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A BEHAVIORAL AND ELECTROPHYSIOLOGICAL STUDY OF SELECTIVE STIMULUS CONTROL IN THE RABBIT

by

© PERRY STUDLEY KINKAIDE

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "A Behavioral and Electrophysiological Investigation of Selective Stimulus Control in the Rabbit", submitted by Perry Studley Kinkaide in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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ABSTRACT

A behavioral and electrophysiological investigation of selective visual stimulus control (VSC) was conducted on albino rabbit during classical eyeblink conditioning. Ss were first differentially conditioned to two non-visual stimuli: CS+ and CS-. VSC was acquired if the visual stimulus was reinforced in compound with CS- but was blocked when reinforced in compound with CS+. These data were consistent with both a CS attenuation or attentional interpretation (Sutherland & Mackintosh, 1971) and a US attenuation or associative interpretation (Rescorla & Wagner, 1972). Conditioning and blocking were demonstrated to both 3-Hz flashes and electrical stimulation of the optic chiasma. Thus, blocking was not attributable to "gating" or "filtering" of afferent input at the peripheral or retinal level (Hernández-Peón, 1964). Extinction and backward conditioning of chiasmic stimulation preceded subsequent retraining of six Ss. Performance was asymptotic within four trials after the visual stimulus was reinforced in compound with CS-, a finding inconsistent with associative theory. A distinction was warranted between attentional and associative factors suggesting that low performance reflected a lack of attention and not necessarily low associative strength.

In conjunction with the behavioral investigations visual evoked potentials (VEPs) were averaged at the striate cortex. Primary activity, particularly the initial surface positive deflection, of the VEP (both amplitude and an energy estimate) was inversely related to the level of non-VSC. If the visual stimulus was paired with CS- as opposed to CS+ primary activity was enhanced. These changes were evident at the

initiation of compound conditioning, or later, when the visual stimulus was presented alone. The enhancement was not attributable to sensitization or to peripheral factors but was interpreted as reflecting disinhibition of the dorsolateral geniculate (LGB) acting to enhance afferent input during selective visual attention. Increments in secondary activity produced by chiasmic stimulation were observed under the same conditions which produced a decrement in secondary activity produced by photic stimulation. These results were attributable to changes in arousal which produced changes in the amount of intracortical recurrent inhibition triggered by the visual stimulus. The data suggest that following CS onset there was a phasic increment in arousal which reached a peak prior to the initiation of a CR and declined during a CR.

The recovery cycles of components of the geniculo-striate VEP subsequent to optic chiasma stimulation were obtained during a Pretest, after suborbital shock, and during VSC and non-VSC. The absolute level of recovery was elevated during VSC over non-VSC and attenuated subsequent to suborbital shock. Recovery rate was enhanced during non-VSC and retarded following suborbital shock. Geniculo-striate excitability appears to be the product of: (1) selective influences augmenting LGB responsiveness during visual attention and attenuating responsiveness during non-visual attention and (2) phasic and/or tonic arousal acting to augment intracortical inhibition. The mechanism mediating these changes is uncertain.

Acknowledgements

In the preparation of this dissertation I have sought and received invaluable assistance from a number of people. It is with sincere gratitude and appreciation that I acknowledge the personal contribution of my supervisor, Dr. R. E. Walley, whose methodological suggestions improved the experimental design. His theoretical guidance clarified my thinking on a number of issues and numerous suggestions greatly improved the content of this manuscript. I am also indebted to Dr. R. Wagner at Yale University for his review and comments regarding the behavioral results. I am particularly grateful for the facilities and services made available to me by Mr. J. Thomsen and Mr. J. Bouvier of Biosciences Animal Services. The diligence and ready assistance of Mr. P. Wong on electrotechnical matters is also acknowledged. Mrs. Rita Edge and Mr. P. DeGroot were particularly helpful in insuring that equipment was promptly available on request. Mr. G. Cole saved me countless hours by performing the majority of the histological work. Mrs. Barbara Patrick and Mrs. Milka Chruma assisted me in the preparation of the figures. I should also like to register my thanks to Miss Merrilyn Greig and Mrs. Annamarie Thornberry who, with patience and skill, typed this thesis. Finally I will be forever indebted to Marian who sustained me through the seemingly never ending "crises" associated with the research reported herein.

Foreword

This thesis was designed to study the electrophysiological activity at the visual cortex of the rabbit during selective visual and non-visual stimulus control of a classically conditioned eyeblink response. The aim was to further understanding of the relationship between changes in attention and associated changes in the visual system. To manipulate stimulus control an experimental paradigm was employed in which S is conditioned to respond to either the visual or non-visual element of a stimulus compound. The reinforced compound stimulus was comprised of a visual stimulus (either flashes or optic chiasma stimulation) and either a differentially reinforced or non-reinforced non-visual stimulus. Rescorla and Wagner (1972) have predicted that under these conditions conditioning of the visual element is blocked or enhanced, respectively. Interpretation of the electrophysiological data was contingent upon the success of these behavioral manipulations. For this reason the experimental paradigm and behavioral results are presented in Chapter I and the electrophysiological material is treated in Chapter II. Several supplementary investigations were carried out to assess relationships which bear on interpretations of the primary research results. These studies are cited and presented in the appendices.

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CHAPTER I

THE BEHAVIORAL MANIPULATION OF SELECTIVE STIMULUS CONTROL

This thesis is designed to study the evoked potential at the striate cortex in albino rabbits during selective visual and non-visual stimulus control of a classically conditioned eyeblink response. The aim is to further understanding of the relationship between selective attention and electrophysiological activity within the visual system. As will be outlined in more detail in Chapter II there are several issues confounding the interpretation of changes in the sensory evoked potential in terms of "selective attention" or "learning". Several investigators suggest that changes in the sensory evoked potential associated with operations designed to manipulate attention or conditioned stimulus control actually reflect non-specific increments in arousal. Also, the behavior of Ss during the recording of electrophysiological activity is often poorly controlled, not clearly described, and often only intuitively related to processes of interest to the investigator. An experimental paradigm, described below, is adopted to overcome these limitations. The design also permits an evaluation of the fundamental issue: Is an "attentional" construct required to adequately explain behavior associated with the employed paradigm? A recent modified-continuity theory (Rescorla & Wagner, 1972) derived from research employing a similar design suggests that associative processes adequately account for data often referenced as support for a process of "selective attention". The aim of Chapter I is: (1) to familiarize the reader with the "blocking" paradigm, (2) to evaluate the effectiveness of the paradigm in establishing and

reversing selective visual and non-visual stimulus control within Ss, (3) to assess the adequacy of two contemporary attentional and associative models in accounting for the behavioral data, and (4) to provide a behavioral framework permitting an appropriate interpretation of simultaneously recorded electrophysiological data. A presentation and discussion of the electrophysiological data is reserved for Chapter II.

In an extensive series of experiments Kamin (1965, 1968, 1969a, 1969b) has investigated the way in which an animal ". . . in some sense selects and chooses those particular elements within an array of stimulus elements which enter into learned associations" (Kamin, 1969b, p. 42). Fundamental to this research has been an assessment of the extent to which the contiguity of events in time is sufficent for establishing an association. The basic experimental paradigm has involved conditioning a stimulus (A) and subsequently reinforcing the stimulus in compound with another stimulus (B). Following reinforcement of the compound (AB), an assessment is made of the stimulus control acquired by stimulus B by presenting stimulus B alone. If contiguity alone is sufficent for conditioning, the associative strength of element A should not influence the degree of conditioning to element B.

The results of Kamin's research along with those of Reynolds (1961); Sutherland and Mackintosh (1964); Miles and Jenkins (1965); Egger and Miller (1962); Wagner (1969a, 1969b); Wagner, Logan, Haberlandt, and Price (1968); and Rescorla (1969a, 1969b, 1970, 1971b) have demonstrated that the stimulus control acquired by element B during AB conditioning is dependent upon the prior amount of excitatory conditioning (i.e., the number of prior CS-US pairings) to element A. The critical observation has been that if B is paired with a stimulus

having a positive associative strength, that is, a stimulus which has been positively correlated with reinforcement, then conditioning of element B is attenuated or "blocked". Evidently CS-US contiguity alone is not sufficient to establish conditioning of element B.

Two models are frequently cited to explain the blocking effect. The CS attenuation or two-process attentional model (e.g., Lashley, 1942, Sutherland, 1964, Mackintosh, 1965, Sutherland & Mackintosh, 1971) was designed to explain discrimination learning in an instrumental situation. However, the model is generalizable to classical conditioning. The position maintains that the conditioning established to element B is inversely related to the attention directed to element A during AB compound conditioning. This hypothesis is derived from two assumptions regarding the nature of attention. First, it is assumed that S has a limited attention capacity but can attend to a limited number of cues simultaneously in proportion to their validity. Secondly, the validity of a cue is a function of the consistent significance of events following the presentation of the cue, i.e. the CS-US contingency. Unfortunately, tests of the CS attenuation model have often assumed a "causal" (Rescorla, 1969a, p. 79) attention position considering attention as an all-or-none process. The often cited models of Mackintosh (1965) and Sutherland (1964) are quite explicit in maintaining that while attention is limited it is not an all-or-none response, e.g. "Whether or not an analyser is switched in is not an all-or-none process, as different analysers can be more or less strongly switched in." (Sutherland, 1964, p. 150); or, to paraphrase Mackintosh (1965, p. 130), attention determines which stimuli will prepotently control the animal's behavior though not in

an all-or-none manner. The second model devised to explain the blocking effect is the US attenuation or modified-continuity model. This model was initially suggested by Kamin (1968, 1969b) and has since been elaborated in some detail by Rescorla and Wagner (1972). According to this position if element A has received prior excitatory conditioning then the reinforcer loses its effectiveness to establish conditioning to element B when element AB is reinforced. Underlying this position is an assumption that the effectiveness of a reinforcer to establish conditioning is inversely related to the degree to which its occurrence is expected. Two points complete the essential outline of this model. First, associative strength can assume any value along a continuum from +1 (a conditioned excitor) to -1 (a conditioned inhibitor) depending on whether a stimulus has positively or negatively associated with reinforcement, respectively. Secondly, as long as a reinforcer remains effective, i.e., is unexpected or unpredicted, two stimuli reinforced in compound will be incremented equivalently regardless of their initial associative strengths. However, once the sum of their associative strengths is equivalent to the level of conditioning supportable by the US intensity, additional conditioning will not alter the associative strengths of the respective elements.

Study 1. Classical Eyeblink Conditioning of Selective Stimulus Control in the Rabbit

Most tests of the CS attenuation and US attenuation positions have employed the Estes and Skinner (1941) conditioned emotional response (CER) paradigm. Disruption of an ongoing instrumental task upon the presentation of a stimulus previously associated with shock is

interpreted as evidence that the stimulus has acquired fear eliciting properties. Wagner (1969b) and Wagner et al. (1968) have demonstrated the generality of results from the CER paradigm to a situation employing a compound stimulus during classical conditioning of the rabbit eyeblink response. The experimental paradigm employed in this thesis represents a modification of Wagner's design. The response eliciting properties of a visual CS are assessed following reinforcement of the visual stimulus in compound with either a differentially reinforced or non-reinforced non-visual stimulus. Each S is used as its own control in successive attempts to block and establish excitatory conditioning of the eyeblink response to the visual element of a compound conditioned stimulus. Other Ss received excitatory conditioning followed by the blocking procedure.

Kamin (1969b, p. 43) has noted that the concept of attention has increased in respectability with advances in neurophysiology which describe complex and rich interrelations between excitatory and inhibitory elements within the nervous system. Several observations make plausible "filtering" or "gating" effects on stimulus input. In this regard, Hernández-Peón (1964) has proposed that centrifugal modification of neural transmission in the retina is a primitive attentional mechanism. A test of Hernández-Peón's contention was incorporated into the design here described. Half of the Ss received flashes as the source of visual stimulation. The other half received electrical stimulation of the optic chiasma as the visual stimulus. This feature provided a control over the influence of receptor adjustments and neural modification of stimulus input within the retina during compound conditioning. The extent to which blocking and

conditioning of the visual stimulus element is dependent upon the method of visual stimulation yields information about the role of peripheral receptor adjustments and retinal mechanisms in mediating these processes.

Method

Subjects

A total of 24 female New Zealand white rabbits were employed. Each S weighed 3-4 kg at the time of surgery.

Surgery

In preparing S for surgery chloroprothixene (.04 ml, sc) was administered one-half hour before sodium pentobarbital (60 mg/cc, iv) was delivered to effect. Xylocaine-HCl with 2%-epinephrine was administered to locally anesthetize the incision line and stereotaxic pressure points on the zygomatic arch. S was also fitted with two stainless steel wire sutures. One suture was secured 1 cm below S's left lower eyelid, the other, 1 cm caudal to the same eye. A midline scalp incision exposed the skull from in front of the saggital suture to the lambdoidal suture. The periosteum was scraped clear and the tissue reflected and lubricated with a topical ointment (Neosporin). The skull was secured in a Trent-Wells stereotaxic instrument such that the lambda was 1.5 mm below the bregma (Sawyer, Everett, & Green, 1954).

Each S received daily post-operative intramuscular injections of 25,000 units of Penicillin and 0.06-gm dehydrostreptomycin sulfate (Derapen-A, Ayherst Laboratories) for three days.

Electrode Placement. A pair of insulated stainless steel recording electrodes with 0.2-mm tip exposure was positioned across

the left striate cortex such that the depth electrode rested 2.7 mm below the dura while the surface electrode rested on the dural surface at a point 5 mm lateral and 9 mm posterior to the bregma. Two stimulating electrodes with 0.2-mm tip exposure were positioned 1.0 mm apart straddling the midline at a point 2.8 mm anterior to the bregma. The electrode pair was lowered to the optic chiasma, approximately 13.5 mm below the dura, and identified by monitoring potentials generated to a 1-Hz flash presented to S's right eye. The optic chiasma was then stimulated and the vertical position of the electrode pair was adjusted to yield maximum cortical response. A stainless steel 00-90 anchor screw was inserted over the frontal pole providing a reference ground lead. Acrylic dental cement secured the electrodes to additional anchoring screws. All electrode leads were soldered to a multi-connector socket and the entire assembly cemented to the skull. The reflected tissue was sutured closely around the base of the acrylic pedestal.

Histological Procedures

Electrode placement was inspected following the completion of behavioral testing. The tip locus of each electrode was marked within the brain by passing 200 μa of anodal current for 45 sec through each electrode. The Ss were sacrificed with pentobarbital and perfused with 0.9%-saline followed by a solution of 10%-buffered formalin and 2%-potassium ferrocyanide. The brains were placed into a 10%-formalin fixative for no less than four weeks. Gum acacia was added to the fixative to reduce shrinkage of the brains. The brains were dehydrated with ethanol, butanol, and toluene and embedded in histowax (Matheson,

Coleman, and Bell, Inc.). Coronal sections 15-μ-thick of the brain were mounted on slides and Klüver stained (Klüver & Barrera, 1953).

Stimulation Apparatus

The experimental conditions for two essentially identical conditioning chambers (39 X 12 X 18 inches) were programmed using a Tally Model 625 tape reader interfaced with BRS Foringer solid state logic. The chambers were painted flat white, sound-proofed, ventilated, and electrically shielded. Each chamber was divided into two compartments by a dimly illuminated 10 inch-high X 12 inch-wide screen of 1/8-inch flashed opal glass. The forward compartment contained a sound insulated and electrically shielded Grass PS2 photostimulator; the glass lens of the stimulator was replaced with flashed opal glass permitting additional flash diffusion. The strobe unit was positioned 3 inches from the center of the divider screen. S was contained in the rear compartment in a restraining stock similar to that described by Gormezano (1966); the stock was oriented at a 30° angle to the divider screen so that S's right eye was oriented toward the stimulator at an approcimate distance of 12 inches. A brass nozzle with a bore of .063 inches cound be inserted into a block mounted 1.5 inches above S's neck on the back of the head stock. The nozzle directed the tactile CS, a puff of compressed nitrogen, to the dorsal surface of S's neck. The apparatus controlling the delivery of the tactile CS was similar to that described by Gormezano (1966, pp. 399-400) for administering an air puff US. A 4-inch speaker was centered at the base of the divider screen for the delivery of tone CSs. An additional 4-inch speaker was centered on the back wall of the rear compartment for the continuous delivery of white masking noise into the

chamber. Chiasma stimulation was generated by a Tektronix 161 pulse generator and Tektronix 162 waveform generator. Shielded US leads were attached via alligator clips to the wire sutures affixed below S's left lower eyelid. A Model S-8 Grass stimulator delivered the shock-US through an isolation transformer (Grass SIU-5), a step-up 10:1 audio-transformer (Hammond 131), and 175-K ohms of resistance. This arrangement provided current regulation of approximately 95%.

Recording Apparatus

Conditioned eyeblink activity was detected by a system employing two photo transistors (Model LS-400, Texas Instruments) in a bridge circuit. The transistors were attached to opposing edges of half a ping-pong ball shell. The shell was mounted on a buss wire frame which was attached to a multiconnector plug. When the multiconnector plug was inserted into S's receptacle plug the frame straddled S's left eye. One transistor was aimed at the upper portion of S's left eye and the other was positioned over fur caudal to the orbit. A small light bulb in the shell's center diffusely illuminated the eye. After the detector was positioned, approximately 2 cm from S's eye, the bridge was balanced. Potential differences between the photo-transistors were amplified by a Grass P-511 B amplifier. The amplifier was adjusted so that the potential generated by the first US of a session corresponded to approximately +12 v. The output of the amplifier was led to both a Schmidt trigger and a Brush chart recorder (Model Mark II). The Schmidt trigger was gated for 850 msec (950 msec for non-reinforced test trials) starting 150 msec after CS onset. Any gated potential exceeding +0.6 v (5% of a US) was defined as a CR and activated the Schmidt trigger which consequently delivered a marker pulse to the

appropriate channel of the chart recorder. The chart recorder operated at 25 mm/sec with a UR corresponding to a 20-mm pen deflection. The pulse marker was also led to two event channels of a four channel Thermionix FM tape recorder. These channels also received coded pulse markers indicating the treatment condition for each recorded trial.

Experimental Procedure

Two replications of 12 Ss each were conducted with particular emphasis directed to maintaining conditions constant between replications. Certain procedures were standard across all sessions of the experiment unless otherwise noted. Each S was run in the same conditioning chamber at approximately the same time each day. Each session consisted of 90 trials with an average intertrial interval of 40 sec and a range of 30-50 sec for each stimulus condition. Thus, each session lasted 60 min in addition to a 5-min adaptation period which initiated each session.

Stimulus Conditions. The various stimulus conditions employed during training and testing were: visual (V), auditory (A), tactile (T), compound audio-visual (AV), and compound tactile-visual (TV) stimulation. All CSs were delivered for 1,000 msec. The US, initiated at CS offset, consisted of a 100-msec train of 5-msec pulses at a rate of 100 Hz and a pulse amplitude of 8 ma. These parameters were found to provide stable performance and acceptable rates of conditioning. Visual stimulation consisted of either four flashes (10-μsec duration each) delivered by the photic stimulator at a rate of 3 Hz and an intensity setting of 4, or four pulses delivered to the optic chiasma

through a stimulus isolation unit. The optic chiasma pulses had a duration of 50 μsec and an amplitude of twice threshold for the generation of a cortical evoked response. During preliminary investigations, optic chiasma intensities exceeding 2.5 times threshold were found to interfere with blocking of the visual stimulus. Auditory stimulation consisted of a 1,000-Hz 75-db tone as measured at S's right ear (Dawe Instruments sound level meter Model 1400F) against a continuous background level of 64-db white noise. The tactile stimulus was a 3.25-lb/inch^2 puff of compressed nitrogen directed to the dorsal surface of S's neck. Intensities in excess of 4.0 lb/inch^2 were found to produce overshadowing (Pavlov, 1927, p. 269). See Appendix A for a discussion of visual and non-visual stimulus parameters and their relationship to blocking and overshadowing.

Training Procedures. Table 1 summarizes the conditioning and test schedules. Preliminary training was initiated no less than seven days after surgery; this training consisted of adapting S to the experimental setting and differential conditioning of the two non-visual stimuli. During the first adaptation session, S was restrained and placed into the conditioning chamber. During all subsequent sessions, the multi-connector plug and CR detector were secured and the US leads attached. During the third session each S's visual evoked responses to photic and chiasmic stimulation were assessed. Those demonstrating good evoked responses to low intensity optic chiasma stimulation were assigned the Central visual treatment. The other Ss constitute the Peripheral group and received photic stimulation as their visual stimulus. A fourth adaptation session intervened before Pretesting. The 45-min Pretest session consisted of 12 non-reinforced presentations of each of the

TABLE 1

Training and Testing Conditions for Study 1

		Group A					Group T				
		Stimulus					Stimulus				
Sessions	Procedure	T	A	V	TV	AV	T	A	V	TV	AV
4	Adaptation	-	-	-	-	-	-	-	-	-	-
1	Pretest	12_o	12_o	12_o	12_o	12_o	12_o	12_o	12_o	12_o	12_o
(to criterion) +3	Differential Conditioning	45_o	45_+				45_+	45_o			
1	Pseudo-cond. Test	39_o	39_+	12_o			39_+	39_o	12_o		
4	TV Compound Conditioning				90_+					90_+	
1	Test TV	12_o		12_o	66_+		12_o		12_o	66_+	
2	(no training)	-	-	-	-	-	-	-	-	-	-
4	Differential Conditioning	45_o	45_+				45_+	45_o			
4	AV Compound Conditioning					90_+					90_+
1	Test AV		12_o	12_o		66_+		12_o	12_o		66_+

Note.---Each cell indicates the number of stimulus presentations per session and whether that stimulus was reinforced (+) or not (o).

stimulus conditions employed during training. No stimulus condition was repeated twice in succession and each stimulus followed every other stimulus three times with the exception of the audio element which initiated the Pretest and followed the visual element twice. The average inter-trial interval for each stimulus condition was 40 sec. across the 60 Pretest trials. Differential conditioning was initiated the session following the Pretest. Each session consisted of 45 presentations of the auditory and tactile elements; the auditory CSs were reinforced for Group A and the tactile for Group T. Six Peripheral and six Central Ss had been assigned to each group. To more nearly equate all Ss' performances and establish stable conditioning each S was conditioned until meeting a criterion of 40 or more CRs to CS+ and 5 or less to CS- for a session. Conditioning was terminated after 11 differential conditioning sessions for two Ss which failed to reach criterion. Preliminary training was then complete. During each differential conditioning session, the order of stimulus presentations was counterbalanced such that each element followed itself 15 times while the reinforced element followed the non-reinforced element 30 times. The non-reinforced element, which initiated each session, followed the reinforced element 29 times. No stimulus occurred more than three times in succession.

Two days after the last S completed preliminary training the following conditioning schedule was begun. Each S was given three additional sessions of differential conditioning. During the last session, identified as the Pseudo-conditioning Test, the visual stimulus was presented alone on twelve trials replacing six reinforced and six non-reinforced CSs. This test provided control data for evaluating the

influence of US presentations and differential conditioning on the response probability to the visual stimulus. During each of the five subsequent conditioning sessions, 90 TV compound stimulus presentations were 100% reinforced. The fifth session comprised a stimulus control test, Test TV, to determine the degree of stimulus control acquired by each element of the stimulus compound during TV compound conditioning. Twenty-four TV compound presentations were replaced by 12 non-reinforced presentations of each of the elements of the compound. These trials were balanced across the session such that each element occurred twice every 15 trials and never less than two trials after the last non-reinforced trial. Upon completion of Test TV, a two day period of no training intervened before the conditioning schedule was repeated. During the repetition, the Pseudo-conditioning Test trials were omitted from the fourth session of differential conditioning and the AV compound stimulus was employed during compound conditioning.

Study 1 thus comprised a repeated measures factorial design with the following factors and levels: Groups (A and T), Visual Conditions (Peripheral and Central), Tests (Pretest, Pseudo-conditioning Test, Test TV, and Test AV), and Stimulus Elements (non-visual, visual, and compound stimuli). The data were analyzed in terms of response probabilities (P_x, where x represents the stimulus condition) which express S's response frequency to a stimulus as a proportion of the number of times the stimulus was presented within a session.

Results

A comparison of the performances recorded during the Pretest and Pseudo-conditioning Test sessions illustrates the degree of differential

stimulus control established prior to compound conditioning. Figure 1 illustrates the mean response probabilities associated with each stimulus element during these two test sessions. An analysis of variance performed on the arc sine transformed ratios associated with the Groups x Visual Conditions x Preliminary Tests x Stimulus Elements factors is summarized in Appendix B1. Responding to each of the non-reinforced stimuli (i.e., T and V for Group A, and A and V for Group T) did not differ significantly within or between tests ($p > .05$, Duncan's Multiple Range Tests) nor was responding to these stimuli found to significantly interact with the method of visual stimulation. The reinforced element's response probability ratio increased significantly ($p < .01$) following differential conditioning for both Groups A and T. The difference between the performances associated with the reinforced audio element for Group A ($P_A = .872$) and the tactile element for Group T ($P_T = .823$) was not significant.

Examining the pattern of results for the two groups following TV and AV compound conditioning (the shaded columns in Figure 2), it is clear that the paradigm was effective in achieving successive reversal of Ss' performances to the visual stimulus element. The two compound conditioning treatments had inverse effects on the two groups and on their performances to the stimulus elements. Group A showed a high P_V (.677) and a low P_T (.153) during Test TV and the inverse relationship during Test AV, a low P_V (.361) and a high P_A (.625); Group T by contrast showed a low P_V (.285) and a high P_T (.708) during Test TV and a high P_V (.701) and a low P_A (.208) during Test AV. These data reflect a three way interaction of Groups x Compound Conditioning Tests x Stimulus Elements which was significant ($F = 80.02$, $df = 1/20$, $p < .01$)

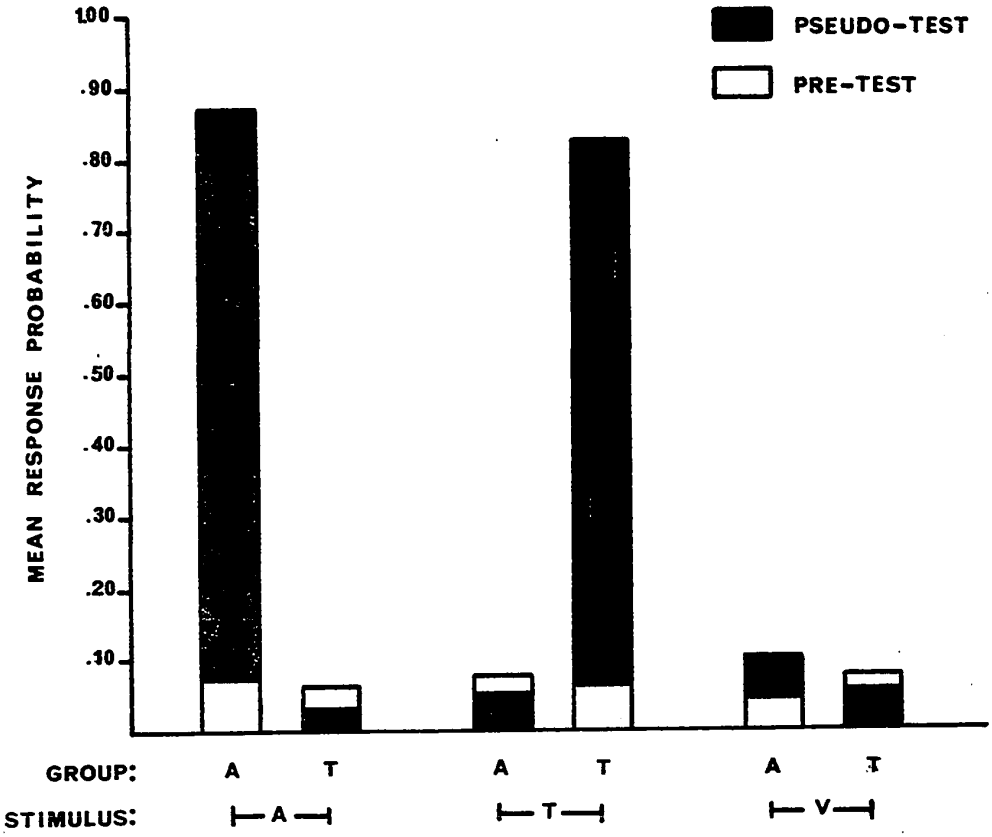

Fig. 1. The mean response probabilities for Groups A and T to the stimulus elements A, T, and V during the Pretest and Pseudo-conditioning tests. Study 1. Behavioral

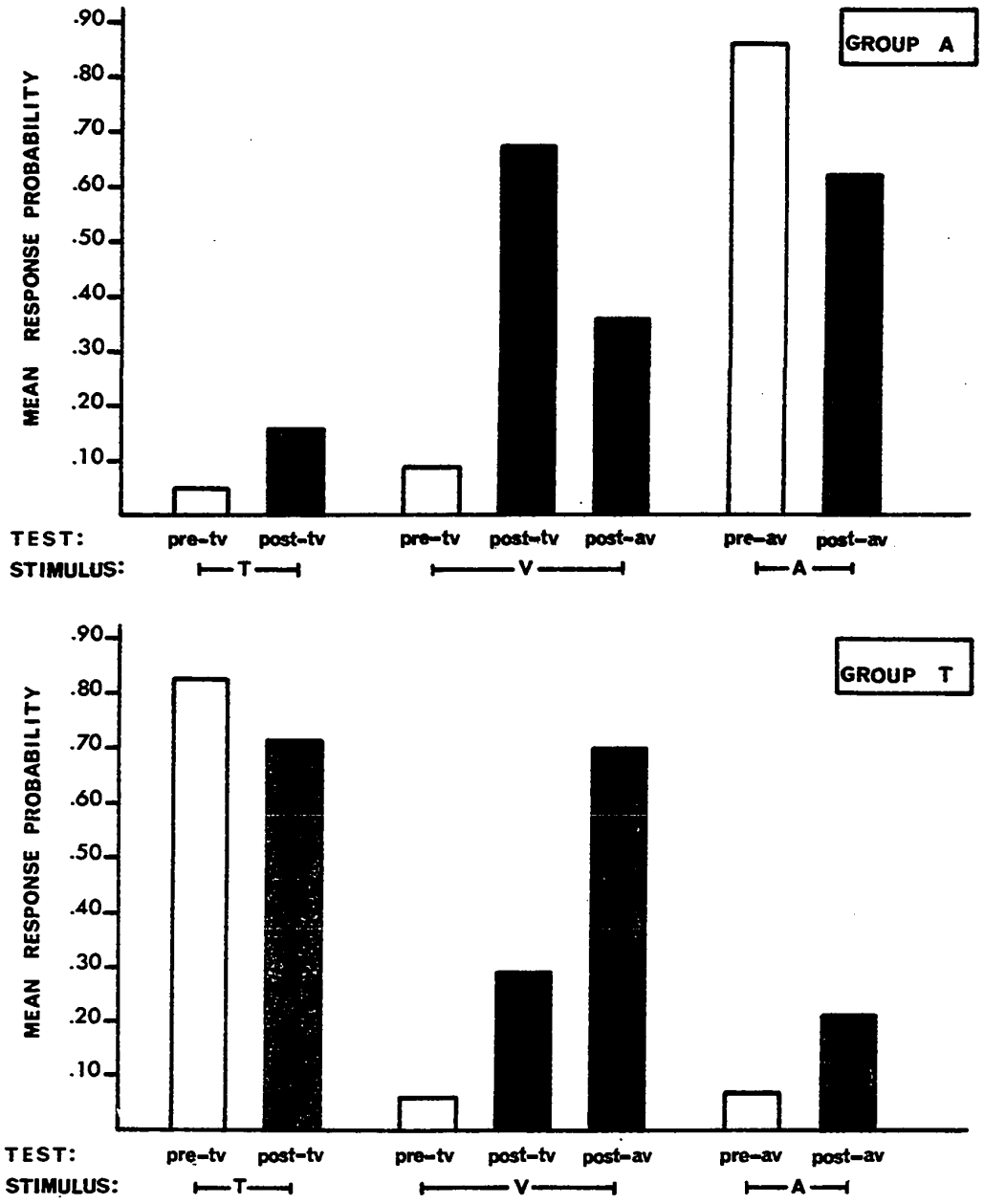

Fig. 2. The mean response probabilities for Group A (upper portion) and Group T (lower portion) to the stimulus elements A, T, and V prior to (pre-) and following (post-) TV and AV compound conditioning. Study 1. Behavioral

as evaluated by an analysis of variance on the transformed response probability ratios assessed during the TV and AV Test sessions (see Appendix B_2). Duncan's Multiple Range Tests between the means of this interaction revealed that the changes in P_V between the two tests, a decrement for Group A and an increment for Group T, were significant ($p < .01$) as were the inverse changes in performance to the non-visual stimuli ($p < .01$).

Figure 2 also illustrates the performance of Groups A and T prior to compound conditioning. The pre-conditioning response probabilities for each element were assessed during the last differential conditioning session prior to the initiation of compound conditioning. The P_V assessed during Test TV was assumed to represent P_V prior to AV compound conditioning. The post-conditioning response probabilities for each element were obtained from the two stimulus control test sessions. The pre- and post-compound conditioning response probabilities associated with the stimulus elements, presented in Figure 2, were subjected to an analysis of variance summarized in Appendix B_3. This analysis enabled an assessment of the significance of changes in the response probabilities of the elements of the compound as a function of each compound conditioning treatment. Of primary interest was a significant Groups x Stimulus Elements interaction ($F = 49.13$, $df = 6/120$, $p < .01$). Multiple comparisons performed on the P ratios associated with this interaction indicate that for Group A there was a significant increment ($p < .01$) in P_V but no change in the response probability associated with the tactile element ($p < .05$) as the consequence of TV compound conditioning; AV compound conditioning significantly reduced both P_A ($p < .01$) and P_V ($p < .01$). Group T, on

the other hand, showed no change in responding to either of the non-visual elements as the consequence of either TV or AV compound conditioning. However, P_V was incremented as the consequence of each of the compound conditioning treatments ($p < .05$ and $p < .01$, respectively). Note that the change in P_V following AV conditioning was significantly incremented above the level of P_V assessed following TV conditioning ($p < .05$).

Figure 3 illustrates P_V for the Peripheral and Central visual conditions during the Pseudo-conditioning Test and following TV and AV compound conditioning. The method of visual stimulation was not found to significantly interact with any factor nor was the main effect significant ($F = 2.19$, $df = 1/20$). P_V was slightly higher under the Central visual condition for Ss in Group A. This reflects the divergent performance of one S (F-55) implanted with an optic chiasma stimulating electrode with faulty insulation. During the Pseudo-conditioning Test P_V was assessed at 50% for this S and 100% during both Test TV and Test AV. This was the only S of Group A under either visual condition not to show a decrement in P_V during Test AV. It should be noted that while this S's behavior was at variance with other Ss relative to the visual stimulus, the P values associated with the non-visual elements remained consistent. The exclusion of this S would only serve to potentiate the effects reported.

Figure 4 (upper portion) illustrates the response probabilities during the first session of compound conditioning for the first seven

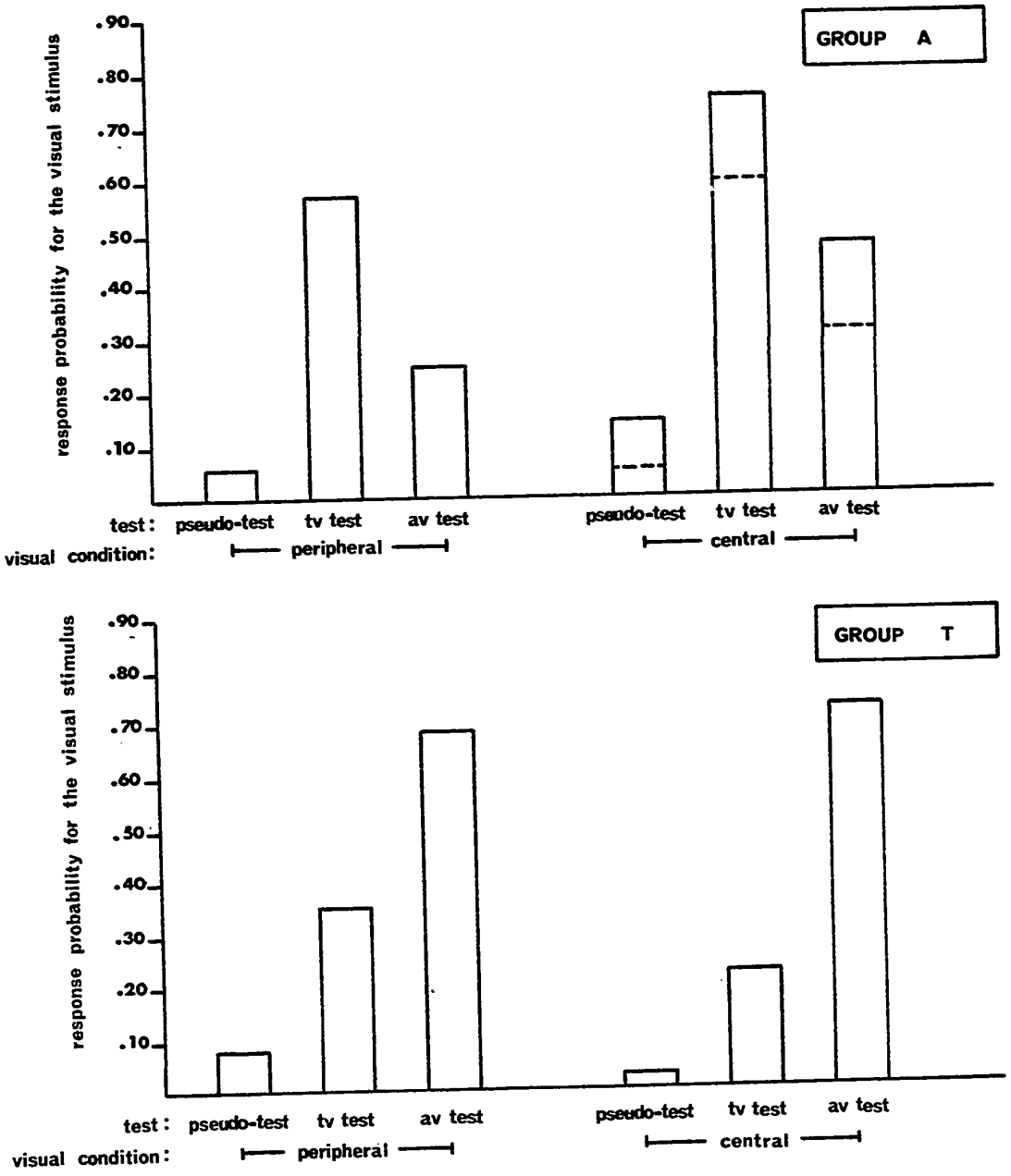

Fig. 3. The mean response probabilities for Group A and Group T to central and peripheral visual stimulation during the Pseudo-conditioning, TV, and AV Tests. The dashes represent the averages after exclusion of a S (F-55) with faulty insulation of a chiasmic stimulating electrode. Study 1. Behavioral

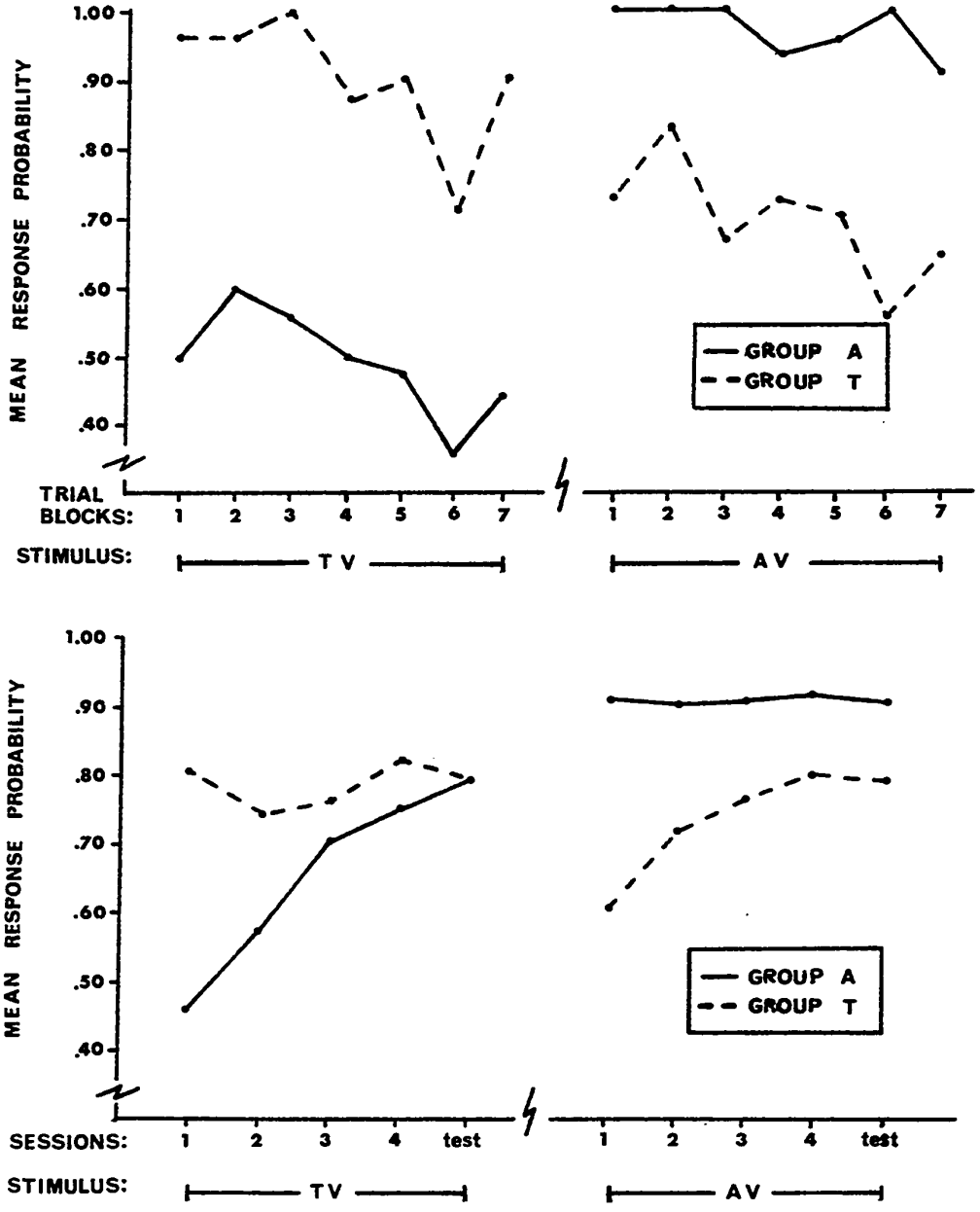

Fig. 4. The mean response probabilities for Group A and Group T to the compound stimuli across the first seven blocks of four trials each during the first TV and AV compound conditioning sessions (upper portion). The lower portion represents the mean response probabilities for Group A and Group T to the compound stimuli during the five TV and AV compound conditioning sessions including the Test sessions.
Study 1. Behavioral

blocks of four trials each. Note a general correspondence between the initial response probabilities and the significance of the non-visual element as established during differential conditioning. Group T at the initiation of AV compound conditioning, however, showed evidence of a carry over of P_V established during TV compound conditioning. The lower graph in Figure 4 illustrates the response probabilities to the compound stimuli for Groups A and T across the five TV and AV compound conditioning sessions. Asymptote, as represented during the test sessions, is similar for each group to each compound (P_{TV} or $_{AV} = \sim .80$) with the exception of Group A which appears to have a higher asymptotic level to the AV compound ($P_{AV} = .909$). This exception is borne out by an analysis of variance on the data represented in the lower portion of Figure 4; the analysis is summarized in Appendix B_4. The Groups x Compound Stimuli x Sessions interaction was significant ($\underline{F} = 15.20$, $\underline{df} = 4/80$, $\underline{p} < .01$). This interaction revealed that Group A exceeded Group T throughout AV conditioning ($\underline{p} < .01$ each session) and achieved a higher asymptotic level during Test AV than evident for either group to TV. Group T exceeded Group A across the initial two sessions of TV compound conditioning ($\underline{p} < .01$); however, P_{TV} for both groups was similar during Test TV. The interaction further reflects the contrasting trends during acquisition by Groups A and T. Group A demonstrated an increasing P_{TV} verified by a significant linear trend ($\underline{F} = 13.81$, $\underline{df} = 1/55$, $\underline{p} < .01$) while showing an asymptotic level of P_{AV} across all sessions of AV conditioning. Group T shows a stable P_{TV} ratio across all sessions of TV conditioning and a significant linear trend ($\underline{F} = 4.33$, $\underline{df} = 1/55$, $\underline{p} < .05$) across AV conditioning sessions.

Discussion

The experimental paradigm was successful in successively manipulating blocking or conditioning of the visual stimulus within groups. Comparable performance was evident to all elements prior to differential conditioning and neither differential conditioning nor the presence of the US altered the response probabilities of non-reinforced stimuli. However, comparable levels of conditioning were evident to the differentially reinforced non-visual stimuli. Conditioning of the visual element was evident following reinforcement in compound with a differentially non-reinforced non-visual element. Blocking of conditioning resulted if the visual element was reinforced in compound with a differentially reinforced element. This was the case following both treatments of compound conditioning.

The results were generally consistent with both the US (Rescorla & Wagner, 1972) and CS (Sutherland & Mackintosh, 1971) attenuation models. In the instance when V was paired with a differentially reinforced stimulus (Group T,Test TV) the response probability associated with element V was low, though significantly increased, and a high response probability was sustained to the non-visual element. The CS attenuation position would maintain that differential reinforcement established attention to the non-visual element and that V was therefore ineffective as a stimulus during compound conditioning. The low P_V would reflect partial blocking of conditioning due to low attention to this element during compound conditioning. The US attenuation position similarly predicts a low P_V maintaining that since conditioning to the differentially reinforced element was at asymptote, the effectiveness of the US would be

attenuated precluding conditioning to V.

The significant increment in P_V when V was paired with the differentially reinforced tactile element is inconsistent with both the US and CS attenuation positions. However, P_V following TV conditioning trials was significantly smaller than P_V after AV trials indicating that at least partial blocking had taken place. Rescorla (1971b) has made a similar observation of incomplete blocking in a conditioned suppression situation. A 2-Hz flashing light was reinforced in compound with a tone which had been previously associated with shock. Incomplete blocking of the visual stimulus, evident after compound conditioning, was explained (p. 118) as the consequence of a stimulus generalization decrement to the tone due to dissimilarity between the preliminary training and compound conditioning situations. This permitted some conditioning to the visual stimulus element. An explanation of partial blocking in terms of stimulus generalization seems unlikely in the present experiment. As an alternative if salience differences between a discontinuous and continuous stimulus are significant then the incomplete blocking reported by Rescorla (1971b) and also noted in our study may be due to a lack of control over this factor. Frey, Englander, and Roman (1971) note that performance is mediated in the serial conditioning of rabbit eyeblink response by CS onset and that learning is dependent on the CS-US contingency. To the extent that the train of visual stimuli was more salient than the continuous tactile stimulus one might argue that the visual stimulus was likely to be attended to when presented alone or in compound thereby causing some disruption of blocking.

When the visual stimulus was paired with a differentially

non-reinforced non-visual element (Group A:Test TV, Group T:Test AV), P_V was incremented and there was no change in responding to the non-visual element. The associative model of Rescorla and Wagner (1972) would maintain that the visual element had a better potential capacity to predict reinforcement than the non-visual element which predicted non-reinforcement (i.e., a conditioned inhibitor with negative associative strength, Rescorla, 1971b, Rescorla and Wagner, 1972) at the initiation of compound conditioning. Reinforcement of the compound would increment the associative strengths of both elements. That the effect of conditioning the non-visual element was not obvious is explainable on the following basis. As a conditioned inhibitor the non-visual stimulus would take proportionally longer than the visual element to acquire excitatory associative strength. Also, once the visual element reached asymptote the effectiveness of the US would be reduced, thus preventing additional conditioning of the non-visual stimulus. Thus, the effect of compound conditioning on the non-visual element would have been disinhibition. Since an assessment of the inhibitory properties of the non-visual element was not incorporated in this design, the extent of disinhibition during compound conditioning cannot be evaluated.

The CS attenuation model is not explicit on what the effect of differential non-reinforcement of a stimulus would be on attention. However, if it is assumed that a differentially non-reinforced stimulus is less likely to be attended to due to the low significance of events subsequent to CS onset, then this would increase the likelihood of S attending to other cues. The consequence would be conditioning to the visual stimulus and little conditioning of the differentially non-reinforced, non-visual stimulus when the two were

reinforced in compound. In this sense, a differentially non-reinforced stimulus (A) when reinforced in compound with a neutral stimulus (B) might initially serve as a cue directing attention to stimulus B, a capacity soon lost as the significance of events subsequent to the compound maintains attention to stimulus B. Independent support for this idea is available in the form of Rescorla's (1971b) demonstration of facilitated excitatory conditioning to a flashing light reinforced in compound with a conditioned inhibitor, a tone. While these speculations are not resolvable on the basis of our data, findings by Rescorla (1969a, p. 74) have been interpreted as evidence that a conditioned inhibitor is attended to. A conditioned inhibitor was shown to reduce "fear" elicited by a conditioned excitor. In any case, the CS-attenuation model seems capable of explaining the performances of Group A:Test TV and Group T:Test AV during which the non-visual stimulus showed little evidence of having become a conditioned excitor.

When both the non-visual and visual elements were paired and had been assessed to have high response probabilities (Group A:Test AV), the test results indicated a decrement in the response probabilities of each element. This is consistent with the US-attenuation model. The effectiveness of a US to establish conditioning is related to the predictability of the US as determined by the associative strength of the CS which precedes the reinforcer. If the associative strength exceeds the level of conditioning supportable by the US intensity then the effectiveness of the US is functionally attenuated with a corresponding diminution of conditioning to the elements. Similarly, if two elements with high associative strengths are paired then the sum of the associative strengths will exceed the asymptote of conditioning

supportable by the US. The effectiveness of the US will be functionally attenuated and a consequent reduction of the associative strengths will ensue during compound conditioning. The results of Group A:Test AV are consistent with this prediction and P_A and P_V were decremented from levels assessed prior to AV conditioning. The decrement in P_V is unlikely to have been the consequence of the non-reinforced presentations of V during the previous Test TV; as will be shown in Study 2, P_V remains unaffected by the few non-reinforced test presentations.

The performance of Group A during Test AV could be explained by two-process attentional theory in the following manner. If two stimuli are paired and each element has a high capacity to elicit attention then S may distribute attention along both dimensions which would result in a decrease in the degree of stimulus control along both dimensions. Alternatively, the number of stimuli to which S is capable of attending may be exceeded. As a consequence only one stimulus would be attended to on any one trial. That is, the more strongly one analyzer is switched in, the less strongly are others switched in (Sutherland, 1964, Rule 2). But since both stimuli have a high attention eliciting capacity then each stimulus has an approximately equal chance of being attended to on any one trial and being reinforced. Under such circumstances S may divide his attention, attending to A on some trials and to V on others. On test trials the reduction in P_A and P_V would be due to S having a "set" (Restle, 1955) to respond to a stimulus which on a certain proportion ($\sim$50%) of the trials would be inappropriate. This of course implies that when stimuli are presented alone they do not elicit attention by overcoming the current "set". Whether S

distributes attention within and/or between trials cannot be assessed here; both interpretations are consistent with the results.

The asymptotic level of AV conditioning for Group A exceeded that observed to other compounds and the elements A and V. Razran (1939) and Weiss (1972) have reviewed research describing an enhancement of response rate or magnitude under similar conditions. Grings (1961); Grings and O'Donnel (1956); Grings and Kimmel (1959); and Grings, Uno, and Fieberger (1965) have noted that when two reinforced elements are paired, the amplitude of the GSR to the compound exceeded the level to either alone and also exceeded the response to a compound comprised of two neutral or a neutral and a positive stimulus. Additive summation has been explained by Weiss (1972) as dependent upon the contingencies predominant during discrimination training. However, the dynamics of additive summation leading to an enhancement of a CR probability remain to be worked out.

Wagner (personal communication) has also noted additive summation of the response probability for a compound comprised of two excitatory elements but that with sustained conditioning the compound response probability assumed a lower level. This latter observation was not apparent with our procedure. Rescorla and Wagner (1972) have maintained that asymptote corresponds with the level of conditioning supportable by the US. Asymptote should, therefore, have been similar for each compound for each group. Additive summation is unlikely to reflect the summation of associative strengths. A likely alternative is that additive summation reflects an enhancement of performance attributable to enhanced attention to a compound comprised of two valid stimuli.

The discussion of the results of AV compound conditioning for

Group A has assumed that little configurational conditioning was established to AV. Several points make configurational conditioning unlikely. If the US was attenuated due to a high P_A additional conditioning would be unlikely. In addition Baker (1968, 1969) has suggested that unless explicit training is introduced to condition a compound then configurational conditioning is unlikely. Egger and Miller (1962) have further demonstrated that little additional conditioning will occur to cues which are redundant predictors of the US; the configuration might be assumed to be redundant in relation to the predictive value of the elements themselves.

In retrospect, the results obtained to the four test situations were consistent with predictions derivable from either the attentional model of Sutherland and Mackintosh (1971) or the associative model of Rescorla and Wagner (1972). In Study 2 both models are tested under a condition in which they make contrasting predictions about performance following extinction and backward conditioning.

The peripheral and central methods of visual stimulation yielded equivalent response probabilities and rates of conditioning throughout all phases of Study 1. Several authors have reported that direct electrical stimulation of the central nervous system yields a higher level of performance and faster conditioning than peripheral stimulation (Patterson & Gormezano, 1968; Colivita, 1969; Loucks, 1938, 1961; Kitai, 1966; Doty, 1961; Doty & Rutledge, 1959). That this was not the case here may have been the result of choosing single electrical pulses of a short duration (50 μsec) rather than trains of longer duration as were generally used in the studies sited above.

The susceptibility of central visual stimulation to blocking and

conditioning suggests that these processes are centrally mediated and do not necessarily involve modification of sensory input at the receptor level. If blocking is to be interpreted in attentional terms and attention is utilized to refer to altered afferent input (Hernández-Peón, 1964), then the relevant modifications must necessarily take place at higher levels in the visual system than the retina. Thus, the modifications in evoked potentials at the optic nerve reported by Hernández-Peón, Guzmán-Flores, Alcaraz, and Fernández-Guardiola (1957) and Palestini, Davidovich, and Hernández-Peón (1959) following manipulation of visual attention do not appear necessary to establish selective blocking or conditioning to visual stimulation. The present data provide no support for Hernández-Peón's hypothesis that attention involves efferent influences which selectively gate sensory input-output relationships at the retinal level.

Study 2. A Partial Replication of Study 1

This study was designed to investigate changes in the geniculostirate excitability cycle associated with alterations in visual stimulus control and arousal. Although the major aim of this study was electrophysiological in nature, the behavioral pardigm is comparable to that employed in Study 1 and warrants inclusion in Chapter I.

Introduction

Six weeks intervened between the conclusion of Study 1 and the first session of Study 2. During the first session each S was subjected to extinction followed by two sessions of backward conditioning with the

visual stimulus. According to the associative model of Rescorla and Wagner (1972) and the research of Rescorla (1971a, 1971b) these procedures should severely reduce the associative strength to the visual stimulus successively producing latent inhibition and conditioned inhibition. Siegal and Domjan (1971) have demonstrated inhibited acquisition of an eyeblink CR in rabbits following exposure to backward CS-US conditioning. However, the two-process attentional model of Sutherland and Mackintosh (1971) would maintain that extinction and backward conditioning would simply reduce S's attention to the visual stimulus leaving associative strength unaltered. The visual stimulus had acquired associative strength during Study 1; reassociating the visual stimulus with the US should redirect attention to the visual stimulus. The consequence would be asymptotic performance early in the initial compound conditioning session.

Method

Subjects

Six Ss from the last replication of Study 1 were selected for inclusion in Study 2. Three Ss were selected from Group A and three from Group T. All Ss had received optic chiasma stimulation as the visual stimulus in Study 1.

Apparatus

The apparatus has been described for Study 1. Only one conditioning chamber, however, was employed in Study 2. Otherwise, only the programming was modified.

Procedure

Six weeks after the completion of Study 1 all six Ss were given

one session of adaptation in the conditioning chamber. An outline of the ensuing training schedule is provided in Table 2. The following session initiated the experimental schedule. Sessions consisted of a five minute adaptation period followed by 96 stimulus presentations. Optic chiasma stimulation at twice threshold was employed as the sole method of visual stimulation. Three 50-μsec pulses were delivered with a 480-msec inter-pulse interval. An additional pulse was introduced between the first and second CS pulses. The latency of this probe stimulus varied in 30-msec steps from 0 to 210 msec after the first pulse. The order of presenting the eight probe intervals was randomized within a session. The probe was introduced for assessing electrophysiological activity initiated by CS_1, the significance of which will be discussed in Chapter II.

The first experimental session comprised a Pretest during which the visual stimulus was presented 96 times without reinforcement. Each of the probe intervals was presented 12 times such that each probe condition followed every other at least once with a 40-sec average intertrial interval for each. The second and third sessions comprised arousal tests during which the Pretest schedule was repeated with each stimulus presentation preceded by a 100-msec presentation of the US. During Arousal Test I the US preceded onset of the visual stimulus by 250 msec. During Arousal Test II the US-CS interval was 2,000 msec. The sessions immediately following Arousal Test II consisted of differential conditioning consistent with S's Group (A or T) assignment. Differential conditioning continued for each S for seven sessions or until a criterion of 43 or more CRs to CS+ and seven or less to CS- for a session was met. TV compound conditioning was initiated the following

TABLE 2

Training and Testing Conditions for Study 2

		Group A					Group T				
		Stimulus					Stimulus				
Sessions	Procedure	T	A	V	TV	AV	T	A	V	TV	AV
1	Adaptation	-	-	-	-	-	-	-	-	-	-
1	Pretest			96_o					96_o		
1	Arousal Test I			$+96_o$					$+96_o$		
1	Arousal Test II			$+96_o$					$+96_o$		
(to criterion)	Differential Conditioning	48_o	48_+				48_+	48_o			
4	TV Compound Conditioning				96_+					96_+	
1	Test TV	8_o		8_o	80_o		8_o		8_o	80_o	
2	(no training)	-	-	-	-	-	-	-	-	-	-
4	Differential Conditioning	48_o	48_+				48_+	48_o			
4	AV Compound Conditioning					96_+					96_+
1	Test AV		8_o	8_o		80_+		8_o	8_o		80_+
1	reTest AV		8_o	8_o		80_+		8_o	8_o		80_+

Note.--Each cell indicates the number of stimulus presentations per session and whether that stimulus was reinforced (+) or not (o). A "+" preceding the stimulus indicates that the US preceded the stimulus as during the two arousal tests. The US preceded V by 250 msec for Arousal Test I and by 2,000 msec for Arousal Test II.

session and continued for five sessions. The fifth session constituted the TV stimulus control Test during which S received eight non-reinforced presentations of T and a single non-reinforced presentation of each of the eight visual stimulus probe intervals. These trials were interspersed among 80 reinforced TV presentations. After a two day rest of no training, differential conditioning was repeated for four sessions followed by AV compound conditioning and Test AV. Test AV was repeated the subsequent session.

Results

Only one CR to the visual stimulus element was recorded among all six Ss within the first ten trials of the Pretest. Figure 5 illustrates the response probabilities associated with the visual stimulus during the Pretest and two arousal Tests. The analysis of variance performed on the arc sine transformed response probabilities is summarized in Appendix C_1. The significance of the Tests main effect ($F = 98.21$, $df = 2/8$, $p < .01$) reflects a significant increment in P_V between the Pretest ($P_V = .012$) and both Arousal Test I ($P_V = .235$, $p < .01$) and Arousal Test II ($P_V = .059$, $p < .05$) and a significant decrement ($p < .01$) between Arousal Test I and II. There were no differences between Groups A and T across the three Tests.

Figure 6 illustrates the performances of Group A (upper portion) and Group T (lower portion) during Test TV and AV (the shaded columns). A comparison of the results of these tests reveals a trend consistent with that observed in Study 1. Group A showed a high P_V (.833) and a low P_T (.000) following TV compound conditioning and the inverse relationship following AV conditioning, a low P_V (.292) and a high

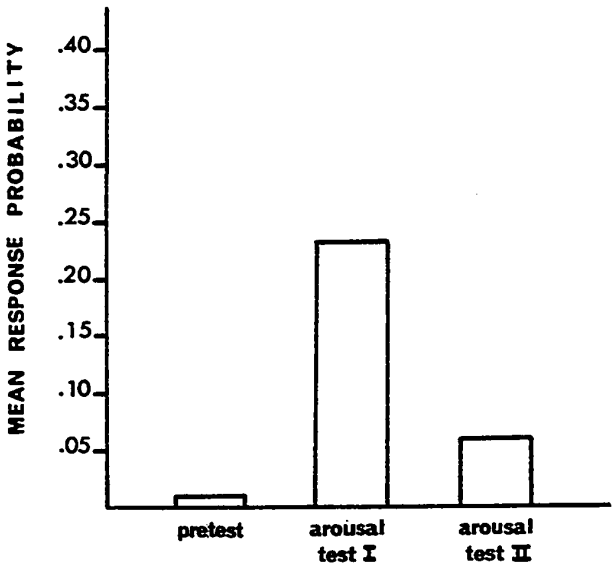

Fig. 5. The mean response probability of all Ss (N = 6) to optic chiasma stimulation during the Pretest (extinction), Arousal Test I (US-CS interval 250 msec), and Arousal Test II (US-CS interval 2,000 msec). Study 2. Behavioral

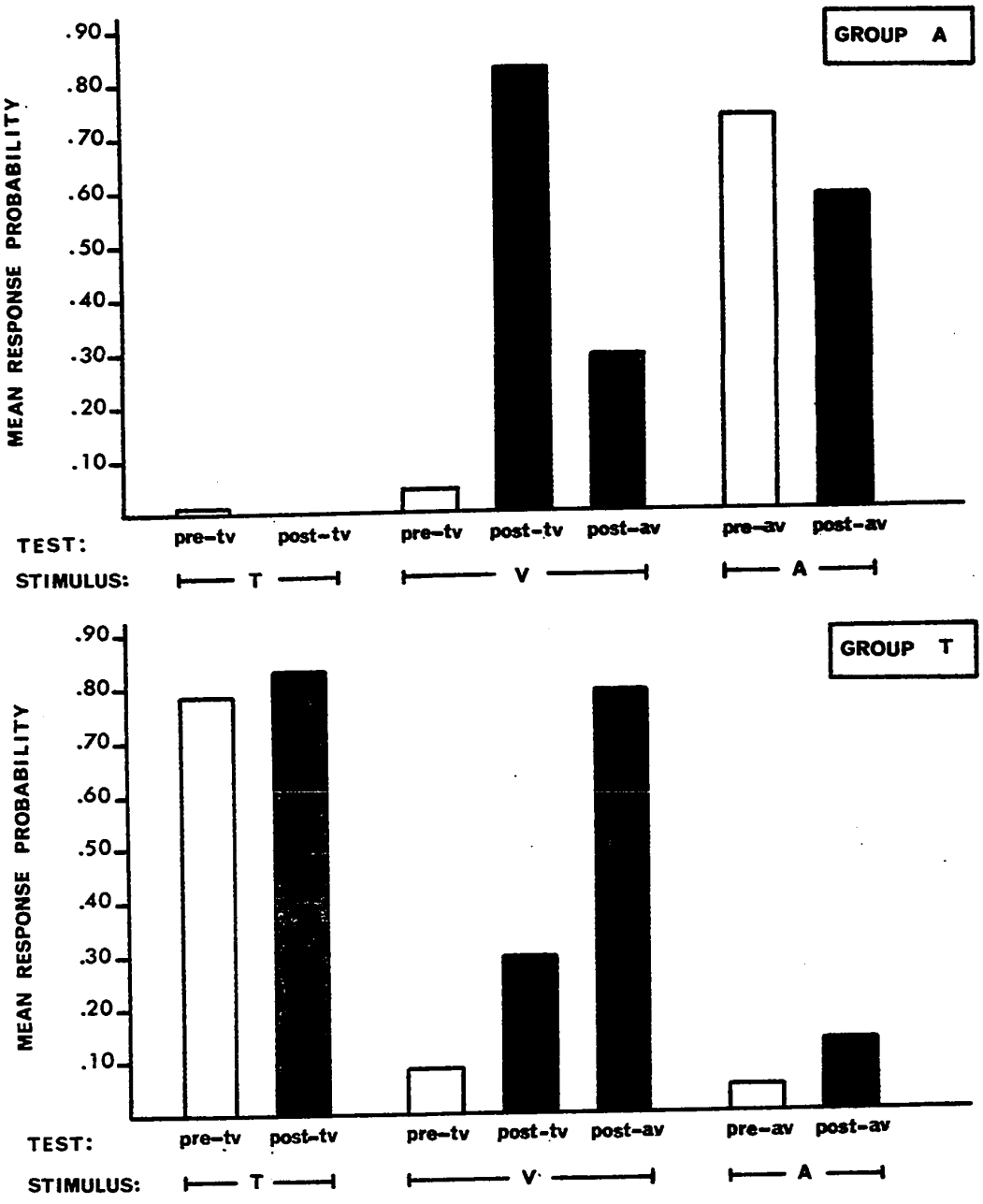

Fig. 6. The mean response probabilities for Group A (upper portion) and Group T (lower portion) to the stimulus elements A, T, and V prior to (pre-) and following (post-) TV and AV compound conditioning. Study 2. Behavioral

PT (.833) following TV conditioning and a high P_V (.792) and a low P_A (.125) following AV conditioning. These observations are borne out by an analysis of variance on the transformed post-compound conditioning response probabilities; a summary of the analysis is presented in Appendix C_2. The Groups x Compound Conditioning Treatments x Stimulus Elements interaction ($F = 38.42$, $df = 1/4$, $p < .01$) confirmed the inverse relationship between Groups A and T on the stimulus elements following the two compound conditioning treatments. The changes in P_V following AV conditioning, a decrement for Group A and an increment for Group T were both significant ($p < .05$). The changes in the non-visual response probabilities following TV conditioning, an increment for Group A and a decrement for Group T, were also significant ($p < .05$).

The data of Figure 6 also reflect the nature of the changes induced in the response probabilities for the stimulus elements following compound conditioning. For lack of a more appropriate measure the response probability associated with V prior to compound conditioning was assessed as the P_V determined during Arousal Test II; this probably inflates P_V somewhat. Following AV conditioning P_A was decremented for Group A though not significantly. P_V, however, was incremented significantly for Group A ($p < .01$) and Group T ($p < .05$) following TV and AV conditioning, respectively. Group T showed a non-significant increment in P_V following TV conditioning while Group A showed a significant decrement ($p < .01$) following AV conditioning. The analysis of variance performed on this data is summarized in Appendix C_3.

The upper portion of Figure 7 illustrates the response probabilities associated with the compound stimuli for Groups A and T

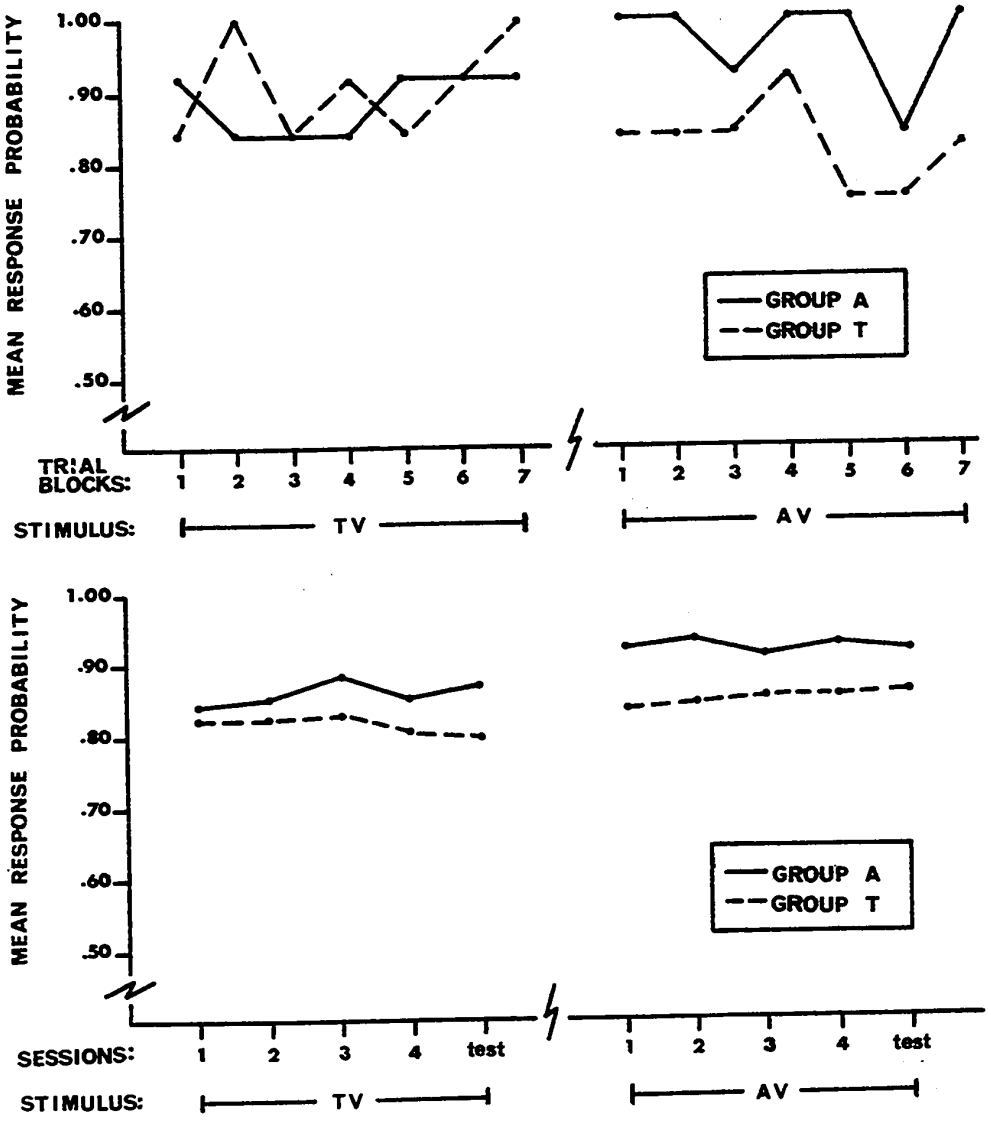

Fig. 7. The mean response probabilities for Group A and Group T to the compound stimuli across the first seven blocks of four trials each during the first TV and AV compound conditioning sessions (upper portion). The lower portion represents the mean response probabilities for Groups A and T to the compound stimuli during the five TV and AV compound conditioning sessions including the test sessions.
Study 2. Behavioral

across the first seven blocks of four trials each for the first TV and AV compound conditioning sessions. It is obvious from this data that Ss in all groups were responding close to asymptote to the compound at the initiation of the session contrasting with what was seen in Study 1 (see Figure 4, top portion, p. 21). Inspection of the lower portion of Figure 7, representing the response probabilities across the five TV and AV conditioning sessions, indicates no obvious changes in the compound response probabilities during conditioning; performance was stable and high across all sessions. An analysis of variance on the arc sine transformed P ratios for the AV and TV compounds (see Figure 7, lower portion) revealed no significant interactions or main effects. This analysis is presented in Appendix C_4. Note that Group A exceeded Group T across all AV conditioning sessions. However, the lack of a significant Groups x Compound Conditioning Treatments x Sessions interaction ($F = 1.28$, $df = 4/16$) suggests that there were no significant differences in the groups' performances across either series of compound conditioning sessions, that is, P_{AV} and P_{TV} were asymptotic across all sessions for each group.

The mean response probabilities for each element for each group as assessed during Test AV and reTest AV are presented in Table 3. The within group's performances on the elements between the two tests were virtually identical.

TABLE 3

Response Probabilities for Test AV and reTest AV

		Group	
Stimulus	Test	A	T
A	Test AV	.583	.125
	reTest AV	.583	.125
V	Test AV	.305	.792
	reTest AV	.292	.792
AV	Test AV	.917	.858
	reTest AV	.917	.854

Discussion

The low response probability to the visual stimulus during the early trials of the Pretest suggests there was little retention of the conditioning to V evident during Study 1. The fact that P_V was incremented when V was preceded by a US is likely the consequence of sensitization or arousal facilitating the eyeblink response. That P_V was inversely related to the US-CS interval between the two arousal tests supports this suggestion although the influence of the order of the tests could have been a contributing factor to the reduction of P_V during the second test. It is unlikely that the increased P_V reflects a secondary eyeblink following the US. Inspection of the CR records during conditioning indicated that post-UCR blinks rarely occurred.

The results of Study 2 generally reconfirm what was observed in Study 1. However, two observations relating the pre- and post-compound conditioning data were inconsistent. When V was paired with A for Group A, both having been assessed as having high associative strength,

A was decremented as in Study 1 though not significantly. The other instance followed TV conditioning; Group T showed an increment in V as in Study 1 but the increment was not significant. This may have been the consequence of employing an inflated preconditioning P_V value assessed during Arousal Test II. Otherwise the directions of change associated with the stimulus elements were consistent with the results and interpretations discussed for Study 1. The above lack of significance may also have been the consequence of employing a small number of Ss ($N = 3$) per group.

The performance of Group A during AV compound conditioning again suggests a summative effect of P_A and P_V thereby yielding a higher P_{AV} to this compound than demonstrated by either group to compounds comprised of at least one element with a low response probability.

The performance of Groups A and T across the compound conditioning trials reflects a different trend than was seen in Study 1. In Study 1, Group A showed an increasing P_{TV} across TV compound conditioning sessions and Group T showed an increasing P_{AV} during AV compound conditioning. During Study 2 no evidence of such a change in responding to the compound across or within the conditioning sessions was observed. Consider Group T during and following AV compound conditioning; prior to AV conditioning P_A was low as assessed during the last differential conditioning session and P_V was low as determined during Test TV. If it is assumed that P_V, as determined during Test TV, reflected a low associative strength then when A and V were reinforced in compound a low compound response probability should have been initially evident with an increasing value across the conditioning trials as V reacquired associative strength. However, as observed, an asymptotic level of P_{AV}

was evident within the first four compound conditioning trials. This would suggest that the associative strength of P_V or P_{AV} was high prior to AV compound conditioning and that the low P_V assessed during Test TV for Group T was due to a performance decrement, i.e. S was not attending to the visual element despite its high associative strength. Similar reasoning may be extended to explain the asymptotic performance of Group A upon the initiation of TV compound conditioning. Both P_T and P_V were assessed prior to TV compound conditioning to be low; V had been subjected to three prior sessions of extinction and backward conditioning. Yet P_{TV} was asymptotic at the initiation of TV compound conditioning and P_V was assessed as high following TV conditioning and P_T was equal to zero. Such a differential response probability would suggest that configurational conditioning did not occur. It follows from these observations that the extinction and backward conditioning procedures did not effect a reduction in associative strength to V established six weeks earlier.

The associative model of Rescorla and Wagner (1972) might argue that the extinction procedure produced latent inhibition of V thereby retarding the subsequent establishment of conditioned inhibition during backward conditioning of V. However, since latent inhibition refers to a reduction in stimulus saliency (i.e., CS attenuation) such an argument incorporates an attentional construct.

The findings of Study 2 are consistent with the argument that performance during the extinction-backward conditioning sessions reflected low attention to the visual stimulus. Reinstating the CS-US contingency restored attention and resulted in an increase in performance, the consequence of redirecting attention to the stimulus

having the higher associative strength, the visual stimulus. The point to be emphasized is that a response probability estimate without additional conditioning data, i.e., acquisition data, does not enable a reliable evaluation of the associative strength of a stimulus when performance is low.

Of additional significance in Study 2 was the observation that there was little change in the response probabilities following Test AV and reassessed during the second AV Test. This indicates that the influence of non-reinforced presentations of the stimulus elements during Test AV was insignificant on the response probabilities of the elements or compound on the subsequent reTest. Thus, the decrement observed to V following AV compound conditioning by Group A in both Study 1 and Study 2 is unlikely to have been the consequence of the non-reinforced test trials of Test TV.

Again the results of Study 2 have been discussed assuming that configurational conditioning was not a significant consideration. The same arguments discussed in connection with Study 1 are applicable here. In addition, if configurational conditioning had occurred in Study 2, it is not apparent in the compound conditioning data; performance was at asymptote immediately upon the initiation of compound conditioning. The possibility that configurational conditioning had occurred in Study 1 and was transferred to Study 2 is also unlikely. The differential performance of the groups to the visual and non-visual stimulus elements on all tests, with the possible exception of Group A to Test AV, indicates that the two elements acquired differential associative strengths, a result inconsistent with what would be expected had the compound acquired associative strength independently

of the elements.

The similarity of the results between the two studies suggests that the experimental paradigm employed is powerful enough to reestablish conditioning and to reverse the response probabilities to the visual element despite: (1) extinction and backward conditioning given to V prior to differential conditioning, (2) previous exposure of all Ss to the experimental paradigm, and (3) inclusion of direct electrical stimulation of the optic chiasma as the exclusive source of visual stimulation.

In the subsequent research on changes in the evoked potential, the data of Group A during Test AV and Group T during Test TV have been pooled constituting what will be referred to as a Non-visual Stimulus Control (NVSC) Test since non-visual control was consistently demonstrated under these conditions to exceed visual stimulus control. Similarly, the data of Group A during Test TV and Group T during Test AV have been pooled constituting a Visual Stimulus Control (VSC) Test.

CHAPTER II

ELECTROPHYSIOLOGICAL ACTIVITY OF THE GENICULO-STRIATE SYSTEM TO OPTIC CHIASMA AND PHOTIC STIMULATION

Visual evoked potentials were averaged during each conditioning session and test session throughout Study 1. The study enabled an assessment of whether changes in the evoked potential accompany changes in visual stimulus control and whether such changes are associated with fluctuations in arousal or with the selective aspect of stimulus control. In addition, to assess the role of peripheral and central structures in modifying components of visual evoked potentials, information was sought on whether any changes depended on the source of visual stimulation.

Study 1. Geniculo-striate Evoked Potentials During Selective Stimulus Control

Central to recent work on selective attention and the analysis of sensory evoked potentials has been a proposition that "some changes must take place in the central nervous system, and perhaps the sense organs, as a result of which the capacity of certain stimuli to evoke a perceptual or behavioral response is diminished" (Horn, 1965, p. 155). Earlier, Adrian (1954) proposed that a controlling mechanism might operate on the transmission of afferent impulses at some subcortical level so that when a stimulus is "attended" irrelevant stimuli would be selectively attenuated thereby excluding the respective sensory impulses from higher perceptual centers in the central nervous system.

Initial support for these speculations was obtained by Hernández-Peón and his colleagues in the 1950's. Hernández-Peón, Guzmán-Flores,

Alcaraz, and Fernández-Guardiola (1957) noted a decrement in photic potentials of the cat at the level of the optic tract if S "attended" auditory or olfactory stimulation. A similar reduction in auditory evoked potentials had been reported at the level of the cochlear nucleus associated with the presentation of a distracting stimulus or reticular stimulation (Hernández-Peón, Scherrer, & Jouvet, 1956). Two severe criticisms have been advanced against these researches and subsequent work attempting to relate changes in the sensory evoked potential to a mechanism capable of selectively "filtering" afferent input on the basis of stimulus relevance.

First, there was a lack of control for changes in receptor adjustments accompanying the presentation of "distracting" stimuli. Changes in pupillary activity (Hess & Polt, 1964; Lynn, 1966; Voronin, Leontiev, Luria, Sokolov, & Vinogradova, 1965), accomodation and ocular fixation (Oswald, 1959), and saccadic eye movements (Barlow, 1952) have each been associated with altered arousal or attention-like orienting activity. Changes in pupil diameter have been associated with altered photic evoked potentials (Naquet, Fischer-Williams, & Fernández-Guardiola, 1960; Fernández-Guardiola, Harmony, & Roldán, 1964; Fernández-Guardiola & Eibenshutz, 1961; Naquet, Regis, Fischer-Williams, & Fernández-Guardiola, 1960; Palestini, Gallardo, & Armengol, 1964; Affanni, Mancia, & Marchiafava, 1962). Further, the orientation of the receptors is logically critical in determining the physical impingement of stimuli on the receptors (Horn, 1960, 1965). Thus, it is not clear whether changes in the photic evoked potential are due to a subcortical filtering mechanism (as suggested by Hernández-Peón,

1964) or simply reflect the dynamics of the peripheral receptor apparatus associated with arousal and/or orienting behavior.

Second, arousal associated with the presentation of a distracting stimulus is often poorly controlled and not entertained as an alternative determinant of the reflected changes in the evoked potential usually attributed to selective attention. Näätänen (1967) reviewed the early "attention" research supporting an attenuation of evoked potentials during "non-attention" or an increment during "selective attention" (e.g., with animals, Hernández-Peón et al., 1956, 1957; Jane, Smirnov, & Jasper, 1962; García-Austt, Bogacz, & Vanzulli, 1964; Horn, 1960; Horn & Blundell, 1959; Palestini, Davidovich, & Hernández-Peón, 1959; Hernández-Peón, 1959; Hernández-Peón & Brust-Carmona, 1961; with humans, Spong, Haider, & Lindsley, 1965; Haider, 1967; Satterfield, 1965; Satterfield & Cheatum, 1964; Chapman, 1965; Chapman & Bragdon, 1964; Davis, 1964). Näätänen notes that in many of these works the stimuli were presented either alternately and/or at regular intervals enabling S to anticipate stimulation. Näätänen demonstrated that if auditory stimuli were presented in sequence so that onset of stimulation could be anticipated or predicted, the auditory evoked potential was facilitated. This was not the case if stimuli were presented in an unpredictable sequence. He suggests that diminished evoked potentials reflected lowered arousal and enhanced evoked potentials reflect increments in arousal:

> Electrophysiological changes taking place in the central nervous system during selective attention, such as reticular activation, alpha blockade, "Expectancy"-waves etc. reflect only the increased non-specific arousal and activation connected with attention states, and not the aspect of selectivity or direction of these states. (Näätänen, 1967, p. 179).

Similarly, Karlin (1970) has suggested that changes in late activity of the evoked potential are correlates (or even artifacts) resulting from changes in the wave of contingent negative variation (CNV) -- a wave he regards as an arousal response in anticipation of significant stimulation.

While there has been little evidence to dispute Näätänen's contention (see Thompson, Patterson, & Teyler, 1972) the relationship between altered levels of arousal and changes in the visual evoked potential is far from clear. A reduction in the striate response to photic stimulation has often been reported as the consequence of arousal induced by reticular stimulation (Bremer & Stoupel, 1958, 1959a, 1959b; Hernández-Peón, Scherrer, & Velasco, 1956; Bremer, Stoupel, & Van Reeth, 1960; Dumont & Dell, 1960; Long, 1959), arousal from sleep (Evarts, Fleming, & Huttenlocher, 1960; Fleming & Evarts, 1959), and arousal induced by novel stimulation (Walley & Urschel, 1972, Skrebitsky, 1962, Hernández-Peón et al., 1957, Dumont & Dell, 1960; see also Appendix D). However, enhanced potentials with increased arousal have also been reported (Lindsley, 1961; Schwartz & Shagass, 1963; Steriade & Demetrescu, 1960, 1962; Gellhorn, Koella, & Ballin, 1954; Mancia, Meulders, & Santibañez, 1959a, 1959b). Fuster and Docter (1962) report an enhancement of the late activity of a photic response following reticular stimulation or the administration of amphetamines to rabbits and an attenuation following barbiturate anesthesia. Eason and Dudley (1971) noted that heart rate, muscular tension, skin conductance, and the primary and secondary phases of the cortical photic potential are increased between low, moderate, and high levels of arousal. Arousal and presumably attention were manipulated by requiring S to passively

observe (low arousal) or to respond to random flashes under no threat (moderate arousal) or threat of shock (high arousal). The level and/or duration of arousal may be crucial in explaining the apparent paradox in the above reports. Further, peripheral and central stimulation have been reported to be differentially affected by increments in arousal (Dumont & Dell, 1958, 1960; Bremer & Stoupel, 1959a, 1959b; Walley & Urschel, 1972). The locus of the recording electrode may also be critical as Thompson, Denny, and Smith (1966) report that the latter components of potentials recorded from the striate cortex of cats are markedly enhanced following stimulation of the frontal cortex while potentials evoked in the association cortex are abolished. They conclude that both enhancement and attenuation occur during increased arousal and attention depending on the region of the cortex from which the potential is recorded. Thompson (1967) has also emphasized that primary and secondary components of the cortical evoked potential may be differentially affected by operations designed to manipulate attention and/or arousal.

The complexity of the mechanism mediating changes in the photic evoked potential is indicated by the work of Palestini, Davidovich, and Hernández-Peón (1959). Photic potentials recorded from the optic tract of the cat were diminished when S was attending a white rat. Intramodal differentiation represents a severe test for any model proposing to account for selective attention. Horn (1960) and Horn and Blundell (1959) have also reported evidence of intramodal effects associated with visual searching in cats. Donchin and Cohen (1967) observed a depression of photic potentials if S was attending a Necker cube but an enhancement if S was asked to count the flashes and ignore the background

Necker cube. To explain intramodal differentiation Hernández-Peón (in Palestini et al., 1959, p. 125) suggested the operation of a "subtle functional organization . . . from the centrifugal fibers to the retina" acting to selectively gate or filter afferent input. Such a mechanism, however, would have to be exceedingly complex at the retinal level and it is not clear how selective gating is initiated to peripherally block irrelevant sensory input at stimulus onset.

Conditioning procedures have also been employed in the investigation of changes in electrophysiological activity associated with selective attention. This procedure has the advantage of reducing habituation and of providing the experimenter with a means of gaining control over the overt and presumably the covert attention of Ss. Palestini et al. (1959) noted a significant increment in the visual evoked potential of cats if flashes were paired with electrical shock. Hearst, Beer, and Sheatz (1960) using a variety of instrumental and classical conditioning procedures with monkey report an enhancement of auditory evoked potentials in a variety of brain regions. The enhancement was most dramatic during the early stages of conditioning and when negative reinforcement was employed. While a pseudo-conditioning test was not employed, EEG desynchrony and increased heart rate were associated with the presentation of the CS; the enhancement seemed specific to the reinforced stimulus. The enhancement of an evoked potential to a conditioned photic stimulus has been reported in several investigations with animals (Macadar, Gines, Bove, & García-Austt, 1963; Jouvet, 1956; Fleming, 1967; Hernández-Peón et al., 1957; Pickenhain & Kingberg, 1965; Klingberg & Grastyán, 1963).

The above conditioning studies generally recorded the evoked

potential prior to conditioning and conclude that an enhancement of the evoked potential to the conditioned stimulus represents a facilitation as the consequence of enhanced stimulus relevance. However, Diamond and Chow (1962), Morrell (1961), Jasper (1961), Thompson (1967), Thompson et al. (1972), like Näätänen (1967), suggest that the consequence of conditioning may be to increment tonic (Sharpless & Jasper, 1956) non-specific arousal. Tonic arousal refers to the more-or-less steady state, background or baseline, level of arousal best assessed just before CS onset. An enhanced potential is interpreted as simply reflecting sensitization such that any stimulus regardless of its significance would be enhanced. In fact, Hall and Mark (1966) and Mark and Hall (1967) noted that facilitation of late activity of auditory evoked potentials generalized to potentials elicited by photic stimulation after establishment of a conditioned emotional response to the auditory stimulus in the rat. Two other studies also controlled for sensitization with similar findings: Gerken and Neff (1963) and Buser, Jouvet, and Hernández-Peón (1958). Galambos, Sheatz, and Vernier (1956) have excluded movement artifacts as a factor mediating enhanced auditory potentials following conditioning of cats paralyzed with Flaxedil. Hall and Mark using semi-restrained rats also ruled out movement artifacts.

A further characteristic of tonic arousal is its apparent susceptibility to habituation (Sharpless & Jasper, 1956). There is substantial evidence indicating that there is a weakening of arousal across trials as conditioning is extended beyond asymptote. Beck, Doty, and Kooi (1958) report an increase in EEG desynchrony only during early leg flexion conditioning in cats; Babiyan (1961) and Thompson and Obrist

(1964) also report maximal EEG changes only during the active or early phases of learning. Andreassi and Whalen (1967) also found an increase in heart rate, GSR, and palamar conductance during learning and a decrease with overlearning of nonsense syllables. Further, Fleming (1967) reports an increase in the late positive-negative component of a photic response during early leg flexion conditioning of cats and a decrease with overtraining and extinction. Enhancements of secondary activity are generally associated with increments in arousal associated with reticular activation (Pickenhain & Klingberg, 1965, Klingberg & Grastyán, 1963, Fuster & Docter, 1962).

A distinction is also warranted between tonic arousal assessed between trials and phasic arousal (Sharpless & Jasper, 1956) initiated subsequent to, or in anticipation of, significant stimulation and peaking at, or prior to, response emission. Kahneman, Turskey, and Criders (1969) noted an increase in arousal (increased heart rate, pupil dilation, and skin resistance) upon the receipt and processing of information on a digit transformation task. The autonomic activity peaked during the verbal report of the transformation and then decreased. Ehrlich and Malmo (1967) report a phasic increase in heart rate peaking at the moment of a bar press for food in rats.

Phasic arousal, particularly if conditioned, could be viewed as a possible explanation for what appear to be selective increases in the amplitude of evoked potentials (e.g., with CS+ and CS- differential reinforcement designs). For example, in a recent study Saunders (1971) reports systematic changes in visual and auditory evoked activity related selectively to the behavioral significance of a stimulus. Cats were trained to avoid shock by performing a "tilting" response to either an auditory or visual stimulus. When the visual stimulus was

employed as the CS the auditory stimulus was presented continuously as a background stimulus. The procedure was reversed if the auditory stimulus served as CS. Performance was monitored and pretest and sensitization tests were performed. Saunders reports an enhancement of late secondary activity at the visual cortex and auditory cortex if visual or auditory stimulation, respectively, was the CS. A reversal of the CS contingencies resulted in a reversal of the enhancement to the new CS during reacquisition. The selective enhancement is likely an electrophysiological correlate of the selective conditioning of a phasic arousal reaction to CS+. The reported changes were, however, transient; secondary amplitudes increased during the early stages of conditioning and decreased to baseline levels subsequent to the establishment of the avoidance response. Tonic arousal apparently decreased upon the attainment of the avoidance response.

The blocking paradigm incorporated in Study 1 has features which should enable a valid assessment of several issues related to alterations of the visual evoked potential and the influence of selective stimulus control and arousal. First, tonic arousal should be constant across conditions subsequent to the Pretest. Secondly, phasic arousal in response to the reinforced compound stimulus should occur regardless of which element of the compound stimulus is exerting stimulus control. Thus, if changes in the visual evoked potential are only due to phasic arousal there should be no difference between the responses to the NVSC and VSC compounds.

The independent variables of interest are the groups, methods of visual stimulation, test conditions, sessions associated with compound conditioning, and a CS factor referring to each stimulus of the 3-Hz

visual stimulus train. The order of establishing VSC and NVSC and/or whether there are differential effects of auditory and tactile stimuli on the visual potential during compound presentations are assessed by comparing the evoked potentials of Ss from Groups A and T. The contribution of receptor modifications on afferent input is assessed by comparing changes in the photic response with changes in the response to electrical stimulation of the optic chiasma. The influence of arousal and stimulus control are assessed by comparing changes in the evoked potential as a function of the various test conditions. The influence of the acquisition of visual stimulus control is assessed across the sessions of compound conditioning. Phasic processes initiated by stimulus onset should be reflected in differential responses across the 3-Hz visual stimulus train.

Method

The Ss, apparatus, and procedures for Study 1 have been elaborated in Chapter I. The only items requiring additional description are the equipment and techniques used in recording, averaging, and evaluating the electrocortical potentials subsequent to visual stimulation.

Recording Apparatus

Cortical activity was led through a Grass high impedence probe to a Grass P-511/D AC-coupled amplifier with a half amplitude bandwidth of 0.1 Hz to 30 kHz. The signals were recorded on a Thermionix FM tape recorder while responses to visual stimulation alone were simultaneously averaged on a CAT 1000 (Technical Instruments Corp.). Digital output onto paper tape was obtained by means of a Model 535 Teletype (Technical Instruments Corp.) interfaced with the CAT. AEPs were also output onto

an X-Y plotter (Model 2-D, F. L. Moseley Co.). Off-line processing of the digital tapes was done on a PDP-8/I general purpose computer (Digital Equipment Corp.).

AEP Analysis

The AEPs were accumulated in the following manner. Each AEP was based on 12 presentations of the visual stimulus alone during a test session or 12 presentations of a compound visual-non-visual stimulus during a compound conditioning session. The CAT was triggered 24 msec prior to the onset of the visual stimulus train. A calibration signal was similarly averaged at the completion of each session which enabled conversion of the AEP to a micro-volt scale. The sweep duration was equal to 1,024 msec corresponding to a dwell time of 2 msec for each of 512 data points. The CAT was employed to average potentials from two Ss simultaneously utilizing 512 data points per S.

Two measures were utilized for evaluation of the primary and secondary activity of the AEPs: (1) amplitude, as measured from baseline, of the initial positive deflection, P_1, and of the late slow wave negative secondary potential, S, and (2) a power measure (Walley & Urschel, 1972) providing estimates of the electrical energy represented in the primary and secondary phases of the AEP. Baselines were established as the average voltage level over 16 msec prior to the occurrence of P_1 for each response (see Figure 8). We have measured S from baseline voltage having noted changes in primary activity which would confound the conventional measurement of S from voltage levels represented in the primary phase. The lack of correspondence between late primary activity to photic and chiasmic stimulation also precluded an alternative measure.

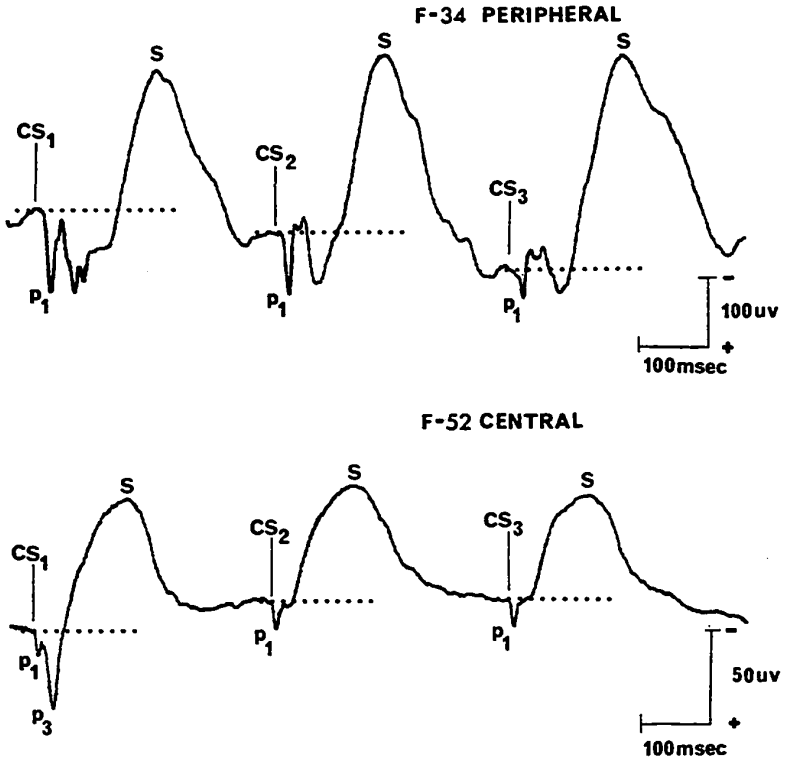

Fig. 8. Representation of two trains of AEPs recorded to visual stimulation. Each train was recorded differentially between the surface and white matter at the depths of the striate cortex in response to twelve 1,000-msec presentations of 3-Hz visual stimulation (CS_1, CS_2, CS_3) during the Pretest. Baselines for each response are represented by dotted lines. Onset of each visual stimulis is indicated by the CS demarcations. The P_1 and S components of each response are indicated. See text for the procedures employed in determining amplitudes and energy estimates of primary and secondary activity. Study 1. Electrophysiological

The amplitude measures enable evaluation of two select components of the AEP. In order to make a more general assessment of the primary and secondary phases of the AEP the power or evoked potential energy estimate (EPE) measure was used. The EPE was obtained by calculating the sum of squares of the voltages corresponding to each data point in the AEP. To employ this measure a criterion had to be established for identifying the primary and secondary phases of the AEP. Two criteria were entertained: (1) a polarity criterion and (2) a latency criterion. The polarity criterion represents the primary phase as that activity after response onset preceding a polarity transition (re: baseline) from positive oscillatory activity to slow wave negativity. The termination of secondary negativity corresponds to that point at which decreasing slow wave negativity exceeds baseline. The latency criterion fixes the primary phase as the 100-msec (photic stimulation) or 50-msec. (chiasmic stimulation) interval after response onset and the secondary phase as the subsequent 230 msec. Pearson product moment correlations between the EPE polarity and corresponding latency data (144 observations consisting of each response to the 3-Hz visual stimulus on the four tests for each of 12 Ss) yielded correlations of +.991 on primary activity and +.994 on secondary activity; the two measures provide redundant estimates of EPE. The polarity criterion was selected in preference to the latency criterion due to the former's interpretive clarity as representing a polarity specific EPE estimate and because the polarity criterion provides an EPE estimate which adjusts to between S variations in AEP latency characteristics.[1] The polarity criterion

[1] The results obtained using the latency criterion, summarized in Appendix E, generally corresponded with the polarity criterion.

also compensates for within S variations in the duration of primary and secondary activity and is analagous to computing the "area under the curve". Coefficients were calculated for each S correlating the number of addresses sampled in calculating each EPE with the corresponding EPE value. The correlations ranged from -.197 to +.299 with a mean correlation of +.075 for primary activity and a range of -.186 to +.230 and a mean of +.011 for secondary activity. These relationships suggest that the EPE estimate is independent of the duration of activity sampled. This is due to the fact that high amplitude components contribute to the EPE more than low amplitude activity proximal to a polarity transition; the logical consequence of summing, across data points, the voltages squared.

The basic statistical treatment consisted of separate analyses of variance on primary and secondary AEP amplitudes for the test and compound conditioning sessions. Corresponding analyses were performed on the primary and secondary EPE data available for the test sessions; hence, a total of six analyses were performed. Duncan's multiple range comparisons for testing the significance of differences between and among means was applied (Edwards, 1963, p. 136). The level of significance was defined as $p < .05$.

The frequency distributions of the EPE and amplitude data were highly positively skewed, resembling distributions of sample variances Pearson product moment correlations on the subject's means and standard deviations were significant ($p < .01$) for both the P_1 amplitude (+.954) and S amplitude (+.908) and primary EPE (+.993) and secondary EPE (+.984) measures. Consequently a logarithmic transformation was employed to normalize the data for each measure (Winer, 1962, p. 221).

Results

Histology revealed that not all Ss were implanted with the cortical depth electrode in the white matter underlying the striate cortex. The depth electrode in some Ss penetrated through the white matter and for four Ss resided above the white matter. Only the results of those 12 Ss (six Peripheral and six Central equally representing Groups A and T) in which the depth electrode clearly resided within the white matter are reported in subsequent AEP analyses. Plate 1 (upper portion) illustrates the placement of the cortical depth electrode in a S representative of those selected for analysis. The lower portion of Plate 1 illustrates the placement of the optic chiasma bipolar stimulating electrodes for this S (F-43).

Pretest Session

The Pretest represents a control session for evaluating changes in the visual AEP as a function of the various experimental manipulations. During the Pretest, S was given initial exposure to each stimulus element presented randomly 12 times with an average ITI of 40 msec. The AEP trains to photic and chiasmic stimulation during the Pretest (refer to Figure 8) generally differed along classically described lines. The photic potential was comprised of an initial positive deflection, P_1, peaking at 21 msec followed by biphasic oscillatory wavelets primarily positive in polarity lasting up to 100 msec and gradually replaced by increasing slow wave negativity. The slow wave negativity peaked between 150 and 200 msec and subsequently returned to near baseline levels before the response to CS_2. The response to the second and third flashes were characteristically similar to the

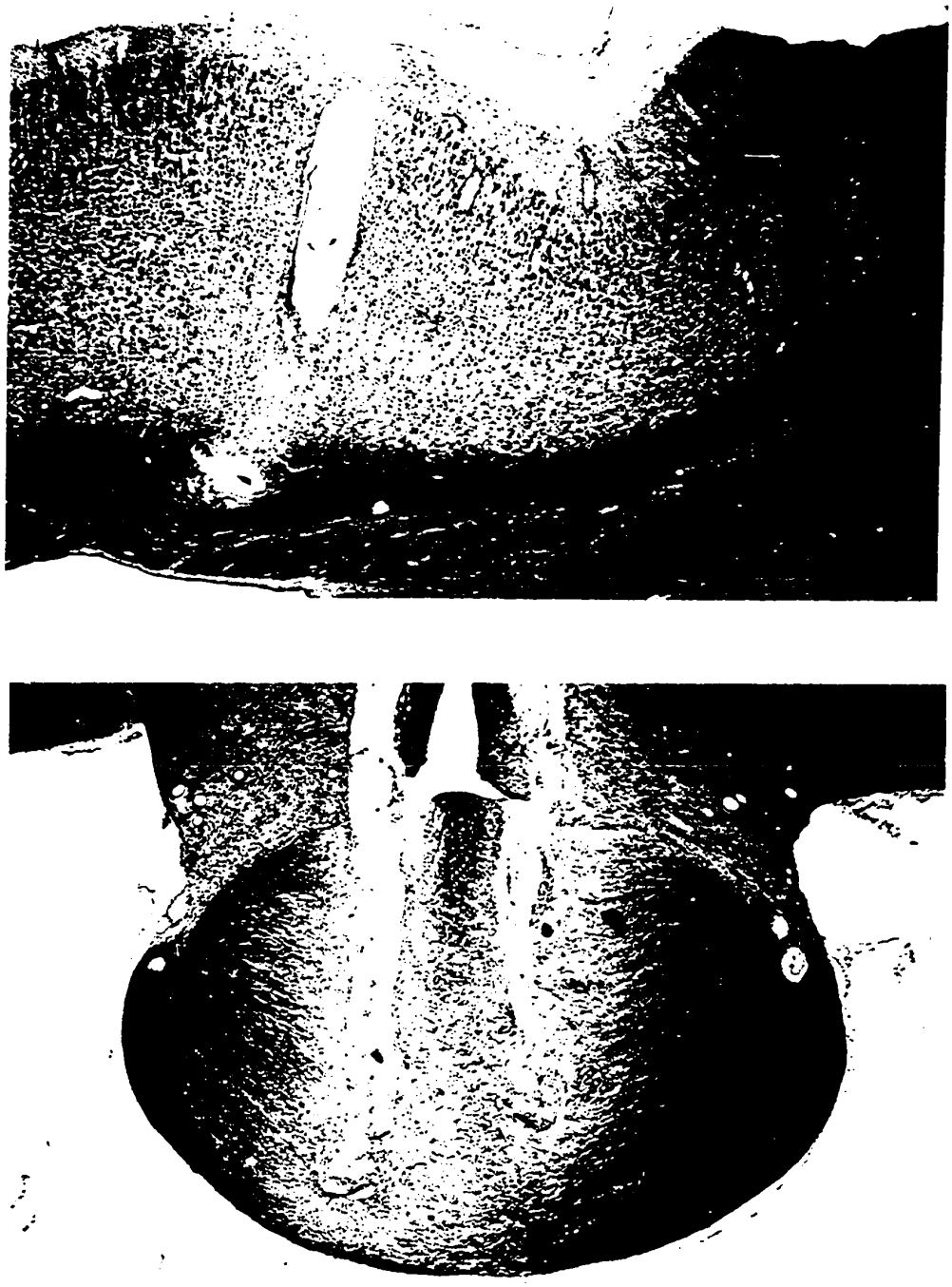

Plate 1. Coronal sections (15-μ-thick) of rabbit brain. The upper section illustrates the cortical depth of electrode tract and tip locus within the white matter at the depths of the striate cortex in the left hemisphere. The surface electrode resided on the dural surface lateral to the splenial sulcus (the cortical indentation). The lower section illustrates the placement of the optic chiasma bipolar stimulating electrodes (Subject F-43). See p. 7 for details of the histological procedure.

response to CS_1. The latency of the initial response to photic stimulation as recorded from the optic chiasma was found to be 15 msec (see Appendix F) indicating a 6 msec transmission time for the photic P_1 response from the optic chiasma to the striate cortex. The responses to chiasmic stimulation were different from the photic responses in several ways. P_1 peaked at about 6 msec and was followed by a small positive deflection, P_2 peaking at about 14 msec , and an extended deep positive deflection, P_3 peaking at about 27 msec.[2] P_3 represented the last positive deflection and was replaced by increasing slow wave negativity peaking at between 100 and 150 msec. The secondary activity returned to near baseline levels before the delivery of successive stimulation. With the exception of P_3, which was rarely evident in response to CS_2 or CS_3, this pattern was repeated to successive pulses of the 3-Hz train. In the subsequent representations, data are expressed as a percentage change from the Pretest session.

Test Sessions

The data from the test sessions enabled an assessment of the selectivity of changes in the AEP to non-reinforced presentations of the 1,000-msec train of 3-Hz visual stimuli. Changes in primary and secondary activity were evaluated when S was under selective non-visual stimulus control (the NVSC Test), selective visual stimulus control (the VSC Test), and subsequent to extensive differential conditioning

[2] If fast sweep speeds at high resolution are used to average the cortical response to chiasmic stimulation three small positive deflections are usually seen on the positive going slope of P_1 corresponding in latency and polarity to components recorded from cats (Malis & Kruger, 1956) but not generally reported in the rabbit. For a more detailed discussion of the correspondence between the latency characteristics of the photic and chiasmic elicited primary activity see Appendix F.

of the non-visual stimuli (the Pseudo-conditioning Test).

Primary Activity. The presentation of the visual stimulus alone when S was under visual stimulus control produced an enhancement of primary activity over activity recorded during both the Pseudo-conditioning and NVSC tests, regardless of the method of visual stimulation. A slight enhancement of primary activity was also evident during these latter conditions relative to the Pretest. Figures 9a-d illustrate the nature of these changes for four Ss, two receiving photic and two chiasmic stimulation. Figure 10 illustrates the average amplitude change of P_1 and primary-EPE (P-EPE) to photic and chiasmic stimulation during each test condition. The correlation coefficient for each S (12 observations, i.e., four tests x three AEPs to the visual stimulus train) between P_1 amplitude and P-EPE was significant ($p < .01$). The range of r between Ss was +.810 to +.963. Analyses of variance on these data revealed a statistically significant Tests main effect for both P_1 amplitudes ($F = 11.89$, $df = 2/16$, $p < .01$) and also for the P-EPEs ($F = 8.60$, $df = 2/16$, $p < .01$; see Appendix $G_{1, 2}$ for summary tables of these two analyses). P_1 and P-EPE were both enhanced during VSC over the Pseudo-conditioning Test ($p < .01$) and NVSC Test ($p < .01$ and $p < .05$, respectively). The enhancement of P_1 during VSC was evident to each stimulus of the visual stimulus train. The duration of primary activity during the Pretest for photic stimulation (72.3 msec) and chiasmic stimulation (29.7 msec) differed significantly ($t = 6.343$, $df = 34$, $p < .01$). However, an analysis of variance on the changes in the duration of primary activity showed no significant changes as a function of either the Visual Conditions, Groups, Tests, or CS factors or their interactions (see Appendix H_1 for the summary table). Thus,

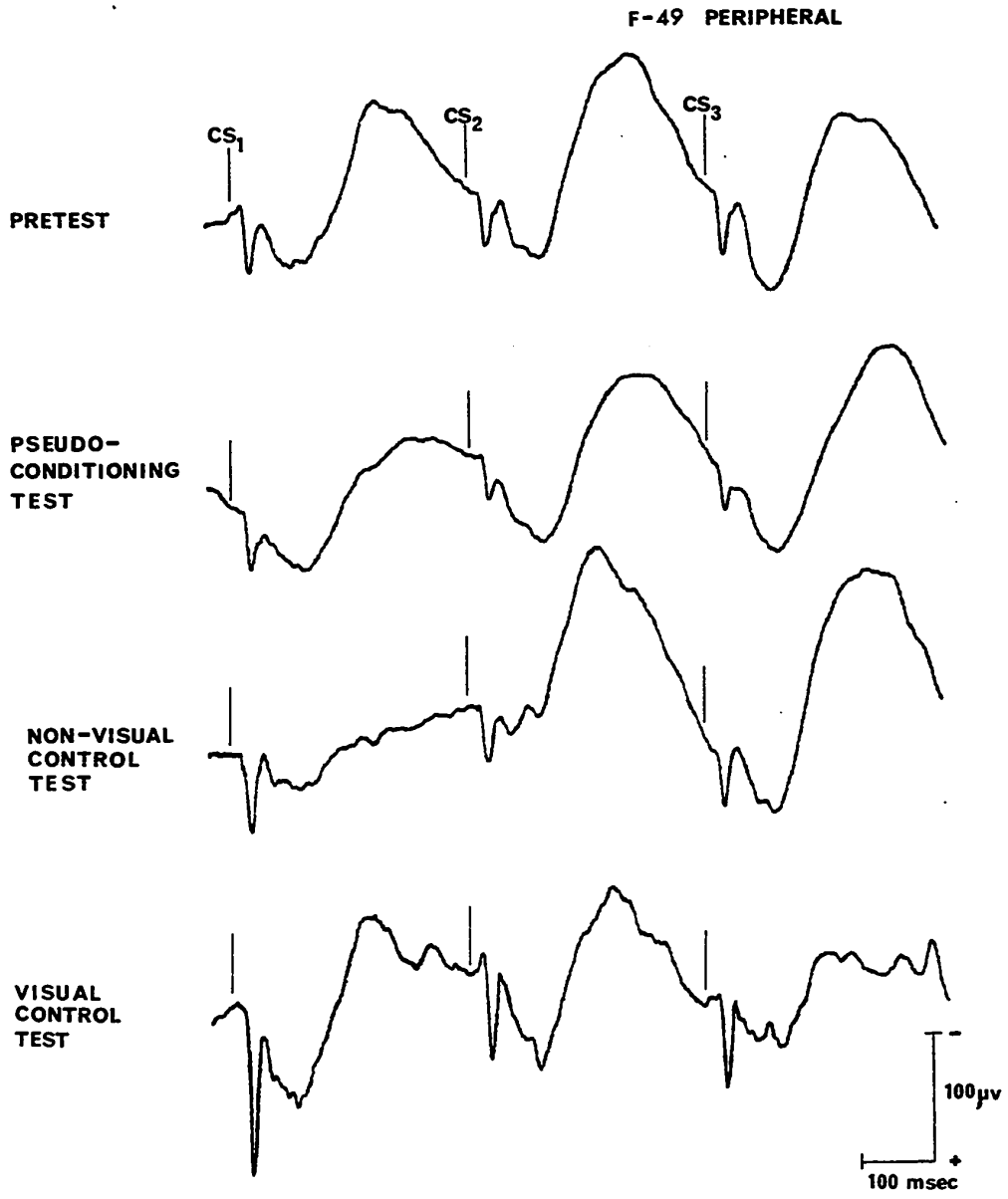

Fig. 9a. The AEPs to 3-Hz photic stimulation alone during each test session (Subject F-49, Group T). Study 1. Electrophysiological

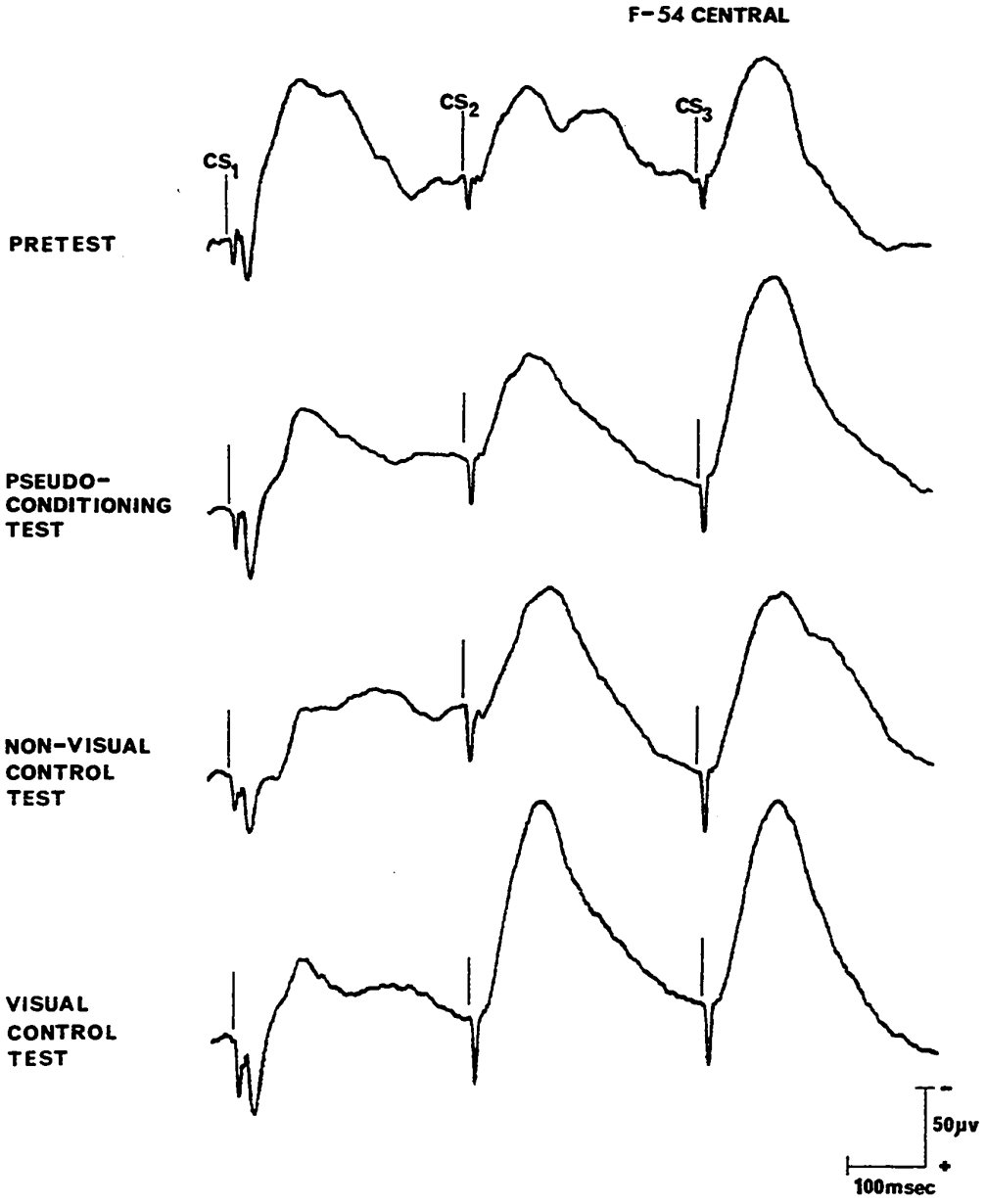

Fig. 9b. The AEPs to 3-Hz optic chiasma stimulation alone during each test session (Subject F-54, Group T). Study 1. Electrophysiological

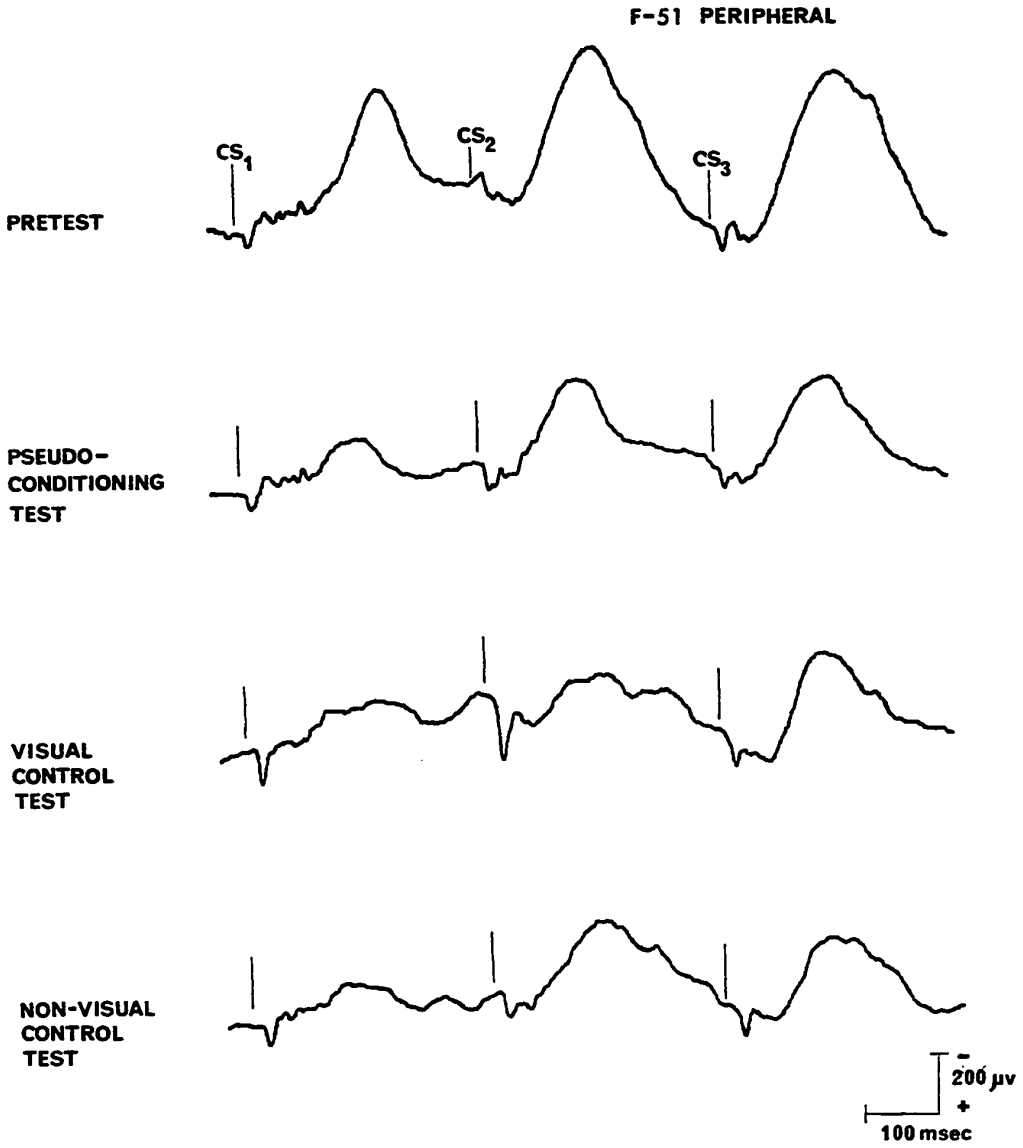

Fig. 9c. The AEPs to 3-Hz photic stimulation alone during each test session (Subject F-51, Group A). Study 1. Electrophysiological

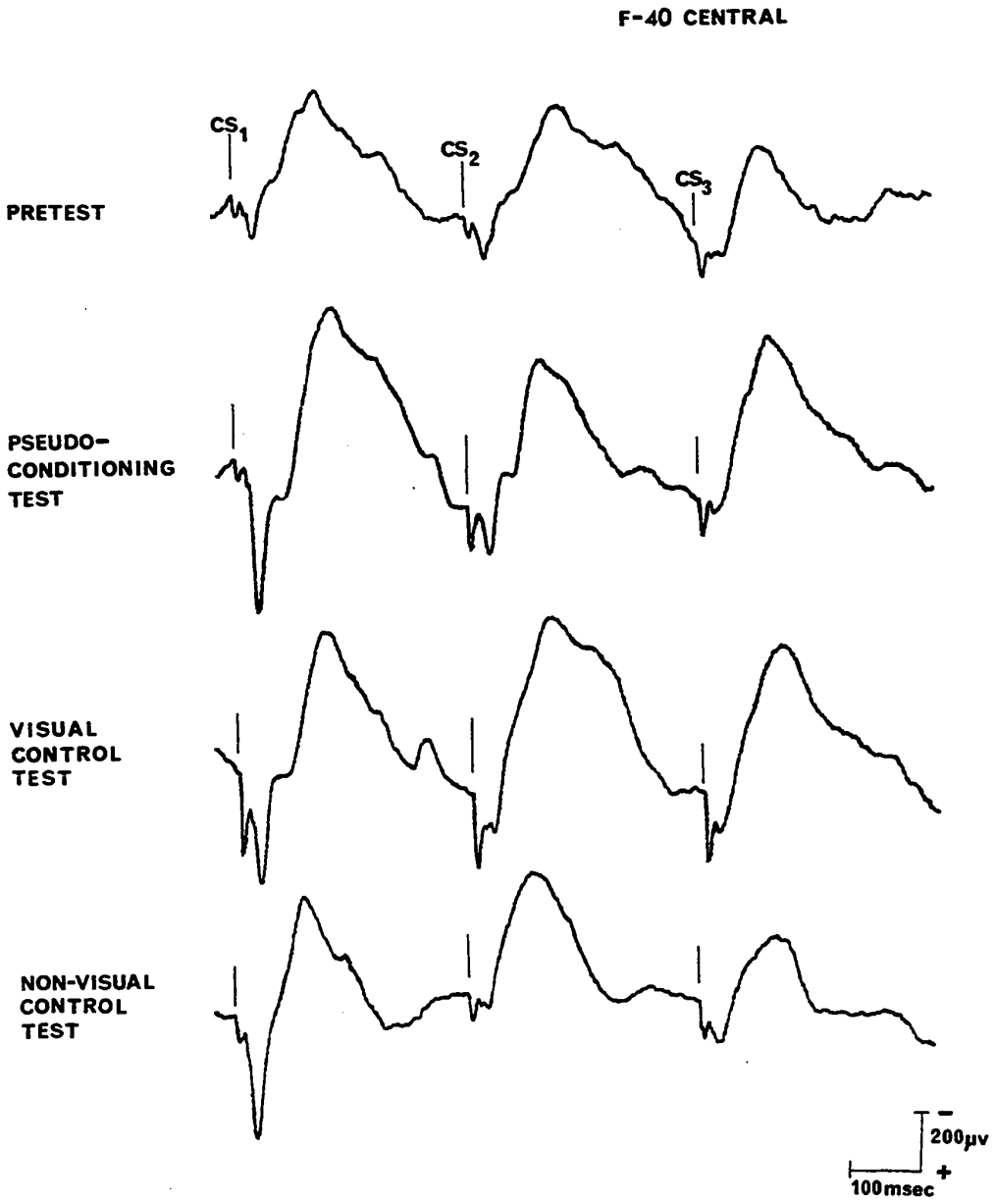

Fig. 9d. The AEPs to 3-Hz optic chiasma stimulation alone during each test session (Subject F-40, Group A). Study 1. Electrophysiological

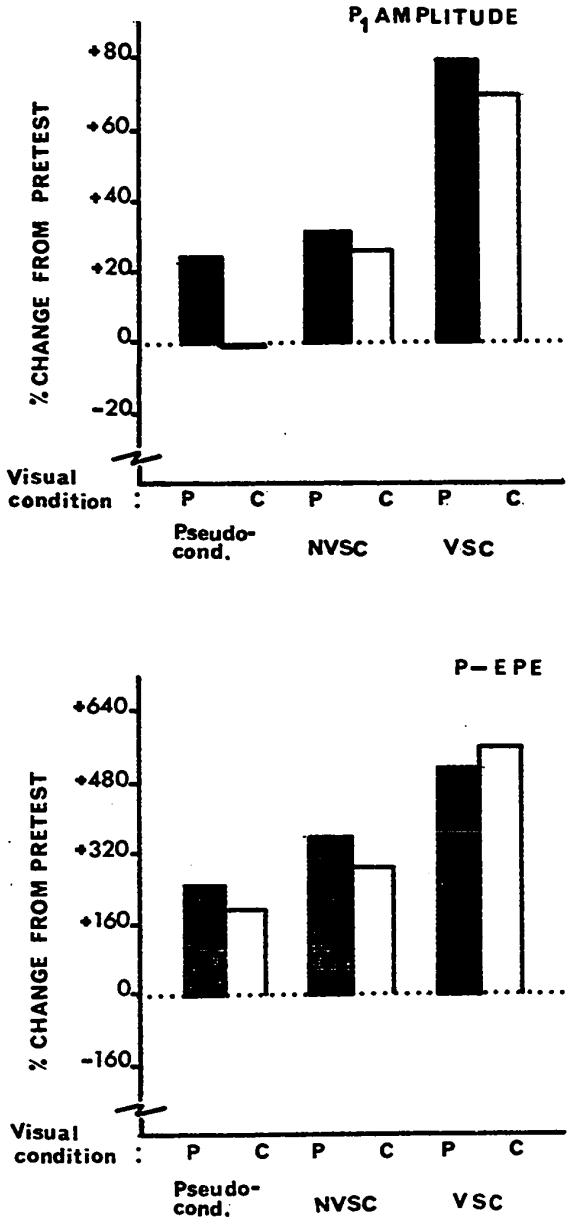

Fig. 10. The mean amplitude changes of P_1 (upper portion) and P-EPE (lower portion) in response to 3-Hz visual stimulation alone during each test condition expressed as a percentage of the Pretest response. P and C refer to peripheral and central visual stimulation groups, respectively. Study 1. Electrophysiological

primary activity and particularly P_1 was enhanced when S was under selective VSC more than when S was under NVSC or before compound conditioning. The enhancement was similar for both photic and chiasmic stimulation.

Secondary Activity. Differential changes in secondary activity were also evident between tests; the changes, however, did not correspond for the two methods of visual stimulation. Analyses of variance revealed a significant Visual Conditions x Tests interaction for changes in both S amplitude and S-EPE ($F = 5.45$, $p < .05$ and $F = 6.26$, $p < .01$, respectively, with $df = 2/16$; see Appendix $G_{3, 4}$ for summary tables of these analyses). The correlation coefficient for each S (12 observations each, i.e., four tests x three AEPs to the visual stimulus train) between S amplitude and S-EPE was significant ($p < .01$). The range of r between Ss was +.947 to +.996. Figure 11 (top) illustrates the Visual Conditions x Tests interaction for changes in S amplitude; S-EPE changes are illustrated in the lower portion of Figure 11. Changes in S amplitude across the tests were inversely represented for the two visual conditions. The S response (both amplitude and EPE) produced by chiasmic stimulation was enhanced over the photic response during both Pseudo-conditioning ($p < .05$) and VSC ($p < .05$, amplitude, and $p < .01$, EPE) tests. During NVSC, S amplitude assumed near Pretest levels. However, during VSC, S amplitude was enhanced ($p < .05$) relative to NVSC levels for chiasmic stimulation and attenuated ($p < .05$) for photic stimulation. S-EPE showed a similar effect for photic stimulation ($p < .05$). No significant main effects or interactions were found involving the Groups or CS factors.

Changes in the duration of S activity were not significant during

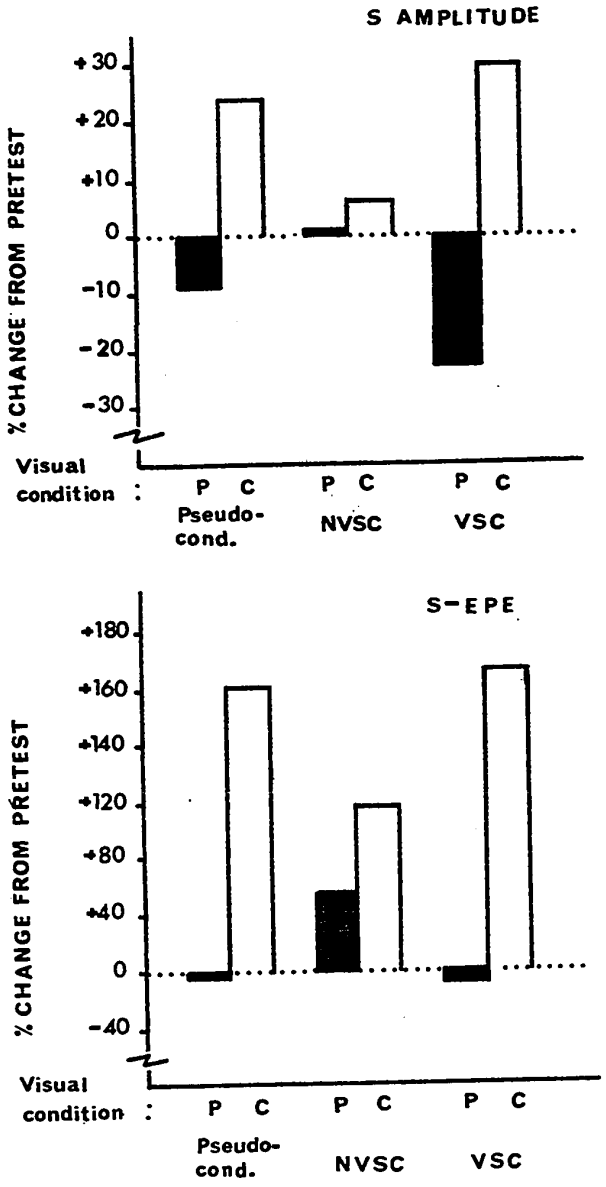

Fig. 11. The mean amplitude changes of S (upper portion) and S-EPE (lower portion) in response to 3-Hz peripheral (P) and central (C) stimulation alone. Each test condition is expressed as a percentage of the Pretest response. Study 1. Electrophysiological

the Pretest for photic (205.2 msec) and chiasmic (253.3 msec.) stimulation (t = 0.633, df = 34). No significant effects were isolated relating changes in the duration of secondary activity to the Visual Conditions, Groups, Tests, or CS factors or their interactions; this analysis is summarized in Appendix H_2.

Compound Conditioning Sessions

The data from the compound conditioning sessions enabled an assessment of the selectivity of changes in the visual AEP to presentations of the visual stimulus in compound with a non-visual stimulus when: (1) S was acquiring VSC or (2) during sustained NVSC. Only the amplitude measures were performed on the compound conditioning data and are expressed as a percentage of the Pretest amplitudes in the following representations.

Primary Activity. As observed during the test sessions there was a greater enhancement of P_1 during the VSC sessions (+74.1%) than during the NVSC sessions (+28.8%). An analysis of variance (see Appendix G_5) on the changes in P_1 amplitude from the Pretest values verified the significance of the difference between VSC and NVSC (i.e., the Compound Conditioning Conditions main effect: F = 27.27, df = 1/8, $p < .01$). A significant Groups x Compound Conditioning Conditions interaction (F = 7.51, df = 1/8, $p < .05$) suggests that the order in which VSC was established was significant. Group A showed a non-significant reduction in P_1 between VSC (+71.9%, compound TV) and NVSC (+43.2%, compound AV) conditioning. However, Group T showed a significant increment in the amplitude of P_1 between NVSC (+14.4%, compound TV) and VSC (+76.4%, compound AV) conditioning. The changes in P_1 amplitude between Groups were apparently independent of the nature of

the non-visual stimulus with which the visual stimulus was paired. Group A showed a significantly higher ($p < .05$) P_1 amplitude to compound TV than did Group T, while Group T showed a significantly higher ($p < .05$) P_1 amplitude to compound AV than did Group A. Figure 12 illustrates the AEPs to photic stimulation for a single S (F-34, Group T) across each session of the two compound conditioning conditions. Note that the enhancement of P_1, and of primary activity in general, was evident at the initiation of compound conditioning designed to establish VSC. The enhancement to both photic and chiasmic stimulation during VSC was equally represented across each compound conditioning session and each CS of the visual stimulus train. Late primary activity was also evidently enhanced across all CSs to photic stimulation during VSC and to CS_1 for the corresponding chiasmic response.

Thus, primary activity associated with the presentation of the visual stimulus in compound with a differentially reinforced (the NVSC condition) or non-reinforced (the VSC condition) stimulus corresponds well with data obtained when the visual stimulus was presented alone.

Secondary Activity. Changes in the amplitude of secondary activity when the visual stimulus was reinforced in compound with a non-visual stimulus were also evident. The Visual Conditions main effect was not significant ($F = 0.88$, $df = 1/10$; see Appendix G_6). There was, however, a tendency for secondary activity to chiasmic stimulation to be enhanced (+32.2%) and for photic activity to be attenuated (-3.5%) relative to the Pretest). Also noteworthy as a function of compound conditioning was a change in the amplitude of secondary activity to the individual CSs of the 3-Hz train of photic or chiasmic stimulation (illustrated in

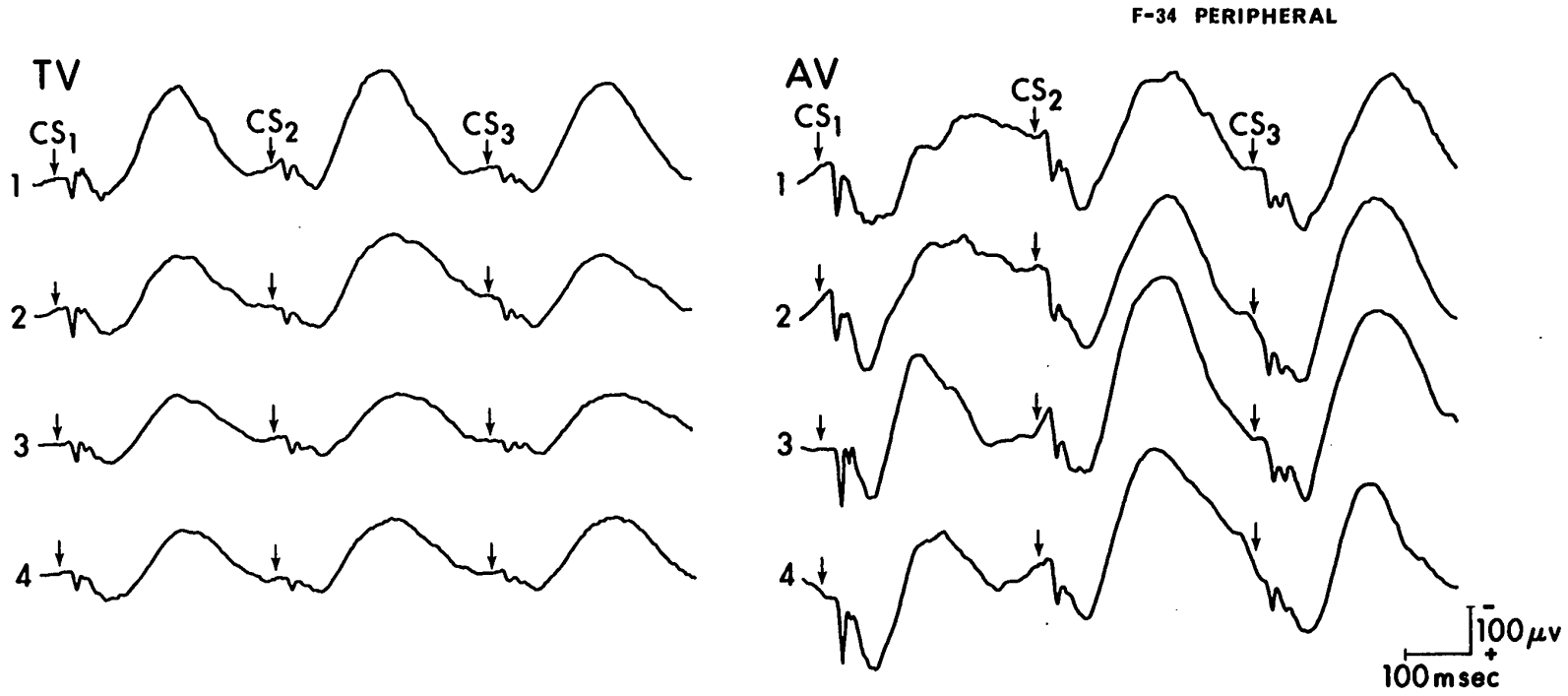

Fig. 12. The AEPs to 3-Hz photic stimulation in compound during four sessions of TV and AV compound conditioning. S (F-34) demonstrated a predominance of NVSC after TV conditioning and VSC after AV conditioning. Study 1. Electrophysiological

Figure 13). An analysis of variance (see Appendix G_6) on changes in the secondary amplitudes revealed a significant Compound Conditioning Conditions x CSs interaction ($F = 9.26$, $df = 2/16$, $p < .01$). The response to CS_1 was reduced (-32.8%) significantly more during NVSC ($p < .01$) than during VSC (-3.4%). However, in response to CS_2, S activity rebounded and was enhanced more ($p < .05$) during NVSC (+80.3%) than during VSC (+50.3%) sessions. The direction of change for each condition as a function of the method of visual stimulation was the same. Note in Figure 13 that the enhancement between CS_1 and CS_2 during NVSC compound conditioning exceeds the enhancement during the NVSC Test when non-significant visual stimulation was presented alone. The response to CS_3 was significantly reduced ($p < .05$) from the response to CS_2 during both NVSC and VSC compound conditioning. The significant CS main effect ($F = 5.15$, $df = 2/16$, $p < .05$) reflects the enhancement of the secondary response to CS_2 (+65.3%) over the response to CS_1 (-18.1%) and CS_3 (+0.03%). No differences were detected significantly relating the method of visual stimulation with any other factor or interaction when the visual stimuli were presented in compound.

Discussion

During all test sessions, AEPs were obtained to presentations of the visual stimulus alone. Since there were differential eyeblink response probabilities to the visual stimulus across these sessions, an independent assessment of the electrophysiological influence of altered visual input to the eye ipsilateral to the cortical recording electrode was undertaken (see Appendix K). Occluding visual input to

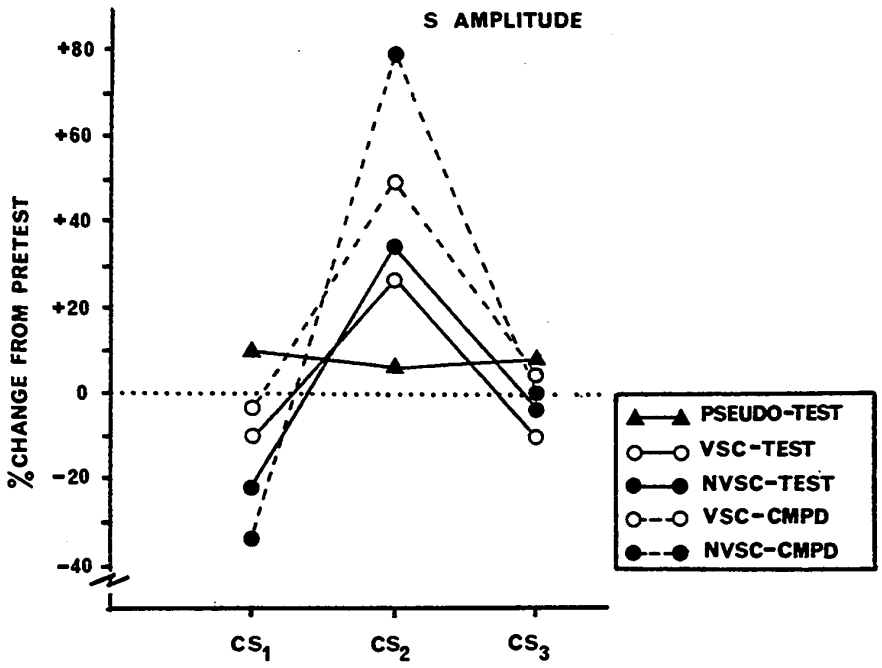

Fig. 13. The mean amplitude change of S in response to each CS of the 3-Hz visual stimulus train during each test condition. The activity representing the compound (CMPD) conditioning sessions reflects the average change of all Ss across the four sessions for each condition. Study 1. Electrophysiological

the eye contralateral to the recording electrode abolished primary components of the AEP to a photic flash at both the lateral geniculate and striate cortex. This was not unexpected as van Hof (1970) has demonstrated that there is little interocular transfer of visual pattern discriminations in rabbit and Giolli and Guthrie (1969) have noted that in albino rabbit only five percent of the optic nerve fibers do not decussate. Thus, an attenuation of photic input to the ipsilateral eye due to conditioned eyeblink activity or eye movements would be expected to have little influence on an ipsilateral photic cortical response. Also, since conditioned eyeblink activity was generally initiated about 550 msec after CS onset (see Appendix L), the influence of such activity would predominate on the potential generated to the third flash of the visual train. There were no obvious differences in the latency of CRs nor, as indicated in Study 1, were there significant differences in CR frequency as a function of the method of visual stimulation.

The significance of the reported changes in primary activity of the visual AEP as a function of the test manipulations must be qualified. The changes were specific for Ss with the transcortical depth recording electrode in the white matter underlying the striate cortex. Similar changes were not detected in Ss for which the depth electrode generally resided outside this region.[3] This discrepancy may be due to the fact that the white matter contains optic radiation fibers projecting from the dorsolateral lateral geniculate (LGB) to the primary receiving area of the occipital cortex. The non-white matter placements were too widely

[3] In fact, in many instances the Ss with depth electrodes in non-white matter reflected changes in primary activity associated with VSC and NVSC which were opposite to those with white matter placements.

distributed to enable a meaningful assessment of AEP changes at other cortical loci.

Of the various oscillatory components of the primary phase of the rabbits' visual AEP only P_1 was reliably elicited to each pulse of 3-Hz photic or chiasmic stimulation. P_1 recorded under fast sweep speeds to photic or chiasmic stimulation consists of three positive deflections riding on the P_1 deflection (see Footnote 2). Probably only the first of these, as in cat (Bishop & O'Leary, 1938; Chang & Kaada, 1950; Bishop & Clare, 1952, 1953; Malis & Kruger, 1956; Bremer & Stoupel, 1956; see also Brindley, 1960) is a radiation spike. Thus, P_1, as measured here, probably reflects both geniculate and cortical activity. However, only significant changes in P_1 were obtained from Ss with white matter placements suggesting that these changes were due to activity arising in white matter, i.e. in radiation fibers, mediated at the geniculate level. This assumption applies to P_1 as produced by either photic or chiasmic stimulation. The response to photic stimulation is transmitted to the optic chiasma in approximately 15 msec in the rabbit and the peak of P_1 in the white matter underlying the striate cortex is evident 6 msec later in response to either photic or chiasmic stimulation. It is suggested that P_1 to photic and chiasmic stimulation as recorded from the white matter is of common origin primarily reflecting action potentials of the optic radiations projecting from the LGB relay cells.

The general enhancement of primary activity during all conditions subsequent to the Pretest is probably attributable to the predominance of US presentations, i.e., tonic arousal, during these sessions. Arousal has been associated with increments in LGB excitability

(Fukuda & Iwama, 1970; Ogawa, 1963; Walsh & Cordeau, 1965; Palestini, Pisano, Rosandini, & Rossi, 1964; Bremer, 1970; Steriade, 1969). The exceptional enhancement of primary activity during VSC conditions over all other conditions, however, excludes a sensitization, i.e., tonic arousal, interpretation. It is apparent that primary activity reflects processes in addition to those associated with the state of tonic arousal.

An increment in phasic arousal to the onset of significant stimulation would account for the difference in primary activity between the VSC and NVSC Tests when the visual stimulus was presented alone. However, no difference should have been evident to the NVSC and VSC compounds. Also, primary activity should have changed across the 3-Hz visual stimulus as phasic arousal increased subsequent to stimulus onset. Further, the enhancement of primary activity during VSC does not appear to be associated with the nature of the non-visual stimulus; the non-visual stimuli were counterbalanced and primary activity was enhanced to compound AV for Group T and compound TV for Group A. Also, any fluctuation in phasic arousal associated with the initial pairing of the visual stimulus with a differentially reinforced or non-reinforced stimulus should have been restricted to the early sessions of compound conditioning. In fact, no significant differences were found relating the sessions factor with alterations in primary activity. The enhancement of primary activity during VSC does not appear attributable to changes in phasic arousal.

Since the enhancement of primary activity during VSC accompanied both photic and chiasmic stimulation, corticofugal regulation of retinal activity (Hernández-Peón, 1964) is not critical. Further, the

enhancement conflicts with Horn's suggestion (1960, 1965) of a negative correlation between the amplitude of the visual evoked potential and "searching". Presentations of the visual stimulus in compound with a non-visual element should have enhanced searching and produced a reduction in primary activity compared to conditions when the visual element was presented alone.

Changes in the primary phase of the visual AEP were in direct correspondence with the performance of Ss relative to the visual element as assessed in Chapter I. Primary activity was inversely related to the level of NVSC throughout compound conditioning and directly related to the level of VSC when the visual stimulus was presented alone. This suggests that the changes in the primary phase, particularly P_1, of the visual AEP as recorded from the primary receiving area, reflect attentional rather than associative factors; S was attending to visual stimulation with a corresponding enhancement of thalamocortical afferent activity at the initiation of VSC compound conditioning even though asymptotic conditioning was not yet evident. Note that during the compound stimulus presentations Group A showed a non-significant reduction in primary activity during NVSC from VSC levels. Recall there was behavioral evidence of a carry-over of VSC from TV to AV (NVSC) compound conditioning for Group A. The high performance of Group A during AV compound conditioning was interpreted as reflecting the consequence of attention to a compound comprised of two valid stimuli. Enhanced phasic arousal to such a compound could likewise account for the lack of a significant reduction in primary activity under this condition.

That primary activity was enhanced to the initial visual stimulus of the 3-Hz train during VSC indicates that the mechanism mediating

primary activity operates prior to stimulus onset corresponding to an attentional set or tonic selective attention. There were no significant changes in primary activity across the three responses to the visual stimulus train under any conditions investigated (the Tests x CS interaction). This suggests that any phasic changes in attention subsequent to onset of the visual stimulus train were not significant.

Dissociation of attentional and arousal factors on primary activity might have been further accomplished by extended non-reinforced presentations of the visual stimulus prior to the Pretest. In this study the Pretest control session represented the first session during which S was exposed to the stimulus elements. Further, there was little fluctuation in primary activity, i.e., attention, as S acquired the conditioned response. Whether the augmentation of primary activity during VSC would have been sustained with overtraining was not assessed.

The following discussion is directed toward the elaboration of a model describing how transmission of activity through the lateral geniculate body (LGB) might be modified during arousal and selective attention (see Figure 14). Central to this proposal is the role of post-synaptic inhibitory interneurones at the level of the LGB acting to modulate excitability of the LGB relay cells (Eccles, 1969, pp. 52-54). Two influences have been found to modulate activity of the inhibitory interneurones: (1) non-specific influences associated with activity of the reticular formation and non-specific thalamic nuclei and (2) specific influences associated with activation of recurrent collaterals of the optic radiation fibers. Bishop and Davis (1960); Bishop, Burke, and Davis (1962); Sefton and Burke (1965); and Burke and Sefton (1966a, 1966b) observed rhythmic waves in the LGB associated with depressed excitability

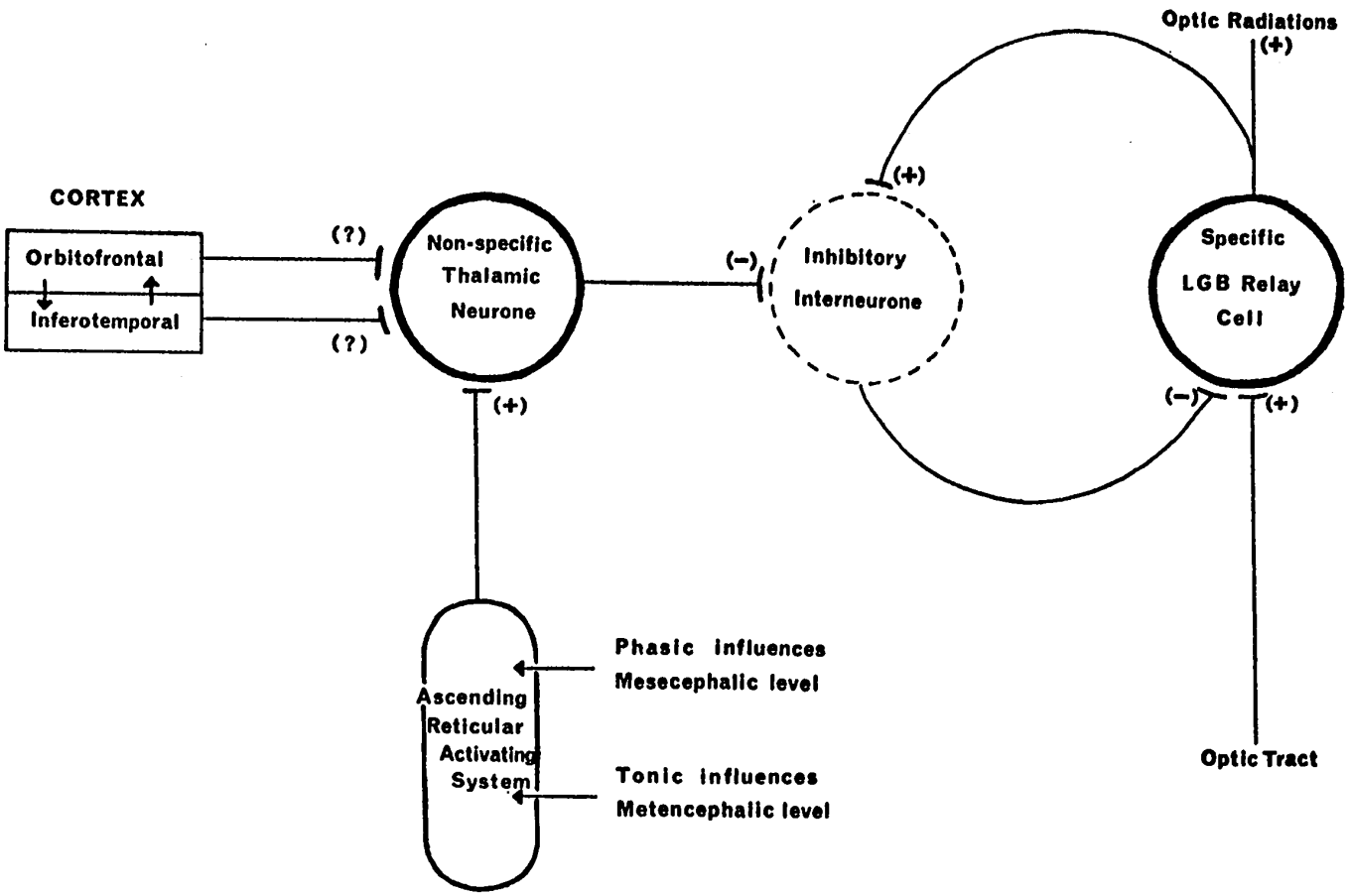

Fig. 14. A model illustrating the influences proposed to predominate on the specific and non-specific thalamic nuclei during selective visual attention. Both inhibitory (-) and excitatory (+) influences are illustrated. See text for further elaboration of the model.

of the relay cells. Fukuda and Iwama (1970) have also noted a reduction in responsiveness of inhibitory interneurones of the LGB following reticular stimulation. Further, Bremer (1970) has suggested that slow wave sleep is characterized by an increase in LGB recurrent inhibition resulting from a decrease in reticular activity. A decrease in LGB recurrent inhibition was associated with reticular stimulation. These findings are consistent with Steriade's (1969) contention that arousal enhances afferent transmission by facilitating synaptic events for all specific thalamic relays.

The data presented here indicate that influences in addition to those associated with arousal operate during selective attention. It would appear that activity of inhibitory interneurones modulates the transmission of afferent input through specific thalamic nuclei. The inhibitory interneurones would be depolarized by recurrent collaterals of the specific relay cells and hyperpolarized via projections from the non-specific nuclei. Normally, tonic excitability of the specific nuclei would be sustained by activity of the diffuse ascending reticular-thalamic activating system. During enhanced arousal hyperpolarization of the inhibitory interneurones would act to enhance afferent input through all specific thalamic nuclei. During attention, influences from the orbitofrontal cortex (Bianchi, 1895; Ferrier, 1890; Jacobsen, 1935; Malmo, 1942; Akert, 1964; see also Warren & Akert, 1964; Spinelli & Pribram, 1967; Skinner & Lindsley, 1967, 1971; Thompson et al., 1966) and/or inferotemporal cortex (Spinelli & Pribram, 1966, 1967; Gerbrandt, Spinelli, & Pribram, 1970) probably interact to selectively alter the excitability of modality specific thalamic nuclei. This could be achieved indirectly via intrathalamic projections of the non-specific thalamic nuclei differentially inhibiting or exciting to the degree

attention is required in any given sense modality. It is unlikely, however, that the fine degree of attention associated with intramodal stimulus selection, e.g., attending a white mouse against a background of flashes (Palestini et al., 1959), is mediated at the thalamic level. The mechanism mediating intramodal stimulus selection must be exceedingly complex. Intramodal effects are probably achieved at a higher level of neurophysiological integration. The findings reported here indicate that some degree of intermodal "selective attention" is achieved at the thalamic level; geniculo-striate transmission of afferent input in the visual mode may be enhanced over afferent auditory or somatosensory activity.

While the nature of the generator of secondary slow wave negativity at the striate cortex is unresolved, its appearance is readily distinguished in AEPs to both photic and chiasmic stimulus trains in the unanesthetized preparation. It is assumed that secondary activity to both photic and chiasmic stimulation is of common origin. The evaluation of changes in secondary activity across conditions was undertaken to test the hypothesis that secondary activity reflects changes in the state of tonic arousal.

There was a general enhancement of secondary activity to chiasmic stimulation during all conditions after the Pretest. The increment is in correspondence with an increment in tonic arousal. That secondary activity to chiasmic stimulation was considerably reduced during the NVSC Test, when the visual stimulus was presented alone, is contrary to a sensitization or tonic arousal interpretation. If, however, it is assumed that NVSC compound conditioning resulted in habituation of phasic arousal, e.g., a reduction of orienting activity, to the visual stimulus train as S maintained an attentional set to non-visual stimulation, then

the results are also consistent with an arousal interpretation. In essence, the lack of attention to visual stimulation during attention to non-visual stimulation would preclude the excitation of phasic arousal when the visual stimulus was presented alone. This is particularly interesting in that it indicates that habituation of arousal may occur to a stimulus when it is reinforced in compound with more significant stimulation. Hence, low performance to the visual stimulus indicative of "blocking" would be correlated with low arousal by, and low attention to, the visual stimulus. Such an effect on secondary activity would not be evident during NVSC compound conditioning due to phasic arousal elicited by the significant non-visual element. The habituation of phasic arousal to the visual stimulus would tend to counter the influence of tonic arousal on secondary activity and may represent a correlate of conditioned inhibition (Rescorla & Wagner, 1972) or sustained attention to other stimuli (Sutherland & Mackintosh, 1971). The enhancement of secondary activity to chiasmic stimulation during VSC could be due to tonic arousal, phasic arousal, or both.

The mechanism mediating arousal has been clarified in recent years by the work of Sharpless and Jasper (1956) and Demetrescu, Demetrescu, and Iosif (1965; see also the reviews of Thompson & Spencer, 1966 and Groves & Thompson, 1970). Demetrescu et al. (1965) have proposed that two communication channels to the reticular formation activate the ascending reticular activating system. Short latency, short duration, phasic arousal is proposed to be initiated at the mesencephalic level via activity in collaterals of the specific sensory and collicular pathways. Activity in a circuitous pathway at the mesencephalic level involving the central grey and posterior hypothalamus is believed to mediate longer latency, longer duration tonic arousal. Sharpless and

Jasper (1965) noted that after severing the brachia of the inferior colliculus the habituation of arousal to auditory stimulation was enhanced, tonal specificity of habituation impaired, the tonic arousal threshold elevated, and phasic arousal abolished. Secondary activity is abolished to photic stimulation following the severing of the brachium of the superior colliculus (Rose & Lindsley, 1968). To the extent that secondary activity to stimulation of the optic chiasma reflects arousal our data would support a distinction between phasic and tonic arousal and their dissociation from mechanisms mediating selective attention.

While enhancements of secondary activity were evident to chiasmic stimulation during arousal, decrements were evident to photic stimulation. This apparent paradox has been noted by other investigators as the consequence of arousal (Bremer & Stoupel, 1959a, Walley & Urschel, 1972). The discrepancy is apparently not mediated by peripheral mechanisms acting to reduce photic input during arousal; during the VSC conditions, although the secondary activity to photic stimulation was reduced, the primary response was enhanced. Further, Bremer (1961) and Bremer and Stoupel (1959a) have found little or no change at the LGB at the same time the cortical response is markedly reduced. Bremer and Stoupel (1959a) explain the attenuation of photic secondary activity during arousal as follows. During arousal the cortical neurones are subliminally activated and therefore partially refractory. The temporally distributed photic potential is incapable of overcoming this refractoriness while the chiasmic potential because it is highly synchronous can "break through" the refractoriness. The position was apparently taken because of an unwillingness to assume there are

inhibitory effects of arousal. That the temporal distribution, i.e., synchrony, of afferent activity is critical is shown by the observation that "the potentials evoked by short (15-40 msec) volleys of high rate stimuli delivered to the optic tract (which elicit discharges less synchronous than single strong pulses) are altered by RF stimulation or continuous retinal illumination in the same way as potentials evoked by flashes" (Demetrescu, 1967, p. 36).

Bremer's hypothesis assumes a neurophysiological mechanism which has never been demonstrated independently (i.e., "breaking through" refractoriness). In addition, Walsh and Cordeau (1965) have found that cortical evoked potentials are initially increased and later decreased with prolonged arousal, a finding clearly contrary to Bremer's hypothesis. Walley and Urschel (1972) suggest that during arousal there is a subliminal facilitation of intracortical recurrent inhibition mediated by the diffuse ascending inhibitory system described by Demetrescu et al. (1965). Since the chiasmic potential is relatively discrete, chiasmic generated afferent input would escape recurrent inhibition. However, the temporally distributed photic potential would be attenuated by recurrent inhibition. Further, the cortical response to chiasmic stimulation, free from the influence of recurrent inhibition, should represent whatever residual intracortical events are initiated by afferent input. It is assumed that cortical surface negativity reflects, in part, hyperpolarization in the depths of the cortex (Jasper & Stepanis, 1956; Andersson, 1965; Creutzfeldt, Watanabe, & Lux, 1966a, 1966b) corresponding to the excitation of recurrent inhibition (Supin, 1966, 1968); additional support for the inhibitory nature of this wave is provided in Study 2. The data are consistent with the hypothesis that arousal

produces a facilitation of intracortical recurrent inhibition reflected in the response to chiasmic stimulation by an augmentation of secondary slow wave negativity. Under the conditions of the present study, the magnitude of this enhancement is apparently independent of the amplitude, but not of the temporal distribution, of primary activity. Secondary activity would reflect the summation of intracortical IPSPs initiated by afferent input but regulated by ascending reticular activity.

The level of secondary activity elicited across the train of visual stimuli was assumed to reflect alterations in phasic arousal initiated by the onset of stimulation. There was a significant increase in secondary activity peaking to CS_2 and decreasing to CS_3 during compound conditioning. Most of this effect was due to the central Ss; there was little change in secondary activity for peripheral stimulation across the CS train (see Appendix G_3). A similar, though not significant, trend was evident during each test session with the exception of the Pretest and Pseudo-conditioning Test when conditioned responses to the visual stimulus were not evident. This would indicate that the phasic increase in arousal was associated with the presentation of significant stimulation. The obvious comparison here is between two conditions differentiated by the presentation of a significant or non-significant stimulus as during NVSC compound conditioning and the NVSC Tests, respectively. The increment in arousal between CS_1 and CS_2 associated with the presentation of non-significant visual stimulation (see Fig. 13, p. 74) was greater when the visual stimulus was in compound with significant non-visual stimulation (NVSC compound) than when presented alone (NVSC Test). This enhancement exceeded a similar effect seen between the VSC Test and VSC compound conditioning. The contrast between

the NVSC Test and NVSC compound conditioning strongly supports the contention that there is a phasic increase in arousal between the onset of significant stimulation and CR emission followed by a reduction in arousal. Similarly, Kahneman et al. (1969) found a phasic increment in several autonomic correlates of arousal up to the point of response emission on a digit transformation task. In the present study, the reduction of secondary activity to CS_3 coincided with CR emission. Pickenhain and Klingberg (1965) have also reported reduced secondary activity when response emission or behavioral activity was associated with the delivery of photic stimulation. Consideration of even such subtle motor activity as a conditioned contralateral eyeblink may be important in interpreting changes in secondary activity. The low significance and few CRs to visual stimulation during the Pretest and Pseudo-conditioning Test would account for the stable level of secondary activity across the train of visual stimulation.

The mechanism mediating the phasic increment in arousal subsequent to the presentation of signficant stimulation has been discussed as involving the activation of the ascending reticular activating system. The attenuation of secondary activity at CR emission suggests that activity coincident with CR emission counters excitatory processes associated with increasing arousal. Attention has recently been directed to the role of the limbic system and particularly the hippocampus as an integral part of the reticulo-cortico-reticular activating system (Gray, 1969). Our data are consistent with the contention that the hippocampus mediates Pavlovian internal inhibition and involves inhibition of the reticular activating system (Douglas, 1967; Kimble, 1969). Thus, the decrease in secondary activity to CS_3

may reflect a decrease in arousal associated with the CR and the initiation of inhibitory activity of the hippocampus. In effect, the initiation of CR activity at a time of intense arousal would initiate a reduction in arousal as activity in the hippocampus effects an active attenuation of reticular activation. The decrease in reticular activity would lead to a decrease in activity in the intracortical inhibitory interneurones responsible for the generation of secondary activity.

Summary

Reinforcement of visual stimulation in compound with a more significant non-visual stimulus appears to attenuate attention, phasic arousal, and performance to the visual stimulus. Reinforcement of visual stimulation in compound with a less significant non-visual stimulus enhances attention, phasic arousal, and performance to the visual stimulus. The enhancement of primary activity of the visual AEP is associated with selective attention to visual stimuli and can be dissociated from facilitatory influences associated with tonic and phasic arousal. This effect occurred for both photic and chiasmic stimulation indicating that the enhancement is mediated by post-retinal influences. Consistent alterations in secondary activity, related to tonic and phasic arousal, were not contingent upon the amplitude of primary activity, i.e., afferent input, though the temporal distribution of the photic potential makes it more susceptible to the attenuating influences of enhanced intracortical recurrent inhibition during arousal. Arousal tends to generally enhance secondary activity to optic chiasma stimulation and to decrement activity to photic stimulation.

The onset of significant stimulation initiates phasic arousal which is associated with a progressive enhancement of secondary activity most evident to chiasmic stimulation. At CR emission an attenuation of the effects of arousal on secondary activity is evident. These data are consistent with the contention that during selective attention there is an enhancement of afferent input through specific thalamic nuclei of the attended modality due to a reduction of inhibition and an attenuation of afferent input through thalamic nuclei of the non-attended modality due to an enhancement of inhibitory influences. At the cortical level there is a general facilitation of intracortical inhibitory processes associated with arousal. An attenuation of intracortical inhibition was evident during CR emission.

Study 2. The Geniculo-striate Recovery Cycle During Arousal and Selective Stimulus Control

The amplitude of visual AEP components was used in Study 1 to measure the excitability of the geniculo-striate complex during arousal and selective attention. While both primary and secondary activity of the visual AEP were found to be responsive to changes in arousal, only primary activity was found to reflect changes in selective attention. Another measure of excitability is the recovery function. This measure is obtained by stimulating, for example, the optic chiasma, with a "conditioning" and a "test" stimulus separated by varying intervals. The recovery function provides a representation of the time course of processes initiated by afferent input, e.g., phasic arousal and recurrent inhibition. To our knowledge the recovery function has never been used to study changes in the excitability of the geniculo-striate complex as a function of peripherally induced arousal and/or selective

stimulus control.

By delivering pulse pairs to the optic pathways the course of the geniculo-striate recovery cycle has been described in the cat by numerous investigators (Bartley, 1936; Bishop & O'Leary, 1940; Marshall, 1949; Clare & Bishop, 1952; Bremer, 1961; Demetrescu et al., 1965, 1966; Malis & Kruger, 1956; see also Gastaut, Gastaut, Roger, Cariol, & Naquet, 1951). The cycle consists of a brief 5 - 10 msec phase of supranormal excitability followed by a 20 to 30 msec phase of absolute refractoriness and gradual recovery. Refractoriness is attributed to active post-synaptic inhibitory activity initiated by stimulus onset. The recovery cycle of the rabbit is not so clear. The duration of the surface positive phase of the cortical potential is approximately 40 msec during which the cortex is absolutely refractory (Pearlman, 1963, Supin, 1966). Recovery of primary activity begins at a latency corresponding to the initiation of the secondary surface-negative wave. Supin (1966) reports that full recovery is retarded until after the termination of the secondary potential. An inhibitory period has often been described for cortical neurones corresponding with the surface negative wave (Baumgartner & Jung, 1955; Akimoto & Creutzfeldt, 1958; Grutzner, Güsser, & Baumgartner, 1958; Li, Ortiz-Galvin, Chou, & Howard, 1960; Li & Chou, 1962; Kondrat'eva, 1964; Krnjević, Randić, & Staughan, 1964; Polyanskii, 1965, 1967; Supin, 1966). Pearlman (1963), however, reports a supranormal recovery phase for the cortical evoked response to

stimulation of the optic nerve coincident with the peak of secondary negativity. The transcortical records of Pearlman show, however, that the supranormal phase is restricted to the early surface positive component. This component may reflect activity in the optic radiations and not intracortical activity (Bishop & Clare, 1952, 1953). In addition, Pearlman reports that the stimulus intensity applied to the optic nerve was higher than adequate to elicit maximal response. Demetrescu et al. (1965, p. 17) stress the importance of working with stimulus intensities near threshold otherwise responsiveness may be distorted.

Alterations in the geniculo-striate recovery cycle have been reported during arousal. Demetrescu (1969) found that arousal induced by midbrain reticular stimulation in encephale isolé cats potentiated the inhibitory phase (see also Steriade & Demetrescu, 1967; Demetrescu et al., 1965, 1966) and also facilitated recovery of the cortical response from this phase. Similarly, Demetrescu (1969) notes that arousal induced by stimulation of the reticular formation tends to bring up ". . . more regular and systematic . . ." unit activity to LGB stimulation and enhance subsequent inhibitory processes triggered by the first cortical discharge. In addition, arousal also shortened the duration of the refractory period, i.e. the rate of recovery.

Demetrescu proposes (in Demetrescu et al., 1965) that arousal constitutes a process activating both diffuse ascending facilitatory and inhibitory processes. An intracortical network of inhibitory neurones is assumed to be activated, i.e., threshold lowered, by diffuse ascending inhibitory influences arising from subcortical structures and excited, i.e., triggered, by any sufficiently strong

event arriving in the cortical primary receiving area. The existence of intracortical facilitation during arousal, however, is contentious. Steriade (1969, pp. 100-101) suggests that apparent facilitative effects were due to increased responsiveness of the stimulated thalamic relay. Steriade maintains that there is an increase in LGB excitability during arousal at the same time that the cortical effects are purely inhibitory. Evarts et al. (1960), Demetrescu et al. (1966), and Demetrescu (1967) found that in the cat intracortical inhibitory processes in the primary visual area are weak during sleep as the cortical response shows rapid recovery. Palestini et al. (1965) noted enhanced excitability of the cortex to optic radiation stimulation during sleep. During the waking state the cortex shows reduced responsiveness and slower recovery from stimulation. Evarts (1964) suggests that during sleep there may be a reduction in intracortical recurrent inhibition. These findings are consistent with the model elaborated in Study 1 suggesting that during arousal there is an attenuation of inhibition at the geniculate level and an augmentation of inhibition intracortically, both the consequence of common ascending subcortical influences.

The above works indicate that the geniculo-striate complex shows a cyclic fluctuation in recovery of excitability subsequent to visual stimulation, and that recovery is dependent upon the state of arousal. Pribram and his colleagues have suggested that attention may also alter the recovery of excitability of the geniculo-striate complex. Visual attention was associated with a large initial positive deflection of monkeys' AEP to LGB stimulation. High attention was associated with slowed flash recovery while low attention was associated with enhanced

recovery (Gerbrandt, Spinelli, & Pribram, 1970). Altered arousal was rejected as an alternative explanation of the changes in recovery. By contrast, Demetrescu et al. (1966) have noted in cat that "visual attentive behavior", like stimulation of the reticular formation, enhances the responsiveness and recovery of excitability of the neothalamo-cortical complex to LGB stimulation. Visual attention was elicited by having the experimenter enter a dimly lit room or by presenting a mouse in a cage. In neither of the above studies was attention referenced to performance.

The relationship of arousal to the geniculo-striate recovery cycle is investigated in Study 2 by subjecting Ss to a suborbital shock prior to the delivery of pulse pairs to the optic chiasma. The response to the conditioning stimulus should reveal the nature of influences associated with noxious somatic stimulation. The subsequent response to the test stimulus establishes the form of the recovery scale. The initial response and recovery cycle are also investigated under conditions in which visual or non-visual stimulation had acquired selective stimulus control. If the geniculo-striate complex is selectively reactive to visual stimulation during VSC then differences in the geniculo-striate recovery cycle might be observed under conditions of VSC and NVSC. The recovery cycles of each of the major components of the geniculo-striate AEP are assessed under the assumption that the greater the latency of the response component the less the influence of thalamic excitability and the greater the influence of intracortical excitability.

Method

The Ss, apparatus, and procedure for Study 2 have been described in Chapter I. Histology revealed that of the six Ss employed in this study all had their cortical depth electrode either within or on the borders of the white matter underlying the striate cortex. The equipment and procedures for recording and averaging the geniculo-striate recovery cycle are described below.

Recording Procedure

Each S received as the sole source of visual stimulation a series of three 50-μsec electrical pulses delivered to the optic chiasma each separated by 480 msec. A 50-μsec test stimulus was introduced at one of seven 30 msec intervals ranging from 30 to 210 msec to probe the recovery cycle after CS_1 onset. Each of the test stimuli was presented 12 times across the 96 trials constituting a session. Changes in the geniculo-striate recovery cycle were evaluated by obtaining the average cortical potential for each test stimulus interval under five conditions: the Pretest extinction session, Arousal Tests I and II, and during the fourth session of both TV and AV compound conditioning.

Recording Apparatus

The cortical activity of S was led through a Grass high impedence probe to a Grass P-511-D amplifier with a half-amplitude band width of 0.1 to 30 kHz. In order to obtain evoked potential averages at each of the test stimulus intervals, two averaging computers were employed. A CAT 1000 (Technical Instruments Corp.) was used to average the cortical response to CS_1 and the test stimuli delivered at 30, 60, or 90 msec after CS_1; each average was obtained across 256 msec with a

resolution of 1 msec per data point. A CAT 400 (Technical Instruments Corp.) was used for averaging the response to test stimuli delivered at 120-, 150-, 180-, or 210-msec intervals after CS_1; each average for the CAT 400 was obtained across 250 msec with a resolution of 2.5 msec per data point. Each CAT was triggered 20 msec prior to the delivery of the test stimulus thus enabling an average baseline of activity to be recorded for the 20-msec interval preceding delivery of the test stimulus. A plot of the average response at each test interval was obtained by means of a Moseley Co., Model 2D X-Y plotter. A calibration signal was also averaged to twelve presentations upon the completion of a test session enabling conversion of the averaged response to a microvolt scale. Digital output onto punched paper tape was obtained with a Model 535 Teletype interfaced with the CATs. The digital records were used for reproducing the averaged responses for illustrative purposes.

AEP Analysis

The electrophysiological potentials were evaluated in the following manner. The average response obtained to onset of the conditioning stimulus (CS_1) was used as a standard. The amplitude of four components of the averaged response were measured and are designated as P_1, P_2, P_3, and S. P_1 corresponds to the initial positive spike-like deflection of the response with a latency of 6-7 msec and was measured as the voltage difference from baseline (B) designated as a point just prior to stimulus delivery ($B\text{-}P_1$, Figure 15). N_1 represents the first negative deflection with a latency of 11-12 msec. P_2 corresponds to the subsequent positive deflection with a latency of 13-15 msec and is measured as the voltage difference from N_1 ($N_1\text{-}P_2$, Figure 15). N_2 corresponds to a negative deflection with a latency of

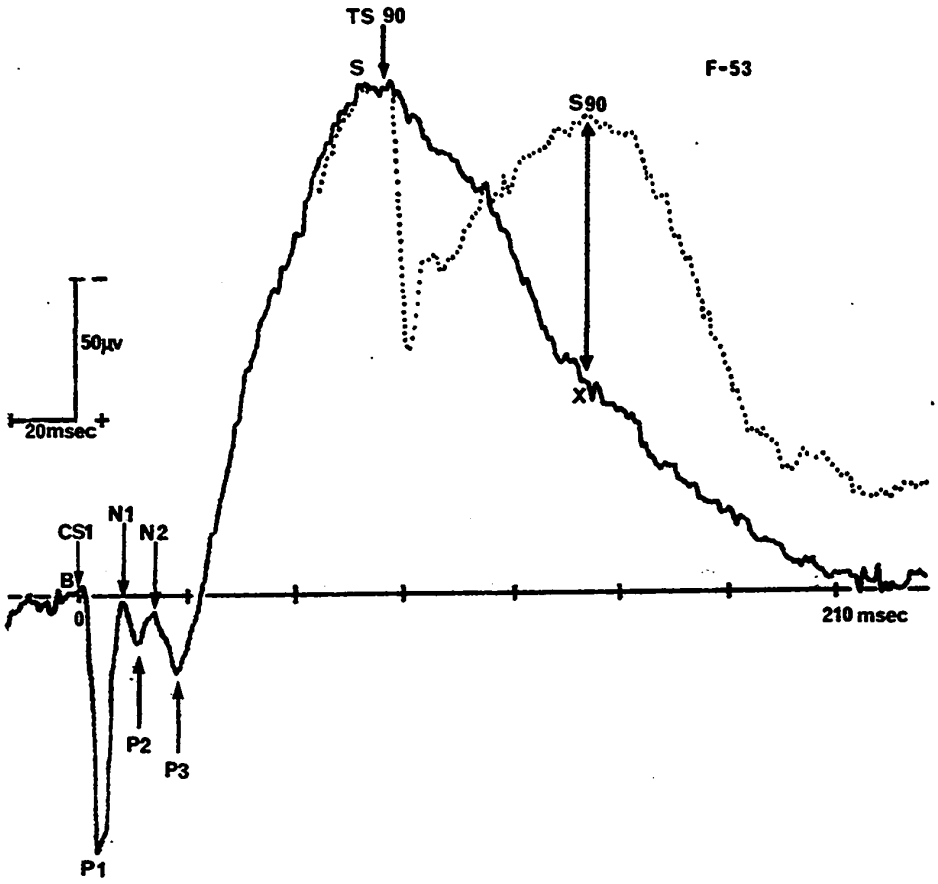

Fig. 15. An averaged (12 sweeps) evoked potential generated to optic chiasma stimulation. Represented on the AEP are the response components of the potential. B represents a baseline of activity just prior to delivery of stimulation, CS_1. The amplitude of P_1 was measured as ($B-P_1$), the amplitude of P_2 as (N_1-P_2). P_3 was measured as (N_2-P_3) and S was measured as (S-B). See text for the methods employed in assessing these response components to a test stimulus (TS) delivered at various intervals after CS_1 (e.g., TS 90 msec). Study 2. Electrophysiological

17-19 msec and P_3 corresponds to a slow positive deflection at 27 msec The voltage of P_3 was evaluated by measuring the voltage difference between the two components (N_2-P_3, Figure 15). These latencies were reliable within and between Ss; therefore, questionable identification of a response component was resolved on the basis of latency. The negative peak of the secondary component (S) had an average latency of 100 msec ranging from 88 to 110 msec between Ss in response to CS_1 during the Pretest. Measurement of the secondary peak of a test response required superimposing the response on the control response at a latency from CS_1 delivery corresponding with the delivery of the test stimulus. The voltage of S was then measured as the voltage difference between the control response and the peak of secondary negativity of the test response. This procedure is illustrated in Figure 15 for determining the amplitude of S to a test stimulus delivered 90 msec (TS_{90}) after CS_1 calculated as S_{90}-X. When the peak of S occurred more than 250 msec after CS_1, the amplitude of S was measured from the baseline of the response to CS_1.

The recovery cycle obtained during the Pretest was regarded as a control for evaluating changes in geniculo-striate excitability subsequent to somatic suborbital shock, i.e., Arousal Test I and II, and conditions of manipulated visual stimulus control. The data of Group A and Group T during the fourth session of TV and AV compound conditioning, respectively, were pooled constituting a "Visual Control Test". Recall that during these tests the AEPs were obtained to reinforced presentations of the compound stimuli. The form of the recovery cycle of the geniculo-striate complex within a test session was assessed by comparing sequential changes in response amplitudes

across the tested intervals and relating the response amplitudes to the control response to CS_1. A Groups (*df* = 2, with three *Ss* in each Group) x Tests (*df* = 5) x Intervals (*df* = 8) analysis of variance was performed on each response component with the response amplitudes subjected to a square root transformation. A square root transformation of (x + 1) was employed as the amplitudes were positively skewed, i.e., the cell means and variances were positively correlated (Winer, 1962, p. 220). Duncan's Multiple Range Tests were performed on the means of significant effects.

Results

Presentation of the results will be primarily directed to the Tests x Intervals interaction for each component considering first, changes between the two arousal tests and the Pretest, and secondly, the two stimulus control tests and the Pretest. The four analyses of variance, one for each component, are summarized in Appendix I; the *F* ratios and *p* levels for each component and within *Ss* effect are presented in Table 4. In the subsequent text the significance of differences between means as assessed by multiple range tests on the Tests x Intervals interaction are shown in parentheses.

TABLE 4

Within Ss Effects from Analyses of Variance on Each Response Component to Optic Chiasma Stimulation

		RESPONSE COMPONENT'S F RATIO			
Effect	df	P_1	P_2	P_3	S
Tests (A)	4/16	3.25**	2.19	6.91*	3.62**
Intervals (B)	7/28	39.73*	4.50*	41.87*	34.73*
A x B	28/112	4.07*	1.61**	10.88*	5.05*

Note: See Appendix I for a complete summary of each analysis conducted on the P_1, P_2, P_3, and S response amplitudes.

*Significance at .01 level of probability
**Significance at .05 level of probability

The Geniculo-striate Recovery Cycle During Arousal

Figure 16 represents the AEPs of a single S to the conditioning stimulus, CS_1, and to each test stimulus delivered after CS_1. Each potential represents the average of 12 responses during the Pretest session. The test responses have been superimposed on the control response at latencies coinciding with the delivery of the test stimulus. Note the refractoriness of the primary components through 60 msec, and recovery at subsequent latencies for the initial component, P_1, and the absolute refractoriness of P_3 through 210 msec. P_2 shows some evidence of recovery at the 90 to 150-msec latencies. Also note that the secondary component is relatively refractory through 150 msec after which full recovery is evident. These observations represent the general trends of the recovery cycles for the individual components under Pretest conditons.

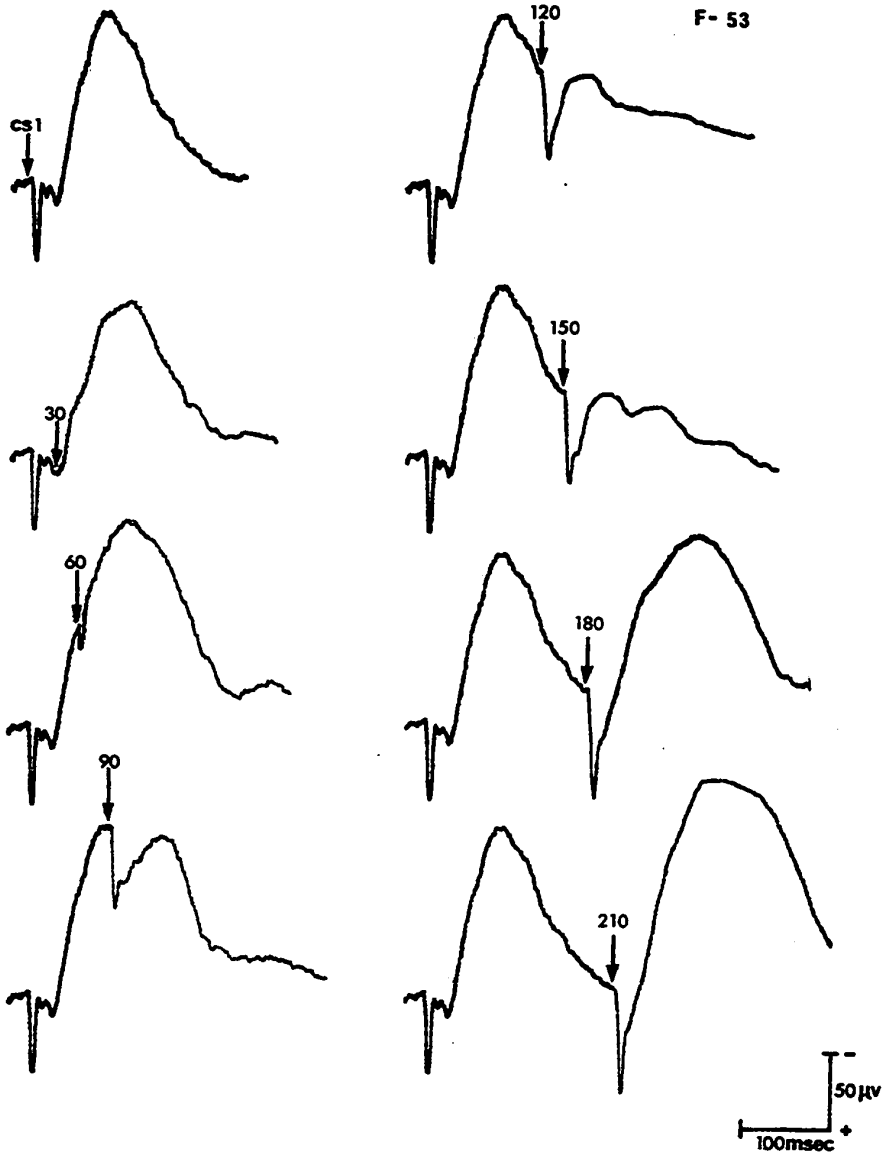

Fig. 16. The evoked potential elicited to the conditioning stimulus, CS_1, and subsequent test stimuli delivered at 30- to 210-msec intervals after CS_1. The test response has been superimposed on the control response at latencies corresponding to the delivery of the test stimulus. Study 2. Electrophysiological

Figure 17 illustrates the control response of two Ss during the Pretest and Arousal Tests I and II. The upper potential represents an S with small response components and the lower respresents an S with distinct components during the Pretest. The average percentage change in the response components to CS_1 between the Pretest and the two arousal tests is shown in Figure 18. The most prominent change during each arousal test was a significant ($p < .01$) increment in the secondary component and reduction in the P_3 component in relation to the Pretest response. Both effects were greater ($p < .05$) during Arousal Test I.

Figure 19 illustrates the relationship between the recovery cycles for each component during the Pretest and the arousal tests. Consider the P_1 component's recovery cycles. During the Pretest, P_1 was absolutely refractory at 30 msec showing evidence of recovery at 60 msec and attaining the control amplitude by 90 msec. By contrast, during the arousal tests, P_1 was refractory through 90 msec attaining the control amplitude at 120 msec during Test II and 150 msec during Test I. P_1 was depressed below Pretest amplitudes at 60 msec ($p < .05$) during Arousal Test I and 90 msec during both Arousal Tests I ($p < .01$) and II ($p < .05$). Note also that there was a reduction in P_1 during both arousal tests at 210 msec ($p < .05$, Test I, and $p < .01$, Test II), a trend initiated after 150 msec. Thus, while the noxious suborbital shock did not significantly alter the amplitude of the initial deflection to the conditioning stimulus, it did retard the rate and level of recovery as P_1 tended to be refractory at latencies greater than 150 msec.

Consider recovery of the P_2 component (Figure 19). P_2 was absolutely refractory at 30 msec and relatively refractory at 60 msec

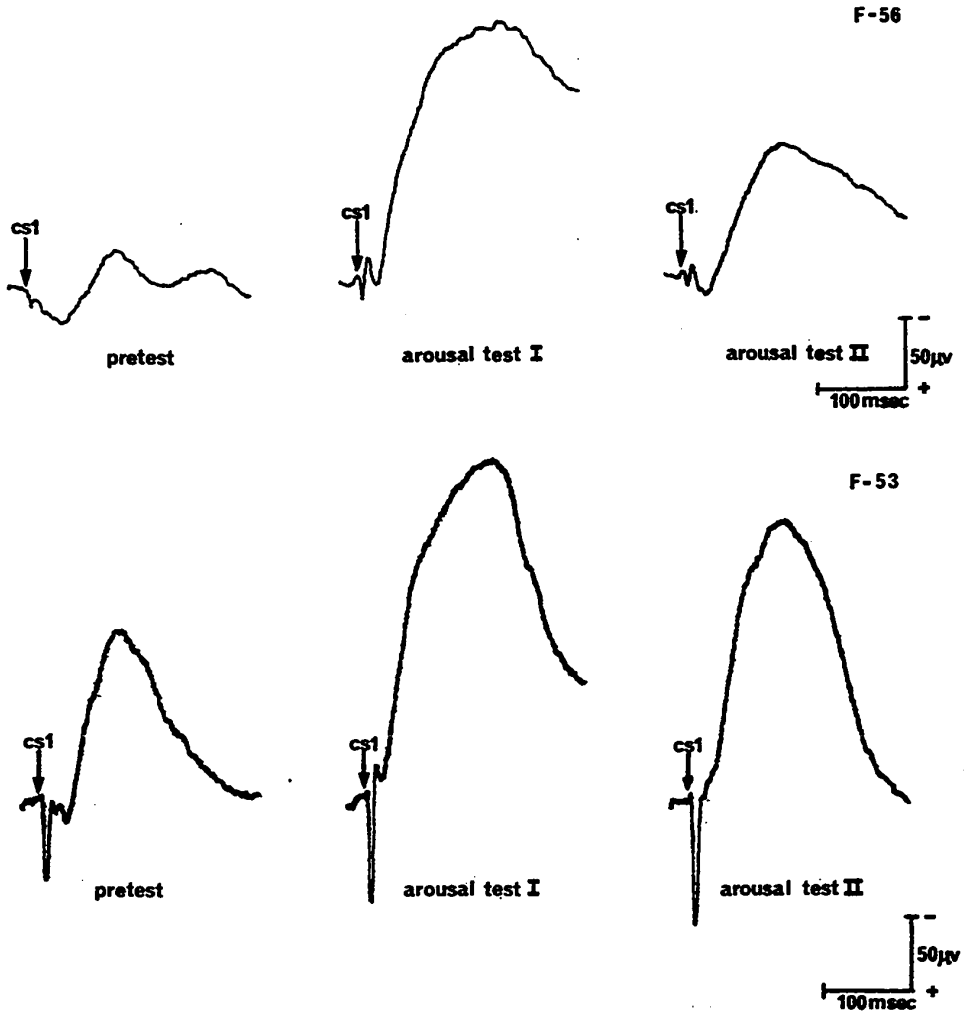

Fig. 17. The geniculo-striate response to the conditioning stimulus, CS_1, as recorded during the Pretest and each arousal test. Represented in the upper portion is an S (F-56) with small response components during the Pretest. The lower traces represent an S with distinct response components during the Pretest (F-53). Study 2. Electrophysiological

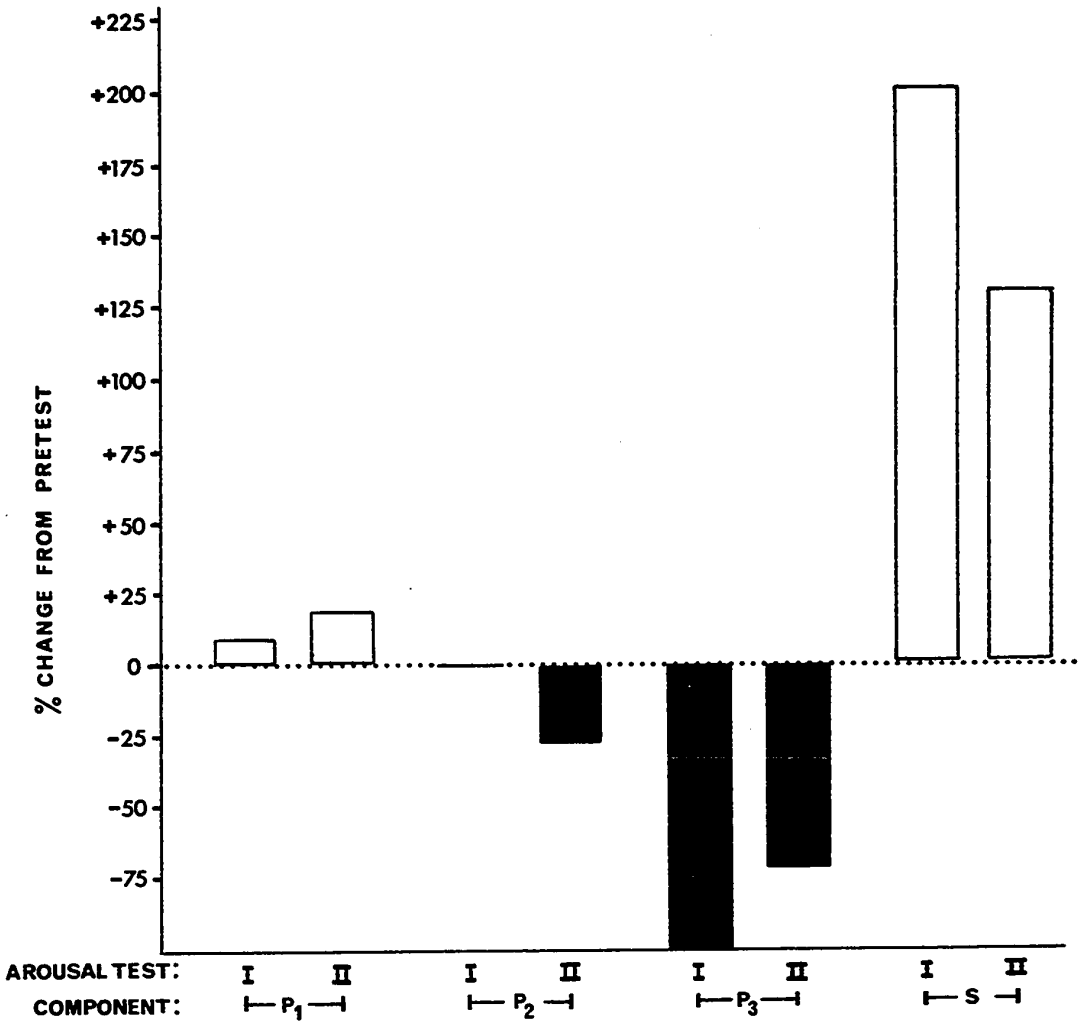

Fig. 18. The mean (N = 6 Ss) percentage change in the amplitudes of the geniculo-striate response components to optic chiasma stimulation during the two arousal tests. The changes are expressed in relation to the Pretest. Suborbital somatic shock preceded chiasmic stimulation by 250 msec (Test I) or 2,000 msec (Test II) during the arousal tests. Study 2. Electrophysiological

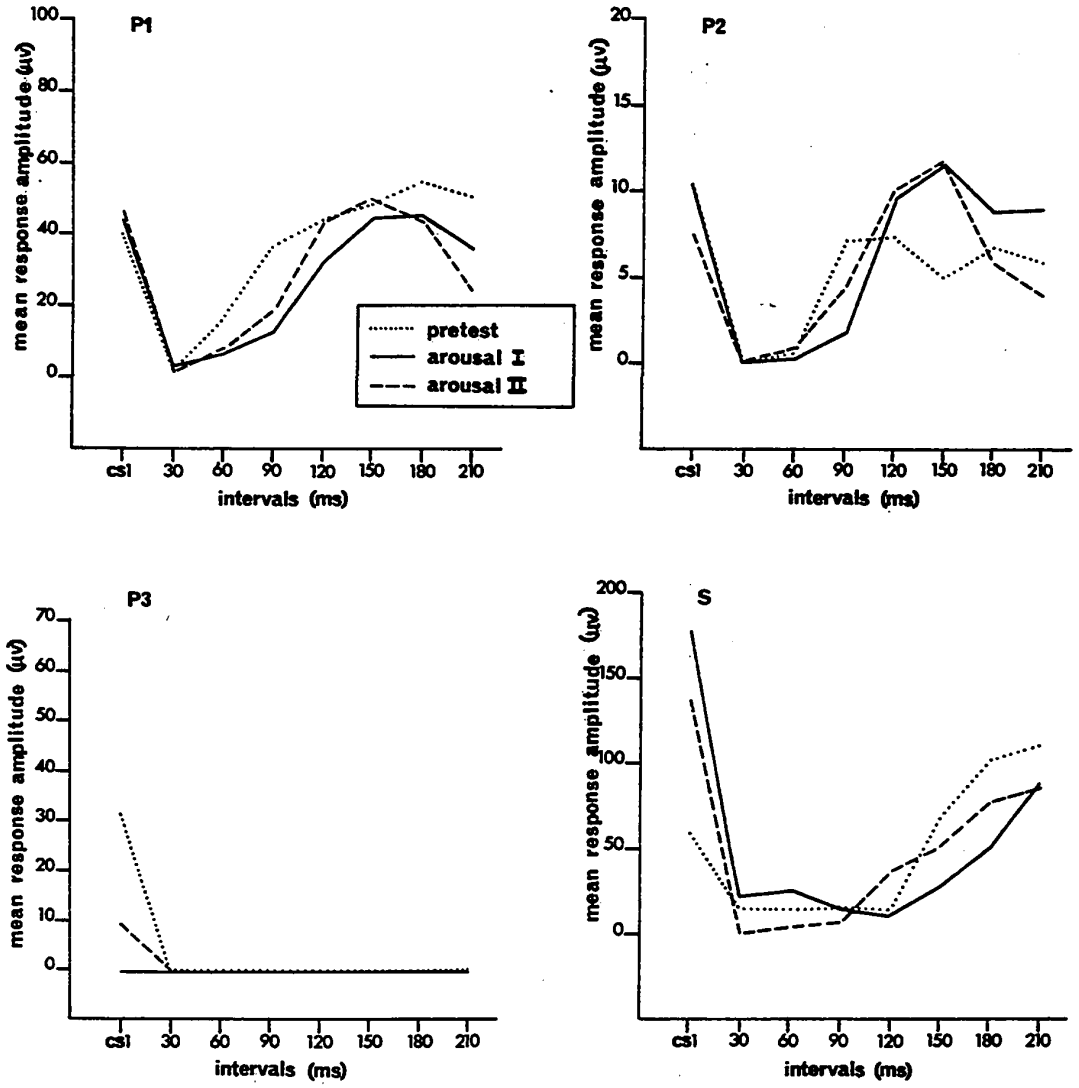

Fig. 19. The geniculo-striate recovery cycles up to 210 msec after onset of the conditioning stimulus, CS_1, for P_1, P_2, P_3, and S as assessed during the Pretest and each arousal test. The response amplitudes to the conditioning stimulus and at each of the seven test intervals represents the mean of six Ss. Study 2. Electrophysiological

during all three tests. Recovery of P_2 was evident during Pretest by 90 msec while during Arousal Test I and to a lesser extent Test II full recovery was not achieved until 120 msec. Thus, while the amplitudes at corresponding latencies did not significantly differ between the Pretest and arousal tests, the form of the recovery cycles suggest that P_2, like P_1, recovered somewhat slower following suborbital somatic shock.

P_3, representing the terminal positive response of primary activity, was more readily apparent in response to CS_1 during Arousal Test II than during Arousal Test I ($p < .01$). There was no evidence of recovery of P_3 during either the Pretest or the two arousal tests.

The recovery of the S component of the cortical response was inversely related to the amplitude of S produced by CS_1. During the Pretest, S declined between CS_1 and 30 msec remaining refractory (re: CS_1) through 120 msec ($p < .01$, at each interval) and recovering to control levels by 150 msec. Subsequent to suborbital shock S was enhanced over Pretest levels in response to CS_1 ($p < .01$, Arousal Test I and $p < .05$, Arousal Test II) but was relatively refractory (re: CS_1) at subsequent latencies through 210 msec for Test I and 150 msec for Test II ($p < .01$). Thus, S showed reduced recovery during the arousal tests. Retarded recovery was more evident when suborbital shock preceded CS_1 by 250 msec (Arousal Test I) than 2,000 msec. Secondary negativity was also reduced in latency to initiation and increased in amplitude, duration, and latency to peak negativity in response to CS_1 during the arousal tests (see Figure 17). These effects were also observed during Pretest recovery of S (see Figure 16).

Thus, during the Pretest, P_1 showed recovery by 90 msec as did P_2. P_3, on the other hand, never showed recovery within 210 msec while S

recovered to supranormal levels after 150 msec. When preceded by suborbital somatic shock recovery of these components was retarded.

The Geniculo-striate Recovery Cycle During Selective Stimulus Control

Changes in response to CS_1, reflecting influences predominant prior to CS_1 delivery, were detected between the Pretest and the two stimulus control tests and are consonant with evidence presented in Study 1. The control responses of two Ss during the Pretest and both stimulus control tests are presented in Figure 20. The upper traces illustrate the responses of an S from Group A; the lower traces illustrate an S from Group T. Figure 21 represents the response components to CS_1 during the two stimulus control tests as the average percentage of the Pretest response. Common to both stimulus control tests was a decrement in P_2; however, only during NVSC was this effect significant ($p < .05$, re: Pretest and NVSC). Thus, with the exception of P_2, the primary activity elicited to CS_1 during VSC exceeded levels observed during all other test conditions. There was little change in S activity associated with either stimulus control test though five of the six Ss showed a higher level of S during the VSC Test than during the NVSC Test. See Appendix I for a representation and discussion of changes in geniculo-striate activity observed during compound conditioning and in response to non-visual stimulation alone as observed during differential conditioning.

Consider the recovery cycle of P_1 during the two stimulus control tests illustrated in Figure 22. The form of recovery during both stimulus control tests resembled that seen during the Pretest, a short refractory period followed by recovery to CS_1 levels by 90 msec. Only in the case of NVSC did recovery rise above CS_1 levels within the test;

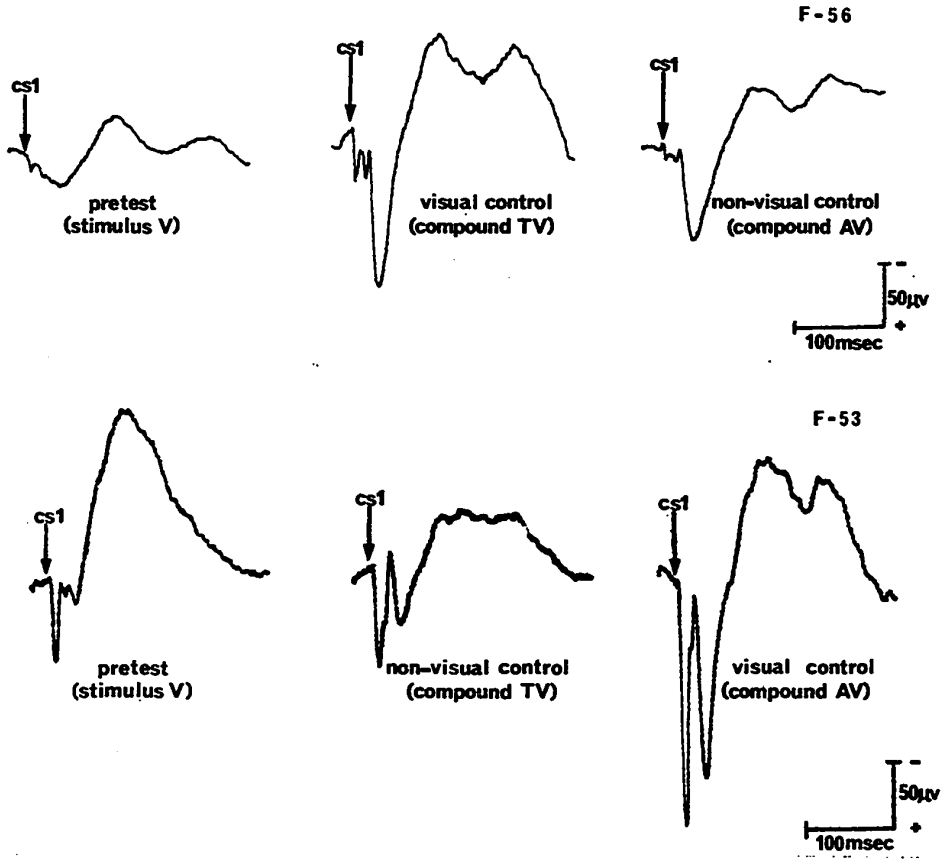

Fig. 20. The geniculo-striate response to the conditioning stimulus, CS_1, during each stimulus control test. Represented in the upper portion is an S (F-56) from Group A. The lower averages represent an S from Group T (F-53). Study 2. Electrophysiological

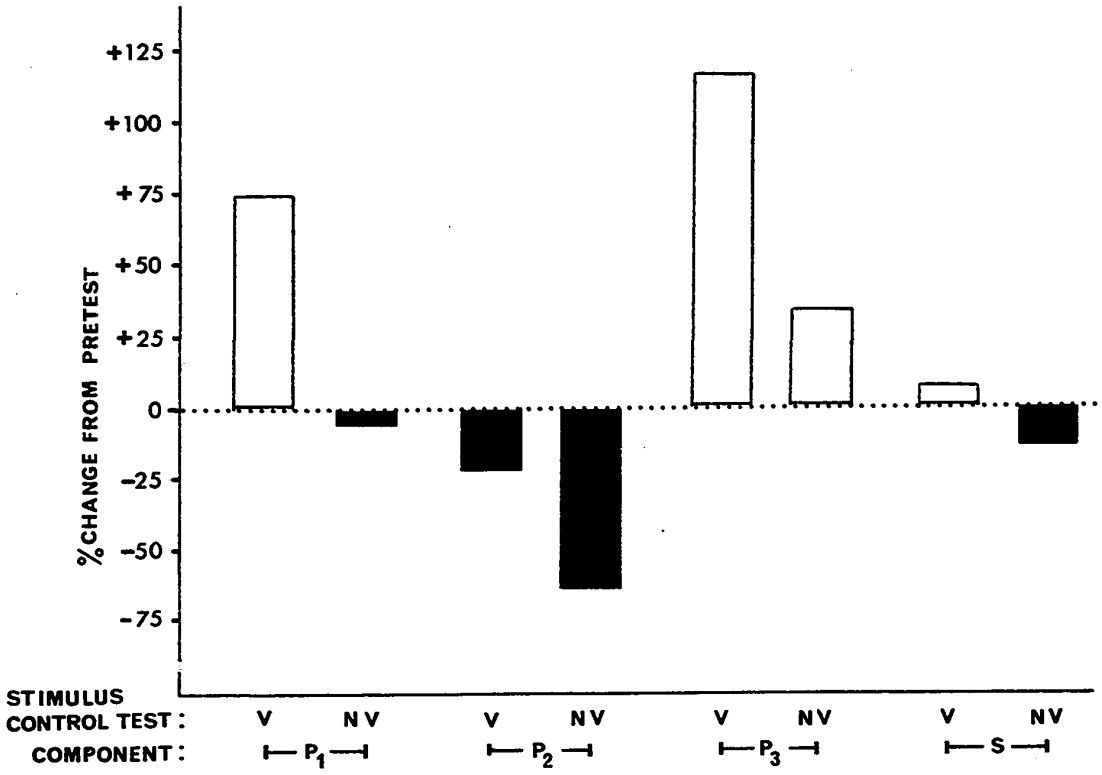

Fig. 21. The mean (N = 6 Ss) percentage change in the amplitudes of the geniculo-striate response components to optic chiasma stimulation during the visual (V) and non-visual (NV) stimulus control tests. The changes are expressed in relation to the Pretest. Optic chiasma stimulation was presented in compound with a differentially non-reinforced audio or tactile element during V and a differentially reinforced element during NV. Study 2. Electrophysiological

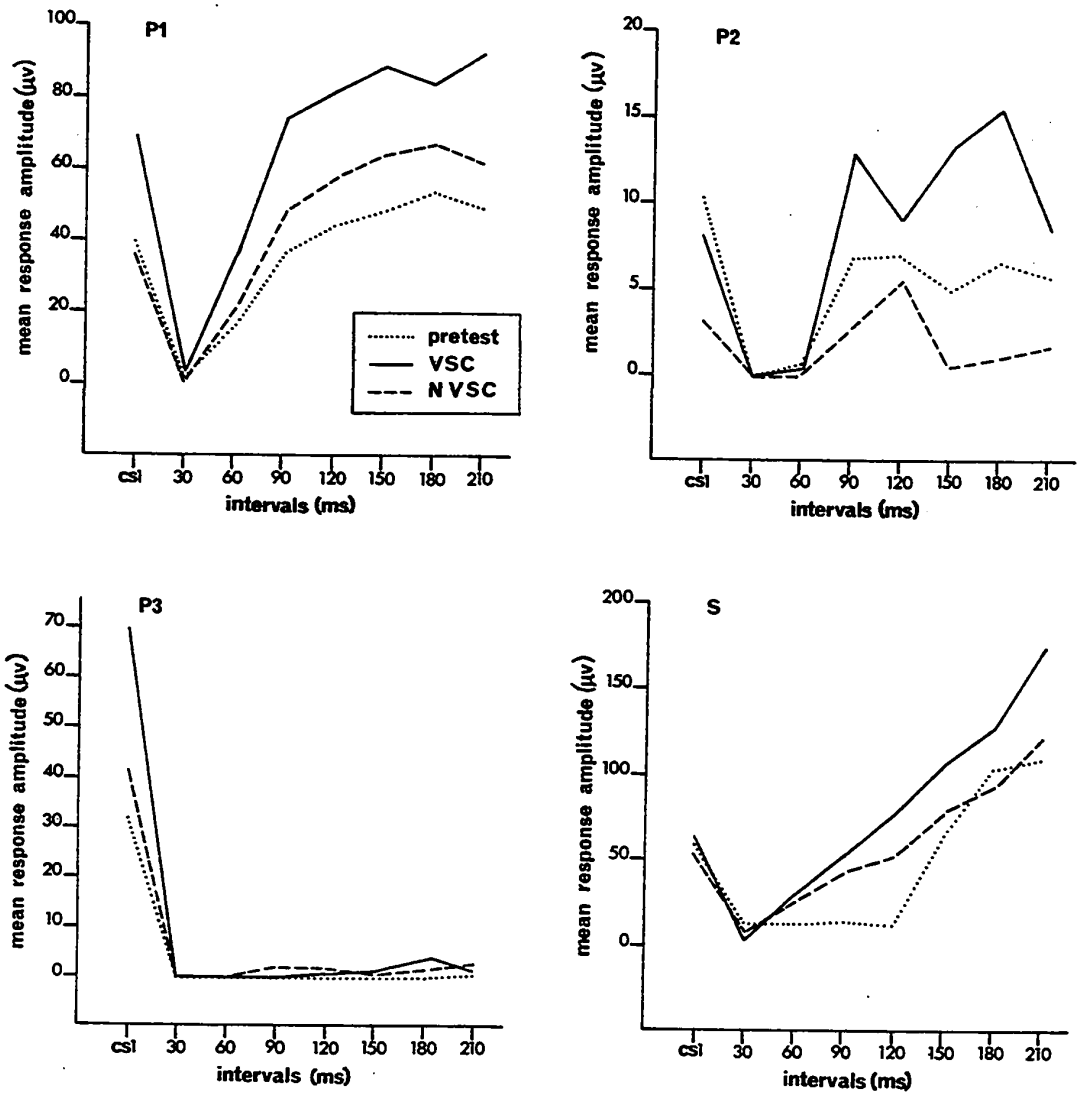

Fig. 22. The geniculo-striate recovery cycles up to 210 msec. after onset of the conditioning stimulus, CS_1, for P_1, P_2, P_3, and S as assessed during the Pretest and each stimulus control test. The response amplitudes to CS_1 and at the seven test intervals represent the means of six Ss. Study 2. Electrophysiological

P_1 response amplitudes at latencies greater than 120 msec exceeded the response to CS_1 ($p < .01$, at each interval). However, the amplitudes of P_1 during NVSC, though consistently higher after 30 msec , were not significantly different from the Pretest amplitudes at corresponding latencies.

P_1 was facilitated to CS_1 during VSC and was significantly higher than corresponding Pretest amplitudes at 60-210 msec ($p < .01$ each) and NVSC Test amplitudes at 90-210 msec ($p < .01$ each). Thus, the geniculo-striate complex was more responsive during VSC, showing the highest absolute level of recovery between all tests. A sustained rate of recovery during NVSC through 120 msec yielded the highest degree of relative recovery within any test condition.

The recovery cycles of P_2 during the two stimulus control tests are illustrated in Figure 22. The level of recovery and the response amplitudes at corresponding latencies after 60 msec during the Pretest were intermediate to levels assessed during the two stimulus control tests. P_2 recovered rapidly during VSC between 60 and 90 msec to higher levels at 150 and 180 msec ($p < .01$ each) than evident at corresponding latencies during the NVSC Test. Thus, there was a significant tendency for P_2 to recover to higher levels during the VSC Test than during NVSC. This tendency was also evident in relation to the Pretest though not significant.

P_3 was absolutely refractory at all intervals up to 210 msec during the Pretest; however, the small response amplitudes initiated after 60 and 90 msec during NVSC and VSC indicate that the geniculo-striate complex was not absolutely refractory for P_3 under conditions of stimulus control.

S activity recovered rapidly to normal by 90 msec during both stimulus control tests while during the Pretest, CS_1 response amplitudes were not evident until 150 msec. Further, the supranormal level of recovery evident at 210 msec was significant during both NVSC ($p < .05$, re: CS_1) and VSC ($p < .01$, re: CS_1). S was also above Pretest levels at 120 msec during VSC ($p < .01$). Thus, during VSC and, to a lesser extent, NVSC, S recovered faster to a higher level than seen during the Pretest.

Generally, the level of recovery of the cortical potential to optic chiasma stimulation was higher during VSC, and to a lesser extent during NVSC, than evident during the Pretest. P_1 showed faster recovery during NVSC while S showed a facilitation in the rate of recovery during both VSC and NVSC.

Discussion

The following discussion is directed to two points: first, what influences predominate on the geniculo-striate complex at stimulus onset during selective stimulus control and arousal? This issue was addressed in part in Study 1 and is briefly treated here. Major discussion is directed to the second point, an interpretation of the recovery functions in light of two hypotheses advanced in Study 1: (1) arousal generally attenuates thalamic recurrent inhibition and augments intracortical recurrent inhibition and (2) selective VSC reduces, while NVSC enhances, the influence of recurrent collateral inhibition at the LGB.

Geniculo-striate Excitability at Onset of Optic Chiasma Stimulation

Only during selective VSC was a general enhancement of primary activity evident to onset of the conditioning stimulus. The dramatic enhancement of P_3 (probably of intracortical origin) is likely attributable to an enhancement of afferent input (P_1 amplitude) to the striate cortex at stimulus onset. These observations are consonant with the hypothesis that during selective VSC the relay cells of the dorsolateral geniculate are disinhibited. The attenuation of P_2 during selective NVSC may be attributed to a somewhat lower level of specific LGB output (P_1) and/or active inhibitory processes associated with selective non-visual attention.

That S was not significantly altered in response to CS_1 during either the VSC or NVSC compound stimulus presentations is consistent with the findings of Study 1. Secondary activity to optic chiasma stimulation increased subsequent to CS onset in correspondence with a slow phasic increment in arousal (intracortical inhibition) peaking at 500 msec or just prior to CR emission.

The lack of a significant enhancement of early primary activity during the arousal tests is inconsistent with the hypothesis that during arousal there is a significant increment in thalamic excitability (Dumont & Dell, 1958; Bremer & Stoupel, 1959; Suzuki & Taira, 1961; Long, 1959; Skinner & Lindsley, 1967, 1971; Ogawa, 1963; Iwama & Yamamoto, 1961; Fukuda & Iwama, 1970; Bremer, 1970). Two alternative explanations seem likely here. The low level of early primary activity may reflect a reduction of LGB responsiveness during non-visual attention to the suborbital shock. Such an effect would act to counter the general facilitating influence of arousal at the geniculate level. In this

instance the so-called "arousal tests" may provide indirect evidence that an active attenuation of LGB excitability occurs during attention to, or distraction by, a non-visual stimulus. The difference between the arousal tests and NVSC may be attributed to the greater saliency of the suborbital shock as opposed to the conditioned non-visual stimuli. This position would also predict a real attenuation of primary activity under conditions of NVSC and low arousal. A second possibility is that early primary activity represents a composite of geniculate and intracortical events and that intracortical inhibition, enhanced during arousal, offsets the enhanced input from the geniculate.

The severe attenuation of P_3 and dramatic enhancement of S subsequent to a suborbital shock provides support for the hypothesis that arousal activates intracortical inhibition. The enhancement of S is not believed to be related to the reflex eyeblink to the somatic shock. The eyeblink was terminated before the onset of the conditioning stimulus. The reduction of S between Arousal Test I (US-CS interval 250 msec) and Arousal Test II (2,000 msec) may reflect the phasic character of shock-elicited arousal. That is, the greater the interval between shock and optic chiasma stimulation, the lower the level of arousal at stimulus onset. This reduction may, however, be due to an order effect such that during Test II there was a reduction in the arousing character of the shock, i.e., habituation to the shock.

Changes in the amplitude of the components of the geniculo-striate AEP are in correspondence with data advanced in Study 1. Controlling for arousal, primary activity appears to reflect modality specific attentional processes associated with an increase in responsiveness of specific thalamic nuclei for the modality under stimulus control. A

reduction in responsiveness is suggested if S is attending another stimulus. These influences can apparently be dissociated from the general enhancing influence of arousal on all specific thalamic nuclei Secondary activity, triggered by afferent input, was independent of the level of associated primary activity and seemed to reflect the level of intracortical inhibition which is enhanced during arousal.

Geniculo-striate Excitability Subsequent to Optic Chiasma Stimulation

Certain variables cannot explain the recorded alterations in the recovery cycles. Since the visual stimulus employed was delivered to the optic chiasma against a background of low illumination, retinal influences may be excluded as mediating the effects. Also, the visual stimulus was paired with auditory or tactile stimuli with the same effect and the order of establishing VSC and NVSC was balanced. Since the recovery cycles were assessed with the visual stimulus in compound with the non-visual stimulus the significance of the compound presentations cannot be assessed. However, as noted in Study 1, compound stimulus presentations tend to reduce the enhancing effects of VSC and the attenuating effects of NVSC. Whether the recovery of excitability is similarly affected or actually potentiated by processes mediating selection of a relevant stimulus from a compound was not evaluated.

Stimulus Control Tests. The absolute refractoriness of initial activity 30 msec after CS_1 is consistent with the hypothesis that afferent input to the LGB triggers recurrent collateral inhibition (Eccles, 1969). The reduction of P_1 between 0 and 30 msec during VSC is attributable to the response to CS_1 exciting recurrent collateral inhibition and briefly overcoming the selective enhancement of LGB excitability associated with visual attention. Input via optic chiasma

stimulation is considered constant; however, the existence of presynaptic inhibition at the level of the LGB is uncertain (Creutzfeldt & Sakmann, 1969; Szentágothai, 1967).

The same influences which during VSC enhance the P_1 response to the conditioning stimulus likely contribute to the absolute level of P_1 during recovery. The supranormal level of early primary activity, i.e., P_1 and P_2, during VSC as compared with NVSC is consistent with the hypothesis that during selective visual attention there is a reduction in tonic activity of inhibitory interneurones at the LGB. Also contributing to P_1 recovery may be influences associated with phasic arousal initiated at stimulus onset. Increasing arousal would result in an increase in LGB responsiveness. Under circumstances during which the CS could be expected to elicit conditioned phasic arousal, i.e., the stimulus control tests, recovery would be expected to exceed Pretest levels and also the level relative to CS_1 within a test. This would account for the facilitation in the rate of P_1 recovery during NVSC over Pretest levels. The lack of a facilitation during VSC, however, is inconsistent with the hypothesis that visual attention effects a reduction in LGB recurrent collateral inhibition. The lack of an evident reduction in LGB recurrent inhibition could be due to a ceiling effect on disinhibition of the LGB associated with visual attention.

An alternative to LGB disinhibition would be that the selective increase in LGB responsiveness during VSC reflects a direct facilitation of LGB neurones or perhaps a reduction in LGB presynaptic inhibition (see Cohen & Vendrick, 1972). Similarly, a facilitated recovery rate

could be attributed to a direct facilitation of LGB excitability or a reduction in LGB presynaptic inhibition associated with conditioned phasic arousal. This position, like LGB disinhibition, however, also leaves unexplained the lack of an enhanced recovery rate during VSC. The data do not enable a clear resolution of the nature of the mechanism mediating changes in P_1 amplitude.

While the level of P_2 in response to CS_1 and throughout recovery was attenuated during NVSC below VSC levels, the level of recovery within either test was not significantly different. Thus, P_2 seems to reflect exclusively those tonic inhibitory influences associated with selective NVSC and is not subjected to the same phasic influences as P_1.

The absolute and relative level of recovery of secondary activity was facilitated during VSC and, to a lesser extent, NVSC. This effect might represent an interaction between: (1) greater specific output of the LGB associated with increased LGB excitability and (2) increasing intracortical inhibition associated with increasing phasic arousal. Supin (1966, 1968) and Polyanskii (1965, 1967) have suggested that secondary activity represents, at least in part, intracortical IPSPs in the depths of the striate cortex.

The general lack of recovery of late primary activity reflected in P_3 could be attributed to increasing intracortical inhibition associated with phasic arousal. A hint of P_3 recovery during the stimulus control tests may be due to greater afferent input to the cortex offsetting the attenuating influence of increasing intracortical inhibition. P_3, it might be added, did not recover during the Pretest session and was

rarely seen under any conditions as late as 330 msec after stimulus onset (see Study 1). While highly speculative, P_3 may reflect activity associated with orienting at the onset of significant visual stimulation.

The above data are consistent with the generalization that the primary phase (0 - 40 msec) of the visual AEP to optic chiasma stimulation as recorded from the striate cortex represents a phase of intense LGB inhibition and increasing intracortical inhibition. At latencies corresponding to the initiation of secondary activity (40 - 60 msec) there is a gradual recovery of LGB excitability while intracortical inhibition intensifies (i.e., the consequence of increasing phasic arousal). At the peak of secondary activity, LGB recovery as evidenced by the amplitude of P_1, is supranormal while intracortical inhibition, as evidenced by the amplitude of P_3 and S, is severe.

It might be noted that the enhanced rate of P_1 recovery during NVSC may not be inconsistent with the findings of Gerbrandt et al. (1970) indicating slowed flash recovery during visual attention. They noted "that the later components of the averaged potentials elicited in the striate cortex by LGB stimulation were increased considerably by allowing the animal [monkey] to look out the front of the experimental box" (p. 148). Concluding that an enhanced EP reflected a high state of attention they proceeded to monitor the EP to LGB stimulation prior to the delivery of pairs of flashes. When a large EP (peak-to-peak amplitude of the first major deflection) was elicited by LGB stimulation, flash recovery rate was retarded; this effect was not evident until 120 msec after onset of the first flash (see Fig. 7 in Gerbrandt et al., 1970, p. 151). The

fact that the recovery functions were based on the first major deflection to a photic stimulus leaves unclear to what degree the recovery functions reflect geniculate or cortical events. If it is primarily cortical, then their results are consistent with the hypothesis of increased cortical recurrent inhibition during arousal. That is, the rate of recovery of intracortical afferent activity would be inversely related to the degree of intracortical recurrent collateral inhibition.

Arousal Tests. The recovery cycle data subsequent to suborbital somatic shock suggest a facilitation of recurrent collateral inhibition at either the LGB or cortex or both. Consider first the recovery of secondary activity again assuming that S reflects IPSPs at the depths of the striate cortex. The depressed S recovery during the arousal tests could be due to subliminal intracortical inhibition activated by suborbital shock and triggered by recurrent activity associated with afferent input. The end effect would be a reduction in subsequent afferent input and, therefore, the generation of stimulus contingent secondary activity. Depressed S recovery may also reflect occlusion as the result of an enhancement and earlier initiation of S in response to CS_1. Contributing also to the slow recovery may be the low level of recovery of primary activity. Further, recall that during the stimulus control tests arousal was increasing after CS onset while during the arousal tests one might expect arousal to be decreasing at least during Arousal Test II, when the CS was delivered 2,000 msec after the arousing shock. The P_3 data are similarly consistent with the inhibition hypothesis; note that the amplitude of P_3 and S were inversely related

throughout this and the preceding study. The amplitude of P_3 was attenuated if S was high and facilitated if P_1 was high and S was low. This further supports the contention that P_3 reflects intracortically transmitted afferent input through the striate cortex evident under conditions of low intracortical inhibition (low S) and abolished if intracortical inhibition is evident (high S regardless of the amplitude of P_1). The recovery of P_2, while somewhat retarded before recovery to supranormal levels, is difficult to interpret; in any case, these data are inconclusive as significant differences from the Pretest were generally lacking. Differences between the two arousal tests, e.g., faster recovery during Arousal Test II, is probably due to the latency differences between shock and CS_1 onset and a consequent lower level of arousal during Test II. As indicated earlier, habituation of arousal between Tests I and II may have been a contributing factor.

The response of P_1 to CS_1 following suborbital shock has been discussed as reflecting a reduction of LGB responsiveness due to attention to noxious somatic stimulation. This effect is also apparent in the recovery of P_1. During the Pretest, P_1 showed recovery by 90 msec while during Arousal Test II recovery was retarded to 120 msec and during Test I to 150 msec. The retardation is interpreted as an attenuation of LGB excitability. Enhanced intracortical inhibition during intense arousal may also retard P_1 recovery to the extent that P_1 reflects some degree of intracortical excitability. Recovery was also below levels evident during the Pretest and stimulus control tests. These data are consistent with the earlier interpretation that the suborbital shock had

specific as well as non-specific effects. While somatic shock induced arousal, it seems to have also elicited attention thereby attenuating influences which during arousal and/or attention to visual stimulation, act to enhance LGB excitability. Whether the attenuation is the consequence of increased recurrent collateral inhibition (i.e., less inhibition of inhibitory interneurones) or the consequence of a direct enhancement of LGB post-synaptic or presynaptic inhibition must await further investigation.

Note we have discussed the results of the stimulus control tests (re: the Pretest) in terms of the influence of arousal while contrasting the two tests in terms of the influence of selective attention. The arousal tests (re: the Pretest) were discussed in relation to the effect of non-visual attention elicited by the suborbital shock while differences between the two tests were attributed to differential levels of arousal. In other words, the stimulus control tests revealed an arousal effect in the LGB, while the arousal tests produced an attention effect. Actually both are attention effects; it depends on which thalamic nucleus one looks at.

The validity of many of the speculations raised in Study 1 and Study 2 must await further investigation seeking to elaborate the origin and character of influences determining the recovery cycle to central afferent stimulation at both the level of the LGB and intracortically to optic radiation stimulation. This might be carried out under conditions of low arousal, peripherally induced arousal, mesencephalic and metencephalic stimulation, orbitofrontal and inferotemporal stimulation, and as a

function of VSC and NVSC and visual and non-visual stimulus distraction. Until such information is available the significance of some of the changes in the cortical evoked potential and recovery cycle of the rabbit AEP to visual stimulation must remain speculative. Particularly critical to such a program of research is the necessity to assess the contribution of geniculate and cortical activity to the initial components of the rabbit cortical AEP to optic chiasma stimulation.

Summary

Pulse pairs were delivered to the optic chiasma subsequent to a suborbital shock and under conditions of selective visual and non-visual stimulus control of a conditioned eyeblink response. The recovery of primary and secondary components of the geniculo-striate AEP to optic chiasma stimulation were assessed. The somatic shock as well as the significant element of a visual-non-visual compound stimulus had apparent specific, attention eliciting, and non-specific, arousing, effects. The recovery functions indicated that: (1) during arousal there is a subliminal enhancement of intracortical inhibition facilitating secondary activity (reflecting intracortical IPSPs) to stimulus onset. There is also a subsequent enhancement of intracortical recurrent inhibition retarding recovery of intracortical excitability. At the geniculate level, arousal effects a general increase in excitability of specific relay cells thereby enhancing primary activity to stimulus onset. Phasic arousal initiated to onset of significant stimulation also appears to enhance geniculate excitability and facilitate recovery.

(2) During visual attention the LGB shows a tonic increase in responsiveness reflected as an increase in early primary activity to visual stimulus onset and an enhancement in the absolute level of recovery of early primary activity. (3) During non-visual stimulus attention (elicited by suborbital shock or during NVSC) the excitability of specific thalamic nuclei for the non-attended modalities is attenuated reducing early primary activity to onset of visual stimulation and reducing the absolute level of recovery of early primary activity. While attention to suborbital shock retarded the recovery rate of primary activity, attention to the conditioned non-visual stimulus element enhanced recovery rate. The enhancement was not evident during VSC, an effect inconsistent with a model described in Study 1. An overall assessment of the data suggest that activity associated with arousal and selective attention interact to modulate geniculo-striate excitability.

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APPENDIX A

BLOCKING AND CS-US PARAMETERS: PILOT WORK

An experimental design was sought which would enable the establishment and blocking of visual stimulus control (VSC) within Ss without altering the reinforcement contingency associated with the visual stimulus. Using rabbits and the conditioned eyeblink response, Wagner, Logan, Haberlandt, and Price (1968) showed that stimulus control acquired by a stimulus A reinforced in compound with stimulus B was a function of the reinforcement contingency associated with stimulus B. If stimulus B was reinforced when alone (P = +1.00, the Correlated condition) then the acquisition of stimulus control by A was blocked during AB compound conditioning. However, if B was reinforced in compound with A but not when presented alone (P = +.50, the Uncorrelated condition) then stimulus A acquired stimulus control. We adopted a similar compound conditioning approach in two pilot studies.

DESIGN I. This design consisted of two phases. During Phase I, six Ss received Correlated eyeblink training during which 4-Hz photic (intensity 4, Grass PS-2 photostimulator) or optic chiasma stimulation (50-μsec pulses at three times threshold) was reinforced in compound with US-correlated clicks (12 Hz at 21 db above 64 db background white noise). The remaining six Ss received Uncorrelated training such that the visual stimulus was reinforced in compound with clicks which were uncorrelated with reinforcement (P = +.50). VSC was assessed by presenting the visual stimulus alone after asymptotic performance was evident to the audio-visual compound. During Phase II the Correlated

and Uncorrelated conditions were reversed for the respective Ss.

Of the twelve Ss four failed to show any evidence of conditioning within five sessions of 90 trials each. Three of these Ss, one receiving photic and the others chiasmic stimulation, were from the Uncorrelated condition and expected to yield VSC. The lack of any conditioning here is probably due to an insufficent intensity of optic chiasma stimulation and/or overshadowing of the visual stimulus by the more salient clicks.

TABLE A-1

Mean Percentage of Responses to the Visual and Auditory Elements and Compound Following Uncorrelated (U) and Correlated (C) Training in Phases I and II

Phase	Training Condition		Stimulus		
	Phase I	Phase II	Visual	Auditory	Compound
I	U		53%	20%	88%
	C		11%	80%	97%
II	U	C	41%	23%	94%
	C	U	19%	12%	91%

As expected, those Ss which demonstrated some conditioning (see Table A-1) showed less VSC in Phase I after Correlated than after Uncorrelated training. This was not the case, however, for Phase II. There was only a 12% decrement in VSC after Correlated training. Further, only an 8% increment in VSC was evident following Uncorrelated training. Partial reversal may indicate that a change in reinforcement contingencies without altering CS characteristics may be less than optimal for full or rapid reversal of stimulus control.

Some degree of stimulus control also appears to have been acquired by the compound stimulus in Phase II for the Uncorrelated condition; neither element shows appreciable stimulus control. This may be due to low salience of the visual stimulus and non-reinforcement of the clicks after stimulus control by the click had been established in Phase I. This combination of circumstances likely favored conditioning to the unique properties of the compound. In any case, the small shifts of VSC between Phases I and II were not particularly encouraging for planned within S comparisons associated with VSC reversal.

Because of the above difficulties and the fact that there were more USs per session (50%) during Correlated than during Uncorrelated conditions, Design I was abandoned. A 1,000-Hz tone was also adopted in place of the 12-Hz clicks. The intensity of the US was also increased. Kamin (1968) has noted that overshadowing is a function of the relative intensities of two stimuli which determine their saliency. However, overshadowing, measured in terms of absolute responses to a stimulus, is attenuated by employing greater US magnitude (Rescorla & Wagner, 1972, Kamin, 1968). An additional modification consisted of the online usage of a CAT 1000 computer of average transients to enable more rapid and reliable determination of the intensity of electrical pulses delivered to the optic chiasma "just" capable of eliciting a response at the striate cortex.

DESIGN II. Each S was repeatedly exposed to several successive sessions of differential conditioning followed by compound conditioning. During the former either the auditory stimulus (A, a 1,000-Hz tone at 85 db) for Group A or the tactile stimulus (T, a 7.5-lb/inch^2 puff of

compressed nitrogen directed to the dorsal surface of the neck) for Group T was reinforced. Visual stimulation interspersed among differential conditioning trials constituted a Psuedo-conditioning Test. During compound conditioning the auditory and visual (AV) or tactile and visual (TV) compound stimuli were reinforced. Successive sessions of compound conditioning were always concluded with a stimulus control test. During such a test the visual stimulus was interspersed among the reinforced compound presentations. The method of visual stimulation was balanced between Ss of each Group.

Figure A-1 illustrates the response probabilities for the various stimuli for Group A (above) and Group T (below) across the respective conditioning and test conditions. The 7.5-lb/inch^2 tactile air puff would appear to have been too intense causing overshadowing of the visual stimulus and thereby causing a retardation of conditioning to V and a facilitation of conditioning to T. Note that Group T showed more rapid conditioning to T than did Group A to element A. When the visual stimulus was paired with the differentially reinforced element performance was asymptotic and the establishment of VSC was blocked. This is evident during Test AV for Group A and for the two successive TV Tests for Group T. On the other hand, acquisition was evident across the three sessions of compound TV and Test TV to the compound TV for Group A. However, the acquisition was for non-VSC by T and not VSC.

A reduction in the intensity of the air puff to 3-lb/inch^2 is indicated by the vertical bar running through the subsequent session of differential conditioning in Figure A-1. Note that for Group T this reduction was associated with a lowered asymptotic level of T. A similar

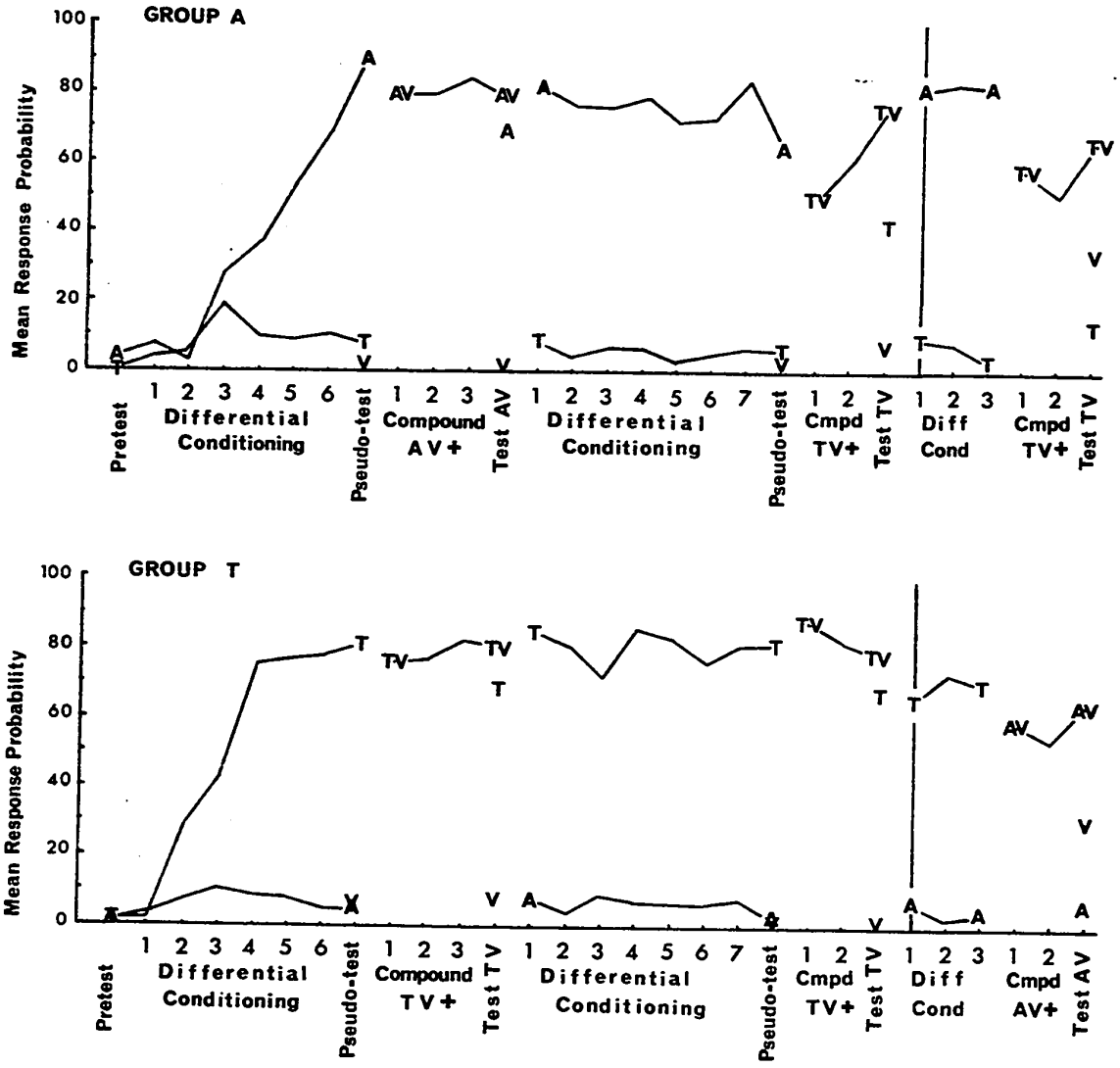

Fig. A-1. The response probabilities for the auditory (A), tactile (T), and visual (V) elements and their compounds (TV and AV) for Group A ($\underline{N}$ = 6 $\underline{Ss}$) and T ($\underline{N}$ = 6 $\underline{Ss}$). Element A was differentially reinforced for Group A and element T for Group T. The vertical bar denotes a change in the intensity of T from 7.5-lb/inch^2 to 3-lb/inch^2.

effect was also evident for Group A; the response probability of T dropped from the preceding Test TV level. During compound TV+ conditioning Group A showed a high response probability for TV and further evidence of acquisition. However, with the intensity of T now lowered, VSC was evident for the last experimental session, Test TV. Similarly, for Group T, when the visual stimulus was reinforced in compound with the differentially non-reinforced auditory element, VSC was evident (see Test AV). Thus, reducing the intensity of the tactile stimulus permitted the reversal of stimulus control from the non-visual tactile element to the visual element when the visual element was reinforced in compound with the differentially non-reinforced tactile element. That T was still salient is evident by the high level of stimulus control demonstrated by Group T during differential conditioning after T was reduced.

On the basis of the above findings we adopted the above Design II with but minor modifications for the stimulus parameters. As seen in the body of this dissertation, Design II permitted the successive manipulation of VSC and non-VSC within Ss.

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APPENDIX B

SUMMARY TABLES OF ANALYSES OF VARIANCE ON THE ARC SINE TRANSFORMED P RATIOS FOR STUDY 1. (BEHAVIORAL)

TABLE B-1

Groups X Visual Conditions X Preliminary Tests Analysis of Variance on the P Ratios for the Auditory, Tactile, and Visual Stimulus Elements

Source[1]	DF	MS	F
Groups (A)	1	0.0212	0.648
Visual Conditions (B)	1	0.0014	0.043
A x B	1	0.0047	0.143
Error	20	0.0328	
Preliminary Tests (J)	1	3.8055	331.265*
A x J	1	0.0253	2.201
B x J	1	0.0080	0.694
A x B x J	1	0.0067	0.586
Error	20	0.0115	
Stimulus Elements (K)	2	0.9277	70.112*
A x K	2	3.0802	232.784*
B x K	2	0.0007	0.559
A x B x K	2	0.0254	1.919
Error	40	0.0132	
J x K	2	0.8488	54.906*
A x J x K	2	3.0791	199.188*
B x J x K	2	0.0040	0.256
A x B x J x K	2	0.0001	0.008
Error	40	0.0155	
Total	143		

* Significant at the .01 level of probability

[1] Source levels: Groups: A and T; Visual Conditions: Peripheral and Central; Preliminary Tests: Pretest and Pseudo-conditioning Tests; Stimulus Elements: Auditory, Tactile, and Visual.

TABLE B-2

Groups X Visual Conditions X Compound Tests Analysis of Variance on the P Ratios for the Non-Visual and Visual Stimulus Elements

Source[1]	DF	MS	F
Groups (A)	1	0.0102	0.796
Visual Conditions (B)	1	0.4701	3.654
A x B	1	0.3900	3.024
Error	20	0.1286	
Compound Tests (J)	1	0.0085	0.368
A x J	1	0.0347	1.508
B x J	1	0.0212	0.921
A x B x J	1	0.0989	4.295
Error	20	0.0230	
Stimulus Elements (K)	1	0.4244	2.367
A x K	1	0.1307	0.729
B x K	1	0.0504	0.281
A x B x K	1	0.0551	0.307
Error	20	0.1793	
J x K	1	0.0798	0.943
A x J x K	1	6.7733	80.028*
B x J x K	1	0.0148	0.175
A x B x J x K	1	0.0101	0.199
Error	20	0.0846	
Total	95		

* Significant at the .01 level of probability

[1] Source levels: Groups : A and T; Visual Conditions: Peripheral and Central; Compound Tests: TV and AV; Stimulus Elements: Non-Visual and Visual.

TABLE B-3

Groups x Visual Conditions Analysis of Variance on the P Ratios for the Stimulus Elements Prior to and Following TV and AV Compound Conditioning

Source[1]	DF	MS	F
Groups (A)	1	0.0001	0.001
Visual Conditions (B)	1	0.2655	2.192
A x B	1	0.3327	2.748
Error	20	2.4218	
Stimulus Elements (J)	6	0.8324	13.214*
A x J	6	3.0951	49.131*
B x J	6	0.0501	0.795
A x B x J	6	0.0429	0.680
Error	120	0.0630	
Total	167		

* Significance at the .01 level of probability

[1] Source levels: Groups: A and T; Visual Conditions: Peripheral and Central; Stimulus Elements: pre-A (pre-AV conditioning), pre-T (pre-TV conditioning), pre-V (pre-TV conditioning), V (post-TV and pre-AV conditioning), post-A (post-AV conditioning), post-T (post-TV conditioning), and post-V (post-AV conditioning).

TABLE B-4

Groups X Visual Conditions X Compound Stimulus Conditions X Sessions Analysis of Variance on the P Ratios for the Compound Stimuli

Source[1]	DF	MS	F
Groups (A)	1	0.0873	0.116
Visual Conditions (B)	1	1.3152	1.750
A x B	1	0.3349	0.446
Error	20	0.7515	
Compound Stimulus Conditions (J)	1	1.8611	21.452*
A x J	1	3.234	37.277*
B x J	1	0.0134	0.155
Error	20	0.0868	
Sessions (K)	4	0.3118	14.195*
A x K	4	0.0136	0.617
B x K	4	0.0142	0.647
A x B x K	4	0.0167	0.762
Error	80	0.0220	
J x K	4	0.0246	1.565
A x J x K	4	0.2390	15.195*
B x J x K	4	0.0226	1.439
A x B x J x K	4	0.0063	0.403
Error	80	0.0157	
Total	239		

* Significance at the .01 level of probability

[1] Source levels: Groups: A and T; Visual Conditions: Peripheral and Central; Compound Stimulus Conditions: TV and AV; Sessions: 1-5 (includes test session).

APPENDIX C

SUMMARY TABLES OF ANALYSES OF VARIANCE ON THE ARC SINE TRANSFORMED P RATIOS FOR STUDY 2. (BEHAVIORAL)

TABLE C-1

Groups X Preliminary Tests Analysis of Variance on the P Ratios for Optic Chiasma Stimulation

Source[1]	DF	MS	F
Groups (A)	1	0.0003	0.161
Error	4	0.0018	
Preliminary Tests (J)	2	0.0828	98.206*
A x J	2	0.0008	0.963
Error	8	0.0008	
Total	17		

* Significance at the .01 level of probability

[1] Source levels: Groups: A and T; Tests: Pretest, Arousal Test I, and Arousal Test II.

TABLE C-2

Groups X Compound Tests Analyses of Variance on the P Ratios for the Non-Visual and Visual Stimulus Elements

Source[1]	DF	MS	F
Groups (A)	1	0.0580	0.405
Error	4	0.1434	
Tests (J)	1	0.1022	1.285
A x J	1	0.0011	0.014
Error	4	0.0796	
Stimulus Elements (K)	1	0.3772	2.696
A x K	1	0.2194	1.568
Error	4	0.1399	
J x K	1	0.0065	0.064
A x J x K	1	3.9638	38.415*
Error	4	0.1032	
Total	23		

* Significance at the .01 level of probability

[1] Source levels: Groups: A and T; Tests: TV and AV; Stimulus Elements: Non-Visual and Visual.

TABLE C-3

Groups X Stimulus Elements Analysis of Variance on the P Ratios for the Non-Visual and Visual Stimulus Elements Prior to and Following TV and AV Compound Conditioning

Source[1]	DF	MS	F
Groups (A)	1	0.0588	0.857
Error	4	0.0686	
Stimulus Elements (J)	6	0.3065	4.491**
A x J	6	1.0518	15.410*
Error	24	0.0683	
Total	41		

* Significance at the .01 level of probability
** Significance at the .05 level of probability

[1] Source levels: Groups A and T; Stimulus Elements: pre-A (pre-AV conditioning); pre-T (pre-TV conditioning); V (post-TV and pre-AV conditioning); post-A (post-AV conditioning); post-T (post-TV conditioning); and post-V (post-AV conditioning).

TABLE C-4

Groups X Compound Stimulus Conditions X Sessions Analysis of Variance on the P Ratios for the Compound Stimuli

Source[1]	DF	MS	F
Groups (A)	1	0.2098	1.288
Error	4	0.1629	
Compound Stimulus Conditions (J)	1	0.1057	1.736
A x J	1	0.0260	0.427
Error	4	0.0609	
Sessions (K)	4	0.0008	0.106
A x K	4	0.0020	0.268
Error	16	0.0074	
J x K	4	0.0046	1.073
A x J x K	4	0.0055	1.279
Error	16	0.0043	
Total	59		

[1] Source levels: Groups: A and T, Compound Stimuli: TV and AV, Sessions: 1-5 (includes Test session).

APPENDIX D

OBSERVATIONS ON THE RELATIONSHIP BETWEEN AROUSAL AND THE PHOTIC RESPONSE TO STIMULUS ONSET AND OFFSET

Arousal is maintained to have a general attenuating influence on the amplitude of sensory evoked potentials to photic stimulation (Bremer & Stoupel, 1958, 1959a, 1959b; Dumont & Dell, 1958, 1960; Evarts, Fleming, & Huttenlocher, 1960; Walley & Urschel, 1972). Accompanying arousal are well documented changes in the peripheral receptor apparatus. The attenuation of the photic potential accompanying arousal, however, appears inconsistent with the constriction of the pupil generally reported to accompany arousal (Lynn, 1966; Voronin, Leontiev, Luria, Sokolov, & Vinogradova, 1965). Fernández-Guardiola, Harmony, and Roldán (1964) and Affanni, Mancia, & Marchiafava (1962) have demonstrated that changes in pupil diameter are associated with alterations in the photic evoked potential. Maintenance of pupil dilation by application of atropine (Fernández-Guardila, Roldán, Fanjul, & Castells, 1961) or placement of a lens over the pupil (Palestini, Gallardo, & Armengol, 1964) prevents visual EP habituation. Barlow (1952) notes changes in the photic potential to changes in saccadic eyemovements which increase during attention and decrease when an object is moved from the field of vision. The significance of eye movements accompanying distraction are logically related to changes in the visual evoked potential due to altered visual input.

Our observations on the rabbit's evoked response to flashes during arousal induced by background auditory stimulation coincide with the preceding reports. We have noted an attenuation of the photic potential

to both light-onset and light-offset under conditions of light and dark adaptation. Transcortical averaged evoked potentials (AEPs) at the striate cortex were accumulated from Ss under light pentobarbital anesthesia and mounted in a stereotaxic to eliminate head movements. Potentials were averaged under four conditions: non-arousal and arousal during either light or dark adaptation. The potentials were averaged under each condition to a light flashing at 1 cps. Assessment of EEG activity at the visual cortex indicated that auditory stimulation from in back and above S at 1,000 Hz. and of continually varying intensity (40-100 db.) was sufficent to initiate and sustain EEG desynchrony during averaging for the arousal conditions.

Fig. D-1 illustrates the AEPs generated from a single S under the four conditions A-D. The following relationships were observed in all Ss investigated. Potentials generated under conditions of arousal were attenuated with respect to the control conditions A and C. Primary positive deflections and secondary slow wave negativity were both reduced to stimulus onset and stimulus offset during arousal. A reliable decrease in the latency to peak secondary activity and disappearance of the late primary positive components also accompanied arousal. The primary activity to stimulus-offset generally exceeded the response to stimulus-onset; this relationship was reversed for secondary activity. Neither the initial primary positive deflection nor secondary negative activity was reliably altered as a function of dark adaptation.

Either a change in pupil dilation or orientation of the receptor apparatus away from the light source could account for the attenuation in the photic AEP during arousal. Pupil changes seem unlikely however; the pupil dilates during arousal. Nor were any eye movements visibly

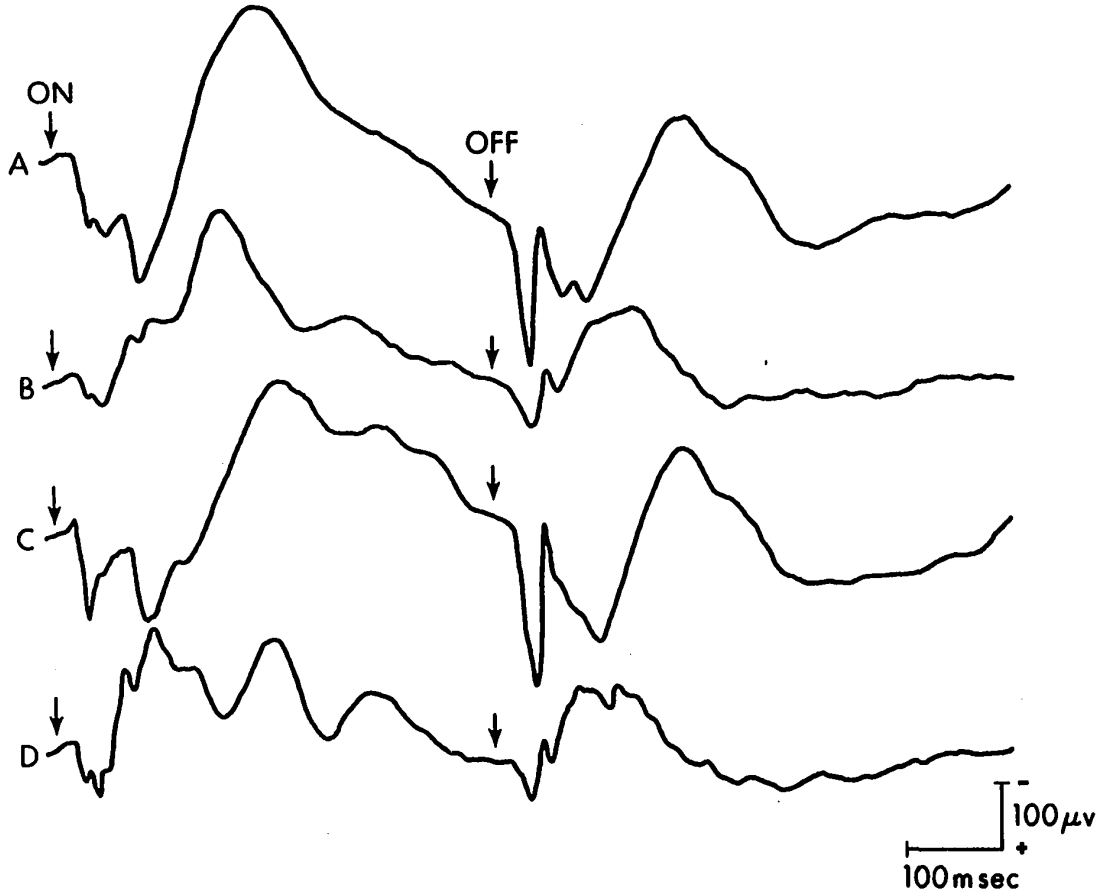

Fig. D-1. Averaged evoked potentials to light-onset and light-offset in a lightly anesthetized albino rabbit as recorded transcortically at the striate cortex. S was light adapted during A and B and dark adapted during C and D. Arousal, EEG desynchrony, during conditions B and D, was effected by variable auditory stimulation. Each potential represents the averaged response to 25 presentations of a light flashing at 1 cps.

evident. That the attenuation in primary activity was replicable under both light and dark adaptation conditions when the pupil was maximally dilated, is evidence against such a peripheral interpretation. Steriade (1968) has reported that the initial primary activity of the photic potential is of retinal origin. The attenuation of the photic AEP accompanying arousal may be the product of enhanced retinal inhibition. That the depression of the photic AEP was similar between light and dark adaptation further suggests that activity associated with adaptation is independent of the mechanism mediating the attenuation of the photic AEP during arousal.

The independence of primary and secondary activity within a test condition further suggests that the amplitude of secondary activity is to some extent independent of the amplitude of primary activity. Primary activity to light-offset exceeded the response to light-onset while secondary activity showed an inverse relationship. This is consistent with the contention that primary activity is a specific response reflecting in part activity of light-on center and/or light-off center units in the specific sensory system and that secondary activity is reflecting non-specific activity associated with the organism's tonic state of arousal or phasic level of arousal initiated by stimulation. Taking this position it would appear that arousal tonically depresses photic activity and that phasic arousal to light-onset exceeds the response to light-offset. The retinal response to light-onset appears to be exceeded by the response to light-offset; this is however probably a relative effect being dependent upon the background illumination as the difference was reduced somewhat when S was dark adapted.

The assessment of visual AEP changes to light-onset and light-offset could provide a useful paradigm for assessing intramodal selective processes, i.e., intramodal selective attention. A US contingeny could be established to light-onset and/or light-offset simultaneously. Sommer-Smith (1967) and Sommer-Smith, Galeano, Piñeyũra, Roig, and Segundo (1962) noted that tone cessation can be employed as a conditioned stimulus though it is less effective than tone onset, the evoked potential was also reported as somewhat lower.

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APPENDIX E

SUMMARY TABLES OF ANALYSES OF VARIANCE ON CHANGES IN LOGARITHMIC TRANSFORMED VISUAL EPEs (LATENCY CRITERION) STUDY 1. (ELECTROPHYSIOLOGICAL)

TABLE E-1

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in Primary EPE Activity

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.0171	0.439
Groups (B)	1	0.0883	2.273
A x B	1	0.0000	0.000
Error	8	0.0389	
Tests (J)	2	0.0176	3.334
A x J	2	0.0034	0.652
B x J	2	0.0170	3.215
A x B x J	2	0.0004	0.078
Error	16	0.0053	
CS (K)	2	0.0117	0.517
A x K	2	0.0023	0.104
B x K	2	0.0061	0.272
A x B x K	2	0.0138	0.612
Error	16	0.0226	
J x K	4	0.0017	0.798
A x J x K	4	0.0039	1.795
B x J x K	4	0.0019	0.869
A x B x J x K	4	0.0036	1.679
Error	32	0.0021	
Total	107		

[1] Source levels: Visual Conditions (Peripheral and Central), Groups A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

TABLE E-2

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in Secondary EPE Activity

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.0432	1.886
Groups (B)	1	0.0211	0.922
A x B	1	0.0158	0.689
Error	8	0.0229	
Tests (J)	2	0.0003	0.099
A x J	2	0.0120	3.567**
B x J	2	0.0025	0.742
A x B x J	2	0.0037	1.090
Error	16	0.0034	
CS (K)	2	0.0303	1.820
A x K	2	0.0065	0.393
B x K	2	0.0138	0.832
A x B x K	2	0.0170	1.022
Error	16	0.0166	
J x K	4	0.0048	1.598
A x J x K	4	0.0016	0.534
B x J x K	4	0.0020	0.678
A x B x J x K	4	0.0006	0.190
Error	32	0.0030	
Total	107		

**Significance at the .05 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

APPENDIX F

THE LATENCY AND POLARITY CHARACTERISTICS OF PRIMARY ACTIVITY ELICITED TO PHOTIC AND OPTIC CHIASMA STIMULATION

Visual evoked responses (VERs) to photic and chiasmic stimulation were averaged across the striate cortex in albino rabbit. High resolution VER averages permitted an assessment of the latency and polarity of the components as a function of the source of stimulation. Figure F-1 represents averaged VERs (48 sweeps each) recorded at low resolution across 1,000 msec. at 1 msec/data point and, simultaneously, at high resolution across 50 msec. at 200 μsec/data point.

By accumulating the VERs to photic and optic chiasma stimulation at high resolution several points became evident which were not readily apparent otherwise. First, a close correspondence is evident between the latency of those early components for the two potentials when retinal transmission time (approximately 15 msec.) is considered. The polarity and latency of the initial "major" components correspond for P_1, N_1, and N_2 and the latency but not the polarity of a major deflection at 26-27 msec. Of even greater import, note the small but distinct positive deflections apparent on the positive going slope of P_1 generated to the chiasmic stimulus. The latency of these components corresponds well with the latency of the initial components of cat VERs to optic nerve stimulation as reported by Bishop and Clare (1952), Malis and Kruger (1956), and Chang and Kaada (1950). Bishop has identified the first spike as the axon potentials of the axons of geniculate cells and the subsequent ones as post-synaptic responses of neurones within the cortex activated serially.

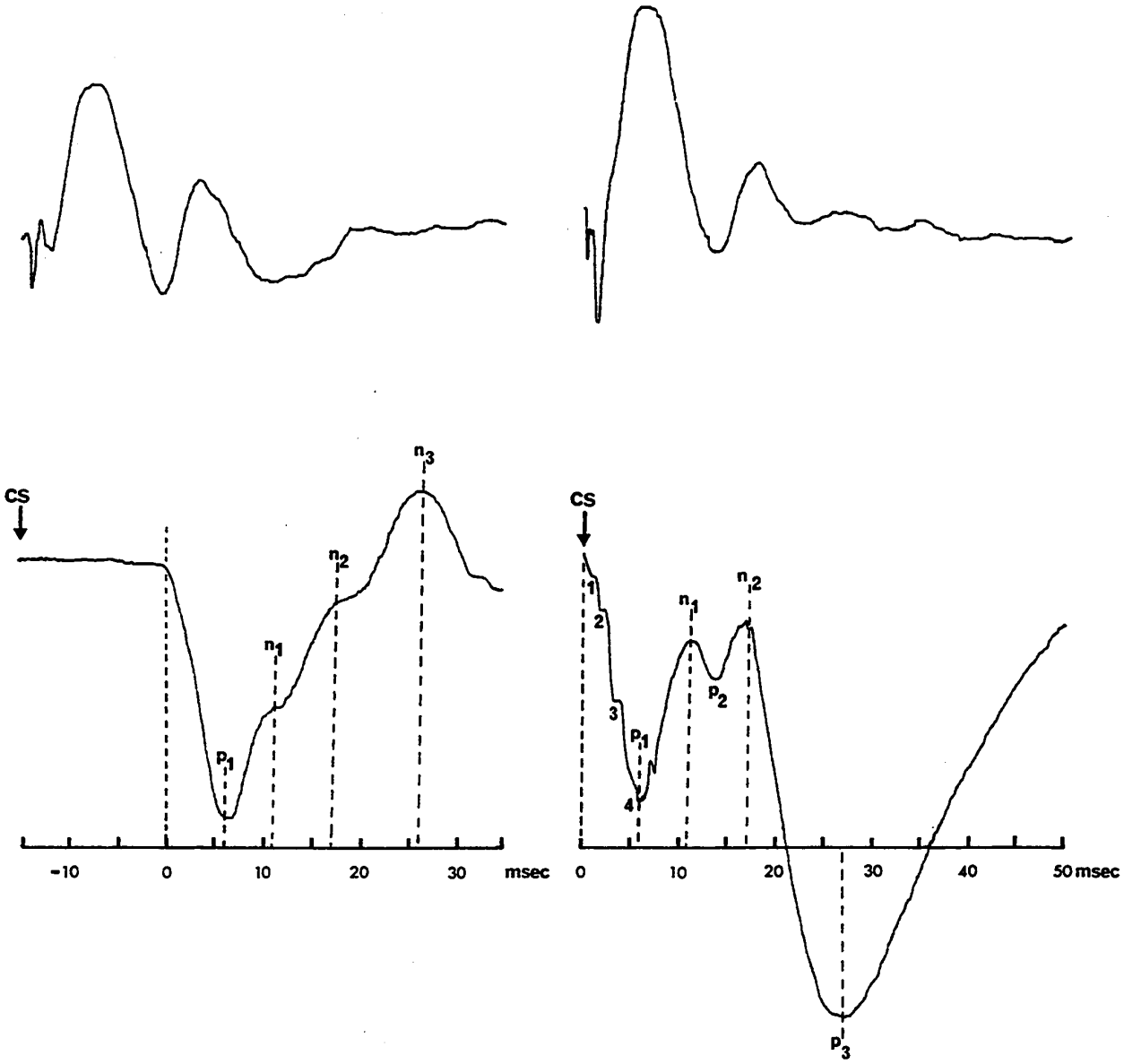

Fig. F-1. The transcortical averaged VERs of unanesthetized rabbit to photic and optic chiasma stimulation. Above. Low resolution VERs averaged (48 sweeps) across 1,000 msec. at 1 msec. per data point. Below. High resolution (200 μsec/data point) display of the initial 50 msec. of the above VERs. Left. Simultaneously averaged VERs to photic flashes. The spikes have been labelled according to their polarity and serial sequence. The time base is referenced in msec. from the initial indication of spike activity after CS onset. Right. Simultaneously averaged VERs to optic chiasma stimulation. The components 1,2,3, and 4 refer to corresponding components seen in cat similarly designated by Malis and Kruger (1956). See text for a discussion of the correspondence of specific components of the VERs to each type of stimulation.

It seems reasonable to compare the initial "major" components of rabbits' VERs elicited to photic and chiasmic stimulation; it is also apparent that early primary components of the rabbit and cat to optic stimulation are of common origin. Most critical, however, may be the locus of the cortical depth recording electrode. The depth electrode for bipolar transcortical recording resided within the white matter at the depths of the striate cortex for those Ss showing the regularties discussed. As in cat (e.g., Bishop and Clare, 1953) the rabbit primary component, P_1, of the VER to either photic or chiasmic stimulation appears to reflect a composite of presynaptic cortical activity corresponding to a "radiation spike" of LGB origin and activity in post-synaptic neurones in the cortex activated by serially conducted action potentials.

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APPENDIX G

SUMMARY TABLES OF ANALYSES OF VARIANCE ON CHANGES IN LOGARITHMIC TRANSFORMED VISUAL AEP ACTIVITY OF Ss WITH CORTICAL ELECTRODE PLACEMENT IN WHITE MATTER STUDY 1. (ELECTROPHYSIOLOGICAL)

TABLE G-1

Visual Conditions X Groups X Tests X CS Analysis of Variance in Changes in P_1 Amplitude

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.0031	0.035
Groups (B)	1	0.0765	0.867
A x B	1	0.1124	1.273
Error	8	0.0882	
Tests (J)	2	0.1571	11.887*
A x J	2	0.0094	0.709
B x J	2	0.0140	1.057
A x B x J	2	0.0174	1.315
Error	16	0.0132	
CS (K)	2	0.0019	0.171
A x K	2	0.0236	2.082
B x K	2	0.0104	0.919
A x B x K	2	0.0103	0.905
Error	16	0.0113	
J x K	4	0.0034	1.106
A x J x K	4	0.0034	1.130
B x J x K	4	0.0019	0.633
A x B x J x K	4	0.0025	0.822
Error	32	0.0030	
Total	107		

*Significant at the .01 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

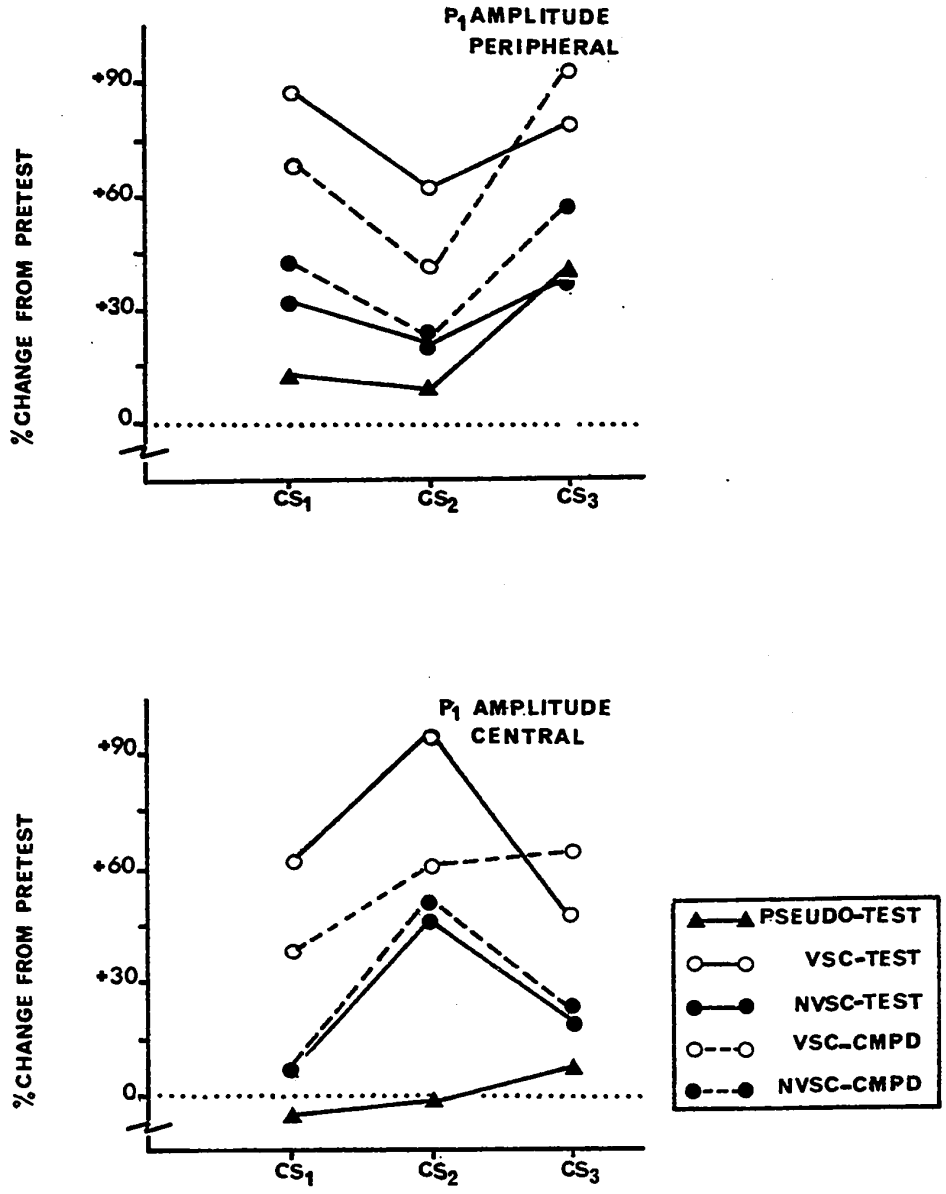

Fig. G-1. The mean amplitude change of P_1 in response to each CS of the 3-Hz. photic (above) and chiasmic (below) stimulus train during each test condition. The activity representing the compound (CMPD) conditioning sessions reflects the average change of all Ss (N= 12) across the four sessions for each condition. Study 1. Electro-physiological

TABLE G-2

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in Primary EPE (Polarity Criterion) Activity

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.1216	1.664
Groups (B)	1	0.0890	1.219
A x B	1	0.1194	1.635
Error	8	0.0731	
Tests (J)	2	0.1048	8.602*
A x J	2	0.0284	2.327
B x J	2	0.0252	2.070
A x B x J	2	0.0046	0.374
Error	16	0.0122	
CS (K)	2	0.0203	0.889
A x K	2	0.0056	0.245
B x K	2	0.0036	0.158
A x B x K	2	0.0396	1.739
Error	16	0.0228	
J x K	4	0.0041	1.143
A x J x K	4	0.0015	0.426
B x J x K	4	0.0023	0.625
A x B x J x K	4	0.0032	0.883
Error	32	0.0036	
Total	107		

*Significant at the .01 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

TABLE G-3

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in S Amplitude

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.1170	2.437
Groups (B)	1	0.0468	0.974
A x B	1	0.0183	0.381
Error	8	0.0480	
Tests (J)	2	0.0041	0.874
A x J	2	0.0253	5.451**
B x J	2	0.0099	2.137
A x B x J	2	0.0078	1.678
Error	16	0.0046	
CS (K)	2	0.0505	1.230
A x K	2	0.0109	0.266
B x K	2	0.0300	0.730
A x B x K	2	0.0346	0.842
Error	16	0.0411	
J x K	4	0.0119	1.688
A x J x K	4	0.0021	0.294
B x J x K	4	0.0056	0.788
A x B x J x K	4	0.0020	0.280
Error	32	0.0071	
Total	107		

**Significant at the .05 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

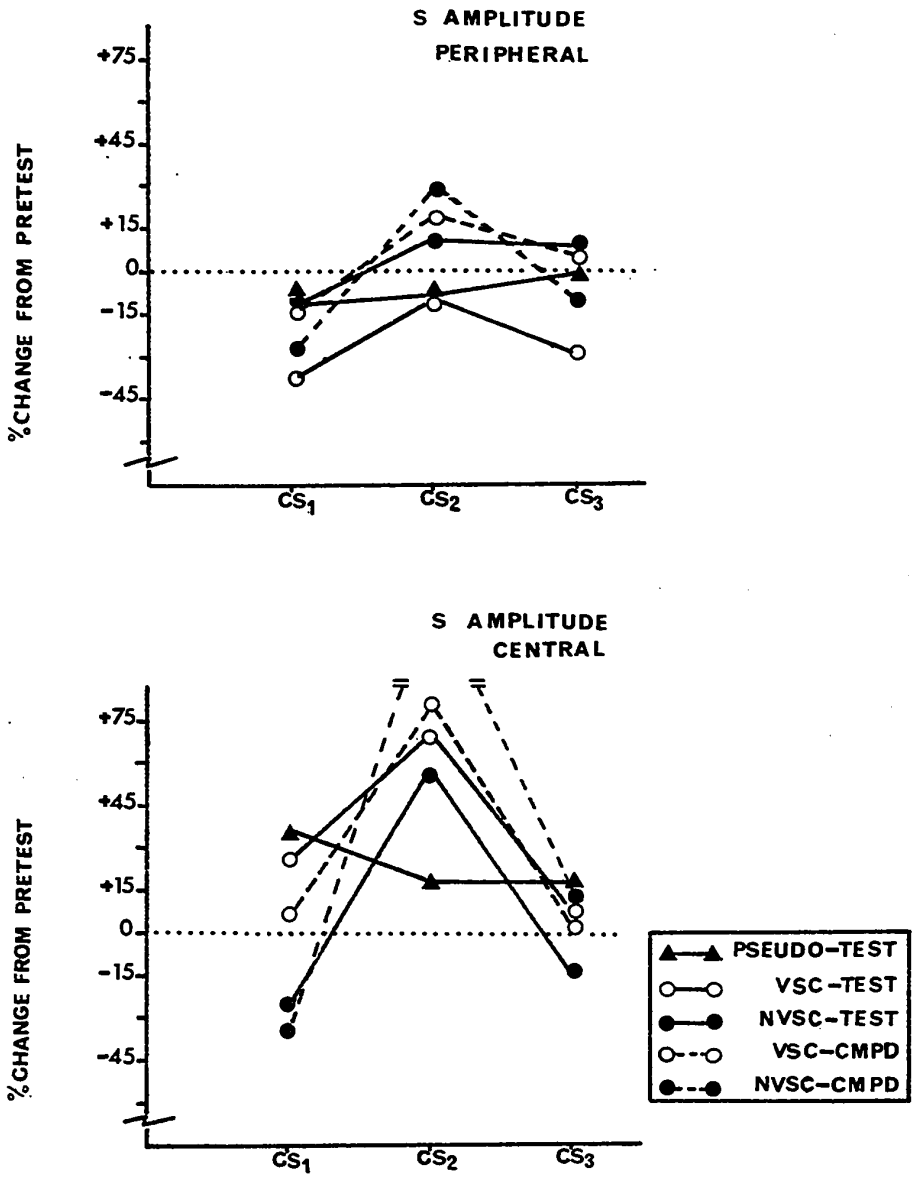

Fig. G-2. The mean amplitude change of S in response to each CS of the 3-Hz. photic (above) and optic chiasmic (below) stimulus train during each test condition. The activity representing the compound (CMPD) conditioning sessions reflects the average change for all Ss (N= 12) across the four sessions for each condition. Study 1. Electrophysiological.

TABLE G-4

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in Secondary EPE (Polarity Criterion) Activity

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.0928	3.372
Groups (B)	1	0.0717	2.607
A x B	1	0.0220	0.801
Error	8	0.0275	
Tests (J)	2	0.0028	0.851
A x J	2	0.0204	6.264*
B x J	2	0.0040	1.217
A x B x J	2	0.0055	1.676
Error	16	0.0033	
CS (K)	2	0.0308	1.101
A x K	2	0.0029	0.103
B x K	2	0.0240	0.867
A x B x K	2	0.0184	0.663
Error	16	0.0277	
J x K	4	0.0054	1.574
A x J x K	4	0.0013	0.377
B x J x K	4	0.0017	0.496
A x B x J x K	4	0.0004	0.105
Error	32	0.0034	
Total	107		

*Significant at the .01 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

TABLE G-5

Visual Conditions X Groups X Compound Conditioning Conditions X CS Analysis of Variance on Changes in P_1 Amplitude

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.0154	0.045
Groups (B)	1	0.0001	0.000
A x B	1	0.6041	1.778
Error	8	0.3397	
Compound Conditioning Condition (J)	1	0.4904	27.268*
A x J	1	0.0080	0.443
B x J	1	0.1351	7.512**
A x B x J	1	0.0295	1.643
Error	8	0.0180	
Sessions (K)	3	0.0047	0.822
A x K	3	0.0095	1.653
B x K	3	0.0109	1.893
A x B x K	3	0.0038	0.657
Error	24	0.0058	
J x K	3	0.0014	1.278
A x J x K	3	0.0049	0.545
B x J x K	3	0.0028	0.310
A x B x J x K	3	0.0039	0.440
Error	24	0.0089	
CS (L)	2	0.0596	1.252
A x L	2	0.1338	2.808
B x L	2	0.0170	0.357
A x B x L	2	0.0046	0.097
Error	16	0.0476	
J x L	2	0.0031	1.409
A x J x L	2	0.0039	1.742
B x J x L	2	0.0019	0.869
A x B x J x L	2	0.0005	0.246
Error	16	0.0022	
K x L	6	0.0033	1.748
A x K x L	6	0.0034	1.805
B x K x L	6	0.0004	0.189
A x B x K x L	6	0.0016	0.859
Error	48	0.0019	
J x K x L	6	0.0012	0.460
A x J x K x L	6	0.0040	1.576
B x J x K x L	6	0.0006	0.233
A x B x J x K x L	6	0.0002	0.076
Error	48	0.0025	
Total	287		

*Significance at the .01 level of probability.

**Significance at the .05 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Compound Conditioning Conditions (VSC and NVSC), Sessions (1-4), and CS (1-3).

TABLE G-6

Visual Conditions X Groups X Compound Conditioning Conditions X CS Analysis of Variance on Changes in S Amplitude

Source[1]	DF	MS	F
Visual Conditions (A)	1	0.1423	0.882
Groups (B)	1	0.2055	1.274
A x B	1	0.0270	0.167
Error	8	0.1613	
Compound Conditioning Condition (J)	1	0.0147	1.511
A x J	1	0.0029	0.297
B x J	1	0.0007	0.073
A x B x J	1	0.0515	5.280
Error	8	0.0097	
Sessions (K)	3	0.0017	0.257
A x K	3	0.0003	0.047
B x K	3	0.0046	0.703
A x B x K	3	0.0030	0.458
Error	24	0.0066	
J x K	3	0.0030	0.497
A x J x K	3	0.0023	0.373
B x J x K	3	0.0061	1.012
A x B x J x K	3	0.0051	0.833
Error	24	0.0061	
CS (L)	2	0.7151	5.146**
A x L	2	0.0702	0.505
B x L	2	0.0269	0.194
A x B x L	2	0.0432	0.311
Error	16	0.1390	
J x L	2	0.0856	9.261*
A x J x L	2	0.0162	1.750
B x J x L	2	0.0156	1.684
A x B x J x L	2	0.0143	1.549
Error			

Continued . . .

*Significance at the .01 level of probability.

**Significance at the .05 level of probability.

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Compound Conditioning Conditions (VSC and NVSC), Sessions (1-4), and CS (1-3).

TABLE G-6 (Continued)

Source	DF	MS	F
K x L	6	0.0078	1.450
A x K x L	6	0.0030	0.548
B x K x L	6	0.0057	1.061
A x B x K x L	6	0.0044	0.807
Error	48	0.0054	
J x K x L	6	0.0028	0.633
A x J x K x L	6	0.0093	2.095
B x J x K x L	6	0.0008	0.175
A x B x J x K x L	6	0.0074	1.667
Error	48	0.0044	
Total	287		

APPENDIX H

SUMMARY TABLES OF ANALYSES OF VARIANCE ON CHANGES IN THE DURATION OF PRIMARY AND SECONDARY (POLARITY CRITERION) VISUAL AEP ACTIVITY STUDY 1. (ELECTROPHYSIOLOGICAL)

TABLE H-1

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in the Duration of Primary EPE (Polarity Criterion) Activity

Source[1]	DF	MS	F
Visual Conditions (A)	1	1.8206	1.062
Groups (B)	1	0.0311	0.018
A x B	1	6.0668	3.540
Error	8	1.7139	
Tests (J)	2	0.9578	1.846
A x J	2	0.7063	1.362
B x J	2	0.5761	1.111
A x B x J	2	0.9576	1.846
Error	16	0.5188	
CS (K)	2	2.0611	2.786
A x K	2	0.7781	1.052
B x K	2	0.9096	1.229
A x B x K	2	1.3140	1.776
Error	16	0.7399	
J x K	4	0.3396	1.093
A x J x K	4	0.1042	0.335
B x J x K	4	0.2157	0.694
A x B x J x K	4	0.3486	1.122
Error	32	0.3107	
Total	107		

[1] Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

TABLE H-2

Visual Conditions X Groups X Tests X CS Analysis of Variance on Changes in the Duration of Secondary EPE (Polarity Criterion) Activity

Source[1]	DF	MS	F
Visual Conditions (A)	1	1.1406	0.707
Groups (B)	1	1.6501	1.023
A x B	1	1.7279	1.072
Error	8	1.6123	
Tests (J)	2	0.1598	1.831
A x J	2	0.2354	2.696
B x J	2	0.0980	1.122
A x B x J	2	0.0778	0.891
Error	16	0.0873	
CS (K)	2	0.5857	0.621
A x K	2	0.9980	1.057
B x K	2	0.5219	0.553
A x B x K	2	1.6342	1.731
Error	16	0.9439	
Total	107		

[1]Source levels: Visual Conditions (Peripheral and Central), Groups (A and T), Tests (Pseudo-conditioning, VSC, and NVSC), CS (1-3).

APPENDIX I

SUMMARY TABLES OF ANALYSES OF VARIANCE ON THE SQUARE ROOT TRANSFORMED AMPLITUDES OF VISUAL AEP COMPONENTS STUDY 2. (ELECTROPHYSIOLOGICAL)

TABLE I-1

Groups X Tests X Intervals Analysis of Variance on the P_1 Amplitudes

Source[1]	DF	MS	F
Groups (A)	1	170.7496	1.5107
Error	4	113.0256	
Tests (B)	4	41.3941	3.2476**
A x B	4	18.5469	1.4551
Error	16	12.7460	
Intervals (C)	7	121.9027	39.7321*
A x C	7	5.7909	1.8874
Error	28	3.0681	
A x B	28	2.7224	4.9657*
A x B x C	28	0.5871	0.8767
Error	112	0.6696	
Total	239		

*Significance at the .01 level of probability
**Significance at the .05 level of probability

[1] Source levels: Groups (A and T), Tests (Pretest, Arousal Test I, Arousal Test II, VSC and NVSC Tests), Intervals (0, 30, 60, 90, 120, 150, 180, and 210 msec. Interstimulus Test Intervals).

TABLE I-4

Groups X Tests X Intervals Analysis of Variance on the S Amplitudes

Source[1]	DF	MS	F
Groups (A)	1	205.5608	5.9928
Error	4	34.3015	
Tests (B)	4	43.6314	3.6217**
A x B	4	22.0296	1.8286
Error	16	12.0472	
Intervals (C)	7	235.9737	34.7322*
A x C	7	11.9327	1.7631
Error	28	6.7682	
A x B	28	22.9944	5.9597*
A x B x C	28	3.6251	0.8321
Error	112	4.3567	
Total	239		

*Significance at the .01 level of probability
**Significance at the .05 level of probability

[1] Source levels: Groups (A and T), Tests (Pretest, Arousal Test I, Arousal Test II, Visual Stimulus Control, Non-Visual Stimulus Control), Intervals (0, 30, 60, 90, 120, 150, 180, and 210 msec. Interstimulus Intervals).

TABLE I-3

Groups X Tests X Intervals Analysis of Variance on the P_3 Amplitudes

Source[1]	DF	MS	F
Groups (A)	1	0.0555	0.0327
Error	4	1.6989	
Tests (B)	4	10.4102	6.9150*
A x B	4	2.0447	1.3582
Error	16	1.5055	
Intervals (C)	7	47.8233	41.8745*
A x C	7	0.3331	0.2916
Error	28	1.1421	
A x B	28	5.7634	10.8806*
A x B x C	28	0.2678	0.5055
Error	112	0.5297	
Total	239		

*Significance at the .01 level of probability

[1] Source levels: Groups (A and T), Tests (Pretest, Arousal Test I, Arousal Test II, VSC, and NVSC Tests), Intervals (0. 30, 60, 90, 120, 150, 180, and 210 msec. Interstimulus Intervals).

TABLE I-2

Groups X Tests X Intervals Analysis of Variance on the P_2 Amplitudes

Source[1]	DF	MS	F
Groups (A)	1	1.5746	0.0340
Error	4	46.2665	
Tests (B)	4	6.3006	2.1906
A x B	4	2.9256	1.0172
Error	16	2.8762	
Intervals (C)	7	12.5224	4.4972**
A x C	7	1.2606	0.4527
Error	28	2.7845	
A x B	28	0.8875	1.6131**
A x B x C	28	0.4325	0.7861
Error	112	0.5502	
Total	239		

**Significance at the .05 level of probability

[1] Source levels: Groups (A and T), Tests (Pretest, Arousal Test I, Arousal Test II, VSC, and NVSC Tests), Intervals (0, 30, 60, 90, 120, 150, 180, and 210 msec. Interstimulus Intervals).

APPENDIX J

GENICULO-STRIATE ACTIVITY DURING COMPOUND CONDITIONING AND IN RESPONSE TO NON-VISUAL STIMULATION ALONE STUDY 2. ELECTROPHYSIOLOGICAL

Inspection of the AEP records during compound conditioning in Study 2 when chiasmic stimulation was paired with non-visual tactile of auditory stimulation revealed several consistencies. Figures J-1 and J-2 illustrate the cortical AEPs across 512 msec. for two Ss to CS_1 for each compound conditioning session. Figure J-1 represents an S (F-56) from Group A and Figure J-2 illustrates an S (F-56) from Group T. If the visual stimulus was relevant as assessed during the fifth compound conditioning session then the facilitation of primary activity seen in response to CS_1 was apparent within the first compound conditioning session. This was also noted in Study 1. By contract if the non-visual stimulus was relevant there was an evident reduction in primary activity in relation to conditions when the visual stimulus was relevant. Particularly striking was the observation that P_1, a component presynaptic to the cortex at least in origin, and P_3, a post-synaptic component, were incremented at the initiation of compound conditioning (re: the Pretest) when the visual stimulus was paired with a differentially non-reinforced element.

Also evident whenever the non-visual and visual stimuli were presented together was a late positive wave noted as "x" in Figures J-1 and J-2 occurring subsequent to the peak of secondary negativity when the visual stimulus was presented alone. Brazier, Killam, and Hance (1961, p. 713) have noted a similar wave associated with the pairing of a photic

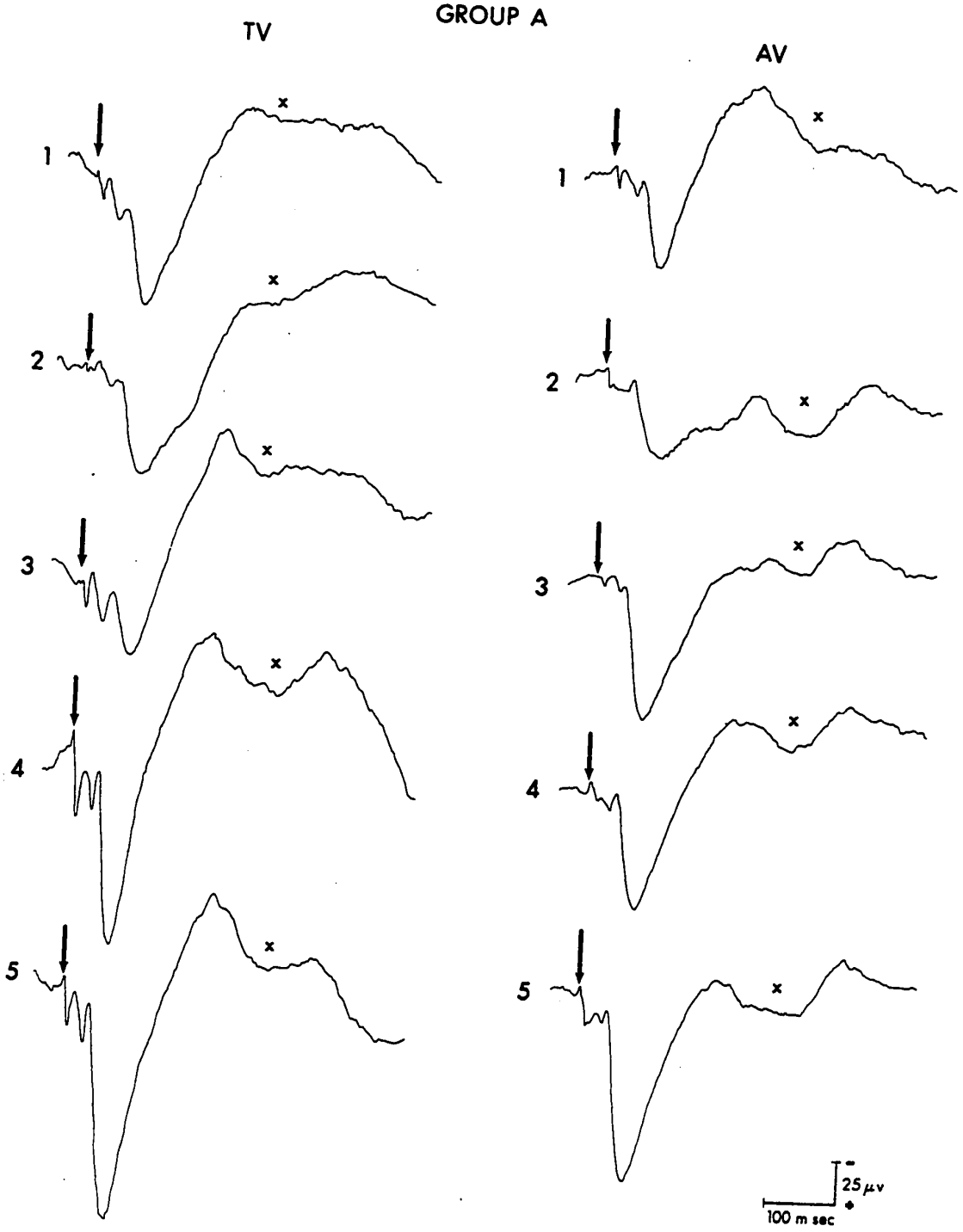

Fig. J-1. Averaged evoked responses to chiasmic stimulation paired with a non-visual stimulus. The potentials were recorded from the striate cortex across 5 successive sessions of TV and AV compound conditioning. The S (F-56) demonstrated visual stimulus control following TV compound conditioning. The "x" indicates a late slow wave positive potential which was often found on visual potentials when a non-visual stimulus was presented. Study 2. Electrophysiological.

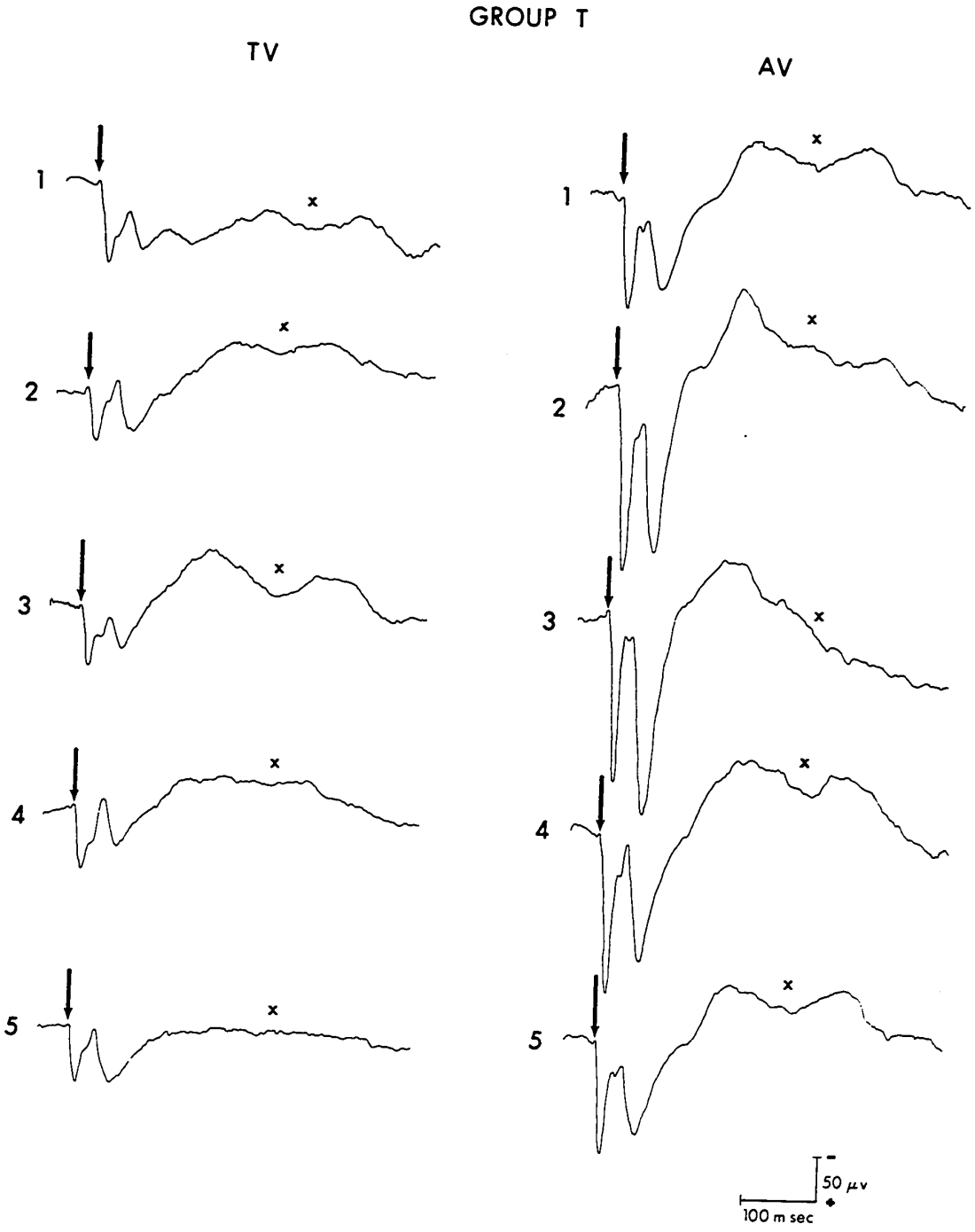

Fig. J-2. Averaged responses recorded under the same conditions reported for Fig. J-1. However, this S (F-53) from Group T demonstrated visual stimulus control following AV compound conditioning. Study 2. Electrophysiological

stimulus and an auditory click in the cat. They note that the potential was recordable monopolarly from the depth and surface of the visual cortex and had a latency similar to the potential reported here to both tactile-visual and audio-visual compound stimuli. While we also noted the potential at the surface and the depth of the striate cortex, the origin and significance of this potential remain to be determined.

Another potential related to non-visual stimulation recordable from the striate cortex was noted during differential conditioning employing non-visual stimuli. Figure k-3 represents the potential as it was recorded simultaneously across 1,024 msec. at the optic chiasma and striate cortex to 45 presentations of the reinforced (+) and non-reinforced (o) auditory and tactile stimuli. These potentials were recorded after five differential conditioning sessions. Features of this response were as follows. The potential, a slow positive wave initiated 50-70 msec. and peaking 100 to 150 msec. after onset of non-visual stimulation, was recordable from either the depth or surface cortical electrode and the optic chiasma. A late negative component was often seem to follow the initial positive potential with a latency of 200 to 300 msec. post-CS onset. This potential was evident as well at the level of the optic chiasma. The potential was recordable in the dark to either auditory or tactile stimulation and was often as large as 200 μv. at the cortex and 20 μv. at the chiasma. There was little change in the potential within Ss across the differential conditioning sessions if the stimulus was non-reinforced. However, if the stimulus was differentially reinforced the potential increased becoming more regular with a longer duration but no apparent change in latency.

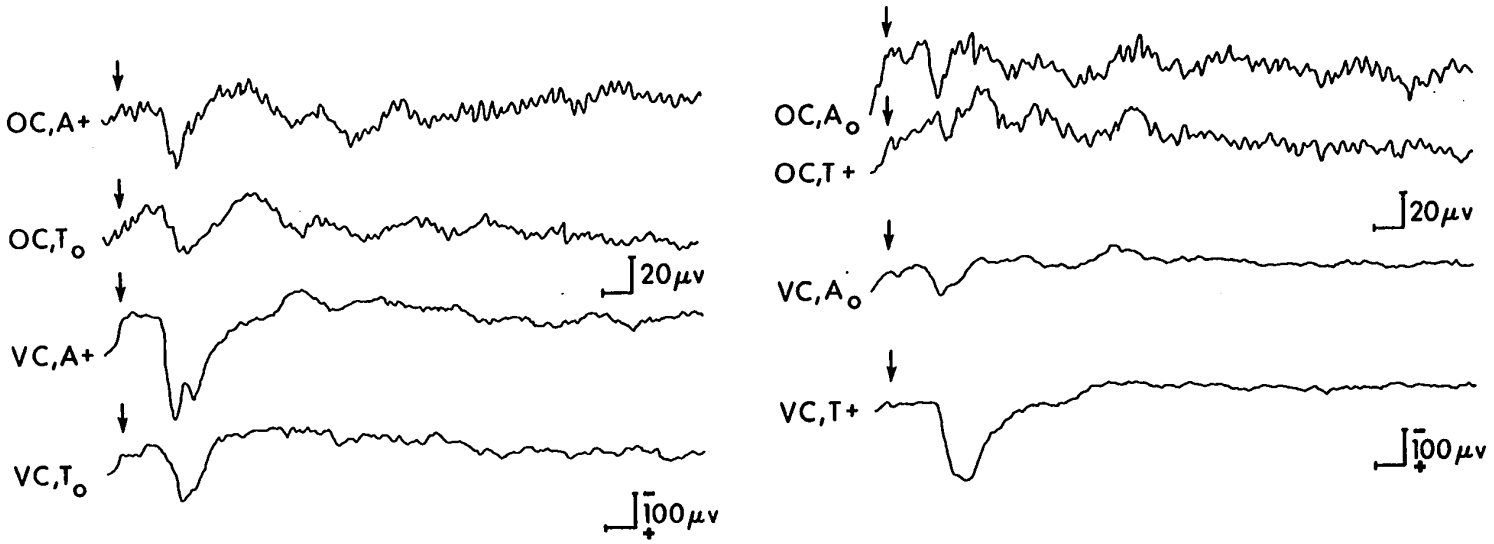

Fig. J-3. Averaged evoked responses recorded from the optic chiasma and striate cortex to 45 presentations of auditory or tactile stimulation alone. The S on the left (F-28) was differentially reinforced to presentations of auditory stimulation and non-reinforced to presentations of the tactile stimulus. Conditions were reversed for the S illustrated on the right (F-22). These potentials were averaged simultaneously from both sites during the sixth session of differential conditioning. Calibration time base is 50 msec. The potentials as represented here have been averaged, however, they were discernable to single stimulus presentations.

Since the potential was recordable only at the onset of non-visual stimulation and from the optic chiasma the potential may represent activity associated with orienting which is facilitated during conditioning.[1] Feldman and Cohen (1968) have noted that with monkeys a rapid movement of the eyes is associated with a monophasic negative potential in the LGB attributed to central oculomotor mechanisms.

Evans in 1949 (Evans, 1952a, 1952b, 1953) and Y. Gastaut (1951) recorded occipital potentials in the EEG record which were later generally denoted as lambda waves. Characterisitics of the potential as recorded from humans are as follows: "lambda waves are characteristically present with eyes open, during scanning of well illuminated contrast patterns, and disappear with closed eyes, in darkness, during steady fixation of gaze, or if the visual field is featureless." Barlow and Ciganek (1969, p. 183). Rhodes, Lanoir, Saeir, and Naquet (1963) and Naquet, Lanoir, Bach-y-Rita, Saeir, and Rhodes (1967), however, report cortical responses to active and passive movements of the eye in cats in darkness. Chatrian (1964) suggested that lambda represents: (1) ocular movements, (2) proprioceptive afferences from oculomotor muscles, and/or (3) visual afferences originating in the retina. A preliminary hypothesis is that the potential as we have recorded it represents lambda as the consequence of proprioceptive afferences from oculomotor muscles participating in orienting activity.

[1]Note that the conditioned eyeblink activity to the reinforced non-visual element did not produce any regular or noticeable artifact on either the chiasmic or transcortical potential.

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APPENDIX K

STUDY A: DIFFERENCES OF IPSILATERAL AND CONTRALATERAL PHOTIC EVOKED RESPONSE IN UNANESTHETIZED ALBINO RABBIT

In Study 1 evoked responses are recorded to a 3-Hz. 1,000-msec. visual stimulus train. The delivery of photic stimulation is often accompanied by monocular conditioned eyeblink activity at latencies which partially occlude visual input (see Appendix L). Several observations suggest, however, that alterations in photic input to the eye should not alter afferent activity recorded at the ipsilateral occipital cortex. Giolli and Guthrie (1969) have revealed that approximately 95% of the optic fibers in albino rabbit decussate at the optic chiasma. In correspondence with this finding, van Hof (1969), using Dutch rabbits--in which 90% of the optic fibers are reported to decussate (Giolli & Guthrie, 1969)--obtained no interocular transfer of a visual pattern discrimination. Further, Creel (1971) noted a severe reduction of primary evoked response activity at the ipsilateral visual cortex after ipsilateral enucleation of albino-like cats (Siamese). The following investigation was undertaken to extend Creel's observations to albino rabbit and to compare the contribution of ipsilateral and contralateral monocular photic input on evoked potentials recorded at various levels of the visual system.

Method

Subjects

Five albino rabbits weighing 3-4 kg. were implanted with bipolar chiasmic and transcortical recording electrodes. The surgical procedure and coordinates have been outlined in Study 1. A single electrode was

placed into the dorsal portion of the lateral geniculate body (LGB) at the following coordinates: AP +5.0 mm., L +6.5 mm., and H -9.00 mm. from the dural surface. Each S was given up to one-month post-operative recovery.

Apparatus

The apparatus described for Study 1 was utilized for this investigation with minor modifications to suit the specific design requirements of this study. A PDP-8/I computer was used for on-line averaging of the visual evoked potentials.

Procedure

Each S was given one hour of adaptation to the experimental chamber and restraining apparatus. The parameters of amplification for each of the recording sites was assessed during this time while randomly presenting S with binocular flashes. Twenty-four hours later S was reintroduced into the chamber and again retrained. Monocular photic stimulation was delivered 24 times for each eye at intensity setting 4 (Grass Instruments PS-2 photostimulator). Occlusion of visual input for monocular stimulation was achieved by taping a black foam rubber pad over the closed eye. Single 10-μsec. flashes were delivered every 10 sec. S was oriented to the stimulus source at a 90^{o} angle. Evoked responses were simultaneously averaged across 500 msec. (2 msec. per data point) from stimulus onset at the three electrode sites and across the initial 100 msec. as well.

Results

Figures K-1 and K-2 illustrate the averaged evoked potentials (AEPs) for two Ss after stimulation of the eye contralateral and ipsilateral to the LGB and cortical electrode sites. The first 100 msec. of each

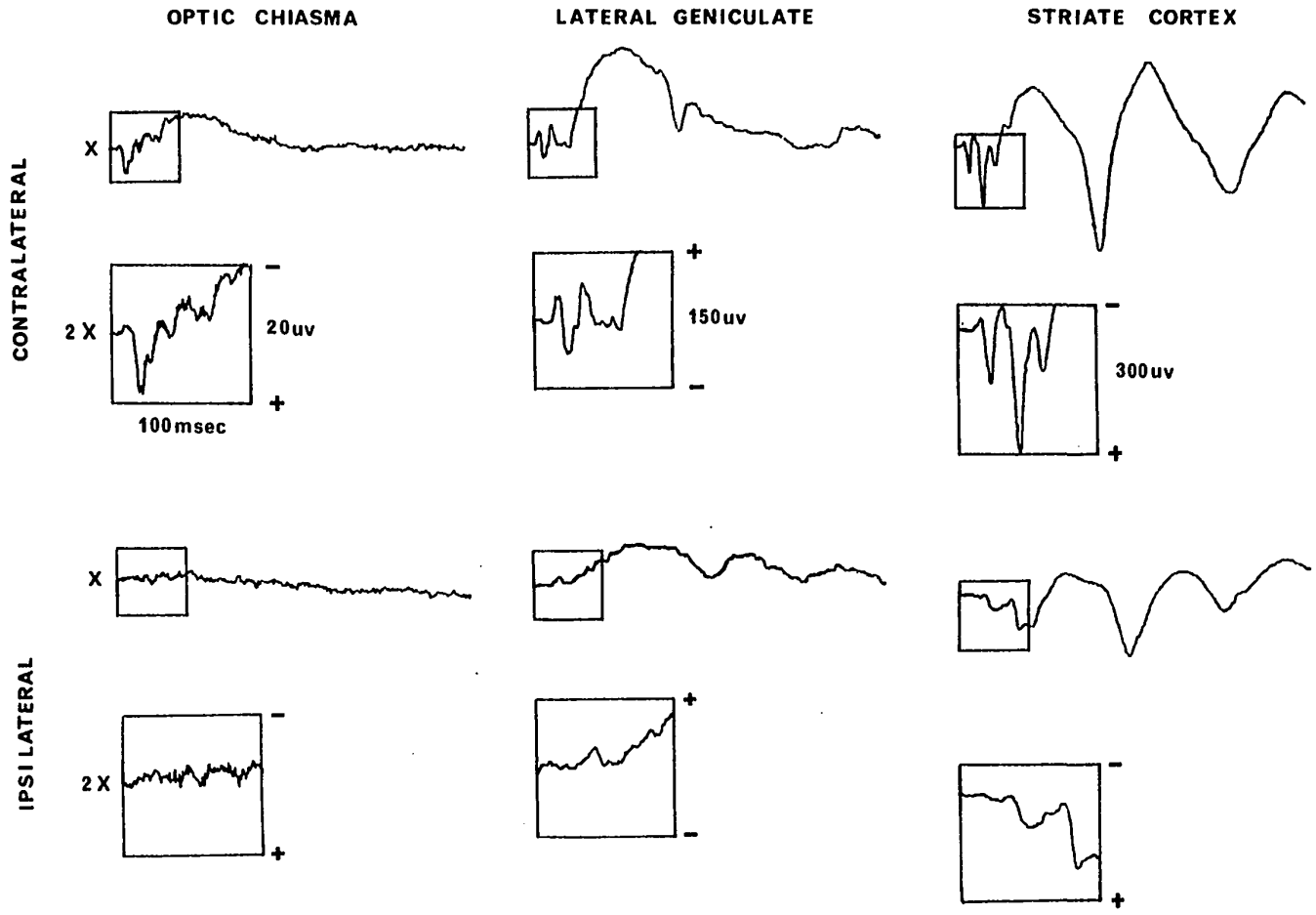

Fig. K-1 Averaged evoked responses as simultaneously recorded at the optic chiasma, dorsolateral geniculate, and striate cortex to contralateral and ipsilateral monocular photic stimulation. The initial 100 msec. of each potential is framed and reillustrated at twice the resolution. Each potential is averaged across 500 msec. Subject PP-5.

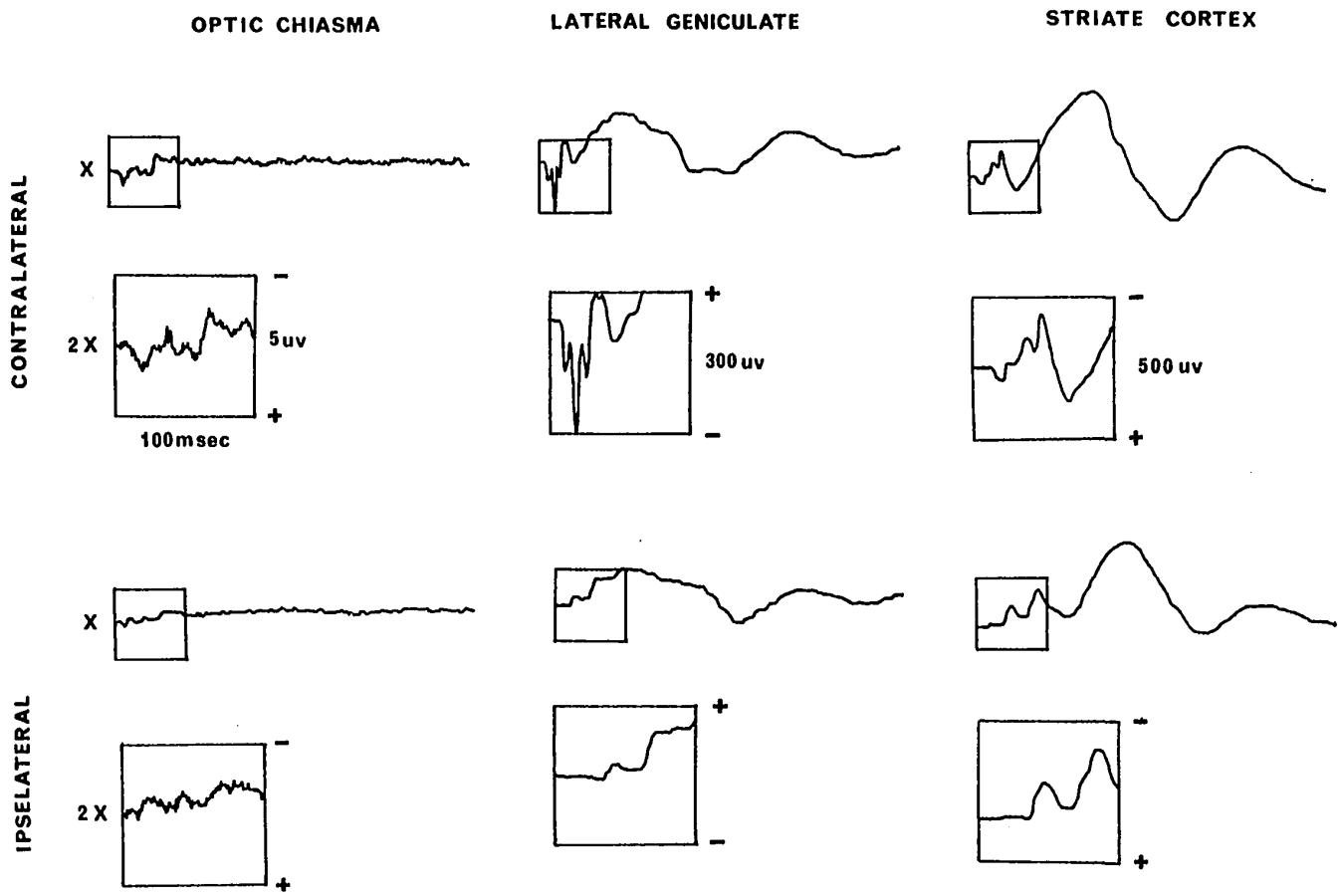

Fig. K-2. Averaged evoked responses as simultaneously recorded at the optic chiasma, dorso-lateral geniculate, and striate cortex to contralateral and ipsilateral monocular photic stimulation. The initial 100 msec. of each potential is framed and reillustrated at twice the resolution. Each potential is averagec across 500 msec. Subject PP-6.

AEP have been framed and illustrated as simultaneously averaged at twice the resolution. Contralateral photic stimulation elicited a characterisitic visual evoked potential at the striate cortex, LGB, and optic chiasma. The potential at the cortex consisted of a 80 msec. series of sharp positive primary deflections. The primary phase was followed by a slow wave negative secondary component. The secondary activity normally peaked at between 100 and 200 msec. and was followed by a series of rhythmic negative afterpotentials. At the LGB negative primary deflections lasting about 50 msec. were replaced by rising positivity peaking at about 90 msec. and followed by a series of slow wave biphasic negative afterpotentials resembling those recorded from the cortex. Potentials at the optic chiasma were variable and often quite weak (<50 uv).

The potentials elicited to ipsilateral stimulation were reduced at the LGB and striate cortex. Changes in optic chiasma potentials were related to electrode placements within the chiasma and will not be considered. Slow wave secondary activity was always evident though the peak amplitude was delayed at both the LGB and cortical sites up to 50 msec. The delay was most evident at the cortical site. Early primary positive deflections, however, were unequivocally absent at both sites. The only primary components which were evident were slower late positive waves generally seen at the cortical level. Of the five Ss investigated none showed any evidence of the sharp positive deflections consistently evident with contralateral stimulation.

Discussion

The results correspond with the findings of Creel (1971) using cat and are consistent with the anatomical reports of Giolli and Guthrie (1969). The lack of primary activity at the level of the LGB to ipsilateral stimulation is also consistent with the contention that little afferent activity is conveyed by the non-decussating fibers of the albino rabbits' optic nerve. Whether these observations are representative of the entire striate cortex or LGB was not assessed. However, it seems reasonable to conclude that fluctuations of photic input to the ipsilateral (left) eye are unlikely to significantly alter primary electrophysiological activity recorded at the contralateral cortex.

Blocking of input to the contralateral eye eliminated primary positive deflections at both the LGB and cortical levels. This is further evidence supporting a contention (e.g., Bishop & Clare, 1952) that the early primary activity represents afferent activity of a specific character relayed through the geniculate to the cortical level That this relationship also holds for electrical stimulation of the optic nerve is suggested by O'Leary and Bishop (1937, 1938) who report that ipsilateral stimulation elicits responses which are significantly lower in amplitude than responses to contralateral stimulation.

STUDY B. DIFFERENCES OF IPSILATERAL AND CONTRALATERAL PHOTIC EVOKED RESPONSES IN ANESTHETIZED ALBINO RABBIT

In Study A we noted the absence of geniculate and cortical representation of afferent activity accompanying ipsilateral photic stimulation. Thompson Woolsey, and Talbot (1950) have, however, recorded ipsilateral early positive deflections to photic stimulation in Ss under deep pentobarbital anesthesia. This activity was of a longer latency than contralateral activity and may represent activity conducted through interhemispheric callosal fibers rather than through the specific system. However, ipsilateral activity "...varied a great deal and had much smaller amplitudes under light anesthesia" (Thompson et al., 1950). Hughes and Wilson (1969) have noted that ipsilateral and contralateral projections of the rabbit are connected by callosal fibers. That these fibers do not conduct behaviorally functional information is suggested by the work of van Hof (1969). He notes that rabbits perform like split-brain animals; they demonstrate little interocular transfer of pattern discrimination which he suggests is due to an insufficency of intra- rather than inter-hemispherial communication. A more parsimonious interpretation of van Hof's findings is that the ipsilateral hemisphere is essentially "untrained" due to a lack of interhemispheric pathways. We would suggest that the bilateral representation of afferent input found by Thompson et al. (1950) was partially the consequence of using anesthetized preparations. If it is assumed that inhibitory processes predominate on interhemispheric callosal transmission during wakefulness but that callosal transmission is released from such influences during deep barbiturate anesthesia, then the findings of Thompson et al. (1950)

which conflict with Creel (1971) and the results of Study A, are resolved. The following study constitutes a replication of Study A except that Ss were pentobarbital anesthetized in an effort to determine whether intracallosal transmission of afferent input is released from subcortical multi-synaptic inhibitory influences.

Method

The same Ss and apparatus employed in Study A were used in Study B. Study B was conducted 24 hours after the completion of Study A. Each S was administered pentobarbital intraveneously (40 mg/kg) with an additional 10 mg/kg administered 30 minutes later between the conditions of contralateral and ipsilateral stimulation. The stimulated eye was taped open.

Results

Figures K-3 and K-4 illustrate the potentials of two Ss to contralateral and ipsilateral stimulation when under pentobarbital anesthesia. The Ss illustrated are the same Ss as illustrated in Figures K-1 and K-2, respectively. In contrast with the unanesthetized conditions of Study A, there was a complete abolition of secondary slow wave negativity to both contralateral and ipsilateral stimulation. This was evident at both the LGB and striate cortex. Several Ss demonstrated an enhancement of the chiasmic potential to both ipsilateral and contralateral stimulation. There was also a specific tendency for the initial positive spike at both the LGB and cortex to be enhanced to contralateral photic stimulation. However, primary spike activity to ipsilateral photic stimulation was never evident at either the LGB or cortical sites; all Ss showed evidence of a further attenuation of ipsilateral primary activity under deep pentobarbital

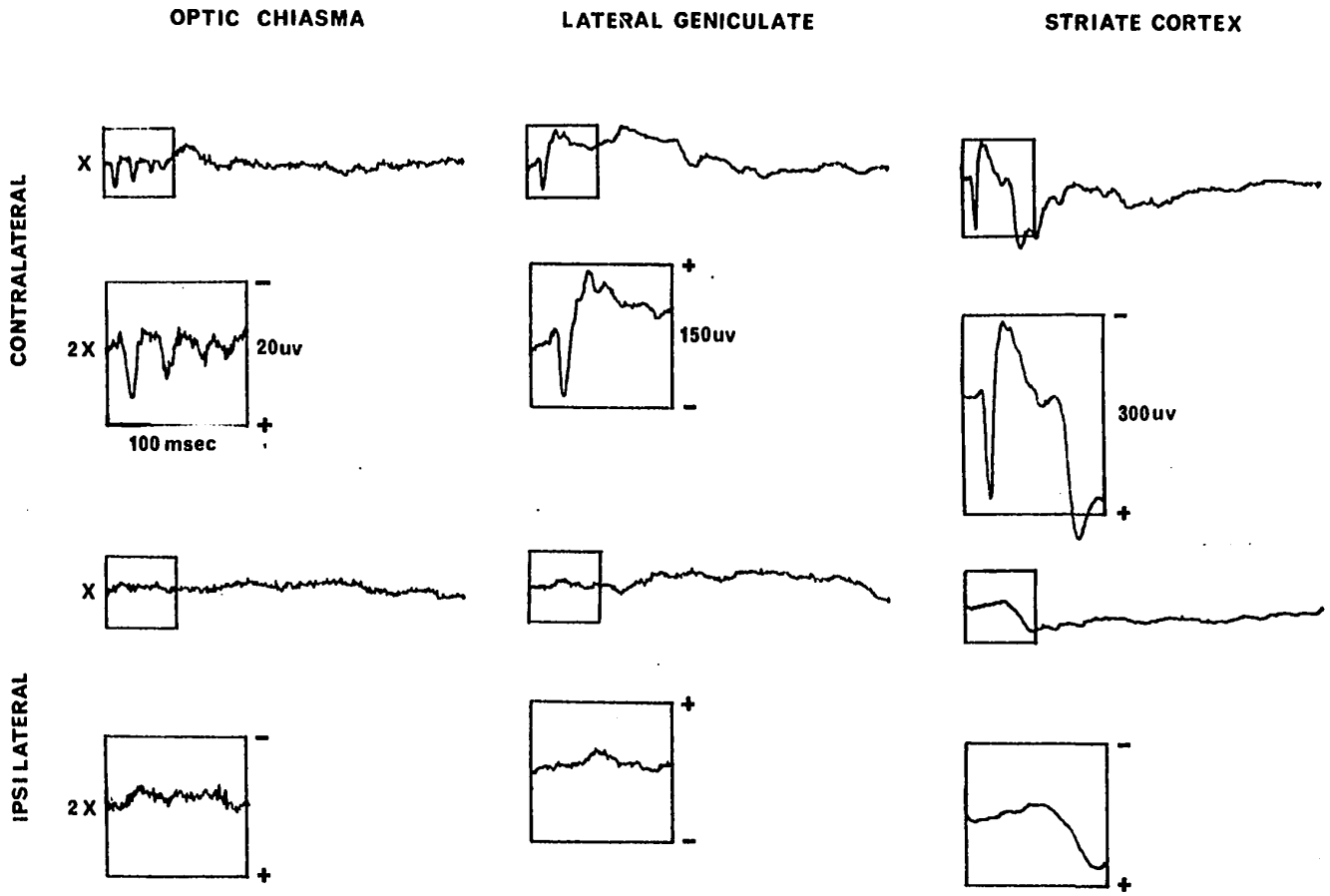

Fig. K-3. Averaged evoked responses recorded under similar conditions as described for Fig. K-1 except that S (PP-5) was under deep pentobarbital (40 mg/kg) anesthesia.

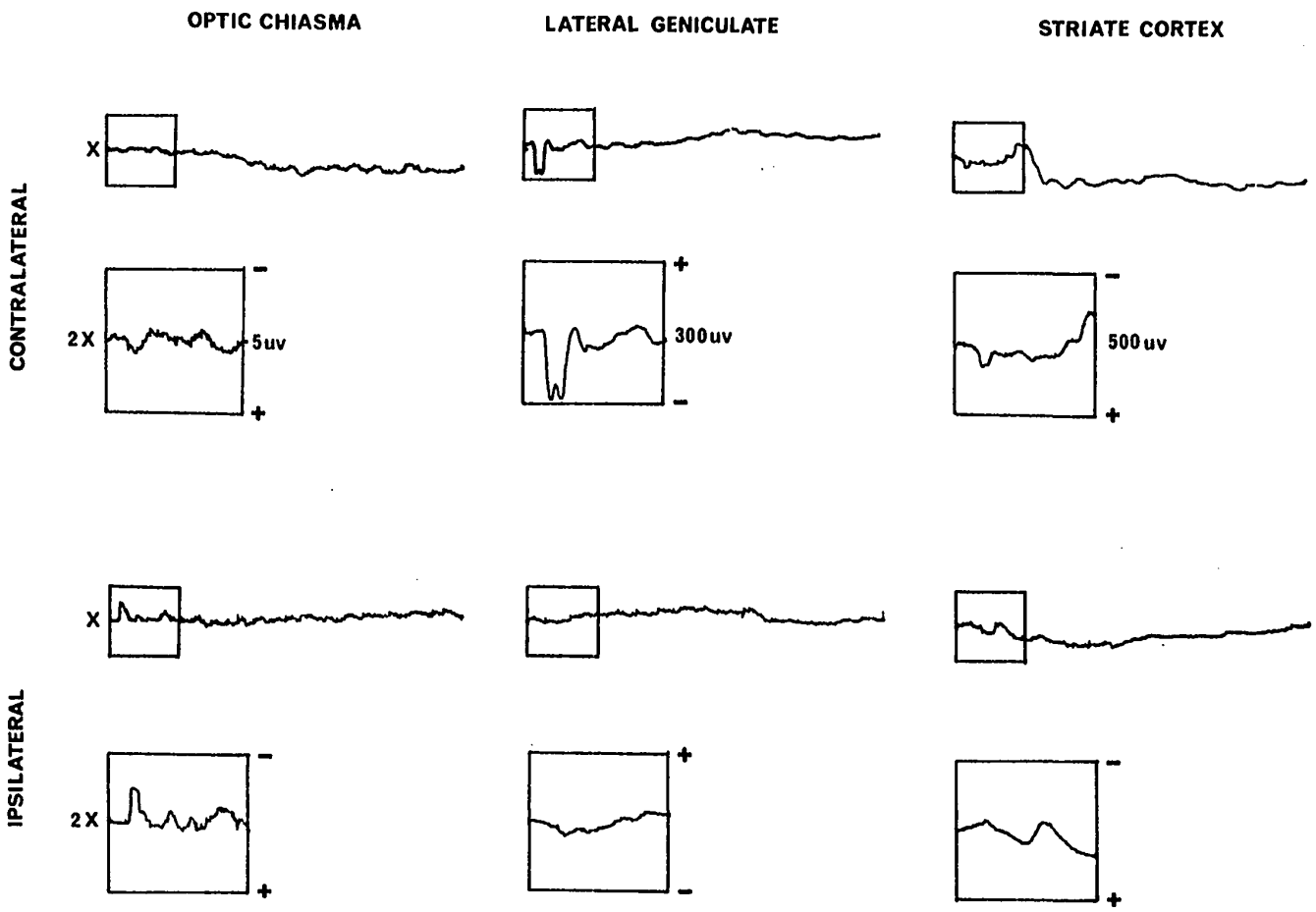

Fig. K-4. Averaged evoked potentials recorded under similar condions as described for Fig. K-2 except that S (PP-6) was under deep pentobarbital (40 mg/kg) anesthesia.

anesthesia.

Discussion

The dramatic attenuation of secondary activity during pentobarbital anesthesia has been reported by several investigators (Fuster & Docter, 1958; Bremer, Stoupel, & Van Reeth, 1960) and has been suggested to be due to the blockage of multi-synaptic transmission of the reticular formation responsible for non-specific secondary activity at the cortical level.

No evidence was found supporting the hypothesis that interhemispheric afferent transmission is released from inhibitory influences with an accompanying enhancement of afferent activity represented ipsilaterally during deep pentobarbital anesthesia. Thus, we would suggest that in the albino rabbit the interhemispheric callosal fibers reported by Hughes and Wilson (1969) are either absent or not functional. This would suggest that the lack of interocular transfer reported by van Hof (1969) is due to the lack of significant afferent input to the ipsilateral cortical primary receiving area. This is most likely the consequence of: (1) a low proportion of non-decussating optic fibers and/or (2) the functional insignificance of interhemispheric callosal fibers, and not necessarily due to a lack of intra-hemispheric transmission.

The findings of Thompson et. al. (1950) indicating a bilateral cortical representation of photic stimulation is inconsistent with the results of Studies A and B. This is apparently not the consequence of using anesthetized preparations. However, we have only sampled one

electrode site at each level of the specific visual system. At the cortical level the locus was adjacent to the lateral border of the splenial sulcus in the medial suprasylvian gyrus and anterior to the peristriate area, a region of low ipsilateral representation according to Thompson.

The disappearance of several late positive primary deflections at the cortex in the anesthetized preparation further supports the contention that late primary activity to photic stimulation represents intracortical multi-synaptic activity within the striate cortex similar to the late positive component to chiasmic stimulation (Bremer, 1961, Bishop & Clare, 1952, see also Appendix F).

The enhancement of the initial primary positive deflection at the LGB and striate cortex suggests that reticulo-retinal inhibitory influences of multisynaptic character are disinhibited during anesthesia. Steriade (1968) has indicated that these potentials are of retinal origin The enhanced chiasmic potential recorded during deep anesthesia is consistent with the observation of enhanced retinal inhibition during arousal noted in Study D. Retinal disinhibition did not apparently enhance activity of homolateral projections as only a late slow wave positive component was represented at the ipsilateral striate cortex.

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APPENDIX L

AVERAGED CONDITIONED EYEBLINK ACTIVITY TO PHOTIC AND CHIASMIC STIMULATION: CR LATENCIES

A 1,000 msec. CS-US interstimulus interval was employed in establishing conditioned eyeblink activity during Study 1 and Study 2. Average latency characteristics of the conditioned eyeblink were assessed to determine where movement artifacts might be expected to occur on AEP activity averaged at the striate cortex. This was accomplished by leading the amplified differential voltage between the two phototransistors of the eyeblink detector to the averaging computer and triggering the averaging process 24 msec. prior to CS onset. An averaged response was accumulated across 2,048 msec. corresponding to 1,000 msec. pre-US and post-US intervals.

Figure L-1 illustrates the conditioned response average for one S (F-19) conditioned to 3-Hz. photic stimulation (lower trace) and another S (F-18) conditioned to 3-Hz. chiasmic stimulation (upper trace). The average latency of response onset was between 500 and 600 msec. after CS_1 onset. The peaks ~40 msec. after delivery of the US correspond to the regularly elicited UR. No reliable differences in response latency or characteristics were seen as a function of the method of visual stimulation. There was some evidence that Ss respond to onset of the CS train by opening their eye slightly as indicated by depression of the averaged activity between CS_1 and CS_2 for F-19. While the eye which received the US was found to react to the CS presentation, no corresponding eyeblink activity was ever observed to the contralateral eye, i.e.,

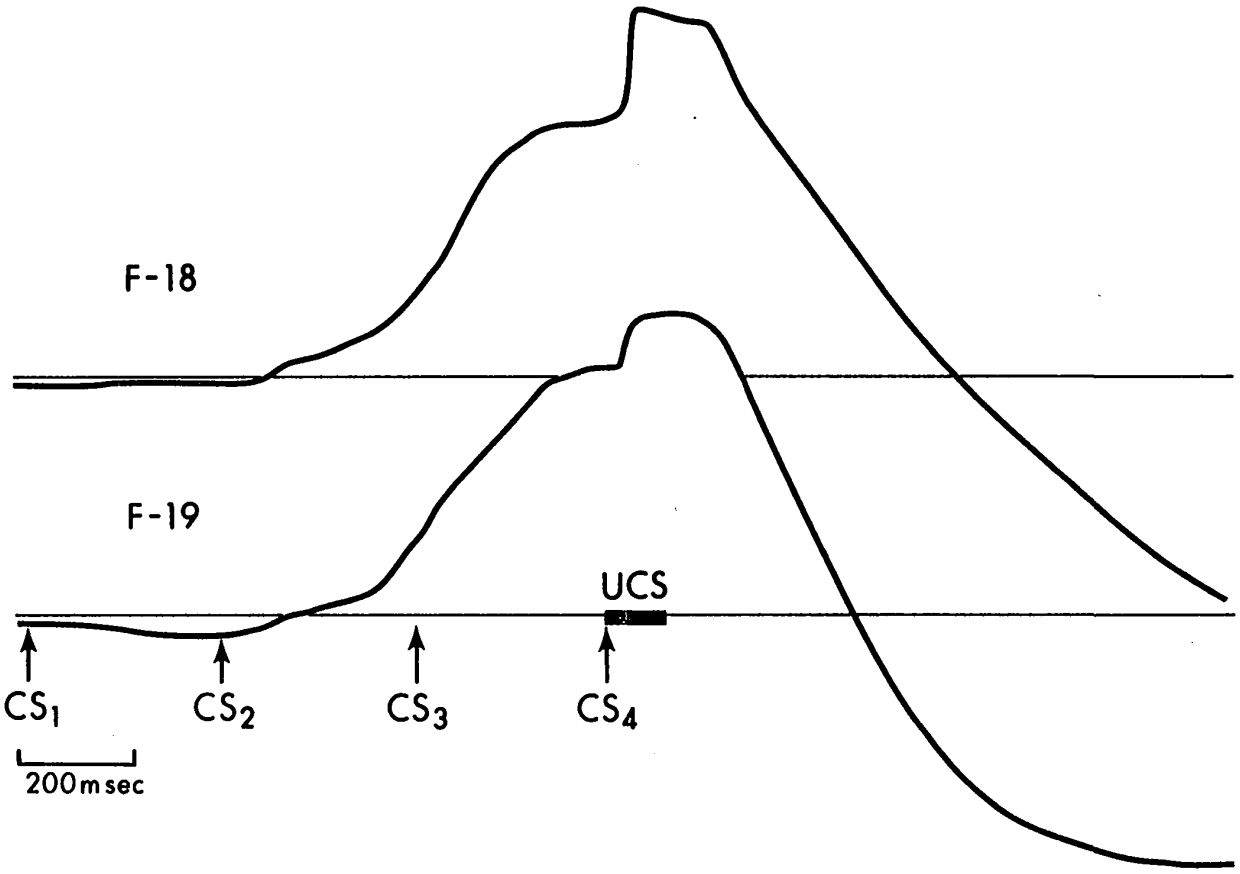

Fig. E-1. Averaged conditioned eyeblink responses to photic (S F-18) and chiasmic (S F-19) stimulation at 3 Hz. Eyeblinks were detected by a phototransistor bridge circuit which monitored the reflectance of light between the pupil and fur caudal to the eye. The differential was balanced and changes accompanying eyeblink activity were amplified and led to a CAT 1000 signal averaging computer for averaging across 2,048 msec, initiated at CS onset. Note the slight deflection between CS_1 and CS_2 by F-19 identifying a regular opening of the eye at onset of CS.

conditioned eyeblinks, were specific for the reinforced eyeblink.

On the basis of these conditioned response latency characterisitics it seems unlikely that conditioned eyeblink motor artifacts would be reflected in the visual AEP to the initial visual stimulus presentation, CS_1. Also, differences between photic and chiasmic generated evoked potentials are unlikely to be attributable to motor artifacts associated with differing conditioned eyeblink response latencies.