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The Neural Multiprocess Memory Model

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The LEAD AI Project

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Abstract: In an Artificial Neural Network (ANN), associative memory corresponds to the synaptic weighting which modulates the efficacy of each ANN element input and has simple (normally perfect) retention characteristics. This paper details the development and operation of a new multistage memory system, referred to as the Neural Multiprocess Memory Model (NMMM). The NMMM supports spontaneous regression and recovery, U-shaped memory retention, and incorporates a new adaptive associability mechanism capable of supporting 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.

The Learning, Evolution, Adaptation, and Development (LEAD) Artificial Intelligence (AI) Project.

INTRODUCTION

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 may have a large impact upon the characteristics of learning and the temporal characteristics of transfer from acquisition to deployment.

Perhaps the most pervasive artificial form of memory is that utilised in digital computers, which is typically characterised by very fast rates of storage and recall (typically requiring only nanoseconds to microseconds), perfect transfer of digital data, and perfect retention for practically unlimited intervals of time. Having separately identified and categorised the phenomenon of memory in digital computers, criteria have evolved for its operation which are absolutely separate from its potential applications. No information processing other than strictly memory processes such as storage and retrieval of data is permitted, so that each "memory module" makes virtually no nonmemory contribution to the mechanisms which alter memory contents.

While this methodology has been particularly effective at producing general purpose computer memory which is application nonspecific, it may have engendered an overly narrow concept of the possible role of memory in biological neural network systems, particularly among the ANN research community. In biological systems, memory appears to be distributed among many adaptive synapses between neurons in a fine-grained manner (Eccles and McIntyre, 1953; Eccles, 1964; Ungar, 1970; John, 1972), and so the mechanisms of acquisition, retention, and deployment may well be more tightly integrated. In such a case it may not be appropriate to separately idealise the characteristics of memory. In fact, it will be discussed below how the "non-ideal" information processing and integration characteristics of biological memory may actually complement and enhance the learning and deployment processes.

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 (384-322 BC), 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. The phenomenon of retrograde amnesia, in which a traumatic event can prevent memory of immediately preceding events, led Muller and Pilzecker (1900) to suggest in their "consolidation theory" that neural pathways must temporarily remain active after associations are initially formed in order for them to consolidate and become permanent.

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 such as the highly influential Atkinson and Shiffrin (1971, 1977) multiprocess memory model, depicted in Figure 1. Atkinson and Shiffrin 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.

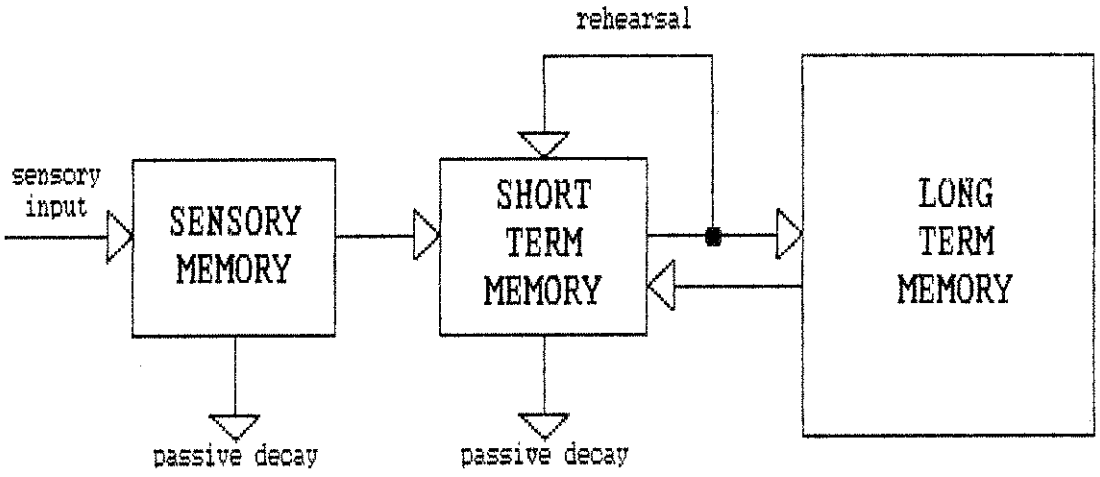


FIGURE 1. The Atkinson and Shiffrin (1971, 1977) multiprocess model of human memory.

In the Atkinson and Shiffrin (1971, 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 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 inadequately understood and specified from a mechanistic viewpoint. 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 STM capacity of between five and nine 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. In this model LTM was assumed to be permanent, and so free of passive decay.

As a generalised model of human memory the Atkinson and Shiffrin (1971, 1977) model is easy to relate to, but it provides virtually none of the mechanistic detail of interest to ANN researchers. However, memory also appears to be a multiphasic process in more humble vertebrates and invertebrates (e.g., Chen, Aranda, and Luco, 1970; McGaugh, 1966, 1969; Menzel, 1984; Menzel, Erber, and Masuhr, 1974; Messenger, 1971; Riege and Cherkin, 1971; Young, 1970; Sanders and Barlow, 1971) in which a mechanistic exploration is facilitated by the reduced complexity of these creatures. Furthermore, in the case of the marine mollusc *Aplysia* learning and memory appears to be supported by a highly localised and relatively complicated synaptic mechanism (Carew, Hawkins, Abrams, and Kandel, 1984; Carew, Hawkins, and Kandel, 1983; Carew, Pinsker, and Kandel, 1972; Carew, Walters, and Kandel, 1981; Hawkins, 1981; Hawkins, Abrams, Carew, and Kandel, 1983; Hawkins, Castellucci, and Kandel, 1981; Kandel and Schwartz, 1982; Pinsker, Kupfermann, Castellucci, and Kandel, 1970; Walters and Byrne, 1983).

As the cellular mechanisms of learning and memory in *Aplysia* slowly yield to experimental analysis, a picture is beginning to emerge in which basic intracellular mechanisms seem to be combined to produce progressively more sophisticated learning behavior (e.g., Hawkins and Kandel, 1984). While the mechanistic detail is still insufficient to permit the development of a fully functioning model of learning and memory in *Aplysia*, the demonstration of basic classical conditioning behavior (e.g., Hawkins, Abrams, Carew, and Kandel, 1983) has encouraged the development of several relatively sophisticated artificial neuronal models of classical conditioning, with varying degrees of correspondence to the known mechanisms in *Aplysia* (e.g., Sutton and Barto, 1981; Klopf, 1987; Gluck and Thompson, 1987; Desmond and Moore, 1988; Card and Moore, 1990; Rogers, 1991). A similar approach is taken here, except that an emphasis is placed upon memory processes rather than learning in general, for which relatively little functional detail is known. Consequently, a combination of empirical results from behavioral animal experiments using various species, and qualitative theoretical argument was used to direct this work.

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 (PRE), and accelerated learning following alternating acquisition/extinction training sessions. The NMMM is designed as a general purpose module for implementation within biologically relevant ANN elements which also incorporate learning mechanisms, temporal eligibility traces, and CS-nonspecific nodal activity equations (e.g., Sutton and Barto, 1981; Gluck and Thompson, 1987; Grossberg and Schmajuk, 1989; Rogers, 1991). Consequently, computer simulation results illustrating the NMMM's full capabilities can only be provided when the NMMM is functioning within a

complete ANN element. While such results are available (Rogers, 1991), this paper focusses upon the characteristics of the NMMM that are independent of the ANN element in which it is integrated. A largely qualitative explanation of the NMMM is therefore provided, though this is supplemented by computer simulation results illustrating the NMMM's spontaneous memory retention characteristics, as these are able to be simulated with the NMMM in isolation.

ASSOCIATIVE ARTIFICIAL NEURAL NETWORK MEMORY

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

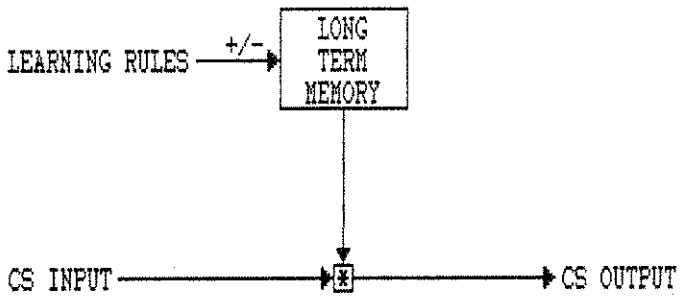


Figure 2. The standard configuration of synaptic memory for ANNs. The operator immediately below LTM is analog multiplication. Individual CS output signals are typically summed at each ANN element. The CS input and CS output signals may be considered to correspond to synaptic input and synaptic output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

When STM is considered in an ANN it is usually implemented by temporarily sustaining the activation of the element so that it slowly passively decays after active inputs terminate. This is sometimes supplemented by reentrant positive feedback connections from the element's output to its inputs (e.g.,

Grossberg, 1988). Such element-based CS-nonspecific STM can help support cooperative-competitive interelement interactions to process parallel streams of input activation. However, this type of STM only stores nonassociative information. Furthermore, since the element activity itself forms the essence of this type of STM, it becomes 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.

The approach adopted here is to develop a multiprocess memory system, incorporating both STM and LTM (and later medium term and adaptive associability memory) to temporally modulate synaptic efficacy. STM and LTM are then distinctly separate to element output or element input activation. This type of synaptic memory system does not appear to have been seriously investigated by other ANN researchers. A combined STM and LTM 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.

SPONTANEOUS REGRESSION AND SPONTANEOUS RECOVERY

Spontaneous regression describes a post-acquisition training partial decline in learned performance (Figure 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.

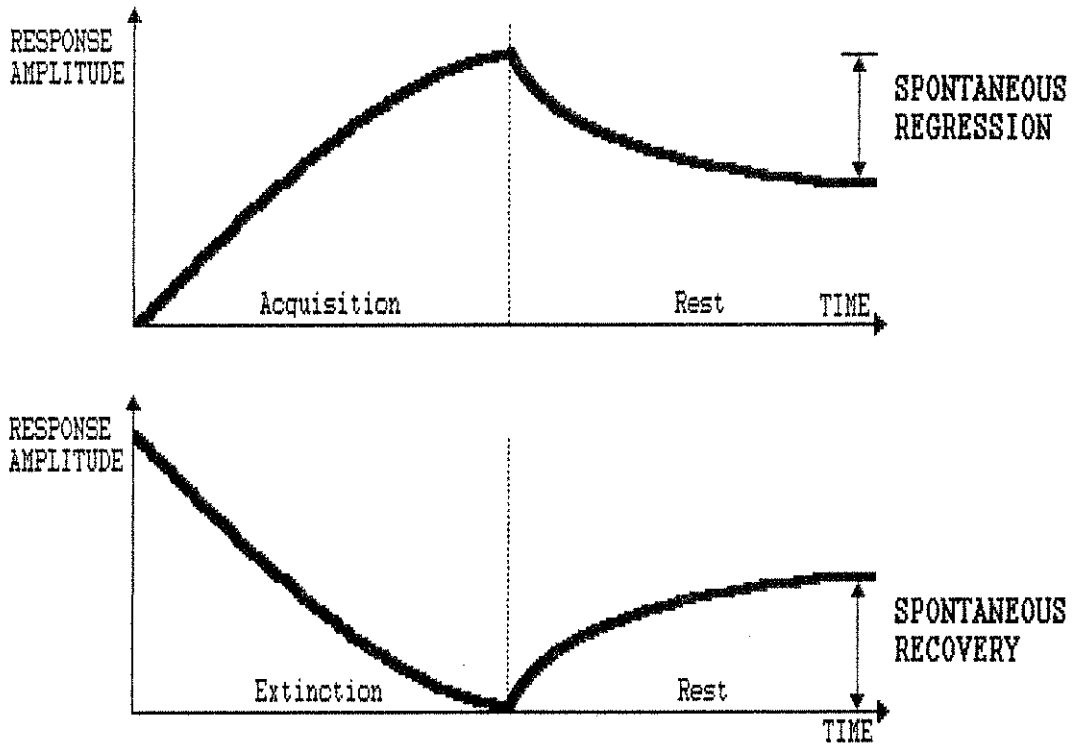


Figure 3. A qualitative illustration of spontaneous regression and spontaneous recovery behavior.

Spontaneous regression and spontaneous recovery behavior have received very little attention from 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 fundamental 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 a system to rapidly adjust its behavior to suit new or changing environmentally imposed contingencies when they are of a consistent nature. This has obvious advantages in situations in which system well-being is threatened. However, if the experienced contingencies are of a statistical nature, as may 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 behavior may temporally combine the 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 (reached sometime after the last trial) being a blend 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 gained the attention of 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 illustrates how associative STM and LTM are arranged within the proposed NMMM. A synaptic (CS-specific) form of STM displaces the synaptic LTM normally used to modulate the efficacy of interelement pathways (Figure 2), while LTM is relegated to a background role in which it is not directly accessible. The impact of LTM upon performance is now only indirectly apparent via its influence upon STM. In turn, LTM value is only indirectly affected by experience via STM. The effect of experience upon STM, through whatever system learning rules are employed, is simulated here by altering the initial value of STM. Since only the spontaneous memory retention and transfer characteristics of the NMMM are able to be simulated with it in

isolation, and experience-induced changes in STM occur at much faster rates, this technique approximately reproduces the response of the NMMM to recent experience.

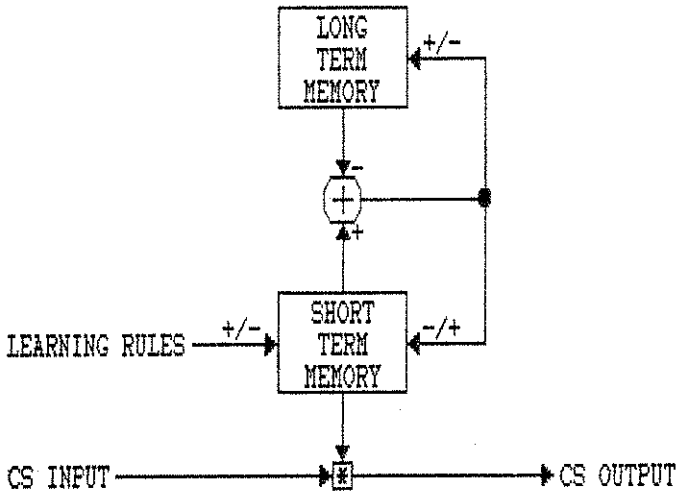


FIGURE 4. The NMMM, integrating associative STM and LTM. STM and LTM may attain positive (excitatory) or negative (inhibitory) values, and all signals are bipolar except for the synaptic input, which is restricted to non-negative values. +/- indicates that the effect of the signal matches its polarity, while -/+ indicates a converse relationship. The operator immediately below STM is analog multiplication, while that immediately above STM is analog summation.

The above NMMM is defined by the following difference equations:

$$STM[T+1] = STM[T] + STMchg.(LTM[T] - STM[T]) \quad [1]$$

$$LTM[T+1] = LTM[T] + LTMacc.pos(STM[T]-LTM[T]) - LTMdep.pos(LTM[T]-STM[T]) \quad [2]$$

Where: STM[T] = synaptic Short Term Memory at time state T.

STM[T+1] = synaptic Short Term Memory at time state T+1.

STMchg = rate of change of STM.

LTM[T] = synaptic Long Term Memory at time state T.

LTM[T+1] = synaptic Long Term Memory at time state T+1.

LTMacc = LTM accumulation rate.

LTMdep = LTM depletion rate.

pos(x) = x, if x >= 0, and pos(x) = 0, if x < 0.

Time between successive time states is 10ms.

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 [1] and [2] therefore describe how the levels of synaptic STM and LTM for the next time state 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 [1] and [2] relate directly to the NMMM schematic diagram (Figure 4), with the addition that a constant is associated with every signal which affects the value of a cumulative quantity. STMchg 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]). LTMacc determines the rate at which LTM accumulates, and LTMdep the rate at which LTM depletes.

This type of nomenclature is standard throughout this paper, with three or four 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 of the variable with which the constant is associated. The standard suffixes "acc", "dep", and "chg" denote that the constant affects the accumulation, depletion, and change (both accumulation and depletion) rates of the associated variable respectively.

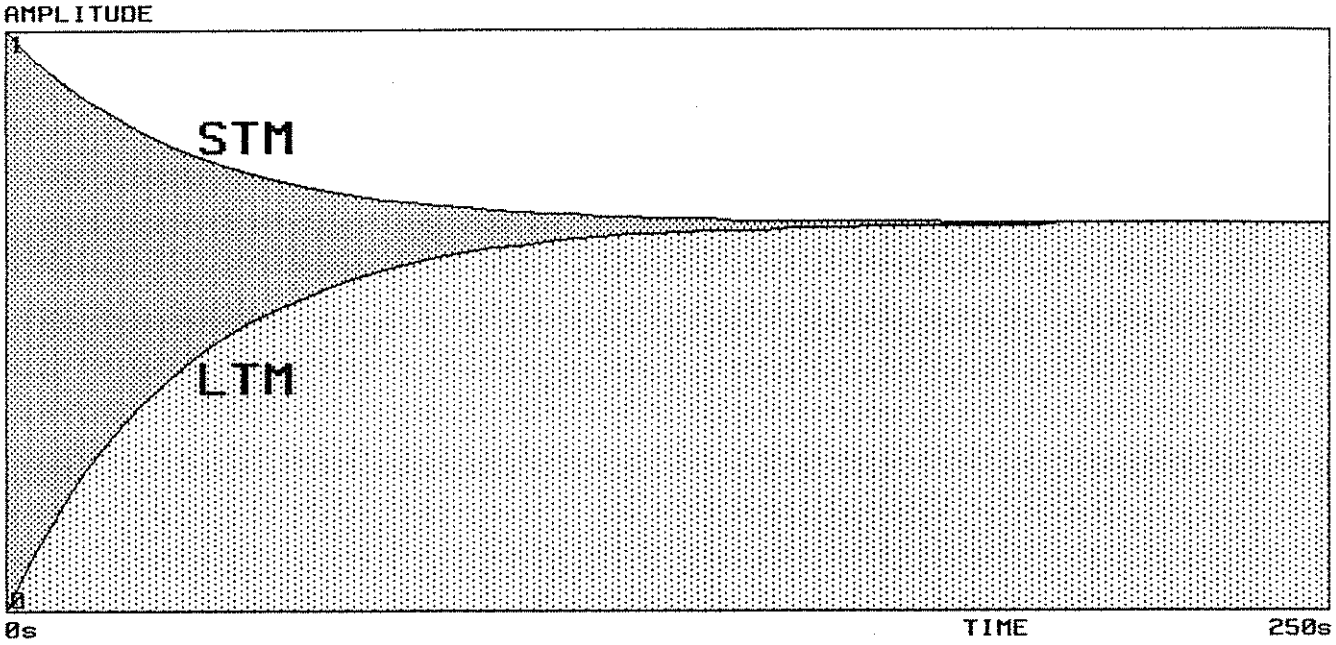
The selection of an interval between successive time states of 10ms is somewhat arbitrary, the main consideration being that it be brief enough to prevent time quantisation effects from becoming apparent. A value much larger than 10ms could have been used to simulate the relatively slow spontaneous interaction between memories in the NMMM, and considerably reduced the number of time states in which the equations needed to be computed. However, as the interval between time states also affects the absolute values of the constants in Equations [1] and [2], and since the NMMM is designed to function as a component of a complete ANN element, the interval between time

states should also be brief enough to support the more rapid internal changes within an ANN element. The value of 10ms is used in the ANN models of classical conditioning of Desmond and Moore (1988) and Rogers (1991), and so was also used here.

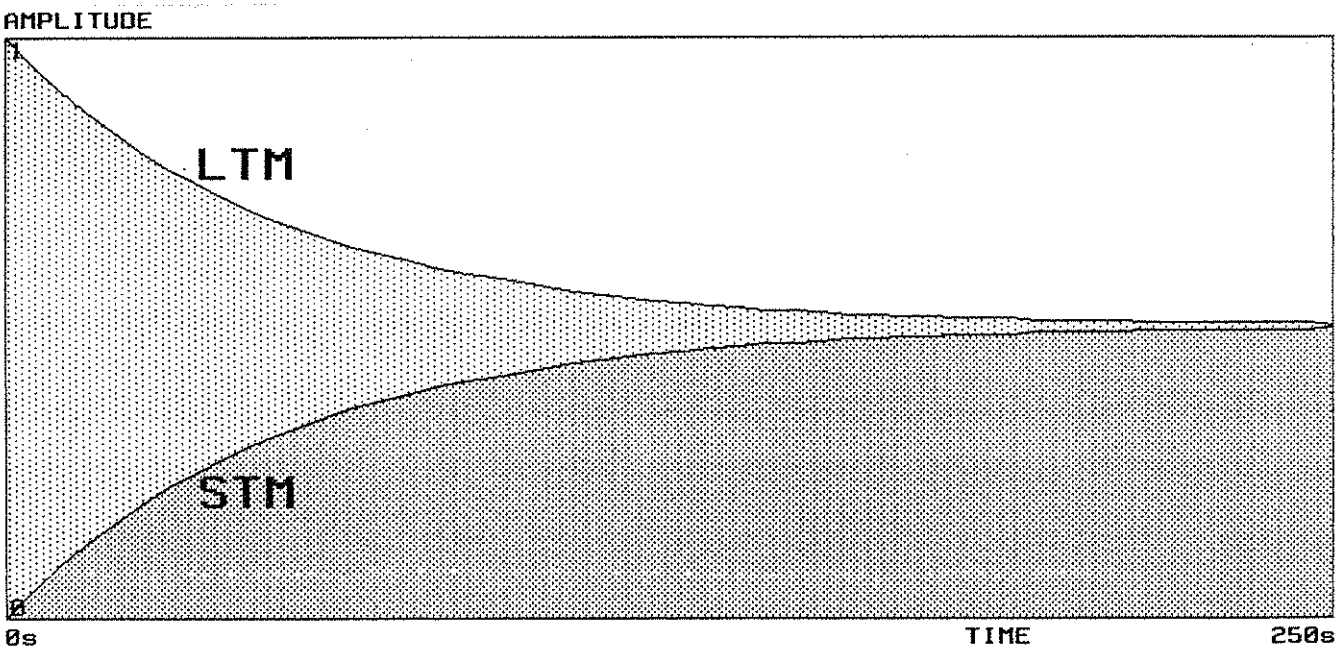
The NMMM illustrated in Figure 4 behaves as follows: After STM is disturbed by a recent experience, STM and LTM interact in a manner which gradually makes them attain the same value, being somewhere in between each of their initial values. The relative rate of change of each memory determines the extent to which each influences the final value. In addition, if different rates of accumulation and depletion are employed (as in Equation [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 by the computer simulation results presented in Figure 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%.

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 (1971, 1977) (Figure 1). In particular, the background role 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 fine-grained operation, the mechanism of interaction between LTM and STM 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 two memory models operate at distinctly different levels, with the Atkinson and Shiffrin (1971, 1977) multiprocess model being essentially *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.



(a)



(b)

FIGURE 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 experience-induced increase in STM from 0.0. (b) Spontaneous recovery behavior. Initially STM = 0.0 and LTM = 1.0, to simulate a rapid experience-induced decline in STM from 1.0. STMchg = 0.0001, LTMacc = 0.0002, and LTMdep = 0.0001, from Equations [1] and [2].

ASSOCIATIVE MTM

The NMMM may be considerably enhanced by incorporating a new adaptive associability mechanism, which is described later below. 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. However, addition of MTM to the NMMM in itself also improves the extent to which the NMMM's memory retention behavior correlates with several additional types of empirical results, as discussed below. Figure 6 illustrates how MTM is integrated within the NMMM.

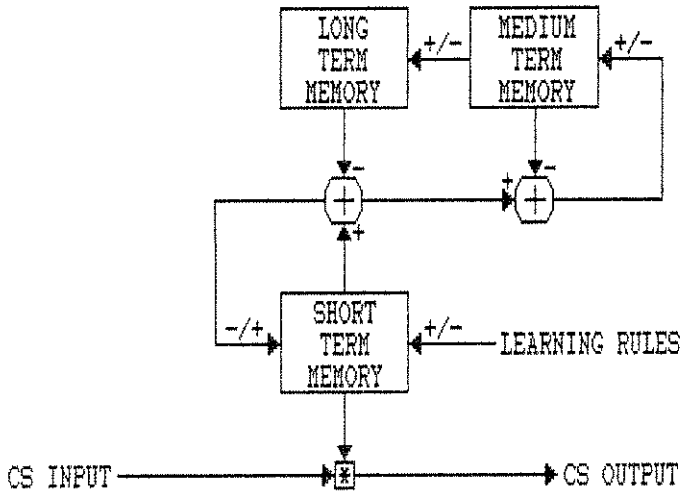


FIGURE 6. MTM-enhanced NMMM, in which changes in LTM are now mediated by changes in MTM.

MTM-enhanced NMMM difference equations:

$$STM[T+1] = STM[T] + STMchg.(LTM[T] - STM[T]) \quad [3]$$

$$MTM[T+1] = MTM[T] + MTMacc.pos(STM[T]-LTM[T]-MTM[T]) - MTMdep.pos(MTM[T]+LTM[T]-STM[T]) \quad [4]$$

$$LTM[T+1] = LTM[T] + LTMacc.pos(MTM[T]) - LTMdep.pos(-MTM[T]) \quad [5]$$

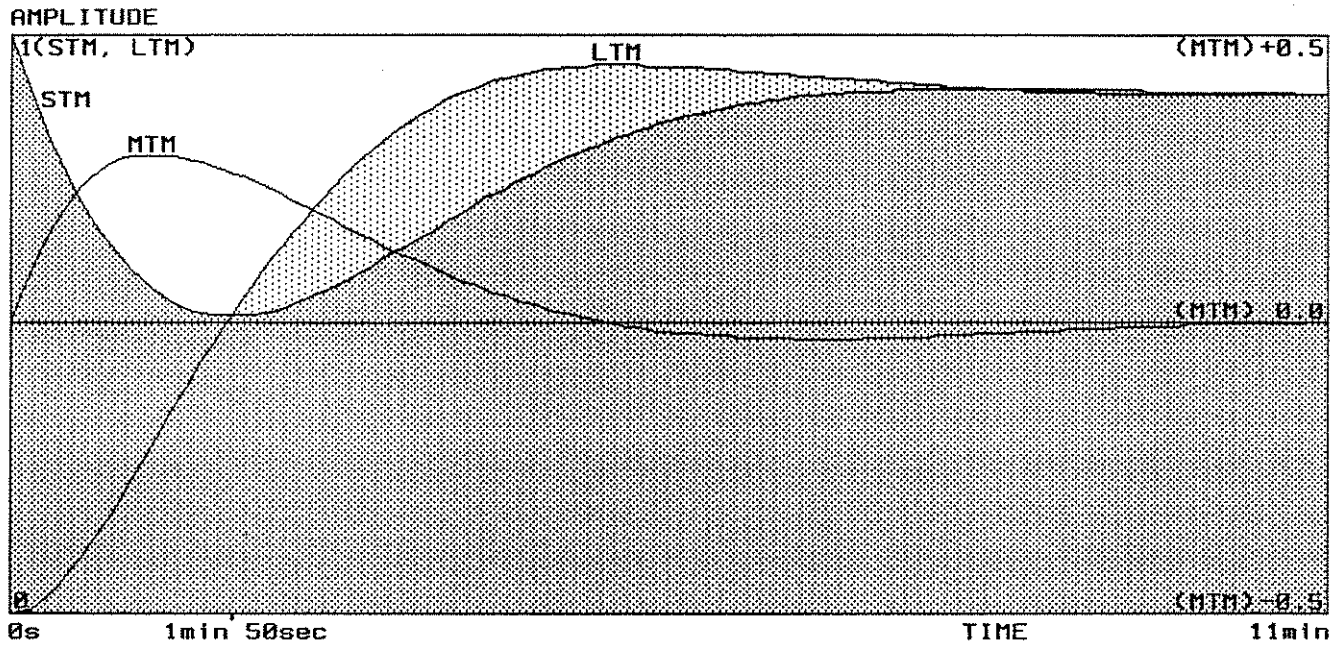
As shown above, the difference between STM and LTM value which used to directly modify LTM, now modifies MTM instead, which in turn drives changes

in LTM. While the basic role of STM and LTM remains unaltered, MTM now requires time to accumulate towards the difference between STM and LTM before LTM can begin to appreciably change. The rate of change of MTM is comparable to that of LTM, so that the effect of relatively rapid learning-rule induced changes in STM upon LTM is delayed.

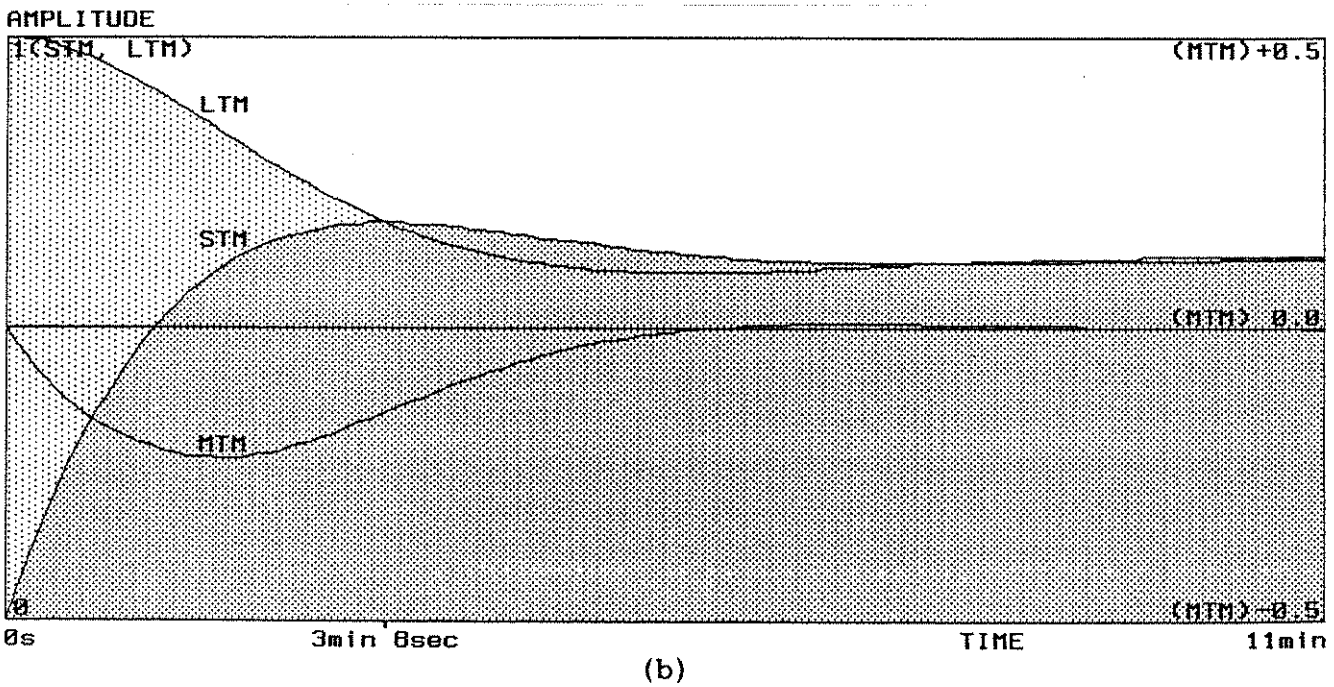
MTM only plays an active role during the transfer of information between STM and LTM. Unlike both STM and LTM, MTM has a steady-state value of zero. Figure 7 illustrates how an experience-induced change in STM value produces a smoothly changing inverted U-shaped deviation in the magnitude of MTM, while STM 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 7a 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 5a). In contrast, the spontaneous recovery illustrated in Figure 7b exhibits only a slight inverted U-shaped response more closely resembling the shape of its counterpart without MTM (Figure 5b), and recovers 62% (compared to 50%).



(a)



(b)

FIGURE 7. Memory retention behavior exhibited by the MTM-enhanced NMMM, 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 [3], [4], and [5].

The U-shaped STM retention of Figure 7a results from the following sequence of events: Immediately after the induced increase in STM from zero to one, LTM is still at its original level of zero. Similarly, MTM has not had sufficient time to react to the difference between STM and LTM, and therefore remains close to its steady state value of zero. STM then starts to gradually decline towards the value of LTM, creating the left half of the U-shaped STM response. However, MTM and LTM also begin to accumulate in this period, so that after approximately 2 minutes, LTM and STM values cross. This corresponds to the point in time at the bottom of the U, and with LTM = STM, would constitute their new steady state values, were it not for the fact that MTM has not had time to deplete back to zero. This causes LTM value to continue to increase and overshoot STM value, 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 7a.

The U-shaped STM behavior of Figure 7a compares very favourably with empirical results (Mercer and Menzel, 1982; Menzel, 1984) depicting the retention in honeybees of excitatory conditioning of color and odor using food reinforcement (Figure 8). As previously described, STM level in the NMMM directly determines the current associative strength of a CS, and therefore current response performance. With due caution in comparing response measurements of a different type, comparison between Figure 7a and Figure 8 indicates that the STM behavior correlates with Menzel's data in the following respects: (a) the position of the bottom of the U, both in time and magnitude; (b) the maximum amplitude attained in the right half of the U; and (c) 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. Alternatively, a gradual passive decay of LTM contents may be responsible, which could easily be added to the NMMM as a slow exponential decay term in Equation [5].

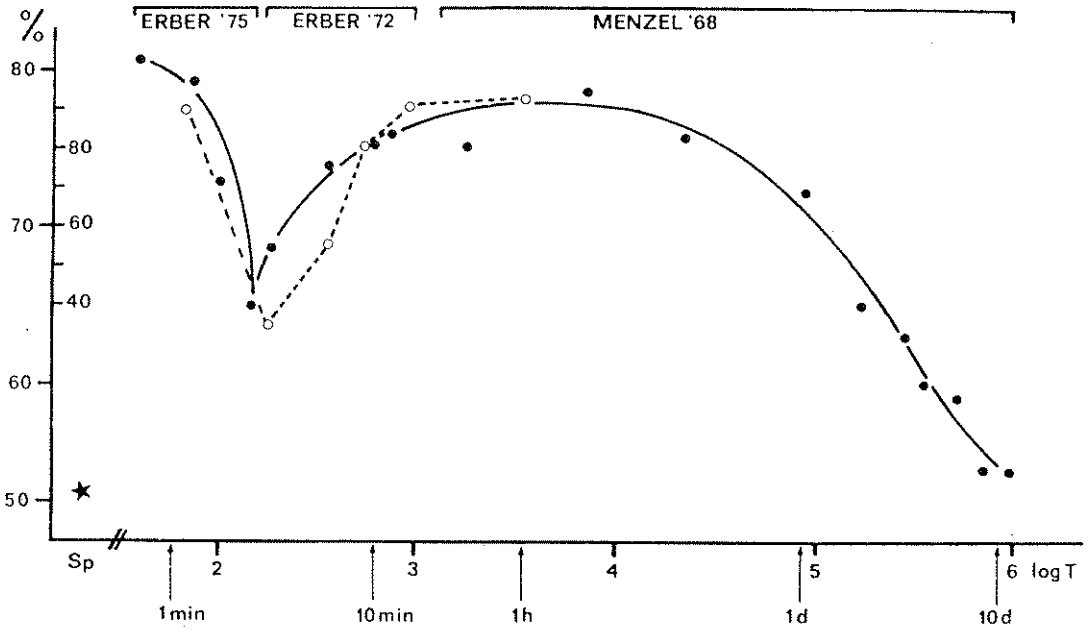


FIGURE 8. Retention of excitatory conditioning of free-flying bees trained to a color (long dashes and closed circles) and fixed bees conditioned to an odor (short dashes and open circles, from Mercer and Menzel, 1982). Note the log scale time axis. The outer ordinate is the percentage of correct choices 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 7a). The difference in time scale for these results may merely result from the different types of response systems being measured.

Addition of MTM into the NMMM may also produce new characteristics which accord with empirical results that were not initially intentionally designed for. The minor STM overshoot depicted in Figure 7a for a single excitatory acquisition trial may be substantially accentuated by massing many excitatory

acquisition trials (i.e. by using a short Inter-Trial Interval (ITI)) to produce a significant 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). The extent of these effects depends upon both the particular values of constants employed in the NMMM equations, and the specific nature of the learning rules which are determined by the ANN element in which the NMMM is deployed.

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)." The possibility exists that such U-shaped retention may be more than just a curiosity, and 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,

depending upon the associability value at the beginning of acquisition training. Computer simulation results to support the above claims are provided in Rogers (1991), when the NMMM is functioning as an integral part of Associative Conditioning Element (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, the temporal characteristics of retention of the level of associability, the precise manner in which associability regulates learning, the conditions controlling its modification, and (if necessary) any additional types of memory required, 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, being specific to every individual CS input.

Effecting Changing Associability

A point of control capable of supporting adaptive associability within the NMMM is the link which enables learning rules to modify STM contents (Figure 9). Using ALTM to directly modulate modification of STM in this way produces a literal implementation of the apparent nature of associability, whereby the ease with which associations are modified is directly controlled.

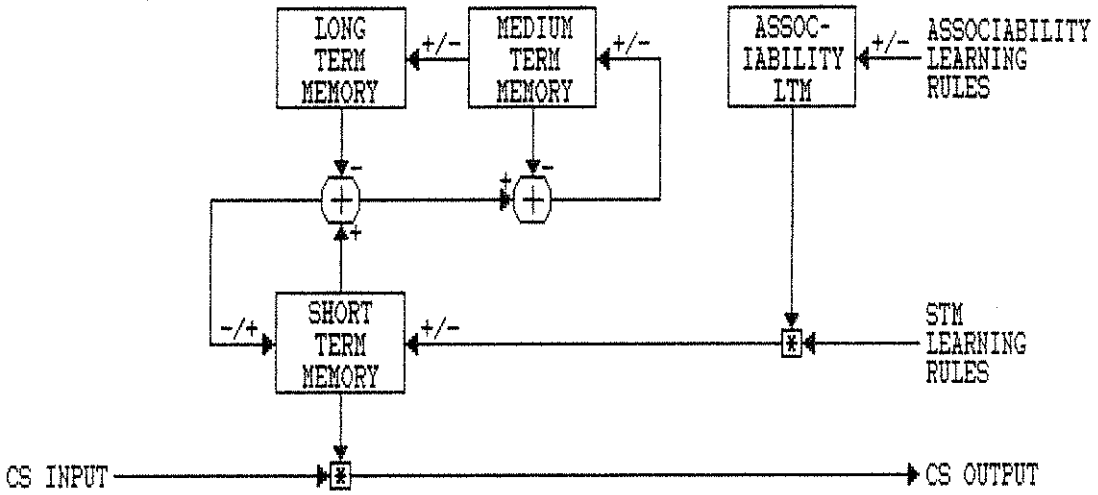


FIGURE 9. The NMMM, now incorporating adaptive associability levels, which are retained in a dedicated Associability Long Term Memory (ALTM). ALTM controls the associability of the CS input by modulating the effect of STM learning rules upon STM contents.

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. The temporal combination of learning rates 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 STM learning rate for the particular environmental contingencies previously experienced. Without such an adaptive associability mechanism, only one STM learning rate could be preset.

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 7) provides a potentially suitable controlling signal for an adaptive associability mechanism that meets the above requirements. As will be discussed below, what is required is a quantity which reflects the type (reinforced or nonreinforced) of trial(s) recently experienced, with the virtual exception of 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 MTM already plays in transferring STM changes to LTM make it almost, but not quite, suitable for retaining a history of recently experienced trials.

As indicated in Figure 7a, 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 LTM or STM 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 zero. This new quantity is called Memory-Gating Short Term Memory, which is hereafter abbreviated to MGM. Figure 10 illustrates how MGM is integrated into the NMMM.

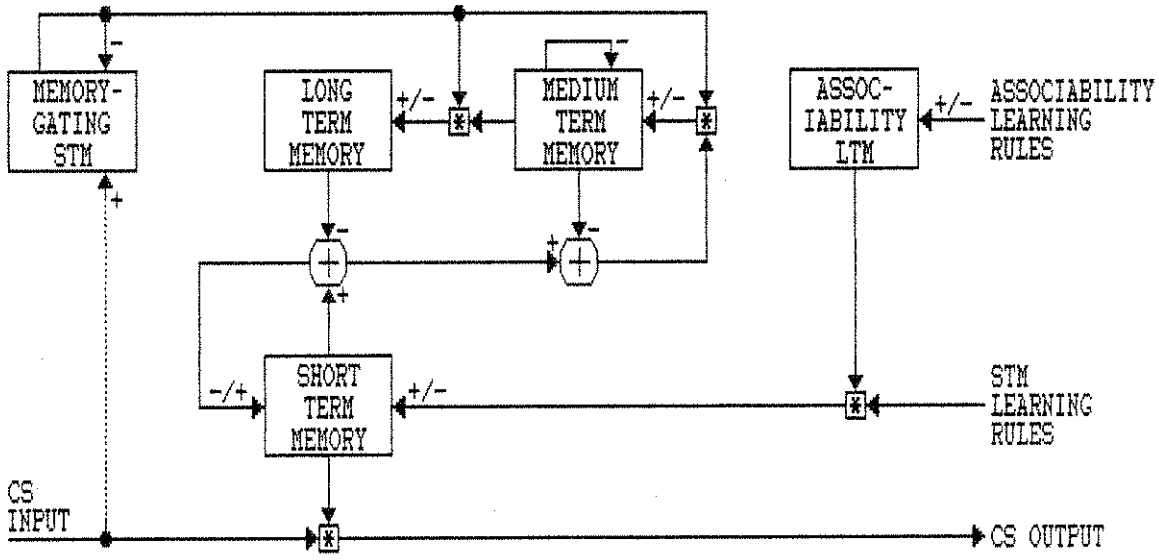


FIGURE 10. The NMMM, now also incorporating Memory-Gating Short Term Memory (MGM) to modify MTM behavior in preparation for its use to produce appropriate changes in ALTM.

Note that in order to counter the effect upon LTM of sustaining MTM contents, it is also necessary to gate changes in LTM using MGM. This prevents sustained non-zero MTM values from producing excessively large changes in LTM value. Also, to prevent MTM from remaining permanently at a non-zero

value as a result of the previous trial, a simple passive decay mechanism is also implemented (Equation [7]) to enable residual MTM levels to gradually dissipate. The rate of MTM decay is determined by the constant MTMchg.

Difference Equations [6], [7], [8], and [9], describe the spontaneous interaction between the NMMM's internal memories, with change in MTM and LTM now gated by MGM. Note that Equation [9] is incomplete in that it does not specify how the CS input increases MGM contents. (A specific example of one suitable technique is provided in Rogers (1991).) It is sufficient for current purposes to assume that CS input activation initially increases MGM contents from zero to one, so that modification of MTM and LTM is enabled (or gated) following CS input activity.

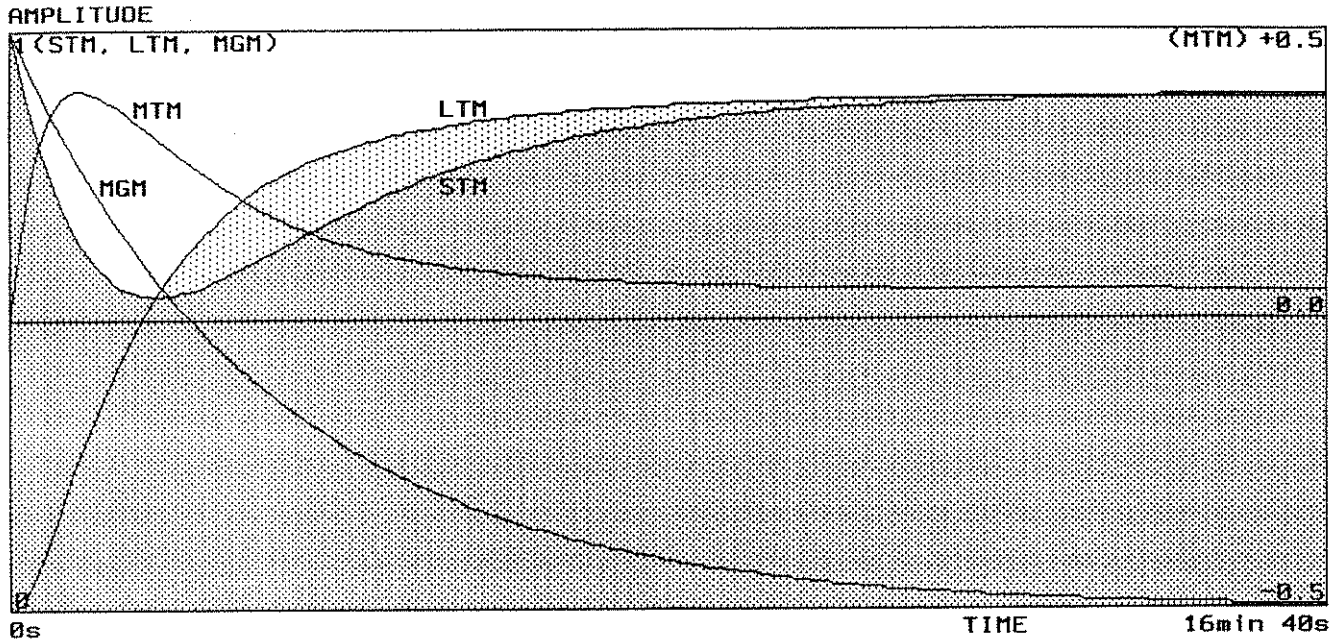
$$STM[T+1] = STM[T] + STMchg.(LTM[T] - STM[T]) \quad [6]$$

$$\begin{aligned} MTM[T+1] = {} & MTM[T] + MTMacc.pos(STM[T]-LTM[T]-MTM[T]).MGM[T] \\ & - MTMdep.pos(MTM[T]+LTM[T]-STM[T]).MGM[T] \\ & - MTMchg.MTM[T] \end{aligned} \quad [7]$$

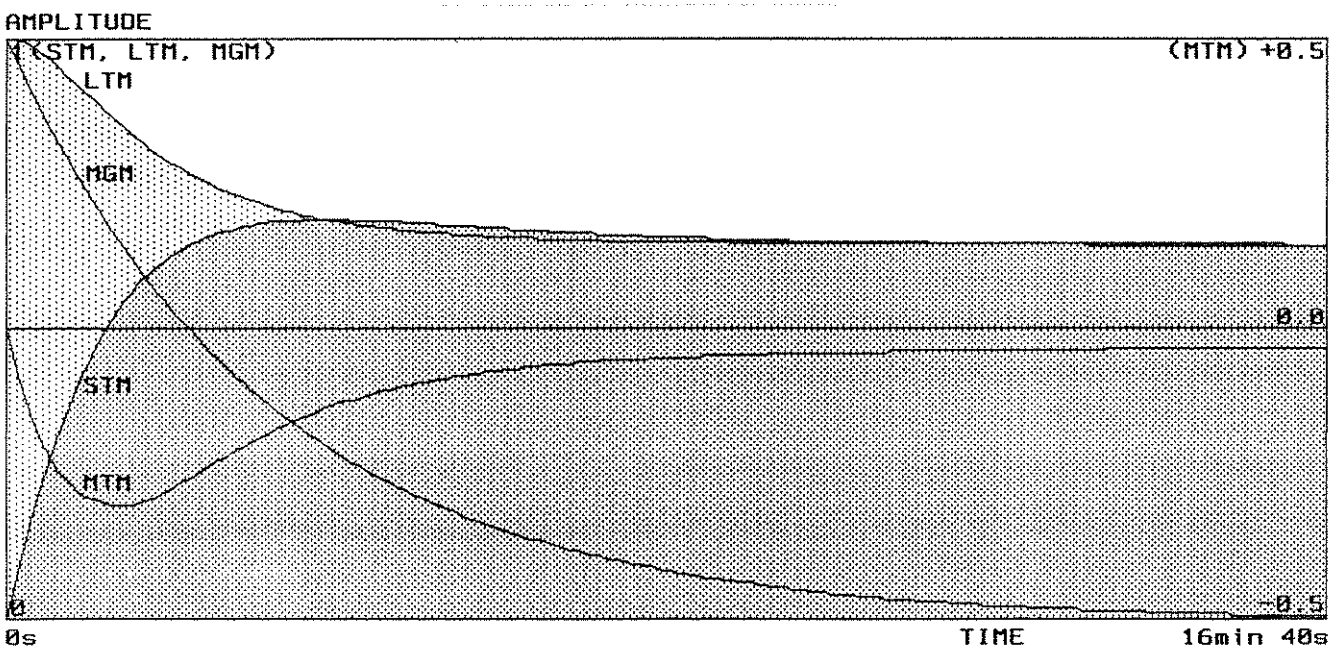
$$\begin{aligned} LTM[T+1] = {} & LTM[T] + LTMacc.pos(MTM[T]).MGM[T] \\ & - LTMdep.pos(-MTM[T]).MGM[T] \end{aligned} \quad [8]$$

$$MGM[T+1] = MGM[T] - MGMdep.MGM[T] \quad [9]$$

As shown in Figure 11a, this new memory configuration is still able to produce memory retention behavior which compares favourably with that of honeybees (Figure 8), when the constants MTMacc and MTMdep are doubled to offset the effect of the decaying MGM value. However, MTM behavior has now been modified so that it consistently maintains a trace of prior reinforcement (Figure 11a) or nonreinforcement (Figure 11b) that is available for hours after the last trial. Also shown in Figure 11 are the MGM traces, which after simulated initial energisation by the CS input, rapidly decay leaving MTM at a significant nonzero value of correct polarity.



(a)



(b)

FIGURE 11. Memory retention behavior exhibited by the MTM-enhanced NMMM, with change in MTM and LTM now gated by MGM. (a) Spontaneous regression behavior. (b) Spontaneous recovery behavior. STMchg = 0.0001, MTMacc = 0.0002, MTMdep = 0.0001, LTMacc = 0.0002, LTMdep = 0.0001, and MGMdep = 0.00005, from Equations [6], [7], [8], and [9].

Although MGM was introduced to make MTM a more suitable source with which to drive changes in adaptive associability, its introduction also has the important effect of isolating STM from both MTM and LTM when the CS input has not been activated recently, which is normally most of the time. This makes STM 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 of Rogers (1991).

Implementing an Adaptive Associability Mechanism

A new adaptive associability mechanism will now be implemented by combining 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 12. As computer simulation results demonstrating the NMMM's operation are only able to be provided when it is functioning within a complete ANN element, all remaining explanation will be restricted to a qualitative discussion. The basic operation and behavioral characteristics of the NMMM's adaptive associability mechanism may be revealed by considering in turn two types of reinforcement schedules that require the acquisition of extreme associability values for optimum learning behavior.

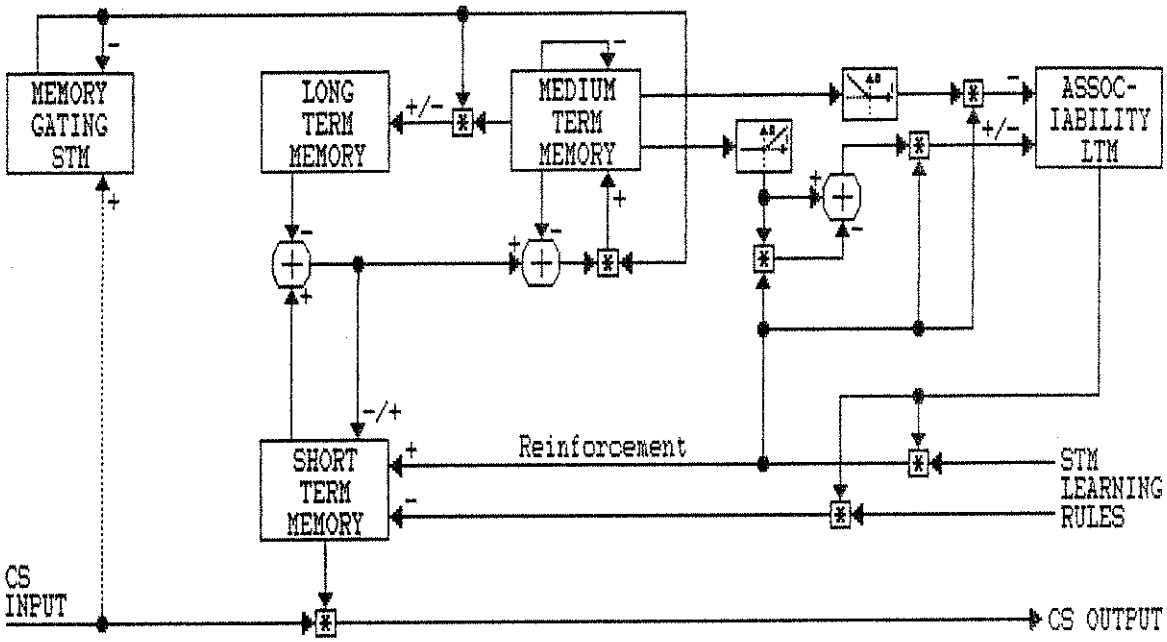


FIGURE 12. The NMMM now utilising MTM to produce behaviorally appropriate adaptation of ALTM. The bipolar STM learning rule signal is now separated into two non-negative signals, with only that which increases STM (corresponding to the effect of reinforcement) gating changes in ALTM. The two new operators towards the upper right corner split the bipolar MTM signal into two 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. The effect of positive MTM values upon ALTM is further regulated to limit the maximum attainable ALTM value.

Consider first the particular 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 R R N N N N N N N N R R R R R R R R R R R R N N N N ...

Under these circumstances, the current type of trial (N or R) indicates with high reliability that the next trial will be of the same type, and the most

appropriate learning rate would be a relatively 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 tend to attain large positive values, by the same mechanism that increases MTM following a single R trial (the effects of which are simulated in Figure 11a). Furthermore, as indicated in Figure 12, 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 an appropriately high level of associability.

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 11b). 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 that 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 11), this effect can be minimised by inserting an additional delay between transitions from extinction to acquisition sessions. In any case, even if no such additional delay is employed, this negative effect upon ALTM level will be insufficient to defeat the positive effect of the acquisition phases because of the relatively large number of consecutive R trials. Consequently, 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 intertrial interval within each session, and the interval between extinction and acquisition sessions (Capaldi, Leonard, and Ksir, 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.

If positive MTM values were simply gated by reinforcement to produce increases in ALTM, then ALTM value would increase during each acquisition session of every acquisition-extinction pair of sessions without ever reaching a maximum limit. Moreover, increases in ALTM enable larger increases in STM, which produce larger MTM values, and which in turn enable progressively larger increases in ALTM. Consequently, a special effort needs to be made to control the positive feedback relationship that exists between increases in ALTM, and increases in STM.

As illustrated above in Figure 12, a special negative feedback loop reduces the extent of the increase in ALTM as the reinforcement signal (which is modulated by ALTM) becomes increasingly larger, when MTM value is positive. While it is simpler to directly use the ALTM value to limit its own growth, the observation that a stronger US increases resistance to extinction (i.e., decreases ALTM) of conditioned suppression after all subjects have reached asymptotic levels of suppression in acquisition (Annau and Kamin, 1961) suggests that the reinforcement signal should be used instead.

When the reinforcement signal is of unity amplitude, no additional increase in ALTM is possible. If the reinforcement signal exceeds unity amplitude, a

negative increase (i.e. a decrease) in ALTM actually results, because the signal which normally acts to increase ALTM now attains a negative value. That this condition is permitted is indicated in Figure 12 by the "+/-" 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, even if arbitrary changes in the effectiveness of reinforcement occur. 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.

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 N R N R N R R R R N N N R N N R R N R R N N R R N R R R N 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 and/or probability being some monotonically increasing function of the 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 previous trial), the adaptive associability mechanism depicted in Figure 12 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 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 (Mackintosh, 1974, pp. 434-467). 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. The more specific observation that for relatively short intertrial intervals (< 20 minutes) N trials only increase resistance to extinction when they are followed by an R trial (Grosslight and Radlow, 1956, 1957; Spivey and Hess, 1968; Mackintosh and Little, 1970; Capaldi and Kassover, 1970) is also supportive of the specific adaptive associability mechanism employed here.

Psychologists interested in the construction of formal models of conditioning sometimes examine the course of acquisition in the expectation that its shape will provide some clue as to its underlying mechanisms. Researchers often attempt to model either a negatively accelerated acquisition curve shape (e.g., Rescorla and Wagner, 1972; Barto and Sutton, 1985), or a sigmoidal curve, with an initial acceleration phase followed by deceleration to an asymptotic maximum level of performance (e.g., Klopf, 1987). However, it is not uncommon for some individual subjects within the same experimental procedure to exhibit sigmoidal acquisition while other individuals do not (Spence, 1956, p. 60). Furthermore, 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 suggest that previous experience is capable of determining the nature of subsequent acquisition, and not just its extent or rate of change. While these aspects are beginning to be addressed by computational models (e.g., Kehoe, 1988) the NMMM addresses them in a new and relatively comprehensive way.

It was discussed above how ALTM may be progressively increased by successive reinforced trials during an acquisition session. If the NMMM is used in combination with learning rules which by themselves normally produce a negatively accelerated acquisition curve (e.g., those based upon the Rescorla-Wagner model (Rescorla and Wagner, 1972)) then the increase in ALTM can be sufficient to produce an initial positive phase of accelerated acquisition, when the initial ALTM level is low (Rogers, 1991). If ALTM is already at its maximum attainable level at the beginning of acquisition, then acquisition proceeds at a fast rate from the very first trial, and the normally expected negatively accelerated acquisition curve is produced. Hence, the new adaptive associability mechanism within the NMMM is also capable of supporting the production of either sigmoidal or negatively accelerated acquisition curves, depending upon the initial associability value.

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), 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; Halgren, 1974; Baker and Mackintosh, 1977). Nevertheless, this effect is still usually referred to as latent inhibition.

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).

If it is assumed that a naive CS may initially produce some excitatory output, as a result of a mildly excitatory initial STM value, then repeated presentation of the CS will result in extinction of its weak excitatory strength. This will reduce its STM level towards zero, and make its MTM value significantly negative. Figure 11b 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 will enable 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, except that the extent of the decrease in ALTM is smaller because of the weaker excitatory strength of the untrained CS. Even though these reductions in ALTM may be small in absolute terms, their effect can be considerable because 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 the NMMM 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. The ALTM level of a CS remains fixed throughout inhibitory acquisition for both Pavlovian and differential conditioned inhibition training procedures because of the absence of temporally contiguous US presentations (Rogers, 1991). 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, 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 MTM over the medium term, may also help account for the confusing evidence regarding short term latent inhibition effects (Mackintosh, 1983, p. 229).

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 two during subsequent conditioning".

Learned irrelevance may be supported by the NMMM 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. 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. Also, the associative aspect of the learned irrelevance effect supported by the NMMM, which results from the associative nature of ALTM, is consistent with Mackintosh's (1973, 1974) observations that the effect is specific to both the CS and the US.

Finally, as suggested by the empirical results from Kremer (1971), latent inhibition and learned irrelevance do not just alter the rate of subsequent acquisition, they also alter the shape of its course. It was discussed above that as the initial ALTM value at the beginning of acquisition is reduced, the shape of ACE's resulting acquisition curve changes from negatively accelerating only to sigmoidal, in accord with Kremer's results. Thus, the NMMM supports both the decline in rate, and the change in shape, of acquisition subsequent to latent inhibition and learned irrelevance procedures.

CONCLUSION

Researchers in the ANN community generally regard associative (or synaptic) memory as merely a repository for the efficacy of each neural input that makes no other active contribution to learning or performance. However, the emerging picture of a complex system of chemical interactions functioning in support of synaptic memory in relatively humble creatures (such as *Aplysia*), and the widespread occurrence of apparently nonideal memory phenomena such as spontaneous recovery and regression in behavioral learning experiments, suggested that a reevaluation of the potential role of associative memory might be profitable. More specifically, the intention of the research documented herein was to explore the extent to which a more sophisticated multistage associative memory system could contribute to the appropriate modulation of the impact experience has upon memory, and its subsequent transfer into altered performance. The result is a computational model of associative memory, referred to as the Neural Multiprocess Memory Model (NMMM). The NMMM has been developed as a general purpose memory module for use in complete ANN elements, where it may replace the standard associative LTM normally employed, and contribute the following utilitarian behavior; spontaneous regression and recovery, U-shaped memory retention, 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.

While this paper focussed upon the NMMM as an isolated module and provided computer simulation results of its spontaneous memory retention behavior, much of the NMMM's more elaborate behavior could only be discussed here because of its dependence upon the learning rules employed within its host ANN element. However, computer simulation results in support of all of the above behavioral claims are available in Rogers (1991), in which the NMMM is deployed within the new Associative Conditioning Element.

The NMMM incorporates synaptic STM 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 the way in which these two memory types interact within the NMMM. The separation between short and long term experience offered by STM 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.

The new adaptive associability mechanism developed within the NMMM also supports preconditioning procedures in which prior nonreinforced CS presentations (in the case of latent inhibition) or noncontingent CS and US presentations (in the case of learned irrelevance) retard subsequent associative conditioning training between the CS and the US. Furthermore, the specific behavioral characteristics of the reduced associability resulting from these procedures also compares very favorably with empirical results.

In conclusion, the NMMM is a psychological multiprocess model of associative memory that unifies a considerable range of empirically observed memory phenomena, and it is also a functioning computational system capable of contributing this additional utilitarian behavior to the ANN element in which it is deployed.

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