Attention and the habituation of human brain potentials

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ABSTRACT

The lability of the late slow biphasic deflection (i.e. N1-P2) of the vertex EP is examined in relation to selective attention and repetitive stimulation in both the visual and auditory modalities. Amplitude effects are of primary interest and a capacity theory of attention is applied, with some neurophysiological justification, throughout the work in order to account for such effects. A range of data analytic techniques relevant to EP research are also evaluated and, in particular, a correlation method for determining the amplitude and latency of individual EPs is described and assessed.

Unlike the auditory modality, the amplitude of the N1 component of the visual EP was found to remain invariant with respect to selective attention, although the subsequent P2 component was enhanced to stimuli on the attended channel. The N1 correlate of auditory selective attention was shown to be a reflection of the greater 'mental work' associated with attended stimuli.

Various aspects of the fast habituation of the visual EP were explored, including its interaction with slow habituation (using a single trial analysis) and the effect of presentation rate. The role of pupillary mechanisms was also evaluated.

The generic term 'fast response decrement' (FRD), which subsumes both the fast habituation and the temporal recovery of the late componentry of the EP, was introduced. Using paradigms based on pairs of stimuli, the mechanisms of both the visual and auditory FRD were investigated. The visual FRD was not affected by dichoptic

presentation and exhibited extensive stimulus generalisation. A refractoriness mechanism for the FRD was finally rejected in favour of a psychological one which represented a marriage of Sokolov's concept of a neuronal model with a capacity theory of attention. Using a novel technique, in which an oscilloscope 'clock' enabled experimental control over temporal uncertainty, it was demonstrated that the inadequacy of the neuronal model in its temporal aspects was the key variable underpinning the FRD, although in the auditory modality a second variable, designated 'firstness', also appeared to be important.

CONTENTS

CHAPTER ONE

INTRODUCTION

The transient electrical response of the human brain to an abrupt change in the sensory array can be extracted from the "muffled polyneural roar" (Rosenblith, 1962, p.4) of the EEG by the technique of signal averaging. The resulting amplitude-versus-time plot of the evoked potential (EP) represents practical access to the neural activity of the intact brain. The opportunity to excavate the neural substrata of human information processing is irresistable and EP studies have proliferated. Much of the research is "undeniably sloppy" (Regan, 1972, p.XIII) where the full range of the inquiry, extending across the "anatomical, biophysical, neurophysiological, statistical and psychological" (Vaughan, 1969, p.75) domains, has not been adequately embraced. Although the present work is no less imperfect, there remains an earnest optimism that EP techniques can afford valuable insights into brain-behaviour relationships.

In accord with Vaughan's prescription for an adequate neuropsychology, the first section of this opening chapter discusses the mechanisms underpinning observed surface potentials with the objective of providing some physiologically respectable basis for interpreting them. Despite the somewhat dogmatic style, the discussion is not intended to be a definitive account but is rather to be regarded as one author's determined attempt, in response to Vaughan's philosophy, to distil from a vast and complex literature an understanding of the EP which can subsequently be translated into the terminology of human information processing and yet which retains some foundation in anatomy, biophysics and neurophysiology.

The second and third sections of the chapter constitute selective reviews of the literature on the EP amplitude effects associated with selective attention and stimulus repetition. The physiological concepts developed in section one are further elaborated and integrated with this material and the chapter finally closes with a brief synopsis of the thesis research programme.

Mechanisms of Scalp-recorded Evoked Potentials

The evoked potential can reasonably be interpreted as representing the algebraic sum at the recording electrode of the extracellular field potentials associated with the various neural assemblies activated by the stimulus. Although quantitative methods do exist for the computation of these potentials, their treatment is beyond the terms of reference of this thesis and a less rigorous exposition is substituted.

Following Thompson (1967, p.59), the tissue of the brain is thought of as a volume-conducting pool of saline containing many sources (origins of current flow) and their associated sinks (termini of current flow). The potential recorded by a pair of gross electrodes reflects their relative locations within the current fields generated by the source-sinks. Consider, for example, the potential recorded from a vertex electrode in response to auditory stimulation to the ear ipsilateral to a 'referential' mastoid placement. The sound waves impinging upon the tympanic membrane ultimately distort the basilar membrane and the attached hair cells in the cochlea. A generator potential develops that in turn activates the auditory nerve fibres. The depolarisation of the hair cells represents a net flow of current into the region of the reference electrode and away from the

more distant scalp location: a short latency positive deflection is thus predicted as the first component of the vertex auditory EP. Jewett and Williston (1971) indeed obtained such a wave at a latency of 1.5 msec, and Picton, Hillyard, Kraus and Galambos (1974) confirm its scalp distribution to be consistent with a generator in the inner ear.

The central problem in the study of evoked field potentials is the reduction of the simultaneous activation of a population, or 'assembly', of cells to that of a single source-sink element, thus allowing an approximate evaluation of the overall average activity of the assembly. In their invaluable simplification of Lorente de No's analysis, Hubbard, Llinas and Quastel (1969) describe the three elementary types of current fields for cell assemblies. Of the three the so-called open field is the most pertinent to this discussion. Such a field designates a cell arrangement in which all the somas are accumulated in a common plane with their dendrites aggregated in a parallel layer (Fig. 1.1a). This type of assembly is substituted by an element having a soma and a single dendritic cylinder orientated in the same direction as the main axis of the dendritic tree (Fig. 1.1b). The pyramidal cells of the cortex, for instance, display this geometry, with their dendrites extending towards the surface. If such a pool is depolarised synchronously via axo-dendritic synapses, as pyramidal cells typically are (Eccles, 1973), the current field (generated between the dentritic sink and the somatic source) spreads throughout the volume of the conductor. A negative field potential is generated at all points on the dendritic side of the zero isopotential surface and a positive potential at all points on the somatic side. A

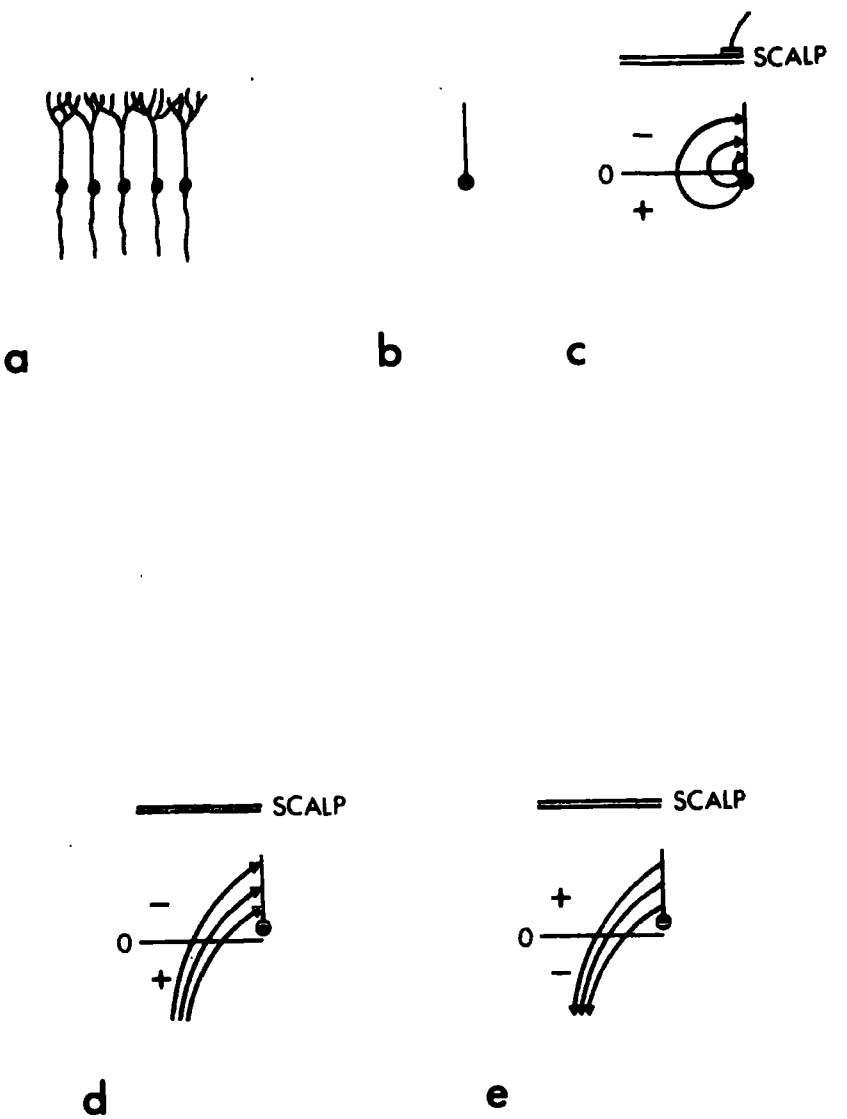

Figure 1.1

Current fields associated with a cell arrangement (a) in which somas and dendrites are arranged in parallel planes. The equivalent element of this arrangement is shown in b. Part c depicts the current field associated with the synchronous depolarisation of the dendritic layer. The current fields generated between the total somatodendritic membrance and the axon and deeper associated with depolarisation and hyperpolarisation of the somatodendritic membrane respectively, are shown in d and e.

scalp electrode located above a layer of so-activated pyramidal cells would thus read negative with respect to an indifferent reference (Fig. 1.1c).

Although this analysis is convincing in theory, the problems of unambiguously interpreting scalp records should not be underestimated: surface negativity may well be associated with the synchronous axo-dendritic activation of pyramidal cells but other mechanisms are theoretically possible. Recurrent inhibitory collaterals, for instance, predominantly synapse close to the somas of pyramidal cells (Eccles, 1973) and would thus also generate a deep source, a superficial sink and ipso facto surface negativity. Thus the relationship of surface potentials to possible underlying neural activities is a one-to-many correspondence, and supplementary information must be recruited before a particular generator can be uniquely inferred.

Fortunately considerable empirical evidence is available to reinforce these speculations (for reviews see Mackay, 1969; and Regan, 1972), the work of Creutzfeldt's group being outstanding (Creutzfeldt, Watanabe and Lux, 1966; Creutzfeldt, Rosina, Ito and Probst, 1969). They point out that although the above dipole model of vertically oriented cortical pyramidal cells is a probable explanation of the fast transients of early evoked activity, the later slower potentials develop over time-periods considerably longer than the time needed for the post-synaptic potential (PSP) to depolarise the entire somadendritic membrane. Potential differences would exist too fleetingly to account for these slow waves, and they propose the current field between the total somadendritic membrane

and the axon as a more likely mechanism. An advantage of such a model is its limited repertoire: the somadendritic membrane is either depolarised or polarised, sink or source; the axon and deeper correspondingly source or sink; and any more superficial locations, including the scalp, negative or positive (Fig. 1.1d,e) respectively. If the model is correct then the polarity of surface slow waves unambiguously indexes the pervading balance of excitation and inhibition within the underlying population of pyramidal cells. Creutzfeldt et al's (1969) findings in the cat visual system support the model with slow surface positivity and negativity coinciding with inhibitory and excitatory PSPs respectively dominating the majority of cortical cells. Although their work has necessarily been limited to animals they tentatively suggest that the principal features of the human EP are due to similar mechanisms. The present research is almost exclusively concerned with the late slow components of the human EP, and Creutzfeldt's model is cautiously adopted.

Before concluding a further point must be considered. So far the distinction between scalp and intracranial recording has not been introduced; it has been assumed for convenience that the scalp record more or less faithfully reflects the underlying cortical potentials. However, as Regan (1972) indicates, the attenuating effect exerted by the cerebrospinal fluid, skull and other overlying material limits the ability of a scalp electrode to resolve localised intracranial sources, and therefore effectively increases the relative contribution to the scalp record of volume-conducted activity from more distant but larger generators. Although Vaughan (1969) in reviewing the literature on mechanisms, including his own

substantial contribution, attributes scalp EPs to localised sources within the primary projection areas, intra-cranial recording demonstrates that evoked activity is rapidly apparent throughout the association cortex and indeed appears even in the depths of the frontal cortex within 25 ms of auditory and tactile stimulation and within 30 ms of a visual stimulus, figures not so different from those for the earliest components of the corresponding responses in the specific cortex (Grey Walter, 1964). It is thus likely, given the above consideration of the relationship between scalp and epicortical recording, that even the evoked activity witnessed by a scalp electrode located above a primary receiving area will contain a substantial contribution from the surrounding non-specific cortex. The intimacy between the scalp record and the activity of the non-specific cortex is further corroborated by Grey Walter's subsequent observation in his 1964 paper that, whereas primary EPs do not habituate both non-specific and scalp responses are typically variable in this sense. It is concluded that human scalp recorded evoked potentials, in reflecting the summated electrical activity of a widespread area of cortex, probably predominantly reflect the activity of the pyramidal cells of the non-specific cortex, although the relative contribution of specific activity will presumably increase with the proximity of the electrode to the relevant primary area.

Eccles (1973, Chapter 6) discusses the essentially columnar organisation of the cerebral cortex in which the cortical neuropil is organised in long cylinders perpendicular to the surface of the cortex. Eccles estimates the cross-sectional area of these 'cortical columns'

to be no more than .2 mm and on this basis the total cortex will comprise no more than one million such columns. Of the 10,000 or so cells within each column the most salient are the few hundred pyramidal cells which Eccles indicates as providing the output of the column to other columns. For Eccles (and indeed many other authors including Mountcastle (1957), Hubel and Wiesel (1962) and Werner (1970) in particular relation to the specific sensory areas, and Walley and Weiden (1973), Shepherd (1974) and Szengothai (see Szengothai and Arbib, 1975) more generally) the cortical column is regarded as the basic unit of cortical functioning. Eccles envisages psychological processes, and perception in particular, as being subserved by the elaboration over relatively long time-courses (for a visual perceptual experience, for instance, Eccles indicates that as long as a fifth of a second may be necessary) of complex patterns of cortical column activity over the whole of the cortex. Thus to the extent that the late slow waves of the EP reflect the general aspects from relatively widespread areas of the cerebrum of the excitation and inhibition of the output neurones of cortical columns (i.e. pyramidal cells), these slow waves will provide an index of the brain activity underlying psychological processes. Indeed, the lability of these waves with respect to psychological variables is well-known, and in particular the sensitivity of the N1 component of the scalp EP to perceptual variables (Regan, 1972) is consistent with both its time-course and polarity which, in the present scheme, correspond to the elaboration of the initial pattern of activation of cortical columns that presumably represents the earliest, i.e. perceptual, stages of processing and possibly even the conscious perceptual experience itself.

Evoked Potentials and Selective Attention

Since Hernández-Peón's pioneer experiments of the mid 1950s research into the neural mechanisms of attention has pullulated and in the ensuing selective review only the most salient of the studies are included as the progress of the EP limb of the enquiry is followed.

The long programme of research of Hernandez-Peon and his group (reviewed in Hernández-Peón, 1966) generated considerable support for his notion that attention was subserved by a mechanism of afferent neuronal inhibition. He suggested that the transmission of afferent signals along the specific sensory pathways is regulated by the efferent inhibitory influence of a central station in the rostral brain stem. In a now classic experiment (Hernández-Peón, Scherrer and Jouvet, 1956) the effect of attention was demonstrated in the attenuation of click evoked potentials (recorded from the cochlear nucleus) that occurred when a cat was distracted by a mouse, the smell of fish or a forepaw shock. The significance of Hernández-Peón's work is further enhanced by the similarity of his physiological hypothesis of afferent neuronal inhibition with the cognitive filter theory of Broadbent (1958). The latter proposed a peripheral nervous system comprising a number of input channels that compete for a single central channel of limited capacity, an intermediate filter determining the input channel to be sampled. Broadbent considered the filter to block completely the unattended channels; however, Treisman (1960) suggests their attenuation as an alternative possibility.

Although at first sight Hernández-Peón's EP decrements do seem to support the hypothesis of afferent neuronal inhibition and evidence the

operation of Broadbent's and Treisman's filter, Worden (1966) in an extensive critique provides cogent methodological and theoretical grounds for scepticism. By delivering the clicks over headphones to his cats the acoustic input was held constant regardless of any movements of the animal, and with this factor controlled he was unable to replicate Hernández-Peón's result. Dismissing the latter's finding as an artifact of the sound field variations that occur when the animal turns his head towards the source of distraction, Worden proceeds with a discussion of the various assumptions that generally underpin most interpretations of EP amplitude changes. His scrutiny of the assumptions a) that larger EP amplitudes invariably reflect facilitated neural activities, and b) that facilitated neural activities indicate increased information handling, is of relevance and will be discussed briefly.

The first of these assumptions is untenable in his view for the simple reason that the potential registered by a gross electrode is the instantaneous sum of both positive and negative fields and that even vigorous neural activity, consisting of excitatory and inhibitory PSPs, could easily summate to a small total. Although Worden is not so explicit in his deliberations over the validity of the second assumption my own reservations are contained in the following consideration: if n input lines are available to the organism and all n respond whatever the stimulus, then the summated activity will always be larger than if the stimulus is uniquely specified by the firing of a particular subset of the lines, and yet the information handled is minimal by comparison. Although the details of the neural coding of information are admittedly largely unknown,

it is felt that EP amplitude may, in general, not prove to be any simple index of the representation of information in the nervous system.

Returning to the logic of the peripheral filter it could be argued, Worden's critique notwithstanding, that evoked potentials from probe stimulation in the same modality as the distracting (attention rousing) stimulus ought to be enhanced in amplitude. Thompson (1967) reviews a number of such studies involving both primary and association cortex EPs. The most common component measured was the initial surface positivity , and rather than increasing, it was virtually abolished when the animal attended to any type of stimulus. Although the significance of this amplitude change is difficult to interpret (see however footnote 1) for the reasons given above, one can certainly argue that its generality suggests some non-specific state change rather than the operation of an attentional mechanism selectively gating peripheral sensory input.

It should be emphasised that the EP components studied in all the work discussed so far represent the earliest evoked activity; later components are, as Thompson (p.150) comments, "much more labile and more related to behavioural conditions". Apart from a study by Picton and Hillyard (1974), which failed to demonstrate any effect of attention upon early components, human selective attention research has been exclusively concerned with these later components.

Fortunately the plethora of such studies (e.g. Davis, 1964; Spong, Haider and Lindsley, 1965; Donchin and Cohen, 1967; Eason, Harter and White, 1969; Smith, Donchin, Cohen and Starr, 1970) has been excellently reviewed and evaluated by Näätänen (1975). He comments (p.265)

that an experiment by Hillyard, Hink, Schwent and Picton (1973) is "the only one among those investigations reviewed so far providing no basis for the subject to predict the order of the relevant and irrelevant stimuli in which a selective enhancement of a relatively early component is reported". His argument, echoed by Karlin (1970), is that any task that allows the subject to differentially prepare himself for relevant events necessarily confounds non-specific state changes with the hypothesised operation of the selective filter, and that any observed attention related EP amplitude changes are thus not admissible as evidence for such a mechanism. He proceeds to quote two studies (Näätänen, 1967 experiment 2; Wilkinson and Ashby, 1974) in which the differential-preparation artifact was not present and in which selective EP enhancements were indeed not obtained. He finally notes, with some regret, that many human EP investigators have even lost sight of the basic question of sensory filtering that motivates the whole enquiry, and expresses the hope (p.183) that with this question kept in mind "experimental planning in EP studies on attention might become more adequate and at least some of the confusion in interpreting research results might be avoided".

In their classic study, Hillyard et al (1973) surpassed previous work simply by being the first to adapt and employ the established procedure of cognitive psychology for investigating auditory selective attention, namely the dichotic listening paradigm. Bleeps were delivered over headphones to the two ears according to a random sequence; the subject being required to detect certain target bleeps in the stimuli arriving at one of the ears, the 'attended' ear. The late negative wave (N1) of the

vertex EP, at a peak latency of between 80 and 110 ms, was found to be enhanced to stimuli on the attended channel. They adduce this finding as evidencing a 'stimulus set' mode of attention whereby sensory input is preferentially admitted from the attended channel for further perceptual analysis whilst inputs arriving over unattended channels are either blocked or attenuated. In short they consider their data as evidencing the operation of the sensory filter.

Before discussing this conclusion any further some consideration of the mechanisms of scalp recorded late potentials, and the vertex EP in particular, is necessary. In the previous section of the chapter, it was argued that the scalp record, and particularly the electrical activity recorded from an electrode located away from the relevant specific sensory area - such as at the vertex, primarily reflects the activity of the non-specific cortex. Thus Hillyard's above conclusion is not regarded as justifiable by this author. It is considered that the existence of a sensory filter can, by definition, only be deduced from changes in the specific sensory input to the cortex, and certainly not from the activation of the non-specific cortex, as embodied in N1, 60 to 90 ms later. Indeed Hillyard recognises the present argument himself in a subsequent paper (Picton and Hillyard, 1974) where it is stated that:

> The stability of the early components of the evoked response would seem to indicate that auditory information is analysed in the lemniscal or primary auditory system in much the same manner regardless of whether it is attended to or not. A secondary system, imperfectly defined but probably comprising the reticular formation, medial thalamus and association cortex, is involved when further evaluation of the significance of the auditory information is required.

They conclude that the evidence shows that human auditory attention is not mediated by a peripheral gating mechanism and revise the function of 'stimulus set' to a preferential streaming of sensory information into the secondary system from that part of the lemniscal system comprising the attended channel. The resulting increased involvement of the secondary system, and its association cortex component in particular, with the processing of attended stimuli is reflected in an enhanced N1 wave, and it is in this way that the amplitude of N1 is considered to index the operation of 'stimulus set'. Although in this formulation the filter is clearly located on the input to the secondary system, Picton et al (1977) make the more reasonable and sophisticated proposition that "selection is likely mediated by means of a thalamic gating system centered in the reticular nuclei of the thalamus and susceptible to both specific organisation by the frontal cortex[2] and general inhibition by the mesencephalic reticular formation concerned with arousal". Näätänen, however, maintains his traditional position of dissent where, after noting the long latency of the observed effects, he suggests (1975, p.285) that the selective effect on N1 "could be a reaction to a particular outcome of some early step in the discrimination process rather than [being] indicative of pre-set filters". For my part though, the value of distinguishing between a gating mechanism and an early discriminatory process loses much of its power when the gating mechanism no longer refers to the peripheral blocking of sensory input but instead designates the manipulation of non-specific thalamic activity.

In his capacity model of attention, Kahneman (1973) provides at least the beginnings of a possible explanation of the above EP amplitude changes

and the neural activity they embody. He states (p.8) that the capacity model "assumes that there is a general limit on man's capacity to perform mental work" and that the investment of this capacity is synonymous with paying attention. He thus distinguishes two ingredients in mental activity; a specific information input and this non-specific input, which to some degree covaries with arousal, that he variously labels 'effort', 'capacity' and 'attention'. It is my contention both that these specific and non-specific inputs correspond to Hillyard's primary and secondary systems and that mental capacity is measured in terms of the number of functional units (cortical columns) of the association cortex that are avilable. It was argued in the previous section of the chapter that the late slow waves of the EP reflect the activity of cortical columns and although it has been suggested in the discussion of Hernández-Peón's work that the relationship between EP amplitude and the representation of information in the nervous system may not, in general, necessarily be a simple one, it is nonetheless my hypothesis that the amplitude of the late componentry of the EP is directly proportional to the number of cortical columns mobilised by a stimulus. Depending upon the location of the recording electrode, the contribution of the cortical columns of the non-specific cortex to the scalp record will to some degree vary and hence also the lability of the EP associated with this recording site with respect to attentional variables. However, an electrode positioned at the vertex is well-placed to monitor the activity of the cortical columns of the non-specific cortex and <u>ipso facto</u> the utilisation of processing capacity by a stimulus. In respect of the N1 component of the vertex EP

in particular, it is recalled from the previous section of the chapter that surface negativity indexes underlying excitation, and hence to the extent that N1 indexes the initial mass activation of the cortical columns of the non-specific cortex (i.e. capacity) its amplitude will covary with the operation of an early selective process such as Picton's thalamic gate, which, via its action upon non-specific cortical input, regulates the allocation of processing capacity to a stimulus and thereby subserves selective attention.

By way of summarising this section a somewhat eclectic neuropsychological theory of selective attention (diagrammatised in Fig. 1.2) is advanced in the four propositions set out below. In preparation for this exposition it should, however, be emphasised that although styled as a theory, these propositions receive no critical test in the thesis research and are best regarded as fulfilling the more modest role of synthesising the diverse research reviewed above into a framework for thinking about selective attention in terms of possible underlying mechanisms. The present theory is detailed as follows: it is proposed I) that the sensory array is faithfully mapped onto the primary receiving areas of the cortex via the specific sensory pathways; II) that collaterals from these pathways enter the reticular formation and the sensory array is transformed (gated) into a surface of activity that will appropriately mobilise the resources of the non-specific cortex; III) that this surface is played up to the cortex, where sensory information is recruited from the specific cortices with an integrated pattern of activity, corresponding to the perception, ultimately prevailing; and finally IV) that selective attention corresponds

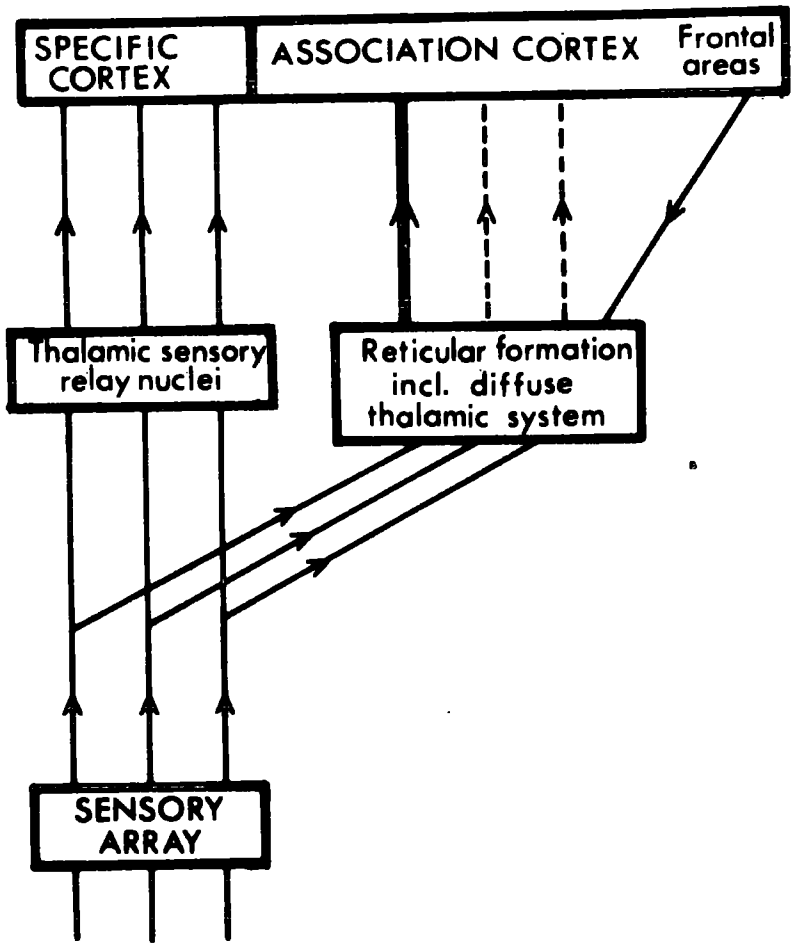

Figure 1.2

Diagrammatisation of the neuropsychological theory of attention elaborated in the text.

to the manipulation of the cortical response in favour of one input and is accomplished by an appropriate transformation of the sensory array at the reticular level, the transformation possibly being computed and supplied by the frontal cortex (see Picton's et al's (1977) quote in the above discussion and also footnote 2).

The Habituation and Temporal Recovery of Evoked Potentials

The term habituation has been applied to a diverse array of response changes in relation to repetitive stimulation from the gill withdrawal reflex in Aplysia (Kandel, 1970) to the serial position effect in man (Hockey and Hamilton, 1977). The most widely accepted definition of the phenomenon is the operational definition proposed by Harris (1943) which simply defines habituation as "a response decrement as a result of repeated stimulation". In their review of habituation in the auditory system, Picton, Hillyard and Galambos (1976, p.348) also define habituation operationally, defining it as the "decline in the magnitude or probability of a response upon repetition of the eliciting stimulus" and go on to indicate that the function of habituation is "to prevent the needless stereotyped response to repeating stimuli" and it thereby (quoting Sherrington, 1906, p.222) "helps to ensure the serial variety of reaction". Habituation, they point out, connotes a change in central nervous system activity, and the various electrophysiological indices of this activity, including EPs, would therefore be expected to demonstrate a corresponding decline in response to repetitive stimulation.

The literature on the habituation of EPs finds its origins, like the attentional literature, in the work of Hernández-Peón's group (Hernández-Peón

et al, 1956; Hernández-Peón, Jouvet and Scherrer, 1957). They recorded EPs to trains of click stimuli (one every 2 secs) and found the EPs to exhibit marked habituation at all levels of the auditory system from the cochlear nucleus to the auditory cortex. Marsh and Worden (1964) again emphasise the importance of controlling the acoustic input, and in their experiment were unable to replicate Herdandez-Peon's results at the cochlear nucleus. They did, however, find a consistent and progressive loss of amplitude of cortical EPs over time. More recently Wickelgren (1968) has corroborated their results, demonstrating that EP habituation in the auditory system is not prominent below the level of the thalamus and Thompson (1975, p.480) states that "this appears to be a general finding in all sensory systems - the ascending relay nuclei do not exhibit habituation". Thompson continues, sensibly remarking that "it would be most surprising if there were marked habituation in sensory systems. If our sensory systems habituated to sensory stimuli, we would rapidly cease to have sensation - a most unadaptive state of affairs". The various studies of the habituation of the human EP have generally not investigated such early sensory activity but have restricted themselves to later components which, given their apparent lability with respect to psychological variables (Regan, 1972), might more plausibly be expected to evidence amplitude changes in response to monotonous stimulation.

Two distinct varieties of the habituation of the late components of the human EP are distinguished in the literature. 'Long-term' or 'slow' habituation is characterised as the gradual monotonic decline in the amplitude of the EP that takes place over an extended period of stimulation.

The phenomenon is easily demonstrated by computing the AEP for successive blocks of trials over the course of an experimental session. 'Short-term' or 'fast' habituation, on the other hand, describes a rapid EP decrement that develops within a train of stimuli separated by short ISIs and which is largely complete with a small number of stimulus repetitions. This phenomenon requires the more sophisticated technique of across-train averaging for its demonstration: short trains of stimuli are presented with long intervals between trains, and then EPs are averaged according to their serial position in the train. It is this latter habituation that preoccupies much of the thesis and which is discussed in the remainder of this introduction. The interaction between slow and fast habituation is, however, the subject of a number of experiments, whereupon the discussion of slow habituation will be resumed.

In their review paper of 1966, Thompson and Spencer delineate a number of criteria which response decrements must satisfy before they can qualify as habituatory. These criteria require that the decrements exhibit spontaneous recovery, potentiation of habituation, generalisation, dishabituation and habituation of dishabituation; that the rate of decline of the response be a negatively accelerated function of the number of stimulus presentations and be steeper for more frequent and less strong stimuli; and finally that the decrements are not produced by peripheral mechanisms, namely receptor adaptation and effector fatigue. Typically, the fast habituation of human EP shows some of these characteristics, but sometimes the criteria are not satisfied or remain

untested. Reviewing the literature briefly, the first notable experiment was conducted by Ritter, Vaughan and Costa (1968) who, by using across-block averaging, were the first to demonstrate short-term decrements in the average EP. They found that with tones delivered every 2 secs there resulted a rapid drop in the amplitude of the P2 component of the EP over the first few stimuli, but that no similar change was found for tones delivered every 10 secs. Since a change in pitch did not dishabituate the response decrement they concluded that the rapid amplitude drop had "only the appearance of habituation" and they considered it to reflect "refractoriness in the auditory system". Fruhstorfer (1971) was also unable to elicit dishabituation of a habituating vertex auditory response using a somatosensory stimulus, and vice versa. He concluded however, that his evidence showed intermodality generalisation of habituation rather than representing a failure to demonstrate dishabituation, and that it supported the hypothesis that auditory and somatosensory information converge in a common corticopetal pathway and hence that the vertex response is mediated by the extra-lemniscal, i.e. non-specific system.

In the first of two earlier experiments, however, Fruhstorfer's group (Klinke, Fruhstorfer and Finkenzeller, 1968) were successful in producing dishabituation by the unexpected interpolation of an additional stimulus in a sequence of somatosensory stimuli. In the subsequent study (Fruhstorfer, Soveri and Jarvilento, 1970) they confirmed the response decrements for both the N1 and P2 wave of the auditory EP and demonstrated that the decrements were a negative exponential function of the number of stimuli, that they developed more rapidly at faster presentation rates and that they exhibited spontaneous recovery.

In completing this review a number of additional studies are included. Ohman and Lader (1972) and Maclean, Ohman and Lader (1975) have investigated the effect of selective attention upon the rate of decrementation of the auditory EP (both fast and slow) and found it to have little influence. Weber (1970), however, reports an enhancement in the habituation of the vertex EP when the duration of the stimulus (a tone-burst) is lengthened. Although Picton et al (1976) supply an undocumented observation indicating an effect of stimulus intensity upon fast habituation, Roth and Kopell (1969), in a properly reported study, were unable to demonstrate any such effect. Although Callaway (1973, p.162) comments that the evidence thus far does provide some reasons "why this phenomena deserves to be considered as a kind of habituation" this latter failure to show an influence of stimulus intensity compounded with the lack of a convincing demonstration of dishabituation and the frequent absence of any control over peripheral mechanisms (such as middle ear muscle contractions, pupillary constrictions, etc.) all leave room for scepticism.

Following Harris (1943) habituation is defined operationally for the purposes of the thesis with the fast habituation of the EP being defined as 'a rapid within-train EP amplitude decrement which develops as a result of repetitive stimulation when ISIs of less than 10 seconds (see Ritter et al, 1968) separate successive stimuli'. What is immediately apparent at this operational level is that this definition subsumes another EP amplitude effect, namely the temporal recovery of the EP. This effect, which refers to the progressive increase in EP

amplitude with lengthening ISI, is also concerned with a response decrement in EP amplitude that develops when short intervals elapse between successive presentations of a stimulus.

The term 'recovery', however, derives from the concept of a refractory period at the level of a single neurone and is used expressly to connote a physiological refractoriness induced by a prior stimulus, in contrast to the psychological bias implied by the term habituation (Callaway, 1973, p.164). As such the defining paradigm for investigating temporal recovery involves the use of pairs, rather than trains, of stimuli (Schwartz and Shagass, 1964; Bess and Ruhm, 1972). However, in much of the work (presumably where averaging across ordinal position was not possible) trains of stimuli have been used (e.g. Davis, Mast, Yoshie and Zerlin, 1966; Gjerdingen and Tomsic, 1970). The AEP is calculated for a run of stimuli separated by a particular ISI, and the presence of refractoriness is inferred from the increase in the amplitude of these AEPs with lengthening ISI. The graph that relates EP amplitude to ISI in this way is referred to as a recovery function, and Fig. 1.3 (adapted from Gjerdingen and Tomsic, 1970) shows such functions for the N1-P2 wave of the vertex EP evoked by visual and auditory stimulation. For both modalities the function is quasi-logarithmic and, although the visual function does appear to asymptote more rapidly, full recovery is complete well within 10 seconds for both types of stimulation.

Thus both the temporal recovery and the short-term habituation of the late components of the EP describe a decrement in the amplitude

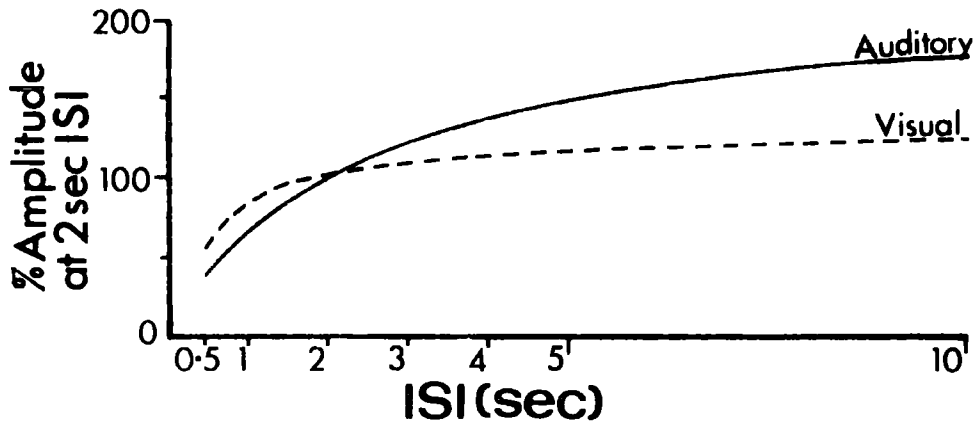

Figure 1.3

Recovery functions of the N1-P2 wave of the visual and auditory vertex evoked potential.

of the EP that develops as a result of stimulus repetition when ISIs of less than 10 seconds elapse between successive presentations of stimuli. Although the term temporal recovery implies an amplitude refractoriness induced by a single preceding stimulus and connotes a physiological investigator bias rather than the psychological bias connoted by the term habituation, operationally the two phenomena are very similar and there is considerable overlap in the procedures used in both cases. Indeed, the studies of such authors as Gjerdingen and Tomsic (1970) directed towards establishing the recovery function of the EP are essentially identical with, for instance, Fruhstorfer's (Fruhstorfer et al, 1970) investigation of the interaction of fast habituation with ISI, except that, in the former case, the findings are used to draw the amplitude-versus-ISI plot of the recovery function. Although discussion of the relationship between temporal recovery and habituation will be resumed later in the thesis, the argument so far allows both phenomena to be subsumed together within a single generic class of EP 'fast response decrements' (FRDs) whose defining characteristic is the depression in EP amplitude that develops when a stimulus is repeated within an interval of 10 seconds of its prior presentation. This generic term is also suggested by Callaway (1973, p.163) and it is relevant to note that both this author and Picton et al (1976) in their discussions of fast habituation freely recruit evidence from temporal recovery studies, and in so-doing provide further support for the present argument that the distinction between these two phenomena is simply terminological.

By subsuming both fast habituation and temporal recovery under a single generic label in this way the pursuit of the mechanism or

set of mechanisms underpinning both is facilitated. Two such possible classes of mechanism have suggested themselves to this author and their exposition and discussion will occupy the remainder of this section. The first and most obvious possibility is that the decrements are simply a manifestation of the refractoriness of the underlying neural networks responsible for the generation of surface recorded potentials. This explanation, in a sense, corresponds to the reduction of the fast habituation of the EP to a temporal recovery phenomenon and as such coincides with the abovementioned view of Ritter (Ritter et al, 1968) in this respect. Roth and Kopell (1969) also apparently regard such an explanation of fast habituation as appropriate when in the introduction to their study (p.302) they question whether "the inhibition [their term for refractoriness] ... of responses to a series of stimuli is complete after the second ... or whether it progresses with the third, fifth or even hundredth stimulus" and in their study they proceed to deal with and discuss EP amplitude decrements across a train of stimuli (i.e. the phenomenon normally described as the fast habituation of the EP) purely in terms of the recovery process.

The second class of mechanism for the FRD proceeds at the psychological (habituation), in contrast to the physiological (refractoriness) level of explanation and can be regarded as representing a reversal of the logical direction of the first mechanism in the sense that it elevates the phenomenon of temporal recovery to the status of an elemental form of fast habituation. In their review Picton et al (1976) discuss a number of types of theory of habituation; namely efferent control theories,

decreased synaptic efficiency theories (similar to the above refractoriness explanation) and finally 'model-making' theories such as Sokolov's. The present psychological explanation of the FRD of the EP is based on Sokolov's work.

The essentials of Sokolov's position, derived from a recent statement (1975), are as follows. Stimulus repetition is held to lead to the forging of a neuronal model of the stimulus in the cortex. To the extent that an anticipated stimulus fits the model a corticofugal inhibition is applied to the reticular activating system. As a result the cortex is activated by a stimulus to a degree that is commensurate with the novelty, i.e. information content, of the stimulus. When a stimulus is repeated, an increasingly accurate neuronal model of it is elaborated, with the result that both novelty and hence the functional state of the cortex (as indexed by EEG desychronisation) decline. In short, habituation is considered to be the cortical inhibition of the reticular arousal response upon stimulus repetition.

The present psychological explanation of the FRD of the EP borrows Sokolov's concept of a neuronal model, but discards the notion of a cortico-reticular system in favour of a purely cognitive mechanism, involving a capacity theory of attention, for the fast response decrement of human brain responses. Although no critical test of Sokolov's theory lay behind this modification of his position, the author's reasons were twofold; firstly, a cognitive account of human brain activity was deemed to be a more appropriate and parsimonious way of talking about this activity than speaking in terms of a hypothetical

mechanism (corticofugal inhibition) derived from animal experimentation; and secondly, an interpretation of EP amplitude effects in terms of a capacity theory of attention has already been argued for in respect of selective attention and does have some basis in neurophysiology and biophysics. This latter theory of EP amplitude was intended to have some generality, and is accordingly retained in the present context.

The author's modification of Sokolov's position apropos the fast response decrement of the late componentry of the human EP runs as follows. It is proposed that, as per Sokolov, the accuracy of a neuronal model of stimulation is the crucial variable with the information content (novelty) of a stimulus being defined in terms of this variable. Given that the organism is concerned about the accuracy of this model, it is argued that the number of cortical columns (i.e. amount of processing capacity) mobilised by a stimulus, and hence the amplitude of the late componentry of the EP, will index the amount of mental work required to extract the necessary information from the stimulus in order to correct the deficiencies of the model. Upon presentation of the first stimulus in the train or, in the case of the defining paradigm of temporal recovery, the pair, the model is maximally deficient and a relatively large investment of capacity is therefore necessary. As a result the model is improved with the consequence that the second stimulus in the train or pair requires less capacity, and hence the amplitude of its EP manifests a decrement. In the case of the fast habituation paradigm, further decrements in EP amplitude are observed across the subsequent stimulus positions in the train as the model achieves an increasingly closer correspondence with reality. The present psychological explanation

of the FRD of the EP thus constitutes a marriage of a capacity theory of attention with the concept of a neuronal model, and is hereafter referred to as an attentional, or, reflecting its neurophysiological underpinnings, a neuropsychological explanation.

A number of interesting implications of this 'model-making' interpretation of the fast response decrement of the EP derive from a consideration of the prevailing sources of novelty (information) in temporal recovery and habituation experimental procedures. Apropos fast habituation, such a perspective suggests a plausible explanation for the difficulties in eliciting dishabituation and the failure of Roth and Kopell (1969) to find any effect of stimulus intensity upon the rate of decrementation. In such procedures, the general organisation of the experimental session (i.e. type of stimulus, number of trains or pairs, number of stimuli per train, etc.) is necessarily outlined to the subject before the session begins, with the result that the content of the coming events is more or less completely known in advance, i.e. event uncertainty is virtually zero. However, whilst the subject knows exactly what will happen in the experiment, his ability to estimate in the time domain is limited and the temporal location of the coming events is thus but imperfectly specified in the neuronal model. Thus it is argued that temporal rather than event uncertainty is the predominant source of novelty in temporal recovery and fast habituation experiments. This hypothesis is supported by the well-established observation that the regularity of ISI is a critical determinant of the rate of habituation (Ohman, Kaye and Lader, 1972; Maclean, Ohman and Lader, 1975) and that this factor also influences the recovery process (Rothman, Davis and Hay,

1970; Nelson, Lassman and Hoel, 1969) with a regular ISI producing, in both cases, more decrementation than an irregular one. Furthermore, if the time of occurrence of the stimulus is of principal interest in establishing the veridical neuronal model then it is perhaps not surprising to find little effect of stimulus intensity upon the rate of habituation. The success in eliciting dishabituation by a change in the temporal characteristics of stimulation (Klinke et al, 1968) but not by a change in its physical characteristics (Ritter et al, 1968; Fruhstorfer et al, 1971) is also neatly and conveniently explained from, and provides support for, this perspective.

The Present Research

The principal objective of the present research is to elucidate the mechanisms underpinning the EP amplitude phenomena associated with selective attention and, in particular, stimulus repetition that were discussed in the preceding two sections. In addition, a number of subsidiary topics within these two general areas and also in the area of EP data-analysis are explored. A brief synopsis of the research programme is provided following this opening paragraph. Although in some of the experiments an occipital placement was employed, the work is primarily concerned with visual and auditory EPs recorded from the vertex, where it has been argued that the EP is optimally sensitive to the variables of interest, namely attentional ones. The conventional nomenclature (Picton and Hink, 1973) is followed, with the late biphasic deflection of the EP (comprising an initial negative deflection at 80 to 150 ms post-stimulus, depending on the modality, followed by a positive trough 80 ms or so later) that preoccupies the thesis being

designated N1-P2. In the opening section of this chapter, the dichotomy between slow surface negativity and positivity was characterised in terms of the excitation and inhibition of cortical columns. In the subsequent sections this notion was meshed with Kahneman's (1973) capacity theory of attention, generating the thesis that the amplitude of the late componentry of the EP (including N1-P2), in reflecting the degree of excitation and inhibition between cortical columns, indexes the utilisation of processing capacity by a stimulus in order to complete the exigent mental work; i.e. that a capacity theory of attention provides a useful general basis for interpreting EP amplitude effects. It should be emphasised that this meshing of the concept of capacity with the conclusions of the mechanisms section of the chapter in order to provide a physiologically and psychologically respectable basis for interpreting EP amplitude is, in the context of the thesis, no more than this. This 'theory' of EP amplitude receives no critical test in the course of the following research, and without such a validation it is conceded that, despite its careful germination over the course of the chapter, it can probably be substituted throughout the thesis by the much cruder statement that 'bigger EPs reflect both more neural and more psychological activity'. It is further recognised that the present 'theory' involves a view of brain function, a theory of attention and a biophysical bridge between the two, any aspects of which can doubtless be criticised or even rejected as they stand. However, in response to Vaughan's exhortations in the opening remarks of the chapter for an adequate multidisciplinary approach to EP data, the present 'theory' is nonetheless retained with the terms 'capacity',

'mental work', 'cortical column', etc. being freely used in relation to EP amplitude effects simply because they represent the elements of an internally coherent conceptual system that facilitates the discussion of these effects in a potentially meaningful way that has some foundation in psychology, biophysics and neurophysiology.

Briefly, the programme of research runs as follows. A preliminary chapter surveys and evaluates a number of potentially useful bioelectric data analytic techniques and indicates those methods which were finally employed in the research and presents the rationale behind their selection. In particular a correlation method for determining the amplitude and latency of individual EPs is described and assessed.

Chapter 3, Part I represents an attempt to extend the work on EPs and auditory selective attention by establishing an analagous correlate in the visual modality. In the second part of the chapter, following a preliminary replication of Hillyard et al's (1973) result, the mechanism of the N1 correlate of auditory selective attention is investigated.

The single trial analysis validated in Chapter 2 is applied in the two introductory studies of the short-term habituation of the vertex visual EP that comprise the first half of Chapter 4. A parametric investigation of the effect of ISI upon the rate of decrementation, and an extension of the inquiry to include a second recording site over the occiput, complete the chapter. A control study which evaluates the role of pupillary mechanisms in fast habituation is also discussed.

The research presented in Chapter 5 remains within the visual modality and evaluates the two alternative hypothetical mechanisms

(i.e. refractoriness and attentional) that were proposed, over the final paragraphs of the preceding section, as explanations of the generic class of EP 'fast response decrements' (FRDs) that subsumes both the short-term habituation and temporal recovery phenomena. It is finally concluded that an attentional mechanism is appropriate, a finding that Chapter 6 attempts to generalise to the auditory modality.

A final chapter comprises an overview and conclusions, and offers some suggestions for future research, including a report of a preliminary investigation in such a programme.

Footnotes - Chapter One

(1) Thompson and Bettinger (1970, p.380) suggest the following explanation based upon the observation that ongoing single cell activity is markedly increased (presumably indicating increased information processing) during novel stimulation: "If the not unreasonable assumption is made that the amplitude of the gross association response ... is proportional to the number of cortical cells activated then ... the number of cells available to be activated by a peripheral probe stimulus (and hence the amplitude of the gross response) is decreased in proportion to the increase in ongoing cellular activity in the system resulting from the novel stimulus presentation."

(2) In reviewing the evidence from his studies on visual recovery cycles (Spinelli and Pribram, 1966, 1967) Pribram (Pribram and McGuinness, 1975) concludes that the frontal cortex does exert considerable control over sensory input channels; influencing, in his terms, the redundancy characteristics of sensory capacity. In an earlier statement Thompson and Bettinger (1970) locate this control as being mediated by the brainstem reticular formation.

CHAPTER TWO

GENERAL METHODOLOGY AND DATA ANALYSIS

Following an initial general methodological section, which includes standard details of procedure and notes on the presentation of results, Chapter Two proceeds with a two-part evaluation of a range of data-analytic techniques relevant to EP research. In Part I the potential of a single trial analysis of human EP data is explored and in Part II the problem of component identification is addressed. The chapter concludes with some general recommendations abstracted from these data analysis sections and indicates those techniques which are to be adopted in the thesis.

Data Collection, the Control of Experiments and the Presentation of Results

The various procedures employed in the recording and transcription of the EEG, and in the control of experiments, remained fairly standard throughout the present research programme. These procedures are summarised in this section and only significant deviations will be noted hereafter.

EEG was recorded monopolarly from the scalp using Ag-AgCl dome electrodes, with reference and ground being provided by either mastoid or clip-on earlobe placements. Electrodes were located on the scalp with reference to the 10/20 electrode system and were affixed with colloidion cement. Neptic electrode gel was injected into the dome to provide the electrolytic medium. Electrode impedances did not exceed 5KΩ and were generally below 1KΩ .

The EEG was amplified by a Grass model 7P58 wide band a.c. pre-amplifier (time const. 0.1 sec) coupled to a 7DAF driver amplifier

(500 hz high frequency cut-off) yielding a total possible gain of x 50000. Analogue-to-digital conversion of EEG epochs was performed on-line by a WDV interface in conjunction with an IBM 1130 computer, and the resulting vector, which included any other information relevant to the epoch (such as trial and block number, reaction time, etc.), was stored on magnetic disk. Although epoch length and sampling rates did vary between experiments they were usually 5 ms/pt and 500 ms respectively. At a subsequent time the data were transferred onto a magnetic disk pack where they could be accessed and analysed by an IBM 370/168 computer.

All the experiments were run under the supervision of the departmental IBM 1130 computer which variously triggered flashes, produced oscilloscope displays, manipulated LED (light emitting diode) matrices, generated bleeps, timed interstimulus and interblock intervals, and timed and recorded response times. Most of the subjects were recruited from the students and staff of the department and were normally remunerated for their services. All the experiments were conducted in a dimly illuminated laboratory allowing a minimum period of 15 minutes for satisfactory dark adaptation.

All the main programmes and most of the subroutines for the control of experiments were written in FORTRAN IV by the author. Some subroutines were of necessity written in ASSEMBLER language, particularly those involved in the digitisation of the EEG where temporal economy was at a premium. Programmes for data-analysis were also written in FORTRAN IV by the author, although packaged programmes (such as SPSS and various analysis of variance programmes) and existing subroutines were employed where possible.

Four levels of statistical significance were recognised and are indicated throughout as follows (the reader is referred back to this key at the first appropriate point in the tables and text of each chapter):

p	$> .05$	ns
p	$\leqslant .05$	*
p	$\leqslant .01$	**
p	$\leqslant .001$	***

Apart from the analysis of the combined data of experiments 7 and 8, in which one between-subject factor appeared, all the designs comprised within-subjects factors exclusively. Whenever a Bartlett test (Winer, 1971) indicated the various subjects x treatments interactions to be homogeneous they were pooled, yielding a common error term. Although a .1 level test of the homogeneity hypothesis was adopted, with a single asterisk indicating a p of $>.1$, the more conservative .25 level (**) suggested by Winer (1971) was generally satisfied. If a pooled term does not appear in the analysis of variance summary table then it can be assumed that pooling was not found to be justified, with individual subjects x treatments error terms for each source being retained. Where such terms are used two figures, separated by a comma, appear in the df column of the analysis of variance summary table. The first of the figures indicates the dfs associated with the source in the normal way, and the second figure indicates the dfs associated with the relevant error term. A further point in relation to tables is that some internal discrepancies may be present due to rounding errors. In some of the analysis of variance summary tables the statistic r^2 is included to index the proportion of the variability due to treatments accounted for by a particular comparison

(see Keppell, 1973, p.107 for details). On such occasions the significance of the residual variation is also usually evaluated in a final column.

Component amplitudes are expressed throughout as the absolute displacement measured in μV of the peak (or trough) from the baseline (0 μV), with reference to the normal direction of the deflection. Thus a positive component, such as P2, peaking, for instance, 2.3 μV positive with respect to the baseline is entered as having an amplitude of +2.3 μV. On the other hand, an N1 2.3 μV positive would be assigned an amplitude of -2.3 μV, and so on. In general, only those treatment means regarded as essential (main effects, notable interactions, etc.) are presented.

DATA ANALYSIS PART I: SIGNAL AVERAGING AND THE SINGLE TRIAL APPROACH

The technique of signal averaging is widely employed in the extraction of evoked activity from the background noise of the EEG. The validity and efficiency of the technique depend upon the following criteria: that the observed waveform is an additive combination of signal (i.e. the evoked potential, EP) and noise; that the noise is produced by a stationary random process with an expected value of zero; and finally that the EP is stable in amplitude and latency. These assumptions have been discussed by a number of authors, notably Donchin (1966) and Regan (1972), the consensus being that they do not obtain in practice. Of particular significance to this research is the question of the invariance of the EP; if the amplitude and latency of the EP vary from trial to trial then potentially valuable information is being lost in the computation of the average EP (AEP). In general this information loss may not be a serious limitation- the AEP, like any statistic, provides an expedient summary of a large quantity of data.

However, in certain areas, such as the habituation of the EP, where trial-by-trial changes are critical, a single trial analysis would be of considerable value. Näätänen, in his recent review (1975, p.298), also alludes to the unexplored promise of the single trial approach; the two studies to be presented in this part of the chapter, inspired by Näätänen, explore this promise in preparation for the single trial analysis of the fast habituation of the EP that occupies the opening experiments of Chapter 4.

Experiment One: Statistical Detection of Individual Evoked Potentials: An Evaluation of Woody's Adaptive Filter

INTRODUCTION

Broadly speaking, the problem of extracting information from single trial records has been approached using either multivariate statistics or cross-correlation. In the former category the work of Donchin's group, using Stepwise Discriminant Analysis to 'recognise' individual EPs, has been outstanding (Donchin, 1969a, b; Donchin and Herning, 1975; Squires and Donchin, 1976). However, as Donchin himself recognises (1969b), methods of this type do continue to assume that the latency of the EP is invariant.

The cross-correlation function on the other hand makes no such assumption, and can serve to indicate both the amplitude and latency of individual EPs. A number of investigators (Palmer, Derbyshire and Lee, 1966; Derbyshire, Dreissen and Palmer, 1967; Woody, 1967; Weinberg and Cooper, 1972a, b; Pfurtscheller and Cooper, 1975) have employed correlation techniques; in particular, Woody describes an adaptive filter system that, through an iterative use of correlation and averaging, locates

repetitive bio-electric signals, such as EPs, and resolves them from their associated noise. Using the framework supplied by Signal Detection Theory (SDT) the present technical investigation describes and evaluates the practical performance of Woody's filter in discriminating components of the visual EP from the background EEG.

METHOD

The EEG was recorded monopolarly from a vertex placement over the course of an experimental session that comprised 400 trials (separated by intervals of 4 secs) arranged in 8 blocks of 50. Trials consisted of a warning bleep followed by a visual stimulus after a random interval of either 600, 725 or 850 ms. Subjects, of whom there were 3 male and 3 female, were directed to respond as quickly as possible to the occurrence of the stimulus, thus assuring their continuing interest in the experiment. A chequerboard stimulus was used (provided by a Grass PS2 photostimulator positioned 12 feet from the subject, intensity setting = max.), as patterned stimuli yield larger EPs (Regan, 1972) and consequently facilitate the detection process. The central area of the chequerboard was marked with a cross which served as a fixation point.

For each flash 300 EEG samples were taken, extending at 2 ms intervals from an origin 60 ms in advance of the stimulus. On half of the trials the stimulus was withheld, i.e. 'catch' trials, but the EEG was sampled as per 'test' trials with 300 points over a 300 ms epoch being collected. Both these 'noise only' catch trials and the 'signal plus noise' (SN) test trials were stored awaiting off-line data-analysis.

Identically cueing both N and SN trials was considered essential in order to justify an assumption crucial to this analysis; namely, that the characteristics of the noise process, the EEG, are equivalent on both types of trial.

The prominent late biphasic deflection (N1-P2), peaking negative and positive at typically 130 and 270 ms respectively, was chosen as the feature of the EP to be filtered. An initial template of this wave was obtained by conventional time-locked averaging, and the adaptive filtering then proceeded as described in the publication of this investigation (Wastell, 1977, p.835).

> The sum of the cross-products between the template and the observed EEG, γ_{k^*}, is computed across a range of time shifts, k = 1, 2, 3 ... etc., extending from some starting point. Template width, scan origin and range were typically 200 ms, stimulus onset and 200 ms respectively. When γ_{k^*} attains a maximum the signal is said to have been detected, with amplitude γ_{k^*} and latency k^*. The procedure may be illustrated by referring to Fig.1 [Fig. 2.1 here] ; when the template is cross-correlated with b, c, and d, values of k^* corresponding to 102, 63 and 116 ms are obtained. The concomitant amplitudes, expressed as the ratio of γ_{k^*} to template variance, are 1.31, 1.68 and 1.41 respectively. Every trial is then shifted the number of points given by k^* and, thus aligned in time, averaging yields an improved 'picture' of the signal. The new correlation-average then replaces the old template and the entire process is repeated. Further iterations, bringing better resolution, are pursued until, on some index, no more improvement is seen.

All the remaining details of the procedure are well covered in the final paragraphs of Wastell (1977, pp.835-6) which are accordingly reproduced here in full.

> Once aligned in time Woody calculates the correlation coefficient between each trial and the template and uses the arithmetic mean of these individual coefficients to index the iteration process. More properly however, Z-transforms of the correlation coefficients ought to enter into the calculation of the mean (Fisher, 1950). The average correlation coefficient

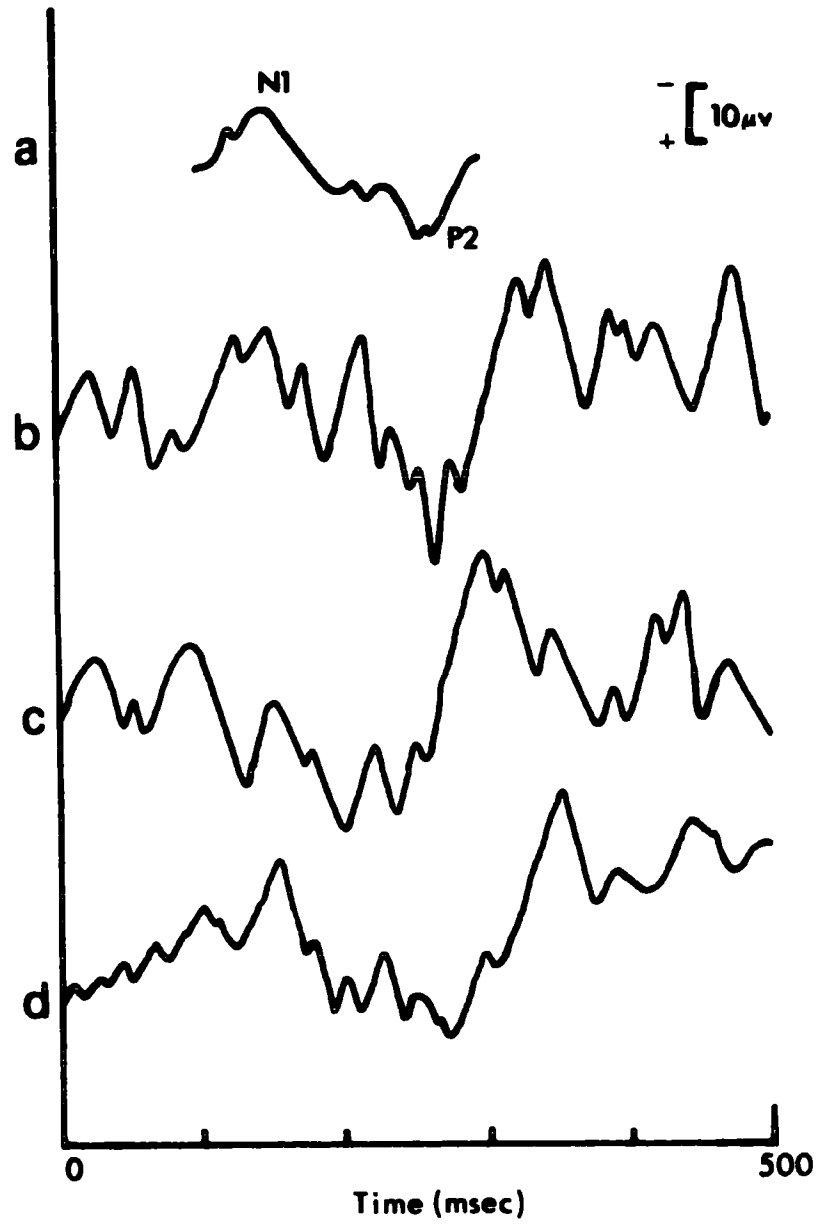

Figure 2.1

b - d are examples of single trial records for subject SW. Also shown (a) is the initial template.

is then given by:

$$\bar{\rho} = Z^{-1}\left(\frac{1}{n}\sum_{i=1}^{n} Z(\rho_i)\right)$$

where ρ_i is the correlation coefficient between the template and the i^{th} trial once aligned and $Z()$ and $Z^{-1}()$ indicate the Z-transform pair.

The present study goes beyond Woody's procedure in another respect: the template was also cross-multiplied by 'noise only' trials across the same range of k and the average correlation coefficient calculated as above. $\bar{\rho}_s$ and $\bar{\rho}_n$ are used to designate $\bar{\rho}$ for SN and N trials respectively.

In Signal Detection Theory terminology γ_{k^*} is an evidence variable: i.e. it can be considered as providing evidence on which, if no other information was available, a decision as to the presence or absence of the signal on any trial could be based. The performance of the filter in resolving signals from noise can thus be characterised as the dissimilarity of the distributions of γ_{k^*} when a signal is present and when there is noise only. Following Green and Swets (1966) the statistic Δm is adopted to describe the extent of this dissimilarity: Δm is defined as the distance between the means of the SN and N distributions scaled in noise standard deviation units. It provides us with a measure of sensitivity combining the information given by $\bar{\rho}_s$ and $\bar{\rho}_n$ into a single index of the filter's ability to distinguish signal from noise.

RESULTS (also reproduced from Wastell, 1977, p.836)

Table 1 [Table 2.1 here] shows changes in $\bar{\rho}_s$ and $\bar{\rho}_n$ as iterations proceed. In both cases the trend is upwards. For 5 subjects the criterion improvement of no change in the second decimal place of $\bar{\rho}_s$ is attained within 4 iterations. For the remaining subject, GJ, increments to $\bar{\rho}_s$ are still observed when the process is eventually curtailed after the sixth iteration.

Changes in Δm over iterations are tabulated in Table 2 [Table 2.2 here] . On the whole sensitivity appears to decline as iterations proceed; in only two subjects, NS and SW, does it improve.

Frequency distributions of k^* were plotted; the examples for 2 subjects shown in Fig.2 [Fig.2.2 here] are typical of those found throughout. As one might anticipate the distribution of k^* in the presence of a signal demonstrates a marked central tendency, whereas on 'noise only' trials, in the

TABLE 2.1: EXPERIMENT 1

The average correlation coefficient, $\bar{\rho}$, on SN. (Upper Table) and N trials (Lower Table) shown for each subject as a function of iteration number.

Subject	Iteration					
	1	2	3	4	5	6
DK	0.788	0.791	0.792	-	-	-
NS	0.772	0.782	0.783	-	-	-
KO	0.710	0.731	0.738	0.742	-	-
LB	0.682	0.697	0.699	-	-	-
SW	0.637	0.658	0.655	-	-	-
GJ	0.553	0.553	0.575	0.604	0.637	0.660

Subject	Iteration					
	1	2	3	4	5	6
DK	0.443	0.442	0.442	-	-	-
NS	0.466	0.483	0.490	-	-	-
KO	0.406	0.416	0.433	0.445	-	-
LB	0.490	0.507	0.515	-	-	-
SW	0.421	0.435	0.436	-	-	-
GJ	0.305	0.360	0.389	0.421	0.455	0.481

TABLE 2.2: EXPERIMENT 1

N1-P2 latency (i.e. k^*) variance ("signal plus noise" trials) tabulated for each subject as a function of iteration number

Subject	Iteration					
	1	2	3	4	5	6
DK	10.5	10.7	11.4	-	-	-
NS	16.9	16.5	16.1	-	-	-
KO	9.9	11.2	11.1	11.2	-	-
LB	17.6	19.0	19.1	-	-	-
SW	15.1	14.1	14.4	-	-	-
GJ	15.4	15.9	16.5	17.4	18.4	18.8

TABLE 2.3: EXPERIMENT 1

Sensitivity, Δm, tabulated for each subject as a function of iteration number

Subject	Iteration					
	1	2	3	4	5	6
DK	4.62	4.62	4.60	-	-	-
NS	2.66	2.68	2.68	-	-	-
KO	3.32	3.29	3.25	3.20	-	-
LB	1.50	1.44	1.40	-	-	-
SW	3.67	3.81	3.83	-	-	-
GJ	1.23	1.23	1.20	1.18	1.16	1.13

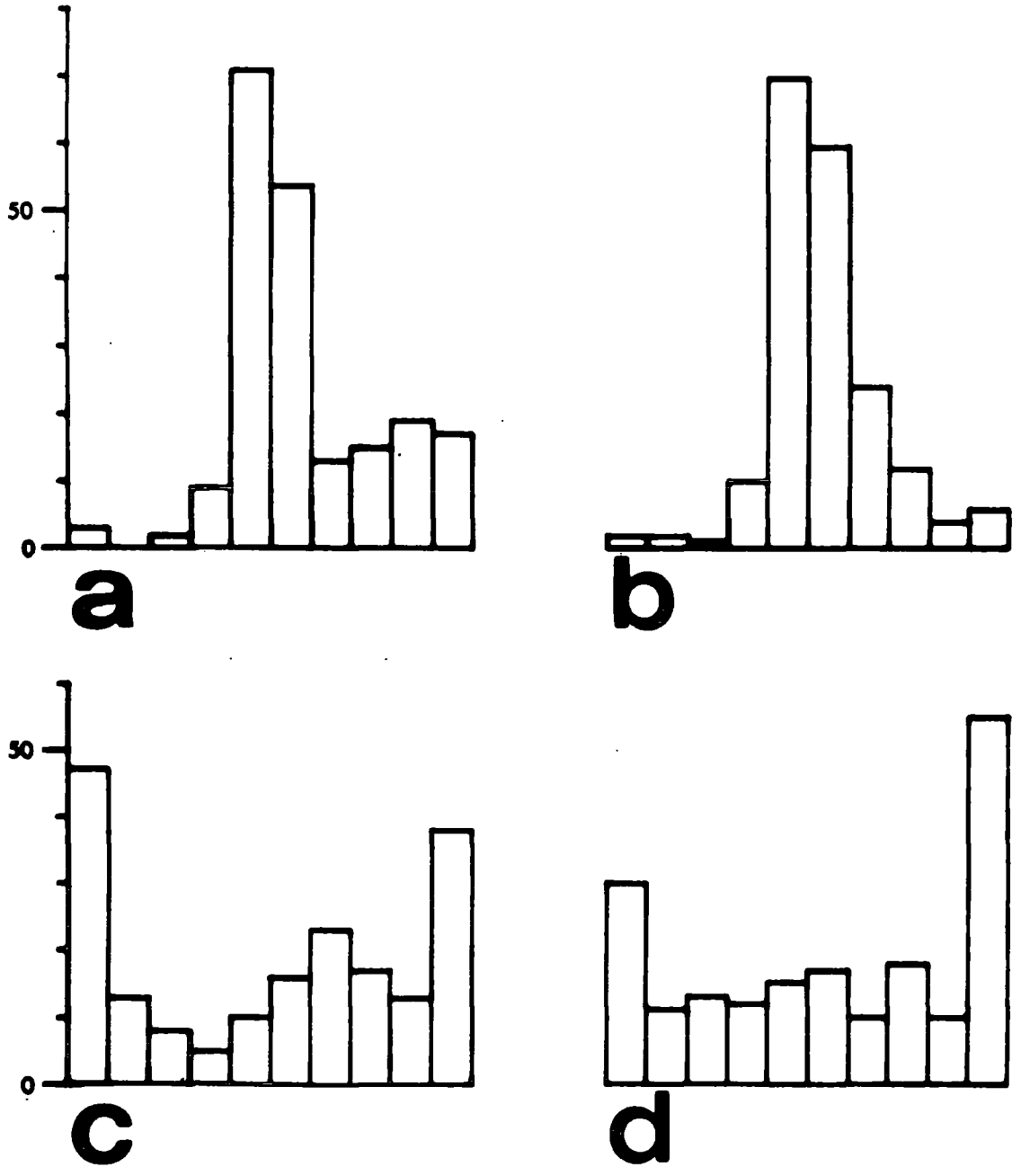

Figure 2.2

Frequency distributions of k* for subjects LB (a,c) and SW (b,d) after 3 iterations. A template, approximately 200 ms wide, was used to scan the EEG for N1-P2 across a range of 200 ms; a and b are the distributions obtained in the presence of a signal, and c and d in its absence. Each bin represents one-tenth of the range, i.e. 20 ms.

absence of any such tendency, clustering in the first and last bins and uniformity over the intervening range is observed. Summary statistics up to the second moment were computed for all subjects for each iteration; standard deviations for SN trials are shown in Table 3 [Table 2.3 here] (note that k^* is measured in 2 ms units). The overall picture is of increasing k^* variance. Interestingly, and intuitively attractive, the 2 subjects who show a decline in k^* variance are the same two who show an improvement in sensitivity.

DISCUSSION

The ability of the cross-covariance function to discriminate individual EPs from the background EEG is well attested in the results of this study. For instance, if we arrange the criterion value of the evidence variable (i.e. some minimum value of γ_{k^*} that must be exceeded before an EP can be said to have been detected) to obtain a 'hit' rate (i.e. detection of an EP when one is present) of 90%, the false alarm rate (detection of an EP when none is present) is as low as 2% for subject DK ($\Delta m = 4.62$). For other subjects, particularly GJ and LB, the false alarm rate runs somewhat higher and a stricter criterion could be set to reduce this rate to a more acceptable level[1].

It would thus appear that if the signal-to-noise ratio in EP research is characterised as the ability of a cross-covariance pattern recogniser to distinguish signal from noise then, as far as the more prominent features of the EP are concerned, the ratio is more favourable than the widespread use of averaging might suggest. Certainly information about N1-P2 amplitude and latency seems to be readily available in individual records and, as the investigation of this wave is the main preoccupation of this thesis, this demonstration of the practicality of a single trial analysis is particularly relevant.

The value of Woody's iterative procedure is, however, cast in some doubt as the discussion of Wastell (1977, p.838) recognises in the following two paragraphs:

> Changes in $\bar{\rho}_s$ are in the main accompanied by corresponding changes in $\bar{\rho}_n$. The suggestion is that as iterations proceed there is an increasing accommodation within the template of aspects of the noise process. The decline in sensitivity and accompanying increasing k* variance observed in 4 of the subjects can also be explained in this way. If iterations beyond the first are to be pursued then it appears desirable to employ a statistic such as Δm to index the operating characteristics of the filter.
>
> Woody's claim that the filter improves signal resolution must be considered carefully in respect of evoked response data. There appears to be some point beyond which further iterations bring only spurious improvement. In the present study, selection of a good initial template meant that this point was reached relatively rapidly; in 4 subjects only one iteration was required. The poor showing of the filter is not a defect in some fundamental respect but rather a reflection of this choice of initial template compounded with the use of an index, $\bar{\rho}_s$, that converges after the limit of real improvement has been passed.

A final comment derives from the possible relationship between the performance of the filter as indexed by the changes in both Δm and the standard deviation of k* over iterations. The latter is considered as such an index in the sense that erroneous detections would tend, because (unlike hits) they are not restricted to a limited central latency range, to increase the overall temporal variance of detection. It is thus gratifying to note that if the performance of the filter is expressed in terms of the net change in both indices (Table 2.4) the correlation between the two measures is in fact significant ($r = .78$, $df = 5$, $2p < .05$).

TABLE 2.4: EXPERIMENT 1

The performance of the filter expressed in terms of the overall change in Δm and the standard deviation of k* tabulated for each subject. The correlation between these two indices is also shown.

	increase in Δm	Decrease in standard deviation of k^*
DK	-.02	-.9
NS	+.02	+.8
KO	-.12	-1.3
LB	-.10	-1.5
SW	+.16	+.7
GJ	-.10	+.1

$r = +.78$, $df = 5$, $2p < .05$

Experiment Two: Evoked Response Correlates of Reaction Time: A Single Trial Analysis

INTRODUCTION

Inspired by the technical success of experiment 1 in extracting the amplitude and latency of N1-P2 from individual records, this second study sought to demonstrate the applied usefulness of such a single trial approach. It will be recalled from the earlier study that reaction times to the flashes were recorded and this second experiment constitutes an investigation of the correlation between this behavioural data and the already presented single trial physiological data. As such it does not really amount to a separate experiment, but is so-classified for convenience of exposition.

Previous studies of EP correlates of RT (e.g. Donchin and Lindsley, 1966; Morrell and Morrell, 1966; Morris, 1971) have typically proceeded by blocking trials together according to speed of response, and then calculating the AEP for each block. Although these studies demonstrate that shorter RTs are associated with a larger amplitude N1-P2, no consistent relationship with N1-P2 latency has emerged. The rationale of this study was that this latter null result was a consequence of the use of AEPs as a data-base rather than indicating the genuine absence of such a relationship. If the correlation between N1-P2 latency and RT is independent of, and weaker than, the amplitude correlation, then one would not expect it to be revealed by the blocking procedure described above. By extracting N1-P2 latencies from individual trials and computing their correlation coefficient with RT it was hoped to provide a less ambiguous insight into the existence of any such relationship. It was

also expected that the amplitude correlation would be corroborated using the single trial data.

METHOD

Although the procedure has already been presented in the method section of the first experiment the main points, plus some additional information regarded as being more pertinent to this investigation, are recapitulated in the following paragraph:

EEG was recorded from the vertex of the six subjects participating in the experiment. The task was a simple reaction time task employing a variable foreperiod; an auditory warning signal, delivered over headphones, preceded the imperative visual stimulus by a random interval in the range 600 to 850 ms. The subject was instructed to key-press as rapidly and accurately as possible; catch trials were included to ensure accurate responding, and a red light (positioned within the photostimulator to eliminate the possibility of post-stimulus eye movements in its direction) was flashed to inform the subject if a response was unacceptably slow.

DATA ANALYSIS

Given the evaluation of the iterative process expressed in Experiment One, the single trial analysis was restricted to one iteration. This simplification of the analysis allows it to be rephrased in the more intelligible form of the succeeding two paragraphs (reproduced from Wastell, 1976). Although the procedure does receive some illustration in Fig.2.1, a second, and more complete, illustration is provided here in Fig.2.3 to support this particular account.

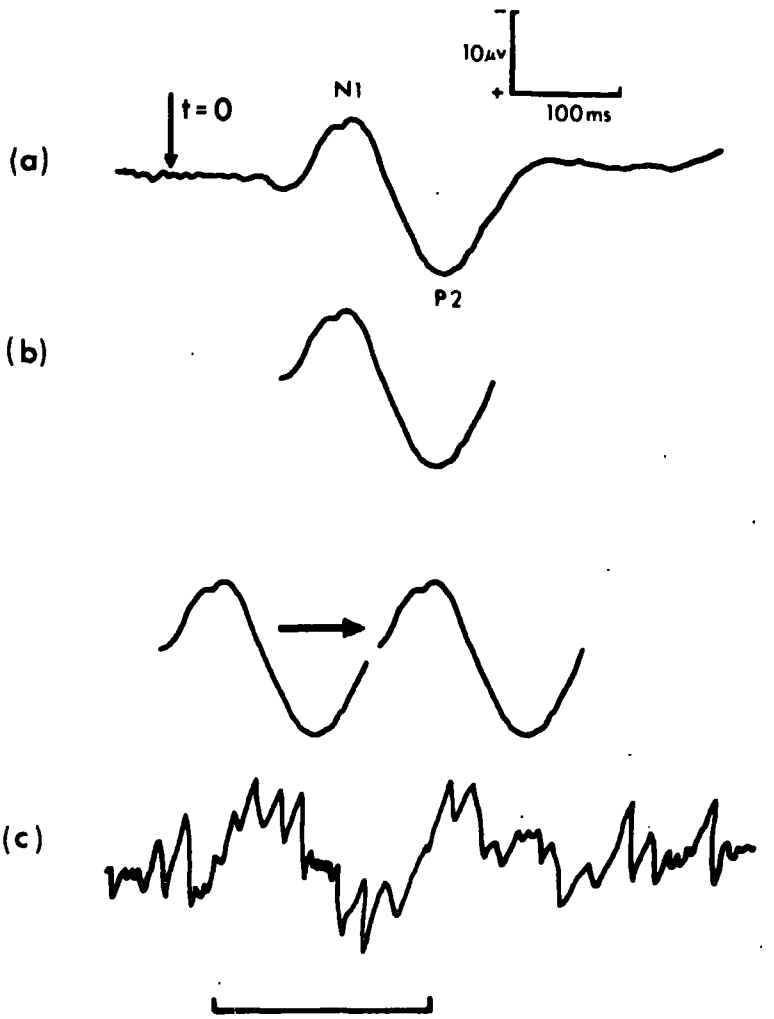

Figure 2.3

Illustration of the single trial analysis with reference to one subject's data. The average EP for subject 1 is shown in a) and the template in b). An example of a single EEG record is presented in c) with the initial and final positions of the template also shown above the record. For each trial the template is stepped across this range with the cross-covariance computed at each position. For this particular trial N1-P2 was detected with the template positioned as indicated by the solid line beneath the record, i.e. 48 ms earlier than its position in the average.

> For each subject the AEP was computed and ... N1-P2 ... identified. The entirety of this feature from the earliest beginnings of negativity at 100 ms to the return to baseline after P2, i.e. about 200 ms later, was extracted and used as a template. The cross-covariance between the template and the EEG is then computed across a range of lags for each trial in turn. When the covariance attains a maximum N1-P2 is taken to have been detected; this maximum and the associated time-point, providing the measures of N1-P2 amplitude and latency respectively for that trial.
>
> Each single trial record of a subject's data is thus scanned for the occurrence of N1-P2. The origin and width of the scan were typically stimulus onset and 200 ms respectively. To give a more immediate impression of the process consider N1 peak latency. Remembering that this peak occurs usually around 40 ms into the template, we are in effect scanning each trial for it in a window extending from 40 ms to 240 ms post-stimulus.

With values for N1-P2 amplitude and latency, and reaction time Pearson product-moment correlation coefficients were calculated for each subject to describe and assess the relationships between these three variables.

RESULTS

The resulting coefficients are compiled in Table 2.5i. Reaction time and N1-P2 latency show a high positive correlation (r_a) in 5 subjects, RT and N1-P2 amplitude a high negative correlation (r_b) in 4 subjects, and N1-P2 amplitude and latency a low negative correlation (r_c) also in 4 subjects.

Partial correlations were also computed and are presented in Table 2.5ii. Although the correlations are generally reduced, the two principal relationships remain substantially intact (r_d, r_e); faster responses are thus independently associated with shorter latency and larger amplitude N1-P2s. However, as the disappearance of r_c when reaction time is partialled out (r_f) indicates, there is a tendency for latency and amplitude to be conjointly associated with RT.

TABLE 2.5: EXPERIMENT 2

Pearson product-moment correlations (i) and partial correlations (ii) between reaction time (RT), N1-P2 latency (LAT) and N1-P2 amplitude (AMPL). For key to significance levels see Section 1 of this Chapter. (Note - only the 5% and 1% levels of significance are distinguished in these tables and ns entries have been omitted for tidiness).

(i)

Subjects	Correlations, df = 200		
	LAT. + RT (r_a)	AMPL. + RT. (r_b)	LAT. + AMPL. (r_c)
DK	0.47**	-0.32**	-0.20**
NS	0.31**	-0.34**	-0.19**
KO	0.38**	-0.11	-0.08
LB	0.45**	0.04	0.05
SW	0.40**	-0.67**	-0.24**
GJ	0.06	-0.39**	-0.15*

(ii)

Subjects	Partial Correlations, df = 200		
	LAT. + RT - AMPL. (r_d)	AMPL. + RT. - LAT. (r_e)	LAT. + AMPL. - RT (r_f)
DK	0.44**	-0.26**	-0.06
NS	0.27**	-0.30**	-0.09
KO	0.37**	-0.09	-0.04
LB	0.45**	0.02	0.04
SW	0.33**	-0.65**	0.04
GJ	0.00	-0.39**	-0.14*

DISCUSSION

The rationale of the present study was clearly vindicated by the results; the relationship between N1-P2 latency and RT is revealed by the single trial analysis. The previous finding of a relationship between N1-P2 amplitude and RT is also corroborated by the analysis, and although these relationships are principally orthogonal, there remains a suggestion (r_c) that earlier N1-P2s are larger in amplitude.

In his review of EP and RT studies, Wilkinson (1967, p.240) comments that in the procedures used "there may be little else to affect reaction time other than variations in the subject's attention", and goes on to conclude that, given the sensitivity of EP amplitude to attentional level, it would be "the amplitude rather than the latency of the evoked response which correlates with variation in reaction time". In the introduction to the experiment, it was similarly speculated, in order to explain the failure of the blocking method of earlier studies (trials are blocked together according to speed of response and the AEP calculated for each block, e.g. Morrell and Morrell, 1966, etc.) to demonstrate a relationship between N1-P2 latency and RT, that this relationship would be weaker than, and independent of, the amplitude correlation. In such circumstances, blocking trials together according to RT would tend to amount to blocking them together according to N1-P2 amplitude rather than latency, and hence the null results in this latter respect. However, the correlations of Table 2.5 indicate, if anything, the N1-P2 latency/RT relationship to be the stronger, implying that the blocking method would indeed be successful in demonstrating this relationship in the data of this study. Accordingly, the median reaction

time was calculated for each subject and the two AEPs representing the trials on either side of this mid-point, were calculated. The amplitude and latency of N1-P2 (the latency being defined as the mean latency of the two components) were determined and are compiled in Table 2.6. The anticipated latency and amplitude differences are both confirmed, with faster responses being associated with both shorter latency and larger amplitude N1-P2s.

Thus, although the single trial analysis of the present study was successful in revealing a correlation between N1-P2 latency and RT where earlier work using signal averaging had failed, this relative success cannot be simply ascribed to the introduction of the single trial analysis. It is relevant, particularly in respect of Wilkinson's above quotations, to recognise that the present study incorporated a range of experimental manipulations, not generally present in the earlier work, designed to minimise fluctuations in attention; namely, the use of a warning signal, penalisation of slow responding, and the presentation of stimuli in short blocks. The earlier null results can thus probably be attributed both to their use of experimental procedures that did not constrain fluctuations in attention, and hence allowed the N1-P2 amplitude/RT relationship to dominate the latency one, compounded with the use of a data-analytic technique, the blocking method, that would necessarily not be sensitive to minor EP-RT correlations.

Although the success of conventional averaging methods in this study does cast some doubt on the necessity of the single trial analysis, three important points should, however, be considered:

TABLE 2.6: EXPERIMENT 2

Mean N1-P2 amplitude (μV) and latency (ms) as a function of reaction time (1 = trials faster than the median RT, 2 = trials slower). Values of Student's t are also shown (one tailed test).

	N1-P2 amplitude	N1-P2 latency
1	22.34	201
2	19.06	213
t,df = 5	2.01*	2.53*

TABLE 2.7: EXPERIMENT 2

Mean correlation coefficients showing the influence of ISI The results of Student's t-tests evaluating this influence are also shown.

	LAT + RT. AMP.	AMP + RT. LAT.	AMP + LAT.
ISI controlled	.32	-.31	-.14
ISI not controlled	.31	-.28	-.11
t,df = 5	-.21 ns	-.49 ns	-1.02 ns

TABLE 2.8: EXPERIMENT 1.

The ratio of the variance of the distribution of γ_{k*} obtained in the presence of a signal to the variance obtained with no signal (F) shown for each subject. Significance levels associated with the F ratios are also presented.

Subject	$F = MS_{SN}/MS_N$	(F distributed as F_{max} with 200 df)
DK	4.10	**
NS	1.39	*
KO	1.25	*
LB	0.75	ns
SW	2.50	**
GJ	1.21	*

firstly, the success of the study retains an intrinsic value in any event as a demonstration of the practicability of extracting usable information from single trials; secondly, whereas the blocking procedure reveals that both N1-P2 amplitude and latency are correlated with RT such an analysis does not permit evaluation of the independence of these correlations; and thirdly, the correlation coefficients generated by the blocking procedure will inevitably over-estimate the underlying brain-behaviour relationships[2].

In his review, Wilkinson (1967, p.235) concludes that the observed relationship between N1-P2 amplitude and RT "is probably due to the influence of attentional factors upon both the behavioural and physiological measures concerned". Following Wilkinson, the inverse correlation between N1-P2 amplitude and RT of this study is reasonably ascribed to the joint correlation of larger amplitude N1-P2s and faster responses with increased attentional level.

Although the correlation between RT and N1-P2 latency is easily and reasonably explained as the association of faster reaction times with an earlier cortical activation, the source of the EP and RT temporal variability is in itself an intriguing question. It is possible that the temporal uncertainty introduced by the variable foreperiod was a contributory factor: Surwillo (1977) has shown that both the latency of the P2 component of the auditory AEP and RT vary inversely as a function of interstimulus interval (ISI).

In order to evaluate the significance of the ISI variable in Experiment Two the data were re-analysed. The correlation coefficients between N1-P2 amplitude, latency and RT were calculated at each ISI and,

by computing the average coefficient across ISI (using the Z-transform), any influence of ISI was eliminated. Average correlations and partial correlations[3] were computed across subjects with the correlations that summarise the results of the study being compiled in Row 1 of Table 2.7. The second row of the table shows the corresponding average correlation coefficients derived from Table 2.5 and, as Student's t-tests (Row 3) do not reveal any significant discrepancies, an explanation of the correlations as a result of the use of a variable ISI can be dismissed.

The negative correlation between N1-P2 amplitude and latency suggests either that the attentional mechanisms can influence the time-course of processing or alternatively that both the level of attention and N1-P2 latency are influenced by some common factor. However, although the correlation was significant in 4 subjects, it is small in magnitude and it appears that, as far as the present task is concerned, N1-P2 latency and amplitude reflect largely independent dimensions of information processing.

Finally, a study by Bostock and Jarvis (1970), which has recently come to the author's attention, should be mentioned. They, in fact, also obtained a positive relationship between the latency of some EP components, including P2, and reaction time using the blocking procedure described above. Although the amplitudes of N1 and P2 in their study were related to RT in the usual way, these associations were shown to be in part attributable to long-term effects developing over the course of the experiment, rather than moment-by-moment fluctuations in alertness. In order to evaluate the dependence of the correlations

of the present study upon time-in-the-experiment, the EP and RT data were differenced (Chatfield, 1975) in order to remove any long-term trends. The average correlation coefficients based on the differenced data were .34 and -.27 for the partial correlations between N1-P2 latency and RT, and N1-P2 amplitude and RT respectively; and -.13 for the correlation between N1-P2 amplitude and latency. These values do not notably diverge from the figures already presented, indicating that the correlations of this work reflect the covariance of trial-by-trial changes in brain evoked activity and behaviour. Presumably, the absence of an influence of time-in-the-experiment is a further reflection of the success of the experimental manipulations in maintaining the subject in a constant attentional state over the sourse of the session. In passing, it is finally interesting to note that Bostock and Jarvis performed a single trial analysis upon the data of two of their 15 subjects who showed a sufficiently high signal-to-noise ratio to permit ready visual identification of individual EPs. No relationships between either N1 or P2 amplitude and RT were obtained, although N2 was significantly correlated with RT. Latency effects were not evaluated.

Supplementary Notes to Experiments One and Two

A. Controls for eye-movements and blinks were not implemented in either of these investigations and their absence represents an obvious criticism of the design. However inspection of the frequency distributions of N1-P2 amplitude for the 6 subjects reveals a consistently continuous unimodal morphology, and it may be deduced that eye artifacts which, representing a different class of event, would produce irregularities

(probably in the form of a cluster of extreme values) in these distributions, were not significantly present. An example from one subject's data is shown in Fig.2.4.

B. Some justification for the use of γ_{k*} as an index of EP amplitude is probably necessary as it could be contended, for instance, that this measure reflects the overall shape of the EP rather than its peak amplitude. Certainly serious doubts over the validity of cross-correlation in this regard would be well-founded, but cross-covariance, being simply the weighted sum of the time-points that encompass the EP, seems better justified. Indeed the broader base of such a measure, its objectivity and the fact that, by virtue of the weighting, it continues to reflect primarily the peak amplitude of the EP, are considered, if anything, to recommend its use as an index of EP amplitude.

C. As already indicated in this chapter, the technique of signal averaging assumes post-stimulus brain activity to represent an additive sum of a constant amount contributed by the signal, the EP, and a random component, the EEG, referred to as noise. If this model is correct then, although the presence of the EP on SN trials would shift the mean value of γ_{k*} (N1-P2 amplitude) above its value on N trials, the variance of γ_{k*} should remain the same under both conditions. However, as Table 2.8 indicates, the variance of the SN distribution is significantly greater in 5 of the 6 subjects and thus, what is a general observation in SDT experiments, that the signal does contribute to the variance of the evidence variable, is confirmed in this less conventional application of the method. Indeed it was this very conviction that the EP was not

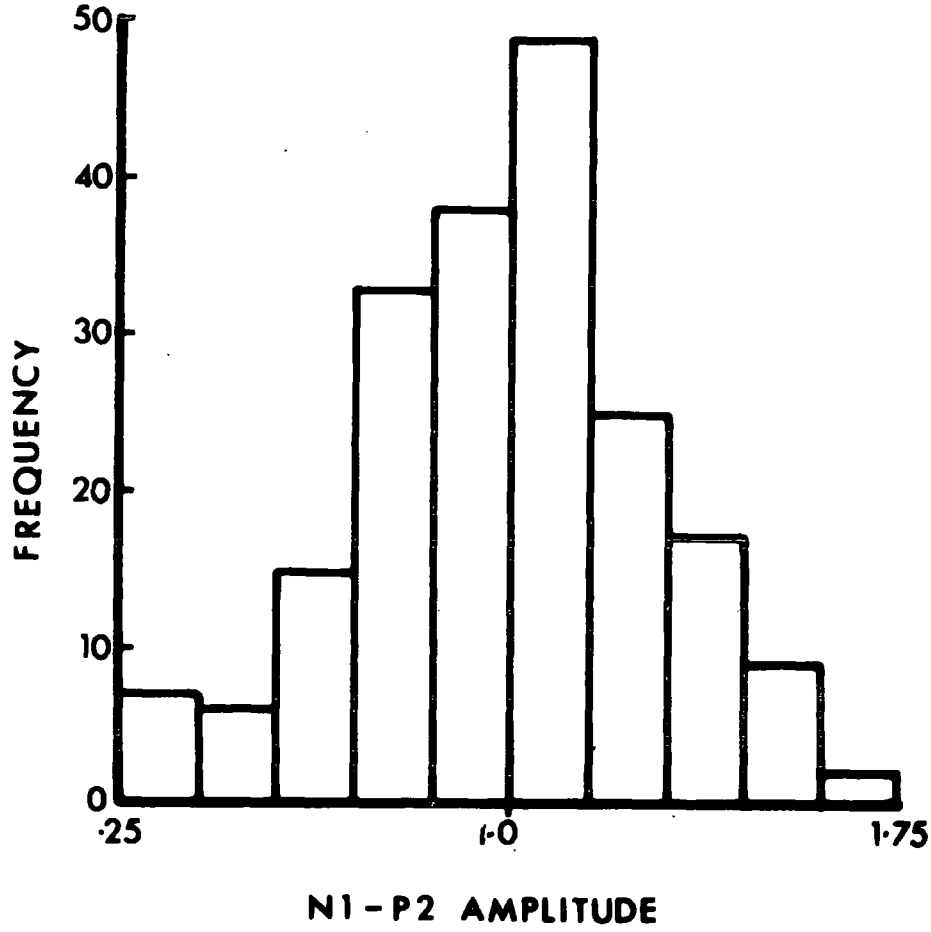

Figure 2.4

Frequency distribution of N1-P2 amplitude (obtained in the presence of a signal) for subject KO. (N1-P2 amplitude is expressed as the ratio of γ_{k*} to the template variance, yielding a measure that expresses the magnitude of the detected EP as a function of the size of the template. An amplitude value of 1.5, for instance, thus indicates the detected EP to be 1.5 'times as big' as the template, and so on. Bin width = .15).

invariant that motivated the research presented in this section. A corollary of this reasoning is that a failure to demonstrate increased variance of the SN distribution implies that significant EP amplitude information will not be forthcoming. This corollary is supported by the fact that the only subject (LB) not to demonstrate such an increase is also associated with the lowest N1-P2 amplitude/RT and N1-P2 amplitude/latency correlations.

DATA-ANALYSIS PART II: THE PROBLEM OF COMPONENT IDENTIFICATION

A recurring problem in analysing average EPs is in the definition of components that facilitate meaningful comparisons between the wave shapes of AEPs obtained under different experimental conditions. Although visual inspection of the AEP is often adequate in this regard, there are occasions when the averaged waveform is so noisy and inconsistent as to render such a facile analysis hazardous, if not impossible. The remainder of this chapter addresses this problem, exploring the potential solutions offered by factor analysis, discriminant analysis and digital filtering. All three methods are evaluated by their success in facilitating the analysis of a common data-set.

The data of this investigation were in fact generated by Experiment 11 of the thesis programme, but for our purposes here all we need note is that the EEG was recorded from a vertex placement in response to weak visual stimulation and that there were three experimental conditions, with 100, 50 and 50 stimuli in conditions A, B, and C respectively. The problem of waveshape comparison can be illustrated by reference to Fig.2.5a where the AEPs in each condition are displayed for one subject, CH. Apart

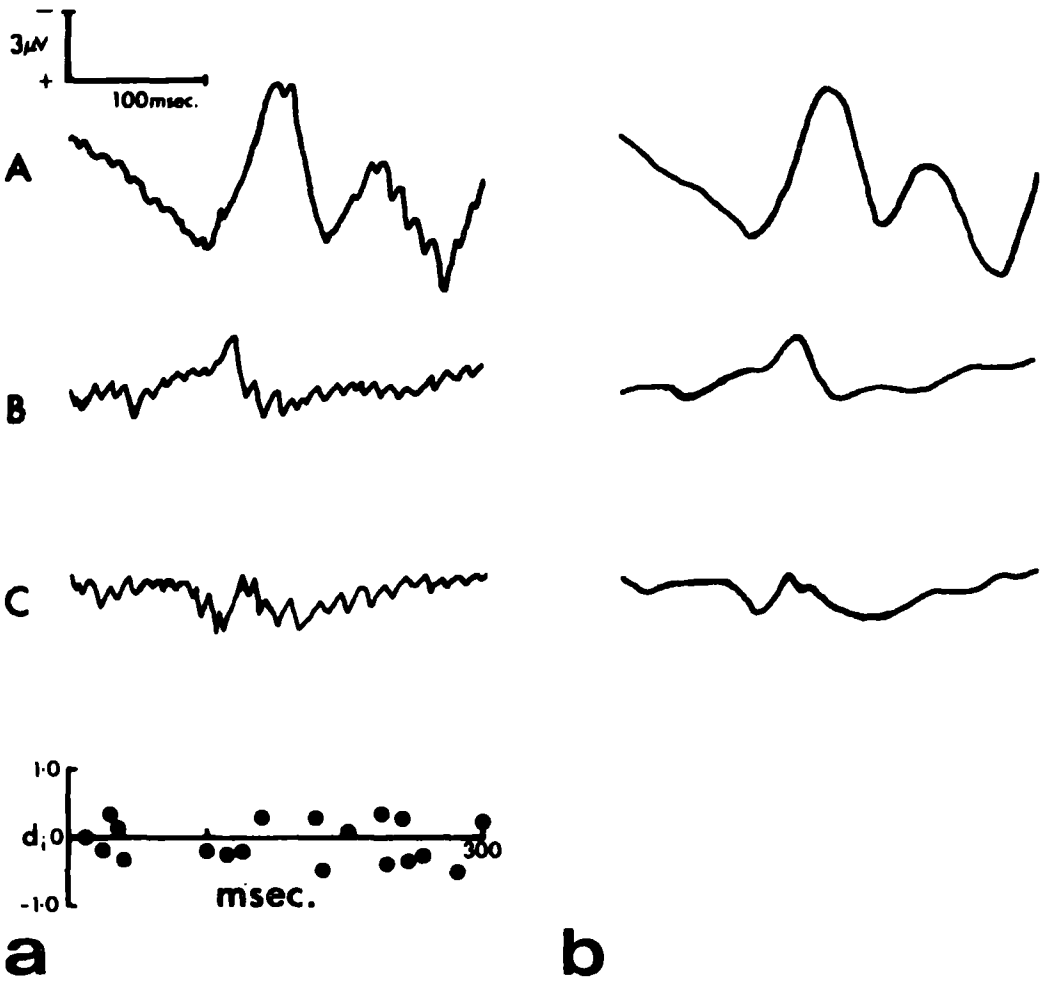

Figure 2.5

The AEPs for subject CH for conditions A, B and C and also the standardised coefficients (d_is) associated with the first discriminant function are compiled in a. The digitally filtered versions of the AEPs are shown alongside in b.

from the impression of a positive trough, peaking at around 200 ms or so, no other components can be consistently and unambiguously identified and some further objective procedure to elucidate the various waveform differences would clearly be of value.

(The major theoretical sources for the material of these subsections were Van de Geer (1971) and Chatfield (1975) for the multi-variate and time-series aspects respectively. The two multivariate analyses were performed using the SPSS (Nie, Hull, Jenkins, Steinbrenner and Bent, 1975) computer programme package.)

A Factor Analysis of Visual Evoked Potentials

THEORETICAL INTRODUCTION

Consider an experiment consisting of n stimulus presentations in which the m time-points of the EEG sampled after each stimulus are compiled in the m x n matrix, X. Post-stimulus EEG, in these terms, can be thought of as an m-dimensional observation on the m time-points. If these dimensions are correlated then factor analysis affords a technique whereby the original m-dimentions can be transformed into a smaller number of orthogonal dimensions (the factors). It was hoped that amongst this set of factors it would be possible to identify a subset of factors that were related to the evoked activity embodied in the AEP. With the AEP objectively and economically summarised in terms of these factors, its waveform description would be facilitated.

Unfortunately there is no unique mathematical solution to the above problem: the orthogonalisation can proceed in many different ways, and any resulting dimension system can then be rotated to an infinity of

different positions. One solution is to locate an initial dimension that accounts for the largest portion of the total observed variance and to select the next dimension in order to account for the maximum share of the remaining variance, and so on until all the remaining variance is exhausted. Such a procedure is known as principal components analysis and is the method used in the present investigation. Although the analysis of only one of the six subjects data is presented here, the results are typical of those found throughout.

RESULTS AND DISCUSSION

Conditions A, B and C were combined in order that the dimensions identified were common to all three conditions and, with 60 EEG time-points encompassing the range 0-295 ms post-stimulus, a 200 x 60 matrix was thus generated. Principal components analysis proceeds by computing the correlation matrix, $R = X'X/n$, the eigenvectors and eigenvalues of which represent the dimensions of the new co-ordinate system, and the variance accounted for by each dimension respectively. This initial factoring was curtailed after seven dimensions, accounting for 60% of the total variance, had been extracted.

One of the problems of the principal components solution is that it tends to produce new dimensions which correlate at a medium level with a large number of the original dimensions rather than at a high level with a small number that would be more easily interpretable. The seven factors were thus rotated on this latter principle to produce the so-called varimax solution which is presented, with the factors expressed in terms of their correlations with the original time-points, superimposed upon the

AEP in Fig. 2.6. What the solution elegantly demonstrates is, quite simply, that scalp recorded brain electrical activity represents a highly auto-correlated process such that, on average, approximately 50% of the activity over a 60 ms epoch can be accounted for in terms of a single factor. Although this result is far from novel, it is nonetheless interesting and reassuring that, despite the daunting complexities of factor analysis, it should produce what was, on reflection, the only sensible solution to the problem it was presented with. No factors that can be associated with evoked activity, as distinct from the auto-correlated noise of the EEG, can be distinguished; most likely because of the variability of such activity is small in comparison with the EEG and possibly also because it possesses similar, and therefore indistinguishable, statistical characteristics. It is conceded, in retrospect, that the rationale of this particular investigation was probably misguided, but it is considered to merit inclusion, first for the elegance of the final solution and secondly for its heuristic value.

It follows that a factor analysis of EPs would better proceed with minimal EEG noise present, i.e. with AEPs constituting the rows of the m x n matrix. Although there have been many successful studies employing such a procedure (e.g. Donchin, 1966; Suter, 1970; Van Hoek, 1974; Kavanagh, Darcey and Fender, 1976; Street, Perry and Cunningham, 1976) it would have been of limited use in the present problem where such a matrix would have only three rows corresponding to the AEPs for conditions A, B and C.

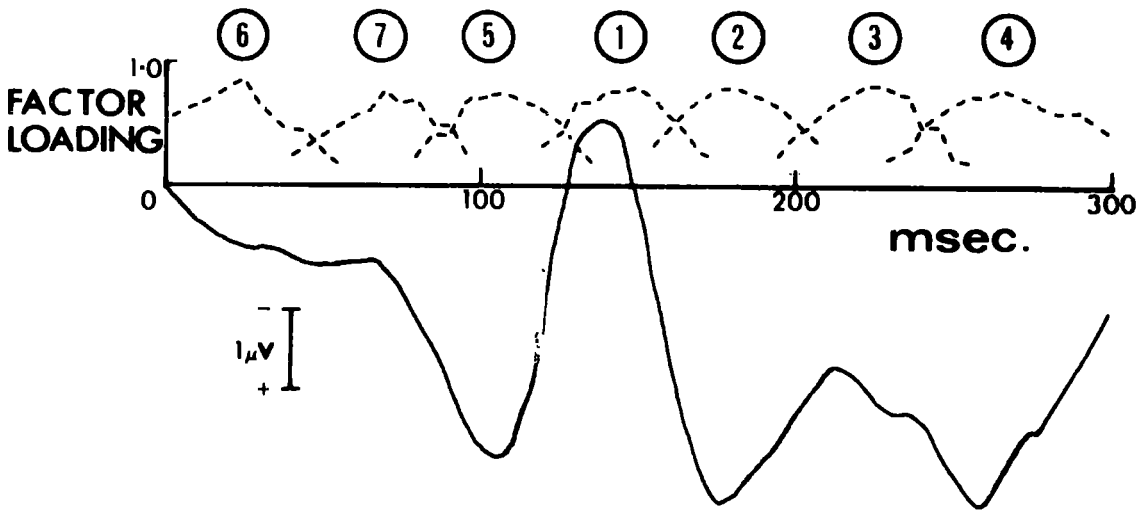

Figure 2.6

The factor loadings of the seven factors derived from the VARIMAX rotation of subject CH's data are shown, in broken lines, superimposed upon the continuous line of the average EP.

Discriminant Analysis of Visual Evoked Potentials

THEORETICAL INTRODUCTION

The second general approach to the problem of component identification involved the use of another multivariate technique, that of discriminant analysis. Again the data-set for each subject is compiled in an m x n matrix but with this technique the dimensionality reducing transformation is motivated to extract linear combinations of the m column variables (i.e. EEG time-points) that maximally discriminate between the various subgroups (in the present case, conditions A, B, C) within the matrix. These discriminant functions are of the form:

$$D_i = d_{i1} Z_1 + d_{i2} Z_2 + d_{i3} Z_3 \ldots.$$

where D_i is the score on the i^{th} discriminant function, the d_i's are weighting coefficients and the Z's are the standardised values of the m column variables. The maximum number of functions (no. new dimensions) that can be derived is one less than the number of groups, which in this example is 3-1 = 2. Although in general terms the analysis solves the eigenvector problem $WA = \lambda Ba$, where B and W are the between- and within-groups sums of squares and cross-products matrices, the following exposition will proceed at a less formal level.

A variety of methods of discriminant analysis are available, of which the present investigation employed the stepwise procedure similar to that already mentioned in connection with the work of Donchin and his group (see Introduction to Experiment One). Rather than including all the m discriminating variables (i.e. column variables, EEG time-points) in the analysis, this method selects a subset of these variables on the

basis of their discriminating power as measured on some index. The index adopted in this study was Rao's V, a generalised distance measure that reflects the overall separation of the subgroups. Discriminating variables are only retained in the analysis if their inclusion represents a significant increase in V, and thus redundant variables (EEG time-points) are eliminated.

RESULTS AND DISCUSSION

For all six subjects the analyses produced discriminant functions which consistently summarised the differences between the three conditions as mainly A being different from both B and C (the first function for all subjects) and, to a lesser degree, that B and C were also discriminable (the second function). The first discriminant function accounted for, on average, four times as much of the total variance as the second, and of the individual EPs (rows of the m x n matric) classified as belonging to condition A, on average 88% were correctly identified. The analysis was however less successful with conditions B and C, where correct detections respectively represented, on average, 57% and 56% of the so-classified trials.

Clearly the analysis indicates the major AEP waveform differences to be between condition A, and conditions B and C taken together, and in the process supports Donchin's contention (e.g. 1969b, p.209) that there is adequate information in the majority of individual EPs to correctly classify them according to source. Although discriminant analysis is manifestly a powerful and valuable technique, it suffers from the two disadvantages that characterise the application of multivariate methods

to the analysis of EP data. Firstly, it is voracious in its appetite for computer time and secondly, the final vectors are often in themselves difficult to interpret and are invariably inconsistent across subjects. This latter point is reinforced by inspection of Fig. 2.5a where the standardised coefficients ($d_i s$) associated with the first discriminant function for subject CH are shown. Any interpretation of the set of coefficients in terms of AEP topography would be highly complex and quite idiosyncratic to this one subject. A simpler and quicker data-analytic technique that reduces all subjects' data to a small number of common meaningful measures is clearly required and such a technique is presented in the final section of this chapter.

Digitally Filtered Average Evoked Potentials

Filtering the EP data was originally rejected as a means of yielding 'cleaner' and therefore more easily analysed AEPs because analogue filtering can produce considerable phase-shifting (Dawson and Doddington, 1973). However digital filters can be easily designed to eliminate unwanted high frequencies without introducing any such distortion, and digitally filtered AEPs were finally adopted as the data-base whenever a single trial analysis was not required.

Effectively the digital filtering of an AEP amounts to the 'smoothing out' of local fluctuations by the computation of a moving average. If x_t represents the unfiltered value of the AEP at time t, then the filtered value, y_t , is given by:

$$y_t = \sum_{k=-q}^{q} a_k \, x_{t-k} \qquad \text{where } (a_k) \text{ are a set of weights and } \sum a_k = 1.$$

In order to plot the gain and phase characteristics of such a filter its transfer function, $H(\omega)$, which is the Fourier transform of the weighting function, h_k, is first computed. The weights used throughout the present research were generated by the expansion of $(1/2+1/2)^{2q}$ where $q = 3$, and y_t, h_k and $H(\omega)$ were thus given by:

$$y_t = (x_{t-3} + 6x_{t-2} + 15x_{t-1} + 20x_t + 15x_{t+1} + 6x_{t+2} + x_{t+3})/64$$

$$h_k = \begin{cases} 1/64 & k = \pm 3 \\ 6/64 & k = \pm 2 \\ 15/64 & k = \pm 1 \\ 20/64 & k = 0 \\ 0 & \text{otherwise} \end{cases} \qquad \ldots\text{a}$$

and $$H(\omega) = \sum_{k=-q}^{q} h_k e^{-i\omega k}$$

$$= (2\cos 3\omega + 12\cos 2\omega + 30\cos\omega + 20)/64 \qquad \ldots\text{b}$$

$H(\omega)$ is generally a complex function of the form

$$G(\omega)e^{i\phi(\omega)}$$

where $G(\omega)$ and $\phi(\omega)$ are the gain and phase functions respectively. However the transfer function of a symmetrical weighting function, such Equation a, happens to be real (Equation b), and so the phase is given by:

$$\phi(\omega) = 0$$

i.e. no phase distortion is introduced by the filter. The gain function for the h_k of Equation a is thus simply given by the expression for $H(\omega)$ of Equation b and is plotted in Fig. 2.7a. For a discrete process, such as the AEP, the maximum frequency that can be resolved is .5 cycles, i.e. π radians, per unit time; and thus the abscissa of the gain

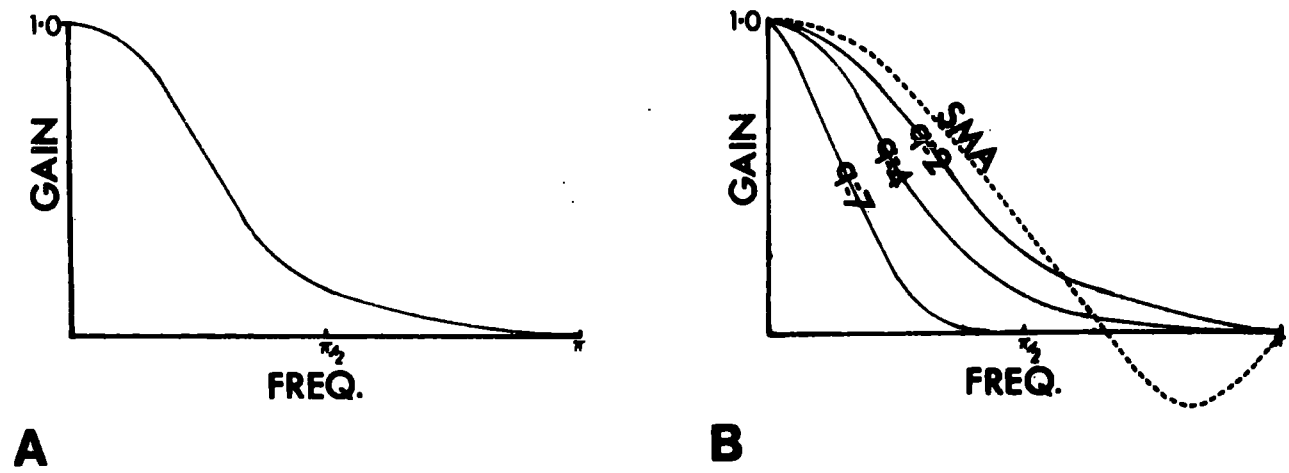

Figure 2.7

Gain functions of the filters based on the weights generated by the expansion of $(\frac{1}{2} + \frac{1}{2})^{2q}$ with q = 3(A); 2,4 and 7(B). The gain function of the simple moving average (SMA) described in the text is also shown in B.

diagram is limited to the frequency range 0 to π . For a sampling interval of Δt this maximum frequency, the so-called Nyquist frequency, is given by $1/2\,\Delta t$. For the sampling rate of 1pt/5 ms generally employed in this research, this means that the filter effectively eliminates frequencies above 50 cps ($\pi/2$), increasingly attenuates those above 15 cps and largely preserves those below 15 cps, i.e. the frequency range of the late components of the AEP.

Fig. 2.7b shows the gain functions of the filters based on the weights generated by the expansion of $(1/2+1/2)^{2q}$ with q = 2,4,7 and it can be seen that the expansion is generating a family of curves of similar shape with the desirable characteristic of positive gain throughout. In contrast the gain function of the simple moving average given by:

$$h_k = \begin{cases} 1/3 & k = -1,0,1 \\ 0 & \text{otherwise} \end{cases}$$

is also plotted and can be seen to go negative at high frequencies, which effectively amounts to the introduction of a 180 degree phase shift.

When the digital filter of this section is applied to the AEPs of Fig. 2.5a the 'clean' waveforms of Fig. 2.5b are produced. The high frequency noise contaminating the unfiltered record, complicating component identification and measurement, is effectively abolished by the filter without significantly distorting the latencies and amplitudes of the AEP components. With the difficulties in component identification thus diminished, positive troughs at around 100 (P1) and 200 ms (P2), and an intermediate negative peak (N1), are readily identifiable. As already indicated, this data-analytic procedure of averaging and smoothing was the one finally utilised for the data of this study, and is also employed extensively throughout the thesis research.

Data Analysis: Summary and Concluding Comments

Part I of this survey of data analytic techniques relevant to EP research demonstrated the efficacy of a simple pattern recognition technique, based on the cross-covariance function, in extracting the amplitude and latency of the N1-P2 wave of the EP from individual trials. In certain areas, such as the investigation of non-stationary processes (including the habituation of the EP), single trial information may be vital, and this technique should therefore prove of considerable value.

However, where signal averaging is considered to be a satisfactory expedient, Part II recommends the use of digital filtering to remove unwanted high frequency noise, thus facilitating component identification. Two multivariate techniques are also evaluated (namely, factor analysis and discriminant analysis), but the difficulty in interpreting the final vectors in such solutions is considered to render them of limited value. Following these recommendations the thesis research thus, in the main, adopts digitally filtered AEP s as its data-base, unless a single trial analysis is specifically required.

Footnotes - Chapter Two

(1) Pfurtscheller and Cooper (1975) suggest the use of correlation procedures in selective averaging; the present treatment would provide a useful framework for describing and manipulating the selection procedure.

(2) In essence, the blocking procedure involves the computation of a correlation coefficient based on a number of pairs of means, the first of which is the mean RT for a particular block (based on the chosen subdivision of the distribution of RT's; quartile, 'quintile', 'decile' or whatever) and the second, the associated AEP-derived measure, also a mean. Designating the individual values underlying these means, X and Y (in the present case, the single trial RT and N1-P2 data respectively) and assuming for simplicity, and without any loss of generality, that both X and Y are standardised, then, where ρ expresses the population correlation between X and Y and μ_b represents the underlying mean value of X for the block (quartile, etc.) of X in question, then we have, for any particular block:

$$E(X) = \mu_b$$

and $$E(Y|\ \mu_b) = \rho\mu_b = \rho E(X)$$

Thus, given n X values in a block, whose mean stabilises with increasing n at $\bar{X}$ (μ_b), the mean of the associated Y values, $\bar{Y}$, converges upon $\rho\bar{X}$. The computed correlation, r, between a number of pairs of $\bar{X}$ and $\bar{Y}$, based upon a large n, is thus given by:

$$r = E(\bar{X}\bar{Y})/\sqrt{VAR(X)VAR(Y)}$$

$$= \rho\, E(\bar{X}^2)/\sqrt{E(\bar{X}^2)E(\ [\rho\bar{X}]^2\ )}$$

$$= \pm 1$$

Intuitively, the effect of blocking and basing correlations on the block means is to increasingly eliminate the error associated with the pairs of observations entering into the computation of the correlation coefficient. Thus the proportion of the total variance explainable in terms of the linear correlation between the two variables, i.e. ρ, increases with n until, ultimately, all the variance is so-explained. At this point it should be noted that this footnote assumes that both covariates are normally distributed, and thus that any relationship between them is exactly linear (Kendall, 1947), i.e. completely described by their correlation coefficient. It is appreciated that certainly reaction times are not generally normally distributed and thus, although the general argument that blocking enhances r, still holds, the asymptotic value of r may be less than ± 1 depending upon the extent of the non-linear component in the relationship between the covariates.

(2) Cont...

However, with one distribution essentially normal (i.e. the EP-derived measure, either N1-P2 amplitude or N1-P2 latency) and the other only slightly non-normal (reaction time) this latter component is not likely to be substantial and the asymptotic value of r will remain close to ± 1.

Thus the blocking procedure commonly employed in RT and EP studies will generate correlations that, at a rate that will depend upon VAR(X), VAR(Y) and ρ , inevitably increase with increasing n, approaching some asymptotic value close to ± 1. Correlations so-derived are thus misleading as quantitative estimates of the underlying relationships and are therefore most prudently ascribed only qualitative status.

To reinforce this footnote the correlation between N1-P2 amplitude and RT was recalculated for one subject, GJ, analoguing the procedure used by Morrell and Morrell (1966). These authors divided the RT distributions for each subject into quartiles and computed the AEP for each quartile. The paired observations that entered into the computation of r consequently represented the means of, in fact, 60-62 scores. Analogously, for GJ's single trial data the mean RT's and amplitudes for each RT quartile (50 trials) were computed and are compiled in the following table:

RT (ms)	N1-P2 amplitude (arbitrary units)
214	67
188	89
173	108
156	113

Although the underlying correlation between the two covariates is in fact -.39 (see Table 2.51), the blocking procedure generates a Pearson product-moment correlation coefficient of -.98.

(The material of this footnote has been largely endorsed by Seheult, A.H and Hayes, R.J. of the Mathematics Department at Durham, and a joint publication is in preparation.)

(3) These averages were computed for descriptive purposes only. With a maximum standard error of .07 ($1/\sqrt{200}$) associated with the individual coefficients they are clearly not homogeneous and the average coefficient is thus not to be considered as an estimate of a common population value.

CHAPTER THREE

SELECTIVE ATTENTION AND EVOKED POTENTIALS

PART I: VISUAL SELECTIVE ATTENTION

The neuropsychological model of attention advanced in Section 2 of Chapter 1 proposes that selective attention is subserved by a pre-cortical gating system regulating the activation of the non-specific cortex by a stimulus. The amplitude of the N1 wave of the EP was argued to reflect the degree of this activation and hence to index the operation of the selective machinery. Although this model is intended to be generally applicable to all sensory modalities, convincing EP evidence for a pre-cortical gating system is restricted to experiments, such as those of Hillyard and his group (Hillyard, Hink, Schwent and Picton, 1973; Schwent and Hillyard, 1975; Schwent, Snyder and Hillyard, 1976; Hink and Hillyard, 1976), that employ an analogue of the dichotic listening pardigm to investigate auditory selective attention. What few studies of visual selectivity there are generally fail to completely exclude the possibility that the subject, at least sometimes, knew approximately when the relevant stimuli would be presented. With reference to a typical study (Donchin and Cohen, 1967), Näätänen (1975, p.249) comments that "the enhancement reported could ... [thus] ... be due to (non-specific) differences in the state of the organism between the moments of occurrence of the two kinds [attended and unattended] of stimuli." One study, however, that does satisfy all Näätänen's criteria, including the requirement that peripheral receptor orientation be under experimental control, was conducted by Harter and Salmon (1972).

In their experiment, subjects were required to selectively attend (either by counting or producing a reaction time)to one out of a pair of visual stimuli that were presented in a random sequence at a rate of approx. 1 or 2 stimuli per second. Four such pairs of stimuli were used in order to vary the extent to which the relevant and irrelevant stimuli fell on identical retinal points. The pairs (described by Harter and Salmon as problems I to IV) were red and blue colours; vertical and horizontal bars; a blue colour and crossed bars; and a circle and a square. A significant enhancement of the occipital EP with attention was obtained and, although there was an interaction between the magnitude of this enhancement and problem type, the effect of attention was least pronounced for those pairs of stimuli with greater disparity in their retinal projections. This latter result strongly suggests that, even if subjects did stray from the central fixation point to facilitate selection, these peripheral changes were not responsible for the EP effects. This finding, in conjunction with their use of a random presentation sequence, leads them (p.610) to the reasonable conclusion that "the effects of attention cannot be attributed to transient changes in arousal level preceding the stimuli ... or peripheral orienting factors, such as visual fixation, or both."

After making this methodological point they go on to dismiss Karlin's (1970) 'reactive change' hypothesis (i.e. that attention-related EP enhancements at longer latencies than the N1 component are most reasonably interpreted as reflecting the drop in non-specific arousal occasioned by the relevant stimulus) as a possible explanation of their

results, pointing out that their earliest effects occurred only "slightly later" than Karlin's N1 deadline. However this author cannot agree that a negative wave peaking at between 225 and 250 ms post-stimulus is only slightly later than the typical N1 latency of around 140 ms, and their final conclusion (p.611) that the amplitude enhancement of this 'early' component reflects the modulation of "sensory impulses ... in the peripheral nervous system" is regarded as bizarre.

The research to be presented in Part I of this chapter was motivated by the desire to demonstrate an unequivocal EP correlate of intra-modality visual selective attention analagous to the correlate already established in the auditory system. It was anticipated that an enhancement of the N1 component to attended stimuli would be found; an enhancement that could reasonably be interpreted, in terms of the neuropsychological speculations of Chapter One, as reflecting the gating of the visual sensory array at the reticular level.

Experiment 3: Evoked Potential Correlates of Visual Selective Attention with Attended and Unattended Channels defined in terms of Stimulus Intensity

INTRODUCTION

Drawing on the work of Näätänen (1967, 1975) and Schwent, Hillyard and Galambos (1976a,b), it was considered that an adequate experimental procedure for investigating visual selective attention should satisfy the following requirements:

a) The arrival of stimuli over the attended and unattended channels must be completely randomised.

b) The subject must be subjected to a high processing load.

Schwent et al (1976a,b) have demonstrated that the failure

of directed attention to influence the auditory vertex EP, as reported in several laboratories, is probably a consequence of the long ISI (greater than .5 sec) used. At such rates of stimulation they make the reasonable claim that subjects would find it difficult to avoid attending to all the sensory channels; only at high processing loads, when rejection of irrelevant material becomes imperative if the task is to be managed, will selectivity be manifest. Schwent et al in their two studies increase processing load in three ways: firstly, by shortening ISI; secondly, by decreasing stimulus intensity; and finally, by increasing the level of background noise. In all cases the effect of attention upon EP amplitude increased as a function of load, and indeed was minimal at the lowest loads. In the present study a high processing load was achieved with a combination of a difficult task and the minimal ISI for its successful performance. More specifically, the attended and unattended channels were defined in terms of stimulus intensity, with subjects being required to count the number of stimuli at one intensity level, whilst ignoring those at the other. The stimuli within both streams were identical, comprising in each case a sequence of ones and zeros. The task thus required the subject to discriminate between two stimuli in the attended stream and accordingly update the appropriate one of the two ongoing totals. The demands of the task were thus exacting and, indeed, preliminary work indicated that the fastest stimulus presentation rate at which the subject could successfully manage it was 2 per second.

c) Peripheral receptor orientation must remain constant throughout.

It is conceded that this restriction is unnatural in the sense that the peripheral mechanisms of head and eye movements are pre-eminent in selective looking; Haber and Hershenson (1973, p.229) comment that they are "the most important means by which we select one part of the visual world over another part to attend to." However the involvement of such mechanisms would necessarily make interpretation of any EP amplitude effects as evidencing thalamic gating difficult to sustain. By using the same stimuli (differing only in their intensity) on both channels, it was hoped to satisfy this final requirement.

METHOD

EEG was recorded monopolarly from the vertex of the five subjects who participated in the experiment. A 5 x 3 matrix of red light emitting diodes (LEDs) was mounted 1 metre from the subject at the far end of a dimly illuminated tunnel and subtended an angle at the eye of approximately 1 degree. The apparatus is depicted in Fig. 3.1.

The LED matrix was controlled by the transistor outputs of the departmental IBM 1130 computer and was used to present a sequence of ones and zeros of either a high or low brightness level. The subject's task was to count the number of 1s and the number of 0s of the intensity specified for the current block of trials. Eight blocks of trials, each block consisting of the randomised presentation of 20 of each type of stimulus (i.e. 80 trials per block), constituted the experimental session. On four of the blocks the subject was instructed to count high intensity

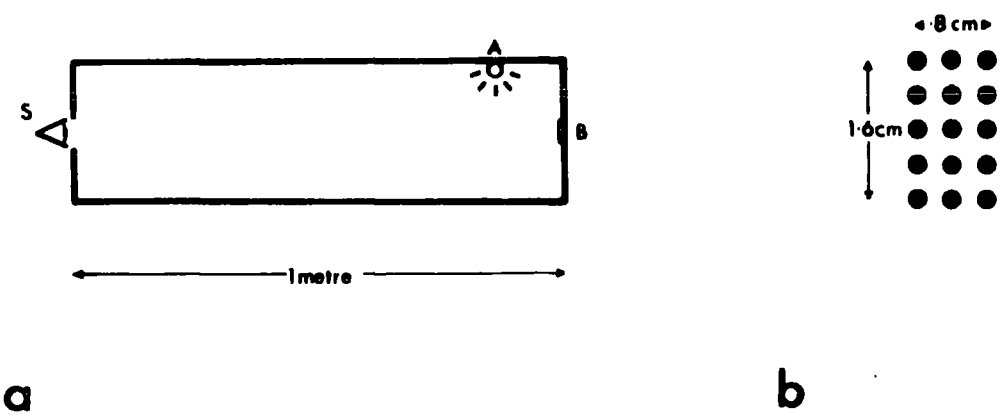

Figure 3.1

The apparatus of experiments 3 and 4 is depicted in a. The 5 x 3 LED matrix (B) is viewed by the subject (S) along a one metre long tunnel, dimly illuminated by the light A. The dimensions of the matrix are indicated in b.

1s and 0s (attend hi condition) and on the remaining blocks to count the low intensity stimuli (attend lo condition), the order of the conditions being counter-balanced within subjects. A constant ISI of 500 ms was used as already indicated, and during this interval the central LED of the matrix remained illuminated. Subjects were required to fixate this point throughout. Before each block began subjects were given a run of practice trials which they terminated when they felt capable of performing the task; and at the end of each block the subject was asked to report the number of 1s and 0s counted. These two totals were noted.

Upon completion of the experiment subjects were asked to describe their relative level of awareness of the unattended stimuli in relation to the attended ones. All subjects reported that they were only fleetingly aware of the 'dim' stimuli on the attend hi blocks, but were fully able to 'see' the dim stimuli when they were looking for them (attend lo condition). On the other hand no corresponding phenomenological attenuation of the bright stimuli on the attend lo condition was reported. These psychological data were regarded as consistent with Schwent et al's (1976b) theorising that selectivity will be reduced at high stimulus intensity levels, and it was anticipated that any effects of attention upon the EP would show a corresponding interaction with stimulus intensity.

RESULTS

Eight average EPs[1] were computed for each subject representing the brain response to each of the four types of stimulation (high intensity, 1, hi 0, low intensity 1, lo 0) under each attention condition. As the

experimental session comprised 640 trials, each of these AEPs was thus based on 80 trials. AEPs for one of the subjects are presented for illustrative purposes in Fig. 3.2. Four measures were derived from these averages: the amplitude and latency of the negative wave peaking at an average latency of 122 ms (N1), and the amplitude and latency of the subsequent positive deflection that peaked on average 78 ms later (P2). A three factor repeated measures analysis of variance (attend vs nonattend, hi vs lo intensity, ones vs zeros being the three factors) was conducted for each measure, with the means expressing the main effects of attention, intensity and spatial structure upon each measure, and the four analysis of variance summary tables being compiled in the five sub-sections of Table 3.1 (for key to significance levels and further presentation details, see Section 1 of the preceding chapter).

Considering N1 amplitude and latency first, neither the main effect of attention nor the interaction between attention and intensity approach significance ($F < 1$ in both cases). It does, however, appear that the N1s evoked by less intense stimuli are both smaller in amplitude ($F(1,28) = 6.51^{*}$) and longer in latency ($F(1,28) = 24.57^{***}$) and there is also evidence that 1s evoke a shorter latency N1 than do 0s ($F(1,28) = 4.33^{*}$). All remaining main effects and interactions in both analyses are non-significant.

A significant main effect of attention ($F(1,28) = 5.18^{*}$) upon the amplitude of the P2 component is revealed by the analysis, with attention producing a .69μV enhancement of the amplitude of this component. No other main effects or any of the two-way interactions

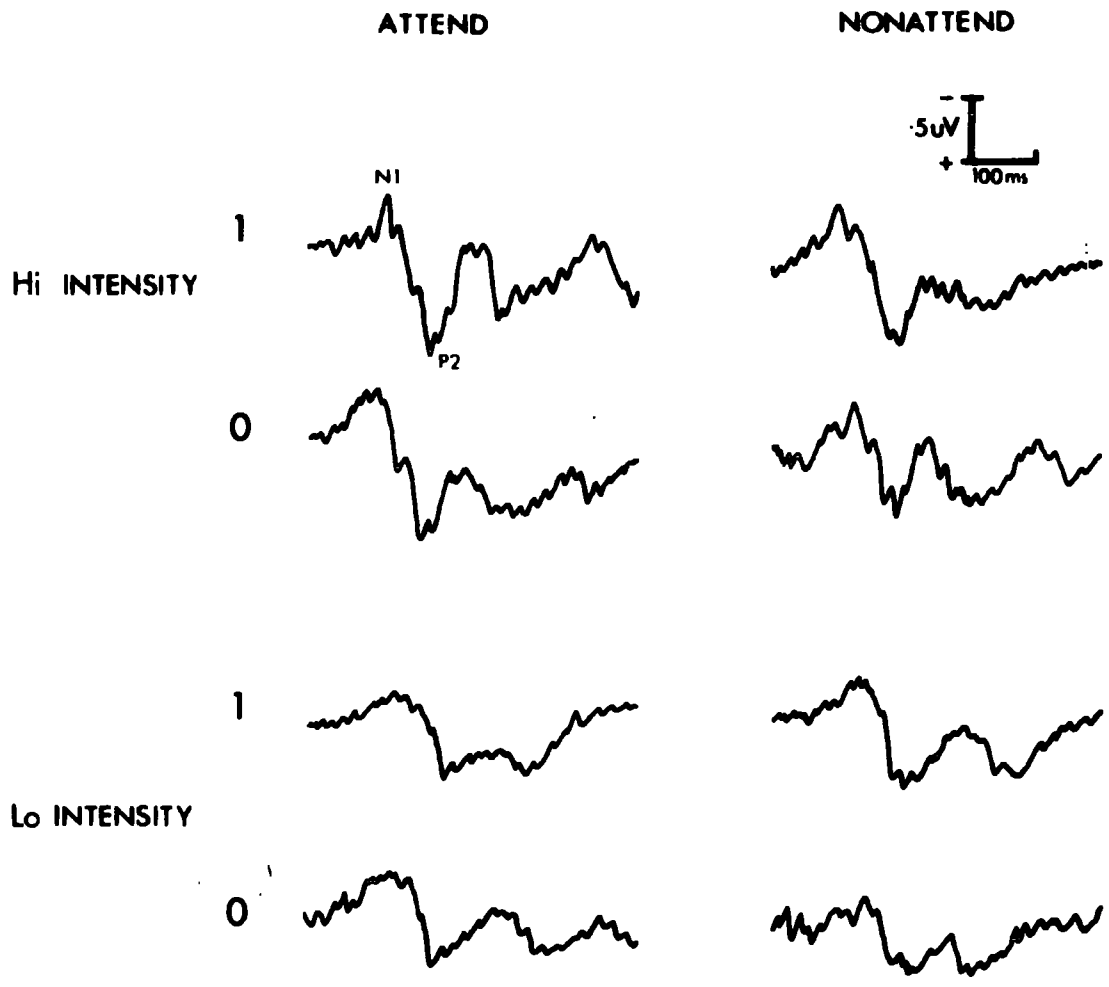

Figure 3.2

Experiment 3: AEPs for subject NS presented as a function of attention condition, intensity and spatial structure.

TABLE 3.1: EXPERIMENT 3

i) Mean EP (N1,P2) amplitude (μV) and latency (ms) as a function of attention (ATT - attend, IGN - nonattend) stimulus intensity (HI,LO) and spatial structure (1,0).

	ATTENTION		INTENSITY		STRUCTURE		
	ATT.	IGN.	HI	LO	1	0	$\bar{X}$
N1 amplitude	1.52	1.27	1.75	1.04	1.55	1.24	1.39
P2 amplitude	5.93	5.24	5.68	5.50	5.80	5.38	5.59
N1 latency	124	120	109	135	117	128	122
P2 latency	204	197	185	216	196	204	200

ii) Summary of the analysis of the effects of attention (A), intensity (I) and structure (S) upon N1 amplitude. For key to significance levels see Section 1 of Chapter 2.

Source	df	SS	MS	F
Subjects	4	52.07		
A	1	.64	.64	<1 ns
I	1	5.13	5.13	6.51 *
A x I	1	.17	.17	<1 ns
S	1	1.00	1.00	1.27 ns
A x S	1	.17	.17	<1 ns
I x S	1	.12	.12	<1 ns
A x I x S	1	2.58	2.58	3.28 ns
Error (pooled **)	28	22.02	.79	
Total	39	83.90		

iii) Summary of the analysis of the effects of attention (A), intensity (I) and structure (S) upon N1 latency.

Source	df	SS	MS	F
Subjects	4	14,242.4		
A	1	144.4	144.4	<1 ns
I	1	6,864.4	6,864.4	24.57 ***
A x I	1	90.0	90.0	<1 ns
S	1	1,210.0	1,210.0	4.33 *
A x S	1	0.4	0.4	<1 ns
I x S	1	608.4	608.4	2.18 ns
A x I x S	1	115.6	115.6	<1 ns
Error (pooled **)	28	7,822.08	279.36	
Total	39	31,100.4		

iv) Summary of the analysis of the effects of attention (A), intensity (I) and structure (S) upon P2 amplitude. The table of means associated with the significant A x I x S interaction is also presented, with P2 amplitude expressed in μV. See i) for key.

Source	df	SS	MS	F
Subjects	4	179.72		
A	1	4.68	4.68	5.18 *
I	1	.32	.32	<1 ns
A x I	1	2.19	2.19	2.42 ns
S	1	1.76	1.76	1.95 ns
A x S	1	.68	.68	<1 ns
I x S	1	.06	.06	<1 ns
A x I x S	1	4.57	4.57	5.06 *
Error (pooled **)	28	25.29	.90	
Total	39	219.27		

ATT:

	1	0
HI	5.49	6.08
LO	6.53	5.62

IGN:

	1	0
HI	6.21	4.93
LO	4.96	4.88

v) Summary of the analysis of the effects of attention (A), intensity (I) and structure (S) upon P2 latency.

Source	df	SS	MS	F
Subjects	4	9,617.6		
A	1	409.6	409.6	1.78 ns
I	1	9,486.4	9,486.4	41.26 ***
A x I	1	129.6	129.6	< 1 ns
S	1	640.0	640.0	2.78 ns
A x S	1	102.4	102.4	1.02 ns
I x S	1	40.0	40.0	< 1 ns
A x I x S	1	360.0	360.0	1.57 ns
Error (pooled **)	28	6,438.0	229.93	
Total	39	27,225.6		

(including the interaction between attention and stimulus intensity alluded to in the final paragraph of the method section) are significant, but the three-way interaction, with an F of 5.06 (df = 1,28), does achieve significance at the 5% level. As this interaction possesses no intrinsic interest and with its interpretation difficult and complex, in the interests of a simple discussion it will not be considered any further. It is nonetheless presented for the reader's inspection in section iv of Table 3.1.

Turning finally to P2 latency it is noted that no attention related latency changes, accompanying the amplitude enhancement, are present ($F(1,28) = 1.78$ ns). Indeed the latency of this component behaves more in the manner of both N1 amplitude and latency in being primarily sensitive to the effect of stimulus intensity ($F(1,28) = 41.26^{***}$). Neither the remaining main effect nor any of the interactions are significant.

DISCUSSION

The results of this experiment, although failing to reveal either a significant main effect of attention or an interaction between attention and stimulus intensity upon N1 amplitude, demonstrate an interesting dissociation between this component and the subsequent positive deflection of P2. Whereas N1 amplitude and latency are influenced by stimulus intensity, with less intense stimuli evoking smaller and later potentials (a result in accord with the literature; Regan, 1972), P2 is not. Conversely P2 amplitude, unlike N1, does appear to index selective attention. The most reasonable interpretation of this dissociation is that N1, being earlier, reflects the earlier

mental events of perceptual processing that would differentiate between intense and less intense stimuli but not necessarily between attended and unattended ones. Post-perceptual processing would, on the other hand, be expected to reflect more of the relevance of stimuli rather than their perceptual content and ipso facto so would any concurrent brain activity, such as is indexed by P2. The observation that the latency of P2 is influenced by stimulus intensity does not contradict this hypothesis for the reason that any delays in the earlier perceptual stages of processing would inevitably be expected to delay all subsequent stages. This reasoning remains valid providing that no significant additional delay in the latency of P2 can be demonstrated, a stipulation that the data appears to support with the delay of 30.8 ms in the latency of P2 being only 4.6 ms longer ($t(df = 4) = 1.38$ ns) than the delay of 26.2 ms in the occurrence of N1 to the low intensity stimuli.

Further discussion of these results will be deferred until the discussion section of the next experiment wherein the implications of the results of both experiments for models of visual selective attention will be discussed together.

Experiment 4: Evoked Potential Correlates of Visual Selective Attention with Attended and Unattended Stimuli defined in terms of Spatial Structure

INTRODUCTION

Although no particular features of experiment 3 were identified as central to its failure to demonstrate an N1 amplitude correlate of visual selective attention, a second experiment was conducted which

exhibited the following modifications:

a) The attended and unattended stimuli were defined in terms of their patterning rather than their intensity. Considering the general psychological literature, selective processes in vision have very largely been investigated in relation to the spatial structure of stimuli, that is in terms of searching for particular letters or forms (Haber and Hershenson, 1973). Although colour has also been employed by some investigators (most notably, Neisser, 1969; Willows and McKinnon, 1973) as the basis of selection, the use of stimulus intensity, as in experiment 3, is a somewhat unusual procedure. The present study, therefore, investigates visual selective attention in a context where selection has been thoroughly investigated, i.e. in relation to character recognition. In short, subjects were required to detect the presence of a single target letter in a stream of letters presented successively in the visual field using the apparatus of experiment 3. As such this experiment is directly related to a long tradition of psychological experimentation, and in particular to the work of Neisser and his various co-workers (e.g. Neisser, 1963; Neisser and Beller, 1965; Neisser and Stoper, 1965). One disadvantage, however, in this adaptation of Neisser's visual search paradigm as against the procedure of experiment 3 is that, with the subject 'looking for' different patterns, his fixation point might vary across conditions. It was considered that with the visual display subtending only 1 degree at the eye, and with a central fixation

point remaining on throughout the experiment, any such variations would be minimal.

b) With the subject being simply required to detect and count a single target item in an ongoing stream, a faster presentation rate than that employed in experiment 3 was necessary in order to induce the high information load that is a necessary condition for producing an effect of selective attention upon N1 amplitude (Schwent et al, 1976a, b). The achievement of an effective processing load by this device is a more proven method than the use of the demanding task in experiment 3, thus representing a further potential improvement over this earlier design.

METHOD

Six subjects participated in the experiment and their EEG was recorded monopolarly from two electrode placements, one at the vertex (Cz) and the second positioned at a point midway between the two occipital lobes (O). The apparatus of experiment 3 (see Fig. 3.1) was used to present a random sequence of the eight letters D, E, F, H, I, O, T and U at a rate of 3/sec. The experiment was comprised of two such sequences of 256 stimuli each (yielding on average 32 relevant stimuli per sequence) with subjects being required to count the number of Is and Es in one sequence and the number of Es in the other. The choice of Is and Es, although somewhat arbitrary, was because, with the apparatus used (that is the 5 x 3 LED display) they were easily recognised and were rarely confused with other letters. The order of presentation of the two sequences was balanced across subjects and practice trials given in

the manner of the previous experiment. It should be noted that unlike experiment 3, where the sequences of stimuli were random permutations of 20 of each of the 4 stimulus types, the sequences in this experiment were completely random. Although this inevitably meant that varying numbers of attended and unattended stimuli would occur in each sequence, this procedure was adopted as it removed a possible basis for the subject to predict the stimuli above chance level.

At the end of each sequence subjects were again asked to indicate the number of relevant stimuli they had counted and their level of awareness of the unattended material. No reliable preference for either the attend-I or the attend-E condition was expressed by the subjects, a finding corroborated by the identical mean error scores of 3.4 (11%) for the two conditions (error being indexed by the absolute discrepancy between the total reported by the subject and the actual number of target stimuli). Subjects were invariably largely unaware of the irrelevant stimulus material in the sense that they could only imprecisely recall the content of the unattended letter set, and even when unattended stimuli intruded into consciousness the number of such occurrences was but vaguely recollected.

RESULTS

Eight digitally filtered average EPs (based on, on average, 32 stimuli each) were calculated for each subject, representing the brain activity evoked by the Is and Es under each attention condition at each electrode placement. For both occipital and vertex AEPs a negative peak (N1) and a subsequent positive through (P2) were identified in all

records and the amplitudes of these components were measured.

Remembering that the experiment comprised two blocks of stimuli, with Is being attended in one block and Es in the other, the following index was adopted in order to express the effect of selective attention upon EP amplitude (AMPL.) for each subject's data:

$$A = (\text{AMPL. attended I} + \text{AMPL. attended E})/2 - (\text{AMPL. nonattended I} + \text{AMPL. nonattended E})/2$$

Not only is the analysis simplified by combining the EP data for Is and Es in this way, but the effect of any general state change (which might add a constant amount to the EP amplitude for all stimuli in a particular block) between blocks is also eliminated.

The effect of attention was thus calculated for both EP components (N1,P2) at each recording site (C_z,O) and evaluated using Student's t-tests (Table 3.2). No effect of attention upon the N1 component at either location was found ($t(df = 5) = 1.28$ ns; $t(df = 5) = -.25$ ns), but, following experiment 3, P2 is enhanced with attention at the vertex ($A = 2.08\mu V$; $t(df = 5) = 4.83^{**}$). There is also a corresponding, but diminished, effect of attention upon the occipital P2 ($A = .83\mu V$; $t(df = 5) = 7.31^{***}$). Although not shown, a further t-test indicated this interaction with electrode location to be reliable ($t(df = 5) = 3.49^{**}$). In order to illustrate these results the four composite AEPs, representing the brain activity collapsed across the six subjects evoked by the attended and nonattended stimuli at each electrode location, were computed and are shown in Fig. 3.3.

One problem in the design of this experiment and the use of the above index of selective attention is that the experimental model is

TABLE 3.2: EXPERIMENT 4

Mean N1 and P2 amplitudes (in μV) to attended Is and Es (ATT) and nonattended Is and Es (IGN) at the vertex and at the occiput. The results of Student's t-tests evaluating the effect of attention are also shown (one tailed test).

	VERTEX		OCCIPUT	
	N1	P2	N1	P2
ATT	.14	2.75	1.15	1.13
IGN	.78	.67	1.23	.30
t,df = 5	-1.28 ns	4.83 **	-.25 ns	7.31 ***

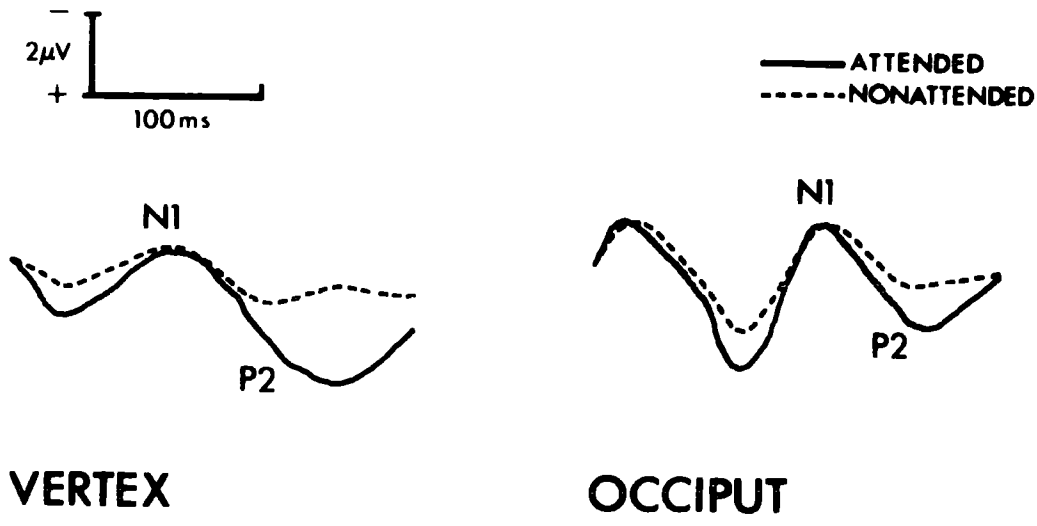

Figure 3.3

Experiment 4: Composite vertex and occipital AEPs associated with attended and non-attended stimuli.

assuming no order effects interacting with the effect of attention between block 1 and block 2. However, such effects are intuitively likely: once a subject has been attending to, say, Is in block 1, for instance, it is not reasonable to assume that they then become just as ignorable in block 2 as the Es were in block 1. In order to assess such effects the data were reanalysed, restricting the analysis to the first block of each subject's data. The effect of attention was thus re-estimated by:

A' = AMPL. attended stimulus block 1 - AMPL. nonattended stimulus block 1

Although this meant that any effects of attention were necessarily confounded with EP amplitude differences due to the different physical characteristics of the attended and nonattended stimuli in any individual subject's data, these latter differences would be cancelled out in the group mean. Statistical analysis of these data revealed essentially the same results as above, with no effect of attention upon N1 amplitude at either the vertex ($t(df = 5) = -.82$ ns) or the occiput ($t(df = 5) = -1.29$ ns). A significant enhancement of P2 amplitude at the vertex ($A' = 2.17\mu V$; $t(df = 5) = 2.21^*$) is again obtained, although the smaller occipital effect disappears ($t(df = 5) = .17$ ns). Thus, although the attention related enhancement of P2 at Cz and the null results for N1 appear to be robust with respect to order effects, the enhancement of P2 at O should be treated with some caution.

DISCUSSION

The results of this experiment confirm those of experiment 3 with selective attention to a visual informational channel being indexed by

the amplitude of the P2 component of the visual EP, but not by the amplitude of the N1 component. It is further shown that this effect of attention is essentially restricted to the more anterior placement of a vertex electrode, suggesting that the allocation of the processing capacity of the more frontal brain regions is less immutable than more posterior areas. Although of interest, further discussion of this interaction is, however, postponed until Chapter Seven.

The theory presented in Chapter One and the introduction to the experiments of this chapter argues that selective attention is accomplished by the pre-cortical gating of non-specific cortical input and that the amplitude of N1 indexes this process. Broadbent (1970) describes this mode of attention as 'stimulus set' connoting its concern with the early perceptual stages of analysis and distinguishing it from the subsequent 'response set' mode in which specific signals within the attended stream are recognised. However, although the evidence of experiment 3 indicates N1 to be sensitive to perceptual variables, neither experiment 3 nor 4 demonstrate any relationship between N1 and stimulus set; and the suggestion, therefore, is that pre-cortical gating of the visual sensory array does not occur. This discrepancy between the auditory and visual EP data questions the generality of the neuro-psychological model of Chapter One Section 2, and indeed is consistent with the aforementioned pre-eminence of peripheral mechanisms in selective looking which implies that central gating mechanisms are less crucial in the visual system, and possibly not even necessary at all. However, Haber and Hershenson (1973, p.245) certainly believe that central selective

processes do operate upon visual information and it is thus possible that the failure of the EP data to evidence gating could be more a reflection of the sensitivity of the EP as an index of brain function in this context than indicating the absence of such a mechanism. A related point is that the deepening of P2 may be reflecting an overall positive shift upon which the enhancement of a superimposed N1 component could have been obscured.

Whether or not the organism is operating a visual stimulus set in the experiments of this chapter, relevant and irrelevant stimuli certainly receive differential processing at some stage. It is proposed that the P2 correlate of selective attention indexes this differential processing. Drawing tentatively on the principles of nervous system function as espoused by Arbib (1972), which suggest that neural information processing is achieved by successive transformations of patterns of neural activity, it is proposed that post-perceptual information processing is accomplished by inhibitory interactions between cortical columns (which produce the surface positivity of P2) which transform and process the initial pattern of cortical activation embodied in N1. The degree of these interactions reflects the depth of processing, and hence task relevant stimuli evoke larger amplitude P2s. In simple terms, given the long latency of P2, its amplitude is regarded as being more related to Broadbent's 'response set' mode of attention which necessarily distinguishes between task relevant and irrelevant stimuli. It is pertinent to note that Hillyard (e.g. Hillyard et al, 1973 , p.179) makes this same distinction between N1 and subsequent positivity, although in his case

he implicates P3 as indexing 'response set'.

Finally, it is recognised that, although the metatheory of the thesis relates both N1 and P2 to the utilisation of processing capacity by a stimulus, i.e. cognitive factors, Karlin's non-cognitive emplanation for long latency (i.e. post N1) attention related EP effects, that they represent a reactive change in some preparatory brain state (such as a drop in non-specific arousal or alertness) occasioned by the occurrence of a relevant stimulus, is clearly applicable to the P2 correlate of this study. Karlin is further supported by the findings of Chapter Seven of the thesis which demonstrate that P2 coincides with the resolution of the CNV and can be considered to be a graph of the gradient of this resolution. It is acknowledged that if this P2-CNV relationship were substantiated, some modification of the relationship between the P2 component of the N1-P2 complex and mental work would be necessary. However such considerations are premature in advance of such substantiation and are not pursued.

PART II: AUDITORY SELECTIVE ATTENTION

Following the failure of Experiments 3 and 4 to demonstrate an N1 correlate of selective attention, it was felt that some sort of replication of Hillyard et al's (1973) result was a necessary pre-requisite before any further research was conducted in this area. Experiment 5 constituted such a replication and permitted a second investigation (Experiment 6) which explored the mechanisms of the effect and which completes the chapter. In preparation for Experiment 5, though, it should be noted that certain short-comings in Hillyard et al's

(1973) study were recognised, and thus, whilst the experiment represented an attempt to replicate their finding of an N1 correlate of auditory selective attention using a procedure based on their adaptation of the dichotic listening paradigm, a number of procedural modifications were nonetheless incorporated.

Experiment 5: Evoked Potential Correlates of Auditory Selective Attention: A Replication of Hillyard et al (1973)

INTRODUCTION

Summarising the essential features of Hillyard et al's now classic 1973 study, it is sufficient to note that subjects received independent binaural sequences of 512 tone pips each, with the left ear pips being at a pitch of 800 Hz and the right ear pips, 1500 hz. A random ISI (250 - 1250 ms) was employed within each sequence and the subject's task was to count the number of target pips (at the slightly higher frequencies of 840 and 1560 Hz for the two ears respectively) at either the left ear (condition A) or the right ear (condition C). These targets were interposed every 3 to 20 stimuli at random throughout the sequence. A third condition (B) required the subject to read a novel and disregard all tones, and was used to reduce carry-over effects between successive attend-left and attend-right conditions. Five subjects received the sequence ABCCBA and five others CBAABC, their EEG being recorded from a vertex electrode. In a subsequent experiment (Schwent and Hillyard, 1975) Hillyard's group went on to demonstrate that the selective enhancement of N1 to the stimuli on the attended channel could not be attributed to peripheral factors at the level of the middle ear or cochlea.

The present procedure diverged from that of Hillyard et al (1973) in the following two important respects:

a) The tone-pips delivered to the attended and unattended ears were of identical physical characteristics. This allowed the effect of attention upon EP amplitude to be evaluated within a single block, and thus any problems introduced by general state changes between blocks are avoided. In an experiment such as Hillyard et al's, where the use of physically different stimuli precludes a within-block comparison between EP amplitude to attended and unattended stimuli, attentional effects can only be deduced from the presence of an interaction between stimulus type and attention condition. If the form of the interaction is a cross-over, such as depicted in Fig. 3.4a, then the evidence for an effect of selective attention is unequivocal. However, if the interaction has the form of Fig. 3.4b then the evidence is less convincing; the interaction might simply indicate a differential effect of some general state change upon the amplitude of the EPs evoked by the two types of stimulus. Such a differential effect could arise, for instance, as a result of the EP to stimulus A being initially closer to its maximum amplitude than the EP to stimulus B.

b) Hillyard et al (1973) used the same stimulus sequence on each of the six blocks that constituted their experimental session. The possibility that subjects learned something of the structure of the sequence, and were thus increasingly able to predict the occurrence of the stimuli above chance level, cannot be discounted and would certainly confound any interpretation of their results as evidencing an effect of selective attention upon EP amplitude

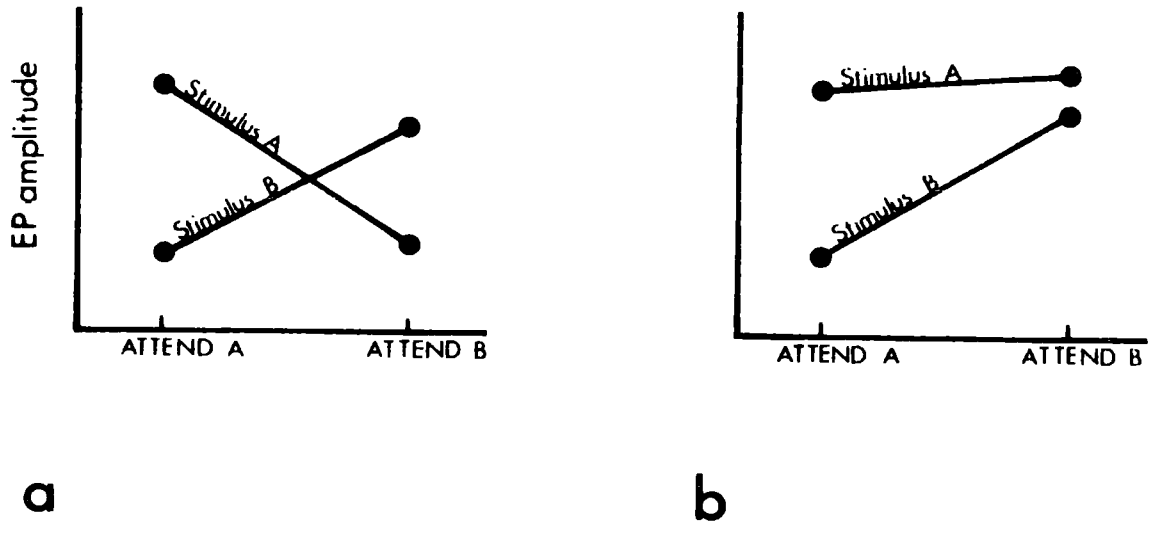

Figure 3.4

Two hypothetical forms (a,b), discussed in the text, which an interaction between stimulus type and attention condition might take.

(Näätänen; 1967, 1975). Although they do not state so, presumably the same stimulus sequence was used for all ten subjects; thus representing a second unfortunate feature of their design. The present study, by employing a different sequence for each subject and with subjects only receiving one sequence, circumvents both the above criticisms.

A related statistical point, which was alluded to in the previous experiment, is that in the present study the randomisation procedure which determined which ear was to be stimulated was completely random, i.e. it did not embody any internal constraints which might improve the subject's ability to predict the occurrence of relevant stimuli above chance level. In Hillyard et al's study, however, such a constraint did operate, with the number of relevant and irrelevant stimuli being constrained to be equal. In such circumstances the predictability of stimuli can diverge significantly from chance if some track of previous stimulation is kept. Operating such a 'gambler's fallacy', for example, the departmental computer was able to predict the occurrence of relevant events in a 'random' sequence containing 100 such events intermixed with 100 irrelevant ones, with an average success rate of 54% over 50 sequences (White, A.P., personal communication).

METHOD

EEG was recorded from a vertex derivation and was referred to either a right or left earlobe placement (randomly chosen with a coin flip). On average, 350 1000 Hz tone pips of 50 ms duration were delivered to the ears over headphones, the stimulated ear being determined by a completely random sequence. Twenty-two replications over a period of 12 months

were conducted. A fast presentation rate (3 per sec), generating a high processing load, and low intensity pips (approx. 60 dB) were used in order to comply with Schwent et al's (1976, a,b) criteria for obtaining selective attention related EP amplitude effects.

The task itself was simplified in relation to that of Hillyard et al (1973). The target bleeps were dispensed with and the subject was simply required to count all the stimuli arriving at one designated ear and to ignore those delivered to the other. A run of practice trials, which the subject terminated when he felt competent to perform the task, preceded the experimental sequence. The attended ear was counter-balanced across replications. It was considered that any confounding EP amplitude differences between the ears not related to the level of attention (due for instance to differences in the acoustic properties of the headphones or ears, positioning of the electrode, etc.) were satisfactorily randomised across replications, and hence that group mean differences in the characteristics of the EP evoked by attended and unattended stimuli could be unambiguously interpreted.

RESULTS

The AEPs to attended and nonattended stimuli were computed for each subject and the N1 wave (mean latency 101 ms) was easily identified on every trace. The combination of the short ISI and the particular software used in this experiment restricted the sampling epoch to 195 ms. This restriction precluded measurements on the P2 component because in some cases this deflection had not reached its peak amplitude within the epoch. Student's t-tests, compiled in Table 3.3, indicated that the N1 wave was significantly enhanced in amplitude (98%; $t(df = 21) =$

TABLE 3.3: EXPERIMENT 5

N1 amplitude (μV) and latency (ms) associated with attended (ATT) and nonattended (IGN) stimuli. The results of Student's t-tests evaluating the effect of attention are also shown (one tailed test).

	N1 AMPLITUDE	N1 LATENCY
ATT	2.23	97
IGN	1.13	105
t,df = 21	4.26 ***	1.70 *

4.26***) and occurred 8 ms earlier (t(df = 21) = 1.7*) to attended versus unattended stimuli.

DISCUSSION

Posner (Posner and Boies, 1971; Posner, Klein, Summer and Buggie, 1973) follows Moray (1969) in distinguishing three major categories in which studies of attention may be grouped; the categories corresponding to the three senses of attention which relate to alertness, selectivity and processing capacity. Reiterating Näätänen's argument (Näätänen, 1967; Näätänen, 1975) presented in Chapter One, early human EP studies, through unsatisfactory procedures, confound the first two of these components. However the work of Hillyard and his group and the present experiment do represent unambiguous investigations of EP correlates of selectivity, and indeed indicate that the amplitude of the N1 wave of the auditory EP, unlike its visual counterpart, indexes the operation of the stimulus set mode of attention whereby task relevant stimuli are preferentially admitted for further processing. In the terms of the neuropsychological model of section two of the introduction, this N1 amplitude effect is explained with reference to Posner's third component of processing capacity: a thalamic gating mechanism is considered to govern the initial allocation of capacity (cortical columns) to a stimulus, with the greater allocation to the relevant stimuli being reflected in an enhanced N1 wave. In this connection it is finally pertinent to note that Hillyard, in a recent publication (Hink, van Voorhis, Hillyard and Smith, 1977), also interprets the N1 correlate of selective attention in capacity terms.

The present study, in addition to replicating Hillyard et al (1973),

goes further in demonstrating that the enhanced N1 to stimuli on the attended channel also occurs at an earlier latency. In general, EP and attention studies have been primarily preoccupied with amplitude effects, although attention related latency changes have been occasionally reported (e.g. Ford, Roth and Koppell, 1976). Given this preoccupation and with the latency shift being apparently only a relatively slight affair (8 ms) that is statistically undramatic (requiring df = 21 before barely achieving significance at the 5% level), it is thus perhaps not surprising that Hillyard et al do not note its presence. Given my speculations in the discussion of the N1-P2 amplitude-latency correlations of Experiment 2, this finding is adduced as further evidence "that the attentional mechanisms can influence the time-course of processing". The fact that the latency shift is not spectacular relative to the amplitude enhancement, is consistent with the small magnitude of the correlation observed in the earlier experiment.

Experiment 6: Evoked Potential Correlates of Auditory Selective Attention: An Investigation of the Mechanisms of the N1 Amplitude Correlate

INTRODUCTION

The results of Experiment 5 indicate that in the auditory modality, apparently unlike the visual, the initial allocation of processing capacity to a stimulus (indexed by N1) can be regulated by a pre-set filter, such that stimuli in the attended stream receive a greater allocation than those on an unattended stream. The present neuro-psychological model of attention indicates that the rationale behind this allocation policy simply reflects the greater processing demands of the task relevant stimuli (in the thesis experiments, counting them).

Thus the model clearly predicts that if this differential mental work can be dissociated from the time of occurrence of the relevant stimuli then the N1 enhancement should not be obtained. It was precisely this prediction that Experiment 6 was designed to investigate: by shortening the ISI in the binaural tone-pip task of the previous experiment from 350 to 200 ms it was envisaged that the subject would be forced to adopt a different counting strategy in order to cope with the increased processing load. An anticipated, subjects' introspections revealed that the short ISI precluded counting responses to individual stimuli; instead relevant stimuli were shunted into some sort of buffer store and at a suitable occasion (such as the non-occurrence of a relevant tone-pip) a running total of relevant stimuli was updated by a quantity that reflected the subject's impression of the number of items in the buffer. The buffer was then cleared.

With the mental work associated with counting thus not time-locked to the occurrence of relevant stimuli, the present theory predicts that the amplitude of N1 will be the same for the attended and unattended stimuli. In analysis of variance terms, Experiment 6 was thus designed to evaluate the significance of the interaction between ISI and attention upon N1 amplitude.

METHOD

Four subjects participated in the experiment and their EEG was recorded monopolarly from a vertex placement. Four random sequences of 200 binaural tone-pips (60 dB, 1000 Hz, 50 ms duration) were delivered over headphones, each sequence being associated with one of the four different experimental conditions: count left ear, 350 ms ISI (condition A);

count right ear, 350 ms ISI (B); count left ear, 200 ms ISI (C); and count right ear, 200 ms ISI (D). The order of conditions was counterbalanced, with subjects corresponding to the rows of the following Latin square:

B D C A
D A B C
A C D B
C B A D

Before each condition the subject was given practice trials in the manner of Experiment 5, and at the end of each sequence the reported count of attended stimuli was recorded. Eight AEPs were computed for each subject, corresponding to the eight combinations of ISI, ear and level of attention; the amplitude and latency of N1 (mean latency 109 ms) was measured in each case.

RESULTS

A three factor Latin square analysis of variance was performed on both the N1 amplitude and latency data; summary tables and the means associated with the various main effects and interactions are compiled in Table 3.4 and Table 3.5. In both analyses the variance associated with the order of conditions was extracted from the residual variation and is designated factor O. The remaining sums of squares, with 18 df, was used as the error term for all the F ratios.

Inspecting Table 3.4 it is noted that none of the main effects of attention, ISI , ear or order are significant; however, the experimental hypothesis that the effect of attention and ISI would interact is confirmed in the data ($F(1,18) = 7.54^{**}$). This interaction is depicted in Fig. 3.5 and an analysis of its simple main effects

TABLE 3.4: EXPERIMENT 6, AMPLITUDE DATA

i) Mean N1 amplitude (μV) as a function of attention (ATT - attend, IGN - ignore) and ISI (350,200 ms). The main effect of ear is also shown.

	ATT	IGN	
200	1.20	1.62	1.41
350	2.68	1.10	1.89
	1.94	1.36	1.65

Right ear - 1.64

Left ear - 1.67

ii) Summary of the analysis of the effects of attention (A), ISI (I) and ear (E; right, left) upon N1 amplitude.

Source	df	SS	MS	F
Subjects	3	3.58		
A	1	2.69	2.69	2.47 ns
I	1	1.84	1.84	1.67 ns
A x I	1	8.00	8.00	7.34 **
E	1	0.01	0.01	< 1 ns
A x E	1	0.98	0.98	< 1 ns
I x E	1	0.18	0.18	< 1 ns
A x I x E	1	1.22	1.22	1.12 ns
Order, O	3	2.76	0.92	< 1 ns
Error (residual)	18	19.63	1.09	
Total	31	40.89		

iii) Simple main effects of the A x I interaction

Source	df	SS	MS	F
A at I = 200 ms	1	.71	.71	< 1 ns
A at I = 350 ms	1	9.99	9.99	9.16 **

TABLE 3.5: EXPERIMENT 6, LATENCY DATA

i) Mean N1 latency (ms) as a function of attention (ATT, IGN) and ISI (350,200 ms). The main effects of ear and order are also shown.

	ATT	IGN	
200	105	109	107
350	109	114	112
	107	112	109

Right ear - 111

Left ear - 107

ORDER: Block 1 - 92

2 - 115

3 - 123

4 - 107

ii) Summary of the analysis of the effects of attention (A), ISI (I) and ear (E; right, left) upon N1 latency.

Source	df	SS	MS	
Subjects	3	6,713.1		
A	1	200.0	200.0	<1 ns
I	1	190.1	190.1	<1 ns
A x I	1	4.5	4.5	<1 ns
E	1	128.0	128.0	<1 ns
A x E	1	180.5	180.5	<1 ns
I x E	1	1.1	1.1	<1 ns
A x I x E	1	78.1	78.1	<1 ns
Order, O	3	4,113.6	1,371.2	4.02 *
Error (residual)	18	6,140.8	341.2	
Total	31	17,749.8		

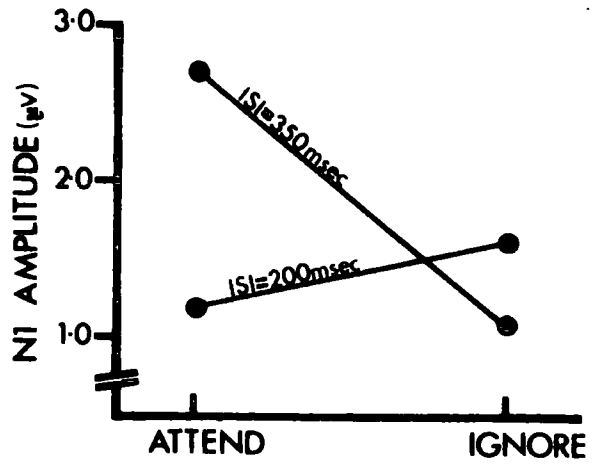

Figure 3.5

Experiment 6: The interaction between attention and ISI.

appears in Table 3.4iii: whereas N1 is significantly enhanced by attention at the 350 ms ISI ($F(1,18) = 9.16^{**}$) no corresponding effect is observed at the shorter ISI ($F < 1$). None of the remaining two-way interactions nor the three-way interaction approach significance.

Table 3.5 reveals no significant main effects or interactions in the latency data[2] apart from the effect of order ($F(3,18) = 4.02^{*}$). No interpretation of this effect is offered, although had it been accompanied by a parallel effect of order upon amplitude an explanation in terms of long-term vigilance changes could have been elaborated.

Considering the absolute discrepancy between the reported total and the actual number of relevant stimuli in the sequence as a measure of performance, subjects performed marginally better at the longer ISI miscounting by, on average, 1.1 stimuli per sequence as against 2.0 at the shorter ISI. However this difference was not statistically significant ($t(df = 3) = 1.53$ ns), and in any event the near perfect performance at both ISIs refutes any explanation of the failure to obtain an effect of attention at the faster rate as a product of the subject's general inability to perform the task at this ISI.

DISCUSSION

The experimental hypothesis, generated by the present neuropsychological orientation, that the N1-correlate of auditory selective attention reflects the increased mental work associated with the arrival of attended stimuli, is confirmed in the data of this study. When this differential mental work is dissociated from the time of occurrence of the attended stimuli, the N1 correlate of stimulus set is accordingly abolished. What remains is to explore the implications of these findings

for current cognitive theories of human attention, and in particular for the concept of a pre-cortical gating mechanism. Deutsch and Deutsch (1963) argue that such a filter requires discriminatory capacities as complex as those used in normal perception and suggest that selection only occurs after all sensory input has been fully analysed at the level of pattern recognition. The filter notion has also been rejected by proponents of what Walley and Weiden (1973) term the 'encoding hypothesis'; this cluster of theories including the work of Neisser (1967) and Hochberg (1970). Common to all these latter theories, including Deutsch and Deutsch, is the idea, which echoes Näätänen's (1975) thesis, that a filter is not necessary in order to explain the selectiveness of attention; i.e. that selection is simply part of the information processing sequence that follows the central registration of sensory input.

On the other hand, the present neuropsychological theory considers selection to be mediated by a filter in the form of a thalamic gating mechanism controlling non-specific cortical input. Clearly neither the encoding theories nor this updated version of Broadbent's filter theory are embarrassed by the findings of the present study, which simply indicate that when additional processing capacity is not immediately required by the relevant stimuli there will be no EP evidence of selectivity. Thus whilst demonstrating the value of a capacity theory of attention in interpreting EP amplitude effects, these findings do not shed any illumination upon the mechanisms subserving the allocation of processing capacity, and hence upon the existence or not of a filter. However, as I have already argued in Chapter 1, once the idea is abandoned

that selective attention is mediated by the blocking (or attenuation) of specific sensory input, the distinction between filters and early cognitive processes loses much of its power and may represent more of a question of terminology than a crucial issue in information processing.

By way of a summary, the results of this study can be formulated as representing an important qualification to Schwent and Hillyard's (1976a,b) heavy processing load criterion for obtaining attention related N1 amplitude enhancements, namely that the increased mental work that produces the enhancement must ensue immediately upon the occurrence of an attended stimulus. Although the failures of Experiments 3 and 4 to obtain such an enhancement have already been discussed, this consideration suggests further possible explanations for these null results: in the first of these experiments the demands of the task were particularly exacting and a counting strategy akin to that employed by subjects in the short ISI condition of the present study may well have been adopted; and in the second, the low post-perceptual load (with a counting response being required to, on average, only 1 in every 8 stimuli) did not constrain the subject to commence the requisite mental work immediately upon receipt of the stimulus. In Hillyard et al's (1973) study, although counting responses were not required to every stimulus on the attended channel, a difficult pitch discrimination was, and it was presumably this mental work that produced the N1 enhancement. The counting response to targets did not superimpose an additional amplitude increment, presumably for similar reasons to those advanced in the above consideration of Experiment 4.

Footnotes - Chapter Three

(1) It should be noted that, as this experiment was conducted prior to the development of the digital filter discussed in the final section of the preceding chapter, these AEPs were not smoothed.

(2) The presence of a noticeable N1 amplitude decrement at the shorter ISI (although not significant) and the virtual absence of any latency shift is consistent with the results of Boddy (1972) but does at first sight appear to contradict Surwillo's (1977) finding of an inverse relationship between the latency of the EP and ISIs of this order. However this latter relationship was not found by Surwillo to be significant for the N1 component and in any event the present experiment was not designed to show such effects and includes manipulations related to attention that might obscure or eliminate them.

CHAPTER FOUR

THE SHORT-TERM HABITUATION OF THE VISUAL EVOKED POTENTIAL

The major points of Chapter One relevant at this juncture of the thesis are recapitualted as follows: an operational definition of habituation is adopted, defining it as a response decrement that develops when a stimulus is repeated. Two classes of the habituation of the late components of human brain responses are distinguished, namely long-term (slow) habituation, which developes gradually over an extended period of stimulation, in contradistinction to short-term (fast) habituation, which describes a response decrement that develops rapidly within a short run of stimuli, and which is complete with a small number of stimulus presentations. This latter genre was discussed extensively in the earlier chapter as it represents the principal concern of the bulk of the thesis. The discussion of slow habituation was, however, not elaborated in any depth, being of limited overall relevance. However, the phenomenon is relevant to the experiments of this chapter and will now be discussed further.

The slow habituation of the EP was first reported by Garciá-Austt and his group (Bogacz, Vanzulli, Handler and Garciá-Austt, 1960 ; Bogacz, Vanzulli and Garciá-Austt, 1962) and has since been the subject of several investigations which have been primarily concerned with elucidating the relationship between this phenomenon and general vigilance factors.

Using visual stimuli, Haider (Haider, Spong and Lindsley, 1964; Haider, 1967) found that the slow habituation of the N160 (i.e. a negative

peak at 160 ms) component of the AEP was accompanied by a corresponding decline in vigilance performance; a finding that Fruhstorfer and Bergstrom (1969) have corroborated in the auditory modality. However studies by Wilkinson, Morlock and Williams (1966) and Roth and Koppell (1969) report no such vigilance deterioration. Callaway (1973) reasonably concludes from this work that it is probable that slow habituation is a complex phenomenon, "the result of a combination of factors" (p.157) including peripheral adjustments (looking away, pupil constriction, inner ear muscle contractions, etc.) and central changes (boredom, fatigue, reduced arousal, etc.). The author has nothing to add to this conclusion.

By way of closing these introductory remarks, it is recalled from Chapter One that the effect of selective attention upon both fast and slow habituation has also been the subject of investigation. It is particularly relevant to the experiments of the preceding chapter to note a comment by Ohman and Lader (1972, p.79) in the first of their two studies in this regard. They remark that the well-established "effect of attention upon AER amplitude could result from less response decrement [i.e. less habituation] in the attending than in the non-attending conditions, rather than from a direct effect of attention". Such an explanation of selective attention related EP enhancements, including the effects of the preceding chapter, is, however, not considered tenable by this author for a variety of reasons, the most significant of which is Ohman and Lader's own subsequent observation that there is, if anything, a tendency for habituation to be more, not less, pronounced at a higher attentional level.

A crude reversal of the logical direction of their proposition in relation to fast habituation generates the suggestion that the fast response decrement (FRD) of the AEP with stimulus repetition is simply a reflection of declining attention. Such an explanation belongs to the class of psychological explanations of the FRD which are explored in the general investigation of mechanisms that occupies the succeeding two chapters. The present chapter, however, is concerned with establishing certain characteristics of the fast habituation phenomenon (such as its interaction with slow habituation and the effects of various other factors, including presentation rate and electrode location) in advance of this subsequent work. In Chapter Two, a method for the single trial analysis of visual EP data was developed. Such an analysis is required in this chapter and accordingly the research to be presented is located in the visual modality. It is thus pertinent to note that, to this author's knowledge, this material constitutes the first intensive investigation of fast habituation in the visual system, with previous work being primarily concerned with auditory, and to a lesser extent, somatosensory stimuli.

Experiment Seven: A Single Trial Analysis of the Short-term Habituation of the Evoked Potential

INTRODUCTION

By way of introducing these opening remarks, the general structure of a fast habituation experiment is recalled from Chapter One. Typically, short trains of stimuli are presented with long intervals between trains and then EPs are averaged according to their ordinal position in the train in order to demonstrate a within-train amplitude decline. In the discussion section of their 1972 paper, Ohman and Lader remark that

"the interpretation of amplitude decrements of the AEP is difficult, since this response cannot be genuinely studied from trial to trial except in subjects with exceptionally clear evoked responses. To infer such changes, indirect methods must be used which invariably introduce restricting assumptions. In averaging across trains to study stimulus-by-stimulus (within-train) changes, these assumptions may not be met if there are lasting decremental effects from one train to the next, since the procedure implies that exactly the same process is operating in each train". If the EP amplitude of the individual trials that comprise the fast habituation experiments could be compiled in an m x n matrix, with the rows of the matrix representing successive trains and the columns indicating the serial position of a trial in a train, then Ohman and Lader are lamenting, in analysis of variance terminology, their inability to assess the interaction of the main effect of fast habituation (as reflected in the column means) with the main effect of any longer term changes that are embodied in the row means. That such long-term between-block effects do operate is certainly clear, as the phenomenon of the slow habituation of the EP amply testifies. In their data, Ohman and Lader (1972) also describe such a long-term effect, which they express as a linear decreasing trend in N1-P2 amplitude over successive blocks. However, even in the absence of such an effect, there are strong theoretical reasons for suspecting a variation in the characteristics of the within-train habituatory decrement as the train is repeated. In detailing their habituation criteria, Thompson and Spencer (1966, p.188) state that "if repeated series of habituation training and spontaneous recovery are given, habituation

becomes successively more rapid" and they designate this defining characteristic of habituation, "potentiation of habituation".

Returning to the logic of the current experiment, although between- and across-block averaging effectively yield the row and column means of the m x n matrix that represent the long-and short-term effects operating in the experiment, a single trial analysis is required in order to fill in the body of the matrix and permit evaluation of their interaction. The present study accordingly employs the single trial method developed in Chapter Two for this purpose.

METHOD

The EEG was recorded monopolarly from the vertex of the three subjects who participated in the experiment. Ten trains, each consisting of ten chequerboard flashes (1 per sec, provided by a Grass PS2 photostimulator positioned 3 metres from the subject, intensity setting = 4) were presented to the subject, with an interval of one minute elapsing between trains. The subject was informed of these details of the organisation of the experimental session at the outset. Ten seconds in advance of each train of stimuli the subject was warned verbally by the experimenter of the imminence of stimulation. He was further instructed to attend passively to the stimuli and to avoid blinking whilst they were occurring, and to fixate a cross that was located in the centre of the chequerboard. Eye artifacts were thus minimised, although some 'rudimentary' blinks, not systematically related to stimulus number or block number, were reported. Subsequent inspection of the data revealed no significant contamination from such artifacts.

The single trial analysis of Experiment Two was used to extract the latency and amplitude of the N1-P2 wave on all of the 100 trials that constituted the experiment, and a two factor (stimulus number, block number) repeated measures analysis of variance was performed on the amplitude data. Analysis of the latency data was withheld until completion of Experiment Eight, whereupon a combined analysis of both experiments was performed. As the covariance measure of N1-P2 amplitude is in relatively meaningless units it was divided throughout by the template variance (a quantity derived from the sum of the squared values of the time-points in the template), yielding a more useful measure that expresses the magnitude of the detected EP as a function of the size of the template. Crudely speaking, an amplitude value of 1.35, for instance, indicates that the EP on that trial is 1.35 'times as big' as the template, and so on.

RESULTS

The N1-P2 amplitude results are depicted in Fig. 4.1, where all three subjects' data are shown plotted as a function of trial and block number. Whereas the earlier applications of the single trial approach in Chapter Two maintained the raw data at a distance, relying upon various statistics to abstract the features of interest, the success of the method in this experiment is immediately obvious upon inspection of this figure. For all three subjects the single trial data, far from being too 'noisy', is certainly 'clean' enough to permit the consistent identification of both within-and between-block effects and even to provoke meaningful discussion of any trial-by-trial events.

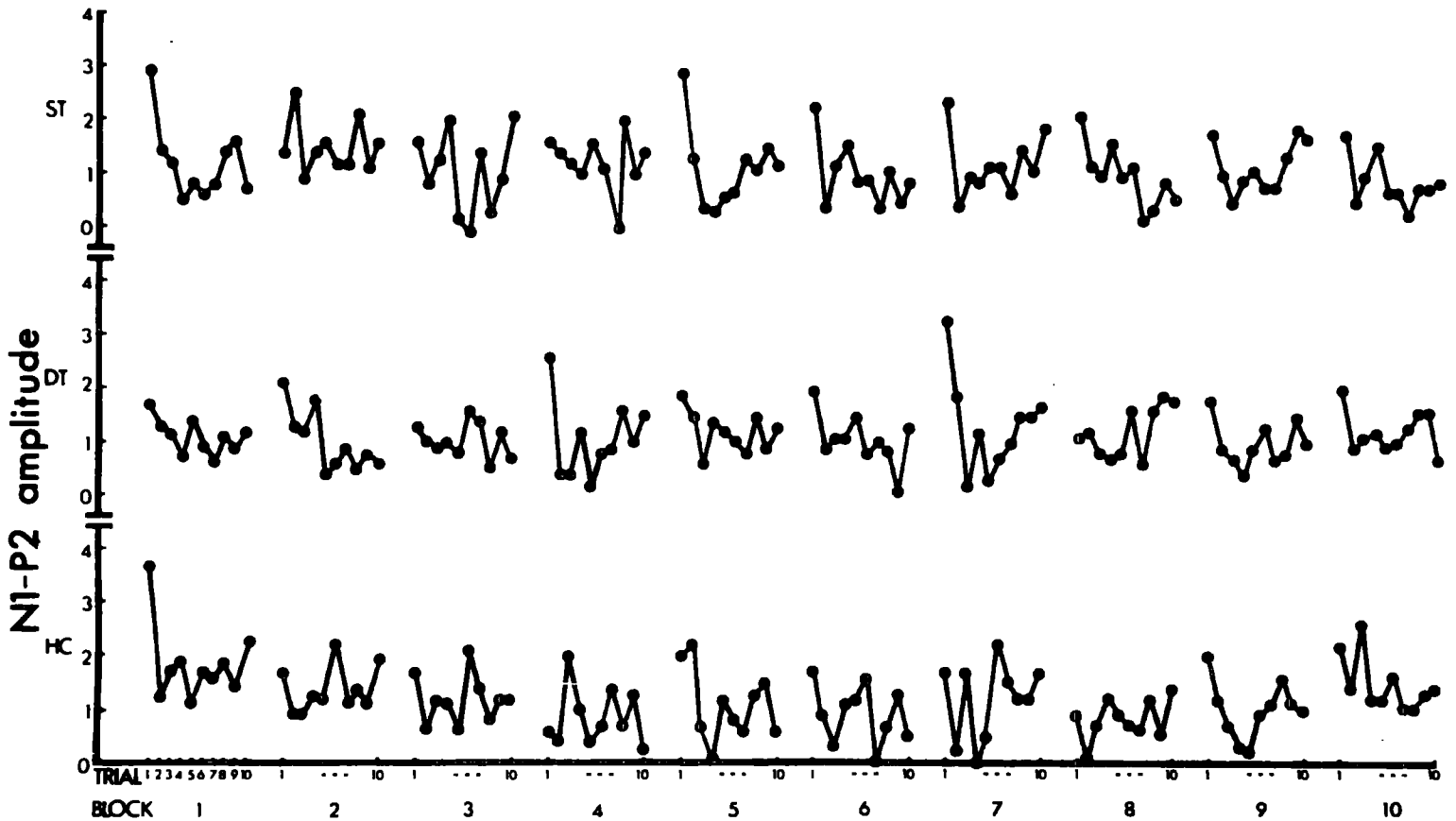

Figure 4.1

Experiment 7: N1-P2 amplitude for individual trials plotted as a function of trial and block number. All three subjects' (HC, DT and ST) data is shown.

The results of the analysis of variance and the tables of means are compiled in Table 4.1 (for key to significance levels and further presentation details see the first section of Chapter Two). Both the main effects of block number ($F(9,198) = 1.91^{*}$) and stimulus number ($F(9,198) = 11.04^{***}$) are significant although their interaction is not ($F < 1$). Both the above main effects are depicted in Fig. 4.2. A linear trend test, although not significant ($F(1,198) = 3.48$ ns), revealed that 20% of the variability of the main effect of block could be accounted for in terms of this comparison. The lack of significance of the residual variation ($F(8,198) = 1.71$ ns) compounded with the absence of any theoretical predispositions, precluded any further analytical comparisons. 87% of the short-term habituation effect (i.e. the effect of stimulus number) was located in the difference between the amplitude of N1-P2 to the first stimulus and the mean N1-P2 amplitude of all succeeding stimuli considered together (Comparison 1, Table 4.1; $F(1,198) = 86.3^{***}$). Apart from a small but significant increase in the amplitude of N1-P2 over the last three stimuli (Comparison 2; $F(1,198) = 8.52^{**}$), no further amplitude effects after the first stimulus are present, with the F ratio of the residual variation being < 1. To support this single trial demonstration of short-term habituation, the across-block averaged AEPs for one subject, ST, are shown in Fig. 4.3 where both the enormous 'first stimulus effect' and the slight upturn towards the end of the train are clearly seen.

TABLE 4.1: EXPERIMENT 7, AMPLITUDE DATA

i) Mean N1-P2 amplitude as a function of block and stimulus number.

Block no.	1	2	3	4	5	6	7	8	9	10
N1-P2 ampl.	1.37	1.29	1.09	1.05	1.14	0.99	1.24	1.01	1.06	1.20

Stimulus no.	1	2	3	4	5	6	7	8	9	10
N1-P2 ampl.	1.96	1.03	1.00	1.04	0.89	1.09	0.89	1.18	1.14	1.23

ii) Summary of the analysis of the effects of block (B) and stimulus (S) number upon N1-P2 amplitude. For key to significance levels see Section 1 of Chapter 2.

Source	df	SS	MS	F
Subjects	2	0.15		
B	9	4.42	0.49	1.91 *
S	9	25.55	2.84	11.04 ***
B x S	81	19.22	0.24	<1 ns
Error (pooled *)	198	50.87	0.26	
Total	299	100.21		

iii) Single df comparisons

Source	Comparison	MS	F	r^2	F residual
B	linear trend	.90	3.48 ns	.20	1.71 (8,198) ns
S	comp 1: $1\ -\frac{1}{9}\ -\frac{1}{9}\ \ldots\ -\frac{1}{9}\ -\frac{1}{9}$	22.18	86.30 ***	.87	
	comp 2: $0\ -\frac{1}{6}\ \ldots\ -\frac{1}{6}\ \frac{1}{3}\ \frac{1}{3}\ \frac{1}{3}$	2.19	8.52 **		<1 (7,198) ns

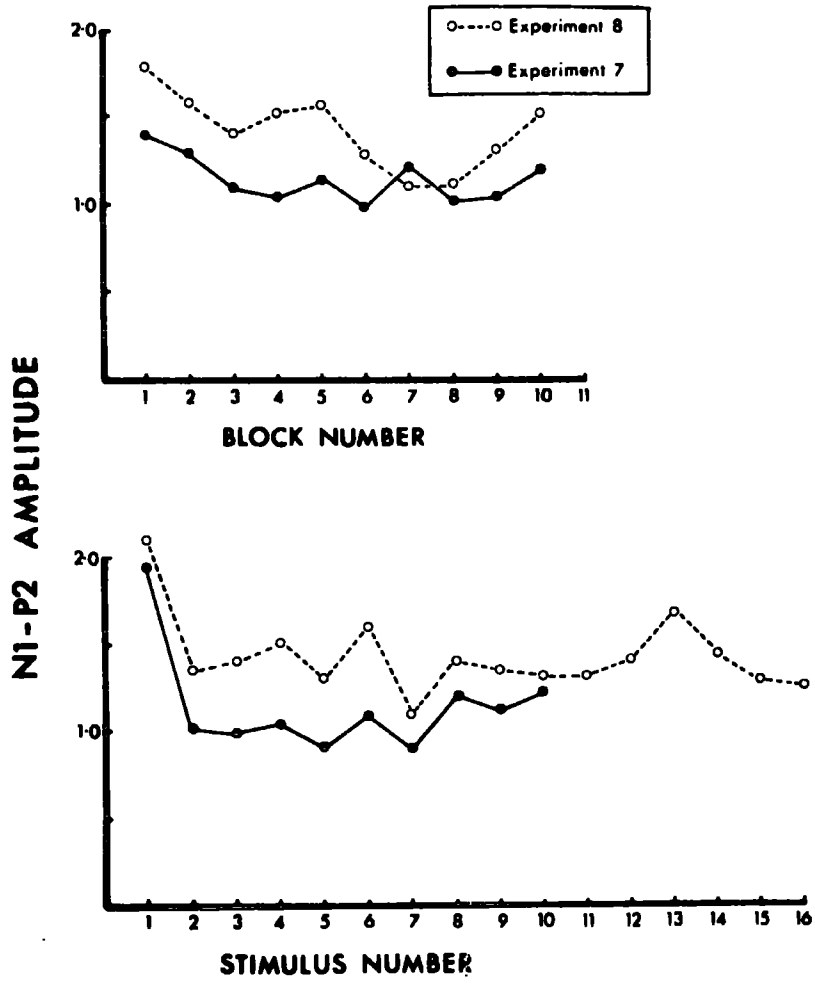

Figure 4.2

Experiments 7 and 8: Mean N1-P2 amplitude plotted as a function of block and stimulus number.

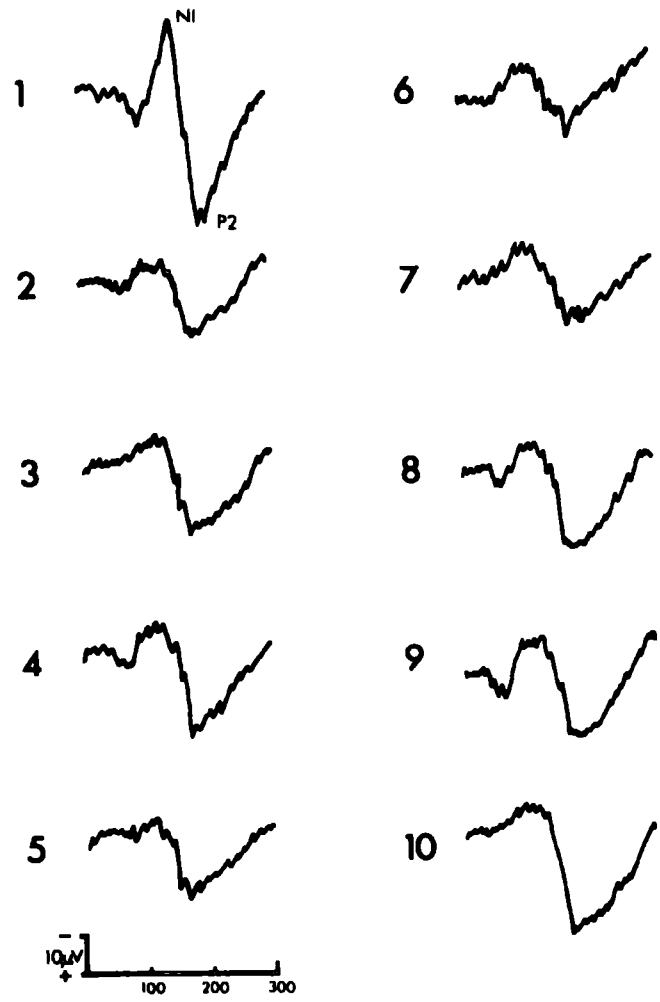

Figure 4.3

Experiment 7: Across-block averaged AEPs for subject ST for ordinal stimulus positions 1 through to 10.

DISCUSSION

The principal aim of this experiment was to employ single trial methods in order to evaluate the interaction between the within-block fast habituation effect and any long-term changes occurring over blocks. In particular, it was anticipated that the phenomenon of 'potentiation of habituation' would be embodied in this interaction. However, no such interaction exists and, whilst casting some further doubt on the validity of describing the fast habituation of the EP as genuine habituation in terms of Thompson and Spencer's (1966) criteria, at least this finding provides reassurance for Ohman and Lader (1972), and indicates that the short-term habituation experiment can be completely described in terms of the two main effects embodied in the two sets of AEPs derived from between- and across-block averaging.

Considering the first of these main effects, it has already been noted that Ohman and Lader (1972) obtained a between-blocks 'slow habituation' effect in their N1-P2 data, which they summarise as a linear decreasing trend over successive blocks. The present investigation also finds a significant between-blocks effect, whose most obvious component is a linear trend. Although not significant, this trend represents some evidence for slow habituation developing over the course of this experiment.

Finally, considering the within-block fast habituatory effect, the rapidity with which it developed, requiring only one stimulus before reaching its asymptotic level, was surprising as many of the earlier studies reviewed in Chapter One typically observe amplitude decrements continuing well into the run. However, the present study differs from

previous studies in two important respects: firstly, it employs visual stimuli, in contrast to the earlier work which is primarily concerned with auditory stimulation; and secondly, the stimulus delivery rate of 1 per second is faster than that generally employed elsewhere. Apropos this latter consideration, Fruhstorfer et al (1970) also found, using auditory stimuli, that with a 1 second ISI the response decrement was completely developed with the second stimulus.

The interpretation of the partial 'recovery' of the amplitude of N1-P2 towards the end of the run will be deferred for the moment as the purpose of Experiment Eight is to investigate the effect in more detail.

Experiment Eight: A Replication of Experiment Seven using a Block Length of 16 Stimuli

INTRODUCTION AND METHOD

The objective of this second study in the chapter was to examine the replicability of the partial 'recovery' phenomenon of Experiment Seven. Assuming the effect to be robust, an extended block length was employed in a tentative investigation of the following hypothesis: if the phenomena has a physiological mechanism, expressing, for instance, the recovery of the depressed neural nets generating the observed potentials, then recovery should commence at the same point in the train, regardless of its greater length, and likely continue after the tenth stimulus. Alternatively if a psychological mechanism, such as anticipation of the end of the sequence, is more appropriate then the commencement of recovery should be related to train length, occurring at a higher stimulus number on the longer train.

Three subjects participated in the experiment, whose method and data analysis is identical to that of Experiment Seven, save that the ten trains of stimuli comprised 16 stimuli each.

RESULTS

The mean N1-P2 amplitude scores for the main effect of block and stimulus number are depicted in Fig. 4.2 and are also compiled in Table 4.2, where the analysis of variance summary table also appears. Although neither main effect nor their interaction are significant ($F(9,18) = 1.26$ ns; $F(15,30) = 1.81$ ns; $F(135,270) = 1.15$ ns), it was considered justifiable to evaluate the two planned comparisons that embody a) a linear long-term habituatory trend and b) the within-block 'first stimulus effect'. These two comparisons are presented in Table 4.2iii with the linear slow habituation trend achieving statistical significance ($F(1,18) = 4.27*$) and accounting for 38% of the between-blocks variation and the 'first stimulus effect', being highly significant (comp. 1: $F(1,30) = 17.42***$), effectively summarising the within-block main effect ($r^2 = .64$, F-resid < 1).

Over both Experiments Seven and Eight, N1-P2 latency data had also been collected and was now analysed in a three factor split-plot design with replications (i.e. Experiment Seven vs Experiment Eight), block and stimulus number as the factors. Replications was not treated as a random factor as it did represent a systematic difference between the two experiments, namely block length. The analysis of the within-block effect was restricted to the first five stimulus positions only, firstly to overcome the fact that the number of levels of this factor was not constant

TABLE 4.2: EXPERIMENT 8, AMPLITUDE DATA

i) Mean N1-P2 amplitude as a function of block and stimulus number.

Block no.	1	2	3	4	5	6	7	8	9	10
N1-P2 ampl.	1.79	1.57	1.40	1.53	1.57	1.28	1.11	1.12	1.30	1.52

Stimulus no.	1	2	3	4	5	6	7	8	9	10
N1-P2 ampl.	2.10	1.36	1.40	1.50	1.29	1.59	1.12	1.40	1.34	1.32

Stimulus no.	11	12	13	14	15	16
N1-P2 ampl.	1.32	1.41	1.69	1.44	1.29	1.26

ii) Summary of the analysis of the effects of block (B) and stumulus (S) number upon N1-P2 amplitude.

Source	df	SS	MS	F
Subjects	2	24.87		
B	9,18	18.69	2.08	1.26 ns
S	15,30	22.75	1.52	1.81 ns
B x S	135,270	87.25	0.65	1.15 ns

iii) Single df comparisons

Source	Comparison	MS	F	r^2	F residual
B	linear trend	7.06	4.27 *	.38	<1 (8,18) ns
S	compl: $1-\frac{1}{15}\ldots-\frac{1}{15}$	14.63	17.42 ***	.64	<1 (14,30) ns

for the two experiments and secondly, because inspection of the mean N1-P2 latencies for subsequent stimulus positions revealed no further systematic trends.

The mean N1-P2 latencies expressing the main effect of block and stimulus number are compiled in Table 4.3. It should be noted that the latency values upon which the analysis of variance was performed represent the displacement of the template when N1-P2 was detected from its original position in the AEP, measured in ms units. Thus, for instance, the entry -5.1 for level 3 of the between-blocks factor indicates that, on average, N1-P2 was detected 5.1 ms earlier for the third experimental block than its position in the AEP. Inspection of the summary table indicates that only the main effect of fast habituation is significant ($F(4,200) = 5.40^{***}$) and that again a 'first stimulus effect', with N1-P2 occurring at a longer latency for the first stimulus in relation to all succeeding stimuli (Comparison 1), dominates, accounting for 83% of the within-blocks variability ($F(1,200) = 18.02^{***}$, $r^2 = .83$, F-resid = 1.19 ns).

The N1-P2 amplitude data was also combined in the same way for the two experiments, with the analysis of variance summary table and the table of means appearing in Table 4.4. Both the main effects expressing slow and fast habituation are significant ($F(9,36) = 2.91^{**}$; $F(4,16) = 11.62^{***}$) with 1 df comparisons revealing significant linear ($F(1,36) = 11.09^{**}$) and quadratic ($F(1,36) = 9.84^{**}$) components in the between-blocks effect, together accounting for 80% of the total variability due to this source. The fast habituation of N1-P2 can exclusively be

TABLE 4.3: EXPERIMENTS 7 AND 8, LATENCY DATA

i) Mean N1-P2 latency as a function of block (1 to 10) and stimulus (1 to 5) number. The latency of N1-P2 is given by the displacement of the template when N1-P2 was detected from its original position in the AEP, measured in 1 ms units. See text for further details.

Block no.	1	2	3	4	5	6	7	8	9	10
N1-P2 latency	1.6	3.2	-5.1	-0.5	2.6	3.7	10.9	-1.1	9.0	4.1

Stimulus no.	1	2	3	4	5
N1-P2 latency	15.2	4.9	-0.9	-3.2	-1.7

ii) Summary of the analysis of the effects of experiment (E), block number (B) and stimulus number (S) upon N1-P2 latency.

Source	df	SS	MS	F
E	1	22.96	22.96	<1 ns
B	9	5,883.70	653.74	1.04 ns
B x E	9	5,024.70	558.30	<1 ns
S	4	13,608.10	3,402.02	5.40 ***
S x E	4	3,062.50	765.62	1.21 ns
B x S	36	23,347.00	648.53	1.03 ns
B x S x E	36	29,647.30	823.54	1.31 ns
Error(pooled**)	200	126,060.00	630.30	
Total	299	207,754.5		

iii) Single df comparison

Source	Comparison	MS	F	r^2	F residual
S	compl: 1 $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$	11,358.93	18.02***	.83	1.19(3,200)ns

TABLE 4.4: EXPERIMENTS 7 AND 8, AMPLITUDE DATA

i) Mean N1-P2 amplitude as a function of block (1 to 10) and stimulus (1 to 5) number.

Block no.	1	2	3	4	5	6	7	8	9	10
N1-P2 ampl.	1.93	1.66	1.32	1.24	1.46	1.26	1.00	1.12	1.14	1.45

Stimulus no.	1	2	3	4	5
N1-P2 ampl.	2.03	1.19	1.20	1.27	1.09

ii) Summary of the analysis of the effects of experiment (E), block number (B) and stimulus number (S) upon N1-P2 amplitude.

Source	df	SS	MS	F
E	1,4	9.05	9.05	9.28 *
B	9,36	21.13	2.35	2.91 **
B x E	9,36	5.86	0.65	< 1 ns
S	4,16	35.05	8.76	11.62 ***
S x E	4,16	0.91	0.23	< 1 ns
B x S	36,144	18.75	0.52	1.24 ns
B x S x E	36,144	21.46	0.60	1.42 ns

iii) Single df comparisons

Source	Comparison	MS	F	r^2	F residual
B	linear trend	8.96	11.09**)	.80	< 1(7,36) ns
	quadratic trend	7.94	9.84**)		
S	compl: 1 $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$	34.07	45.18***	.97	1.31(3,16) ns

described as a 'first stimulus effect' with comparison 1 ($F(1,16) = 45.18^{***}$) explaining 97% of the within-blocks sums of squares. Finally, although the main effect of replications is demonstrated to be significant ($F(1,4) = 9.28^{*}$), with the amplitude of N1-P2 larger in Experiment Eight, none of the replications x treatments interactions are so. It appears that both the N1-P2 latency and amplitude habituatory effects are replicable, despite effective procedural variations between the two experiments.

DISCUSSION

The principal findings of Experiments Seven and Eight can be summarised in the statement that the human visual EP recorded from the vertex exhibits a decline in amplitude and a shortening of latency in response to repetitive stimulation; and that, at a stimulation rate of 1 per sec, these changes require only one stimulus to attain their asymptotic level. The partial recovery of N1-P2 amplitude over the last three stimuli of the train of length 10 used in Experiment Seven was not replicated with the longer train length of Experiment Eight. A physiological mechanism for this recovery can thus be reasonably rejected, and a psychological mechanism, suitably labile with respect to changes in experimental procedure, posited instead. Sutton (1969) discusses the problems of specifying psychological variables in human AEP experiments. In the present case the author is unable to identify conclusively the precise nature of the relevant psychological mechanism, or indeed the effective procedural differences between Experiment Seven and Eight, save to point out that even subtle changes in the manner of the

experimenter and/or the instructions given to subjects could have profound ramifications in terms of the expectations and attentional set of the subject, and hence upon EP amplitude.

The latency effects observed in Experiments Seven and Eight are not novel: Ohman and Lader (1972) report that N1 occurs at an increasingly earlier latency with successive stimulus presentations, although no corresponding shift emerged for P2. However in an experiment presented in their subsequent paper (Maclean et al, 1975) the latency of all the components measured, including both N1 and P2, decreased with stimulus repetition.

In addition to the fast habituatory decrements, more gradual train-by-train EP amplitude changes were noted. Over the course of the experiment EP amplitude first waned, and then waxed as the end of the experimental session was approached, but overall followed the slow decaying course that has frequently been observed elsewhere and is appropriately labelled slow habituation. As indicated in the method section of Experiment Seven, subjects were made aware of the overall organisation of the experiment at the outset, and presumably the above waxing in EP amplitude reflects some attentional change related to delivery from the monotony of the experimental situation.

Experiment Nine: Effects of Electrode Location and ISI upon the Fast Habituation of the Visual EP. The Contribution of Pupillary Changes is also evaluated.

INTRODUCTION

Before proceeding with an investigation of the mechanisms underpinning the fast response decrement of the EP, a parametric study of

the short-term habituation of the visual EP was conducted, wherein the effects of ISI and electrode location were scrutinised. In the former case it was hoped to replicate, using visual stimuli, the well-established result in the auditory system (Fruhstorfer et al, 1970; Ohman and Lader 1972; Roth and Koppell, 1969) that EP attenuation is more pronounced at faster stimulus presentation rates. As far as electrode location is concerned, no particular theoretical motivation inspired the extension of the investigation to include a second recording site over the occiput, although some interaction of the fast habituation phenomenon with electrode location, reflecting the different information processing preoccupations of the posterior and more anterior cortical areas, was anticipated.

Prior to the main study, a brief preliminary investigation was conducted in order to examine and compare the waveform of the occipital EP with the EP concurrently recorded from a vertex electrode. Four subjects participated in this preliminary study and their EEG was recorded from two monopolar derivations on the midline, one at the vertex (C_z) and a second midway between the two occipital lobes (O). Fifty chequerboard stimuli (provided by a Grass photostimulator 3 metres away, intensity setting = 4) were presented at a rate of 1 per 4.5 seconds (a largely arbitrary rate, although it did, to some extent, represent a compromise between temporal economy and the amplitude depression that would increasingly be incurred at faster rates, as indicated by the visual recovery function - see Fig. 1.3) and the subject was directed to fixate the centre of the chequerboard, paying 'passive' attention throughout.

For each subject the vertex and occipital AEPs were computed and digitally filtered, the waveforms for one subject being shown in figure 4.4. Given the superficial isomorphism of the two AEPs , the occipital deflection corresponding to the N1-P2 wave of the vertex response was itself simply labelled N1-P2. Thus the amplitude and latency of both N1 and P2 was extracted from both AEPs and subjected to statistical analysis. In the case of EP amplitude a two factor analysis of variance, with components and electrode location as the factors, was deemed appropriate; but for the latency data, where the main effect of component is predetermined by the fact that N1 necessarily precedes P2, two t-tests evaluated the effect of scalp location upon each component separately. The various means and the results of these analyses are compiled in Table 4.5.

The main effects upon EP amplitude of component and electrode placement, and their interaction were all significant ($F(1,9) = 14.54^{**}$, 9.87^{**}, and 5.25^{*} respectively) indicating, in words, EP amplitude to be larger at C_z, and P2 to be larger than N1 and particularly so at C_z. The t-tests revealed a notable, but nonsignificant, tendency for N1 to be earlier at C_z ($t(df = 3) = 1.6$, $p \approx .1$) and a corresponding latency shift for P2, although this latter difference did not even approach statistical significance ($t(df = 3) = .22$, $p > .3$).

The overall morphological similarity of C_z and O visual AEPs is consonant with previous findings (e.g. Kooi and Bagchi, 1964; Gastaut and Regis, 1965; Groves and Eason, 1967) and is convenient for the purposes of analysis allowing comparisons to be made between the two locations in terms of a common set of measures. However the observation that the

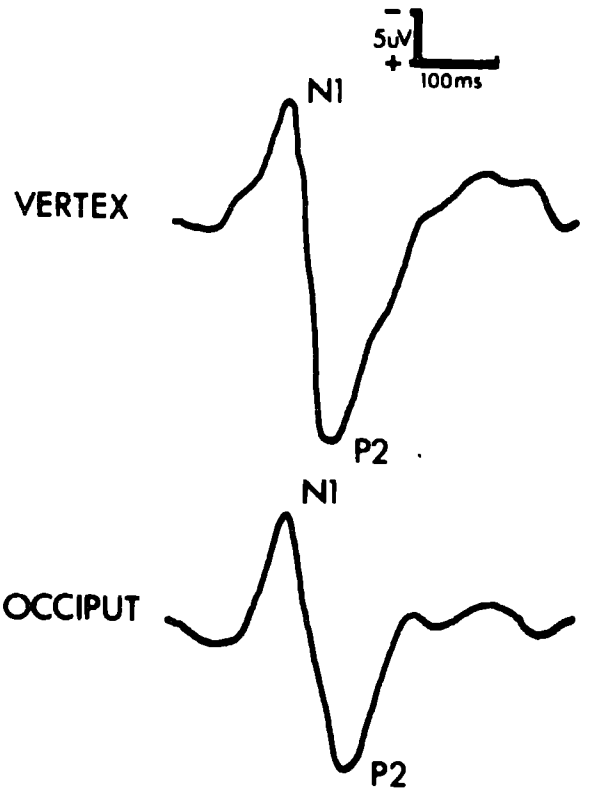

Figure 4.4

Experiment 9, Preliminary study: Simultaneous vertex and occipital EPs for subject RS.

TABLE 4.5: EXPERIMENT 9, PRELIMINARY STUDY

i) Mean N1,P2 amplitude (μV) and latency (ms) as a function of electrode placement (C_z, O). The Student's t-tests (two-tailed) associated with the latency data are also shown.

Amplitude:

	N1	P2	
C_z	5.86	17.32	11.59
O	4.26	7.12	5.69
	5.06	12.22	8.64

Latency:

	N1	P2
C_z	126	206
O	141	211
t,df = 3	1.60 ns	.22 ns

ii) Summary of the analysis of the effects of component (C;N1,P2) and electrode location (E;C_z,O) upon EP amplitude.

Source	df	SS	MS	F
Subjects	3	268.17		
C	1	205.06	205.06	14.54 **
E	1	139.24	139.24	9.87 **
C x E	1	73.96	73.96	5.25 *
Error (pooled **)	9	126.91	14.10	
Total	15	813.34		

vertex responses were both earlier and larger than those at the occiput is not in agreement with much published material, with all the above studies, for instance, indicating the occipital response to be larger. More significantly though, the various studies that have employed chequerboard stimuli (e.g. Harter and White, 1968, 1970; Eason, White and Bartlett, 1970; Harter, 1971; White, 1969) generally show the N1 wave of the occipital EP to occur at a latency of around 100-120 ms, i.e. in advance of the 126.25 ms of the vertex N1 of the present study and well before the 141.25 ms of the occipital N1. However, given the numerous physical and procedural differences (filter settings, stimulation variables, experimental purpose, subject's task and instructions, etc.) between these studies and the present one, these discrepancies are not regarded as serious.

With this preliminary study complete the main experiment was undertaken.

METHOD

Four subjects participated in the experiment and their EEG was recorded from 2 monopolar derivations, at the vertex and at the occiput. The experimental session consisted of 4 blocks, each of 10 trains of 10 chequerboard stimuli, with a different ISI of .5,1,2 or 3 seconds for each block. An interval of one minute elapsed between trains and the order of blocks was counterbalanced across subjects. As per all the experiments in this chapter, the stimuli were provided by a Grass PS2 photostimulator (intensity setting = 4) positioned three metres from the subject. Again as usual, subjects were informed of the

overall organisation of the experiment at the outset. Ten seconds warning was given (verbally, by the experimenter) in advance of each train and subjects were instructed to attend passively to stimuli, and to maintain a fixation, without blinking, upon the central cross on the chequerboard whilst the stimuli were occurring. Comments over eye artifact are as per Experiment Seven. A single trial analysis was not performed on the data as only the main effect of stimulus number was of interest: digitally filtered across-train averaged EPs for trials 1 to 10 at each ISI thus represented the data-base. The amplitude and latency of N1 and P2 were extracted from the AEPs for serial positions 1 to 5 (no further within-train decrements were observed beyond this point) and subjected to statistical analysis.

RESULTS

The amplitude data was subjected to a four factor repeated measures analysis of variance; with electrode, component, ISI and stimulus number as the factors. The means for the various main effects and the summary table are compiled in Table 4.6. It is conceded that the treatment of electrode location and EP component as factors, rather than representing multiple measures, is unusual, but, as they are both expressed in terms of a common dependent variable (namely the amplitude of brain electrical activity in microvolts) with their main effects and interactions with other factors both meaningful and of experimental interest, this 'grand analysis' was considered justified. The components factor, for instance, with its levels expressed as measurements on a dependent variable at different points in time, is

TABLE 4.6: EXPERIMENT 9, AMPLITUDE DATA

i) Mean EP amplitude (μV) as a function of component and electrode location.

	N1	P2	
C_z	8.90	15.56	12.23
O	7.06	8.23	7.64
	7.98	11.89	9.93

Mean EP amplitude (μV) as a function of ISI (sec) and stimulus number (1 to 5)

ISI	.5	1	2	3
Amplitude	8.90	9.44	10.54	10.86

Stimulus no.	1	2	3	4	5
Amplitude	13.50	10.44	8.94	8.21	8.57

ii) Summary of the analysis of the effects of electrode location (E), component (C), ISI (I) and stimulus number (S) upon EP amplitude.

Source	df	SS	MS	F
Subjects	3	2,866.56		
E	1,3	1,680.64	1,680.64	2.31 ns
C	1,3	1,224.96	1,224.96	34.40 **
E x C	1,3	602.56	602.56	53.82 **
I	3,9	204.61	68.20	8.19 **
I x E	3,9	108.29	36.10	5.62 *
I x C	3,9	69.44	23.15	2.85 ns
I x E x C	3,9	22.48	7.49	2.85 ns
S	4,12	1,204.22	301.06	20.16 ***
S x E	4,12	111.67	27.92	6.27 **
S x C	4,12	129.75	32.44	1.55 ns
S x E x C	4,12	6.56	1.64	<1 ns
I x S	12,36	164.74	13.73	1.01 ns
I x S x E	12,36	33.11	2.76	1.22 ns
I x S x C	12,36	123.88	10.32	1.17 ns
I x S x E x C	12,36	29.42	2.42	1.00 ns

iii) Single df comparisons

Source	Comparison	MS	F	r^2	F residual
I	linear trend	196.31	23.58***	.96	< 1(2,9) ns
S	comp 1: 1 $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$	1,020.28	68.36***	.85	< 1(2,12) ns
	comp 2: 0 1 $-\frac{1}{3}$ $-\frac{1}{3}$ $-\frac{1}{3}$	167.87	11.24**	.14	
	comp 3: 1 0 $-\frac{1}{3}$ $-\frac{1}{3}$ $-\frac{1}{3}$	1,166.95	78.16***	.98	< 1(3,12) ns
I x S	comp 4: M	57.67	4.24*	.35	< 1(11,36) ns

where M is a matrix of coefficients expressing a linear declining trend with ISI in the quasi-exponential trend that embodies the main effect of stimulus number. More formally,

$$M = (3\ 1\ -1\ -3)^{T}.\ (1\ 0\ -\tfrac{1}{3}\ -\tfrac{1}{3}\ -\tfrac{1}{3})$$

essentially no different from a factor whose levels are trials, blocks or days, etc. Interactions between the component factor and other factors are clearly of interest and wherever this factor does not appear in a source (i.e. is collapsed across) this effectively and conveniently constitutes measurements on the peak-to-peak deflection, N1-P2.

Consistent with the results of the preliminary study, N1-P2 was larger at the vertex (although not significantly so, $F(1,3) = 2.31$ ns); and P2 was larger than N1 ($F(1,3) = 34.40$**), and particularly so at C_z ($F(1,3) = 53.82$**). The significant main effect of ISI ($F(3,9) = 8.19$**) constitutes a linear increase in N1-P2 amplitude with lengthening ISI (Table 4.6iii: $F(1,9) = 23.58$**; $r^2 = .96$, F-resid < 1) which the interaction with electrode ($F(3,9) = 5.62$*) indicates to be more prominent at the vertex. The interaction with component and the three-way interaction between ISI, electrode and component, although neither is significant ($F(3,9) = 2.84$, $p \approx .1$ in both cases), demonstrate a tendency for the effect of ISI to be more pronounced upon P2 amplitude and especially so at the vertex.

The fast habituation of the AEP is clearly seen in the data of this study ($F(4,12) = 20.16$***) and although again the 'first stimulus effect' predominates (Table 4.6iii; Comp. 1, $r^2 = .85$), the second stimulus in the train also evokes a larger amplitude EP than the succeeding stimuli (Comp. 2; $F(1,12) = 11.24$**). No further significant within-train amplitude effects are present (F-resid < 1) and the quasi-exponential trend coefficients of 1 0 $-\frac{1}{3}$ $-\frac{1}{3}$ $-\frac{1}{3}$ are

adopted as summarising the variability of this source (Comp. 3; $r^2 = .98$). The significant interaction of the fast habituation phenomenon with electrode placement ($F(4,12) = 6.27^{**}$) indicates that the phenomenon is better evidenced at a vertex recording site.

One of the principal objectives of the present experiment was to investigate the effect of ISI upon the development of fast habituation. The ISI x stimulus number interaction is depicted in Fig. 4.5, and although fast habituation is apparently more pronounced at the faster presentation rates, the interaction falls well short of statistical significance. It should be pointed out, though, that a) much of this interaction is expressed in the main effect of ISI and b) that 12 df are associated with it, even though the experimental hypothesis of a declining trend in the magnitude of the fast habituation phenomenon with lengthening ISI can be expressed in one single df analytical comparison. By formulating the hypothesis more economically in this way, i.e. as a monotonic declining trend in the quasi-exponential trend that embodies the within-train effect (Comparison 4, Table 4.6iii), a significant interaction with ISI is indeed obtained ($F(1,36) = 4.24^{*}$) accounting for 35% of the variability of the ISI x stimulus number interaction. (Effectively, this technique is a two-stage process. Firstly, the above quasi-exponential trend coefficients are applied to the effect of stimulus number at each level of the ISI factor in the ISI x stimulus number interaction yielding four values which represent the magnitude of the fast habituation effect at each presentation rate. These values were 6.59, 4.61, 5.24 and 3.28μV for the .5,1,2 and 3 second ISIs respectively. The second stage of the technique completes the hypothesis that the magnitude

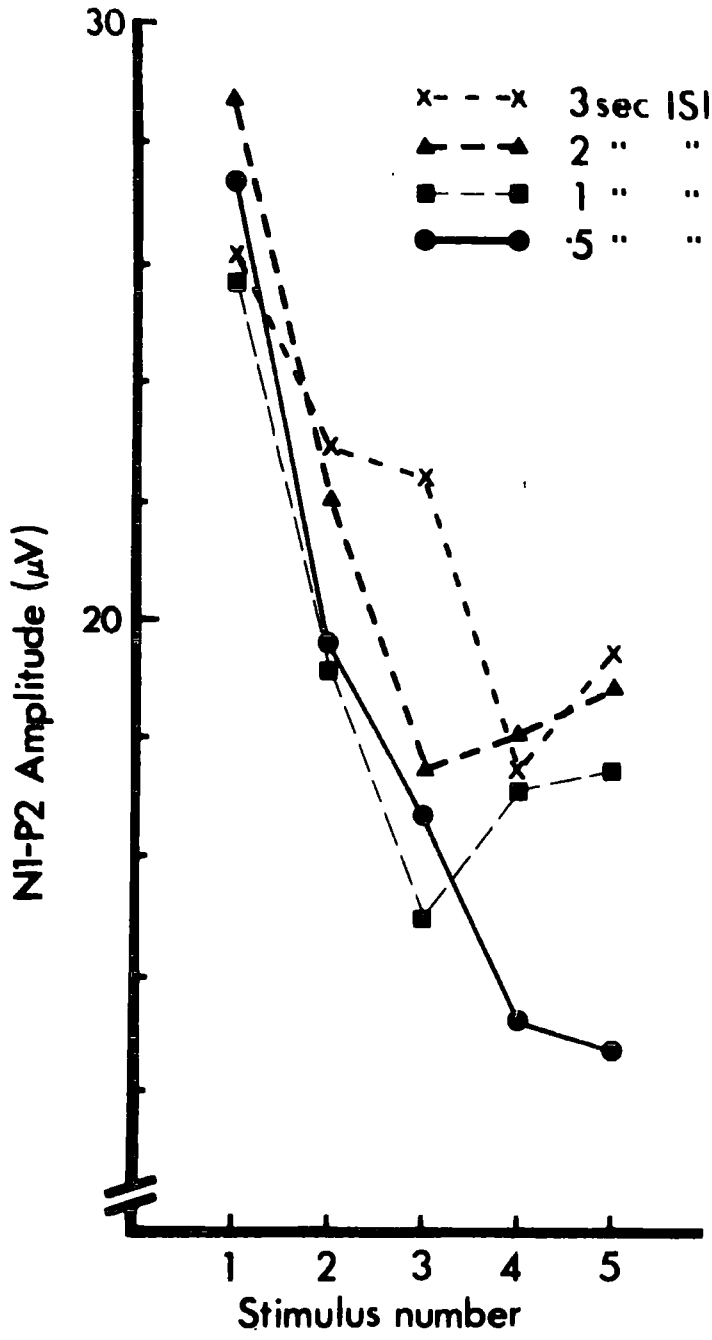

Figure 4.5

Experiment 9: The interaction between ISI and stimulus number.

of fast habituation will increasingly decline as ISI lengthens by applying standard linear trend coefficients to these four values, yielding a single quantity whose mean square is calculated in the usual way for single df comparisons. See also Footnote 1.) Completing this appraisal of Table 4.6, it is noted that no further two-or three-way interactions managed to achieve significance, and without any further specific planned comparisons in mind the analysis of the amplitude data is concluded at this point.

Separate statistical analyses were performed on the N1 and P2 latency data for the reasons given in the presentation of the results of the preliminary experiment. The mean latencies associated with the various main effects and the analysis of variance summary tables for N1 and P2 are compiled in Table 4.7, Parts i, ii and iii respectively. The non-significant tendencies observed in the preliminary study for N1 and P2 to be earlier at the vertex, and N1 more so than P2, are confirmed in the data of the main experiment with N1 and P2 at the vertex occurring 14 ms and 6 ms respectively before their occipital counterparts ($F(1,117) = 85.57^{***}$; $F(1,117) = 5.12^{*}$). No main effects of ISI were obtained although the interaction between electrode and ISI was significant for the P2 component ($F(1,117) = 5.62^{*}$). Analysis of the simple main effects of this interaction revealed a significant increasing linear trend in P2 latency with lengthening ISI at the vertex, but not at the occiput ($F(1,117) = 14.41^{***}$; $F(1,117) = 1.96$ ns).

Application of the quasi-exponential trend coefficients $1\ 0\ -\frac{1}{3}\ -\frac{1}{3}\ -\frac{1}{3}$ demonstrated a within-train shortening of latency for both N1

TABLE 4.7: EXPERIMENT 9, LATENCY DATA

i) Mean N1 and P2 latency (ms) as a function of electrode location, ISI (sec) and stimulus number (1 to 5).

	C_z	O
N1	137	152
P2	206	211

ISI	.5	1	2	3
N1 latency	144	144	145	145
P2 latency	209	204	209	213

Stimulus no.	1	2	3	4	5
N1 latency	147	146	142	145	143
P2 latency	225	211	202	202	202

ii) Summary of the analysis of the effects of electrode location (E), ISI (I) and stimulus number (S) upon N1 latency, including single df comparisons.

Source	df	SS	MS	F
Subjects	3	6,324.00		
E	1	8,193.91	8,193.91	85.57 ***
I	3	86.72	28.91	<1 ns
I x E	3	140.47	46.82	<1 ns
S	4	472.81	118.20	1.23 ns
S x E	4	370.94	92.73	<1 ns
S x I	12	1,054.68	87.89	<1 ns
S x E x I	12	554.05	46.17	<1 ns
Error (pooled *)	117	11,202.75	95.75	
Total	159	28,400.33		

Single df comparisons

Source	Comparison	MS	F	r^2	F residual
S	IN: 1 0 $-\frac{1}{3}-\frac{1}{3}-\frac{1}{3}$	318.75	3.33	.67	1.61(3,117) ns
SxE,S	2N: 1 0 $-\frac{1}{3}-\frac{1}{3}-\frac{1}{3}$ at C_z	675.02	7.05 *		
	3N: 1 0 $-\frac{1}{3}-\frac{1}{3}-\frac{1}{3}$ at O	0.00	<1 ns		
SxExI,S	4N: M (see table 4.6) at C_z	534.75	8.19 **		
	5N: M at O	11.50	<1 ns		

iii) Summary of the analysis of the effects of electrode location (E), ISI (I) and stimulus number (S) upon P2 latency, including single df comparisons

Source	df	SS	MS	F
Subjects	3	16,386.70		
E	1	1,237.65	1,237.65	5.12 *
I	3	1,647.95	549.32	2.27 ns
I x E	3	4,069.00	1,356.33	5.62 **
S	4	13,190.00	3,297.50	13.66 ***
S x E	4	7,103.75	1,775.94	7.35 ***
S x I	12	2,926.25	243.85	1.01 ns
S x I x E	12	2,442.50	203.54	<1 ns
Error (pooled **)	117	28,255.50	241.50	
Total	159	77,259.30		

Single df comparisons

Source	Comparison	MS	F	r^2	F residual
I x E, I	linear trend at C_z	3,481.00	14.41 **		
	linear trend at 0	473.00	1.96 ns		
S	1P: 1 0 $-\frac{1}{3}$ $-\frac{1}{3}$ $-\frac{1}{3}$	12,921.00	53.50 ***	.98	1.11(3,117) ns
S x I	2P: M'	1,281.75	5.31 *	.44	<1 (11,117) ns
S x E x I	3P: M' at C_z - M' at 0	25.25	<1ns		

where M' is a matrix of coefficients expressing the hypothesis that the quasi exponential trend that embodies the main effect of S is most pronounced at the .5 sec ISI. More formally,

$$M' = (1 \ -\tfrac{1}{3} \ -\tfrac{1}{3} \ -\tfrac{1}{3})^T \cdot (1 \ 0 \ -\tfrac{1}{3} \ -\tfrac{1}{3} \ -\tfrac{1}{3})$$

(Comp. 1N) and P2 (Comp. 1P) which accounted for 67% and 98% of the variability of the main effect of stimulus number upon each component respectively. This within-train effect was markedly more prominent upon the P2 component ($F(1,117) = 53.5^{***}$) where it produced a net forward - latency - shift of 23 ms, in comparison to the shift of 4 ms for N1, which in fact fails to achieve statistical significance ($F(1,117) = 3.33$ns). The significant interaction between stimulus number and electrode location for P2 ($F(1,117) = 7.35^{**}$) indicated the forward-latency-shift to be more pronounced at the vertex, and indeed inspection of the corresponding, but non-significant, interaction for N1 demonstrated a substantial and, in fact, statistically significant exponential forward shift of 7.5 ms at the vertex (Comp. 2N; $F(1,117) = 7.05^{*}$) but no shift whatsoever at the occiput (Comp. 3N, $F < 1$).

Both the interaction between ISI and stimulus number, and the three way interaction between ISI, stimulus number and electrode placement that complete the analyses were non-significant for each component. However, following a similar approach to that outlined above in connection with the ISI x stimulus number interaction in the amplitude data, analysis of the simple main effects of the electrode x ISI x stimulus number interaction for N1 revealed a significant linear declining trend in the magnitude of the exponential forward-latency-shift that summarises the effect of stimulus number (see preceding paragraph) with lengthening ISI at the vertex (Comp. 4N; $F(1,117) = 8.19^{**}$) but not at the occiput (Comp. 5N, $F < 1$). Similarly for the P2 component, although a linear declining trend in the forward-latency-shift with ISI

was not significant, the effect was still related to ISI, being significantly larger at the fastest presentation rate (Comp. 2P; $F(1,117) = 5.31^{*}$). However this interaction was only marginally more pronounced, but not significantly so, at C_z (Comp. 3P, $F < 1$).

DISCUSSION

The rapid decline in visual EP amplitude and the shortening of its latency within a train of repetitive stimuli corroborate the findings of Experiments Seven and Eight, although it is recognised that the within-train changes of this study do constitute more than a simple 'first stimulus effect', with a quasi-exponential trend best summarising them. Inspection of Fig. 4.5 indicates that this trend appears at each of the presentation rates used in the present study, and thus an explanation of this discrepancy as reflecting the contribution of a decellerated rate of habituation at the longer ISIs to the overall main effect is not tenable. It is, however, possible that the use of several presentation rates in this study, as against the single rate in the earlier work, influenced the subject's attention to the second stimulus in the train, which would, for instance, confirm that the specified rate for the current block was indeed in use. Any suspicions that the discrepancy reflected some fault in the single trial analysis of the earlier studies were allayed by the re-analysis of the N1-P2 amplitude date of Experiments Seven and Eight (summarised in Table 4.8) using across-train averaging. In agreement with the single trial analysis (Table 4.4), the within-train response decrement is best expressed as a 'first stimulus effect', with the coefficients $1\ -\frac{1}{4}\ -\frac{1}{4}\ -\frac{1}{4}\ -\frac{1}{4}$ accounting for 96% of the main effect of stimulus number.

TABLE 4.8: EXPERIMENTS 7 AND 8.

Mean N1 - P2 amplitude (μV) as a function of stimulus number (S: 1 to 5) derived from the data of experiments 7 and 8 using across-train averaging. Also shown is the analysis of variance summary table and the 1 df comparisons

Stimulus no.	1	2	3	4	5
N1-P2 ampl.	31.76	16.40	15.84	16.16	15.28

Source	df	SS	MS	F
Subjects	5	2,218.43		
S	4	1,205.03	301.26	15.37 ***
Subjects x S	20	392.02	19.60	
Total	29	3,725.49		

Single df comparisons

Source	Comparison	MS	F	r^2	F residual
S	1 $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$	1,200.80	61.26	.996	<1(3,20) ns
	1 0 $-\frac{1}{3}$ $-\frac{1}{3}$ $-\frac{1}{3}$	1,148.80	58.61	.95	<1(3,20) ns

In general, the expectation that ISI would influence the development of the fast habituation of the visual EP is confirmed in the data; it appears that the magnitude of both the amplitude decrement and the forward-latency-shift are greater at faster stimulus presentation rates. In part this influence is reflected in the main effect of ISI, with trains incorporating a longer ISI generating a larger amplitude N1-P2 and a longer latency vertex P2, but is also apparent in the various ISI x stimulus number interactions. In order to summarise and simplify what was a complex data analysis section in this respect, the influence of ISI upon fast habituation is further illustrated in Figure 4.6, where the effect of stimulus number is depicted (for the vertex amplitude and latency data only, where such effects are most pronounced) at a fast presentation rate (the .5 and 1 second ISIs combined) in contrast to the slower rate yielded by the combination of the 2 and 3 second ISIs. Two components are distinguishable in the effect of ISI; firstly and most obviously, a lowering of the final asymptotic value of the habituation function and secondly, an acceleration in the rate at which this asymptotic value is approached. The quasi-exponential trend coefficients $1\ 0\ -\frac{1}{3}\ -\frac{1}{3}\ -\frac{1}{3}$, which are applied to the effect of stimulus number in the data of this experiment, are predominantly sensitive to the former of these components and hence the finding of the results section that fast habituation (as embodied in these coefficients) is more pronounced at shorter ISIs primarily reflects the lowering of the asymptote of the habituation function rather than a change in the rate of habituation. However, adopting the percentage of the full habituation effect achieved with the second presentation of the stimulus as a measure of the rate of habituation, it is apparent from Fig. 4.6 that this rate is accelerated at shorter ISIs, with figures of approximately 70% and 50% (for both the amplitude and latency data) expressing the rate of habituation at the fast and slow presentation rates respectively.

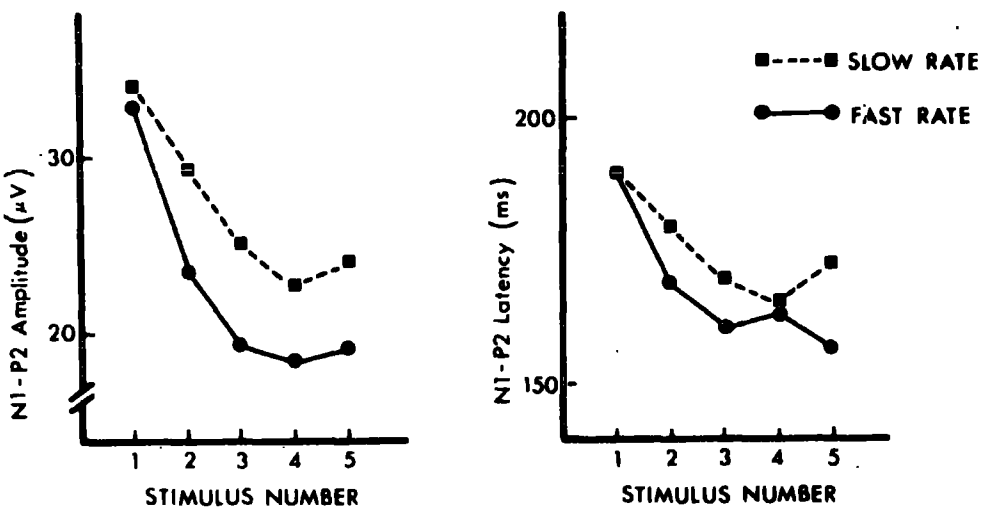

Figure 4.6

Experiment 9: Mean N1-P2 amplitude and latency as a function of stimulus number for the .5 and 1 sec ISIs combined (fast presentation rate) and the 2 and 3 sec ISIs combined (slow presentation rate). Note that the single value expressing the latency of N1-P2 represents the mean of the two latencies for its two constituent components.

This finding that both the rate as well as the asymptotic value of the habituation of the N1-P2 wave of the visual EP are influenced by ISI is consistent with similar research in the auditory modality (see, for instance, Fruhstorfer et al, 1970), although latency effects have typically received less attention elsewhere. The demonstration of an acceleration of habituation at faster stimulus presentation rates gains further significance with reference to Thompson and Spencer's (1966) delineation of the defining characteristics of habituation wherein they specifically discuss the effect of ISI in terms of the rate (rather than, for instance, the final asymptotic value) of habituation.

The extension of the present inquiry to include a second electrode placement over the occiput generated many fascinating effects, which can be assembled into the general statement that the fast habituatory EP amplitude and latency effects are more pronounced at a vertex recording site; and indeed, supplemented by the observation that N1-P2 is both larger and earlier at this site, it must be concluded that the anterior cortical regions are more actively involved in the processing of information in experimental procedures that employ repetitive stimulation and elicit habituation. This finding is consistent with Pribram's [2] theorising that the frontal brain is concerned with the orienting reaction, the habituation of which has been extensively described by Sokolov (1975). The generators of occipital potentials, which presumably lie in the primary visual cortices and surrounding areas of the posterior association cortex, are apparently less active. Pribram associates the function of the posterior association cortex with discriminatory processes and the relative insensitivity of the occipital EP can thus be reasonably ascribed to the secondariness of such

processing in habituation procedures, compounded with the presence of a specific, and therefore non-habituating (Grey Walter, 1964), contribution to the occipital EP from the primary visual cortex. Of the two components investigated, the P2 deflection, in general, demonstrates more sensitivity to the experimental manipulations: however, the author can find no ready explanation of this finding.

In their discussion of the evidence for the habituation of the visual EP, Thompson and Spencer (1966, p.22) point out that "where habituation has been reported, it may be due to alterations in ... pupillary constriction", and they suggest the use of atropin and an artificial pupil to control for this possibility. This is an important caveat as response decrements occurring as a result of receptor mechanisms do not qualify as habituatory in their system. Unfortunately, the unpleasant side-effects of atropin and its classification as a Schedule 1 poison (Pharmacy and Poisons Act, 1933) combined to preclude its use by this experimenter. However, the use of an artificial pupil is a simple measure and affords quite adequate control over pupillary changes. Accordingly, a brief control study employing such a device was run in order to evaluate the contribution of pupillary changes to the fast habituation of the visual EP.

Six subjects participated in the control study. Apparatus and procedural details are essentially the same as per Experiment Seven with subjects being directed to attend passively to 10 trains of 10 chequerboard flashes (ISI = 1 sec, inter-block interval = 1 min). The flashes were again provided by a Grass PS2 photostimulator (intensity setting = 4)

which the subject viewed monocularly through an artificial pupil (diameter = 2 mm), the other non-stimulated eye being covered with an eye-patch. It is worthwhile recalling from Chapter 2 that all the thesis experimentation, including this control study, was carried out in a dimly illuminated laboratory, allowing a minimum period of 15 minutes for dark adaptation. Pupil diameter would thus be satisfactorily dilated in relation to the diameter of the artificial pupil used.

Vertex EPs associated with the first five stimulus positions in the sequence were computed using across-train averaging, allowing the data of this control study to be compared with the across-train averaged data of Experiments Seven and Eight already presented in Table 4.8. Mean N1-P2 amplitude for the control study is shown as a function of stimulus number in Table 4.9, and as with Experiments Seven and Eight, the analysis of variance summary table (also shown) indicates a significant within-train decrement ($F(4,20) = 5.72^*$) that is best summarised as a 'first stimulus effect', with the coefficients $1\ -\frac{1}{4}\ -\frac{1}{4}\ -\frac{1}{4}\ -\frac{1}{4}$ accounting for 93% of the main effect of stimulus number. Although there is an overall drop in N1-P2 amplitude in the control study relative to Experiments Seven and Eight (presumably reflecting the decrease in effective stimulus intensity resulting from the use of the artificial pupil) the rate of decrementation remains the same, with the amplitude of N1-P2 to stimulus No.1 being, on average, 97% and 86% greater than the mean amplitude of N1-P2 to the succeeding four stimuli in Experiments Seven and Eight, and the control study respectively ($t(df = 5) = .32$ ns). The data of this control study thus reassuringly indicate that the fast habituation of the human visual EP cannot be attributed to a pupillary

TABLE 4.9: EXPERIMENT 9, CONTROL STUDY

Mean N1-P2 amplitude (μV) as a function of stimulus number (S: 1 to 5). Also shown is the analysis of variance summary table and the 1 df comparison.

Stimulus no.	1	2	3	4	5
N1-P2 ampl.	19.27	10.09	11.49	12.08	9.43

Source	df	SS	MS	F
Subjects	5	120.29		
S	4	373.27	93.32	5.72 *
Subjects x S	20	326.37	16.32	
Total	29	819.93		

Single df comparison

Source	Comparison	MS	F	r^2	F residual
S	1 $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$ $-\frac{1}{4}$	346.04	21.21 ***	.93	1.67 (3,20) ns

mechanism, and also corroborate the findings of Roth and Koppell (1969) in the auditory system, who also failed to observe any effects of stimulus intensity upon the rate of fast habituation.

The remaining experimentation of the thesis research programme is devoted to further elucidating the mechanisms of the fast habituation phenomenon; firstly, remaining within the visual modality (Chapter Six) before extending the investigation to include auditory stimulation as well (Chapter Seven). All that remains in the present chapter is to note in passing two attempts to study the habituation of the early sensory components, rather than the later non-specific activity, of the human scalp-recorded EP.

A number of investigators (Jewett, Romano and Williston, 1970; Jewett and Williston, 1971; Lev and Sohmer, 1972; Picton, Hillyard, Krausz and Galambos, 1974) have reported a series of small positive deflections occurring at 1 ms intervals over the first 8 ms of the scalp recorded auditory EP, that presumably correspond to the relaying of sensory impulses along the auditory pathway. If Thompson's afore-mentioned (p.19) statement in summarising the evidence for habituation in specific sensory pathways is correct, i.e. that "the ascending relay nucleii do not exhibit habituation", then these early sensory components of the human EP should not demonstrate any amplitude decrements with repetitive stimulation. However, although a general positive-going trend over the initial 10 ms after stimulation was consistently noted in a number of pilot subjects (e.g. subject LW depicted in Fig. 4.7) the above mentioned series of distinct components was not obtained

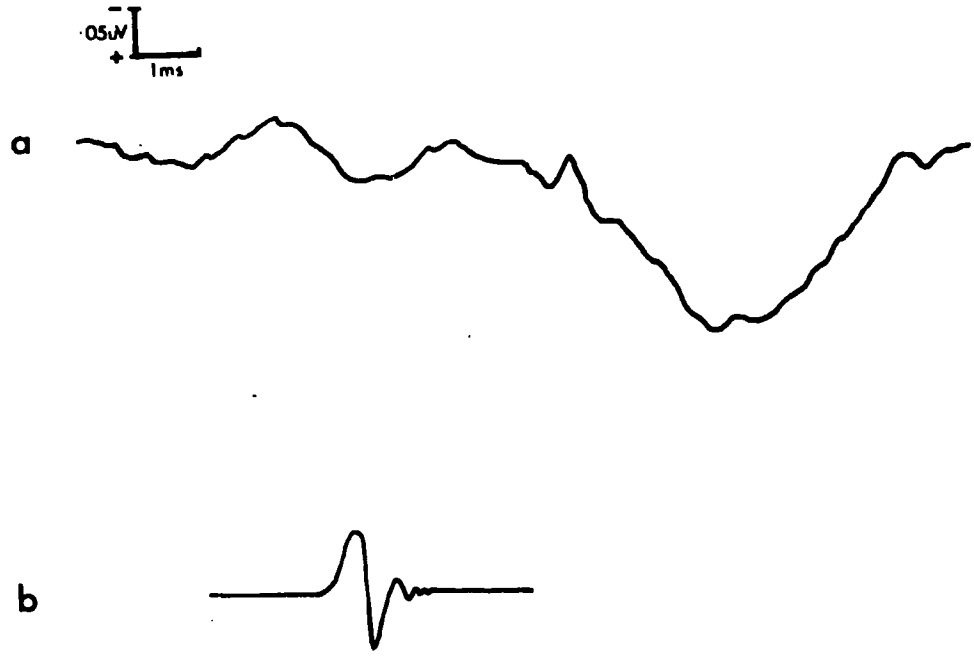

Figure 4.7

The AEP (based on 1,000 trials) showing the early vertex activity evoked by a brief click for subject LW is shown in a. Clicks were delivered to the right ear at a rate of 5/sec and a right mastoid reference was used. The high frequency cut-off of the amplifier was set at 40k Hz. The simultaneous audio record of the click is shown in b.

with any reliability and the investigation was finally abandoned.

A second line of enquiry began with the proposition that, if frequency coding operates in the auditory system for low frequencies (Wever, 1949) then it should be possible to extract a nervous system response of the same frequency as a steady stimulating tone. If such a sensory response could be extracted then it would be interesting to observe whether it habituated over the course of prolonged exposure to the tone.

In a preliminary investigation in this laboratory, a Dawe audio oscillator was used to provide a signal of the requisite frequency, which was then used to generate the steady tone and concurrently trigger a Biomac 500 averaging computer. A brain response that reflected the frequency of the tone was, in fact, obtained at a variety of recording sites for several subjects for tones at various frequencies in the range 150 to 400 Hz. However, upon further inspection of the literature it was discovered that such a 'frequency-following response' (although not to continuous stimulation) had been first described in cats by Marsh and Worden (1968) and Worden and Marsh (1968) and subsequently has been successfully recorded from the human scalp by a number of investigators (Moushegian, Rupert and Stillman, 1973; Gerken, Moushegian, Stillman and Rupert, 1975; Smith, Marsh and Brown, 1975; Marsh, Brown and Smith, 1975, Sohmer and Pratt, 1977). Owing to the uncertainty surrounding the generators responsible for this response (and in particular its relation to the cochlear microphonic) and also the difficulties in recording it with appropriate controls for contamination by stimulus artifact, the investigation of its habituation was not pursued.

Footnotes - Chapter Four

(1) In retrospect, this technique of interrogating interactions with 1 df comparisons that encapsulate experimental hypotheses could usefully be applied to the interaction of the fast and slow habituatory effects that was of central interest in Experiments Seven and Eight. In particular the phenomenon of 'potentiation of habituation' could be re-expressed in a single df comparison that embodies a systematic decline in the magnitude of the 'first stimulus effect' over blocks (see Chapter 13 of Kandel (1976), and in particular Fig. 13.8B). Accordingly, the magnitude of this effect was evaluated for each of the blocks 1 to 10 in the combined amplitude data of Experiments Seven and Eight, and a linear trend test applied to these values was found to be statistically significant, with $F(1,144) = 3.91^{*}$. However, further inspection of this linear trend indicated it to be simply a product of the 'first stimulus effect' being most pronounced on the first block of the session, with no significant decline thereafter ($F(1,144) = 10.11^{***}$), and that this effect was, in turn, due to the EP evoked by the very first stimulus in the session being exaggeratedly large. Nonetheless, some sort of 'potentiation of habituation' is apparently operating in Experiments Seven and Eight. However, although a case for discarding the first block of trials in a habituation experiment could be made if it be required that the across-block averaged EPs exactly mirror the effect of stimulus number in each block, the overall distortion introduced by this interaction is not serious, constituting simply a marginal over-estimate of the amplitude of the brain potential evoked by the first stimulus in the sequence. Discarding the first block of each subject's data in Experiments Seven and Eight in this way, for instance, leads to a value for N1-P2 for stimulus number 1 of 1.89, i.e. only 7% short of the value of 2.03 that appears in Table 4.4i.

(2) Pribram and McGuiness, 1975.

CHAPTER FIVE

MECHANISMS OF THE FAST RESPONSE DECREMENT OF THE VISUAL EVOKED POTENTIAL

In the introductory discussion of the habituation of EPs in Chapter One, the phenomenon of the temporal recovery of the EP was described. This phenomenon refers to the EP amplitude refractoriness induced by a single preceding stimulus and implies a physiological rather than a psychological investigator bias. However, given the considerable procedural overlap between investigations of the temporal recovery and the fast habituation of the EP[1], and the fact that temporal recovery is classifiable as habituation under an operational definition of the same, it was suggested that both phenomena (i.e. temporal recovery and fast habituation) be subsumed together within a single generic class of EP fast response decrements (FRDs). Both phenomena, it was argued, describe an EP amplitude decrement induced by a recent (i.e. within a previous interval of the order of 10 seconds) prior stimulus, and the present chapter and the next investigate the mechanisms of this decrement. Although both fast habituation and temporal recovery define a decrement in response strength (EP amplitude) with stimulus repetition, the forward-latency-shift of the EP that was observed to accompany the amplitude decrements in the last chapter and was subsumed, for convenience, under the mantle of habituation as a secondary characteristic thereof, will also receive some attention.

In this investigation of mechanisms, paradigms based on pairs of stimuli are used extensively, mainly from necessity but also for

reasons of temporal economy. Although this simplification presents no difficulties in applying any conclusions deriving from this research to the temporal recovery of the EP (whose defining paradigm, after all, involves pairs of stimuli) it may be felt, however, in respect of fast habituation that the truncation of the long trains of stimuli that are employed in this context, to effectively trains of length 2, somehow represents a qualitative departure from this standard procedure. Although difficult to refute conclusively, this objection is countered at this juncture with the following two points: firstly, the fast habituation phenomenon is essentially complete after the first two stimuli of the train, with no novel trends developing thereafter; and secondly, as the results of experiment 10 will show, the same degree of response attenuation occurs to the second stimulus of a pair (59%) as to the second stimulus of a train (Experiments 7 and 8, which employed the same data-analysis as Experiment 10, demonstrated the near identical figure of 60%). This latter evidence in particular strongly suggests that the same process is operating over both pairs and trains of stimuli.

However, it is recognised that although fast habituation is essentially a 'first stimulus effect', further response decrements beyond stimulus two are often observed (e.g. Experiment 9). However, once the mechanism of the major decrement between stimulus nos. 1 and 2 has been isolated and identified, a powerful logical case may then be possible to extend this mechanism to account for any subsequent effects.

Experiment 10: A Preliminary Investigation in order to Establish a Satisfactory Inter-pair Interval

INTRODUCTION

The objectives of this preliminary study were firstly, to verify that the second stimulus of a pair shows the same amplitude attenuation as the second stimulus in a train and secondly, to investigate the interaction of this decrementation effect with inter-pair interval (IPI). The definition of the FRD on page 166 of this chapter indicates that an interval between stimuli of less than 10 seconds is required to produce this decrement, thus suggesting that a highly economical IPI of 10 seconds could be safely employed without distorting the within-pair effect. Indeed the recovery function data presented in Fig. 1.3 suggests that, for the visual EP, an IPI as short as 5 seconds may well be satisfactory. The main purpose of this study was thus to confirm that an IPI of 10 seconds, or even less, would indeed be satisfactory for the present work.

METHOD

The EEG was recorded monopolarly from the vertex of the three subjects who participated in the experiment. Sixteen pairs (ISI = 1 sec) of chequerboard flashes (stimulation details as per the experiments of Chapter Four), separated by an IPI of either 5, 10, 20 or 60 seconds, constituted the experimental session. Each IPI occurred 4 times in a sequence balanced for order effects. The subjects were directed to pay passive attention to the stimuli; to avoid blinking and to fixate the central cross on the chequerboard throughout.

The single trial analysis employed in Experiments 2, 7 and 8 was

again used to assess the latency and amplitude of N1-P2 for all of the 32 stimuli in the experiment. The mean N1-P2 amplitude for the first and second stimuli of the four pairs associated with each IPI was calculated, and subjected to a two-factor (IPI, stimulus position) repeated measures analysis of variance.

RESULTS AND DISCUSSION

The means for the two main effects, their interaction and the analysis of variance summary table are compiled in Table 5.1 (for key to significance levels and further presentation details, see the first section of Chapter Two). The significance of the main effect of stimulus position ($F(1,14) = 46.67^{***}$) indicates the second stimulus of a pair to be depressed relative to the first; and the magnitude of this effect, as already indicated, is virtually identical to that observed (using the same ISI and data-analysis) between the first and second stimuli in the trains of flashes used in Experiments 7 and 8.

Neither the main effect of IPI, nor the IPI x stimulus position interaction are significant ($F<1$ in both cases), indicating that short IPIs do not distort the FRD. However, there is a slight, although non-significant, tendency for the effect to be diminished at the 5 second IPI relative to the 10 second interval (comp. 1; $F(1,14) = 1.54$ ns). Thus, to be on the safe side, the results of the study are taken to recommend an interval of the order of 10 rather than 5 seconds as the minimum satisfactory IPI.

If the FRD observed in the present study for pairs of stimuli is indeed the quintessence of the fast habituation effect of the previous chapter, then it should not only display the same magnitude at a given ISI

TABLE 5.1: EXPERIMENT 10

i) Mean N1-P2 amplitude as a function of interpair interval (IPI, secs) and stimulus position (S; 1 = first in the pair, 2 = second).

		S		
		1	2	
IPI	5	1.36	.89	1.13
	10	1.56	.79	1.17
	20	1.43	.91	1.17
	60	1.40	.85	1.13
		1.44	.86	1.15

ii) Summary of the analysis of the effects of IPI (I) and stimulus position (S) upon N1-P2 amplitude. For key to significance levels see Section 1 of Chapter 2.

Source	df	SS	MS	F
Subjects	2	.108		
I	3	.012	004	<1 ns
S	1	2.007	2.007	46.67 ***
I x S	3	.078	.026	<1 ns
Error (pooled **)	14	.600	.043	
Total	23	2.805		

iii) Single df comparison

Source	Comparison	MS	F
I x S, S	compl:(effect of S at 5 sec IPI)-(effect of S at 10 sec IPI), i.e. .47 - .77	.07	1.54 ns

but should also demonstrate the same properties. One of the properties of the within-train effect is its interaction with electrode placement, with a more posterior occipital location manifesting less decrementation. A short experiment involving four subjects was conducted in which their EEG was recorded from an occipital lead during the presentation of 50 pairs of chequerboard flashes (ISI = 1 sec, IPI = 10 sec). Two AEPs were computed for each subject, representing the brain response to the first and second stimuli of a pair. The amplitude of the N1-P2 wave was measured for both stimulus types, and, although the amplitude to the first stimulus was marginally larger than that to the second (11.82 μV as against 11.34 μV) this difference was far from being statistically significant ($t(df = 5) = .19$ ns). Thus the findings of experiment nine, which indicate that the fast response decrement of the visual EP over a train of stimuli is predominantly a vertex phenomenon, is corroborated here using pairs of stimuli. Finally, it should also be noted that, although latency data was not analysed in experiment 10, the N1-P2 evoked by the second stimulus of a pair, like the second of a train, is generally observed to occur at an earlier latency than that evoked by the first stimulus (e.g. in the data of experiment 11 , for instance, N1-P2 is found to be 19.5 ms earlier; $t(df = 5) = 2.45$*).

Summarising the findings presented and discussed in connection with this experiment, they can be formulated, in terms of the relationship between fast habituation and the use of pairs of stimuli, as indicating that the fast habituation of the EP, a phenomenon normally associated with long trains of stimuli separated by intervals of the order of a

minute or so, can indeed be essentially reduced to the response decrement that develops to the second stimulus of a pair with an interval of around 10 seconds elapsing between pairs. Thus the mechanisms of both subclasses of genus of EP FRDs (i.e. fast habituation and temporal recovery) can apparently be satisfactorily investigated with a single paradigm based on pairs of stimuli separated by an IPI of the order of 10 seconds.

Experiment 11: An Investigation to Determine whether the Fast Response Decrement of the Visual EP is a Peripheral or a Central Effect

In the third section of the introduction, two alternative hypotheses of the mechanism underpinning the fast response decrement of the EP were proposed. It was considered that the FRD had either a simple physiological explanation in terms of the refractoriness of certain of the neural nets involved in processing the stimulus information, or on the other hand, that a psychological explanation in terms of the decline in information (novelty) with stimulus repetition was more appropriate. The term refractoriness is specifically used to distinguish an invariant physiological property of neural nets from a response decrement that represents the operation of a labile psychological mechanism that governs the allocation of processing capacity, this latter mechanism being denoted psychological in the sense that it underpins the psychological construct of attention. Of the two alternatives the refractoriness mechanism, being the more parsimonious, is adopted as the initial standpoint of the thesis.

A standard procedure for investigating the centrality of visual phenomena (e.g. masking; Turvey, 1973) involves the use of dichoptic presentation. In the case of EP FRDs, if the second stimulus of a pair

is presented to the eye contralateral to the eye stimulated by the first stimulus, and the EP response decrement equal to that obtained with ipsilateral stimulation, then a locus for the refractory nets responsible for the FRD beyond the point of binocular convergence can be inferred. In man, although some interactions between inputs from the two eyes do exist at the level of the lateral geniculate body (Szentagothai and Arbib, 1975) binocular convergence is generally associated with a central cortical level of processing. Thus if contralateral stimulation produces an EP response decrement, then a precortical origin for the effect can be rejected in favour of a central one. Thompson and Spencer (1966) point out that response decrements may be due to receptor mechanisms such as pupillary diameter or receptor adaptation; the control study already presented in connection with experiment 9 controls for the first of these mechanisms, whilst the present dichoptic procedure does represent a control for the possibility of EP FRDs being produced by the second.

If, on the other hand, the FRD is demonstrated to depend upon which eye is stimulated by the second stimulus then, although a central locus cannot be categorically rejected, as centrally located nets could in some way be responsible for this interaction, a peripheral origin is certainly suggested.

The purpose of experiment 11 was therefore to employ a dichoptic stimulus presentation in order to establish whether the neural nets whose refractoriness produces the fast response decrement of the visual EP have a peripheral or a central locus.

METHOD

The EEG was recorded monopolarly from the vertex of the six subjects who participated in the experiment. 100 pairs of stimuli (IPI = 10 seconds, ISI = 1 sec), presented in two blocks of 50, constituted the experimental session. Using the apparatus depicted in Fig. 5.1, either eye could be monocularly stimulated with an LED flash (10 ms duration). Two red LEDs were mounted on opposite sides of a midline at the focal length of the two lenses A1 and A2. Parallel light from both sources was arranged to be directed onto a third lens, B, whose focal plane coincided with the subject's frontal plane. From the subject's viewpoint stimulation consisted of the brief uniform illumination of the aperture O. The red background light D remained illuminated throughout obscuring any unwanted stray light, including that scattering off the front surface of B.

Four types of stimulus pair, corresponding to the combinations left eye first stimulus, right eye second (LR); left first, left second (LL); right first, left second (RL) and right first, right second (RR) were used; the 100 pairs of the session representing a random permutation of 25 of each type. Subjects were required to fixate the aperture throughout and to maintain a constant level of passive attention. Prior to each session the brightness of the LEDs was adjusted such that right and left monocular stimulation was identical. One of the features of monocular stimulation is that the subject is not able to tell which eye, per se, is being stimulated and thus, if the two monocular stimuli are physically the same, as in the present experiment, all stimuli, whether right of left eye, will be perceptually equivalent. Thus the contralateral second stimulus should generate the same percept and processing as an ipsilateral one, allowing

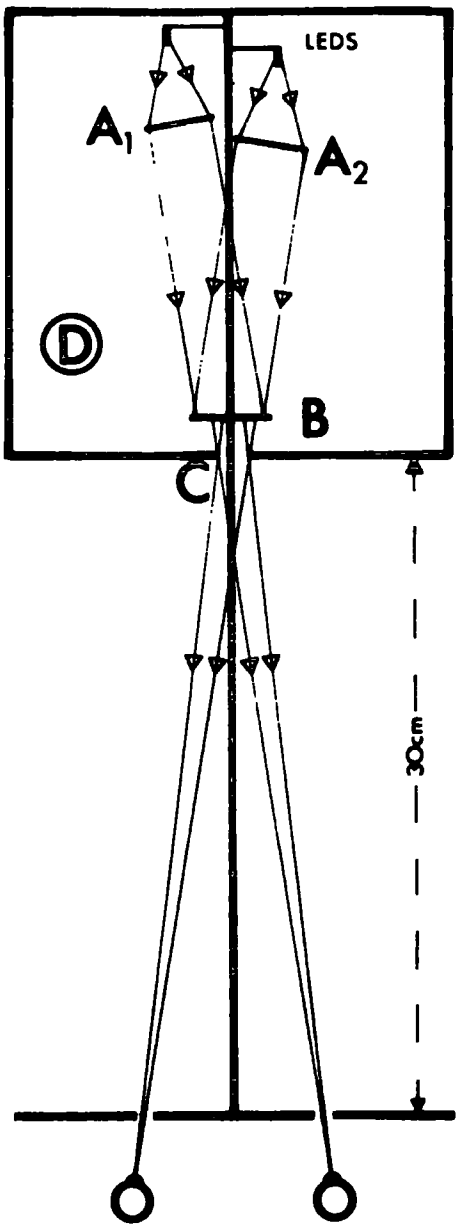

Figure 5.1

The apparatus of experiment 11 viewed from above. See text for description.

unambiguous comparisons to be made between the two stimulus types. When asked at the end of the session subjects confirmed that they had not been aware of any systematic differences between stimuli; and were surprised to learn that, what to them was a sequence of essentially identical stimuli, in fact was comprised of two fundamentally distinct (right vs left eye) types of stimulation.

RESULTS

Three AEPs were computed for each subject representing the brain activity evoked by A) the first stimulus of a pair (1), B) the second stimulus of a pair delivered to the eye ipsilateral to the first stimulus (2I) and C) the second stimulus of a pair delivered to the eye contralateral to the first (2C). Owing to the 'noisiness' of these records various data-analytic techniques, including factor analysis and discriminant analysis, were applied to the data. The technique finally adopted involved a conventional component analysis of the AEPs, using digital filtering to facilitate the identification of components. The filtered waveforms for one of the subjects have already been presented in Fig. 2.5b. The results for the two further subjects are shown in Fig. 5.2, and the general impression that emerges, which is supported by the discriminant analysis discussed in Chapter Two, is that the AEP evoked by the first stimulus (A,1) is quite distinct from the AEPs to both an ipsilateral (B,2I) and a contralateral (C,2C) second stimulus, and that these latter two AEPs are themselves not dissimilar. This statement is further supported by the component analysis that occupies the next two paragraphs.

The amplitude and latency of N1 and P2 was extracted from the AEP for each condition and subjected to statistical analysis. The various

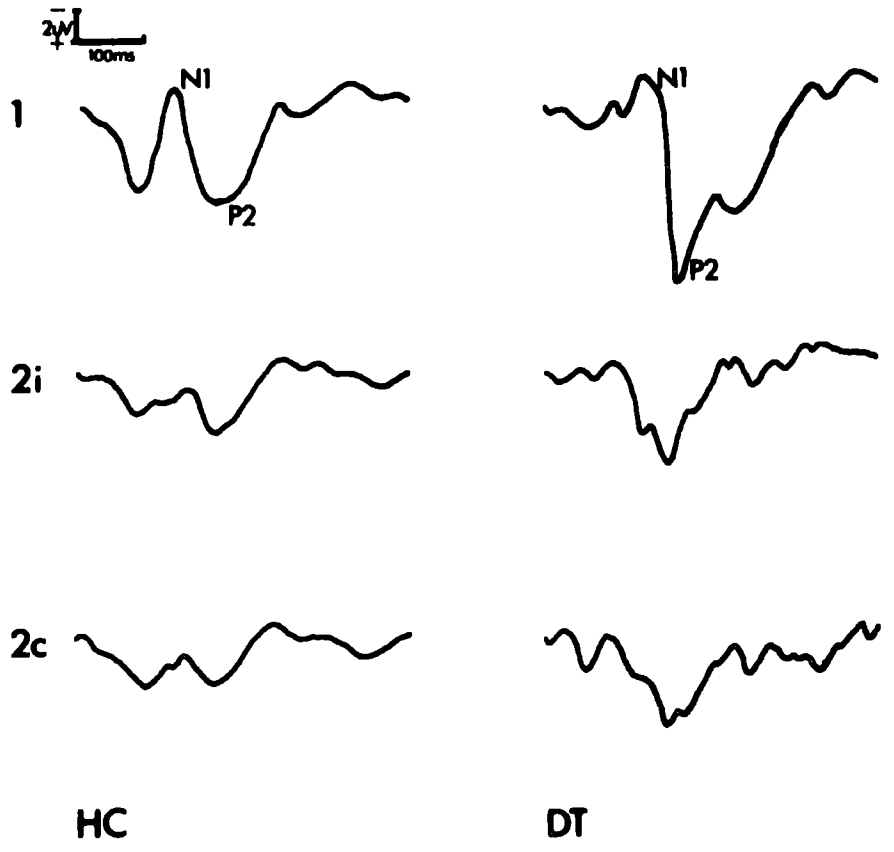

Figure 5.2

Experiment 11: Digitally filtered AEPs for subjects HC and DT associated with the first stimulus of a pair (1), an ipsilateral second stimulus (2i), and a contralateral second stimulus (2c).

means and analysis of variance summary tables are assembled in Table 5.2. The amplitude of N1-P2 is significantly larger for the first stimulus of a pair irrespective of the laterality of the second (Comp. 1; $F(1,25) = 13.19^{**}$) and this response decrement is as equally developed for contralateral stimulation as it is for ipsilateral (Comp. 2, $F<1$). Although P2 is significantly larger than N1 ($F(1,25) = 198.54^{***}$) the stimulus type x component interaction fails to approach significance ($F<1$).

The analysis of the latency data reveals much the same picture, with the latency of both N1 (Table 5.2iii, Comp. 3; $F(1,10) = 4.57^{*}$) and P2 (Table 5.2iv, Comp. 5; $F(1,10) = 14.57^{***}$) being larger for the first stimulus. Again no laterality effects are evident (Comp. 4, $F<1$; Comp. 6, $F(1,10) = 2.33$ ns).

DISCUSSION

The failure to demonstrate an interaction of the FRD (and the concomitant latency shift) of the N1-P2 component of the visual EP with the laterality of the second stimulus provides good evidence that the networks whose refractoriness is responsible for the FRD are located centrally. Indeed, if a peripheral sensory mechanism was the case, then, intuitively, it would be expected that the FRD would be more evident at an electrode more proximate to the primary visual receiving areas. The results of experiment 9, which show this not to be the case, are thus also not consistent with a peripheral mechanism and hence provide further support for the findings of this study. As far as identifying which cortical networks are involved it is possible, for instance, that the refractoriness is a property of the networks directly underlying N1-P2.

TABLE 5.2: EXPERIMENT 11

i) Mean EP amplitude (μV) and latency (ms) as a function of component (N1,P2) and stimulus type (1 = first stimulus, 2i = 2nd stimulus ipsilateral to the first and 2c = 2nd stimulus contralateral to the first).

Amplitude:

	N1	P2	
1	1.03	6.21	3.62
2i	-.24	4.69	2.23
2c	-.61	4.92	2.15
	.06	5.28	2.67

Latency:

	1	2i	2c	mean
N1	145	118	128	130
P2	209	189	197	198

ii) Summary of the analysis of the effects of component (C) and stimulus type (T) upon EP amplitude, including single df comparisons.

Source	df	SS	MS	F
Subjects	5	24.21		
C	1	245.24	245.24	198.54 ***
T	2	16.39	8.20	6.61 **
T x C	2	.55	.28	<1 ns
Error (pooled **)	25	30.88	1.24	
Total	35	317.27		

Orthogonal comparisons

Source	Comparison	MS	F
T	comp 1: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	16.36	13.19 **
	comp 2: 0 1 -1	.03	<1 ns

iii) Summary of the analysis of the effect of stimulus type (T) upon N1 latency, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	5	4,250	850.0	
T	2	2,325	1,162.5	2.63 ns
S x T	10	4,425	442.5	
Total	17	11,000		

Orthogonal comparisons

Source	Comparison	MS	F
T	comp 3: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	2,025	4.57 *
	comp 4: 0 1 -1	300	<1 ns

iv) Summary of the analysis of the effect of stimulus type (T) upon P2 latency, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	5	2,000	400.0	
T	2	1,225	612.5	8.45 *
S x T	10	725	72.5	
Total	17	3,950		

Orthogonal

Source	Comparison	MS	F
T	comp 5: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	1,056	14.57 *
	comp 6: 0 1 -1	169	2.33 ns

However, any cortical net earlier in the information processing sequence could also be responsible. In the auditory modality Ruhm and Hess (1972), using dichotic presentation, obtained similar results to those of this visual study for the N1-P2 wave of the auditory EP, and they too conclude that their findings are an expression of the temporary "inexcitability" (refractoriness) of binaurally driven (and therefore cortical) neuronal populations that is produced by a prior signal. They further point out that the magnitude of the response decrement produced by a contralateral second stimulus will be a function of the overlap in the cortical projections from the two ears, and thereby deduce that, because an ipsilateral auditory second stimulus produces a greater decrement than a contralateral one, these projections are not entirely common. However, the visual data of this study indeed demonstrates that the response decrement to a contralateral second stimulus is equal to that obtained for ipsilateral stimulation. Following Ruhm and Hess (ibid), the suggestion is that, unlike the ears, there is complete overlap in the projections from the two eyes and thus one might tentatively deduce that information about which eye is being stimulated will not be available to the subject, this latter deduction being in agreement with the subject's introspections.

Experiment 12: A Brief Experiment to examine the Refractoriness Explanation of the Visual EP

INTRODUCTION AND METHOD

If the FRD of the EP is a reflection of the refractoriness of cortical neural nets then it is reasonable to argue, as per Ruhm and Hess (1972), that the magnitude of the response decrement will be a function of the degree of overlap between the populations of cells activated

by the first and second stimulus. With complete overlap, i.e. identical stimuli, the decrement will be at a maximum, but will decline as the dissimilarity in the patterns of neural activity increases. In the language of habituation, this function constitutes a gradient of generalisation of habituation and the purpose of experiment 12 was to investigate whether such a gradient exists.

Four subjects were recruited and their EEG was recorded monopolarly from a vertex placement. Two blocks of stimuli comprised the experimental session, each block consisting of 25 pairs of visual stimuli (ISI = 1 sec, IPI = 8 seconds). Two types of pair were used for the two blocks: a chequerboard second stimulus (used to maintain continuity with the bulk of the FRD research already presented) was preceded by either an identical chequerboard (condition A) or a stimulus of quite different spatial structure and retinal projection, namely, an annulus (condition B). The stimuli were presented using a tachistoscope (stimulus duration = 40 ms) and the order of conditions was counterbalanced across subjects. Figure 5.3 depicts the chequerboard and the annulus, and it will be noted that retinal projections of the two stimuli will be quite distinct providing the subject maintains a central fixation. Such a fixation point (a dark spot on a white background) was provided and the subject was instructed to fixate it throughout, whilst passively attending to the stimuli.

RESULTS AND DISCUSSION

Three AEPs were computed for each subject corresponding to the brain activity evoked by the chequerboard stimulus under the three circumstances in which it occurred, i.e. first (1); second, following its presentation

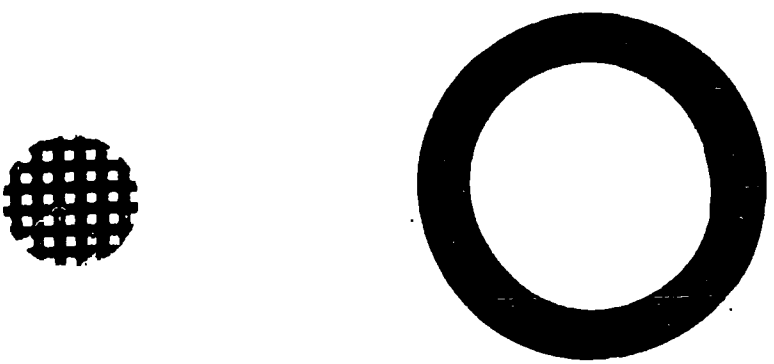

Figure 5.3

The chequerboard and the annulus used in experiment 12. These stimuli, which are drawn to scale, were mounted on white cards and presented tachistoscopically.

as the first stimulus (2S); and second, following the annulus (2D). The amplitude of both N1 and P2 was measured for each AEP and subjected to statistical analysis, the analysis of variance summary table and the table of means appearing in Table 5.3 (latency data was not collected).

Inspection of Table 5.3 reveals that the amplitude of N1-P2 is larger for the first stimulus of a pair (Comp. 1; $F(1,15) = 6.26^{*}$) and that the FRD is equally developed irrespective of the congruence between the two stimuli of the pair (Comp. 2, $F<1$). Although P2 is significantly larger than N1 ($F(1,15) = 74.16^{***}$), the stimulus type x components interaction is not significant indicating the FRD to be comparable for both components.

The failure of the present study to demonstrate a diminution in the FRD when the second stimulus of a pair, having a quite different spatial structure and retinal projection, presumably generates a substantially distinct pattern of neural activity, engenders serious doubts over the validity of interpreting the FRD as a simple refractoriness phenomenon. The results of a study by Boddy (1973) which employed pairs of stimuli separated by comparable ISIs (although in a different theoretical context) are also germane to this discussion. In his investigation of the relationships between EPs, reaction time and foreperiod duration he found that amplitude of N1-P2 evoked by an auditory imperative stimulus was identical, irrespective of the modality (auditory or visual) of the preceding warning signal. Although it is possible that the cortical refractoriness induced by prior stimulation generalises completely to all succeeding stimuli, whatever their form or modality, such a notion is

TABLE 5.3: EXPERIMENT 12

i) Mean EP amplitude (μV) as a function of component (N1,P2) and stimulus type (1 = chequerboard in first position of pair, 2S = chequerboard following chequerboard first stimulus, and 2D = chequerboard following annulus first stimulus).

	N1	P2	
1	1.58	5.78	3.68
2S	-1.56	5.16	1.80
2D	-2.06	5.56	1.75
	-0.68	5.50	2.41

ii) Summary of the analysis of the effects of component (C) and stimulus type (T) upon EP amplitude.

Source	df	SS	MS	F
Subjects	3	28.05		
C	1	229.15	229.15	74.16 ***
T	2	19.37	9.68	3.13 ns
T x C	2	12.57	6.27	2.03 ns
Error (pooled **)	15	46.34	3.09	
Total	23	335.58		

iii) Orthogonal comparisons

Source	Comparison	MS	F
T	comp 1: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	19.35	6.26 *
	comp 2: 0 1 -1	.02	<1 ns

neither considered likely nor useful. Certainly such a general refractoriness does obtain at shorter ISIs, being manifest in the delay in producing a reaction time to the second of two closely-spaced stimuli (Smith, 1967) but not at intervals as long as 1 second. Furthermore this latter refractoriness is reflected in a corresponding delay rather than foreshortening in the latency of N1-P2 (Surwillo, 1977); and indeed, the very fact that the amplitude decrements that have pre-occupied the last two chapters of the thesis are associated with a forward latency shift in itself suggests that a refractoriness explanation is inappropriate.

Experiment 13: A Neuropsychological Explanation of the Fast Response Decrement of the Visual EP

INTRODUCTION

In this final section of Chapter Five, the neuropsychological explanation of the FRD of the EP, outlined in the third section of the introductory chapter and briefly recapitualted in the introduction to Experiment 11, is evaluated. Although this explanation involves attentional factors (in the form of a capacity theory of attention), a simple attentional explanation of the FRD (in terms of say, arousal) is clearly not adequate, as indicated by the tendency for reaction time to improve rather than lengthen as the amplitude of the EP declines over the course of a sequence of repetitive stimuli (Ohman and Lader, 1972). For the sake of clarity, it is emphasised that by attention in the context of this experiment (and those of the next chapter), the selectivity of attention in the sense of the operation of a pre-set filter upon information from an unpredictable source (as in the

experiments of Chapter Three) is not meant. It has already been shown in this earlier chapter that one of the two EP components of interest here, namely N1, does not reflect this mode of attention in the visual system. Rather, attention is used to denote the investment of processing capacity by the subject in those circumstances in which he can specifically prepare himself in advance of the occurrence of stimuli. In these circumstances the lability of both the N1 and P2 components of the visual EP with respect to attention is well known (e.g. Spong et al, 1965).

Recapitulating the arguments of the introductory chapter, a neuropsychological explanation of the FRD of the EP a) introduces the concept of a neuronal model of a stimulus; b) embraces a capacity model of attention, indicating the amplitude of the late components of the EP to index the allocation of processing capacity to a stimulus; and c) argues that, to the degree that the cortex is concerned with updating the neuronal model of the stimulus, the allocation of capacity (and hence the amplitude of the EP) corresponds to the extent that this internal model is deemed inadequate. The FRD in the amplitude of the EP with stimulus repetition is accounted for, in these attentional terms, as follows. Upon the presentation of the first stimulus in a pair or longer sequence, the neuronal model is held to be maximally deficient and a relatively large investment of processing capacity is therefore required in order to extract the relevant information from the stimulus in order to remedy this deficiency. As a result of this processing the model is 'improved', with the consequence that less mental work is generated by subsequent stimuli, and the amplitude of the late componentry of the EP,

which indexes mental work, therefore shows a corresponding decrement. It is further relevant to follow through an additional implication of this orientation; namely, that, with the neuronal model better 'tuned' to the stimulus after its first presentation, it is reasonable to suggest that, not only will the necessary mental work decline, but such work as is required will be completed more speedily, thus explaining the forward-latency-shift of the EP that is found to accompany the amplitude decrement. The facilitation of RT with stimulus repetition in Ohman and Lader's (1972) study may be similarly explained.

The findings of Experiment 11 of the thesis provide further support for the contention advanced in Chapter One that in habituation and temporal recovery experiments the neuronal model is primarily inadequate in its temporal aspects. In such experiments the content of the stimulus events themselves are completely known to the subject (his being informed of the general organisation of the experiment at the outset) and are therefore specified in the model; it is their precise location in time that is only imperfectly known. Thus in these experiments the occurrence of a stimulus resolves temporal, not event uncertainty. Since the temporal uncertainty associated with a chequerboard succeeding another chequerboard is equal to the temporal uncertainty associated with a chequerboard succeeding an annulus (the ISIs being the same), the amplitude of their respective EPs will also be identical. The independence of the FRD from the physical characteristics of the prior stimulus, as observed and discussed in Experiment 11, is thus explained.

Recapitulating then, the FRD of the EP is considered to be simply a reflection of the variation between stimuli in the deficiencies of the neuronal model of the temporal aspects of stimulation (i.e. temporal uncertainty) and hence in the necessary investment of capacity to remedy these deficiencies. The amplitude of the EP is held to index the investment of capacity and hence it covaries with temporal uncertainty producing the FRD as follows. The temporal uncertainty associated with the first stimulus of a train or pair is necessarily greater (and hence also the amplitude of its EP) than that associated with succeeding stimulation; and in the case of trains of stimuli, as the train progresses temporal uncertainty (and EP amplitude) increasingly declines until some minimum value, which represents the uncertainty that inevitably re-accumulates over the ISI, is reached. This minimum value will be proportional to the length of the ISI^2, thus accounting for the effects of ISI upon the asymptotic value of the fast habituation of the EP that were observed in Experiment 9. In general, the temporal uncertainty associated with a stimulus will be proportional to the length of the interval that has elapsed since the last stimulus (see Footnote 2 again), thus generating the FRD-versus-time plot of the temporal recovery of the EP. It would appear from this graph (see Fig. 1.3) that although temporal uncertainty accumulates rapidly immediately after a stimulus, it reaches an asymptotic maximum in terms of its effects upon EP amplitude within 10 seconds.

Reconstructing the logic of the current experimentation, the mechanisms of both the short-term habituation and the temporal recovery of the EP are investigated using pairs of stimuli, i.e. with procedures

that logically reduce the FRD to the relationship between two types of stimulus event: a 'first' stimulus, which follows a long silent interval (of the order of 10 seconds or longer) and a 'second' stimulus which occurs within a few seconds of prior stimulation. The theory outlined above indicates that the EP amplitude depression of a 'second' stimulus relative to a 'first' (i.e. the FRD) is a function of the greater temporal information content, and therefore attentional value, of the latter. It therefore follows that, if a 'second' stimulus were equated with a 'first' in terms of its temporal uncertainty, then no EP amplitude difference between the two should be obtained, i.e. that the FRD will be abolished. Experiment 13 evaluates the theory of this section with an investigation of this prediction.

METHOD

The EEG was recorded monopolarly from the vertex of the five subjects who were recruited for the experiment. The experimental session was comprised of 140 trials arranged in three blocks of 40, 50 and 50 trials, the first block of 40 being for practice purposes. Trials were constituted as follows (see Fig. 5.4): a spot was driven at a uniform speed across an oscilloscope screen, the time taken to complete a single traverse being 10 seconds. A second stationary spot marked the end of the traverse and when the moving spot reached this destination on a random 50% of trials a chequerboard was briefly flashed onto the screen. The subject was required to key-press as rapidly as possible when the chequerboard occurred, but to avoid responding in its absence.

The same chequerboard was additionally presented in the same position on the screen on half of the trials (again at random) at a

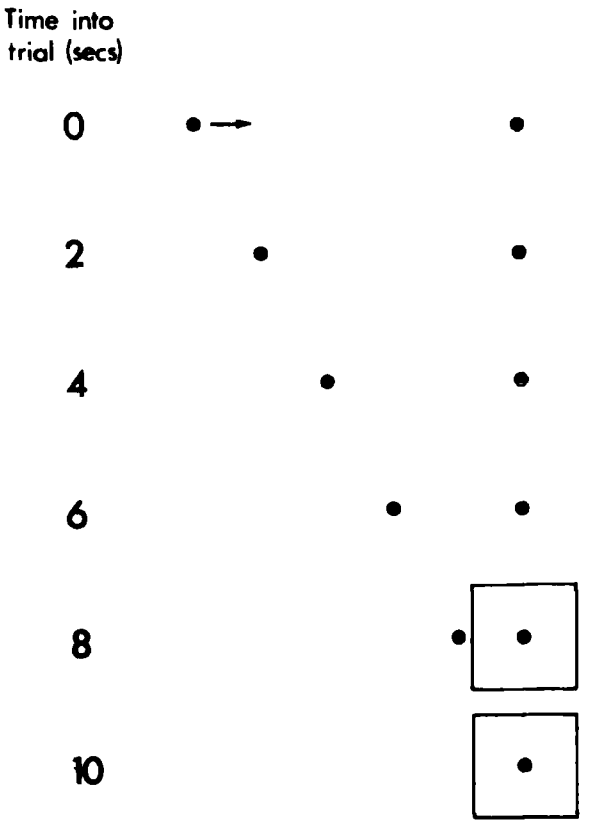

Figure 5.4

The 'clock' paradigm of experiment 13 is illustrated in the 'typical' trial depicted above. A spot is driven across an oscilloscope screen towards a stationary spot. On this particular trial the chequerboard (represented by the square) is flashed onto the screen twice: at the end of the spot's traverse (the imperative 'second' stimulus-event 2P) and 2 seconds before this point (the irrelevant prior stimulus - event 1). See text for further details.

random interval 1 to 3 seconds (i.e. within the range over which Experiment 9 has already demonstrated the visual FRD to operate) prior to the end of the spot's traverse. The subject was informed that such an event was purely a distraction requiring no response, and that it provided no information pertaining to the presence/absence of an imperative stimulus on the current trial. Thus the experimental procedure generated three types of stimulus event: an irrelevant chequerboard (1), an imperative chequerboard preceded by an irrelevant chequerboard (2P) and an imperative chequerboard on its own (1S). The comparison of interest is between these latter two events, i.e. between the 'second' stimulus of 2P and the 'first' stimulus of 1S. Informationally they are equivalent, both in terms of event uncertainty (the irrelevant chequerboard has no predictive value) and temporal uncertainty, this latter equivalence having been achieved by the complete elimination of temporal uncertainty by the provision of the oscilloscope 'clock'[3]. The requirement of an immediate key-press to both events, a measure included to obtain some behavioural feedback on the subject's attentive state, would also tend to equate the events in terms of their respective processing demands. The arguments outlined above thus predict that, despite the presence of a prior stimulus on event 2P, the late components of the AEPs evoked by 2P and 1S should be equal in amplitude (and latency). The time of occurrence of event 1 was randomised to remove the possible signalling utility that a constant temporal relationship with the end of the traverse could provide. Such a utility could have introduced a difference in the subject's use of the 'clock' on trials with an irrelevant stimulus, thus jeopardising the equivalence of events 2P and 1S.

Subjects were instructed to key-press accurately and rapidly, and to avoid tracking the moving spot by maintaining a fixation upon the stationary spot. Both the irrelevant and imperative chequerboards were flashed onto the screen centred around this point of fixation. The subjects were seated five feet from the oscilloscope and the total display area used subtended 3 degrees at the eye. A period of 1 second was allocated for the subjects response before the moving spot was reset and the next trial begun. The early stages of the 'clocks' cycle represented a 'silent' time for AEP recovery of 7-9 seconds, a period considered satisfactory with reference to the recommendations of Experiment 10.

RESULTS

The AEPs (digitally filtered) for stimulus events 1, 2P and 1S are shown for two typical subjects in Fig. 5.5; and the overall visual impression is that, whereas the AEP associated with 1 is distinct from both 2P and 1S, these latter two events evoke similar brain potentials. The amplitude and latency of N1 and P2 was extracted from the AEPs of all five subjects and subjected to statistical analysis, the tables of means and analysis of variance summary tables being shown in Table 5.4.

Considering the amplitude data first, an initial 2 factor repeated measures analysis with stimulus type (T; 1, 1S, 2P) and component (C) as the factors revealed a significant T x C interaction; and as such an interaction complicates the presentation of the results, this analysis was substituted by two 1 factor analyses, one for each component. Comparisons 1 and 3 (Table 5.4ii,iii) indicate that the amplitude of N1

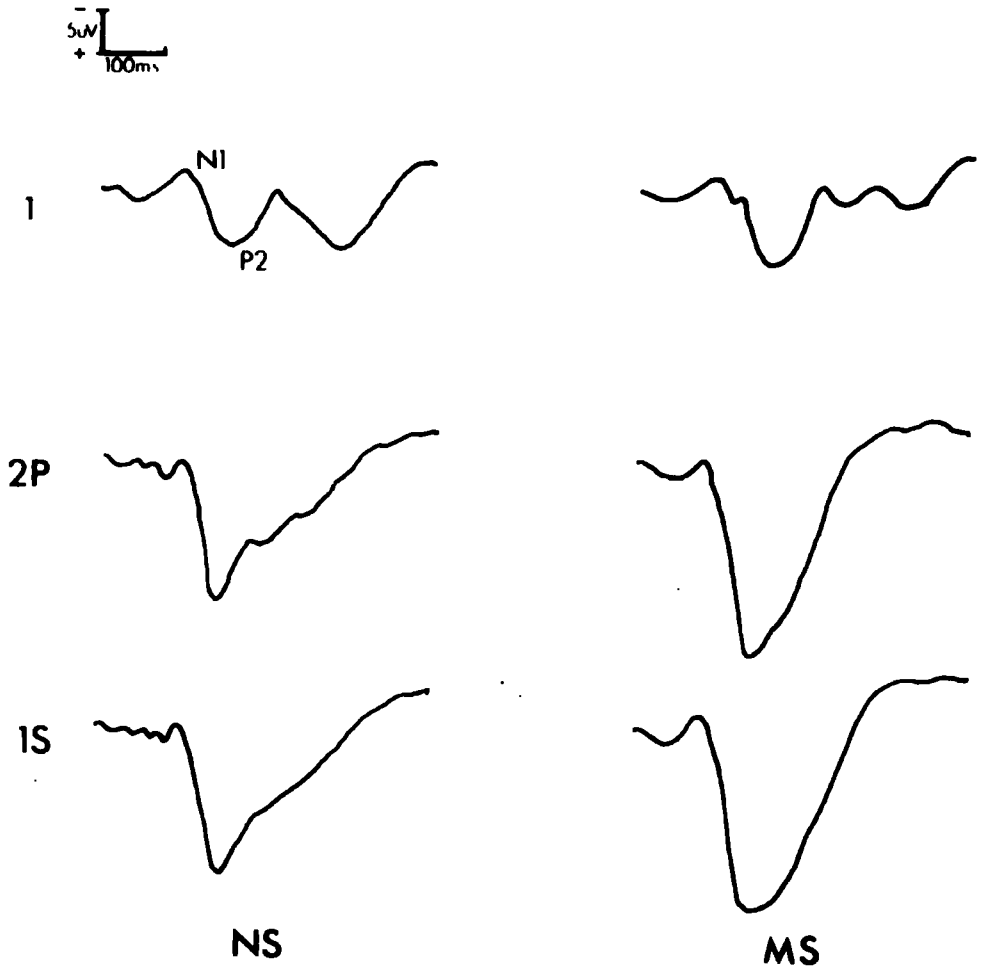

Figure 5.5

Experiment 13: Digitally filtered AEPs for subjects NS and MS associated with stimulus events 1, 2P and 1S.

TABLE 5.4: EXPERIMENT 13

i) Mean EP (N1,P2) amplitude (μV) and latency (ms) as a function of stimulus type (1,1S,2P - see text for details).

	1	1S	2P	
N1 amplitude	2.96	-1.20	-2.19	-0.14
P2 amplitude	7.87	18.66	18.66	15.06
N1 latency	134	112	114	120
P2 latency	216	236	231	228

ii) Summary of the analysis of the effect of stimulus type (T) upon N1 amplitude, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	4	36.16		
T	2	74.72	37.36	5.56 *
T x S	8	53.73	6.72	
Total	14	164.61		

Orthogonal comparisons

Source			
T	comp 1: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	72.76	10.75 **
	comp 2: 0 1 -1	2.46	<1 ns

iii) Summary of the analysis of the effect of stimulus type (T) upon P2 amplitude, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	4	180.46		
T	2	387.65	193.82	20.91 ***
T x S	8	74.16	9.27	
Total	14	642.27		

Orthogonal comparisons

Source	Comparison	MS	F
T	comp 3: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	387.65	41.82 ***
	comp 4: 0 1 -1	0	<1 ns

iv) Summary of the analysis of the effect of stimulus type (T) upon N1 latency, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	4	13,533.33		
T	2	1,480.00	740.00	11.03 **
T x S	8	536.67	67.08	
Total	14	15,550.00		

Single df comparison

Source	Comparison	MS	F	F residual
T	comp 5: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	1,470.00	21.91 ***	<1 (1,8) ns

v) Summary of the analysis of the effect of stimulus type (T) upon P2 latency.

Source	df	SS	MS	F
Subjects (S)	4	10,743.33		
T	2	1,083.33	541.67	1.22 ns
T X S	8	3,566.67	445.83	
Total	14	15,393.33		

is greater for event 1 than for either event 2P or 1S ($F(1,8) = 10.75^{**}$) and that the converse holds for the amplitude of P2 ($F(1,8) = 41.82^{***}$). Events 2P and 1S themselves evoke equal amplitude N1s and P2s, with the F ratios for comparisons 2 and 4 failing to achieve significance ($F<1$ in both cases).

The latency data expresses much the same trends. No differences in the latencies of either N1 or P2 evoked by 2P and 1S are apparent, with the only significant effect being the delay in the latency of N1 evoked by event 1 (Table 5.4iv, Comp. 5; $F(1,8) = 21.91^{***}$). Analysis of the performance data reveals no reaction time differences between events 2P and 1S (RT2P = 227.2, RT1S = 225.2 ms; $t(df = 4) = .94$ ns), inferring that the experimental manipulations were successful in equating the two events. This conclusion is further supported by the introspections of the subjects who reported that the events were indeed 'psychologically equivalent'.

DISCUSSION

The neuropsychological orientation elaborated in Chapter One and in the introduction to this experiment proposes the notion that the amplitude of N1-P2 simply indexes the necessary allocation of processing capacity (i.e. attention) to a stimulus in order to complete the required mental work. Thus if the mental work associated with a stimulus is specified, then the amplitude of N1-P2 is completely determined, and any other factors that do not influence this work (such as a redundant prior stimulus) will equally not affect the amplitude of N1-P2. The FRD of the EP is attributed to the decline in the mental work (attention) associated with repetitive stimulation as the neuronal model becomes more adequate. In temporal recovery and habituation procedures,

it is argued that this mental work is primarily concerned with abstracting temporal information from stimuli, and it follows that if temporal uncertainty is eliminated then, event uncertainty and other aspects of information processing being equal, no EP amplitude variations should be obtained. Employing a paradigm in which the quintessence of the FRD of the EP is encapsulated in the relationship between a 'first' stimulus and a 'second' stimulus, the results of this experiment confirm this prediction with no amplitude depression developing between the first stimulus of 1S and the attentionally equivalent second stimulus of 2P.

Other explanations and interpretations are, of course, possible. It could be, for instance, that the alerting properties (Loveless and Sanford, 1974; Loveless, 1977) of the 'irrelevant' prior stimulus produce an amplitude enhancement that exactly compensates for the FRD. However, the failure to find facilitated reaction times to the 2P event suggests such an explanation to be unlikely, and indeed it would be improbably fortuitous for any such effects to compensate exactly for the depression. Alternative interpretations in terms of other psychological constructs could also be advanced. However, all such interpretations reduce to a common logical crux, i.e. that the amplitude of the EP evoked by events 2P and 1S is the same because the events themselves are the same in terms of the chosen psychological construct; i.e. the subject is equally prepared, aroused or whatever. However, although the author recognises considerable potential in the preparation construct in particular, with the application of this construct in this context receiving some greater consideration in the general discussion section

of Chapter Seven, the interpretation in terms of the capacity model of attention is nonetheless maintained; firstly, because it represented the theoretical a priori of the author and secondly, because of its generality in explaining EP amplitude effects across a wide range of enquiry.

The finding that the amplitude of N1 is smaller and P2 larger for stimulus events 2P and 1S in comparison to event 1 can be attributed to the development of a greater overall positivity, possibly when (anticipating the results of Experiment 18 of Chapter Eight) a prior state of readiness is resolved. The increased processing demands associated with the imperative stimuli could also have produced such an overall positive shift (Ohtani and Yagi, 1971; Poon, Thompson and Marsh, 1976) and indeed the temporal uncertainty of stimulus event 1 could have contributed to the enhancement of its N1. The observation that N1 peaks significantly later for stimulus event 1 is consistent with both its temporal uncertainty and the absence of an imperative to process quickly.

In conclusion then, the data of this study are adduced as supporting an attentional mechanism for the FRD of the visual EP. A physiological refractoriness mechanism, which would predict that the EP evoked by stimulus event 2P should be smaller as an inevitable consequence of the refractoriness induced by the prior stimulus, can confidently be rejected.

However, before closing this chapter a post hoc control study is discussed. Considering Experiment 13 in retrospect it was recognised that, in addition to the provision of the 'clock', the procedure included two further innovations over the preceding experiments of the thesis

(namely, an irregular ISI and a behavioural response) which, either separately or in combination, could have been effective in removing the amplitude depression hitherto associated with prior stimulation. Although Ohman and Lader included both of these features in their 1972 experiment, and yet nonetheless obtained the usual response decrement, this experiment is not entirely satisfactory as a control here, as, unlike the current thesis experimentation, their work used auditory stimuli. Accordingly, a brief control study was conducted in order to assess the effect of an irregular ISI and the requirement of behavioural responding upon the FRD of the visual EP.

EEG was recorded from the vertex of the 6 subjects who participated in the study. Twenty pairs of chequerboard stimuli (IPI = 9 secs; ISI = random, in the range 1 to 3 seconds) were presented to the subject, a response being required to each member of the pair. Brain potentials associated with the first and second stimulus positions were averaged separately, filtered, and the amplitude of N1-P2 measured. Values for the two stimulus positions were 21.36 and 17.49μV respectively, representing an amplitude decrement in the EP to the second stimulus of 3.89μV, which proved to be statistically significant ($t(df = 5) = 2.97^{*}$). Thus it can be concluded that the complete elimination of differences in temporal uncertainly between stimuli via such a mechanism as the oscilloscope clock of Experiment 13 is a necessary condition for the abolition of the visual FRD.

Footnotes - Chapter Five

(1) As already indicated in the introductory discussion of the temporal recovery of the EP in the third section of Chapter One, some recovery function studies (e.g. Davis et al, 1966; Gjerdingen and Tomsic, 1970), presumably where within-block averaging is the only method available, indeed employ trains of stimuli. The AEP is calculated for a run of stimuli separated by a particular ISI and temporal recovery is inferred from the increase in the amplitude of this AEP with lengthening ISI. Such an indirect method is clearly inferior to across-pair averaging, and indeed the effect itself corresponds to the main effect of ISI observed in Experiment 9 of the thesis. Gjerdingen and Tomsic (1970) express their recovery functions as the percentage increase in the amplitude of the EP produced by doubling the ISI. For the N1-P2 wave of the vertex visual EP the increase comes to 16%, a figure not dissimilar to the 12% increase that can be derived from the data of Experiment 9.

(2) Certainly absolute error in time estimation is proportional to the duration to be estimated (Snodgrass, Luce and Galanter, 1967).

(3) This method of controlling temporal uncertainty using an oscilloscope 'clock', although seemingly complicated, represented, in the author's view the only truly satisfactory solution to this problem. Certainly, any simple device employing discrete 'warning' or 'prompting' stimuli of even quite different physical characteristics to the experimental stimuli would not be satisfactory given the extensive generalisation of the FRD discussed in experiment 12.

CHAPTER SIX

MECHANISMS OF THE FAST RESPONSE DECREMENT OF THE AUDITORY EVOKED POTENTIAL

In the previous chapter, the argument of the introductory chapter, that both the short-term habituation and the temporal recovery of the EP could be subsumed together within a single generic class of EP fast response decrements (FRDs) thereby permitting the investigation of the mechanisms common to both, was recapitulated. Employing paradigms in which the quintessence of the FRD is summarised as the amplitude decrement that develops between the first and second stimuli of a pair, it was eventually able, in experiment 13, to demonstrate that the variable producing this decrement was the greater temporal uncertainty associated with the first stimulus. It was thus concluded that an attentional explanation of the visual FRD, in which the attention paid to a stimulus (indexed by EP amplitude) reflects its information content (defined in terms of the deficiencies of the neuronal model of the stimulus), was appropriate.

The experiments of this chapter attempt to generalise this conclusion to the auditory modality, where much of the temporal recovery work and most of the fast habituation research has been conducted. The prototype 'clock' paradigm of experiment 13, which permits investigation of the relationship between the two types of stimulus event that summarise the FRD (the 'first' stimulus and the 'second' stumulus) with temporal uncertainty under experimental control, is employed throughout this chapter in varying degrees of refinement. Again, it is recognised that, although any conclusions deriving from the use of a paradigm based

on pairs of stimuli can be readily applied to the temporal recovery of the auditory EP, any such generalisation to the fast habituation phenomenon, where trains of stimuli are used, is more fraught. However, where in these latter procedures further decrements beyond stimulus number two are obtained, once, say, temporal uncertainty is identified as the mechanism of the decrement between stimulus numbers one and two, a powerful logical case can then be made out that these additional decrements reflect the operation of this same mechanism. Indeed, considering a neuronal model of a stimulus deficient in its temporal aspects, it would be unreasonable to preclude the possibility of further improvements in the model after the presentation of the second stimulus.

Experiment 14: An Investigation of the Mechanisms of the FRD of the Auditory EP using a modified version of the 'clock' paradigm of Experiment 13

INTRODUCTION AND METHOD

Experiment 14 constituted a replication of Experiment 13 using tone-bursts instead of flashes, but with the following important modification. It was attempted to attentionally equate all three constituent stimulus events by locating event 1 (in Experiment 13, the irrelevant prior stimulus) with the same temporal precision as events 2P (the 'second' stimulus) and 1S (the attentionally equivalent 'first' stimulus), and requiring the subject to produce a reaction time to it. The rationale behind this modification was that the abolition of the FRD between two successive stimuli (i.e. 1 and 2P) would be a more convincing demonstration of the validity of the current theory than its absence between 2P and 1S.

Five subjects participated in the experiment, their EEG being recorded monopolarly from a vertex placement. An oscilloscope clock was again employed, this time incorporating two moving spots and a single stationary spot. Trials consisted of the slow progression of the mobile spots across the screen towards and passing through the stationary spot. As each mobile spot coalesced with the stationary one, a tone-burst (approx. 88 dB, 1000 Hz, 50 ms duration) could occur with a probability of .5. Subjects were required to key-press as rapidly as possible to such an occurrence and to avoid making false positives. In order to maintain the subject's performance at a constant high level, slow responses (longer than 200 ms) and errors were indicated on the oscilloscope display at the end of each trial. At the beginning of the session, which comprised 3 blocks of 64 trials (the first block being for practice), the subject was allocated 256 points and one point was deducted from this total whenever a slow response or an error was registered.

The two mobile spots were separated by a constant distance that corresponded to an interval of 3 seconds between their arrival at the stationary spot. The main rationale behind this choice of length of interval was a practical one, namely to achieve a reasonable spatial separation between the two mobile spots. The question of whether an auditory FRD would normally be expected with this ISI is answered in the affirmative by reference to the recovery function data of Fig. 1.3. Indeed, in the studies of the short-term habituation of the EP reviewed in Chapter 1 an ISI of 3 seconds is frequently employed, and to satisfactory effect. Following the terminology of Experiment 13, a

bleep associated with the arrival of the first of the spots was designated stimulus event 1. Depending on the prior occurrence of an event 1 on the trial, a bleep coinciding with the arrival of the second spot was classified as either stimulus event 2P or 1S. The initial stages of the spot-pair's traverse prior to the arrival of the first spot occupied 8 seconds, permitting virtually full recovery of the EP (see Gjerdingen and Tomsic's (1970) recovery function data presented in Fig. 1.3 of Chapter One. It is perhaps more relevant to note that Bess and Ruhm (1972) whose work, like the present, was based on pairs of stimuli indicate an interval of 6 seconds to be satisfactory in this respect), and the spot-pair continued to move for a further 2 seconds after the arrival of the second spot. The 'strip cartoon' of Fig. 6.1 illustrates the major events in a trial.

RESULTS

Digitally filtered average EPs, and also median EPs (MEP; Borda and Frost, 1968) were computed for each stimulus event. The results for one typical subject are shown in Fig. 6.2. It can be seen that the morphology of the MEPs and AEPs are essentially the same, indicating the bioelectric data to be substantially free from trials heavily contaminated with artifact.

The amplitude and latency of N1 and P2 was extracted from the AEPs and subjected to statistical analysis; the various analysis of variance summary tables and treatment means being compiled in Table 6.1. Comparison 1 (Table 6.1iii) indicates that the amplitude of N1-P2 is larger for events 1 and 1S than for 2P ($F(1,8) = 7.44^{*}$); but no amplitude difference between events 1 and 1S is found (Comp. 2, $F \approx 1$). Althoug

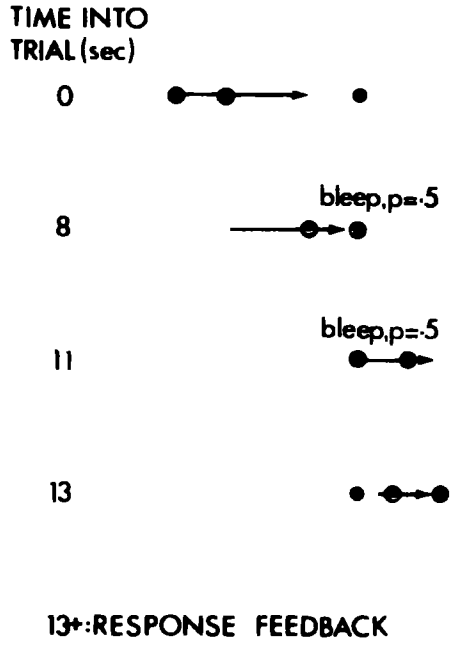

Figure 6.1

Illustration of the 'clock'paradigm of experiment 14. See text for details.

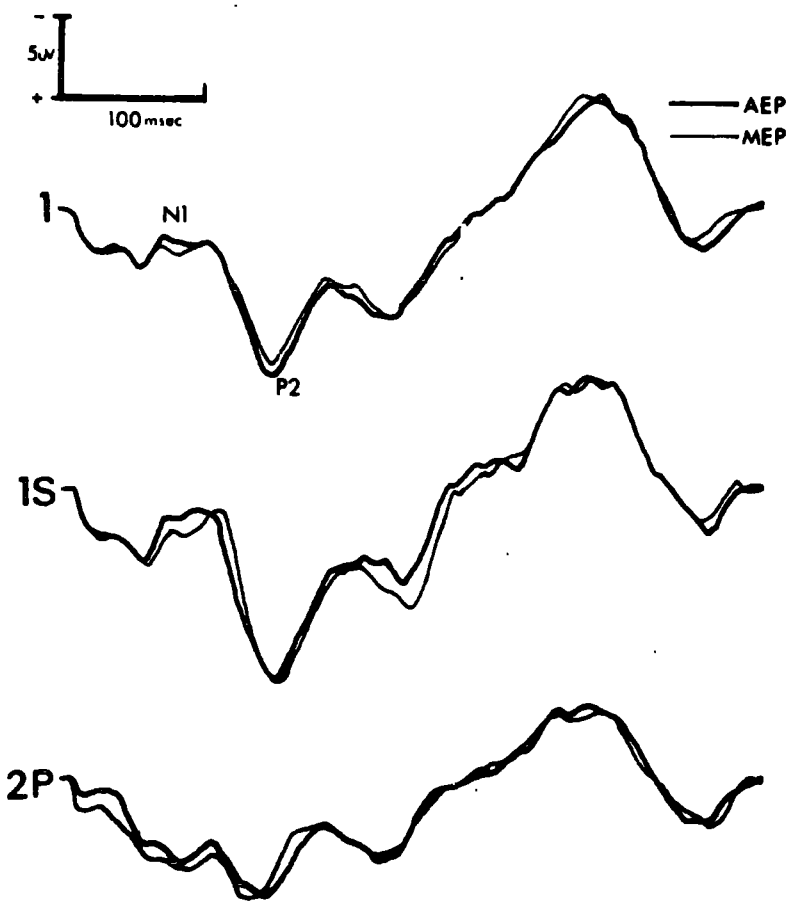

Figure 6.2

Digitally filtered average EPs and median EPs for subject DW associated with stimulus events 1, 1S and 2P in experiment 14.

TABLE 6.1: EXPERIMENT 14

i) Mean EP amplitude (μV) and latency (ms) as a function of component (N1,P2) and stimulus type (1,1S,2P - see text for details). Performance data also shown.

Amplitude:

	N1	P2	
1	2.00	17.39	9.70
1S	3.73	17.58	10.66
2P	.40	15.62	8.01
	2.04	16.86	9.45

Latency and median reaction time:

	N1	P2	RT (ms)
1	94	171	145
2S	92	182	145
2P	93	168	144
	93	174	144

ii) Summary of the analysis of the effect of component (C) and stimulus type (T) upon EP amplitude, including single df comparisons. For key to significance levels see Section 1 of Chapter 2.

Source	df	SS	MS	F
Subjects	4	376.09		
C	1,4	1,647.54	1,647.54	149.32 ***
T	2,8	35.94	17.97	4.27 *
T x C	2,8	3.53	1.77	1.18 ns

Orthogonal comparisons

Source	Comparison	MS	F
T	comp 1: $\frac{1}{2}$ $\frac{1}{2}$ -1	31.33	7.44 *
	comp 2: 1 -1 0	4.61	1.09 ns

iii) Summary of the analysis of the effect of stimulus type (T) upon N1 latency.

Source	df	SS	MS	F
Subjects (S)	4	2,506.67		
T	2	10.00	5.00	<1 ns
T x S	8	273.33	34.17	
Total	14	2,790.00		

iv) Summary of the analysis of the effect of stimulus type (T) upon P2 latency.

Source	df	SS	MS	F
Subjects (S)	4	18,456.67		
T	2	543.33	271.67	1.23 ns
T x S	8	1,773.33	221.67	
Total	14	20,773.33		

v) Summary of the analysis of the effect of stimulus type (T) upon median RT.

Source	df	SS	MS	F
Subjects (S)	4	2,519.60		
T	2	4.80	2.40	<1 ns
T x S	8	125.20	15.65	
Total	14	2,649.60		

N1 is reliably smaller than P2 ($F(1,4) = 149.3^{***}$), the stimulus type x components interaction fails to achieve significance ($F(2,8) = 1.18$ ns). No effects of stimulus type upon either N1 (Table 6.1iii, $F<1$) or P2 latency (Table 6.1iv, $F(1,8) = 1.23$ns) are noted.

The performance data were also analysed, with the median RT of 144 ms remaining constant for the three stimulus events (Table 6.1v, $F<1$). On average the false positive rate ran at 1%; and 2.5% of responses were slow. Analysis of the MEPs yielded the same overall picture as the AEPs.

DISCUSSION

The hypothesis that the FRD of the auditory EP is produced by the same mechanisms as the visual FRD is not supported by the data of this study. Despite the behavioural and informational equivalence of 1, 2P and 1S (which is reflected in the RT data), the N1-P2 wave of the EP evoked by event 2P was depressed relative to the corresponding evoked activity associated with both 1 and 1S. However, before abandoning an attentional explanation of the auditory EP, it should be noted that the magnitude of the response decrement in this study is significantly less than has been found elsewhere. The amplitude of N1-P2 evoked by the second stimulus of a pair remains at 83% of the amplitude of the first stimulus which, for instance, compares with figures of 69% and 54% for the same ISI which can be derived from the recovery function data of Gjerdingen and Tomsic (1970) and Davis et al (1966) respectively, and with the figure of 37% which represents the EP amplitude depression to the second stimulus in a train relative to the

first (under comparable conditions, i.e. subject attending, ISI = 3 seconds) in Fruhstorfer's 1970 study of the fast habituation of the auditory EP. It is also significant that the forward-latency-shift that typically occurs to the second stimulus of a pair is not present in this data, and it would appear that the FRD of the auditory EP does contain a component related to temporal uncertainty that the experimental manipulations were successful in eliminating. This point will be returned to in the discussion section of the next experiment.

By equating all three stimulus events, the present procedure was effective in removing the disparity in the degree of overall positivity of the EP which produced the component x stimulus type interaction of Experiment 13. A final point is that the absence of any further EP recovery between stimulus events 1 and 1S indicates that full recovery had indeed developed by the time of occurrence of event 1.

Experiment 15: A Replication of Experiment 13 in the Auditory Modality

INTRODUCTION AND METHOD

Before finally rejecting the notion that the FRD of the auditory and visual EP were produced by similar mechanisms, a closer replication of Experiment 13, i.e. without the modification to event 1 that was incorporated in Experiment 14, was conducted. Eight subjects were recruited, and their EEG was recorded from a vertex electrode. The procedure was essentially identical to that employed in the earlier study (i.e. Experiment 13) except that 1000 Hz tone-bursts (88 dB, 50 ms duration) were used instead of flashes. Briefly, trials consisted of a spot traversing an oscilloscope screen towards a fixed point (9 secs)

and continuing on for a further 2 seconds. Bleeps occurred with a probability of .5 with the mobile spot at each of two positions, either at the fixed point (the imperative bleep) or at a random interval of between 2 and 4 seconds (i.e. the same mean interval as Experiment 14) before this point (the irrelevant bleep). Three stimulus events were thus generated corresponding to an irrelevant bleep (1), an imperative bleep preceded by an irrelevant bleep (2P) and an imperative bleep on its own (1S); the comparison of interest being between the AEPs evoked by the 'first' stimulus of event 1S and the attentionally equivalent 'second' stimulus of 2P. Unlike Experiment 13, feedback (indicating false positives and responses slower than 200 ms) was provided via the oscilloscope display at the end of each trial. The experimental session consisted of three blocks of 64 trials, the first block being for practice. At the outset subjects were allocated 128 points and informed that they should respond accurately and rapidly as a point would be deducted for every slow or erroneous response. On average subjects produced 3.38 slow responses (5.27%) and .25 errors (1.39%) per session.

RESULTS

The amplitude and latency of N1 and P2 was extracted from the smoothed AEPs associated with the three types of stimulus event; the group means and their statistical analysis being assempled in Table 6.2. As in Experiment 13, an overall positive shift in the activity evoked by both 2P and 1S produced a stimulus type x component amplitude interaction and for simplicity separate single factor repeated measures analyses of variance were performed upon the amplitude data.

TABLE 6.2: EXPERIMENT 15

i) Mean EP (N1,P2) amplitude (μV) and latency (ms) as a function of stimulus type (1,1S,2P - see text for details).

	1	1S	2P	
N1 amplitude	12.20	10.53	7.69	10.14
P2 amplitude	13.44	22.88	19.86	18.73
N1 latency	89	88	87	88
P2 latency	159	153	153	155

ii) Summary of the analysis of the effect of stimulus type (T) upon N1 amplitude, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	7	744.70		
T	2	83.19	41.59	3.84 *
T x S	14	151.78	10.84	
Total	23	979.67		

Single df comparisons

Source	Comparison	MS	F	F residual
T	comp 1: $\frac{1}{2}$ $\frac{1}{2}$ -1	72.03	6.64 *	1.03 (1,14) ns
	comp 2: 0 1 -1	32.26	14.03 (MS_{error} = $MS_{S \times comp\ 2}$ = 2.30)	

iii) Summary of the analysis of the effect of stimulus type (T) upon P2 amplitude, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	7	601.58		
T	2	371.87	185.93	12.80 ***
T x S	14	203.37	14.53	
Total	23	1176.82		

Orthogonal comparisons.

Source	Comparison	MS	F
T	comp 3: -1 $\frac{1}{2}$ $\frac{1}{2}$	335.39	23.09 ***
	comp 4: 0 1 -1	36.48	2.57 ns

iv) Summary of the analysis of the effect of stimulus type (T) upon N1 latency.

Source	df	SS	MS	F
Subjects (S)	7	1,007.29		
T	2	25.00	12.50	<1 ns
T x S	14	358.33	25.60	
Total	23	1,390.62		

v) Summary of the analysis of the effect of stimulus type (T) upon P2 latency, including single df comparisons.

Source	df	SS	MS	F
Subjects (S)	7	3,257.29		
T	2	208.33	104.17	1.61 ns
T x S	14	908.33	64.88	
Total	23	4,393.95		

Single df comparison

Source	Comparison	MS	F	F residual
T	comp 5: 1 $-\frac{1}{2}$ $-\frac{1}{2}$	208.33	3.21	<1 (1,14) ns

vi) Summary of the analysis of the effects of component (C: N1,P2) and stimulus type (T:2S,2P) upon EP amplitude.

Source	df	SS	MS	F
Subjects	7	750.63		
C	1,7	1,202.46	1,202.46	21.73 **
T	1,7	68.68	68.68	11.39 **
T x C	1,7	0.06	0.06	<1 ns

Inspection of Table 6.2ii reveals that events 1 and 1S evoke a larger N1 than event 2P (Comp. 1; $F(1,14) = 6.64^{*}$). Although the residual variation was not significant, a further non-orthogonal comparison (Comp. 2) was made specifically to evaluate the N1 amplitude difference between 2P and 1S. This comparison was found to be significant at the 1% level with $F = 14.03$ and $df = 1,7$. An F ratio of < 1 indicated that the tendency of N1 (Table 6.2iv) to occur later to event 1 was not statistically significant. Comparison 3 (Table 6.2iii, $F(1,14) = 23.09^{***}$) indicates P2 to be deeper for both 2P and 1S, and although not significant there are tendencies a) for 1S to evoke more positivity than 2P (Comp. 4; $F(1,14) = 2.57$, $p < .25$) and b) for the P2 evoked by stimulus event 1 to be delayed (Table 6.2v, Comp. 5; $F(1,14) = 3.21$, $p < .1$).

A further two factor analysis of variance, also presented in Table 6.2 (part vi), with components and a stimulus type factor restricted to 2P and 1S was conducted in order to simplify the main findings of the experiment in respect of EP amplitude. This analysis showed N1-P2 to be significantly larger for stimulus event 1S than 2P ($F(1,7) = 11.39^{**}$). Although N1 was reliably smaller than P2 ($F(1,7) = 21.37^{**}$), the components x stimulus type interaction did not approach significance ($F < 1$). Analysis of the performance data demonstrated that the group mean median RTs of 159.5 and 157.25 ms to events 1S and 2P respectively were not significantly different ($t(df = 7) = .775$ ns).

DISCUSSION

Despite the apparent success of the experimental manipulations, as evidenced in the RT data, in equating stimulus events 2P and 1S, the brain activity evoked by 2P was nonetheless depressed relative to 1S. This result corroborates the findings of the previous experiment, and it is concluded that an attentional explanation of the FRD of the auditory EP in terms of temporal uncertainty alone is inadequate. It is still considered though, although without satisfactory proof as yet, that temporal uncertainty is a component, but, unlike the visual modality, other factors beyond the control of the present procedure are clearly operating. Indeed the different shape of the auditory recovery function (see Fig.1.3) in itself suggests that the FRD of the visual and auditory EPs do not share identical mechanisms. The author was reluctant to resort to the notion of auditory refractoriness for such an additional component and a second psychological factor, complementing temporal uncertainty, was hypothesised. This factor is introduced in the next experiment and is explored in the remaining two experiments of this chapter.

By way of closing this section it is noted that the AEP differences between the irrelevant stimulus and the imperative stimuli are similar to those observed in Experiment 13, with the late components evoked by stimulus 1 tending to be less positive and to be delayed. These effects were discussed in the earlier experiment and rather than duplicate this discussion the reader is referred back to the appropriate paragraph of the discussion section of Experiment 13.

Experiment 16: The Role of 'Firstness' in the Fast Response Decrement of the Auditory EP

INTRODUCTION

In their investigation of the primacy effect in serial position recall, Hockey and Hamilton (1977, p.50) conclude that the effect originates from the "stronger 'perceptual impact' for the first one or two items in a sequence." Although Hockey and Hamilton proceed to relate this perceptual impact to preparation, the notion conveys to the present author the suggestion that items early in a train of stimuli, and the first stimulus in particular, possess an intrinsic psychological quality, difficult to define but being something to do with their earliness *per se*. This quality is designated 'firstness', and is the psychological factor anticipated in the discussion section of the previous experiment. No precise definition of this concept is offered, save to indicate that it embraces any significance that the first one or two stimuli in a sequence might have for the organism above their objective informational content.

Thus the FRD of the auditory EP is considered to be a function of the decrease in attention associated with both declining 'firstness' and temporal uncertainty over a train (fast habituation) or pair (temporal recovery) of stimuli. The AEP associated with stimulus event 2P is depressed relative to the AEP evoked by event 1S in Experiment 15, and events 1 and 1S in Experiment 14 because, although equivalent in terms of objective information, the 'firstness' of 2P is attenuated by its being the second stimulus of a pair. Although the introduction of an imprecise concept in order to expediently explain away unwanted findings

is not the most satisfactory of manoeuvres, fortunately the above theorising generates an eminently testable hypothesis: namely, that the FRD of the auditory EP will be abolished if informationally equivalent stimuli of equal 'firstness' are used. Experiments 16 and 17 examine this hypothesis.

METHOD

In order to equate the 'firstness' of stimuli, the discrete trials of the earlier experiments (they were delimited by the beginning and end of the spot's traverse) were replaced by a single long sequence of stimuli. Thus, after the first few stimuli, 'firstness' would cease to operate and comparisons could be safely made, free from the influence of this variable. The sequence was 'clocked' to eliminate temporal uncertainty and the experimental procedure is described in the following paragraph and illustrated in Fig. 6.3a.

Subjects were instructed to fixate a stationary spot on the oscilloscope screen. A second spot was continuously in motion, wheeling at a constant angular velocity around a circular pathway that brought it, once a revolution, close to the stationary spot. At this point a bleep (88 dB, 1000 Hz, 50 ms duration) occurred with a probability = .5, to which the subject was directed to respond. Rapid, but accurate, performance was encouraged. A complete orbit of the moving spot required 3 seconds, and thus the experiment generated a sequence of stimuli of equal temporal and event uncertainty, separated in time by varying multiples of 3 seconds. 180 revolutions of the 'clock' constituted the sequence and, allowing the first 30 revolutions for practice and the dissipation of any

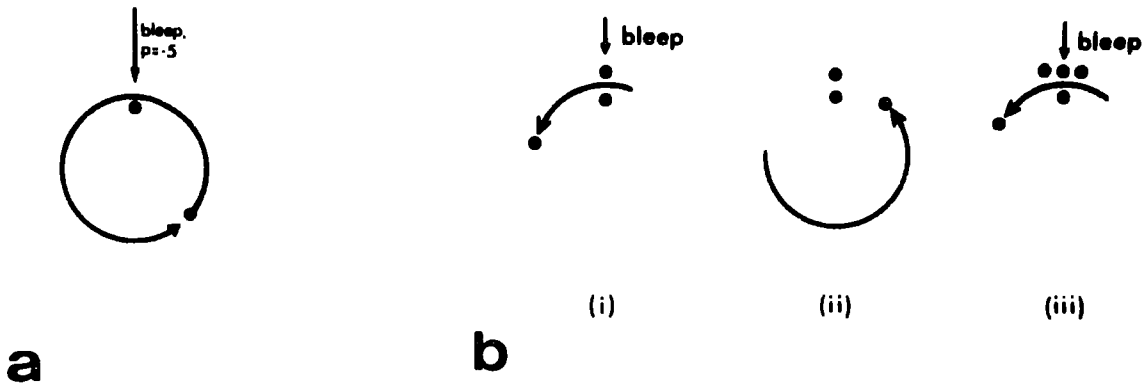

Figure 6.3

a) Illustration of the 'clock' paradigm of experiment 16. See text for details.

b) The modified version of the 'clock' paradigm of experiment 16 employed in experiment 17 is illustrated with reference to a 'typical' sequence of events. In i) the moving spot has just passed above the stationary spot and a bleep has occurred. The appearance of the single spot above the stationary one coincided with the bleep and indicates that the next bleep will occur at the end of this current cycle, i.e. in 3 seconds time. Approximately 2 seconds or so later the moving spot has circled round to the position shown in ii) and shortly afterwards, iii), it passes above the stationary spot and the expected bleep occurs. 3 spots appear this time indicating that 3 cycles (i.e. 9 seconds) will elapse before the next bleep.

'firstness', data was collected for the last 150. Thus the procedure generated, on average, 37.5 stimuli associated with a 3 second ISI (event A) and 37.5 stimuli associated with a 6 second ISI or longer (event B). Recalling the definition of the FRD of the EP as an amplitude depression associated with recent prior stimulation, i.e. with ISIs of the order of a few seconds, then under this definition, the amplitude of the AEP associated with event A (Experiments 14 and 15 both show an amplitude decrement at this ISI) would normally be expected to exhibit a depression in relation to the AEP amplitude associated with event B (50% of which includes ISIs of 9 seconds or longer where from Fig. 1.3 little or no decrement would be expected, and the remainder of which represents an ISI of 6 seconds, where again relatively little decrement is found; indeed, Bess and Ruhm (1972) report no decrement at this ISI.). Translating the 'first' stimulus/'second' stimulus dichotomy that is held to encapsulate the essence of the FRD into ISI terms, a 'first' stimulus is reduced to a stimulus occurring after a long 'full recovery' ISI and a 'second' stimulus to a stimulus associated with more recent prior stimulation, i.e. a short ISI. In these terms, events A and B correspond to a 'second' stimulus and a 'first' stimulus respectively and the experimental hypothesis was therefore that, with 'firstness' and temporal uncertainty under experimental control, the AEPs for the 'second' stimulus of event A and the 'first' stimulus of B would not differ, i.e. that no EP amplitude depression would be exhibited at the shorter ISI. Thus, with no evidence for a fast response decrement with both 'firstness' and information content under

control, the results of the experiment would be advanced as supporting the current attentional mechanism for the FRD of the auditory EP.

EEG was recorded from the vertex of the eight subjects who participated in the experiment. The oscilloscope was positioned 5 feet from the subject and the diameter of the spots orbit (= .4 inch) thus subtended an angle of less that .5 of a degree at the eye. The whole of the display could thus be comfortably absorbed from the designated fixation point. At the end of the experiment the subject was informed of his median RT and the number of false positives he had recorded. On average 1.38 false positives were produced per subject.

Finally, the correspondence between this procedure and that employed by Roth, Krainz, Ford, Tinklenberg, Rothbart and Koppell (1976) in their investigation of the recovery function of the EP is noted. Roth et al also used long runs of stimuli with their various ISIs occurring according to a random sequence, commenting (p.623) that "the customary method of presenting stimuli in blocks of the same ISI is particularly liable to influence ... by fluctuations in arousal or attention." By varying the ISI randomly within a single run they hoped that such influences would fall more equally on each ISI. The present procedure thus coincides with established recovery function procedures and indeed apparently possesses a number of desirable features in this respect.

RESULTS

Digitally filtered AEPs were computed for each subject, representing the brain activity evoked by events A and B. The

amplitude and latency of N1 and P2 were measured for each AEP and subjected to statistical analysis: the various treatment means, analysis of variance summary tables and values of Student's t being assembled in Table 6.3.

Considering the amplitude data first, the analysis of variance indicates both main effects to be significant, with P2 larger than N1 ($F(1,7) = 43.83^{***}$) and the N1-P2 evoked by event B larger than its counterpart evoked by event A ($F(1,7) = 10.90^{**}$). The ISI x component interaction was not significant ($F(1,7) = 1.41$ ns), indicating that the tendence of N1 to be more sensitive to the effect of ISI is not reliable.

Although Student's t-tests revealed no significant latency effects for P2 ($t(df = 7) = 1.0$ ns), N1 occurred significantly earlier at the shorter ISI ($t(df = 7) = 1.99^{*}$). Analysis of the performance data reveals no significant difference in the median RT to events A and B (188,186 ms; $t(df = 7) = .32$ ns).

DISCUSSION

The experimental hypothesis that the amplitude of N1-P2 would not be related to ISI when the significance for the organism of the evoking stimuli was held constant does not appear to be supported in the results of this study. The term 'stimulus significance' is introduced here to denote the total import of the stimulus for the organism and includes its informational content and those less easily defined qualities, such as 'firstness'. The attention paid to a stimulus, i.e. the allocation of capacity to it and hence the amplitude of the EP,

TABLE 6.3: EXPERIMENT 16

i) Mean EP (N1,P2) amplitude (μV) and latency (ms) and reaction time (ms) as a function of ISI (A = 3 sec, B = >3 secs - see text for further details). The results of Student's t-tests evaluating the latency and RT effects are also shown (one tailed test).

Amplitude:

	N1	P2	
A	3.02	16.64	9.83
B	6.59	18.27	12.43
	4.81	17.46	11.13

Latency and median RT:

	N1	P2	RT
A	82	158	188
B	86	156	186
t,df=7	1.99*	1.0 ns	.32 ns

ii) Summary of the analysis of the effects of component (C) and ISI (I) upon EP amplitude.

Source	df	SS	MS	F
Subjects	7	415.12		
C	1,7	1,280.18	1,280.18	43.83 ***
I	1,7	54.08	54.08	10.90 **
I x C	1,7	7.53	7.53	1.41 ns

is held to be determined by this quantity alone. With this quantity remaining constant over stimuli, it is predicted that no EP amplitude effects should be observed, despite variations in the ISI. However typical FRD effects are noted, with the amplitude of N1-P2 being attenuated at the short ISI of event A relative to the long ISI of event B. Although the current preoccupation of the thesis is with EP amplitude effects, it is noted that the forward-latency-shift in N1 at the shorter ISI is also consistent with the presence of an FRD (see Experiment 10 and also Callaway, 1973; p.162, Fig.1).

At first sight the results of this study appear to represent a third consecutive demonstration of the inadequacy of an attentional explanation of the auditory FRD. However, the design of the experiment includes a serious flaw which derives from a confusion of subjective and objective probability. Although the objective probability of an imperative bleep remains constant throughout at .5, subjective probability necessarily waxes and wanes according to the recent experience of the subject. Thus a number of cycles without a stimulus will bring the subject to a pitch of anticipation in contrast to the incredulity which presumably developes over a run of consecutive imperative events. Such systematic variations in the subject's state will certainly affect the significance of stimuli and therefore the amplitude of their EPs.

Considering the three cycles leading up to the occurrence of an imperative stimulus, 8 antecedent events are distinguishable. These events are as follows, with 0 denoting the absence and 1 the presence

of an imperative stimulus on a particular cycle: 000, 100, 010, 110, 001, 101, 011, 111. Thus an imperative stimulus can be classified into one of 8 classes according to the recent experience of the subject. With, on average, 9 or so of each type of class occurring per sequence an acceptable AEP could be and was computed for each class.

Considering the variation in subjective probability induced by the 8 types of prior event, all the middle-order events (i.e. 100...011) are unexceptional occurrences and such variations as will exist are difficult to anticipate and are likely to be unimportant. Only the extreme eventualities of 3 or more consecutive stimuli (event 111) or stimulus omissions (000) allow a clear-cut prediction to be formulated, namely that the subjective probability of an imperative stimulus on the current cycle following 111 will be less than .5, and following 000, greater than .5. The following 1 df comparison (X) was accordingly elaborated to evaluate the presence of an N1-P2 amplitude trend related to subjective probability.

Event	000	100...011	111	
Coefficient	1	All 0	-1	X

On the other hand the recovery function of the EP indicates that full recovery is virtually complete within 6 seconds, and thus a temporal recovery prediction of the variation of N1-P2 across the 8 'histories of prior stimulation' is best expressed in the following 1 of comparison (Y):

Event	000,100,010,110	001,101,001,111	
Coefficient	All 1	All -1	.....Y

which amounts to the original event A versus event B comparison. The flaw in the design of the experiment is that the effect of subjective

probability also predicts an AEP difference between A and B, i.e. comparisons X and Y are confounded in the main effect of treatments (ISI) in Table 6.3. However, with the effect of treatments expressed in terms of the 8 types of prior event 000 to 111, a decision between a subjective probability and a temporal recovery explanation can be made. Quite simply, the comparison that accounts for the most of the variability due to treatments can be regarded as indicating which of the two explanations is the more appropriate.

The amplitude of N1 and P2 was extracted from the AEPs associated with each of the eight types of antecedent event, and subjected to a 2 factor repeated measures analysis of variance; the summary table of which is shown with the various treatment means in Table 6.4. A significant effect of prior event upon N1-P2 amplitude is noted ($F(7,49) = 3.59^{**}$) and although comparisons X and Y are both significant ($F(1,49) = 19.48^{***}$, 16.00^{***} respectively) the subjective probability explanation accounts for more of the variability of this main effect that does the temporal recovery explanation ($r^2 = .78, .64$ respectively). The analysis also shows N1 to be significantly smaller than P2 ($F(1,7) = 41.76^{***}$); however the T x C interaction does not approach significance with an F ratio of little over 1.0.

Summarising, although the results of Experiment 16 initially appeared to repudiate an attentional explanation of the FRD of the auditory EP, the reanalysis of the last few paragraphs indicates that the amplitude increase of N1-P2 with ISI is better explained in terms of subjective probability than EP recovery. It is considered that the extreme subjective

TABLE 6.4: EXPERIMENT 16, RE-ANALYSIS

i) Mean EP (N1,P2) amplitude (μV) as a function of prior event type (8 types, 000 ..... 111 - see text for details). The coefficients associated with comparisons X and Y (see text) are also shown.

Prior event	000	100	010	110	001	101	011	111
EP ampl.	15.19	12.58	12.64	12.69	10.76	11.02	11.51	9.70
comp X	1	0	0	0	0	0	0	-1
comp Y	1	1	1	1	-1	-1	-1	-1

ii) Summary of the analysis of the effects of component (C; N1,P2) and prior event type (T) upon EP amplitude.

Source	df	SS	MS	F
Subjects	7	1,578.73		
C	1,7	5,171.45	5,171.45	41.77 ***
T	7,49	311.42	44.49	3.59 **
T x C	7,49	93.57	13.37	1.12 ns

iii) Single df comparisons

Source	Comparison	MS	F	r^2	F residual
T	X	241.56	19.48 ***	.78	<1 (6,49) ns
	Y	198.40	16.00 ***	.64	1.52 (6,49) ns

reactions of 'it must be this time' or 'there can't be another one' invest the occurrence of a stimulus after three or more consecutive omissions and occurrences with an enhanced or attenuated degree of significance respectively, inducing corresponding variations in N1-P2 amplitude. It is thus tentatively concluded that, had subjective probability been held constant, no effect of ISI upon N1-P2 amplitude would have been obtained. This conclusion is examined in the final experiment of this chapter.

Although the effect of expectancy upon the waveform of the EP has been the subject of numerous studies (e.g. Sutton, Braren, Zubin and John, 1965; Tueting, Sutton and Zubin, 1971; Tueting and Sutton, 1973; Squires, Wickens, Squires and Donchin, 1976; Duncan-Johnson and Donchin, 1977), experimental interest has been largely confined to the P3 component, with N1-P2 apparently showing little lability in this respect. The contribution of both a priori probabilities and sequential effects to 'expectancy' and the amplitude of P3 was evaluated in the studies of Squires et al (1976) and Duncan-Johnson and Donchin (1977) and it is pertinent to note that the amplitude of P3 covaries with stimulus sequence in the same manner as the N1-P2 wave of this study. Although neither of the above studies looked at the effect of stimulus sequence upon pre-P3 components, inspection of the AEPs presented in Fig.2 of Squires et al reveals a decline in N1 amplitude with stimulus repetition. It is further significant that P3s were not observed in the waveforms of the present study suggesting that the P2 component here may include the later positivity of P3 shifted forward in time, possibly reflecting the facilitation of processing by the 'clock'. Thus the effects of subjective

probability upon N1-P2 amplitude of this study are not at variance with the established literature.

Experiment 17: An Attentional Explanation of the Fast Response Decrement of the Auditory EP Finally Confirmed

INTRODUCTION AND METHOD

The experimental procedure of Experiment 17 was identical to that of Experiment 16, save in the following two respects.

a) Variations in subjective probability were eliminated by providing the subject with complete information about the number of clock cycles between stimuli by means of a row of spots that appeared above the stationary spot after the occurrence of a stimulus. Either 1 or 3 such spots appeared, representing ISIs of 3 and 9 seconds respectively. The subject was informed of the fact that the imperative stimulus actually occurred at a random interval (0 to 150 ms) on either side of the stationary spot on the designated cycle, thus ensuring that responses would remain contingent upon the bleep rather than becoming simply time-locked to the arrival of the moving spot at the stationary one. On average only 1 anticipatory response was recorded per subject, indicating this manipulation to be successful. This modified version of the procedure of Experiment 16 is illustrated in Fig. 6.3b.

b) Subjects were presented with a second train of imperative bleeps, but without the benefit of the oscilloscope clock. Again a random sequence of 3 and 9 second ISIs was used. The order with which subjects received this unclocked control condition (UC) and the above clocked condition (C) was counterbalanced across subjects.

100 clock cycles constituted the sequences; data being collected from the last 80, the initial 20 being for practice and the dissipation of 'firstness'. Six subjects were run. It was considered that the procedure of Experiment 17 (condition C) represented the perfection of the 'clock' paradigm; and that with the temporal uncertainty associated with the 3 second and the 9 second ISI equal, and with such complicating factors as 'firstness' and subjective probability under control, no EP amplitude decrement at the short ISI would be obtained. It was further anticipated that the control condition would reveal the usual FRD effect, and finally that due to the greater temporal uncertainty of the stimuli in the unclocked sequence, it was hypothesised that the late components of their EPs would be both larger and later than those observed in the clocked condition.

RESULTS

Digitally filtered AEPs were computed for each subject for the 3 and 9 second ISI under both experimental conditions (C and UC); two subjects' data are presented for illustrative purposes in Fig.6.4. The amplitude and latency of N1 and P2 was extracted from these averages and subjected to statistical analysis. The various tables of means and analysis of variance summary tables are compiled in Table 6.5.

Considering the amplitude data first, a three factor repeated measures analysis of variance was conducted with components (C), clocked vs unclocked (E) and ISI (I) as the factors. All three main effects were significant with P2 larger than N1 ($F(1,5) = 9.40^{*}$), and N1-P2 larger in the unclocked condition ($F(1,5) = 18.5^{**}$) and for the

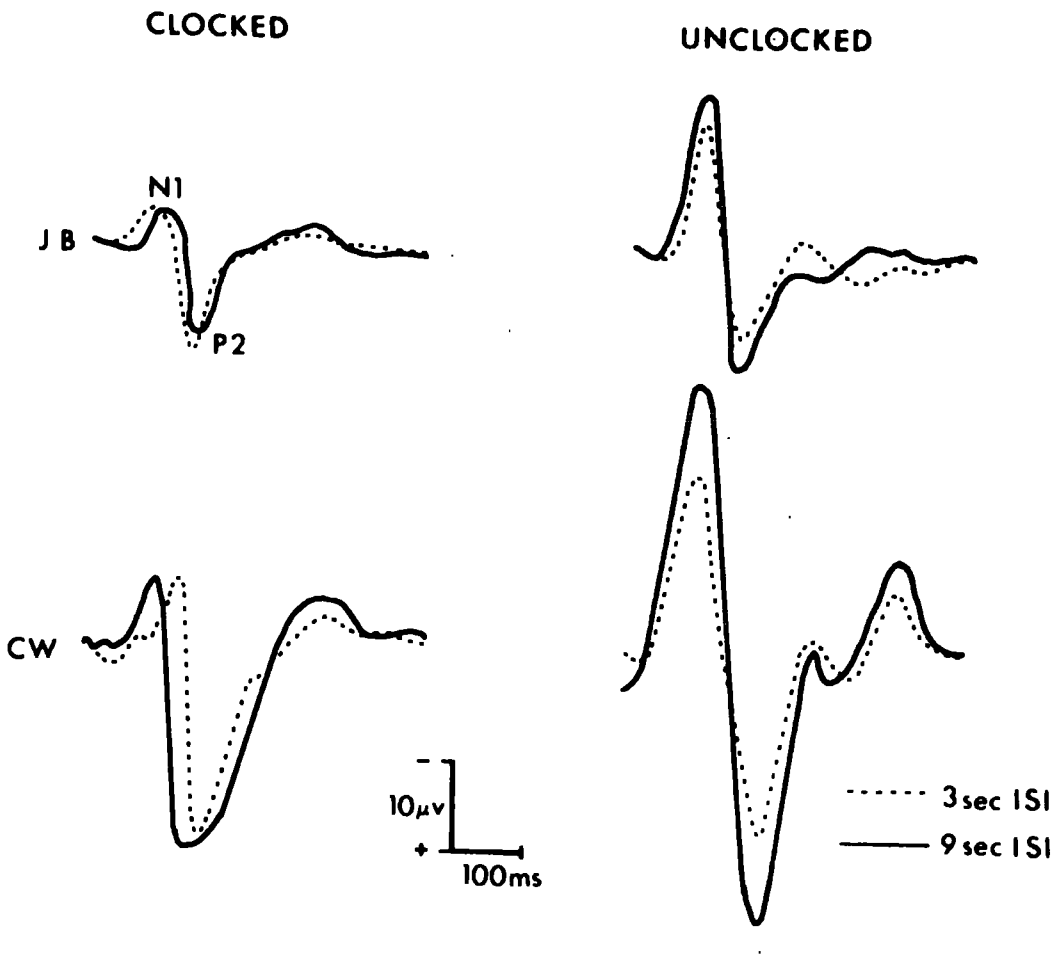

Figure 6.4

Experiment 17: Digitally filtered AEPs for subjects JP and CW for the 3 and 9 second ISIs under 'clocked' and 'unclocked' conditions.

TABLE 6.5: EXPERIMENT 17

i) Mean EP (N1,P2) amplitude (μV) and latency (ms) and median reaction time (ms) as a function of ISI (I; 3,9 secs) and experimental procedure (E; C = clocked, U = unclocked).

Amplitude:

		E		
		C	U	
ISI	3	10.02	15.47	12.75
	9	10.41	20.00	15.21
		10.22	17.74	13.98

		N1	P2
E	C	4.09	16.33
	U	17.43	18.05
		10.76	17.19

N1 latency:

		ISI		
		3	9	
E	C	78	80	79
	U	95	98	97
		86	89	88

P2 latency:

		ISI		
		3	9	
E	C	146	143	144
	U	176	175	175
		161	159	160

Median RT:

		ISI		
		3	9	
E	C	179	181	180
	U	240	235	237
		209	208	209

ii) Summary of the analysis of the effects of component (C; N1,P2), ISI (I) and experimental procedure (E) upon EP amplitude.

Source	df	SS	MS	F
Subjects	5	1,015.23		
C	1,5	496.14	496.14	9.40 *
I	1,5	72.81	72.81	8.09 *
I x C	1,5	.79	.79	<1 ns
E	1,5	679.21	679.21	18.50 **
E x C	1,5	405.08	405.08	18.66 **
I x E	1,5	51.09	51.09	8.79 *
I x E x C	1,5	4.36	4.36	2.49 ns

Analysis of simple main effects of I x E interaction.

Source	df	SS	MS	F
I at E = C	1,5	.96	.96	<1 ns
I at E = U	1,5	122.94	122.94	21.16 **

iii) Summary of the analysis of the effects of ISI (I) and experimental procedure (E) upon N1 latency.

Source	df	SS	MS	F
Subjects	5	768		
I	1	51	51.00	<1 ns
E	1	1,926	1,926.00	19.22 ***
I x E	1	1	1.00	<1 ns
Error (pooled **)	15	1,503	100.20	
Total	23	4,249		

iv) Summary of the analysis of the effects of ISI (I) and experimental procedure (E) upon P2 latency.

Source	df	SS	MS	F
Subjects	5	2,068		
I	1	26	26.00	<1 ns
E	1	5,859	5,859.00	60.15 ***
I x E	1	10	10.00	<1 ns
Error (pooled **)	15	1,461	97.40	
Total	23	9,424		

v) Summary of the analysis of the effects of ISI (I) and experimental procedure (E) upon reaction time.

Source	df	SS	MS	F
Subjects	5	1,993.33		
I	1,5	16.67	16.67	<1 ns
E	1,5	19,952.67	19,952.67	47.28 ***
I x E	1,5	60.17	60.17	1.24 ns

longer ISI ($F(1,5) = 8.09^{*}$). The significant C x E interaction ($F(1,5) = 18.66^{**}$) indicates the enhancement of EP amplitude in the unclocked condition to the greater for N1 than P2. Analysis of the simple main effects of the significant E x I interaction ($F(1,5) = 8.79^{*}$) reveals that the N1-P2 amplitude increase with ISI only occurs in the unclocked condition ($F(1,5) = 21.16^{**}$) with the F ratio for the clocked condition being less than 1. Neither the C x I interaction nor the three-way interaction between E x I and the components factor were significant ($F<1$, $F(1,5) = 2.49$ ns). This latter interaction is depicted in Fig. 6.5 were its non-significance is seen to indicate that the introduction of the 'clock' was successful in abolishing a response decrement of approx. 4 to 5 μV at the 3 second ISI that was present for both N1 and P2 in the unclocked condition.

Analysis of the latency data (Table 6.5 iii, iv) revealed that both N1 and P2 occurred later in the unclocked condition ($F(1,15) = 19.22^{***}$, 60.15^{***} respectively). However, neither the main effect of ISI, nor the I x E interaction approached significance for both N1 and P2 latency, with F ratios of <1 throughout. Similarly no effect of either I or I x E upon RT was noted (Table 6.5v; $F<1$, $F(1,5) = 1.24$ ns), although in the absence of the 'clock' RT was significantly delayed by 58 ms ($F(1,5) = 47.28^{**}$).

DISCUSSION

An attentional explanation of the fast response decrement of the auditory EP is finally confirmed by the data of this study. With the variable of 'stimulus significance', a term introduced in the previous experiment to denote the total import of the stimulus for the organism

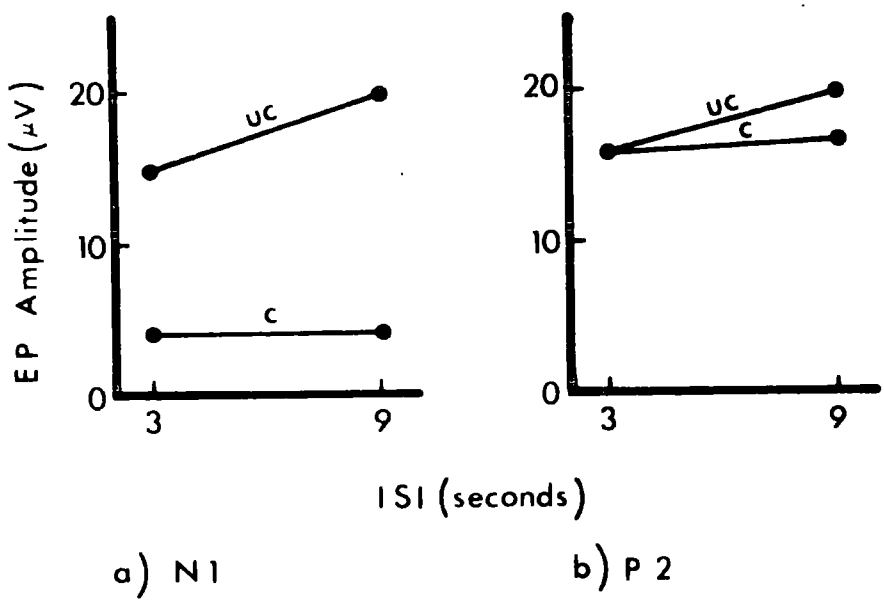

Figure 6.5

Experiment 17, Amplitude data: The three-way interaction between component (N1,P2), ISI (3,9 seconds) and experimental condition (c - clocked, uc - unclocked).

including its information content and certain less easily defined qualities such as 'firstness', brought under adequate experimental control, EP amplitude shows no variation with ISI, with the N1-P2 evoked after a 3 second interval being equal to the N1-P2 evoked after the much longer 9 second 'full recovery' silent time. It thus appears that the FRD of the auditory EP can be completely accounted for in terms of the variations in the allocation of processing capacity (i.e. attention) to stimuli, this allocation being commensurate with the significance of the stimuli for the organism. Unlike the visual FRD, however, stimulus significance is apparently not simply a function of the inadequacy of the neuronal model but a further variable, loosely designated 'firstness', also appears to be relevant. Using a single long sequence of stimuli, however, this latter complication can be eliminated, and the auditory FRD reduced to neuronal model considerations. When the deficiencies of the neuronal model are largely removed by the provision of virtually complete information about the time of occurrence of relevant events, little mental work is necessary when the events occur, and hence the amplitude of N1-P2 is correspondingly small. No ISI effects are obtained under these 'clocked' conditions because the model is no less adequate after a 9 second than a 3 second ISI.

However, in the absence of such certainty (i.e. in the unclocked condition), the occurrence of events conveys considerable information, with relatively large brain potentials reflecting the extensive mobilisation of capacity that is necessary to extract this information

and update the neuronal model[1]. Inevitably the adequacy of the model progressively declines as the ISI lengthens and, reflecting this increasing temporal uncertainty, the amplitude of the EP becomes correspondingly larger. The FRD of the EP, which refers to an amplitude depression associated with short ISIs, is thus produced with N1-P2 amplitude being smaller at the 3 second ISI than at the 9 second one in the unclocked condition.

In this way the present neuropsychological orientation accounts for the major results of this study, and it is concluded that an attentional mechanism for the FRD of the auditory EP is appropriate. Before proceeding, however, the necessity is recognised for some sort of recapitulation of the experimental logic that permits the results of this final study to be adduced as supporting this latter conclusion. Although the relationship of the current paradigm to the temporal recovery subclass of the genus of EP fast response decrements is straightforward, with this paradigm having indeed been employed elsewhere (e.g. Roth et al, 1976) in this respect, its relationship to fast habituation is more obscure. However, when it is remembered that the definition of the FRD of the EP as an amplitude decrement associated with a recent prior stimulus (i.e. with an ISI of a few seconds) covers both fast habituation and temporal recovery, this relationship becomes clearer. No (Ritter et al, 1968) or little (Ohman and Lader, 1972) fast habituation develops, for instance, when ISIs of the order of 10 seconds elapse between stimuli. The present work simply demonstrates that the fast habituation paradigm can be reduced to the relationship

between two types of stimulus event; namely, a 'first' stimulus which occurs after a long ISI (the results of Experiment 10 and the various recovery function data indicate that, with negligible further increments to EP amplitude at intervals longer than the order of 10 seconds, an ISI of this order satisfactorily substitutes the inter-train interval of a minute or so normally encountered in fast habituation studies) and a 'second' stimulus which occurs after a short ISI of a few seconds only. By equating these two stimulus types in terms of temporal uncertainty and, in the case of auditory stimuli 'firstness', this work shows that the amplitude decrement of the 'second' in relation to the 'first' can be abolished. Having identified the mechanism of the amplitude depression of a 'second' stimulus relative to a 'first' in this way, the standard fast habituation paradigm can be reconstructed at least as far as the second stimulus in a train without difficulty. However, as already noted, further decrements beyond the second stimulus in a train are often obtained and recourse to a logical argument must be made at this point. Having identified the decline in stimulus significance ('firstness', temporal uncertainty) as responsible for the amplitude decrement between stimulus numbers 1 and 2, this mechanism can be powerfully applied to any subsequent decrements between stimulus numbers 2 and 3, 3 and 4 and so on. It has already been argued in the introduction to the chapter that, as far as the deficiencies of an internal model of the temporal aspects of stimulation are concerned, it would be unreasonable to rule out the possibility of further improvements in the model (i.e. decrements in temporal

uncertainty) after the second stimulus. Further corresponding decrements in 'firstness' might also reasonably be expected as the beginning of the sequence recedes further and further into the past. Thus both of these components of the mechanism empirically shown in the current experimentation to be responsible for the amplitude decrement in the auditory EP over stimulus positions 1 and 2 in a train of 2, can logically be applied to account for subsequent decrements in a longer train.

To complete this discussion, a number of points are considered concerning the following three issues: a) the E x C interaction, b) the RT and EP latency data and c) 'firstness'. In respect of the first of these issues, inspection of the C x E x I interaction depected in Fig. 6.5 indicates that the significant E x C interaction reflects the relative insensitivity of P2 to the elimination of temporal uncertainty in the clocked condition, although it is nonetheless sensitive to the within-condition increase in temporal uncertainty between the 3 and 9 second ISIs in the unclocked condition. Although at first sight this finding appears to complicate the relationship between P2 and temporal uncertainty, it should be pointed out that the between-conditions difference in temporal uncertainty is confounded with any other general between-conditions differences (task difficulty, alertness, etc.) that might be, and presumably are, present. It is, for instance, possible that the provision of precise information over the time of occurrence of stimuli in the clocked condition encourages a greater allocation of effort (capacity) by the subject to some other

aspect of processing indexed by P2 (such as, possibly, response production) thus obscuring an underlying P2 amplitude decrement.

Both the facilitation of RT and the shorter latency of N1 and P2 in the clocked condition are regarded as consistent with a speedier commencement of processing when the time of occurrence of a stimulus is more or less precisely known. It is surprising though that no similar effects are noted accompanying the reduced temporal uncertainty at the short ISI in the unclocked condition. Although latency shifts in the EP accompanying the amplitude decrement at short ISIs have been previously noted in the thesis research, no ready explanation of the failure of this study to find such effects occurs to the author.

As far as the apparent absence of an effect of 'firstness' in the investigation of the mechanisms of the FRD of the visual EP of Experiment 13 is concerned, my suggestion is that the omnipresent visual stimulation of the oscilloscope 'clock' might be attenuating any perceptual impact that the evoking stimuli would otherwise have. Alternatively, it may be t that the visual stimuli used, or indeed any visual stimuli, simply intrinsically lack the impact of an auditory tone-burst. These suggestions are admittedly tentative and in the absence of a definitive answer this question will be left open. A further related point refers back to the discussion section of Experiment 15 where the mechanism of auditory refractoriness was decided against in favour of 'firstness' as the second component of the auditory FRD. Although this decision was somewhat arbitrary at this earlier stage, it is clearly vindicated in the subsequent research at the level of both experimental rationale and results:

a refractoriness mechanism would neither have inspired the paradigms of Experiments 16 and 17, which were ultimately successful in abolishing the FRD, nor indeed can such a mechanism, which predicts response decrements as the inevitable consequence of the refractoriness induced by a recent prior stimulus, cope with this latter success.

Footnote - Chapter Six

(1) Papakostopoulos, Cooper and Crow (1975) and Papakostopoulos (1977) note that self paced stimulation evokes smaller late potentials than externally paced stimulation. They suggest that the efferent activity preceding sensory stimulation in the self paced condition somehow depresses the cortical reaction to those stimuli. However, the results of this study indicate the reduced temporal uncertainty associated with the self paced condition as a more plausible mechanism.

CHAPTER SEVEN: OVERVIEW AND CONCLUSIONS

In this final chapter the thesis research is summarised, firstly in the concrete with a chapter-by-chapter account of its major substantive findings before proceeding to a fuller discursive recapitulation of the major general conclusions that derive from this work. A number of additional generalisations are also abstracted and discussed. It is recalled from Chapter One that the principal objective of the thesis was the elucidation of the mechanisms underpinning the EP amplitude effects associated with selective attention and repetitive stimulation, and a capacity theory of attention has been applied, with some neurophysiological justification, throughout the thesis in order to account for these effects. A general discussion section discusses this interpretation of EP amplitude and also a number of residual theoretical issues and points of interest. By way of rounding off the thesis some suggestions for further research are offered, including a report of a preliminary investigation deriving from these suggestions.

SUMMARY OF THE RESEARCH

Chapter 2: General Methodology and Data Analysis

Following the theoretical introduction of Chapter One, Chapter Two dealt with general methodology and evaluated a number of data analytic techniques. Experiments 1 and 2 demonstrated that N1-P2 amplitude and latency information can be extracted from single trials using a correlation method. Using such single trial information, correlations between N1-P2 latency and RT (positive) and N1-P2 amplitude and RT (negative) were established. N1-P2 amplitude and latency showed a

weak negative correlation. The use of digital filtering to facilitate the identification of components in the average EP was also recommended, although factor analysis and discriminant analysis were judged to be of limited value in this respect.

Chapter 3: Selective Attention and Evoked Potentials

Part I: No evidence of an N1 correlate of visual selective attention was found, although P2 at the vertex was enhanced to attended stimuli. On the other hand, the vertex N1 appeared to be sensitive to sensory variables, such as stimulus intensity, whereas P2 was immutable in this respect.

Part II: The vertex N1 was enhanced in amplitude and occurred at an earlier latency to attended stimuli in a binaural listening task. However these effects were abolished at a faster presentation rate, even though performance was not markedly impaired.

Chapter 4: The Short-term Habituation of the Visual Evoked Potential

The interaction between the fast habituation of the visual EP and long-term between-block effects was investigated with a single trial analysis. The interaction was not significant. Conventional across-block averaging demonstrated that the within-train exponential N1-P2 amplitude decline and accompanying forward-latency-shift that characterised the fast habituation of the visual EP, developed more rapidly and proceeded to a greater overall magnitude at faster presentation rates. In general, vertex evoked activity and the P2 component of the N1-P2 complex were more labile than occipital activity and the N1 component. A final control study indicated that pupillary mechanisms were not responsible for the fast habituation

of the visual EP and, as a by-product, that a reduction in effective stimulus intensity did not affect the rate of habituation.

Chapter 5: Mechanisms of the Fast Response Decrement of the Visual Evoked Potential

The term 'fast response decrement' (FRD), which subsumes both fast habituation and temporal recovery, was re-introduced from Chapter One. In this present chapter and the next the mechanisms of the FRD of the EP were explored using paradigms based on pairs of stimuli in which the FRD is summarised as the amplitude depression of a 'second' stimulus relative to a 'first'. The experiments of Chapter Five indicated that the visual FRD was not affected by dichoptic presentation, and that it exhibited extensive stimulus generalisation. However, when temporal uncertainty was controlled using the oscilloscope 'clock' of Experiment 13 it was abolished.

Chapter 6: Mechanisms of the Fast Response Decrement of the Auditory Evoked Potential

The FRD of the auditory EP appeared to include a second psychological component, denoted 'firstness'. When 'firstness' was eliminated by presenting stimuli in a single long sequence, controlling temporal uncertainty was effective in abolishing the auditory FRD.

GENERAL CONCLUSIONS

The major general conclusions deriving from the thesis research are compiled as follows.

1) A single trial analysis of EP data represents a practical alternative to the use of signal averaging

The single trial analysis developed in Experiment 1 of Chapter Two was successfully applied in order to extract N1-P2

amplitude and latency from the individual visual EPs in the reaction time study of Experiment 2 and the early habituation studies of Chapter Four, and where trial-by-trial changes in brain activity are of particular interest this technique should prove of considerable value. In general though, this degree of resolution is not necessary and the AEP, like any other statistic, will thus provide a satisfactory and expedient data-base which, in the face of the bedimming complexities of Donchin's recent work (see Duncan-Johnson and Donchin, 1977) is to be recommended for its immediacy, and indeed by this comparison achieves a simple elegance.

2) <u>The N1 correlate of auditory 'stimulus set' reflects the greater mental activity ensuing upon the arrival of an attended stimulus</u>

In his distinction between the 'stimulus set' and 'response set' modes of selective attention, Broadbent (1970) makes it clear that selective processes can be associated with any information processing stage. However, whereas it is trivially obvious that we can make post-perceptual decisions about the relevance of stimuli, the existence of pre-perceptual selective processes, whether in the form of a pre-cortical gating mechanism or an early cognitive decision stage, is not in any way obvious and must be ascertained empirically. In Chapter One it was argued that the amplitude of the N1 wave of the scalp EP indexes this latter 'stimulus set' mode of attention and the results of Experiments 3 and 4, whilst evidencing the subsequent 'response set' mode of selectivity, suggest that such early selective processes do not operate in the visual system. On the other hand, Experiment 5 does indicate the presence of early selective machinery in the auditory modality.

This apparent dissociation between the two modalities is interesting and indeed suggests that a simple cognitive explanation of 'stimulus set', such as Naatanen's (and indeed the "encoding theories" (Walley and Weiden, 1973) more generally), is inappropriate as it is difficult to see why such a general mechanism as a cognitive process should only be available to the auditory modality. If early cognitive processes can be applied to auditory sensory information then why not visual? On the other hand, it is correspondingly easy to appreciate that a specific piece of neural machinery, such as a thalamic gate, may or may not be a component part of the construction of an input system in the same way that any specific component part can be included or not in any overall assemblage. Presumably, as was discussed in Chapter Two, the absence of a filter in the visual system reflects the preeminence of peripheral processes in selective looking, rendering such an additional 'piece of machinery' superfluous.

Considering the underlying mechanisms of the N1 correlate of auditory 'stimulus set', the neuropsychological model of attention presented in Chapter One argued that the amplitude of the late componentry of the EP reflects the allocation of processing capacity to a stimulus, and that the selective attention related enhancement of EP amplitude is due to the greater allocation of processing capacity necessary to complete the more demanding mental work associated with attended stimuli. Thus it was predicted that the N1 correlate of selectivity would be abolished if the differential mental work associated with task relevance could be dissociated from the time of occurrence of attended stimuli. This prediction was confirmed in Experiment 6.

3) The fast response decrement of N1-P2 is an attentional phenomenon

A refractoriness explanation of the FRD of the vertex potential (i.e. N1-P2) was eventually rejected in favour of an attentional one, in which it is argued that the attention paid to a stimulus reflects its significance for the organism[1]. In FRD studies (fast habituation, temporal recovery)'stimulus significance' can be principally related to the inadequacies of a neuronal model of the stimulus (which are mainly temporal) and also, in the auditory modality, to a less well defined variable of 'firstness'. When 'stimulus significance' is adequately controlled the FRD is abolished. However, it is recognised that the experiments demonstrating the success of such control were restricted to the use of paradigms based on pairs of stimuli with ISIs in the range 1 to 3 seconds for the visual modality, and 3 seconds only for the auditory modality. In comparison, the FRD of the vertex N1-P2 is associated with a diverse variety of experimental procedures (including the use of trains of stimuli in the fast habituation paradigm) and a wider range of ISIs. However, it has been reasonably well-argued that the quintessence of the FRD can, although the terminology is somewhat clumsy, be encapsulated in the amplitude depression of a 'second' stimulus relative to a 'first' and that a powerful logical case can be made out to extend the mechanism responsible for this amplitude depression to any further decrements that may, for instance, be observed in the trains of stimuli employed in fast habituation studies. Furthermore, the ISIs used in this work are considered to be adequately representative of the range of ISIs of interest (i.e. from 500 ms up to a few seconds) across which range the present thinking, (at least in respect of the variable of

central interest - namely, temporal uncertainty) is supported by a variety of circumstantial evidence already discussed at various points in the thesis in connection with the extensive stimulus generalisation of the FRD, the effects of stimulus regularity and intensity, dishabituation, and the presence of a forward-latency-shift accompanying the FRD. However, it is recognised that at intervals of less than 500 ms both intuition and the appearance of a reaction time and EP latency delay suggest that some sort of physiological refractoriness variable may become of increasing importance. Thus the present finding that 'stimulus significance', and its temporal uncertainty component in particular, is the crucial variable, under albeit limited conditions, in FRD procedures employing ISIs of >500 ms is considered to justify the following general account of the major characteristics of the temporal recovery and the fast habituation of N1-P2.

In the defining paradigm of temporal recovery, pairs of stimuli are employed and, reflecting their respective 'significance', the amplitude of the second of the stimuli exhibits an amplitude depression in relation to the first. As the ISI lengthens, the temporal uncertainty associated with the second stimulus increases and hence its significance and the amplitude of N1-P2 also increase. The so-called temporal recovery and the amplitude-versus-ISI plot of the recovery function are thus generated. The fast habituation of the EP is explained as the progressive decline in temporal uncertainty and, in the case of auditory stimuli, 'firstness' over a train of stimuli. The reaccumulation of temporal uncertainty over the ISI limits the ultimate extent of the

decline, and because this reaccumulation is necessarily proportional to the length of the ISI, the effect of presentation rate upon the final asymptotic value of the habituation function is explained. The 'recovery' effects in the early temporal recovery studies of Davis et al (1966) and Gjerdingen and Tomsic (1970), which also employed trains of stimuli, are explained in a similar manner, with the AEPs for the various trains gaining in amplitude as the within-block ISI, and hence the temporal uncertainty and EP amplitude associated with the constituent stimuli, increases.

It is recognised that those aspects of this discussion relating to the covariation of the FRD with ISI in temporal recovery and fast habituation situations is based on the temporal uncertainty component of the 'stimulus significance' variable only. However, 'firstness' may also be influenced by ISI, although such a relationship is considerably more speculative. What is worth noting though is that, whereas 'firstness' is always potentially present in fast habituation paradigms its importance in temporal recovery experiments will vary depending upon the particular experimental procedure used, ranging from a minimal contribution in Roth et al's method and in the early work to a fully developed contribution in the pairs of stimuli used in the defining paradigm.

In addition to these major conclusions, the following three less important generalisations can also be abstracted from the research.

1) N1 reflects perceptual and P2 post-perceptual aspects of processing

Although components x treatments interactions were often not found to be significant, a number of interesting dissociations between N1 and P2 did emerge. It was concluded in Experiment 3 that the sensitivity of N1 to sensory variables and P2 to task variables indicated N1 to index perceptual and P2 post-perceptual processing. The greater lability of P2 in Experiment 9 can be attributed to the absence of a RT to standardise post-perceptual processing.

In Chapter One discussion of the functional dichotomy between the slow surface negativity and positivity of N1 and P2 in terms of the excitation and inhibition of cortical columns was not developed to any extent although it was suggested that the initial elaboration of a widespread coherent pattern of cortical column activation embodied in N1 subsumes the subject's perception of the world at the time of the stimulus. In Chapter Three it was more tentatively argued that the subsequent inhibitory interactions between columns that transform and process this pattern of activation subserve the post-perceptual information processing operations indexed by P2. It is acknowledged that the findings of Experiment 18, which correlate P2 with the resolution of the CNV, would, if substantiated, necessitate modification of this account and indeed question the relationship between the amplitude of the P2 component of the N1-P2 complex and mental work. However, for the sake of tidiness as well as caution these considerations are deferred for the moment pending substantiation of the P2-CNV relationship.

2) The vertex visual EP is more labile with respect to attention than its occipital counterpart

The capacity theory of attention embraced in the present research models attention as the investment of processing capacity. In Chapter One it was argued that the cortical columns of the non-specific cortex represented the neural substratum of processing capacity, and hence that the lability of the EP with respect to attention would be a function of the distance of the recording electrode from the specific sensory cortical areas, with a vertex electrode being particularly well-positioned in this respect. The findings in the visual modality of Experiment 4 and, in the light of an attentional mechanism for the FRD, Experiment 9 are consistent with this theorizing with the vertex EP being a more sensitive index of variations in attention than the EP concurrently recorded from the occiput. In respect of visual selective attention the fact that attentional effects are greater at the vertex provides further evidence that selective attention is not mediated by the gating of specific sensory input and, in the case of the visual FRD, a similar logical argument was also directed (in Chapter Five) against a peripheral mechanism for this effect. In general, the author finds such interactions between electrode location and information processing variables upon the evoked potential of considerable fascination and feels that such topographic studies, and in particular a recent study by Simson, Vaughan and Ritter (1977), signpost a profitable direction for future research into the elucidation of brain-behaviour relationships.

3) The time-course of processing is reflected in the latency of N1-P2

Although latency effects were found throughout the thesis associated with both the FRD and selective attention, and were adduced as evidence for an earlier or later onset of processing, this putative relationship is only convincingly demonstrated when corroborated by behavioural data. Experiment 2 in particular, and also Experiment 17, both indicate that earlier N1-P2s are associated with earlier reaction times.

GENERAL DISCUSSION

The principal objective of the thesis research programme was to elucidate the mechanisms underpinning the N1-P2 amplitude effects associated with selective attention and repetitive stimulation. In this respect a capacity theory of attention, whose neurophysiological and biophysical foundations were argued for in Chapter One, has been successfully applied to both of these categories of EP amplitude effects. This 'theory' of the amplitude of the late componentry of the EP was intended to have some generality when it was introduced in the first chapter. Given these subsequent successful applications in respect of N1-P2 it is thus tempting to argue that the 'theory' provides a complete account of EP amplitude effects in this latency range, i.e. that the amplitude of the late waves of the EP (more specifically, N1-P2) is completely determined by the attention paid (i.e. capacity allocation) to a stimulus, which in turn is determined by the prevailing importance of various specific factors (task demands, 'firstness', objective information content, expectancies, salience, general arousal, etc.) whose influence is

expressed via the general construct of 'stimulus significance'. One advantage of this perspective is that it draws attention to the dynamic psychological determinants of the amplitude of the late components of the EP and away from static physiological determinants, such as refractoriness in the particular context of this thesis but also, by implication, physiological determinants more generally, including, for instance, specific sensory activity. However, it is conceded that the determinants of 'stimulus significance' are conveniently diverse, difficult to quantify and potentially arbitrary. Nonetheless the 'theory' does generate a general prediction which is eminently testable: namely, that if 'stimulus significance', and hence attention, is adequately controlled then any amplitude effects should be abolished. It was this prediction that motivated the crucial experiments of the thesis and was ultimately confirmed for both the amplitude effects associated with selectivity and stimulus repetition, enabling the particular determinants of these effects to be identified.

However, despite these confirmations of the present theory's general prediction apropos selective attention and the FRD, it is recognised that this success, although gratifying, does not conclusively validate the theory although it does confirm it as a useful and valuable perspective. Apart from any other considerations, the EP almost certainly contains a contribution from the relevant specific sensory area which, being presumably determined by the physical properties of the stimulus, cannot be regarded as attentional. The literature on the influence of such sensory variables upon the EP is extensive (see Regan, 1972) and indeed the vertex EP itself exhibits sensory effects (see, for instance,

Experiment 3). However, before diluting the present position to accommodate (non-attentional) sensory influences over EP amplitude it is pointed out a) that the scalp record has been primarily associated with the activity of the non-specific cortex and b) that sensory effects such as those, for instance, related to stimulus intensity could, although somewhat unparsimoniously, be attributed to intervening attentional variables, such as in this case 'salience'. Although this latter statement is admittedly difficult to test, it does receive some support from the finding that the relationship between EP amplitude and stimulus intensity is not invariant. Soskis and Shagass (1974) report that in some subjects an increase in intensity produces a decrement in the amplitude of the late components of the EP.

Although the success of Experiments 13 and 17 in abolishing the FRD, as has been recognised, does not conclusively validate an attentional (i.e. capacity) theory of EP amplitude nor indeed prove that a neuronal model of the stimulus exists, these experiments certainly point to temporal uncertainty as the key variable underpinning the FRD. Although the mediation of the effects of temporal uncertainty upon EP amplitude via the conceptual vehicle of a 'neuronal model which can recruit mental work' is logically coherent and relates to the established habituation literature, it was conceded in the discussion of Experiment 13 that other forms of explanation of the abolition of the FRD when temporal uncertainty is controlled are possible. In particular, the construct of preparation was indicated as a powerful one in this respect. If the amplitude of the EP is related to the subject's preparedness then it is plausible that

the elimination of temporal uncertainty in this work is effective in eliminating differences in preparedness, and hence EP amplitude. Certainly it is not inconceivable that the amplitude of N1 and P2 are related to certain characteristics of the ambient slow potential changes that map-out the time-course of preparation (for instance, N1 could be related to the final amplitude of the CNV via some sort of ceiling effect and P2 related to the rate of resolution of this d.c. shift) which the experimental control over temporal uncertainty effectively regulate. However, without the relevant d.c. records and, more importantly, a theoretical predisposition in respect of the preparation construct which would, in particular, precisely explain how preparation mediates the observed effects of temporal uncertainty upon N1 and P2 amplitude, this construct is not pursued any further and the present attentional orientation maintained.

However, although the present research identifies temporal uncertainty as the key variable in FRD studies other variables are also important. Indeed the experiments of Chapter Six indicate that, in the auditory modality, a second variable, loosely designated 'firstness', also appears to be relevant. Moreover, although the FRD, as discussed in Experiment 12, does display extensive stimulus generalisation, there is some evidence of specificity with respect to the physical characteristics of stimulation. The work of Butler (1972a,b), for instance, indicates some specificity of the auditory vertex potential with respect to spatial position and pitch; and although in the discussion section of Experiment 12 the evidence recruited from a study by Boddy (1973) suggested complete cross-modality generalisation of the

FRD, the work of Davis et al (1972), specifically directed at this issue, indicates some modality specificity, with a prior flash or shock producing a 30 to 40% reduction in the amplitude of the auditory vertex potential as compared with the figure of 60% for a prior auditory stimulus. However, although temporal uncertainty is apparently not the 'whole story', identification of this variable is important as it elucidates why the FRD is sensitive to stimulus presentation rate, a finding which is not easily explicable from a simple model-making theory of habituation that only makes recourse to considerations of the physical characteristics of stimulation. Why should a neuronal model of the physical characteristics of the stimulus be any more adequate after a shorter ISI than a longer ISI? In respect of this question, the implicit thinking in the literature would appear to run as follows. Because we have classified the FRD of the EP as a type of habituation, and because habituatory response decrements develop more rapidly at faster stimulus presentation rates then the sensitivity of the FRD to presentation rate is 'explained'. Thus the operationally defined phenomenon of habituation is conveniently elevated to the status of an explanatory construct in order to explain away a characteristic of the phenomenon and finesse an awkward, but nonetheless interesting, question.

Finally, on the subject of the role of temporal uncertainty in FRD studies the work of Ohman and his group receives some attention. Having isolated this variable as the key component of the FRD, it is disquieting to note that in the various studies of the fast habituation of the EP conducted by this group a red light was used throughout to warn the subjects

of the arrival of the trains of auditory stimuli, and that this light came on at a foreperiod equal to the within-train ISI. The various other habituation studies discussed in the thesis do not warn their subjects with any such proximity or precision. Given that temporal uncertainty is the central variable in fast habituation experiments, the use of this warning device is clearly unwise and would be expected to remove much of the 'first stimulus effect' from their data. This latter expectation is confirmed upon comparison of their data with the data of other fast habituation studies. This comparison reveals that their within-train decrements look like the decrements obtained by other authors from stimulus number 2 onwards, i.e. excluding the first stimulus. For instance, under comparable experimental conditions (i.e. ISI = 3 seconds, subject attending) Maclean, Ohman and Lader (1975) find that the amplitude of N1-P2 evoked by the second stimulus in their train remains at approximately 80% of the amplitude of that evoked by the first stimulus. In contrast, Fruhstorfer et al's (1970) data evidences figures of approximately 40% and 70% for the corresponding ratios between stimulus numbers 1 and 2, and 2 and 3 respectively. Thus it is apparent that the use of the warning device in Ohman's work seriously attenuates the magnitude of the decrement between the first and second stimulus positions in a train to a degree that is comparable to that normally obtained between the second and third stimulus positions. This effective elimination of the 'first stimulus effect' that contributes so substantially to the fast habituation of the EP confounds the integration of their findings with the main body of the literature and indeed casts doubt over the validity

of their work. To give one example, in a study in 1972 Ohman, Kaye and Lader find no evidence in their data for an exponential within-train N1-P2 amplitude decline with irregular stimulation (see Figure 1 of this paper) and thereby deduce that regular stimulation is a necessary condition for this effect. However, this null result should be treated with some caution given that their warning device eliminates the locus of much of the exponential aspects of fast habituation, i.e. the 'first stimulus effect', and the possibility must remain that such an exponential trend would indeed have been found had they not employed this device.

On the subject of this particular paper a further minor criticism is dealt with. Ohman et al point out, quite reasonably, that a neural refractoriness mechanism cannot easily account for the effects of the temporal characteristics of stimulation upon the FRD. Although they only refer to effect of stimulus regularity in this respect, to this author's mind Klinke et al's (1968) success in eliciting dishabituation with a shortening of ISI is also difficult to explain from a refractoriness viewpoint. However, this author would like to take issue with their argument (p.277) that a refractoriness mechanism predicts no effect of stimulus regularity. I would argue that, if anything, the reverse of what is observed, i.e. a more not less pronounced response decrement, is in fact predicted for the reason that, given the asymmetry of the recovery process about any position on the recovery function, the mean 'recovery' of a range of ISIs would be less than the recovery associated with the mean ISI of this range.

Finally, considering the question of the qualification of the fast

habituation of the EP as genuine habituation in the purely operational terms of Thompson and Spencer's (1966) criteria, the thesis produces some further evidence in this regard. Ohman, Kaye and Lader (1972) indicate potentiation of habituation as an important criterion for recognising genuine habituation and the material presented in Footnote 1 of Chapter Four to some extent reveals potentiation of habituation in the visual fast habituation data of Experiments 7 and 8. The demonstration that peripheral mechanisms do not significantly influence the FRD (see the control study associated with Experiment 9, and also Experiment 12) represents a further contribution to this debate in favour of genuine habituation, although the failure of the above control study to show an effect of stimulus intensity upon the rate of habituation leaves another of Thompson and Spencer's criteria unfulfilled.

SUGGESTIONS FOR FURTHER RESEARCH

Although a diverse and multitudinous array of research suggestions derive from the thesis research, the author will restrict himself to those associated with the application of the 'clock'paradigm to control temporal uncertainty. Certainly, the relationship between the length of the ISI *per se* (i.e. without the confounding variable of temporal uncertainty) and EP amplitude could be mapped out across a wider range of ISIs, and in particular at intervals much shorter than those employed here (i.e. < 500 ms) where the psychological literature (Smith, 1967) certainly suggests the presence of a state of general cortical refractoriness. In general, the investigation of a variety of aspects of performance

(including reaction time) would be enhanced with temporal uncertainty under experimental control.

The development of the slow negative potential shift of the CNV in advance of an imperative stimulus has been related to the elaboration of motor preparation (Loveless, 1977). The hypothesis that the finer control over preparation afforded by a 'clock' should be reflected in the concomitant CNV is of interest. The results of varying parameters of the 'clock'(speed, acceleration, etc.) might also be explored.

The author is also particularly interested in the possibility of employing the paradigm to investigate emitted potentials. Such potentials consist primarily of a positive deflection occurring at a latency of 250 to 500 ms after the time of an expected but absent stimulus. The 'shallowness' of these potentials has been generally ascribed to their greater latency variability and various sophisticated data-analytic procedures (see Weinberg, Walter and Crow, 1970; and Ruchkin and Sutton, 1977) have been applied in order to provide a better estimate of their amplitude. The 'clock' paradigm affords a simpler solution to this problem: the arrival of the moving spot at a 'target' position would precisely time-lock brain potentials whether a stimulus occurred or not. Experiment 18 explores this possibility.

Experiment 18: Emitted Potentials, the CNV and Temporal Uncertainty

INTRODUCTION AND METHOD

In this pilot experiment, in addition to evaluating the use of the 'clock'paradigm to time-lock emitted potentials, the hypothesis that the CNV would reflect the finer control over preparation afforded by a 'clock' was also investigated. It was anticipated that, with the time of

occurrence of the imperative stimulus precisely known, preparation could be, and presumably would be, 'put off' until the last possible moment and hence that a more concave CNV would be obtained than in an 'unclocked' condition.

Two subjects were recruited and their EEG recorded from a vertex electrode referred to a mastoid placement. A second contralateral mastoid placement provided the ground. The EEG was amplified by a Grass 7P1D d.c. preamplifier (set to d.c.) coupled to a 7DAF driver amplifier, and also by the usual a.c. system, yielding simultaneous a.c. and d.c. records.

Trials were as follows. A spot appeared on an oscilloscope screen and after a random interval in the range .5 to 2 seconds disappeared again. On 'clocked' trials (illustrated in Figure 7.1) the spot immediately reappeared and began to move at a uniform speed towards a pair of stationary spots which formed a gate through which it eventually passed 2 seconds later[2]. A chequerboard was flashed onto the screen at this point with a probability of .5, the subject being directed to respond as quickly as possible to such an event. The spot continued to move on for a further 1.5 seconds before the trial was concluded. In the 'unclocked'condition the spot did not reappear and the chequerboard simply occurred, with $p = .5$, 2 seconds after the initial disappearance of the spot. Again a reaction time was required. Thus four types of trial were generated; clocked/stimulus present (CS), clocked/no stimulus (CN), unclocked/stimulus present (US), and unclocked/no stimulus (UN). The experiment consisted of a practice session followed by a single run of 64 trials, comprising a random

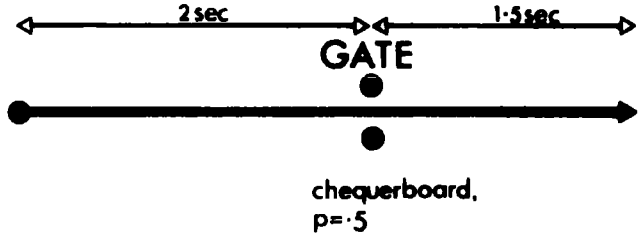

Figure 7.1

Illustration of the 'clock' paradigm of experiment 18. See text for details.

permutation of 16 of each of the four types. Subjects were instructed to fixate the 2 stationary spots, which remained on throughout, and were exhorted not to track the moving spot at any cost. The total display area subtended less than 1.5 degrees at the eye, allowing it to be completely absorbed from the fixation point[3].

RESULTS AND DISCUSSION

The d.c. brain activity over a 4.5 second epoch, extending from 1 second before the initial disappearance of the spot until 1.5 seconds after the imperative stimulus, was averaged for each type of trial and then digitally filtered. The resulting waveforms for one subject (SW) are compiled in Figure 7.2a. The corresponding averaged a.c. records for a one second epoch after the time of occurrence of the imperative stimulus for the four trial types are also shown for the same subject in Figure 7.2b. The reaction times for both subjects were significantly faster in the clocked condition; for subject SW by 20 ms (225,245 ms; Mann-Whitney U = 24***) and subject AM by 35 ms (245,280 ms; U = 49***).

Figure 7.2a indicates that the facilitation of reaction time in the 'clocked' condition is associated with a more concave CNV morphology for subject SW. Subject AM's data, although not presented, showed the same effect and the experimental hypothesis was thus deemed confirmed. Intuitively, uncertainty about the exact time of occurrence of the imperative stimulus is considered to dictate that a state of readiness to respond be achieved comfortably in advance of its approximate position. As already indicated, certainty of the temporal location of the imperative stimulus would, on the

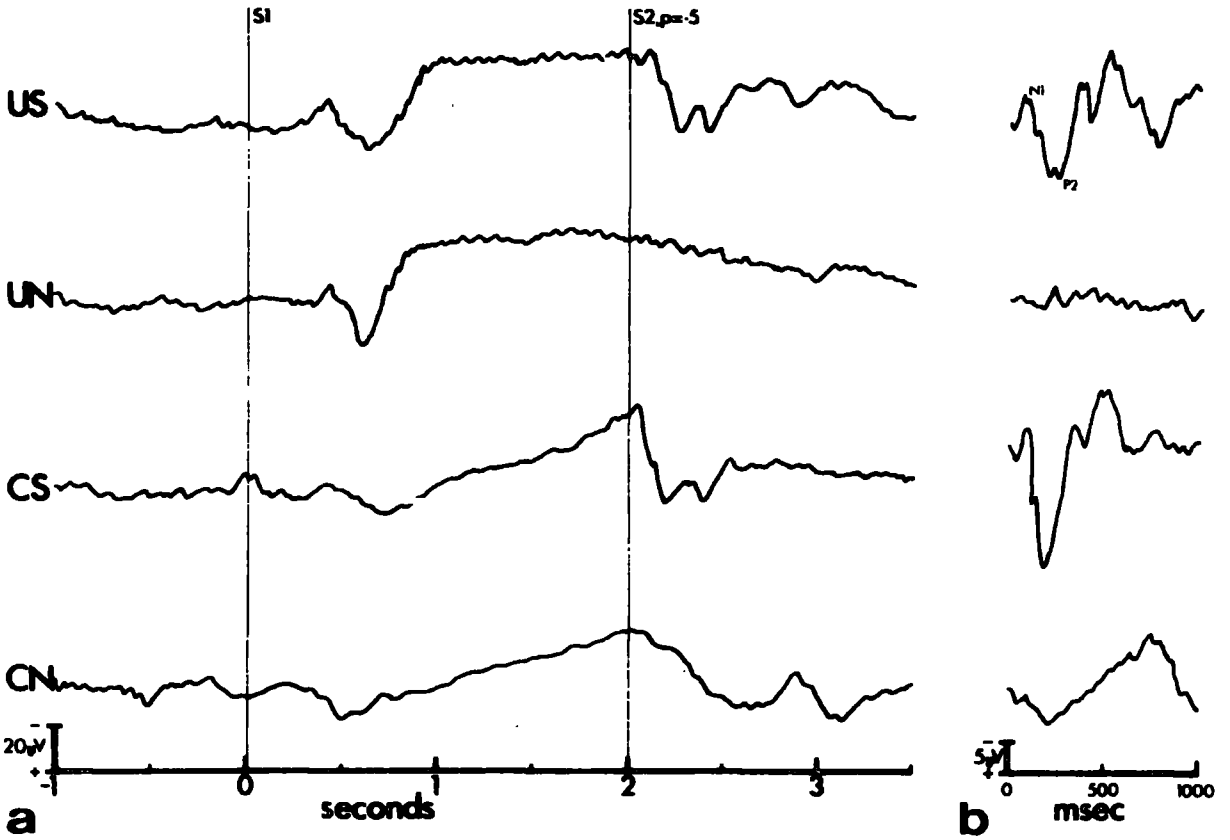

Figure 7.2

The d.c. brain activity for subject SW averaged over a 4.5 second epoch extending from 1 second before the initial disappearance of the spot (S1 - the warning signal) until 1.5 seconds after the time of occurrence of the imperative stimulus (S2, 2 seconds after S1) is shown in part a for each of the four trial-types of experiment 18 (i.e. CN, CS, UN, US - see text). The corresponding averaged a.c. records for a 1 second epoch after the time of occurrence of S2 are also shown in part b.

other hand, allow preparation to be deferred until some later point in time. The CNV morphological differences in this experiment would appear to be consistent with these putative differences in motor preparation between the 'clocked' and the 'unclocked' conditions.

As hoped, an emitted positive event is noted at a latency of 250 ms for the clocked/no stimulus record in Figure 7.2b. No corresponding deflection is noted in the corresponding 'unclocked' condition and it thus appears that the paradigm has some value in time-locking emitted potentials. Subject AM's results were essentially similar with a shallow positive wave (latency, 375 ms) in the CN but not the UN condition. Inspection of both subjects' d.c. and a.c. records together for the CS, CN and US conditions indicated that, in all cases, the late positivity of the a.c. record coincides with the resolving CNV, with the peak of this positivity coinciding with the point at which this resolution is steepest. This can be most clearly seen in Figure 7.2 (SW) in the CN condition where the CNV resolution is conveniently stretched out. For the two evoked potentials (CS,US) this late positivity, being the first positive deflection after N1, constitutes the P2 component of the AEP in the terminology of the thesis. It was suggested in the discussion section of Experiment 3 that P2 could represent a reactive change on the subject's part: the data of this study appear to support this suggestion.

Thus, it is tentatively argued that the late positivity of evoked and emitted brain potentials reflects the gradient of resolution of the CNV which, because the a.c. record is simply the differentiation of the underlying d.c. record, necessarily intrudes as a positive deflection.

Inspecting the d.c. record in the CN condition for subject SW it is apparent that the average CNV resolves less rapidly than in either stimulus present condition. The second subject's data showed the same effect. Examination of superimposed single trials indicated that the more gradual nature of this resolution in the main reflected the morphology of the individual constituent CNVs rather than, for instance, being an artifact of their greater latency variability (although they were slightly more variable, indicating the 'clock'paradigm to be not entirely successful). It thus appears that the non-occurrence of a stimulus is not as effective in resolving the CNV, and that the shallowness of emitted potentials is largely a reflection of this phenomenon.

Of course, numerous studies have addressed the relationship between the CNV and late positivity in the EP in the latency range of that observed in the present study (i.e. peaking at around 300 ms), although the label P300, rather than P2, is generally attached to this positivity in these studies. Donchin, Tueting, Ritter, Kutas and Heffley (1975) conclude that because the amplitude of the CNV and P300 can be dissociated in terms of a) sensitivity to experimental variables and b) scalp topography, they are independent processes, and this appears to be the concensus of opinion. Although the present findings are apparently inconsistent with this conclusion, it seems to this author that the established literature may have wrongly concerned itself with the relationship between the amplitude of the CNV and the late positivity of the EP when the gradient of CNV resolution is manifestly the appropriate variable. Clearly, more research is required.

Footnotes - Chapter Seven

(1) The term 'stimulus significance' is also used by Ruchkin and Sutton (1973) with reference to the P3 component of the EP. The amplitude of this component, as already noted in the discussion section of Experiment 16, is held to reflect the unexpectedness or surprisal value of a stimulus. Again the concept of an internal model of the world has been introduced, with Duncan-Johnson and Donchin (1977 , p.466) commenting that when "the unexpected happens, the model of the operating context must be revised. It is hypothesised that this context revision process is manifested by P300". Typically in P3 studies, the P3 component of the EP to infrequent events in a sequence is enhanced relative to its amplitude in response to the frequent events, but no effects of stimulus probability upon N1-P2 amplitude are obtained (e.g. Roth and Kopell, 1973). Thus although the amplitudes of both N1-P2 and P3 can be related to 'stimulus significance', this latter dissociation indicates that the two components are sensitive to different aspects of this variable.

Where N1-P2 correlates with 'stimulus significance' in selective attention and fast habituation paradigms, the occurrence of a stimulus is an informative but not a surprising event. The mental work associated with this occurrence is specified in advance of the stimulus and is simply released when the stimulus occurs. In the P3 studies, however, the eventuality of an unexpected stimulus is, by definition, an event for which the organism is not prepared and the mental reaction is therefore less immediate and, as a consequence, is reflected in a component developing at a longer latency than N1-P2, i.e. P3. However, in terms of the temporal inadequacies of the neuronal model, with which the pre-set aspects of the stimulus evoked mental work are concerned, expected and unexpected events in the P3 studies are equivalent, with the stimuli being presented in long sequences separated by constant ISIs. The theory of this thesis is thus not embarrassed by the findingsof these studies that P3, but not N1-P2, covaries with differences in 'stimulus significance' related to the surprisal value of the stimulus.

(2) It should be emphasised that the spot does 'pass through' the gate. It did not coalesce with either of the stationary spots as such an event might arguably be construed as an evoking stimulus.

(3) It was thus hoped that the number of eye artifacts would be minimal. No recording manipulations to control for any such artifacts were in fact included, although it was noted that the vertex-mastoid electrode configuration used is not especially vulnerable in this respect. This deficiency is not intended to prescriptive but rather reflects the preliminary nature of the investigation.

BIBLIOGRAPHY

Arbib, M.A. (1972). The Metaphorical Brain: an Introduction to Cybernetics as Artificial Intelligence and Brain Theory. Wiley, New York.

Bess, J.C. and Ruhm, H.B. (1972). Recovery cycle of the acoustically evoked potential. Journal of Speech and Hearing Research, 15, 507-517.

Boddy, J. (1972). The psychological refractory period and vertex evoked Potentials. Quarterly Journal of Experimental Psychology, 24, 175-192.

Boddy, J. (1973). Evoked potentials in reaction time with a variable foreperiod. Quarterly Journal of Experimental Psychology, 25, 323-334.

Bogacz, J., Vanzulli, A. and Garciá-Austt, E. (1962). Evoked responses in man. IV. Effects of Habituation, Distraction and Conditioning upon auditory evoked responses. Acta Neurologica Latinoamericana, 8, 244-252.

Bogacz, J., Vanzulli, A., Handler, P. and Garciá-Austt, E. (1960). Evoked responses in man. II. Habituation of visual evoked response. Acta Neurologica Latinoamericana, 6, 353-362.

Borda, R.P. and Frost Jnr., J.D. (1968). Error reduction in small sample averaging through the use of the median rather than the mean. Electroencephalography and Clinical Neurophysiology, 25, 391-392.

Bostock, H. and Jarvis, M.J. (1970). Changes in the form of the cerebral evoked response related to the speed of simple reaction time. Electroencephalography and Clinical Neurophysiology, 29, 137-145.

Broadbent, D.E. (1958). Perception and Communication. Pergamon, London.

Broadbent, D.E. (1970). Stimulus set and response set: Two kinds of selective attention. In Mostofsky, D.I. (Ed.), Attention: Contemporary Theory and Analysis. Appleton-Century-Crofts, New York, 51-60.

Butler, R.A. (1972a). The influence of spatial separation of sound sources on the auditory evoked response. Neuropsychologia, 10, 219-225.

Butler, R.A. (1972b). Frequency specificity of the auditory evoked response to simultaneously and successively presented stimuli. Electroencephalography and Clinical Neurophysiology, 33, 277-282.

Callaway, E. (1973). Habituation of averaged evoked potentials in man. In Peeke, H.V.S. and Herz, M.J. (Eds.), Habituation, II: Physiological Substrates. Academic Press, London, 153-174.

Chatfield, C. (1975). The Analysis of Time series: Theory and Practice. Chapman and Hall, London.

Creutzfeldt, A., Rosina, M., Ito, M. and Probst, W. (1969). Visual evoked response of single cells and of the EEG in primary visual area of the cat. Journal of Neurophysiology, 32, 127-139.

Creutzfeldt, O.D., Watanabe, S. and Lux, H.D. (1966). Relations between EEG phenomenon and potentials of single cortical cells. I. Evoked responses after thalamic and epicortical stimulation. Electroencephalography and Clinical Neurophysiology, 20, 1-18.

Davis, H. (1964). Enhancement of evoked cortical potentials in humans related to a task requiring a decision. Science, 145, 182-183.

Davis, H., Mast, T., Yoshie, N. and Zerlin, S. (1966). The slow response of the human cortex to auditory stimuli: Recovery process. Electroencephalography and Clinical Neurophysiology, 21, 105-113.

Davis, H., Osterhammel, R.A., Weir, C.C. and Gjerdingen, D.B. (1972). Slow vertex potentials: Interactions among auditory, tactile, electric and visual stimuli. Electroencephalography and Clinical Neurophysiology, 33, 537-545.

Dawson, W.W. and Doddington, H.W. (1973). Phase distortion of biological signals: Extraction of signal from noise without phase error. Electroencephalography and Clinical Neurophysiology, 34, 207-211.

Derbyshire, A.J., Driessen, G.J. and Palmer, C.W. (1967). Technical advances in the analysis of single acoustically evoked potentials. Electroencephalography and Clinical Neurophysiology, 22, 476-481.

Deutsch, J.A. and Deutsch, D. (1963). Attention: Some theoretical considerations. Psychological Review, 70, 80-90.

Donchin, E. (1966). A multivariate approach to the analysis of average evoked potentials. IEEE Transactions on Biomedical Engineering, 3, 131-139.

Donchin, E. (1969a). Discriminant analysis in average evoked potential studies: The study of single trial data. Electroencephalography and Clinical Neuropnysiology, 27, 311-314.

Donchin, E. (1969b). Data analysis techniques in averaged evoked potential research. In Donchin, E. and Lindsley, D.B. (Eds.), Average Evoked Potentials. NASA SP-191, Washington, 199-217.

Donchin, E. and Cohen, L. (1967). Averaged evoked potentials and intramodality selective attention. Electroencephalography and Clinical Neurophysiology, 22, 537-546.

Donchin, E. and Herning, R.I. (1975). A simulation study of the efficacy of stepwise discriminant analysis in the detection and comparison of event related potentials. Electroencephalography and Clinical Neurophysiology, 38, 51-68.

Donchin, E. and Lindsley, D.B. (1966). Average evoked potentials and reaction times to visual stimuli. Electroencephalography and Clinical Neurophysiology, 20, 217-223.

Donchin, E., Tueting, P., Ritter, W. Kutas, M. and Heffley, E. (1975). On the independence of the CNV and the P300 components of the human averaged evoked potential. Electroencephalography and Clinical Neurophysiology, 38, 449-461.

Duncan-Johnson, C.C. and Donchin, E. (1977). On quantifying surprise: The variation of event-related potentials with subjective probability. Psychophysiology, 14, 456-467.

Eason, R.G., Harter, R.M. and White, C.T. (1969). Effects of attention and arousal on visually evoked cortical potentials and reaction time in man. Physiology and Behaviour, 4, 283-289.

Eason, R.G., White, C.T. and Bartlett, N. (1970). Effects of chequerboard pattern stimulation on evoked cortical responses in relation to check size and visual field. Psychonomic Science, 2, 113-115.

Eccles, J.C. (1973). The Understanding of the Brain. Macgraw-Hill, New York.

Fisher, R.A. (1950). Statistical Methods for Research Workers. Oliver and Boyd, Edinburgh.

Ford, J.M., Roth, W.P. and Kopell, B.S. (1976). Attention effects on auditory evoked potentials to infrequent events. Biological Psychology, 4, 65-77.

Fruhstorfer, H. (1971). Habituation and dishabituation of the human vertex response. Electroencephalography and Clinical Neurophysiology, 30, 306-312.

Fruhstorfer, H. and Bergstrom, R.M. (1969). Human vigilance and auditory evoked responses. Electroencephalography and Clinical Neurophysiology, 27, 346-355.

Fruhstorfer, H., Soveri, P. and Jarvilehto, T. (1970). Short-term habituation of the auditory evoked response in man. Electroencephalography and Clinical Neurophysiology, 28, 153-161.

Gastaut, H. and Regis, H. (1965). Visually evoked potentials recorded transcranially in man. In Procter, L.D. and Adey, W.R. (Eds.), The Analysis of Central Nervous System and Cardiovascular Data using Computer Methods. NASA, Washington, 7-34.

van der Geer, J.P. (1971). Introduction to Multivariate Analysis for the Social Sciences. Freeman, San Francisco.

Gerken, G.M., Moushegian, G., Stillman, R.D. and Rupert, A.L. (1975). Human frequency - following responses to monaural and binaural stimuli. Electroencephalography and Clinical Neurophysiology, 38, 379-386.

Gjerdingen, D.B. and Tomsic, R. (1970). Recovery functions of human cortical potentials evoked by tones, shocks, vibration and flashes. Psychonomic Science, 19, 228-229.

Green, D.M. and Swets, J.A. (1966). Signal Detection Theory and Psychophysics. Wiley, New York.

Groves, P.M. and Eason, R.G. (1967). Visually evoked cortical potentials from different cortical regions in humans. Psychonomic Science, 9, 19-20.

Haber, R.N. and Herchenson, M. (1973). The Psychology of Visual Perception. Holt, Rinehart and Winston, New York.

Haider, M. (1967). Vigilance, attention, expectancy and cortical evoked potentials. Acta Psychologica, 27, 246-252.

Haider, M., Spong, D. and Lindsley, D.B. (1964). Attention, vigilance and cortical evoked potentials in humans. Science, 145, 180-182.

Harris, J.D. (1943). Habituatory response decrements in the intact organism. Psychological Bulletin, 40, 385-422.

Harris, E.K. and Woody, C.D. (1969). Use of an adaptive filter to characterise signal-noise relationships. Comput. Biomed. Res., 2, 242-273.

Harter, M.T. (1971). Visually evoked cortical responses to the on-set and off-set of patterned light in humans. Vision Research, 11, 685-695.

Harter, M.R. and White, C.T. (1968). Effects of contour sharpness and check-size on visually evoked cortical potentials. Vision Research, 8, 701-711.

Harter, M.R. and White, C.T. (1970). Evoked cortical responses to chequerboard patterns: Effect of check-size as a function of visual acuity. Electroencephalography and Clinical Neurophysiology, 28, 48-54.

Harter, M.R. and Salmon, L.E. (1972). Intra-modality selective attention and evoked cortical potentials to randomly presented patterns. Electroencephalography and Clinical Neurophysiology, 32, 605-613.

Hernández-Peón, R. (1966). Physiological mechanisms in attention. In Russell, R.W. (Ed.), Frontiers in Physiological Psychology. Academic Press, New York, 121-147.

Hernández-Peón, R., Jouvet, M. and Scherrer, H. (1957). Auditory potentials at cochlear nucleus during acoustic habituation. Acta Neurologica Latinoamericana, 3, 144-156.

Hernández-Peón, R., Scherrer, H. and Jouvet, M. (1956). Modification of electrical activity in cochlear nucleus during 'attention' in unanaesthetized cats. Science, 123, 331-332.

Hillyard, S.A., Hink, R.F., Schwent, V.L. and Picton, T.W. (1973). Electrical signs of attention in the human brain. Science, 182, 177-180.

Hink, R.F. and Hillyard, S.A. (1976). Auditory evoked potentials during selective listening to dichotic speech messages. Perception and Psychophysics, 20, 236-242.

Hink, R.F., van Hoorhis, S.T., Hillyard, S.A. and Smith, T.S. (1977). The division of attention and the human auditory evoked potential. Neuropsychologia, 15, 597-605.

Hochberg, J. (1970). Attention, organisation and consciousness. In Mostofsky, D.E. (Ed.). Attention: Contemporary Theory and Analysis. Appleton-Century-Crofts, New York, 99-124.

Hockey, R. and Hamilton, P. (1977). The basis of the primacy effect: Some experiments with running memory. Quarterly Journal of Experimental Psychology. 29, 49-63.

van Hoek, L.D. (1974). Multivariate vectorial analysis of the visual evoked response. Kybernetik, 15, 65-72.

Hubbard, J.I., Llinas, R. and Quastel, D.M.J. (1969). Electrophysiological Analysis of Synaptic Transmission. Arnold, London.

Hubel, D.H. and Wiesel, T.N. (1962). Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. Journal of Physiology, 160, 106-154.

Jewett, D.L., Romano, M.N. and Williston, J.S. (1970). Human auditory evoked potentials: Possible brain stem components detected on the scalp. Science, 167, 1517-1518.

Jewett, D.L. and Williston, J.S. (1971). Auditory-evoked far fields averaged from the scalp of humans. Brain, 94, 681-696.

Kahneman, D. (1973). Attention and Effort. Prentice-Hall, New Jersey.

Kandel, E.R. (1970). Nerve cells and behaviour. In Progress in Psychobiology - Readings from Scientific American. Freeman, San Francisco, 281-292.

Kandel, E.R. (1976). Cellular Basis of Behaviour: An Introduction to Behavioural Neurobiology. Freeman, San Francisco.

Karlin, L. (1970). Cognition, preparation and sensory evoked potentials. Psychological Bulletin, 73, 122-136.

Kavanagh, R.N., Darcey, T.M. and Fender, D.H. (1976). The dimensionality of the human visual evoked scalp potential. Electroencephalography and Clinical Neurophysiology, 40, 633-644.

Kendal, M.G. (1947). The Advanced Theory of Statistics, I. Griffin, London.

Keppel, G. (1973). Design and Analysis: A Researcher's Handbook. Prentice-Hall, New Jersey.

Klinke, R., and Fruhstorfer, H. and Finkinzeller, P. (1968). Evoked responses as a function of external and stored information. Electroencephalography and Clinical Neurophysiology, 25, 119-122.

Kooi, K.A. and Bagchi, B.K. (1964). Visual evoked responses in man: Normative data. Annals of the New York Academy of Sciences, 112, 254-269.

Lev, A. and Sohmer, H. (1972). Sources of averaged neural responses recorded in animal and human subjects during cochlear audiometry (electrocochleogram). Arch. klin. exp. Ohr.-, Nas.- U. Kihlk. Heilk., 201, 79-90.

Loveless, N.E. (1977). Event-related brain potentials in selective response. Biological Psychology, 5, 135-149.

Loveless, N.E. and Sandford, A.J. (1974). Slow potential correlates of preparatory set. Biological Psychology, 1, 303-314.

Mackay, D.M. (1969). Physiological correlates of evoked potentials (EPs). In Neurosciences Research Program, 7, 189-204.

Maclean, V., Ohman, A. and Lader, M. (1975). Effects of attention, activation and stimulus regularity on short-term 'habituation' of the averaged evoked response. Biological Psychology, 3, 57-69.

Marsh, J.T., Brown, W.S. and Smith, J.C. (1975). Far-field recorded frequency-following responses: Correlates of low-pitch auditory perception in humans. Electroencephalography and Clinical Neurophysiology, 38, 113-119.

Marsh, J.T. and Worden, F.G. (1964). Auditory potentials during acoustic habituation: Cochlear nucleus, cerebellum and auditory cortex. Electroencephalography and Clinical Neurophysiology, 17, 685-692.

Marsh, J.T. and Worden, F.G. (1968). Sound evoked frequency-following responses in the central auditory pathway. Laryngoscope (St. Louis), 68, 1149-1163.

Moray, N. (1969). Listening and Attention. Penguin, Harmondsworth.

Morrell, L.K. and Morrell, F. (1966). Evoked potentials and reaction times: A study of intra-individual variability. Electroencephalography and Clinical Neurophysiology, 20, 567-575.

Morris, C.J. (1971). Electroencephalographic and evoked potential correlates of reaction time and visual discrimination performance. Psychonomic Science, 23, 193-195.

Mountcastle, V.B. (1957). Modality and topographic properties of single neurones of cat's somatic sensory cortex. Journal of Physiology, 20, 408-434.

Moushegian, G., Rupert, A.L. and Stillman, R.D. (1973). Scalp-recorded early responses in man to frequencies in the speech range. Electroencephalography and Clinical Neurophysiology, 35, 665-667.

Näätänen, R. (1967). Selective attention and evoked potentials. Annales Academiae Scientiarium Fennicae, B151, 1, 1-226.

Näätänen, R. (1975). Selective attention and evoked potentials in humans - a critical review. Biological Psychology, 2, 237-307.

Neisser, U. (1963). Decision-time without reaction-time: Experiments in visual scanning. American Journal of Psychology, 76, 376-385.

Neisser, U. (1967). Cognitive Psychology. Appleton-Century-Crofts, New York.

Neisser, U. (1969). Selective Reading: A method for the study of visual attention. Paper presented at the symposium on Attention: Some Growing Points in Recent Research. XIX International Congress of Psychology, London.

Neisser, U. and Beller, H.K. (1965). Searching through word lists. British Journal of Psychology, 56, 349-358.

Neisser, U. and Stoper, A. (1965). Redirecting the search process. British Journal of Psychology, 56, 359-368.

Nelson, D.A., Lassman, F.M. and Hoel, R.L. (1969). The effects of variable-interval and fixed-interval signal presentation schedules on the auditory evoked response. Journal of Speech and Hearing Research, 12, 199-209.

Nie, N.H., Hull, C.H., Jenkins, J.G., Steinbrenner, K. and Bent, D.H. (1975). Statistical Package for the Social Sciences. McGraw-Hill, New York.

Norman D.A. (1976). Memory and Attention, 2E. Wiley, New York.

Ohman, A., Kaye, J.J. and Lader, M. (1972). Regular interstimulus interval as a critical determinant of the short-term 'habituation' of the auditory averaged evoked response. Psychonomic Science, 27, 275-278.

Ohman, A. and Lader, M. (1972). Selective attention and 'habituation' of the auditory averaged evoked response in humans. Physiology and Behaviour, 8, 79-85.

Ohtani, A. and Yagi, A. (1971). Average evoked potentials as a measure of the mental load. Paper presented at the 17th International Congress of Applied Psychology.

Palmer, C.W., Derbyshire, A.J. and Lee, A.W. (1966). A method of analysing individual cortical responses to auditory stimuli. Electroencephalography and Clinical Neurophysiology, 18, 204-206.

Papakostopoulos, D. (1977). Attention and neurophysiology. Some discrepancies. Paper presented at the Fifth Scientific Meeting of the Psychophysiology Group, Institute of Psychiatry, London.

Papakostopoulos, D., Cooper, R. and Crow, H.J. (1975). Inhibition of cortical evoked potentials and sensation by self-initiated movement in man. Nature, 258, 321-325.

Perry, N.W. and Childers, D.G. (1969). The Human Visual Evoked Response: Method and Theory. Thomas, Springfield.

Pfurtscheller, G. and Cooper, R. (1975). Selective averaging of the intracerebral click evoked responses in man: An improved method of measuring latencies and amplitudes. Electroencephalography and Clinical Neurophysiology, 38, 187-190.

Picton, T.W., Campbell, K.B., Barbibeau-Braun, J. and Proulx, G.B. (1977). Abstract of a paper entitled "The neurophysiology of human attention: A tutorial review" to be published in Attention and Performance VII. MRC human performance reports, List 22, Cambridge, p.30.

Picton, T.W. and Hillyard, S.A. (1974). Human auditory evoked potentials. II: Effects of attention. Electroencephalography and Clinical Neurophysiology, 36, 191-199.

Picton, T.W., Hillyard, S.A. and Galambos, R. (1976). Habituation and attention in the auditory system. In Keidel, W.D. and Neff, W.D. (Eds.), Handbook of Sensory Physiology V/3. Springer-Verlag, Berlin, 343-389.

Picton, T.W., Hillyard, S.A. Krausz, H.I. and Galambos, R. (1974). Human auditory evoked potentials. I: Evaluation of components. Electroencephalography and Clinical Neurophysiology, 36, 179-190.

Picton, T.W. and Hink, R.F. (1973). Evoked potentials: How? What? and Why? American Journal of EEG Technology, 14, 9-44.

Poon, L.W., Thompson, L.W. and Marsh, G.R. (1976). Average evoked potential changes as a function of processing complexity. Psychophysiology, 13, 43-49.

Posner, M.I. and Boies, S.J. (1971). Components of attention. Psychological Review, 78, 391-408.

Posner, M.I., Klein, R., Summers, J. and Buggie, S. (1973). On the selection of signals. Memory and Cognition, 1, 2-12.

Pribram, K.H. (1971). Languages of the Brain: Experimental Paradoxes and Principles in Neuropsychology. Prentice-Hall, New Jersey.

Pribram, K.H. and McGuiness, D. (1975). Arousal, activation, and effort in the control of attention. Psychological Review, 82, 116-149.

Regan, D. (1972). Evoked Potentials in Psychology, Sensory Physiology, and Clinical Medicine. Chapman and Hall, London.

Ritter, W., Vaughan, H. and Costa, L.D. (1968). Orienting and habituation to auditory stimuli: A study of short-term changes in average evoked responses. Electroencephalography and Clinical Neurophysiology, 25, 550-556.

Rosenblith, W.A. (Ed.). (1962). Processing Neuroelectric Data. MIT, Cambridge.

Roth, W.T. and Kopell, B.S. (1969). The auditory evoked response to repeated stimuli during a vigilance task. Psychophysiology, 6, 301-309.

Roth, W.T. and Kopell, B.S. (1973). P300 - an orienting reaction in the human auditory evoked response. Perceptual and Motor Skills, 36, 219-225.

Roth, W.T., Krainz, P.L., Ford, J.M., Tinkelberg, J.R., Rothbart, R.M. and Kopell, B.S. (1976). Parameters of temporal recovery of the human auditory evoked potential. Electroencephalography and Clinical Neurophysiology, 40, 623-632.

Rothman, H.H., Davis, H. and Hay, I.S. (1970). Slow evoked cortical potentials and temporal features of stimulation. Electroencephalography and Clinical Neurophysiology, 29, 225-232.

Ruchkin, D.S. and Sutton, S. (1973). Visual evoked and emitted potentials and stimulus significance. Bulletin of the Psychonomic Society, 2, 144-146.

Ruchkin, D.S. and Sutton, S. (1977). Emitted P300 potentials and temporal uncertainty. Unpublished manuscript.

Schartz, M. (1976). Average evoked responses and the encoding of perception. Psychophysiology, 13, 546-553.

Schwartz, M. and Shagass, C. (1964). Recovery functions of human somatosensory and visual evoked potentials. Annals of the New York Academy of Sciences, 112, 510-525.

Schwent, V.L. and Hillyard, S.A. (1975). Evoked potential correlates of selective attention with multi-channel auditory inputs. Electroencephalography and Clinical Neurophysiology, 38, 131-138.

Schwent, V.L., Hillyard, S.A. and Galambos, R. (1976a). Selective attention and the auditory vertex potential. I. Effects of stimulus delivery rate. Electroencephalography and Clinical Neurophysiology, 40, 604-614.

Schwent, V.L., Hillyard, S.A. and Galambos, R. (1976b). Selective attention and the auditory vertex potential. II. Effects of signal intensity and masking noise. Electroencephalography and Clinical Neurophysiology, 40, 615-622.

Schwent, V.L., Snyder, E. and Hillyard, S.A. (1976). Auditory evoked potentials during multichannel selective listening: Role of pitch

and localisation cues. Journal of Experimental Psychology: Human Performance and Perception, 3, 313-325.

Shepherd, G.M. (1974). The Synaptic Organisation of the Brain. Oxford University Press, New York.

Sherrington, C.S. (1906). The Integrative Action of the Nervous System. Yale University Press, New Haven.

Simpson, R., Vaughan, H.G. and Ritter, W. (1977). The scalp topography of potentials in auditory and visual discrimination tasks. Electroencephalography and Clinical Neurophysiology, 42, 528-535.

Smith, D.B.D., Donchin, E., Cohen, L. and Starr, A. (1970). Auditory averaged evoked potentials in man during selective binaural listening. Electroencephalography and Clinical Neurophysiology, 28, 146-152.

Smith, J.C., Marsh, J.T. and Brown, W.C. (1975). Far-field recorded frequency-following responses: Evidence for the locus of brainstem sources. Electroencephalography and Clinical Neurophysiology, 39, 465-472.

Smith, M.C. (1967). Theories of the psychological refractory period. Psychological Bulletin, 67, 202-213.

Snodgrass, J.G., Luce, R.D. and Galanter, E. (1967). Some experiments on simple and choice reaction time. Journal of Experimental Psychology, 75, 1-17.

Sohmer, H. and Pratt, H. (1977). Identification and separation of acoustic frequency-following responses (FFRs) in man. Electroencephalography and Clinical Neurophysiology, 42, 493-500.

Sokolov, E.N. (1975). The neuronal mechanisms of the orienting reflex. In Sokolov, E.N. and Vinogradava, O.S. (Eds.), Neuronal Mechanisms of the Orienting Reflex. Lawrence Erlbaum Associates, New Jersey, 217-235.

Soskis, D.A. and Shagass, C. (1974). Evoked potential tests of augmenting-reducing. Psychophysiology, 11, 175-190.

Spinelli, D.N. and Pribram, K.H. (1966). Changes in visual recovery functions produced by temporal lobe stimulation in monkeys. Electroencephalography and Clinical Neurophysiology, 20, 44-49.

Spinelli, D.N. and Pribram, K.H. (1967). Changes in visual recovery function and unit activity produced by frontal cortex stimulation. Electroencephalography and Clinical Neurophysiology, 22, 143-149.

Spong, P., Haider, M. and Lindsley, D.B. (1965). Selective attentiveness and cortical evoked responses to visual and auditory stimuli. Science, 148, 395-397.

Squires, K.C. and Donchin, E. (1976). Beyond averaging: The use of discriminant functions to recognise event related potentials elicited by single auditory stimuli. Electroencephalography and Clinical Neurophysiology, 41, 449-459.

Squires, K.C., Wickens, C., Squires, N.C. and Donchin, E. (1976). The effect of stimulus sequence on the waveform of the cortical event-related potential. Science, 193, 1142-1146.

Street, W.J., Perry, N.W. and Cunningham, W.R. (1976). A factor analysis of visual evoked responses. Psychophysiology, 13, 352-356.

Surwillo, W.W. (1977). Cortical evoked response recovery functions: Physiological manifestations of the psychological refractory period? Psychophysiology, 14, 32-39.

Suter, C.M. (1970). Principal components analysis of average evoked potentials. Experimental Neurology, 29, 317-329.

Sutton, S. (1969). The specification of psychological variables in average evoked potential experiments. In Donchin, E. and Lindsley, D.B. (Eds.), Average Evoked Potentials. NASA SP-191, Washington, 237-262.

Sutton, S., Braren, M., Zubin, J. and John, E.R. (1965). Evoked potential correlates of stimulus uncertainty. Science, 150, 1187-1188.

Szentagothai, J. and Arbib, M.A. (1975). Conceptual Models of Neural Organisation. MIT Press, Massachusetts.

Thompson, R.F. (1967). Foundations of Physiological Psychology. Harper and Row, New York.

Thompson, R.F. (1975). Introduction to Physiological Psychology. Harper and Row, New York.

Thompson, R.F. and Bettinger, L.A. (1970). Neural substrates of attention. In Mostofsky, D.I. (Ed.), Attention: Contemporary Theory and Analysis, Appleton-Century-Crofts, New York. 367-402.

Thompson, R.F. and Spencer, W.A. (1966). Habituation: A model phenomenon for the study of neuronal substrates of behaviour. Psychological Review, 73, 16-43.

Treisman, A. (1960). Contextual cues in selective listening. Quarterly Journal of Experimental Psychology, 12, 242-248.

Tueting, P. and Sutton, S. (1973). The relationship between prestimulus negative shifts and poststimulus components of the average evoked potential. In Kornblum, S. (Ed.), Attention and Performance IV. Academic Press, New York, 187-207.

Tueting, P., Sutton, B. and Zubin, J. (1971). Quantitative evoked potential correlates of the probability of events. Psychophysiology, 7, 385-394.

Turvey, M.T. (1973). On peripheral and central processes in vision: Inferences from an information processing analysis of masking with patterned stimuli. Psychological Review, 80, 1-52.

Vaughan, H.G. (1969). The relationship of brain activity to scalp recording of event-related potentials. In Donchin, E. and Lindsley, D.B. (Eds.), Average Evoked Potentials. NASA SP-191, Washington, 45-94.

Walley, R.E. and Weiden, T.D. (1973). Lateral inhibition and cognitive masking: A neuropsychological theory of attention. Psychological Review, 80, 284-302

Walter, D.O. (Ed.) (1976). Digital processing of bioelectrical phenomenon. Handbook of Electroencephalography and Clinical Neurophysiology, 4B. Elsevier, Amsterdam.

Walter, W.G. (1964). The convergence and interaction of visual, auditory, and tactile responses in the human nonspecific cortex. Annals of the New York Academy of Sciences, 112, 320-361.

Wastell, D.G. (1976). Evoked response correlates of reaction time: A single trial analysis. Paper prepsented at the Fourth Scientific Meeting of the Psychophysiology Group, Bedford College, London.

Wastell, D.G. (1977). Statistical detection of individual evoked responses: An evaluation of Woody's adaptive filter. Electroencephalography and Clinical Neurophysiology, 42, 835-839.

Weber, B.A. (1970). Habituation and dishabituation of the averaged auditory evoked response. Journal of Speech and Hearing Research, 13, 387-394.

Weinburg, H. and Cooper, R. (1972a). The use of correlational analysis for pattern recognition. Nature (London), 238, 292.

Weinburg, H. and Cooper, R. (1972b). The recognition index: A pattern recognition technique for noisy signals. Electroencephalography and Clinical Neurophysiology, 33, 608-613.

Weinburg, H., Walter, W.G. and Crow, H.J. (1970). Intracerebral events in humans related to real and imaginary stimuli. Electroencephalography and Clinical Neurophysiology, 29, 1-9.

Werner, G. (1970). The topology of body representation in the somatic afferent pathway. In Quarton, G.S., Melnechuk, T.M. and Schmitt, F.O. The Neurosciences, II. Rockefeller University Press, New York, 605-616.

Wever, E.G. (1949). Theory of Hearing. Wiley, New York.

White, C.T. (1969). Evoked cortical responses and patterned stimuli. American Psychologist, 24, 212-214.

Wickelgren, W.O. (1968). Effect of acoustic habituation on click-evoked responses in cats, Journal of Neurophysiology, 31, 777-785.

Wilkinson, R.T. (1967). Evoked response and reaction time. Acta Psychologica, 27, 235-245.

Wilkinson, R.T. and Ashby, S.M. (1974). Selective attention, contingent negative variation and the evoked potential. Biological Psychology, 1, 167-179.

Wilkinson, R.T., Morlock, H.C. and Williams, H.L. (1966). Evoked cortical responses during vigilance. Psychonomic Science, 4, 221-222.

Willows, D.M. and Mackinnon, G.E. (1973). Selective reading: Attention to unattended lines. Canadian Journal of Psychology, 27, 292-304.

Winer, B.J. (1971). Statistical Principles in Experimental Design, 2nd Ed. McGraw-Hill, Kogakusha, Tokyo.

Woody, C.D. (1967). Characterisation of an adaptive filter for the analysis of variable latency neuroelectric signals. Med. Biol. Engng., 5, 539-553.

Worden, F.G. (1966). Attention and auditory electrophysiology. In Stellar, E. and Sprague, J.M. (Eds.), Progress in Physiological Psychology, 1. Academic Press, London, 45-116.

Worden, F.G. and Marsh, J.T. (1968). Frequency-following (microphone-like) neural responses evoked by sound. Electroencephalography and Clinical Neurophysiology, 25, 42-52.