channel to acquire and produce an inhibitory output signal (INH). In this respect it is similar to the basic configuration proposed by Konorski (1948), and later refined by Rescorla and Wagner (1972). However, it should be noted that, unlike the original Rescorla–Wagner model (1972), ACE will not cause a CS– to extinguish when it is presented alone, because negative OUT values are not allowed, and so in this respect is consistent with empirical results (e.g., Zimmer-Kart and Rescorla, 1974).

A single CS input channel previously trained over 400 trials of massed excitatory acquisition training at an ISI of 250ms, and with an ROC setting of 0.1 to make its CST coincide approximately with US onset, was used to provide the pretrained CS1+. STM[1] and ALTM[1] identify in Figure 8–12 the STM and ALTM levels respectively for the CS input channel (number [1]) employed for CS1+. CS input channel number [2] was used for CS2–, which as illustrated in Figure 8–12a has initial STM[2] and ALTM[2] values of 0.0 and 0.1 respectively, corresponding to the standard initial values used for all naive CS input channels.

For graphical presentation, the negative STM[2] values are shown inverted in Figure 8–12 to emphasise the difference between excitatory CS1+ strength and inhibitory CS2– strength as acquisition proceeds. The negative STM[2] curve is shaded with a heavy hatched pattern to help differentiate it from all of the other positive quantities graphed in Figure 8–12.

It is clear from Figure 8–12 that CS1+ retains most of its excitatory strength, as indicated by its almost level STM[1] curve. The gradual growth in negative STM[2] amplitude indicates the expected growth in conditioned inhibition for CS2–. An interesting feature of Figure 8–12f is that the negative STM[2] curve intersects the excitatory STM[1] curve, and continues to climb at a fixed rate. With ACE implementing a modified intratrial version of the Rescorla–Wagner model (1972), it might be expected that the strength of CS2– would level out as it approaches that of CS1+. The fact that it does not, the reason why, and the behavioral utility of this are discussed later below.
FIGURE 8-12. STM and ALTM levels for both the CS+ and the CS− during massed acquisition using Pavlov's conditioned inhibition paradigm, over successive groups of 200 training trials at an ISI of 250ms, when the 2 CS input channels have ROC settings of 0.1. Note that STM[2] (hatched), being inhibitory, is actually a negative quantity. (a) Trials 0–200. (b) Trials 200–400. Figures 8-12c, d, e and f are on the following pages.
FIGURE 8-12. STM and ALTM levels for both the CS+ and the CS− during massed acquisition using Pavlov's conditioned inhibition paradigm, over successive groups of 200 training trials at an ISI of 250ms, when the 2 CS input channels have ROC settings of 0.1. Note that STM[2] (hatched) is a negative quantity. (c) Trials 400–600. (d) Trials 600–8069. Figures 8-12a and 8-12b are on the previous page, Figures 8-12e and 8-12f are on the next page.
FIGURE 8-12. STM and ALTM levels for both the CS+ and the CS- during massed acquisition using Pavlov's conditioned inhibition paradigm, over successive groups of 200 training trials at an ISI of 250ms, when the 2 CS input channels have ROC settings of 0.1. Note that STM[2] (hatched) is a negative quantity. (e) Trials 800–1000. (f) Trials 1000–1200. Figures 8-12a, b, c and d are on the previous pages.
Figure 8-12 also reveals that the rate of acquisition for CS2- is many times slower than that of CS1+ (Figure 8-11a), even allowing for the fact that 2 trials in Figure 8-12 provide only 1 acquisition trial for CS2-. The unequal rates of accumulation and depletion of MIM and LTM, which were determined in Chapter 4, are partly responsible for this asymmetry in acquisition rates. The accumulation rates for both MTM and LTM are twice as fast as their depletion rates, in order to produce greater than 50% spontaneous recovery, and a U-shaped excitatory memory retention curve which correlates well with empirical results.

Another reason why inhibitory acquisition proceeds more slowly than excitatory acquisition within ACE is that the associability level of CS2- (ALTM[2]) remains at its original low level throughout training, as illustrated in Figure 8-12. The absence of US presentations following CS2- presentation prevents ALTM changes from occurring, which in any case would be reductions and not increases, because the decreases in STM[2] produce negative MTM[2] values. The acquisition rate of CS2- should be able to be increased by prior massed excitatory acquisition and then extinction, as this would elevate ALTM[2].

A striking feature of Figure 8-12a is the rapid reduction in the associability level of CS1+ (ALTM[1]) at the beginning of training. This, along with the above proposition regarding prior elevation of ALTM[2], represent clearly testable predictions arising from the operation of ACE, for which empirical results could be readily obtained. The dramatic drop in ALTM[1], combined with the extensive spontaneous recovery and the reinstatement effect of US presentation supported by ACE, contribute to the minimal reduction in the excitatory strength of CS1+ during early training. This common type of result (e.g., Rescorla, 1984, p. 39) is obtained despite the fact that CS1+ effectively experiences a sudden change from a 100% to a 50% reinforcement schedule at the beginning of acquisition training far CS2-.

The unexpected linear progression in the strength of the CS- beyond that of the CS+ employed to train it, revealed in Figure 8-12f, motivated a search for the mechanism responsible. As noted above, with ACE implementing a modified
intratrial version of the Rescorla–Wagner model (1972), the CS− strength would be expected to level off as it approaches that of the CS+, since expectation then comes to match the outcome for both reinforced and nonreinforced trials. The additional mechanism or mode of operation responsible for the additional conditioned inhibition accruing to the CS− will now be discussed.

Consider first that CS input channel activated by the CS+, with an ROC setting which makes its CST signal peak approximately at US onset. The STM in the NMMM provides the CS−specific temporal buffer required to compare expected with actually delivered reinforcement. While the HNC feedback signal, which conveys the potentially reinforcing effect of US presentation, is temporally concentrated primarily at US onset and terminates with US offset (Chapter 6), the expectation of reinforcement conveyed by the OUT signal is sustained over a much greater duration (e.g., Figure 8–9). When the total integrated expected and delivered reinforcement are equal, there is no net change in the STM level associated with the CS+ from trial to trial, in accordance with the Rescorla–Wagner model (1972). However, the different temporal profiles of expected and actual reinforcement make the CS+’s STM level significantly deviate from its average asymptotic value. Although the process is complicated by the operation of several mechanisms, the STM levels tend to behave as follows:

After CS+ onset, but before US onset, almost half of the expectation of reinforcement has been produced, significantly reducing the STM level of the CS+. When the US is then presented, its reinforcing effect (in addition to its reinstatement effect) elevates STM well above its average asymptotic value. Then, after the US terminates, the lingering expectation of reinforcement gradually brings STM back below its average asymptotic value again. This then combines with the negative influence upon STM immediately after CS onset at the beginning of the next trial, making STM reach a definite low point just before US onset.

The unique design of ACE extends the effect of the above process so that it also affects all other CS input channels via the nonassociative extinction and reinstatement mechanisms described in Chapter 7. All other CS input channels
within the same ACE experience cycles of depressed STM levels, which are then reinstated to their previous long term values by each US presentation. In the special case of those CS input channels which are activated by CS presentations on alternate nonreinforced trials, such as the CS– in the Pavlovian conditioned inhibition paradigm, this mechanism provides the additional depression of STM required to produce the ongoing development of conditioned inhibition apparent in Figure 8–12f.

In order to isolate this particular cyclic effect from that produced by the standard overall discrepancy between expectation and delivery of reinforcement, another set of results were obtained using a different conditioned inhibition paradigm. If trials in which CS1 is reinforced are randomly intermixed with trials in which CS2 is nonreinforced, CS1 becomes a CS+, while CS2 may be established as a CS– (Konorski and Szwejkowska, 1952; Szwejkowska and Konorski, 1959; Rescorla and LoLordo, 1965; LoLordo, 1967). This is a type of successive discrimination procedure (Mackintosh, 1983, p. 241), and is usually referred to as differential conditioning (Mackintosh, 1974, p. 34).

Figure 8–13 shows the computer simulation results generated using differential conditioning, in which reinforced CS1+ trials are alternated with nonreinforced CS2– trials. The same pretrained CS1+ employed in Figure 8–12 is used in Figure 8–13 to facilitate comparison between them. Early in differential training of CS2–, its conditioned inhibition is acquired at a substantially slower rate than that early in training using the Pavlovian conditioned inhibition paradigm, results for which were presented in Figure 8–12. As training proceeds, the accumulation rate for the Pavlovian procedure slows down until, when the CS2– strength exceeds the CS1+ strength, it comes to equal that for the differential procedure. In other words, the Pavlovian procedure includes the negatively accelerated acquisition produced by the Rescorla-Wagner model (1972), in addition to the almost linear acquisition of conditioned inhibition produced by the particular internal processing of ACE, the effect of which is separately revealed in Figure 8–13.
FIGURE 8-13. STM and ALTM levels for both the @S+ and the CS− during massed differential conditioning over successive groups of 200 training trials at an ISI of 250 ms when the 2 CS input channels have ROC settings of 0.1. Note that STM[2] (hatched), being inhibitory, is actually a negative quantity. (c) Trials 0–200. (d) Trials 200–400. Figures 8–13c, d, e and f are on the following pages.
FIGURE 8-13. STM and ALTM levels for both the CS+ and the CS− during massed differential conditioning over successive groups of 200 training trials at an ISI of 250 ms when the 2 CS input channels have ROC settings of 0.1. Note that STM[2] (hatched) is a negative quantity. (c) Trials 400–600. (d) Trials 600–800. Figures 8-13a and 8-13b are on the previous page, Figures 8-13e and 8-13f are on the next page.
FIGURE 8-13. STM and ALTM levels for both the CS+ and the CS− during massed differential conditioning over successive groups of 200 training trials at an ISI of 250ms when the 2 CS input channels have ROC settings of 0.1. Note that STM[2] (hatched) is a negative quantity. (e) Trials 800–1000. (f) Trials 1000–1200. Figures 8–13a, b, c and d are on the previous pages.
Animal experiments comparing the 2 types of procedures support the above result that Pavlov's conditioned inhibition paradigm is much more effective at establishing CS2 as a CS− than the simpler differential conditioning procedure (e.g., Marchant and Moore, 1974; Rescorla and Holland, 1977). Note that the effectiveness of differential conditioning for ACE will be substantially reduced when ITIs larger than the 10s employed here are used. ITIs of approximately 60s are more typical in experiments using the NMR preparation.

The attempted explanation usually advanced to account for the ability of the above differential conditioning to develop conditioned inhibition is that conditioned excitation is developed to situational stimuli during the reinforced trials, which then provides the necessary expectation of reinforcement during the nonreinforced trials to establish the CS−. While plausible, this explanation is unlikely to account for all of the conditioned inhibition established (Mackintosh, 1983, p. 188). ACE therefore provides a new possible explanation for the full effectiveness of differential conditioning to establish conditioned inhibition.

Figure 8–13 also indicates that CS1+ retains all of its excitatory strength during differential conditioning, a result expected since CS1+ is always reinforced fully when presented. The slower reduction in the associability of CS1+ (ALTM[1]) for this differential conditioning procedure, compared to that resulting from Pavlov's conditioned inhibition paradigm, is another testable prediction emanating from ACE's operation.
THE PARTIAL REINFORCEMENT EFFECT

The Partial Reinforcement Effect (BRE) refers to the reduced rate of extinction of a CS previously reinforced during some fraction of its excitatory acquisition trials, compared to the rate of extinction of a CS previously continuously reinforced on every acquisition trial. The extent of the PRE is usually greatly enhanced by the use of a multistage procedure in which all subjects are initially continuously reinforced until a high level of responding is achieved. In the next stage some subjects are partially reinforced, while others are continuously reinforced. All subjects are then exposed to a series of CS-alone extinction trials, and their rate or extent of extinction are compared to reveal the magnitude of the PRE.

The PRE is supported within ACE by its new adaptive associability mechanism, which as a result of partial reinforcement, reduces the associability of a CS with the US. Subsequent reacquisition of the partially reinforced CS would therefore also be expected to occur at a reduced rate - a prediction for which empirical evidence could readily be obtained. The way in which the NMMM supports the PRE was described in the final pages of Chapter 4. However, computer simulation results demonstrating ACE's capability to support the PRE were not able to be presented at that stage, as the adaptive associability mechanism interacts with most other mechanisms within ACE, which were documented in later chapters.

In Chapter 4 it was described how the following specific empirically observed characteristics of the PRE are supported by the NMMM (from Mackintosh, 1974, pp. 443-447):

(i) N trials only increase resistance to extinction when they are followed by an R trial, and become less effective as the interval between them is increased beyond several minutes.

(ii) Early in training, resistance to extinction is primarily determined by the number of N-R transitions.

(iii) Later in training, and at short ITIs (less than 20min), the number of successive N trials (or "N-length") preceding each R trial in a given session is primarily responsible for determining the extent of the PRE. However, at
long ITIs (greater than 20 min), the proportion of N trials in acquisition is a more important factor than N-length.

Although an adaptively regulated maximum ALTM mechanism was later introduced in Chapter 7, the explanations provided in Chapter 4 for all 3 of the above characteristics remain valid for experimental procedures in which the ITI is much greater than the 10s used here, as is usually the case. This results in the maximum ALTM level being limited more by the asymptotic STM limit than by the adaptive maximum ALTM level.

Empirical results are reproduced below from 2 experiments conducted by Gibbs, Latham, and Gormezano (1978). These experiments assess the effects of Partial Reinforcement (PR) schedules on the maintenance and resistance to extinction of the rabbits' NMR. Both experiments consisted of three stages. In Stage I all subjects received continuously reinforced acquisition training. Stage II consisted of different types of PR schedules for each group of subjects, and revealed the extent to which responding was maintained. Stage III consisted entirely of CS-alone extinction training for all subjects, and revealed resistance to extinction.

In Experiment 1, 6 groups of subjects were employed. In Stage II of Experiment 1, each group received a PR schedule with a probability of reinforcement of 100%, 50%, 25%, 15%, 5%, or 0%. Each group is referred to by "C ", followed by its corresponding percentage probability of reinforcement. The mean percentage of CRs for each group is reproduced in Figure 8-14. After reaching comparable levels of responding in Stage I, the performance maintained by each group was a monotonically increasing function of the probability of reinforcement. For Groups C 100, C 50, C 25, and even C 15, the level of responding appears to drop rapidly by a small amount within the first block of trials, and then remain relatively constant for the rest of Stage II. Although Group C 5 shows a definite progressive decline in responding, it is markedly better than that of the extinction control Group C 0. In Stage III Groups C 50 and C 25 produce the highest response performance, suggesting the presence of a substantial PRE.
Computer simulation results of ACE’s operation when subject to a similar 3 stage procedure to that employed by Gibbs, Latham, and Gormezano (1978) in Experiment 1 are shown in Figure 8-15. Despite differences in experimental procedure and response measurement, the results illustrated in Figure 8-15a compare very favorably with the empirical results reproduced in Figure 8-14, with respect to the main observations described above. After starting from the same conditions following continuously reinforced acquisition, the response strength of a single CS input channel when subject to similar PR schedules exhibits a similar rapid decline to a marginally lower level, and then only a very slow decline throughout the partially reinforced stage. In the extinction stage, all PR schedules, and in particular the 50% and 25% ones, soon-produce stronger responses than the fully reinforced group, illustrating a strong PRE.
FIGURE 8–15. Massed training of 1 CS input channel, previously continuously reinforced for 400 trials, then subject to each of 6 different partial reinforcement schedules over 100 trials, then 100 extinction trials. Partial reinforcement schedules of 100%, 50%, 25%, 10%, 5%, and 0% were used. ROC □ 0.1, ISN □ 250ms. (a) STM levels. (b) ALTM levels.
The graph of ALTM values for each CS input channel separately trained, shown in Figure 8-15b, provides an additional insight into the means by which ACE produces a **PRE**. As indicated in Figure 8-2, changes in ALTM are gated by the Enable Associability Change (EAC) signal, which only attains positive nonzero values when US presentation occurs while the CS Trace (CST) signal is still above zero following CS presentation. In other words, EAC only enables changes in ALTM when a *CS* is reinforced by subsequent US presentation.

The ALTM level of the completely nonreinforced CS input channel, labelled 0% in Figure 8-15b, therefore remains fixed at the elevated level produced by prior continuously reinforced massed excitatory acquisition. The ALTM levels of all of the partially reinforced CS input channels drop substantially with each reinforced trial. The extent of the drop in ALTM value depends upon the preceding **number** of nonreinforced trials. Hence, while those *CS* input channels receiving a lower probability of reinforcement experience fewer reinforced trials, the extent of the change in ALTM with each reinforced trial tends to be greater. Consequently, all partially reinforced CS input channels tend to attain similarly **low** ALTM values during the PR stage. In the final extinction stage, all ALTM levels remain unaltered due to the complete absence of reinforcing US presentations, which ensures that the PRE produced remains effective for an indefinite period in extinction.

With all PR schedules producing **similarly** low ALTM values, the extinction rates they exhibit in Figure 8-15a are so dramatically slower than that following continuous reinforcement that it is difficult to tell which probability of reinforcement produces the greatest resistance to extinction. Figure 8-15b is of some assistance in this regard, since it clearly indicates that the 25% PR schedule produces the lowest ALTM value, with the 50%, 10%, and 5% PR schedules producing progressively higher ALTM values. This compares very well with the 25%, 50%, 15%, and 5% order obtained by Gibbs, Latham, and Gormezano (1978, Figure 2), when the groups are sorted in order of decreasing average percentage of **CRs** produced over the entire extinction stage of Experiment 1.
The steps in each STM curve of Figure 8-15a are produced by the localised temporal effect of each reinforced and nonreinforced trial. In contrast, Gibbs, Latham, and Gormezano (1978) found no evidence of such immediate reinforcement-dependent changes in response frequency for each individual subject. Their empirical results suggest that the direct relationship between STM and response strength utilised within ACE could possibly be enhanced by the addition of new cumulative quantities which temporally buffer the effect of changes to STM upon response performance.

The apparent absence of decrements in CR maintenance that are able to be localised to the immediate effects of reinforcement in Experiment 1 led Gibbs, Latham, and Gormezano (1978) to consider more generalised effects of nonreinforcement. More specifically, they considered stimulus generalisation hypotheses, for which they cited Capaldi (1966) and Sheffield (1949) as examples. With the intention of assessing the possible implications of such stimulus generalisation hypotheses, they devised and conducted a second experiment in which a group of subjects received progressively lower probabilities of reinforcement in Stage I, thus avoiding an abrupt transition between continuous and partial reinforcement schedules.

In Experiment 2, Gibbs, Latham, and Gormezano (1978) employed three groups of subjects. Group C (Continuous reinforcement) was almost continuously reinforced with 95% probability in Stage II, while Group E (Extinction) received CS-alone trials only. Group P (Partial reinforcement) was reinforced with a probability of 85%, 75%, 65%, 55%, 45%, 35%, 25%, 15%, and 5%, for successive sessions of 60 trials in Stage II. The mean percentage of CRs for each group are reproduced in Figure 8-16. The response probability of Group P remained remarkably high during Stage II, being indistinguishably close to that of Group C until the last 1/3 of Stage II, as the probability of reinforcement approached 5%. In Stage III, in which all 3 groups received CS-alone extinction trials, Group P response performance converged and overlapped with that of Group C. Since the response probability of Group C fell from a higher level at the end of Stage II than that of Group P, this is also suggestive of a significant PRE.
FIGURE 8-16. Percentage CRs as a function of 2-day blocks of 120 trials in Stages I, II, and III. Stage I is a fully reinforced acquisition period. Stage II consists of a progressively reducing partial reinforcement schedule of 85%, 75%, 65%, 55%, 45%, 35%, 25%, 15%, and 5%, for each successive 2-day block of trials (Group P). Stage III is a period of extinction training. Also shown are the percentage CRs for 95% (Group C), and 0% (Group E) reinforcement schedules during Stage II. Reprinted from Gibbs, Latham, and Gormezano (1978) - Experiment 2.

A 3 stage procedure similar to that employed by Gibbs, Latham, and Gormezano (1978) in Experiment 2 was devised, for which computer simulation results of ACE's behavior are shown in Figure 8-17. The specific sequence of R and N trials used in the Stage II is indicated in Table 8-2. A striking similarity exists between the trend of the STM curve illustrated in Figure 8-17a, and that of Group P in Figure 8-16, during Stage III of each procedure. In Stage III, in which CS-alone trials are experienced, the computer simulation results of Figure 8-17 also reveal the expected PRE. As with Figure 8-15, the PRE is of greater prominence than that exhibited by the empirical results reproduced in Figure 8-16, which is primarily a result of the decision to employ the relatively short 10s ITI to generate the computer simulation results (p. 8-6).
Comparison between Figure 8-17 and Figure 8-15 indicates that the progressively declining PR schedule achieves an AETM level as low as that produced by the fixed 10% PR schedule, but with a response strength at the end of Stage I as high as that produced by the fixed 25% PR schedule. In other words, ACE adapts more favorably to a declining PR schedule than to an abrupt change to one of low probability of reinforcement.

The extent of the similarity between the trends of the computer simulation results and the empirical results, for both Experiments 1 and 2 of Gibbs, Latham, and Gormezano (1978) suggest that ACE is capable of accounting for the empirically observed PRE and the maintenance of response. Although it may be possible to interpret the internal operation of ACE in terms of some form of stimulus generalisation hypothesis, recourse to such mechanistically vague theoretical accounts may no longer be required.
FIGURE 8–17. Massed training of 1 CS input channel (shaded), previously continuously reinforced for 400 trials, over 100 partial reinforcement trials, then 100 extinction trials, when partial reinforcement is progressively reduced from 80% to 5% as detailed in Table 8–2. Also shown (unshaded) for reference are the results for fixed 100%, 59%, and 0% PR schedules. ROC = 0.1, PSI = 250 ms. (a) STM levels. (b) ALTM levels.
ALTERNATE ACQUISITION-EXTINCTION SESSIONS

If subjects are given training which consists of alternating sessions of fully reinforced acquisition trials and then extinction trials, it is well established that the rate of both reacquisition and reextinction progressively increases (e.g., Bullock and Smith, 1953; Gonzalez, Holmes, and Bitterman, 1967; Davenport, 1969).

It was discussed in the final pages of Chapter 4 how ALTM is increased by alternate acquisition-extinction sessions, and how the extent of the increase in ALTM is increased as the interval between extinction and acquisition sessions is made progressively longer than the ITI within each session, as empirically observed by Capaldi, Leonard, and Ksir (1968). The latter results from the medium term retention characteristics of MTM, the value of which is affected by changes in STM produced by acquisition and extinction, and in turn determines the direction and extent of change in ALTM.

Computer simulation results have been presented in this chapter which illustrate that ALTM level is actually increased by massed excitatory acquisition (Figures 8–5b and 8–8), and that ALTM level remains unaltered during extinction (Figures 8–15b and 8–17b). Although the ALTM levels approach their adaptively regulated maximum levels in each of the massed excitatory acquisition sessions for which results are presented, only partial increases in ALTM would be produced if each acquisition session included fewer trials, or if the ITI was much greater than the 10s ITI used here. Under these circumstances ALTM level would be progressively increased by each of the acquisition sessions early in training.

Results from the computer simulations intended primarily to reveal the PRE also reveal an additional role for alternate acquisition-extinction sessions that was not originally designed for, or considered. Figure 8–15b reveals that the ALTM level of a continuously reinforced CS does in fact decline, albeit at a very slow rate. The absence of any such decline in Figure 8–5b indicates that it does not occur until massed excitatory acquisition is well advanced, or in
other words until the total integrated expectation of reinforcement is nearly equal to that subsequently delivered.

The mechanism responsible for the decline in ALTM is associated with that responsible for differential inhibitory conditioning, and is most easily described in terms of the behavior of SIM in a single CS input channel activated by a reinforced CS+. The periodic minimum SIM level occurring prior to US onset also produces small negative MTM values at this time, as STM acquisition nears completion. These small negative MTM values produce small reductions in AETM level each time reinforcement is experienced, producing the observed progressive decline in associability.

A behavioral implication of this is that if a CS+ is continuously reinforced with extensive massed training, then the very high SIM level attained by the CS+ with the assistance of a rapidly increasing ALTM value, will eventually tend to become locked into memory by a subsequently decreasing ALTM value. The massed training need not consist of a single massive unbroken session, as many brief sessions will produce a similar result, albeit less efficiently. Intuitively, when a CS-US association in STM and LTM has been consistently and extensively reinforced, it is appropriate that it attain some degree of relative permanence. If an unexpected outcome is subsequently experienced following presentation of such a previously reliable CS+, then its low associability will tend to protect its excitatory associative strength while other new CSs are trained to expect the deviation in outcome.

Within ACE, alternate acquisition–extinction training sessions therefore not only help to establish an appropriately high level of associability, they are also required to maintain a high level of associability. The latter role is another prediction for which empirical results may be readily obtained, though its behavioral utility is apparent with or without such empirical support.
LATENT INHIBITION AND LEARNED IRRELEVANCE

If a CS is presented to a subject without reinforcement prior to excitatory acquisition training, then the subsequent rate of conditioning to the CS will be retarded compared to that of a nonpreexposed CS. The effect was called "latent inhibition" by Lubow and Moore (1959), who first studied it using excitatory classical conditioning of the leg flexion CR in sheep and goats. They referred to the effect as latent inhibition, because they reasoned that the preexposed CS acquired inhibitory properties which retarded subsequent excitatory conditioning, since inhibition opposes excitation. However, it was later demonstrated that a preexposed CS does not become inhibitory, and that subsequent inhibitory conditioning can also be significantly retarded by such CS-alone preexposure (Rescorla, 1971; Hailgren, 1974; Baker and Mackintosh, 1977). Despite this, the now somewhat misleading name given to this effect is still commonly used.

Latent inhibition has been demonstrated in several different response systems in other animal species, establishing it as a general phenomenon (Lubow, Markman and Allen, 1968; Carlton and Vogel, 1967; Anderson, O'Farrell, Formica and Caponigri, 1969; Lubow and Siebert, 1969; Siegel, 1969a, b; Kremer, 1971; Chacto and Lubow, 1967). Latent inhibition is increasingly effective with increases in the number of nonreinforced presentations of the CS (Lubow, 1965; May, Tolman, and Schoenfeldt, 1967; Siegel, 1969a), and appears to have the greatest impact during the initial training stages (Chacto and Lubow, 1967; Lubow, Markman, and Allen, 1968; Siegel, 1969b; James, 1971).

It seems reasonable to consider that a naive CS may initially produce some excitatory output (EXC) from the CS input channel it activates, as a result of generalisation from previously trained excitors, or a mildly excitatory SIM starting value. Repeated presentation of an untrained CS will then result in extinction of the small excitatory strength it has, reducing its SIM level towards zero, and making its MTM value significantly negative. Figure 4-13b indicates that such induced negative values in MTM may be sustained for hours. While this in itself produces no change in ALTM level, the reinforcing US presentations in subsequent excitatory acquisition training enables the
negative non-zero levels of MTM to decrease ALTM, and so reduce associability level. This process is virtually identical to that occurring at the transition between acquisition and extinction sessions in alternate acquisition–extinction training, described in the final pages of Chapter 4, except that the extent of the decrease in ALTM is smaller because of the weaker excitatory strength of the untrained CS. However, while these reductions in ALTM may be small in absolute terms, their effect can be considerable because, as illustrated in Figure 8–2, changes in ALTM are modulated by the current ALTM level. In other words, subsequent rates of increase in ALTM are also reduced by a decrease in ALTM level.

The extent of the latent inhibition effect produced by this process will increase with the number of CS-alone presentations, at least until extinction is almost complete. The spontaneous recovery also supported by ACE (Chapter 4) will mean that many CS-alone presentations are required to approach complete extinction. Since the ALTM level controls the rate of both increases and decreases in STM, the latent inhibition effect supported by ACE will retard both subsequent excitatory and inhibitory acquisition. Although making a naive CS less excitatory will tend to facilitate subsequent inhibitory acquisition, this will be overshadowed by the enduring effect of a reduced ALTM level, which as indicated in Figures 8–12 and 8–13 remains fixed throughout acquisition for both Pavlovian and differential procedures. Subsequent excitatory acquisition is retarded both by the reduced ALTM level, and to a lesser extent the reduced STM level of the preexposed CS. However, both of these quantities are increased dramatically as excitatory acquisition proceeds (e.g., Figure 8–11), and so their retarding effect is restricted primarily to the beginning of excitatory acquisition training. All of these characteristics are consistent with the above mentioned empirical observations regarding latent inhibition, The inverted U behavior of ACE’s MIM over the medium term, may also help account for the confusing evidence regarding short term latent inhibition effects (Mackintosh, 1983, p. 229).

Preexposure of a CS without reinforcement (i.e., latent inhibition) is neither the only, nor the most effective technique for retarding subsequent associative conditioning. A learned irrelevance procedure, in which US
Presentations are randomly correlated with CS presentations, can be dramatically more effective at retarding subsequent acquisition than latent inhibition (Gamzu and Williams, 1971; Kremer, 1971). Mackintosh (1973) confirmed this result, and also showed that learned irrelevance is specific to the reinforcer used. Mackintosh (1974, p. 40) states that "animals may specifically learn that a particular CS and UCS are uncorrelated (that the CS predicts no change in the probability of the UCS), and that this learning interferes with the establishment of an association between the 2 during subsequent conditioning".

Learned irrelevance may be implemented by ACE in a very similar manner to latent inhibition. The now interspersed US presentations provide an occasional source of reinforcement, when they happen to occur shortly after a CS presentation, which enables multiple decreases in ALTM prior to subsequent acquisition. The effect upon ALTM is similar to that illustrated in Figures 8-15b and 8-17b during partial reinforcement, except that the reinstatement effect of noncontiguous US presentations, and the reduced STM level of the naive CS, will tend to reduce the size of the reductions in AETM that occur with each contiguous US presentation. This additional process, by which ALTM may be reduced during the preexposure procedure, accounts for the increased effectiveness of learned irrelevance to reduce associability compared to latent inhibition.

Finally, as indicated in the empirical results from Kremer (1971), reproduced in Figure 8-4, latent inhibition and learned irrelevance do not just alter the rate of subsequent acquisition, they also alter the shape of its course. Figure 8-5a indicated that as the initial ALTM value at the beginning of acquisition was reduced, the shape of ACE's resulting acquisition curve changes from negatively accelerating only to sigmoidal, in accord with Kremer's results. Thus, ACE supports both the decline in rate, and the change in shape, of acquisition subsequent to latent inhibition and learned irrelevance procedures.
GENERAL DISCUSSION

The generality of classical conditioning is apt to lead the uninitiated to think that animals are able to associate a CS of any modality with reinforcement of any nature with equal facility. However, there has for some time been ample empirical evidence to indicate that this is not so. Garcia and Koelling (1966) demonstrated that rats were able to strongly associate the flavour of water with induced sickness but not with electric shock, and auditory or visual stimuli with shock but not with sickness. Garcia, McGowan, Ervin and Koelling (1968) showed that rats would associate the taste of food with induced sickness, but the visual appearance of food with electric shock. Rozin (1969) demonstrated that rats would associate the taste of a liquid with induced sickness more easily than the location of the liquid.

These are 2 main processes which may establish predispositions concerning the ease with which some stimuli are associated with particular types of reinforcers. First, phylogeny may equip an animal species with neural interconnections which favor the association of CSs from particular modalities with particular types of USs. In terms of ACE, a CS and a US must converge upon at least one ACE to enable associative conditioning between them, and conversely absence of such convergence prevents association. Second, ontogeny may lead individuals to more readily associate particular CSs with particular USs, based upon their personal experience. In other words, individuals may incorporate an adaptive associability capability. This general idea is by no means new, and receives strong support from Mackintosh (1983, pp. 222–239).

The development of ACE has taken the basic concept of adaptive associability, and created a specific functioning implementation of it. Furthermore, the role of ACE's adaptive associability behavior has been extended so that it not only supports latent inhibition and learned irrelevance – it also plays a pivotal role in the selective production of sigmoidal acquisition, the PRE, and accelerated learning following alternate acquisition–extinction sessions. In addition, ACE's adaptive associability behavior implements two effects to which most people can directly relate. First, when an excitatory association has been established
by massed acquisition which is maintained until performance or recall is strong (e.g., undergraduate studies), and then the association is apparently lost over subsequent years, subsequent reacquisition of this knowledge proceeds much more rapidly than the original acquisition. Second, when massed excitatory acquisition has been carried out well beyond the point at which maximum associative strength has been attained, then the original association becomes relatively permanent (e.g., rote learning of multiplication tables).

The latter behavioral effect of ACE's adaptive associability serves as a good example as to how apparently dissociated mechanisms may combine to produce advantageous adaptive behavior. In Chapter 4, development of the NMMM made synaptic STM available as a temporal buffer within which expectation and delivery of reinforcement could be compared, despite possible differences in their temporal profile. In Chapter 5, the CSTC was developed to produce a temporally broad trace of CS activation that compared well with the mean topography of response of the NMR. In Chapter 6, RSTM was introduced into the output stage of ACE, ostensibly to make the effectiveness of reinforcement increase with US duration in a manner consistent with empirical NMR results. RSTM also plays an important role in the implementation of empirically relevant temporal discrimination and generalisation behavior by providing the most intense reinforcing effect at US onset. The essentially brief reinforcing effect of US presentations, combined with the more sustained effect of expectation of reinforcement generated by the CSTC and the retention characteristics of STM, are responsible for the spontaneous emergence of the ability of extensively overtrained CS+s to eventually attain relative permanence.

Note that this behavior is not necessarily at odds with the overtraining reversal effect (Mackintosh, 1974, pp. 602-607), or the overtraining extinction effect (Mackintosh, 1974, pp. 423-427), in which the subsequent learning rate may be increased by increasing the amount of acquisition training. This is because ACE exhibits an inverted U-shaped ALTM curve as massed excitatory acquisition proceeds. Thus, subsequent learning rates are initially increased, and then slowly decreased, as the amount of acquisition training is increased. Precisely when the peak ALTM value is reached, if at all, depends upon many
factors such as the ISI, the ITI, the duration and intensity of the CS and the US, previous experience, and the effects of any other compound stimuli.

The selection of computer simulation results presented in this chapter illustrate how ACE produces consummatory classical conditioning behavior not adequately addressed by previous theories or models. In Chapter 6 it was described how a real-time intratrial modified version of the Rescorla-Wagner model (Rescorla and Wagner, 1972) is implemented within ACE. This feature enables ACE to exhibit all of the important advantageous behavior supported by the Rescorla-Wagner model, which includes stimulus amplitude effects, overshadowing, compound conditioning effects (e.g., blocking and unblocking), and discriminative stimulus effects. A more complete appreciation of ACE’s behavioral capability may be obtained by reading their original work (Rescorla and Wagner, 1972). A conscious decision was made to focus in this thesis upon the novel aspects of ACE, and so computer simulation results illustrating behavior directly attributable to the Rescorla-Wagner model were generally not included.

Preceding chapters may have given the impression that ACE would be merely a conglomeration of many isolated and sometimes apparently inconsequential mechanisms. In other words, that ACE might be akin to a large and fragile patchwork of tenuously interconnected mechanisms. However, from the very beginning, the aim was to produce a highly localised, integrated, and robust system of complementary subsystems to produce both a range and a depth of behavior normally associated with biological systems, but beyond the reach of most existing theories and models of associative learning and performance. While ACE has gone a long way towards achieving this aim, it still undoubtedly represents a simplified account of the type of empirical behavior with which it has been compared and assessed.

The use of computer simulation of system behavior facilitated the development of a much more complicated and interactive system than would have otherwise been achievable using theoretical conceptualisation alone. The development process was initially very slow and difficult, with a great deal of time spent exploring what later turned out to be blind alleys. However, as ACE’s internal
architecture started to take shape, the pace of development increased, and aspects of behavior not previously considered began to spontaneously emerge. ACE's development seemed to converge towards one stable solution in which a single set of mechanisms interactively produce all of the required behavior. Each "different" type of behavior is generated using the same set of mechanisms, with the different input conditions corresponding to each type of behavior emphasising particular aspects of their operation.

ACE selectively draws upon many existing theoretical constructs and empirical interpretations normally separately categorised and studied in isolation, and implements them in new ways using complex combinations of simple types of interactions with the result that they overlap and merge together. ACE suggests that memory, performance, expectation, and selective attention behavior (in the form of adaptive associability) may have been inappropriately mechanistically separated in the past, and that more integrated approaches to the understanding and production of natural intelligence behavior by artificial means appears to be a fruitful direction for future research.
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CONCLUSION
INTRODUCTION

The development, operation, and performance of a new nonlinear analog system designed to exhibit a wide range of (mainly) consummatory classical conditioning behavior has been documented herein. This new system, referred to as the Associative Conditioning Element (ACE), may be categorised as a functioning Artificial Neural Network (ANN) element, as a neuronal model of consummatory classical conditioning, or as a psychological theory of animal learning and memory, depending upon the perspective of the reader.

ACE produces behavior attributable to a single biological neuron, or small group of neurons. Despite the widespread ANN view that neural systems consist of densely interconnected networks of very simple elements, the demonstration of basic classical conditioning behavior by presynaptic facilitation in the marine mollusc Aplysia (Carew, Abrams, Hawkins, and Kandel, 1984), and the sparsely interconnected organisation of relatively humble creatures in general, suggests that individual neurons may indeed have considerable information processing capability. Neurophysiologists have for some time been aware of the mechanistic complexity of neurons, a thorough understanding of which may still be many years away. However, only recently is it starting to be more generally appreciated that much of this mechanistic complexity may be functionally instrumental in the production of similarly complex and utilitarian adaptive behavior.

ACE is a major departure from the established ANN view that neuronal models should be simple, and that complex behavior should somehow emerge from the interaction of many simple units. ACE extracts and appropriately utilises the many interrelationships that may exist among its inputs, and forms a distinctly self-contained ANN element of unusually large behavioral capability and mechanistic complexity. In so doing, ACE begins to bridge the gap between the excessive simplicity of standard ANN elements, and the considerable complexity of biological neurons.

The performance specifications for ACE consist entirely of empirical results from behavioral animal experiments, primarily focussing upon the Nictitating
Membrane (third eyelid) Response (NMR) of the rabbit. However, the widespread applicability of classical conditioning means that ACE's behavior also generalises to many other response systems and animal species, and where appropriate NMR results were not available, those from other response systems were utilised. ACE has been assessed mainly by comparing the results of computer simulations of its operation with corresponding empirical results. Where appropriate, comparisons were also made with the performance of other ANN models.

RESEARCH APPROACH

The approach used in the research described herein consisted first of minimising the input-output configuration of ACE to form the smallest possible element capable of exhibiting useful, and biologically relevant, associative conditioning behavior. A critical evaluation of the basic relationship between the inputs and the output then led to the view that the US input functions exclusively to reinforce CS-US associations. These CS-US associations are used by ACE to produce a "CS expects US" output that is subsequently available for generation of the CR, and as a source of conditioned reinforcement for both higher-order and instrumental conditioning.

The next stage in the approach used here was to maximise the complexity of the now highly localised problem, in the expectation that additional constraints and criteria would reveal aspects of the underlying mechanism that otherwise remain inaccessible. Although accorded only a small proportion of this thesis, the problem of selecting precisely which additional constraints should be included from the plethora of diverse empirical results obtained this century was a difficult and time consuming task.

After having decided to specifically target consummatory classical conditioning of the rabbit's NMR, it was then decided to address the extent of the functional role that memory may play in an adaptive system. This was partly motivated by the observation that most aspects of associative conditioning behavior also exhibit a range of short and long term retention effects. Memory, in its various types, is also one of the most poorly understood and
inadequately addressed aspects of associative conditioning behavior in the field of ANNs. However, the main reason for addressing memory phenomena in relative isolation at this early stage in the research was more pragmatic. It was considered that the development of an underlying mechanism for memory might provide a new substrate upon which mechanisms supporting other desired behavior could more easily be developed. This in fact turned out to be the case.

A fresh exploration of the functional relationship between memory and learning yielded a new nonlinear subsystem of interacting CS–specific (or synaptic) memory types, collectively referred to here as the Neural Multiprocess Memory Model (NMMM). The NMMM was progressively developed from the standard ANN adaptive synaptic weighting, initially producing spontaneous regression and recovery behavior, then U–shaped memory retention, and finally comprehensive adaptive associability behavior. The adaptive associability mechanism supports both negatively accelerated and sigmoidal acquisition curves, latent inhibition, learned irrelevance, the Partial Reinforcement Effect (PRE), and accelerated learning following alternating acquisition/extinction training sessions.

An associative Short Term Memory (STM), made available by the NMMM, is utilised to support a highly modified and enhanced intratrial version of the Rescorla–Wagner (1972) model. STM enables expectation of reinforcement and actual reinforcement subsequently experienced to be compared to determine the required change in associative strength, despite the asynchronous nature of CS and US presentation, and their different temporal profiles. This allows ACE to exhibit all of the advantageous behavior supported by the Rescorla–Wagner model, which includes stimulus amplitude effects, acquisition of conditioned excitation and inhibition, extinction, overshadowing, compound conditioning effects (e.g., blocking and unblocking), and discriminative stimulus effects.

Before moving directly onto the mechanisms which control experience–induced changes in STM, it was decided to next address fundamental problems associated with timing. Like memory, timing is also an issue left largely unaddressed within the ANN research community, though it is beginning to
receive more attention. Some aspects of timing were already addressed in the development of the NMMM. These pertained to the time-course over which the various types of associative memories spontaneously change as a result of their interaction, and the way in which time is required for consolidation of acquired knowledge to occur. However, aspects of timing of a more functionally critical nature also need to be addressed. These relate to the way in which the acquired strength and timing of a CR is dependent upon the duration of the CS and the US, and the temporal relationship between them.

Consequently, a new nonlinear Conditioned Stimulus Trace Circuit (CSTC) was developed with sufficient complexity to accurately model the shape of the mean NMR topography, and the way in which it changes when subject to variations in CS duration and amplitude. The CS "trace" output signal produced by the CSTC is available for generation of an appropriately timed CR, and to selectively gate the effect of experience subsequent to CS presentation upon the associative strength of the CS. The latter role enables the acquisition of a CR which is timed to peak approximately at the time reinforcement is expected, the production of anticipatory CRs, and the implementation of trace conditioning. It also goes some way towards supporting a relationship between rate of acquisition, and the ISI between CS and US onset, which correlates well with empirical results from animal experiments.

A relatively simple, and conventional, common output stage was then developed upon which the multiple CS input channels converge. This combines the excitatory and inhibitory output signals from the CS input channels to form a positive compound expectation of reinforcement signal, which corresponds to ACE's single "CS expects US" output signal. The output stage also processes the US input to provide the greatest reinforcement effect at US onset, and distributes both this and the output signal back to all of the CS input channels so that they may determine how their individual associative strengths and associabilities should be altered.

A new set of learning rules was then developed which control the relatively rapid changes in associative SIM that result directly from experience. These were classified into three discrete mechanisms which primarily support
extinction, reinstatement, and reinforcement. A surprising feature of the extinction mechanism is that it is distinctly nonassociative, and as a result, is able to support a new form of secondary extinction which fully spontaneously recovers. ACE can therefore capture a sense of the prevailing temporal context of US expectation and availability. This enables ACE to support, for example, the acquisition of a conditioned inhibitor by successive discrimination, without relying upon conditioned excitation to background stimuli. Unlike most ANN elements, which do not seriously address memory or timing issues, the learning rules within ACE do not dominate its design, but instead assume a more integrated role.

Development of the STM learning rules was essentially the last step in the development of ACE. These interconnected all of the previously developed subsections to form a complete version of ACE. The final task was to test and document the behavior of ACE as a complete unit, and to compare its performance with that obtained from animals. A completely new software environment was specifically developed using Microsoft C to facilitate sequencing of the 5 inputs, the selective display and logging of the 56 system variables, and management of the 25 system constants.

RESULTS

ACE is completely specified mathematically by a single set of difference equations, and a single set of associated constants whose values remain fixed for all tests. Computer simulations of ACE's operation using these equations have been presented which directly, or in some cases indirectly, indicate that the following behavior is supported:

(a) A family of excitatory acquisition curve shapes ranging from strictly negatively accelerating to strongly sigmoidal, depending upon the initial level of adaptive associability.

(b) Appropriate adaptation of associability over a wide range of acquisition conditions, including relatively rapid increases and then very gradual decreases during massed continuously reinforced excitatory acquisition, less increase (or even a decrease) in associability following partial reinforcement,
progressive increases in associability with alternate acquisition-extinction sessions, and a decreased associability level for acquisition subsequent to CS-alone (i.e. latent inhibition) or uncorrelated CS-US (i.e. learned irrelevance) presentations, with learned irrelevance tending to produce a much larger decline in associability than latent inhibition.

(c) The Partial Reinforcement Effect (PRE), with a relatively high level of response strength retained.

(d) The strength of CR in acquisition as an inverted U-shaped function of ISI.

(e) The appropriate modulation of both performance and learning when CS or US amplitude or duration are varied.

(f) Both trace and delayed conditioning, with appropriate degrees of strength of acquired association and level of associability for each type of conditioning procedure.

(g) The acquisition of conditioned inhibition, from both the Pavlovian conditioned inhibition paradigm, and purely successive discrimination (or "differential") training.

(h) A mean topography of response shape that compares very favourably with that of the rabbits' NMR, for both trace and delayed CS inputs.

(i) Discrimination and generalisation of response production in the time domain, supported by combinations of excitatory and inhibitory associations at appropriate times after CS onset.

(j) Spontaneous regression and spontaneous recovery.

(k) Reinstatement of recently diminished excitatory strength by US-alone presentations.

(l) U-shaped memory retention curves.
Consolidation of memory contents over time as information is transferred from STM to LTM.

The development of ACE, a system of considerable mechanistic and behavioral complexity, was facilitated by the decision to develop it as a functioning ANN element, rather than a less specific theoretical model. The computer simulation results of ACE's operation also facilitated relatively direct comparison with empirical results, and revealed useful behavior that was not originally considered. Some of the behavior which spontaneously emerged related directly to existing empirical results. The best example of this is the direct support of differential inhibitory conditioning without reliance upon conditioned excitation to background stimuli. Other aspects of behavior revealed in the computer simulation results constitute predictions that may be tested by specially conceived animal experiments, and include:

(i) The rapid reduction in the associability of the CS+ from its high level previously established by massed excitatory acquisition, during the early stages of inhibitory acquisition of the CS− using Pavlov's conditioned inhibition paradigm.

(ii) The gradual reduction in the associability of a CS+ during massed reinforced training that is maintained well beyond the point at which the strength of the corresponding CR approaches its asymptotic limit.

(iii) The dependence of changes in the associability of a CS upon paired US presentations during, or subsequent to, previous CS presentations. For example, no change should occur in the associability of a CS− during inhibitory acquisition training, or in that of a CS+ during extinction training, provided they are not followed by reinforced CS presentations within a day of the last training trial.

ACE also exhibits all of the above mentioned advantageous behavior supported by the Rescorla–Wagner model. However, it was decided to concentrate in this thesis upon those aspects of ACE’s behavior not able to be adequately accounted for by other models or theories. While the extent, accuracy, and stability of the consummatory classical conditioning behavior exhibited by ACE
is greater than that of previous neuronal models, what really sets ACE apart is the breadth of the type of behavior it exhibits. In particular, the extent to which timing and memory phenomena are also addressed is highly unusual. That such a wide range of behavior may be simultaneously supported by a single highly localised element suggests that the prevailing tendency to subdivide mechanisms according to apparently distinct types of behavior may not be entirely appropriate. Indeed, the judicious selection of a wide range of behavioral specifications can facilitate development of a unifying set of underlying mechanisms.

FUTURE WORK

The substantial degree of original work embodied within the research described herein has opened up two main avenues for future work. The most obvious direction involves the testing and further development of ACE within a specific real-time environment, and possibly its subsequent fabrication in analog electronic hardware. This would also more completely reveal the extent of ACE's behavioral capabilities, and possibly lead to further mechanistic enhancements.

ACE is the first of many different ACE-like components that will be required to interact in order to produce a completely operational and independent adaptive system, and primarily addresses consummatory classical conditioning behavior. New ACE-like elements, with varying degrees of mechanistic similarity to ACE, also need to be developed to specifically address preparatory classical conditioning, instrumental conditioning, and higher-order behavior. The development of these other elements, and subsequently of a system comprising them, is the other more lateral direction for future research.
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APPENDIX 1: ACE EQUATIONS
Appendix 1: ACE Equations

SIGNALS:

CSTC Equations:

ROC = Rate Of Change input (preset).

\[ \text{CS}[T, N] = \text{Conditioned Stimulus input to channel N at time state T.} \]

NMMM Equations:

\[ \text{EXC}[T, N] = \text{pos}(\text{STM}[T, N]).\text{CST}[T, N] \quad [A1-2] \]
\[ \text{INH}[T, N] = \text{pos}(-\text{STM}[T, N]).\text{CST}[T, N] \quad [A1-3] \]
\[ \text{CCST}[T, N] = \text{CST}[T, N]/(\text{CST}[T, N] + \text{CCSTreg}) \quad [A1-4] \]
\[ \text{EAC}[T, N] = \text{INC}[T].\text{CST}[T, N].\text{ALTM}[T, N] \quad [A1-5] \]

Output Stage Equations:

\[ \text{US}[T] = \text{Unconditioned Stimulus input to the output stage at time state T.} \]
\[ \text{OUT}[T] = \text{pos}(\text{sum}(N=1 \text{ to } n: \text{EXC}[T, N]) - \text{sum}(N=1 \text{ to } n: \text{INH}[T, N])) \quad [A1-6] \]
\[ \text{INC}[T] = \text{pos}(\text{US}[T] - \text{RSTM}[T]) \quad [A1-7] \]
\[ \text{DEC}[T] = \text{OUT}[T] \quad [A1-8] \]

CUMULATIVE QUANTITIES:

CSTC Equations:

\[ - \text{Adep}.\text{pos}(A[T, N] - \text{CS}[T, N]).\text{ROC} \quad [A1-9] \]
\[ C[T+1, N] = C[T, N] \]
\[ D[T+1, N] = D[T, N] + \text{Dchg}.(C[T, N] - D[T, N]).\text{ROC} \quad [A1-12] \]

NMMM Equations:

\[ \text{RGM}[T+1, N] = \text{RGM}[T, N] \]
\[ + \text{RGMacc}.\text{pos}(\text{CCST}[T, N] - \text{RGM}[T, N]) \]
\[ - \text{RGMdep}.\text{pos}(\text{RGM}[T, N] - \text{CCST}[T, N]) \quad [A1-13] \]
Appendix 1: ACE Equations

\[ \text{MGM}[T+1, N] = \text{MGM}[T, N] \]
\[ + \text{MGMacc.pos(RGM}[T, N] - \text{MGM}[T, N]) \]
\[ - \text{GMdep.MGM}[T, N] \quad [A1-14] \]

\[ \text{STM}[T+1, N] = \text{STM}[T, N] \]
\[ + \text{STMacc.INC}[T], \text{CCST}[T, N], \text{ALT}[T, N], \text{RGM}[T, N] \]
\[ - \text{STMdep.OUT}[T], \text{ALT}[T, N] \]
\[ + \text{STMch.g.(LTM}[T, N] - \text{STM}[T, N]) \]
\[ + \text{STMsen.pos(LTM}[T, N] - \text{STM}[T, N]), \text{INC}[T] \quad [A1-15] \]

\[ \text{MTM}[T+1, N] = \text{MTM}[T, N] \]
\[ + \text{MTMacc.pos(STM}[T, N] - \text{LTM}[T, N] - \text{MTM}[T, N]), \text{MGM}[T, N] \]
\[ - \text{MTMdep.pos(STM}[T, N] + \text{LTM}[T, N] - \text{STM}[T, N]), \text{MGM}[T, N] \]
\[ - \text{STMch.g.MTM}[T, N] \quad [A1-16] \]

\[ \text{LTM}[T+1, N] = \text{LTM}[T, N] \]
\[ + \text{LTMacc.pos(STM}[T, N]), \text{MGM}[T, N] \]
\[ - \text{LTMdep.pos(STM}[T, N]), \text{MGM}[T, N] \quad [A1-17] \]

\[ \text{ALTM}[T+1, N] = \text{ALTM}[T, N] \]
\[ + \text{ALTMacc.pos(STM}[T, N]), (\text{ALTmreg} - \text{EAC}[T, N]), \text{EAC}[T, N] \]
\[ - \text{ALTMdep.pos(STM}[T, N]), \text{EAC}[T, N] \quad [A1-18] \]

Output Stage Equations:

\[ \text{RSTM}[T+1] = \text{RSTM}[T] + \text{RSTMacc.(US}[T] - \text{RSTM}[T]) - \text{RSTMdep.RSTM}[T] \quad [A1-19] \]

Where:

- \( \text{pos}(x) = x, \text{if } x \geq 0, \text{ and } \text{pos}(x) = 0, \text{if } x < 0. \)
- Number of CS input channels \( n = 4. \)
- \( T = \text{time state number}. \)
- The interval between successive time states is 10 ms.

And:

\[ \text{STMacc} = 0.032 \quad \text{MTMacc} = 0.0002 \quad \text{LTMacc} = 0.0002 \quad \text{ALTMacc} = 2.0 \]
\[ \text{STMdep} = 0.002 \quad \text{MTMdep} = 0.0001 \quad \text{LTMdep} = 0.0001 \quad \text{ALTMdep} = 2.0 \]
\[ \text{STMch.g} = 0.0001 \quad \text{MTMch.g} = 0.000001 \quad \text{ALTMreg} = 0.5 \]
\[ \text{STMsen} = 0.5 \]
\[ \text{RGMacc} = 0.2 \quad \text{MGMacc} = 0.5 \quad \text{RSTMacc} = 0.30 \quad \text{CCSTreg} = 0.2 \]
\[ \text{RGMdep} = 1.0 \quad \text{MGMdep} = 0.00005 \quad \text{RSTMdep} = 0.01 \]
\[ \text{Aacc} = 0.5 \quad \text{Bacc} = 0.020 \quad \text{Chg} = 2.8 \]
\[ \text{Ad}ep = 0.5 \quad \text{Bdep} = 0.005 \quad \text{Dch}g = 0.4 \]

\( 0.01 \leq \text{ROC} \leq 0.50 \text{ typically}. \)
APPENDIX 2:
SPECTRAL TIMING MODEL EQUATIONS
The following difference equations describe the operation of the Spectral Timing Model developed by Grossberg and Schmajuk (1989). Some symbols have been altered to facilitate comparison between Equations [A2-1], [A2-2], and [A2-3], and those of the CSTC which is developed in Chapter 5 and described by Equations 15-11 to [5-5] (also reproduced in Appendix 1 as Equations [A1-1], [A1-9], [A1-10], [A1-11], and [A1-12]).

**SIGNALS:**

CS[T, N] = Conditioned Stimulus input to channel N at time state T.

US[T] = Unconditioned Stimulus input at time state T.

R[T] = Output signal at time state T.

**Output Signal Equation:**

\[
R[T] = \text{pos}(\sum_{N=1}^{n} F(X[T, N]).Y[T, N].Z[T, N]) - R\text{thr} \tag{A2-1}
\]

**CUMULATIVE QUANTITIES:**

X[T, N] = Cell activity or potential of input channel N at time state T.

Y[T, N] = Depletable neurotransmitter of input channel N at time state T.

Z[T, N] = LTM level of input channel N at time state T.

**Spectral Activation Equation:**

\[
X[T+1, N] = X[T, N] + Xch[g][N].((1-Xacc.X[T, N]).CS[T, N] - Xdep.X[T, N]) \tag{A2-2}
\]

Transmitter Gate Equation:

\[
Y[T+1, N] = Y[T, N] + Yacc.(1-Y[T, N]) - Ydep.F(X[T, N]).Y[T, N] \tag{A2-3}
\]

Associative Learning (LTM) Equation:

\[
Z[T+1, N] = Z[T, N] + Zch[g].F(X[T, N]).Y[T, N].(US[T] - Z[T, N]) \tag{A2-4}
\]

Where:

\[ F(x) = \frac{(x^P)}{(B^P + x^P)}; \quad \text{And } P = 8, \quad E = 0.8 \]

\[ \text{pos}(x) = x, \quad \text{if } x \geq 0. \]

\[ \text{pos}(x) = 0, \quad \text{if } x < 0. \]

Number of CS input channels n = 80 (cf. n = 4 for ACE).

T = time state number.

Interval between successive time states is 1ms (cf. 10ms for ACE).

And:

\[ X\text{acc} = 1.0 \quad Y\text{acc} = 8.0001 \quad Z\text{chg} = 0.01 \quad R\text{thr} = 0.0 \]

\[ X\text{dep} = 1.0 \quad Y\text{dep} = 0.1250 \]

\[ X\text{ch}g = 0.2/i \quad \text{for } i = 1, 2, \ldots 80; \quad (\text{cf. the 4 ROC settings for ACE}). \]
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REFERENCES


*Journal of Comparative and Physiological Psychology, 86,* 74-78.


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