EFFECTS OF RETICULAR FORMATION, PULVINAR, AND SUPERIOR COLLICULAR STIMULATION UPON VISUAL CORTICAL RESPONSIVITY IN THE CAT

by

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INTRODUCTION

Sensory systems have been classically conceptualized as consisting of a receptor generating impulses which are relayed through a number of synapses to the cortex for information processing. Conceived in this way, the sensory system is a rather inflexible mechanism of information transmission, since it is assumed that the total input to the receptor is conveyed relatively unchanged to cortex via specific projection systems. In recent years, however, a more dynamic view of sensory systems has emerged, namely, that activity along the specific pathways can be modified by the so-called "diffuse" systems of the brain. In particular, the reticular activating system has been shown to play an important role in the modification of sensory activity at various levels along the neuroaxis.

The amplitude of single evoked responses or the recovery cycle have been traditionally utilized in evaluating the responsivity of sensory systems with macroelectrode recordings (Chang, 1959; Bremer, 1959). The recovery cycle may be defined as the amplitude of the second of two successive responses studied as a function of the interval between the two eliciting stimuli. Studies in several sensory modalities have shown that both of these measures may be modified by stimulation of the reticular formation. In brief, it has generally been reported (Hernandez-Peon, Scherrer, and Velasco, 1956; Bremer and Stoupel, 1959; Dumont and Dell, 1960), that when an adequate stimulus is used to evoke cortical responses, prior reticular tetanization tends to depress the amplitude of single-evoked responses. When an inadequate (shock) stimulus is utilized, the amplitude of the cortical evoked response is enhanced by such tetanization (Bremer, 1960; Long, 1959; Steriade and Demetrescu, 1963). On the

other hand, the recovery cycle is potentiated with adequate and inadequate stimuli. For example, Lindsley (1958) has shown that the evoked response to the second of two light flashes at an interval of 50 millisecond is normally depressed, but increases in amplitude following tetanization of the reticular formation. Similar data have been reported when paired geniculate shocks were used to evoke cortical responses (Steriade and Demetrescu, 1967; Demetrescu, Demetrescu and Iosif, 1965).

It has generally been assumed that the effects of reticular stimulation upon the recovery cycle are independent of reticular effects on single evoked responses (Schwartz and Shagass, 1963; Shagass and Ando, 1970; Steriade and Demetrescu, 1962). No study however, has systematically investigated the effects of reticular tetanization on single responses (at various temporal intervals) and on the recovery cycle in the same preparation. Such an approach was used in the present study in order to determine the relative magnitude of these effects and their respective time course. In addition, the influence of other variables such as intensity of subcortical tetanization and geniculate stimulation, and Nembutal anesthesia were also investigated.

It is also well known that stimulation of a number of brain structures outside the reticular formation can also influence the amplitude of single evoked responses; e.g., superior colliculus, pulvinar, hippocampus, hypothalamus, and the intralaminar thalamic nuclei. Accordingly, it is reasonable to suspect that the recovery cycle may also be affected by stimulation of such subcortical structures, either because they are functionally connected with the reticular formation or by more direct inputs

to cortex. Thus, in addition to the mesencephalic reticular formation, the effects of pulvinar and superior collicular tetanization were also investigated in most preparations. These subcortical structures were chosen in particular, since it has been suggested that the pulvinar (Battersby and Oesterreich, 1963) and the superior colliculus (Brown and Marco, 1967) may mediate their effects on single responses via the reticular formation. This hypothesis could be tested in the present study by determining if stimulation of all three structures produces analogues effects on both, single evoked responses and the recovery cycle. In addition, the functional interconnections among these structures were studied more directly by lesioning one of these structures and then retesting the effects of tetanizing the remaining structure(s) following the lesion.

In the present study, single and paired brief shocks of the lateral geniculate body were used to evoke cortical responses. Such stimulation produces a response with a characteristic waveform which permits some analysis of the site of origin of its components (see Method). In most experiments the effects of reticular formation, superior collicular and pulvinar tetanization were determined on the responses elicited by single and paired (recovery cycle) geniculate shocks. Since barbituates are known to depress cortical activity (Curtiss, 1940; Marshall, Talbot and Ades, 1943; Wagman and Battersby, 1964) and the functioning of the reticular formation (Arduini and Arduini, 1954; French, Verzeano, and Magoun, 1953; Goodman and Mann, 1967), locally anesthetized, acute preparations were utilized.

LITERATURE REVIEW

Since Moruzzi and Magoun's (1949) discovery of the reticular activating system, a number of studies have shown that stimulation of the reticular formation produces a modification of sensory activity along specific pathways. In the visual system, there is evidence for reticular modulation of afferent activity at the receptor, lateral geniculate body, and the cortical projection areas.

At the retinal level, Granit (1955) demonstrated that reticular stimulation causes either a facilitation or inhibition of light responses in ganglion cells. Similar effects were obtained by Hernandez-Peon, Scherrer and Velasco (1956) with macroelectrode recordings of optic tract activity. Additional evidence for peripheral effects of reticular stimulation was provided by Jacobson and Gestring (1958), who found that tetanization of the bulbar reticular formation reduced ERG amplitude for over two minutes. More recently, Nakai and Domino (1968) showed a reduction of reticular facilitation of lateral geniculate and cortical responses to optic tract stimulation following bilateral enucleation. These data were interpreted as showing that reticular disinhibition of inhibitory retinal discharges is an important mechanism in the reticular modulation of the visual system. Although the above studies have been cited as evidence for centrifugal fibers originating from the reticular formation (French, 1959; Livingston, 1958; 1959), the anatomical basis for this contention is questionable (see Ogden, 1968 for recent review).

The responsivity of the lateral geniculate body has also been shown to be altered by the activation of the reticular system. The

geniculate evoked response following optic tract stimulation consists of a presynaptic and postsynaptic component. Two studies (Suzuki and Taira, 1961; Okuda, 1962) have reported facilitation of the postsynaptic component following reticular stimulation, while no change was found in the presynaptic response. However, more recent studies (Pecci-Saavedra, Wilson and Doty, 1966; Angel, Magni and Strata, 1965) have shown an increase in geniculate presynaptic inhibition resulting from reticular stimulation. When a photic stimulus is used, geniculate evoked responses have been found to be inhibited by reticular tetanization (Hernandez-Peon, et. al., 1956).

Reticular effects on the lateral geniculate body have also been studied with microelectrodes. Arden and Soderberg (1959) showed that stimulation of the brain stem reticular formation produced both increased and decreased resting discharge levels in the rabbit geniculate. Furthermore, units that did not respond to flickering light could be induced to do so by reticular stimulation. Similar effects were obtained with a natural arousing stimulus. Satinsky (1968) reported similar data in the cat, while Ogawa (1963) found only excitatory effects of reticular stimulation.

In addition to the lateral geniculate body, changes in responsivity induced by reticular stimulation of other subcortical structures associated with visual function have also been investigated. Thus, superior collicular responses to optic nerve or tract shocks were found to be facilitated following reticular activation (Marchiafava and Pepeu, 1966; Sumitomo and Vasumasa, 1967; Chi and Flynn, 1969). Recently it was reported that single unit responses of the pulvinar elicited by

optic nerve stimulation may also be enhanced following tetanization of the reticular formation (Suzuki and Kato, 1964). Furthermore, some units of the pulvinar would only respond to optic nerve shocks when these were preceded by reticular activation. From these data it was hypothesized that reticular and retinal fibers may converge on the same cells in the pulvinar.

Experiments investigating the influence of the reticular system on single cortical evoked responses have shown that reticular stimulation can both facilitate and diminish the evoked response amplitude. In general, when an adequate sensory stimulus is used, prior reticular stimulation diminishes the peak to peak amplitude of the primary cortical response (Hernandez-Peon, et. al., 1956; Bremer and Stoupel, 1959; Bremer, 1959) and at the same time the secondary discharge or "b" wave is facilitated (Steriade, Belekhova, and Apostal, 1968). However, when an electrical shock is applied to the visual system, then reticular stimulation has been found to facilitate the cortical response (Cavaggioni and Goldstein, 1965; Chi and Flynn, 1968; Dumont and Dell, 1960; Nariskashvili and Moniava, 1965; Redding, 1967). This effect has been shown to be dependent on the frequency of reticular stimulation (Long, 1959). The above findings may be specific to the cat, since in the rabbit, reticular stimulation had no effect on the primary flash response, while the secondary response was consistently enhanced (Fuster and Docter, 1962). Also in monkey, cortical responses to both peripheral and central stimuli were enhanced by reticular stimulation (Bartlett and Doty, 1970).

Two points should be discussed regarding the interaction of

the reticular formation with the visual system. First, it should be noted that the effects of reticular stimulation obtained at cortex are independent of those recorded from the lateral geniculate body. This has been demonstrated by studies in which opposing effects were obtained from lateral geniculate and cortical recordings (Long, 1959; Steriade and Demetrescu, 1960). Similarly, Bremer (1959) has reported that reticular tetanization may have no effect on geniculate responses to optic tract shocks, while at the same time cortical responses are greatly enhanced. These data suggest that the reticular formation may have at least two separate inputs to the visual system. A direct reticulo-geniculate input has also been demonstrated anatomically (Scheibel and Scheibel, 1958).

The second point concerns the manner in which the reticular formation mediates its effects, and in this area two hypothesis have been advanced. Bremer (1959) postulates that reticular stimulation activates cortical neurons, thereby, causing them to be in a relative refractory state. A peripheral (adequate) stimulus, because of its "insufficient density" is unable to overcome this refractoriness. Thus, following reticular stimulation only a limited number of neurons are available to contribute to the evoked response and this results in a diminished response. A shock stimulus on the other hand, is highly synchronous and it can overcome the refractoriness of these units, thereby evoking a response to which are added neurons from the subliminal fringe resulting in an increased response amplitude. A similar explanation has been proposed by Nariskavili and Moniava (1965).

Support for this hypothesis was provided by Steriade and Demetrescu (1960) who showed that evoked responses to flickering light below 5 cps are reduced in amplitude by reticular stimulation; however, when flicker rates were increased, thereby producing synchronous responses, the cortical occlusion was changed to facilitation. Similar data were reported for the auditory modality (Steriade and Demetrescu, 1962).

Since the magnitude of the subliminal fringe varies inversely as a function of stimulus intensity (Marshall, 1949), another procedure for testing Bremer's hypothesis is evident. This consists of varying the intensity of a central visual stimulus while maintaining the intensity of reticular stimulation at some constant level. Bremer's explanation for the facilitation of centrally evoked responses by reticular stimulation predicts that submaximal central stimuli should be facilitated proportionally to a greater extent than supramaximal stimuli.

The second explanation of the effects of reticular activation postulates that synaptic inhibitory or facilitatory processes are involved in the modulation of specific sensory systems. Thus, Hernandez-Peon (1959) states that the depression found in the evoked response with reticular stimulation at the lateral geniculate lasts for too long a period to be explained by neuronal refractoriness, and therefore, synaptic inhibition must be at work. Synaptic inhibition was also proposed by Purpura (1955) to explain the reduction of direct cortical responses by reticular stimulation. Support for this hypothesis is also provided by Jung's (1958, 1959) contention of direct convergence of reticular

fibers on visual cortical neurons. Jung divides the neurons of the primary visual cortex into five types (A,B,C,D, and E) depending on their response to diffuse light. Jung has shown that reticular stimulation tends to facilitate the characteristic response pattern of each neuronal type. Thus, for example, neurons which typically respond to diffuse light throughout the presence of a stimulus (type B), will increase their rate of response with reticular stimulation. Similarly, D units, which fire only to light offset, will also increase their firing rate to the offset of light following activation of the reticular formation. Although these findings have been essentially confirmed in the rabbit by Fuster (1961), they do not necessarily indicate direct convergence of reticular and visual fibers. The only direct test of this hypothesis, would be to record intracellularly the interaction of reticular and visual stimulation.

The previously reviewed studies demonstrate that reticular stimulation significantly changes cortical responsivity when the amplitude of a single evoked response is used as the index of the responsivity of the cortex. However, in addition to the single evoked response, the recovery cycle or cortical temporal resolution has also been frequently utilized as a measure of local cortical responsiveness (Chang, 1959). The recovery cycle may be defined as the amplitude of a response to a test stimulus measured as a function of the delay from a preceding conditioning stimulus (Bartley, 1968). In the visual system it has been demonstrated that the evoked response to a test stimulus is briefly enhanced from one to five milliseconds and then it becomes markedly depressed at interstimuli intervals of 50 to 100 milliseconds. At longer delays, the response to the second stimulus begins

to recover reaching full amplitude at delays of 300 to 500 milliseconds (Marshall, 1949; Clare and Bishop, 1952; Schoolman and Evarts, 1959; Sturr and Battersby, 1966).

A few papers have been concerned with the effects of reticular stimulation on cortical temporal resolution. Lindsley (1959) first reported that following five second tetanization of the reticular formation the visual cortex could resolve two light flashes 50 milliseconds apart, whereas prior to reticular stimulation only the response to the first flash was observed. Evoked responses in the optic tract and lateral geniculate failed to show this effect. Subsequently, this phenomenon was demonstrated in audition (Steriade and Demetrescu, 1961) and somesthesis (Schwartz and Shagass, 1963, Shagass and Ando, 1970). More recently, the effects of reticular stimulation on paired geniculate shocks has also been studied (Demetrescu, Demetrescu, and Iosif, 1965; Steriade and Demetrescu, 1967). Generally it has been assumed that reticular mechanisms affecting single response amplitudes are independent of the mechanisms potentiating the recovery cycle (Schwartz and Shagass, 1963; Shagass and Ando, 1970; Steriade and Demetrescu, 1963). However, no study has investigated the effects of reticular stimulation on single responses (at various temporal intervals) and the recovery cycle in the same preparation.

Aside from the reticular system discussed above, there are other structures which may affect the corticopetal transport of visual afferent activity. These include the midline thalamic nuclei (Jasper and Ajmone-Marsan, 1952; Jasper, Ajmone-Marsan and Stoll, 1952; Jung, 1958,

Long, 1959), certain regions of the hypothalamus (Chi and Flynn, 1968), the lateral posterior thalamic complex (Long, 1959; Morillo, 1961; Battersby and Oesterreich, 1962; Brown and Marco, 1967) and the superior colliculus (Brown and Marco, 1966, 1967). In these studies single stimuli have generally been employed, therefore, possible changes in cortical temporal resolution obtained by stimulation of non-specific structures other than the reticular formation have not been investigated in the visual system.

It had been speculated by Battersby and Oesterreich (1963) that the pulvinar may mediate its effects on single photic responses indirectly through the intralaminar nuclei, superior colliculus and the midbrain reticular formation. Brown and Marco (1967), however, showed that stimulation of the pulvinar and the superior colliculus has differential effects on a cortical response evoked by photic stimuli. Thus, stimulation of the pulvinar potentiates the cortical evoked response, while superior collicular stimulation diminishes the primary response and enhances the secondary discharge. Stimulation of both structures facilitates a cortical response evoked by a brief shock to the lateral geniculate body. These data were interpreted as indicating that the pulvinar effects on cortical responses are not conveyed via the superior colliculus. In addition, it was noted that the superior colliculus is functionally related to the mesencephalic reticular formation since stimulation of both structures has the same effect on photic and shock evoked responses. Therefore, according to Brown and Marco (1967) the effects of pulvinar stimulation are probably also not

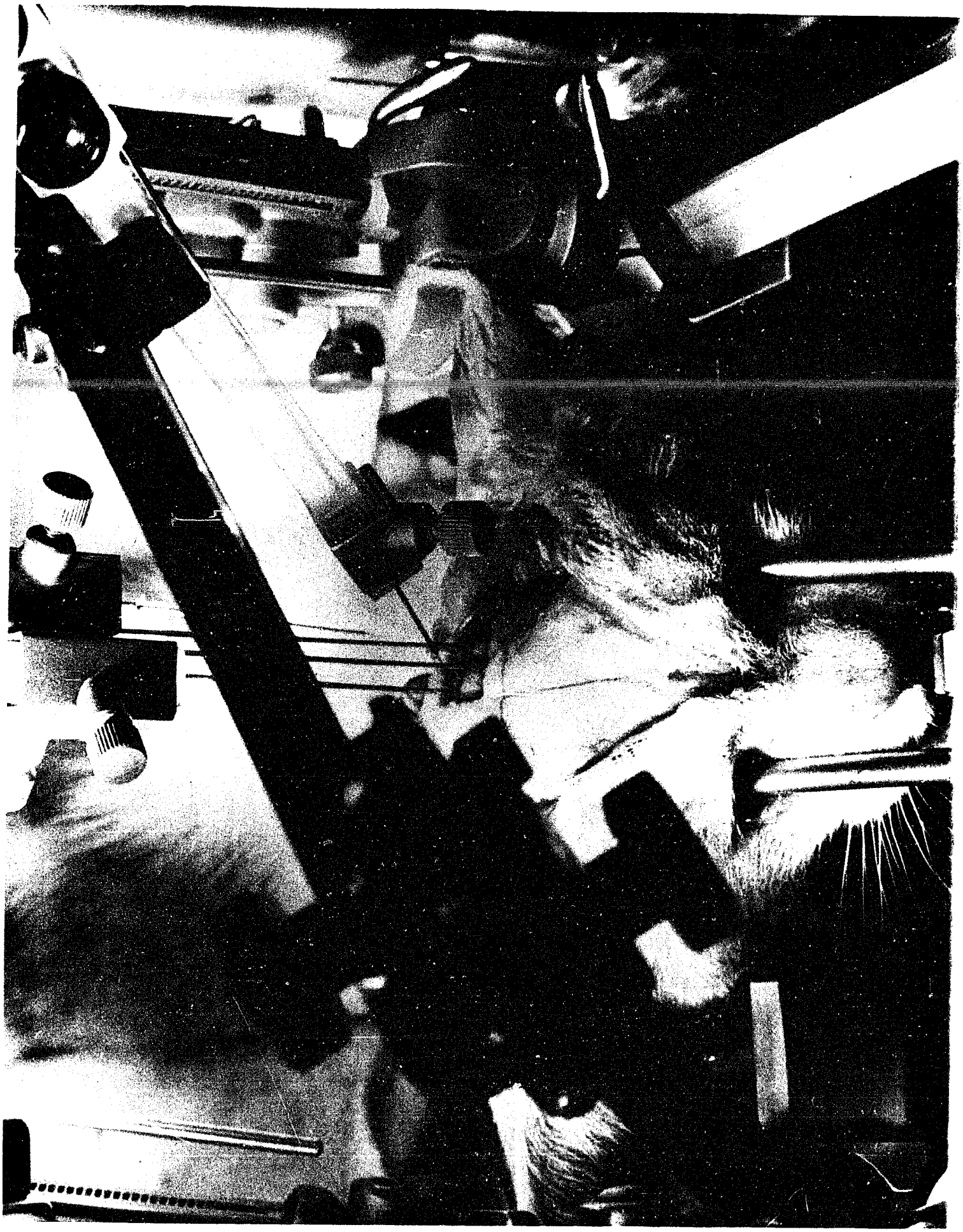
mediated by way of the reticular formation, while those obtained by stimulating the superior colliculus may be conveyed in this manner. Anatomically, there are reciprocal connections between the superior colliculus and the reticular formation (Nauta and Kyper, 1958; Altman and Carpenter, 1961; Altman, 1962). No direct pathways from the pulvinar to the reticular formation have been reported, although a recent study has suggested inputs from the mesencephalic reticular formation to the pulvinar (Suzuki and Kato, 1969).

METHODS AND MATERIALS

Surgery and Histology. Observations were obtained on a total of 55 adult cats, each weighing between 2-3 kilograms. Following ether induction in an airtight box, a trachial cannula was inserted, and the animal thereafter maintained on an ether bottle at a moderate level of narcosis. After alignment in a stereotaxic apparatus, the left saphenous vein was cannulated for subsequent infusion of gallamine triethiodide (Flaxedil), a mdiline skin incision was made, and the temporal muscles reflected bilaterally to expose the dorsal calvarium. A single trephine hole on the left side was then rongeured to make an opening extending rostral-caudally from approximately A 15 to A -5, and laterally to about 15 millimeters from midline. The dura was then reflected exposing portions of the lateral and suprasylvian gyri, and the cortex thereafter kept moist with warm saline.

In most animals, four bipolar stimulating electrodes were directed at the following structures according to the stereotaxic coordinates of Jasper and Ajmone-Marsan (1954): lateral geniculate body at A 6.5, L 10.0; pulvinar at A 6.5, L 6.0; mesencephalic reticular formation at A 3.0, L 2.5; and superior colliculus at A 1.0, L 2.5. The reticular formation and superior collicular electrodes were always placed on a common electrode carrier, while the other two (geniculate and pulvinar) were always independently manipulated (see figure 1). After all wounds and pressure points were liberally infiltrated with Citanest (prilocaine), ether was withdrawn, Flaxedil infused into the saphenous vein to immobilize the preparation, and the animal then placed on artificial respiration at about 35 cc of air per stroke, 28 strokes per minute. A heating blanket set at body temperature was wrapped around

Fig. 1 Illustration of preparation in stereotaxic instrument. Dorsal exposure of left visual cortex. Stimulating electrodes impale lateral geniculate body, pulvinar, mesencephalic reticular formation, and superior colliculus. Also shown are two bipolar recording electrodes resting on the pial surface, one on marginal and the other on the suprasylvian gyrus.



the animal's abdomen and if necessary the rear of the animal was slightly elevated to prevent stasis.

At the termination of the experiment, subcortical stimulating loci were anodally fulgarated by passing a DC current of 0.5 milliamperes between the electrode tip and the earbars of the stereotaxic instrument (cathode) for 25 to 30 seconds. The animal was then sacrificed by an intravenous injection of Nembutal, the brain removed, rinsed and then placed in a 10 percent buffered formalin solution for 7 to 14 days. Frozen sections were subsequently cut at 48 micra thickness and every fifth section stained with a Kluver-Barrera stain. The stained sections were microscopically examined in order to reconstruct the stimulating positions of all electrodes in each cat.

Electrodes and equipment. In most animals electrical activity was recorded with silver balled tipped tungsten electrodes placed on the pial surface. The electrodes monopolarly recorded the voltage oscillations in cortex with a saline soaked cottonoid referent placed either on the cranial base or a muscle edge. Two recording electrodes were used simultaneously, one on marginal and the other on the suprasylvian gyrus. Cortical activity picked up by the electrodes was fed through two Grass cathode followers, and two differential Grass AC preamplifiers each with a band pass of 7 cps to 10 kc (3 db cutoff), and was then displayed on a split beam oscilloscope (Tektronix 561) and a slave storage scope (Tektronix 564). Five or more responses were superimposed on the latter scope and photographed for later data analysis. In a number of early animals, bipolar tungsten electrodes

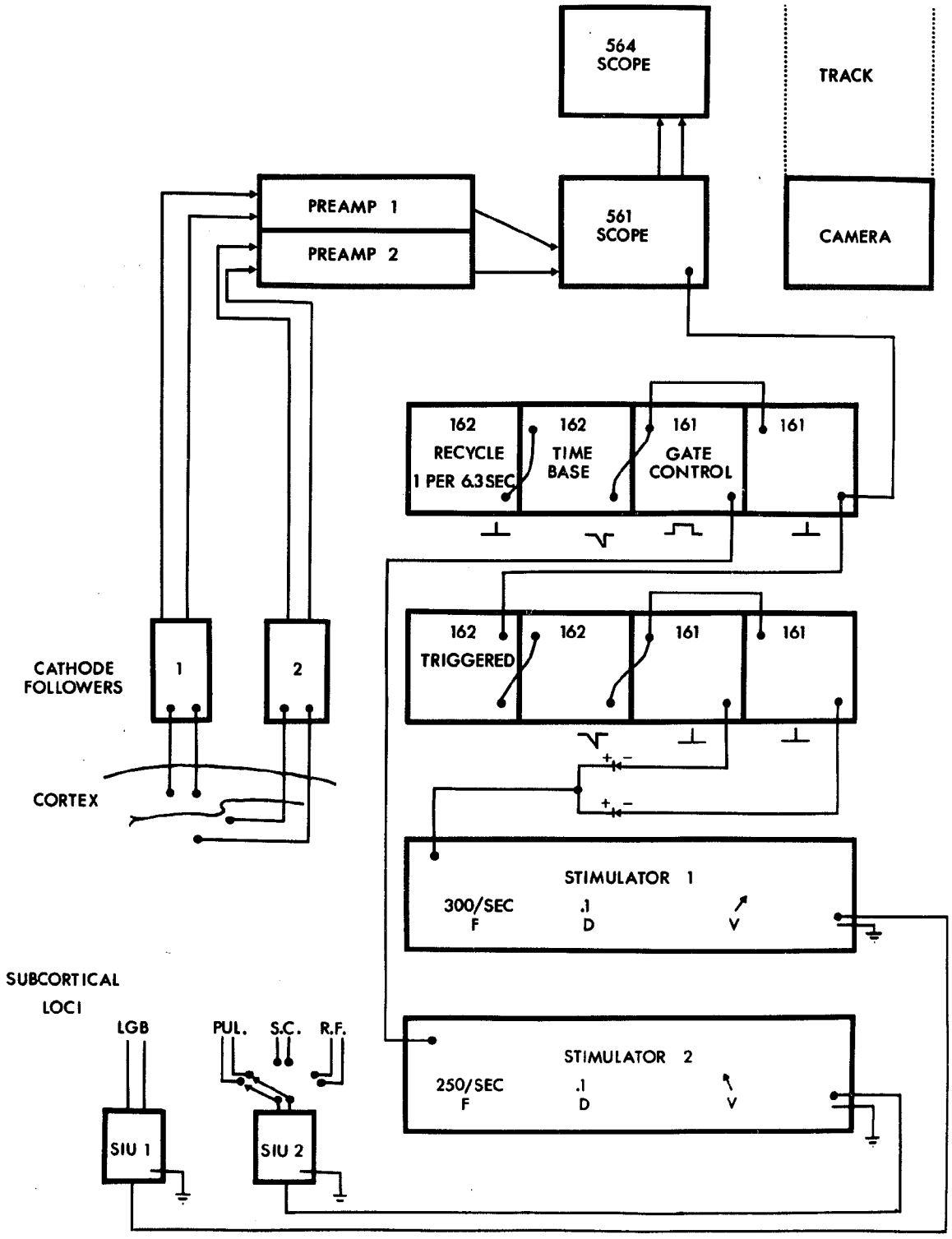
were used, the balled tips being separated by 1.0-2.0 millimeters.

Stimulating electrodes consisted of two pieces of 0.005 inch tungsten wire (formvar insulated except for the flush cut and polished ends) encased in a 20 gauge stainless steel tube also insulated with formvar. The tips of the stimulating wires extended approximately 2 millimeters beyond the edge of the tube and were horizontally separated by about 0.5 millimeters. In most animals, electrodes were lowered into the lateral geniculate, pulvinar, superior colliculus and the mesencephalic reticular formation.[1] In several early animals, only lateral geniculate stimulation was used in conjunction with reticular formation and/or collicular tetanization.

Overall timing control for stimulation of the various subcortical loci and for recording the cortical responses was accomplished by a Tektronix Waveform (Model 162) generator. Every 6.3 seconds it triggered another waveform generator which served as a time base for two pulse generators (Model 161). One of the pulse generators gated a Grass S-4 stimulator for activation of the reticular formation, pulvinar or superior colliculus, while the other triggered the scope tracings and another set of waveform and pulse generators. This second set of generators controlled another S-4 stimulator through a diode-coupled cable, thereby permitting independent variation of the delay between two pulses used for lateral geniculate stimulation. Figure 2 presents a flow chart

[1]The resistance between the tips of the stimulating electrodes was determined periodically in physiological saline and was found to be approximately 50,000 ohms.

Fig. 2 Schematic of electrophysiological stimulating and recording system.


564
SCOPE
TRACK
PREAMP 1
PREAMP 2
561
SCOPE
CAMERA
162
RECYCLE
1 PER 6.3SEC
162
TIME
BASE
161
GATE
CONTROL
161
162
TRIGGERED
162
161
161
CATHODE
FOLLOWERS
1
2
CORTEX
STIMULATOR 1
300/SEC
F
.1
D
V
SUBCORTICAL
LOCI
LGB
PUL.
S.C.
R.F.
SIU 1
SIU 2
STIMULATOR 2
250/SEC
F
.1
D
V

of the stimulating and recording equipment.

In a few experiments the lateral geniculate body was tetanized and this was followed at various delays by a single geniculate shock. Both, the tetanizing train and the single pulse were delivered through the same electrode by connecting the isolation unitsin series.

Procedure. All experiments were conducted at least one hour following the discontinuation of ether anesthesia in a dark, shielded room. The subcortical stimulating electodes were lowered toward their respective stimulating sites, the particular final locus of stimulation for each electrode being determined by a combination of calculated stereotaxic coordinates and electrophysiological results. The lateral geniculate electrode was first lowered in 0.5 millimeter steps, and at each position the cortical response evoked at the marginal gyrus by a 0.1 millisecond shock was assessed. That locus, which when stimulated produced a reliable four or five component response, was finally chosen and used throughout the remainder of the experiment. The pulvinar electrode was then lowered in 0.5 millimeter steps; with each advance, stimulation was effected with 4 or 5, 0.1 millisecond pulses at a frequency of 8-12 cps. That electrode position which produced an augmenting response of maximum amplitude in the suprasylvian gyrus was then determined and fixed. The reticular formation-superior colliculus electrodes (which were on a common carrier), were then slowly lowered into the midbrain, and a high frequency (250-300 cps), 100 millisecond train of stimuli was used to test for the possible effects on the subsequent cortical response elicited by a single geniculate shock. The position at which stimulation

with each electrode produced a reliable facilitation of the cortical response was determined and then used for subsequent experimentation. In addition, to the evoked response facilitation, reticular formation stimulation typically produced a marked bilateral pupillary dilation. This effect was greatly minimized or absent with superior collicular stimulation and it was never observed with stimulation of the pulvinar. Pupillary dilation was thus additionally utilized as a guide for the placement of the electrode in the reticular formation.

Following placement of the electrodes, conditioning (S_c) and test (S_t) shocks were delivered to the lateral geniculate body at inter-shock intervals ranging from 5 to 150 milliseconds in order to determine the recovery cycle. (In a few instances S_c-S_t delays of up to 500 milliseconds were employed). High frequency stimulation of the reticular formation, superior colliculus or pulvinar was presented prior to the onset of S_c to determine its effect on the recovery scale. S_c onset was generally synchronous with the end of subcortical tetanization. The effects of subcortical activation on single evoked responses were also investigated in each experiment by omitting the presentation of S_c.

Several other experiments were performed to investigate the influence of several variables on the potentiation produced by subcortical tetanization. These included subcortical lesions, intensity of geniculate and subcortical stimulation, and Nembutal anesthesia. In 15 animals, after determining the effects of pulvinar, reticular and collicular stimulation on single evoked responses and the recovery cycle, a destructive lesion was placed in one of these structures, and the stim-

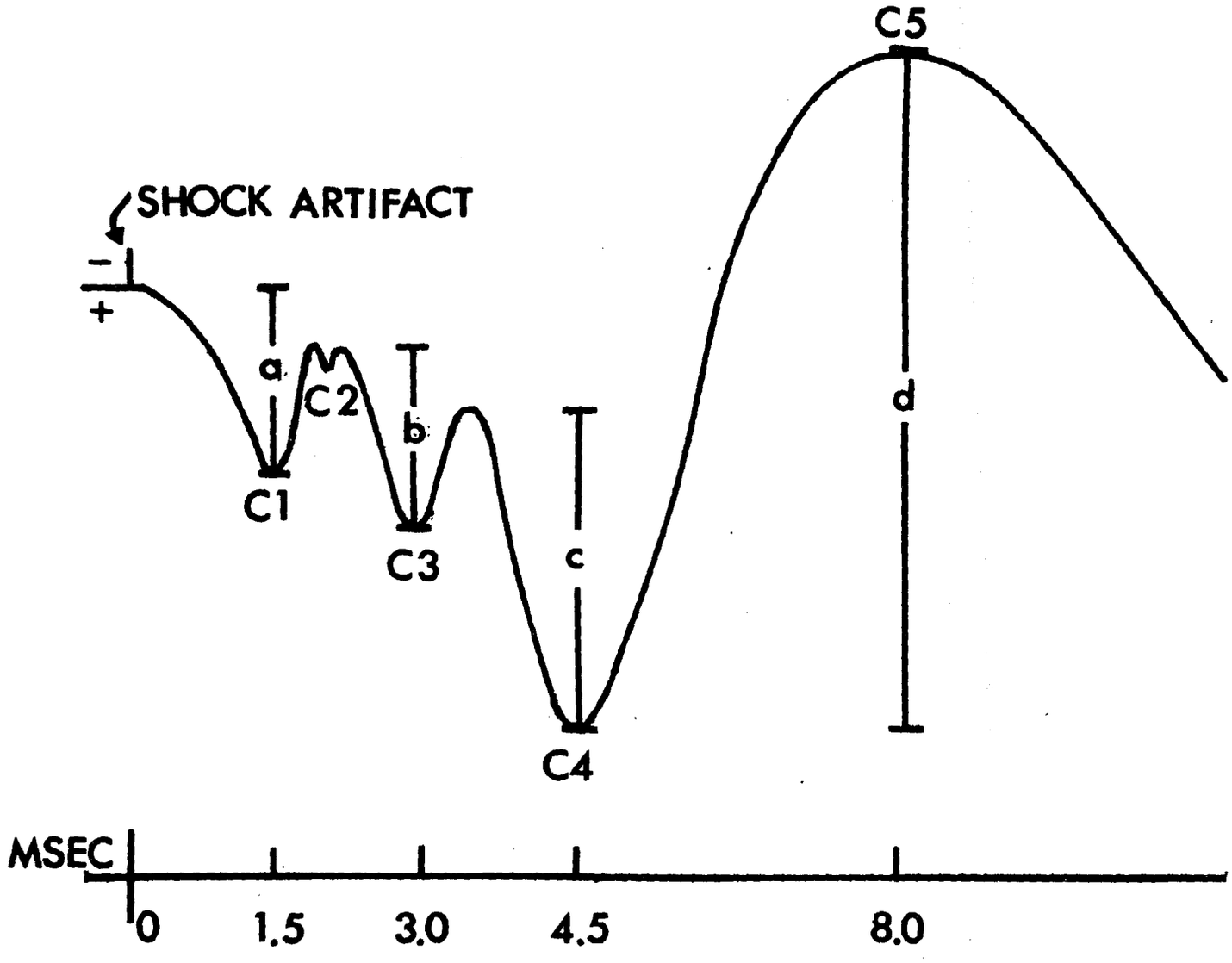
ulation effects of the remaining structures were retested. The lesion was made by passing a DC current, usually of 1 to 6 milliamperes between the ear bars and the stimulating electrodes for 1 to 4 milliamperes, 0.5 milliamperes apart.[2] The effects of stimulating the remaining structure(s) was usually retested 30 to 60 minutes after the lesioning. In investigating the influence of geniculate shock intensity, the voltage level was systematically varied in four or five steps from a threshold intensity to over four times threshold. For each intensity level, the effects of pulvinar, reticular and collicular tetanization were determined. In other experiments, the geniculate shock intensity was maintained constant while reticular, collicular and pulvinar tetanizing intensities were varied. In 5 animals successive dosages of Nembutal were injected intravenously and the effects of subcortical stimulation were investigated at each dosage level. Finally, in five experiments the lateral geniculate body was tetanized with the same parameters used in the stimulation of the other subcortical structures and the effects on single evoked responses at various temporal intervals were observed.

Analysis of data. For each cat, the filmed data of five or more superimposed tracings in response to S_t in every condition were analyzed. In most animals, only peak to peak amplitude was determined and this was then expressed in terms of the percent of the control (S_t

[2]Since the reticular and collicular electrodes were on a common carrier, when the reticular formation was fulgarated a high current (5-6 mamps) was used at only one or two loci to prevent movement of the collicular electrode. In those few instances where a collicular electrode was not used during the experiment, a destructive lesion of the reticular formation was made by using a low current (1-3 mamps) at 4 or more successive loci, each 0.5 millimeter apart. Even though the current was kept constant, the first locus fulgarated resulted in a larger lesion than lower loci (see fig. 23, top).

alone) condition. In other experiments a component analysis of the evoked response was performed. Figure 3 shows a schematic representation of a typical cortical response evoked by a geniculate shock and the criterion used to analyze the amplitude of components 1,3,4, and 5. (Component 2 is usually too small and inconsistent to be reliably measured in this manner). The amplitude of the overall response was measured by determining the peak displacement from the largest positive component (usually C4) to the peak negative (C5) deflection.

Fig. 3 A schematic of the typical shock evoked response on visual cortex of the cat. C1, C2, C3, C4, and C5 denote the five components of the response, while letters a-d indicate the criterion used to measure the amplitude of each component.



LATENCY OF COMPONENTS

RESULTS

Influence of stimulus intensity on response potentiation. Early in the study it was found that the degree of potentiation of the evoked response obtained upon tetanizing subcortical structures varied directly with the intensity of tetanization over a small voltage range. Increasing the intensity level above the upper limits of this range resulted in either no further increases, or in some cases, in a slight decrease in the enhancement of the evoked response. Figure 4 illustrates this finding for reticular tetanization. A similar relationship between intensity of subcortical stimulation and enhancement of cortical responses was found for superior collicular and pulvinar activation. In view of this finding, the intensity of reticular, pulvinar or collicular stimulation was always set at a level just sufficient to produce maximum facilitation. For example, for the animal whose data are shown in figure 4, an intensity level of 4 volts was used to study the effects of reticular tetanization. With stimulation levels determined in the foregoing manner, it was generally found that the intensity necessary for facilitation from reticular formation stimulation was lower then that for superior collicular or pulvinar activation. The mean intensity of reticular stimulation overall animals was 4 volts (range 1.5-8 volts), while 9 (range 3-15) and 11 (range 4-15) were the respective mean intensities for collicular and pulvinar activation.

In addition to intensity of subcortical tetanization, the strength of the test shock was also found to be an important variable in determining the degree of potentiation obtained upon sub-

Fig. 4 Potentiation of a single cortical response at four intensities of reticular tetanization are illustrated by scope tracings and a graphical summary for a single animal. Top trace is the response recorded from the marginal gyrus, while the bottom trace shows supra-sylvian recordings.

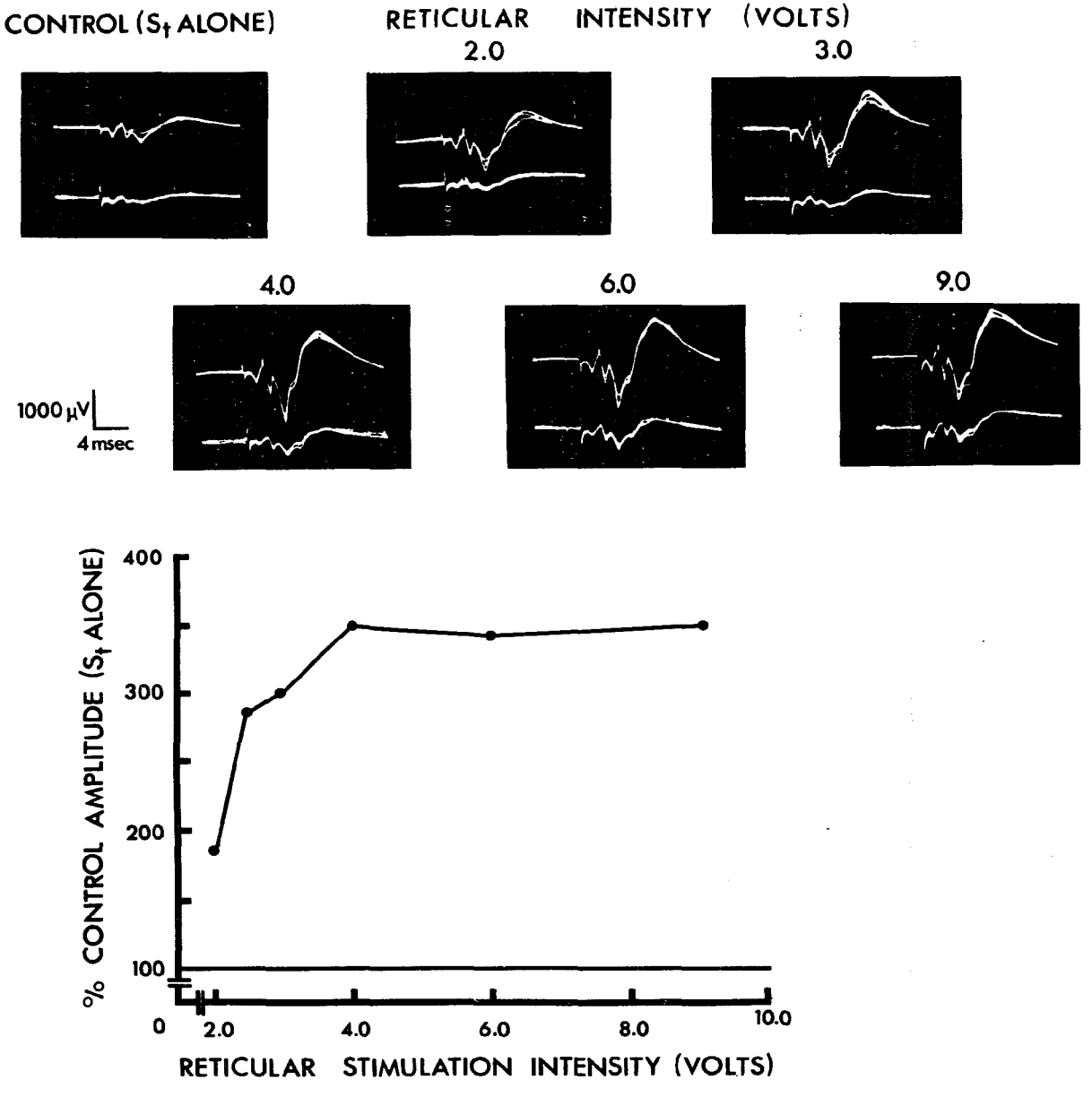
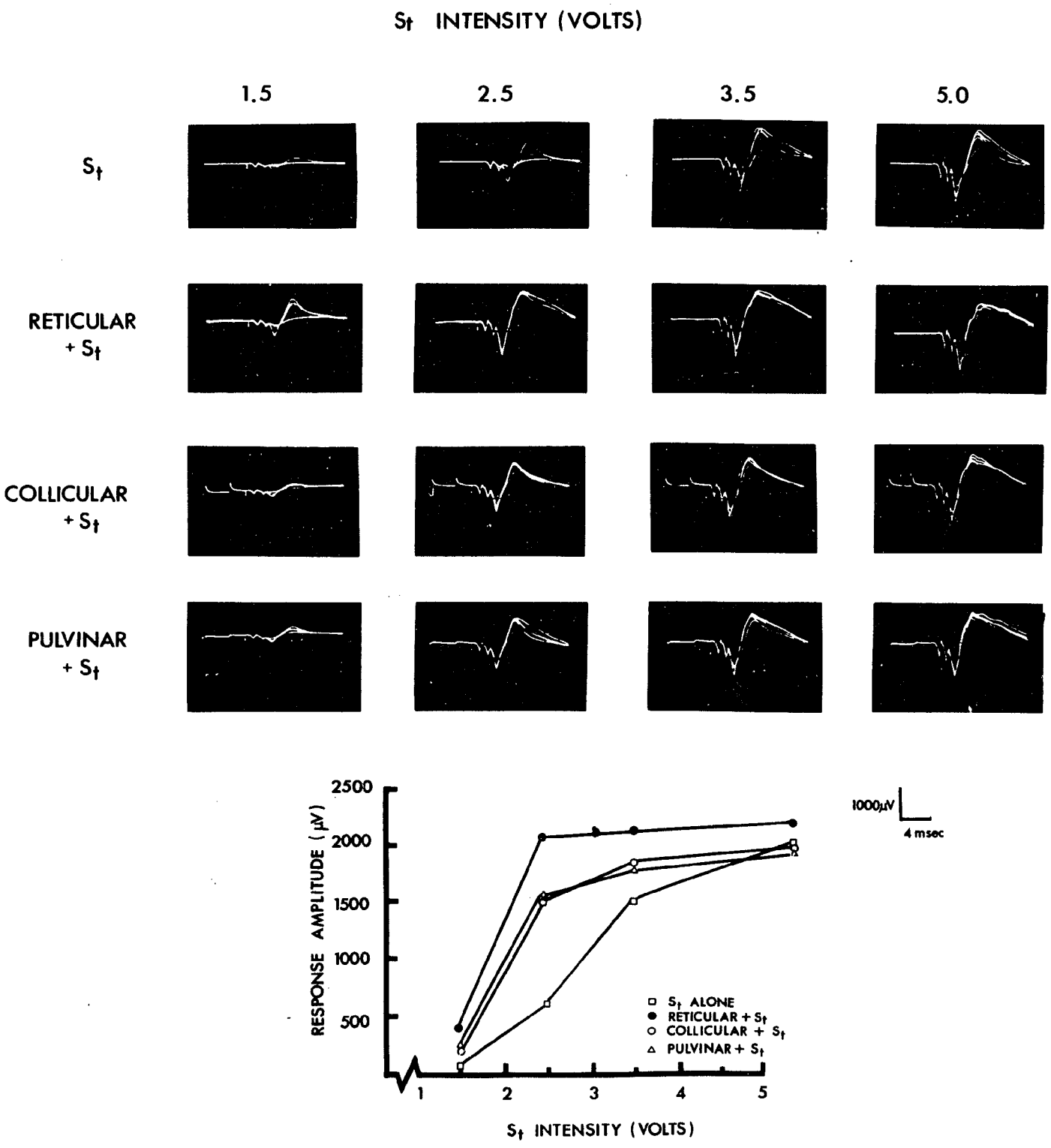

CONTROL (S1 ALONE)
RETICULAR INTENSITY (VOLTS)
2.0
3.0
4.0
6.0
9.0
1000 μV
4 msec
% CONTROL AMPLITUDE (S1 ALONE)
400
300
200
100
0
2.0
4.0
6.0
8.0
10.0
RETICULAR STIMULATION INTENSITY (VOLTS)

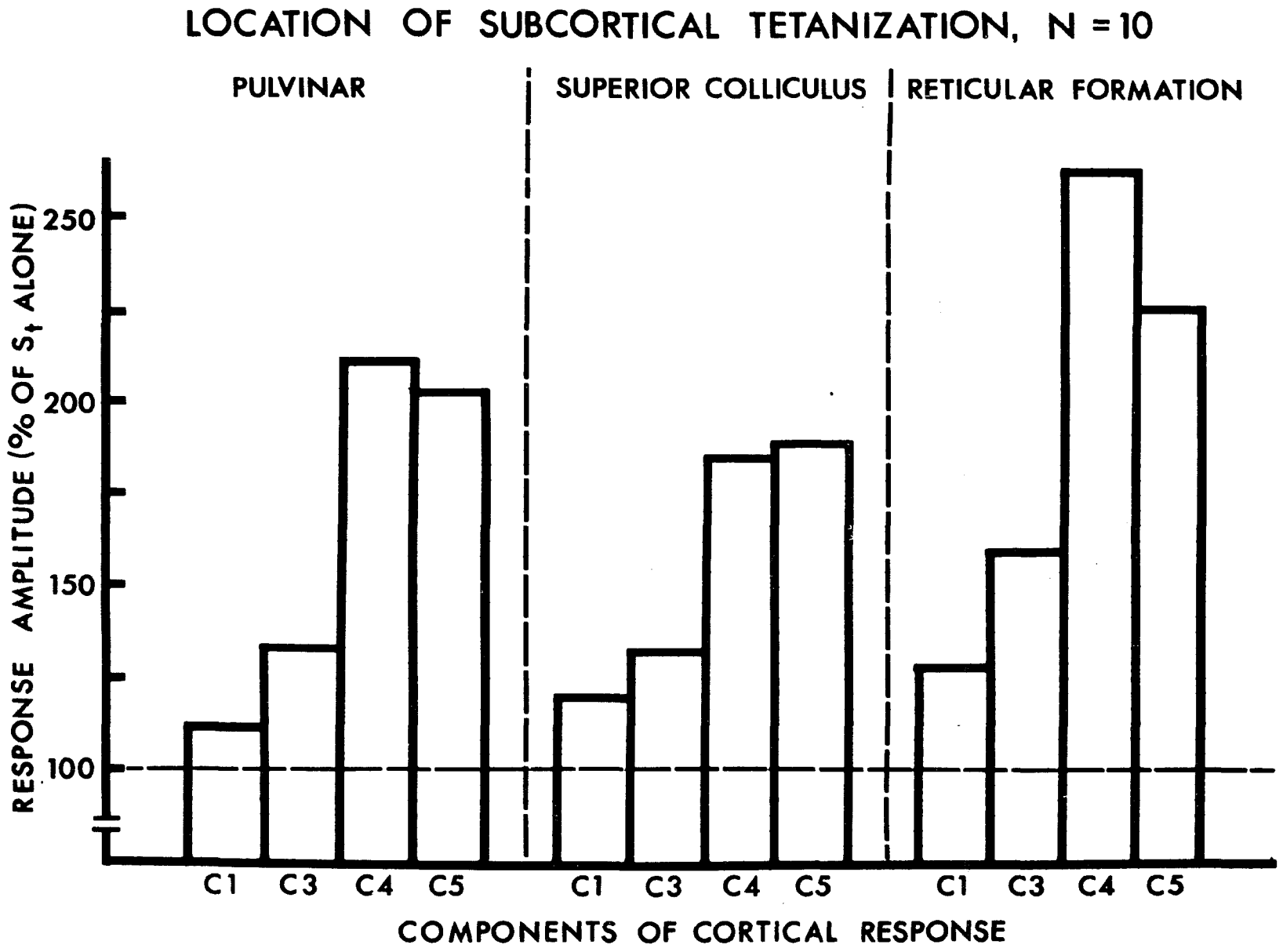
Fig. 5 Effects of reticular, collicular and pulvinar tetanization on a cortical response evoked by four shock intensities ranging from threshold (1.5 volts) to over three times threshold (5.0 volts).


St INTENSITY (VOLTS)
1.5
2.5
3.5
5.0
St
RETICULAR + St
COLLICULAR + St
PULVINAR + St
RESPONSE AMPLITUDE (μV)
2500
2000
1500
1000
500
1
2
3
4
5
St INTENSITY (VOLTS)
St ALONE
RETICULAR + St
COLLICULAR + St
PULVINAR + St
1000μV
4 msec

cortical stimulation. Figure 5 shows the effects of reticular formation, superior collicular and pulvinar stimulation on the cortical responses evoked by four different intensities of geniculate shock which ranged from threshold (1.5 volts) to over three times the threshold intensity (5 volts). Note that the evoked response changes from submaximal to supramaximal levels as the intensity of geniculate shock is increased. In addition, the potentiation elicited by reticular, collicular, or pulvinar stimulation is minimal at threshold, greatly increases at suprathreshold levels, and then either diminishes again or is absent, at the highest level of geniculate shock intensity. Similar data were obtained in four other experiments. As a result of these findings, geniculate stimulation levels of 10 to 20 percent above threshold were consistently used throughout the study; threshold being defined as the lowest stimulation intensity (in volts) which reliably evoked all five components of the response in marginal gyrus.

Effects on components of the geniculate shock response. Figure 6 shows the mean data (N=10 cats) for the effects of pulvinar, collicular, and reticular tetanization on all four components (C1, C3, C4, and C5) of the marginal gyrus response to a geniculate shock ten to twenty percent above threshold. In all cases the delay between the end of subcortical tetanization and geniculate test shock was held constant at zero milliseconds. These data show that activation of all three structures produced the greatest increment in the late components (C4 and C5) normally attributed to cortical origins (Bishop and Clare, 1951, 1952; Chang and Kaada, 1950; Malis and Kruger, 1956). The fact that reticular

Fig. 6 Mean potentiation (N=10) of components C1, C3, C4, and C5 elicited by pulvinar, collicular, and reticular formation tetanization.


LOCATION OF SUBCORTICAL TETANIZATION, N = 10
PULVINAR
SUPERIOR COLLICULUS
RETICULAR FORMATION
RESPONSE AMPLITUDE (% OF S1 ALONE)
250
200
150
100
C1 C3 C4 C5
C1 C3 C4 C5
C1 C3 C4 C5
COMPONENTS OF CORTICAL RESPONSE

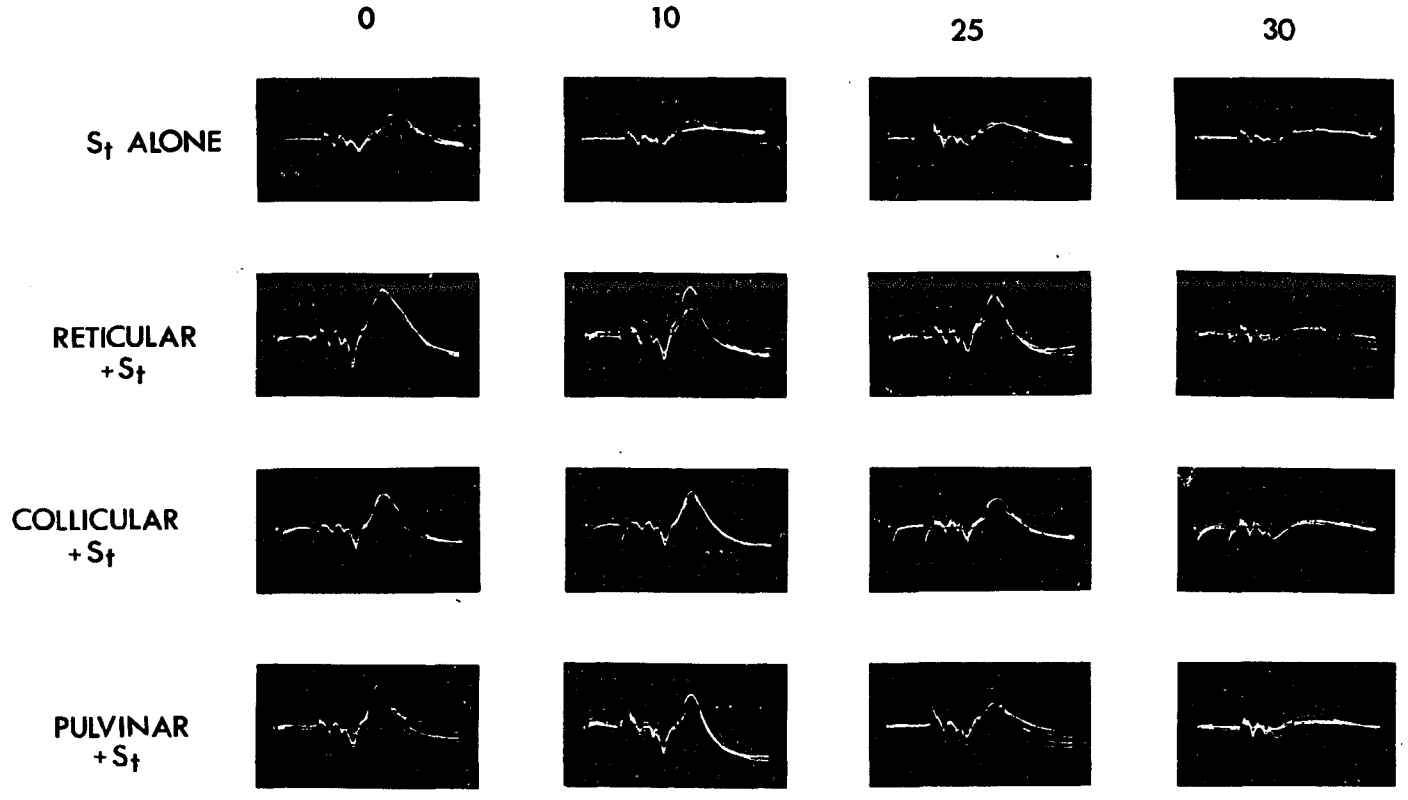
stimulation primarily enhances these cortical components has been noted previously (Bremer and Stoupel, 1959; Dumont and Dell, 1960; Nakai and Domino, 1968; Redding, 1967; Steriade and Demetrescu, 1960, 1967). To date, however, there have been no studies showing that pulvinar or collicular stimulation can affect the various components of the shock response in an analogous fashion. Although, it should be noted that collicular tetanization enhanced both cortical components to an equal extent, while pulvinar and reticular activation potentiated C4 to a greater degree than C5.

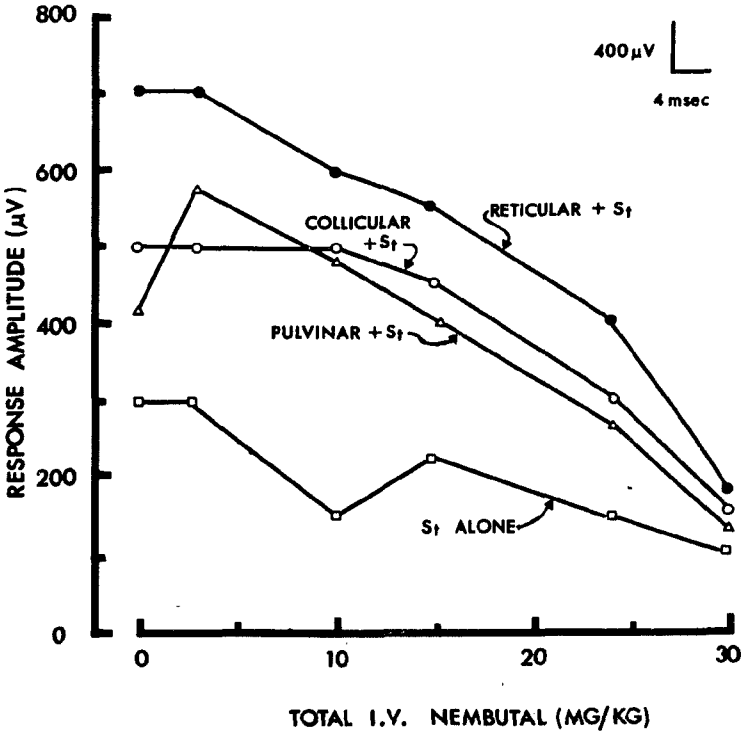
Effects of anesthesia on response potentiation. It is well known that many anesthetic agents, particularly barbituates, can affect the functioning of the reticular formation (Bremer and Stoupel, 1959; Long, 1959; King, Naquet, and Magoun, 1957). No prior studies, however, have determined whether the effects obtained upon pulvinar or collicular stimulation are also influenced by the administration of barbituates. Accordingly, small doses of sodium pentobarital (Nembutal) were administered intravenously in 5 animals, and the effects of pulvinar, collicular, and reticular stimulation on the cortical response to geniculate shock were then observed. For an illustrative animal, figure 7 shows that increasing doses of Nembutal (in mg/kg of body weight) produced decreasing potentiation of the evoked response following tetanization of all three structures. At the highest dosage level, only minimal potentiation was observed in the animal shown in figure 7; in other preparations no enhancement was observed at all. At the higher Nembutal levels the cortical evoked response to the geniculate shock alone (top

Fig. 7 Influence of successive doses of Nembutal anesthesia (mg/kg) on the potentiation of the cortical response elicited by reticular, collicular, and pulvinar stimulation.

CAT 258

TOTAL I. V. NEMBUTAL (MG/KG)





row of figure 7) was markedly modified. In particular, as noted by prior studies (Storck and Battersby, 1970; Schoolman and Evarts, 1959), two types of changes were observed. First, the evoked response amplitude was less variable in the deeply anesthetized state, and secondly, there was a decrease in the amplitude of the response.

Temporal aspects of response potentiation. The data thus far presented utilized a zero millisecond delay between the end of subcortical stimulation and the onset of the geniculate test shock. In most experiments, however, this delay was systematically varied from 0 to at least 150 milliseconds (in some instances up to 500 milliseconds), and the peak to peak amplitude of the cortical response plotted as a function of the delay interval (figure 8). Points plotted are the means, the height of the bars $\pm$ 1 SD, for the magnitude of response potentiation, following reticular (N=35), pulvinar (N=18), and collicular (N=22) stimulation. Every delay was not studied in every animal, but all data points are based on a minimum N of 6. These data show that stimulation of the reticular formation (top of figure 8) and pulvinar (bottom) produced a monotonic decreasing function between test shock response amplitude and temporal interval. Superior collicular stimulation (middle of figure 8), however, potentiated the cortical response up to a maximum at a test shock delay of 60 milliseconds, this was followed by a gradual decrease of the potentiation effect. At every delay interval the potentiation produced by reticular stimulation was greater or equal to that produced by tetanization of the other structures, even though, as previously noted, the lowest voltage level was used for reticular activation. Conversely, although the pulvinar was generally stimulated with the high-

Fig. 8 Mean potentiation of a single evoked response by reticular (N=35), collicular (N=22), and pulvinar (N=18) tetanization as a function of the S_t delay. The bars indicate 1 standard deviation above and below each mean.

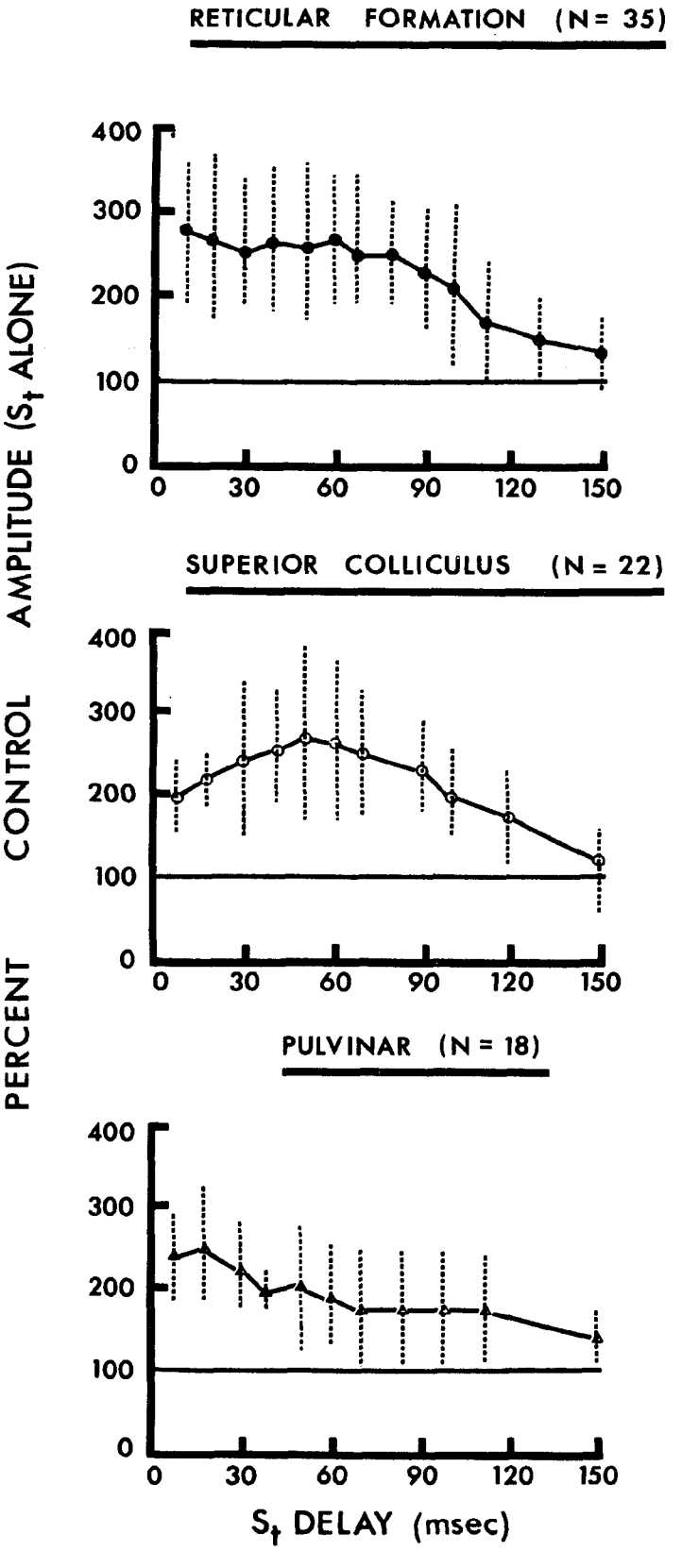
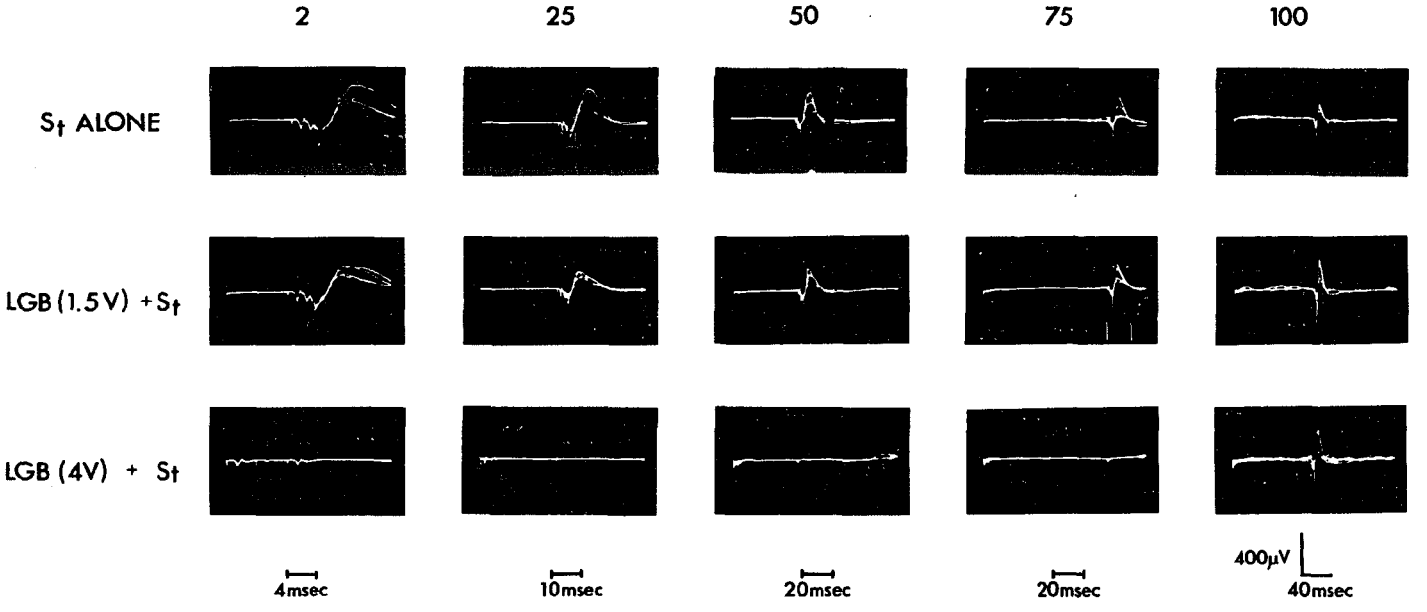

RETICULAR FORMATION (N = 35)
SUPERIOR COLLICULUS (N = 22)
PULVINAR (N = 18)
PERCENT CONTROL AMPLITUDE (S_t ALONE)
400
300
200
100
0
0
30
60
90
120
150
S_t DELAY (msec)

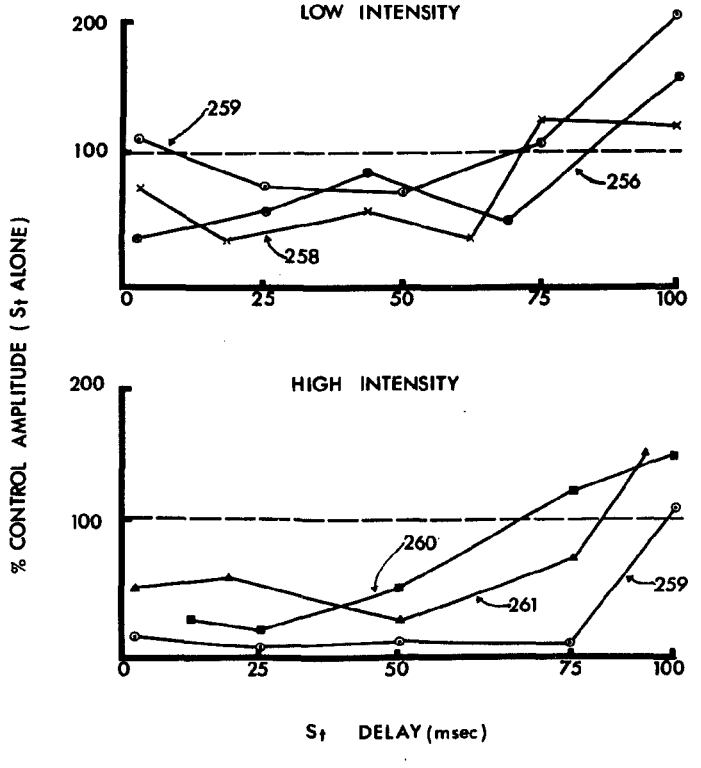
Fig. 9 The tracings illustrate the effects of tetanizing the lateral geniculate body at low (1.5 volts) and high (4 volts) intensity levels on a cortical evoked response at various temporal intervals. These data, and the results from four other cats (each identified by a number) are shown in graphic form.

CAT 259

POST TETANIC POTENTIATION

S_t DELAY (msec)





est intensity level, pulvinar stimulation resulted in the smallest potentiation of the evoked response at all test shock delays less than 100 milliseconds. At longer test shock delays, pulvinar tetanization effects were slightly greater than those produced by collicular stimulation and equal to the reticular potentiation.

It is well known (Hughes, 1958) that an increase in cortical responsiveness can be obtained following tetanization of the lateral geniculate body; i.e. post-tetanic potentiation. It was a necessary control, therefore, to compare the facilitation in amplitude of the evoked response elicited by activation of the reticular formation, superior colliculus and the pulvinar, with that which could be produced by tetanizing the lateral geniculate body itself. Figure 9 presents illustrative tracings and individual data for five animals (indicated by numbers) in terms of amplitude change in the cortical response following tetanization of the geniculate at two intensity levels. (The parameters of stimulation were identical with those used for pulvinar, collicular, and reticular stimulation.) Confirming earlier reports (Hughes, Marshall, and Evarts, 1956) at both low and high voltage levels, a depression of the cortical response is initially apparent, followed by a potentiation of the evoked response at test shock delays of 60 to 100 milliseconds. This excitability function is markedly different from that produced by reticular, pulvinar and collicular stimulation, since the slopes are in the opposite direction. Hence, it does not appear likely that the potentiating effects described in preceding

sections can be attributed to a direct spread of current to the optic tract, lateral geniculate body or radiation fibers.

Potentiation of recovery cycle to dual geniculate shocks. Figures 10, 11, and 12 illustrate for one typical animal the effects of tetanizing the reticular formation, pulvinar, or superior colliculus on the cortical responses to dual geniculate shocks; i.e. temporal resolution at cortex. In each figure, illustrative tracings are shown at the top and a graphical summary at the bottom. Three curves are presented; one shows the amplitude of the response to S_t as a function of the delay from S_c (the recovery cycle); a second illustrates the potentiation of the recovery cycle by tetanization of the reticular formation (figure 10), the superior colliculus (figure 11), or the pulvinar (figure 12). Still a third curve illustrates the magnitude of potentiation, elicited by stimulation of these same structures, of the cortical response to a single geniculate shock. For all curves, response amplitude is expressed as a percent of the control (S_t alone) response.

Facilitation of the recovery cycle was observed in 32 of 38 animals upon activation of the mesencephalic reticular formation. Superior collicular stimulation produced potentiation of the recovery cycle in 21 of 25 animals, and pulvinar stimulation affected the recovery cycle in 11 of 20 cats. In all cases, the recovery cycle was considered to be facilitated if subcortical tetanization produced an increase in the response to S_t when preceded by S_c (not necessarily an amplitude greater

Fig. 10 Effects of reticular tetanization on the recovery cycle and single evoked responses (at 4 temporal intervals) are illustrated by the tracings at the top for one typical animal. Below, these data and additional S_t delays are summarized graphically.

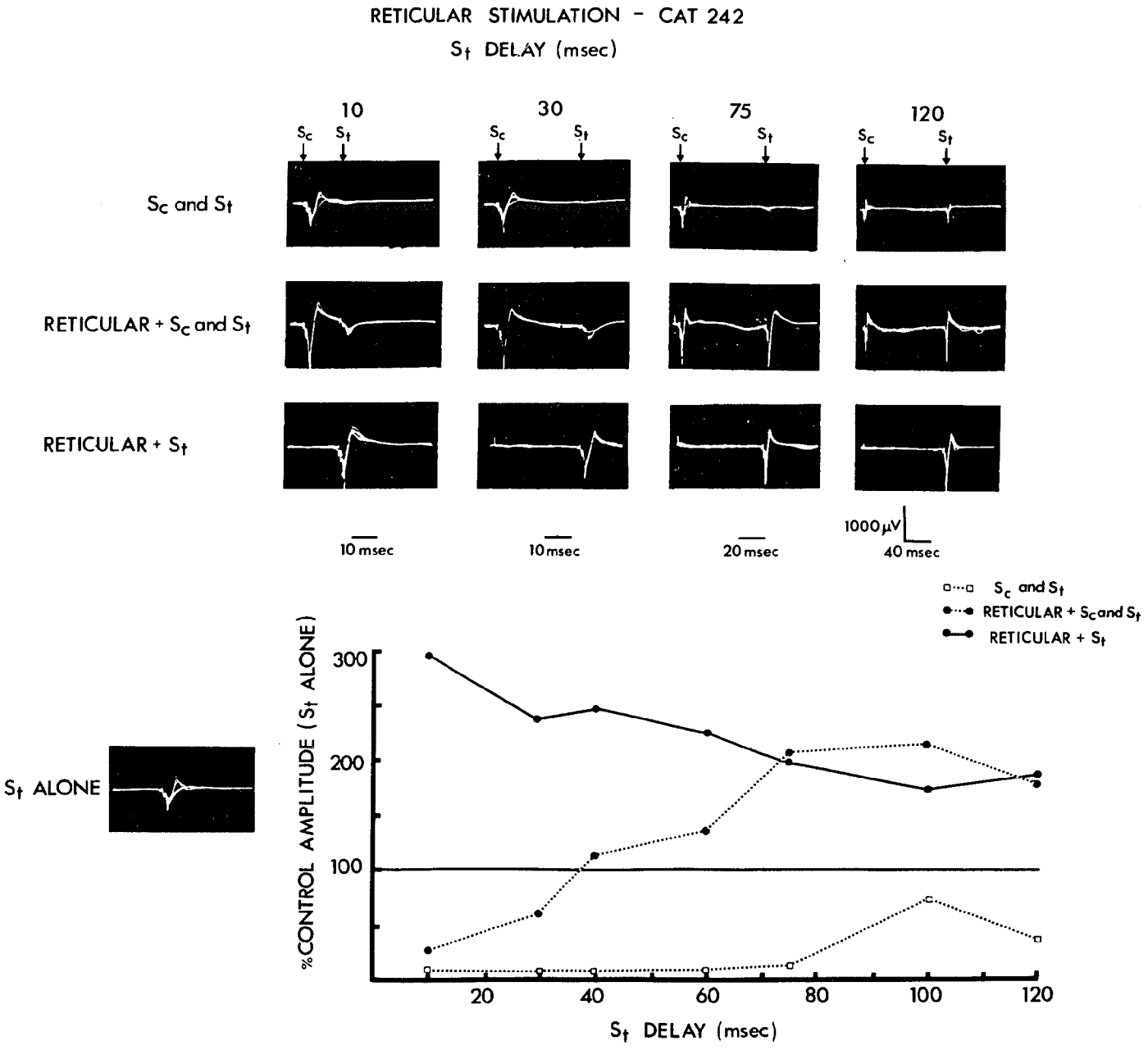

RETICULAR STIMULATION - CAT 242
St DELAY (msec)
10
30
75
120
Sc
St
Sc and St
RETICULAR + Sc and St
RETICULAR + St
10 msec
10 msec
20 msec
1000 μV
40 msec
St ALONE
Sc and St
RETICULAR + Sc and St
RETICULAR + St
% CONTROL AMPLITUDE (St ALONE)
300
200
100
20
40
60
80
100
120
St DELAY (msec)

Fig. 11. Effects of collicular tetanization on the recovery cycle and single evoked responses. Same preparation as in fig. 10.

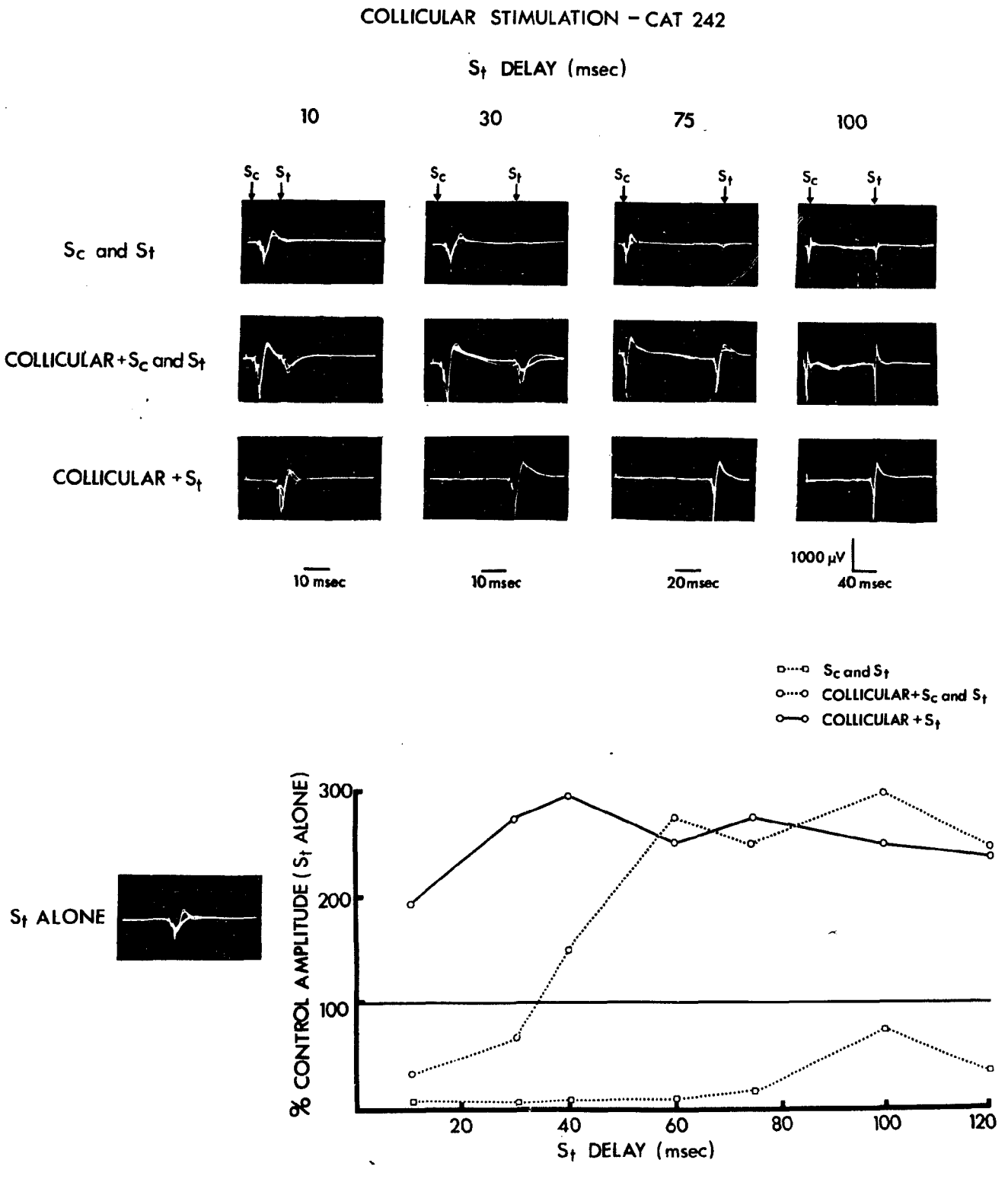
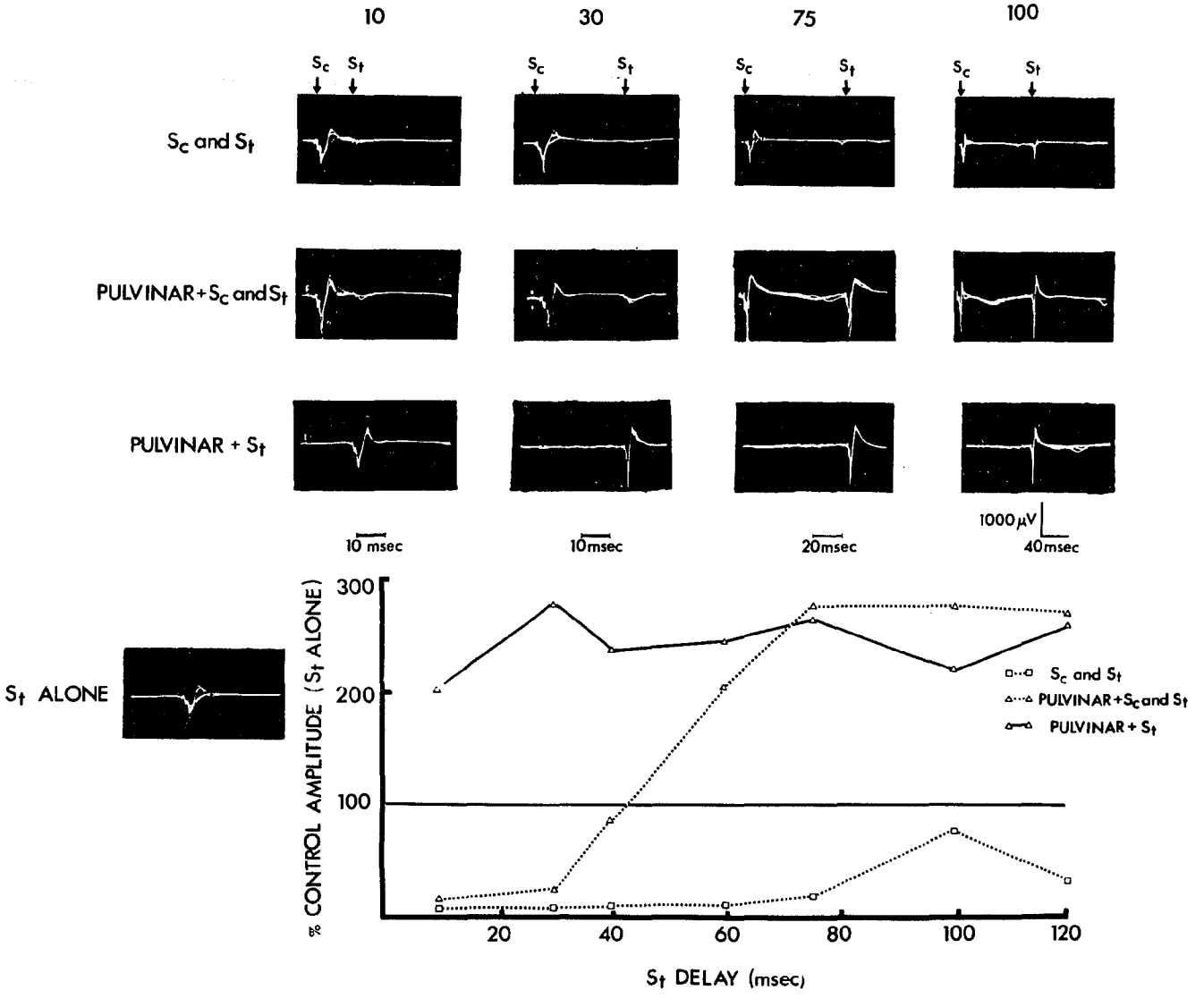

COLLICULAR STIMULATION – CAT 242
St DELAY (msec)
10
30
75
100
Sc and St
COLLICULAR + Sc and St
COLLICULAR + St
Sc
St
10 msec
10 msec
20 msec
1000 μV
40 msec
Sc and St
COLLICULAR + Sc and St
COLLICULAR + St
St ALONE
% CONTROL AMPLITUDE (St ALONE)
300
200
100
20
40
60
80
100
120
St DELAY (msec)

Fig. 12 Effects of pulvinar tetanization on the recovery cycle and single evoked response. Same preparation as in figs. 10 and 11.

PULVINAR STIMULATION – CAT 242



than the S_t alone value). In all animals, stimulation of the subcortical structures produced potentiation of single evoked responses. Thus, facilitation of single evoked responses was more readily obtained than potentiation of the recovery cycle; stimulation of the pulvinar being least effective in potentiating the recovery cycle.

The magnitude of the effects of subcortical tetanization on the recovery cycle can be quantified by subtracting for each individual animal, the response amplitude (in percent of control) to S_t when preceded by S_c, from the S_t response when preceded by both S_c and subcortical tetanization; i.e., the ordinate differences between two of the curves shown in figures 10, 11, and 12. The recovery cycle potentiation was thus calculated for all S_c-S_t delays, for each experiment in which subcortical tetanization did produce recovery cycle potentiation (see appendix). Table 1 shows the average magnitude and standard deviation of recovery cycle potentiation produced by reticular, pulvinar or collicular stimulation. Each mean is based on data obtained from at least six experiments.

The data of these few animals (N=5) in which stimulation of all three subcortical structures potentiated the recovery cycle were used in order to evaluate most precisely the magnitude and time course of the potentiation (figure 13). It may be readily seen, that stimulation of each structure produced only minimal effects on the recovery cycle at S_c-S_t intervals of 0 to 20 milliseconds. This was followed by a marked enhancement of the recovery cycle for the next 50 to 60 milliseconds, and then a gradual decline in the degree of potentiation is

TABLE ONE

Mean Magnitude and Standard Deviation of Recovery Cycle Potentiation Elicited by Subcortical Tetanization

Reticular Formation															
S_t Delay	5	10	20	30	40	50	60	70	80	90	100	110	120	130	150
$\bar{X}$	60	31	71	106	128	92	128	145	112	76	103	93	127	64	103
S.D.	88	81	69	77	91	89	76	87	90	39	88	73	76	87	77
Pulvinar															
$\bar{X}$	59	18	29	52	78	96	72	91	70	--	59	70	43	62	15
S.D.	76	68	35	59	72	74	54	64	68		46	62	40	53	12
Superior Colliculus															
$\bar{X}$	--	25	51	86	137	118	113	115	160	138	87	--	92	--	59
S.D.	--	69	59	100	139	110	80	50	68	41	42	--	56	--	53

evident. As shown in figure 13, collicular tetanization resulted in the greatest potentiation of the recovery cycle at all S_t delays, while stimulation of the pulvinar produced the least potentiation. In similar fashion, figure 14 presents the mean effects of those more numerous experiments in which stimulation of any two subcortical structures resulted in recovery cycle potentiation. Thus, figure 14 compares the stimulation effects of the superior colliculus and pulvinar (N=7), reticular formation and pulvinar (N=9), and reticular formation and superior colliculus (N=12). Collicular and reticular activation still produce greater potentiation of the recovery cycle than pulvinar stimulation. However, when more animals are included in the analysis, differences between reticular and collicular effects are substantially reduced, although the peak potentiation elicited by stimulation of the colliculus is still greater than that which is obtained by reticular tetanization. Furthermore, it should be noted, that although the magnitude of the potentiation elicited by tetanization of each subcortical structure exhibits considerable variability at any given S_c-S_t interval, the general time course of the recovery cycle potentiation remains relatively constant.

While most of the data in the present study were obtained from marginal gyrus recordings, in several animals similar potentiation was recorded from the suprasylvian gyrus. This was especially evident in Cat 245, where stimulation of the ventral portion of the lateral geniculate body produced responses of greater amplitude at the supra-

Fig. 13 Comparison of the mean recovery cycle potentiation (N=5) elicited by collicular, reticular, and pulvinar tetanization. The ordinate indicates the difference (expressed in percent of S_t alone) between the recovery cycle and the changes elicited in this function by subcortical tetanization. In each experiment stimulation of all three subcortical loci resulted in recovery cycle potentiation.

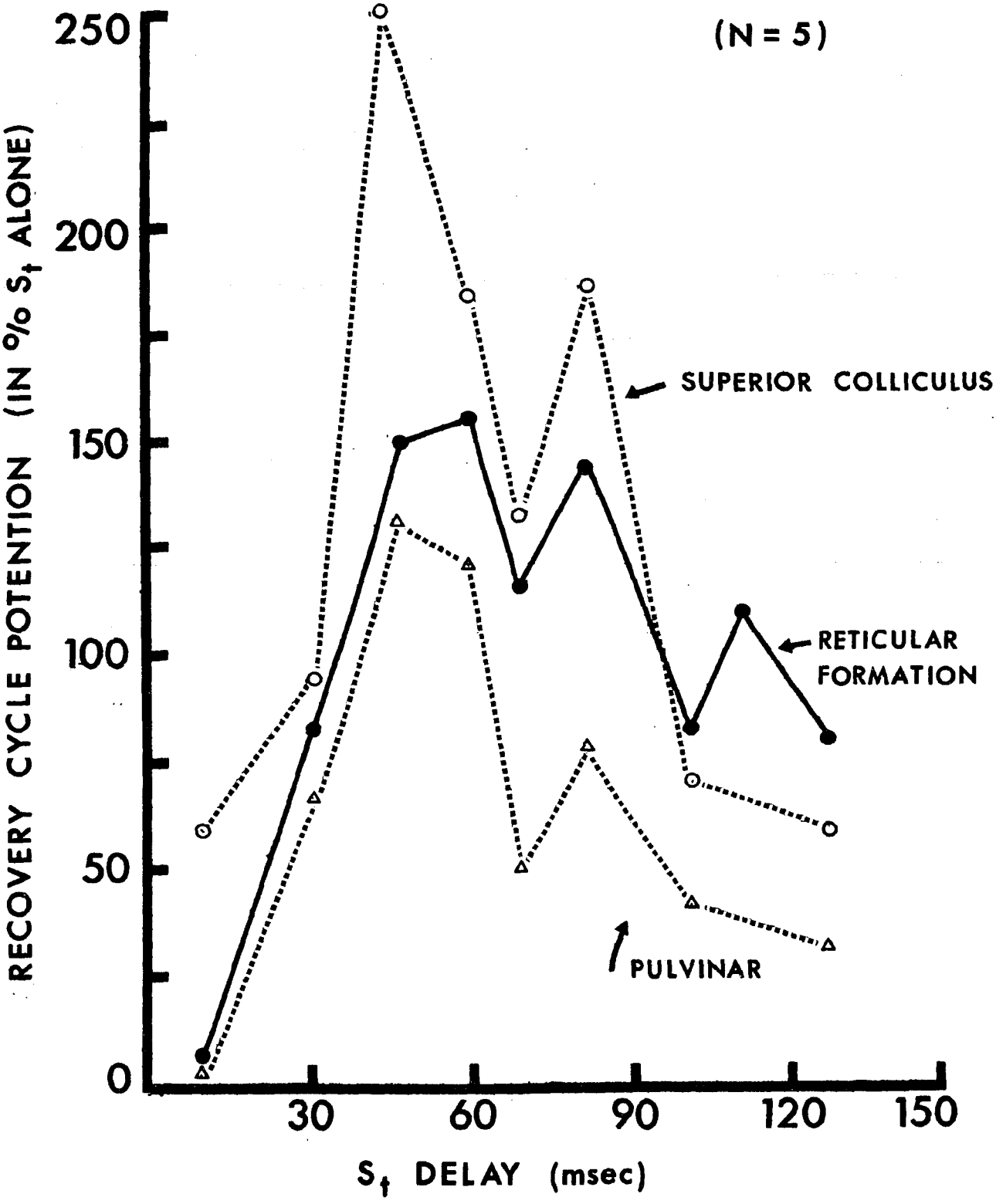

(N = 5)
RECOVERY CYCLE POTENTION (IN % S_t ALONE)
250
200
150
100
50
0
SUPERIOR COLLICULUS
RETICULAR FORMATION
PULVINAR
30
60
90
120
150
S_t DELAY (msec)

Fig. 14 Comparison of mean recovery cycle potentiation elicited by stimulation of pulvinar and reticular formation (N=10), top; pulvinar and colliculus (N=7), middle; and colliculus and reticular formation (N=12), bottom. In each comparison, stimulation of both structures resulted in potentiation of the recovery cycle in a single experiment.

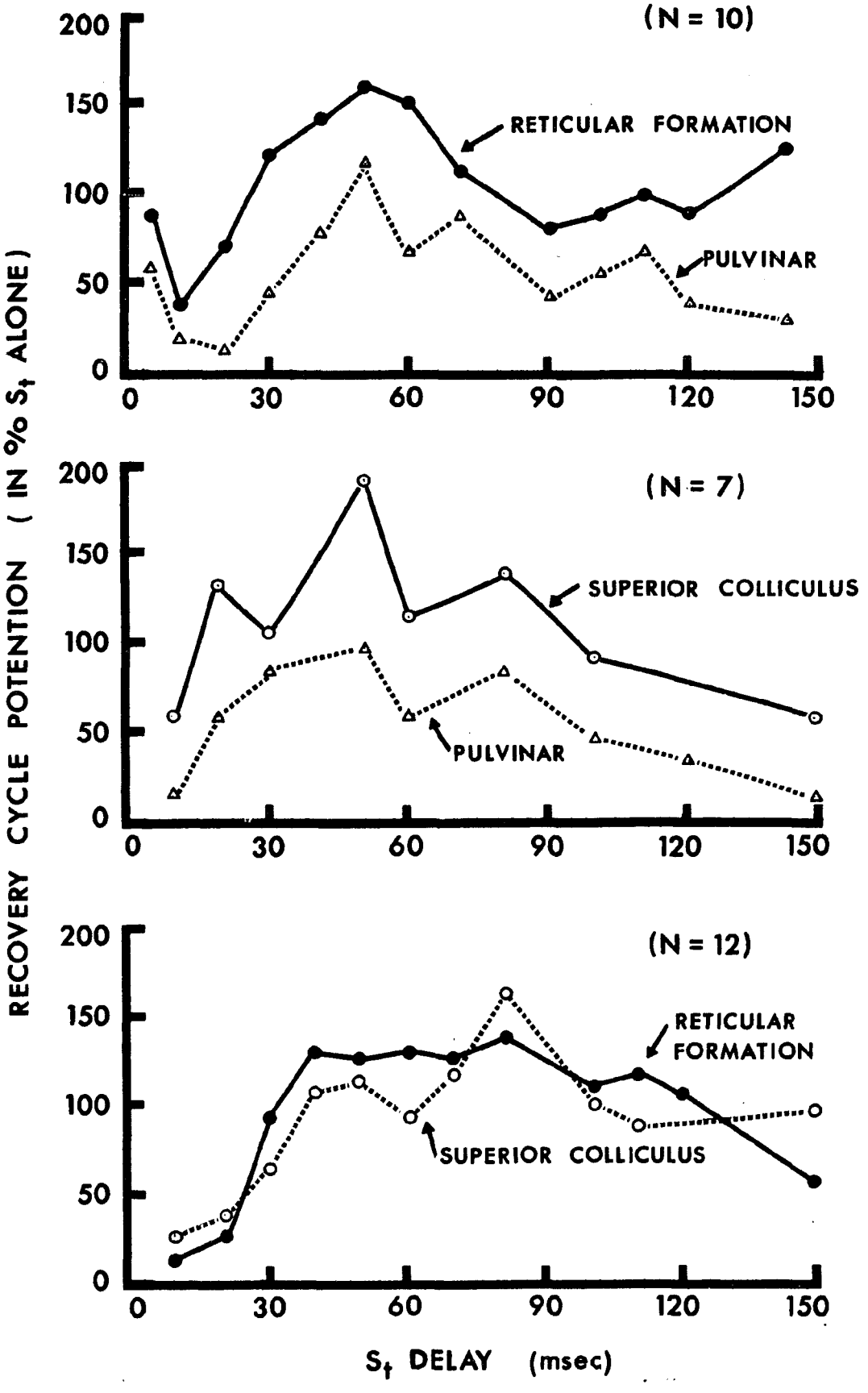
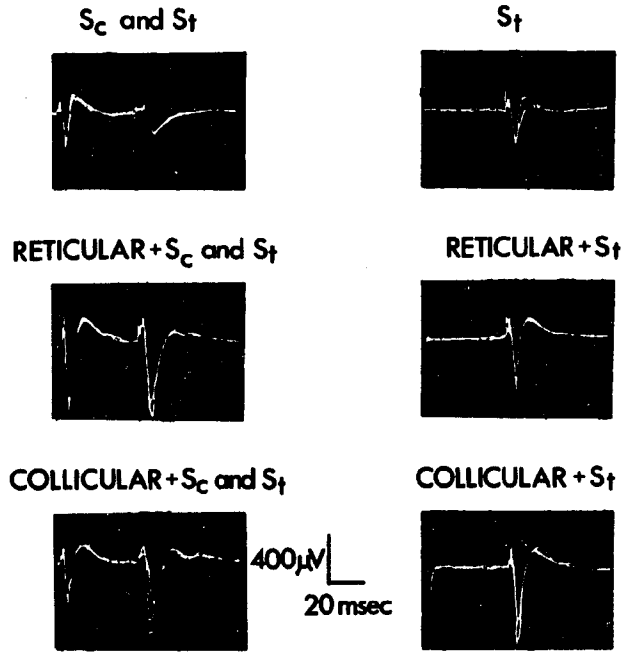
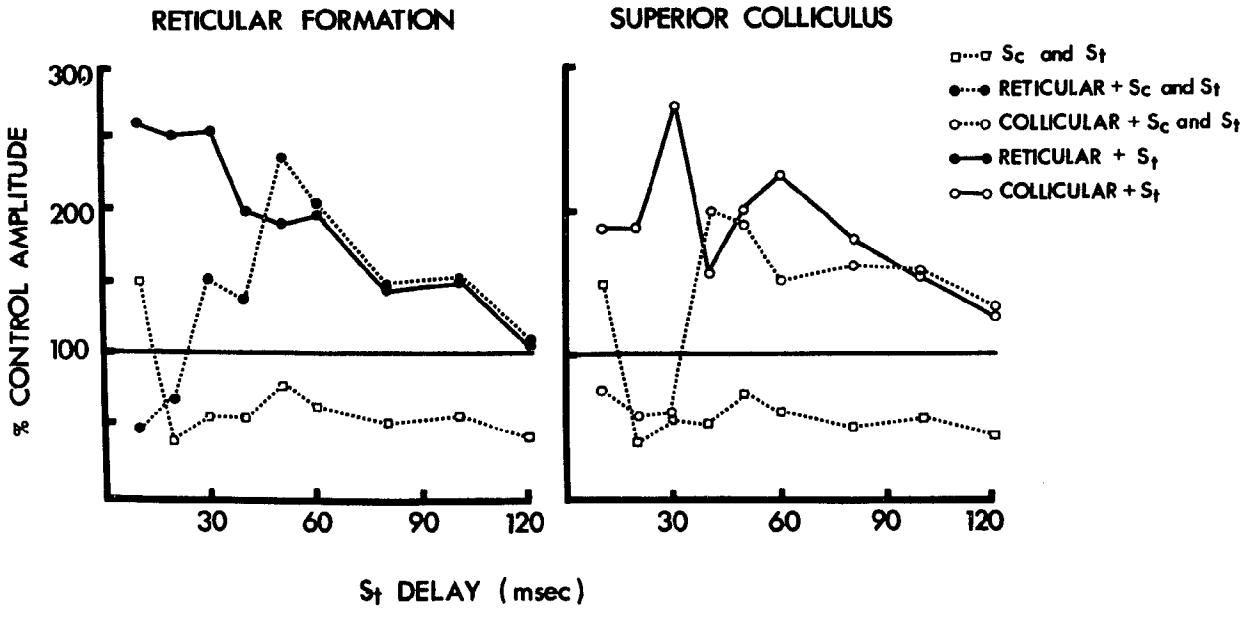

(N = 10)
RETICULAR FORMATION
PULVINAR
(N = 7)
SUPERIOR COLLICULUS
PULVINAR
(N = 12)
RETICULAR FORMATION
SUPERIOR COLLICULUS
200
150
100
50
0
0
30
60
90
120
150
RECOVERY CYCLE POTENTION (IN % S_t ALONE)
S_t DELAY (msec)

Fig. 15 Potentiation of the recovery cycle and single evoked responses recorded at the suprasylvian gyrus by collicular and reticular tetanization. Presented at the top are representative traces from this experiment.

SUPRASYLVIAN RECORDING – CAT 245





sylvian than at the lateral gyrus.[3] Figure 15 shows that both reticular formation and superior collicular activation in this preparation enhanced single evoked responses and the recovery cycle at the suprasylvian gyrus in the same way as were responses recorded at the marginal gyrus in other preparations. Generally however, geniculate stimuli evoked suprasylvian responses which were much smaller than those in the marginal gyrus (see figure 5 for comparison).

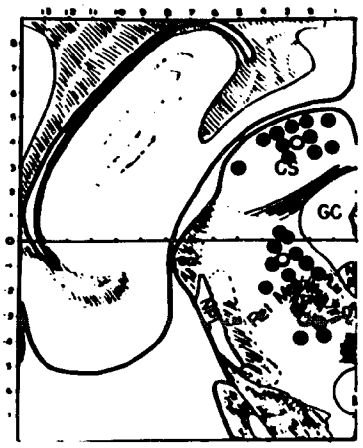
Factors influencing magnitude of recovery cycle potentiation. As already noted, recovery cycle potentiation was elicited less frequently by subcortical tetanization than potentiation of single evoked responses. Figure 16 indicates the loci in the reticular formation, superior colliculus, and the pulvinar which when stimulated either facilitated (closed circles) or failed to facilitate (open circles) the recovery cycle. Stimulation of all of these loci, however, produced enhancement of the single evoked response. In general, there is no evident anatomical differentiation in the reticular formation and the superior colliculus of the areas which produce enhancement of the recovery cycle from those that fail to do so. In the pulvinar, however, there is a tendency to obtain facilitation of the recovery cycle by stimulating the dorsal rather than the ventral parts of the pulvinar-lateral posterior complex, but there is definite overlap and no firm conclusion can be arrived at.

[3]Bishop and Clare (1955) and more recently Nariskavili et. al. (1969) have shown that projections from the ventral laminar (B layer) of the cat lateral geniculate terminate in the suprasylvian gyrus.

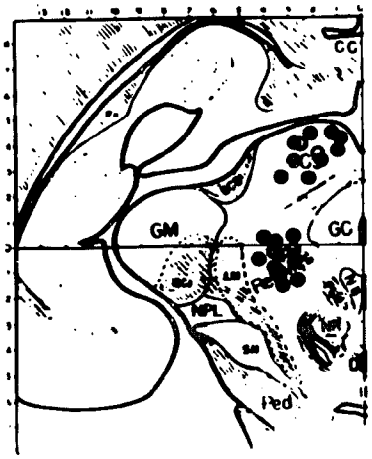
Fig. 16 Serial coronal sections from the cat brain atlas of Jasper and Ajmone-Marsan (1954) showing positive loci in the pulvinar, colliculus and reticular formation (closed circles), stimulation of which resulted in recovery cycle potentiation, and negative loci (open circles) stimulation of which failed to potentiate the recovery cycle.

POTENTIATION OF RECOVERY CYCLE

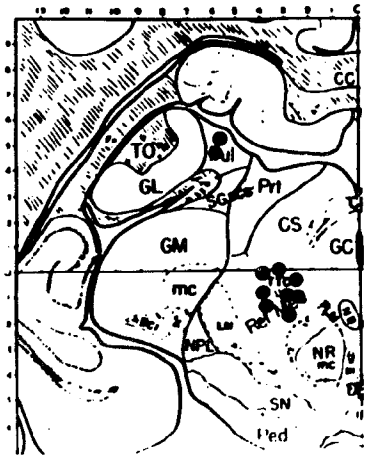
FR. 2.0



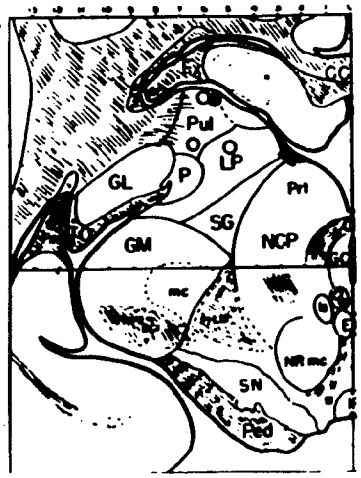
FR. 3.0



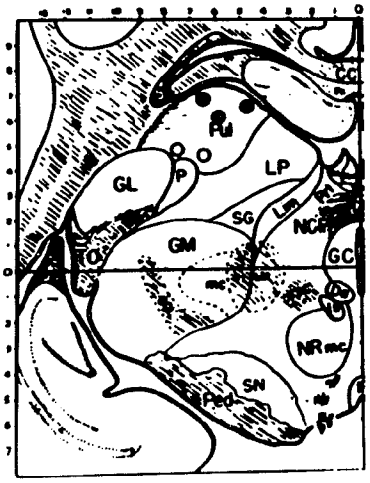
FR. 4.0



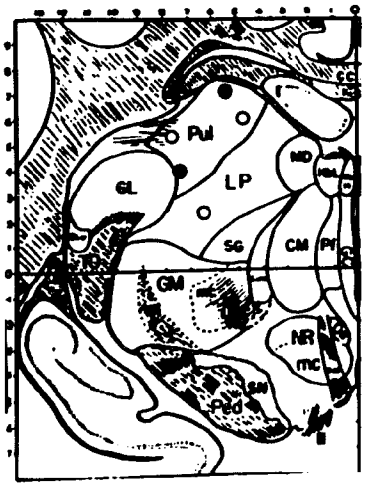
FR. 5.0



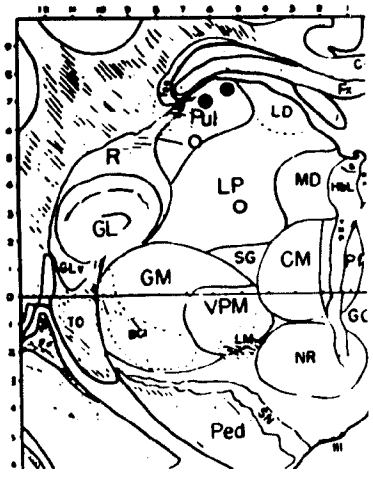
FR. 6.0



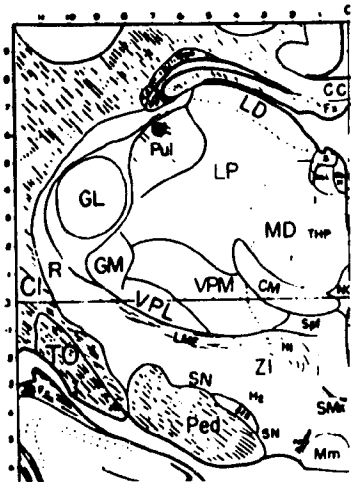
FR. 6.5



FR. 7.0



FR. 8.0



● POSITIVE LOCI

○ NEGATIVE LOCI

5 mm

In the six experiments where stimulation of the reticular formation failed to enhance the recovery cycle, the pupils of these animals appeared to be more dilated than those of animals in which potentiation of the recovery cycle could be demonstrated. In addition, in these six animals, tetanization of the superior colliculus or of the pulvinar also failed to produce an effect on the recovery cycle. The extent of pupillary dilation has been reported to be indicative of an animal's arousal level (Naquet, Regis, Fischer- Williams, and Fernandez-Guardida, 1960), and it is possible that the failure to obtain enhancement of the recovery cycle by stimulation of subcortical structures, may be due, in part, to the constant high arousal level of these animals during the experiment (Schwartz and Shagass, 1963). Such an explanation, however, would not hold for those instances in which pulvinar tetanization failed to enhance the recovery cycle, since in the majority of these experiments stimulation of the superior colliculus and/or the reticular formation did produce recovery cycle potentiation.

Although single evoked response enhancement was more frequently elicited by subcortical tetanization than recovery cycle potentiation, it was generally found, that when the latter effect was obtained it was equal or greater in magnitude than the facilitation of single evoked responses. (This occurred usually for S_c-S_t intervals longer than 30 milliseconds, see figures 10, 11, and 12). Thus, potentiation of the recovery cycle was greater or equal to that of the single evoked response in 26 of 32 experiments with reticular, 16 of 21 with collicular, and 8 of 11 with pulvinar tetanization. Detailed study in 3 animals showed, however, that a higher intensity of stimulation was necessary to enhance

the recovery cycle than the single evoked response. (The relationship between the intensity of subcortical stimulation and the facilitation of the single evoked response have been previously discussed, see figure 4). In these experiments, the tetanization intensity was progressively increased resulting in increases of the recovery cycle potentiation, while no further enhancement of the single evoked response was produced by the increased stimulation intensities. This is shown in figure 17, where the effects on single evoked responses and the recovery cycle were compared at three reticular stimulation intensities. Figure 18 presents the evoked responses of 3 animals where similar observations were recorded with reticular, collicular and pulvinar stimulation. In those experiments where pulvinar tetanization produced no potentiation of the recovery cycle, it was found, as shown in figure 19, that increasing pulvinar stimulation to higher levels still resulted in no recovery cycle potentiation. As indicated in figure 16, these loci were usually in the ventral portions of the pulvinar-lateral posterior complex.

In three experiments the recovery cycle was selectively potentiated by subcortical tetanization, in that, minimal or no facilitation of single evoked responses was elicited by such stimulation. This finding is illustrated for reticular stimulation in figure 20, while similar data were obtained by tetanizing the colliculus and pulvinar in other preparations.

Most data in the present study were obtained by using a zero millisecond delay between the offset of subcortical tetanization and the onset of the first geniculate shock (S_c delay). In a few experiments this delay was varied $\pm$ 50 milliseconds in order to determine

Fig. 17 Changes in recovery cycle potentiation as a function of reticular tetanization intensity (in volts). Note, that at the same time no significant changes were found in single response potentiation.

CAT 232
RETICULAR INTENSITY (VOLTS)

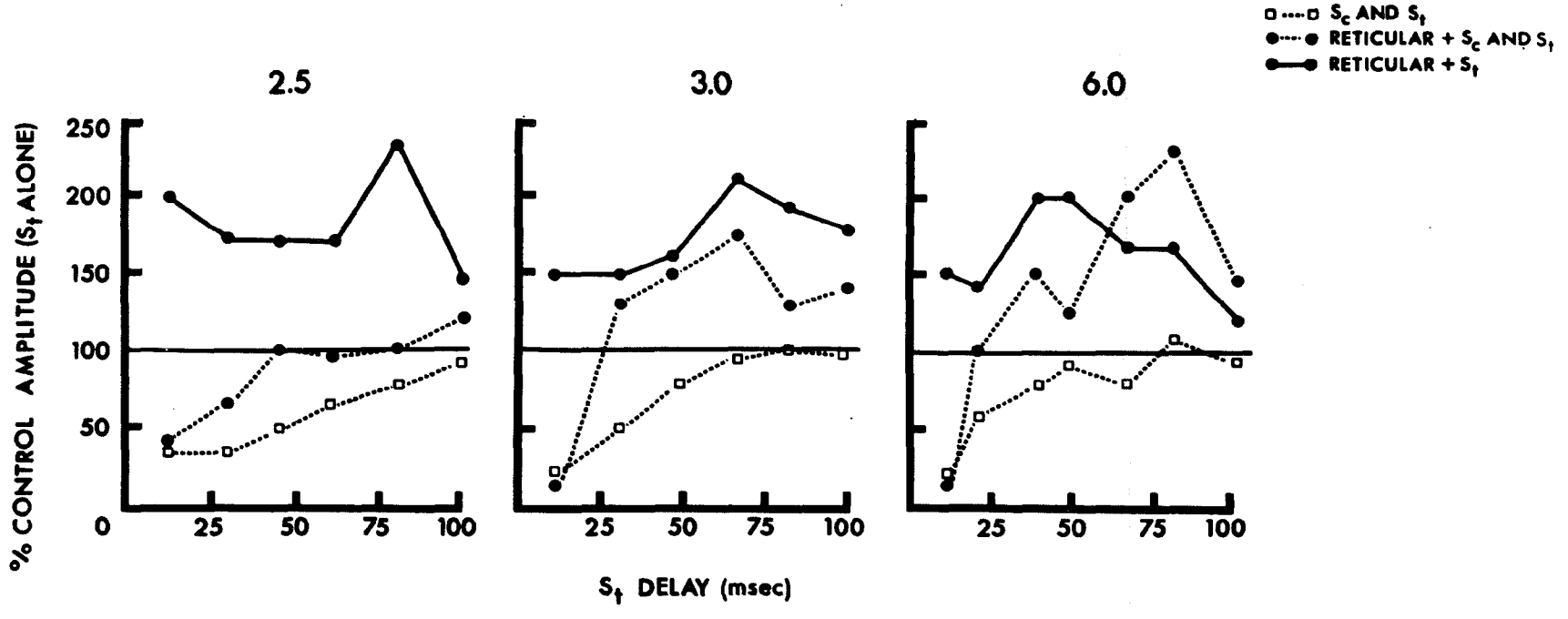


Fig. 18 Tracings from three experiments in which increasing the intensity of reticular (Cat 260), collicular (Cat 250), or pulvinar (Cat 253) tetanization resulted in increased potentiation of the recovery cycle, but minimal effects on single response potentiation.

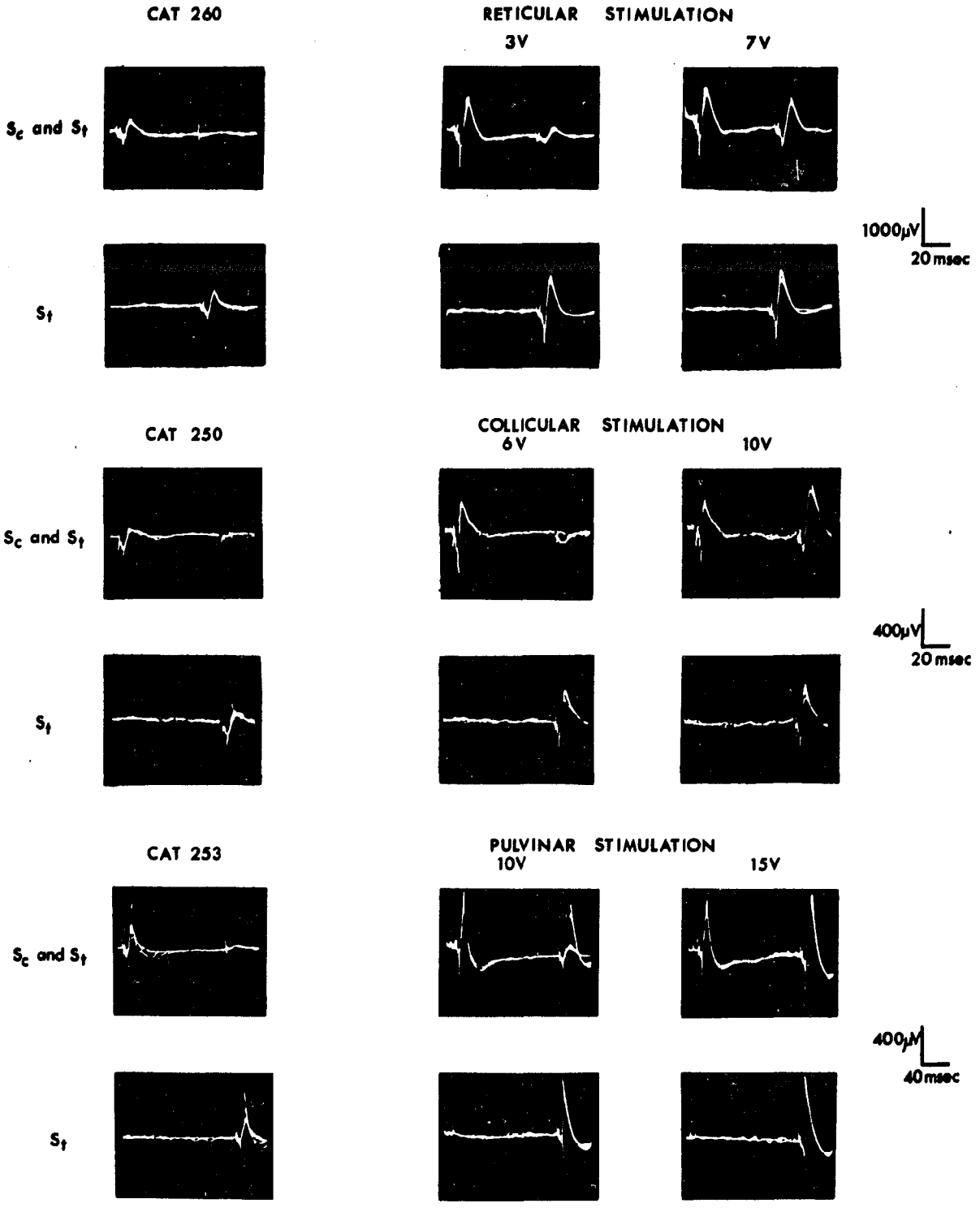

CAT 260
RETICULAR STIMULATION
3V
7V
S_c and S_t
S_t
1000μV
20 msec
CAT 250
COLLICULAR STIMULATION
6V
10V
S_c and S_t
S_t
400μV
20 msec
CAT 253
PULVINAR STIMULATION
10V
15V
S_c and S_t
S_t
400μV
40 msec

Fig. 19 In this preparation (Cat 233), tetanizing the pulvinar with 7.5 volts (left), or 15.0 volts (right) produced no effects on the recovery cycle.

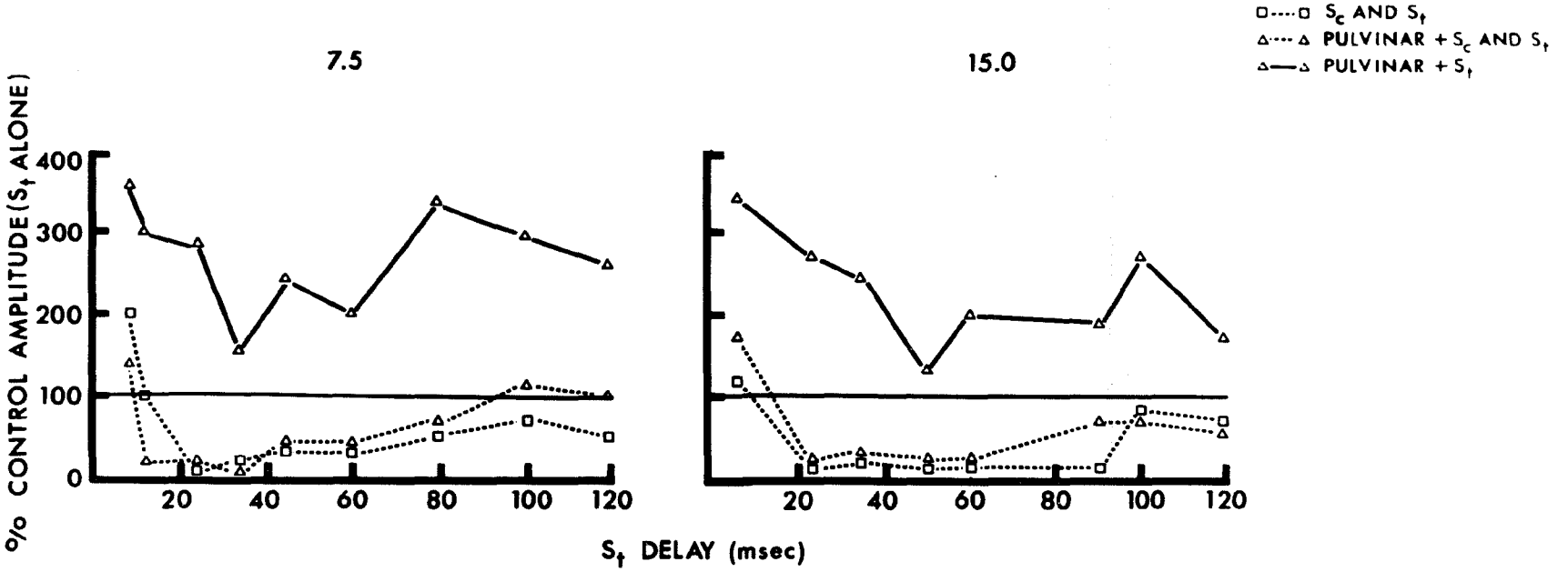
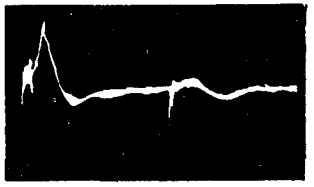

CAT 233
PULVINAR INTENSITY (VOLTS)
7.5
15.0
□----□ S_c AND S_t
△---△ PULVINAR + S_c AND S_t
△—△ PULVINAR + S_t
% CONTROL AMPLITUDE (S_t ALONE)
400
300
200
100
0
20
40
60
80
100
120
S_t DELAY (msec)

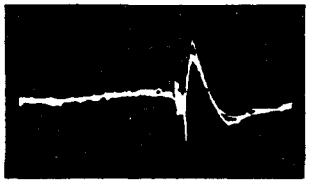
Fig. 20 Tetanizing the reticular formation in this experiment (Cat 255) resulted in minimal facilitation of the single evoked response while the recovery cycle was significantly potentiated.

RETICULAR STIMULATION – CAT 255

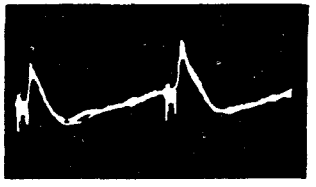
S_c AND S_t



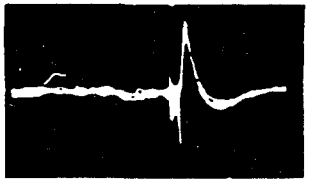
S_t



RETICULAR + S_c AND S_t



RETICULAR + S_t



400μV
20 msec

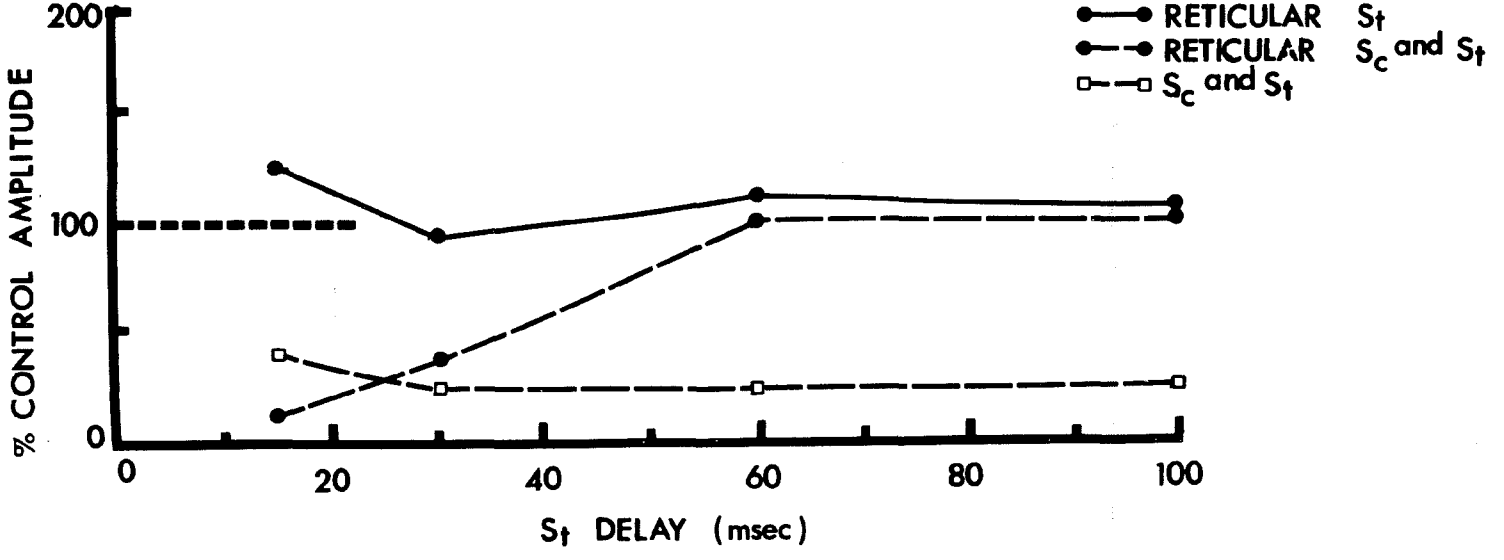
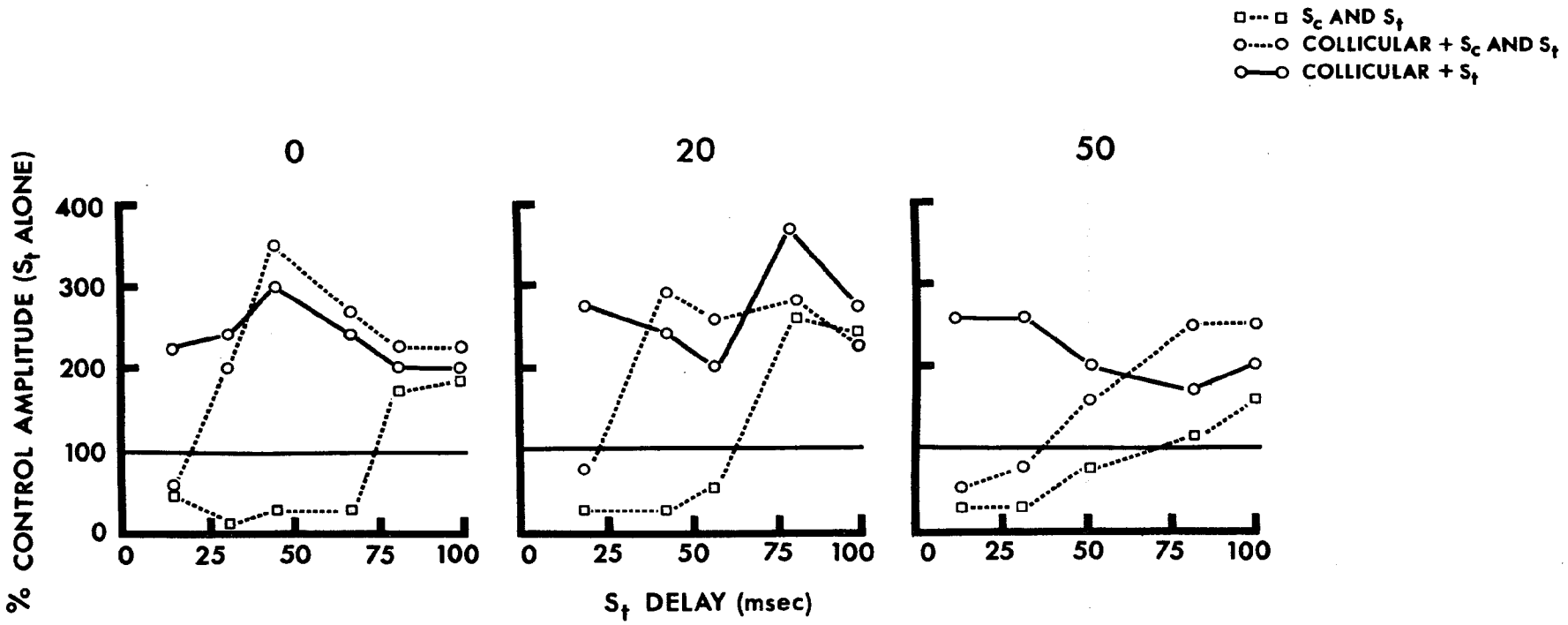


Fig. 21 Single evoked responses and recovery cycle potentiation elicited by collicular tetanization are shown at three S_t delays (interval between the end of subcortical tetanization and onset of the first geniculate shock).

CAT 237

COLLICULAR STIMULATION — S_c DELAY (msec)



if this variable was important in obtaining recovery cycle potentiation. Data in figure 21 are presented only for superior collicular stimulation, and show that the facilitation of the recovery cycle was not substantially changed at three S_c delay settings (0, 20, and 50 milliseconds). Reticular stimulation also showed no evident changes when the S_c delay was varied within these limits, while pulvinar stimulation was not systematically investigated.

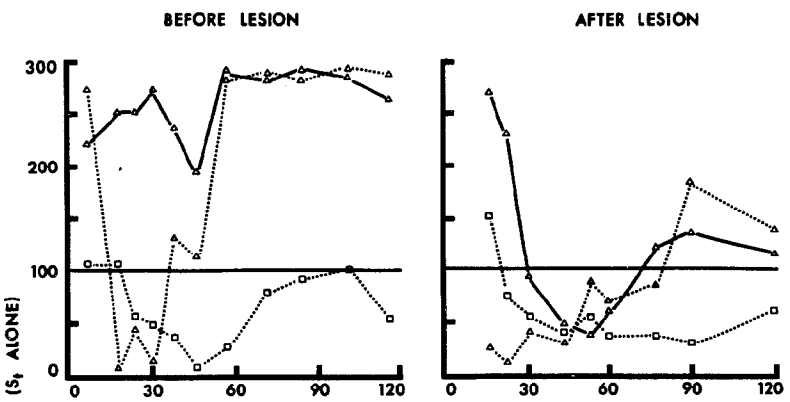
Influence of subcortical lesions on potentiation effects. The results of the present study indicate that stimulation of the reticular formation, pulvinar, and superior colliculus can affect cortical responsivity in similar ways, suggesting that these structures may form a functional system. If this be true (see Literature Review), then it should be possible to disrupt the functional integrity of this system by an appropriately placed lesion in one of these structures. Hence, in fifteen animals, after determining the effects of subcortical stimulation on cortical responsivity, a lesion was made in one of the structures studied, and the effects of stimulating the remaining structure(s) was then retested.

The reticular formation was electrolytically lesioned in twelve animals (5 milliamperes for 20 to 30 seconds at 1 to 4 successive loci spaced 0.5 milliamperes apart). In eight of these, the effects of pulvinar stimulation on cortical responsivity were evaluated. In 5 of these 8, an alteration in the effects of pulvinar activation was found following such lesions as indicated at the top of figure 22. Note that the time course of recovery cycle and single evoked response potentiation

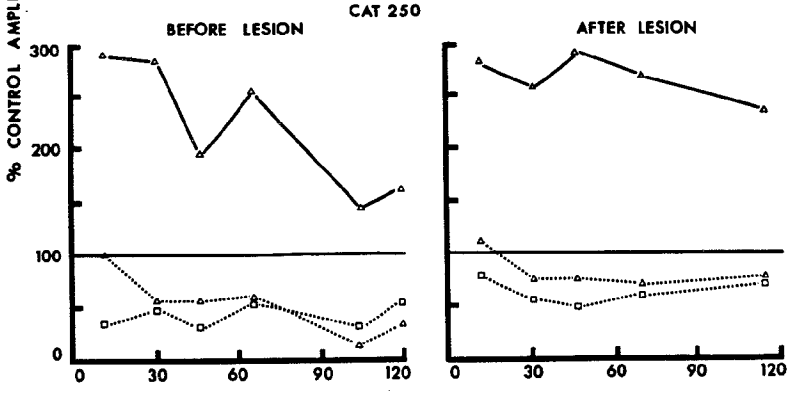
Fig. 22 At the left are the results of two experiments showing the effects of pulvinar tetanization on single evoked responses and the recovery cycle before and after lesions of the mesencephalic reticular formation. On the right are the accompanying photomicrographs indicating the locus and extent of subcortical lesions (outlined by stripped tape).

EFFECTS OF RETICULAR LESIONS ON PULVINAR STIMULATION

CAT 228



CAT 250



% CONTROL AMPLITUDE (S_t ALONE)

S_t DELAY (msec)

□----□ S_c AND S_t
△----△ PULVINAR + S_c AND S_t
△—△ PULVINAR + S_t

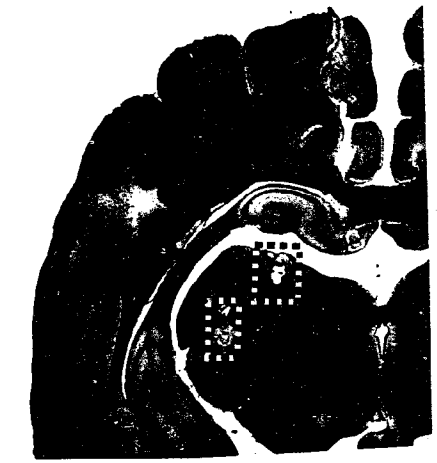
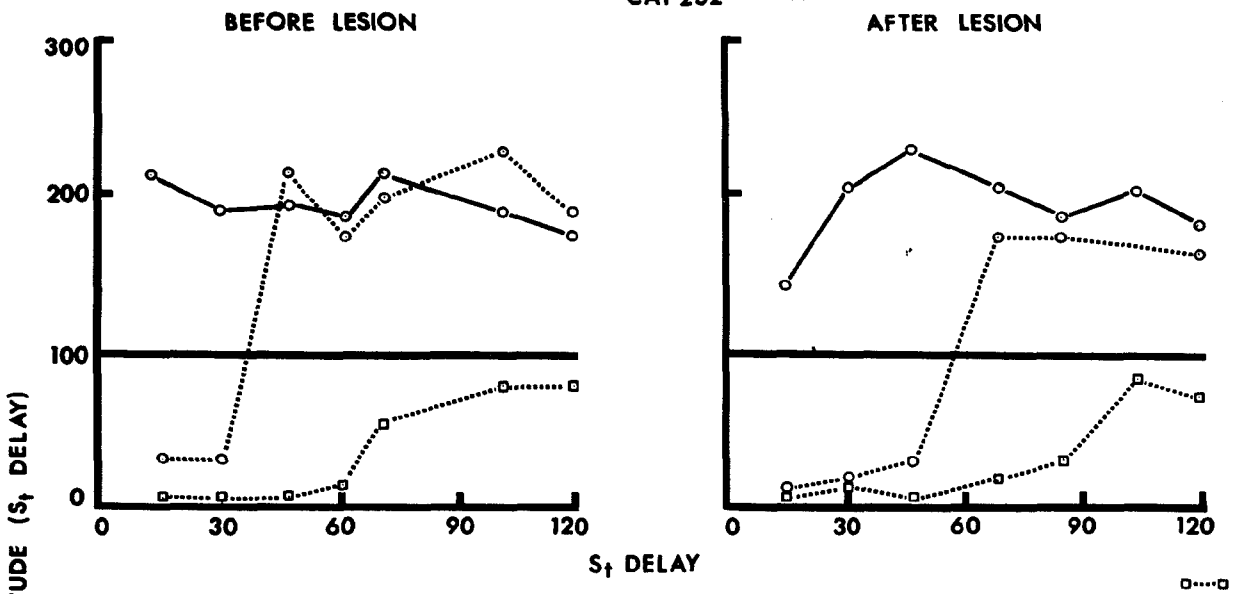


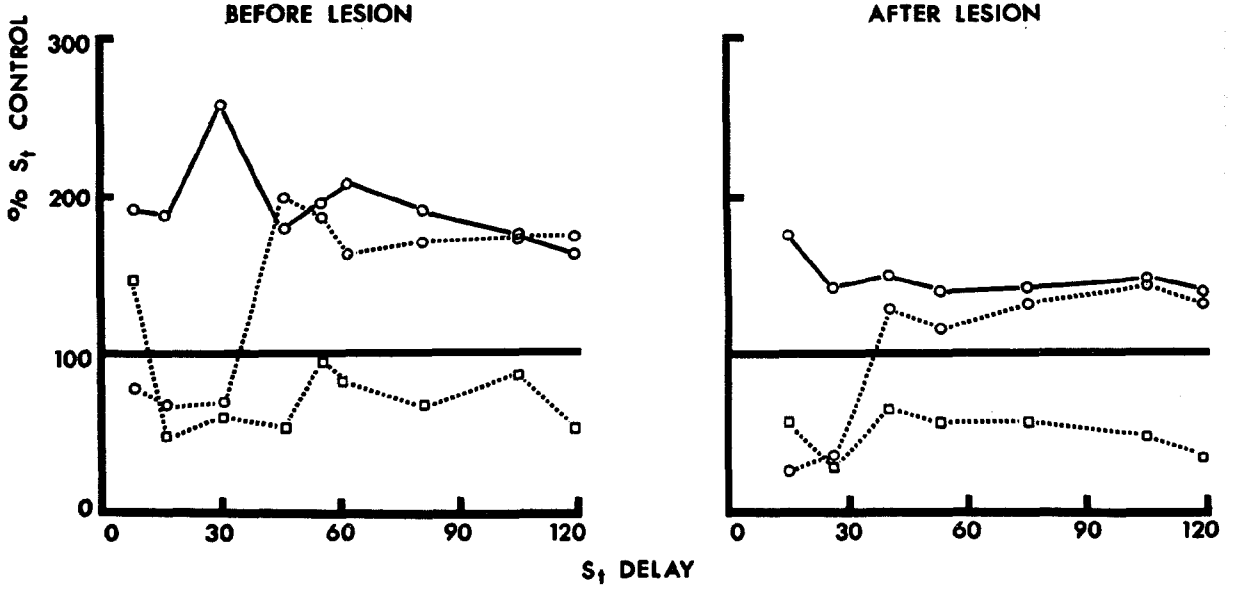
Fig. 23 In the experiment on the top (Cat 252) a lesion of the reticular formation resulted in only minimal changes in the effects of collicular tetanization on single evoked responses and the recovery cycle. At the bottom (Cat 245) reticular lesion decreased the magnitude of single evoked response and recovery cycle potentiation. For both experiments, the locus and extent of reticular lesions, and the site of collicular stimulation are identified in the accompanying photomicrographs.

EFFECTS OF RETICULAR LESION ON COLLICULAR STIMULATION

CAT 252



CAT 245



was markedly altered by reticular lesions in this animal. Such changes were observed (although to a lesser extent) in two other preparations. Only single response potentiation was tested, and found to be altered after reticular lesions in two additional experiments. Reticular lesions, however, were never found to differentially influence recovery cycle or single response potentiation. In three animals where lesions of the reticular formation did not modify the effects of pulvinar tetanization, recovery cycle potentiation was not elicited by pulvinar stimulation (as shown at the bottom of figure 22) prior to reticular lesions.

In four experiments, the effects of superior collicular stimulation on cortical responsivity were studied following lesions of the reticular formation. Such lesions produced minimal changes in the effects of collicular stimulation on single evoked response and recovery cycle potentiation in one animal (top of figure 23); in a second experiment the recovery cycle was unchanged, but single evoked potentiation was even increased somewhat after the lesion. In two other preparations, the magnitude of both single response and recovery cycle potentiation was decreased following lesioning of the reticular formation (bottom, figure 23). Reticular lesions, however, did not produce any changes in the typical time course of the potentiation effects elicited by collicular stimulation. This observation contrasts with the results obtained in the majority of experiments with pulvinar stimulation following similar lesions.

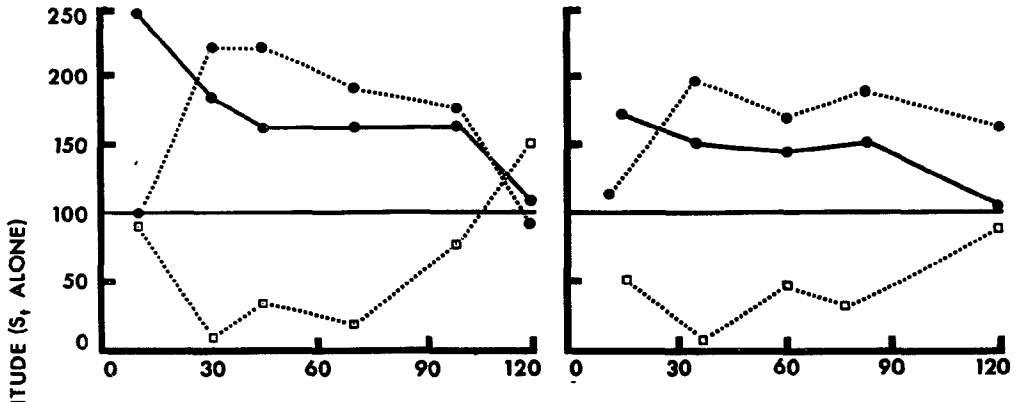
In two animals the superior colliculus was lesioned and in both experiments this had no influence on either pulvinar or reticular

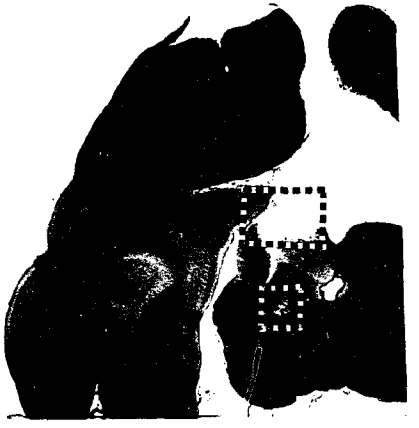
Fig. 24 Effect of reticular (top), and pulvinar (bottom) tetanization on single evoked responses and the recovery cycle, before and after collicular lesions. Photomicrographs at left indicate locus of pulvinar, geniculate and reticular stimulating electrodes, as well as the extent of the destructive collicular lesion.

CAT 247

EFFECTS OF COLLICULAR LESION

RETICULAR STIMULATION





S_c AND S_t

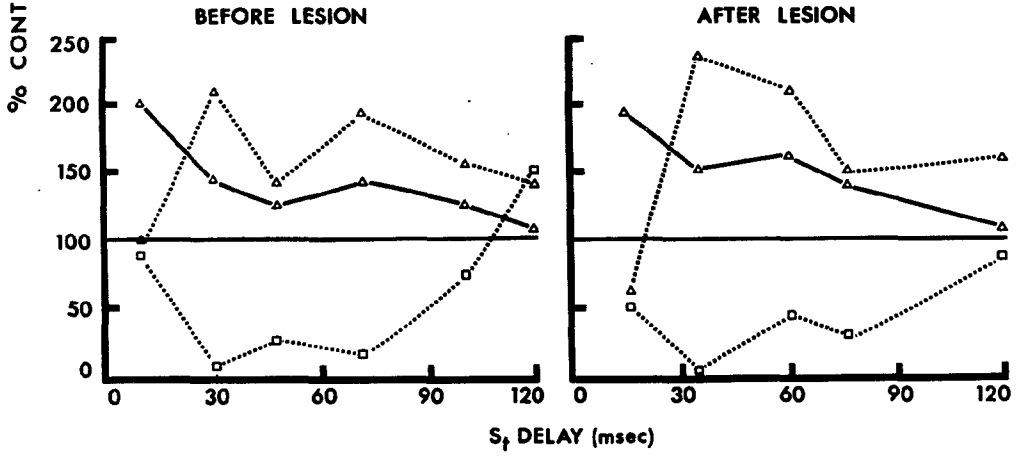
RETICULAR + S_c AND S_t

PULVINAR + S_c AND S_t

RETICULAR + S_t

PULVINAR + S_t

PULVINAR STIMULATION



stimulation effects (figure 24). In one animal, the pulvinar was excessively damaged by electrolytic lesions and this was also found not to modify the effects of reticular stimulation (figure 25). Finally, in two experiments, the lateral dorsal nucleus was inadvertently stimulated instead of the pulvinar. (Stimulation of this nucleus with low frequency shocks also evoked an incremental response at cortex). In both experiments, tetanization of the lateral dorsal nucleus produced the typical enhancement of single evoked responses and the recovery cycle. Furthermore, lesions of the reticular formation in one experiment, and the superior colliculus in another, did not modify these effects of lateral dorsal activation.

The results from this series of experiments indicate that the only marked modification of the effects of subcortical activation were observed with pulvinar tetanization (in 5 of 8 animals) following lesions of the reticular formation. It should be noted, however, that in these animals, reticular lesions did not influence the augmenting response elicited by low frequency stimulation of the pulvinar (figure 26). This suggests that the cortical inputs of the pulvinar underlying the augmenting response are different from those which mediate potentiation of cortical responsivity.

Fig. 25 Single evoked response and recovery cycle potentiation by reticular tetanization before (left) and following (right) pulvinar lesions. At the bottom are shown the extent of the destructive lesion in the pulvinar and the locus of the reticular electrode.

CAT 224

EFFECTS OF PULVINAR LESION ON RETICULAR STIMULATION

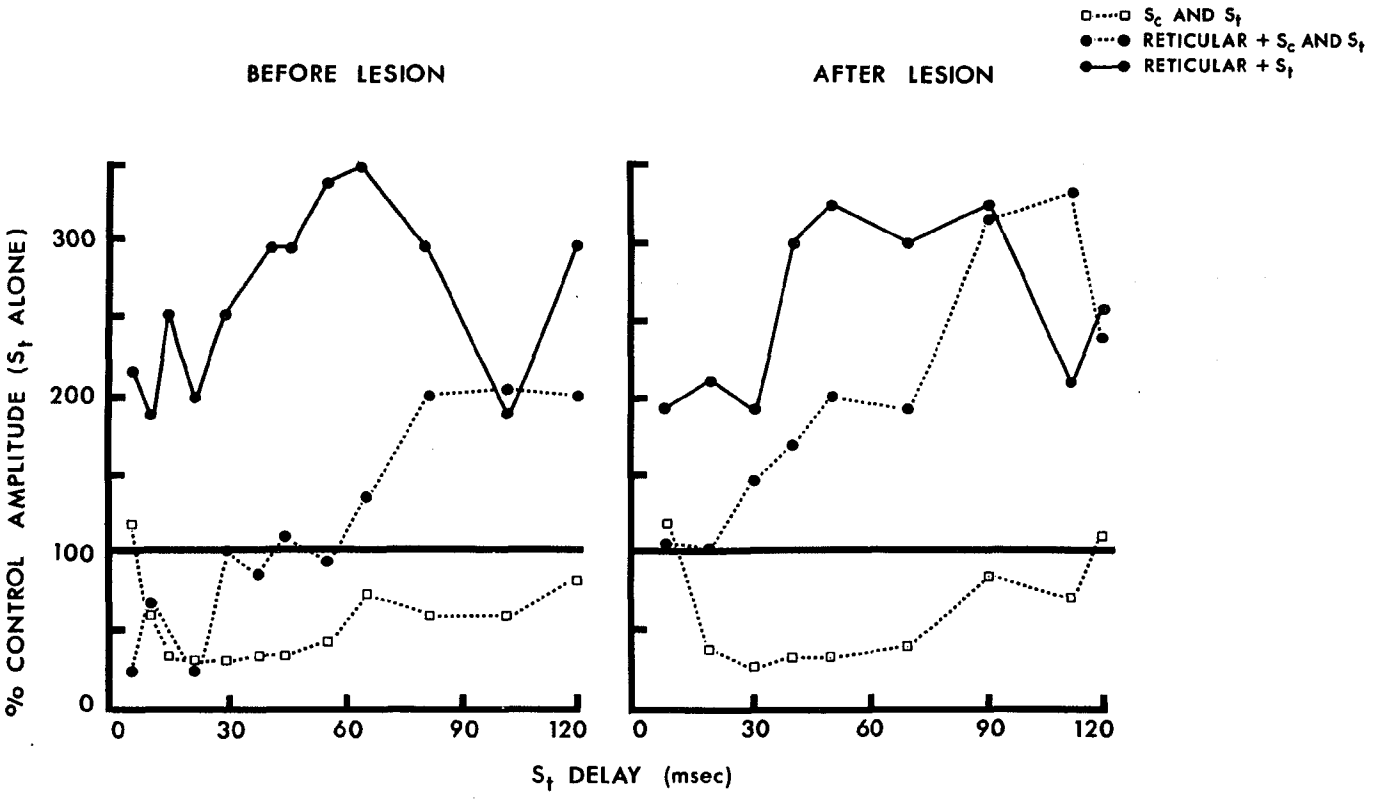


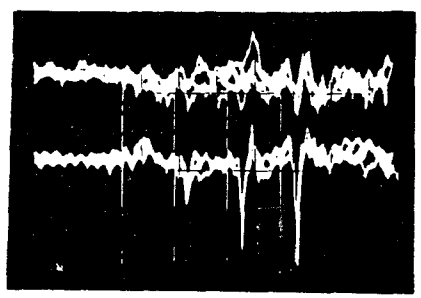
Fig. 26. At the left are shown cortical responses recorded in two experiments at the suprasylvian (bottom of each trace) and marginal (top) gyri to low frequency stimulation of the pulvinar. At the right, these augmenting responses were recorded following lesion of the reticular formation.

EFFECTS OF RETICULAR LESIONS ON AUGMENTING RESPONSES

BEFORE LESION

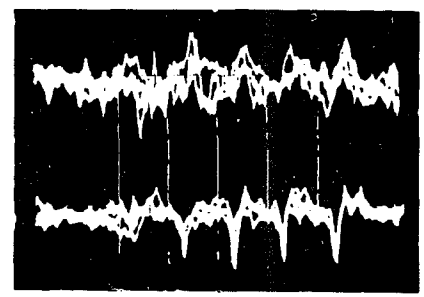
AFTER LESION

CAT 228

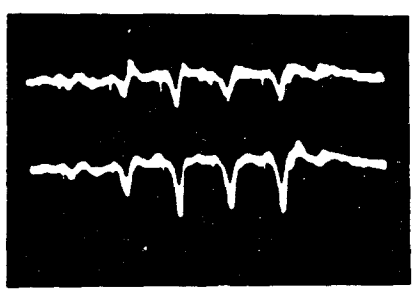


100μV 200μV

100 msec

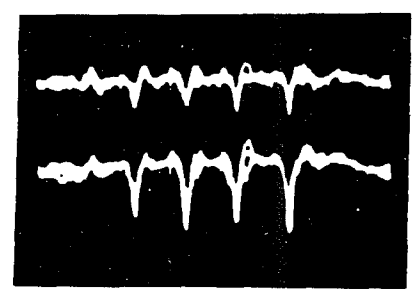


CAT 260



400μV

100 msec



DISCUSSION

Potentiation of single evoked responses. The results of the present study indicate that tetanization of the reticular formation, superior colliculus, and pulvinar can increase the amplitude of a single evoked response for at least 150 milliseconds following the offset of subcortical tetanization (figure 8). Prior investigators have usually presented their results only in qualitative terms and hence the data in figure 8 cannot be directly compared with that of previous studies. The few exceptions which have presented quantitative data on the effects of reticular activation on the amplitude of the evoked response, have either studied responses at the geniculate level (Suzuki and Taira, 1961; Okuda, 1962), or else have used entirely different parameters of reticular stimulation (Nakai and Domino, 1968). Regarding prior pulvinar studies, it has been reported that tetanization of the pulvinar produces an enhancement of the evoked response whose excitability cycle is polyphasic and lasts for about one second (Battersby and Oesterreich, 1963). However, a photic stimulus was used to evoke cortical responses and this precludes a comparison with the present data. Effects of pulvinar and superior collicular stimulation on cortical responses to optic nerve stimuli were investigated by Brown and Marco (1967), who found that stimulation of both structures doubled the amplitude of the evoked response; systematic excitability curves, however, were not determined.

As shown in figure 6, tetanization of the reticular formation, colliculus and pulvinar potentiated all four major components (C1, C3,

C4, and C5) of the evoked response, the greatest effect being produced in the latter two components. A number of studies have been concerned with determining the site of origin of the cortical evoked response components (Bishop and Clare 1951, 1952; Bishop and O'Leary 1938; Bremer and Stoupel 1958; Chang, 1952; Changeand Kaada, 1950; Landau, Bishop and Clare, 1964; Malis and Kruger, 1956; Marshall, Talbot, and Ades, 1943; Schoolman and Evarts, 1959). Most investigators agree that C1 represents the presynaptic input to cortex, while C4 and C5 are the result of postsynaptic cortical activity.[4] Collectively, the data of the present study suggest that subcortical tetanization influenced the cortically recorded response at presynaptic as well as postsynaptic levels. This is not surprising since prior studies have reported that retinal (Granit, 1956), optic nerve (Hernandez-Peon, et. al., 1956), as well as geniculate responses (Okuda, 1967; Satinsky, 1968) can all be modified by reticular stimulation. Furthermore, anatomical evidence has been presented showing that there are reticular inputs to the lateral geniculate body (Scheibel and Scheibel, 1958), as well as to the retina itself (reviewed by Ogden, 1968). Although pulvinar and collicular stimulation also enhanced the presynaptic component of the evoked response, there is no evidence for direct connections of these structures with either the lateral geniculate body or retina (Meikle and Sprague, 1967).

The magnitude of evoked response potentiation produced by subcortical tetanization was shown to be dependent on three variables; viz, intensity of subcortical tetanization, geniculate shock intensity,

[4]A controversy exists, however, regarding the origin of C2 and C3. Chang has maintained that C2 and C3 are afferent cortical inputs; Bishop has held that both represent postsynaptic cortical activity, while Malis and Kruger reported that C2 is of presynaptic and C3 of postsynaptic origin.

and level of Nembutal anesthesia. The least effective of these was the intensity of subcortical tetanization, since the magnitude of evoked response potentiation varied over only a small voltage range. This finding parallels the results reported by Okuda (1962), who studied the effects of different intensities of reticular stimulation on geniculate responses. Intensity of geniculate test shock, on the other hand, was shown in the present study to produce definite changes in the magnitude of potentiation (figure 5). Evoked response potentiation was minimal with a threshold geniculate shock, increased markedly at supramaximal intensities, and then diminished again or was absent at the highest shock levels. This finding has been previously noted for reticular activation by Nariskavili (1963). Bremer has suggested that reticular tetanization potentiates the evoked response amplitude by activating the subliminal fringe which in turn summates with the response to the sensory stimulus. Since Marshall (1949) reported that the extent of the subliminal fringe varies inversely with the intensity of a sensory stimulus, it is apparent that the data obtained with supra-threshold stimuli are compatible with Bremer's hypothesis. Lack of significant potentiation with threshold shocks, however, fails to conform to the results predicted by the hypothesis. Confirming prior studies (Bremer and Stoupel, 1959; Long, 1959; King, Naquet, and Magoun, 1957), successive doses of Nembutal were found to decrease, and in some cases to abolish, the potentiation of evoked responses elicited by reticular tetanization. The similar influence of barbituates on the effects of pulvinar and collicular stimulation, however, have not been previously reported. Since it is well known that many barbituates depress reticular (Arduini and Arduini, 1954; French, Verzeano, and Magoun, 1953; Goodman and Mann, 1967),

as well as cortical activity (Curtiss, 1940; Marshall, Talbot and Ades, 1943; Battersby and Wagman, 1964), it is not possible to determine which of these factors is responsible for the influence of Nembutal on evoked response potentiation.

Potentiation of dual responses (recovery cycle). The present study has shown that the recovery cycle can also be potentiated by tetanization of reticular formation, superior colliculus or pulvinar. As indicated in figures 13 and 14 the magnitude of the potentiation varied systematically with the S_c-S_t interval, minimal potentiation being generally obtained around 60 milliseconds and then returning toward normal levels by 150 milliseconds. These data confirm and extend the findings of Steriade and Demetrescu (1967) and Demetrescu, Demetrescu, and Iosif (1969) who also used paired geniculate shocks to study the effects of reticular tetanization on the cortical recovery cycle. The particular time course of recovery cycle potentiation shown in figures 13 and 14, may be modality specific, since Schwartz and Shagass (1963) using somesthetic stimuli, reported greatest potentiation at the shortest S_c-S_t intervals. Modification of the recovery cycle by pulvinar or collicular stimulation has not been previously investigated in the visual modality, although Schwartz and Shagass (1963) did note that pulvinar activation produced little or no effect on the somesthetic recovery cycle.

Recovery cycle potentiation was less often elicited by tetanizing subcortical structures than enhancement of single evoked responses. This was most evident from the data obtained with pulvinar stimulation since only 55 percent of the preparations exhibiting single response potentiation also showed potentiation of the recovery cycle.

The comparable percentage for both reticular and collicular stimulation was 84. There was no anatomical differentiation in the reticular formation and colliculus between the loci which produced potentiation of both recovery cycle and single responses, as compared to those that only affected the latter measure (figure 16). There was some indication that the failure to elicit recovery cycle potentiation may have been due in part to a high arousal level of these animals, since their pupils were markedly dilated during the experiment. (Schwartz and Shagass (1963) have previously reported that potentiation of somesthetic recovery cycle was not elicited in aroused preparations, where the arousal level was judged by the EEG). Regarding the pulvinar, stimulation of loci in the dorsal pulvinar tended to produce potentiation of the recovery cycle more often than those in the ventral pulvinar (figure 16). There were exceptions to this differentiation, however, and the only conclusion which may be made, is that certain areas in the pulvinar (particularly in the ventral pulvinar) do not elicit recovery cycle potentiation even when high tetanizing intensities are used (figure 19). It is of interest to note that neurons in the "upper half" of the pulvinar have recently been reported to have "visual properties" (Suzuki and Kato, 1969), while those in the ventral half have been attributed with auditory properties (Buser, Bornstein, and Bruner, 1959).

When potentiation of the recovery cycle was elicited by tetanization of subcortical structures, the magnitude of this effect usually equalled, and in some cases was greater, than the amplitude of single response potentiation. The time course of these two effects was different, in that minimal recovery cycle potentiation was observed at short

S_t delays (0-30 milliseconds), while single responses were substantially potentiated during this period. The respective mean magnitudes and time courses of these two effects may be seen by comparing the data in figure 14 with that of figure 8. (In order to equate the ordinates of these two figures, 100 percent should be subtracted from the ordinates of figure 8).[5] In general, it should be noted, that a higher tetanization level was usually necessary to elicit potentiation of the recovery cycle than of the single response (figures 17 and 18). In a few instances, however, minimal potentiation of single responses was observed but the recovery cycle was definitely potentiated (figure 20). This latter observation confirms the reports of Schwartz and Shagass (1963) and Steriade and Demetrescu (1965) who found that reticular stimulation occasionally did not affect the response to the first of two stimuli, but did affect the second (recovery cycle potentiation).

Comparison of the potentiation effects of reticular, collicular, and pulvinar stimulation. The present study has shown that stimulation of the reticular formation, superior colliculus, and pulvinar could potentiate the amplitude of both single responses and the recovery cycle. Reticular formation and collicular tetanization generally produced potentiating effects of equal magnitude, while the effects of pulvinar stimulation were usually smaller. Subcortical tetanizing intensity (figure 4), geniculate shock intensity (figure 5), and Nembutal anesthesia (figure 7), influenced the

[5]In figure 8 the potentiation is expressed in terms of percent control (S_t alone); thus 100 percent indicates no potentiation. The ordinate in figure 7, however, shows a difference between the curves presenting the recovery cycle and the change in this function produced by subcortical stimulation. In this case 0 percent indicates no difference between these two curves, thus zero potentiation of the recovery cycle.

potentiating effects of all three subcortical structures in similar ways. There was some indication, however, of differences between the effects of collicular stimulation as compared to those produced by reticular or pulvinar tetanization. Thus, collicular activation equally potentiated components 4 and 5 of the evoked response (figure 6), while both pulvinar and reticular activation increased component 4 to a greater extent than component 5. In addition, pulvinar and reticular potentiating effects of single evoked responses decreased monotonically as a function of the S_t delay, while the effects of collicular stimulation reached a peak at 60 milliseconds and then gradually declined. These results indicate that stimulation of most loci in the pulvinar produced potentiating effects which were closely analogous (although lower in magnitude) with those elicited by reticular stimulation. Effects of collicular stimulation, on the other hand, were similar to those elicited by stimulation of the other structures, but also showed the two differences discussed above. In general, the pulvinar and reticular system could be producing their effects through a common system, but a more unique or independent system might be involved for the mediation of the effects of collicular stimulation.[6]

[6]Current spread as a possible factor in the results of the present study should be briefly discussed. Potentiation of cortical responsivity by spread of current to the specific visual system may largely be ruled out, since it was shown that the time course of post-tetanic potentiation was markedly different from the potentiation elicited by collicular, pulvinar or reticular stimulation. It is also doubtful that tetanization of the pulvinar could activate the mesencephalic reticular formation (or vice versa) by a passive spread of current. This is indicated by the few experiments in which an error in electrode calibration resulted in stimulation of loci two or three millimeters lateral to the reticular formation. In these instances no potentiation of cortical responsivity was elicited even at high stimulation intensities (15 volts). Evoked response potentiation could be observed in these preparations if the electrode was removed from the brain, recalibrated, and then reinserted into the reticular formation.

Mechanisms mediating potentiating effects. Brown and Marco (1967) have suggested that the effects of collicular stimulation on cortical responsivity may be mediated via the mesencephalic reticular formation. In support of this hypothesis are electrophysiological data indicating that these two structures are functionally related (Brown and Marco, 1966, 1967), and anatomical evidence showing reciprocal tectal-reticular fiber connections (Nauta and Kuypers, 1958; Altman and Carpenter, 1961; Altman, 1962). In the present study lesions of the reticular formation did reduce somewhat the magnitude of the potentiation effects elicited by collicular tetanization (in 2 of 4 experiments), but no changes were observed in the typical time course of these effects (figure 23). A similar observation was made when a lesion was made in the superior colliculus and the effects of reticular formation stimulation were retested. In general, the positive findings suggest that reciprocal tectal-reticular connections may be involved in producing potentiation in cortex elicited by either collicular or reticular activation. On the other hand, the negative instances indicate that such connections do not serve as the sole mediating basis for these effects. Tectal efferents also project to the pre-tectum, centre median, the supragenicular nuclei of the thalamus and the zona incerta (Bucher and Burgi, 1950; Altman and Carpenter, 1961). Since the mesencephalic reticular formation also projects to many of these structures (Nauta and Whitlock, 1959; Scheibel and Scheibel, 1959), it is possible that a common pathway for the effects of collicular and reticular stimulation may arise in one or more of these structures.

In 5 of 8 preparations, reticular lesions resulted in major disruptions in the magnitude and time course of the effects of pulvinar activation. These positive data suggest that the pulvinar produces its effect on cortical responsivity via projections through the mesencephalic reticular formation. Is is doubtful, however, that the pulvinar projects directly to the mesencephalic reticular formation, since Altman (1962) has reported no degeneration in the reticular formation following lesions of the pulvinar. Battersby and Oesterreich (1963) have speculated that the pulvinar may project to the reticular formation by way of the superior colliculus. This hypothesis is supported by Altman's finding of collicular degeneration after pulvinar lesions, but is inconsistent with the data of the present dtudy which showed no alteration in the effects of pulvinar stimulation after collicular lesions (figure 24). It is well known that the pulvinar projects to the suprasylvian gyrus (Waller and Barris, 1937), and that the mesencephalic reticular formation receives inputs from this cortical region (Niemer and Jimenez-Castellanos, 1959). Thus, an alternative manner by which the pulvinar may connect with the reticular formation is by corticofugal fibers from the suprasylvian gyrus. This hypothesis also accounts for the finding that reticular lesions which altered the effects of pulvinar activation on cortical responsivity did not influence the augmenting response (figure 26). Not all areas in the pulvinar when stimulated activate the reticular formation. This is indicated by the finding that tetanization of some loci (usually in the ventral pulvinar) even with the highest voltage levels used in the present study, produced no recovery cycle

potentiation; in the same animal these effects were elicited by reticular stimulation. In addition, lesions in the reticular formation in three such experiments did not disrupt the effects of pulvinar activation. It should be noted that the effects of subcortical lesions were investigated in a relatively small number of experiments. Therefore, the conclusions suggested by these data are offered as likely hypothesis for future studies, rather than as definitive answers to the anatomical interconnections among the reticular formation, colliculus, and pulvinar.

In conclusion, the present study, in agreement with prior investigations, has shown that both, the single evoked response and the recovery cycle may be potentiated by stimulation of the reticular formation. In addition, it was found that analogous effects could be elicited by tetanizing the superior colliculus and pulvinar. Single evoked response potentiation, however, was more frequently observed with subcortical tetanization and lower tetanizing voltages were usually adequate to elicit this effect. Reticular and collicular stimulation was generally equally effective in potentiating both measures of cortical responsivity. Stimulation of the pulvinar, on the other hand, resulted in less frequent recovery cycle potentiation and the magnitude of both recovery cycle and single response potentiation was lower with pulvinar tetanization. Since in most experiments lesions of the mesencephalic reticular formation disrupted the effects of pulvinar stimulation, it was suggested that the effects of pulvinar activation may in part, be mediated via the mesencephalic reticular formation.

Behavioral implications. The behavioral significance of the present data should be briefly discussed. Hernandez-Peon (1959) originally hypothesized that the reticular formation functions as a filtering

mechanism which monitors sensory inputs and then determines which sensory stimuli should be inhibited and which facilitated. More recently a similar approach has been taken by Kilmer and McCulloch (1969), who use a computer simulation model to explain how the reticular system may "command" an organism's mode of behavior. A number of studies can be cited to support the general concept that the reticular formation is involved in the behavioral functions of attention and habituation (Galamos, Sheatz, and Vernier, 1956; Hernandez-Peon, 1955; Hernandez-Peon, Guzman-Flores, Alarez, and Fernandez-Guardiala, 1957). On the other hand, when the size of the pupils is maintained constant, habituation of visual evoked responses was greatly minimized (Naquet, et. al., 1960). The role of the reticular formation in attention has also been questioned, since in acute (Nariskavili and Moniava, 1963) as well as chronic preparations (Horn, 1960), tetanization of the reticular formation does not selectively enhance evoked responses of one sensory modality while inhibiting those of other modalities.

Lindsley (1958) has proposed that the reticular formation is closely involved in perceptual discrimination. He originally showed that the temporal resolution of two photic responses at cortex can be facilitated by tetanization of the reticular formation, and these data were presented in support of the concept that the reticular system is uniquely involved in perceptual discrimination. The present study has shown that recovery cycle potentiation may also be elicited by collicular and pulvinar stimulation. In any event, the behavioral significance of the foregoing data should be interpreted with caution, since the rela-

tionship between cortical evoked responses and perceptual processes has not been clearly established. In recent years an extensive literature has developed on the behavioral significance of brain potentials, and according to some, there is a high correlation between averaged evoked responses and perceptual processes, such as attention (Donchin and Cohen , 1967), temporal resolution (Donchin and Wicke, 1963), and backward masking (Fehmi, Adkins, and Lindsley, 1969). However, recently the significance of the evoked potential for perceptual processes has been questioned (Clark, Butler, and Rosner, 1969), and the suggestion has been made that the secondary discharge may be more significant for sensory function that the primary evoked response (Libet, Alberte, Wright and Feinstein, 1967).

Suggestions for future experiments. At least three additional experiments are suggested by the results of the present study. First, the interconnections between the pulvinar and the mesencephalic reticular formation should be studied in more detail. It was suggested in the present study that the pulvinar porjects to the reticular formation by way of the suprasylvian gyrus. This hypothesis could be tested by interfering with the function of the syprasylvian gyrus (by local cooling, topical KCL, or sub-pial aspiration), and then determining whether effects of pulvinar activation are altered by such a procedure. Secondly, it would be of interest to investigate the effects of subcortical activation on the recovery cycle at the unit level. This could be performed with extracellular recordings, although recently intracellular techniques have also been used to record from visual neurons in the cat cortex (Creutzfeldt and

Ito, 1969). Such a procedure would permit a more precise understanding of the mechanism by which subcortical structures influence cortical responsivity. Thirdly, as already noted, the behavioral significance of the present data should also be investigated. Reticular stimulation has been shown to facilitate the percentage of correct responses in a visual discrimination task and to decrease the reaction time of these responses (Fuster, 1958; Fuster and Uyeda, 1962). The results of the present study suggest that activation of the pulvinar, colliculus, as well as the reticular formation, may behaviorally potentiate temporal resolution. This suggestion should be tested in future studies using chronic preparations with cortical and subcortical implanted electrodes. In this way, the effects of subcortical stimulation on behavioral and cortical temporal resolution could be simultaneously determined, thereby bridging the gap between behavior and electrophysiology.

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APPENDIX

TABLE TWO

Individual Data for Recovery Cycle Potentiation Elicited by Subcortical Tetanization

Reticular Formation
S_t Delay

Cat Number	5	10	20	30	40	50	60	70	80	90	100	110	120	130	140	150
197	-45	-30	100	90												
200	70	100	115	35	10	10	-25									
204	70	-80	120	155	200		210	265	290	65	35			-25		25
206	60	35	120	155	85	20		-45	-45		-70					
214		135	50	40	65	70	110	115	45		65			120	25	10
215	115	275	90	230	205	245	220		130	105		135			90	
216	100	0	-25	0	40	185	175	115		75		0				10
217	220	-30	25	110	260	280	140	230	250		245			175		
219	-60	-10	170	60	130	70	70		20		-25	10	50			
224	-85	0	60	60	70	50	80	125	100		155		105		150	
225	5	230	55	200	200	230					300		325		200	
228	200	90	180	245	225	200	245		160		175	220			115	
232		-10	45		75	35	90		65	85		50				
233	90		0	50		35	145		170		115		150			
234		5	5		0		0		0		80		220		250	150

Reticular Formation (Continued)

S_t Delay

Cat Number	5	10	20	30	40	50	60	70	80	90	100	110	120	130	140	150
214				30		65	70	110	145		155		100		115	90
235		25		205	250		175	100		150			125		0	
236		-5	0	90	135		165		185	200		145				
237			0	195	325		250		85		35		75		-25	
238		0			0	0	30	50	95		75					
242		260		295	410	310	220		280		80	110	10			
244		0	25	20	70	90		205			180		110			
245		-65	25	0	140	100	85		105		85		110			
247		0		130		170		185			60		-30			
250		70		100		100		115			80		120			
251		40		10		25		80		100			110			
252		20		50			0			100			150			
255		0		5	20		20				100					
256			165	300		350			225		80					
257				10		20					90					100
258		10	90	20		70	75									
259		10		5			150									
260		10		-5	10			75					60			
261		0			5						25					75

Pulvinar

S_t Delay

Cat	Number	5	10	20	30	40	50	60	70	80	90	100	110	120	130	140	150
	215	130	200	10	85	70	125	115		170	75		55		20		-5
	216	15	25	0	0	5	55	85	85		75		-5				-5
	219	-35	0	70	45	85	85		0	25		0		10			
	228	165	-90	-10	-30	85	270	170		155		155	205			145	
	234		0	0		5		0		0		80		100		95	60
	242	20			90	220	80	115		25		60	25	5			
	247		20		195		100		180			75			-10		
	252		-10		30			5			-20			70			
	259		5		5			80									
	260		0		5			20		30				30			

Reticular Formation (Continued)

Cat	Number	S_t Delay 5	10	20	30	40	50	60	70	80	90	100	110	120	130	140	150
	236		-5	85	90	335		245		180		200	135				
	238		10	40	70	150	120	95	160			50		120			
	242		-40		235	230	180	155		160		100	100	80			
	244		0	25		30	40	70		220		180			100		
	245		-95	25	95	90	150	130		80		95		85			
	247		20		205		180		140			60			-50		
	249		25		145	100	140			60		20		25			
	250		90		220		250		255			100		125			
	252			-25	75			60			125			110			
	255			-20	10	50		75				75					
	258		25	10	10			150									
	259		25		10			250									
	260		20		20		10		90					120			
	261			0			0				0						100
	198	40	20	105	50												

AUTOBIOGRAPHICAL STATEMENT

Name: Leo M. Chalupa

Date of birth: March 28, 1945.

Marital Status: Married

Education:

1960-1963	Stuyvesant High School
1963-1966	Queens College of the City University of New York
1966-1970	City University of New York (Ph.D., Neuropsychology, 1970)

Graduate Appointments:

1966-1967	Research Assistant (Queens College)
1968-1970	Trainee in Neuropsychology
1969-1970	Lecturer (part-time) in Psychology (Queens College)

Professional Societies:

Optical Society of America
American Association for the Advancement of Science
American Psychological Association