The Effect of Temporal Predictability on Habituation: Empirical Studies and
Connectionist Models

by

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ABSTRACT

This thesis reviews existing models of habituation of the orienting response (OR) and presents four experiments designed to clarify contentious issues in the existing literature. A new connectionist model of habituation is developed and its performance in various experimental situations simulated.

Models created to account for habituation can be classified into two types:

1) Comparator models in which the subject compares an internal trace of past stimulation with current stimulation and the orienting response is an index of the disparity between the two.

2) The dual-process model in which habituation is the result of two independent processes of inferred habituation and inferred sensitisation acting within the stimulus-response (S-R) pathway.

A major difference between the two types of model is that comparator models of habituation (Öhman, 1979; Sokolov, 1960; Wagner, 1981) postulate that the internal trace encodes extrapolatory or temporally predictive information about the stimulus series, while the dual-process model (Groves & Thompson, 1970) does not suppose that temporal information is encoded. Both the elicitation of a response by stimulus omission, and the effect of ISI variability on the rate of habituation, are vital indicators of the merit of each approach. Only if both phenomena are demonstrated to exist must a valid habituation model incorporate a mechanism of temporal encoding.

Prior evidence for a response to stimulus omission and an effect of ISI variability on rate of habituation was inconclusive. Four experiments were conducted to examine these and related phenomena in habituation of the skin conductance response (SCR). It was concluded, from the results of these experiments, that both response to stimulus omission and an effect of ISI

variability on the course of habituation had a significant effect at short ISIs (1-2s) but no significant effect was at longer ISIs (>10s).

A model of OR habituation was devised that was capable of incorporating the new results as well as other published results. A neural network or connectionist modelling framework was chosen for this purpose, for its quantitative nature, ease of simulation and neurobiological plausibility. Five different connectionist models were constructed and simulations were performed to assess the performance of each model in experiments testing various temporal phenomena. It was concluded that a model incorporating delta-rule learning of physical features plus learning of temporal relationships by the learning of the interval between stimuli could most satisfactorily simulate the observed empirical results. The connectionist models and their performance in experimental simulations were related to traditional models of habituation.

ACKNOWLEDGMENTS

CONTENTS

LIST OF FIGURES

CHAPTER 1

Habituation of the Orienting Response

1.1 Definitions

Much of the initial work describing and defining the properties of the orienting
response (OR) was performed by Soviet physiologists and psychologists, with
Pavlov (1927, cited in Graham, 1973) being the first to use the term, and
Sokolov (1960, 1963a, 1963b, 1966, 1969, 1975, 1989) formulating one of the most
influential current theories of its elicitation and habituation.

A suitable place to start the description of the orienting response is with
Sokolov's (1975) definition:

> The orienting reflex is characterised by a low threshold, extensive
> generalisation of the excitatory process, absence of any specific
> reflexogenic zone, uniform responses to the stimulus when it is switched
> on or switched off, and development of extinction which proves to be
> selective with respect to the parameters of the repeated stimulus. (p. 217)

An OR is a particular type of unconditioned response (UR), that is a
physiological reflex elicited naturally by an external stimulus, rather than a
conditioned response which may be paired with a stimulus which would not
normally elicit that response.   Not all unconditioned responses can be classified
as orienting responses and there has been much work on differentiating
orienting responses from both defensive responses (DR) and startle responses
(SR). Graham (1979) asserts that the four crucial criteria for distinguishing the
orienting and defense reflexes are that "(1) Offset should elicit an OR and not a
DR; (2) high and low intensities of stimulation should evoke different reflexes;
(3) rates of habituation of OR and DR should differ ; (4) an habituated OR but
not a DR should recover with a stimulus change that does not increase stimulus
intensity" (p. 138). Turpin (1983) writes that an OR is characterised by
non-specificity in regard to the intensity of the stimulus and selectivity of

extinction of various properties of the stimulus with repeated presentations while a DR is characterised by elicitation only by high intensity stimuli. Physiological changes accompanying an OR include an increase in skin conductance with decreases in heart rate, digital pulse amplitude and cephalic blood content indices. A reasonable conclusion is therefore that changes in skin conductance (the dependent measure used in this series of experiments) elicited by stimuli of moderate intensity are an index of an orienting response rather than a defense or startle response.

Maltzman and colleagues (Maltzman, 1979; Maltzman & Pendery, 1988) distinguish between voluntary and involuntary ORs, each elicited by different processes within the subject. Involuntary ORs are, like those described above, elicited by stimulus change and are the subject of the theories developed in this thesis. Voluntary ORs are however elicited when signal value is given to the stimulus, for example by instruction or classical conditioning, or by other problem solving processes used by the subject. This thesis will however be largely concerned with involuntary ORs with physical stimulus changes being used to elicit ORs.

Habituation is a widespread behavioural phenomenon which is most simply defined as the decrement of response to a stimulus with repeated stimulus presentations. More specifically, response habituation is defined as a response decrement which occurs in the absence of receptor adaptation, effector fatigue or infringement of neuronal refractory periods (Siddle & Packer, 1987). Thompson and Spencer (1966) list nine properties necessary to define habituation: a) repeated stimulation results in a (normally negative exponential) decrease in response, b) spontaneous recovery of response after the stimulation is stopped, c) long term effects – repeated habituation series cause greater habituation, d) habituation is directly proportional to stimulus frequency, e) habituation is inversely proportional to stimulus intensity, f) below-zero

habituation may occur (i.e., further stimulus presentations following response habituation will result in slower recovery), g) habituation may exhibit stimulus generalisation, g) presentation of another (usually strong) stimulus results in recovery of the habituated response (dishabituation), h) the effect of dishabituation itself habituates if it is repeated. These definitions of habituation apply not only to the orienting response but also many other aspects of behaviour.

1.2 Theories of Elicitation and Habituation of the OR

The basic requirement of a valid theory of habituation and elicitation of the OR is provision of a method by which the repeated presentation of a stimulus results in a decrement in the magnitude of the orienting response, that is, a mechanism by which the orienting response habituates. All the theories to be discussed fulfil this requirement but differ in the mechanism by which habituation is implemented and in the predicted performance of the model under various patterns of stimulation.

Theories of habituation can be basically divided into two general types, comparator theories and non-comparator theories. Comparator theories including those of Sokolov (1960), Wagner (1976, 1978) and Öhman (1979) postulate that the elicitation of an OR follows the comparison of afferent stimulation with an internal trace of past stimulation. The magnitude of the orienting response is then directly related to the degree of disparity between the internal trace and afferent stimulation. Non-comparator theories, notably the dual process theory of Thompson and colleagues (Groves & Thompson, 1970; Thompson, Berry, Rinaldi, & Berger, 1979), do not postulate a specific trace of previous stimulation but rather suggest that the behavioural outcome of repeated stimulation is the result of the interaction of the independent processes of inferred habituation and inferred sensitisation within the stimulus-response pathway.

1.2.1 Sokolov's Comparator Theory

E.N. Sokolov has been possibly the most influential theorist in the area of

habituation of the orienting response, publishing prolifically over a period of

almost 30 years (Sokolov, 1960, 1963a, 1963b, 1966, 1969, 1975, 1989; Graham,

1989). This excerpt taken from his 1989 paper succinctly states the essence of

his comparator model of habituation:

> In discussing the selective nature of habituation of the orienting response
> (OR), we can make use of an abstract parameter representing the
> independent variables, and a corresponding abstract measure of the
> response. After habituation of responses to a specific stimulus, a
> presentation of a stimulus characterized by those specific parameters
> evokes no response. But we get responses to deviations from that
> stimulus. . . . We can say that during repeated presentations of the
> stimulus, the nervous system is elaborating a selective filter which is a
> "band-reject" filter, meaning that the specific stimulus is not passing
> through the filter to the mechanism triggering the response. . . . The next
> step in the study of selective habituation is to use two-dimensional
> combinations of parameters, e.g., intensity and frequency. Using a
> specific combination of these parameters, and a specific response
> measure, we can find also a selective filter for the combination of
> parameters. If we change the intensity or the frequency we get a
> response. Finally, we can also include time in the stimulus. The three
> parameters define a three-dimensional space, in which a particular
> habituating stimulus is represented by a point of no response which is
> surrounded by a response-eliciting area. The distance of a test stimulus
> from the standard point influences the magnitude of the response. We
> can think then of surfaces in three dimensional space which represent
> equal responses. Thus a repeatedly-presented stimulus results in a
> multidimensional model of the stimulus in the brain. Such a
> multidimensional trace, established in the nervous system during
> repeated stimulus presentations, can be called a *stimulus neuronal model*.
> The stimulus neuronal model differs from the sensory image in not being
> represented subjectively. At the same time, it is a very precise replica of
> the repeatedly presented stimulus. The mismatch between the neuronal
> model and a novel stimulus is a triggering factor of the OR. (p. 143).

Sokolov's comparator model of habituation thus states the elicitation of an OR is dependent upon the disparity between the afferent stimulation and an internal neuronal model constructed during previous stimulation. The function of the OR is to facilitate the extraction of information from the environment through increases in the activity of 'analyser' systems including the visual system (Sokolov, 1963, 1975; Spinks & Siddle, 1983) and an increase in the signal-noise ratio of neurons for which the information is important (Sokolov, 1969).

The early formulations of the comparator model of habituation by Sokolov included two distinct units, the neuronal model described above, and an amplifying system. The two units were connected as shown in Figure 1.1.
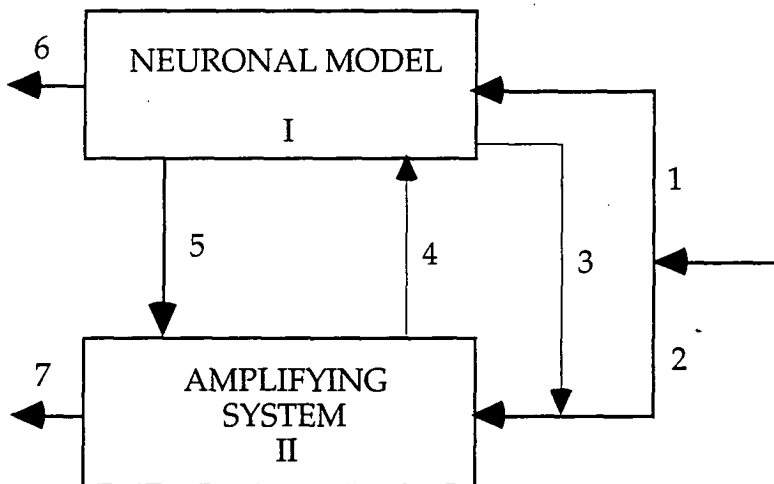
Figure 1.1. Comparator model of elicitation of the orienting reflex. I. Neuronal model of past stimulation. II. Amplifying System. The sensory input is transmitted via specific pathways (1) to the cortex where a neuronal model of the stimulus is built up. The afferent stimulation also activates the reticular formation via collateral pathways (2). when the stimulus is novel, the stimulation will not match the characteristics of the current neuronal model, and excitatory impulses reach the reticular formation via cortico-reticular pathways (5). Activation of the reticular formation in this manner elicits the

autonomic and somatic components of the OR via the specific efferent pathways (7), while specific responses are transmitted via the specific pathways (6). At the same time, the reticular formation has an activating influence on the discriminatory power of the modelling system via pathway (4). Stimulus repetition leads to the formation of a neuronal model in the cortex. The similarity of afferent stimulation and this neuronal model then produces a negative feedback via pathway (3) onto the afferent collaterals to the reticular formation. This blocks impulses to the reticular formation and inhibits the OR. (Stephenson & Siddle, 1983 p.190, originally adapted from Sokolov, 1960).

Later formulations of the model (e.g., Sokolov, 1975, 1989) include some changes to the brain structures and connections postulated to be involved in the habituation process but maintain the basic notion that habituation of the OR occurs because the elicitation of the responses constituting an OR is modulated by connections from neuronal model or filter which, over a number of presentations, encodes the dimensions of the afferent stimulation. As the activity encoded in the neuronal model increasingly more accurately encodes the afferent stimulus/stimuli the inhibition of the amplifying/response generating systems increases, thus reducing the magnitude of the OR elicited.

A feature of the comparator model as proposed by Sokolov is that the habituation and subsequent recovery of the OR when the stimulus is changed can only occur with respect to stimulus dimensions encoded by the neuronal model of a particular subject. A trivial example of this argument would be to imagine a blindfolded subject presented with a complex stimulus consisting of a tone accompanied by a flash of light. After a number of presentations of the stimulus the subject would, because the neuronal model had encoded the stimulus and was inhibiting the response generating systems, habituate and no OR would be elicited by the stimulus. The experimenter now changes the stimulus in one of two dimensions, alterations of the pitch or intensity of the

tone causes an OR to again be elicited while alteration of the colour or brightness of the light stimulus elicits no OR. Not surprisingly the experimenter concludes that pitch and intensity of the tone were dimensions encoded by the neuronal model of this subject, and thus compared to afferent stimuli when they were presented, while colour and light intensity were not being encoded by this particular subject. In this example it is obvious that the reason why colour and light intensity were not encoded was because the subject was physically prevented from perceiving these dimensions of the compound stimulus. In other experiments however it is possible that while the subject is presumably physically able to perceive particular stimulus dimensions, alterations of the stimulus along these dimensions does not result in recovery of the OR, suggesting the dimension was not being encoded by the neuronal model. The aim of the experimental research presented in this thesis can be seen as an attempt to use this type of paradigm in an attempt to further specify both the extent and mechanism of encoding of various (in particular temporal) stimulus dimensions by the neuronal model (or alternative structures included in alternative models).

The neuronal model proposed by Sokolov encodes (at least) the stimulus dimensions of stimulus intensity, stimulus duration, tone pitch, stimulus modality, interstimulus interval (ISI, the time interval between successive stimuli), colour, location (Sokolov, 1969) and the temporal order of successive stimuli (Sokolov, 1975). Sokolov (1969) further stresses that the neuronal model is not static but rather "extrapolatory" in nature, with the neuronal model predicting not only the physical dimensions of the stimulus but also the time at which it will be presented. It is also likely, although not explicitly stated by Sokolov in the papers referenced, that the neuronal model declines or decays over time if a stimulus is not presented to it, resulting in less reliable prediction of the stimulus by the neuronal model when the stimulus is next presented.

If the dimensions listed above are indeed encoded by the neuronal model then the predictions outlined in Table 1.1 can be made:

TABLE 1.1

Predictions of Sokolov's comparator theory of elicitation and habituation of the OR.

1. A change in stimulus intensity, tone pitch, colour, position, duration or modality should result in the recovery of a previously habituated OR.

2. Presentation of a stimulus at a shorter ISI will result in more rapid habituation (requiring fewer presentations) than the presentation of the same stimulus at a longer ISI.

3. A change in the temporal relationship between successive stimuli should result in the recovery of a previously habituated OR.

4. A change in the ISI of a stimulus series should result in the recovery of a previously habituated OR.

5. Presentation of a stimulus at a variable ISI should result in less rapid habituation (requiring more presentations) than the same stimulus presented at a constant ISI.

6. Omission of a stimulus which has previously been presented for a number of presentations will result in the elicitation of OR at the time the stimulus would otherwise have been presented.

Prediction 1 follows simply from the theory in that stimulus intensity, pitch, colour, position, duration and modality are stimulus dimensions postulated by Sokolov to be encoded by the neuronal model. Change in one or more of these dimensions should therefore result in the recovery of a previously habituated OR. Interestingly, the arrangement of representation of each of these dimensions should allow experimenters to predict the magnitude of the recovered OR. For example, tone pitch is known to be organised in the human

cochlea and auditory cortex as a tonotopic (i.e., in order of frequency) map. If a subject has been repeatedly presented with a 1000 Hz tone stimulus, and then is presented with a tone of a different frequency then the proximity of the new tone to 1000 Hz will determine the size of the OR. Thus a new tone with a frequency of 1600 Hz should elicit a larger OR than one of 1050 Hz. Results consistent with this prediction have been reported by Ben-Shakhar and Lieblich (1982).

The prediction that ISI duration will have an effect on the rate of habituation can be explained in two ways from the model. The first is simply to assume that the mechanism by which ISI is encoded by the neuronal model is more complex for longer ISIs than it is for shorter ISIs. Therefore, it would be expected that it would take more stimulus presentations for the subject to accurately predict the time of stimulus presentation in the longer ISI condition. An alternative explanation is that the neuronal model decays between presentations. The longer ISI condition allows more decay of the model between stimulus presentations and thus more stimulus presentations are required before the neuronal model accurately predicts the time of stimulus presentation and the OR habituates.

Of most relevance to the current research are predictions 3, 4, 5 and 6. These predictions all result from the ability of the neuronal model to encode and extrapolate both the ISI of a series of presentations of an individual stimulus and the temporal relationships between multiple stimuli. If, after a number of stimulus presentations with a constant temporal pattern, one aspect of this pattern is altered then recovery of the OR results.

It is predicted that if a stimulus is presented at a constant ISI then the neuronal model will require fewer presentations before it can accurately predict the time of presentation of the stimulus than if the stimulus is presented at a variable ISI, resulting in less rapid habituation in the variable ISI condition.

The prediction of the eliciting of an OR when a stimulus is omitted following a number of presentations is commonly explained by suggesting that, at the time the stimulus would have been presented, there will be a disparity between the neuronal model and the afferent stimulation (ie. no stimulus) and an OR will be elicited. The existence or otherwise of this response to stimulus omission is considered a vital indicator of the presence or otherwise of extrapolatory temporal encoding in the neuronal model. Hall (1989) however states that the presence of extrapolatory temporal encoding in a model is not in itself a sufficient process enabling the prediction of a response to stimulus omission. For a response to stimulus omission to occur there also needs to be some method by which a mismatch between the neuronal and afferent stimulation can directly elicit a response. In Figure 1.1 it was seen that the main function of mismatch in the model was to prevent or "gate" the elicitation of a response which would normally have been elicted by a stimulus presented to the subject. It is not entirely clear that a mismatch itself elicits any response although it is possible that this might occur via the response pathway directly connected to the neuronal model. It appears that Sokolov did assume that a mismatch could actually elicit a response in the absence of any external stimulation and his model is assessed in this spirit. It is however also clear that the precise method by which this occurs was not clearly described in the works cited.

1.2.2 Wagner's Priming , SOP and AESOP Models

Priming theory (Wagner, 1976, 1978) and the later incarnations of the basic concept as the SOP which stands for "sometimes opponent process" (Wagner, 1981; Whitlow & Wagner, 1984) or "standard operating procedures" (Hall, 1991) and AESOP (affective extension of SOP, Wagner & Brandon, 1989) theories provide an alternative to Sokolov's comparator theory of habituation while maintaining some common elements with the earlier theory.

The basic tenet of priming theory (Wagner, 1976,1978) is that there will be no response to a stimulus if it is pre-represented or primed in a limited capacity short term store (STS). There are two mechanisms by which a stimulus may become primed in the STS. The first is self-generated priming in which the representation of the stimulus is activated by a recent presentation of a stimulus. The second is retrieval-generated or associative priming in which the activation of the stimulus representation in the STS is primed by the presentation of contextual cues which have been associated with the stimulus in the past. The parallels with Sokolov's theory are immediately apparent in that the STS could conceivably be construed as Sokolov's neuronal model while the analogues of self-generated and retrieval-generated priming can be proposed as particular cases of processes involved in the formation and maintenance of the neuronal model.

One feature of habituation explored in Wagner's work which was not addressed by Sokolov's theory is the distinction between short-term (within-session) and long-term (between-session) habituation. It has been suggested by Stephenson and Siddle (1983) that self-generated priming is responsible for short-term habituation while retrieval-generated priming is largely responsible for long-term habituation. Wagner, however, proposed that retrieval generated-priming does indeed play a part in short-term habituation (Wagner, 1976) and it seems plausible that an effect like the response to S2 omission following the presentation of a series of S1-S2 pairs which has been observed during short-term habituation studies can be more easily explained by an associative retrieval-generated priming mechanism than a non-associative self-generated priming mechanism.

Wagner (1981) and Whitlow and Wagner (1984) extended the basic ideas of priming theory into the quantitative model known as SOP. In SOP memory is conceptualised as a graph structure consisting of representative memorial

nodes complexly interconnected via directional associative links. Each node is not actually a single entity but rather a set of informational elements allowing for the notions included in stimulus sampling theory (Estes, 1955a, 1955b). Different spaces of the memory structure are distinguished from each other depending on whether or not the nodes are currently active or inactive. The active/inactive distinction is analogous to the short term memory/working memory/long term memory/attention distinctions used in other theories. In particular SOP has three possible states: inactivity (I), and two states of activity, primary activity (A1) and secondary activity (A2).

The memory system is connected to the sensory environment via a "sensory register" so that presentation of a stimulus will tend to activate the components of the corresponding memory node that are currently inactive. This unconditioned effect moves the corresponding elements from the I state to the A1 state, from which (if not further stimulated) they will eventually decay to first the A2 state and eventually back to the I state.

The memory system is also connected to a "response generator" where the response, R_j emitted by the animal is calculated by the formula:

$$R_j = f_j (W_{1,j} \, p_{A1} + W_{2,j} \, p_{A2})$$

where p_{A1} and p_{A2} are the proportions of nodal elements in the subscripted states (based on stimulus sampling theory), $W_{1,j}$ and $W_{2,j}$ are linear weighting factors and f_j is a mapping function appropriate to the response measure.

The processes important in the habituation of the response to a particular stimulus are provided in SOP by the definition of rules by which a node may move between the various states. These rules are depicted in Figure 1.2.
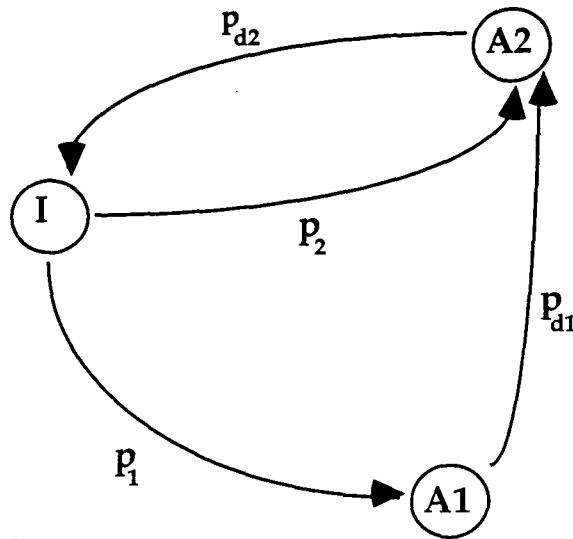
Figure 1.2. Possible routes of state transformation for a memorial node in SOP. (adapted from Wagner, 1981, p.17).

The elements within a node can thus only move between the states I, A1 and A2 in particular circumstances. The variable p assigned to each pathway is the probability that that particular state transition will occur at that time, with each value of p dependent on the nodal environment at that moment. It can be seen that the only way a node can reach the A1 state is if it is first in the I state and p_1 has a nonzero value, a situation which only occurs if the corresponding unconditioned stimulus is presented (the magnitude of p_1 is directly related to stimulus intensity). The node activity will then decay back to the A2 state with probability p_{d1}, and eventually from A2 to I with a probability p_{d2}. Wagner (1981) approximated that because of capacity differences $p_{d1}=5p_{d2}$ resulting in the assumption that nodes would decay from A1 to A2 relatively rapidly but from A2 to I more slowly. Interference of further distractor stimuli increase the values of p_{d1} and p_{d2} and therefore result in faster decay from both the A1 and A2 states. Finally, it is possible for a node to move from the I state to the A2 state directly when $p_2 \neq 0$. This will occur when activity in another node or set of nodes, connected to the depicted node, passes activity to the depicted node.

This process, analogous to a conditioned stimulus (CS) or other contextual element being presented and then activating the elements representing the US, is a more formal description of retrieval-generated priming.

A node is assumed to produce its maximal response if it is in the A1 state. The A2 state may also produce a response but this contribution is smaller, and in some cases in an opposite direction to that produced by the elements in the A1 state. Given this assumption and the possible state transformations described above it is a simple matter to derive a number of basic habituation phenomena. Habituation occurs due to successively fewer elements of the node representing a stimulus reaching an A1 state as the stimulus is repeatedly presented. The reason that the number of elements reaching the A1 state decreases as the stimulus is repeatedly presented is that progressively more of the elements representing that node are in the A2 state and thus unable to move to the A1 state when the stimulus is presented. This increase in the number of elements in the A2 state occurs firstly because some of the elements have yet to decay from A1 following the previous stimulus presentation, and that this proportion of nodes in the A2 state will increase on successive stimulus presentations (Stephenson and Siddle (1983) argued , however, if this non-associative or self-generated priming process is the only one involved in short-term habituation and the decay rates are held constant then the proportion of elements in the A2 state will eventually asymptote at a some level, leaving a residual number of elements in the I state and thus able to emit a response to further stimulus presentations), and an increase in retrieval-generated or associative priming of elements into the A2 state by an increase in association between the stimulus and other nodes representing the stimulus context. While associatively generated decreases in response magnitude are generally thought to predominate in long term habituation it is, as described above, necessary that it plays some role in short-term habituation if the SOP model is to avoid a small

residual response to repeated stimulation and be able to exhibit complete habituation.

SOP (and its variants) can be thought of as a type of comparator theory in that each stimulus is represented by a certain pattern of elements in the memory space of the subjects. Repeated presentations of the same stimulus or presentations of a new stimulus, activate the same or alternative units and the response to the stimulus depends solely on the activity within the activated units. Thus there is some notion of comparison of the afferent stimulation with the trace of previous stimulation which in the case of SOP is represented as the pattern of activity the elements of the memory space at a given moment.

One prediction which can be made from the SOP theory which was not predicted by Sokolov is the phenomenon of conditioned diminution of the UCR. This phenomenon (Kimble & Ost, 1961; Kimmel, 1966) refers to a decrease in the size of response (and more rapid habituation) to a stimulus (normally the US in a conditioning study) when it is reliably preceded by a CS, compared to the response elicited when the same stimulus is not preceded by a predicting stimulus. While this effect does not always occur, and occasionally may instead be a conditioned facilitation of the UCR (Hupka, Kwaterski & Moore, 1970), SOP is able to explicitly predict all of these results by the associative process of moving some elements of the node representing the US into the A2 state by presentation of the CS which has become associated with the US. Conditioned diminution is explained by saying that the elements now in the A2 state cannot move into the A1 state and cause a strong response, while conditioned facilitation is explained by saying that the elements in the A2 state actually contribute very strongly to the response and thus the response is facilitated by the associative mechanism described above.

The phenomena of recovery of response to changes in stimulus intensity, tone pitch, colour, position, duration or modality can be accommodated by SOP.

The elements of the node representing a particular stimulus could conceivably store information regarding any of these stimulus dimensions. As was stated earlier , after a number of stimulus presentations the particular elements representing the current stimulus will no longer move to the A1 state when the stimulus is presented and therefore no response will be emitted. If, however, one of these dimensions is altered in some way then elements other than those previously habituated will be stimulated. These "fresh" elements will be in the I state and therefore, on stimulation, move to the A1 state and a response will be emitted.

The effects of changes in temporal properties on response habituation (and more specifically habituation of the OR) are generally less confidently predicted in SOP than in Sokolov's comparator theory. The prediction that stimuli presented at a shorter ISI will habituate more rapidly than the same stimulus presented at a longer ISI is easily accommodated by SOP because, at the shorter ISI, elements representing the stimulus will have less time to decay from the A1 and A2 states to the I state and will thus not be available to move to the A1 state and contribute to the response. At the longer ISI more elements will have decayed to the I state by the time the next stimulus is presented and thus move to the A1 state and consequently result in a larger response and corresponding less rapid habituation.

A change in the temporal order of stimuli would be expected to result in response recovery under the SOP model. Following habituation the presentation of a CS (in a CS-US pair) would prime the elements representing the US by moving them into the A2 state, reducing the ability of the US to evoke a response. It is important to note that Wagner (1981, 1984) argued that the capacity of different sections of the memorial system were limited and thus the movement of the elements representing the US into the A2 state means that other elements representing other stimuli would be more likely to decay to the I

state. Presentation of the US before the CS, or later than usual would mean that the elements representing the US would be more likely to be in the I state and thus able to move into the A1 state, causing a recovery of response. Interestingly, the model would predict that presentation of the US earlier than usual should result in a decreased response, rather than a recovery of response, because it is likely that more elements representing the US would be in an A2 state and thus unable to move to the A1 state at this time.

The question of whether ISI variability will affect the rate of habituation is not clearly predicted by SOP. The prediction can be made if one of the contextual elements encoded in the memorial system is ISI. If this is the case then the passage of time itself should be able to associatively prime the elements representing the US at the time the stimulus would regularly be presented. Presentation of the stimulus series at a variable ISI would then mean that the associative link between time and the US presentation would require more presentations to form (if it formed at all) and the resulting lower level of temporal priming at the time of US presentation would result in the continuation of a larger response than if the same US was presented at a constant ISI. Wagner (1981, 1984) however does not speculate upon the existence of temporal information as a contextual component in SOP.

The actual mechanism of encoding of the ISI of a series of presentations of a single stimulus is troublesome for SOP as it stands. There are essentially two methods by which ISI could be encoded within SOP. The first is to simply assume that ISI is encoded by each individual element within each node, that is, the probability that an element will be in a particular state varies with time and is learnt by that individual node with the result that the node is most likely to be in the A2 state at the time the stimulus would normally be presented, thus reducing its ability to respond. The second method is to rely entirely on associative priming as the means of ISI encoding. This method assumes that

associative priming is of the nature "event Y will occur Z seconds after event X" and explicitly encodes time intervals rather than simply ordinal information such as "event Y occurs after event X". The second method is an ideal candidate for encoding such variables as CS-US intervals, but is not as effective at explaining ISI encoding for a single repetitively presented stimulus. If the CS and US are separate stimuli then SOP has provided connections by which the appropriate information can be stored. If, however, the series consists of presentations of a single stimulus then that stimulus must serve as its own context. This suggests connections from each element recurrent upon itself, not mentioned in the formulation SOP, but perhaps analogous to the "expectancy loops" described in Öhman's (1979) theory described below. While Wagner (1981, 1984) postulates the existence of connections between nodes representing different stimuli he does not however speculate on the existence of the recurrent connections connecting one node to itself described above.

The question of response to stimulus omission is a complex issue for SOP. Omission responses can be divided into two types; the first is response to complete stimulus omission, emitted when one of a series of presentations of a single stimulus is omitted; the second is stimulus omission in the situation where an S2 of an S1-S2 pair is omitted following the presentation of a number of S1-S2 pairs. Both types of omission response must be considered separately when discussing SOP. In the case of complete stimulus omission it is unclear that SOP has a mechanism by which the response can be elicited. As Hall (1989) states, the basic process behind habituation and response elicitation in SOP is that nodes normally emitting a given response become primed and *prevented* from eliciting that response as habituation proceeds. Thus, when a stimulus is omitted there is no means by which a response can be evoked, even if there is some mismatch between the primed activity and the afferent stimulation. Hall directs this criticism at all comparator theories, and indeed Sokolov's model also suggests that the mismatch did not directly elicit a

response but rather gated activation of the amplifying system. However, Sokolov (see Figure 1.1) did suggest a response pathway directly from the comparator which could conceivably serve this purpose.

If response to complete stimulus omission does then occur, comparator or comparator-like theories of habituation must explicitly include a means of direct response elicitation by the mismatch detector, in place of or additional to the current response prevention theories. There is one process by which SOP could, in its present form, account for response to complete stimulus omission. It was postulated earlier that, at the time the omitted stimulus would normally have been presented, many of the elements of the node representing it would be in the A2 state. The response of that node depends in part upon the proportion of elements of the node in the A1 and A2 states. While the constant applied to the A2 proportion is normally negative (ie. nodes in A2 state reduce overall level of response) it can be positive in some response systems. If this were the case then the proportion of elements of the node in the A2 state (primed by some temporal cue) would emit a response at the time the stimulus would have been presented. This explanation, effectively an account of temporal conditioning, introduces complications of its own; it is difficult to see how such a system would ever habituate to a series of stimuli presented at a constant ISI in that the primed elements in the A2 would be eliciting a response after any number of presentations.

The case of response to S2 omission following presentation of a number of S1-S2 pairs in SOP is open to the same criticisms as the account of response to complete stimulus omission. While it is simple to see how the node representing the S2 can be primed by the S1 presentation thus resulting in a mismatch when the S2 is omitted, it is not easy to see how this mismatch can actually elicit a response.

AESOP (Wagner & Brandon, 1989) is the so called affective-emotional SOP. Its basic differences with SOP are that stimuli are now represented not by a single node, but by separate sensory and emotive nodes. This distinction does not significantly affect the performance of SOP in the conditions relevant to this dissertation and will not be discussed further.

For comparison with the predictions of Sokolov's comparator theory, some relevant predictions of SOP are summarised in Table 1.2.

TABLE 1.2

Predictions of SOP applied to elicitation and habituation of the OR.

1. A change in stimulus intensity, tone pitch, colour, position, duration or modality should result in the recovery of a previously habituated OR.

2. Presentation of a stimulus at a shorter ISI will result in more rapid habituation (requiring fewer presentations) than the presentation of the same stimulus at a longer ISI.

3. A change in the temporal relationship between successive stimuli should result in the recovery of a previously habituated OR.

4. A change in the ISI of a stimulus series will result in the recovery of a previously habituated OR. *

5. Presentation of a stimulus at a variable ISI should result in less rapid habituation (requiring more presentations) than the same stimulus presented at a constant ISI. *

6. Omission of a stimulus which has previously been presented for a number of presentations will result in the elicitation of OR at the time the stimulus would otherwise have been presented. *

7. A stimulus presented as the S2 of a series of S1-S2 pairs will habituate more rapidly than the same stimulus presented as a series of single stimuli (conditioned diminution of the UCR).

* Prediction inconclusive, see text.

It is interesting to note that the theoretical criticisms of SOP (particularly concerning the effects of ISI variability, omission responding and initial versus change OR responses) are only possible because of the rigid formal nature of the model. For example, it has been shown that if a comparator-like model of habituation is to predict a response to stimulus omission it must provide both a

method of encoding ISI *and* a method by which a mismatch can directly elicit a response. In the case of Sokolov's model it is said that ISI is encoded and a pathway exists which might conceivably provide a direct response route when a mismatch is detected by the comparator. This leads us to suppose that the predictions made in Table 1.1 are a valid reflection of the model. In SOP all processes of response elicitation, priming and associative connections between stimuli are clearly defined. This leads us to directly assess that temporal encoding is not explicitly predicted by the model and that there is no simple method by which a mismatch can independently elicit a response, concluding that the prediction of a response to stimulus omission and a larger change than initial OR can not be easily predicted from the model as it stands (although it could be argued that the model can accommodate both processes with appropriate assumptions).

Assuming that these effects are empirically valid it would seem that Sokolov's comparator model was more valid than SOP. However it can also be argued that this is not the case, rather the level of formal statement of SOP allows it to be assessed fairly and clearly in the face of the evidence. Sokolov's model, on the other hand does not sufficiently define the nature of the mechanisms described within it in such a way to allow such stringent testing and it can be argued that predictions commonly made concerning Sokolov's model always require the application of assumptions (in particular that there is a direct route by which a mismatch can elicit a response and that temporal parameters of a stimulus series are encoded) which were not clearly expounded in Sokolov's original writing, while assessment of SOP is limited by the more formal statement of this theory. The difference in approaches is described thus by Wagner:

> One reaction I anticipate is the following: Do we really need to go through all this complex mathematizing? . . . The answer, of course, is that, casually, one can anticipate anything. Whatever one's theoretical

predilections, there is a need to develop *some* determinate account. . . One might wish that the model appealed to fewer processes and involved a smaller number of parameters. But one should not confuse the attempt to articulate presumed processes in a careful manner, or to specify the parametric choices that must be made, with their invention. In the absence of a relatively formal model such as SOP, we can more surely "fit" any outcome to our theoretical viewpoint, by undisciplined appeal to the multiple processes and parametric variation abundantly acknowledged in our more casual treatment of memory phenomena. (Wagner, 1981, p.43)

It will be seen, as the reader proceeds through this thesis, that the author's sympathies lie mostly with Wagner's approach. Wherever possible processes will be outlined and tested in mathematical form (with accompanying verbal explanations). In this way it is hoped that the theory which will eventually be developed will share, with SOP, the virtue of stringent and simple testability by future workers in the area.

1.2.3. Öhman's Information Processing Theory

A dimension of Öhman's (1979, 1992) theory not discussed previously in this thesis is the concept of the signal value of a stimulus and its effect on elicitation of the OR. In addition to the non-signal stimuli assumed in the theoretical discussions above (e.g., innocuous lights, tones sensations etc.) Öhman also discusses the OR to stimuli known to have pre-defined significant consequences to the organism. An example of a stimulus with a high signal value is a phobic object relevant to a particular subject. As Öhman (1992) points out the necessary conditions for OR elicitation by non-signal stimuli are fundamentally opposed to those necessary for OR elicitation by significant stimuli. In the former case the OR is elicited if there is a mismatch between the afferent stimulus and the contents of some memory store while in the latter case the response is elicited if there is a *match* between the stimulus and a predetermined template of significant stimuli. Thus, if it is assumed that there

is basically only one type of OR, there must be some level at which these two disparate processes are integrated.

Öhman (1979, 1992) argues that the unifying feature of both types of OR is that both indicate the need for further information processing of the stimulus.
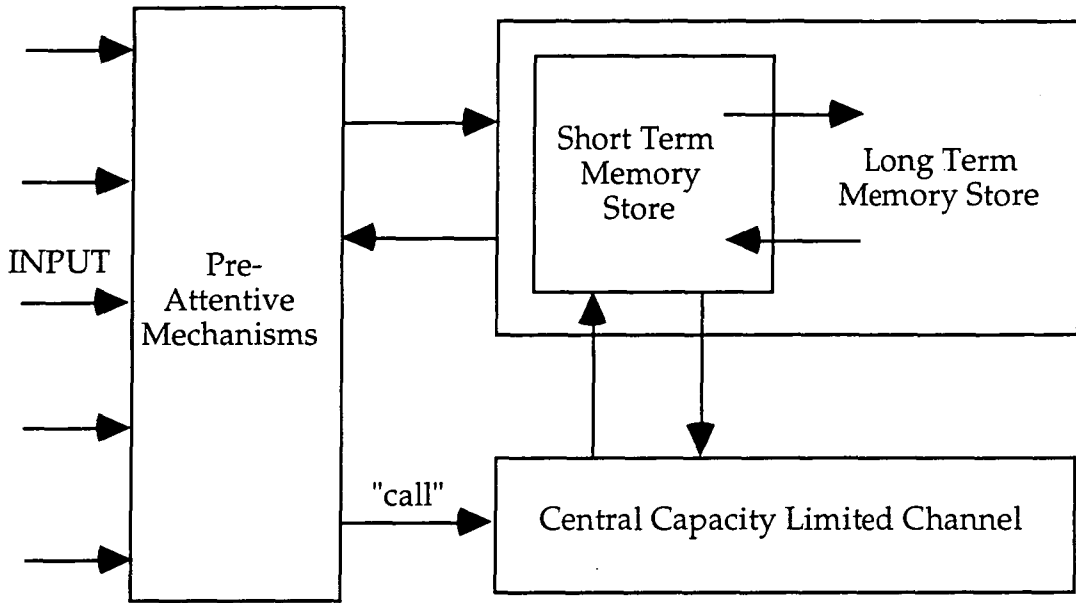
Figure 1.3. Öhman's information processing model of elicitation and habituation of the orienting response (Öhman, 1992, p. 265).

The OR in this model (see Figure 1.3) is conceptualised as the "call" to a capacity-limited central processing channel by pre-attentive processing mechanisms. Öhman (1992) describes the OR as the "gateway to consciousness". Öhman states that the OR can be elicited in one of two ways, both of which result from the comparison of afferent activity with memory held in a short-term store (STS), a store defined by Öhman to be an active section of a long-term store. If there is no match between the input stimulus and the elements of the STS the nonsignal route to OR elicitation is activated and controlled processing resources are allocated to further stimulus analysis. If, however, the afferent stimulation matches an element of the STS *tagged as*

significant the signal route to OR elicitation is activated and control is transferred to the controlled processing level for response selection.

In both cases the OR is elicited as a reflection of the call to the controlled processing channel, and the stimulus enters the focus of attention. This call is furthermore assumed to be reflected with autonomic responses such as the skin conductance response (Öhman, 1979). The later formulation of the model (Öhman, 1992) however argues that the OR does not in fact reflect the call for processing but rather the controlled processing itself and can thus be affected by the availability of processing resources. The general aspects of the model however remain as in the earlier formulation.

While the distinction between signal and non-signal stimuli is interesting and necessary, particularly with regard to some studies using phobic objects as stimuli (e.g., Öhman, Dimberg & Esteves, 1989), this thesis is largely concerned with the habituation of the OR to non-signal stimuli. In the case of Öhman's model this means that the focus of discussion and criticism will be limited to the non-signal pathway to elicitation of the OR. This pathway consists of a pre-attentive comparison of the afferent stimulus with the existing contents of the STS with an OR being elicited if a representation of the stimulus is not already activated in the STS. Like other comparator models of habituation the predictions concerning habituation of the OR to nonsignal stimuli then becomes a question of the extent and processes involved in the building up of representations in the STS, and the comparison of this representation to the incoming stimuli.

Physical parametric changes (e.g., tone pitch, intensity, colour) would be predicted to cause the recovery of a previously habituated stimulus, as the altered stimulus would not be pre-represented in the STS. This prediction is identical to that of the previously outlined comparator theories.

Encoding of temporal information is discussed in Öhman (1979). Both order and interval information is encoded by "expectancy loops". Öhman discusses these in terms of CS-US series where the presentation of the CS initiates the expectancy loop which results in the US presentation being represented in the STS at the time the US is presented. The loop is described by Öhman:

> The loop consists of a waiting state and of tests of occurrence of the specific UCS and the passing of the ISI. If the UCS is changed or omitted, a memory mismatch occurs, an OR is elicited . . . Similarly, if the ISI is changed an OR occurs either to the UCS, if it is presented earlier than normal, or to the point in time where it normally occurs, if it is presented later than normal. (Öhman, 1979, p. 451).

While Öhman specifically refers to CS-US pairs it can be argued that the same expectancy loops could encode the ISI of a series of presentations of a single stimulus. While both Sokolov's and Wagner's theories could be criticised in that in both cases the neuronal model acted as a gate on response elicitation and thus the omission response could not be explicitly predicted from these theories (while there was a mismatch there was no afferent stimulus, and it was not clear that a mismatch itself could elicit a stimulus), Öhman (1979) avoids this problem. In Öhman's theory the OR is postulated to be either the call for central processing or else the actual processing resulting from this call. It is not necessary for a stimulus to be presented for this call to be made, a point made in the excerpt quoted above where the simple representation of the US in the STS (cued by the CS), and subsequent omission of the US is said to be a sufficient condition for the elicitation of an OR.

While the purely mismatch initiated OR is an advantage in predicting the existence of an omission response it provides a number of problems of its own. As Stephenson and Siddle (1983) point out this mechanism suggests that every situation in which a stimulus ceases to be presented or when afferent stimulation does not completely match all the representations in STS (e.g., environment change) will elicit an OR, a situation which would appear to be

more sensitive than empirical results suggest. In a personal communication to the authors (cited in Stephenson & Siddle, 1979, p. 203) Öhman further elaborates the theory to account for this point. It is suggested that expectancy has a number of varieties. Quality expectancy refers to the type of stimuli normally associated with particular stimuli and disruption of this does not lead to an OR. Both temporal expectancy and event expectancy predict with high probability the presentation and time of presentation of a particular event and disruption of either results in the elicitation of an OR. Öhman, however, does not elaborate on how these differences in expectancy are represented within his model.

Most comparator models are, in their present formulations, vulnerable to either the criticism that the mismatch itself cannot elicit a response, and thus the omission response cannot be elicited, or that if the mismatch itself can elicit an OR then why do not all changes which could reasonably result in a mismatch evoke an OR? It would appear that the latter criticism could be more effectively countered than the former, perhaps by the application of a non-linear function relating mismatch to the OR magnitude.

The question of ISI duration effects on within-session habituation rate are in accord with the theories of Sokolov and Wagner. Öhman (1979) states that the content of the STS decays over time, therefore shorter ISIs result in a greater pre-representation of an iterated stimulus in STS at the time it is next presented, resulting in a smaller OR and thus more rapid habituation than at longer ISIs.

The predictions of Öhman's theory for the habituation of non-signal stimuli are outlined in Table 1.3. The predictions concerning these particular effects are the same as those of SOP, however the mechanisms by which the omission response is thought to be elicited are different, a point that will become more relevant later in the dissertation where formal models are constructed.

TABLE 1.3

Predictions of Öhman's information processing model applied to elicitation and habituation of the OR to non-signal stimuli.

1. A change in stimulus intensity, tone pitch, colour, position, duration or modality should result in the recovery of a previously habituated OR.

2. Presentation of a stimulus at a shorter ISI will result in more rapid habituation (requiring fewer presentations) than the presentation of the same stimulus at a longer ISI.

3. A change in the temporal relationship between successive stimuli should result in the recovery of a previously habituated OR.

4. A change in the ISI of a stimulus series should result in the recovery of a previously habituated OR.

5. Presentation of a stimulus at a variable ISI should result in less rapid habituation (requiring more presentations) than the same stimulus presented at a constant ISI.

6. Omission of a stimulus which has previously been presented for a number of presentations will result in the elicitation of OR at the time the stimulus would otherwise have been presented.

7. A stimulus presented as the S2 of a series of S1-S2 pairs will habituate more rapidly than the same stimulus presented as a series of single stimuli (conditioned diminution of the UCR).

1.2.4. *Other Comparator-like/Associative Models of Habituation*

A number of modifications of the basic "OR as indexed by mismatch between a trace of previous stimulation and afferent stimulation" have been presented by other authors. Many of these were however constructed to account for effects beyond the scope of this dissertation, notably the signal/non-signal difference.

The current work is concerned with the effect of various manipulations of temporal predictability on the habituation of *non-signal* stimuli. For this reason models such as those proposed by Gati and Ben-Shakhar (1990) and Cowan (1988) will not be discussed further.

1.2.5. Dual-process theory

Dual-process theory (Groves & Thompson, 1970; Thompson et al., 1979; Thompson, Groves, Teyler, & Roemer, 1973) takes a fundamentally different approach to response habituation to the theories mentioned above. Rather than suggesting that responses are elicited when there is a mismatch between a trace of previous stimulation and current stimulation, they propose that observed responses are a result of the superimposition of the two independent processes of inferred habituation and inferred sensitisation. Inferred habituation occurs only in the direct stimulus-response (S-R) pathway, as a result of iterated stimulation, while inferred sensitisation takes place in a separate "state system". Of course an animal can not exhibit both habituation and sensitisation of the same response system simultaneously so the actual behavioural outcome is the result of interaction of the separate systems of *inferred* habituation and *inferred* sensitisation, expressed behaviourally via pathways such as the motoneuron (motor neuron) in Figure 1.4.

Inferred habituation is said to develop, in the S-R pathway, exponentially to an asymptote at a rate inversely related to ISI. The inferred habituation process is said to generalise to stimuli sharing common elements with the stimulated S-R pathway. When stimulation ceases the inferred habituation process decays, allowing spontaneous recovery of the response when it is next stimulated. Repeated series of habituation sessions however increase the time needed for full recovery, introducing a means by which long-term habituation can be incorporated into the model.

Inferred sensitisation develops in the state system of the organism. This means that while inferred habituation is specific to a particular pathway (with generalisation to a small proportion of similar pathways), inferred sensitisation occurs in both stimulated and non-stimulated pathways to increase the general level of excitation of the animal. The time course of inferred sensitisation is different to that of inferred habituation with inferred sensitisation to repeated presentations of a moderate stimulus proposed to first increase, but later decrease. The specific time course of this change depends on the intensity of the stimulus, with there being a larger amount of sensitisation in the system if a more intense stimulus is being presented. Inferred sensitisation may not decrease after many iterations if the stimulus is sufficiently intense, a condition which also produces continuing behavioural sensitisation.
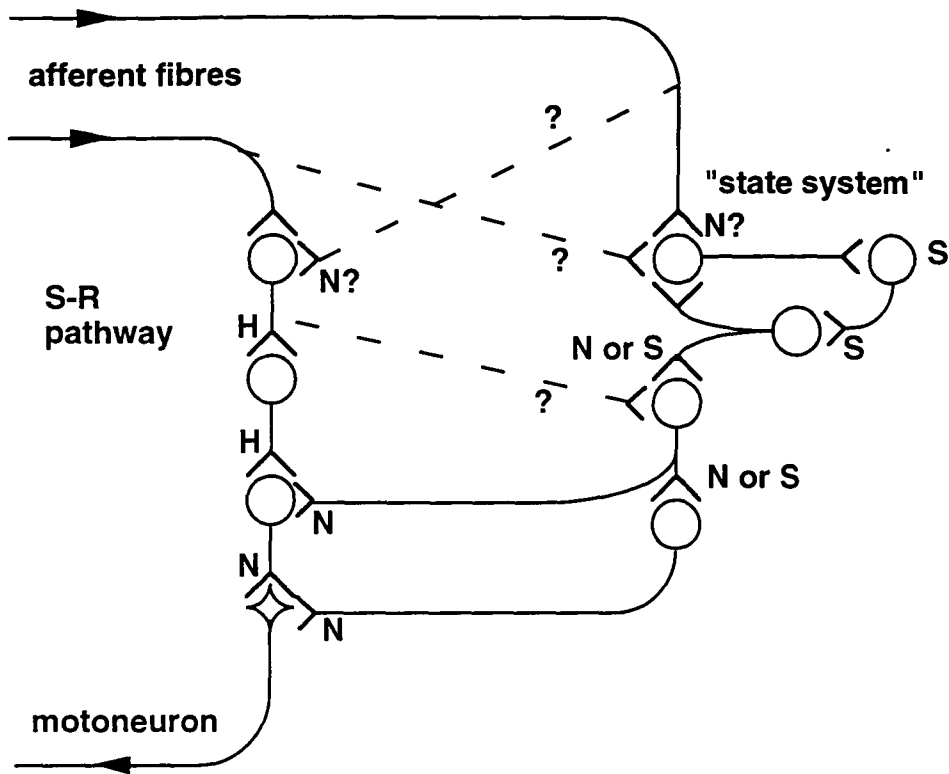
Figure 1.4. Schematic diagram of possible neuronal substrate of habituation and sensitisation. N indicates nonplastic synapses, H indicates habituating synapses, and S indicates sensitising synapses. Dashed lines indicate possible pathways of interaction between the S-R pathway and state system. (From Groves & Thompson, 1970, p. 436).

The processes of inferred habituation and sensitisation are hypothesised to interact along the neuronal pathways outlined in Figure 1.4. While this figure was initially developed to represent neurons in the spinal cord (Groves & Thompson, 1970) the basic general tenets of the model can be easily seen in it. Indeed the model could be simplified with afferent neurons leading to the S-R pathway and a single state system neuron, with each of these neurons synapsing directly on the motor neuron. The model illustrated however is more neurobiologically accurate, including a number of interneurons in both systems and making the state system a multineuronal system.

The dual-process theory has been praised as a clearly defined, parsimonious model capable of accounting for many of the observed habituation phenomena (Stephenson & Siddle, 1983). Furthermore, there is strong neurobiological evidence supporting the existence of both habituation and sensitisation at a neuronal level in both vertebrates and invertebrates. Kandel and colleagues (Castelluci & Kandel, 1976; Kandel, 1976; Kandel, 1979) have identified both processes in the invertebrate *Aplysia*. Both habituation and sensitisation of the gill and siphon withdrawal reflexes of *Aplysia* are caused by alterations in the calcium ion current of the pre-synaptic cell of the sensory to motor neuron synapse of the reflex loop. Habituation is caused within the S-R pathway by a decrease in the amount of neurotransmitter released at the pre-synaptic membrane of the synapse connecting the sensory neuron directly to the motor neuron, a result of a decrease in the influx of Ca+ ions into this cell. Sensitisation at the same synapse is thought to be caused by an increase in the effect of calcium on the presynaptic neuron. Specifically the depolarisation of this neuron is prolonged resulting in a broadening of the resultant action potential. This effect can be artificially induced by introducing increased levels of either serotonin or cyclic AMP into the sensory neuron.

Thompson et al. (1979) cite the existence of this neurobiological evidence as a strong point in favour of dual-process theory, and one area in which alternative theories are generally deficient. Thompson et al. (1979) argue that for comparator theories of habituation to provide a viable alternative to dual-process theory there needs to be neurobiological evidence of the neuronal model of previous stimulation central to these theories. This evidence would take the form of neural structures in which activity would increase as further iterations of a stimulus were presented. At the time of writing Thompson et al. (1979) concluded that there was no available evidence which suggested that the repeated presentation of a single, moderate stimulus resulted in an increase in activity of *any* neural structure. This failure to find neurobiological structures

capable of supporting the neuronal trace required by comparator theories of habituation must be considered a weakness of such theories.

Dual-process theory then is a simple and strong account of habituation which successfully predicts many of the basic habituation phenomena, and is supported by clear neurobiological evidence. If we refer to the list of predictions in Tables 1.2 and 1.3 it is evident that predictions made from it are quite different to those made from the comparator theories discussed previously.

The prediction that the OR will recover when a previously habituated stimulus is altered is easily accounted for by dual-process theory. The change in the stimulus simply causes some non-habituated S-R pathways to be stimulated resulting in an increased response to the new stimulus.

That a stimulus presented at a short ISI should habituate more rapidly than the same stimulus presented at a longer ISI is explicitly predicted by dual-process theory as the amount of inferred habituation in the S-R pathway decays over time. If the interval between successive presentations is increased then there will be less inferred habituation in the stimulated pathway when the stimulus is next presented, resulting in a larger behavioural response and correspondingly less rapid habituation.

Predictions which require the cuing/priming of one stimulus by another (e.g., changes in S1-S2 order, conditioned diminution of the UCR) can not be made from dual-process theory. The model was not, however, intended to deal with such situations, rather it was developed as a model of habituation of a single stimulus.

The major difference in predictions between dual-process theory and the comparator theories, is in the prediction of phenomena which require the encoding of ISI, in particular the response to stimulus omission

(missing-stimulus effect) and the effect of ISI variability on habituation. Groves and Thompson (1970) noted that, at the time, evidence for an omission response was meagre and did not include a process capable of prediciting the response as an integral part of their theory. They did however propose a mechanism by which the response could be provided utilising the process of temporal conditioning.

Temporal conditioning is the phenomena by which repeated presentation of a stimulus at a constant ISI results in the organism responding at the time the stimulus would normally have been presented, even if the stimulus itself is not presented. Groves and Thompson (1970) argued that the omission response can be explained by saying that temporal conditioning occurs within the state system as the stimulus is repeatedly presented. Recalling that response was thought to be a result of inferred habituation in the S-R pathway with inferred sensitisation in the state system, when the stimulus is omitted the conditioned activity in the state system results in only the sensitisation component of this response being present and thus the response appears to recover. While this is a plausible explanation of the omission response two problems arise; firstly, temporal conditioning is by no means a well documented phenomenon, and secondly, the need to resort to temporal conditioning in the state system does not seem qualitatively different to the other forms of neuronal models proposed by comparator theories and criticised by Thompson et al. (1979). Thus, while a response to stimulus omission can be predicted from dual-process theory the prediction is strained and requires a fundamental break with the simple definition of the theory initially stated by Groves and Thompson (1970).

The lack of an integral process capable of encoding the ISI of stimulus presentation means that dual-process theory does not predict that ISI variability should have any effect on the rate of habituation. Nor, it can be argued, can dual process theory predict the response to stimulus omission without

modifications which result in it becoming open to similar criticisms to the comparator theories regarding the neurobiological evidence for modelling of previous stimulation by the nervous system. For the purposes of this thesis it is then suggested that dual process theory be adopted in the simple form first outlined in this section. While this removes the ability of the theory to predict any response to stimulus omission or that there is an effect of ISI variability on habituation rate, the initial formulation of the theory has the benefits of simplicity and supporting neurobiological evidence. The predictions in Table 1.4 are then based upon this simplified version of dual-process theory, rather than a special modification of the theory designed to account for a particular effect.

TABLE 1.4

Predictions of dual-process theory applied to elicitation and habituation of the OR.

1. A change in stimulus intensity, tone pitch, colour, position, duration or modality should result in the recovery of a previously habituated OR.

2. Presentation of a stimulus at a shorter ISI will result in more rapid habituation (requiring fewer presentations) than the presentation of the same stimulus at a longer ISI.

3. A change in the temporal relationship between successive stimuli will not result in the recovery of a previously habituated OR.

4. A change in the ISI of a stimulus series will not result in the recovery of a previously habituated OR.

5. Presentation of a stimulus at a variable ISI will not result in less rapid habituation (requiring more presentations) than the same stimulus presented at a constant ISI.

6. Omission of a stimulus which has previously been presented for a number of presentations will not result in the elicitation of OR at the time the stimulus would otherwise have been presented.

1.3 Summary

The examination of the various theories of habituation presented in the preceding pages raises a number of points requiring further investigation. Firstly, all the theories discussed are able to provide a clear mechanism by which simple habituation of response to repeated presentation could proceed. Secondly, all the theories provide alternative, but equally effective mechanisms by which a change in physical stimulus parameter can result in a recovery of a previously repeated response. All theories also provided a mechanism which

predicted the generalisation of habituation to stimuli sharing similar physical parameters.

The different theories provided disparate predictions regarding the effect of various experimental manipulations on habituation and recovery of the OR. Conditioned diminution of the UCR is only predicted by theories in which the presentation of one stimulus can eventually come to cause the pre-representation of a subsequent stimulus in the internal store. Both Wagner's SOP theory and Öhman's information processing theory can predict the conditioned diminution of the UCR, Sokolov's comparator theory can not, while dual-process theory was not designed to be applied to such situations and effectively makes no relevant prediction.

The major difference between the theories is in their treatment of temporal predictability on the course of habituation. Empirically, the encoding of ISI would be evidenced by a result of less rapid habituation when a stimulus is presented at a variable ISI compared to when the same stimulus is presented at a constant ISI, and the emission of an OR to the omission of a stimulus previously presented at a constant ISI. The encoding of ISI has been included to some extent in all of the theories discussed. It is however clear that none of the theories discussed have proposed a plausible mechanism by which ISI encoding can occur and/or a response to stimulus omission can be elicited.

Sokolov's comparator theory predicts both effects but stops short of providing a clear statement of a mechanism by which ISI is encoded in the neuronal model. Prediction of an omission response also causes problems for this model in that, even if there is a mechanism for detecting a mismatch between the neuronal model and afferent stimulation, there is no mechanism by which a mismatch per se can actually elicit an OR if there is no afferent stimulation.

Wagner's SOP theory possesses some advantages over Sokolov's comparator theory in that its explicit quantitative form allows a more stringent test of its

various predictions. It is, with certain assumptions, possible to see how ISI may be encoded in SOP but again it does not seem clear that a mismatch alone, with no afferent stimulation, could cause an OR to be elicited, thus making it difficult to see how an omission response could be predicted.

Öhman's information processing theory specifically proposes the encoding of ISI by expectancy loops and also suggests that the OR is a reflection of a call to processing. This conception of the OR provides a possible mechanism by which an OR can be elicited by mismatch alone, thus allowing for an omission response. Such a mechanism, however, causes problems in that it would mean that all instances of discrepancy between the neuronal model and the afferent stimuli would result in the emission of an OR when this is not the case.

Dual-process theory does not explicitly predict the encoding of ISI although it does allow for such a mechanism in its "state system". Groves and Thompson (1970) however did not speculate on the mechanism of this encoding of ISI in the state system and it is thus difficult to evaluate the validity of the model in the circumstances described.

In conclusion, it would appear that a promising area for further elaboration of models of habituation is that of temporal encoding and associated phenomena. This is the focus of the current thesis. The next chapter will review the empirical status of the various phenomena predicted by the aforementioned models.

CHAPTER 2

Some Empirical Studies of Habituation

This chapter outlines the results of experiments performed in the past to test the predictions of the various theories listed in the previous chapter.

2.1. Recovery of the OR to physical stimulus changes and generalisation of habituation

Changes in the physical parameters of a previously habituated stimulus have been shown to result in recovery of a previously habituated OR. Siddle, Stephenson and Spinks (1983) report that changes in modality (e.g., light $\Rightarrow$ tone, Houck & Mefferd, 1969; Furedy, 1968, both cited in Siddle, Stephenson, & Spinks, 1983), tone pitch (e.g., O'Gorman, 1972, cited in Siddle, Stephenson, & Spinks, 1983), both increases and decreases in intensity (e.g., Kimmel, 1960; Siddle & Heron, 1977, both cited in Siddle, Stephenson, & Spinks, 1983) all result in the recovery of a previously habituated OR. The results regarding stimulus duration are described as equivocal by Siddle, Stephenson and Spinks (1983) although there is some evidence of OR recovery to both lengthening and shortening of stimulus duration (e.g., Koepke & Pribram, 1966, cited in Siddle, Stephenson, & Spinks, 1983). While Sokolov (1969) also asserts that changes in the colour and location of a habituated stimulus results in recovery of the OR a search of the literature did not find reference supporting this assertion.

An issue related to the representation of physical stimulus parameters is the generalisation of habituation. Ben-Shakhar and Lieblich (1982) performed an experiment in which tones of 1000, 1004, 1016, 1064 and 1256 Hz were presented to subjects in sequences where presentations of one tone followed varying numbers of presentations of a different tone. The results of the experiments showed that generalisation of habituation between stimuli was greatest when the stimuli were psychologically indistinguishable (1000 Hz compared to 1004 and 1016 Hz tones) and least when they were clearly

different (1000 Hz compared to 1064 and 1256 Hz tones). These results are consistent with the known tonotopic organisation of the auditory system and provide evidence that the representational elements in the various habituation models should be organised in the same way as real biological systems.

2.2. ISI duration

It has generally been reported that within-session or short-term habituation is more rapid (requires less presentations) if the stimulus is presented at short ISIs rather than at longer ISIs (Gatchel & Lang, 1975; Schaub, 1965). Both Schaub (1965) and Germana (1969) report virtually no short term habituation at ISIs of 120 and 240 seconds respectively. The results with respect to long-term or between-session habituation are usually reported to be the opposite with longer ISIs resulting in greater habituation if response is tested in a later session (Davis, 1970; File, 1973; Gatchel, 1975). The difference in results for the two different methods is thought to be that in the case of short-term habituation, more rapid habituation occurs with shorter ISIs because these ISIs allow less time for the internal stimulus trace to decay between stimulus presentations. In the case of long-term habituation the longer ISI allows more rehearsal of the stimulus between presentations resulting in a stronger trace in long-term memory.

2.3. Conditioned Diminution of the UCR

As noted in the discussion of SOP in an earlier chapter, conditioned diminution of the UCR has been demonstrated by Kimble and Ost (1961) with human eyeblink conditioning and by Kimmel (1966) with human GSR (galvanic skin response, alternative name for SCR) conditioning.

2.4. ISI Variability

The empirical status of both the effect of ISI variability on habituation and also the response to stimulus omission are of central importance to the theories

developed in this thesis. The literature in each of these areas will therefore be reviewed in more depth than the effects discussed above.

The effect of ISI variability on habituation rate of autonomic responses is an area where empirical study has proved to be largely ineffective in answering theoretical questions. In the most comprehensive review of the literature Graham (1973) remarks:

> The available data thus afford little basis for testing theories that depend on concepts of temporal conditioning or of uncertainty as these are reflected by differences in autonomic response to stimuli presented at variable or constant intervals. Graham (1973, p. 189).

A similar review by Stephenson and Siddle (1983) concludes that "the evidence concerning this issue is meagre and conflicting so the question remains open" (Stephenson & Siddle, 1983, p. 213).

It will be noted that all the papers to be discussed are published pre-1975. An examination of Psychological Abstracts from then until 1994 revealed no additional papers investigating this phenomenon between these years.

One reason that fixed versus constant ISI habituation experiments have not been successfully performed and replicated is thought to be the unknown contribution of temporal conditioning to autonomic responses observed in experiments performed using this paradigm. Pavlov (1927, cited in Graham, 1973) suggests that presentation of a stimulus at a fixed interval leads to increased responding near the point of the UCS occurrence and reduction of responding in the intertrial interval. Such an effect, if it can be shown to exist, is then contrary to Sokolov's theory of habituation in that a constant ISI does not induce more accurate temporal encoding within the neuronal model and thus faster habituation. Presentation at a constant ISI rather has the opposite effect of preserving activity at the time the UCS is presented and thus retarding habituation.

The question of whether temporal conditioning occurs is also difficult to empirically test in that if the conditioned response occurs at exactly the same time or slightly later than the unconditioned response then it will normally be masked by the UCR. It can thus only be detected if the UCR is eliminated, that is by omitting the UCS. The obvious problem here is that such a CR will be indistinguishable from an omission response. It is however possible that temporal conditioning may manifest as an anticipatory response occurring slightly before the UCR. If this is the case it is predicted that the anticipatory response will increase as the number of trials increase and the CR becomes more strongly conditioned.

It is therefore important to note that when experiments using fixed or variable ISIs are being examined that evidence of temporal conditioning should be considered as well as evidence of faster habituation in the fixed ISI condition. One possibility, which could make this task much simpler, is that temporal conditioning and orienting to stimulus omission may not confound each other due to differences in the intensity of stimuli which normally elicit each behaviour.

The first study to be considered is Rouse (1934, reported in Hull, 1934, 1943). Rouse presented 30 uncued shock stimuli at a fixed 38.5 second ISI. Rouse found that when the shocks were no longer presented a number of subjects responded at about the time the UCS would have been presented. This response however disappeared after only a few trials. This result is considered by some to be evidence of temporal conditioning (or possibly an omission response) but a closer examination of Rouse's procedure reveals that this conclusion may not be warranted. Rouse counted as a conditioned response any response which occurred within 10 seconds before or after when the UCS would have occurred. Thus any response within a 20 second window was counted as a CR. It may be expected that, at least in some subjects, spontaneous

responses would have occurred within this time and have been counted as CRs. It is also remarked by Lockhart (1966) that after a shock stimulus there is a refractory period directly following the shock presentation. This may also increase the likelihood of spontaneous responding being more likely in the time window where Rouse would have scored it as a CR. Therefore, while Rouse's results may, on first sight, provide evidence for temporal conditioning and/or omission responding they are, on a closer examination, less compelling.

Lockhart (1966) performed a study where subjects received either 50 two second duration shocks at a fixed 40 second ISI or variable ISIs of 20, 30, 40, 50 or 60 seconds so that the average was 40 seconds. Lockhart then measured the GSR both before the UCS was presented to check for an anticipatory response and in a 10 second (5 seconds before and after) window around the time of UCS presentation on trials where the UCS was omitted. Lockhart reported that both the early (anticipatory) and maximal response within this window were significantly higher in the constant ISI condition. This was interpreted as evidence of greater temporal encoding in the constant ISI group. No data on habituation rate in the two groups was reported. Unfortunately, Lockhart also reported an extremely unusual piece of data, in that when the UCS was no longer presented the CR or omission response showed no diminution over 10 extinction (no UCS) trials. Lockhart reported that there were no significant differences in spontaneous responding at the beginning of the acquisition period. However, Lockhart employed a masking procedure in his experiment in an attempt to prevent his subjects from counting the time between stimuli. This task consisted of listening to a tape of music and responding to a written questionnaire about the tape. Badia and Harley (1970) remark that such a procedure, which gave rise to "singing, keeping time with the rhythms, commenting on the selections" (Lockhart, 1966, p.444) may also be likely to elicit GSR responses. Lockhart, however, stated that tape presentation was

synchronised across both groups and it is difficult to see how such an effect could occur differentially between the groups.

Schaub (1965) presented subjects with eighteen 64 dB white noise stimuli of 8 seconds in duration at either a fixed ISI of 70 seconds or variable ISIs of 30, 60 and 180 seconds. Schaub reported a non significant trend for the fixed ISI group to habituate more quickly than the variable ISI group and also a significant effect relating GSR response to the immediately preceding ISI. The second effect was replicated in experiments by Grings and Schell (1969). It should be noted that Schaub's experiments only had 6 subjects in each group, a relatively small number which raises questions of adequacy of the statistical power of this study.

Pendergrass and Kimmel (1968) performed a study where one group received 40 trials of one second duration, 90 dB, 1000 Hz tones, either at a constant ISI of 40 seconds or ISIs varying between 20, 30, 40, 50 and 60 seconds. They also split these two groups into groups where one group had to judge the intensity of the tone after it was presented and the other did not. There were 60 subjects in all (15 per cell). The results showed a difference in an anticipatory GSR response with the fixed ISI group showing a greater response than the variable ISI group. However this result was reported as "approaching significance [F (1,56)=3.57, .05<p<.10]". This difference was only found in the group which had to judge the intensity of the stimulus. There was also a significant difference in response when the UCS was omitted in the direction of being larger in the fixed ISI group but only when the subjects had to judge the intensity of the stimulus. If the subjects did not have to judge the stimulus intensity then the result was reversed with the variable ISI group showing greater anticipatory response during extinction. Kimmel (1973) asserts that these results show how habituation and conditioning may be affected by cognitive factors. Considering the habituation data from this experiment, it was found that in the

judgement condition response to the UCS in the variable ISI condition did not habituate while the response in the fixed ISI condition did habituate. In the non-judgement condition the result was reversed.

Temporal conditioning in this experiment was supported by the increased (but not significant) tendency for greater anticipatory responding in the fixed ISI group. Close examination of Figure 1a of Pendergrass and Kimmel (1986), however, reveals that there was a substantial difference in anticipatory responding after the first two acquisition trials, a difference unlikely to have arisen from conditioning effects. In the conditions most like a basic nonsignal habituation task discussed in this thesis, that is the non-judgement conditions, there is no evidence for greater anticipatory responses in the fixed ISI group, and further there is a tendency for the variable ISI condition to habituate more quickly. In the judgement condition the tendency was for the fixed ISI condition to habituate more quickly. Kimmel (1973) suggested that this is because in the non-judgement task, the fixed ISI interferes with habituation and thus the variable ISI condition habituates more quickly, while in the judgement condition the judgement task interfered more with the variable ISI condition than the fixed ISI condition, thus the fixed ISI condition habituated more quickly.

An alternative explanation to this is that temporal conditioning is not very well evidenced by anticipatory responses, but is rather evidenced by preservation of the UCR. Thus in the non-judgement condition the fixed ISI condition showed more temporal conditioning and habituated more slowly than the variable ISI condition. When subjects were asked to judge the stimulus intensity temporal conditioning was unable to proceed, due to the limitations on processing resources imposed by the judgement task. The results in the judgement condition were therefore mostly due to habituation processes and the fixed ISI condition habituated more quickly in accordance with predictions from

Sokolov's theory. Such an interpretation would predict higher responding for the fixed group during extinction for the judgement condition, a result also seen in these data.

Badia and Harley (1970) gave subjects 40 shocks of 0.5 seconds duration at a fixed ISI of 40 seconds. They had 3 levels of shock intensity and also split the subjects into stimulus intensity judgement and non judgement conditions. Sixty subjects participated in the study. Badia and Harley reported no evidence of anticipatory responding in any condition and also reported nearly identical patterns of responding for the judgement and non judgement groups. This study, while not employing variable ISIs, is important in that it raises some doubts about the interpretations of temporal conditioning offered by both Lockhart (1966) and Pendergrass and Kimmel (1968).

Gatchel and Lang (1974) attempted to avoid the problem of confounding ISI variability in habituation with temporal conditioning by comparing variable ISI conditions where the degree of variability was manipulated. The stimuli used in their study were 20, 700 Hz, 65dB, two second duration tones presented at varying ISIs. Group 1 received the tones at ISIs of 15, 20 and 25 seconds, group 2 at 50, 60 and 70, and group 3 at 90, 100, 110 seconds. These three groups were termed low ISI variability groups. Group 4 received tones at ISIs of 20, 60 and 100 seconds and was the high ISI variability group. There were 14 subjects in each group. Results indicated that, as expected, groups with shorter ISIs habituated more quickly than the groups with longer ISIs. However, there was no significant difference between groups 2 and 4, that is the high and low ISI variability groups with an average ISI of 60 seconds. Gatchel and Lang concluded that:

> ISI variability, therefore, does not appear to be an important variable affecting SCR habituation. (Gatchel and Lang, 1974, p.803)

Reviewing these studies highlights the problems associated with deciding whether temporal encoding is important in habituation. The studies of Rouse (1934, cited in Hull, 1934), Lockhart (1966) and Schaub (1965), for various reasons are inconclusive. Badia and Harley (1970) and Gatchel and Lang (1974) seem more conclusive in a finding of no evidence of either an effect of stimulus intensity judgement, temporal priming or variability of ISI in habituation. This leaves the work of Pendergrass and Kimmel (1968), which, while it may have some unusual results, does not seem as though it can be completely dismissed. With the phenomenon being such a vital indicator of the temporal encoding parameters of a successful habituation theory it would seem that more research is required to better determine the standing of this effect.

2.5. Response to Complete Stimulus Omission

Further insight into the question of temporal encoding in habituation may come from considering that the fixed ISI/temporal conditioning task is the same as or very similar to tasks used in the investigation of the response to complete omission of a single stimulus. As has been previously stated, these omission studies also report mixed results. However, there have been some relatively successful attempts to relate the results to systematic differences in the subject population. These studies will now be reviewed and an attempt made to relate their results to the ISI variability and temporal conditioning studies.

Of the studies concerning complete omission of a single stimulus it seems that only a subset of subjects show positive results. Cooper, Ashe and Weinberger (1978), in a study on anaesthetised cats, reported omission responses in 31% of cases, a number they say is "uncomfortably low for those supporting a "neural network" theory, but at the same time too high to be attributable to chance alone" (Cooper et al., 1978, p. 4). Of the studies using human subjects, O'Gorman and Lloyd (1976) and O'Gorman (1989) used auditory stimuli and alpha blocking as the dependent variable; Siddle and Heron (1976), O'Gorman

and Lloyd (1984), Barry (1984), Barry and O'Gorman (1987) and O'Gorman (1989) all used auditory stimuli with SCR as the dependent measure.

Siddle and Heron (1976) reported an omission response (as measured by greater dishabituation on the stimulus presentation following the omitted stimulus) in 43% of subjects; O'Gorman and Lloyd (1976) reported an alpha blocking omission response in 60% of subjects; O'Gorman and Lloyd (1984) an SCR omission response in 48% of subjects; Barry (1984) an SCR omission response in 45% of subjects; Barry and O'Gorman (1987) reported rates of 80% in their first experiment and 55% in their second experiment while O'Gorman (1989) provided new data (Experiment 3) with 34% of subjects showing a response to stimulus omission.

Given these results, it could be that there is a systematic difference in either experimental paradigms eliciting the omission response or in subjects exhibiting these responses.

Cooper et al. (1978) suggested ISI as a variable which may mediate the emission of an omission response. Four human studies used ISIs of 13 (Barry, 1984) and 20-21 seconds (O'Gorman and Lloyd, 1976; O'Gorman and Lloyd, 1984; Siddle & Heron, 1976). ISI differences, however, cannot account for only a percentage of subjects within a paradigm showing the omission response. The same criticism could be levelled at variables such as stimulus intensity and duration, although Siddle and Heron (1976) showed that ISI and stimulus intensity have no significant effect on the amount of response recovery caused by stimulus omission.

Another variable which has been suggested to account for the percentages of subjects showing the response to stimulus omission is individual electrodermal lability, measured by the number of spontaneous SCR responses within a pretraining adaptation period. Both Siddle and Heron (1976) and O'Gorman and Lloyd (1984) reported significantly more omission responding in

electrodermally labile subjects than non labile (often termed "stabile") subjects. Siddle and Heron (1976) reported that 80% of labiles showed the response while only 18% of stabiles did, while O'Gorman and Lloyd (1984) reported 75% of labiles and only 29% of stabiles as showing the response.

Barry (1984) performed his analysis of the omission response by comparing response during the omission period with the response during a control period between the last stimulus and the time the omitted stimulus would have been presented. Seventeen of 48 subjects (11 of them stabiles) failed to respond in either of these two periods and 22 of the remaining 31 responded during the omission period with 11 of these being labiles and 11 being stabiles. Therefore, Barry did not show any differential advantage for labile subjects in exhibiting the omission response.

A possible explanation for these results is that omission responses are spontaneous responses which happen to fall in the correct latency window. The labile subjects by definition show more spontaneous responses and thus are more often scored as having made an omission response. However, Barry (1984), O'Gorman and Lloyd (1984) and O'Gorman (1989) scored omission responses by comparing responses within the latency window corresponding to the time the stimulus would have been presented, to responses in a control window. An omission response was only scored if the responses in the omission window were larger than those in the control window. If the so-called omission responses were really only spontaneous SCRs then they would be expected to be randomly distributed in both time of occurrence and magnitude. The scoring of omission responses then would be expected to be much lower than the percentages reported in these papers.

Siddle and Heron (1976) avoided the problem of scoring omission responses per se by indexing omission as a greater response to the trial following the omitted trial. Such an effect, which they termed stimulus recovery, directly

follows from Sokolovian habituation theory in that the omission of a stimulus presumably will result in some degradation of encoding of the neuronal model and then, on the next stimulus presentation, a larger orienting response. This method of scoring greatly reduces the possibility of spontaneous SCRs being scored as omission responses. O'Gorman and Lloyd (1984) also used this definition of omission response in their study and reported responses in 87% of subjects. However, unlike Siddle and Heron (1976), they reported no significant difference between the labile and stabile groups.

Of the two scoring techniques, the comparison of responses in the omission latency window and a control window seems acceptable, but does not remove completely the possibility of scored omission responses being chance events. For example, if spontaneous responses occur randomly in time and with randomly varying amplitudes then the choice of any two latency windows will result in omission responses being scored in 50% of cases where the subjects show a response in either window, a percentage near that found in most of these studies. Labiles may be expected to show a greater percentage of omission responses because many subjects, particularly stabiles, will show no response in either of these latency windows and thus can not be scored as having responded to stimulus omission. Only a subset of subjects, mainly consisting of labiles, are able to be part of the analysis comparing the two latency windows. Then even if the response is an artefact of chance it will manifest more often in labiles as a majority of labiles take part in the part of the analysis comparing response magnitude in the two windows.

The scoring of omission responses by checking for facilitated response to the next presented stimulus as used by Siddle and Heron (1976) and O'Gorman and Lloyd (1984) seems less liable to interference from spontaneous SCRs. The necessary greater passage of time between the pre-omission and post-omission presentations however means that this increase in response may reflect

spontaneous recovery of the OR rather than any effect specific to stimulus omission.

In summary, it seems probable that electrodermally labile subjects are more likely to show responses to complete omission of single stimuli than are electrodermally stabile subjects. This difference may be due to better encoding of temporal stimulus parameters in labile subjects, or more likely, to a more sensitive response mechanism in these subjects. That is, temporal encoding is present in *all* subjects, but only those with sensitive response systems are able to manifest this encoding in their electrodermal activity.

There is some evidence suggesting that response to stimulus omission is a voluntary rather than involuntary OR, that is, a result of complex higher cortical processes rather than lower automatic processes, and that this might account for the reported fragility of the effect. Barry and O'Gorman (1987) support this hypothesis with the result that ORs to stimulus omission have a longer latency than those elicited by a physical stimulus change, allowing for greater processing in the intervening period. Wilson (1989), however, criticises the methodology and theoretical conclusions of the paper with claims that the longer latency of a response is a reflection of inaccuracies in the ISI trace maintained by the subject with an omission response not being emitted until the subject is completely sure that the stimulus has been omitted, a suggestion rejected by Barry and O'Gorman (1989). O'Gorman (1989) however provides evidence contrary to the conclusion that response to stimulus omission reflects a voluntary OR, presenting results in which modifications in stimulus significance do not significantly affect the elicitation of an omission response.

2.6. Omission of S2 following a series of S1-S2 presentations

A second paradigm which is sometimes referred to as stimulus omission involves the presentation of a number of S1-S2 pairs followed by an S1 alone presentation, optionally followed by further S1-S2 presentations (Gliner, Harley

& Badia, 1971; Siddle, 1985; Siddle, Booth & Packer, 1987; Siddle & Hirschorn, 1986; Siddle & Packer, 1987). This paradigm is fundamentally different to the complete omission experiments described above in that the S2 can be explicitly cued by the presentation of the S1 in this case, whereas in the case of complete omission the cuing of the stimulus is less clear.

The emission of a response to S2 omission in this situation is, as in the case with response to complete stimulus omission, not shown by all subjects. Gliner et al. (1971) state that "some" of their subjects exhibited an OR at the time the S2 would normally have been presented. In four separate experiments reported in Siddle, Remington, Kuiack and Haines (1983) and Siddle (1985) the frequency of responses to S2 omission was reported as 50%, 33%, 75% and 66%, which, while not very high percentages, are generally greater than those reported in the case of complete stimulus omission. Furthermore, the SCR at S2 omission was significantly larger than the SCR to an S2 presented at the same time in a control series (using a between-group design) in three experiments reported in Siddle et al. (1987), in both experiments utilising SCR reported in Siddle and Packer (1987), and in two of three experiments in Siddle (1985). Thus, while S2 omission may also be restricted to subsets of subjects it appears that the basic existence of the phenomenon is better supported than that of response to complete stimulus omission.

2.7. Summary

The existing literature regarding the effect of ISI variability on habituation leaves much to be desired. The studies of Rouse (1934, cited in Hull, 1934), Lockhart (1966) and Schaub (1965) have methodological problems preventing too much weight being placed on their conclusions. The studies of Pendergrass and Kimmel (1968) and Gatchel and Lang (1974) are better designed but fail to provide conclusive evidence for the presence or otherwise

of the effect. This is one of the questions which the experiments presented in this thesis seek to answer.

The response to complete stimulus omission is also open to further research. Of the studies employing the assessment of an omission response by comparing response to stimulus omission to response in a control window (Barry, 1984; O'Gorman & Lloyd, 1984; O'Gorman, 1989) only Barry (1984) reported the frequency of subjects showing a larger response in the control compared to the omission window. It is also clear that response to complete stimulus omission occurs in only a subset of subjects. The experiments presented in this thesis seek to further delineate the situations in which a response to complete stimulus omission can be evoked and also further refine the techniques used to score this phenomenon.

Given the results of these and other experiments described in this chapter it is clear that one area needing clarification within the habituation literature is the question of temporal encoding, in particular the status of response to both complete stimulus omission and omission of the S2 in a series of S1-S2 pairs (omission of an element of a complex stimulus), and the effect of ISI variability on the course of habituation. The following experiments are largely an attempt to investigate these phenomena, and to delineate the conditions under which each effect occurs.

CHAPTER 3

Experiment 1

The Effect of ISI Variability and Complete Stimulus Omission on Habituation at a Long ISI

The aim of this experiment was to examine the effect of ISI variability on the course of habituation and to test for the presence of a response to complete stimulus omission. The design was therefore simple with a single, moderate intensity tone stimulus presented either at a constant ISI or at a variable ISI for 18 trials, then omitted for one trial and finally re-presented at the time it would have been presented for the twentieth trial if the nineteenth presentation had not been omitted.

A feature of this experiment, which will assume more importance as the thesis progresses, was the choice of ISI. The dependent variables used in this experiment (trials to habituation, absolute rate of habituation and SCR magnitude to stimulus omission) all required that the SCR to each individual stimulus presentation be identified and measured. If a very short ISI was used then the responses to individual stimuli would tend to influence or obscure each other and independent assessment of magnitude would not have been possible (Grings & Schell, 1969). The stimuli in this experiment were therefore presented at an average ISI of 21s, allowing for the shortest ISI in the variable ISI condition to be 14s, a length which Grings and Schell (1969) suggest is sufficient to allow relatively accurate scoring of SCR intensity independent of the influence of preceding stimuli.

METHOD

Subjects

Seventy-eight undergraduate volunteers participated in the experiment. Subjects were randomly assigned to the fixed and variable ISI conditions, thirty-nine to each. There were 55 female and 23 male subjects.

Apparatus

Skin conductance was recorded by applying a constant voltage of 0.5V across domed Ag-AgCl electrodes with .05M NaCl electrolyte. The electrodes were placed on masked areas on the distal phalanges of the index and second fingers of the subject's left hand. The electrodes were connected to a custom-built battery powered bridge which was connected to a Grass 7 DAG preamplifier with a recording sensitivity of 0.02 μS/mm pen deflection. Respiration was recorded using a Phipps and Bird pneumatic bellows connected to a Grass 7PRTE transducer and 7DAG preamplifier.

The auditory stimulus was a 1000 Hz tone with a 30 ms risetime. It was presented binaurally through Sony DR-7 stereophonic headphones at an intensity of 70 dB (SPL) which was calibrated by a Bruel and Kjaer model 2205 sound level meter. Stimulus presentation was controlled by an IBM compatible personal computer.

Procedure

Subjects were seated in a semi-reclined padded chair in a darkened room with an ambient temperature of 23°C and an illumination level of 0.2 cd/m^2. The stimulus presentation and response recording equipment was situated in an adjoining room. Prior to the experiment subjects were informed that the first part of the experiment would be a rest period during which they were to relax. Following the test period there would be a series of tones. The subjects were

instructed that they would not be asked to remember the tones or to respond to them consciously, they were simply required to relax and stay awake.

The 4.5 minute pre-stimulation period consisted of a 1.5 minute period to allow the experimenter to stabilise the recording, and a 3 minute period in which non-specific responses (NSR) would be counted to give an index of subject lability. Following this period all subjects received 18 tone stimuli at either a fixed or variable ISI followed by a period during which no stimulus was presented (stimulus omission trial). Finally, all subjects received a re-presentation trial of the original stimulus.

The duration of the tone stimulus was three seconds for all presentations in both conditions. The fixed ISI group received the 18 pre-omission trials with a constant offset to onset ISI of 21 seconds. The re-presentation trial was presented 45 seconds after the offset of eighteenth stimulus presentation for both the fixed and variable ISI groups. The variable ISI group received the 18 pre-omission trials at varying offset to onset ISIs of 14, 21 and 28 seconds. There were six ISIs of 14 seconds, six of 21 seconds and five of 28 seconds. All subjects received these ISIs in the order: 28-14-21-14-21-28-21-21-14-14-28-21-28-14-21-14-28.

Scoring

Non -specific responses (NSRs) were scored by counting the number of SCRs greater than 0.02 μS occurring in the 3 minute rest period before the first stimulus. Subjects were split at the median into stabile (below median) and labile (above median) lability groups.

Responses considered to be due to stimuli during the habituation series were SCRs greater than 0.02 μS occurring during the 1 to 5 seconds after stimulus onset. Responses associated with unusual respiratory activity were excluded from further analysis.

Responses to stimulus omission were scored by comparing the size of the largest SCR in a time window where an omission response might be expected to occur ,to the largest SCR in a control window where it would not be expected that a response would be elicited (O'Gorman & Lloyd, 1984). The omission window was defined as 1 to 9 seconds after the time the stimulus would normally have been presented with "the time the stimulus would normally have been presented" defined to be 21 seconds (the average ISI of the habituation series for both groups) after the offset of the stimulus 18. The control window was defined as the interval from six to fourteen seconds before stimulus 18.

Trials to habituation, an index of the rate of habituation, was scored as the presentation number after which the following three stimulus presentations did not evoke responses greater than 0.02 μS. The uncorrected habituation rate was calculated as the slope of the regression line predicting the skin conductance response from log trial number over the eighteen pre-omission trials (Lader & Wing, 1966).

RESULTS

TABLE 3.1.

Mean trials to habituation and absolute rate of habituation for stabile and labile groups under constant and variable ISI conditions.

	Constant ISI		Variable ISI	
	Stabile	Labile	Stabile	Labile
	n = 20	n=19	n=19	n=20
Trials to Habituation (SD)	6.15 (5.84)	10.15 (5.04)	5.26 (3.23)	11.05 (5.52)
Absolute Rate of Habituation (SD)	-.357 (.392)	-.748 (.628)	-.291 (.275)	-.602 (.431)

1. Trials to habituation

A 2x2 Condition (fixed ISI/variable ISI) x Electrodermal Lability (stabile/labile) analysis of variance (ANOVA) was performed revealing a significant main effect for electrodermal lability [F (1,74)=18.48, p=.0001] with electrodermally labile subjects taking more presentations to habituate than the stabile subjects.. Both the condition main effect [F(1,74)=0.00, p=.99) and the condition x electrodermal lability interaction did not approach statistical significance [F (1,74)=.61, p=.44).

2. Uncorrected rate of habituation

An 2x2 Condition x Electrodermal Lability ANOVA was also performed with the uncorrected habituation rate as the dependent variable. Again a significant electrodermal lability main effect was found [F(1,74)=11.89, p=.0009] but both

the condition main effect [F (1,74)=0.66, p=.39) and condition x electrodermal lability interaction [F (1,74)=2.29, p=.13) were not significant.

3. Omission response

Subjects showing a greater maximum response in the omission window compared to the control window were scored as having emitted an omission response while subjects showing a larger response in the control window than in the omission window were scored as having emitted a "control window response".

TABLE 3.2

Frequency of stabile and labile subjects emitting omission or control window responses in constant and variable ISI conditions.

	Constant ISI		Variable ISI		
	Stabile	Labile	Stabile	Labile	Total
Response Type	n = 20	n=19	n=19	n=20	78
Omission	2	6	5	6	19 .
Control Window	4	7	0	9	20

Thirty-nine (50%) of the subjects emitted a response in at least one of the omission or control response windows. In 19 (49 %) of cases the response in the omission window was larger than that emitted in the control window while in 20 cases the opposite result was observed, a difference clearly not reaching statistical significance. Following the variable ISI stimulus series 11 subjects emitted a larger response in the omission window compared to the control window with 9 subjects showing the reverse pattern. These frequencies are not significantly different (Binomial test p>.10). The results for subjects receiving

the constant ISI series were similar with 8 subjects showing a larger response in the omission window and 11 showing a larger response in the control window. These frequencies were not significantly different (Binomial test p>.10).

The ratio of subjects showing larger responses in the omission and control windows was 7:4 for the stabile subjects and 12:16 for the labile subjects. In neither case is there a significant difference between the two frequencies by the binomial test. A χ^2 analysis failed to provide evidence of significant differences in the proportion of omission responses compared to control window responses for the four electrodermal lability/ISI variability conditions (χ^2 (df=3)=6.32, p=.09).

In summary, analysis of responses in the omission and control windows provided no significant evidence for an omission response. There was no significant tendency for subjects to respond more strongly in the omission window than in the control window.

DISCUSSION

This study does not support the notion that presentation of stimulus at a variable ISI retards habituation when compared to the same stimulus presented at a fixed ISI. Neither the trials to habituation nor the uncorrected habituation rate measures showed a significant difference between the fixed and variable ISI conditions.

There is no evidence for the presence of an omission response in this study. When compared to a control window placed between previous stimuli there is no evidence that there is a tendency for responses to be more likely to occur in the omission window. The most likely explanation of this data is that the responses are simply non-specific responses which are randomly distributed in time and that the omission window is no more likely to contain these responses than any other window which is not related to a stimulus.

The analysis used in this study is slightly different to that used by O'Gorman and Lloyd (1984). Of their total of 48 subjects 25 (18 labiles, 7 stabiles) responded more strongly in the omission window compared to the control window. O'Gorman and Lloyd (1984) however do not report the number of people responding more strongly in the control window compared to the omission window. Without reporting this number these data do not conclusively suggest the presence of an omission response.

Barry (1984) using a similar analysis to the present study, however, found that significantly more subjects (22 of 31 responding in either interval) responded more strongly in the omission compared to the control window. The results of the current experiment are at odds with these results with only 19 of 39 subjects emitting a response in either window showing the larger response in the omission window. One difference between Barry (1984) and the current study is that Barry used an onset to onset interval of 15s while the corresponding interval in the current study was 24 seconds. It is possible that the shorter interval in the Barry (1984) study led to a stronger temporal trace than the current study, which resulted in a more reliable elicitation of the orienting response.

The suggestion that electrodermally labile subjects would have more sensitive temporal encoding processes than electrodermally stabile subjects is not supported by this experiment. While labile subjects emitted more responses in the control and omission windows, there was no tendency for labile subjects to differentially emit more responses in the omission window compared to the control window. Similarly, the results of the ISI variability manipulation on rate of habituation do not show a significant interaction between condition and electrodermal lability, a result which would have been expected if the electrodermally labile subjects possessed particularly sensitive mechanisms of temporal encoding.

With regard to the various habituation theories discussed earlier, these results are consistent with the notion that temporal encoding is not an important part of habituation of the OR. Briefly, these results suggest that a valid model of habituation need not necessarily provide an extrapolatory temporal trace of previous stimulation, thus removing one of the largest advantages of comparator-type habituation models over the dual-process model of habituation. Final conclusions however must be reserved until the completion of the series of experiments, which further tested the necessity of temporal encoding processes in a valid model of OR habituation.

CHAPTER 4

Experiment 2

The Effect of ISI Variability and Omission of S2 Following S1-S2 Pairs at a Long ISI

Experiment 2 was a more sophisticated attempt to answer the same questions posed in Experiment 1. Experiment 2 utilised pairs of stimuli (S1-S2 pairs) as these allow more specific conclusions regarding the type of temporal encoding (if any) implicit in the internal processes concerned with habituation of the OR.

Postulated processes of temporal encoding in theories of habituation of the orienting response can be classified according to two types of mechanism. The first type is encoding of the period of stimulus presentation where the resulting expectancy is that "stimulus X is presented at a period of 30 seconds". The second type is the encoding of the time of stimulus presentation by reference to a cuing event or context, resulting in the expectancy that "stimulus X is presented 5 seconds after stimulus Y". If, as in comparator theories of habituation (Öhman,1979; Sokolov, 1969; Wagner,1978), the magnitude of the orienting response is proportional to the difference between afferent stimuli and the expected stimulation predicted by an internal neuronal model, then either of these mechanisms will lead to the results that; (a) the orienting response to stimuli presented at a variable or irregular interstimulus interval (ISI) will habituate more slowly than stimuli presented at a constant or regular interstimulus interval, and (b) that an orienting response elicited by stimulus omission will be larger in the constant interstimulus interval condition than in the variable interstimulus interval condition. The existence or otherwise of the effect of interstimulus interval variability on habituation rate and the presence and magnitude of the response to stimulus omission are both vital indicators of the type of mechanism of temporal encoding used by humans.

The use of a series of presentations of a single stimulus in the study of the effects of both interstimulus interval variability and omission responses, as in Experiment 1, did not allow differentiation of the type of temporal encoding process, if evidence for one had been observed. This is due to the fact that the processes of encoding by period and encoding by reference to a cuing event are identical in the case of a series of presentations of a single stimulus. For example a single stimulus presented at a period of 30 seconds necessarily occurs 30 seconds after a salient cue (the last stimulus presentation). The current experiment seeks to address this problem by the use of a stimulus series involving pairs of stimuli. Using this method the effects of both variable period of stimulation and variable temporal cuing by a salient event on rate of habituation and omission responding will be independently assessed.

METHOD

Subjects

Seventy-two undergraduate volunteers participated in the experiment. There were 54 female and 18 male subjects.

Apparatus

Skin conductance was recorded by applying a constant voltage of 0.5V across domed Ag-AgCl electrodes with .05M NaCl electrolyte. The electrodes were placed on masked areas on the distal phalanges of the index and second fingers of the subject's left hand. The electrodes were connected to a custom-built battery powered bridge which was connected to a Grass 7 DAG preamplifier with a recording sensitivity of 0.02 μS/mm pen deflection. Respiration was recorded using a Phipps and Bird pneumatic bellows connected to a Grass 7PRTE transducer and 7 DAG preamplifier.

The auditory stimulus was a 1000 Hz tone with a 30 ms risetime and a duration of 4 seconds. It was presented binaurally through Sony DR-7 stereophonic

headphones at an intensity of 70 dB (SPL) which was calibrated by a Bruel and Kjaer model 2205 sound level meter. The light stimulus had an intensity of 142 cd/m^2 and was produced using a Kodak Carousel slide projector with a Polaroid filter and Gerbrands model G1166 tachistoscopic shutter. The light stimulus also had a duration of four seconds. The stimulus was back projected onto a ground glass screen (15 X 18 cm) set into the wall of the experimental cubicle. The screen was situated 160 cm in front of the subject at eye level. The ambient light intensity of the subject room was 0.9 cd/m^2 while the ambient temperature was 23°C. Light intensity was measured by a Tektronix light meter. Stimulus presentation was controlled by an IBM compatible personal computer.

Procedure

Upon arrival in the laboratory subjects were told that skin conductance and respiration would be measured and that the experiment would consist of an approximately five minute rest period which would be followed by the presentation of some lights and tones. Subjects were instructed that they would not to be required to remember anything of the experiment or make any deliberate response to the stimuli, they merely had to relax with their eyes open while the experiment was in progress.

All subjects initially received a four minute prestimulation period during which the experimenter stabilised the physiological recordings and assessed electrodermal lability.

Subjects then received 16 preomission S1-S2 pairings followed by one S1 alone (S2 omission trial) and one S1-S2 re-presentation trial. The stimulus onset asynchrony (onset S1 to onset S2, SOA) and S2 period (onset S2 to onset S2) varied between the control and two experimental groups. For all subjects S1 was the light and S2 was the tone.

The design of the experiment involved the comparison of the habituation of the S2 and response to S2 omission in three different experimental conditions. The first or control condition involved the presentation of the stimulus pairs at a constant interstimulus interval and S2 period. The second or variable period condition preserved a constant SOA for all stimulus pairs but the S2 period varied. The third or variable SOA condition maintained a constant S2 period for all stimulus pairs but used variable SOAs.

For the control group the SOA for the preomission and re-presentation trials was 8 seconds. The S2 period for all pairs (except for the omission and re-presentation trials) was 33 seconds. The time interval between the omission and re-presentation trials (offset S1 alone to onset S1) was 29 seconds.

The first experimental group (variable period) also had a constant SOA of 8 seconds for all trials except for the S1 alone trial. The interstimulus interval for the 16 preomission stimulus pairs was 26 seconds for five instances, 33 seconds for five instances and 40 seconds for the remaining five presentations. The order of the three interstimulus intervals was randomised in triplets (where each period appeared once in the first three periods, once in the second three and so on). Once this sequence had been generated two additional sequences were created by substituting firstly, 26 for 33, 33 for 40 and 40 for 26, and secondly, 26 for 40, 40 for 33 and 33 for 26 in the original sequence. Each sequence was used for one-third (8) of the subjects in this group. The S1 alone omission trial was presented 21 seconds after the offset of the S2 of the preomission trial 16 and the re-presentation S1-S2 trial 29 seconds after the offset of the S1 alone trial.

The second experimental group (variable SOA) had the same interval of 33 seconds from the onset of one S2 to the onset of the next S2 for all 16 preomission trials. The SOAs for these 16 trials varied between five trials of 5 seconds, six trials of 8 seconds and five trials of 11 seconds. The first trial

always had an SOA of 4 seconds while the remaining 15 SOAs were randomised in triplets with three counterbalanced series generated by the same method described in reference to the first experimental group. The S1 alone omission trial was presented 21 seconds after the offset of the S2 of the preomission trial 16 and the re-presentation trial 29 seconds after the offset of the S1 alone trial.

Figure 4.1 shows diagrams of the stimulation regimens for the three experimental groups.
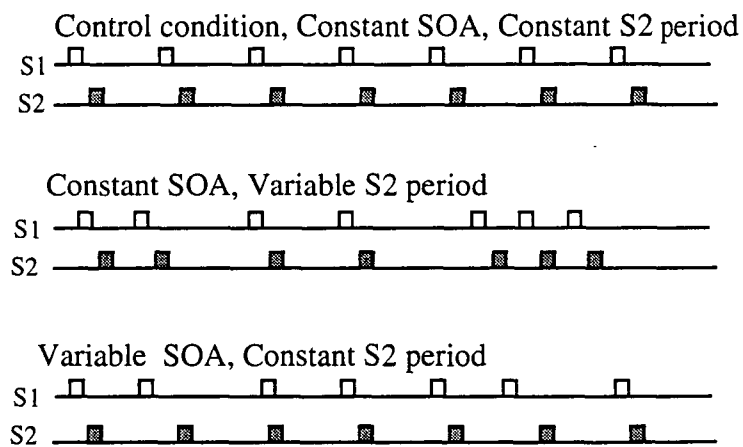
Figure 4.1. Temporal relationship between S1 and S2 stimuli presentations for control, variable period and variable SOA conditions.

Scoring

Non -specific responses (NSRs) were scored by counting the number of electrodermal responses greater than 0.02 μS occurring in the 3 minute rest period before the first stimulus. Subjects were split at the median NSR score to form two lability groups: stabile (below median) and labile (above median).

Responses considered to be due to stimuli during the habituation series were SCRs greater than 0.02 μS occurring 1 to 5 seconds after stimulus onset.

Responses associated with unusual respiratory activity were excluded from further analysis. Trials to habituation for the S2 was scored as the presentation number of the S2 where the following three S2 presentations did not show responses greater than 0.02 μS. The uncorrected habituation rate was calculated as the slope of the regression line predicting the skin conductance response to the S2 from log presentation number across all 16 pre-omission trials (Lader & Wing, 1966).

Responses to stimulus omission were measured by comparing the maximum electrodermal response in a time window during which an omission response is hypothesised to occur to the maximum electrodermal response in a control window where no response would be expected to occur (Barry, 1984, O'Gorman & Lloyd, 1984). The omission window was defined from five to thirteen seconds after the offset of the S1 on the S1 alone omission trial while the control window was in the non-stimulated interval from one to nine seconds following the offset of the S2 presentation on trial 16. The control window thus occurs the same interval following the previous stimulus as does the omission window and controls for differences in responding which could be due to proximity of preceding stimuli (Grings & Schell, 1969). A subject showing a larger response in the omission window than in the control window was scored as exhibiting an omission response while subjects showing a larger response in the control window were scored as exhibiting a control window response.

RESULTS

A preliminary analysis of variance was performed to determine if there were significant differences in the levels of electrodermal lability in the three experimental conditions. A significant difference in electrodermal lability could have resulted in contamination of both the habituation rate and omission response measures used in this experiment. The mean frequency of non-

specific responses for all subjects was 11.03 (SD=9.86). The mean frequency of non-specific responses in each of the three conditions are presented in Table 4.1. Differences between the three conditions did not approach significance [$F_{(2,69)}=.16$, $p>.85$].

TABLE 4.1

Mean (SD) frequency of non-specific responses for the three conditions.

Condition	n	Mean Frequency
Control	24	11.96 (10.68)
Variable ISI	24	10.58 (8.92)
Variable SOA	24	10.58 (10.24)

1. Rate of Habituation

Figure 4.2 shows the SCR in each condition for the sixteen pre-omission presentations for subjects in each condition and lability group.
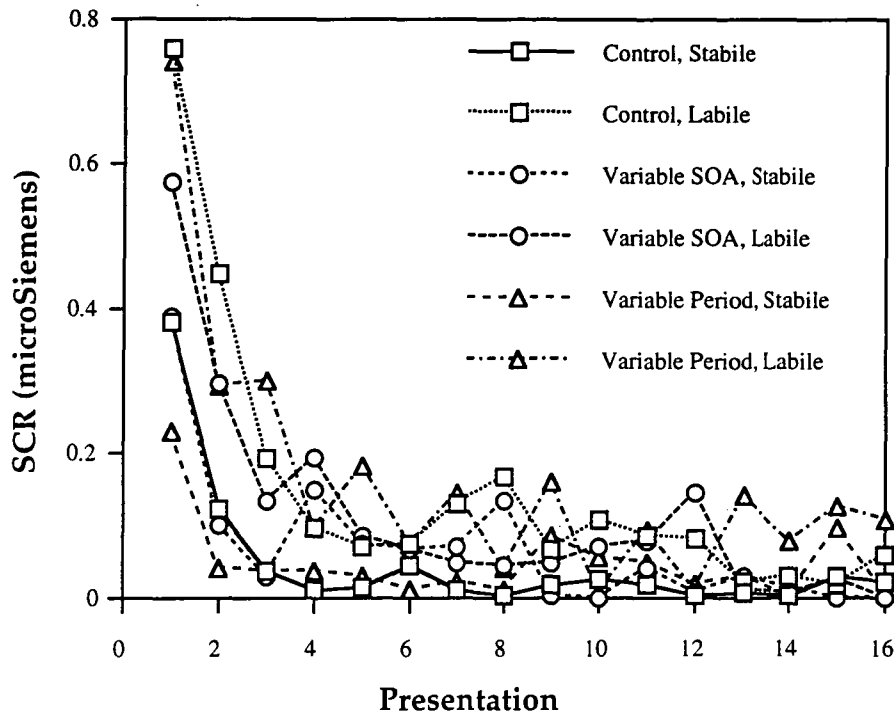
Figure 4.2. SCR on each S2 presentation for all conditions and both electrodermal lability groups (n=12 in each cell).

Separate 2 x 3 Lability (Stabile/Labile) x Condition (Control/Variable period/Variable SOA) analyses of variance were performed for the dependent variables of trials to habituation and uncorrected habituation rate.

The analysis of variance for trials to habituation revealed a significant main effect for electrodermal lability [$F(1,66)=17.22$, $p<.0001$] while the main effect for condition [$F(2,66)=1.14$, $p=.33$] and the condition x electrodermal lability interaction [$F(2,66)=.53$, $p=.59$] were both non-significant. These results are illustrated in Figure 4.3 and Table 4.2 which show that there was a significant tendency for trials to habituation to be higher in the labile group compared to the stabile group but no significant differences in the scores for the three experimental conditions.
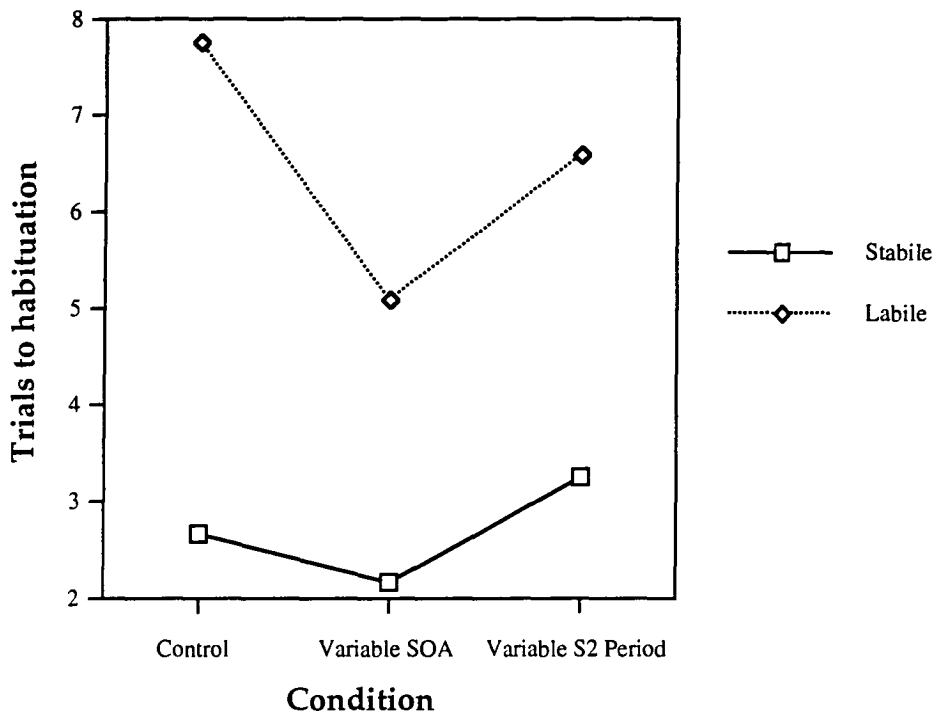
Figure 4.3. Trials taken to reach habituation for electrodermally stabile and labile subjects under each experimental condition.

TABLE 4.2

Mean (SD) trials to S2 habituation for each lability group and experimental condition.

	Stabile Subjects (n=12)	Labile Subjects (n=12)
Control	2.67 (2.57)	7.75 (5.12)
Variable SOA	2.17 (2.08)	5.08 (3.73)
Variable Period	3.25 (4.69)	6.58 (4.06)

A similar analysis of variance was performed with uncorrected habituation rate as the dependent variable. The group means of uncorrected rate of habituation

for each level of electrodermal lability for the three experimental groups are presented in Figure 4.4 and Table 4.3.
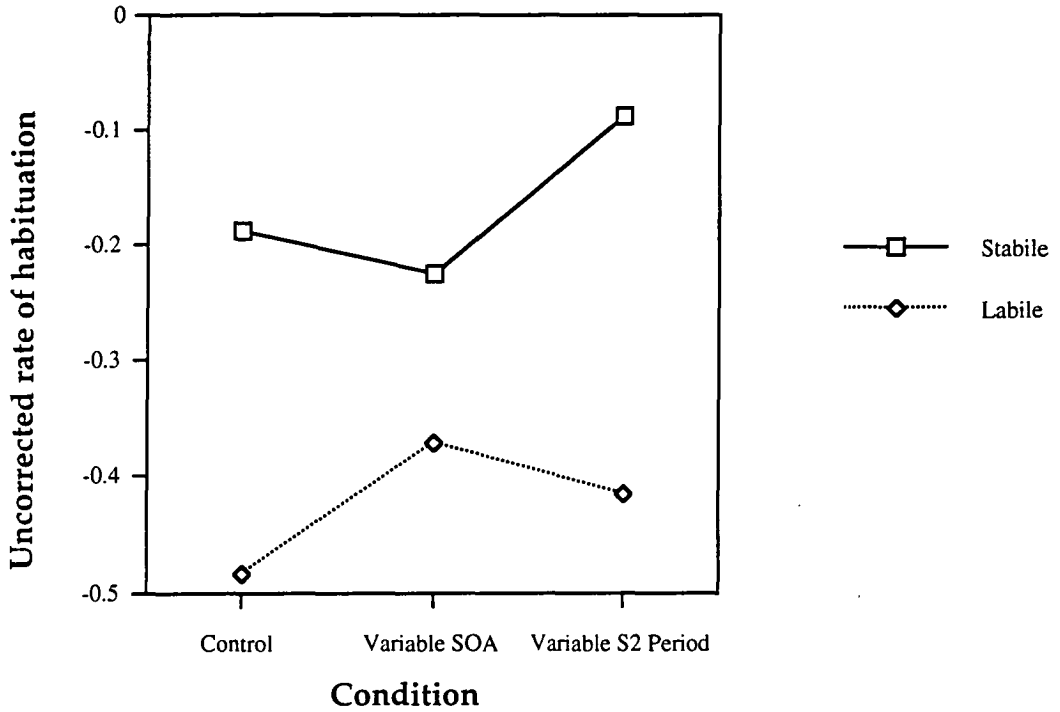
Figure 4.4. Uncorrected rate of habituation for electrodermally stabile and labile subjects under each experimental condition.

TABLE 4.3

Mean (SD) uncorrected rate of habituation for each lability group and experimental condition.

	Stabile Subjects (n=12)	Labile Subjects (n=12)
Control	-.188 (.271)	-.485 (.283)
Variable SOA	-.226 (.267)	-.372 (.368)
Variable Period	-.090 (.125)	-.416 (.436)

Again there was a significant main effect for electrodermal lability [F(1,66)=12.56, p=.0007] but non significant effects for experimental condition [F(2,66)=.44, p=.64] and the condition X electrodermal lability interaction [F(2,66)=.59, p=.55]. Figure 4.4 however indicates a slightly surprising result with the steepest rate of habituation being found in the labile group rather than the stabile group. It would have been expected that the condition with the lowest number of trials to habituation would have shown the steepest rate of response decrement. Two reasons for the unexpected result can be found. Firstly, there was a higher initial response in the labile group compared to the stabile group which would provide a steeper rate of response decrement if both groups habituated in the same number of presentations. Secondly, the uncorrected rate of habituation was calculated across all stimulus presentations (Lader & Wing, 1966). While the stabile subjects may have shown a steeper rate of response decrement on the first few presentations the long "tail" of zero responses following habituation would tend to result in attenuation of the rate of S2 decrement. There were no significant effects of experimental condition on the uncorrected rate of habituation.

2. Response to stimulus omission

Table 4.4 shows the number of subjects in each experimental group exhibiting either an omission response or a control window response.

TABLE 4.4

Frequency of omission and control window responses for each experimental group (n=24 per group).

Condition	Omission response	Control Window Response
Control	8	5
Variable ISI	4	2
Variable SOA	5	4

These figures indicate that 28 (39%) of the subjects in this study exhibited a response greater than 0.02 μS in either the control or omission response windows. Seventeen (61%) of these subjects showed a larger response in the omission window compared to the control window. This proportion did not reach statistical significance (Binomial test p= 0.17). The proportions of omission and control window responses in the three experimental conditions were not significantly different (χ^2 =.66, df=2, p>.05).

These results are somewhat at odds with those reported in earlier studies. The SCR at S2 omission was reported to be significantly larger than the SCR to an S2 presented at the same time in a control series to a second group of subjects in three experiments reported in Siddle et al. (1987), in both experiments utilising SCR reported in Siddle and Packer (1987), and in two of three experiments in Siddle (1985). If a similar analysis is performed on the current data, in which the magnitude of the largest response in the omission window is compared to the largest response in the control window, no significant difference is found. Two possible differences between this and the current study are the use of a between-subjects compared to a within-subjects design and the interval between the S1 and S2 components of a pair in each study. In each of Siddle et

al. (1987), Siddle and Packer (1987) and Siddle (1985) the onset of the S2 was concurrent with the offset of the S1, while in the current study there was, on average, a 4 second interval between the S1 and S2. It is possible that the failure of the current study to provide significant evidence of a response to omission of an S2 following a number of S1-S2 pairs is due to the SOA utilised in the study. The longer interval between the S1 and S2 in this study may lead to the omission of the S2 not being as significant an event as it was in the other studies cited.

A further analysis of stimulus omission was conducted to see if the amount of dishabituation caused by the omission of the second stimulus on the omission trial was equal for the three experimental conditions. Dishabituation caused by the stimulus omission was measured by subtracting the response to the S2 on the pre-omission trial (trial 16) from the response to the S2 on the post omission trial (trial 18). A total of 24 subjects showed a positive amount of dishabituation. Nine received the control condition, eight the variable interstimulus interval condition and seven the variable stimulus onset asynchrony condition. The differences in proportions were not significant. The mean amount of dishabituation across all subjects was .051 μS (SD=.195). This result was significantly different from zero at the .05 level of significance [$F(1,71)=4.85$, $p<.05$]. An electrodermal lability X condition analysis of variance with the amount of S2 dishabituation (response to S2 on post-omission trial-response to S2 on pre-omission trial) as the dependent variable was used to determine if the amount of dishabituation was systematically related to electrodermal lability or experimental condition. The analysis revealed a non-significant main effect for electrodermal lability [$F(1,66)=3.47$, $p=.067$], and no significant main effect for condition and no significant lability X condition interaction. Therefore, it was concluded that the amount of dishabituation of the response to the S2 caused by the omission of the S2 on the seventeenth trial,

and measured by the response to the re-presentation of the S2 was not significantly affected by the experimental treatments.

A similar analysis was performed on dishabituation of the response to the S1 following the omission trial. Twenty-four subjects showed a positive amount of S1 dishabituation, eight from the control group, twelve from the variable interstimulus interval condition and four from the variable stimulus onset asynchrony condition. A one-group analysis of variance showed that the mean amount of S1 dishabituation across all subjects was not significantly different to zero [$F(1,71)=.142$, $p>.05$].

DISCUSSION

The main conclusion that could be drawn from this experiment was that altering the temporal predictability of a stimulus by period encoding or encoding by temporal cuing by another event does not significantly affect the rate at which the orienting response to that stimulus habituates. The non-significant tendency for subjects to respond more strongly in the S2 omission window was surprising but may have been a result of the increased S1-S2 interval compared to previous studies. There was, however, significant dishabituation of the response to the S2 caused by its omission. The magnitude of this dishabituation was not related to the variations in temporal predictability of the S2 examined in this study. Electrodermal lability was found to be linked to the rate of habituation, as evidenced by the trials to habituation and uncorrected rate of habituation measures.

The existence of an effect of interstimulus interval variability on habituation rate is not supported by this experiment and leads to the conclusion that the mechanism of temporal encoding employed in the formation of a neuronal model for elicitation of the orienting response does not precisely encode either the period between successive stimulus presentations or the delay between a

stimulus and a significant cuing event at the time intervals used in this experiment.

The finding that there was significant dishabituation of the electrodermal response to the S2 when it was re-presented on the trial following the omission trial is in agreement with the data of Siddle (1985), Siddle et al. (1983), Siddle and Packer (1987), Siddle and Hirschorn (1986) and Siddle, Remington, Kuiack and Haines (1983) and appears to be a well documented finding in the case of omission of one element of a stimulus pair.

On the basis of the results obtained in this experiment it was concluded that there was no effect, given the parameters used in this experiment, of reducing the temporal predictability of a stimulus' presentation by either variation of its period of presentation or by variation of its temporal predictability from a cuing stimulus, on the habituation rate of that stimulus. It was also concluded that the omission of a stimulus results in dishabituation of the response to the omitted stimulus when it is re-presented. These results are compatible with the various comparator based theories of habituation proposed by Sokolov (1963), Öhman (1979) and Wagner (1978) but they indicate that the mechanisms of the temporal encoding used in these theories to construct their internal trace of past stimulation, and hence expectancies of future stimulation, are not temporally precise. It is possible that these expectancies are either temporally imprecise and able to accept large variations in the time of presentation of a stimulus without creating large discrepancies between the internal trace of past stimulation and the current afferent stimulation or that the expectancies are simply ordinal rather than explicitly temporal. It may be sufficient that S2 eventually occurs after S1 rather than there being any requirement that the temporal relationship between the two is exactly the same as on previous presentations. While it could be argued that the re-presentation of the S1 after the omission trial also breaks the ordinal relationship encoded by the previous

presentations (the previous S1s, except the first, had all been preceded by S2s) and may thus be expected to exhibit significant dishabituation it may be that this ordinal expectation is, by virtue of the longer S2-S1 latency compared to the S1-S2 latency, is not as strongly encoded as the S1-S2 expectation and thus no significant dishabituation is observed when the expectation is violated.

CHAPTER 5

A change in methodology

The overall conclusion drawn from the first two experiments was that there seemed to be little evidence of extrapolatory encoding of temporal information in habituation of the OR. In Experiment 1 there was no effect for ISI variability on rate of habituation and no evidence of response to complete stimulus omission. Experiment 2 showed that manipulation of the predictability of a stimulus by varying either its own period, or varying the interval between it and a cuing stimulus, did not affect the rate of habituation. Experiment 2 however did provide evidence that, following a number of S1-S2 pairs, omission of the S2 resulted in dishabituation of response to the S2 when it was next presented.

It was stated earlier that the ISIs chosen for these first two experiments were chosen with the proviso that they were sufficiently long so that the response to one stimulus presentation would not overlap the response to the next. Experiments using event-related potentials (ERPs) as the dependent measure have however been generally conducted with much shorter (commonly 1-2 s) ISIs. Some components of the ERP share some properties with autonomic measures of the OR (Barry, Cocker, Anderson, Gordon, & Rennie, 1992; Kenemans, Verbaten, Sjouw, & Slangen, 1988; Näätänen & Lyytinen, 1989) and there is some evidence that certain ERP components may be sensitive to stimulus omission (Simson, Vaughan & Ritter, 1976). It would therefore be useful if studies using SCR as the dependent variable could be conducted at shorter ISIs than was previously the case.

It is also theoretically plausible that short ISIs could be encoded by subjects more easily, rapidly and precisely than longer ISIs. For example, if ISI was encoded by alterations in the spontaneous firing rate of a pacemaker neuron then there could be bounds on the degree of alteration possible from the

spontaneous firing rate, these bounds could enable the encoding of short ISIs but not longer ISIs. Alternatively, ISI could be encoded by delay or expectancy loops which were incapable of encoding ISIs longer than a certain duration. A more complete examination of temporal predictability and habituation of the OR would include experiments using both short and long ISIs rather than the longer ISIs of the previous studies.

To achieve the aim of investigating the effect of temporal predictability on habituation of autonomic indices of the OR at short ISIs a change in the normal methodology was required. In Experiments 3 and 4 a new scoring technique was used in which it was not necessary to relate each response to a particular stimulus, removing the requirement that stimuli are separated by long time intervals.

The essence of the technique is similar in concept to, but not derived from, signal-detection theory in that all responses occurring within an interval of a given length are scored and then the measures obtained from this interval are compared with indices scored in other intervals of equal duration. For example, in an experiment investigating the effect of a variable ISI on the OR at ISIs of the order of 1s, there might be a series of stimuli presented at a constant ISI which is then replaced with a series of stimuli presented at a variable ISI. With such a short ISI it is impossible to accurately relate each SCR to a particular stimulus presentation so we would rather define two equal length scoring windows, say the final ten seconds of the constant ISI series and the first ten seconds of the variable ISI series. We would then compare the various SCR indices within each window to test if the change from a constant ISI to a variable ISI resulted in a significant change in SCR activity. The SCR indices measured in each scoring window employed in the studies are maximum SCR amplitude (the largest response), the total SCR activity (sum of all SCRs in

scoring window) and the number of SCRs in the window greater than a particular threshold (0.02 μS in the current studies).

One advantage of this scoring technique when it is coupled with a repeated-measures design is that the occurrence of non-specific SCRs (NSRs) does not differentially affect one condition compared to another. If NSRs occur randomly over time then they are just as likely to fall in the scoring window corresponding to any of the different conditions and tend simply to add a random, background level of activity to the SCR scores which is equal in all conditions and does not affect the differences between the conditions. In between-subjects designs, subjects with different levels of electrodermal lability (number of NSRs emitted in a given interval) may be differentially assigned to conditions and either affect the testing of the effect of the independent variable, or necessitate statistical adjustment of the scores.

Barry, Feldmann, Gordon, Cocker and Rennie (1993) presented a technique that was also designed to allow the scoring of SCRs at short ISIs. While their technique was developed independently of the one used in this thesis there are some points of overlap between the two methods. The technique of Barry et al. (1993) has two parts. The first is, like the normal ERP scoring technique, the averaging of a number of SCR traces to calculate an average trace which is then scored for features. The second part of the technique involves the extrapolation of the scoring baseline from a falling line resulting from an early large response. This second part of the technique requires an assumption which is also employed in the technique developed in this thesis, that is, that the observed level of skin conductance is the sum of any responses which may be contributing to it. In the case of Barry et al. (1993) this assumption means that the difference between the extrapolation of the falling baseline and the actual skin conductance level can be attributed to a response to a stimulus. In the technique developed in this thesis the assumption allows any difference in the

size or number of responses between one scoring interval and another interval of equal duration can be attributed to differences in stimulation in the two windows, assuming equal frequency of non-specific responses in each interval.

The differences between the technique devised by Barry et al. (1993) and that devised in this thesis are worthy of discussion. One advantage claimed by Barry et al. is that their technique allows the identification of response to individual stimuli, even at short ISIs. It is difficult, however, to see how this is possible when the possible scoring window for each stimulus in their study was only 1.14 seconds. Scoring windows for SCRs are commonly of the order of two to five seconds in duration (e.g., O'Gorman & Lloyd, 1984; Barry, 1984). Barry et al. suggest an interval of one to three seconds after stimulus onset (Barry, 1990, cited in Barry et al. 1993). If the response latency exceeds the scoring window duration of 1.14 seconds then the response would be misallocated to a stimulus in the Barry et al. scoring system.

A second area of concern with the technique reported in Barry et al. (1993) lies in the effect of non-specific responses on very small responses attributed to stimuli in their study. Responses reported in their paper are of the order of 1-10 nS (nanoSiemens), while the lowest criterion for a response to a stimulus in other studies is usually of the order of 0.01 or 0.02 μS. Barry et al. are, therefore, scoring responses up to ten times smaller than those normally scored in electrodermal studies. Each response in Barry et al. (1993) is the result of the averaging of fifteen stimulus trains. A non-specific response of 0.10 μS occurring in just one of these fifteen stimulus trains would, after averaging, result in a response of six nS being observed in the average trace. In Experiment 2 it was observed that subjects emitted an average of 11 non-specific SCRs in a three minute period, with their amplitude often exceeding 0.10 μS. Given that the total scoring period for each subject in Barry et al. (1993) was approximately 195 seconds in duration (15 trains, each of 13

seconds duration) and no adjustment was made for the occurrence of non-specific responses, it is possible that a considerable number of the responses attributed to the stimuli in this study were in fact non-specific responses.

The technique devised for the current thesis was expected to be more robust to the occurrence of non-specific responses than that proposed by Barry et al. (1993). The comparison of responses in two scoring windows from a single subject should, assuming that non-specific responses are randomly occurring in time, mean that any observed difference between the two windows is due to differences in the response to actual stimuli. The current technique cannot attribute responses to individual stimuli but it is uncertain that this is successfully accomplished by Barry et al. It is argued, therefore, that the technique presented in the current thesis provides a more robust test of the electrodermal response to series of stimuli presented at a short ISI than that proposed by Barry et al. (1993).

For the reasons outlined above the final two experiments of this series utilised short ISIs, repeated-measures designs and used the scoring techniques just described. This methodological combination allowed the investigation of the hitherto unknown effects of temporal predictability on habituation of the SCR at short ISIs.

Because of the change in scoring necessitated by the shorter ISIs employed in the final two experiments there is a possibility that any observed difference in results between the long and short ISI experiments may be a reflection of the difference in methodology rather than a real difference in the phenomena at the different ISIs. The data from the first two longer ISI experiments were therefore re-analysed using similar techniques to those devised for the shorter ISI experiments.

While the first two experiments were designed as between-subjects designs and the following two experiments were designed as within-subjects designs the scoring technique can be adapted to both designs. In the case of Experiment 1 two groups of subjects received series of single stimuli at either a constant or a variable ISI. If the subjects were encoding the ISI of the series in a neuronal model and comparing the contents of the neuronal model with the afferent stimulation, then it would be expected that when the series was presented at a variable ISI there would be a greater number of SCRs (compared to the constant ISI series) emitted when the contents of the neuronal model failed to match the afferent stimulation. There would similarly be expected to be a larger total sum of SCR activity in the variable ISI condition. The maximum SCR magnitude would not necessarily be expected to be larger in either condition, it would probably occur in the first few stimulus presentation, before temporal predictability could be established.

The scoring window used in the re-analysis of Experiment 1 was defined as the entire duration of the experiment, from the onset of presentation 1 to the offset of presentation 18 (the pre-omission presentation). To reduce skewness the raw values of maximum SCR, total SCR activity and number of SCRs were square-rooted before analysis. The mean values of each variable for each condition and lability group are presented in Table 5.1.

TABLE 5.1

Mean (SD) square-rooted maximum SCR, total SCR and SCR number for constant and variable ISI groups and stabile and labile subjects.

		Constant ISI		Variable ISI	
	Unit	Stabile	Labile	Stabile	Labile
Maximum SCR	$\sqrt{\mu S}$	.65 (.42)	1.05 (.34)	.71 (.33)	1.01 (.26)
Total SCR	$\sqrt{\mu S}$	1.29 (1.13)	2.77 (1.33)	1.22 (.67)	2.76 (1.17)
Number of SCRs		2.83 (1.63)	5.54 (1.41)	2.99 (1.41)	5.72 (1.36)

2 x 2 Condition (Constant ISI/Variable ISI) x Lability (Stabile/Labile) analyses of variance were performed for each dependent variable. In each case there was a significant main effect for electrodermal lability (Square root maximum SCR: $F(1,74)=21.25$, $p=.0001$; Square root total SCR: $F(1,74)=36.07$ $p=.0001$; Square root SCR number: $F(1,74)=67.84$, $p=.0001$) but a non-significant main effect for condition (Square root maximum SCR: $F(1,74)=.02$, $p=.89$; Square root total SCR: $F(1,74)=.03$, $p=.87$; Square root SCR number: $F(1,74)=.26$, $p=.61$) and a non-significant Condition x Lability interaction (Square root maximum SCR: $F(1,74)=.38$, $p=.54$; Square root total SCR: $F(1,74)=.02$, $p=.89$; Square root SCR number: $F(1,74)=.001$, $p=.98$). In summary, these results are in agreement with those obtained when Experiment 1 was analysed by the more traditional methods. There was no evidence to suggest that temporal encoding was being performed by a neuronal model as the stimulus series was being presented. The existing analysis of the response to stimulus omission manipulation in Experiment 1 is similar to the revised technique so the original data were not re-analysed .

The re-analysis of Experiment 2 was conducted in a very similar way. Maximum SCR magnitude, total SCR activity and number of SCRs were recorded for the entire duration from the onset of the first stimulus pair to the offset of the last pair before the omission of the S2 (refer to the description of Experiment 2 for a full description of the stimulus series). Table 5.2 shows the mean of each variable for the control, variable SOA and variable S2 period conditions.

TABLE 5.2

Mean (SD) square-rooted maximum SCR, total SCR and SCR number for control, variable SOA and variable S2 period conditions for both stabile and labile subjects.

	Unit	Control		Variable S2 Period		Variable SOA	
		Stabile	Labile	Stabile	Labile	Stabile	Labile
Maximum SCR	$\sqrt{\mu S}$	.76 (.29)	1.00 (.25)	.78 (.28)	.99 (.24)	.77 (.25)	1.07 (.24)
Total SCR	$\sqrt{\mu S}$	1.53 (.81)	3.41 (.90)	1.67 (.97)	2.96 (1.00)	1.66 (.88)	3.71 (1.13)
Number of SCRs		3.53 (1.89)	7.14 (1.62)	3.44 (1.31)	6.18 (1.54)	3.84 (1.81)	7.04 (1.70)

3 x 2 Condition (Control/Variable ISI/Variable SOA) x Lability (Stabile/Labile) analyses of variance were calculated for each dependent variable. In each case there was a significant main effect for lability (Square root maximum SCR: $F(1,66)=17.26$, $p=.0001$; Square root total SCR: $F(1,66)=59.95$, $p=.0001$; Square root SCR number: $F(1,66)=66.49$, $p=.0001$) but no significant main effect for condition (Square root maximum SCR: $F(2,66)=.16$, $p=.85$; Square root total SCR: $F(2,66)=.93$, $p=.40$; Square root SCR number: $F(2,66)=.99$, $p=.38$) and no significant condition x lability interaction (Square root maximum SCR: $F(2,66)=.19$, $p=.83$; Square root total SCR: $F(2,66)=.1.03$, $p=.36$; Square root SCR number: $F(2,66)=.41$, $p=.67$). The omission data were not re-analysed.

It was clear that the use of the altered scoring technique did not result in different conclusions in the first two experiments. In both cases the level of temporal predictability does not significantly effect the level of SCR activity

when assessed by any of the three measures. It was then possible to utilise the scoring techniques at shorter ISIs, and draw direct comparisons with the same measures scored during experiments with longer ISIs.

CHAPTER 6

Experiment 3

The Effect of ISI Variability and Complete Stimulus Omission on Habituation at a Short ISI

Experiment 3 is essentially a replication of Experiment 1 but using a shorter ISI. That is, the experiment tests the effects of ISI variability and complete stimulus omission on the electrodermal response with ISIs in the order of 1 to 2 seconds rather than the 10 to 20 seconds of previous studies. It was also designed to test whether the administration of instructions suggesting to the subject that the stimulus series would have the properties of a clock would have an effect on temporal processing. Such contextual cuing may be predicted to have an effect on the neuronal model by a mechanism like the retrieval-generated priming of Wagner's priming theory (1978) or alternatively affect the significance attached to the series by the subject and mediate the elicitation of a response to stimulus omission (Barry & O'Gorman, 1987).

METHOD

Subjects

Subjects were 26 University student volunteers. There were eleven males and 15 females.

Apparatus

Skin conductance was recorded by applying a constant voltage of 0.5V across domed Ag-AgCl electrodes with .05M NaCl electrolyte. The electrodes were placed on masked areas on the distal phalanges of the index and second fingers of the subject's left hand. The electrodes were connected to a custom-built

battery powered bridge which was connected to a Grass 7 DAG preamplifier with a recording sensitivity of 0.02 μS/mm pen deflection. Respiration was recorded using a Phipps and Bird pneumatic bellows connected to a Grass 7PRTE transducer and 7DAG preamplifier.

The tone stimulus was a 1000 Hz tone with a rise time of 0 ms. It had an intensity of 69 dB as measured by a Dawe Type 1408E sound level indicator. The tone was presented binaurally via Sennheiser type HD222 headphones. The stimulus duration was 0.1 seconds for all presentations.

Procedure

On arrival at the laboratory subjects were informed that electrodermal activity and respiration measurements would be taken . Alternate subjects were assigned to either contextually cued or non-contextually cued conditions.

Subjects in the contextually cued condition were instructed:

" When you are in the subject room you will hear some tones made by the ticking of a clock. You do not have to make any conscious response to the ticking because we are interested in the automatic responses made by your skin during the experiment rather than your conscious responses. You can simply sit and relax and listen to the ticking. You may close your eyes if you wish."

Subjects in the non-contextually cued condition were instructed:

" When you are in the subject room you will hear some tones. You do not have make any conscious response to the tones because we are interested in the automatic responses made by your skin during the experiment rather than your conscious responses. You can simply sit and relax and listen to the tones. You may close your eyes if you wish."

To ensure that the subject was fully habituated to the stimulus before it was omitted the experiment began with 80 stimulus presentations with an onset to

onset ISI of 1 second. There were then three repetitions of the experimental block described below followed by a further 40 presentations of the stimulus at a constant onset to onset ISI of one second.

Each of the three repetitions of the experimental block consisted of the following components: 1) A constant ISI series consisting of 40 stimulus presentations at the one second onset to onset ISI. 2) An omission period consisting of an 8.9 second non-stimulation period corresponding to the omission of eight stimulus presentations and ensuring the re-presentation of the next stimulus is in phase with the previous constant ISI series. 3) A second constant ISI series identical to the first. 4) A variable ISI series consisting 40 stimuli presented with varying ISIs of either 0.5, 1 or 1.5 seconds. Over the 40 trials each subject received 12 ISIs of 0.5 seconds, 15 ISIs of 1 second and 12 ISIs of 1.5 seconds, giving an average ISI of one second. The order of ISIs was devised by random draw and was the same for all subjects: 0.5, 1, 1.5, 1.5, 0.5, 1.5, 1, 1, 0.5, 1, 0.5, 1, 1.5, 1.5, 0.5, 1.5, 1, 1, 0.5, 1, 0.5, 1, 1.5, 1.5, 0.5, 1.5, 1, 1, 0.5, 1, 0.5, 1, 1.5, 1.5, 0.5, 1.5, 1, 1, 0.5 for all subjects. 5) An 8.9 second omission period.

Scoring

The short ISI used in this experiment precluded the scoring of SCRs for each individual stimulus presentation. Rather, the scoring and analysis consisted of scoring of the maximum SCR response, the number of SCR responses greater than 0.02 μS and the total amount of SCR response (sum of all responses) within theoretically relevant 9 second time windows. The windows for the omission response measurement were the nine seconds preceding the omission period (either constant or variable ISI), the nine second omission period and the first nine seconds of the constant ISI re-presentation series. If the neuronal model was encoding the temporal parameters of the stimulus series then it would be expected that there would be greater activity in both the omission window and the post-omission window, compared to the pre-omission window. It may be

further expected that this effect would be more marked when the omission period was preceded by the predictable constant ISI series, rather than when it was preceded by the variable ISI series.

For the constant to variable ISI manipulation the windows were the last nine seconds of the constant ISI series and the first nine seconds of the variable ISI series. If the neuronal model was encoding the temporal properties of the stimulus series it would be expected that there would be an increase in all three SCR measures when the constant ISI series was immediately followed by the variable ISI series.

Electrodermal lability was measured by counting the number of responses greater than 0.02 µS emitted during the three minute pre-stimulation period. The subjects were split at the median into electrodermally stabile (low activity) and labile (high activity) groups for analysis.

To reduce skewness square root transformations were performed for both maximum SCR and total SCR indices before analysis. Greenhouse-Geisser epsilon corrections were used for effects containing repeated measures.

Results

Stimulus Omission

A 2 x 2 x 3 x 2 x 3 Instruction (Clock, No Clock) x Electrodermal Lability (Stabile, Labile) x Block (First, Second, Third) x ISI Variability (preceded by constant or variable ISI series) x Window (Pre, Omission, Re-Presentation) mixed model analysis of variance (ANOVA) was performed for each dependent variable (maximum SCR, number of SCRs, total SCR). Clock/No Clock was a between subjects factor while the other factors were repeated measures.

The clock/no clock factor had no significant main effect, nor was it implicated in any significant interactions and is therefore not discussed further. Table 6.1

shows the main effects and interactions that reached or approached statistical significance in the analysis of each dependent variable.

TABLE 6.1

F and Geisser-Greenhouse epsilon adjusted p-values for significant effects for each dependent variable.

Dependent Variable	Effect	df	F	Adjusted p	ε
SQRT Maximum SCR	Window	2, 44	4.91	.0167	.85
	Lability	1, 22	7.29	.0131	
	Window x ISI Variability	2, 44	5.55	.0079	.96
SQRT Total SCR	Window	2, 44	4.86	.0160	.85
	Lability	1, 22	6.99	.0148	
	Window x ISI Variability	2, 44	4.55	.0165	.96
	Block x ISI Variability	2, 44	3.71	.0338	.92
Mean Number of SCRs	Window	2, 44	4.53	.0200	.85
	Lability	1, 22	6.86	.0156	
	Window x ISI Variability	2, 44	6.84	.0031	.96

It is clear that the pattern of significant effects involving the omission trial manipulation is similar for each dependent variable.
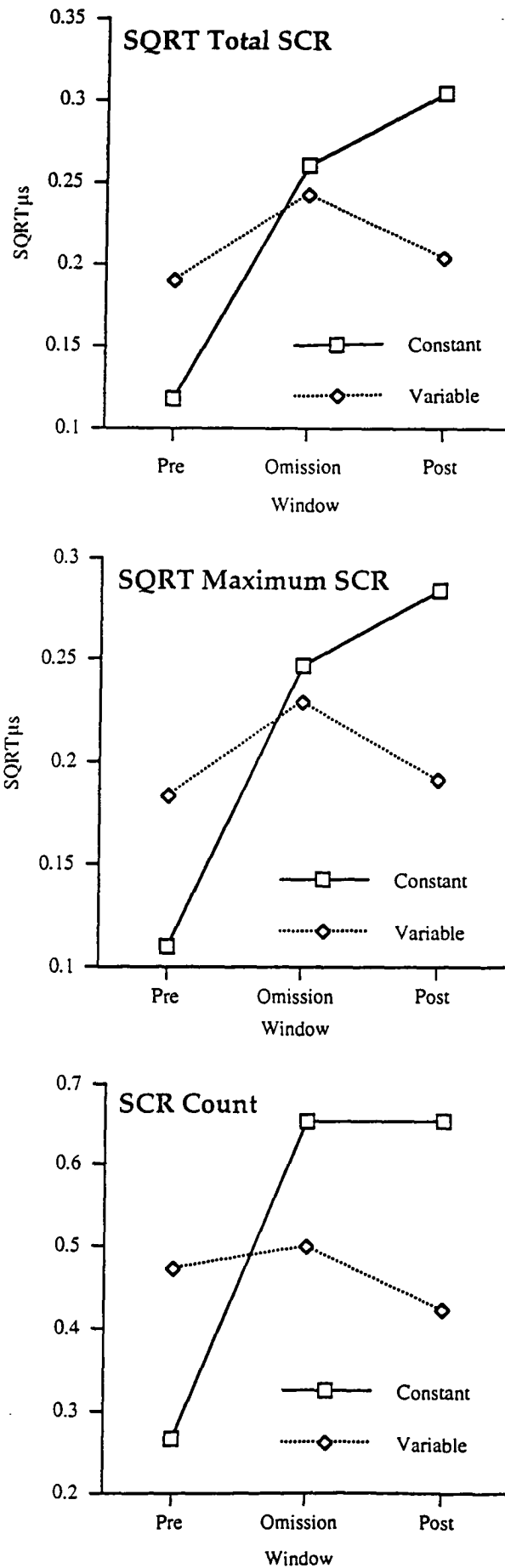
Figure 6.1. Maximum SCR, total SCR and number of SCR variables for pre-omission, omission and post-omission windows following both constant and variable ISI stimulus series.

TABLE 6.2

Mean (SD) maximum SCR, total SCR and number of SCR variables for pre-omission, omission and post-omission windows following both constant and variable ISI stimulus series.

	Following Constant ISI Series			Following Variable ISI Series		
	Pre	Omission	Post	Pre	Omission	Post
SQRT Total SCR Activity	.112 (.271)	.260 (.355)	.303 (.416)	.190 (.346)	.243 (.382)	.204 (.375)
SQRT Maximum SCR	.110 (.253)	.247 (.331)	.284 (.372)	.184 (.325)	.229 (.352)	.192 (.339)
Number of SCRs	.269 (.596)	.654 (.819)	.654 (.770)	.474 (.785)	.500 (.734)	.423 (.694)

Post hoc comparisons were calculated within the ISI variability x Window interaction testing the simple main effect of window at each level of variability for each dependent variable. For each dependent variable there was a significant increase in the SCR measures, compared to the preceding control interval, at both the time of stimulus omission and the time at which the stimulus is re-presented if the preceding stimuli had been presented at a constant ISI (Omission window compared to pre-omission window: SQRT Total SCR $F_{(1,44)}=12.37$, $p=.001$; SQRT Maximum SCR $F_{(1,44)}=14.74$, $p=.0005$; Number of SCR $F_{(1,44)}=14.47$, $p=.0001$; Pre-omission window compared to post-omission window: SQRT Total SCR $F_{(1,44)}=21.13$, $p=.0001$; SQRT Maximum SCR $F_{(1,44)}=24.17$, $p=.0001$; Number of SCR $F_{(1,44)}=20.04$, $p=.0001$).

If the preceding stimuli were presented at a variable ISI there was no significant increase in response at time of stimulus omission or upon re-presentation of the omitted stimulus (Omission window compared to pre-omission window: SQRT Total SCR $F(1,44)=1.86$, $p=.17$; SQRT Maximum SCR $F(1,44)=1.71$, $p=.19$; Number of SCR $F(1,44)=.17$, $p=.66$; Pre-omission window compared to post-omission window: SQRT Total SCR $F(1,44)=.11$, $p=.74$; SQRT Maximum SCR $F(1,44)=.04$, $p=.83$; Number of SCR $F(1,44)=.25$, $p=.60$). Figure 6.1 and Table 6.2 illustrate the activity before, during and after the omission period for both variable and constant ISI conditions for each dependent variable. These results are consistent with the notion that during the presentation of the constant ISI stimulus series the neuronal model encoded the ISI of the stimulus, and therefore when the stimulus was omitted there was a disparity between the activity of the neuronal model and the afferent stimulation and orienting responses were elicited. If the preceding stimuli were presented at a variable ISI the temporal encoding in the neuronal model did not occur to the same extent, and thus the disparity between the activity of the neuronal model and the afferent stimulation was not as great at the time of stimulus omission as in the constant ISI condition.

The presence of a significant block x ISI variability interaction in the case of total SCR activity is due to there being increased SCR activity on the three omission trials following the constant ISI series compared to those following variable ISI series in the first two experimental blocks but a reversal of this effect on the third experimental block. Given the failure of the block x ISI variability interaction to reach statistical significance in the cases of maximum SCR activity or SCR number measures this interaction may be no more than chance variation.

The significant main effects for electrodermal lability for each dependent variable were due to the labile subjects showing more average electrodermal

activity for each measure. There was no evidence that electrodermal lability significantly interacted with any other factors.

With regard to the number of subjects emitting an omission response, the maximum SCR data from the first experimental block were also analysed non-parametrically to examine the frequencies of subjects emitting particular patterns of responses under different conditions.

Of the 52 observations (26 subjects receiving both constant and variable ISI series), 22 showed no response in either of the pre-omission control window or the omission time window while one case showed non-zero equal responses in each window. The remaining 29 observations were analysed to test the likelihood of the emission of a response to stimulus omission following stimuli presented at either a constant or variable ISI. When the subjects had received the preceding stimuli at a constant ISI 13 (87% of non-zero observations) cases showed a larger response in the omission window compared to the preceding control window while only 2 (13%) cases showed the reverse. These frequencies were significantly different by the binomial test ($p<.01$). Following the variable ISI series, 8 cases (57% of non zero responses) showed a larger responses in the omission window while 6 cases (43%) showed the reverse, frequencies which are not significantly different by the binomial test. This suggested that the likelihood of emitting an omission response was greater following a constant ISI series than a variable ISI series.

These results are in some ways similar to those reported by O'Gorman (1989) in that while there was some evidence for an SCR to complete stimulus omission this was not the case in all subjects. Closer examination of the results of this experiment may give more cause for optimism. Considering the data for the first experimental block, of the 26 subjects, 13 showed a larger response during the omission window than the preceding control window when the omission followed the constant ISI series, a 50% score compared to estimates of between

34% and 52% reported by O'Gorman (1989). However, only two subjects showed the reverse effect while 10 others showed no responses in either window. If there was indeed no evidence of temporal encoding it would be expected that of the subjects emitting some response in either of the omission or control windows there would be a 50:50% chance of the larger response occurring in either window, not 87:13% as reported by this study for responses following the constant ISI stimulus series. The results of the scoring period following the variable ISI stimulus series are 57:43% a ratio consistent with a condition where the stimulus was less temporally predictable. It is argued that the reason why the percentage of subjects exhibiting omission responses was not higher is not because temporal prediction was not being performed by the subject but rather that some subject's response systems were not sufficiently sensitive to emit the response.

When the same data were analysed by level of electrodermal lability it was found that, of the subjects emitting a response in the control or omission windows following either constant and variable ISI series in the first experimental block, 12 stabile subjects showed a larger response in the omission window with 2 showing a larger response in the control window. The differences in frequencies was significant (Binomial test p=.02). For the labile subjects 10 showed a larger response in the omission window, and 6 a larger response in the control window, a non-significant difference (Binomial test p>.05). Note that the total number of responses was larger than the total number of subjects because each subject was scored following both the constant and variable ISI series.

In summary, the stimulus omission results indicated that following the constant ISI stimulus series the size and number of SCRs increased both during periods of complete stimulus omission and on re-presentation of the omitted stimulus. There was no evidence of a significant increase in activity if the preceding

stimuli had been presented at a variable ISI. Surprisingly, the response to complete stimulus omission was more marked in the stabile group than in the labile group.

Constant ISI v Variable ISI Stimuli Series

To assess the effect of changing (without an intervening omission period) from stimuli presented at a constant ISI to the same stimulus presented at a variable ISI 2 x 2 x 2 x 3 Instruction (Clock, No Clock) x Electrodermal Lability (Stabile, Labile) x ISI (Constant/Variable) x Experimental Block (1,2,3) mixed model ANOVAs were performed for each dependent variable. Clock/No Clock was a between subjects factor while the other factors were repeated measures. It will be recalled that scoring windows were defined as the last nine seconds of a constant ISI section of the stimulus series and the immediately following nine second first part of the variable ISI portion of the series.

Results were again very similar for the three dependent variables with the only effects reaching or approaching statistical significance for each dependent variable being the main effects for ISI variability and electrodermal lability. The F and p values for the electrodermal lability main effect were: Maximum SCR ($F(1,22)=5.43$, $p=.0293$), Total SCR ($F(1,22)=5.09$, $p=.0343$ and SCR Count ($F(1,22)=3.33$, $p=.0818$) indicating generally more electrodermal activity in the labile subjects than the stabile subjects. Electrodermal lability was not involved in any significant interactions.

Table 6.3 summarises the magnitude and significance of the ISI variability effect for each dependent variable.

TABLE 6.3

Means (SD), F and p values for constant versus variable ISI stimulus series for each dependent variable.

Dependent Variable	Unit	Constant	Variable	df	F	p
SQRT Maximum SCR	$\sqrt{\mu S}$	.118 (.215)	.294 (.397)	1, 22	12.81	.001
SQRT Total SCR	$\sqrt{\mu S}$	.141 (.246)	.295 (.408)	1, 22	7.71	.01
Mean Number of SCRs		.487 (.769)	.679 (.747)	1, 22	4.66	.04

These results clearly show that when the ISI of the stimulus series was changed from constant to variable the subjects showed an increase in SCR activity reflecting disparity between their previously encoded internal neuronal models and the afferent stimuli.

DISCUSSION

The results of this experiment are consistent with a theory of the orienting response in which the neuronal model is capable of encoding the ISI of afferent stimuli. If the orienting response is considered to be a measure of the amount of disparity between the afferent stimulus and the activity of the neuronal model then the results of this experiment can be simply explained.

When the stimulus was omitted then there was some disparity between the activity of the neuronal model (encoded during the preceding presentations of the stimulus) and the afferent stimulus and thus an orienting response was elicited. The omission response is thus a response elicited by the non-presentation of a stimulus and is due entirely to the disparity between the neuronal model and the afferent activity. When the stimulus is re-presented after the omission period (in phase with previous presentations) the activity of

the neuronal model at the time the stimulus is presented is less than it was just before the stimulus was omitted and thus the disparity between the activity of neuronal model and the afferent re-presented stimulus is greater and a larger orienting response is omitted.

The response to complete stimulus omission was much more marked when the omission followed a series of stimuli presented at a constant ISI than when it followed a series of stimuli presented at a variable ISI. Of the subjects who responded in either the omission or control period scoring windows, nearly 90% of them showed a larger response in the omission window if the preceding stimuli had been presented at a constant ISI. The percentages were much closer to 50% if the stimuli immediately preceding the omission had been presented at a variable ISI. On the first experimental block 13 of the 26 subjects showed an omission response following the constant ISI series. This percentage is similar to those reported in previous studies but higher than that in found in Experiment 1 (24%). It was therefore concluded that an omission response was more likely to be elicited when the preceding stimuli were presented at a shorter rather than a longer ISI.

The result that not all subjects showed a response to complete stimulus omission is often considered to be a flaw of comparator theories of habituation. The results of this experiment provide some means of reply to this criticism. Many subjects did not respond at all in either of the control or omission scoring windows. This result could easily be accounted for in a comparator theory of habituation by including a mechanism where a threshold of mismatch is required before a response is emitted, and allowing this threshold to vary between subjects. Electrodermal lability may be an index of this threshold but does not directly influence the ability of a subject to encode the temporal parameters of a stimulus series. If anything the results of this experiment suggest that stable subjects are more likely than labile subjects to emit a larger

response at the time of complete stimulus omission rather than during a control interval. It was also seen that if the subjects which are not responding in either the omission or control windows, presumably subjects with high response thresholds, are ignored, then following the constant ISI series 87% of the remaining subjects showed what was scored as an omission response. This high percentage indicates that in optimum conditions with a constant ISI series and a short ISI an omission response is emitted by most of the subjects who are responding at all. This result raises no insurmountable problems for a modified comparator model of habituation.

The results of the manipulation from a fixed to a variable ISI series also suggest that subjects encode the temporal parameters of a stimulus series as it is presented. When the ISI was changed from constant to variable the average size and number of SCRs was increased, consistent with the hypothesis that the afferent stimuli no longer fell at the same time as the peaks in activation encoded by the neuronal model, resulting in an increase in SCR activity.

CHAPTER 7

Experiment 4

The Effect of ISI Variability and Omission of S2 Following S1-S2 Pairs at a Short ISI

While the results of the previous experiment provided strong evidence of temporal encoding within the internal neuronal model involved in elicitation of the orienting response, they do not immediately suggest a process by which the encoding occurs. Similarly to Experiment 2, this experiment sought to more finely define the mechanism of temporal encoding used in habituation of the OR.

Postulated processes of temporal encoding in theories of habituation of the orienting response can be classified according to two types of mechanism. The first type is encoding of the period of stimulus presentation where the resulting expectancy is that "stimulus X is presented at a period of 10 seconds". The second type is the encoding of the time of stimulus presentation by reference to a cuing event or context, resulting in the expectancy that "stimulus X is presented 5 seconds after stimulus Y".

The use of a series of presentations of a single stimulus in Experiments 1 and 3 did not allow differentiation of the type of temporal encoding process. This was because the processes of encoding by period and encoding by reference to a cuing event are identical in the case of a series of presentations of a single stimulus. For example, a single stimulus presented at a period of 30 seconds necessarily occurs 30 seconds after a salient cue (the last stimulus presentation). Experiment 4 addressed this problem by the use of a stimulus series involving pairs of stimuli. Using this method the effects of both variable period of stimulation and variable temporal cuing by a salient event on rate of habituation and omission responding could be independently assessed.

The basic design of the experiment was to assess the relative contributions of period encoding and temporal cuing to the neuronal model by presenting series of tone pairs in which the second element could be predicted by either its period or cued by the first element. Comparison of the SCR activity during the different series would then show which method of encoding was employed by the neuronal model.

METHOD

Subjects

Subjects were 18 University student volunteers. There were 5 males and 13 females.

Apparatus

Stimulus one (S1) was a 950 Hz tone while stimulus two (S2) had a frequency of 775 Hz. Both tones had a rise time of 0 ms, a duration of 0.1 seconds and an intensity of 69 dB as measured by a Dawe Type 1408E sound level indicator. The tones were presented binaurally via Sennheiser type HD222 headphones. The ambient light intensity of the subject room was 0.9 cd/m^2 while the ambient temperature was 23°C.

Psychophysiological measurements were as in Experiment 3.

Design of the Stimulus Series

The terminology used to describe the stimulus series is that the period of a stimulus refers to the interval between successive presentations of the same stimulus (e.g., onset S2 to onset next S2) while stimulus onset asynchrony (SOA) will be used to refer to the interval from the onset of the S1 to the onset of the S2 of a stimulus pair. The stimulus series presented to the subjects consisted of three distinct sections which were presented in different orders.

Figure 7.1 illustrates the relationship between the S1 and S2 in the three different series.

Control condition, Constant SOA, Constant S2 period

Constant SOA, Variable S2 period

Variable SOA, Constant S2 period
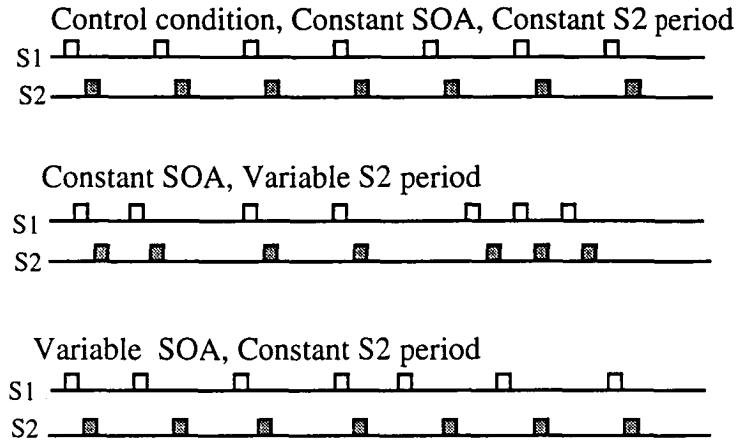
Figure 7.1. Temporal relationship between S1 and S2 presentations for the control, variable period and variable SOA conditions.

The control sections were constant period/constant SOA sections where the subjects were presented with 30 stimulus pairs at a constant period of 2.5 seconds for both stimuli and a constant SOA of 0.7 seconds.

The variable period condition consisted of 30 pairs where the SOA was constant at 0.7 seconds while the period of both S1 and S2 presentations varied randomly between 1.9, 2.5 and 3.1 seconds. Order of periods for all subjects was 1.9, 2.5, 2.5, 2.5, 1.9, 3.1, 3.1, 1.9, 1.9, 3.1, 2.5, 3.1, 1.9, 3.1, 2.5, 1.9, 2.5, 2.5, 2.5, 1.9, 3.1, 3.1, 1.9, 1.9, 3.1, 2.5, 3.1, 1.9, 3.1.

The variable SOA section consisted of 30 pairs where the SOA between the S1 and S2 was varied between 0.5, 0.6 and 1.1 seconds. The interval between the S2 and the next S1 presentation was adjusted so that the period of S2 presentation was constant at 2.5 seconds while the period of the S1 varied.

Order of SOA presentation for all subjects was 0.7, 0.2, 1.2, 0.7, 1.2, 0.2, 1.2, 0.2, 0.7, 1.2, 0.7, 0.2, 0.7, 0.2, 0.7, 0.2, 1.2, 0.7, 1.2, 0.2, 1.2, 0.2, 0.7, 1.2, 0.7, 0.2, 0.7, 0.2, 0.7.

It will be noted that if longer time intervals were used, as was the case in Experiment 2, then it would have been possible to measure responses to the S2 alone and, because both the SOA and period were independently manipulated with respect to S2, independently assess the merits of encoding by period or temporal cuing to the prediction of S2 by the neuronal model. While this experiment used the same arrangement of stimuli as Experiment 2, the inability to score responses to individual stimuli at these short intervals means that conclusions drawn from the experiment are more complex. Any responses scored during a reasonable time window in the current experiment must be a combination of responses to both S1 and S2 and thus manipulations should be considered with respect to both stimuli, rather than just S2. The predictions of this experiment are presented in Table 7.1, which outlines the predicted SCR activity which would result during the variable SOA or period series if either or both of encoding by period or encoding by temporal cuing were the methods of temporal encoding being used by the neuronal model to predict the time of presentation of the S1-S2 pairs.

TABLE 7.1

Predicted pattern of results under combinations of period encoding or temporal cuing mechanisms.

Encoding Method	Stimulus Series	Expected SCR activity when variable stimulus series follows control series
None	Variable SOA	SCR activity same as in control series because temporal information is not coded and thus can not be disrupted.
	Variable Period	As for Variable SOA
Period Encoding	Variable SOA	S2 is reliably predicted by its period but S1 is not, moderate SCR activity results.
	Variable Period	Neither S1 nor S2 predicted reliably , large SCR activity results.
Temporal Cuing	Variable SOA	Neither S1 nor S2 predicted reliably , large SCR activity results.
	Variable Period	S2 is reliably predicted by temporal cuing but S1 is not, moderate SCR activity results.
Both Period and Temporal Cuing	Variable SOA	S2 is reliably predicted by its period but S1 is not, moderate SCR activity results.
	Variable Period	S2 is reliably predicted by temporal cuing but S1 is not, moderate SCR activity results.

Therefore, if there are differences in SCR activity between the variable series and the control series then some form of temporal encoding must exist in the neuronal model. Differences in the amount of activity during the variable SOA and period series will indicate the presence of one or the other of encoding by period or temporal cuing with the direction of the difference indicating which is dominant. Equal activity in both variable SOA and variable period conditions

will indicate that both period encoding and temporal cuing are being utilised by the neuronal model. These conclusions are however valid only if the effects of unpredictability of either stimuli are additive, if the effects can have a non-additive relationship then the results are not as predictable as outlined in Table 7.1.

Procedure

On arrival at the laboratory subjects were informed that electrodermal activity and respiration measurements would be taken . Subjects were instructed that they would be hearing tones through the headphones, that they did not have to count or remember them and that they could close their eyes if they wished. All subjects then received a stimulus series consisting of combinations of the sections in one of the two orders described in Table 7.2. The adaptation consisted of a three minute period with no stimulation during which the experimenter counted non-specific SCRs as a measure of electrodermal lability.

TABLE 7.2

Order of presentation for Experiment 4.

Order A	Order B
Adaptation	Adaptation
Control	Control
Control	Control
Variable Period	Variable SOA
Control	Control
Variable SOA	Variable Period
Control	Control
Variable Period	Variable SOA
Control	Control
Variable SOA	Variable Period
Control	Control

Scoring

Measures were similar to those in Experiment 3 with maximum SCR, total SCR activity and SCR counts being scored in experimentally relevant time windows. The windows were all defined with respect to a variable SOA or variable S2 period stimulus series with each series having three windows associated with it. The control-pre window was defined as the last 15 seconds of the previous control window, the varying window was the first 15 seconds of the variable SOA or period stimulus series and the control-post window was defined as the first 15 seconds of the control window immediately following the variable SOA or period stimulus series. The choice of these windows allowed the testing of whether there was an increase in electrodermal activity when the stimulus

series was altered from control to one of the variable conditions, and whether the activity returned to its previous levels when the series was changed back to the control condition. Electrodermal lability was scored as in Experiment 3. Maximum SCR and Total SCR data were square-rooted before analysis.

RESULTS

2 x 2 x 2 x3 Lability (Stabile/Labile) x Block (First/Second) x Variability Type (SOA/IPI) x Window (Control-pre/Varying/Control-post) mixed model ANOVAs were performed on each dependent variable. Lability was a between-subjects factor while the other factors were repeated measures. Greenhouse-Geisser epsilon corrections were applied to effects containing repeated measures.

The results were almost identical for each dependent variable with only the electrodermal lability and window main effects reaching or approaching the .05 level of significance. The F and p values for these effects are summarised in Table 7.3. Means for each condition are displayed in Figure 7.2 and Table 7.3.

TABLE 7.3

Mean (SD) maximum SCR, total SCR and number of SCR measures for pre-varying, varying and post-varying windows for both variable SOA and variable S2 period manipulations.

	Variable SOA			Variable S2 Period		
	Pre	Varying	Post	Pre	Varying	Post
SQRT Total SCR Activity	.361 (.522)	.520 (.661)	.367 (.474)	.396 (.566)	.534 (.621)	.416 (.531)
SQRT Maximum SCR	.290 (.399)	.369 (.398)	.292 (.367)	.281 (.359)	.391 (.423)	.314 (.381)
Number of SCRs	1.57 (2.63)	1.97 (2.78)	1.36 (1.89)	1.56 (2.30)	2.11 (2.40)	1.42 (1.93)
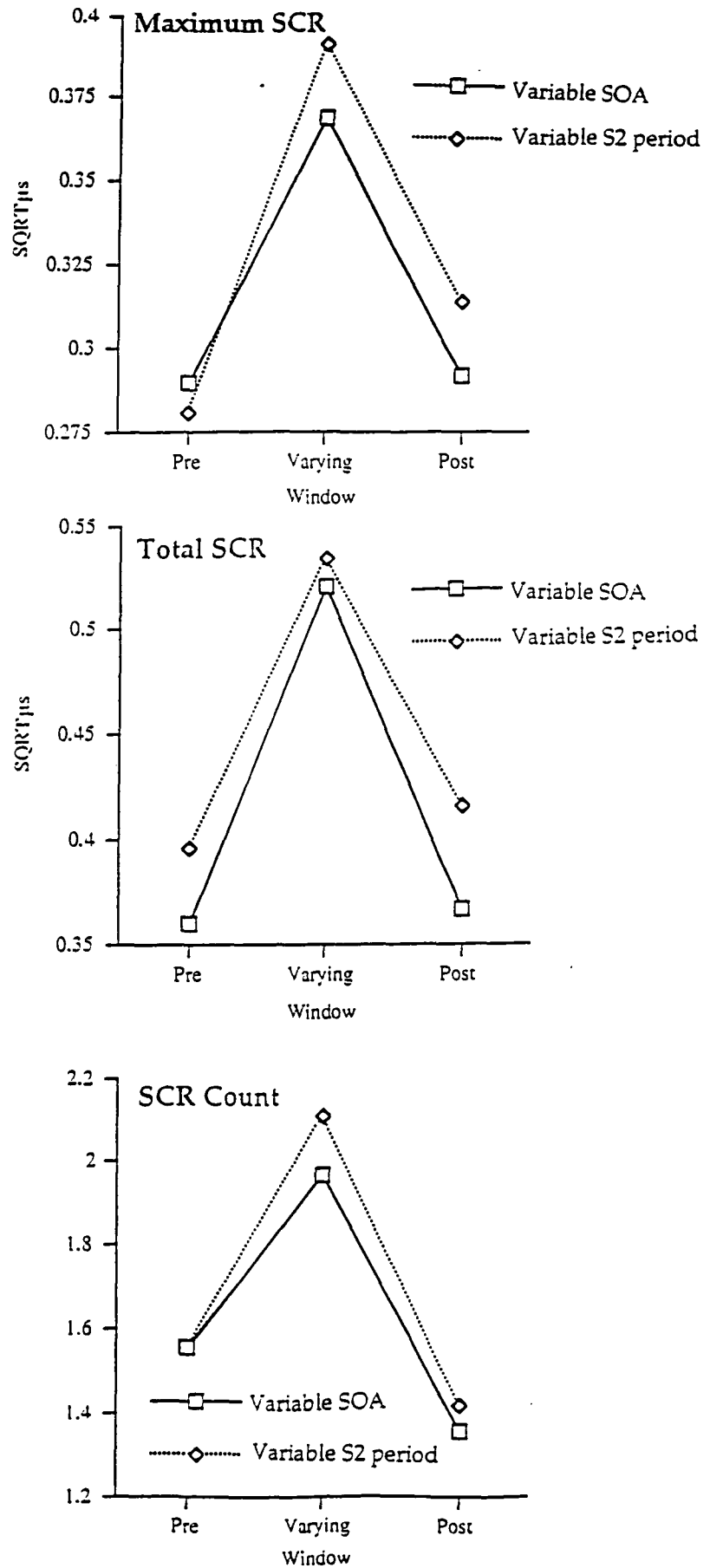
Figure 7.2. Maximum SCR, total SCR and number of SCR results for pre-variation, varying and post-variation scoring windows for variable SOA and variable period conditions.

TABLE 7.4.

F and Geisser-Greenhouse epsilon adjusted p-values for electrodermal lability and window effects for each dependent variable.

Dependent Variable	Effect	df	F	Adjusted p	ε
SQRT Maximum SCR	Window	2, 32	3.375	.0529	.956
	Lability	1, 16	9.357	.0075	
SQRT Total SCR	Window	2, 32	5.311	.0113	.956
	Lability	1, 16	8.107	.0116	
Mean Number of SCRs	Window	2, 32	6.783	.0059	.956
	Lability	1, 16	10.175	.0057	

The significant lability main effects resulted from the electrodermally labile subjects showing significantly greater values for each dependent variable across all scoring windows. The failure of electrodermal lability to be involved in any significant two or three way interactions indicates that the pattern of variation across the scoring windows was not significantly different for the two lability groups.

The significant window main effect indicates that there was a higher level of electrodermal response during the varying window compared to the control-pre and control-post windows (see Figure 7.2). Post hoc F-tests within this effect reveal that the differences between the control-pre and varying windows and also the varying and control-post windows were significant for each dependent variable. There was however no significant difference between the control-pre and control-post scoring windows indicating that responses return to control-pre levels once the varying stimulus series reverts to the control series.

These results clearly indicate that the subjects were encoding the temporal parameters of the stimulus series during the control presentations, and thus eliciting larger SCRs when the variable SOA and variable period series provided input disparate with the activity encoded by the neuronal model.

It was previously hypothesised that if encoding of period or ISI were the sole basis of temporal encoding in the neuronal model then there would be a difference between the electrodermal activity in the varying window in the variable SOA condition compared to the variable period series. F-tests between these two windows for each dependent variable reveal that these differences were not significant and they are in fact extremely similar (p>0.35 in each case). This suggests that neither SOA or period encoding is predominant in the neuronal model.

Although the Block x Window interaction was not statistically significant, exploratory comparisons were performed testing if the same pattern of results across the windows was found in both the first and second experimental blocks of variable SOA and variable period series. The pattern of results for each dependent variable was again the same with there being a significant difference between the varying window and the two control windows during the first experimental block while there was no significant difference between the three windows in the second experimental block.

To test for the possibility of a ceiling effect on response during the varying window a final series of comparisons was performed to test whether the average magnitude of the response during the varying window was significantly different to that when the stimuli were first presented at the beginning of the first control series at the commencement of the experiment. The results for all three dependent variables indicated that the average level of response was significantly lower in the varying windows than at the commencement of the experiment (Number of SCRs: Control=3.167,

Varying=2.042, F(1,17)=10.251, p=.0052; SQRT Maximum SCR: Control=.762,

Varying=.380, F(1,17)=33.408, p=.0001; SQRT Total SCR: Control=.970,

Varying=.527, F(1,17)=32.600, p=.0001).

DISCUSSION

The results of Experiment 4 provide some insights into the nature of temporal

encoding in habituation to stimuli pairs presented at a short time intervals. The

results are in agreement with the notion that as the stimuli pairs are presented

the neuronal model builds a representation of the temporal relationships

between them and eventually response to the stimuli habituates. When the

stimulus series becomes temporally unpredictable there is a consequent

disparity between the neuronal model and the afferent stimuli and the response

to the stimuli dishabituates, as evidenced by the increase in the size and

frequency of the SCRs in the variable SOA and variable period windows.

When the stimulus series reverts to the predictable control series the afferent

stimuli are again synchronised with the neuronal model and the size and

frequency of SCRs decreases.

The finding that the control-post activity is not significantly different to the

control-pre activity implies that the presentation of the SOA or period variable

series does not significantly degrade the neuronal model built up during the

initial control stimulus series. Conversely the fact that the difference between

the three scoring windows largely disappears by the second block of

experimental series, and that this result is due to an increase in both pre and

post-variation activity and a decrease in the varying window activity suggests

that there is an overall decrease in the quality of temporal information

contained in the neuronal model across the first experimental block caused by

the presentation of the SOA and period variable series. It may alternatively

have been that the overall pattern of predictable and unpredictable series

making up the experimental blocks itself became partially predictable toward the end of the experiment.

The result that the level of response was very similar in both the variable SOA and variable period series suggest that both the temporal cuing and encoding by period are utilised by the neuronal model in predicting the time of presentation of afferent stimuli. As was outlined in Table 7.1, if only one of these methods was used exclusively then the amount of electrodermal activity during the variable SOA and variable period series would have been different. The suggestion that the equality of responses during the two temporally unpredictable stimulus series was due to a ceiling effect and thus was insensitive to differences in processing would seem unlikely given that the average response during the varying windows was significantly less than that during the initial presentations of the same stimuli. This result suggests that the subjects were indeed capable of emitting a larger response if the disparity between the neuronal model and the afferent stimulation was large enough.

Finally, it is proposed that the mechanisms of encoding by period and temporal cuing need not necessarily be separate mechanisms, this being only the case if a stimulus is unable to serve as a temporal predictor of itself. If the mechanism by which the S1-S2 interval is also able to encode the S1-S1 interval then both period and SOA are thus encoded by the same mechanism. It is however possible that the two properties may be independently encoded by separate neural mechanisms, although the results of this experiment do not suggest that this is necessary.

CHAPTER 8

Conclusions from the empirical studies

The results of the final two experiments provided evidence of the presence of temporal encoding utilised by the subject in the elicitation of the orienting response. Published evidence investigating the presence of both a response to complete stimulus omission and an increase in response during variable ISI stimulus series had been mixed. The results of the current experiments confirm the validity of these effects in some subjects and further suggest that these phenomena are more likely to be exhibited at shorter ISIs rather than longer ISIs. It is possible that the shorter intervals make it easier for the subjects to encode the temporal parameters of a stimulus series and that this is the reason that the current results have been positive where others have not.

It was then possible to extend the list of known effects of various experimental manipulations on habituation of the OR with more certain information regarding the effects of temporal predictability of the OR. Table 8.1 presents a summary list of various phenomena, their empirical status and the ability of the various theories of habituation to predict the observed results.

Table 8.1

Habituation phenomena, their empirical status and the theories that predict them.

Effect	Empirical Status	Comments	Sokolov	Wagner (SOP)	Öhman	Dual-process
				Theories		
Recovery of OR to physical stimulus change	√	Including tone pitch, intensity etc.	√	√	√	√
Generalisation of habituation	√		√	√	√	√
Conditioned diminution of the UCR	√		X	√	√	X b
Effect of ISI duration on habituation	√	Shorter ISIs producing more rapid habituation	√	√	√	√
Effect of ISI variability on habituation	√	Variable ISI producing less rapid habituation	√	√	√	X b
Response to complete stimulus omission	√	More reliable with short ISIs	√a	√a	√a	X b
Response to S2 omission after S1-S2 series	√	With 0s S1-S2 interval but not with 4s S1-S2 interval	√a	√a	√a	X b

√ = Clear evidence for this effect/prediction.
X = Clear evidence against this effect/prediction.
Notes:
a: Can only emit an omission response if disparity between the neuronal trace and afferent stimulus can itself elicit an orienting response.
b: Requires assumption of ISI encoding elsewhere in the "state system" of the subject, an assumption which seems an unreasonable addition to the basic dual-process theory.

It can be seen then that each of the existing models of habituation is lacking in their ability to predict at least one of the phenomena listed. Often it was found that on first sight the theories appear to predict a particular effect, but on closer examination this prediction cannot be sustained without recourse to additional assumptions which are variously troublesome or at odds with the original spirit of the theory.

The weight of empirical evidence, including that presented in this thesis, allows the conclusion that the presentation of a series of stimuli at a variable ISI does result in the production of greater SCR activity (and hence less rapid habituation) than the same series presented at a constant ISI. There was also evidence of a response to complete stimulus omission. Both of these phenomena were found at shorter ISIs but not at longer ISIs. The question of a response to omission of an S2 after the presentation of a number of S1-S2 pairs is more uncertain. Siddle et al. (1987) and Siddle and Packer (1987) found quite strong evidence of this phenomena when there was no interval between the offset of the S1 and the onset of the S2 while Experiment 2 of this thesis found no evidence of the effect when the S1 and S2 were separated by a 4 second interval. It was therefore concluded that the occurrence of this effect was dependent on the interval between the S1 and S2, with no significant effect occurring if the interval was 4 seconds.

The remaining chapters of the thesis will outline the formulation and testing of an explicit, quantitative theory capable of reliably displaying the pattern of effects displayed in Table 8.1.

CHAPTER 9

Neural Network Models in Psychology

9.1. Introduction to neural network models

The growth in neuroscientific knowledge and the availability of relatively inexpensive and powerful computers have contributed to the rise in popularity of neural network models in recent times. Neural network models or variations of them which may be variously called connectionist models, parallel distributed processing (PDP) systems, artificial neural systems/networks or neuromorphic models (Gluck & Rumelhart, 1990) are most generally defined as computational models which are loosely based on actual neural structures, or are at least constructed of elements loosely based on actual neural structures. Approaches to the development of models falling in this group range from the neurobiologically and behaviourally driven models like Gluck and Thompson's (1987) model of associative learning or Wagner's SOP model (1981), to models designed to optimally solve particular computational problems like Rumelhart, Hinton and Williams' back propagation model (1986) or Ackley, Hinton and Sejnowski's "Boltzmann machine" network (1985).

Neural network models share a common basic arrangement where the emergent properties of the network are a result of the interaction of a number of separate elements, called nodes, neurons or units, linked together by inhibitory or excitatory connections. According to Rumelhart, Hinton and McClelland (1986) a parallel distributed processing model typically has eight major aspects: 1) a set of processing units; 2) a state of activation; 3) an output function for each unit; 4) a pattern of connectivity between the units; 5) a propagation rule for propagating patterns of activities through the network of connections; 6) an activation rule for combining the inputs impinging on a unit with the current state of that unit to produce a new level of activation for the unit; 7) a learning

rule whereby patterns of connectivity are modified by experience; 8) an environment within which the system must operate.

The set of processing units forms the substrate for all processing performed by the network. Each unit, also termed a node, may represent neural elements or assemblies, or non-neural objects such as a psychological concepts or an elements of language. Not all the units in a model are necessarily identical. They may be differentiated in both their internal function and their connections to other units. Neural network models of the type discussed in this thesis are inherently parallel with many units carrying out computations simultaneously. Rumelhart et al. (1986) classify units into three basic types: input units, output units and hidden units. Input units receive inputs from outside the model system while output units send signals out of the system. Hidden units are invisible from outside the model system and are connected only to other units within the network. Hidden units do not directly interface with the environment external to the model.

The state of activation of a network refers to the concept that, at any instant, each unit has a particular level of activity often represented by a real number, a_i, assigned to that unit. To preserve clarity in the current thesis the activations of input, hidden and output units will be denoted by a_{Ii}, a_{Hi} and a_{OUTi} respectively. The possible range of activation of a unit differs between different models. It may be binary or continuous and if continuous it may be bounded or unbounded. A common feature of many models is that an activation level of 0 indicates that the particular unit is inactive.

Related to the idea of activation is the way that computation proceeds when a network is actually simulated. The activation level of the input units is supplied externally as a vector of values a_{Ii} representing some external input , the input units therefore provide the interface by which the experimenter provides the input for the network. The activation from the input units then

propagates through the connections to the hidden (if present) and output units, following the rules outlined below. The vector of values a_{OUTi} representing the activity of the units in the output layer is then the "answer" or product produced by the network when the particular input is presented to it. Processing generally proceeds in discrete time steps; when an input is presented as the activity in the nodes in the input layer all the calculations required to calculate a new output vector and update any values within the network are completed before the next input vector is read in to the input layer.

Each unit emits an output which may either be transmitted along connections to other units or, if the unit is an output unit, output from the entire model as a result of processing. Each unit has an output function which relates the level of activity of a unit to the output emitted by that unit. This can be written as

$$O_i = f_i(a_i)$$

where O_i is the output of unit i, a_i is the activation of unit i and f_i is the output function for that unit. This function may simply be an identity function where the output equals the activity level, a threshold function where an output will only be emitted if the activity exceeds a certain value or some other function, chosen to impart desirable characteristics to the model.

The units of a particular model are arranged in a specific pattern of connectivity which can be devised to imitate putative neurological or psychological structures or alternatively provide a general substrate upon which the network may evolve emergent properties. Figure 9.1 illustrates the pattern of connectivity for a simple neural network model.
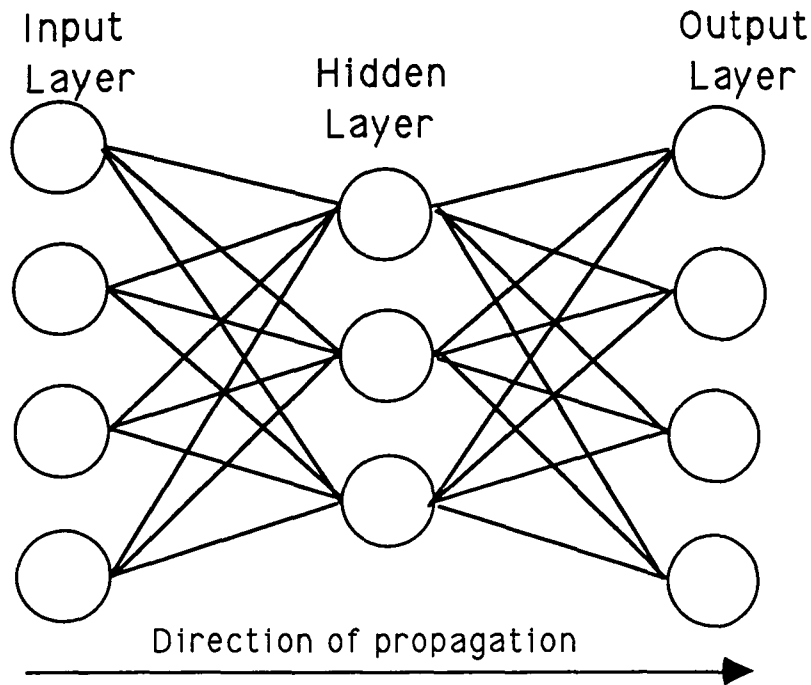
Figure 9.1. Pattern of connectivity of a simple neural network model.

In the pattern of connectivity in Figure 9.1 three layers are shown. A layer of four input units have connections to the three units of the hidden layer. The hidden units themselves have connections to the four units of the output layer.

The rules of propagation define the functions governing the way the model transmits the outputs of each unit along the connections to other units. The concept implemented by the rules of propagation is that the influence of one node on another is mediated by a connection weight, that is that the connections in Figure 9.1 are not all of equal strength. Generally, the connection weight from node i to node j is denoted by w_{ij}. In most models connection weights can take both positive and negative values. Positive values can be thought of as implying an excitatory effect of node i on node j while a negative connection weight implies an inhibitory connection.

The rule of activation defines how the combined inputs to a unit from other units affect the activation of that unit. The most common rule of activation is that the activity of a unit is a function of the weighted sum of the outputs of the

units connected to that unit. Rumelhart et al. (1986) describe this process of the calculation of the activation of a unit by defining an intermediate step where a parameter net_i is calculated as the weighted sum of the outputs of the j units connected to unit i. That is:

$$net_i = \sum_j w_{ji} O_j$$

where it is assumed that unit i is connected to j other units, w_{ji} is the connection weight of the connection from unit j to unit i and O_j is the output of unit j. For example, in the case of the network shown in Figure 9.1 net_i for each of the units in the hidden layer would be the sum of the output of each input unit multiplied by the weight of the connection between the respective input and hidden units.

An activation rule is then applied to net_i to produce a new level of activation for unit i. Generally we have:

$$a_i = g(net_i, a_i')$$

where g is a function relating the new value of a_i to net_i and a_i' which is the previous value of a_i. In many cases, including the models simulated in this thesis, a_i' is not included in the calculation of a_i and thus:

$$a_i = g(net_i)$$

where g is a monotonically increasing bounded function. This means that as net_i increases a_i increases but does not exceed some limit. This limit may, for example, be physiologically determined.

The learning rule by which connections between units are modified by experience can take one of two forms. In the first case, connections could be deleted from the model or new connections added. This method of plasticity is not very commonly used in neural network models. The second and more

commonly used method of modification is to alter the connective strengths or weights of existing connections between the units.

Most learning rules used in connectionist models can be viewed as modifications of the Hebbian learning rule (Hebb, 1949). The Hebbian rule basically asserts that if one unit receives input from another unit then, if both units are active at the same time (i.e., within a single processing iteration), the weight or strength of the connection between those units is increased (Rumelhart et al., 1986). In the simplest case of the Hebbian rule the amount of change in the weight w_{ij} connecting units i and j is given as

$$\Delta w_{ij} = d a_j O_i$$

with d being the learning rate.

A consequence of repeated application of the Hebbian learning rule is that both the magnitude of the connection weight between two units i and j and the activation of unit j will increase infinitely if unit i is repeatedly stimulated (assuming a that w_{ij} initially was $\neq$ zero). This can not happen in a real biological system where both the connection weight and the level of activation must have physical limits. A biologically valid learning rule must therefore, if it is to be based on Hebbian learning, add a means of controlling this inflation of connection weights.

A common modification of the Hebbian rule is given by the formula

$$\Delta w_{ij} = d(t_j - a_j) O_i$$

where t_j is a teacher or target activation for unit j. t_j may be explicitly defined by the experimenter and represents the desired activity of node j given the current input vector. For example, if the network is being used to perform a classification task then t_j may be used to define the desired value in particular output nodes when specific input vectors are presented to the network. This rule is known as the Widrow-Hoff rule (Widrow & Hoff, 1960) or alternatively as the delta rule (Rumelhart et al., 1986). In the delta rule, the amount of

change in the connection strength is proportional to the actual activation achieved by a unit and the target activation defined by the teaching input. Weights adjusted according to the delta rule will generally stabilise when the output of a unit reaches the target activation. The use of an excessively large value of d may however prevent this stabilisation from occurring. There are many other possible learning rules for connection weights within neural network models and these will be described as they arise in the discussion.

Any connectionist model, or indeed any model of a psychological process, must be placed within its environment. In particular, it is necessary to indicate how stimuli are presented to a model and how outputs are emitted from it. When a model is designed the researcher must specify how each input and output pattern relates to real or theoretical constructs, for example, the way in which each pattern input to or generated by the network can be interpreted as relating to psychological events.

9.2. The problem of evaluation of models constructed by workers in different disciplines.

Connectionist models have been developed by workers in a number of disciplines. Computer scientists, engineers, psychologists, neuroscientists and mathematicians have all been responsible for developments in neural network models. Each discipline has its own priorities in the development of a particular flavour of model. To generalise, computer scientists and engineers measure the performance of their models in terms of efficiency and accuracy on a specific problem, normally some type of classification or recognition problem. The ultimate goal in these disciplines is to develop a model which can perform a particular task perfectly in all situations, in many cases a super-human feat. Psychologists and neuroscientists, on the other hand, seek not perfect accuracy of classification but rather that their models mimic as closely as possible the results observed in their model organism.

Researchers hoping to apply neural network models to their area of research therefore have to be careful to assess the performance of their and other workers' models by criteria appropriate to their particular goals. An example of this may be if a psychologist develops a model of organisation of knowledge into semantic categories. The performance of humans in experiments on semantic organisation indicates that their organisation is not optimal. Errors are sometimes made in classification and some items are classified more rapidly than others. A psychologist might hope to incorporate these biases and errors into the model and would feel successful if the model accurately reflected human performance. A computer scientist assessing the model may, however, view these phenomena as inadequacies of the model, remove them , and report the increase in correct classifications as an improvement in the model. In this thesis the models eventually developed will be assessed by the criterion that "the more closely the performance of the model mimics human performance then the more successful is that model". Thus, while the models reported in

the thesis may not be the most efficient methods of performing the tasks required of them they will perform similarly to humans. The models may make errors and show surprising limitations in their performance, but this is appropriate and acceptable provided that empirical results indicate that humans exhibit similar errors and limitations in the same situations.

A second area of difficulty in the comparison of connectionist models across disciplines, or even different models within the same discipline, is the wide variation in the scale of the various models. In some models the units or nodes represent psychological concepts, words or objects. At a finer scale are models in which the units represent supposed neural assemblies or specific artificial neurons. These intermediate scale models however do not identify specific neuronal structures corresponding to those in their model and are often based on fairly crude analogues of neural physiology. The final class of models are conceived as accurate simulations of actual neural performance and their performance and mechanisms are often correlated with measurements from intact neural systems.

The models developed in the current thesis lie between the second and third levels of scale. They are based on neuron-like units and pay some respect to neuronal plausibility. There is, however, less emphasis on correlation of the neuronal structures proposed in the models with known neurological structures than has been performed with the most detailed neuroscientific models.

9.3. Habituation and neural network models

9.3.1. Neural network models of conditioning

One area in which there has been a successful description of phenomena at all levels of abstraction from largely verbal behavioural descriptions, through behaviourally based quantitative models and finally as identification of the

actual neurobiological processes, is classical conditioning. In the earliest chapters of the thesis we saw that associative (classical conditioning) and non-associative (habituation) processes could sometimes be incorporated into a single theory (e.g., SOP, Wagner, 1981). A neural network explanation of classical conditioning will now be discussed with the intention that issues raised will be useful in formulation of neural network model of habituation.

The basic phenomenon of classical conditioning is that a conditioned stimulus (CS) is repeatedly paired with an unconditioned stimulus (US). Initially, the US elicits some natural or reflexive unconditioned response (UR). For example, in Pavlov's initial experiments the US was the presentation of food to a dog which elicited an UR of salivation. The CS in these experiments was a bell or light which itself did not elicit a salivatory response. However, after a number of CS-US pairs the presentation of the bell or light CS alone (in the absence of the food US) was enough to elicit a salivatory response in the dog. This response which became paired to the CS was called a conditioned response (CR). The essential component of all theories of conditioning is therefore the provision of a process by which repeated pairing of the CS and US will eventually result in the presentation of the CS alone eliciting the response (CS) previously elicited by presentation of the US.

A succinct quantitative account of classical conditioning is given by the Rescorla-Wagner theory (Rescorla & Wagner, 1972). The theory is expressed in cognitive terms as:

> Organisms only learn when events violate their expectations. Certain expectations are built up about the events following a stimulus complex; expectations initiated by the complex and its component stimuli are then only modified when consequent events disagree with the composite expectation. (Rescorla & Wagner, 1972, p. 75)

The Rescorla-Wagner theory is described mathematically as:

$$\Delta V_A = \alpha_A \beta [\lambda - V_{AX}]$$

where

ΔV_A = the change in associative strength between the CS (denoted by A) and the US

λ = the asymptotic level of associative strength possible with the current US

V_{AX} = the composite associative strength already present between the US and other stimuli including the current CS

α_A and β are constants which depend on the particular CS and US

The essence of the Rescorla-Wagner theory is that the connection weight or associative strength between the CS and US will be modified when events such as the US (represented by λ) differs from the composite associative strength (represented by the sum of associative strengths already associated with the US) (Sutton & Barto, 1981). The fact that λ is limited leads to the effect known as blocking (Kamin, 1969) where in the first phase of the experiment a CS1 (e.g., a light) is repeatedly paired with a US. In the second phase a second CS2 (e.g., a bell) is added, with exactly the same temporal relationship to the US as the CS1 , that is, the CS1 is replaced by a CS1 +CS2 complex. It is observed that although the CS2 is paired with the US equally as well as the CS1 there is little increase in the associative strength between the CS2 and the US. This is explained by the Rescorla-Wagner theory because in the first stage the associative strength between the CS1 and the US reached the asymptotic level of λ. When the CS2 was added in the second phase there was no change in associative strength because the total association between the US and other stimuli (i.e., CS1) was equal to λ.

A relevant point about the Rescorla-Wagner theory is that it is mathematically identical to the Widrow-Hoff or delta rule described earlier (Sutton & Barto, 1981). The change in connection weights in Widrow-Hoff rule was given by the formula $\Delta w_{ij} = d(t_i - a_i)O_j$. To show correspondence between this and the

Rescorla-Wagner theory let the learning rates $(\alpha+\beta)=d$, $t_i=\lambda =1$ if the US is present or 0 of the US is not present, $O_j=1$ (or alternatively a real value indicating the activity in the unit representing the CS) if the CS is present or 0 otherwise. The substitution of a_i for V_{AX} is because a_i is calculated by summing the weighted activations of all the other units connected to unit including those representing the CS which is clearly related to the sum of the associations between the US and all other units connected to it.

Sutton and Barto (1981) remark that the finding that the Widrow-Hoff and Rescorla-Wagner rules are identical is striking , given that one (Widrow-Hoff) was designed as a mathematical algorithm with no thought to its predictions to learning theory. Conversely, the Rescorla-Wagner theory was designed solely as a model of animal learning with its utility in mathematics not being a consideration. There is however much more to a successful neural network model of conditioning than a simple set of units linked by connections where modifications in weight are mediated by the Widrow-Hoff rule. The two areas which are of particular relevance to the current thesis are: 1) neurobiological plausibility of the learning rule, and 2) representation of temporal relationships between stimuli.

9.3.2. The novelty detector as a model of habituation

A process closely resembling habituation is a feature of many neural network models. Kohonen (1984) describes a processing unit called a novelty detector. The novelty detector is similar to the units common in many models and its performance provides useful hints to the construction of a neural network model of habituation.

The novelty detector unit is described as follows.

Let the output η, be a scalar which in the present context corresponds to the magnitude of the OR.

Then η is defined by

$$\eta = m^T x$$

where

α = the learning rate, $\alpha > 0$ in this case

x is a row vector containing the inputs to the unit

m is a column vector containing the weights of the input connections to the unit

The learning rule for the weights is

$$\frac{dm^T}{dt} = -\alpha\eta x^T \qquad \text{(Kohonen, 1984, p.107)}$$

where

t is time

The output of the network, η, is the inner product of the memory and input vectors and is a measure of orthogonality between the two vectors, with $\eta=0$ when the vectors are orthogonal. Kohonen (1984) shows that if the input vector (or a set of up to n input vectors, where n is the number of input units) remains constant then the memory vector becomes orthogonal to the set of input vectors and the output, η, monotonically tends to zero. If however a new input is presented to the unit then the output will be non-zero. As Kohonen (1984) notes the performance of the novelty detector, if it was observed in experimental psychology, would be termed habituation.

The novelty detector makes a simple translation to a simple model of habituation because its output is a measure of error between the desired state of the network and the current state: η is small when a constant stimulus has been presented for a number of iterations, and large when a novel stimulus is presented to the unit. The novelty detector is not however unique in this respect. While other network models do not normally define their output function so that it tends toward zero as the units receive more training presentations, it is almost always the case that the dissimilarity between the

current output and some desired output does in fact decrease as training progresses. If a measure of this dissimilarity or some parameter proportional to it is taken as the dependent measure then most common neural network models can be used more or less effectively as models of habituation. This assumption is central to the models which will be developed and tested in the remaining chapters of the thesis. In particular, it is postulated that the dissimilarity between the current input and the desired output of the network corresponds to the orienting response. We will now investigate further the advantages and shortcomings of some potential neural network models of psychological phenomena with particular reference to the effect that each factor has on the suitability of a the network as a model of habituation.

9.4. Choice of learning rules

Both Hebbian and Widrow-Hoff learning rules have advantages and disadvantages.

The major points supporting the Hebbian learning rule are:

1) Does not require a special teacher input.

2) Supported by neurobiological data (Gluck & Granger, 1993; Hawkins, Kandel & Siegelbaum, 1993; Sejnowski & Tesauro, 1989).

While the Widrow-Hoff rule has the following features:

1) More computationally powerful and efficient than the Hebbian learning rule (McLaren, 1989).

2) Some neurobiological support (Gluck & Granger, 1993; Gluck, Reifsnider & Thompson, 1990; McLaren, 1989; Mitchison, 1989).

3) Identical to Rescorla-Wagner theory of conditioning (Sutton & Barto, 1981).

These points will now be expanded upon.

The Hebbian learning rule is less complex than the Widrow-Hoff or delta rule. It was demonstrated earlier that the Widrow-Hoff rule could be considered a special case of a Hebbian learning rule by adding an input from a teacher input which provides a target activation for a unit. Sutton and Barto (1981) suggest that the Widrow-Hoff learning rule necessarily requires a more complex neuronal arrangement than the Hebbian learning rule. The provision of a teacher input in any network can provide difficulties in that it is often difficult to imagine an arrangement by which the some part of the system can know the target activity for a set of units. The Hebbian learning rule has the definite advantage of neurobiological support. The Hebbian learning rule can be seen in real nervous systems as long-term potentiation (LTP) (e.g., Gluck & Granger, 1993; Kirkwood & Bear, 1994). Hawkins et al (1993) reviews the evidence for LTP and reports that LTP in the CA1 region of mammalian hippocampus resembles Hebbian learning. For LTP to occur in CA1 there must be coincident activity in the postsynaptic pyramidal neuron and presynaptic neurons. If the postsynaptic neuron is blocked from firing, then LTP will not proceed and maximal LTP is produced if activation of the pre- and post-synaptic neurons is almost simultaneous. Gluck & Granger (1993) report that LTP has also been reported in olfactory paleocortex, dentate gyrus of the hippocampus, and in motor, visual and somatosensory neocortex. While the biochemical mechanism of this change is beyond the scope of this thesis the evidence for LTP shows that Hebbian learning can occur at a single synapse within the mammalian brain. The evidence for error-correcting learning rules such as the Widrow-Hoff and delta rules is most clearly understood in the cerebellum (Gluck & Granger, 1993). In the case of eye-blink conditioning in the rabbit, information about the CS travels along mossy fibre projections to the cerebellum via the pontine nuclei. The reinforcement or teacher input representing the US travels along climbing fibres originating in the inferior olive. The interpositus nucleus of the cerebellum is thought to be the site where the CR is originated. The example of

rabbit eye blink conditioning clearly illustrates a possible disadvantage of the error-correcting rules compared to the Hebbian learning rule. An error-correcting rule necessarily requires a more complex neural substrate than the Hebbian rule. While the Hebbian learning rule can be seen to be implemented in a synapse between a pair of neurons, an error-correcting rule necessarily requires an additional afferent to the target cell to provide the teacher or desired level of activity and determine the change in strength between the input and output (Gluck & Granger, 1993). In conclusion, the neurobiological evidence for Hebbian and error-correcting learning rules can be summarised by stating that Hebbian learning requires a more simple neuronal substrate than an error-correcting rule and has been observed in mammalian hippocampus and neocortex. Error-correcting rules are necessarily more complex but have also been observed in real systems, in particular the cerebellum of the rabbit.

McLaren (1989) points out that error-correcting rules such as the delta rule have some advantages over the Hebbian learning rule. It has already been stated that the Widrow-Hoff rule is identical to the Rescorla-Wagner theory of conditioning and this can be seen as an advantage in the ability of the rule to account for observed psychological data. Furthermore, McLaren (1989) asserts that error-correcting rules, when compared to the Hebbian rule, can store more information in the same sized network and are more powerful in that they can normally be expected to arrive at some least-squares solution to a problem.

A final point to consider is the possibility that a neuronal assembly capable of implementing an error-correcting learning rule may be devised from the combination of a number of Hebbian elements. The existence of such an assembly would render the question of the validity of either rule somewhat less crucial than it presently is. Both McLaren (1989) and Mitchison (1989) attempt but do not completely succeed in providing this link. Both of their formulations

require modification of the usual Hebbian learning rule to allow the implementation of an error-correcting algorithm. It is however not impossible that either these modifications to the Hebbian learning rule are incidental and not in themselves neurobiologically implausible or that future workers may devise an assembly of Hebbian units capable of implementing an error-correcting learning rule.

9.5. Timing in neural network models

The aspect of timing particularly relevant to the current thesis, and investigated in the psychophysiological studies, is the question of how can a neural network learn and store the interval between successive presentations of a single stimulus, or the interval between two different stimuli.

Two approaches to this problem will be discussed.

1) The use of delays (Longuet-Higgins, 1989; Mitchison, 1989) and tapped delay lines (Desmond, 1990).

2) Pacemaker learning model (Torras, 1985, 1986).

9.5.1 Delay lines

The use of delay lines to encode and store the interval between two inputs to a network (representing either two different stimuli, or two presentations of a single stimulus) is an example of what was termed in earlier chapters as temporal encoding by a cuing event. The animal learns the interval between two inputs which are repeatedly presented with a regular temporal relationship and is consequently able to predict the time presentation of the second event from the first event. Both Longuet-Higgins (1989) and Mitchison (1989) describe similar models for the learning of these intervals. The essence of these delay line models is shown diagrammatically in Figure 9.2.
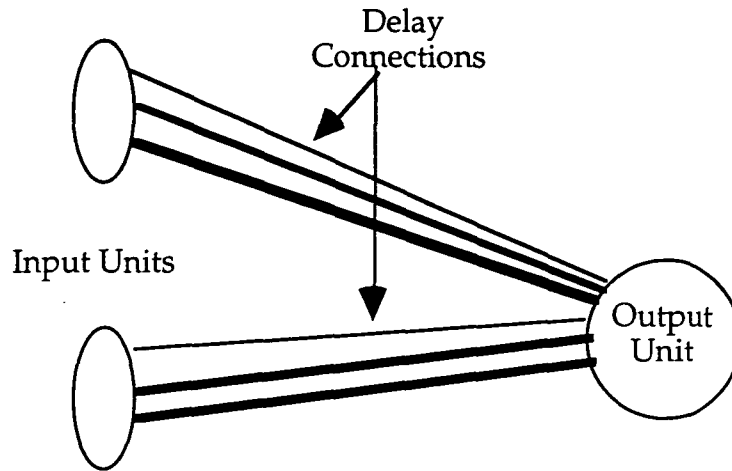
Figure 9.2. A delay line model of temporal encoding.

The basic idea of the delay line model is that each input unit is connected to each output unit (assuming the single-layer architecture illustrated) by a number of connections which conduct the neural impulses at different speeds. Therefore activity from one input node will reach the output node at a number of instants spread over time via the various delay connections. If two input nodes are stimulated with some interval (or one interval is stimulated twice) and the impulses from both reach the output node at the same time (or within a pre-defined time window) then a synapse between the two lines is activated (not shown in diagram). The modification of this synapse may follow the Hebbian learning rule. A relevant facet of the delay line model is that it would be a simple matter to implement a means by which the intervals could be encoded only within a certain range. For example, the experiments reported earlier in this thesis indicated that temporal encoding of this type seemed to have a significant effect at ISIs of 1-2 seconds but not at an ISI of greater than 14 seconds. A delay line model could easily accommodate this result by having delay lines with a range of conduction times ranging from 0 to 10 seconds. In this case there is no way that the impulses from two units stimulated 15 seconds apart could possibly coincide at the output unit.

The principal objection to delay line theories of interval encoding and storage (Mitchison, 1989; Sutton & Barto, 1981) is the problem of neurophysiological plausibility. While it is simple to devise possible mechanisms, such as the use of different axon diameters or a number of additional synapses to slow conduction, to provide a variety of delay values for the delay lines, it is uncertain that these methods could provide the sufficiently wide variety of values necessary for all observed phenomena (Mitchison, 1989). There also does not seem to be a real example of a population of neurons displaying the properties necessary to form a delay line model.

Desmond (1991) proposes a similar theory of interval encoding utilising tapped delay lines. Rather than a stimulus evoking impulses in a number of delay lines with differing rates of conductivity, Desmond's (1991) model postulates that the onset or offset (each is represented by separate sets of units) of a stimulus itself sets off a sequential pattern of activity in a group of neurons associated with that stimulus, a process analogous to the formation of a stimulus trace. For example, the trace of a particular stimulus may be represented by n units. At the time the stimulus is presented to the network ($t=1$) unit 1 will be active, after one time step ($t=2$) unit 2 would be active while the activity in unit 1 would have decayed to zero. The "wave of activity" then propagates along the units until at $t=n$ unit n would be active. At $t=n+1$ none of the units would be active and no trace of the stimulus would persist. Similarly to the delay line theories described earlier, each of the n units of the stimulus trace is connected to an output unit which receives input from other stimuli. The connection weights connecting the units representing the stimuli are only incremented when the activity from units in the stimulus trace of each coincide. Like the delay line theory above this learning process means that the assembly will eventually learn the interval between the onsets or offsets of the two stimuli. Desmond (1991) does not however report any evidence of the existence of real neurons with the properties required by his theory.

Both the delay line and tapped delay line theories of ISI encoding are compatible with the basic model of habituation of the orienting response described earlier in the chapter. In the model the magnitude of the orienting response was related to the distance or dissimilarity between the input and output layers at any instant. Both of the delay methods of encoding could be incorporated into this basic structure with the result that the presentation of either one or a number of stimuli with a regular temporal relationship would enable the model to predict future presentations of the stimulus. For example, a series of presentations of an S1 followed five seconds later by an S2, would be expected to increment the weights of the network in such a way that presentation of the S1 to the network would result in the activation of the output nodes connected to the S2 five seconds later. The use of delay line theories of ISI encoding can also be considered a possible mechanism for the retrieval-generated priming of Wagner's priming (1978) and SOP (1981) theories.

A final point to consider with the delay line theories is one which was raised in the discussion of the empirical studies in earlier chapters. If time intervals are learned and stored by cuing from another input to the network, as they are in the delay line theories, then are the units representing a stimulus allowed to cue future presentations of themselves? The theories presented seem more easily visualised if they are representing intervals between two different stimuli. They do not however preclude that a stimulus can cue a future presentation of itself. If a delay theory is to be used in the simple case of learning the ISI of a series of presentations of a single stimulus then it needs to expressly include a mechanism by which a stimulus can cue future presentations of itself.

9.5.2. Pacemaker learning model

Torras (1985, 1986) suggests a model of learning of temporal intervals rather different to the delay line models. Rather than storing the time interval by use

of delay lines Torras' model stores time intervals by changes in intra-unit parameters which allow a single unit to store the period of its own activation internally. In essence the model requires that firstly the nodes in the output layer (representing the internal trace of past stimulation) spontaneously fire at some regular rate. The reason for this firing is proposed to be that a gradual leaking of ions through the cell membrane results in a regular series of action potentials being elicited over time. When the membrane potential exceeds a given threshold the neuron emits an action potential or response. Torras' model utilises exponential functions for both the rise of the membrane potential and the fall of the threshold as well as considering the membrane potential at any instant as a stochastic process. For the purpose of this thesis, the details of the model are modified somewhat with the threshold having a constant level throughout the experiment and the rise of the membrane potential being linear.

The basic processes of the simplified model are shown diagrammatically in Figure 9.3.

OUTPUT OF NODE
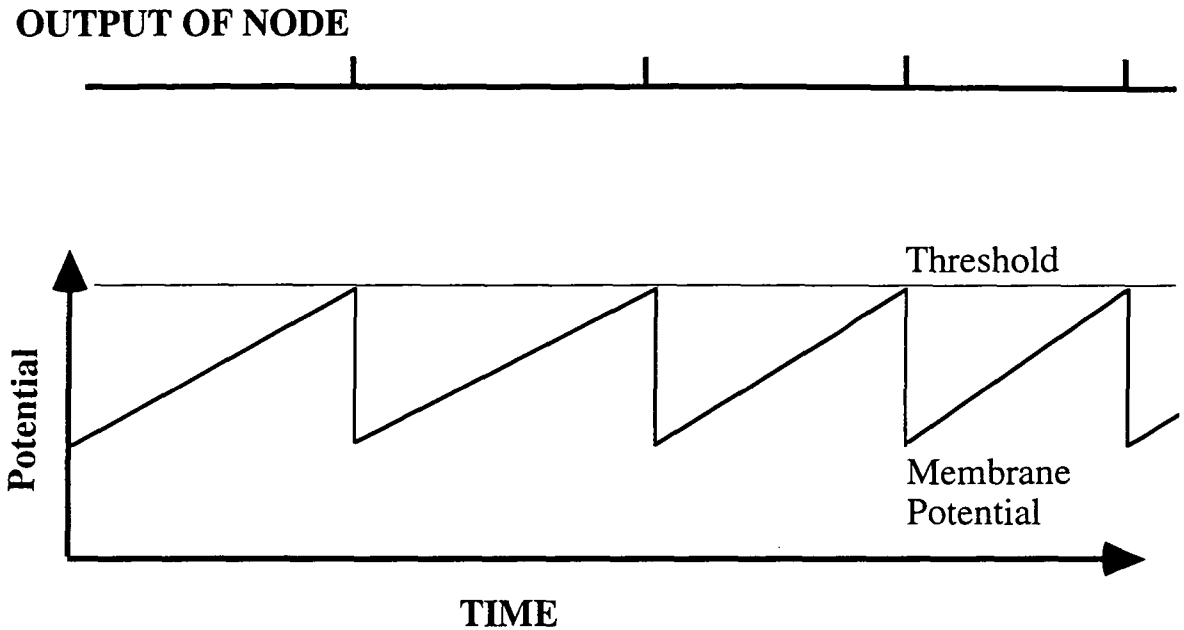
Figure 9.3. Mechanism of spontaneous firing of output nodes in Tempo model.

The spontaneous period of firing of each output node is, by the application of a learning rule, entrained to the period of stimulation of the input node connected to it. The result of this entrainment of output nodes is that an output node is at maximum activity (i.e., an action potential is emitted) at the same time that the input node connected to it would, assuming a regular ISI, be expected to be stimulated. In particular, the rate of rise of the membrane potential of the unit is altered to adjust the period of output of the simulated neuron. A fuller description of a modified version of Torras' model called the Tempo model is provided in the next chapter.

The Tempo mechanism of ISI encoding, while it may perform well in many situations suffers from a lack of neural plausibility. Pacemaker cells exhibiting

both plasticity of the rate of rise of membrane potential and change of phase of output are known to occur in heart cells (Katz, 1992) and in ganglia in *Aplysia* (Torras, 1985). The existence of cells with these properties in the mammalian brain is uncertain. The Tempo rule as formulated in this study requires that each output node has the ability to store and retrieve the time since last input and time since last output as well as the period of stimulation. The storage of these parameters within the network architecture described is troublesome. One potential mechanism by which these parameters could be stored is by concentrations of particular substances related to the input to and output from the cell. For example, the concentration of a substance produced when the cell receives action potentials could be used as a measure of the time interval from the time of the last stimulation to the present. Similarly, the concentration of a substance produced during the firing of a cell could be used as an index of the interval since the last output.

Putting aside the question of neuronal plausibility the pacemaker entraining method of time interval learning has some important differences to the delay line theories described earlier. In the delay line theories it was necessary to specifically allow for a mechanism by which the units representing a stimulus could learn to predict future presentations of themselves, if the model was to successfully encode the ISI of single stimulus. The pacemaker learning rule can easily learn the ISI of a single stimulus as each pacemaker neuron becomes entrained to the period of input to it. The pacemaker rule, however, is unable to provide a method by which the repeated presentation of two different stimuli with a regular temporal relationship will lead to the presentation of the first stimulus predicting the occurrence of the second stimulus. The pacemaker model lacks connections enabling the associative linking of the representations of two stimuli of the delay line theory. Nevertheless, the pacemaker theory of temporal encoding is interesting in that it requires no additional specialised

connections to be added to existing, non-temporally sophisticated networks yet still endows them with some ability to learn and store temporal sequences.

9.6. Relationship to other models of habituation

The question of whether the traditional habituation models described in chapter 1 can easily be reformulated as neural network models is now addressed.

It will be recalled that Sokolov's (1960, 1963) comparator model , Wagner's Priming (1978) and SOP (1981) models and Öhman's (1979) information processing models of habituation were based on the notion that an orienting response would only be elicited if there was a mismatch between the afferent stimulation and the activity in a pre-existing neuronal model (Sokolov's model) or short term store (Wagner's and Öhman's models). It is suggested in this thesis and demonstrated in the next chapters that the neuronal model or short term store can simply be envisaged as a set of units in a neural network model. The dissimilarity of the activity in the units representing the neuronal model and the units receiving the afferent input can then be calculated and interpreted as the orienting response. The sensitivity of the particular model to recovery of the neuronal model due to changes in the nature of stimulation is then dependent on the particular architecture and learning rule/s utilised in the network model.

Wagner (1981) proposes a neural network implementation of SOP designed to produce various phenomena observed in classical conditioning. In particular, he specifies stimuli with reference to the CS and US, a convention unnecessary in the discussion of habituation. It must be remembered that the prime focus of the current thesis is the effect of temporal predictability on habituation. The major relevant difference between Wagner's theories and Sokolov's theory was Wagner's specification of retrieval-generated and self-generated priming. Some of the models tested in the current thesis include processes analogous to

retrieval and self-generated priming but implement these processes differently to Wagner's model. Therefore, conclusions which may be drawn from the simulations presented in the next few chapters do not necessarily apply to SOP itself, but rather to any theory of OR elicitation which utilises processes which could conceivably be regarded as retrieval-generated and self-generated priming.

Dual-process theory (Groves & Thompson, 1970; Thompson et al., 1979) has the advantage of strong supporting neurobiological evidence (see Chapter 1) for both the processes of inferred habituation and inferred sensitisation. It will also be recalled from Chapter 1 that a simple circuit encapsulating dual process theory could be that depicted in Figure 9.4.
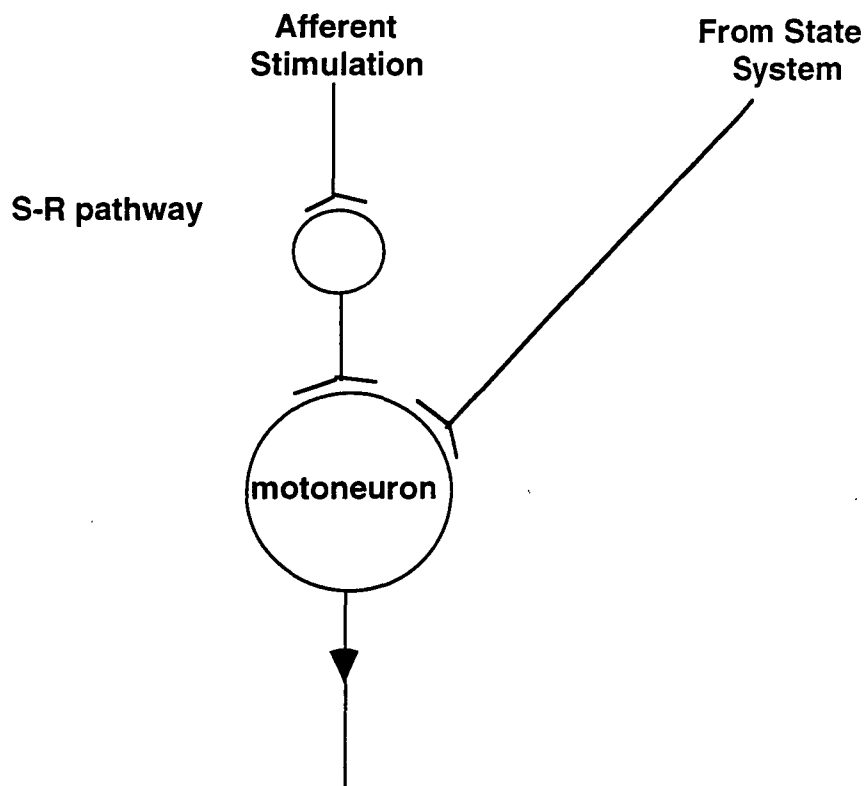
Figure 9.4. Simple neuron arrangement for dual-process theory

It will be recalled that resultant output from the motoneuron was due to the superimposed activity from the afferent neurons stimulated by the input, and

from the internal state system. Response habituation was a result of the development of inferred habituation in the S-R pathway while inferred sensitisation could develop in the state system. The modelling of the dual-process theory as a neural network model is thus, at one level, already complete. All that is required is to suggest the functions which relate the input to inferred habituation (Gluck & Thompson, 1987, present a formula suggesting that inferred habituation could be represented by the connection weight of the S-R pathway and simply decays linearly with rate β_2 with each presentation of the same stimulus), the formulae defining the development of inferred sensitisation in the state system and finally the function linking the actual input, inferred habituation and inferred sensitisation to the observed response. The nature of these functions is however open to some speculation and it is possible that one or other of the neural network models could play some part in these functions. In particular, it was noted that the state system was suggested by Thompson et al. (1979) as the locus of ISI encoding which could then lead to the response to stimulus omission. Perhaps, while the dual-process model is significantly different in conception to the comparator-like models of habituation there is still some place for a comparator-like mechanism in the state system portion of the dual-process theory.

The next chapters will describe an attempt to devise and simulate some neural network models of habituation capable of accounting for the results of the experiments reported earlier in the dissertation. While none of the models is a faithful implementation of the models discussed in Chapter 1, it will be shown that many of the processes and features contained in them could be considered translations of the notions of the earlier models.

CHAPTER 10

Simulation of Five Models of Habituation

10.1. Specifications of the models

The five connectionist models of habituation tested in this thesis can be most simply considered as one architecture, to which is applied one of five different learning rules with each rule being a mathematical description of the process and limits of plasticity implicit within a particular theoretical approach. The architecture and processes common to all models are described first, followed by the specific concepts and processes of each individual model. Each model is described informally with a formal statement of each model following.

10.1.1. General architecture and processes

All simulations utilised the same basic neuronal architecture although some individual models added extra connections to the basic structure. The architecture can be visualised as consisting of four layers. The first two layers (the input and trace layers) represent the model's input interface with the outside world and the trace of previous stimulation respectively. Layers 3 and 4 are concerned with the calculation of the measure of dissimilarity between the input and trace layers with the fourth layer containing a single output unit, the activation of which represents the OR. The configuration of layers 3 and 4 is not altered between the five models presented in this chapter. All differences between the five models are implemented through alterations of the learning rules and range of connections in the input and trace layers.

In each of the five models 20 input nodes, denoted by I, were connected directly to 20 trace nodes denoted by $\emptyset$. The ith input node was connected only to the ith trace node. During development of the model more extensive connections were tested (e.g., connecting the ith input node to trace nodes i-1 ,i and i +1) but these were rejected in favour of the current simpler arrangement. Each

input node and trace node was additionally connected to the third layer of units to allow calculation of the dissimilarity measure. The rationale behind the architecture is that the activity in the input layer is used to represent the properties of afferent stimulation with the activity in the trace layer representing an internal trace of previous stimulation. The orienting response was represented by a measure of dissimilarity between the activity in the input and trace layers. The models were, therefore, essentially comparator-like models of habituation.

Representation in the input layer was topographic with similar stimuli represented by near or overlapping nodes while less similar stimuli were represented by more distant nodes. The concept of overlapping of the representations of similar stimuli was similar to that in SOP (Wagner, 1981) or stimulus sampling theory (Estes, 1955a, 1955b). Different modalities were represented by different sets of the input nodes. In the current model with 20 input nodes, light stimuli were represented by nodes 1-7, with nodes 8-20 representing tone stimuli. A representation of a single tone or light stimulus may be represented as activity in a number of input nodes, but can not include nodes in both the light and tone sections of the input layer. Alternatively, the single input layer could be envisaged as a number of separate input layers with each representing stimuli of a different modality. The activity of a particular node in the input layer, a_i, may take any value between 0 (no activity) and 1 (maximum activity). Stimulus intensity was not varied in the current simulations so inputs were classified only as active (1) or inactive (0).

An important point to note is that in all the nodes of the network models in the thesis the output function was the identity function with $O_i=a_i$ for all nodes. The formulae presented use a_i in preference to O_i but the reader should keep in mind that both values are identical for a given node.

An example of the architecture of the models is shown in Figure 10.1. Only two input nodes and their connections are shown.
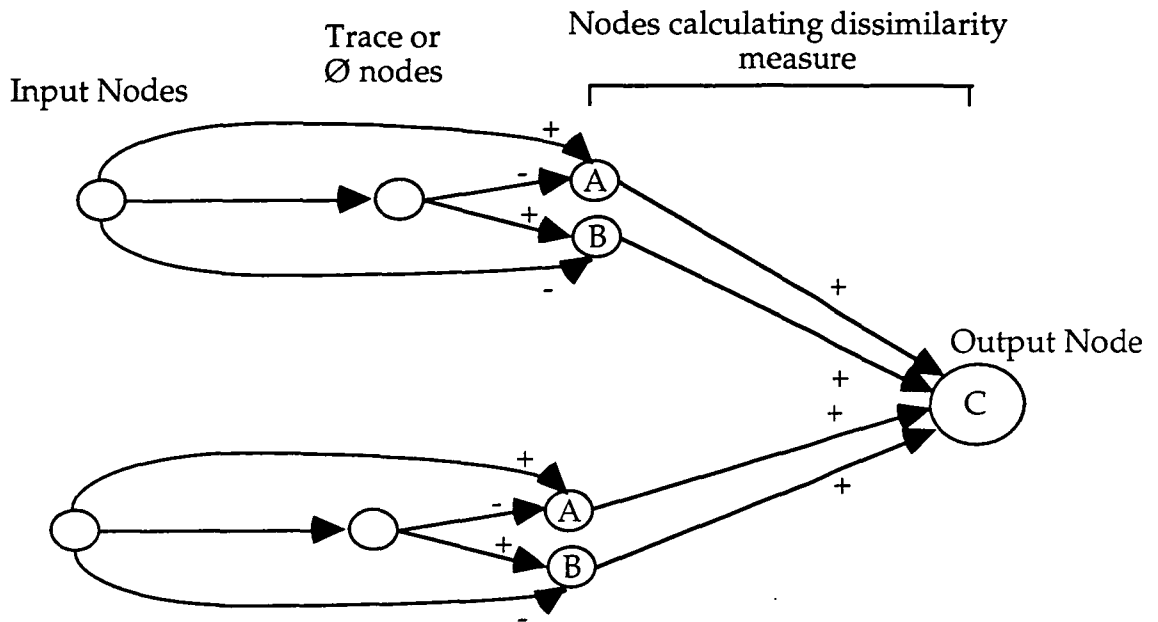
Figure 10.1. Basic architecture of connectionist models of habituation

The connections or weights, w_{ij}, between node i in the input and node j in the trace layer are initialised with small random values. They may be adjusted during the course of the experiment depending on the learning rule being used.

Recalling the notation of a_{Ii} and $a_{\emptyset i}$ for the activation of node i in the input and trace layers respectively activity in a trace layer node $a_{\emptyset i}$ in the basic model was given by the formula

$$a_{\emptyset i} = tanh \sum_{j} w_{ji}a_{Ij}$$

where

a_{Ij} = activity in input node j

w_{ji} = weight of connection between input node j and trace node i

a_{Oi} = activity in trace node i

The activity in a trace node was thus a weighted function of the activity in all the input nodes. In the current architecture the only non-zero connections were those connecting input nodes to single trace nodes where $i=j$ (e.g., connecting input node 10 to trace node 10). The formula for a_{Oi} in these simulations could therefore be simplified to

$$a_{Oi} = tanh\ (w_{ii}\ a_{Ii})$$

The *tanh* function was chosen to give desirable characteristics to the activation function, in particular to give an activation of 0 when $a_i=0$ and to limit the maximum activation of a trace node to 1.

The orienting response was modelled in the simulations by a measure of the dissimilarity between the input and trace layers. If the activity in both layers was identical then no response would be elicited whereas any differences in activity between the input and trace layers would result in the elicitation of a response. The orienting response, D, varied in direct proportion to the degree of mismatch between the activity in the input and trace layers. In the simulations the value of the response was calculated by

$$D = \frac{\sqrt{\sum_i (a_{\emptyset i} - a_{Ii})^2}}{\sqrt{n}}$$

with $n=20$ and the other the variables as above.

The question of neurobiological plausibility of this method of calculation of the orienting response can be partially answered by the neuronal arrangement of layers 3 and 4 in Figure 10.1. The connection weights from the input and trace layer nodes to layer 3 nodes, as well as the connections between layers 3 and 4 were not changed during an experiment.

Units in layer 3 are classified into two types, A and B, with each type receiving different connections from the input and trace layers and possessing different activation functions. In the arrangement shown in Figure 10.1 all input nodes I_1-I_n have excitatory synapses (denoted with a + sign) on nodes A and inhibitory synapses (denoted by a - sign) on nodes B. Each trace layer node $\emptyset_1$-$\emptyset_n$ had an inhibitory synapse on nodes A and an excitatory synapse on nodes B. A and B nodes had activations S defined by

$$S_A = max[\left(\sum_j a_{Ij} - \sum_j a_{\emptyset j}\right), 0] \quad \text{for A}$$

$$S_B = max[\left(\sum_j a_{\emptyset j} - \sum_j a_{Ij}\right), 0] \quad \text{for B}$$

where

a_{Ij} = activity of input node j

Both *A* and *B* nodes were connected to the comparator node *C* of layer 4, the output of which may be called *D*, or the dissimilarity between the activity in the input and trace layers.

The neuronal assembly described above was capable of calculating a measure of the difference between the input and trace vectors, regardless of the direction of the disparity. The formula for *D* calculated in this fashion could be written

$$D = \frac{1}{\sqrt{n}} \sum_i \sqrt{(a_{\emptyset i} - a_{li})^2}$$

The $\frac{1}{\sqrt{n}}$ term was a constant scaling factor used to restrict the maximum dissimilarity in the current simulations. The above descriptions outline the basic network architecture which was central to each of the five habituation models which were simulated. Each model will now be described in turn. It must be noted that the differences between the five models lie only in the processes in the units of the input and trace layers and the connections between those layers. The same dissimilarity measure was used for all models.

10.1.2. Delta model

This model is so called because it utilises the delta rule (Rumelhart et al., 1986) first suggested by Widrow and Hoff (1960) as the means of learning the internal representation of previous stimulation. The course of learning with the delta rule is that it is assumed that some "supervisor" has an accurate knowledge of the desired output of each trace node when a particular input pattern is presented to the network. As patterns of input are repeatedly presented the weights of the connections between each node in the input and trace layers are adjusted in a direction, and by an amount, dependent upon the difference between the current activity in the trace node and the desired activity in that node and the level of activity in the input node associated with that connection. In the current model the target input for each trace node was defined as the activity (and given that the output function of the all nodes in the current

models was the identity function then activation=output for all nodes) of the input node connected to that trace node. The amount of adjustment Δw_{ij} for each connection in the network was calculated by the formula

$$\Delta w_{ij} = d(a_{li} - a_{\emptyset j})a_{li}$$

with

d = learning rate for the Delta model = 0.7 in the current model

and the other symbols having their previous meanings.

Given that in the current architecture each input node was connected to only one trace node we can see that the learning rule in the Delta model is such that, given no further modification, after a number of iterations the weights would be adjusted so that the activity in the trace layer resembles that presented to the input layer. In the first few iterations the dissimilarity D between the patterns of activity in the input and trace layers would be greatest and a large "orienting response" would be elicited, while after a greater number of iterations the dissimilarity between the two layers would decrease and the response would be seen to habituate.

In addition to the delta learning rule, the connection weights in the Delta model were subject to decay between each iteration of the experimental simulations. This decay term was included in the Delta model to simulate a simple form of forgetting and provide a simple mechanism of simulating the effect of ISI duration on the rate of habituation.

The course of calculation of the Delta model is described below.

Initialisation occurs once at the beginning of each simulated experiment.

$$w_{ij}=0.10$$

where

$w_{ij}=$ the connection weight of the connection from input node i to trace node j

The following calculations were performed for each iteration of the simulated experiment.

Input vector A to network

$$A = (a_{I1}, a_{I2}, ..., a_{I19}, a_{I20})$$

Calculate Activation of Trace Units

$$a_{\varnothing j} = tanh \sum_j w_{ji} a_{Ii}$$

Calculate Dissimilarity

$$D = \frac{\sqrt{\sum_i (a_{\varnothing i} - a_{Ii})^2}}{\sqrt{n}}$$

Adjust and Decay Weights

$$w_{ij} = \begin{cases} w_{ij}' + d(a_{Ii} - a_{\varnothing j})a_{Ii} - p & w_{ij} > 0 \\ w_{ij}' + d(a_{Ii} - a_{\varnothing j})a_{Ii} + p & w_{ij} < 0 \\ w_{ij}' + d(a_{Ii} - a_{\varnothing j})a_{Ii} & w_{ij} = 0 \end{cases}$$

where

$d=$ learning rate for delta rule learning$= 0.7$

$p =$ decay constant $= 0.01$ in these simulations

$w_{ij}' =$ previous value of connection weight

The Delta model could de considered to be analogous to a comparator model of habituation in which the internal trace of past stimulation encodes only the physical stimulus parameters, including intensity, tone frequency and modality. There was however no means by which the internal trace in the Delta model could encode the ISI of a stimulus, or the temporal relationship between a number of stimuli.

10.1.3. Tempo model

The Tempo model incorporates a pacemaker learning algorithm allowing the internal trace of past stimulation to include temporal information, specifically the ISI of a stimulus. The Tempo model was based on Torras' (1985) work on models of entrainment of pacemaker neurons. The calculations for the Tempo model were as follows:

Initialisation:

$$w_{ij} = 0.10$$

$$R_{\emptyset j} = R_{min} + Random(R_{max} - R_{min})$$

$$R_{in\emptyset j} = R_{min} + Random(R_{max} - R_{min})$$

$$t_{in\emptyset j} = 1$$

$$t_{out\emptyset j} = 1$$

$$\omega_{in\emptyset j} = 1$$

$$\omega_{out\emptyset j} = 1$$

where

$R_{\emptyset j}$= the rate of rise of membrane potential of trace node j

$R_{in\emptyset j}$= the estimate of the rate of rise of membrane potential of the input to trace node j (see below)

$t_{out\emptyset j}$= the number of iterations since the last non-zero activation of trace node j

$t_{in\emptyset j}$= the number of iterations since the last input to trace node j

$\omega_{out\emptyset j}$= period of firing of trace node j

$\omega_{in\emptyset j}$= the period of the input to trace node j

R_{max}= the upper limit of the rate of rise of membrane potential=9 (mV/s)

Rmin= the lower limit of the rate of rise of membrane potential=15 (mV/s)

In the Tempo model it was assumed that only the trace nodes have the ability to encode the period of stimulation. The following calculations were performed on each iteration of the simulated experiment:

Input vector A to network

$$A = (a_{I1}, a_{I2}, \ldots\ldots\ldots\ldots, a_{I19}, a_{I20})$$

Calculate temporary activity in trace nodes: For each trace node a temporary value $a_{temp\emptyset i}$ was calculated which corresponded to the activity evoked in a trace node by activity propagating from the input nodes. $a_{temp\emptyset i}$ was later combined with activity contributed by the periodic rise in membrane potential of the Tempo rule to give a final $a_{\emptyset i}$.

$$a_{temp\emptyset i} = \sum_j w_{ji} a_{Ij}$$

A series of calculations were then performed to update, and if necessary modify, the parameters which defined the cyclical firing of the trace nodes in the Tempo model.

Firstly, the membrane potential $P_{\emptyset j}$ of each trace node was incremented by an interval defined by the rate of potential rise $R_{\emptyset j}$ and the value of each time step in the simulation.

$$P_{\emptyset j} = P_{\emptyset j}' + R_{\emptyset j} s$$

where

$P_{\emptyset j}$ = new value of membrane potential of trace node j

$P_{\emptyset j}'$ = previous value of membrane potential

s = value of timestep for each iteration = 1 in current simulations

(i.e., each iteration represents 1 second of real experimental time)

The Tempo parameters for each trace node were then updated and modified.

If the membrane potential of a node was below the threshold H and there was no input to the node via connections from the input layer, then the parameters containing the time since the last input and output of that node were incremented by 1.

If $P_{\emptyset j} < H$ and $a_{tempo j} = 0$, that is, membrane potential below threshold and no input

$t_{in\emptyset j} = t_{in\emptyset j}' + 1$

$t_{out\emptyset j} = t_{out\emptyset j}' + 1$

with

H = the threshold membrane potential necessary for the node to fire = -10 (mV)

$t_{in\emptyset j}'$ = the previous value of $t_{in\emptyset j}$

$t_{out\emptyset j}'$ = the previous value of $t_{out\emptyset j}$

If the membrane potential exceeded the threshold then the node would output with a value $a_{T\emptyset j}$. $a_{T\emptyset j}$ represents the contribution of the Tempo learning to the activity of the node and was later combined with $a_{temp\emptyset j}$ to give a resultant value of $a\emptyset j$. If the membrane potential exceeded the threshold at a time when there is no external input to the node then the rate of rise of the membrane potential was adjusted to alter the period of firing of the trace node to more closely resemble that of the external stimulation to the node.

If $P_{\emptyset j} \geq H$ and $a_{temp\emptyset j} = 0$, that is, membrane potential above threshold and no input

$$a_{T\emptyset j} = \begin{cases} \dfrac{1}{|t_{in\emptyset j} - t_{out\emptyset j}|} & t_{in\emptyset j} \neq t_{out\emptyset j} \\ 1 & t_{in\emptyset j} = t_{out\emptyset j} \end{cases}$$

$t_{in\emptyset j} = t_{in\emptyset j}' + 1$

$t_{out\emptyset j} = 1$

$P_{\emptyset j} = P_0$

$$R_{\emptyset j} = \begin{cases} R_{\emptyset j}' + d_T(R_{in\emptyset j} - R_{\emptyset j}') & R_{min} < R_{\emptyset j}' < R_{max} \\ R_{min} & R_{\emptyset j}' < R_{min} \\ R_{max} & R_{\emptyset j}' > R_{max} \end{cases}$$

where

d_T = the learning rate for the Tempo model = 0.35

P_0 = the level of membrane potential immediately following the firing caused by the membrane potential exceeding the threshold = -100 (mV)

$R_{\emptyset j}'$ = previous value of the rate of rise of membrane potential

Alternatively, a node may not have a sufficiently high membrane potential to fire but received input via the connections from the input layer. In this case the rate of rise of membrane potential was again modified to alter the period of firing to more closely approach that of the period of stimulation. In addition, the actual level of the membrane potential was modified to move the phase of firing of the node closer to that of the stimulating input.

If $P_{\varnothing j} < H$ and $a_{temp\varnothing j} \neq 0$, that is, membrane potential below threshold and input

$O_{T\varnothing j} = 0$

$\omega_{in\varnothing j} = t_{in\varnothing j}$

$t_{in\varnothing j} = 0$

$t_{out\varnothing j} = t_{out\varnothing j}' + 1$

$R_{in\varnothing j} = \dfrac{H - P_0 + 1}{\omega_{in\varnothing j} + 1}$

$$R_{\varnothing j} = \begin{cases} R_{\varnothing j}' + d_T(R_{in\varnothing j} - R_{\varnothing j}') & R_{min} < R_{\varnothing j}' < R_{max} \\ R_{min} & R_{\varnothing j}' < R_{min} \\ R_{max} & R_{\varnothing j}' > R_{max} \end{cases}$$

$\theta_{\varnothing j} = H - (R_{\varnothing j}(\omega_{in j} + 1)$

$P_{\varnothing j} = P_{\varnothing j}' - \dfrac{d_T(P_{\varnothing j} - \theta_{\varnothing j})}{\sqrt{\dfrac{t}{\omega_{in\varnothing j}}}}$

where

$\theta_{\varnothing j}$ = the estimate of the membrane potential of the input node stimulating node j

t = the number of iterations elapsed since the start of the experiment

$P_{\varnothing j}'$ = previous value of $P_{\varnothing j}$

$t_{out\varnothing j}'$ = previous value of $t_{out\varnothing j}$

Finally, if the membrane potential exceeded the threshold level and there was an input to the node via the connections from input nodes then the node fired and the potential returned to its post firing level. The rate of rise of membrane potential was also modified if it was not already the same as that estimated for the stimulating input.

If $P_{\emptyset j} \geq H$ and $a_{temp\emptyset j} \neq 0$, that is, membrane potential above threshold and input

$$\omega_{out\emptyset j} = \begin{cases} t_{out\emptyset j} & t_{out\emptyset j} \neq 0 \\ 1 & t_{out\emptyset j} = 0 \end{cases}$$

$$\omega_{in\emptyset j} = \begin{cases} t_{in\emptyset j} & t_{in\emptyset j} \neq 0 \\ 1 & t_{in\emptyset j} = 0 \end{cases}$$

$$t_{in\emptyset j} = 0$$

$$t_{out\emptyset j} = 1$$

$$R_{in\emptyset j} = \frac{H - P_0 + 1}{\omega_{in\emptyset j} + 1}$$

$$O_{T\emptyset j} = \begin{cases} \dfrac{1}{|t_{in\emptyset j} - t_{out\emptyset j}|} & t_{in\emptyset j} \neq t_{out\emptyset j} \\ 1 & t_{in\emptyset j} = t_{out\emptyset j} \end{cases}$$

$$P_{\emptyset j} = P_0$$

Having updated all the Tempo rule parameters the final activation $a_{\emptyset j}$ for each trace node was calculated and the dissimilarity between the input and trace patterns calculated.

Recalculate trace node activity

$$a_{\emptyset j} = tanh(a_{temp\emptyset j} + a_{T\emptyset j})$$

Calculate Dissimilarity measure

$$D = \frac{\sqrt{\sum_i (a_{\emptyset i} - a_{Ii})^2}}{\sqrt{n}}$$

The essential feature of learning in the Tempo rule is that each trace node becomes entrained to the cycle of input by modification of both the rate at

which the membrane potential rises (which adjusts the period of firing of the trace node) and making direct changes to the level of the membrane potential (adjusting the phase of the firing cycle). The variety of ISIs which can be encoded by a trace node is determined by the minimum and maximum values which can be taken by the membrane potential of that node.

Assuming that the Tempo model allows each trace node to encode its ISI of stimulation (and remembering that each trace node is connected to only one input node) the Tempo rule could be expected to show some sensitivity of habituation to the temporal predictability of a stimulus series. This would be shown in experimental situations by a slowing of habituation (the response to a stimulus would be preserved for a greater number of trials) when a stimulus is presented at a variable ISI compared to the same stimulus presented at a constant ISI. There would also be expected to be a response to complete stimulus omission. This would occur because the trace nodes representing the stimulus would, after a number of presentations, have learned to produce an output at the time the stimulus is presented. When the stimulus is omitted the trace nodes will continue to fire at the expected time of presentation resulting in a disparity between the input and trace activity patterns of the network, which is reflected as in increase in dissimilarity interpreted as a response to stimulus omission.

ISI duration would not necessarily be anticipated to have an effect on the course of habituation in the Tempo model. Learning of ISI should not be more rapid (requiring less stimulus presentations) for short or long ISIs, however there remains the possibility that the initial spontaneous rate of rise in the membrane potential of each node may favour particular ISIs. For example, a node with an initially rapid spontaneous rise in membrane potential would have a short initial spontaneous ISI and would be expected to learn a shorter ISI more rapidly than a longer ISI.

Because the connection weights are not adjusted within the Tempo model it is difficult to see how the model can encode the physical aspects of the stimulus presented to it. The Tempo mechanism however enables the network to perform similarly to a purely physical encoder in a number of ways. The learning results in the stimulated trace nodes being more likely to respond at the time of stimulus presentation as the number of presentations increases, this results in a decrease in dissimilarity between the input and trace nodes which is interpreted as habituation of the orienting response.

An aspect of the Tempo functions that deserves closer examination is the calculation of the output of each trace node as a function of the difference between the intervals from the last input to and last output from that node. In simulations where a number of presentations of one stimulus are input to a network, many trace nodes will not be stimulated at all during an experiment. The time since last output will therefore increase with each time step of the simulation. The time since last output cannot increase indefinitely because it is limited by the lower bound on the rate of rise of membrane potential for each node. Therefore, if the node is not stimulated, will eventually settle to an output period determined by this lower bound. The result of the ever increasing value of t_{in} means that the output from trace nodes which are never stimulated will tend to decrease as the experiment proceeds. This means that there is a further reduction of the dissimilarity between the input and trace nodes on each input presentation because not only will the stimulated nodes become more likely to respond at the correct time as more stimuli are presented but non-stimulated nodes will also tend to emit smaller responses as the length of the experiment increases. In an alternative formulation of the model (not reported in the thesis), the output of each trace node when was simply 1 whenever the membrane potential exceeded the threshold. The result of this modification was that in the non-stimulated nodes the rate of rise of the membrane potential eventually settled at its minimum level. Because there was

no stimulation of these nodes there was no learning of the phase of stimulation to these nodes so the output from them tended to form a cyclical pattern which persisted throughout the simulation. When the dissimilarity between the input and trace at each presentation was measured this manifested as a cyclical and relatively high residual level of dissimilarity at each input presentation, presumably a result of non-stimulated nodes emitting outputs at the same time the stimulus was presented. With the current learning rule, the output from the non-stimulated nodes decreased later in the experiment and the major determinant of the dissimilarity at the time of presentation was the level of prediction of the input by the stimulated nodes.

10.1.4. DelTempo model

The DelTempo model was intended as a modification of the Tempo model in which the input-trace layer connections could be modified through experience and decay between trials. It was therefore expected that the DelTempo model would show both the characteristics of the Tempo model such as the sensitivity to ISI variability and the response to complete stimulus omission as well as the characteristics of the Delta model such as the sensitivity to ISI duration. It was also expected that the DelTempo model would display generalisation of habituation, as would be expected when the Delta and Tempo models were simulated individually.

The calculations for the DelTempo model were as follows. All parameters have their previously defined meanings and/or values.

Initialisation:

$$w_{ij}=0.10$$

$$R_{\varnothing j}=R_{min}+Random(R_{max}-R_{min})$$

$$R_{in\varnothing j}=R_{min}+Random(R_{max}-R_{min})$$

$$tin_{\emptyset j}=1$$

$$tout_{\emptyset j}=1$$

$$\omega in_{\emptyset j}=1$$

$$\omega out_{\emptyset j}=1$$

The following calculations were performed on each iteration of the simulated experiment:

Input vector A to network

$$A = (a_{I1}, a_{I2}, \ldots\ldots\ldots\ldots, a_{I19}, a_{I20})$$

Calculate temporary activity in trace nodes: For each trace node a temporary value $a_{temp\emptyset i}$ was calculated which corresponded to the activity evoked in a trace node by activity propagating from the input nodes. $a_{temp\emptyset i}$ was later combined with activity contributed by the periodic rise in membrane potential of the Tempo rule to give a final $a_{\emptyset i}$.

$$a_{temp\emptyset i} = \sum_j w_{ji} a_{Ij}$$

A series of calculations were then performed to update, and if necessary modify, the parameters which defined the cyclical firing of the trace nodes in the Tempo model.

Firstly the membrane potential $P_{\emptyset j}$ of each trace node was incremented by an interval defined by the rate of potential rise $R_{\emptyset j}$ and the value of each time step in the simulation.

$$P_{\emptyset j} = P_{\emptyset j}' + R_{\emptyset j} s$$

where

$P_{\emptyset j}$ = new value of membrane potential of trace node j

$P_{\emptyset j}'$ = previous value of membrane potential

s = value of timestep for each iteration = 1 in current simulations

(i.e., each iteration represents 1 second of real experimental time)

The Tempo parameters for each trace node are then updated and modified.

If the membrane potential of a node was below the threshold H and there was no input to the node via connections from the input layer, then the parameters containing the time since the last input and output of that node were incremented by 1.

If $P_{\emptyset j} < H$ and $a_{temp\emptyset j} = 0$, that is, membrane potential below threshold and no input

$t_{in\emptyset j} = t_{in\emptyset j}' + 1$

$t_{out\emptyset j} = t_{out\emptyset j}' + 1$

with

H = the threshold membrane potential necessary for the node to fire = -10 (mV)

$t_{in\emptyset j}'$ = the previous value of $t_{in\emptyset j}$

$t_{out\emptyset j}'$ = the previous value of $t_{out\emptyset j}$

If the membrane potential exceeded the threshold then the node fired with a value $a_{T\emptyset j}$. $a_{T\emptyset j}$ represents the contribution of the Tempo learning to the activity of the node and was later combined with $a_{temp\emptyset j}$ to give a resultant value of $a\emptyset j$. If the membrane potential exceeded the threshold at a time when there was no external input to the node then the rate of rise of the membrane potential was adjusted to alter the period of firing of the trace node to more closely resemble that of the external stimulation to the node.

If $P_{\emptyset j} \geq H$ and $a_{temp\emptyset j} = 0$, that is, membrane potential above threshold and no input

$$a_{T\emptyset j} = \begin{cases} \dfrac{1}{|t_{in\emptyset j} - t_{out\emptyset j}|} & t_{in\emptyset j} \neq t_{out\emptyset j} \\ 1 & t_{in\emptyset j} = t_{out\emptyset j} \end{cases}$$

$$t_{in\emptyset j} = t_{in\emptyset j}' + 1$$

$$t_{out\emptyset j} = 1$$

$$P_{\emptyset j} = P_0$$

$$R_{\emptyset j} = \begin{cases} R_{\emptyset j}' + d_T(R_{in\emptyset j} - R_{\emptyset j}') & R_{min} < R_{\emptyset j}' < R_{max} \\ R_{min} & R_{\emptyset j}' < R_{min} \\ R_{max} & R_{\emptyset j}' > R_{max} \end{cases}$$

where

d_T = the learning rate for the Tempo model=0.35

P_0 = the level of membrane potential immediately following the firing caused by the membrane potential exceeding the threshold=-100 (mV)

$R_{\emptyset j}'$ = previous value of the rate of rise of membrane potential

Alternatively, a node may not have had a sufficiently high membrane potential to fire but received input via the connections from the input layer. In this case the rate of rise of membrane potential was modified to alter the period of firing to more closely approach that of the period of stimulation. In addition, the actual level of the membrane potential was modified to move the phase of firing of the node closer to that of the stimulating input.

If $P_{\emptyset j} < H$ and $a_{tempoj} \neq 0$, that is, membrane potential below threshold and input

$O_{T\emptyset j} = 0$

$\omega_{in\emptyset j} = t_{in\emptyset j}$

$t_{in\emptyset j} = 0$

$t_{out\emptyset j} = t_{out\emptyset j}' + 1$

$R_{in\emptyset j} = \dfrac{H - P_0 + 1}{\omega_{in\emptyset j} + 1}$

$R_{\emptyset j} = \begin{cases} R_{\emptyset j}' + d\tau(R_{in\emptyset j} - R_{\emptyset j}') & R_{min} < R_{\emptyset j}' < R_{max} \\ R_{min} & R_{\emptyset j}' < R_{min} \\ R_{max} & R_{\emptyset j}' > R_{max} \end{cases}$

$\theta_{\emptyset j} = H - (R_{\emptyset j}(\omega_{inj} + 1)$

$P_{\emptyset j} = P_{\emptyset j}' - \dfrac{d\tau(P_{\emptyset j} - \theta_{\emptyset j})}{\sqrt{\dfrac{t}{\omega_{in\emptyset j}}}}$

where

$\theta_{\emptyset j}$ = the estimate of the membrane potential of the input node stimulating node j

t = the number of iterations elapsed since the start of the experiment

$P_{\emptyset j}'$ = previous value of $P_{\emptyset j}$

$t_{out\emptyset j}'$ = previous value of $t_{out\emptyset j}$

Finally, if the membrane potential exceeded the threshold level and there was an input to the node via the connections from input nodes then the node fires and the potential returned to its post firing level. The rate of rise of membrane potential was also modified if it was not already the same as that estimated for the stimulating input.

If $P_{\emptyset j} \geq H$ and $a_{temp\emptyset j} \neq 0$, that is, membrane potential above threshold and input

$$\omega_{out\emptyset j} = \begin{cases} t_{out\emptyset j} & t_{out\emptyset j} \neq 0 \\ 1 & t_{out\emptyset j} = 0 \end{cases}$$

$$\omega_{in\emptyset j} = \begin{cases} t_{in\emptyset j} & t_{in\emptyset j} \neq 0 \\ 1 & t_{in\emptyset j} = 0 \end{cases}$$

$$t_{in\emptyset j} = 0$$

$$t_{out\emptyset j} = 1$$

$$R_{in\emptyset j} = \frac{H - P_0 + 1}{\omega_{in\emptyset j} + 1}$$

$$O_{T\emptyset j} = \begin{cases} \dfrac{1}{|t_{in\emptyset j} - t_{out\emptyset j}|} & t_{in\emptyset j} \neq t_{out\emptyset j} \\ 1 & t_{in\emptyset j} = t_{out\emptyset j} \end{cases}$$

$$P_{\emptyset j} = P_0$$

Having updated all the Tempo rule parameters the final activation $a_{\emptyset j}$ for each trace node was calculated and the dissimilarity between the input and trace patterns was calculated.

Recalculate trace node activity

$$a_{\emptyset j} = tanh(a_{temp\emptyset j} + a_{T\emptyset j})$$

Calculate dissimilarity of input and trace patterns

$$D = \frac{\sqrt{\sum_i (a_{\emptyset i} - a_{li})^2}}{\sqrt{n}}$$

In addition to the preceding calculations which were identical to those for the Tempo model, the DelTempo model also includes adjustment of weights of the

connection weights between the input and trace nodes according to the delta learning rule.

$$w_{ij} = \begin{cases} w_{ij}' + d(a_{li} - a_{\emptyset j})a_{li} - p & w_{ij}' > 0 \\ w_{ij}' + d(a_{li} - a_{\emptyset j})a_{li} + p & w_{ij}' < 0 \\ w_{ij}' + d(a_{li} - a_{\emptyset j})a_{li} & w_{ij}' = 0 \end{cases}$$

10.1.5. Prime model

The Prime model of habituation was designed to include the associative cuing or priming concepts of the Priming (Wagner, 1978), SOP (Wagner, 1981) and information processing (Öhman, 1979) models of habituation. In essence, it provided the basic model of habituation with the ability to learn the interval between two inputs to the network. The interval may be between inputs to two different nodes, or between successive inputs to one node.

A modification to the basic network architecture was required to allow the implementation of the Prime model. Horizontal (or lateral) connections were defined linking each input node with all other input nodes. These horizontal connections were used to store the cuing information between pairs of input nodes. Each connection had two parameters which were adjusted during the course of learning. These parameters were the delay between the pair of inputs to the two nodes and the strength of the connection between the pair of nodes. The connection strength between a pair of nodes increased each time the nodes were stimulated with a consistent time interval between them. The delay between the stimulation of each unit was learnt with one pair of presentations to the network and only modified if the relevant pair of nodes were stimulated at a different interval.

The function of the lateral connections was to provide a mechanism of cuing of the presentation of one stimulus (e.g., S2) by the presentation of a preceding

stimulus (e.g., S1) that had previously reliably cued the presentation of the S2. If both the S1 and S2 are in fact repeated presentations of a single stimulus then the lateral connections become encoders of the ISI of that stimulus. The lateral connections were therefore agents of retrieval-generated priming. It was mentioned earlier in the thesis when the Priming and SOP models were reviewed that it was uncertain if retrieval-generated priming was intended to act both with one stimulus cuing the presentation of a second stimulus, and also with repeated presentations of a single stimulus cuing each other. The Prime model implemented here explicitly allows that a stimulus presentation can be a cue for future presentations of the same stimulus.

Cuing or priming is implemented in the Prime model as a process which is active entirely in the input layer. If a cuing stimulus has been presented to the network then, after an appropriate time interval, the nodes representing that stimulus will pass activation along horizontal connections to the nodes representing the cued stimulus. This additional cuing or priming activation will be added to any afferent stimulation at the nodes representing the cued stimulus and processed through the vertical connections to the trace nodes as if it was an external stimulus.

It was seen in the empirical studies that the habituation phenomena requiring encoding of the temporal characteristics of a series of stimuli were more marked at shorter ISIs than at longer ISIs. The Prime model, like the Tempo model, therefore required a mechanism which specifically limits the temporal cuing ability of the horizontal connections in the input layer to encode temporal information to a particular range of ISIs. In the current formulation of the Prime model this was achieved by allowing a modification of lateral connection weights to occur only if the interval between two stimulus presentations was within an eligibility period of 10 seconds.

The Prime learning rule learns similar properties to the delay line theories discussed in the previous chapter and a delay line architecture could be used to implement the calculations necessary for the Prime model. In the current example the horizontal input layer connections were assumed to perform all these calculations themselves. It may be more neurobiologically plausible to replace each horizontal connection with a set of delay lines but it was considered that the benefits of this substitution were not sufficient to outweigh the added complexity. It would be surprising if the use of a delay line model provided qualitatively different performance to the Prime model as it stands.

In the basic Prime model where the vertical input layer to trace layer connections were not modified during the course of the experiment the introduction of an eligibility period produces some unusual phenomena. If we simply consider the repeated presentation of a single stimulus at a regular ISI then, from empirical studies, it would be expected that habituation would occur across a wide range of ISIs. With the Prime model habituation is predicted to occur only if the ISI of the stimulus series is shorter than the eligibility period. In this situation the presentation of a stimulus will eventually come to prime the next presentation of the same stimulus, resulting in a decrease in the dissimilarity between the input and trace layers which results in habituation across a number of presentations. If, however, the ISI of the stimulus series is greater than the eligibility period then there will be no cuing between successive stimulus presentations. Because there is no plasticity of the vertical input to trace layer connections, the network will not change throughout the experiment and no habituation will be observed.

The calculations for the Prime model are described below.

Initialisation:

$$wij=0.10$$

$$tout_{Ii}=10000$$

$$alast_{Ii}=0$$

$$wP_{iIj}=0.00$$

$$\partial_{Iij}=10000 \qquad \text{n.b. 10000 is an arbitrary large integer}$$

where

$alast_{Ii}$=size of previous input to input node i

wP_{iIj}=weight of priming connection between input nodes i and j

∂_{Iij}=interval between inputs to input nodes i and j

and the other symbols have their previous meanings

On each iteration:

Input vector A to network

$$A = (a_{I1}, a_{I2}, \ldots\ldots\ldots\ldots, a_{I19}, a_{I20})$$

The priming connections in the input layer were then adjusted and the amount of contribution of priming to the activation of each input node calculated.

First, the time since the last input to each input node was incremented or reset to 1.

$$tout_{Ii} = \begin{cases} tout_{Ii}'+1 & a_{Ii} = 0 \\ 1 & a_{Ii} \neq 0 \end{cases}$$

The connection weights between the input nodes were then adjusted according to the following rules. If there was an input to input node j and the time since the last input to node i was within a prescribed eligibility period E then the priming weight between nodes i and j was increased. If the time since an input was presented to node i was equal to the encoded offset between nodes i and j and no input was presented to node j then the priming weight between nodes i and j was decreased.

$$w_{Pij} = \begin{cases} min(w_{Pij}'+d_P,1) & a_j \neq 0, t_{outii} < E \\ max(w_{Pij}'-d_P,0) & a_j = 0, t_{outii} = \partial_{ij} \\ w_{Pij}' & otherwise \end{cases}$$

where

d_P = the learning rate for the priming rule=0.002

w_{Pij}' = the previous value of w_{Pij}

E=the eligibility period within which prime learning may occur=10 iterations (seconds)

The offset ∂_{ij} between input nodes i and j was modified if there was an input to node j and the time since an input was presented to node i was within the eligibility period. ∂_{ij} was then set to the interval between the two inputs.

$$\partial_{ij} = \begin{cases} t_{outii} & a_{ij} \neq 0, t_{outii} < E \\ \partial_{ij}' & otherwise \end{cases}$$

where

∂_{ij}' = the previous value of ∂_{ij}

A quantity P_{Ii} was then calculated which represented the augmentation of the activity of a particular input node due to priming from other input nodes.

$$P_{Ii} = \left\{ \sum_j a_{last_{ij}} w_{P_{ij}} \qquad \textit{if } t_{out_{ij}} = \partial_{ij} \right.$$

The activity in each trace node was then calculated according to the formula

$$a_{\varnothing_j} = \tanh \sum_i \left(w_{ij} a_{Ii} + P_{Ij} \right)$$

This formula is slightly different to that intended when the model was conceived as P_{Ij} is included i times in the summation of the weighted inputs to node j. This was however the formula used in the construction of the program used in calculating the simulations and it appeared to have no ill effects on the functioning of the model. Because of the architecture of the model where each input node was connected to only one trace node the formula used still takes account of any priming of the input nodes representing the stimulus from input nodes which have previously been stimulated within the eligibility period. A more intuitively plausible formula for the calculation of $a_{\varnothing_j}$ taking into account the weighted inputs and priming from other nodes would however be:

$$a_{\varnothing_j} = \tanh \left(\sum_i \left(w_{ij} (a_i + P_{Ii}) \right) \right)$$

The dissimilarity between the input and trace activity patterns was then calculated as before

$$D = \frac{\sqrt{\sum_i (a_{\varnothing_i} - a_{Ii})^2}}{\sqrt{n}}$$

Finally the weights of the horizontal priming connections in the input layer were decayed

$$wP_{ij} = wP_{ij}' - \frac{dP}{100}$$

10.1.6. PrimeDelta model

The Prime Delta model was a modification of the Prime model in which the vertical input-trace layer connections were able to learn using the Delta rule. This provides the Prime model with the sensitivity to ISI duration of the Delta model while maintaining the sensitivity to ISI variability, response to complete stimulus omission and generalisation of habituation of the Prime model. The calculations for the PrimeDelta model are shown below. All symbols have their previously defined meanings and/or values.

Initialisation:

$w_{ij}=0.10$

$tout_{Ii}=10000$

$alast_{Ii}=0$

$wP_{iIj}=0.00$

$\partial_{Iij}=10000$ *n.b. 10000 is an arbitrary large integer*

where

$alast_{Ii}$=size of previous input to input node i

wP_{iIj}=weight of priming connection between input nodes i and j

∂_{Iij}=interval between inputs to input nodes i and j

and the other symbols have their previous meanings

On each iteration:

Input vector A to network

$$A = (a_{I1}, a_{I2}, \ldots\ldots\ldots\ldots, a_{I19}, a_{I20})$$

The priming connections in the input layer were then adjusted and the amount of contribution of priming to the activation of each input node calculated.

First, the time since the last input to each input node was incremented or reset to 1.

$$t_{outIi} = \begin{cases} t_{outIi}'+1 & a_{Ii} = 0 \\ 1 & a_{Ii} \neq 0 \end{cases}$$

The connection weights between the input nodes were then adjusted according to the following rules. If there was an input to input node j and the time since the last input to node i was within a prescribed eligibility period E then the priming weight between nodes i and j was increased. If the time since an input was presented to node i was equal to the encoded offset between nodes i and j and no input was presented to node j then the priming weight between nodes i and j was decreased.

$$w_{PIij} = \begin{cases} min(w_{PIij}'+d_P,1) & a_j \neq 0, t_{outIi} < E \\ max(w_{PIij}'-d_P,0) & , a_j = 0, t_{outIi} = \partial_{ij} \\ w_{PIij}' & otherwise \end{cases}$$

where

d_P= the learning rate for the priming rule=0.002

w_{PIij}' = the previous value of w_{Pij}

E=the eligibility period within which prime learning may occur=10 iterations (seconds)

The offset ∂_{ij} between input nodes i and j is modified if there is an input to node j and the time since an input was presented to node i is within the eligibility period. ∂_{ij} is then set to the interval between the two inputs.

$$\partial_{ij} = \begin{cases} t_{outii} & a_{Ij} \neq 0, t_{outii} < E \\ \partial_{ij}' & otherwise \end{cases}$$

where

$\partial_{ij}' =$ the previous value of ∂_{ij}

A quantity P_{Ii} was then calculated which represented the augmentation of the activity of a particular input node due to priming from other input nodes.

$$P_{Ii} = \begin{cases} \sum_j a_{lastij} w_{Pij} & \text{if } t_{outIj} = \partial_{ij} \end{cases}$$

The activity in each trace node was then calculated according to the formula

$$a_{\emptyset j} = \tanh \sum_i \left(w_{ij} a_{Ii} + P_{Ij} \right)$$

Calculate input-trace layer dissimilarity

$$D = \frac{\sqrt{\sum_i (a_{\emptyset i} - a_{Ii})^2}}{\sqrt{n}}$$

Decay weights of horizontal priming connections

$$w_{Pij} = w_{Pij}' - \frac{d_P}{100}$$

Adjust and Decay Delta Weights

$$w_{ij} = \begin{cases} w_{ij}'+d(a_{li}-a_{\varnothing j})a_{li}-p & w_{ij}'>0 \\ w_{ij}'+d(a_{li}-a_{\varnothing j})a_{li}+p & w_{ij}'<0 \\ w_{ij}'+d(a_{li}-a_{\varnothing j})a_{li} & w_{ij}'=0 \end{cases}$$

10.2. Description of simulated experiments

The simulated experiments performed in the study were designed to accurately simulate the temporal and physical stimulus relationships used in human autonomic studies of habituation. In particular, this required the representation of stimuli, physical differences between stimuli and the passage of time. Preliminary results of some of these simulations were reported by Daniels (1993).

The input files for each simulation were constructed by quantizing a real-time experiment into one second intervals or time slices. For each interval a vector was defined representing the input to the network during that interval. An example of part of an input file is shown in Figure 10.2.

```
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 1 1 1 1 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 1 1 1 1 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
```

Figure 10.2. Part of an example input file for neural network simulations.

In the example input fragment the first four lines (representing four seconds of real experimental time) represent four seconds of no stimulation. The fifth line represents a stimulus of intensity 1 presented to nodes 9, 10, 11 and 12. There

are then four seconds of no stimulation followed by another stimulus presentation. The ISI (offset to onset) of the example stimulus series is therefore 4 seconds. During the simulations a line of the input file was used as input to the network , all the calculations which were defined as occurring for each iteration were performed, then the next line of input was used and so on.

Different input files were constructed for each experimental simulation. Although the program used for the simulations was capable of processing inputs of real numbers all inputs used during the simulations were in fact binary, taking values of 0 or 1.

The program used for the simulations was written in Turbo Pascal 6 on an IBM compatible personal computer running the MS-DOS operating system. A listing of the program is included as Appendix B.

The program was written so as to allow the easy calculation of the input-trace dissimilarity measure at any instant. The dissimilarity D was calculated for each simulation according to the formulae provided in the description of each learning rule. For each new run of the simulations the processes described in the initialisation section of each learning rule were performed. Each data point in the results section of the simulations is the average of 21 simulations, with the network re-initialised between each simulation.

10.2.1. Generalisation of habituation

This set of simulations sought to test the ability of each model to exhibit the phenomenon of generalisation of habituation. The simulations were based on an experimental design where a number of presentations of a stimulus are presented to a subject, followed by a single test stimulus. The test stimulus may be more or less similar than the previously presented habituation stimulus. In the simulations the similarity of the test and habituation stimuli was indexed by the overlap of the representations of the two stimuli in the input layer. The representations of similar stimuli had many overlapping nodes while less similar stimuli were represented by sets of nodes with fewer or no overlapping units. It was predicted that the size of the response (indexed by the measure of dissimilarity between the activity in the input and trace units) would be inversely proportional to the number of overlapping units in the representations of the habituation and test stimuli.

METHOD

Four input files were constructed. Each consisted of 16 presentations of a habituation stimulus followed by one presentation of a test stimulus. The habituation stimulus was an input of 1 to input bits 9,10,11 and 12 with the other input bits set to zero. The habituation stimulus was presented at an ISI of five seconds. Four test stimuli were constructed, the representations of which overlapped the habituation stimulus by 3, 2, 1 or 0 units (the stimuli were inputs of 1 to bits 10,11,12,13; 11,12,13,14; 12,13,14,15 and 17,18,19,20 respectively). The test stimulus was presented at the time that the seventeenth habituation stimulus would have been presented. Twenty simulations of 21 replications each were computed, with each of the five learning rules being presented with series using each of the four different test stimuli.

RESULTS

A 5 x 4 Rule x Overlap (0, 1, 2 or 3 units) ANOVA was calculated with

dissimilarity on the test stimulus presentation as the dependent variable. The

means for each rule for each overlap value are presented in Table 10.1.

TABLE 10.1

Mean (SD) dissimilarity on test trial for each overlap value under each learning rule.

Learning Rule	Overlap between habituation and test stimuli (units)			
	0	1	2	3
Delta	.447 (0.00)	.388 (0.00)	.318 (0.00)	.227 (0.00)
Tempo	.592 (.021)	.518 (.015)	.416 (.024)	.304 (.017)
DelTempo	.618 (.025)	.531 (.028)	.438 (.027)	.311 (.034)
Priming	.552 (0.00)	.478 (0.00)	.390 (0.00)	.276 (0.00)
PrimeDelta	.579 (0.00)	.501 (0.00)	.409 (0.00)	.289 (0.00)

It can clearly be seen that with each learning rule the dissimilarity on the test

trial decreased as the number of overlapping units between the habituation and

test stimuli increased. The main effects for learning rule ($F(4,400)=890.6$,

$p<.0001$) and overlap ($F(3,400)=6164.7$, $p<.0001$) were both highly significant, as

was the Rule x Overlap interaction ($F(12,400)=17.99$, $p<.0001$). The significant

interaction indicated that the rate of decrease in dissimilarity with each increase

in overlap was not uniform across the learning rules. This result was not

surprising in that the learning rules were not constructed with the intention of

equal parametric performance in all situations, rather they were devised to

exhibit general experimental phenomena.

In summary, the results of the simulations of the effects of generalisation

indicated that each of the learning rules tested were capable of exhibiting

generalisation of habituation to test stimuli, if the representations of the test stimuli overlapped with the representation of the habituated stimulus. In addition, the simulation results indicated that the greater the degree of overlap between the two representations the greater the generalisation of habituation. If overlap of representation is accepted to be an analog of similarity then these results were in concordance with Ben-Shakhar and Lieblich (1982).

10.2.2. *Effect of ISI variability and duration on habituation*

This set of simulations sought to test the effects of duration and ISI variability on the course of habituation under each model as well as each model's ability to emit a response to complete stimulus omission.

METHOD

The performance of the five models in the cases of the effect of ISI variability on the course of habituation, the effect of ISI duration on the course of habituation and the response to complete stimulus omission were tested by simulation using a set of four input files. The four files were constructed to simulate four experimental conditions and are listed below.

a) Input file 5REG: Sixteen presentations of a four bit stimulus (bits 9,10,11,12 set to 1, all other bits set to 0) were presented at a constant ISI of 5 seconds (five all zero stimuli presented between each non-zero stimulus), the seventeenth stimulus was omitted (replace non-zero stimulus with all zero bits), and the eighteenth stimulus re-presented at the time it would have been presented if the seventeenth stimulus had not been omitted.

b) Input file 15REG: The same sequence of stimulation as 5REG with a 15 second ISI rather than 5 seconds.

c) Input file 5IRREG: The same sequence of stimuli as 5REG but with variable ISIs. ISIs between the first 16 presentations were either 3, 5 or 7 seconds. Three

different series were constructed with the following 15 ISIs between the first 16 presentations. The order of ISIs for each series were:

a. 7,7,5,3,5,5,3,5,7,3,7,3,5,3,7

b. 3,3,7,5,7,7,5,7,3,5,3,5,7,5,3

c. 5,5,3,7,3,3,7,3,5,7,5,7,3,7,5

Thus, if 3n simulations were run, with each ISI series being used n times, any single stimulus presentation would have been preceded an equal number of times by a 3, 5 or 7s ISI. Each of the 5IRREG series had the same total duration (measured at the sixteenth presentation) as the 5REG series.

d) Input file 15IRREG: As for 5IRREG but with the 3, 5 and 7s ISIs substituted by 12, 15 and 18s ISIs respectively.

Twenty-one replications of the constant ISI and variable ISI (7 each of orders a, b and c) series were computed for each model.

The effect of ISI duration and variability on the course of habituation across the first 16 trials was tested by the calculation of three dependent variables for each input file. Total response magnitude and number of non-zero responses during the habituation period were scored as described in the analyses of Experiments 3 and 4. The uncorrected habituation rate was calculated as the slope of the regression line predicting the skin conductance response from log trial number (Lader & Wing, 1966). These variables were calculated over the interval from the presentation of the first stimulus up to, and including the sixteenth stimulus presentation. The interval containing the control and omitted stimuli was excluded from the calculation of these variables as the intention was to examine the performance of the models under different habituation conditions, not their response to stimulus change.

It was expected, from the results of the experiments performed in earlier chapters, that there would be more rapid habituation (ie. a faster rate of response decrement) in the 5s constant series compared to the 5s variable series (indicating an effect for ISI variability) and the 15s constant series (indicating an effect for ISI duration). Furthermore, there should also have been an effect for ISI duration when the two variable ISI duration conditions were compared, with the shorter 5s ISI exhibiting a more rapid rate of habituation.

RESULTS AND ANALYSIS

The average dissimilarities on each of the 16 presentations for each experimental situation for each learning rule are presented in the following pages. The distance (dissimilarity) on each presentation or during the omission and control periods is assumed to be an index of the orienting response.
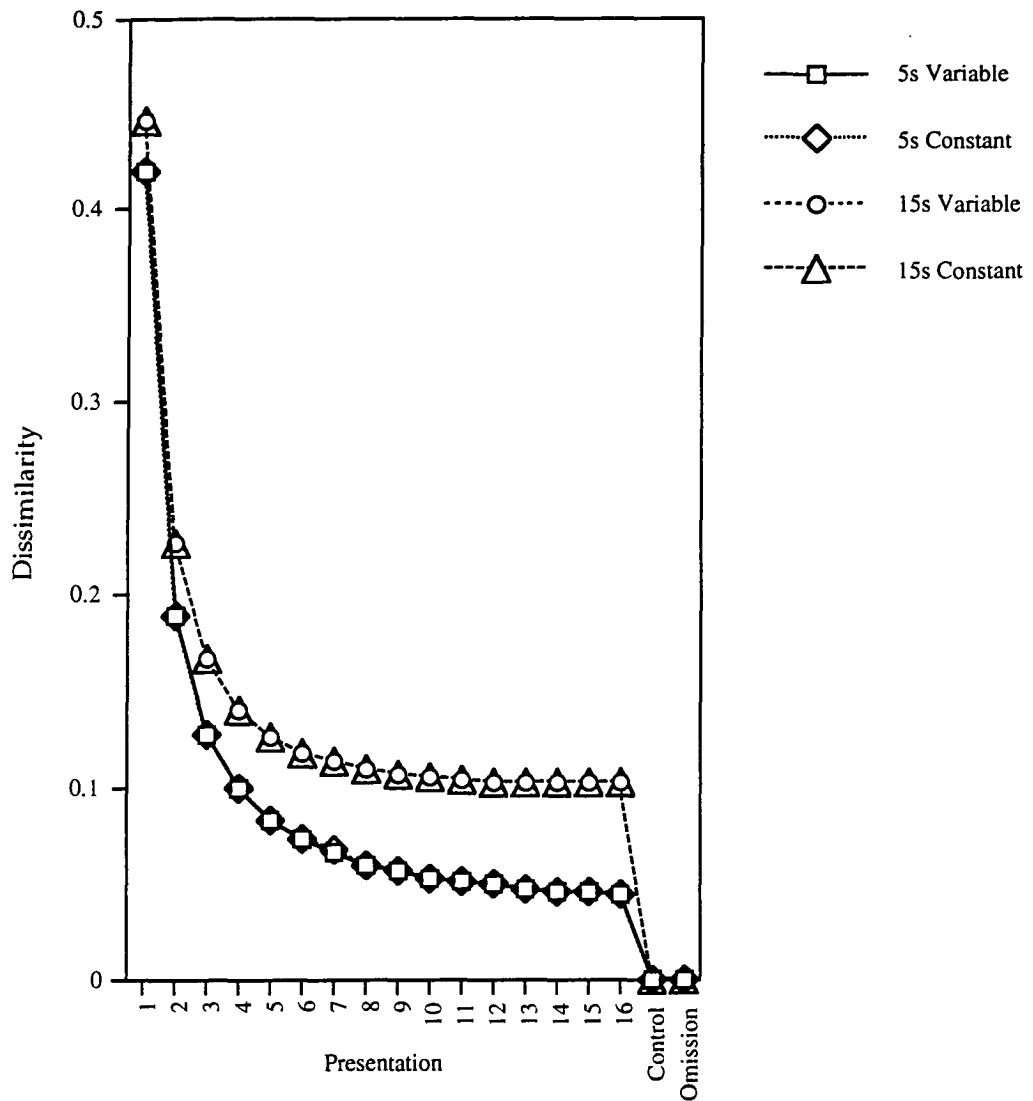
Figure 10.3. Delta model results for mean dissimilarity on each presentation

and stimulus omission at each level of ISI variability and average ISI duration.
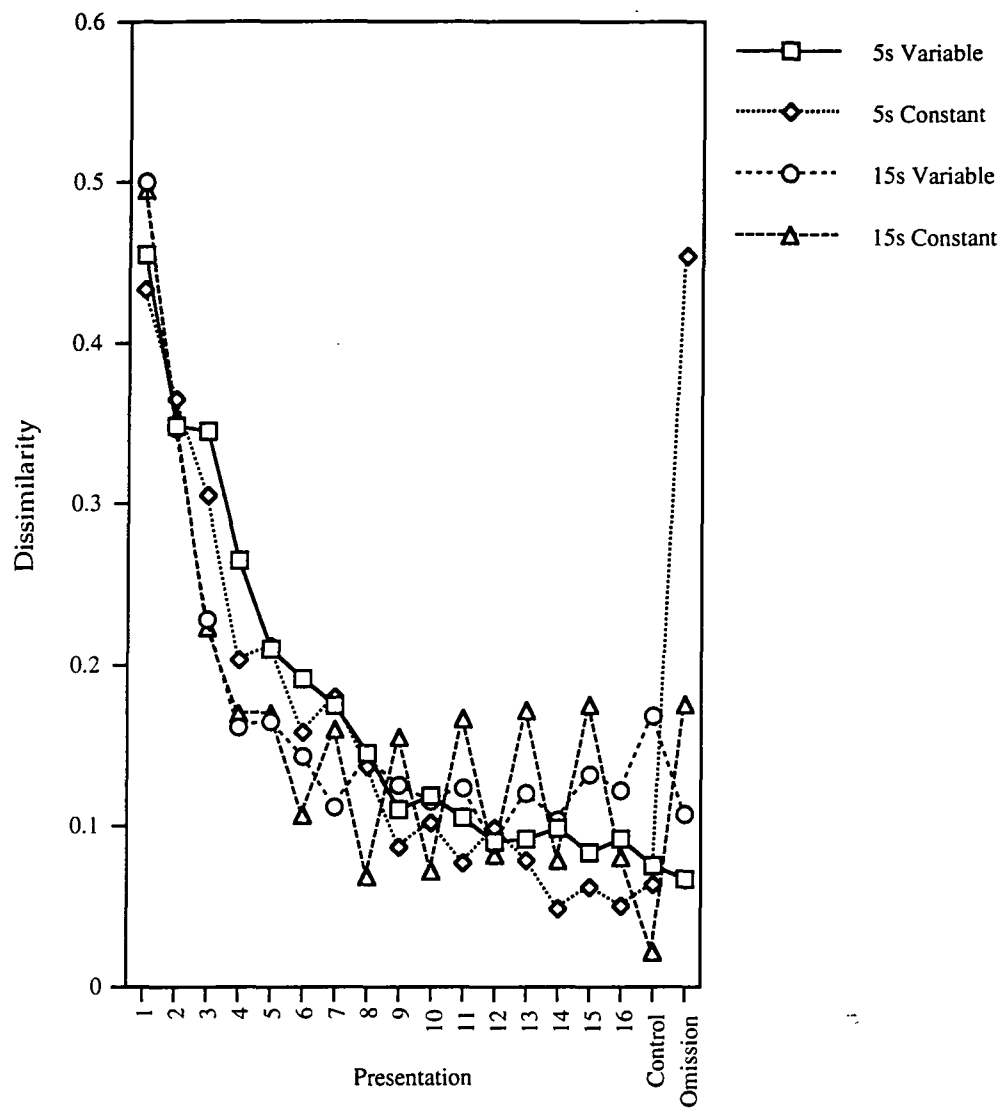
Figure 10.5. DelTempo model results for mean dissimilarity on each presentation and stimulus omission at each level of ISI variability and average ISI duration.
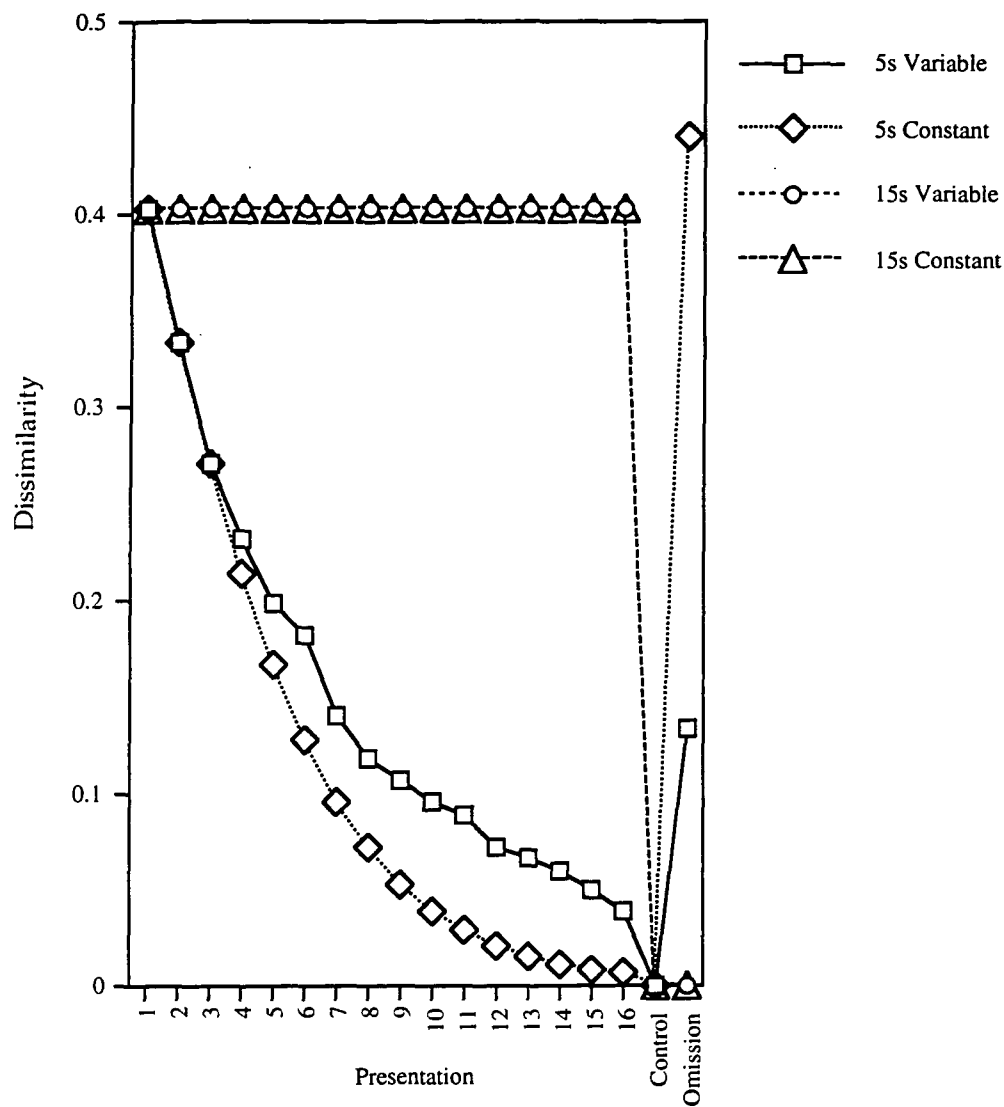
Figure 10.6. Prime model results for mean dissimilarity on each presentation and stimulus omission at each level of ISI variability and average ISI duration.
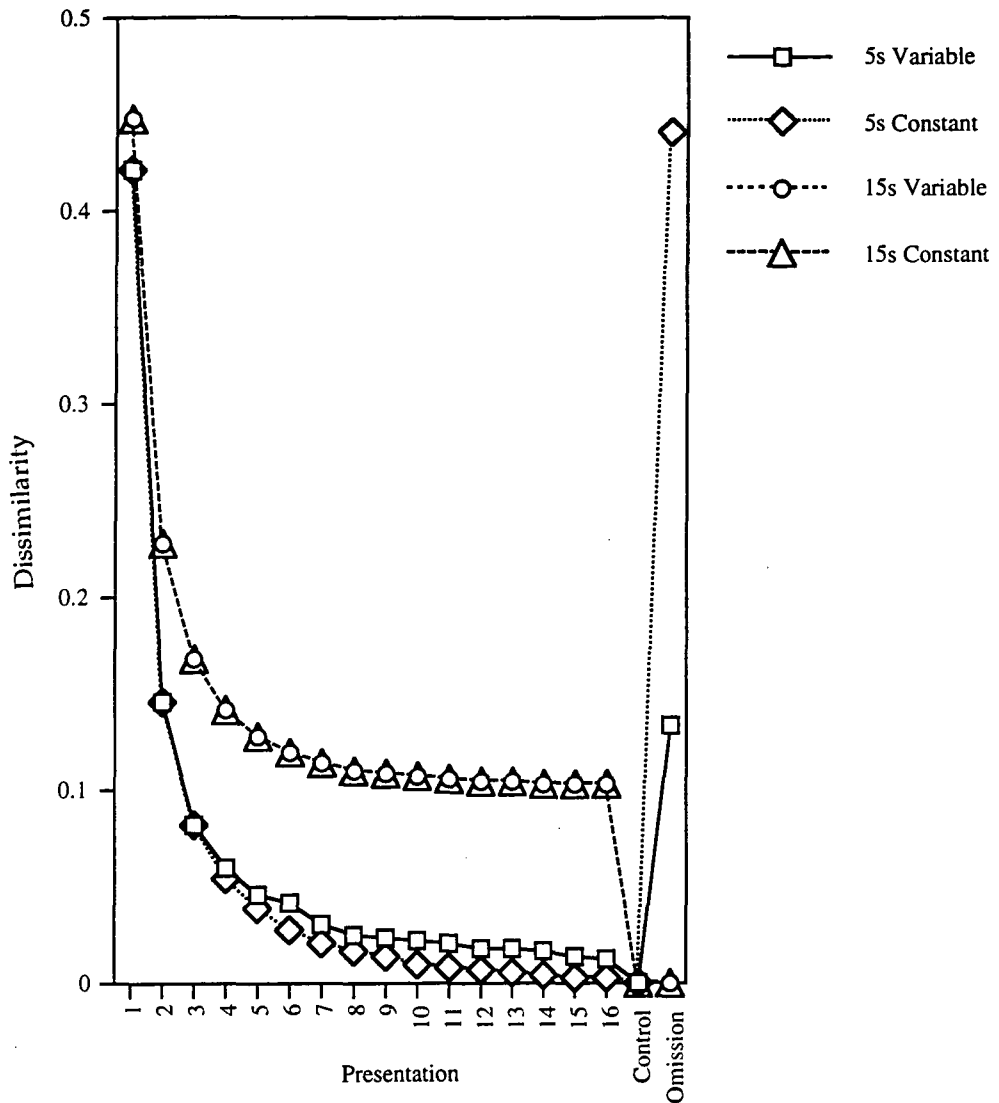
Figure 10.7. PrimeDelta model results for mean dissimilarity on each

presentation and stimulus omission at each level of ISI variability and average

ISI duration.

Three indices of habituation were calculated for each set of simulations (where a set of simulations means 21 simulations using one input file and one model). Uncorrected rate of habituation was calculated as the slope of the regression predicting the observed dissimilarity measure from log(presentation number). Both total response magnitude and the number of non-zero responses were calculated over the interval from the beginning of the experiment up until and including the sixteenth stimulus presentation. The total response was the sum of all non-zero responses occurring in this interval.

The results for each dependent variable were analysed by a three way 2 x 2 x 5 ISI duration (5 second average/15 second average) x ISI variability (Constant/Variable) x Rule (Delta/Tempo/DelTempo/Prime/PrimeDelta) ANOVA. Following the ANOVA, planned comparisons were performed testing the simple effect of ISI variability at each level of ISI duration and the simple effect of ISI duration at each level of ISI variability, within each learning rule. The group means for each dependent variable and the results of each analysis are presented and discussed below.

It will be noted that some cells in the following tables of means have zero (to 3 decimal places) variance. The question of the the appropriateness of ANOVA in this situation was considered with the following points being relevant. The first is that while the variance with some combinations of learning rule and input file is zero, the underlying random factors included in the models mean that the variance is not constrained by the models to be zero. ANOVA is generally robust to moderate violations of the homogeneity of variance assumption providing that cell sizes are equal and greater than five. This being the case in the current studies, ANOVA was considered to be an appropriate choice of analysis.

Uncorrected rate of habituation

TABLE 10.2

Mean (SD) of uncorrected rate of habituation for each learning rule for each level of ISI duration and variability (n=21 in each case).

Learning Rule	Constant ISI		Variable ISI	
	5s ISI	15s ISI	5s ISI	15s ISI
Delta	-.244 (.000)	-.219 (.000)	-.244 (.004)	-.219 (.006)
Tempo	-.399 (.042)	-.219 (.039)	-.058 (.030)	-.081 (.036)
DelTempo	-.335 (.029)	-.280 (.028)	-.327 (.039)	-.283 (.029)
Priming	-.365 (.000)	.000 (.000)	-.315 (.003)	-.000 (.000)
PrimeDelta	-.263 (.000)	-.219 (.000)	-.251 (.002)	-.219 (.006)

TABLE 10.3

F and p values for each effect in ISI x Variability x Rule ANOVA for uncorrected rate of habituation.

Effect	df	F	p
ISI	1, 400	2000.6	.0001
Variability	1, 400	548.9	.0001
Rule	4, 400	395.6	.0001
ISI x Variability	1, 400	130.1	.0001
ISI x Rule	4, 400	628.7	.0001
Variability x Rule	4, 400	400.7	.0001
ISI x Variability x Rule	4, 400	60.2	.0001

TABLE 10.4

Results of planned comparisons for uncorrected rate of habituation.

Learning Rule	Contrast (df = 1,400 in each case)			
	Effect of ISI duration at constant ISI	Effect of ISI duration at variable ISI	Effect of ISI variability at 5s duration	Effect of ISI variability at 15s duration
Delta	F=11.29	F=11.27	F=.00	F=.00
	p=.0009	p=.0009	p=1.0	p=.9938
Tempo	F=537.80	F=9.74	F=2094.90	F=378.70
	p=.0001	p=.0019	p=.0001	p=.0001
DelTempo	F=54.84	F=35.52	F=1.02	F=.19
	p=.0001	p=.0001	p=.3141	p=.6619
Priming	F=2392.9	F=1780.2	F=45.24	F=.00
	p=.0001	p=.0001	p=.0001	p=1.0
PrimeDelta	F=34.63	F=18.46	F=2.50	F=.00
	p=.0001	p=.0001	p=.1147	p=.9938

It should be noted that in this and the other tables in this chapter that rounding of the F values means that a tabled F of 0.00 does not necessarily correspond to a p value of 1. Any disparity is however minor (<.01) and does not affect the interpretation of the analyses of variance.

The results for the effect of ISI duration and variability of the uncorrected rate of habituation were generally as would be expected given the way the models were defined.

For the Delta rule, ISI variability had no significant effect at either ISI duration, a result which was expected given that there was no mechanism of temporal encoding present in this rule. Average ISI duration had a significant effect at

both constant and variable ISIs, reflecting the greater amount of weight decay between each stimulus presentation at the longer 15s average ISI.

The results for the Tempo rule indicated a significant effect for ISI variability at both 5s and 15s average ISIs with the constant ISI series showing a faster rate of response decrement in each case. It was not expected that there would be any difference between the constant and variable ISI series at the 15s average ISI. A possible explanation for this result is that, if a node was not stimulated for the duration of the experiment, then under the Tempo rule it would fire at a maximum ISI determined by the minimum rate of rise of the membrane potential. In the current series this maximum ISI was 10 seconds. Therefore, given an input series with a constant ISI of 15 seconds, the spontaneous firing of even an untrained node coincided with the input on every second presentation. If this was the case then the 15s constant series will appear to habituate to some extent even though the nodes are not actually encoding a 15 second ISI but rather are simply firing at a maximum, untrained ISI of 10 seconds. For both the constant and variable ISI series the 5s ISI duration series habituated more rapidly than the 15s series, a result expected because the Tempo rule should learn, or in the case of the variable ISI series partially learn, the ISI and accurately predict the presentation of the stimuli in the 5s series, but not the 15s series.

The results for the DelTempo rule showed a significant effect for ISI variability at the 5s average ISI duration only with the 5s constant series showing a greater rate of response decrement than the 5s variable series. For both constant and variable ISI series the rate of response decrement was not significantly different in the 5s average ISI series compared to the 15s average ISI series.

The Prime rule exhibited a significant effect for ISI variability at the 5s average ISI duration only with the 5s constant series showing a greater rate of response decrement than the 5s variable series. For both constant and variable ISI series

the rate of response decrement was faster for the 5s average ISI series compared to the 15s average ISI series.

The PrimeDelta rule failed to show a significant effect for ISI variability at the 5s average ISI duration when the uncorrected rates of habituation were compared. This result seems inconsistent with the results in Figure 10.8 and suggests that the uncorrected rate of habituation may not be sensitive to the small but consistent differences observed between the two series. There was an effect for ISI duration (with more rapid habituation at the 5s average ISI) when both the constant and variable ISI series were compared.

Total response magnitude

TABLE 10.5

Mean (SD) of total response magnitude for each learning rule for each level of ISI duration and variability (n=21 in each case).

	Constant ISI		Variable ISI	
Learning Rule	5s ISI	15s ISI	5s ISI	15s ISI
Delta	1.523 (.000)	2.288 (.000)	1.572 (.006)	2.424 (.005)
Tempo	16.708 (1.238)	31.175 (1.662)	25.199 (.778)	37.793 (2.266)
DelTempo	15.239 (.791)	28.735 (.990)	20.218 (.651)	33.547 (1.536)
Priming	1.864 (.000)	6.442 (.000)	5.137 (.019)	6.845 (.000)
PrimeDelta	.858 (.000)	2.288 (.005)	3.487 (.146)	2.424 (.005)

TABLE 10.6

F and p values for each effect in ISI x Variability x Rule ANOVA for total SCR.

Effect	df	F	p
ISI	1, 400	5435.9	.0001
Variability	1, 400	1537.6	.0001
Rule	4, 400	18928.8	.0001
ISI x Variability	1, 400	100.6	.0001
ISI x Rule	4, 400	1259.9	.0001
Variability x Rule	4, 400	280.4	.0001
ISI x Variability x Rule	4, 400	9.99	.0001

TABLE 10.7

Results of simple effects of ISI variability and duration on total response magnitude.

Learning Rule	Contrast (df = 1,400 in each case)			
	Effect of ISI duration at constant ISI	Effect of ISI duration at variable ISI	Effect of ISI variability at 5s duration	Effect of ISI variability at 15s duration
Delta	F=8.51	F=10.55	F=.03	F=.27
	p=.0037	p=.0013	p=.8529	p=.6153
Tempo	F=3041.90	F=2305.10	F=1047.90	F=636.50
	p=.0001	p=.0001	p=.0001	p=.0001
DelTempo	F=2647.60	F=2209.30	F=375.40	F=336.50
	p=.0001	p=.0001	p=.0001	p=.0001
Priming	F=304.60	F=42.40	F=155.70	F=2.36
	p=.0001	p=.0001	p=.0001	p=.1252
PrimeDelta	F=29.72	F=16.44	p=100.50	F=.27
	p=.0001	p=.0001	p=.0001	p=.6053

The results for total response magnitude for the Delta rule indicated a significant effect for ISI duration (greater total response in 15s ISI condition) for both variable and constant ISIs. There were no significant effects for ISI variability at either the 5s or 15s average ISI durations.

The results for the Tempo and DelTempo rules also indicated a significant effect for ISI duration (greater total response in 15s ISI condition) for both constant and variable ISIs as well as effects for ISI variability (greater total response in the variable ISI condition) at both 5s and 15s average ISIs.

The Prime rule results showed a significant effect of ISI variability at the 5s average duration but not at the 15s duration. There was a significant effect for ISI duration when stimuli were presented both constant ISI and variable ISIs.

The PrimeDelta rule showed an effect for ISI variability at the 5s average ISI duration but not at the 15s average ISI duration. There was a significant effect of ISI duration for both the variable and constant ISI series.

Number of non-zero responses

TABLE 10.8

Mean (SD) of Number of non-zero responses for each learning rule for each level of ISI duration and variability (n=21 in each case).

	Constant ISI		Variable ISI	
Learning Rule	5s ISI	15s ISI	5s ISI	15s ISI
Delta	16.00 (.00)	16.00 (.00)	16.00 (.00)	16.00 (.00)
Tempo	84.91 (4.46)	225.95 (15.39)	99.38 (5.02)	263.57 (11.99)
DelTempo	82.67 (4.58)	222.52 (9.39)	100.28 (3.05)	258.05 (16.41)
Priming	16.00 (.00)	16.00 (.00)	23.00 (.00)	16.00 (.00)
PrimeDelta	16.00 (.00)	16.00 (.00)	23.00 (.00)	16.00 (.00)

TABLE 10.9

F and p values for each effect in ISI x Variability x Rule ANOVA for number of responses during habituation series.

Effect	df	F	p
ISI	1, 400	8797.50	.0001
Variability	1, 400	413.60	.0001
Rule	4, 400	13750.70	.0001
ISI x Variability	1, 400	16.03	.0001
ISI x Rule	4, 400	3568.60	.0001
Variability x Rule	4, 400	85.53	.0001
ISI x Variability x Rule	4, 400	23.90	.0001

TABLE 10.10

Results of simple effects for number of responses.

Learning Rule	Contrast (df = 1, 400 in each case)			
	Effect of ISI duration at constant ISI	Effect of ISI duration at variable ISI	Effect of ISI variability at 5s duration	Effect of ISI variability at 15s duration
Delta	F=.00	F=.00	F=.00	F=.00
	p=1.0	p=1.0	p=1.0	p=1.0
Tempo	F=5081.9	F=6802.7	F=61.18	F=361.5
	p=.0001	p=.0001	p=.0001	p=.0001
DelTempo	F=4996.5	F=6277.3	F=88.55	F=322.4
	p=.0001	p=.0001	p=.0001	p=.0001
Priming	F=0.00	F=55.79	F=54.43	F=.00
	p=1.0	p=.0001	p=.0001	p=1.0
PrimeDelta	F=0.00	F=55.79	F=54.43	F=.00
	p=1.0	p=.0001	p=.0001	p=1.0

The number of non-zero responses results for the Delta rule showed no differences between the four conditions.

The Tempo rule results indicated a significant effect for ISI duration (with a greater number of responses at the 15s ISI duration) in both constant and variable ISI conditions, and significant effects for ISI variability at both durations. The DelTempo results showed the same pattern of significant effects.

The Prime and PrimeDelta rules showed identical results after analysis of the number of responses. In both cases there were significant effects for ISI variability at the 5s average ISI duration and a significant effect for ISI duration (with a greater number of responses at the 5s ISI) for the variable ISI conditions.

In conclusion, it must be assessed, for each rule, whether there is an effect of ISI variability on the course of habituation, and whether this effect is different at the 5s and 15s ISIs. It must also be determined if there is an effect of ISI duration on the course of habituation and whether this effect is different for the constant and variable ISI series. To answer these questions the four dependent variables, as well as the descriptive results must all be combined into a consistent form.

To firstly consider the question of the effect of ISI duration. The dependent variables of total response and number of responses were not useful in assessing this question. This is because both of these variables may simply reflect the necessarily longer total duration of the 15s average ISI duration input files. This decision was therefore largely made on the results of the analysis of the uncorrected rate of habituation and inspection of the course of response habituation across the 16 trials.

For the Delta, rule it was clear from Figure 10.3 and from the results of the analysis of the uncorrected rate of habituation, that the series presented at the shorter 5s ISI showed a more rapid decrement of response, and finally reached a lower level of response, when compared to the 15s ISI series. It was therefore concluded that the ISI duration did have an effect on the course of response habituation under the Delta rule with stimuli presented at a shorter ISI habituating more rapidly than those presented at a longer ISI. This effect was independent of ISI variability.

The results for the Tempo rule indicated that when the stimuli were presented at a constant ISI the habituation proceeded more rapidly at the 5s ISI compared to the 15s ISI. This effect was reversed when the stimuli were presented at a variable ISI.

The DelTempo rule results indicated a greater rate of response decrement at the shorter average ISI duration for both variable and constant ISI series.

The results of the Priming and PrimeDelta rule simulations showed the same pattern of results with faster habituation at the shorter average ISI for both constant and variable ISI series.

It was concluded that the Delta, DelTempo, Prime and PrimeDelta rules all performed as expected with regard to the effect of average ISI duration on the rate of habituation. In each case the shorter 5s average ISI duration conditions showed more rapid habituation than the 15s average ISI duration for both the constant and variable ISI conditions.

Turning to the effect of ISI variability, it was now possible to include in the assessment of the performance of each rule the total response and number of non-zero responses data. The reason for this is that both constant and variable ISI series at each ISI duration were controlled for total duration and should therefore show no differences in total response magnitude or number of responses, if responses were simply randomly occurring over time. This was not the case in the comparisons across different durations.

The results for the Delta rule showed no effect of ISI variability at either the 5s or 15s average ISI duration. The results for total response, number of responses, uncorrected rate of habituation all concur in this conclusion.

The Tempo rule showed an effect for ISI variability at both 5s and 15s ISIs, with variable ISIs showing a retarded rate of habituation compared to the constant

ISI conditions. These results were consistently confirmed by the analyses of uncorrected rate of habituation, total response and number of responses.

The results for the DelTempo rule showed an inconsistent effect across the three dependent variables. Both total response magnitude and number of response measures showed a significant effect for ISI variability at both 5s and 15s average ISI durations, while the uncorrected rate of habituation result failed to show a significant difference at either ISI duration. Examination of Figure 10.5 does not clearly resolve the disparity between the findings and the performance of the DelTempo ruled in this case will have to be left, to some extent, inconclusive.

The results for the Prime rule regarding ISI variability showed a clear effect with the constant ISI series showing significantly more rapid habituation than the variable ISI series at the 5s average ISI but not at the 15s average ISI. The results were consistent across the uncorrected rate of habituation, total response magnitude and number of responses dependent indices.

The results for the PrimeDelta rule were almost as conclusive with a similar pattern of results to the Prime rule with the exception that, as was discussed earlier, the comparison of the uncorrected rate of habituation results failed to show a significant effect for ISI variability at the 5s ISI. The other indices as well as the descriptive results in Figure 10.7, however, clearly indicated that there was an effect of ISI variability at the 5s average ISI but not at the 15s average ISI duration.

When the results of these simulations were compared to the empirical results presented in the earlier chapters it was concluded that only the Prime and PrimeDelta models were able to accurately simulate the effect of ISI variability on habituation observed in humans. These were the only two models which were able to simulate an effect for ISI variability at the short average ISI but not at the longer average ISI.

10.2.3. *Response to complete stimulus omission*

These simulations were implemented to examine the performance of each model when a stimulus was omitted following a series of presentations. Complete stimulus omission refers to the case where the pre-omission series consists of presentations of a single stimulus. This is opposed to omission of S2 in an S1-S2 pair where the pre-omission series consists of a series of S1-S2 pairs.

METHOD

The input files for the evaluation of the response to complete stimulus omission were the same four files that were used to examine the effects of ISI variability and duration. ie. 5REG, 5IRREG, 15REG and 15IRREG. The omission trial consisted of the presentation of no stimulus at the time the seventeenth presentation would have been expected to be presented. In the case of the two variable ISI series, the ISI from the sixteenth to the seventeenth stimulus was the same as tin he constant ISI series with the same average duration.

RESULTS

The response to stimulus omission was measured by comparison of the dissimilarity at the time the seventeenth stimulus would have been presented, with the dissimilarity at a control time three seconds before the time of stimulus omission (at which no stimulus was presented, or would be expected to be presented if the ISI of previous stimulation had been encoded).

The means for difference in dissimilarity from the control to omission scoring instants for each ISI, variability and rule are presented in Table 10.11. Positive values indicates that the dissimilarity was larger at the time of stimulus omission than at the control time, while a negative value indicates the opposite.

TABLE 10.11

Mean (SD) (dissimilarity on omission trial - dissimilarity at control instant) for each level of ISI variability, average ISI duration and learning rule (n=21 for each cell).

	Constant ISI		Variable ISI	
Learning Rule	5 s ISI	15 s ISI	5 s ISI	15 s ISI
Delta	.000 (.000)	.000 (.000)	.000 (.000)	.000 (.000)
Tempo	.392 (.026)***	.136 (.124)***	-.003 (.029)	-.042 (.262)
DelTempo	.389 (.033)***	.152 (.127)***	-.009 (.038)	-.062 (.251)
Priming	.440 (.000)#	.000 (.000)	.133 (.193)**	.000 (.000)
PrimeDelta	.440 (.000)#	.000 (.000)	.133 (.193)**	.000 (.000)

*$p<.05$, ** $p<.01$, ***$p<.001$ when t-test (df=19) used to compare mean to hypothesised value of zero.

Because SD = 0 in these conditions t-tests could not be performed. The means are however clearly significantly different to zero.

A 2 x 2 x 5 ISI (5 s/15s) x Variability (Constant ISI/Variable ISI) x Rule ANOVA was computed with the difference in dissimilarity between the control and omission scoring instants as the dependent variable. Each of the main effects and 2- and 3-way interactions were highly significant and are listed in Table 10.12.

TABLE 10.12

F and p values for each effect in ISI x Variability x Rule ANOVA for dissimilarity on omission trial.

Effect	df	F	p
ISI	1, 400	260.1	.0001
Variability	1, 400	280.7	.0001
Rule	4, 400	24.92	.0001
ISI x Variability	1, 400	89.41	.0001
ISI x Rule	4, 400	24.80	.0001
Variability x Rule	4, 400	26.48	.0001
ISI x Variability x Rule	4,400	6.86	.0001

Because of the significant three-way interaction a set of contrasts was performed to elucidate the effect of ISI variability at both 5s and 15s average ISIs, and the effect of average ISI with both variable and constant ISIs. The contrasts were performed for each learning rule and are summarised in Table 10.13.

TABLE 10.13

Results for planned comparisons on dissimilarity on omission trial for effects of ISI variability and duration for each learning rule.

	Contrast (df = 1,400 in each case)			
Learning Rule	Effect of ISI duration at constant ISI	Effect of ISI duration at variable ISI	Effect of ISI variability at 5s duration	Effect of ISI variability at 15s duration
Delta	F=.00	F=.00	F=.00	F=.00
	p=1.0	p=1.0	p=1.0	p=1.0
Tempo	F=56.64	F=1.33	F=135.10	F=27.59
	p=.0001	p=.2491	p=.0001	p=.0001
DelTempo	F=49.04	F=2.39	F=137.90	F=39.49
	p=.0001	p=.1228	p=.0001	p=.0001
Priming	F=167.90	F=15.39	F=81.67	F=.00
	p=.0001	p=.0001	p=.0001	p=1.0
PrimeDelta	F=167.90	F=15.39	p=81.67	F=.00
	p=.0001	p=.0001	p=.0001	p=1.0

It was concluded from these results are that the simulations using the Delta rule alone did not emit a response when the stimulus was omitted. This result followed from the output of this model being a function of current input only, with no method of encoding the temporal properties of previous stimulation.

The Tempo and DelTempo learning rules both showed a similar pattern of results. For each rule there were significant effects of ISI variability (with the omission response following the constant ISI series being significantly greater

than that elicited when the omission followed a variable ISI series) at both 5 and 15 s average ISI durations. For both rules there was a significant effect of ISI duration (with the omission response being larger when the preceding stimuli were presented at an average ISI of 5 seconds than when they were presented at an average ISI of 15 seconds) for the constant ISI series but not following the variable ISI series.

For both the Priming and PrimeDelta rules, significant omission responses were omitted following both the 5s constant and 5s variable ISI series. The omission response was however significantly larger when the omission followed the constant ISI series than when the omission followed the variable ISI series. Neither of the series presented at the 15s average ISI showed a non-zero response to complete stimulus omission.

10.2.4. Omission of S2 in an S1-S2 pair and conditioned diminution of the UCR

These simulations investigated the performance of each model under conditions where the presentation of the S2 was preceded by an S1. In particular, these simulations allowed the testing of whether the reliable cuing of the S2 by the S1 resulted in more rapid habituation compared to the case where the same S2 was presented in a series of single stimuli. Secondly, the simulations allowed the testing of whether the effect of a variable S2 period differed when the S2 was reliably cued by an S1 compared to when the S2 was presented alone. Finally the simulations allowed the testing of each model's ability to emit a response when the S2 was omitted following a series of S1-S2 presentations.

METHOD

The performance of the models under these conditions was tested by the use of four input series. 5REG, 15REG, 5IRREG and 15IRREG described earlier were used and served as the uncued series where the presentation of the stimulus was not cued by the presentation of another non-overlapping stimulus.

Four new series were constructed which were modifications of the uncued series. The new files incorporated cuing of the stimulus (S2) which was presented in 5REG and 15REG by a non-overlapping, different stimulus (S1) which reliably preceded the presentation of the S2. The S1 consisted of setting the activity of input bits 1, 2, 3 and 4 to 1 for a single iteration. In the new files the S1 preceded the normal S2 presentation by two iterations with a non-stimulated iteration between the S1 and S2 for all 16 pre-omission presentations. In the terminology of the experiments conducted with paired stimuli (Experiments 2 and 4) and SOA between the S1 and S2 in each file was 2 seconds. The S2 period took the same value as the ISI in the uncued series used earlier, that is of the four files one had a constant S2 period of 5s, the second had a constant S2 period of 15s, the third had a variable S2 period with an average duration of 5s while the fourth had a variable S2 period with and average of 15s.

Where a constant S2 period was used 21 simulations were performed for each model with each input file. When there was a variable S2 period three variations of each input file were created using the procedure outlined in section 10.2.2. Each variation was used as the input file 7 times for each model when these input files were used.

RESULTS

Response to S2 omission

The response to S2 omission was assessed similarly to the test of response to complete omission. On the seventeenth presentation of the cued series (5CUEDS2 and 15CUEDS2) the S1 was presented as in the previous 16 presentations, but no S2 was presented. The dissimilarity at the control time (two iterations before the seventeenth S1 presentation, a time at which no response was presented or would be expected to be elicited) was subtracted from the dissimilarity at the time the S2 was omitted to give a measure of the omission response compared to background activity.

The mean response to the omitted S2 at both 5s and 15s ISIs for each model are presented in Table 10.14. The probabilities reported in the body of the table refer to one group t-tests testing the null hypothesis that the mean response is equal to zero in each case. F tests were used to test the significance of the difference in the magnitude of the response to S2 omission between the 5s and 15s ISIs.

TABLE 10.14

Mean (SD) (dissimilarity on S2 omission trial - dissimilarity at control instant) for each level of average ISI duration, ISI variability and learning rule.

	Constant ISI		Variable ISI	
Learning Rule	5 s ISI	15 s ISI	5 s ISI	15 s ISI
Delta	.000 (.000)	.000 (.000)	.000 (.000)	.000 (.000)
Tempo	.411 (.023)***	.173 (.120)***	-.167 (.259)**	.096 (.206)
DelTempo	.405 (.022)***	.148 (.126)***	-.147 (.278)*	.032 (.148)
Priming	.447 (.000)#	.442 (.000)#	.295 (.216)***	.442 (.000)#
PrimeDelta	.447 (.000)#	.442 (.000)#	.295 (.216)***	.442 (.000)#

*p<.05, ** p<.01, ***p<.001 when t-test (df=19) used to compare mean to hypothesised value of zero.
Because SD = 0 in these conditions t-tests could not be performed. The means are however clearly significantly different to zero.

A 2 x 2 x 5 ISI (5 s/15s) x Variability (Constant ISI/Variable ISI) x Rule ANOVA was computed with the difference in dissimilarity between the control and omission scoring instants as the dependent variable. Each of the main effects and 2- and 3-way interactions were highly significant and are listed in Table 10.15.

TABLE 10.15

F and p values for each effect in ISI x Variability x Rule ANOVA for dissimilarity on omission trial - dissimilarity at control instant.

Effect	df	F	p
ISI	1, 400	64.29	.0001
Variability	1, 400	502.0	.0001
Rule	4, 400	153.69	.0001
ISI x Variability	1, 400	305.0	.0001
ISI x Rule	4, 400	24.49	.0001
Variability x Rule	4, 400	39.16	.0001
ISI x Variability x Rule	4, 400	19.07	.0001

Because of the significant three-way interaction, a set of contrasts were performed to elucidate the effect of ISI variability at both 5s and 15s average ISIs, and the effect of average ISI at both variable and constant ISIs. The contrasts were performed for each learning rule and are summarised in Table 10.16.

TABLE 10.16

Results of contrasts testing the effect of ISI variability and duration on the response to omission of S2 following presentation of S1-S2 pairs.

Learning Rule	Contrast (df = 1,400 in each case)			
	Effect of ISI duration at constant ISI	Effect of ISI duration at variable ISI	Effect of ISI variability at 5s duration	Effect of ISI variability at 15s duration
Delta	F=.00	F=.00	F=.00	F=.00
	p=1.0	p=1.0	p=1.0	p=1.0
Tempo	F=49.43	F=44.30	F=324.60	F=18.76
	p=.0001	p=.0001	p=.0001	p=.0001
DelTempo	F=57.59	F=40.70	F=303.90	F=12.00
	p=.0001	p=.0001	p=.0001	p=.0005
Priming	F=.03	F=185.80	F=190.10	F=.00
	p=.8734	p=.0001	p=.0001	p=1.0
PrimeDelta	F=.03	F=185.80	p=190.10	F=.00
	p=.8734	p=.0001	p=.0001	p=1.0

The results clearly show that the response to S2 omission occurred only in the rules using either the Tempo or Prime methods of temporal encoding. In the case of the two rules using the Tempo method a significant positive response to S2 omission only occurred following the constant ISI series, with the response being significantly larger at the shorter ISI duration. With the variable ISI series, at the 5s average ISI there was a significantly larger response at the control instant compared to the instant of S2 omission, while there was no

significant difference between the responses at each instant at the 15s average ISI.

It will be recalled that there was no significant evidence of a response at the time of S2 omission in Experiment 2 of this thesis. There are however reports in the literature which suggested that such an effect could be observed in some situations and that this was more likely if the interval between the S1 and S2 was 0 seconds rather than the 4 seconds of Experiment 2. The results of the current simulations are consistent with this conclusion with the S1-S2 interval being 1 second, a duration at which a response to S2 omission might be reasonably expected, although this was not tested directly in the experimental portion of this thesis.

For the two models utilising the Prime method there was a significant response to S2 omission following all four conditions. The response was however significantly smaller following the 5s variable condition compared to the 5s constant and 15s variable conditions. In the 5s variable condition the S2 would have been primed by both the S1 presentation and the preceding S2 presentation but the priming by previous S2 presentation would have been disrupted and the response to S2 omission was correspondingly reduced. At the 15s average ISI duration, there would not be any cuing of the S2 by previous S2 presentations (because the S2 to S2 interval exceeded the maximum priming interval) and the S2 was therefore be equally well primed (relying only on priming by the S1) in both the 15s constant and 15s variable conditions.

Conditioned diminution of the UCR

This was assessed by calculating the uncorrected rate of habituation of the S2 across the 16 pre-omission presentations. The dissimilarity on each presentation for each of the cued/uncued and 5s/15s ISI duration conditions for each learning rule are displayed in Figures 10.8 to 10.12.
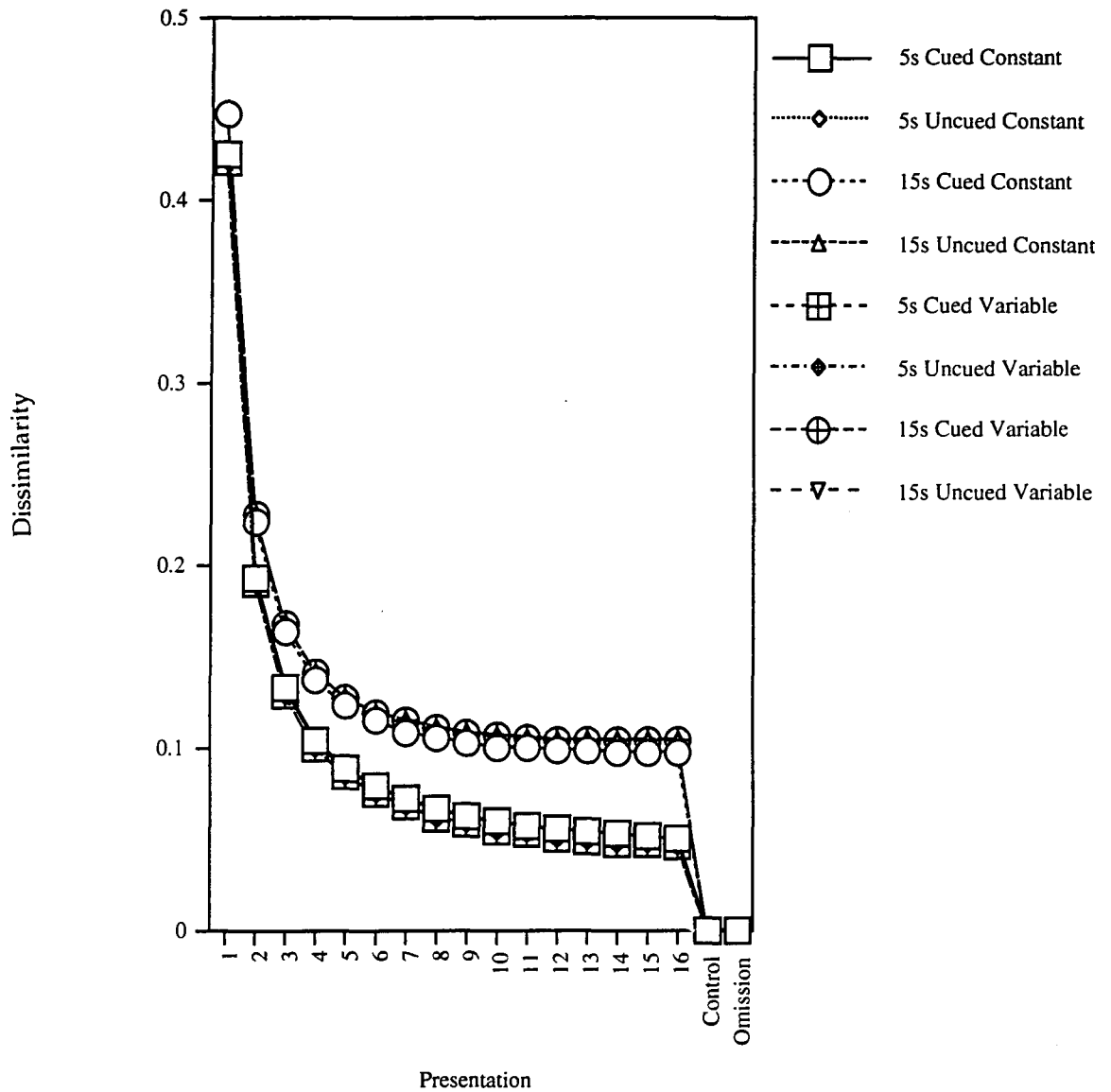
Figure 10.8. Delta model results for mean dissimilarity on each presentation

and stimulus omission at each level of ISI variability, average ISI duration and

cuing by a preceding stimulus.
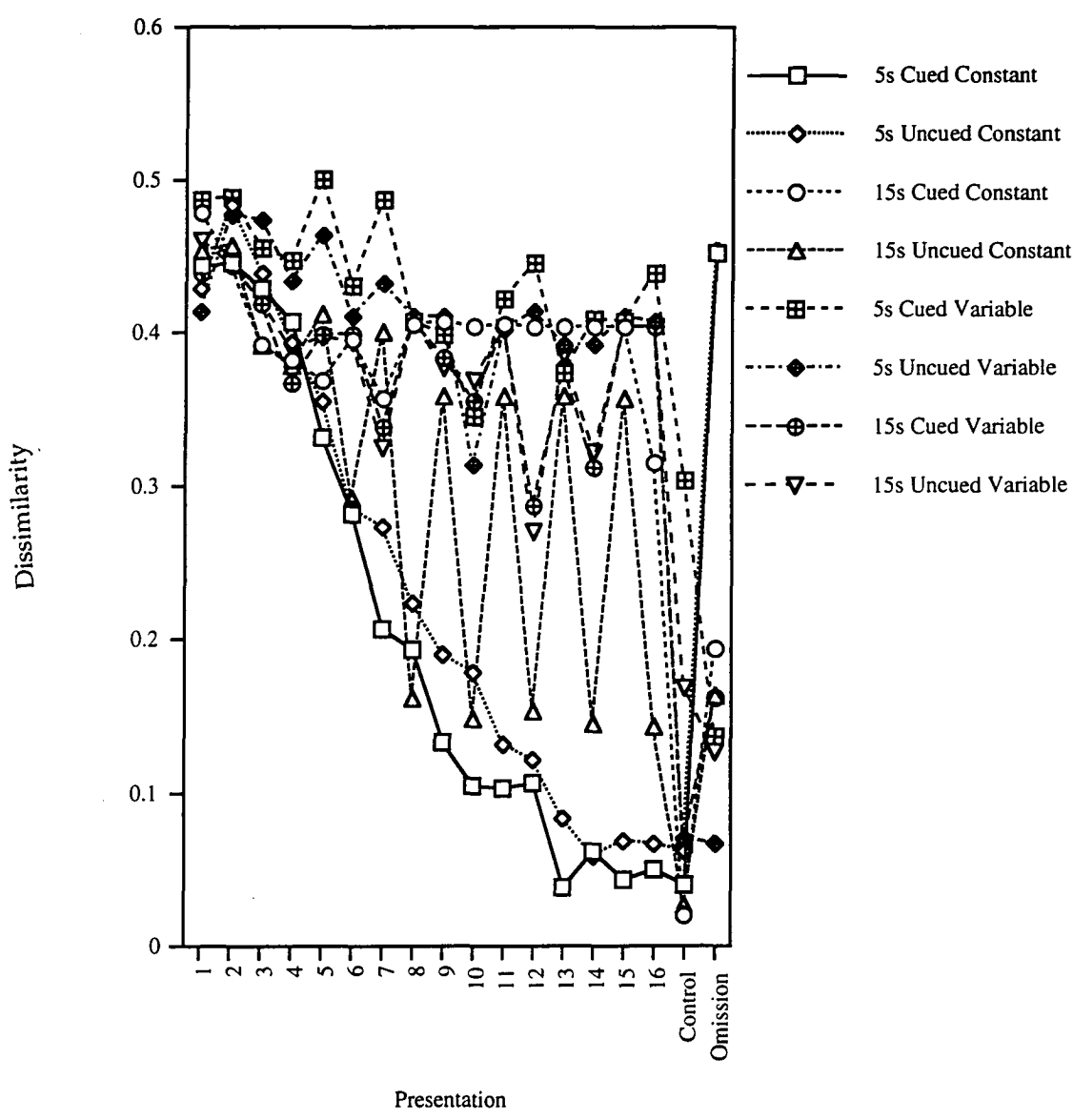
Figure 10.9. Tempo model results for mean dissimilarity on each presentation and stimulus omission at each level of ISI variability, average ISI duration and cuing by a preceding stimulus.
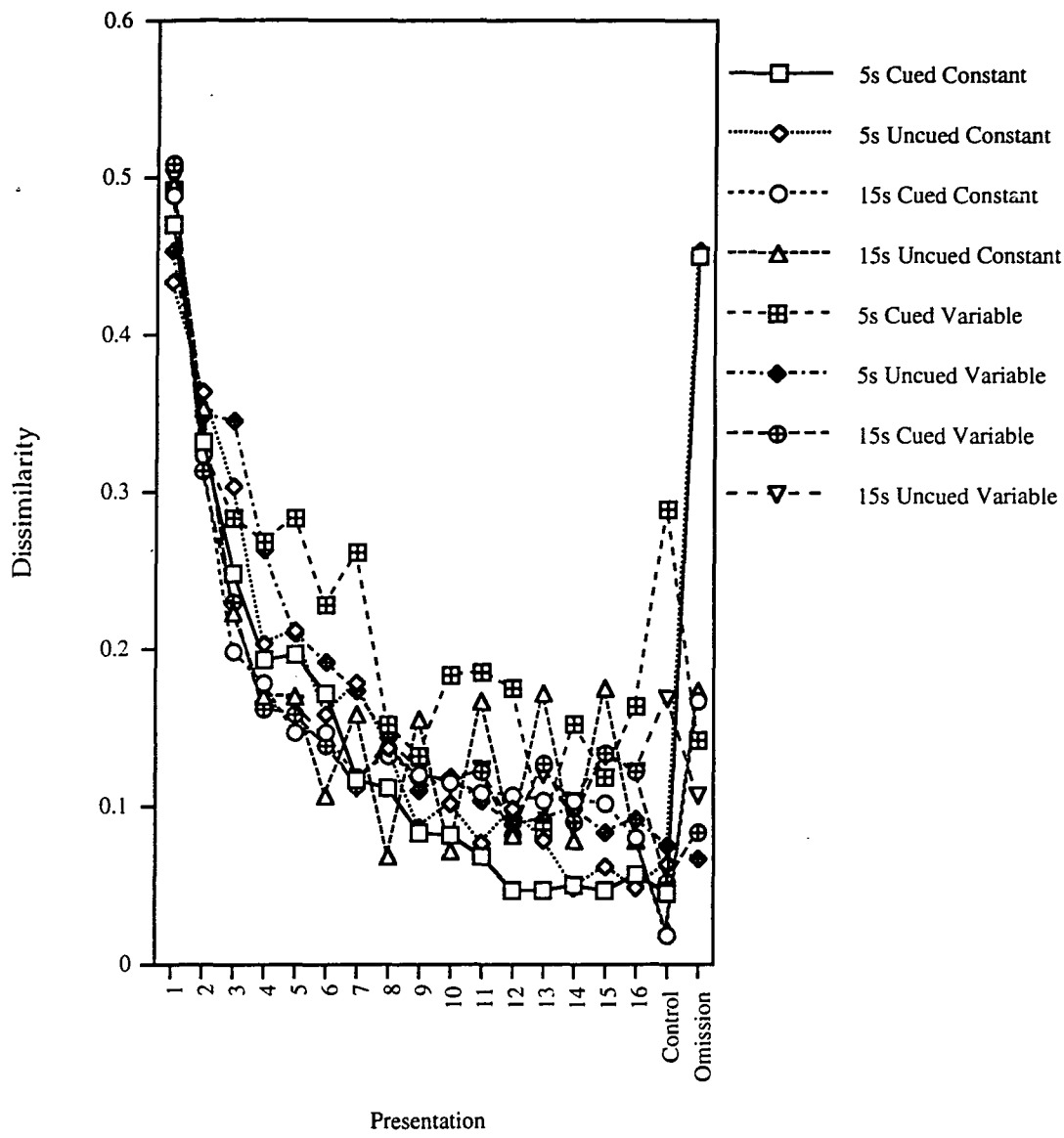
Figure 10.10. DelTempo model results for mean dissimilarity on each presentation and stimulus omission at each level of ISI variability, average ISI duration and cuing by a preceding stimulus.
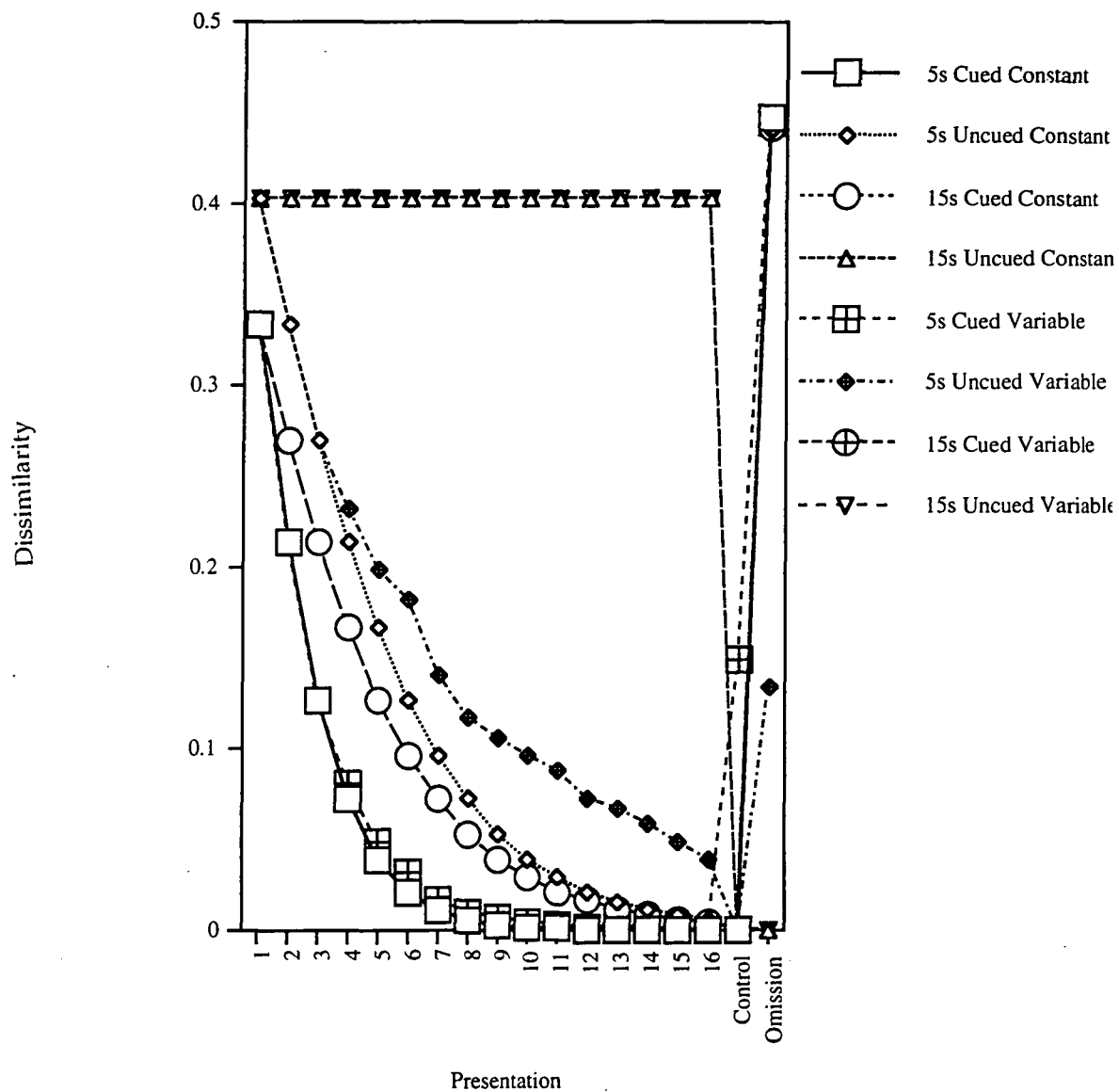
Figure 10.11. Prime model results for mean dissimilarity on each presentation and stimulus omission at each level of ISI variability, average ISI duration and cuing by a preceding stimulus.
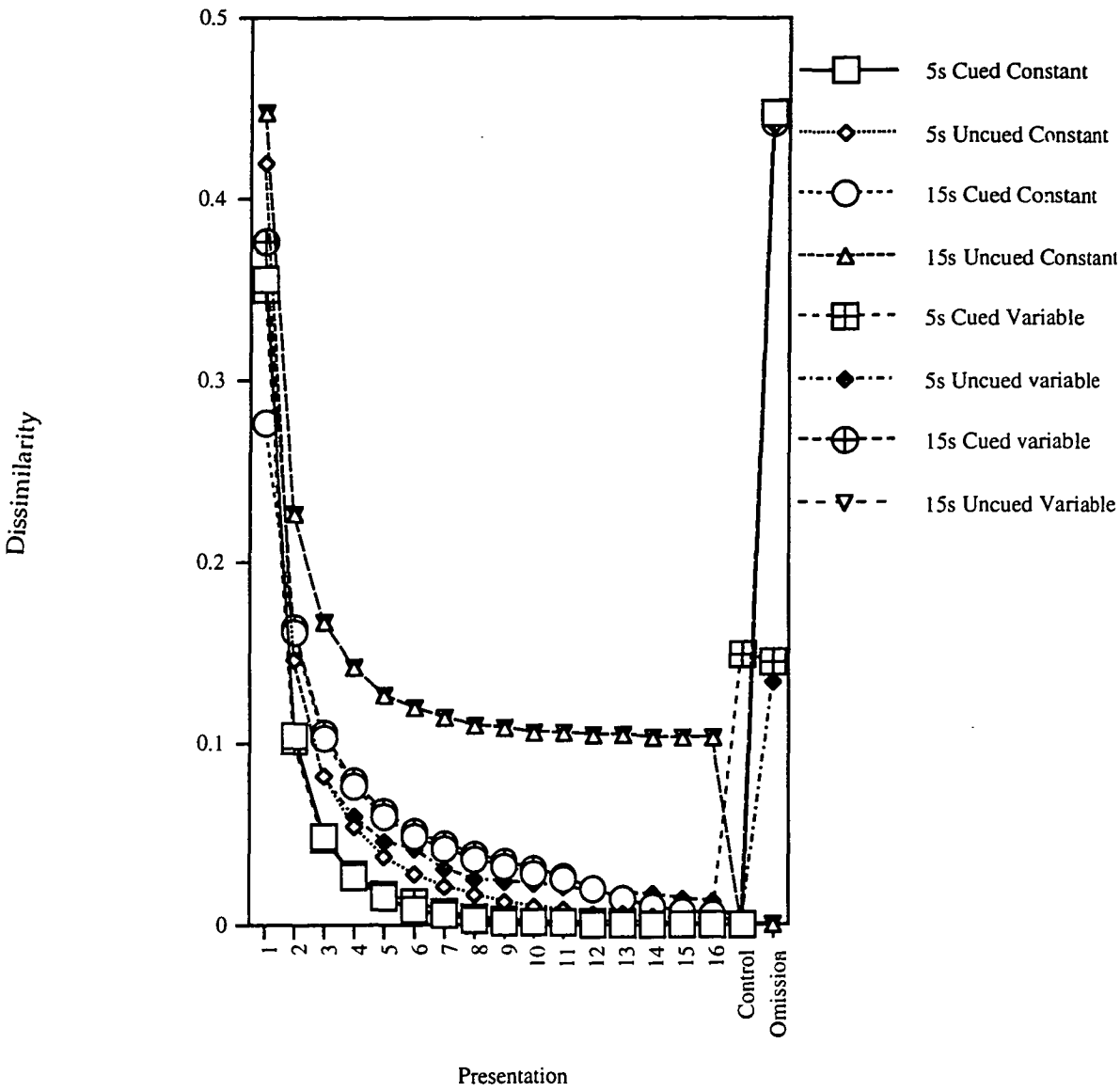
Figure 10.12. PrimeDelta model results for mean dissimilarity on each presentation and stimulus omission at each level of ISI variability, average ISI duration and cuing by a preceding stimulus.

A 2x2x2x5 Cued/Uncued x ISI duration x ISI variability x Rule ANOVA was calculated, followed by planned comparisons testing the effect of cued/uncued at each level of ISI duration and variability for each rule and the effect of ISI duration on the rate of habituation of the cued stimuli for each rule.

Uncorrected rate of S2 habituation

TABLE 10.17

Uncorrected rate of S2 habituation for both cued and uncued S2s with 5 and 15s ISIs and constant and variable S2 periods.

Learning Rule	5s ISI				15s ISI			
	Cued		Uncued		Cued		Uncued	
	Constant	Variable	Constant	Variable	Constant	Variable	Constant	Variable
Delta	-243	-244	-244	-244	-223	-219	-219	-219
	(.000)	(.004)	(.000)	(.004)	(.000)	(.006)	(.000)	(.006)
Tempo	-432	-082	-399	-058	-058	-071	-226	-081
	(.042)	(.000)	(.000)	(.003)	(.037)	(.043)	(.060)	(.036)
DelTempo	-347	-274	-355	-327	-283	-279	-280	-283
	(.035)	(.078)	(.000)	(.039)	(.031)	(.035)	(.028)	(.029)
Priming	-260	-260	-365	-315	-298	-298	.000	.000
	(.000)	(.002)	(.000)	(.003)	(.000)	(.000)	(.000)	(.000)
PrimeDelta	-244	-210	-263	-251	-244	-246	-219	-219
	(.000)	(.001)	(.000)	(.002)	(.000)	(.004)	(.000)	(.000)

A 2 x 2 x 2 x 5 ISI (5 s/15s) Cued/Uncued x Variability (Constant ISI/Variable ISI) x Rule ANOVA was computed with the uncorrected rate of habituation for the S2 as the dependent variable. Each of the effects were highly significant and are listed in Table 10.18.

TABLE 10.18

F and p values for each effect in ISI x Variability x Rule ANOVA for uncorrected rate of habituation of the S2.

Effect	df	F	p
Duration	1, 800	1287.20	.0001
Cued/Uncued	1, 800	37.40	.0001
Variability	1, 800	620.70	.0001
Rule	4, 800	417.90	.0001
Duration x Cued/Uncued	1, 800	331.80	.0001
Duration x Variability	1, 800	322.80	.0001
Cued/Uncued x Variability	1, 800	11.69	.0001
Duration x Rule	4, 800	239.50	.0001
Cued/Uncued x Rule	4, 800	161.70	.0001
Variability x Rule	4, 800	410.10	.0001
Duration x Cued/Uncued x Variability	1, 800	17.40	.0001
Duration x Cued/Uncued x Rule	4, 800	449.70	.0001
Duration x Variability x Rule	4, 800	183.30	.0001
Cued/Uncued x Variability x Rule	4, 800	21.90	.0001
Duration x Cued/Uncued x Variability x Rule	4, 800	23.40	.0001

A set of planned comparisons were then performed to test whether there was a significant effect of cuing of the S2 by the S1 on the uncorrected rate of habituation of the S2 at each of the ISI duration and variability combinations. That is, the rate of uncorrected habituation of the S2 in each condition was compared to the rate observed when the same stimulus was presented in the corresponding uncued series.

TABLE 10.19

Results of planned comparison of the effect of cuing S2 on uncorrected rate of habituation for each combination of ISI variability, average ISI duration and learning rule.

Learning Rule	Contrast (df = 1,800 in each case)			
	Effect of S2 cuing at 5s constant ISI.	Effect of S2 cuing at 5s variable ISI.	Effect of S2 cuing at 15s constant ISI.	Effect of S2 cuing at 15s variable ISI.
Delta	F=.010	F=.00	F=.26	F=.00
	p=.9192	p=1.0	p=.6127	p=1.0
Tempo	F=14.03	F=7.68	F=369.20	F=1.29
	p=.0002	p=.0057	p=.0001	p=.2557
DelTempo	F=2.09	F=37.78	F=.12	F=.18
	p=.1482	p=.0001	p=.7313	p=.6756
Priming	F=145.70	F=39.01	F=1167.50	F=1167.30
	p=.0001	p=.0001	p=.0001	p=.0001
PrimeDelta	F=32.57	F=22.54	p=8.47	F=9.46
	p=.0001	p=.0001	p=.0037	p=.0022

The results for conditioned diminution of the UCR for the Delta rule indicated, as expected, no effect of reliable cuing of the S2 by the S1 on the observed rate of response habituation.

The Tempo rule results indicated a significantly faster rate of response decrement in the cued condition compared to the uncued condition at the 5s ISI for both the constant and variable ISIs. At the 15 s constant ISI condition

however there was significantly faster habituation in the uncued condition compared to the cued condition while there was no difference of cuing on habituation rate for the 15s variable ISI conditions. Coupled with the somewhat erratic performance of the model at the longer ISI for both cued and uncued conditions it was concluded that the Tempo model did not clearly produce an effect of conditioned diminution of the UCR.

The DelTempo were surprising in that there was an effect for S2 cuing on the uncorrected rate of habituation only at the 5s variable condition, this effect was however in the opposite direction to the expected effect with the uncued series showing a faster rate of habituation compared to the cued S2 series. It was expected that this rule would not have showed an effect for temporal cuing at either level of ISI duration or variability. Examination of the figures for both the Tempo and DelTempo rules however do not show as clear an effect for temporal cuing as those of the Prime and PrimeDelta rules, and it is possible that the uncorrected rate of habituation results were not indicative of a clear effect in these cases.

Examination of the figures of the results for the Prime and PrimeDelta rules show a clear effect for the cued stimuli to habituate more rapidly and to a finally greater extent than the uncued stimuli presented at the same ISI. In both cases however these results are reflected by the analysis of the uncorrected rate of habituation results only at the 15s ISI. The 5s ISI results would appear to be in the opposite direction. In the case of both the Prime and PrimeDelta rules this result reflected the situation where the 5s cued condition reached an almost-zero level of dissimilarity after about 10 presentations while an asymptotic level was not reached by the 5s uncued condition after 16 trials. It appears that the different shapes of these two curves affected the calculation of the uncorrected rate of habituation and resulted in values inconsistent with the evidence in Figure 10.13. It was therefore concluded that both the Prime and

PrimeDelta rules provided clear evidence for an effect of conditioned diminution of the UCR at both the 5s and 15s ISIs.

10.3. Discussion of simulations

It is useful at this stage to return to and expand the table of empirical results and theoretical predictions presented in Table 8.1. Table 10.20 adds to this table the results of the simulations described earlier in the chapter.

TABLE 10.20

Habituation phenomena, their empirical status and the theories and neural network models that predict them.

Effect	Empirical Status	Previous Theories				Neural Network Models				
		Sokolov	Wagner (SOP)	Öhman	Dual-process	Delta	Tempo	DelTempo	Prime	PrimeDelta
Recovery of OR to physical stimulus change	√	√	√	√	√	√	√	√	√	√
Generalisation of habituation	√	√	√	√	√	√	√	√	√	√
Conditioned diminution of the UCR	√	X	√	√	√b	X	X	X	√	√
Effect of ISI duration on habituation[1]	√	√	√	√	√	√	X	√	√	√
Effect of ISI variability on habituation[2]	√	√	√	√	√b	X	X	X	√	√
Response to complete stimulus omission[3]	√	√a	√a	√a	√b	X	√	√	√	√
Response to S2 omission after S1-S2 series[4]	√	√a	√a	√a	√b	X	X	X	√	√

√ = Clear evidence for this effect/prediction.
X = Clear evidence against this effect/prediction.
Notes:
1: For the connectionist models to be scored as exhibiting this effect they must have displayed more rapid habituation at the shorter ISI for both the constant and variable ISI series.
2: For the connectionist models to be scored as exhibiting this effect they must have performed similarly to the observations of the experiments performed in earlier chapters, ie. with an effect of ISI variability on rate of habituation at the short ISI but not at the longer ISI.

3: For the connectionist models to be scored as exhibiting this effect they must have performed similarly to the observations of the experiments performed in earlier chapters, ie. with a larger omission response following constant ISI series compared to variable ISI series, and a larger omission response following a short ISI series compared to a longer ISI series.

4: For the connectionist models to be scored as exhibiting this effect they must have show a response to S2 omission in each of the four simulated ISI variability and duration combinations.

a: Can only emit an omission response if disparity between the neuronal trace and afferent stimulus can itself elicit an orienting response. As we saw in Chapter 1 this is a problematic assumption.

b: Requires assumption of ISI encoding elsewhere in the "state system" of the subject, an assumption which seems an ad hoc addition to the basic dual-process theory rather than a natural elaboration.

The presentation of the results in this way provides a valuable indicator of the validity of each connectionist formulation as a model of habituation as well as the links between the new models and the previous models of habituation. It is necessarily a limited test of validity in that the ability of each model to perform under changing conditions of temporal predictability has been emphasised in the current thesis.

Within these limitations, the performance of the simulated models reflects the predictions that could have been made given the knowledge of their specifications. The Delta model, as expected, failed to show any evidence of temporal encoding and thus did not perform well in a number of the simulated experiments.

The Tempo and DelTempo models used the Tempo or period encoding method of temporal encoding where each trace node individually encoded its own ISI of stimulation . These models performed well in the case of complete stimulus omission being able to replicate the pattern of responses observed in Experiment 3 but were less successful in the simulations of the omission of an S2 following a series of S1-S2 presentations and in conditioned diminution of the UCR. It was also sometimes observed that the course of habituation in these models was very erratic with large oscillations in the size of response observed from one presentation to the next, a pattern of responses unlike that

generally seen in human experiments. A possible factor in this unexpected pattern of results is that each node, if not stimulated, would eventually respond at a minimum frequency defined by the minimum level of rate of rise of membrane potential. In the current simulations this minimum rate of firing corresponded to a "resting" ISI of 10 seconds. In many cases there were instants where the nodes happened to fire even at the same time as it was stimulated, simply because the node must fire at least once every 10 seconds. At these coincidental instants the dissimilarity between the input and trace and therefore the observed response will be reduced, causing the low responses which were sometimes observed unexpectedly in the models utilising the Tempo method of temporal encoding.

Both the Prime and PrimeDelta models which used the temporal cuing method were, as would be expected from their definitions, able to successfully produce all of the habituation phenomena simulated in the thesis. This result superficially suggested that the Delta learning rule added no improvement in performance when added to the existing Prime rule. This conclusion does not however bear deeper consideration. The Prime model can, in its current form, be dismissed as a valid model of habituation in that if presented with a series of stimuli with an ISI of greater than the maximum priming eligibility interval the Prime model will not show any response habituation, a result clearly at odds with the real world situation.

The PrimeDelta model is therefore the only one of the five which can produce all of the habituation phenomena tested in the thesis. In the next chapter the PrimeDelta model will be discussed in the context of the previous models of habituation and the novel empirical results presented in the thesis.

CHAPTER 11

Conclusions and future directions

11.1 Results of Psychophysiological Experiments

To conclude and assess the merit of the work described in the preceding chapters it must be shown that the work is both novel and more importantly, that it adds to the body of knowledge already existing in the area. To this end an attempt will now be made to place both the empirical and theoretical contributions of the thesis within the existing literature.

In the earlier chapters of the thesis it was shown that a vital area of uncertainty in theories of habituation of the orienting response is the encoding of the temporal parameters of a stimulus series. Two phenomena which help define the presence and mechanism of this encoding are the effect of ISI variability on the course of habituation and the emission of an orienting response to complete stimulus omission. The existing empirical evidence for both of these phenomena was however conflicting and left their status unclear. Four experiments were then conducted in attempt to elucidate the parameters of these phenomena.

The four experiments can be best considered as two pairs of experiments, the first pair at a longer ISI and the second pair at a shorter ISI. To facilitate experimentation at short ISIs a new scoring method was developed based on the comparison of activity in control and experimentally-relevant scoring windows. It was argued that this method compares favourably with the procedure of Barry et al. (1993) which was also designed for scoring SCR activity at short ISIs. Significant evidence of temporal encoding was found in the experiments conducted with short ISIs (Experiments 3 and 4) but not in the experiments conducted at longer ISIs (Experments 1 and 2). More specifically, it was found that a decrease in the rate of habituation when a single stimulus

was presented at a variable ISI compared to when it was presented at a constant ISI, as well as the response to complete stimulus omission were significant at the shorter ISI (Experiment 3) but not at the longer ISI (Experiment 1). An effect of ISI variability on the course of habituation in the paired stimulus condition was observed at the short (Experiment 4) but not at the longer (Experiment 2) ISI.

11.2. The PrimeDelta model in theoretical context

The importance of ISI as a determinant of the ability of subjects to encode the temporal parameters of a stimulus series was a feature not present in the theories of habituation described in the first chapter of the dissertation. A new theory of habituation of the orienting response was therefore formulated which would be capable of incorporating this effect. It was shown in the previous chapter that the PrimeDelta model which used cuing by a preceding stimulus as its method of temporal encoding was the only connectionist model of the five tested which was able to reproduce both the phenomena investigated in the thesis as well as selected phenomena taken from the existing literature. Of particular interest is the better performance of the PrimeDelta model compared to the DelTempo model. This result suggests that encoding of ISI by the learning of the period of input of each unit is not a satisfactory method of temporal encoding in a model of habituation.

There was a marked departure in the approach taken to modelling of habituation in this thesis compared to the other models discussed in the first chapter. Rather than the largely qualitative approach taken by Sokolov (1963), Öhman (1979) and Groves and Thompson (1970) and to a lesser extent by Wagner (1979, 1981) the current research utilised a neural network or connectionist style of modelling to provide a quantitative approach. The two approaches are compatible and models of either type may be translated into the other. It is asserted that not only are the connectionist models presented in the

thesis compatible with the qualitative models of habituation but also that rather than being simple translations of the existing models, the new models add a greater degree of clarity of explanation and testing of hypotheses than the existing models as well as possessing specific advantages in the explanation of the effect of temporal predictability on habituation.

The connectionist models of habituation defined in the current thesis are essentially comparator models and therefore easily compared with Sokolov's (1963, 1975) model. The neural assembly consisting of the nodes in the input and trace layers and their connections of each connectionist model described in the thesis (Delta, Tempo, DelTempo, Prime or PrimeDelta) can be thought of as particular versions of the neuronal model component of Sokolov's comparator model of habituation. In the original model Sokolov postulated a number of parameters which were encoded by his neuronal model but did not provide a quantitative mechanism showing how the these stimulus parameters were learned over repeated stimulus presentations. In the current thesis, however, specific mechanisms and processes were defined which are performed within the neuronal model, a modification which allowed the a simpler testing of the validity of the model than did the earlier qualitative formulation. A second difference between the current models and Sokolov's (1963, 1975) model is in the way that the orienting response is generated. In Sokolov's model (See Figure 1.1) an orienting response is elicited when the current input fails to correspond to the activity in the neuronal model. As Hall (1989) points out, this mechanism will fail to produce a response at any stage when there is no afferent stimulus and can not therefore account for the elicitation of a response to complete stimulus omission. While there may well be a disparity between the afferent stimulation and the activity of the neuronal model at the time of stimulus omission Sokolov failed to clearly include a mechanism by which this disparity could result in the elicitation of an orienting response. The current models, of which the PrimeDelta proved the most satisfactory, avoid this

shortcoming by allowing the orienting response to be a direct index of the mismatch between the learned trace of previous stimulation stored as the pattern of connection weights and activities within the network, and the current afferent stimulation to the input layer of the network. In summary, the PrimeDelta rule could be considered, although this is not how it was originally conceived, as a modified version of Sokolov's comparator theory with some refinements. The encoding of ISI is specifically defined to be by the learning of the time intervals between a stimulus and other events preceding it within a certain eligibility period while the orienting response is directly elicited by a mismatch between the trace of previous stimulation and the current stimulation. In Sokolov's original theory the OR was elicited by a more complex route where the mismatch between the afferent stimulus and neuronal model acted as a gate between the input and a second system which generates the OR. The definition of the OR as a direct measure of disparity of the activity patterns in the input and trace layers is, however, susceptible to a criticism that any small disparity would result in the elicitation of an OR. These responses may be analogous to the non-specific responses observed in human studies or alternatively could be suppressed in the PrimeDelta model by the application of a threshold or other function during the calculation of the OR from the activity in the input and trace layers.

Öhman's (1979) information processing theory has a more complex set of mechanisms which interact to provide an account of elicitation of the orienting response under varying conditions. The PrimeDelta model and other connectionist models of habituation are complementary to Öhman's theory rather than competitors for it. Öhman (1979) proposed two routes to the elicitation of an OR, a signal route when a stimulus is recognised as significant and a non-signal route, in which similar to Sokolov's theory the OR is elicited by a mismatch between the afferent stimulation and a trace of previous stimulation stored in what Öhman termed a short-term store. The OR itself was

elicited directly by either this mismatch in the case of the non-signal route or the match in the case of the signal route, unlike Sokolov's theory where an OR was elicited only if an external stimulus was presented. The PrimeDelta and other connectionist theories described in this thesis with Öhman's theory the connectionist models provide alternative explicitly defined substrates and mechanisms for the non-signal route to OR elicitation with the dissimilarity measure as an analog of Öhman's call to processing definition of the OR.

Wagner's priming (1978) and SOP (1981) theories also rely upon the notion of the OR as an index of the disparity between the current stimulation and the activity in a trace of previous stimulation again termed the short term store (STS). A stimulus would not evoke an orienting response if it was already pre-represented or primed in the STS. A stimulus could become primed in the STS via two mechanisms, self-generated priming which results from recent presentation of the same stimulus and retrieval-generated priming resulting from the presentation of stimuli or a context which have previously cued the presentation of the primed stimulus. The PrimeDelta theory has a number of parallels with SOP but possesses additional features which extend the performance of SOP in certain situations. Self-generated priming can be thought of as being implemented by the Delta rule learning in the PrimeDelta model while the Prime rule learning provides an analog of retrieval-generated priming.

There are important differences between the PrimeDelta model and SOP. The first is that, like Sokolov's comparator theory, SOP states that the OR is an index of the disparity between the afferent stimulation and the pre-represented activity in the STS but provides no mechanism by which the disparity alone can elicit an OR in the absence of stimulation. The PrimeDelta model avoids this criticism by the OR being a direct index of this disparity. The second difference between SOP and the PrimeDelta model is not so much a difference as a

clarification. In SOP it was not clear that the ISI of a stimulus could be learnt and thus prime the STS at the time of stimulus presentation. If a number of assumptions were made allowing the presentation of a stimulus to use retrieval-generated priming to prime its own next presentation then this could be used as a method of ISI encoding. In the PrimeDelta model these assumptions were made explicit with lateral connections in the input layer encoding the intervals between the stimulation of different nodes connecting each input node with all input nodes including itself, providing an explicit substrate for the encoding of the ISI of a series of presentations of a single stimulus.

The parallels between dual-process theory (Groves & Thompson, 1970; Thompson et al., 1979) and the connectionist models presented in this thesis are fewer than was the case for the other theories. In dual-process theory inferred habituation occurs in the S-R pathway resulting in a decrease in response magnitude as a stimulus is repeatedly presented while no analogous connections can be found in the PrimeDelta theory. A possible place that the processes within the PrimeDelta model could be incorporated into the dual-process model is in the state system part of dual-process theory where inferred sensitisation is postulated to occur. If the state system possessed the ISI encoding properties of the PrimeDelta model then this would provide the dual-process model with a capability of exhibiting a response to stimulus omission and an effect of ISI variability on the rate of habituation. The relative arguments for the dual-process and comparator theories were discussed in Chapter 1 and will not be reiterated here. Suffice to say that the PrimeDelta model is open to many of the criticisms levelled at comparator theories, most significantly the difficulty in locating neural structures showing the physiology necessary to perform the functions of the model. It is however clear that the empirical results reported in the earlier chapters of the thesis could not be

accommodated by the dual-process theory without some modification. The PrimeDelta model suggests a basis for some of these modifications.

The PrimeDelta theory therefore can be considered a refinement of, or a partial substitute for, many of the existing theories of habituation. It is most closely allied in its conception to the comparator-like theories of Sokolov (1963), Wagner (1978, 1981) and Öhman (1979) but is more than a simple translation of any of them. To all of these theories it adds a specific assumption that ISI is encoded by the learning of the intervals between a stimulus presentation and a cuing event and that this learning is limited to a range of intervals (0-10 seconds in the current example). To Sokolov's and Wagner's theories it adds the assumption that the disparity between the trace of previous stimulation and the current afferent stimulation is sufficient to elicit an OR, whether an external stimulus is present or not. The PrimeDelta model also adds to all these theories an arguably simpler and more conclusive method of testing its predictions under simulated experimental conditions, a feature which will aid future investigators assessing the validity of the model.

One area which has not been developed in the PrimeDelta model is the question of individual differences in habituation of the OR. Differences in learning rates between individual simulations/subjects would more closely imitate the results seen in psychophysiological experiments. The introduction of different degrees of randomness in the action of the models would be expected to result in the elicitation of non-specific responses which would enhance the realism of the simulated results.

11.3. Future directions

The work presented in this dissertation has placed much emphasis on the application of novel techniques to the study of SCR habituation and the formulation of models of habituation. Three particular points stand out as areas which could benefit from further investigation.

The first is the use of the novel scoring techniques used for Experiments 3 and 4. With the increasing proliferation of ERP research in psychophysiology it is vital that autonomic nervous system research develops in such a way that the measurement of both indices is feasible within a single experiment. Experiments utilising ERP indices are generally performed at shorter ISIs than are experiments using autonomic measures. The measures developed in this thesis provide one way of analysing SCR at shorter ISIs than are commonly used. Further experiments contrasting the validity and parameters of both these techniques as well as alternatives such as that proposed by Barry et al. (1993) would be of a benefit to the integration of central and autonomic system studies in the future. In addition, the new techniques extend the study of SCR to shorter ISIs than was previously possible, a property which proved useful in Experiments 3 and 4 and would likely prove useful in future studies.

The second major point of interest of the thesis is the conclusion that the effect of ISI variability on the course of habituation and the response to complete stimulus omission are ISI dependent effects which are present at short ISIs (approximately 1-2 seconds) but are not present at longer (> 15 seconds) ISIs. Further experimentation could be conducted to confirm or extend these findings. The status of a response to omission of an S2 following a series of S1-S2 presentations, and its possible correlation with the duration of the S1-S2 interval, would also benefit from further investigation.

The third area which would benefit from further study is in the elaboration of the PrimeDelta model and its validity as a model of habituation. Particular areas of interest include the search for neural structures corresponding to those proposed in the model or conversely alteration of the model (e.g., replacement of the delta learning rule with a Hebbian learning rule) to more truly reflect known neural structures, the investigation of different functions for the dissimilarity measure, and the delineation of the parallels between the

PrimeDelta model, other models of habituation and connectionist models of other types. The addition of mechanisms allowing the simulation of individual differences in habituation would also be valuable.

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APPENDICES

APPENDIX A

Publications arising from the work presented in this thesis:

Daniels, B.A. (1992). *Habituation of the skin conductance response (SCR) to different temporal sequences.* Paper presented at the Australasian Psychpohysiology Conference. Nelson Bay, New South Wales

Daniels, B.A. (1993). A neural network implementation of Sokolov's model of habituation of the orienting response. In P. Leong & M. Jabri (Eds.), *Proceedings of the Fourth Australian Conference on Neural networks* (pp. 57-60). Sydney: Sydney University Electrical Engineering.

Daniels, B.A., & Davidson, J.A. SCR dishabituation to complete stimulus omission and ISI variability change with short ISIs. Manuscript in preparation.

APPENDIX B

Scores for each subject on selected dependent variables for Experiment 1.

Key:

Subject Number: Subject Identification Number.

Condition: Variable or fixed ISI condition.

Max. SCR: Maximum SCR observed in the pre-omission series.

Total SCR: Total SCR observed in the pre-omission series.

N of SCR: Number of SCRs > 0.02μS in magnitude in pre--omission series.

NSR: Number of non-specific responses scored in pre-stimulation scoring period.

Lability: Electrodermal Lability group obtained by median split of NSR.

T hab: Trials to habituation.

Habit. Rate: Uncorrected rate of habituation. i.e., value of b in regression equation; Response = a + b log(presentation number) calculated across all pre-omission stimulus presentations.

Subject Number	Condition	Max SCR	Total SCR	N of SCR	NSR	Lability	T hab	Habit. Rate
1	fixed	.92	2.73	8	3	Stabile	5	-.8753
2	fixed	.09	.19	4	2	Stabile	3	-.0500
3	fixed	.52	1.32	9	10	Labile	4	-.2380
4	fixed	.88	6.96	48	28	Labile	18	-.6177
5	fixed	.54	4.68	45	28	Labile	7	-.3078
6	fixed	.44	.79	5	5	Stabile	5	-.2366
7	fixed	.06	.20	6	0	Stabile	5	-.0302
8	fixed	1.05	7.53	36	20	Labile	4	-.5748
9	fixed	1.00	3.31	21	9	Labile	4	-.0101
10	fixed	.07	.09	2	1	Stabile	7	-.0406
11	fixed	.86	4.35	21	15	Labile	10	-.5110
12	fixed	.42	2.04	18	19	Labile	6	-.3129
13	fixed	.97	1.43	5	11	Labile	5	-.4897
14	fixed	.79	7.20	35	29	Labile	8	-.6149
15	fixed	1.00	10.95	49	28	Labile	7	-.5234
16	fixed	.63	3.68	32	15	Labile	10	-.3327
17	fixed	2.40	22.45	35	32	Labile	11	-1.5219
18	fixed	2.00	5.72	8	1	Stabile	6	-1.0579
19	fixed	.96	8.64	41	20	Labile	18	-.5596
20	fixed	.03	.07	3	0	Stabile	3	-.0409
21	fixed	1.00	4.99	15	5	Stabile	5	-.7747
22	fixed	2.50	14.82	44	28	Labile	14	-1.1714
23	fixed	1.01	6.84	20	6	Stabile	18	-.7968
24	fixed	2.50	39.15	59	31	Labile	8	-1.9200
25	fixed	.92	8.15	33	6	Stabile	4	-.4622
26	fixed	.70	4.90	21	17	Labile	18	-.3079
27	fixed	2.40	13.21	33	16	Labile	10	-1.6441
28	fixed	.95	3.09	20	4	Stabile	6	-.5354
29	fixed	.38	.41	2	3	Stabile	2	-.1703
30	fixed	1.85	14.61	27	5	Stabile	18	-1.1716
31	fixed	.68	2.61	23	0	Stabile	13	-.1509
32	fixed	.40	2.25	23	10	Labile	13	-.3413
33	fixed	.95	7.09	27	8	Stabile	18	-.5949
34	fixed	2.50	18.26	43	30	Labile	18	-2.2140
35	variable	.70	5.69	39	23	Labile	18	.5631
36	variable	.43	2.09	17	11	Labile	14	-.3470

Subject Number	Condition	Max SCR	Total SCR	N of SCR	NSR	Lability	T hab	Habit. Rate
37	variable	1.00	7.12	29	14	Labile	8	-.4934
38	variable	.30	.71	9	6	Stabile	6	-.1807
39	variable	.05	.05	1	0	Stabile	2	-.0397
40	variable	.65	5.11	29	12	Labile	10	-.4789
41	variable	.53	1.35	6	4	Stabile	6	-.2888
42	variable	.90	8.18	53	31	Labile	18	-.6135
43	variable	.42	1.31	10	4	Stabile	6	-.3071
44	variable	.03	.03	1	0	Stabile	1	-.0170
45	variable	.12	.12	1	0	Stabile	1	-.0511
46	variable	.98	7.03	38	19	Labile	10	-.5594
47	variable	1.00	5.27	28	16	Labile	11	-.7434
48	variable	.75	2.48	19	7	Stabile	12	-.3396
49	variable	1.00	1.86	4	1	Stabile	1	-.0544
50	variable	.72	4.03	21	4	Stabile	4	-.2780
51	variable	1.10	40.85	84	35	Labile	18	-.5861
52	variable	.29	.62	5	0	Stabile	5	-.1820
53	variable	.18	1.46	20	5	Stabile	10	-.1633
54	variable	.94	5.46	27	7	Stabile	7	-.4800
55	variable	.58	2.69	17	2	Stabile	6	-.2735
56	variable	.45	4.36	29	10	Labile	4	-.3174
57	variable	.80	4.89	29	19	Labile	7	-.5519
58	variable	.07	.07	1	2	Stabile	1	-.0298
59	variable	1.25	11.44	47	21	Labile	18	-.6964
60	variable	.57	2.52	28	10	Labile	8	-.2668
61	variable	2.00	4.98	12	9	Labile	4	-1.4105
62	variable	1.45	14.23	43	25	Labile	15	-1.1750
63	variable	.89	3.00	15	1	Stabile	9	-.7255
64	variable	.46	2.12	14	11	Labile	7	-.2887
65	variable	1.00	4.82	17	11	Labile	5	-.4496
66	variable	.55	1.18	11	8	Stabile	3	-.0775
67	variable	1.05	5.62	30	11	Labile	4	-.4438
68	variable	2.05	18.76	51	23	Labile	18	-.8440
69	variable	2.05	4.61	8	0	Stabile	8	-1.1013
70	variable	1.10	1.43	5	5	Stabile	5	-.5440
71	variable	1.45	5.83	27	27	Labile	6	-1.0246
72	variable	2.50	16.82	44	17	Labile	18	-1.3033

Subject Number	Condition	Max SCR	Total SCR	N of SCR	NSR	Lability	T hab	Habit. Rate
73	variable	.80	3.85	25	3	Stabile	7	-.3987
76	fixed	.02	.02	1	0	Stabile	1	-.0085
77	fixed	.02	.08	4	0	Stabile	1	-.0056
78	fixed	.19	.19	1	0	Stabile	1	-.0809
79	fixed.	.08	.08	1	1	Stabile	1	-.0341
80	fixed	.04	.04	1	2	Stabile	1	-.0170

Scores for each subject on selected dependent variables for Experiment 2.

Key:

Subject Number: Subject Identification Number.

Condition: Control, Variable SOA or Variable S2 Period.

SCR Max.: Maximum SCR observed in the pre-omission series.

SCR Total: Total SCR observed in the pre-omission series.

SCR N: Number of SCRs > 0.02µS in magnitude in pre--omission series.

NSR: Number of non-specific responses scored in pre-stimulation scoring period.

Lability: Electrodermal Lability group obtained by median split of NSR.

TRIALS TO HAB: Trials to habituation.

Habit. Rate: Uncorrected rate of S2 habituation. i.e., value of b in regression equation; Response = a + b log(presentation number) calculated across all pre-omission stimulus presentations.

Control Window: Maximum response in Control scoring window.

Omission Window: Maximum response in Omission scoring window.

S2 t18- s2 t16: Difference in response to S2 on trials 16 (pre-omission) and 18 (post-omission).

S1 t18- s1 t16: Difference in response to S1 on trials 16 (pre-omission) and 18 (post-omission).

	Subject Number	Condition	SCR Max.	SCR Total	SCR N	Sqrt Max SCR	Habit. Rate	Control Window
1	1	Offset (SOA) V...	.08	.08	1	.283	-.038	0.000
2	2	Offset (SOA) V...	1.00	5.48	17	1.000	-.122	0.000
3	3	Control	.95	6.05	39	.975	.153	.080
4	4	Control	1.06	10.65	53	1.030	-.412	0.000
5	5	Offset (SOA) V...	1.08	19.11	16383	1.039	-.823	.320
6	6	Period (ISI) Va...	1.12	3.01	•	1.058	-.180	0.000
7	7	Offset (SOA) V...	1.00	3.85	0	1.000	-.490	0.000
8	8	Offset (SOA) V...	1.60	11.85	0	1.265	.067	0.000
9	9	Offset (SOA) V...	.65	1.27	0	.806	-.307	0.000
10	10	Control	1.08	3.99	16382	1.039	-.596	0.000
11	11	Offset (SOA) V...	.34	1.34	0	.583	-.066	0.000
12	12	Control	.56	1.54	-1660	.748	-.265	0.000
13	13	Period (ISI) Va...	.76	1.33	-26777	.872	-.090	0.000
14	14	Period (ISI) Va...	1.75	16.55	11745	1.323	-1.057	.360
15	15	Offset (SOA) V...	.47	1.83	-21482	.686	-.074	0.000
16	16	Offset (SOA) V...	.25	.51	16383	.500	-.009	0.000
17	17	Control	.79	5.49	0	.889	-.451	0.000
18	18	Offset (SOA) V...	1.15	10.41	-31800	1.072	-.284	0.000
19	19	Offset (SOA) V...	.54	4.35	-18295	.735	-.002	0.000
20	20	Control	1.06	2.36	23760	1.030	-.213	0.000
21	21	Offset (SOA) V...	.57	1.78	12661	.755	-.041	0.000
22	22	Offset (SOA) V...	.78	8.02	16383	.883	-.370	.040
23	23	Control	1.25	31.39	0	1.118	-.418	.300
24	24	Control	.03	.07	-31483	.173	-.014	0.000
25	25	Period (ISI) Va...	2.50	23.94	-32472	1.581	-1.075	.150
26	26	Offset (SOA) V...	.94	5.88	3644	.970	-.025	0.000
27	27	Offset (SOA) V...	1.35	7.95	-13371	1.162	-.321	0.000
28	28	Control	.70	9.00	16383	.837	-.271	.020
29	29	Control	.90	10.26	0	.949	-.439	0.000
30	30	Offset (SOA) V...	1.80	15.41	-30858	1.342	-1.050	0.000
31	31	Period (ISI) Va...	.80	1.06	25662	.894	-.095	0.000
32	32	Offset (SOA) V...	.96	11.85	-8238	.980	-.580	.180
33	33	Period (ISI) Va...	.47	1.49	-23401	.686	-.244	0.000
34	34	Control	2.50	15.15	16383	1.581	-.789	0.000
35	35	Offset (SOA) V...	1.35	16.50	.0	1.162	-.636	0.000
36	36	Period (ISI) Va...	.70	3.45	•	.837	-.225	0.000

	Omission Window	NSR	Lability	TRIALS TO HAB	S2 t18-S2 t16	s1 T18 - S1 T16	Input Column
1	0.00	0	Stabile	1	0.000	0.00	
2	0.00	1	Stabile	2	0.000	0.00	
3	.02	4	Stabile	6	.020	.17	
4	0.00	20	Labile	9	.020	-.23	
5	.02	21	Labile	15	.530	-.85	
6	0.00	0	Stabile	1	0.000	-.38	
7	0.00	1	Stabile	3	.130	-.06	
8	0.00	26	Labile	1	0.000	0.00	
9	0.00	1	Stabile	1	0.000	0.00	
10	0.00	8	Stabile	3	0.000	0.00	
11	0.00	2	Stabile	4	0.000	0.00	
12	0.00	4	Stabile	1	0.000	0.00	
13	0.00	3	Stabile	1	0.000	-.09	
14	.08	29	Labile	5	0.000	.33	
15	0.00	14	Labile	5	0.000	.22	
16	0.00	1	Stabile	1	0.000	0.00	
17	.07	7	Stabile	7	.210	0.00	
18	0.00	7	Stabile	1	0.000	0.00	
19	.02	14	Labile	5	0.000	-.03	
20	0.00	6	Stabile	1	0.000	0.00	
21	0.00	2	Stabile	2	.030	0.00	
22	.03	10	Labile	2	0.000	0.00	
23	.74	32	Labile	11	.230	-.41	
24	0.00	2	Stabile	1	0.000	0.00	
25	0.00	24	Labile	9	.750	0.00	
26	0.00	12	Labile	2	.060	-.02	
27	0.00	6	Stabile	1	0.000	0.00	
28	.04	14	Labile	12	.360	.12	
29	0.00	14	Labile	3	0.000	0.00	
30	0.00	40	Labile	4	.160	.05	
31	0.00	3	Stabile	1	0.000	0.00	
32	0.00	11	Labile	6	.020	.66	
33	0.00	1	Stabile	3	0.000	0.00	
34	.06	26	Labile	3	.250	1.06	
35	.61	24	Labile	7	0.000	-.50	
36	0.00	10	Labile	3	.050	.07	

	Subject Number	Condition	SCR Max.	SCR Total	SCR N	Sqrt Max SCR	Habit. Rate	Control Window
37	37	Period (ISI) Va...	.88	4.08	17	.938	-.013	0.000
38	38	Period (ISI) Va...	1.00	22.46	72	1.000	-.505	.820
39	39	Period (ISI) Va...	2.05	19.06	65	1.432	-.932	0.000
40	40	Period (ISI) Va...	.30	.55	5	.548	-.009	0.000
41	41	Period (ISI) Va...	.88	7.36	41	.938	.054	0.000
42	42	Period (ISI) Va...	.68	11.03	43	.825	.088	0.000
43	43	Period (ISI) Va...	1.10	14.12	44	1.049	-.501	0.000
44	44	Offset (SOA) V...	.36	6.36	45	.600	-.107	.040
45	45	Control	1.83	17.36	66	1.353	-1.035	.090
46	46	Period (ISI) Va...	1.00	17.51	51	1.000	-.633	0.000
47	47	Offset (SOA) V...	1.70	12.77	46	1.304	-.652	0.000
48	48	Offset (SOA) V...	.16	.45	5	.400	-.057	0.000
49	49	Period (ISI) Va...	.96	13.90	76	.980	-.088	.120
50	50	Period (ISI) Va...	.60	2.55	36	.775	-.164	0.000
51	51	Control	.87	1.04	2	.933	-.080	0.000
52	52	Period (ISI) Va...	.46	7.15	46	.678	.016	.030
53	53	Offset (SOA) V...	.64	2.20	18	.800	-.055	0.000
54	54	Control	.50	5.73	34	.707	-.047	.840
55	55	Period (ISI) Va...	.88	25.51	109	.938	.162	.130
56	56	Control	.69	14.01	81	.831	-.264	.150
57	57	Control	.93	6.08	24	.964	-.804	0.000
58	58	Control	.75	4.76	32	.866	-.051	0.000
59	59	Period (ISI) Va...	.55	4.08	26	.742	-.214	.090
60	60	Control	.90	5.88	26	.949	-.607	0.000
61	61	Offset (SOA) V...	1.00	2.21	7	1.000	-.209	0.000
62	62	Period (ISI) Va...	1.02	5.42	26	1.010	-.323	0.000
63	63	Period (ISI) Va...	1.05	12.56	29	1.025	-.028	0.000
64	64	Control	.14	.37	4	.374	-.052	0.000
65	65	Period (ISI) Va...	.65	2.50	17	.806	.002	.080
66	66	Control	.94	13.12	48	.970	-.505	.020
67	67	Control	1.00	6.47	39	1.000	-.719	.130
68	68	Control	.51	9.81	62	.714	-.225	.040
69	69	Control	.20	.22	2	.447	-.009	0.000
70	70	Offset (SOA) V...	1.00	8.33	20	1.000	-.923	0.000
71	71	Period (ISI) Va...	.02	.02	1	.141	-.014	0.000
72	72	Control	.39	2.94	23	.624	.047	0.000

	Omission Window	NSR	Lability	TRIALS TO HAB	S2 t18-S2 t16	s1 T18 - S1 T16	Input Column
37	0.00	6	Stabile	1	0.000	0.00	
38	.64	20	Labile	5	-.020	.13	
39	0.00	27	Labile	11	.940	.70	
40	0.00	5	Stabile	1	0.000	0.00	
41	0.00	13	Labile	5	-.700	.22	
42	0.00	7	Stabile	0	0.000	.14	
43	0.00	19	Labile	7	0.000	.02	
44	.32	9	Labile	5	0.000	0.00	
45	.63	34	Labile	16	.100	0.00	
46	.02	12	Labile	3	0.000	.22	
47	0.00	24	Labile	7	.020	.21	
48	0.00	4	Stabile	1	0.000	0.00	
49	.30	24	Labile	8	-.070	.12	
50	0.00	12	Labile	6	.070	.08	
51	0.00	3	Stabile	1	0.000	.02	
52	.10	1	Stabile	16	.030	.42	
53	0.00	3	Stabile	1	0.000	0.00	
54	0.00	11	Labile	2	0.000	0.00	
55	.34	12	Labile	16	.340	.02	
56	.02	35	Labile	16	-.030	.42	
57	0.00	18	Labile	6	-.060	-.03	
58	0.00	0	Stabile	7	0.000	.07	
59	0.00	6	Stabile	4	.030	0.00	
60	0.00	14	Labile	2	0.000	0.00	
61	0.00	15	Labile	2	0.000	0.00	
62	0.00	2	Stabile	9	0.000	0.00	
63	0.00	10	Labile	1	0.000	0.00	
64	0.00	0	Stabile	1	0.000	0.00	
65	0.00	8	Stabile	1	.020	0.00	
66	.50	13	Labile	8	0.000	.50	
67	0.00	4	Stabile	3	.040	.04	
68	0.00	12	Labile	5	.360	0.00	
69	0.00	3	Stabile	1	0.000	0.00	
70	0.00	5	Stabile	8	0.000	0.00	
71	0.00	0	Stabile	1	0.000	0.00	
72	.14	3	Stabile	0	-.240	-.21	

	Subject Number	Condition	SCR Max.	SCR Total	SCR N	Sqrt Max SCR	Habit. Rate	Control Window
37	37	Period (ISI) Va...	.88	4.08	17	.938	-.013	0.000
38	38	Period (ISI) Va...	1.00	22.46	72	1.000	-.505	.820
39	39	Period (ISI) Va...	2.05	19.06	65	1.432	-.932	0.000
40	40	Period (ISI) Va...	.30	.55	5	.548	-.009	0.000
41	41	Period (ISI) Va...	.88	7.36	41	.938	.054	0.000
42	42	Period (ISI) Va...	.68	11.03	43	.825	.088	0.000
43	43	Period (ISI) Va...	1.10	14.12	44	1.049	-.501	0.000
44	44	Offset (SOA) V...	.36	6.36	45	.600	-.107	.040
45	45	Control	1.83	17.36	66	1.353	-1.035	.090
46	46	Period (ISI) Va...	1.00	17.51	51	1.000	-.633	0.000
47	47	Offset (SOA) V...	1.70	12.77	46	1.304	-.652	0.000
48	48	Offset (SOA) V...	.16	.45	5	.400	-.057	0.000
49	49	Period (ISI) Va...	.96	13.90	76	.980	-.088	.120
50	50	Period (ISI) Va...	.60	2.55	36	.775	-.164	0.000
51	51	Control	.87	1.04	2	.933	-.080	0.000
52	52	Period (ISI) Va...	.46	7.15	46	.678	.016	.030
53	53	Offset (SOA) V...	.64	2.20	18	.800	-.055	0.000
54	54	Control	.50	5.73	34	.707	-.047	.840
55	55	Period (ISI) Va...	.88	25.51	109	.938	.162	.130
56	56	Control	.69	14.01	81	.831	-.264	.150
57	57	Control	.93	6.08	24	.964	-.804	0.000
58	58	Control	.75	4.76	32	.866	-.051	0.000
59	59	Period (ISI) Va...	.55	4.08	26	.742	-.214	.090
60	60	Control	.90	5.88	26	.949	-.607	0.000
61	61	Offset (SOA) V...	1.00	2.21	7	1.000	-.209	0.000
62	62	Period (ISI) Va...	1.02	5.42	26	1.010	-.323	0.000
63	63	Period (ISI) Va...	1.05	12.56	29	1.025	-.028	0.000
64	64	Control	.14	.37	4	.374	-.052	0.000
65	65	Period (ISI) Va...	.65	2.50	17	.806	.002	.080
66	66	Control	.94	13.12	48	.970	-.505	.020
67	67	Control	1.00	6.47	39	1.000	-.719	.130
68	68	Control	.51	9.81	62	.714	-.225	.040
69	69	Control	.20	.22	2	.447	-.009	0.000
70	70	Offset (SOA) V...	1.00	8.33	20	1.000	-.923	0.000
71	71	Period (ISI) Va...	.02	.02	1	.141	-.014	0.000
72	72	Control	.39	2.94	23	.624	.047	0.000

	Omission Window	NSR	Lability	TRIALS TO HAB	S2 t18-S2 t16	s1 T18 - S1 T16	Input Column
37	0.00	6	Stabile	1	0.000	0.00	
38	.64	20	Labile	5	-.020	.13	
39	0.00	27	Labile	11	.940	.70	
40	0.00	5	Stabile	1	0.000	0.00	
41	0.00	13	Labile	5	-.700	.22	
42	0.00	7	Stabile	0	0.000	.14	
43	0.00	19	Labile	7	0.000	.02	
44	.32	9	Labile	5	0.000	0.00	
45	.63	34	Labile	16	.100	0.00	
46	.02	12	Labile	3	0.000	.22	
47	0.00	24	Labile	7	.020	.21	
48	0.00	4	Stabile	1	0.000	0.00	
49	.30	24	Labile	8	-.070	.12	
50	0.00	12	Labile	6	.070	.08	
51	0.00	3	Stabile	1	0.000	.02	
52	.10	1	Stabile	16	.030	.42	
53	0.00	3	Stabile	1	0.000	0.00	
54	0.00	11	Labile	2	0.000	0.00	
55	.34	12	Labile	16	.340	.02	
56	.02	35	Labile	16	-.030	.42	
57	0.00	18	Labile	6	-.060	-.03	
58	0.00	0	Stabile	7	0.000	.07	
59	0.00	6	Stabile	4	.030	0.00	
60	0.00	14	Labile	2	0.000	0.00	
61	0.00	15	Labile	2	0.000	0.00	
62	0.00	2	Stabile	9	0.000	0.00	
63	0.00	10	Labile	1	0.000	0.00	
64	0.00	0	Stabile	1	0.000	0.00	
65	0.00	8	Stabile	1	.020	0.00	
66	.50	13	Labile	8	0.000	.50	
67	0.00	4	Stabile	3	.040	.04	
68	0.00	12	Labile	5	.360	0.00	
69	0.00	3	Stabile	1	0.000	0.00	
70	0.00	5	Stabile	8	0.000	0.00	
71	0.00	0	Stabile	1	0.000	0.00	
72	.14	3	Stabile	0	-.240	-.21	

APPENDIX D

Raw data for Experiments Three and Four, and for the neural network simulations are too voluminous to include here. They are available from the author.

APPENDIX E

Listing of TurboPASCAL program used for simulations presented in this thesis.

A copy of this program on floppy disk is available from the author.

```pascal
program BDnet;

uses graph,dos,crt;

Const
    Inputnodes=20;
    outputnodes=20;
    bgcolor=7;
    maxinput=1;
    minInput=0;
    learnrate=0.7;
    spread=0;

    PrimeLearn=0.002;
    eligibility=10;

    maxoutput=1;
    minoutput=0;

    MaxDecay=5;

    MinWeight=0.000000001;

    MaxInitWeight=0.10;

    S1=10;
    s2=1;

    MaxIterations=10000;

    {tempo Net constants}
    Threshold=-10;      {mV}
    PostFire=-100;       {mV, value of potential after firing}
    minrate=9;          {mV/s, i.e. period of 10s}
    maxrate=30;          {mV/s, i.e. period of 3s}
    step=1;              {s, steps are in 1 second increments}
    adapstep=15;
    MaxOutCount=3;

Type
    InputRange= 1..Inputnodes;
    InputVectortype= array [InputRange] of real;
    Weightstype=array[1..Outputnodes,1..Inputnodes]of real;
    OutputVectorType=array [1..Outputnodes] of real;
    point=array [1..2] of integer;
    Inputfile=file of Inputvectortype;
    InputBufferType=Array[1..3] of InputVectortype;
    PotentialType=array [1..Outputnodes] of real;
    RateType=array [1..Outputnodes] of real;
    ElapsedTimeType=array [1..OutputNodes] of integer;
    Periodtype=array[1..Outputnodes]of integer;
    SOAType=Array[1..InputNodes]of Integer;
    OffsetType=Array[1..InputNodes,1..Inputnodes] of integer;
    PrimeWeightsType=Array[1..InputNodes,1..Inputnodes] of real;
    PointType= array [1..2] of integer;
    DisplayBufferType= array [1..Maxiterations] of pointType;
```

```
var
    i,j,k,m,openerror,subjectstorun,subjectstodo,rulechoice,rulechosen: integer;
    input,PrimeInput:inputVectortype;
    Maxresp,totalresp,avresp:real;
    Nresp:integer;
    f,g:text;
    stopSumm:integer;
    ss,inputfilename,outputfilename,markbit:string;
    weights:weightstype;
    output,expected,Tempooutput:OutputVectorType;
    epoch:integer;
    totaladjustment,distance:real;
    ch:char;
    lastadjust:point;
    LastStim:point;
    tabuffer:displaybuffertype;
    TimeSinceLastFire:SOAType;
    PrimeOffsets:OffsetType;
    LastStimSize:InputVectorType;
    PrimeWeights:PrimeWeightsType;
    Potential:PotentialType;
    Rate,inrate,Inphase:Ratetype;
    tout,tin:ElapsedTimeType;
    inperiod,outperiod: PeriodType;
```

```
(********************** Utility Functions **************************)
Function max(x,y:real):real;
{returns the larger of two numbers}

begin
    if x>=y then begin
        max:=x;
    end else begin
        max:=y;
    end;
end; {max}
```

```
Function min(x,y:real):real;
{returns the smaller of two numbers}

begin
    if x<=y then begin
        min:=x;
    end else begin
        min:=y;
    end;
end; {min}
```

```
(********************** Graphics Setup Procedures *****************)
procedure VGAInit;
```

{This procedure initialises the graphics to VGA 16 colour mode and calls initgraph
the parameter "drivername" must give the name and location of the graphdriver}
{The procedure also draws the screen which will be used to present the simulations}

```pascal
var
  i:integer;



Procedure VGASetup(DriverName:String);
{from Turbo Pascal library reference manual (Register BGI entry)}
var
  ErrorCode,i:Integer;
  DRIVERF:file;
  Driver, Mode:Integer;
  Driverp:pointer;

begin
{open, driver file, read into memory, register it}

    assign(driverf, DriverName);
    reset(driverf,1);
    getmem(driverp,filesize(driverf));
    blockread(driverf,driverp^,filesize(driverf));
    if registerbgidriver(driverp) < 0 then begin
       writeln('Error registering driver: ', grapherrormsg(graphresult));
       readln;
       halt(1);
    end;

    {init graphics}
    driver :=VGA;
    mode:=VGAhi;
    initgraph(driver,mode,'');
end; {of VGASetup}


(****************** Main Section of VGA init ********************)

begin {Program}
    VGAsetup('F:\home\bdaniels\egavga.bgi');
    setbkcolor(bgcolor);
    cleardevice;
    setcolor(9);
    setLineStyle(solidln,0,thickwidth);
    line(0,getmaxy div 2, getmaxx, getmaxy div 2);
    line(getmaxx div 2, getmaxy div 2, getmaxx div 2, getmaxy);
    setfillstyle(solidfill,13);
    bar3d(0,0,640,30,3,topoff);
    setcolor(15);
    settextjustify(centertext,centertext);
    outtextXY(getmaxx div 2, 15, 'Brett''s Nets Habituation Model Simulation Program');
end; {of procedure VGAInit}

(*********************** Data input procedures *********************)

procedure ReadASecond(var f:text; var Input:InputVectorType; var markbit:string);
{reads one line from a specified text file and puts it into the array
Input}

var
  i:integer;
```

```pascal
      temp:char;

begin
    markbit:='frog';
    FOR I:= 1 to inputnodes do begin
        read(f,input[i]);
    end;
    readln(f,markbit);

end; {procedure ReadASecond}




Procedure OpenInputFile(var f:text; var inputfilename:string);
{Opens for reading the text file containing the input for a simulation,
will continue to ask until the name of an existing file is entered.}

var
   openerror:integer;

begin
    write('Please type in the name of the file containing the data: ');
    readln(Inputfilename);
    assign (f,inputfilename);
    {$I-}
    Reset(f);
    {$I+}
    {writeln('IOResult= ',ioresult,'  (0 indicates no I/O error)');}
    openerror:=ioresult;
    while openerror <> 0 do begin
        writeln('No input file found with that name.');
        write('Please type another: ');
        readln(Inputfilename);
        assign (f,inputfilename);
        {$I-}
        Reset(f);
        {$I+}
        openerror:=ioresult;
    end;
end; {of OpenInputFile}
```

(********************* Graphic Output Procedures *****************)

```pascal
Procedure DrawInputvector(var input:InputVectorType);
{ This procedure draws a representation of the input vector in use at the current
time}

const
    Inputscale=40;

var
   i:integer;
   lastInput:inputvectortype;

begin
    setcolor(4);
    settextjustify(Lefttext,centertext);
    OuttextXY(10,250,'Input:');
```

```pascal
        OuttextXy(302,310-InputScale*maxinput,'1');
        outtextxy(302,310,'0');
        setLineStyle(solidln,0,normwidth);

        {hide last input}

        setfillstyle(emptyfill,bgcolor);
        bar(40,310,300,310-InputScale*maxinput);

        {draw new input}
        setcolor(1);
        setfillstyle(solidfill,1);

        For i := 1 to Inputnodes do begin
            if (input[i] <= maxinput) and (input[i] >= mininput) then begin
            bar(40+(i-1)*(260 div Inputnodes),310,
                40+(i)*(260 div Inputnodes) ,trunc(310-(InputScale*input[i])));
            end;
        end;

        lastinput:=input;

end; {of DrawInputVector}

Procedure DrawOutputvector(var output:outputVectorType);
{ This procedure draws a representation of the output vector in use at the current
time}

const
    outputscale=40;

var
   i:integer;

begin
    setcolor(4);
    settextjustify(Lefttext,centertext);
    OuttextXY(10,320,'Output:');
    OuttextXy(302,370-OutputScale*maxinput,'1');
    outtextxy(302,370,'0');
    setLineStyle(solidln,0,normwidth);

    {hide last input}

    setfillstyle(emptyfill,bgcolor);
    bar(40,370,300,370-outputScale*maxinput);

    {draw new output}
    setcolor(3);
    setfillstyle(solidfill,3);

    For i := 1 to outputnodes do begin
    bar(40+(i-1)*(260 div outputnodes),370,
        40+(i)*(260 div outputnodes) ,trunc(370-(outputScale*output[i])));

    end;
```

end; {of DrawoutputVector}

Procedure DrawEpoch(epochs,subjectstodo,subjectstorun:integer);

var
  s,ss,sss:string;

begin
    setfillstyle(emptyfill,bgcolor);
    bar(340,270,640,245);
    setcolor(4);
    settextjustify(Lefttext,centertext);
    str(epochs,s);
    str(subjectstodo,ss);
    str(subjectstorun,sss);
    s:='Epoch Number '+ s;
    ss:='Replication '+ss+' of '+sss;
    OuttextXY(340,260,ss);
    outtextxy(340,280,s);

end;

Procedure DrawTotalAdjustment(ta:real);

var
  s:string;

begin
    setfillstyle(emptyfill,bgcolor);
    bar(340,305,640,275);

    setcolor(4);
    settextjustify(Lefttext,centertext);
    str(ta:8:3,s);
    s:='Distance = '+ s;
    OuttextXY(340,300,s);

end;

Procedure DrawInfile(fn,ofn:string; rulechoice:integer);

begin
    setfillstyle(emptyfill,bgcolor);
    bar(340,340,640,305);
    setcolor(4);
    settextjustify(Lefttext,centertext);
    fn:='Input File Name: '+ fn;
    OuttextXY(340,320,fn);
    setfillstyle(emptyfill,bgcolor);
    bar(340,360,640,325);
    setcolor(4);
    settextjustify(Lefttext,centertext);
    fn:='Output File Name: '+ ofn ;
    OuttextXY(340,340,fn);
    setfillstyle(emptyfill,bgcolor);
    bar(340,380,640,345);
    setcolor(4);
    settextjustify(Lefttext,centertext);

```
        case rulechoice of
            1: fn:='Learning Rule: Delta Rule';
            2: fn:='Learning Rule: Tempo Rule';
            3: fn:='Learning Rule: Deltempo';
            4: fn:='Learning Rule: Priming';
            5: fn:='Learning Rule: PrimeDelta';
        end;
        OuttextXY(340,360,fn);
end;

Procedure PlotTotalAdjustment(var ta:real; var lastAdjust:point; epoch:integer;
var tabuffer:displaybuffertype);
{plots a graph of total weight adjustment against epoch}

const
    tascaley=80;
    tascalex=1;
    rtmargin=600;
    lftmargin=160;
    topmargin=150;
    bottommargin=200;
var
  oldcolor:word;
  s:string;
  epochsToPlot,EpochsToCrop,i:integer;

begin
    tabuffer[epoch,1]:=lastadjust[1];
    tabuffer[epoch,2]:=lastadjust[2];
    lastadjust[1]:= (tascalex*epoch)+lftmargin;
    lastadjust[2]:=bottommargin-trunc(ta*tascaley);
    OuttextXY(20,200,'Distance');
    SetLineStyle(Solidln,0,Normwidth);
    oldcolor:=getcolor;
    setfillstyle(1,bgcolor);
    bar(rtmargin+100,bottommargin+15,lftmargin-5,topmargin);
    setcolor(12);
    epochstoplot:=(rtmargin-lftmargin) div tascalex;
    if epochstoplot>=epoch then begin
      epochstocrop:=1;
      end else begin
    epochstocrop:=epoch-epochstoplot;
    end; {of if}
    for i := epochstocrop to (epoch-1) do begin
      if i mod 50 = 0 then begin
        str(i,s);
        outtextxy(tabuffer[i,1]-(epochstocrop*tascalex),bottommargin+10,s);
      end;
      moveto(tabuffer[i,1]-(epochstocrop*tascalex),tabuffer[i,2]);
      lineto(tabuffer[i+1,1]-(epochstocrop*tascalex),tabuffer[i+1,2]);
    end;
    setcolor(oldcolor);
end; {PlotTotalAdjustment}



Procedure PlotInput(var input:inputVectortype; var epoch:integer; var LastStim:point);
{plots a graph of stimulus activity against epoch}
```

```
const
    inscaley=50;
    tascalex=1;
    lftmargin=160;
    rtmargin=600;
    topmargin=40;
    bottommargin=120;
var
  oldcolor:word;
  inbuffer:displaybuffertype;
  i,epochstoplot,epochstocrop:integer;
  s:string;


begin
    oldcolor:=getcolor;
    settextjustify(Lefttext,centertext);
    OuttextXY(20,90,'Stimulus 1');
    inbuffer[epoch,1]:=laststim[1];
    inbuffer[epoch,2]:=laststim[2];
    laststim[1]:= (tascalex*epoch)+lftmargin;
    laststim[2]:=120-trunc(input[s1]*inscaley);
    oldcolor:=getcolor;
    setfillstyle(1,bgcolor);
    bar(rtmargin+100,bottommargin+15,lftmargin-5,topmargin);
    setcolor(5);
    epochstoplot:=(rtmargin-lftmargin) div tascalex;
    if epochstoplot>=epoch then begin
      epochstocrop:=1;
    end else begin
      epochstocrop:=epoch-epochstoplot;
    end; {of if}
    for i := epochstocrop to (epoch-1) do begin
      if i mod 50 = 0 then begin
        str(i,s);
        outtextxy(inbuffer[i,1]-(epochstocrop*tascalex),bottommargin+10,s);
      end;
      moveto(inbuffer[i,1]-(epochstocrop*tascalex),inbuffer[i,2]);
      lineto(inbuffer[i+1,1]-(epochstocrop*tascalex),inbuffer[i+1,2]);
    end;
    setcolor(oldcolor);
end; {PlotTotalAdjustment}



Procedure DrawExpectedVector(var expected:outputVectorType);
{ This procedure draws a representation of the output vector in use at the current
time}

const
    outputscale=40;

var
  i:integer;

begin
    setcolor(4);
```

```
settextjustify(Lefttext,centertext);
OuttextXY(10,400,'Expected:');
OuttextXy(302,450-OutputScale*maxinput,'1');
outtextxy(302,450,'0');
setLineStyle(solidln,0,normwidth);

{hide last input}

setfillstyle(emptyfill,bgcolor);
bar(40,450,300,450-outputScale*maxinput);

{draw new output}
setcolor(3);
setfillstyle(solidfill,2);

For i := 1 to outputnodes do begin
     bar(40+(i-1)*(260 div outputnodes),450,
     40+(i)*(260 div outputnodes) ,trunc(450-(outputScale*expected[i])));

end;


end; {of DrawExpectedVector}

(******************* Delta Rule  Net Procedures  ********************)

Procedure InitialiseWeights( var weights:WeightsType);
{This procedure initialises the weights of the net to small random values}

var
  i,j:integer;

begin
   {set new random seed}
   randomize;

   for i := 1 to outputnodes do begin
      for j:= 1 to inputnodes do begin
         weights[i,j]:=0.0;
      end;
   end;

   for i := 1 to outputnodes do begin
      for j:= (i-spread) to (i+spread) do begin
         if i=j then begin
            weights[i,j]:=MaxInitWeight;
         end else begin
            weights[i,j]:=(1/sqrt(sqr(j-i)))*MaxInitWeight;
            weights[i,j]:=weights[i,j]+(maxinitWeight/(random(5)+1));
         end;
      end;
   end;

end; {of Initialise weights}


Procedure TypeWeights(weights:weightstype);
{types the current weights on the screen}
```

```pascal
var
  i,j:integer;

begin
  for i := 1 to outputnodes do begin
    for j:= 1 to inputnodes do begin
      write( weights[i,j]:3:2,' ');
    end;
    writeln;
  end;

end;  {of TypeWeights}

Procedure TypePrimeWeights(weights:Primeweightstype);
{types the current weights on the screen}
var
  i,j:integer;

begin
  for i := 1 to outputnodes do begin
    for j:= 1 to inputnodes do begin
      write( {weights[i,j]:2:1,' ',} PrimeOffsets[i,j],' ');

    end;
    writeln;
  end;

end;  {of TypeWeights}

Procedure CalculateOutput(weights:Weightstype; Input:inputVectorType;
        var output:OutputVectorType);

{ calculates outputs using tanh function}

var
  i,j:integer;
  x:real;

begin
  for i:= 1 to outputNodes do begin
    output[i]:=0;  {initialise output for each node}
    for j := 1 to inputnodes do begin
      output[i]:=output[i]+weights[i,j]*input[j];
    end;
    output[i]:=1/(1+exp(-1*output[i]));
    x:=output[i];
    x:=(exp(x)-exp(x*-1))/(exp(x)+exp(x*-1));
    output[i]:=x;

  end;
end;  {calculate output}


Procedure CalculateWeightedOutput(weights:Weightstype; Input:inputVectorType;
        var output:OutputVectorType);

{ calculates outputs using tanh function, on weights and input}
```

```pascal
var
  i,j:integer;
  x:real;  ·

begin
    for i:= 1 to outputNodes do begin
        output[i]:=0;  {initialise output for each node}
        for j := 1 to inputnodes do begin
           output[i]:=output[i]+weights[i,j]*input[j];
        end;
        x:=output[i];
        x:=(exp(x)-exp(x*-1))/(exp(x)+exp(x*-1));
        output[i]:=x;

    end;
end;  {calculate weighted output}




procedure Typeoutput(output:Outputvectortype);

var
  i,j:integer;

begin
    writeln('Output Vector:');
    for i := 1 to outputnodes do begin
        write( output[i]:3:2,' ');
    end;
    writeln;

end;  {of Typeoutput}

procedure TypeInput(input:inputvectortype);

var
  i,j:integer;

begin

    writeln('Input Vector:');
    for i := 1 to Inputnodes do begin
        write( Input[i]:3:2,' ');
    end;
    writeln;
    writeln;

end;  {of Typeinput}

procedure adjustweights(var weights:weightstype; input:inputVectorType;
        Output:outputVectorType; var totaladjustment:real; rule:string);

{uses the Widrow Hoff equations (delta rule) from page 218 of Wasserman (1989) to
increment weights in net}

var
  i,j:integer;
  error:real;
```

```pascal
    dlearnrate:real;

begin
    totaladjustment:=0;
    dlearnrate:=learnrate;
    for i := 1 to inputNodes do begin
        error:= input[i]-output[i];
        for j:= (i-spread) to (i+spread) do begin
            weights[j,i] :=weights[j,i] + dlearnrate*input[i]*error;
        end;
    end;
end; {adjustWeights}


Procedure Checkweight(var weight:real);
{checks that a given weight has not fallen below MinWeight, sets to MinWeight
if it has}


begin
    if weight <= Minweight then weight := minweight;
end;{Checkweight}


Procedure DecayWeights(var weights:Weightstype; var TotalAdjustment:real);
{decays all the weight values in the net by a random value}


var
    i,j:integer;
    oldweight,decay:real;


begin
    for i := 1 to inputNodes do begin
        for j:= (i-spread) to (i+spread) do begin
            oldweight:=weights[j,i];
            decay:=maxdecay/500;
            if weights[j,i] > 0 then weights[j,i]:=weights[j,i]-decay;
            if weights[j,i] < 0 then weights[j,i]:=weights[j,i]+decay;
        end;
    end;

end; {decayweights}



Procedure CalculateExpected(var weights:weightstype; var expected:outputVectorType);
{calculates the expected output as determined by the current input weights}


var
    i,j:integer;
    DummyInput:inputVectorType;


begin
    for i := 1 to inputNodes do begin
        DummyInput[i]:=1;
    end;
    CalculateOutput(weights,dummyInput,Expected);
end; {CalculateExpected}


(*********************** Tempo Net Procedures ********************)
Procedure InitTempo(var Potential:PotentialType; Var Rate:ratetype;
```

```
            var tout,tin: ElapsedTimeType; inperiod,outperiod:PeriodType);

{initialises the values of relevant Tempo net variables to reasonable values}

Var
  i,j:integer;

begin
    for i:= 1 to OutputNodes do begin
        Potential[i]:= Postfire + random(Threshold-postfire);
        rate[i]:= minrate+random(maxrate-minrate);
        Tin[i]:=1 ;
        Tout[i]:= 1;
        inperiod[i]:=1;
        outperiod[i]:=1;
    end;
end; {inittempo}


Procedure Tempo(input:inputVectortype; weights:weightstype;
        var TempoOutput:outputVectortype; epoch:integer;
        var Potential:PotentialType;
        var Rate,inrate,Inphase:Ratetype;
        var tout,tin:ElapsedTimeType;
        var inperiod,outperiod: PeriodType);

const
    Crate=0.35;      {learning rate for rate of potential rise}
    PhaseRate=0.35;  {Learning rate for phase}

var
  i,j:integer;
  denom:integer;
  tempinput:OutputVectortype;
  s,t,r,q,v:string;
  oldcolor:word;

begin

{if it is the first epoch initialise Tempo values}
    if epoch=1 then begin
        InitTempo(Potential,Rate,tout,tin,inperiod,outperiod);
    end;

{Input to the Tempo net is the output of thecalculated using weighted
outputs from from input to output layers. i.e. activity must pass along
these connections to reach output nodes which can then learn the frequency
of response}
    CalculateWeightedOutput(weights,Input,Tempinput);

  For i:=1 to Outputnodes do begin
        tempinput[i]:=input[i];

        {Increment potential 1 timestep}
        potential[i]:=potential[i]+(rate[i]*step);


        {if potential is too low to fire and there is no input, simply
```

```
    increment relevant variables and make output[i] = 0}
    if (Potential[i] < Threshold) and (Tempinput[i]=0) then begin
        Tout[i]:= tout[i]+step;
        Tin[i]:= Tin[i]+step;
        TempoOutput[i]:=0;
    end;

    {if node fires (potential >threshold) but there is no input to the
    node then estimate period of output and increment rate to make it
    nearer to period of input}
    if (potential[i]>= threshold) and (Tempinput[i] = 0) then begin
        outperiod[i]:=tout[i];
        if tin[i]-tout[i]>0 then denom:=tin[i]-tout[i];
        if tin[i]-tout[i]<0 then denom:=tout[i]-tin[i];
        if denom = 0 then denom:= 1;
        TempoOutput[i]:=1/denom;
        Tin[i]:= tin[i]+step;
        Tout[i]:=1;
        potential[i]:=postfire;
        rate[i]:= rate[i]+((inrate[i]-rate[i])*Crate);
        if rate[i] < minrate then rate[i] := minrate;
        if rate[i] > maxrate then rate[i] := maxrate;
    end;

    {if there is an input to a node when it is not firing then estimate
    period of input, alter rate to change period of output,
    and also alter potential to alter phase of output}
    if (potential[i]<threshold) and (tempinput[i]<> 0) then begin
        inperiod[i]:=tin[i];
        TempoOutput[i]:=0;
        tout[i] := tout[i]+step;
        tin[i]:=0;
        inrate[i]:=(threshold-postfire+1)/(inperiod[i]+1);
        rate[i]:= rate[i]+((inrate[i]-rate[i])*Crate);
        if rate[i] < minrate then rate[i] := minrate;
        if rate[i] > maxrate then rate[i] := maxrate;
        Inphase[i]:=threshold-(rate[i]*(inperiod[i]+1));
        potential[i]:=potential[i]-((potential[i]-
inphase[i])*(phaserate*(1/sqrt(epoch/inperiod[i]))));
    end;

    if (potential[i]>= threshold) and (tempinput[i]<>0) then begin
        inperiod[i]:=tin[i];
        outperiod[i]:=tout[i];
        tout[i]:=1;
        tin[i]:=0;
        if outperiod[i]=0 then outperiod[i]:=1;
        if inperiod[i] = 0 then inperiod[i] :=1;
        if tin[i]-tout[i]>0 then denom:=tin[i]-tout[i];
        if tin[i]-tout[i]<0 then denom:=tout[i]-tin[i];
        if denom = 0 then denom:= 1;
        TempoOutput[i]:=1/denom;
        inrate[i]:=(threshold-postfire+1)/(inperiod[i]+1);
        potential[i]:=postfire;
    end;
  end;
end; {tempo}
```

```pascal
Procedure CalculateTempoOutput(weights:Weightstype;
        Input:inputVectorType; var output, tempoOutput:OutputVectorType);

{ calculates outputs using tanh function, on weights and input}

var
   i,j:integer;
   x:real;

begin
    for i:= 1 to outputNodes do begin
       output[i]:=0;  {initialise output for each node}
       for j := 1 to inputnodes do begin
          output[i]:=output[i]+(weights[i,j]*input[j])+ tempooutput[i];
       end;
       x:=output[i];
       x:=(exp(x)-exp(x*-1))/(exp(x)+exp(x*-1));
       output[i]:=x;

    end;
end;  {calculate tempo output}


(*********************** Priming Procedures **************************)
Procedure InitPrime(var timeSinceLastfire:SOAType;
        var LastStimsize:InputVectorType; var Primeweights:PrimeWeightsType;
        var PrimeOffsets:OffsetType);

var
   i,j:integer;

begin
    for i:= 1 to inputnodes do begin
       TimeSinceLastfire[i]:=Maxiterations div 3;
       LastStimsize[i]:=0;
       for j:= 1 to inputnodes do begin
          PrimeWeights[i,j]:=0.0;
          PrimeOffsets[i,j]:=Maxiterations;
       end;
    end;
end;  {initPrime}


Procedure Prime(var epoch:integer; var input,PrimeInput:inputvectortype;
        var TimeSinceLastFire:SOAType; var PrimeOffsets:OffsetType;
     var LastStimSize:InputVectorType; var PrimeWeights:PrimeWeightsType);


var
   i,j,k,l,m,o,p:integer;
   oldweight,decay:real;


begin
    if epoch=1 then InitPrime(TimeSinceLastFire,LastStimSize,PrimeWeights,PrimeOffsets);
    for k:= 1 to inputnodes do begin
       for l:=1 to inputnodes do begin
```

```pascal
        if (input[k] <> 0) and (timeSinceLastFire[l] <= eligibility) then begin
            PrimeWeights[k,l]:=PrimeWeights[k,l]+PrimeLearn;
            PrimeOffsets[k,l]:=TimeSinceLastFire[l];
        end;

        {decay the priming weights}

        Primeweights[k,l]:=Primeweights[k,l]*(1-(PrimeLearn/100));
    end;
  end;

  for o:= 1 to inputnodes do begin
    primeInput[o]:=0;
    for p:=1 to inputnodes do begin
        if (timeSinceLastFire[p]=PrimeOffsets[o,p]) then begin
            PrimeInput[o]:=PrimeInput[o]+(laststimsize[p]*PrimeWeights[o,p]);
            if input[o] = 0 then begin
                Primeweights[o,p]:=max(PrimeWeights[o,p]-(PrimeLearn),0.00);
                PrimeOffsets[o,p]:=PrimeOffsets[o,p];
            end;
        end;
    end;
  end;

  for m:= 1 to inputnodes do begin
    TimeSinceLastFire[m]:=TimeSinceLastFire[m]+1;
    if input[m]<>0 then begin
        LastStimSize[m]:=Input[m];
    end;                            {
    if (primeinput[m]<>0)then begin
        timesincelastfire[m]:=1;
    end;                            }
    if (input[m]<>0) then begin
        timesincelastfire[m]:=1;
    end;
  end;


end;



Procedure CalculatePrimeOutput(weights:Weightstype; PrimeWeights:PrimeWeightsType;
        Input,PrimeInput:inputVectorType; var output:OutputVectorType);

{ calculates outputs using tanh function, on weights and input}

var
  i,j:integer;
  x:real;

begin
  for i:= 1 to outputNodes do begin
    output[i]:=0;  {initialise output for each node}
    for j := 1 to inputnodes do begin
        output[i]:=output[i]+(weights[i,j]*input[j])+primeinput[i];
    end;
    x:=output[i];
    x:=(exp(x)-exp(x*-1))/(exp(x)+exp(x*-1));
```

```
        output[i]:=x;


     end;
  end;  {calculate weighted output}




(*********************** Similarity Measures ***********************)
Procedure DoCorrelation(input:inputvectortype; output:outputvectortype;
                           var correlation:real);
{Calculates the similarity between the input and output vectors, defined
as 1-correlation(input,output).  Formula from Welkowitz.Ewen and Cohen, p.179}

var
   i,j,n:integer;
   sigmaxy,sigmax,sigmay,sigmax2,sigmay2,upperline,lowerline:real;

begin
     n:=OutputNodes;
     sigmax:=0;
     sigmay:=0;
     sigmax2:=0;
     sigmay2:=0;
     sigmaxy:=0;

     {calculate sigmax,sigmay,sigmaxy}

     for i:= 1 to Outputnodes do begin
        output[i]:=output[i]+1;
        input[i]:=input[i]+1;
        sigmax:=sigmax+input[i];
        sigmay:=sigmay+output[i];
        sigmax2:=sigmax2+sqr(input[i]);
        sigmay2:=sigmay2+sqr(output[i]);
        sigmaxy:=sigmaxy+(input[i]*output[i]);
     end;

     {calculate  formula}

     upperline:=(n*sigmaxy)-(sigmax*sigmay);
     lowerline:=sqrt((n*sqr(sigmax2)-sqr(sigmax))*(n*sqr(sigmay2)-sqr(sigmay)));

     correlation:=upperline/lowerline;

     correlation:=1-correlation;
end;

Procedure DoDistance(input:inputvectortype; output:outputvectortype;
        var Distance:real);
{Calculates the Euclidean distance between the input and output
vectors}

var
   i:integer;
```

```pascal
begin
    distance:=0;
    for i := 1 to Outputnodes do begin
        distance:=distance +sqr(input[i]-output[i]);
    end;
    distance:= sqrt(distance) /sqrt(Outputnodes);  {Outputnodes is maximum value for distance}
end; {doDistance}



Procedure WriteDistance(var g:text; epoch:integer; distance:real;
        markbit,rule:string);

begin
    if (markbit<>'') and (markbit<>' ') then begin
        write(g, distance:4:4,markbit,' ',rule,' ');
    end;
end;

Procedure InitSummStats(Var maxresp,totalresp,avresp:real; var nresp:integer);

begin
    Maxresp:=0;
    Totalresp:=0;
    Avresp:=0;
    Nresp:=0;
end;

Procedure CalculateSummStats(markbit:string; var stopSumm:integer; distance:real;Var
maxresp,totalresp,
        avresp:real; var nresp:integer);
begin
    if (distance > 0) and (stopSumm=0) then begin
        maxresp:=max(distance,maxresp);
        totalresp:=totalresp+distance;
        nresp:=nresp+1;
        avresp:=totalresp/nresp;
    end;
    if markbit = ' last' then stopSumm:=20;

end;

Procedure WriteSummStats(var g:text; maxresp,totalresp,avresp:real;
                    nresp:integer);
begin
    write(g,'MAXRESP= ',MAXRESP:6:3,' TOTRESP= ',TOTALRESP:6:3,
    ' AVRESP= ',AVRESP:6:3,' NRESP= ',NRESP);
end;

(************************* Main Program *************************)


begin


    OpenInputFile(f,inputfilename);
    write('Type in name of Outputfile: ');
    readln(outputfilename);
```

```pascal
write('Enter number of simulations(subjects): ');
readln(subjectstorun);
writeln('Choose Learning Rule: 1)Delta 2)Tempo 3)Deltempo 4)Priming 5)PrimeDelta
6)All: ');
read(rulechosen);
outputfilename:=outputfilename+'.OUT';
Assign(G,outputfilename);
Rewrite(g);
VGAInit;
rulechoice:=rulechosen;
if rulechoice = 6 then begin
  rulechosen:=1;
end;
subjectstodo:=0;
while (subjectstodo <= subjectstorun) do begin
  Randomize;
  DrawInfile(inputfilename,OUTPUTFILENAME,rulechosen);
  initialiseweights(weights);
  InitSummStats(Maxresp,TotalResp,Avresp,Nresp);
  stopSumm:=0;
  lastadjust[1]:=160;
  lastadjust[2]:=200;
  LastStim[1]:=160;
  LastStim[2]:=120;
  readasecond(f,input,markbit);
  totaladjustment:=0;
  epoch:=1;
  subjectstodo:=subjectstodo+1;
  write(g,'Input:',inputfilename,' Simulation ',subjectstodo,' ');

  while not eof(f) and (rulechosen <> 6) do begin
    readasecond(f,input,markbit);
    DrawEpoch(epoch, subjectstodo,subjectstorun);
    Case Rulechosen of
    1:begin   {Delta rule chosen}
       calculateweightedoutput( weights,input,output);
       adjustweights(weights,input,output,totaladjustment,'delta');
       DrawInputVector(input);
       DrawOutputVector(output);                      _
       Dodistance(input,Output,Distance);
       CalculateExpected(weights,expected);
       drawExpectedVector(expected);
       decayweights(weights,totaladjustment);
       DrawTotalAdjustment(Distance);
       PlotTotalAdjustment(Distance,lastAdjust,epoch,tabuffer);
       plotinput(input,epoch,LastStim);
       writedistance(g,epoch,distance,markbit,'Delta');
       CalculateSummStats(markbit,stopsumm,distance,maxresp,totalresp,avresp,nresp);
       epoch:=epoch+1;
    end;

    2:begin   {Tempo Rule Chosen}
       Tempo(input,weights,Tempooutput,epoch,Potential,Rate,inrate,
       Inphase,tout,tin,inperiod,outperiod);
       calculateTempoOutput(weights,input,output,TempoOutput);
       Dodistance(input,Output,Distance);
       DrawInputVector(input);
       DrawOutputVector(output);
```

```
          DrawTotalAdjustment(Distance);
          PlotTotalAdjustment(Distance,lastAdjust,epoch,tabuffer);
          plotinput(input,epoch,LastStim);
          writedistance(g,epoch,distance,markbit,'Tempo');
          CalculateSummStats(markbit,stopsumm,distance,maxresp,totalresp,avresp,nresp);
          epoch:=epoch+1;
end;


  3:begin    {Deltempo rule chosen}
          Tempo(input,weights,Tempooutput,epoch,Potential,Rate,inrate,
          Inphase,tout,tin,inperiod,outperiod);
          calculateTempooutput( weights,input,output,TempoOutput);
          DrawInputVector(input);
          DrawOutputVector(output);
          doDistance(input,output,distance);
          { calculateweightedoutput(weights,input,output); }
          adjustweights(weights,input,output,totaladjustment,'Deltempo');
          decayweights(weights,totaladjustment);
          DrawTotalAdjustment(Distance);
          PlotTotalAdjustment(Distance,lastAdjust,epoch,tabuffer);
          plotinput(input,epoch,LastStim);
          writedistance(g,epoch,distance,markbit,'Deltempo');
          CalculateSummStats(markbit,stopsumm,distance,maxresp,totalresp,avresp,nresp);
          epoch:=epoch+1;
       end;


  4:begin    {Priming rule chosen}
        Prime(epoch,input,Primeinput,TimeSinceLastFire,PrimeOffsets,
        LastStimSize,PrimeWeights);
        calculatePrimeoutput( weights,PrimeWeights,input,PrimeInput,output);
        Dodistance(input,Output,Distance);
        DrawInputVector(input);
        DrawOutputVector(output);
        CalculateExpected(weights,expected);
        drawExpectedVector(expected);
        DrawTotalAdjustment(Distance);
        PlotTotalAdjustment(Distance,lastAdjust,epoch,tabuffer);
        plotinput(input,epoch,LastStim);
        writedistance(g,epoch,distance,markbit,'Prime');
        CalculateSummStats(markbit,stopsumm,distance,maxresp,totalresp,avresp,nresp);
        epoch:=epoch+1;
        end;


   5:begin    {Prime Delta rule chosen}
        Prime(epoch,input,PrimeInput,TimeSinceLastFire,PrimeOffsets,
        LastStimSize,PrimeWeights);
        calculatePrimeoutput( weights,Primeweights,input,primeinput,output);
        adjustweights(weights,input,output,totaladjustment,'Primedelta');
        DrawInputVector(input);
        DrawOutputVector(output);
        Dodistance(input,Output,Distance);
        CalculateExpected(weights,expected);
        decayweights(weights,totaladjustment); {decay delta weights}
        drawExpectedVector(expected);
        DrawTotalAdjustment(Distance);
        PlotTotalAdjustment(Distance,lastAdjust,epoch,tabuffer);
        plotinput(input,epoch,LastStim);
        writedistance(g,epoch,distance,markbit,'Primedelta');
```

```pascal
          CalculateSummStats(markbit,stopsumm,distance,maxresp,totalresp,avresp,nresp);
          epoch:=epoch+1;
          end;
        end; {Case statement}

      if (rulechoice = 6) and (rulechosen=5) and (subjectstodo=subjectstorun) then begin
        subjectstodo:=subjectstorun+1;
      end;


      if KeyPressed then begin
        ch:=readkey;
        if ch='a' then begin
          close(f);
          close(g);
          closegraph;
          typePrimeweights(primeweights);
          halt;
        end; {if ch= 'a'}
      end; {if keypressed}
    end; { while not eof(f)}

    WriteSummStats(g,maxresp,totalresp,avresp,nresp);
    writeln(G);
    reset(f); {put pointer at start of input file}

    if (subjectstodo=subjectstorun) and (rulechoice = 6) then begin
      subjectstodo:=0;
      writeln(g);
      rulechosen:=ruleChosen+1;
    end;


  end; {while subjects to run}


  write(chr(7));
  readln;
  close(f);
  close(g);
  closegraph;

end.
```