OPERANT CONDITIONING OF AUDITORY EVOKED POTENTIAL AMPLITUDE IN HUMANS: THE ROLE OF NON-ASSOCIATIVE AND MYOGENIC FACTORS

by

WILLIAM DAVID KOSNIK

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Supervisor: Dr. David R. Walker

ABSTRACT

The operant conditioning of human scalp recorded evoked potentials was investigated in the absence of electromyographic (EMG) artifact and non-associative factors. One subject was trained to decrease the N1-P2 amplitude of the auditory evoked potential and three subjects were trained to increase it by receiving contingent reinforcement for the production of criterion amplitudes. Specification of the criterion neural signal and reinforcement contingencies were under on-line control of a small laboratory computer. Non-contingent reinforcement was given in baseline and an extinction period was used to control for non-associative factors.

One subject significantly decreased the N1-P2 amplitude of the average evoked potential (AEP) as a result of the response-reinforcement contingency. Conditioning effects were specific to the criterion component and suggested functional independence of the components of the AEP.

Evoked potentials collaterally recorded from the contralateral hemisphere indicated that conditioning involved a change in evoked activity in both hemispheres. The mechanism of conditioning was related to an attentional gating mechanism that directed neural processing away from the

stimulus.

Reasons for the failure of the subjects in the Increase condition to learn were attributed to an ambiguous specification of the neural component being conditioned, a long delay in the reinforcement delivery and other factors.

Operant conditioning of neural events in humans as a result of the reinforcer-response contingency alone should be interpreted with caution until further research substantiates and expands the present findings.

Examining Committe[redacted]

Dr. David R. Walker

[redacted]

Dr. R. Leslie D. Wright

[redacted]

Dr. David Docherty

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REVIEW OF THE LITERATURE

Although the problems of brain research are as complex and formidable as any in science, the number of methods by which researchers have attacked these problems is relatively small, with each providing a unique approach to highly specialized problems. These methods include the disciplines of neuroanatomy, neuropsychology, neuropharmacology, and electrophysiology. An advantage of the electrophysiological study of the nervous system lies in the ability of the researcher to see with the help of electronic equipment the dynamic functioning of the nervous system in an intact organism, generally with a minimum of experimenter interference. Modern techniques such as permanently implanted electrodes and cannulae are optimally suited for the study of neural functioning in a normal, behaving animal.

The basic interest of the electrophysiologist concerns knowledge of the structure and function of the nervous system as it can be derived from the study of its electrical activity. Here, the emphasis is usually on function because electrophysiological techniques are suited to observing processes of individual neurons and populations of neurons.

By measuring the electrical activity of the nervous system, electrophysiologists have tried to formulate laws that govern its functioning. Walker (1974) has outlined

two areas in which laws have been sought. The first area is concerned with the way the nervous system encodes information about the environment and how this information is communicated to other regions of the nervous system. The second area is concerned with the formulation of laws governing the neural basis of behavior, that is, learning, perception, memory, attention, motivation, etc.

Traditionally, the method of study used to determine these laws has been the neural correlate approach. In this approach a recorded neural parameter [spike potential, slow wave (SW), evoked potential (EP)] is associated with either external events, such as sensory stimulation, molar behavior, a learning task, or internal events such as memory, attention, and arousal. It is then hoped that circumstances will prevail which permit the inference that the particular neural parameter in some way encodes or represents some aspect of that sensory stimulus, behavior, or experience. Impressive neurophysiological laws have been developed from the correlation of sensory stimulation with the responses of neurons at various levels of the sensory pathway.

One classical law of information coding of sensory stimulation is the frequency of occurrence code (Adrian, 1928; Perkel, 1970; Stein, 1967). This law states that the quantity of a stimulus is represented by a function of the number of neural impulses per unit time. Many different functions have been determined for specific types of sensory

stimulation at various levels of the sensory system. For example, it has been known for some time that some auditory nerve fibers fire in a linear fashion to tonal frequencies up to approximately 4000 cps (Davis, 1961; Evans, 1975). A power relationship exists between the degree of joint movement and spike frequency for certain cells in the ventro-basal complex of the thalamus (Mountcastle, Poggio & Werner, 1963). In the first order mechanoreceptors innervating the glabrous skin of the monkey's hand, intensity of skin indentation is encoded as a linear function of the number of impulses (LeMotte & Mountcastle, 1975; Mountcastle, Talbot, Darian-Smith & Kornhuber, 1967). A dominant pulse interval encodes the intensity of flutter vibration in first order mechanoreceptor afferents that innervate the glabrous skin of the monkey (Mountcastle et al., 1967). There is a direct relationship between the rate of firing in muscle spindle afferents and the degree of muscle tension (Matthews, 1964). The rate of firing in second order dorsal spinal-cerebellar tract neurons increases linearly with increasing stretch of the innervated muscle (Jansen & Walløe, 1970). The initial discharges of a cat retinal ganglion cell increases linearly in response to logarhythmic increases in light intensity (Creutzfeldt, 1970).

Thus the correlation of stimuli and neural activity has yielded impressive laws for transforming stimulus energy into neural codes. Other non-classical codes have

been proposed to fulfill this purpose as well (Perkel, 1970; Perkel & Bullock, 1968; Segundo & Perkel, 1969).

Another classical code first formulated by Müller (1838) is the doctrine of specific nerve energies, or as it was later termed, labelled-line coding (Mountcastle, 1967). The principle states that an essential aspect of the information about a sensation is embodied in the particular fibers which are active. Although seemingly trivial at the level of the peripheral nervous system (PNS; optic nerves always convey visual information; auditory nerves always convey auditory information), this principle has had profound influence on the way researchers have viewed and interpreted the complex functioning of the CNS.

The concept that unique areas of the brain subserve different functions is at least as old as Gall's "science" of Phrenology. This assumption survives today although in a more elaborate and complex form as the basis of modern neuropsychology. Workers in this field have correlated complex behavioral and sensory deficits with diverse areas of the brain that have been destroyed or impaired in an attempt to construct a model of the brain as a complex functional system of concertedly working zones (Luria, 1973).

Support for localizationist theory of brain function (i.e., labelled-line theory applied to the brain) has also come from numerous studies of the somato-sensory and motor

areas of the cortex. A topographical organization of the somatic afferents from the body skin surface has been mapped out on the cortical surface posterior to the Rolandic fissure; a similar topographical organization has been found to represent muscle groups and muscle movements in the cortical area anterior to the Rolandic fissure (Penfield & Rasmussen, 1950; Walsh, 1943; Werner, 1970; Woolsey, 1958).

In single cell studies of the visual cortex in cats, Hubel and Wiesel (1962, 1965, 1968) have proposed that the encoding of complex stimuli is built on a hierarchical model of cellular connections in the cortex and the optic pathway. Cells at lower levels of the structure signal with abrupt increases in firing rates the presence of simple features of the stimulus. This activity in turn impinges on higher order cells that respond to more complex features of the stimulus. Presumably, the anatomical connections of these cells determine their function. Such findings have been taken to mean that certain cells in the brain act as feature extractors, reliably reporting with high firing rates the occurrence of a specific event in the environment.

It must be pointed out, however, that the question of the degree of localized function at the cellular level even in so-called primary sensory and motor areas is still highly debated. For example, Woolsey, Travis, Barnard, and Ostinso (1953) have found that stimulation of the primary somatic sensory area of the cortex yielded muscle movements even

after ablation of all primary and supplementary motor cortex and elimination of U fiber interconnections between sensory and motor cortex. In contrast to Hubel and Wiesel's work, a number of other studies (Burns & Smith, 1962; Creutzfeldt, 1970; Doty, 1961; Gerstein & Kiang, 1964; Morrell, 1967; O'Brien & Fox, 1969; Spinelli & Weingarten, 1966) have stressed the unreliability and the unspecificity of responsiveness of single cells in the brain. John (1972) has stated, "Activity in a particular cell, per se, cannot be uniquely interpreted. Whether concerned with sensory report or motor control, the single cell is unreliable and ambiguous" (p. 853).

Studies that have supported the localizationist theory of sensory and motor function have also served to support the connectionist theory of learning and memory. If one ascribes to the notion that cells or brain regions perform specific functions, then learning might occur by the establishment of a new connection between one set of cells and another or one brain region and another. Practice would increase the excitability or decrease synaptic resistance along the new pathway; remembering would involve the discharge of those particular cells which constituted the new pathway.

By recording the electrical activity in a particular pathway or neural location during the acquisition and remembering process, one could presumably associate

differences in the activity of those cells with the learning and remembering process. This is precisely what the nueral correlate approach has attempted to do.

This approach was expected to bridge the gap between brain function and complex behaviors such as learning, memory, attention, language, motivation, and so forth. Yet a host of problems has plagued the neural correlate approach to behavior that were not in evidence when correlating stimulus variables with neural activity at the peripheral level.

One major problem is the inadequate knowledge of the behavior being measured. Often only one or a few endpoint responses during learning are measured, ignoring an indefinite set of collateral responses that may be undergoing conditioning at the same time (Fox, 1970). In a review of the neurophysiology of learning Thompson, Patterson, and Teyler (1972) noted little advancement in the past ten years. They stated, "In our opinion this dearth of data and paucity of substance is due mainly to the failure to utilize the necessary behavioral controls and well delineated behavioral paradigms. . . . The systematic investigation of brain activity accompanying learning necessitates the consistent use of behavioral systems in which the behavior can be accurately measured" (p. 85).

Another problem, the inadequate control of stimuli, has been noted with regard to visual EP correlates of

perception. In a review of the topic, MacKay and Jeffreys (1973) stated, "It would not perhaps be too much to say that nearly all the investigations to date could profitably be repeated now under more closely controlled conditions and with a more sophisticated choice of stimuli. . . . The outcome of these experiments has been valuable and encouraging; but their value has perhaps been greatest in the methodological lessons they have taught in retrospect" (p. 669). Fox (1970) has noted the inadequate knowledge of exact stimulus conditions in learning studies as well. In studies of perception and learning the response of each animal to a stimulus is unique, determined by the specific situation and by past experience. As a result, the aspects of the stimulus that are behaviorally relevant to the animal and responsible for the measured changes in electrical activity may be different for each animal. Knowledge of the relevant stimulus may remain hidden from the experimenter.

A third major difficulty is the identification of relevant neural signals to correlate with behavior in the midst of generalized brain activity (Fox, 1970; John & Killam, 1959; MacKay, 1970). In single cell studies of feature extractors in the visual cortex (Emerson & Gerstein, 1977a, 1977b; Hubel & Wiesel, 1962, 1965, 1968; Leventhal & Hirsch, 1975; Poggio, 1975) and single unit correlates of behavior (Adams, 1968; Mountcastle, Lynch, Geogopaulos,

Sakata & Auna, 1975) the maximal firing of a cell is taken as a response to the experimental manipulation. This practice is often criticized because cells in the brain are constantly firing spontaneously and because most cells in the brain fire to a variety of stimuli and experimental situations (Burns & Smith, 1962; Chalupa, Macadar & Lindsley, 1975; Doty, 1961; O'Brien & Fox, 1969).

The correlation of activity at a particular brain site with behavior cannot be assumed to be a unique representation of the behavior (Fox, 1970). Such correlations may be misleading because of the demonstrated plasticity of neurons throughout the brain to alter their firing patterns under a variety of situations (Ben-Ari & LaSalle, 1972; Chalupa et al., 1975; Segal & Olds, 1972; Sparks & Travis, 1968). Furthermore, the general conclusion reached in studies of neural correlates of learning is that both cortical and sub-cortical regions undergo a variety of changes throughout the conditioning process (John, Bartlett, Shimohacki & Klienman, 1973; John & Killam, 1959; John & Klienman, 1975; John & Morgades, 1969; Thompson et al., 1972). These findings support Fox's (1970) contention that, "Continuation of the acquisition processes may depend on the shifting of bioelectrical processes to new configurations of micro-behaviors that characterize new stages of behavioral acquisition" (p. 248).

Finally, the correlation of brain activity with behavior is difficult to reconcile in view of the large differences in time bases. Brain responses that are measured in terms of milliseconds are usually correlated with behaviors of much longer duration (Fox, 1970; Walker, 1974).

The disappointing conclusions regarding neural correlates of behavior have led to a new methodological approach to the brain-behavior problem (Fox, 1970; Fox & Rudell, 1968). This approach involves the study of the bioelectric signal itself. Since many aspects of the signal can be reliably measured (amplitude, frequency, discharge rate, latency, etc.), these neural parameters can be manipulated directly by making reinforcement contingent upon their occurrence.

The basic paradigm for the operant control of neural events (OCNE; Fox, 1970; Fox & Rudell, 1968) is to select a particular bioelectric event, determine its baseline probability of occurrence, and reinforce its occurrence during training. With reinforcement the probability of occurrence should increase. *It is assumed that a neural signal can be treated like any other behavioral operant of the organism only if it represents neural information in the nervous system.* This critical assumption raises the question of what is a relevant signal in the CNS?

A relevant signal that represents the activity of populations of neurons is the slow wave potential. Evidence indicates that the source of the slow wave is the summation of excitatory and inhibitory post-synaptic potentials of a neural population (Eccles, 1951; Purpura, 1959, 1967; Purpura & McMurty, 1965). Thus, SW parameters may give information about the moment-to-moment excitabilities of large neural populations which the unreliable spike potential cannot provide. However, information about cell discharge in the CNS can be derived from SW parameters. High correlations have been obtained between the probability of a unit discharge and the average evoked potential, that is, the frequency distribution of the firing of a cell over many stimulus presentations corresponded to the average waveform of the evoked potential (Fox & O'Brien, 1965; John & Morgades, 1969; Landeau, 1967). Similarly, high correlations were found between the probability of a single cell firing and the EEG amplitude (Creutzfeldt, Watanabe & Lux, 1966a, 1966b; Fox & Norman, 1968; Krekule & Walker, 1971). In these studies, both the SW and unit discharges were recorded from the same microelectrode. Furthermore, it has been asserted that if

a bioelectric signal can be manipulated under reinforcement control for adaptive purposes, then it must represent behavioral or neural information in the nervous system (Fox, 1970; Walker, 1974).

Many bioelectric signals have been brought under operant control. These include the rate of hypothalamic unit discharge (Olds, 1965), the rate of cortical unit discharge (Fetz & Finocchio, 1971), lateral geniculate spikes (Linnstaedter & Perachio, 1974), hippocampal theta activity (Black, 1971; Black, Young & Batenchuk, 1970), visual cortical unit activity (Shinkman, Bruce & Pfingst, 1974), the sensorimotor rhythm (Chase & Harper, 1971; Wyrwicha & Sterman, 1968), movement evoked cortical potentials (Rosenfeld & Fox, 1971, 1972a, 1972b), fast (Walker, 1974) and slow (Fox & Rudell, 1968, 1970; Rosenfeld & Hetzler, 1973; Rosenfeld, Hetzler, Birkel, Kowatch & Antoinetti, 1976; Rosenfeld & Owen, 1972; Rudell, 1970; Rudell & Fox, 1972), components of the sensory evoked potential-human alpha rhythms (Beatty, 1971, 1972; Beatty & Kornfeld, 1973; Brown, 1970, 1971; Hart, 1968; Hord & Berber, 1971; Kamiya, 1969, 1974; Lynch, Paskewitz & Orne, 1974; Martindale & Hines, 1975; Mulholland, 1973; Mulholland & Peper, 1971; Nowlis & Kamiya, 1970; Paskewitz & Orne, 1973; Peper, 1970; Peper & Mulholland, 1970; Plotkin & Cohen, 1976; Prewitt & Adams, 1976; Travis, Konko & Knott, 1974, 1975; Woodruff, 1975), human beta rhythms (Beatty, 1972; Beatty & Kornfeld, 1973;

Brown, 1971), human theta rhythms (Beatty, Greenberg, Piebler & O'Hanlon, 1974; Brown, 1971), human sensory-motor rhythms (Sterman, 1973; Sterman, Macdonald & Stone, 1974), and the human sensory evoked potential (Rosenfeld, Rudell & Fox, 1969). Summaries (Black, 1972; Fox, 1970) and extensive compilations of OCNE research have been published (Chase, 1974; DiCara, Barber, Kamiya, Miller, Shapiro & Stovya, 1974-1976).

In operant conditioning studies, either behavioral or neural, the critical feature lies in the demonstration that the animal has made the appropriate association or connection between the reinforcer and the response. The execution of the behavior is the experimenter's only indication that the animal has learned. The the experimenter is also obliged to show that the response would not have occurred or increased in frequency had it not been paired with the reinforcer. In this way naturally occurring changes in the response that may look like the animal has learned the association can be detected and ruled out.

Some non-associative factors operating during conditioning that could result in a change in the neural parameter are the passage of time or the mere introduction of the reinforcer. A number of procedures have been used to rule out these non-associative effects of conditioning. Either the animal is trained to demonstrate a high degree of discriminative control or other animals are used in

yoked control or non-contingent reinforcement control groups. In OCNE studies involving humans, the former is often used because the latter raises ethical questions and the extra subjects needed to serve as controls are often too costly in terms of time and expense.

Most conditioning studies of human scalp potentials have relied on bidirectional control of the neural parameter as a demonstration of the associative effects of conditioning. With this procedure both an increase and a decrease in the neural parameter are conditioned. Alpha conditioning studies have reported successfully conditioned decreases in alpha activity (Brown, 1970, 1971; Kamiya, 1969; Nowlis & Kamiya, 1970; Peper & Mulholland, 1970). Reported increases in alpha rhythm activity (Brown, 1970; Kamiya, 1969, 1974; Nowlis & Kamiya, 1970) have been attributed to changes in subjective mood states. However, Lynch and Paskewitz (1971) have attributed increases in alpha to non-associative factors such as: (a) a naturally rising baseline over time, or (b) the reduction of the initial novelty, anxiety, and arousal of the subjects during the course of the experiment. It is clear that the control of alpha activity as an operant learning phenomenon cannot be supported by bidirectional conditioning alone. Mechanisms operating during the increase sessions may be different from those operating in the decrease sessions.

Other methods of demonstrating learning of the alpha response are no feedback controls, non-contingent feedback controls and extinction periods. Studies that have employed these controls have yielded conflicting results. Lynch, Paskewitz, and Orne (1974) and Hart (1968) found alpha increases in non-contingent feedback groups while Beatty (1971, 1972) and Travis, Condo, and Knott (1974) found no alpha increases in both yoked and non-reinforced controls. Yet, the latter study and Kamiya (1969) found no extinction of the "learned" response. Based on these data, some workers (Lynch & Pasketitz, 1971; Lynch et al., 1974; Travis et al., 1974, 1975) have concluded that demonstrated control over alpha rhythms cannot be considered learning in the instrumental sense.

This is not to say that previously demonstrated control of alpha is of no value to the researcher. It does indeed seem that at least alpha blockage is readily controlled by the individual (Brown, 1970; Kamiya, 1969). Some hypotheses that attempted to account for this type of alpha control are: (a) attending to a stimulus or direct visual stimulation (Adrian & Matthews, 1934), and (b) activation of the oculomotor system (Mulholland, 1973; Mulholland & Peper, 1971; Peper, 1970; Peper & Mulholland, 1970). In addition, the disinhibition of attentional processes has been suggested as a possible mediator of alpha increases (Lynch et al., 1974; Paskewitz & Orne, 1973). Even though operant

learning per se has not been demonstrated, these studies have attempted to understand the function of alpha rhythms in terms of attentional processes and the oculomotor system.

With such refined control of alpha activity at the disposal of the subject, the contingency between the reinforcer (or feedback) and the alpha response may be superfluous. Some studies have noted very fast acquisition times (Brown, 1970; Nowlis & Kamiya, 1970). One study has shown alpha and beta control when subjects were given only instructional strategies (Beatty, 1972). Another type of learning, other than instrumental, may be responsible for such immediate results.

In order to demonstrate true operant conditioning of a human scalp potential it may be necessary to use a less easily controlled neural parameter. It is the primary purpose of the present study to investigate whether a human scalp potential can be brought under direct reinforcement control at the same time eliminating the possibility of non-associative mediators. By direct reinforcement control it is meant that the contingency between the neural parameter and the reinforcer is necessary for learning. The neural parameter selected in this experiment was the amplitude of a late component of the auditory evoked potential. An example of an auditory averaged evoked potential is shown in Appendix A. Presumably, such a signal evoked by a stimulus would have fewer neural (or non-neural) mecahnisms to mediate its occurrence, as opposed to the alpha rhythm which

has at least three semi-independent foci on the human cerebrum that might be related to three different bring processes (Lehman, 1971).

Only one other study has attempted conditioning of a component of a human evoked potential (Rosenfeld et al., 1969). In this study, 12 subjects were rewarded with money for generating an amplitude of an EP component 1 SD above the mean amplitude of the component obtained in baseline. This was alternated with blocks of trials to suppress the criterion amplitude. The researchers reported that the subjects were able to modestly but significantly increase the number of criterion responses in training over the total of combined baseline and suppression criterion responses. Since this study was not concerned with demonstrating the necessity of the contingency between the reinforcer and the response for learning, many methodological controls were omitted. No bidirectional conditioning was attempted, only an increase in the criterion response over baseline was conditioned. Non-contingent reinforcement was not employed during baseline to control for non-specific effects of the introduction of the reinforcer.

In order to demonstrate the necessity of the reinforcer-response contingency for learning, the following minimal controls must be implemented in the present experiment. Non-contingent reinforcement and pseudoconditioning were used during collection of baseline data. Both an increase

and a decrease in response amplitudes from baseline were required for conditioning. Finally, an extinction period was included to show extinction of the response with removal of reinforcement.

When using an evoking stimulus it may be possible for the subject to generate criterion responses by selectively moving toward or away from the stimulus. This would alter the stimulus energy and hence alter the amplitude of the sensory EP. In animal studies where a photic flash was used, it was found that selective orientation to the stimulus was neither a necessary (Fox, 1970; Rudell, 1970) nor preferred (Rosenfeld, Hetzler & Kosnik, 1974) method of obtaining reinforcement. However, since a positive correlation has been found between auditory stimulus intensity and the amplitude of the major components of the auditory AEP (Rapin, Schimmel, Tourk, Krasnegor & Pollack, 1966), subjects could alter the amplitude of the criterion component by selectively moving toward or away from the stimulus. To eliminate this source of non-associative mediation, auditory stimuli were delivered through a pair of headphones, maintaining constant stimulus energy directed to the receptor.

A major source of interference during conditioning is through myogenic (EMG) contamination of the EEG record. Interference related to muscle activity can be classified into two types. High frequency activity (30-200 Hz) results

from the spatial average of the action potentials of muscle groups. Common sources of EMG artifact picked up by an EEG electrode are contraction of neck and facial muscles, swallowing, eye blinks, eye movements, and movements of the jaw. When muscle groups are rhythmically activated the activity may be mistaken for beta activity (Lindsley & Wicke, 1974). Low frequency EMG activity arises from a change in the position of the recording electrode relative to some underlying tissue, such as field potentials produced by movement of the eyeball. Low frequency artifact can be recorded by EEG electrodes from some distance of the source via volume conduction.

The extent to which EMG related artifact either promotes or interferes with conditioning is rarely determined. In studies that conditioned beta activity (Beatty, 1971, 1972; Brown, 1971) no controls for possible EMG mediation were instituted.

Conditioning of slow wave EEG or low frequency components of the EP could also be mediated or contaminated by EMG activity. In the Rosenfeld et al. (1969) study, muscle artifact was not explicitly controlled. By running separate artifact trials after training it was asserted that subjects could not have obtained reinforcement through artifact alone.

Two items indicate, however, that myogenic artifact may have had a detrimental effect on conditioning. First, Rosenfeld reported that the increase in successful responses

was small, at best 14% greater than baseline. Second, average EP amplitude increases were not significant. It may be that controlling EMG artifact during the training period itself would enlarge conditioning effects and show corresponding changes in the neural component amplitude.

In the present experiment the following controls were used to minimize EMG contamination. A late (90-170 msec) component of the auditory EP was selected as the criterion response. Such a component is not affected by reflexive muscle activity induced by the auditory stimulus (Picton, Hillyard, Kraus & Galambos, 1974) as are the middle latency components (Bickford, Jacobson & Cody, 1964; Picton et al., 1974). All components, however, are affected by voluntary contractions of neck and facial muscles and by movements of the eyelid and eyeball (Picton et al., 1974). The former results in high frequency contamination. This artifact was minimized by instructing the subject to relax, providing a fixation point, and by recording the electro-oculogram (EOG), eliminating trials where artifact activity was excessive.

Slow potential shifts lasting many seconds can affect the absolute voltage level of the EEG. Slow potential shifts have many sources, among them are ocular movement artifact, arousal, trains of sensory stimuli, and movement of the electrode-scalp interface (Hillyard, 1974). To eliminate possible mediation of the criterion amplitude from these artifactual sources the difference between a

negative peak (N1) and a positive peak (P2) was selected as the criterion amplitude, rather than an absolute voltage level.

OCNE studies involving animals have tended to be more methodologically sound than human studies. In-depth recordings obviate EMG activity as an artifactual mediator. Successful conditioning employing yoked controls, non-contingent reinforcement, bidirectional conditioning, and extinction periods (see Black, 1972; and Fox, 1970 for reviews) lends optimism that the same can be accomplished in human studies.

The OCNE strategy has been applied to many brain-behavior problems. The early studies sought to correlate the conditioned neural event with behavior. It was reasoned that as the neural response was acquired the behavior or behaviors mediating the response would become evident. No behavioral correlates were found for the conditioned component of sensory evoked potentials (Fox & Rudell, 1968, 1970; Rosenfeld et al., 1969; Rudell, 1970; Rudell & Fox, 1972). Chase (1974) has attempted to correlate behavioral inhibition with sensory motor rhythm (SMR) conditioning, while others (Brown, 1970, 1971; Green, Green & Walters, 1970; Kamiya, 1969, 1974; Nowlis & Kamiya, 1970) have tried to correlate subjective moods with the generation of different frequencies of the human EEG. These studies are still subject to many of the pitfalls that were discussed with

regard to the neural correlate research. They have simply reversed the independent and dependent variables.

One way to assess the strength of these correlations is to use a dissociative paradigm. A neural event is reinforced when it occurs in the absence of the suspected mediator. If the neural event is elicited in the absence of the behavior (and vice versa) then the occurrence of one is not necessary for the occurrence of the other. This technique has been used to assess the relationship between cortical unit spikes and peripheral muscle contraction (Fetz, 1974; Fetz & Finocchio, 1971) and the relationship between lateral geniculate spikes and eye movements (Linnstaedter & Perachio, 1974).

The dissociative technique has application in the study of human scalp potentials. The strength of already established relationships between various cognitive and perceptual states and the EP or other slow wave activity could be assessed more vigorously. Such assessments could include the relationships between the contingent negative variation and expectation (Walter, Cooper, Aldridge, McCallum & Winter, 1964), EP correlates of hemispheric specialization for language (Davis & Wada, 1974; Morrell & Salamy, 1971), the amplitude of the N1-P2 component of the auditory EP and attention (Davis, 1964; Pictor & Hillyard, 1974), the P300 component of the sensory EP and information delivery (Sutton, Teuting, Zubin & John, 1966), and P300

and task related variables (Picton & Hillyard, 1974; Ritter & Vaughan, 1969; Squires, Squires & Hillyard, 1975).

Clinical applications, such as the operant control of EEG patterns to help reduce epileptic seizures are already being investigated (Hanley & Nirenberg, 1974; Sterman, 1973; Wyler, Lockard, Ward & Finch, 1976).

Another powerful application of the OCNE strategy is to study the interactions of different neural populations in the brain during the conditioning process. In this way the function of these neural populations, their interrelationships, as well as the delineation of their anatomical organization might be better understood. For example, in conditioning various early and late components of the visual EP in cats, Fox and Rudell (1968, 1970; Rudell & Fox, 1972) found that changes in the criterion component occurred in the absence of any changes in other components. This indicated that cortical components were statistically independent and that each component was probably generated by independent synaptic organizations.

Other investigations have been directed toward the mecahnisms of conditioning and the extent to which other brain regions are involved in conditioning. The effects of conditioning a given brain site on the activity at other cortical sites have been investigated by making simultaneous recordings at other brain sites during conditioning. Rosenfeld and Owen (1972) have asserted, ". . . knowledge of

degree of spread of conditioning effects may cast new light on mechanisms of the neural conditioning phenomenon" (p. 851).

In the first study of this kind, Rudell (1970) conditioned visual EPs in cats and found that changes in activity took place in a collaterally recorded cortical site 12 mm away from the trained cortical site. These changes corresponded to changes in the trained site. Rudell concluded, "The implication of these data is that the trained response represents a rather massive alteration in the cortex, such that these changes can be recorded from wide areas of the cortex" (p. 80). It was unclear, however, if the corresponding neural activity at collateral and trained sites was due to two different neural generators responding similarly or a single generator simultaneously recorded at two sites via volume conduction.

Rudell also recorded EPs from the lateral geniculate nucleus (LGN). Since changes in the conditioned neural component were not reflected by similar changes in the EP at the LGN, it was concluded that the mediation of the criterion response came from changes in the receptivity of cortical neurons or from extra-classical pathways. The visual input system was probably not responsible for changes in the neural component.

Rosenfeld and Owen's (1972) findings have supported Rudell's results and have also found corresponding changes

in EP activity in the hemisphere contralateral to the training site. Corresponding EP activity at trained and collateral sites was not apparent during baseline. That relationships between EPs in several brain sites appeared only in training suggested that training led to new processes which were not active in the naive animal during baseline. In other words, the cortical state or excitation that produced the conditioned neural changes was different from the cortical state that produced the criterion responses during baseline. Other work has supported this conclusion (Rosenfeld & Hetzler, 1973).

Finally, Rosenfeld and Owen were interested in determining if the generation of criterion responses was mediated by discrete behavioral or autonomic responses, "whose central efferent command signals of somato-sensory feedback signals might constitute the criterion response" (p. 85). Alternatively, mediation could have occurred through central processes in which a tonic cortical state, i.e., one existing in advance of the evoking stimulus, produced criterion responses. The stimulus would serve to set the timing for this state to produce the criterion response.

The behavioral mediation hypothesis was ruled out by using a random intertrial interval. It was reasoned that knowledge of when to make a discrete behavioral response could be gained from a regular intertrial interval. The proposition that changes in the criterion component of the

visual EP were mediated by central, not peripheral, events was confirmed when successful conditioning was obtained using a random intertrial interval. Other evidence has supported this hypothesis (Rosenfeld et al., 1976).

A secondary aim of the present experiment involved the investigation of possible mechanisms of conditioning the auditory EP. This was broken down into four smaller areas of inquiry.

First, the temporal specificity of conditioning was examined by comparing changes in the criterion component with changes in the earlier components of the EP. Statistical independence of individual human evoked potential components should exist since it has been shown in animal studies (Fox, 1970; Fox & Rudell, 1968, 1970; Rudell & Fox, 1972). These functional relationships of the human auditory evoked potential generators were investigated.

Secondly, EP activity at another electrode site in the contralateral hemisphere was recorded. The effects of the spread of conditioning to this site and the possible sources of the changes in activity at this site were investigated.

Third, in order to support the hypothesis that conditioned neural changes resulted from alterations of central neural activity, rather than from neural activity associated with a discrete peripheral behavior, a random intertrial interval was used.

Finally, it is possible that voluntary selective synchronization and desynchronization of the EEG could produce overall changes in amplitude that could mediate the amplitude changes of the criterion component of the EP. In order to determine if control of the EEG has mediated the amplitude changes in the criterion component, the peak-to-trough amplitude of the prestimulus EEG for each trial was collected during the experiment. Any changes in the criterion were compared with the prestimulus EEG for possible mediational effects.

METHOD

Subjects

Three males and one female took part in the study. The males were all undergraduate students at the University of Victoria. The female subject was a retired stenographer from the community. Because of the length (approximately 22 days) of the training task, only highly motivated subjects who expressed a great deal of interest in participating in a biofeedback experiment were used. Each of the subjects passed a screening procedure for electromyographic artifact and a well defined EP.

Apparatus

The experimental chamber consisted of two adjacent rooms. A small 12 x 12 in. clear plexiglass window was situated in the wall separating the two rooms. The room occupied by the subject was electrically shielded and dimly illuminated by light from the adjoining room. Equipment in the subject room included the electrode apparatus, 3 preamplifiers, passive filters, and shielded cables. A Kodak carousel slide projector fitted with a tachistoscopic lens shutter (Gerbrands) and power supplies for the preamplifiers were located in the anteroom.

A third room housed a DEC PDP 8/e computer. Peripheral devices interfaced to the computer included a disk drive

(Iomec series 3002) and controller (Iomec series 3800) with 3.33 million words of storage, an ASR-33 teletype, an oscilloscope (Tektronix D10), a voltage level comparator, eight analog to digital (A-D) converters, and two D-A converters.

Shielded recording cables were led from the output of the preamplifiers to the computer room and were input to the computer A-D and to an FM recording adapter (Vetters 4 channel) for recording on a tape recorder (Sony 4 channel). Output from the tape recorder played through the FM adapter and also served as input to the computer.

Other equipment in the computer room included a second tape recorder (Roberts) on which triggering pulses were taped, a storage oscilloscope (Tektronix D11) on which all biological signals were displayed during the experiment, a stimulus generator and an amplifier used to amplify the stimulus signal.

The stimulus was a 1000 Hz square wave pulse generated by a square wave oscillator. Stimulus duration was 50 msec.

For the males, reinforcement was both color and black and white slides of nude females and landscapes. Eighty slides were placed in mixed order in a carousel slide tray. For the female subject, reinforcement was 80 travelogue slides of India.

The electrode apparatus consisted of a cap fashioned out of nylon material. Glued onto the cap in an array corresponding to the 10-20 International electrode placement

system (Jasper, 1958) were hollow plastic cylinders, 1/2 in. in length and 3/8 in. in interior diameter oriented vertically to the cap surface. The electrodes were composed of hollow plastic cylinders 1-1/2 in. in length and 1/4 in. in interior diameter and were filled with an electrolytic gel.

Protruding slightly from one end of the cylinder was a small sponge that communicated between the electrolyte and the scalp. Glued onto the other end was a miniature electrode (Beckman 11 mm) with the sensing element in contact with the electrolyte. The sponge electrodes were fitted inside the cap cylinders and secured with a set screw. The advantages of this system are: (a) the same electrode locations can be reliably found day after day, (b) the electrolyte does not dry out, hence maintaining constant impedences of 1.5k-3.0k ohms throughout the experiment, and (c) the electrodes are not disturbed by head or scalp movements. This system helped to reduce a large part of the artifact associated with scalp recording. Other electrodes used were a pair of Beckman miniature skin electrodes and two gold disk electrodes linked to a common lead.

Procedure

Subject preparation. Each subject participated in the experiment individually. The subject was seated in the shielded room in a chair facing a wall approximately 4 ft. away on which the reinforcing slide was presented.

Subjective hearing threshold for a 1000 Hz tone was determined for each subject by the method of limits. Stimuli were presented 60-70 dB above subjective threshold. Hearing threshold for any subject did not differ by more than 5 dB between ears. At 60-70 dB the middle ear muscle reflex is not activated (Jepsen, 1963).

Stimuli were presented binaurally through a set of headphones. This provided the most equitable representation of the stimuli to both hemispheres since it has been shown that the amplitude of the auditory averaged evoked potential is positively biased in favor of input from the contralateral fibers (Ruhm, 1971).

For the three male subjects, sponge electrodes were placed at scalp locations C4 and C3 (Jasper, 1958), each referenced to linked gold disk electrodes attached to the earlobes. A sponge electrode at CZ served as ground. On the female subject, sponge electrodes were placed at CZ and C3, each referenced to O2. Linked disk electrodes attached to the earlobes served to ground the subject. CZ was used in place of C4 because of the presence of a predominant 9-14 Hz saw-toothed rhythm, described as the *en arceau* (Chatrian, Peterson & Lazarte, 1959; Ciganek, 1959), that partially obscured the EP at C4. A bipolar recording configuration (CZ-O2 and C3-O2) was used to test if differential recording of two active electrode sites would facilitate the subject's control over her EP.

The EOG was differentially recorded from two Beckman miniature electrodes placed above and at the outside corner of the right eye. This configuration was sensitive to eye movements, eye blinks, gross facial muscle contractions, swallowing, teeth gritting, and gross head movements.

Signal amplification and recording procedure. The EOG signal was amplified with a gain of 10k. Filter settings were 3 dB down at 10 Hz and 100 Hz. A flat frequency response was obtained between these two cut off frequencies. Cephalic signals were amplified 25k for subjects INC1, INC2, and INC3, and 50k for DEC1. Filter settings for all cephalic signals were 3 dB down at 1 Hz and 35 Hz.

Signals from the EOG preamplifier and the two EEG preamplifiers were conducted by shielded cable to the computer room where they were recorded on magnetic tape by first passing through the FM adapter. All biological signals were displayed on an oscilloscope for continual monitoring by the experimenter.

Control for contamination of the EEG by muscle activity was accomplished by analysis of the EOG record. While the experiment was in progress the EEG channel designated for conditioning was grounded whenever the (+ or -) amplitude of the EOG signal exceeded a prespecified threshold. Off-line, the same procedure was used during input of the non-conditioned EEG channel (C3).

The conditioning EEG signal was input to the computer for on-line analysis. The EEG was digitized at one sample every 2 msec. For each trial 512 msec of data were digitized. Each 512 msec epoch was stored in one 256 word buffer in the computer memory. For each trial 100 msec of data were digitized before stimulus onset. These data were called the prestimulus EEG activity.

Trials were initiated when the computer was triggered by a pretaped pulse from a tape recorder. Eight interpulse intervals of 2.4, 2.5, 2.7, 2.9, 3.1, 3.3, 3.5, and 3.7 sec randomly recorded on tape were used.

All experimental manipulations were under computer control. A specially written program was used to trigger the stimulus, input the EEG, make reinforcement decisions and identify each disk stored wave both for its reinforcement status and whether or not artifact was detected. The same procedure was used to input the other EEG channel off-line.

Identification of waves for artifact took place in two stages. On-line, the computer examined the EEG epoch between 120 and 220 msec for a grounded signal. If a grounded signal was detected, the wave was marked as an artifact trial. Since the above time interval corresponded to the latency of the criterion response the on-line detection procedure ensured that a response was never rewarded that might have been mediated by artifact. Off-line, the

computer re-examined all waves for the full 512 msec epoch for artifact. EPs in which artifact was detected were marked as such and omitted from analysis.

Selection of parameters for conditioning. Before participating in the experiment each subject went through a screening process to determine if his/her auditory EP was sufficiently well defined. A sample of 600 EPs were taken for each subject and averaged. The three criteria for acceptance in the study were: (a) the components P1, N1, P2, and N2 of the EP (Davis & Zerlin, 1966) had to be detectable in the AEP; (b) the component N1-P2 had to be detectable in at least 65% of the single sweeps; (c) no more than 30% of the trials could be contaminated by artifact. The first four subjects tested passed these criteria and subsequently participated in the experiment.

The neural event upon which reinforcement was contingent was the amplitude of the N1-P2 component of the auditory EP (Davis & Zerlin, 1966). The value of the amplitude of N1-P2 in each EP was computed in the following manner.

Each subject's average EP was used to determine the latency of the N1 and P2 peaks. Since the latency of these peaks occurs with some variability in single sweeps, the window for detecting N1 was expanded by calculating the midpoint of the segment formed by N1 and the preceding turning point on the wave and the midpoint of the segment

formed by N1 and the following turning point. The latencies of the two midpoints became the limits of the window for N1. The same method was used to calculate the window for P2. For any single sweep the value of the most negative peak in the first window was taken as the amplitude of N1 and the value of the most positive peak in the second window was taken as the amplitude of P2. The difference between N1 and P2 was calculated as the amplitude of the N1-P2 component. If either peak was not detected the computer registered no N1-P2 amplitude for the trial and marked the wave accordingly.

Conditioning: general. Each subject participated in a baseline, training and extinction session. The order in which subjects were run is DEC1, INC1, INC2, and INC3. The experience of the first subjects served to provide a basis for changing the treatment of later subjects in order to try to maximize the probability of conditioning. These changes are described in the procedure as they occurred.

Each subject was trained to either increase or decrease the amplitude of N1-P2, depending upon the experimental condition to which the subject was assigned. At first there were two subjects per condition. When neither subject in the Increase condition was successfully conditioned, subject INC3 was moved from the Decrease condition to the Increase condition in order to attempt to obtain successful conditioning in both directions.

Baseline. In order to determine which values of the amplitude responses were to be rewarded, amplitude histograms were compiled for each subject during the baseline period. Six hundred EPs per day were collected for two days from C4 and C3 for DEC1, INC1, and INC2; CZ and C3 for INC3. The last 900 artifact-free EPs from C4 (CZ for INC3) were compiled into amplitude histograms. In the Decrease condition criterion responses were defined as amplitudes below the 20th percentile of the total distribution. Criterion responses in the Increase condition were amplitudes above the 80th percentile for INC1 and INC2 and above the 50th percentile for INC3.

Thus, the a priori probability of the occurrence of a criterion response for subjects DEC1, INC1, and INC2 was 0.20 and 0.50 for INC3. INC3 was started on a high success rate so that she could be "shaped" into making responses of lower probability.

Data collection during baseline occurred under a pseudo-conditioning paradigm in which subjects thought they were obtaining practice in biofeedback by trying to manipulate a component other than the criterion component. Non-contingent reinforcement was delivered at levels determined by each subject's a priori probability of reinforcement.

Subjects were instructed to find a state of mind that would yield as many reinforcements as possible. They could expect to start out at a certain level of performance, then

they were to try to increase the number of rewards above that level. They were instructed to refrain from any unnecessary movements and to keep their eyes fixed on the crosshairs positioned at eye level on the wall.

Slides were flashed on the wall centered on the crosshairs 550 msec after a stimulus onset for 750 msec. The delay to reinforcement was calculated as the time between the last point in the P2 window and the reinforcement onset. For DEC1 the reinforcement delay was 224 msec and for INC1, INC2, and INC3 reinforcement delay was 240 msec. Subjects were allowed to scan the slide and blink while the reinforcer was on.

For DEC1's data analysis, 2006 artifact-free trials were collected over 4 days. For INC1, INC2, and INC3, 900 trials were collected over 2 days.

Training. Subjects were given the same instructions for the training period as baseline. However, they were told that a new component (N1-P2) was selected and that they should not develop any expectations based on their performance in baseline.

The training site, at which the reinforcement contingency operated, was C4 for DEC1, INC1, and INC2; CZ for INC3. For all subjects EPs from the contralateral hemisphere were recorded at C3.

Three blocks of 200 trials each were run per day. Short rest periods were given between blocks of trials. At

the end of each block subjects were informed of the percentage of reinforcements obtained. One daily session lasted about 1—1-1/2 hours.

Additional feedback was supplied to INC2. Three light-emitting diodes were located in a vertical column on the wall in front of the subject and were illuminated according to the response amplitude produced. If the N1-P2 amplitude was below the 40th percentile of INC2's baseline amplitude histogram, a red light was illuminated. If it fell between the 40th and 60th percentile, a yellow light was illuminated. A green light was illuminated when the response was between the 60th and 80th percentile. A slide was presented when a criterion response was made. If no N1-P2 component was detected or artifact occurred then no light was illuminated. Thus, the subject was aware of the approximate size of his response amplitude on every trial. It was thought that the more information the subject had about the behavior of his response, the more likely he would be able to learn to control it.

Reinforcement probability for INC3 was set at 50% at the outset of training. As soon as INC3 could demonstrate control at this reinforcement level stricter criterion levels were gradually introduced. This procedure was not instituted, however, since the criterion responses did not increase over 50%, but dropped instead. Criterion amplitudes were recalculated each day so that the reinforcement

did not drop appreciably below 50%. This procedure was designed to maintain a high level of motivation in the subject. The percentage of criterion responses were recalculated at the levels set at baseline for the data analysis.

Training lasted 12 days for DEC1 and INC3 and for 15 and 16 days for INC2 and INC1 respectively. Length of training depended on different factors for different subjects. DEC1's training was terminated when no further significant increase in rewards were obtained. Training for INC2 and INC1 was discontinued when it became apparent that no increase in rewards above baseline would be obtained and because of time commitments of the subjects. INC3's training had to be cut short because of an unexpected family matter that called her out of town.

Data that were used in the analysis were collected over the last 4 days for DEC1. Altogether, 1800 artifact-free EPs were collected. Nine hundred EPs were collected over 2 days for INC1, INC2, and INC3.

Extinction. Because of other commitments of the subjects, the extinction period was restricted to a few days. In order to facilitate the extinction of the learned response, three of the subjects (DEC1, INC1, and INC2) were run with the reinforcement contingency removed. The percentage of criterion responses continued to be recorded by the computer but no reinforcements were delivered. These subjects were instructed to sit quietly and listen to the tones. INC3,

however, was given non-contingent reinforcement during the extinction period, so that to her knowledge the experimental situation did not change from the training period to the extinction period.

Extinction lasted 3 days for DEC1, INC1, and INC2; 4 days for INC3, with 600 EPs collected per day. The last 900 artifact-free EPs of each subject were used in the data analysis.

Software. The following programs were used in the collection and analysis of the data:

1. Average. This program computes the average and standard deviation (SD) of a set of single EPs and stores the AEP and its SD in separate buffers. Options are available to selectively input waves based on parameters such as whether or not the EP met criterion.

2. DCP (Distribution of Criterion Potentials). This program marks waves and makes histograms using parameters specified by the operator. For example, EPs that have met criterion are so marked by DCP and then the amplitudes of those EPs are compiled into a histogram.

3. Debbie. This program controls the running of the experiment. It inputs data, triggers stimulus and reward mechanisms, makes reinforcement decisions, and marks waves according to their reinforcement and artifact status.

4. Display. This program displays the contents of two 256 word buffers simultaneously on the oscilloscope.

This is the equivalent of two EPs. The operator can make a variety of manipulations on the displayed waveforms. The contents of the two display buffers can be independently increased or decreased in gain. The buffers can be high or low pass filtered. They can be added together or subtracted. Each buffer can be integrated or differentiated. The contents of a set of buffers can be output over a phone line for storage in the university's IBM system 370 computer.

Waves and histograms were plotted on a Calcomp plotter.

RESULTS

Data Description

Figure 1 shows some illustrative tracings of single evoked potentials (traces a-e). Each tracing is 512 msec in duration. The amplitude is expressed in microvolts (μV) with negative going potentials producing an upward deflection. The auditory stimulus was presented at 100 msec, making the first 100 msec prestimulus EEG activity. An inspection of traces a-f in Figure 1 shows that there is considerable variability in the human EP. Reliable aspects of the EPs are enhanced by computing average evoked potentials, the result of which appears in trace f of Figure 1.

Several reliable components stand out in the AEP. These are the components P1, N1, P2, and N2 and are labelled on the AEPs. The peak latencies of these components for DEC1 are 36 msec, 114 msec, 202 msec, and 276 msec poststimulus time (PST), respectively. For INC2 the peak latencies for these four AEP components are 36, 92, 182, and 302 msec PST. The component peak latencies for INC1, whose AEPs are not shown in Figure 1, are 29, 94, 158, and 254 msec PST, respectively; for INC3 they are 45, 100, 186, and 338 msec, respectively. The AEP calculated from the first 600 EPs in baseline for each subject was used as a template to determine the latency windows in which the computer searched

FIGURE 1. Illustrative tracings of single EPs and AEPs for one subject each in the Increase (I) and Decrease (II) condition. Stimulus onset is at 100 msec (arrow). (Hits = criterion amplitudes; Misses = non-criterion amplitudes; No N1-P2 = EPs in which N1-P2 was not detected. Vertical lines mark the range of the N1 and P2 windows in the AEPs.)

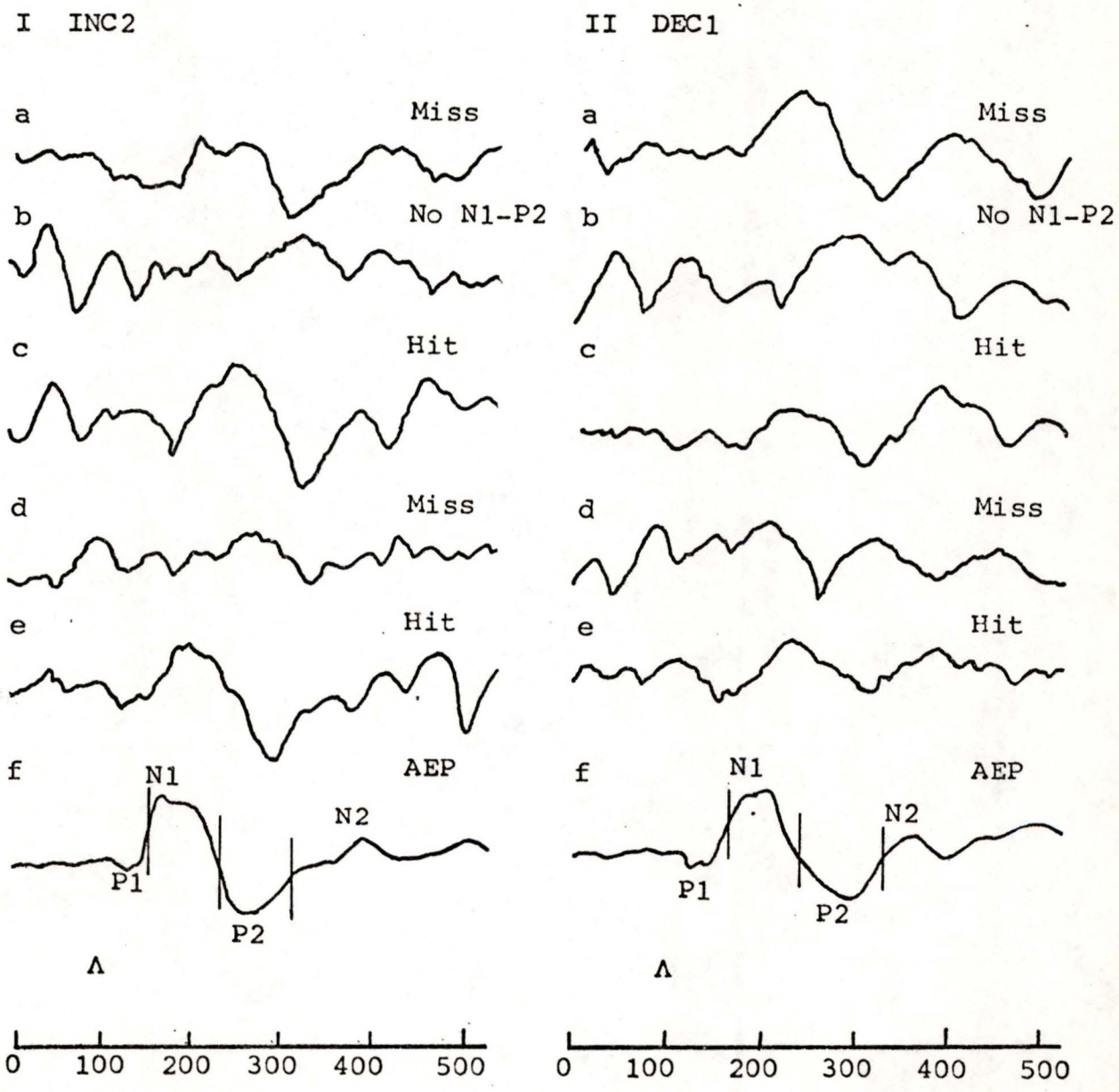
I INC2
II DEC1
a
Miss
b
No N1-P2
c
Hit
d
Miss
e
Hit
f
AEP
N1
P1
N2
P2
Λ
0
100
200
300
400
500
MSEC

FIGURE 1

for the N1 and P2 components in the single EPs. The latency windows are the segments between the three vertical lines in the AEPs of Figure 1 (traces If and IIf). The interval between the first two lines corresponds to the N1 window and the interval between the middle and third lines corresponds to the P2 window. The range of the N1 window was from 80-150 msec PST for DEC1, 80-126 msec PST for INC1, 74-144 msec PST for INC2, and 87-126 msec PST for INC3. The range of the P2 window was 150-226 msec PST for DEC1, 126-210 msec PST for INC1, 144-210 msec PST for INC2, and 126-210 msec PST for INC3.

In some single EPs the N1 or P2 components or both were not present in the latency windows as in Figure 1, Ib and IIb. If both components were detected by the computer the difference amplitude between them was calculated and checked to determine if it reached a criterion. Examples of criterion amplitudes in single EPs of the Increase condition are shown in Ic and Ie of Figure 1. Examples of criterion amplitudes from the Decrease condition are shown in IIc and IIe. Criterion amplitudes are hereafter referred to as *hits*. N1-P2 amplitudes that did not meet criterion are hereafter referred to as *misses*.

Histograms of 900 N1-P2 amplitudes from the baseline period recorded from the training site (C4 or CZ) were formed for each subject to establish the values for criterion amplitudes. Figure 2 illustrates these amplitude

FIGURE 2. Amplitude histograms of the N1-P2 component recorded from the trained hemisphere (C4-right, CZ-vertex) from baseline for each subject. Each histogram contains 900 trials. Cross-hatched are criterion amplitudes. The data point at amplitude 0 indicates the number of EPs in which N1-P2 was not detected.

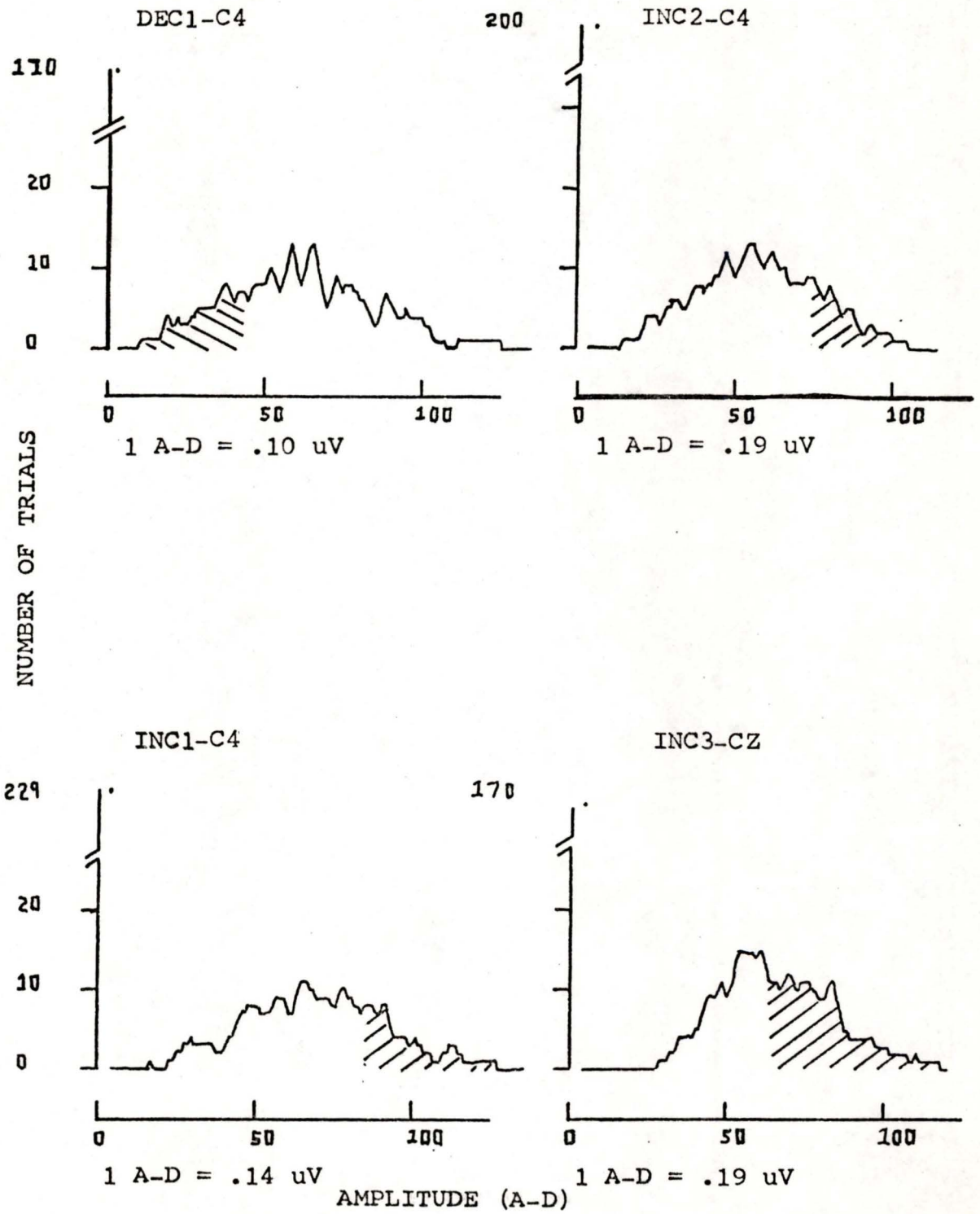
DEC1-C4
200
170
20
10
0
0
50
100
1 A-D = .10 uV
INC2-C4
0
50
100
1 A-D = .19 uV
NUMBER OF TRIALS
INC1-C4
229
170
20
10
0
0
50
100
1 A-D = .14 uV
INC3-CZ
0
50
100
1 A-D = .19 uV
AMPLITUDE (A-D)

FIGURE 2

histograms for the four subjects. The amplitudes were calculated in analog to digital conversion units that ranged from 0-255. The conversion from A-D units to μV are shown for each subject in Figure 2. Vertical lines separate hits from misses with the cross-hatched area denoting hits in each of the histograms. For DEC1 hits were defined as those values below the 20th percentile of the distribution. For INC1 and INC2 hits were defined as those values above the 80th percentile. For INC3 values above the 50th percentile were defined as hits.

In the histograms the data point at amplitude 0 represents the number of waves in which no N1-P2 amplitude was detected within 900 EPs collected. In all conditions the number of waves in which no N1-P2 amplitudes were found never exceeded 27% of the total number of EPs collected.

In Figure 3, histograms of prestimulus EEG amplitudes are displayed for all subjects. Each amplitude was calculated in A-D conversion units as the difference between the maximum and minimum points in the 100 msec prestimulus period of each epoch. The range of EEG amplitudes was 0-255 A-D units. The calibration for those A-D units are the same for the calibration for the A-D units of the N1-P2 amplitudes. Each histogram contains 900 EEG amplitudes. A vertical line in the distributions separates those amplitudes that were indicative of desynchronized activity (left side) from synchronized activity.

FIGURE 3. Amplitude histograms of the maximum peak to trough difference of the prestimulus EEG period for 900 trials recorded in baseline from the training site for each subject. The cross-hatched areas denote amplitudes associated with desynchronization.

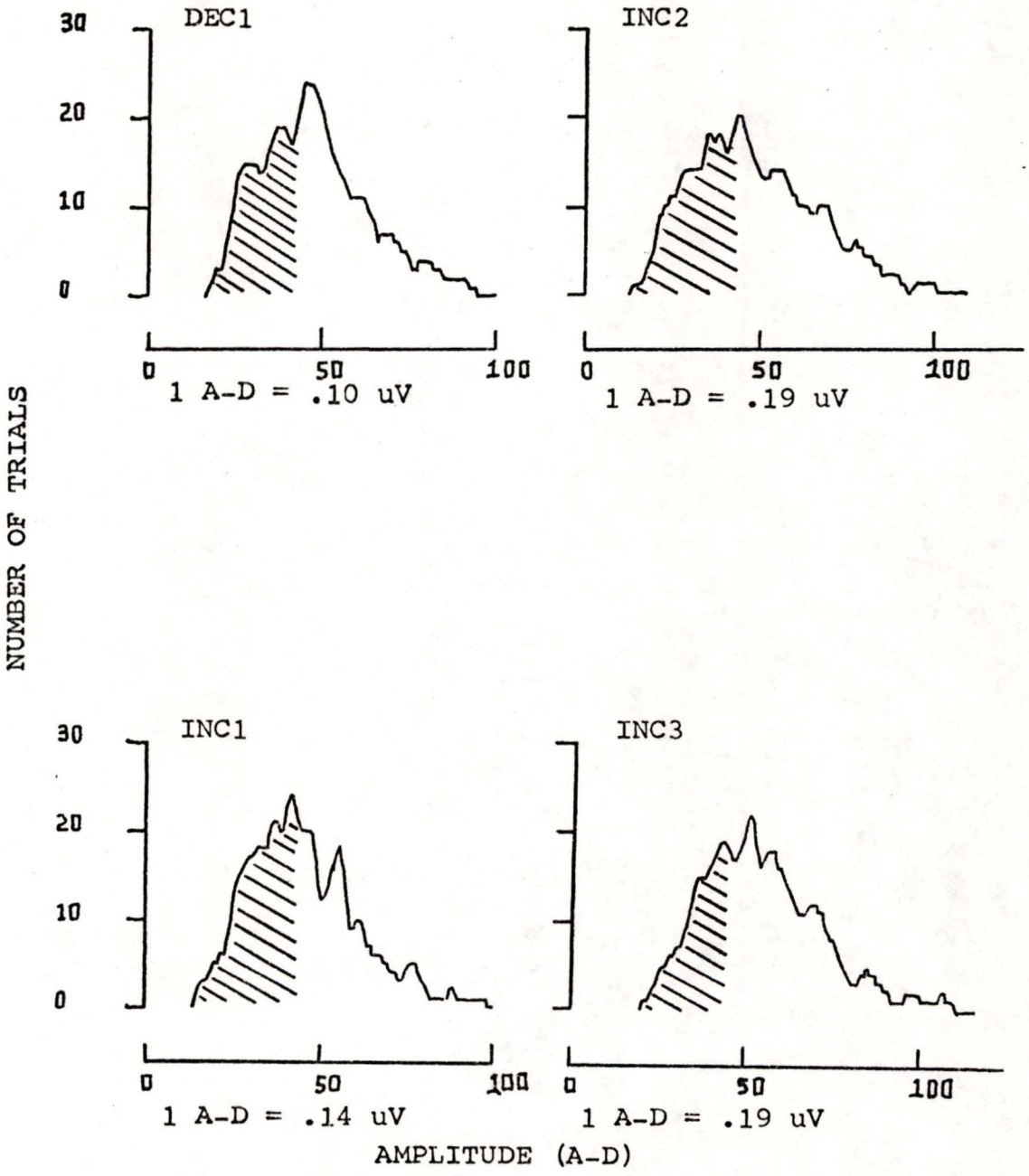
DEC1
INC2
INC1
INC3
30
20
10
0
50
100
1 A-D = .10 uV
1 A-D = .19 uV
1 A-D = .14 uV
1 A-D = .19 uV
NUMBER OF TRIALS
AMPLITUDE (A-D)

FIGURE 3

Data analysis. In order to maximize the probability of learning, the procedure varied somewhat between subjects. Thus, the data from each subject were treated as an individual experiment and analyzed separately.

Subjects were selected to be in the experiment based on the reliability of their evoked potential. This fact, together with the small number of subjects, does not make it possible to statistically generalize these results to the population.

Since many observations (trials) were taken from each subject, both within and across days, a test of the dependency between observations was needed. Dependency would be evident if hits tended to be followed by hits or, conversely, if hits and non-hits tended to alternate, or if the probability of a hit changed over repeated observations. Alternatively, the observations were said to be independent, at least for the purposes of this analysis, if none of the above situations existed.

The Wald-Wolfowitz Runs Test (Conover, 1971) was conducted to determine if the sequences of hits and non-hits could be considered random. The data were collected during the baseline period which lasted from 2-4 days. The results are shown in Table 1. For every subject the pattern of hits and non-hits in the baseline period was statistically not different from expectancy in sampling randomly from a binomial population. Thus, the probability of a hit on any

Table 1

Wald-Wolfowitz Runs Test of the Randomness

of Hits in the Baseline Period

Subject	Runs	Hits	Trials	*T*
DEC1	546	339	1998	456*
INC1	281	174	900	281*
INC2	256	167	900	265*
INC3	439	455	900	439*

*Not significant.

particular trial was the same for all trials and independent of other trials. Since independence of observations was found in the baseline period, it was assumed that observations taken during other phases of the experiment were also independent. This, of course, does not necessarily apply to the training period where the reinforcement contingency would presumably increase the probability of responses. Therefore, an analysis of variance design for independent groups was used for hypothesis testing. The baseline, training, and extinction periods were the levels of the independent variable and observations were treated as the subjects variable.

It was decided prior to the experiment that the test for learning would compare the number of hits obtained during the last 900 baseline and 900 extinction trials with the number of hits obtained in the last 900 training trials. Each set of trials was grouped into 9 blocks of 100 trials each. The percentage of hits was calculated for each block of 100 trials. Thus, 18 percentage scores from baseline and extinction were compared against 9 percentage scores from training. The total percentages of hits obtained by each subject under all phases of the experiment together with the results of the t-tests are reported in Table 2.

One subject, DEC1, significantly increased the percentage of hits in the training period [$t(25) = -4.5$, $p < .0005$, one-tailed test]. No significant differences between

Table 2

t Tests of the Mean Percentage of Criterion Responses (Hits) Averaged Over the Last Nine Blocks of 100 Trials in Each of the Three Treatment Conditions for Subjects Trained to Increase and Decrease the EP

	Percentage of Hits					
Subject	Baseline	Training	Extinction	B+E[a]	*df*	*t*
DEC1		24.5		17.9	25	-4.5***
	18.0		17.9		16	0.1
INC1		14.4		12.7	25	0.6
	19.3		6.0		16	7.8**
INC2		14.5		13.9	25	-0.3
	18.5		9.2		16	4.7**
INC3		43.7		45.3	25	0.5
	50.5		40.1		16	2.6*
No. of Trials	900	900	900	1800		

*$p < .02$

**$p < .001$

***$p < .0005$

[a]B+E is the percentage of hits averaged together from baseline and extinction.

training and the combined baseline and extinction periods were found for any subject in the Increase condition.

In order to test for any changes in the percentage of hits over time, that might be operating in addition to the reinforcement contingency, a comparison was made between the percentage of hits in baseline and extinction. For DEC1 the difference was not significant. The percentage of hits in baseline was significantly greater than the percentage of hits in extinction for all subjects in the Increase condition, $p < .02$, two-tailed test.

The results for DEC1 indicated that the percentage of hits increased significantly during training and then returned to baseline proportions during extinction. The results for subjects in the Increase condition indicated that not only were they unable to increase the percentage of hits above baseline but also that other factors were operating to decrease the percentage of hits over time.

The percentage of hits for each subject are graphed in Figure 4. Percentages are reported in blocks of 100 for the last 900 trials of baseline, training, and extinction. The graph for DEC1 illustrates a moderate increase in hits during training. This subject attained a 36.4% average increase in hits above baseline performance. Graphs of the other subjects' performances are characterized by a downward trend through all phases of the experiment.

FIGURE 4. The percentage of hits obtained by each subject over baseline, training, and extinction. Each point represents the percentage of hits in the last 900 trials of each treatment period.

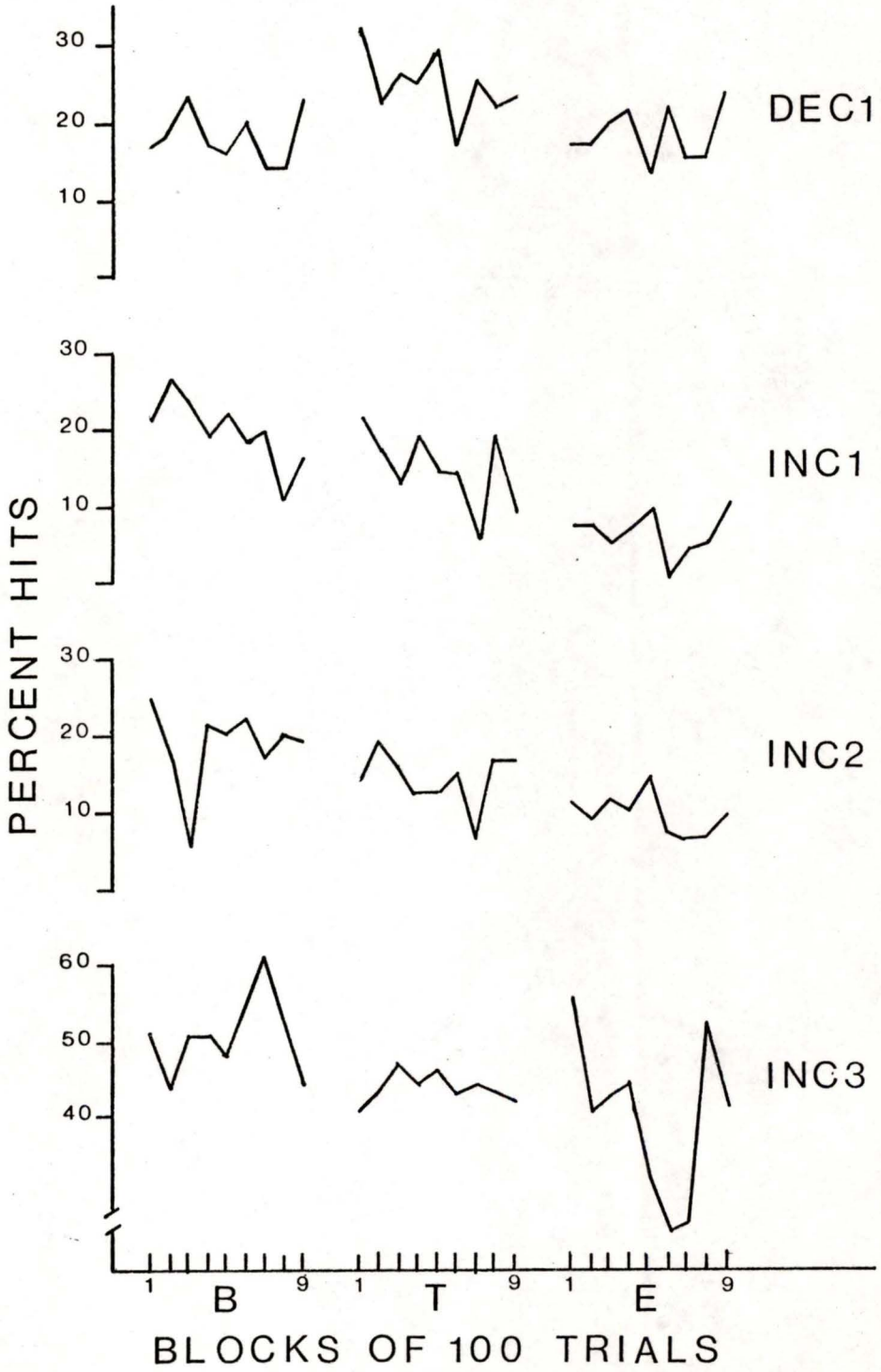
30
20
10
DEC1
30
20
10
INC1
30
20
10
INC2
60
50
40
INC3
PERCENT HITS
1
B
9
1
T
9
1
E
9
BLOCKS OF 100 TRIALS

FIGURE 4

Amplitude analysis. Histograms of N1-P2 amplitudes at the two recording sites, C4 (or CZ) and C3 for each experimental period were computed for each subject. Table 3 shows the mean amplitudes for the N1-P2 amplitude histograms. Reinforcement decisions were based on these amplitudes.

Table 4 shows the mean N1-P2 amplitudes of the AEPs for each experimental period at the two recording sites for each subject. The mean N1-P2 amplitude was calculated as the difference between N1 and P2 measured from the AEP.

In all but one subject (INC2) the direction of amplitude changes across all experimental periods was consistent for the two methods of calculation. For INC2 the three cases in which the direction of amplitude changes did not match up were from baseline to training at C3 and from training to extinction at C4 and C3.

Mean amplitudes calculated from the histograms are generally twice as large as the mean amplitudes calculated from the AEPs. The reason for this is that the histogram amplitudes were obtained by selecting the maximum and minimum points on each EP within the N1 and P2 windows whereas the amplitudes of N1 and P2 in the AEP are determined by averaging EP amplitudes at a particular point in time.

Since the reinforcement contingency was formally operating on the histogram N1-P2 amplitudes at C4 (or CZ) changes in amplitude due to the treatment effects were assessed by analysis of these amplitudes.

Table 3

Mean Amplitudes of the N1-P2 Component in

Microvolts (μV) Calculated from the Histograms

Subject	Site[a]	Mean Amplitudes (μV)			
		Baseline	Training	Extinction	B+E[b]
DEC1	C4	24.6	21.3	24.2	
	C3	26.5	20.7	24.0	
INC1	C4	40.3	32.2	31.0	35.6
	C3	47.2	38.4	35.4	41.4
INC2	C4	46.2	42.8	39.7	43.0
	C3	51.2	47.1	41.1	46.1
INC3	CZ	51.1	47.1	45.5	48.3
	C3	54.4	50.2	52.1	53.3

[a]Electrode site: C4 (right hemisphere) = trained site; C3 (left hemisphere) = collateral site; CZ (Vertex) = trained site.

[b]B+E = average of the combined baseline and extinction periods.

Table 4

Mean N1-P2 Amplitudes in Microvolts (μV) Calculated as the Difference between N1 and P2 of the AEP

Subject	Site[a]	Baseline		Training		Extinction	
		$\bar{X}$	N	$\bar{X}$	N	$\bar{X}$	N
DEC1	C4	11.9	1621	8.6	1362	12.1	656
	C3	10.5	1687	6.8	1410	10.7	680
INC1	C4	23.5	671	17.9	686	14.1	676
	C3	27.7	721	20.4	691	16.1	693
INC2	C4	18.5	700	17.4	665	19.7	684
	C3	15.8	720	17.6	728	18.6	746
INC3	CZ	24.4	735	23.5	717	22.9	741
	C3	24.9	770	23.0	757	23.4	760

[a]Electrode site: C4 (right hemisphere) = trained site; C3 (left hemisphere) = collateral site; CZ (vertex) = trained site.

Graphs of the changes in the mean amplitudes of the histograms over baseline, training, and extinction from C4 (or CZ) and C3 are shown for each subject in Figure 5.

For DEC1 there was a decrease in the mean N1-P2 amplitude of 3.3 μV from baseline to training [$t(2981) = 8.9$, $p < .0005$, one-tailed test]. The difference between the mean baseline and extinction amplitudes of 0.5 μV was not significant.

From Figure 5 it is evident that the mean amplitudes decreased throughout the experiment for all subjects in the Increase condition. This trend is opposite to the direction of amplitude changes predicted for this group. It is possible, however, that a continually declining baseline was operating and that training served to interrupt the rate of this decline. Therefore, the combined baseline and extinction mean amplitude was compared to the mean amplitude in training. The training amplitude was not significantly greater than the non-training amplitude for any of the subjects.

Since planned comparisons were not significant, post hoc comparisons were made to test for other differences between means. An overall F-test indicated significant differences between mean N1-P2 amplitudes for each subject, [INC1: $F(2, 2030) = 92.6$, $p < .001$; INC2: $F(2, 2046) = 26.2$, $p < .001$; INC3: $F(2, 2190) = 24.4$, $p < .001$]. Comparisons between treatment means were assessed using a Tukey test for

FIGURE 5. The change in the mean N1-P2 amplitude in microvolts from the amplitude histograms over baseline, training, and extinction for both the trained hemisphere (C4-right or CZ-vertex) and the contralateral hemisphere (C3-left) for each subject.

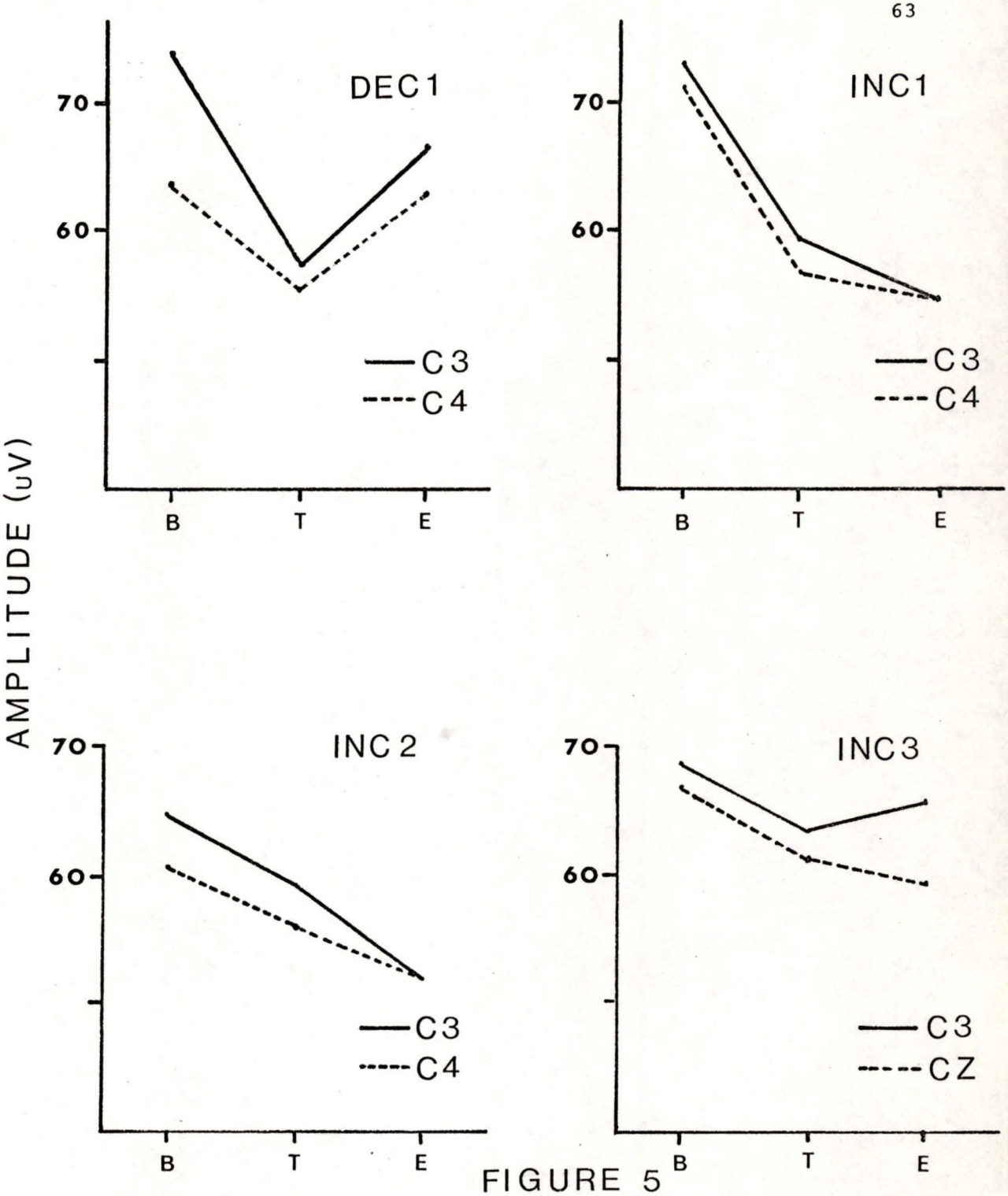

FIGURE 5

unequal Ns to control for the experimentwise error rate. There was a significant decrease in mean amplitude from baseline to traiing for all three Increase subjects at the $p < .01$ level of significance. For INC2 there was a significant decrease in amplitude from training to extinction, $p < .01$. For INC2 and INC3 the decrease from training to extinction was not significant ($p > .05$).

In summary, a general decrease in histogram mean amplitude was noted for all subjects from baseline to training. For INC2 this decrease continued into extinction while for INC1 and INC3 it stayed the same as training. For DEC1 the mean amplitude in extinction was the same as baseline.

Tracings of the AEPs from the last 900 EPs of each of the three experimental periods are shown in Figure 6 for each subject. The AEPs of baseline training and extinction are superimposed for each subject. The changes in the AEPs generally reflect the changes described in the statistical analysis. For DEC1 the N1-P2 amplitude decreased during training to 72% of baseline. During extinction the amplitude returned to baseline proportions.

N1-P2 amplitudes of the other subjects also decreased from baseline to training. The AEP N1-P2 amplitude of INC1 during training was 79% of baseline. Smaller decreases were noted for the other two subjects. The training amplitude was 94% and 96% of baseline for INC2 and INC3 respectively.

FIGURE 6. Superimposed AEPs from baseline (B), training (T), and extinction (E) for each subject from the training site (C4-right or CZ-vertex). Components are labelled. Each tracing is 512 msec. Stimulus onset is at 100 msec (arrow). Negative is up.

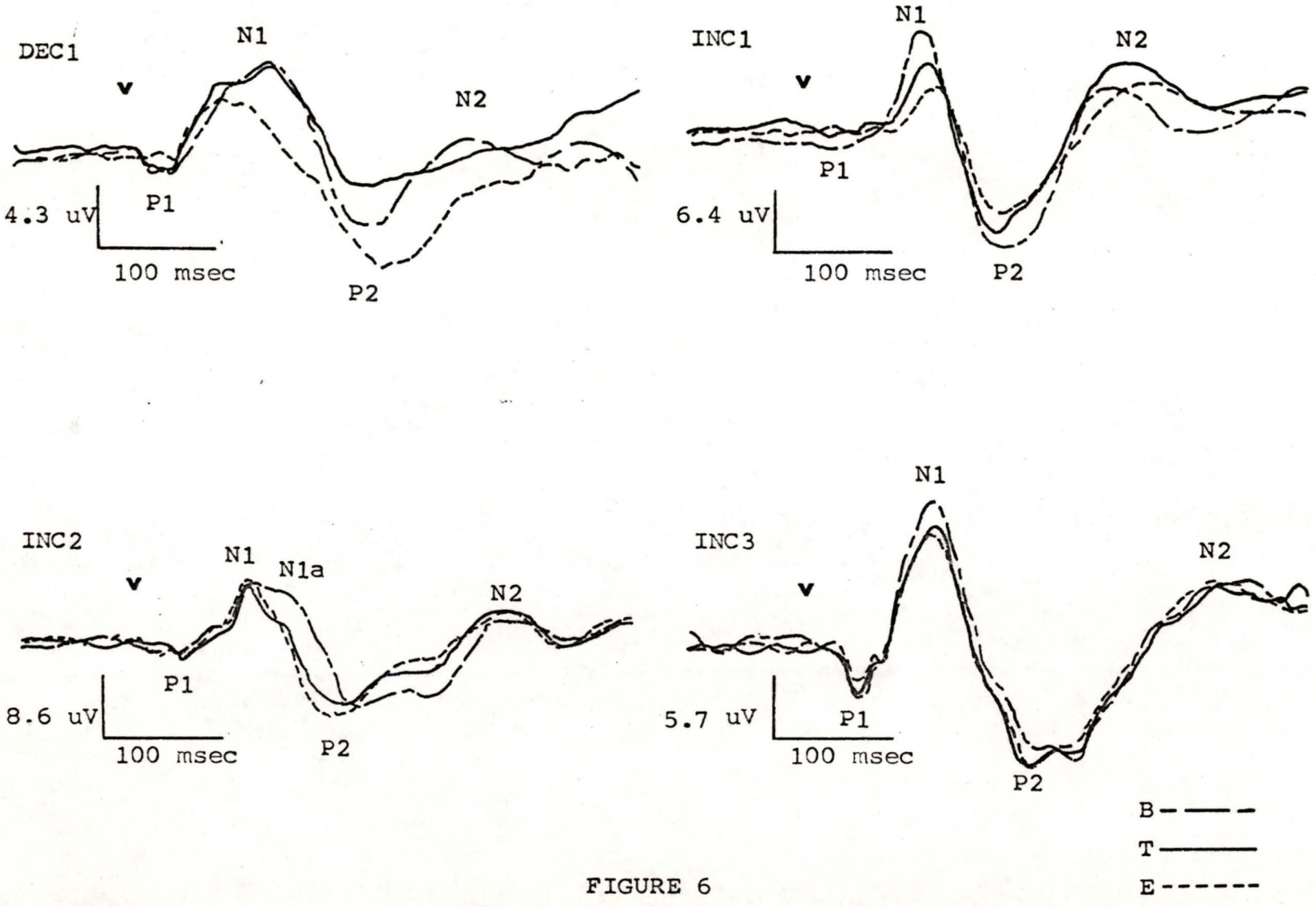
DEC1
N1
N2
P1
4.3 uV
100 msec
P2
INC1
N1
N2
P1
6.4 uV
100 msec
P2
INC2
N1
N1a
N2
P1
8.6 uV
100 msec
P2
INC3
N1
N2
P1
5.7 uV
100 msec
P2
B
T
E

FIGURE 6

Comparison of the N1-P2 amplitude histograms and the AEP N1-P2 amplitudes. For INC3 there was a slight decrease in amplitude from training to extinction. This decrease was not significant in the N1-P2 amplitude histogram analysis. A larger decrease between training and extinction was noted for INC2, but this decrease was not significant in the N1-P2 amplitude histogram analysis. For INC2 the AEP amplitude increased from training to extinction. This is in contrast to the significant decrease noted in the N1-P2 amplitude histograms for these two periods.

This apparent contradiction can be reconciled by noting the component labelled N1a in the baseline AEP in Figure 6. This component is reduced in training and essentially disappears during extinction. This component has a peak latency of 125 msec PST which is within the time window in which the computer looked for N1. Therefore, N1a had an influence on the distribution of N1-P2 amplitudes whenever its amplitude exceeded that of N1, which was localized earlier in the window. The fact that there were actually two components in the window became evident when N1a dropped out of the AEP during training and extinction. Up to this point, this segment of the AEP was considered one component. When N1a dropped out of the AEP in training and extinction it caused a reduction of the mean of the amplitude histogram without affecting the N1-P2 amplitude of the AEP. This explains the difference in the two measures of N1-P2 in

INC2's extinction period.

Although all subjects exhibited a decrease in their N1-P2 amplitudes from baseline to training, the amplitude changes from training to extinction were unique for DEC1 because his amplitude at extinction returned to baseline while for the others it continued to decrease or stayed the same as training. For this reason it is possible that these changes might be considered learned while for the others they may be due to unlearned factors. Therefore an analysis of the possible mechanisms of conditioning was undertaken in order to look for additional evidence for the hypothesized conditioning effects for INC2.

EEG analysis. Changes in EP amplitude during the experiment may have been a result of an overall change in the EEG amplitude. The possibility that conditioned changes in DEC1's AEP amplitude may have been a result of a reduction in EEG amplitude, such as desynchronization, was investigated by examining the EEG amplitude histograms during baseline, training, and extinction.

The means and SDs of the EEG histograms for all treatment conditions and for all subjects are listed in Table 5. Overall *F*-tests indicated significant differences between the means across the experimental periods for each subject. The results are reported in Table 6. Tukey tests were used to assess differences between means. For one subject, INC3, there was a significant decrease in mean EEG amplitude from

Table 5

Means and Standard Deviations (SD) of the EEG Amplitude Distributions for Each Treatment Condition Recorded from C4*

Subject	Treatment Condition	$\bar{X}$	SD	N
DEC1	Baseline	18.3	6.9	2006
	Training	18.4	6.1	1800
	Extinction	20.6	7.7	900
INC1	Baseline	27.4	11.3	900
	Training	26.7	11.4	900
	Extinction	24.4	10.4	900
INC2	Baseline	38.6	16.3	900
	Training	38.2	16.3	900
	Extinction	35.0	13.1	900
INC3	Baseline	43.2	15.3	900
	Training	38.8	12.5	900
	Extinction	37.2	12.6	900

*CZ for INC3.

Table 6

F Tables of the Analysis of the EEG Across Baseline, Training and Extinction for Each Subject

Subject	Source	*df*	MS	*F*-Ratio
DEC1	Between	2	22450.89	50.31**
	Within	4703	313.27	
INC1	Between	2	6527.96	17.35*
	Within	2697	376.20	
INC2	Between	2	6122.07	15.87*
	Within	2697	385.68	
INC3	Between	2	15043.14	48.02**
	Within	2697	313.24	

*$p < .001$

**$p < .0005$

baseline to training, $p < .01$. For DEC1, INC1 and INC2 there was no significant difference between baseline and training mean EEG amplitudes. From training to extinction the mean EEG amplitude significantly decreased for INC1, INC2 ($p < .01$), and INC3 ($p < .025$). For DEC1 the mean EEG amplitude significantly increased from training to extinction ($p < .01$).

For three subjects, DEC1, INC1, and INC2, large decreases in mean N1-P2 amplitude between baseline and training were associated with no differences between mean EEG amplitudes. For INC3, however, a decrease in mean N1-P2 amplitude was associated with a similar decrease in mean EEG amplitude from baseline to training.

EEG histograms from baseline and training are presented for each subject in Figure 7. These histograms have been digitally smoothed for the purpose of clarity. For DEC1, INC1 and INC2 the distributions are very similar. For INC3, however, there are many more amplitudes in training below the mode of baseline.

For DEC1 from training to extinction the increase in the mean EEG amplitude paralleled the increase in the N1-P2 mean amplitude. For INC2 both the mean EEG and the N1-P2 amplitude decreased from training to extinction. For INC1 and INC3 there were no significant differences in either the mean EEG amplitudes or the mean N1-P2 amplitudes from training to extinction.

FIGURE 7. EEG amplitude histograms from baseline (B) and training (T) for each subject.

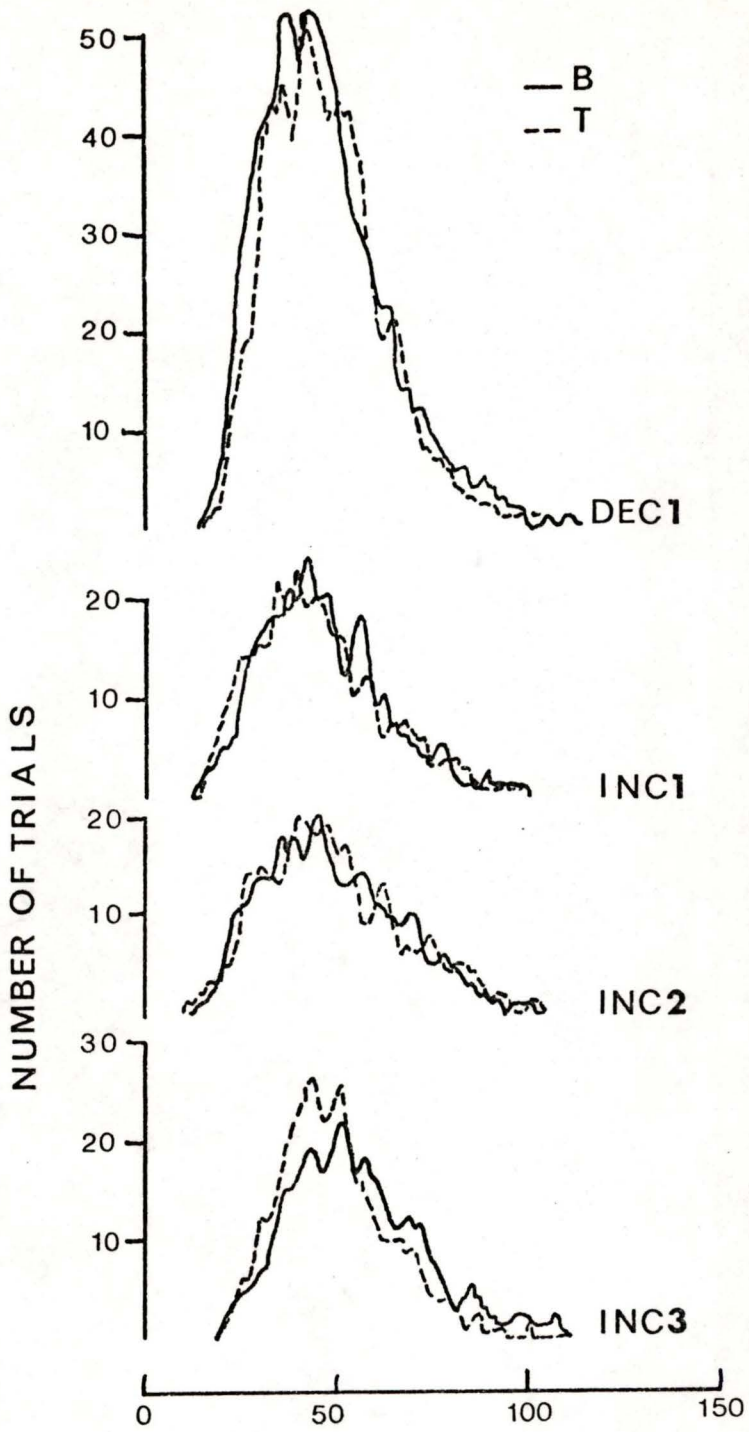
50
40
30
20
10
B
T
DEC1
20
10
INC1
20
10
INC2
30
20
10
INC3
NUMBER OF TRIALS
0
50
100
150
AMPLITUDE (A-D)

FIGURE 7

The relationship between the EEG and the N1-P2 amplitude was inconsistent. In INC3's case the changes in the EEG paralleled the changes in the N1-P2 amplitude throughout the experiment. EEG changes paralleled N1-P2 amplitude changes in two of the remaining three subjects only from training to extinction. Therefore, this relationship was further investigated by analyzing the state of the EEG when a hit was obtained. For DEC1, if the EEG was being manipulated to obtain hits, one would expect a greater proportion of hits when the EEG was desynchronized, i.e., low amplitude EEG, compared to the total proportion of hits. Similarly, for subjects in the Increase condition one would expect more hits when the EEG was synchronized compared to the total proportion of hits.

The proportion of hits under desynchronization for DEC1 and under synchronization for INC1, INC2, and INC3 are reported in Table 7 together with the total proportion of hits obtained under each treatment condition. First, in order to ascertain if such a relationship exists in the non-conditioned subject, a Z-test for two sample proprotions was conducted on the baseline and extinction data.

For DEC1 the proportion of hits during desynchronization was compared against the total proportion of hits. In neither baseline nor extinction was there any significant difference between the two portions, $p > .05$. For each subject in the Increase condition the proportion of hits

Table 7

Relationship of the Proportion of Hits During EEG Desynchronization for DEC1 and During EEG Synchronization for INC1, INC2 and INC3 to the Total Proportion of Hits for Each Subject Under Each Treatment Condition

Subject	Baseline		Training		Extinction	
	H/T	DH/DT	H/T	DH/DT	H/T	DH/DT
DEC1	.190	.190	.238	.262	.169	.192
	H/T	SH/ST	H/T	SH/ST	H/T	SH/ST
INC1	.193	.208	.111	.147	.060	.091
INC2	.186	.225	.132	.165	.092	.101
INC3	.506	.514	.447	.447	.401	.449

Note. Abbreviations: H = Hits; T = Total; DH = number of hits when the EEG was desynchronized; DT = total of desynchronized trials; SH = synchronized hits; ST = synchronized total.

during synchronization was compared to the total proportion of hits in baseline and extinction. There was no significant difference between the two proportions for any of the subjects in either baseline or extinction, $p > .05$. Thus, the state of the EEG (either synchronization or desynchronization) was not associated with the occurrence of a hit in the non-training periods for any subjects.

It was then determined if subjects manipulated EEG amplitude in order to obtain hits during the training period. No differences were found between proportions for any of the subjects.

Changes in activity in the contralateral hemisphere. The means of the N1-P2 amplitudes at C3 calculated from the amplitude histograms and the AEPs are shown in Tables 3 and 4, respectively.

For DEC1 the mean N1-P2 amplitude histogram significantly decreased by 5.8 μV from baseline to training [$t(3095) = 15.5$, $p < .000$, one-tailed test]. The baseline mean amplitude was also significantly greater than extinction [$t(2365) = 5.5$, $p < .000$, one-tailed test]. A post hoc comparison between the mean amplitudes of training and extinction using a Tukey test yielded a significant increase of 3.3 μV, $p < .01$.

Since subjects in the Increase condition did not successfully complete the task, post hoc comparisons were used to test differences between means. Overall *F*-tests are

shown in Table 8. These tests indicated significant differences between the treatment means for each subject. The Tukey test for multiple comparisons was used to correct for the increased experimentwise error rate.

There was a significant decrease in the mean N1-P2 amplitude from baseline to training for INC1, INC2 and INC3, $p < .01$. For INC1 and INC2 there was a significant decrease in the mean N1-P2 amplitude from training to extinction, $p < .01$. For INC3 there was a significant increase in the mean N1-P2 amplitude from training to extinction, $p < .05$.

Changes in activity at the training site (C4 or CZ) were generally paralleled by similar changes in the contralateral site (C3). The similarity of activity at the two sites is shown in Figure 5.

Only trivial inconsistencies were found in the data. For DEC1 the extinction mean amplitude did not reach baseline levels at C3 as it did at C4. The extinction mean amplitude was greater than the training mean amplitude at C3 and was similar to C4 in this respect. For INC1 the decrease in the extinction mean amplitude from training was significant at C3 but not at C4. For INC3 the mean amplitude increased from training to extinction at C3 but stayed the same at C4.

Tracings of the AEPs superimposed across each of the three treatment conditions from C3 for all subjects are shown in Figure 8. AEPs were averaged from the last 900 EPs

Table 8

F Tables of the Analyses of the N1-P2 Amplitudes at C3 Across the Baseline, Training and Extinction Periods for Each Subject

Subject	Source	*df*	MS	*F*-Ratio
DEC1	Between	2	71987.69	87.63**
	Within	3773	821.47	
INC1	Between	2	62794.66	102.12**
	Within	2102	614.89	
INC2	Between	2	30124.55	59.11**
	Within	2191	509.60	
INC3	Between	2	5446.15	11.55*
	Within	2284	471.14	

*$p < .001$

**$p < .0005$

FIGURE 8. Superimposed AEPs from baseline (B), training (T), and extinction (E) for each subject from the contralateral site (C3-left). Each tracing is 512 msec in duration. Stimulus onset is at 100 msec (arrow). Negative is up.

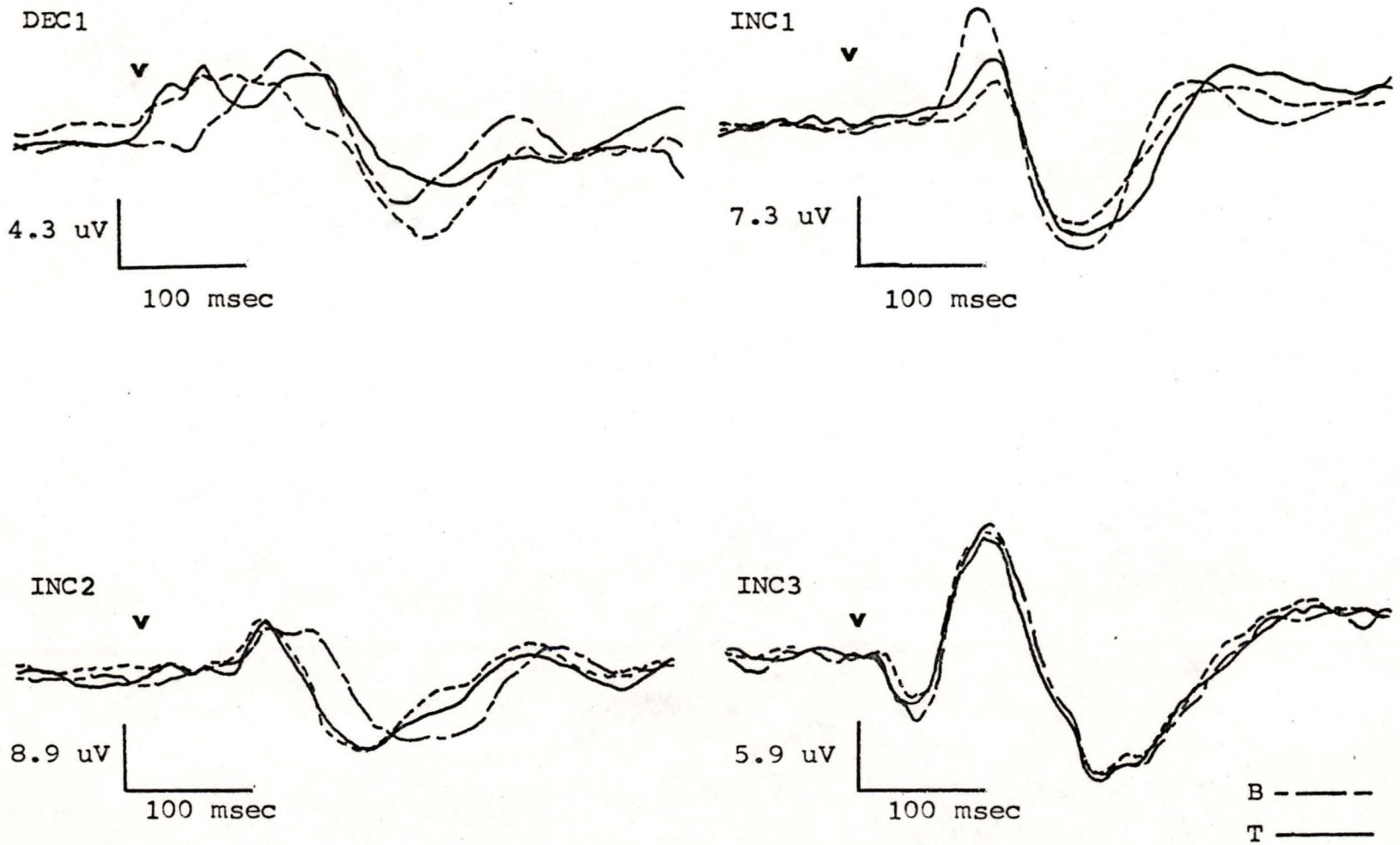
DEC1
v
4.3 uV
100 msec
INC1
v
7.3 uV
100 msec
INC2
v
8.9 uV
100 msec
INC3
v
5.9 uV
100 msec
B
T
E

FIGURE 8

collected from baseline, training, and extinction. For DEC1 and INC1 the changes in AEP amplitude at C3 were similar to the changes noted at C4. For INC3, however, a discrepancy was noted between C4 and C3 during extinction. At C4 the AEP mean amplitude decreased by .6 μV and at C3 it increased by .4 μV. The largest discrepancy in AEP N1-P2 amplitude for INC2 was in baseline where the amplitude at C3 was uncharacteristically small compared to C4.

In general, it was found that the changes in N1-P2 amplitude at C4 and C3 were similar across experimental periods. No unique changes in activity were noted for DEC1 as a result of the conditioning process.

The possibility of unique conditioning effects for DEC1 were further investigated by sorting single EPs from the collaterally recorded site (C3) on the basis of the reward status of the corresponding EP at the trained site (C4). EPs from the collateral site associated with a hit at the trained site were averaged. EPs associated with misses at the trained site were averaged. Figure 9 shows superimposed averages of hits and misses from both recording sites from each phase of the experiment.

In baseline the pairs of AEPs recorded from C3 have waveforms similar to the pairs of AEPs from C4. There is a high correspondence between the evoked activity occurring at C4 and the evoked activity at C3 such that amplitudes of low voltage tended to occur together at both sites and

FIGURE 9. Superimposed AEPs from the trained (C4-right or CZ-vertex) and contralateral hemispheres (C3-left) for baseline (B), training (T), and extinction (E) for DEC1. Solid line AEPs are associated with misses, dashed line AEPs are associated with hits at the trained site. Each tracing is 512 msec. Stimulus onset is at 100 msec (arrow). Negative is up.

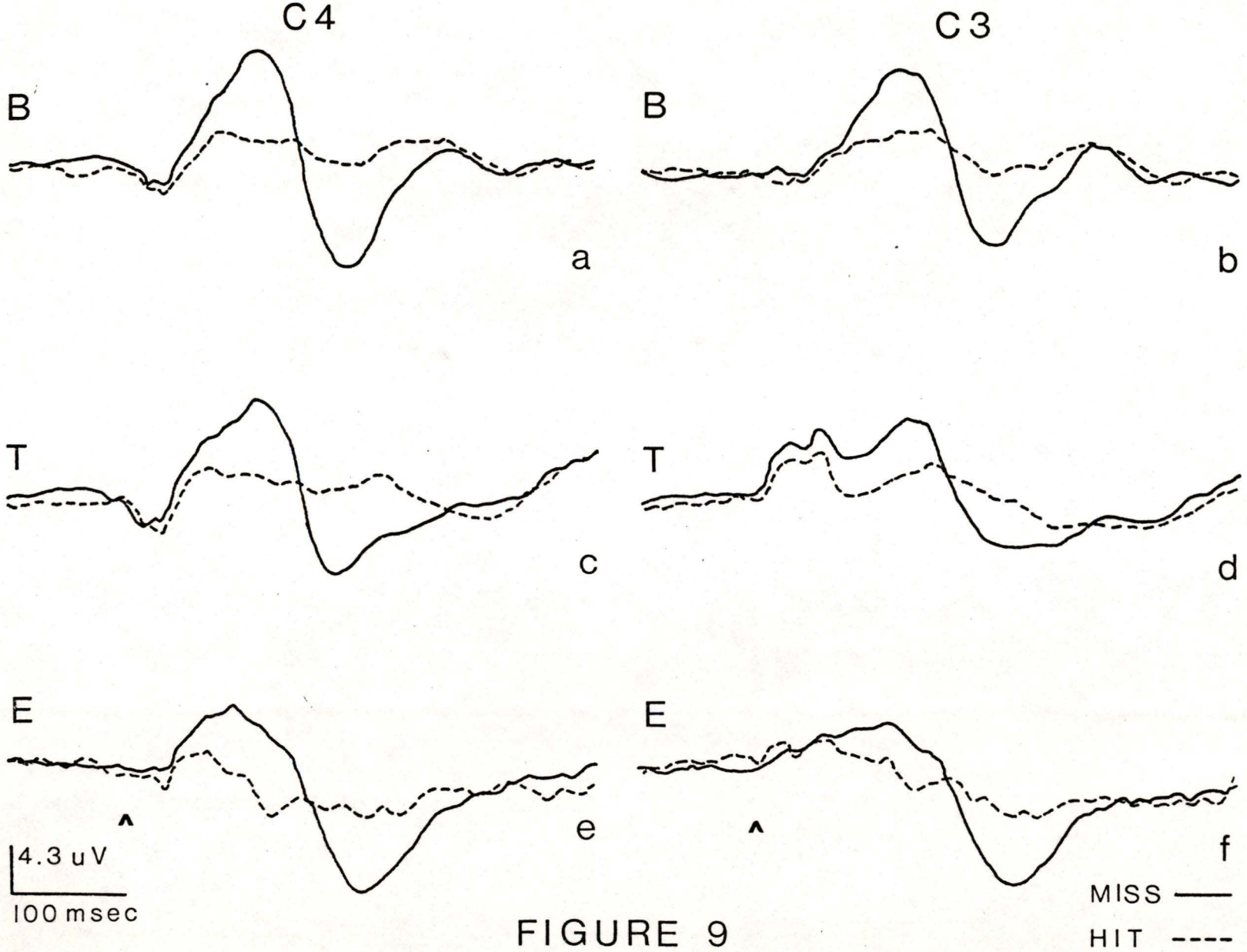

FIGURE 9

amplitudes of high voltage tended to occur together. The fact that the pairs of hit and miss AEPs were not any different in training compared to baseline with respect to N1-P2 suggests that no new patterns of activity were generated in order to modify the N1-P2 amplitude. It seems that the type of activity that generated low voltage amplitudes simply occurred more often.

A high degree of correspondence in EP activity was also noted for the non-conditioned subjects. Pairs of hit and miss AEPs are shown in Figure 10 for one representative subject. EP activity of large amplitude at C4 tended to be associated with large amplitudes at C3. Amplitudes of low voltage at C4 also tended to be associated with amplitudes of low voltage at C3. These relationships were noted for all three subjects in the Increase condition for baseline, training, and extinction.

This result is taken as evidence of similarity of evoked activity across hemispheres regardless of conditioning effects.

Corresponding activity at C3 diverges from activity at C4 during training for DEC1 with respect to the primary component P1 (Figure 9). This component which peaks in amplitude at 56 msec PST reverses polarity and increases in amplitude in the collaterally recorded hemisphere compared to P1 in the trained hemisphere. During baseline the polarity of P1 at C3 and C4 is the same.

FIGURE 10. Superimposed AEPs from the trained (C4-right or CZ-vertex) and contralateral hemispheres (C3-left) for baseline (B), training (T), and extinction (E) for INCl. Solid line AEPs are associated with misses, dashed line AEPs are associated with hits at the trained site. Each tracing is 512 msec. Stimulus onset is at 100 msec (arrow). Negative is up.

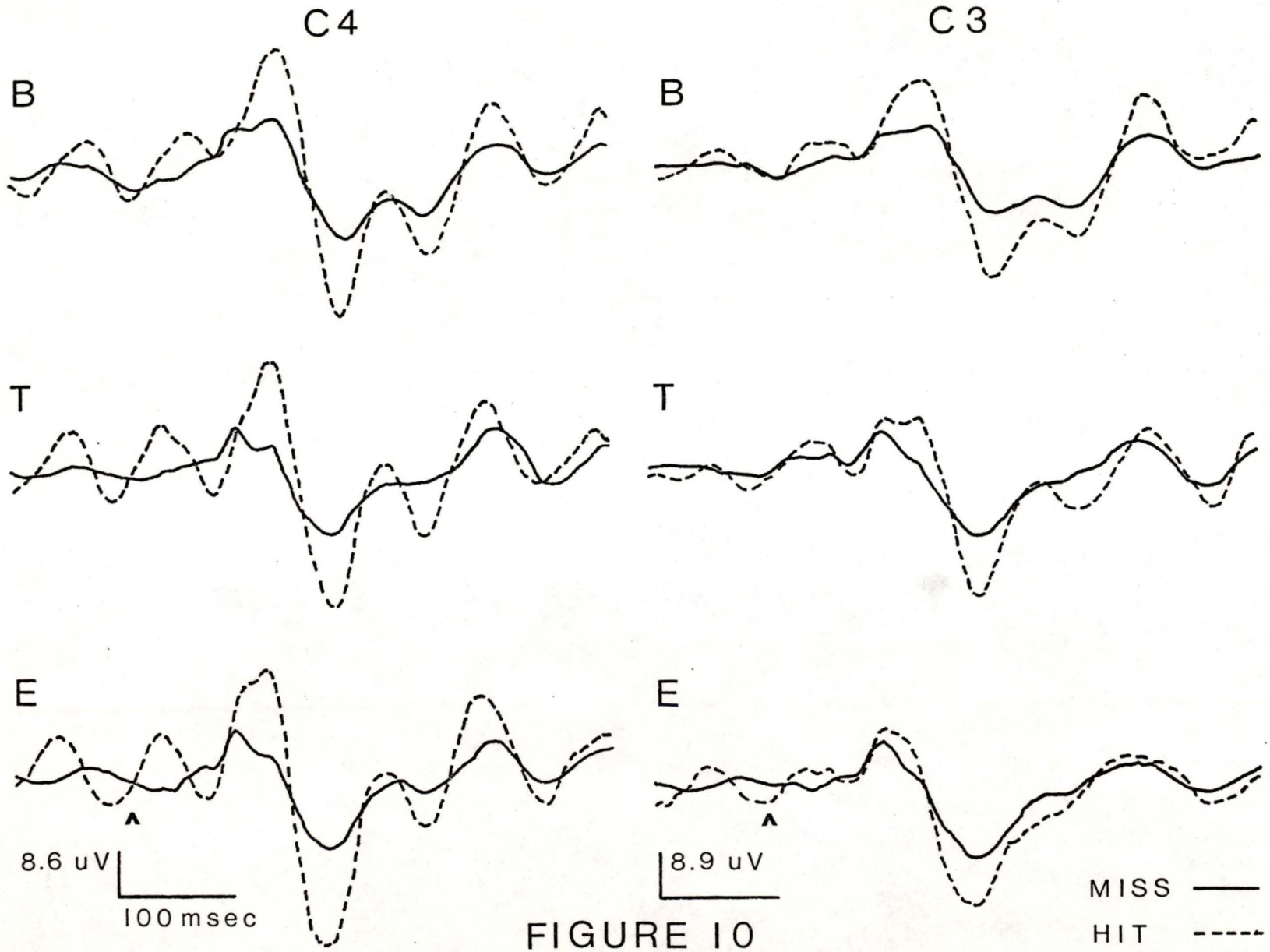

FIGURE 10

The changes in polarity are also evident in the total AEP of the training period at C3. The AEPs of baseline, training, and extinction from C3 and C4 are shown in Figure 11. While P1 at C4 has the same polarity in training and baseline, the polarity of P1 at C3 is reversed. This effect was unique to DEC1 and only occurred during training. The polarity reversal of P1 could be a result of new constraints imposed on the generating cells by conditioning. However, the fact that the significant reorganization of activity occurred at C3 and not at the trained site is puzzling. Furthermore, the polarity reversal of P1 does not seem to be specific to the production of hits since it also occurred in AEPs associated with misses (see Figure 9).

Temporal independence of EP components. Two types of statistical independence of components were identified in the AEPs. First, statistical independence between the primary (P1) and secondary (N1-P2) components was noted in non-conditioned EPs. For example, DEC1's AEPs in Figure 9a and 9c very nearly overlap at P1 independently of the extreme variation in N1-P2 caused by sorting the waves into hit and miss categories. In addition to DEC1, INC3 showed evidence of statistical independence of AEP components. When single EPs were sorted into hit and miss categories, both early (P1, 42 msec PST) and late (N2, 318 msec PST) components were unaffected (see Figure 12).

FIGURE 11. Superimposed pairs of AEPs from the trained hemisphere (C4) and the contralateral hemisphere (C3) for baseline (B), training (T), and extinction (E) for subject DEC1. Stimulus onset is at 100 msec (arrow). Negative is up.

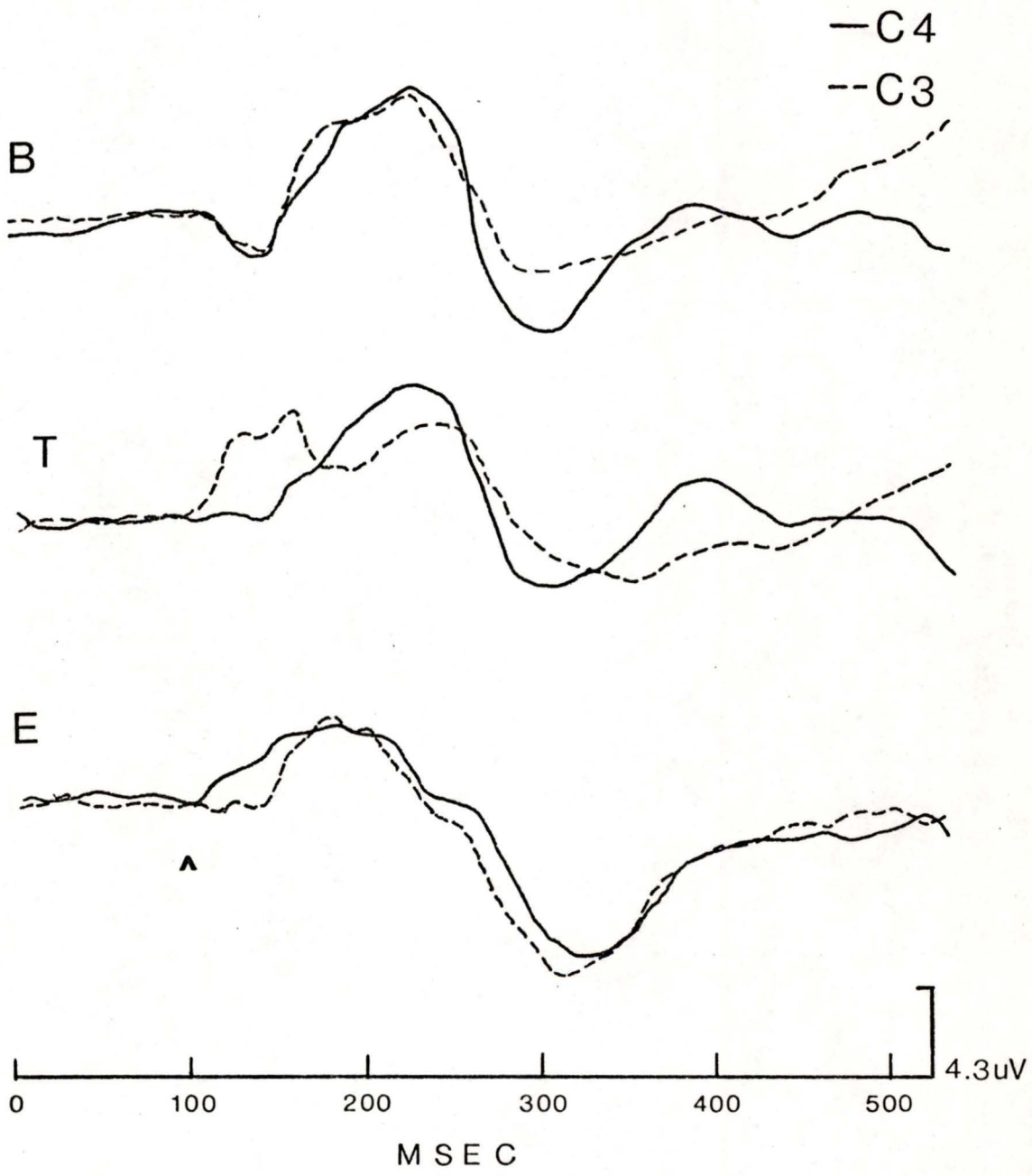

FIGURE 11

FIGURE 12. AEPs of hits (dashed lines) superimposed on AEPs of misses (solid lines) for two subjects, INC2 (top) and INC3. Components are labelled. Stimulus onset is at 100 msec (arrow). Each tracing is 512 msec. Negative is up.

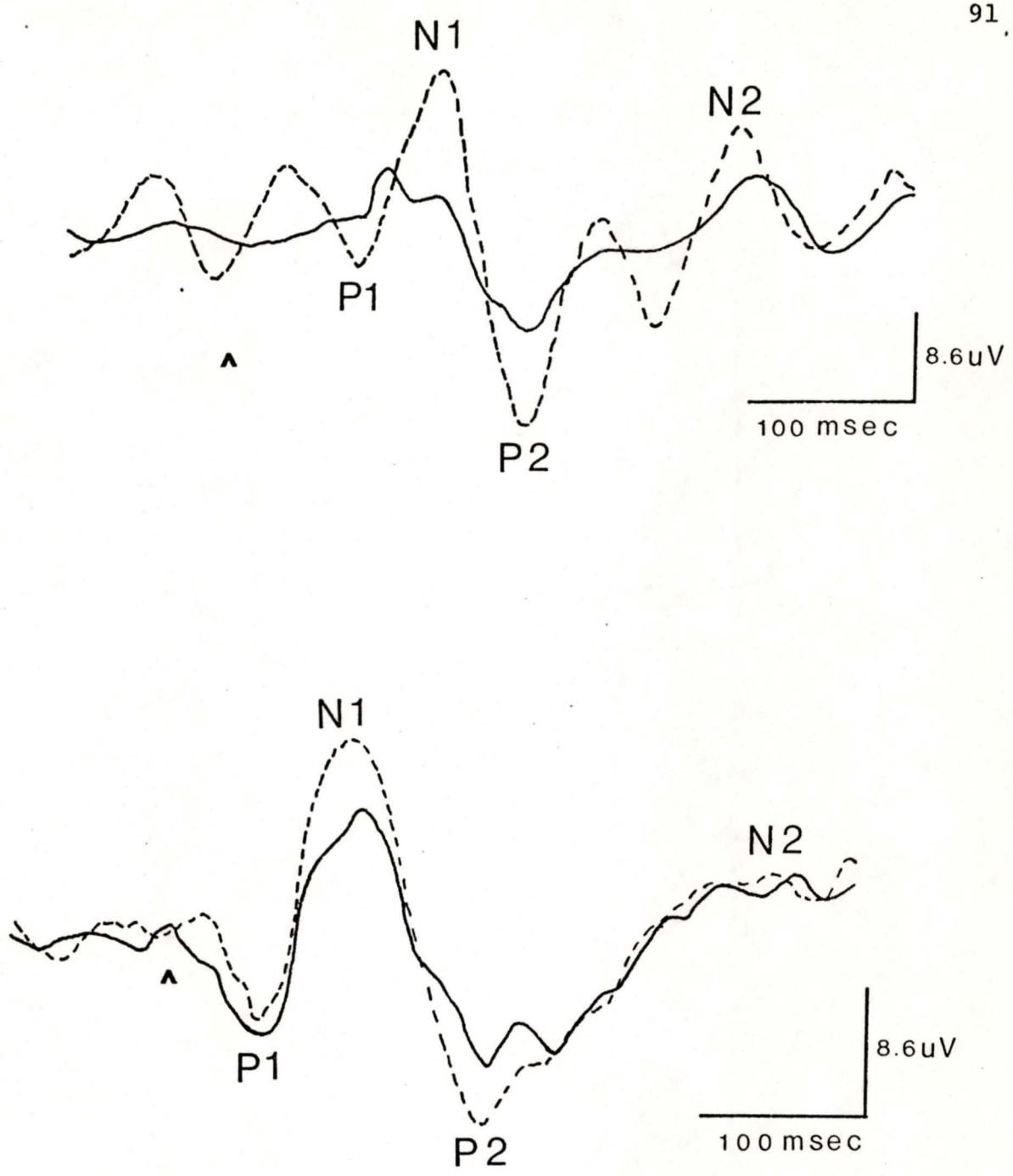

FIGURE 12

Statistical independence does not strictly hold for all subjects, however. For INC2, an overall dependency is evident between components (see Figure 12). The amplitudes of all components were diminished when N1-P2 was sorted into the miss category.

A second type of independence was noted in the specificity of conditioning the N1-P2 amplitude of DEC1. Referring to Figure 5 again, the AEPs in baseline and training show a high degree of correspondence in waveform from stimulus onset to N1 (114 msec PST) such that the two waveforms are nearly superimposable up to this point. Any deviations in the waveforms are minor compared to the conditioned changes in N1-P2. This specific reduction in AEP amplitude is in contrast to the waveforms of INC1, in which a non-conditioned reduction of amplitude of the AEP is most obvious (Figure 5). For INC1, it appears that the amplitude reduction is more generalized, affecting other components. The specific reduction of N1-P2 in DEC1's AEP is evidence in support of conditioning.

DISCUSSION

Conditioning

The results have shown that one subject (DEC1) was able to operantly control the N1-P2 amplitude of the auditory EP. This control was demonstrated by a significant increase in the number of criterion responses obtained in training. The conditioning effects were also noted as a significant decrease in the N1-P2 amplitude of the auditory AEP during training. This was accomplished by reducing the positive amplitude of the P2 component (i.e., increasing the negativity) while the amplitude of N1 remained unchanged.

In the study by Rosenfeld et al. (1969), no statistically significant changes in AEP amplitude were noted. Two possibilities were hypothesized for the lack of amplitude changes in the Rosenfeld study. First, the lack of control for EMG contamination may have obscured the otherwise conditioned changes in the AEP amplitude. Second, the modest conditioning effects may not have been strong enough to produce a noticeable change in the AEP amplitude.

The subjects in the Rosenfeld study at best did not exceed 30% successful responses when chance success was 16%. In the present study the subject at best obtained 32% successful responses when chance performance was 18%. Thus, the reason for the marked conditioned changes in AEP

amplitude in the present experiment and the lack of such changes in the Rosenfeld study is attributed to the explicit control of EMG artifact in the present study. With an increased signal to noise ratio the conditioning effects on the AEP amplitude became apparent.

Conditioned increases in negativity of an EP component have been reported as more robust and as having faster acquisition rates compared to positivity training (Fox & Rudell, 1968, 1970; Rudell, 1970; Rudell & Fox, 1972). These results correspond with the results in this experiment and may in part explain the failure of the three subjects in the Increase condition to learn. As noted before, DEC1 decreased the N1-P2 amplitude by increasing the negativity at P2. The amplitude at N1 was unchanged. For subjects in the Increase condition an increase in the N1-P2 amplitude would have required either increased positivity at P2 or increased negativity at N1. If the findings from the animal studies can be applied to humans it may mean that increased positivity at P2 may be more difficult to achieve than negativity.

Another strategy would have been to increase the negativity at N1. Given that increased negativity is easier to condition than increased positivity, this would be the expected strategy subjects would use. However, subjects may not have been able to accomplish this due to the relationship of N1 and P2 to the time of reinforcement. Since P2

intervened between N1 and the reinforcer, it is possible that the large part of the response-reinforcement contingency was between P2 and the reinforcer. This hypothesis is somewhat supported by the work of Rudell and Fox (1972). When they conditioned both increases and decreases in an amplitude difference between a reference potential and a later occurring potential (P1, 24-28 msec PST) on the EP waveform, the major part of the amplitude changes were accounted for by the change in amplitude at the component closest in time to the reinforcer, i.e., P1. However, amplitude changes were also noted in the earlier occurring reference potential in the desired direction. Whether these changes were significant was not reported. Thus, the failure to learn the task might be accounted for by the interruption of the N1-reinforcement contingency by P2.

Another reason for the failure of subjects to learn may be due to the delay of reinforcement. At best the reinforcement was delayed 240 msec from the completion of the response. It could be argued that the activity being reinforced was not the criterion component but the activity just prior to the occurrence of the reinforcer. Other studies have reported successful conditioning with reinforcement delays of the size reported here, however. Rudell (1970) conditioned an early potential in cats with a reinforcement delay of 336 msec. Rosenfeld et al. (1976) has reported successful conditioning in one of two rats when 800 msec

reinforcement delays were used. Although short reinforcement delays are probably more conducive to learning, the systematic reinforcement of a particular kind of neural event is no doubt a crucial factor in successful conditioning.

As was seen with INC2, more than one kind of activity was being reinforced. The window of N1 was large enough to include N1a. The failure of INC2 to be conditioned might have been a result of the inconsistent reinforcement of N1 and N1a. If N1 and N1a represent two different processes in the brain (which is likely since one seems to occur independently of the other), then conditioning would have been impaired by not systematically rewarding the same kind of activity on every trial.

This line of reasoning may also explain the other subjects' failure to condition. The latency windows of N1 and P2 were necessarily wide, varying from 46 to 84 msec in length. Such a width was necessary to adequately specify the component in a large percentage of the trials. A maximum point in one part of the window may have different significance to the nervous system compared to a maximum point in another part of the window. Westerberg (1977), for example, has shown that latency variations are meaningful in the sense of being conditionable. Thus, reinforcement of the maximum point anywhere within the window is in fact rewarding many different types of neural activity.

Other studies (Fox & Rudell, 1970; Walker, 1974) have been interested in the investigation of the minimal information bearing unit of the EP by placing the reinforcement contingency on components of very short duration. If the component is conditionable, then it must represent significant neural information to the nervous system. Similarly, the maximum information bearing unit can be determined by the same strategy. It is undoubtedly the case that a 46 to 84 msec segment of the EP is of complex informational significance to the nervous system and that rewarding the maximum point in this period is rewarding indiscriminantly many different neural processes.

It is likely that a combination of the above factors (i.e., the reinforcement of two components, the reinforcement of a wide band of activity, and a long reinforcement delay) contributed to the failure to condition. The additional manipulations performed on individual subjects, such as the attempted shaping procedure on INC3 and the increased informational feedback to INC2 were not sufficiently powerful enough to overcome the other debilitating factors.

Control of Artifactual Mediators

Whatever the reasons for learned changes in one direction and not the other, the controls instituted in this experiment rule out certain factors as artifactual mediators of the changes in the AEP amplitude of DEC1. The control of

the EOG has ruled out myogenic mediation. The use of the headphones obviated selective orientation to the stimulus as a mediational mechanism. Non-contingent reinforcement and pseudo-conditioning in the baseline period has ruled out the non-associative effects of the reinforcer or changes in the arousal of the subject as the mediator of the conditioned response.

Two procedures were used to control for naturally occurring changes in the criterion amplitude over time. First, an extinction procedure was used within subjects and second, bidirectional conditioning was employed across subjects. Since conditioning was obtained in only one direction, bidirectional control was not effective in controlling for non-associative changes in the criterion component over time. For DEC1 the extinction period did yield a return of the amplitude to baseline levels.

It must be pointed out that the mean amplitude of the N1-P2 distribution (i.e., the amplitudes upon which reinforcement was contingent) decreased for all subjects from baseline to training. Also, the return of the N1-P2 amplitude to baseline for DEC1 was confounded by the fact that non-contingent reinforcement was not employed during extinction. It is possible that the changes in extinction were a result of a change in state of the subject rather than removal of the reinforcement contingency. However, data from the other subjects suggest that this is unlikely. The

use of either non-contingent reinforcement (for INC3) or simply removal of the reinforcement (for INC1 and INC2) in extinction did not produce a return of the N1-P2 amplitude to baseline levels. Therefore, the changes in the N1-P2 amplitude of DEC1 were due to the response-reinforcement contingency. Until more subjects are run in both the Increase and Decrease conditions, these conclusions should be accepted with caution.

Conditioning Mechanisms

Certain lines of evidence indicate possible mechanisms of the conditioning effects for DEC1. The use of a difference amplitude as the criterion component ruled out slow potential shifts (Hillyard, 1974) as the mechanism of conditioning. The use of a random intertrial interval eliminates the possibility of mediation by discrete behavioral movements whose proprioceptive feedback or efferent command activity could have produced the changes in the EP. Knowledge of when to make a discrete behavioral response could only be obtained from a regular intertrial interval or from the onset of the stimulus itself. Having ruled out the regular intertrial interval as the cue, it is still possible that discrete behavioral movements could have been cued by the stimulus. This would have required a reaction time of less than 226 msec. Although human reaction time is within this limit, a proprioceptive neural feedback loop would add

more time, thus making it unlikely that such a feedback loop could produce the conditioning effects.

Moreover, the degree of specificity of the conditioned changes argues against mediation by a behavioral response. Figure 6 shows the locus of change is restricted to P2. It is hard to imagine behavioral responses that the subject would be unaware of whose neural concomitants could demonstrate such a high degree of specificity. Thus, the results support a tonic mediation hypothesis (Rosenfeld & Owen, 1972; Rosenfeld et al., 1976; Rudell & Fox, 1972). It is proposed that the generator cells of the neural criterion amplitude are preset before the stimulus is presented. The stimulus itself simply serves to probe the generator cells to respond at the appropriate time.

Relationship of the EEG Amplitude to the N1-P2 Amplitude

Conditioning effects for DEC1 do not appear to be related to changes in the EEG amplitude. When the mean N1-P2 amplitude decreased from baseline to training, the mean EEG amplitude remained unchanged. From training to extinction, however, an increase in the mean N1-P2 amplitude was paralleled by an increase in the mean EEG amplitude. Further investigation revealed that the proportion of hits during desynchronization did not deviate significantly from the total proportion of hits in any of the treatment conditions. It may be concluded that although there might be

some relationship between EEG and EP amplitude, particularly in the later stages of conditioning, the conditioned decreases in EP amplitude were not accomplished by voluntary desynchronization of the EEG. This finding supports the results of Rudell (1970) in which the generation of criterion responses was not a result of changes in cortical arousal of the EEG.

The assertion that manipulation of the EEG amplitude was not a mechanism of conditioning is also supported by the specificity of the conditioned changes in the AEP. If the EEG was decreased in amplitude one would expect a uniformly decreased AEP across all components during training. Since the amplitude decrease appears only at P2, it is unlikely that the manipulation of EEG amplitude was responsible for changes in P2 (see Figure 6).

Changes in EEG amplitude sometimes paralleled changes in N1-P2 amplitudes for other subjects. For INC3 changes in EEG amplitude paralleled changes in the N1-P2 amplitude through all phases of the experiment. For INC2 and INC3 parallels were found from training to extinction. However, analysis of the proportion of hits during synchronization of the EEG compared to the total proportion of hits yielded no statistically significant differences. It is concluded that although changes in EEG amplitude are related to changes in N1-P2 amplitude, subjects in the Increase condition did not manipulate EEG amplitude to generate criterion amplitudes.

EPs Recorded from the Contralateral Hemisphere

AEPs recorded from the two hemispheres were similar in waveform for all subjects and under all treatment conditions. Other studies (Picton et al., 1974) have reported similarities between auditory AEPs from homologous recording sites across hemispheres.

No unique changes in the contralateral hemisphere of the conditioned subject could be attributed to the conditioned changes in the trained hemisphere. It was found for all subjects that hits tended to occur at the same time in both hemispheres. The same was found for misses. This relationship was the same for both baseline and training.

This finding is in contrast to the results reported by Rosenfeld and Owen (1972) in animals. These researchers found that conditioning effects from the trained site spread to other sites in the brain during training. In the naive animal the neural activity that was to be rewarded in the trained site did not occur simultaneously in other brain sites. The researchers concluded that the neural criterion response in training may not represent the same event as the criterion response in baseline. This has led Rosenfeld (1974) to propose that operant conditioning of neural events involves novel states of the cortex, such that new processes are involved that were not present in baseline.

The findings in the present study suggest that for human EP conditioning the neural activity that produced a criterion response in baseline was the same activity that produced criterion responses in training. The activity at the trained site was not qualitatively different from activity at the contralateral site in either baseline or training. No new neural processes emerged as a result of conditioning. It appears that the neural response present in baseline was simply produced more often in training.

One finding from DEC1's data does suggest unique changes in brain activity that may have been due to conditioning. As shown in Figure 11, the polarity of the P1 component at C3 was reversed during training. Although this component was not directly related to the conditioning process it is possible that the reorganization of brain activity required for conditioning led to the changes at P1. Why such a significant change should occur at a remote site and not at the trained site is perplexing. Still such dramatic changes in evoked potential configuration were not seen in the subjects who did not condition.

Functional Independence of the Auditory AEP Components

Temporal independence of the auditory AEP components was found for both conditioned and non-conditioned subjects. Averages of hits and averages of misses, when superimposed showed major changes in the criterion components and

relatively minor changes elsewhere on the waveforms. The temporal independence of the major components of the AEP agrees with similar findings reported by others (Fox & Rudell, 1970; Rosenfeld et al., 1976). Furthermore, temporal independence of the components in conditioned AEPs has also been described here and in other studies (Rosenfeld et al., 1976; Rudell & Fox, 1972). These researchers have described the independence of components of evoked potential waveforms in animals. The present study has confirmed the independence of auditory evoked potential components in humans. It appears that the conditioning process is able to capitalize on the temporal independence of the AEP components to produce specific changes in one component independently of other components of the AEP. It is clear that changes in evoked potential waveforms can be quite specific to the reinforced parameter in EPs recorded in both humans and animals. These results suggest a functional independence of the sequential components of sensory evoked potentials in general and that components of the AEP are produced by a number of independent neural generators. The sensory evoked potential waveform is probably best represented as a series of relatively independent configurations in time, each having unique functional significance.

It must be noted that one subject (INC2) did not demonstrate relative independence of the AEP components (see top of Figure 12). All components were reduced when the AEPs

were averaged on the basis of hits and misses.

Attention as a Mediator of Conditioning

The N1-P2 amplitude of the auditory EP has been related to both arousal and attentional aspects of the organism (Naatanen, 1975). The relative contributions of non-specific arousal and specific attentional processes to the N1-P2 amplitude have been a source of much research (Naatenen, 1975). Haider, Spong and Lindsley (1964) have reported a decrease in visual evoked potential amplitude with decreased attentiveness. Attentiveness may have been manipulated by DEC1 to obtain reinforcement. Indeed, the subject reported that just ignoring the stimulus was not sufficient to increase the number of rewards over baseline. He had to actively ignore the tone stimulus by mentally practicing guitar chords. Simple reduction in attentiveness to the stimulus and hence a reduction in the AEP N1-P2 amplitude probably occurred in baseline. Further reduction in N1-P2 required more than inattentiveness to the stimulus, but an active cognitive blocking mechanism. Results from the EEG analysis indicate that the conditioned effects were not due to non-specific arousal of the subject. Thus, the results support the notion that reduction in the N1-P2 amplitude is sensitive to active attention gating mechanisms.

It is possible that the decreases noted for DEC1 were due to long term habituation or other processes (discussed

in the next section). However, Maclean, Ohman, and Lader (1975) have reported that further decreases were obtained beyond habituational decreases by manipulation of attention. This observation in conjunction with subjective reports and the subject's inability to decrease the N1-P2 amplitude in the first 6 days of training suggest that habituation was not the mediating factor.

It is interesting to note that the subjects in the Increase condition were not able to produce an increase in their N1-P2 amplitudes by either focusing attention on the stimulus or increasing arousal. These were the first strategies employed by these subjects. These factors have been invoked to explain increases in N1-P2 amplitude in studies of attention (Garcia-Aust, Bogacz & Vanzulli, 1964; Naatanen, 1975), particularly when subjects were habituated to the stimulus. Focusing of attention to the stimulus did not seem to be an effective mechanism for increasing the N1-P2 amplitude in the present study. This casts doubts on the strength of the relationship between the AEP N1-P2 amplitude and simple attention. The N1-P2 amplitude may be more strongly related to selective attention (Naatanen, 1975) or to such processes as those discussed in the next section.

Long Term Decrements of the Auditory AEP

Long term decreases were noted for INC1 in the N1-P2 amplitude of the AEP. Also long term decreases were noted

for the N1a component of the AEP for INC2. That such components of the AEP decrease over a long period of time (21 days) has not been reported in the literature, to this author's knowledge. Evidence from human studies have reported short term decrements (one session) of 2-10% of the maximum AEP amplitude (Callaway, 1973). With habituation runs of 144 min. Ritter, Vaughan, and Costa (1968) found decrements in N1-P2 to be 10% of maximum AEP amplitude. No further decreases were noted after one-fourth of the total elapsed time of the habituation run.

The decrements in AEP amplitude reported here are different from those commonly described as habituation. The decreases were over a longer period of time (21 days) and the magnitude of the decrease (23%) is much greater than usually reported.

The neurophysiology of habituation is rather confused in the animal literature (Worden, 1966, 1973). Aside from the highly inconsistent results and the methodological inadequacies noted by Worden, EP amplitude changes have not been found to demonstrate the most common characteristics of behavioral habituation: dishabituation and spontaneous recovery (Westenberg, Paige, Golub & Weinberger, 1976). This has led researchers to be more cautious about describing just any AEP amplitude decrement as habituatory (Westenberg & Weinberger, 1976; Worden, 1973).

Callaway (1973) has described two types of habituation that result in a decrease in the human AEP N1-P2 amplitude. Fast habituation occurs in the first few trials of the habituation run and slow habituation usually has a time course of a half hour. Thus, the changes described here are qualitatively different from those described by Callaway.

Perhaps the best interpretation of the AEP amplitude decrements noted for INC1 and INC2 is not one of habituation but of greater efficiency of the nervous system to process the information concerning the stimulus (Worden, 1973). This is consistent with the long term decrements described here. Habituation of the AEP amplitude seems to be more aptly applied to initial decrements in AEP amplitude. Short term changes also tend to coincide better with behavioral indices of habituation.

Conclusions

A conditioned decrease in the N1-P2 amplitude of the human auditory AEP has been demonstrated. The operant control of this component was demonstrated in the absence of myogenic and behavioral mediators. The results reported for the single subject here must be supported by further study. Until the time that supporting evidence is available, these results should be accepted with caution.

Possible conditioning mechanisms were the modification of the neural generators of the criterion response so that

the synaptic excitabilities were preset before the arrival of the stimulus. These neural generators may have been modified in response to a central attentional gating mechanism in which attention was actively gated away from the stimulus.

Conditioning was specific to a single component of the AEP and implies functional independence of the AEP components. This conclusion was also supported by the demonstration of temporal independence of AEP components of subjects who did not condition.

Possible reasons for the failure of the subjects in the Increase condition to learn were attributed to the failure to specify the criterion component in terms of a single neural process and the confounding effects of long term decrements in the AEP amplitude over time. The long term decrements in AEP amplitude noted here are the first to be reported in the literature and are thought to be related to increased efficiency in processing information about the stimulus rather than an habituation effect.

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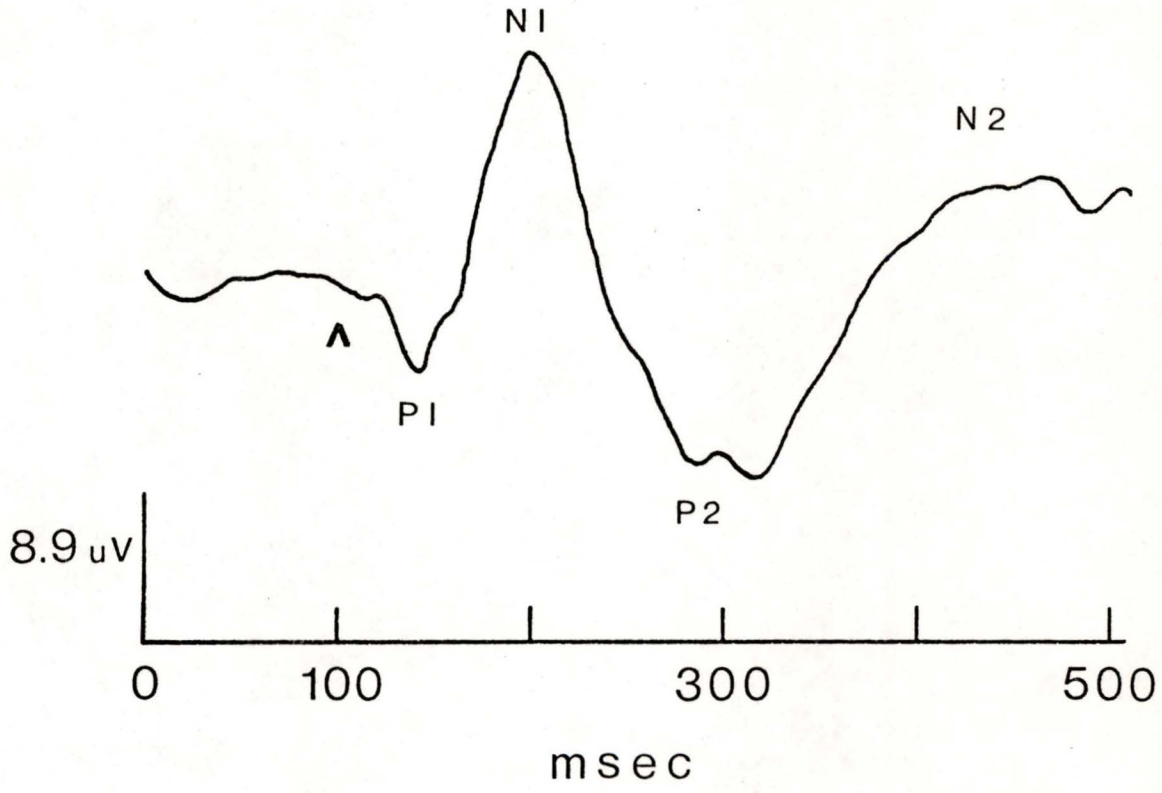
APPENDIX A



An example of an auditory averaged evoked potential recorded from the human scalp. Stimulus onset is at 100 msec (arrow).

APPENDIX B

GLOSSARY OF ABBREVIATIONS

A-D	analog to digital conversion computer units
AEP	average evoked potential
C3	electrode site on human scalp, left hemisphere
C4	electrode site on human scalp, right hemisphere
CZ	electrode site on human scalp, vertex
CNS	central nervous system
D-A	digital to analog conversion computer units
dB	decibels
DEC1	first subject in the Decrease condition, JG
EEG	electroencephalography
EMB	electromyographic
EOG	electrooculogram
EP	evoked potential
FM	frequency modulated
Hit	a criterion evoked potential
Hz	Herz, cycles per second
INC1	first subject in the Increase condition, NW
INC2	second subject in the Increase condition, DE
INC3	third subject in the Increase condition, MR
LGN	lateral geniculate nucleus
Miss	EP whose amplitude did not reach criterion
Msec	millisecond

No N1-P2	an EP in which no N1 or P2 component was detected by the computer
N1	first negative component of an EP
N2	second negative component of an EP
OCNE	operant control of neural events
O2	electrode site on human scalp, right hemisphere
PNS	peripheral nervous system
PST	post-stimulus time
P1	first positive component of an EP
P2	second positive component of an EP
P300	a late positive component of an EP occurring at 300 msec PST
SD	standard deviation
SMR	sensory motor rhythm
SW	slow wave
μV	microvolts

VITA

Surname: KOSNIK Given Names: WILLIAM DAVID

Place of Birth: GENEVA, ILLINOIS, U.S.A.

Date of Birth: 20 JUNE 1951

Educational Institutions Attended,
with Dates of Entering and Leaving:

NORTHWESTERN UNIVERSITY, EVANSTON, ILL.	1969	to	1973
UNIVERSITY OF VICTORIA, B.C.	1974	to	1977
		to	
		to	

Degree, Diplomas, Etc., Awarded,
with Dates and Names of Institutions:

B.A.	1973	Northwestern University

Honours and Awards:

Publications:

Rosenfeld, J. P., Hetzler, H. B., & Kosnik, W. D. Operant photic evoked potential control unmediated by selective orientation. *Physiology and Behavior*, 1974, 13, 479-482.

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OPERANT CONDITIONING OF AUDITORY EVOKED POTENTIAL AMPLITUDE IN HUMANS: THE ROLE OF NON-ASSOCIATIVE AND MYOGENIC FACTORS

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Signature

WILLIAM DAVID KOSNIK
Name

23rd June, 1977
Date