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INFLUENCE OF SUPERIOR COLLICULUS ON THE SPATIAL GRADIENT OF RESPONSES IN CAT VISUAL CORTEX

by

Marian D. Sekuler

A dissertation submitted to the Graduate Faculty in
Psychology in partial fulfillment of the requirements
for the degree of Doctor of Philosophy, The City
University of New York.

1973

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July 26, 1973
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Abstract

The effects of activating the superior colliculus (SC) on the spatial distribution of responses elicited in visual cortex (V1) by single and double "test" shocks to the lateral geniculate body (LGB) were examined in 38 adult cats locally anesthetized and maintained on N_2O and O_2. Results show: (1) Tetanizing the ventral portions of SC increased the amplitude of the single test response, at both the topologically related (maximally responsive) locus, and at surrounding submaximal loci in V1, with the latter effect being proportionally the larger. (2) Prior SC tetanization altered the spatial interaction of responses to dual shocks by reducing the temporal period of interaction between them; in some cases it exerted a "sharpening" effect on the distribution of cortical responses to the second ("test") shock. (3) Similar enhancement effects were obtained by tetanizing SC on the side contralateral to the V1 cortex being mapped, and such effects survived large lesions of the ipsilateral SC alone, or in combination with topical KCl on the contralateral V1 and V2 cortex. (4) SC enhancement survived ipsilateral Pulvinar (Pul) lesion, was slightly reduced following ipsilateral MRF lesion, and was completely abolished by bilateral MRF lesions, or by ipsilateral Pul and MRF lesion. These findings demonstrate the influence of SC on spatial processes in V1 cortex, presumably by way of known tecto-reticular pathways, and are discussed in terms of current concepts of SC function.

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INTRODUCTION

The function of the superior colliculus has classically been considered twofold: 1) as a sensorimotor integration center for oculomotor function (Schäfer, 1888), and, 2) as the mediator of residual visual functions following cerebral lesions, i.e., "collicular vision" (see Marquis, 1935). Unfortunately, neither of these concepts has direct anatomical support. Fibers from the superior colliculus to the third, fourth, and sixth cranial nerve nuclei have yet to be reliably described (Altman and Carpenter, 1961), and in any event, ablation of the superior colliculus yields only a transient oculomotor paresis (Pasik and Pasik, 1964). With respect to residual visual function, visual intensity or pattern discriminations in the cat are still present after lesion of the visual cortex plus serial or simultaneous removal of the superior colliculus (Sperry et al, 1955; Norton and Clark, 1963; Fishman and Meikle, 1965; Urbaitis and Meikle, 1968). In the primate, some residual vision persists following complete striatectomy, plus collicular lesion, and it is only abolished following bilateral lesions of the accessory optic tract (Pasik and Pasik, 1971). Furthermore, current theories have stressed two other aspects of collicular function: first, an ancillary visual projection system in parallel with the geniculo-striate projection and second, a spatial integration center mediating visual orienting behavior (see Schneider, 1969).

While recent studies have supported neither of the two classical concepts of collicular function, much has been revealed about its connections with other central nervous system structures. For example, the superior colliculus in the cat receives one topological projection from the retina, and another from the visual cortex, the two inputs arriving at the same discrete region within the dorsal layers of the superior colliculus (Garey, 1965; Garey et al, 1968). The ventral layers of the superior colliculus receive somesthetic inputs from the spinal cord via the medial spinothalamic tract (Anderson and Berry, 1959), plus an auditory input from the inferior colliculus (Moore and Goldberg, 1963). From the functional point of view, Altman and Malis (1962) have shown that evoked responses and unit discharges to photic or optic nerve stimulation were maximal at the dorsal levels of the superior colliculus. Visual, auditory and somesthetic stimulation can all elicit gross evoked responses in the superior colliculus which can also be modified by cortical influences (Jassik-Gerschenfeld and Ascher, 1963; Marchiafava et al, 1968). More recent studies have demonstrated both modality specific and multimodal responses in individual collicular cells (Jassik-Gerschenfeld, 1965; Wickelgren, 1971; Stein and Arigbede, 1972b; Gordon, 1973) with visually specific cells dominating the dorsal layers, multimodal responsive cells the ventral layers (Stein and Arigbede, 1972b). Some of these properties are subject to change, however, since the response properties of visually specific cells are alterable by cortical influences (Wickelgren and Sterling, 1969; Rizzolatti et al, 1970; McIlwain and Fields, 1970; Hoffman and Straschill, 1971; Palmer et al, 1972; Stein and Arigbede, 1972b).

The superior colliculus (SC) also receives afferent fibers originating in the Pulvinar (Altman, 1962), the mesencephalic reticular formation (MRF) (Anderson and Berry, 1959) and from numerous cortical locations including the marginal, posterolateral, suprasylvian and sigmoid gyri (Sprague, 1963) and the ectosylvian gyrus as well (Sprague, 1963; Paula-Barbosa and Sousa-Pinto, 1973). A number of efferent pathways originate from the superior colliculus. For example, the SC gives rise to fibers projecting to the opposite colliculus, to the MRF, to the interstitial nuclei of Cajal and N. of Darkschewitsch, the pons, spinal cord, pars ventralis of the lateral geniculate body, medial geniculate body, pulvinar-lateralis posterior complex (Pul-LP), suprageniculate n., pretectum, Forel's field H1 and the reticular nucleus of the thalamus (Altman and Carpenter, 1961). Unfortunately, some of these efferent pathways have yet to be well delineated with the more sophisticated histological techniques now available. In any case, some of these structures (in particular Pul-LP and MRF) are known to respond to photic stimuli (French et al, 1952; Chalupa et al, 1972) and these same structures are also known, from other stimulation studies, to be part of a diffuse system which can influence the visual cortex either directly

(Chalupa et al, 1973) or through projection to LGB (Okuda, 1962; Bremer, 1959). While the SC obviously has numerous efferents, their functional significance has not been assessed in any detail. Prior studies have shown that stimulation of the SC diminishes the cortical evoked response to a photic stimulus and enhances the evoked response to a geniculate shock (Bremer, 1961, 1966a, 1966b; Brown and Marco, 1966; Chalupa et al, 1973); in the latter case the enhancement is bilateral in cortex (Bremer, 1966a, 1966b). Some of these effects could be mediated through the MRF, Pul, and/or intralaminar nuclei, structures functionally and anatomically related to the SC, or alternately through the opposite colliculus. This latter possibility of an intercollicular effect has been stressed by Sprague (1966), but exclusively on the basis of ablation evidence.

The behavioral significance recently ascribed to the superior colliculus, is that of a mediating center for visually guided behavior (Sprague and Meikle, 1965; Sprague, 1972). This concept is based on recent findings in the cat (Sprague and Meikle, 1965; Norton and Lindsley, 1971), hamster (Schneider, 1969), tree shrew (Casagrande et al, 1972) and monkey (Denny-Brown, 1962) indicating that orienting and following responses are severely altered by SC lesion while stimulation of SC (in cats) produces eye and head movement responses toward the contralateral side (Hess et al, 1946; Hyde and Eliasson, 1957; Syka and Radil-Weiss, 1971). On the other hand, it should be pointed out that detailed studies on the behavioral effects of SC lesion have not yet been carried out within higher species and, in fact, there is some negative evidence for the monkey (Pasik et al, 1966). The concept of a visual orienting function of the SC has heuristic value, however, and has guided more recent electrophysiological work. For example, it appears significant that collicular units are most responsive to moving stimuli, and that they possess directional sensitivity (Straschill and Taghavy, 1967; McIlwain and Buser, 1968; Sterling and Wickelgren, 1969; Stein and Arigbede, 1972a), topological specificity (Straschill and Hoffman, 1968, 1969), and that their discharge can be correlated with the onset of spontaneous eye movements (Straschill and Hoffman, 1970). In addition, collicular unit discharges attenuate markedly to repeated stimulation (Sprague et al, 1968; Straschill, 1971; Stein and Arigbede, 1972a), this unit habituation being the neuronal correlate of the behavioral habituation of the orienting response (Straschill, 1971). Since orienting involves head and neck movement, it is significant to note that Anderson et al, (1971) demonstrated EPSP's from contralateral neck motoneurons in the cat following stimulation of the deep layers of SC and found them to be reduced following interruption of the tectoreticulospinal pathways. Finally, recent observations on the tree shrew (Casagrande et al, 1972) suggest that a functional distinction can be made between the dorsal layers of SC which may mediate form discrimination, and the ventral layers which may subserve spatial orientation.

Collectively, the above anatomical, electrophysiological and behavioral data indicate that the spatial aspects of visual stimulation may be in part dependent upon collicular functioning, either via its efferents, or possibly by processes intrinsic to SC itself. It would follow from this consideration that one way of assessing the influence of SC activation would be in terms of its influence on spatial projection in the geniculo-striate system. Recent studies from this laboratory in the unanesthetized cat have shown that a single LGB shock stimulus elicits a maximal cortical response fairly localized in visual cortex, if shortest latency and maximum amplitude are used as criteria. This maximal locus is always surrounded, however, by a more widespread distribution of smaller amplitude, longer latency responses which while submaximal are still functionally significant since temporal interaction between responses to paired LGB shocks can be demonstrated (Storck et al, 1972). Another study (Chalupa et al, 1973) has shown that activation of SC, MRF, or Pul can alter the response amplitude of the cortical potential evoked by a LGB test shock(s). In the present study, the influence of activating the SC on the amplitude distribution of responses across cortex (the cortical gradient), as elicited by single or double shocks to LGB was examined in the locally anesthetized, immobilized cat. In an effort to determine the pathways mediating collicular influences on cortical responsiveness, SC effects were compared with those produced by activation of Pul and MRF, as well as by the opposite colliculus. In addition, the effects on cortical responsiveness of lesions in these various structures was also assessed.

METHODS AND MATERIALS

Surgery. Quantifiable data were obtained from 38 adult male cats, each weighing 2 to 3 kilograms. Ether anesthesia was induced in an airtight box, the trachea cannulated, and the animal thereafter maintained on Halothane anesthesia (1 to 2 per cent depending on physiological signs) and N_2O analgesia (1 liter per minute), supplemented with O_2 (0.6 liters per minute). After mounting in a stereotaxic instrument (Kopf), the saphenous vein was cannulated for later infusion of gallamine triethiodide (Flaxedil), the temporal muscles reflected bilaterally to expose the dorsal and lateral parts of the cranium (particularly on the left side), and two trephine holes were made on the left side, one 2 to 3 millimeters from the midline at about stereotaxic A-P plane 0 and another one just rostral and dorsal to the earbar (for LGB stimulation), i.e., the region just overlying the anterior sylvian gyrus. A large left craniectomy or in some cases a bilateral one was made exposing the marginal and posterolateral gyri and most of the suprasylvian gyrus extending laterally approximately 20 millimeters from the midline (see Fig. 1).

General Procedure. Using a calibrated electrode carrier, the cranium bordering the cortical exposure on either side was marked in 5 millimeter intervals from A+20 to P-10, to facilitate accurate subsequent placement of cortical recording electrodes. After the dura was reflected, the cortex was kept moist with saline at about 37° centigrade, and

Fig. 1 Illustration of preparation in stereotaxic instrument. Dorsal exposure of left marginal and suprasylvian gyri: three stimulating electrodes, held together on the same carrier, penetrated the dorsal surface to impale SC, MRF, and Pul and eight recording electrodes rested on the pial surface 2 to 3 millimeters from the midline. Near the left earbar, four horizontally-oriented electrodes impaled LGB through the trephine opening.

partially covered with Saran Wrap to form a moist chamber.

A comb of four, horizontally oriented, stimulating electrodes was directed medially towards the left LGB through the trephine hole just anterior to the left ear (see Fig. 1). Each of these concentric electrodes was formed of a central 5 mil tungsten wire, insulated with Formvar except for the flush-cut and polished tip, which was inserted into a 24 guage hypodermic tubing, similarly insulated except for the concentric tip. The wire tips extended 0.5 millimeter from the edge of their tubes and were spaced 1 millimeter apart. Electrode impedances were frequently monitored in normal saline and were of the order of 20 to 50 kohms. A double-pole selector switch permitted selection of any one of the four electrodes for bipolar stimulation. Additional electrodes fashioned from two 5 mil tungsten wires separated by 0.5 millimeter were directed via a common carrier towards the SC, either on one side or both, and also towards the MRF, again either unilaterally or bilaterally, and in some cases at the Pul as well. All coordinate values used are expressed according to the atlas of Jasper and Ajmone-Marsan (1954). Before each experiment the impedance of the bipolar depth electrodes was measured in normal saline, and found to be approximately 50 to 100 kohms. Occassionally the stimulating current passing through the LGB or SC-MRF-Pul electrodes was monitored in vivo with an inductively-coupled current probe, and found to be in the range of 100 to 200 microamperes. Eight silver monopolar recording electrodes, with 0.5 millimeter balled tips, were mounted on ball joints in a Grass electrode holder and then disposed along the marginal and posterolateral gyri, about 2 to 3 millimeters from the midline, i.e., the 17-18 border or the presumed vertical meridian according to the published results of Otsuka and Hassler (1962) and Woolsey (1971). All recordings were monopolar, the temporal muscle or bone at the occipital ridge serving as a reference (see Fig. 1).

Following electrode placement, all wound edges and pressure points were infiltrated with propitocaine (Citanest), Flaxedil was infused, and the animal placed on artificial respiration at about 35 cc per stroke, 26 strokes per minute. Halothane was then completely withdrawn or, on occasion, reduced to 0.25 per cent. A DC powered heating blanket wrapped around the animal's abdomen helped maintain body temperature at about 37° centigrade as described in prior reports from this laboratory (Battersby and Oesterreich, 1963). Finally, the animal's hindquarters were elevated to prevent stasis, and periodically the trachea was aspirated to prevent fluid accumulation.

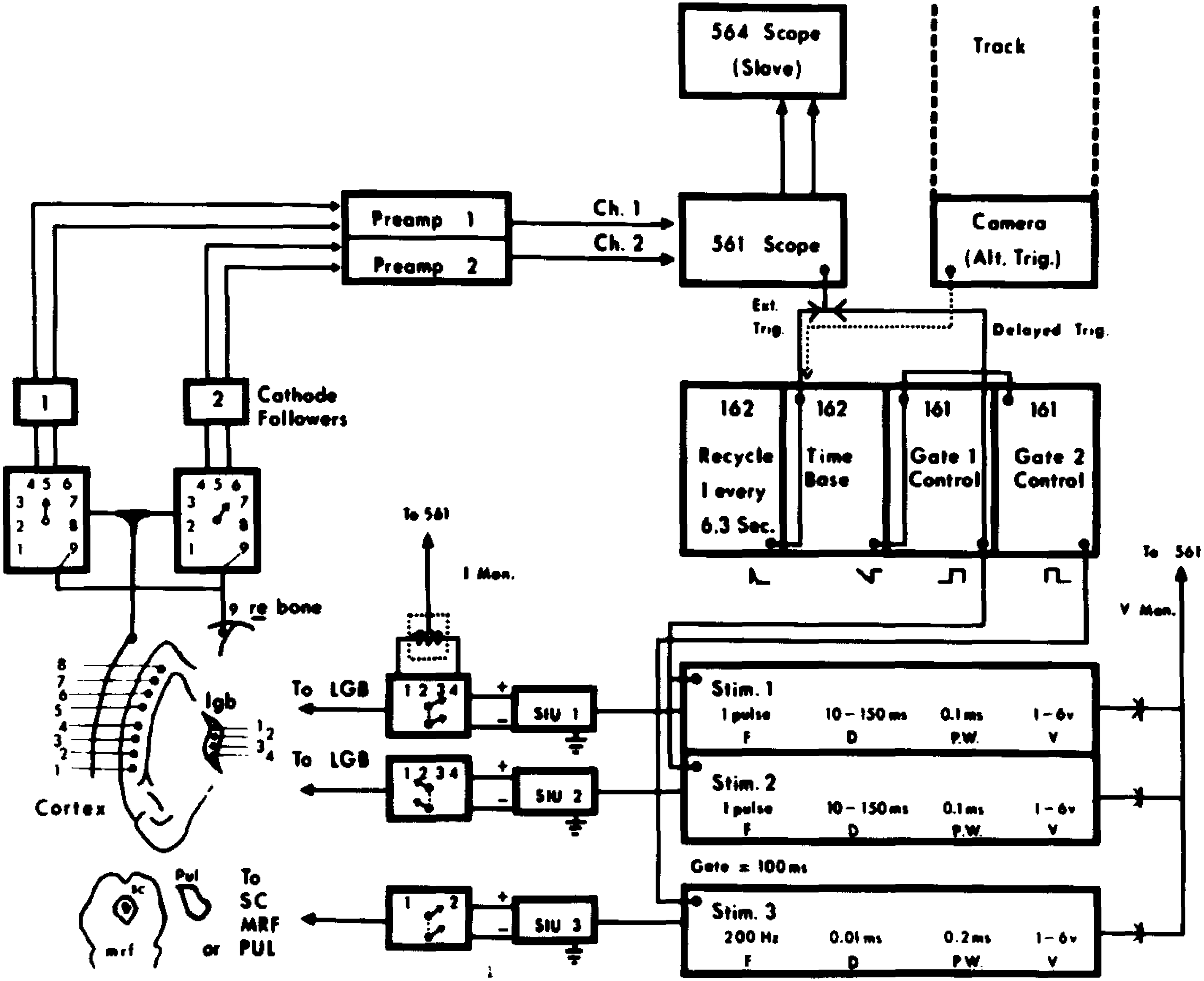
At the end of each experiment, the animal was sacrificed with an overdose of Nembutal (I.V.), subcortical loci were anodally fulgurated with 0.1 to 0.4 milliamperes for 15 to 20 seconds, and the brain removed and placed in a 10 per cent buffered, formalin solution. Frozen, coronal sections were subsequently cut at 50 micra thickness and every fifth or tenth section stained with the Klüver-Barrera

(1953) stain.

Apparatus. The timing control for all stimulation and recording was accomplished using Tektronix Waveform (162) and Pulse (161) Generators and Grass Stimulators, as outlined in Fig. 2. A waveform generator, acting as a recycling timer, triggered another waveform generator every 6.3 seconds whose sawtooth output provided the time base for two subsequent pulse generators. The first pulse generator gated on two Grass S-4 stimulators each of which could be switch-selected to stimulate any one of four loci in LGB. The onset of this gate was also used to provide a delayed trigger to the CRO. The second pulse generator gated a Grass S-4 stimulator to produce stimulation of either SC, MRF, or Pul depending upon switch selection. The LGB stimulus was a 0.1 millisecond duration square pulse of 1.0 to 6.0 volts delivered to either one or two spatially discrete LGB loci. Activation of SC, MRF, or Pul consisted of a brief, 20 pulse train (200 hertz) of 0.1 millisecond duration square pulses and an intensity of 1.0 to 6.0 volts for MRF, 2.0 to 6.0 volts for SC, and 3.0 to 6.0 volts for Pul, each being independently varied for maximum effect as described below.

The cortical activity at any two of the eight recording sites was switch selected and fed through two differential Grass AC preamplifiers (-3 dB at 7 cps and 10 kc) and displayed on a split-beam oscilloscope (Tektronix 561) and a slave storage scope (Tektronix 564). Five superimposed traces were recorded and photographed with a Kymograph camera.

Fig. 2 Flow diagram of stimulating and recording equipment. LGB, lateral geniculate body; SC, superior colliculus; MRF, mesencephalic reticular formation; Pul, pulvinar; SIU, stimulus isolation unit; I Mon., current monitor; Stim., stimulator; F, frequency; D, duration; P.W., pulse width; V. volts; V Mon., voltage monitor; ms, milliseconds.


564 Scope
(Slave)
Track
Preamp 1
Preamp 2
Ch. 1
Ch. 2
561 Scope
Camera
(Alt. Trig.)
Ext. Trig.
Delayed Trig.
1
2
Cathode Followers
4 5 6
3 7
2 8
1 9
162
Recycle
1 every
6.3 Sec.
162
Time Base
161
Gate 1 Control
161
Gate 2 Control
To 561
I Mon.
To 561
V Mon.
9 re bone
8 7 6 5 4 3 2 1
lgb
1 2 3 4
Cortex
To LGB
1 2 3 4
To LGB
1 2 3 4
+
−
SIU 1
SIU 2
SIU 3
Stim. 1
1 pulse F
10 – 150 ms D
0.1 ms P.W.
1 – 6v V
Stim. 2
1 pulse F
10 – 150 ms D
0.1 ms P.W.
1 – 6v V
Gate = 100 ms
Stim. 3
200 Hz F
0.01 ms D
0.2 ms P.W.
1 – 6v V
1 2
sc
Pul
mrf
or
To SC MRF or PUL

Experimental Procedure. Experiments were conducted at least 30 minutes after the withdrawal of Halothane. The LGB stimulating comb was advanced medially in 0.5 millimeter steps until stimulation at each of two or more stimulating sites produced cortical evoked potentials. The SC-MRF-Pul electrodes (all on the same carrier) were then lowered in 1.0 millimeter steps until activation of each of the three subcortical loci with the brief 20 pulse train produced the maximal enhancement of the LGB shock-evoked cortical potential with minimal stimulating voltage. Two physiological criteria were utilized in positioning the depth electrodes: (a) pupillary dilatation was found to be most marked (of large amplitude and short latency) when the area ventral to the superior colliculus and extending caudally into the MRF was stimulated. Stimulation of SC, in contrast, was found to produce little (of slight magnitude and longer latency), if any, pupillary dilatation, while activation of the pulvinar produced none; (b) the Pul placement in most animals could be verified by the elicitation of large augmenting responses in the postero-lateral or suprasylvian gyri upon low frequency stimulation.

The delay between the end of the activating train to SC, or MRF and onset of the LGB test shock (S_t) was constant for any experiment and varied for different preparations from 25 to 50 milliseconds depending upon the interval producing maximal enhancement of the shock evoked potential. In experiments where the effects of Pul activation were compared with those of SC or MRF an interval of 15 milliseconds was used for all conditions.

In 11 cats where spatial interaction in cortex was examined, two LGB shocks, the first a conditioning (S_c) and the second a test (S_t) shock, were always delivered to two different loci in LGB, separated by at least 2 millimeters. The delay between S_c and S_t ranged from 5 to 200 milliseconds. The effect of activating either SC, MRF, or Pul on the response to S_t alone or when preceded by S_c was examined for each S_t delay.

In 15 cats the effects of subcortical lesions of SC, MRF, or Pul were examined. These lesions were made, usually in successive stages, by anodally fulgurating for 30 seconds the SC with 2 to 4 milliamperes and the MRF or Pul with 4 to 5 milliamperes. Halothane anesthesia was always increased to 1 to 2 per cent prior to such fulgurations, then terminated, and the effects of stimulating the remaining structures retested 10 to 20 minutes later. In some experiments, in addition to subcortical lesions, 5 per cent KCl-soaked filter paper was topically applied to the marginal and posterolateral gyri contralateral to the cortical recording region. Surrounding strips of 5 per cent $MgCl_2$-soaked filter paper were used to restrict the spread of KCl.

Analysis of the data. In each cat, the peak to peak amplitude of the response elicited by LGB shock alone (S_t) was read from the filmed data of 5 superimposed tracings before and after subcortical activation. This corresponds with the amplitude of the peak positive to peak negative deflection (components

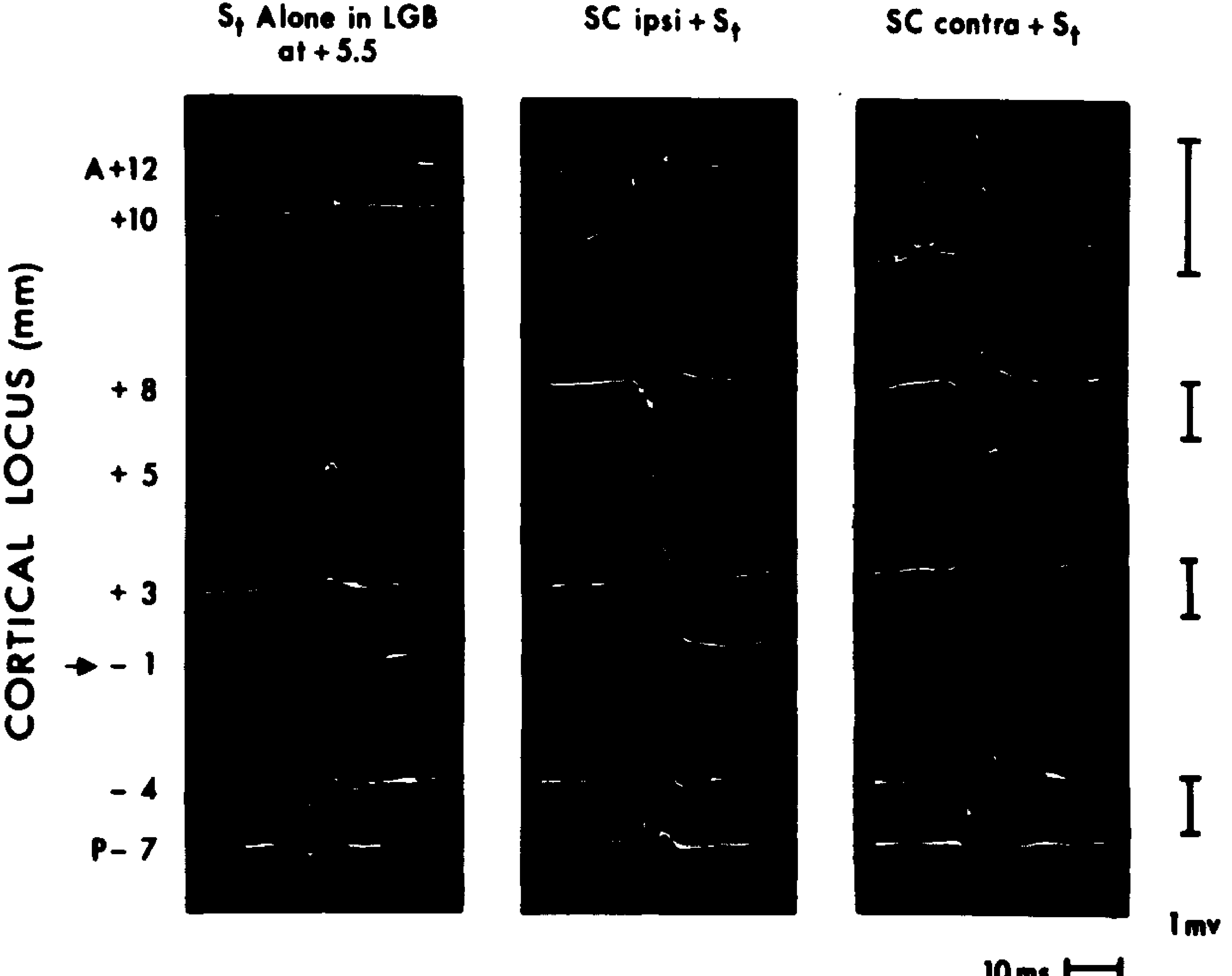
4 to 5 of Malis and Kruger (1956)), and usually fell in the range of 50 microvolts to 5 millivolts. In most cases, the amplitude of this test response when preceded by subcortical activation (SC, MRF, or Pul) was then expressed as a per cent of control, i.e., the test response amplitude without prior subcortical activation was considered 100 per cent.

RESULTS

<u>Influence of superior collicular activation on the cortical gradient of responses elicited by LGB shock</u>. In 34 animals the influence of activating the ipsilateral (N=26) or contralateral (N=24) superior colliculus on the spatial distribution of LGB shock-evoked responses across cortex was examined. The results for Cat 285 (Figs. 3 and 4) are representative of the findings for all animals. Figure 3 illustrates with oscilloscope tracings the spatial distribution of evoked responses to LGB stimulation alone at Fr. 5.5 (left column) or when preceded by activation of the ipsilateral (middle column) or contralateral (right column) superior colliculus. In general, activating the colliculus on either side produced a marked enhancement in the peak to peak amplitude of the test response at all cortical locations, with the largest percentage increase, however, occurring at submaximal locations. Figure 4 presents the complete data for Cat 285 in graphical form. Four families of curves are given, each of them representing a different stimulating locus in LGB (Fr. 8.5, 7.5, 6.5, 5.5). As indicated, the maximal response in cortex (indicated by the arrow on the abscissa) shifts posteriorly as the stimulating locus in LGB shifts posteriorly from Fr. 8.5 to Fr. 5.5, an observation consistent with the topological organization of the geniculo-cortical system as recently described by Storck et al (1972). Figure 4 also illustrates the variety of spatial gradients observed after stimulating different LGB loci.

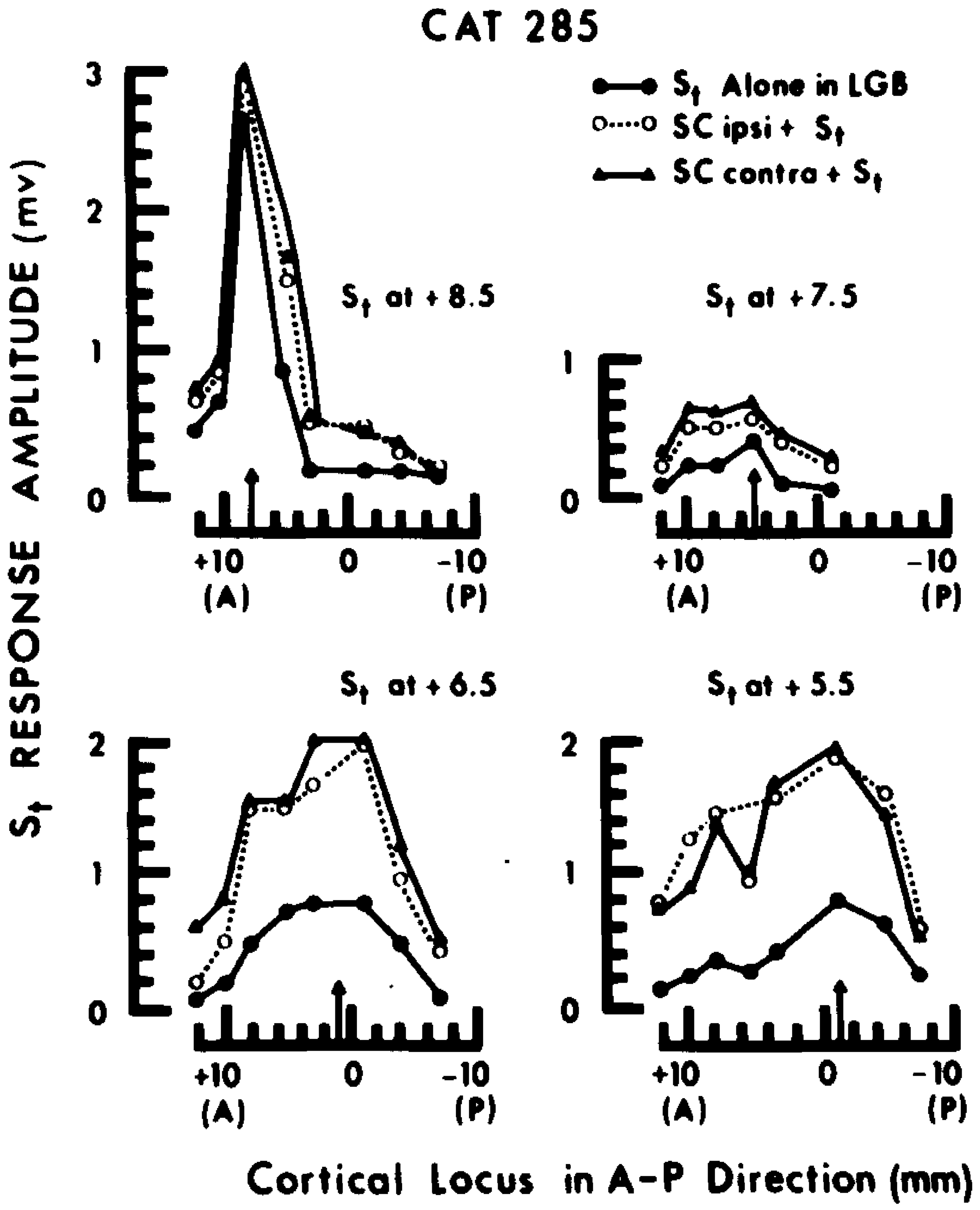
Fig. 3 The responses across cortex to a single shock at Fr. 5.5 in LGB (left column of tracings) and enhancement of responses produced by ipsilateral (middle column) or contralateral (right column) SC activation. The arrow at the left indicates the location of the maximal cortical response. Note change in gain calibrations at right. Surface negativity indicated by upward deflection in this and all subsequent tracings.

CAT 285



S_t Alone in LGB at + 5.5
SC ipsi + S_t
SC contra + S_t
CORTICAL LOCUS (mm)
A+12
+10
+ 8
+ 5
+ 3
- 1
- 4
P- 7
1 mv
10 ms

Fig. 4 Ipsilateral and contralateral SC enhancement of responses across cortex for four different cortical gradients obtained by stimulating four different LGB loci (Fr. 8.5, 7.5, 6.5, 5.5). In each graph the arrow on the abscissa indicates the location of the maximal cortical response.

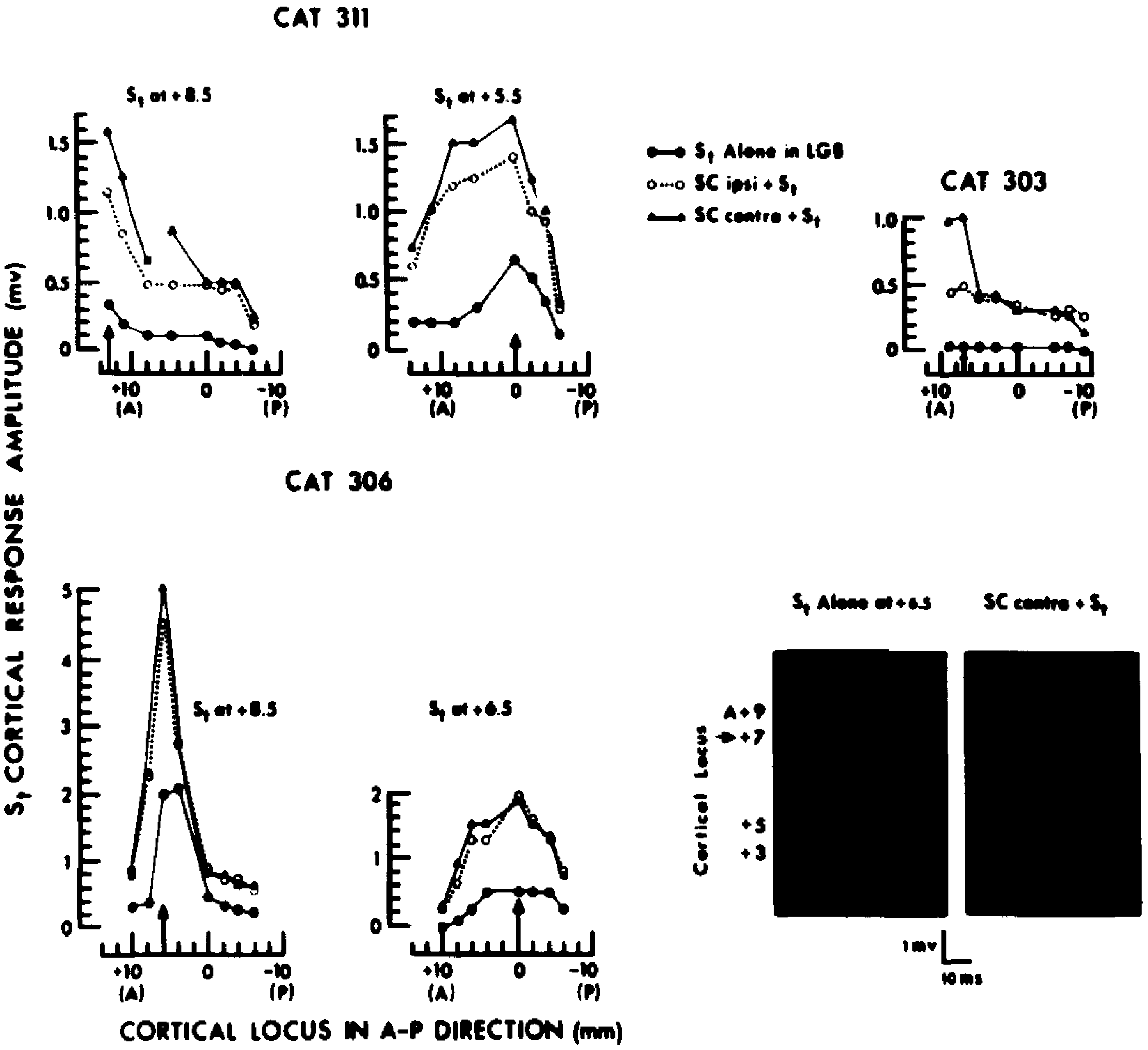

CAT 285
S$_t$ Alone in LGB
SC ipsi + S$_t$
SC contra + S$_t$
S$_t$ RESPONSE AMPLITUDE (mv)
S$_t$ at + 8.5
S$_t$ at + 7.5
S$_t$ at + 6.5
S$_t$ at + 5.5
0
1
2
3
+10
(A)
0
-10
(P)
Cortical Locus in A-P Direction (mm)

One LGB site elicited a relatively peaked distribution on cortex (Fr. 8.5) while all others produced broader cortical gradients (Fr. 7.5, 6.5, 5.5). In all cases, however, activation of either colliculus increased test response amplitude at all cortical locations, both at maximal and submaximal loci.

The variety of cortical gradients elicited with LGB stimulation is further illustrated in Fig. 5. In the case of both Cats 311 (top left) and 306 (bottom left) it was found that one of two stimulating loci in LGB produced a peaked spatial gradient while the other stimulating locus produced a broader spatial gradient. Once again, however, activation of the superior colliculus on either side enhanced the test response at all cortical locations, the greatest relative enhancement occurring at submaximal locations. With Cat 303 (right side of Fig. 5) stimulating at Fr. 7.0 in LGB produced a flat distribution of low amplitude, longer latency responses across all cortex. In this case ipsilateral or contralateral collicular activation increased S_t response amplitude at all cortical locations, but in so doing produced a spatial gradient with a well defined maximal cortical locus at A+7.0. This is illustrated in the oscilloscope tracings below. Similar results were obtained in all eight animals in which the effects of SC activation on intrinsically flat types of spatial distributions were examined.

The relative enhancement of LGB elicited cortical responses for both peak and flat distributions can be best

Fig. 5 The effects of ipsilateral and contralateral SC activation on peak and broad distributions of LGB elicited responses (Cats 311 and 306) and for a flat distribution of responses (Cat 303). Sample tracings for Cat 303 are shown at right.


CAT 311
S_1 at + 8.5
S_1 at + 5.5
S_1 Alone in LGB
SC ipsi + S_1
SC contra + S_1
CAT 303
CAT 306
S_1 at + 8.5
S_1 at + 6.5
S_1 Alone at + 6.5
SC contra + S_1
A + 9
+ 7
+ 5
+ 3
Cortical Locus
1 mv
10 ms
S_1 CORTICAL RESPONSE AMPLITUDE (mv)
CORTICAL LOCUS IN A-P DIRECTION (mm)
1.5
1.0
0.5
0
5
4
3
2
1
+10
(A)
0
-10
(P)

analyzed if the data are normalized by plotting response amplitude after SC activation as a percentage of the control (test response to S_t alone) amplitude. The above data in Fig. 5 are represented in this manner in Fig. 6 for Cats 311 and 303. These data show that in the peak distributions for Cat 311 (top), the greatest proportion of enhancement after either ipsilateral or contralateral SC activation occurred at the submaximal cortical locations. For each stimulating site in LGB, Fr. 8.5 and Fr. 5.5, the proportion of enhancement tended to increase with increasing distance from the maximal locus (indicated by the arrow on the abscissa), reached a maximum value (at P-4 for LGB locus Fr. 8.5 and at A+8 for LGB locus Fr. 5.5) and then decreased slightly. In the flat cortical distribution of LGB elicited cortical responses for Cat 303 (bottom), the greatest proportion of enhancement produced by ipsilateral or contralateral SC activation occurred at the maximal cortical location (A+7), and then declined with increasing distance from the maximal cortical location.

Table 1 presents the indidence of histologically identified locations in LGB in terms of A-P plane (Fr. 4.0 to 9.0) and lamina (A or A', B) for two classes of distributions, those judged to be relatively peaked (at the top) and those judged to be flat (at the bottom). In general, LGB loci producing peak gradients were located throughout the A-P extent of LGB but tended to be somewhat anterior (around Fr. 7.0). Of the 43 tabulated, 35 were clearly

Fig. 6 Data taken from Fig. 5 for Cats 311 and 303 are normalized so that response to test shock (S_t) following SC activation is expressed as a percentage of control (response to S_t alone). Arrow on abscissa in each graph indicates location of maximal cortical response. Proportionate enhancement across cortex depends upon type of distribution: peak (Cat 311) or flat (Cat 303) as originally illustrated in Fig. 5.

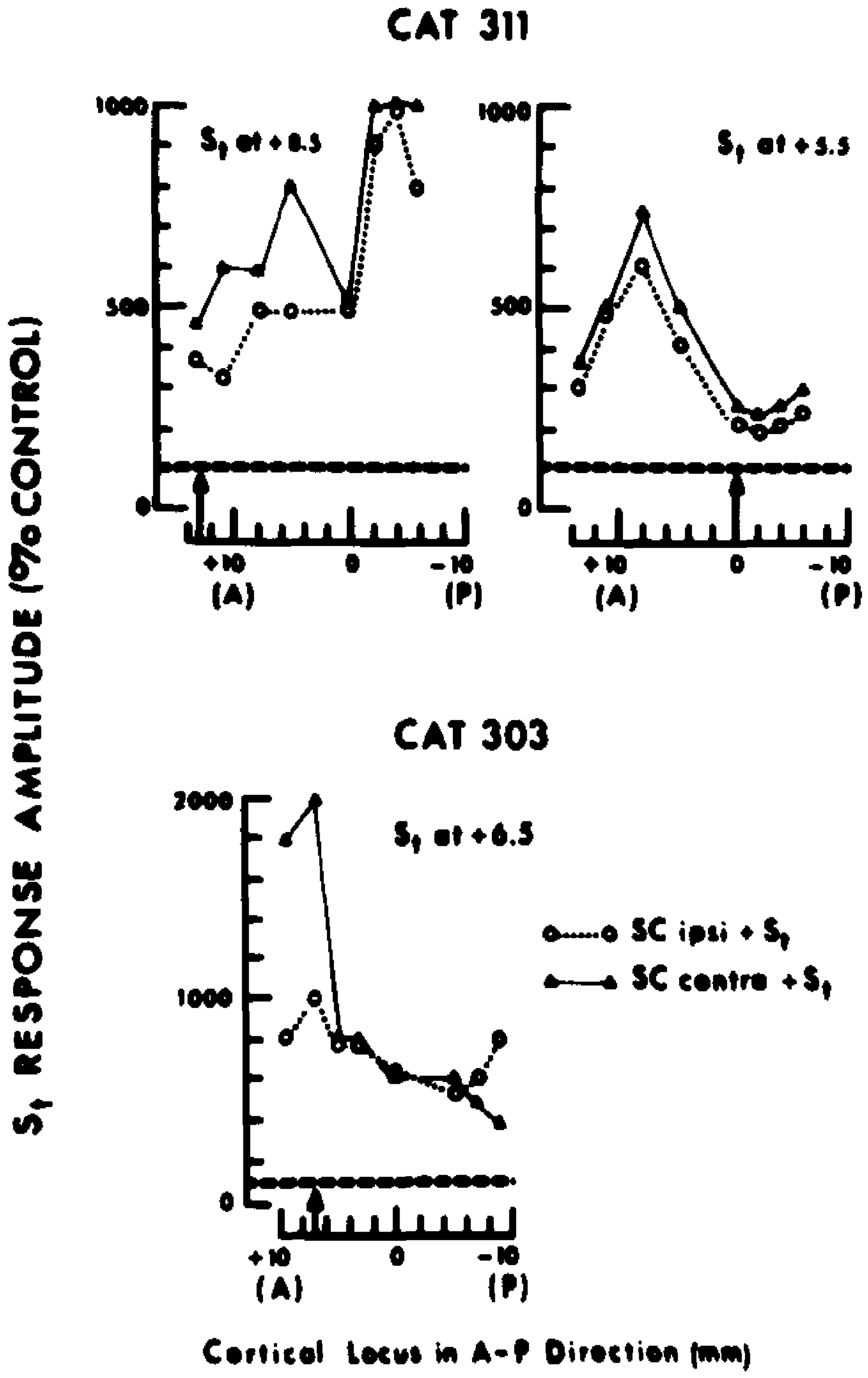

CAT 311
1000
500
0
S₁ at +8.5
S₁ at +5.5
+10
0
-10
(A)
(P)
CAT 303
2000
1000
0
S₁ at +6.5
SC ipsi + S₁
SC contra + S₁
S₁ RESPONSE AMPLITUDE (% CONTROL)
Cortical Locus in A-P Direction (mm)

TABLE ONE

Incidence of LGB Electrode Sites Yielding Peak or Flat Distributions by A-P Plane and Lamina

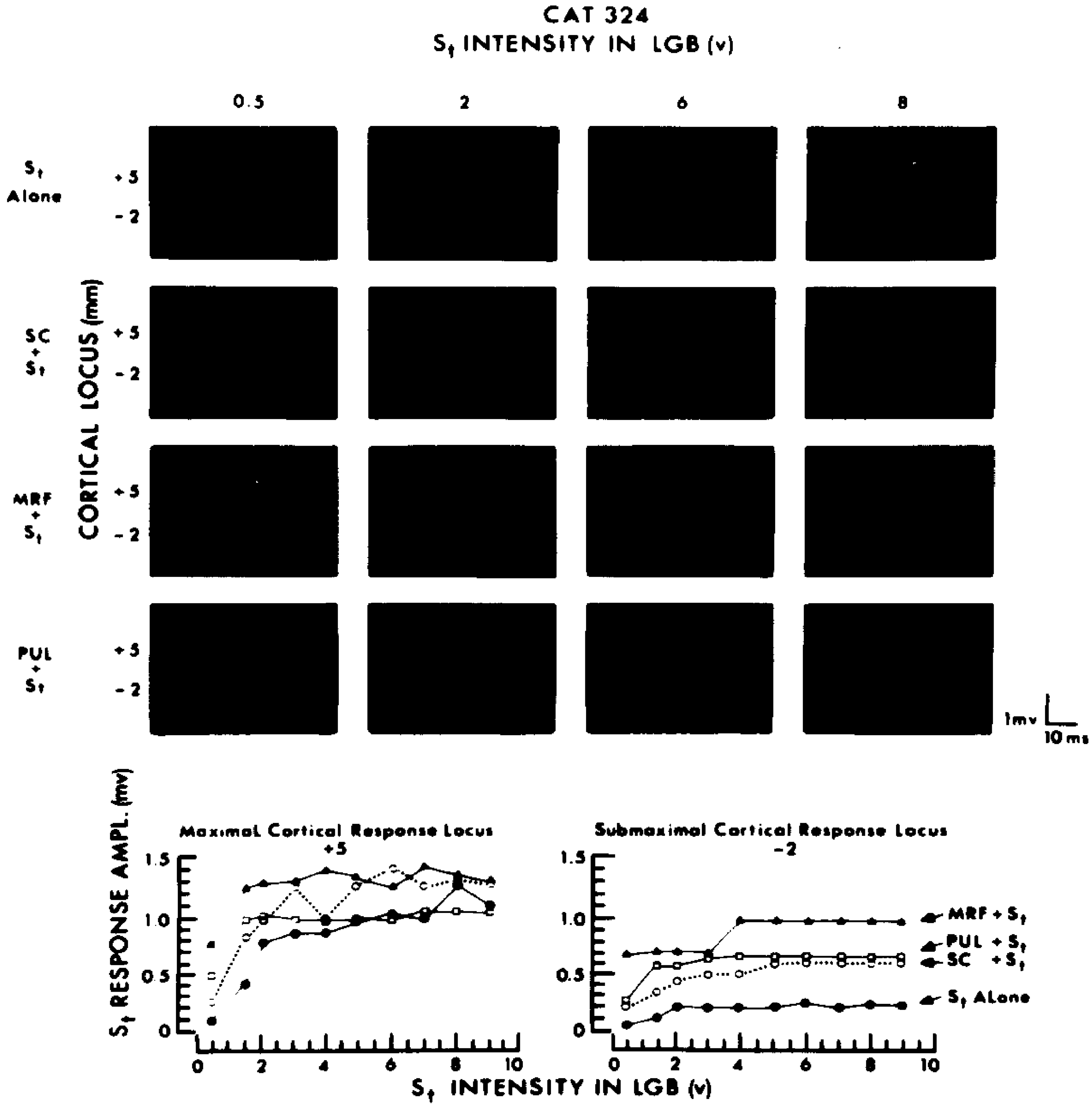
Distribution	A-P Plane						Lamina		
	+4.0	5.0	6.0	7.0	8.0	9.0	A/A'	B	Unclassified
Peak (N=43)	1	6	8	12	11	5	35	2	6
Flat (N=18)	1	7	7	3	0	0	1	11	7

identified as being in lamina A or A', only 2 in lamina B, 6 unclassifiable because electrolytic markings were too large, invading more than one lamina. LGB loci producing flat distributions, on the other hand, tended to be posteriorly (and ventrally) located in LGB, and of the 18 tabulated, 11 were clearly identified as being in lamina B, 1 in lamina A, and 7 others being unclassified because of reason noted above. In general, it would appear that the projections from A or A' are more topologically specific than those from lamina B (see Discussion below).

In summary, it was observed that the spatial distribution of cortical responses evoked by LGB shock could be either peaked or flat, with the maximal response in cortex, when present, being topologically related to the site of LGB stimulation. Any combination of distributions could be obtained in the same animal (eg., Cat 285), the nature of the distribution depending in part upon the exact site in LGB stimulated. For all types of spatial distributions, activation of either the ipsilateral or the contralateral colliculus increased the test response amplitude at both maximal and submaximal cortical locations, with the greatest proportionate enhancement occurring at submaximal locations in peaked distributions, and at maximal locations in flat distributions.

Influence of stimulus intensity on enhancement of maximal and submaximal responses. Since strength of test shock has been found to be an important variable determining degree of enhancement (Chalupa et al, 1973), the influence of stimulus intensity was systematically investigated in four animals. The findings for all animals were similar and are illustrated in Fig. 7 for Cat 324. Cortical responses to a test shock at varying voltages in LGB (given in column headings at top), are shown for LGB test shock alone (top row) and when preceded by activation of SC (second row), MRF (third row) or Pul (bottom row). At both the maximal (top tracings marked +5) and submaximal (bottom tracings marked -2) locations in cortex, response amplitude increases with increments in shock intensity, with the magnitude of this increase being greater at the maximal locus. Activation of SC, MRF, or Pul enhanced the magnitude of the response to the test shock, but the greatest effect was at the submaximal locations. These effects are more clearly indicated in the two graphs at the bottom of Fig. 7 which show that SC, MRF, or Pul activation produced an increase in response amplitude much greater than control (S_t alone) at the submaximal locus (marked -2 at right) than at the maximal locus (marked +5 on the left) in cortex; i.e., percentage of increase was greater at submaximal loci. In most experiments, the LGB stimulating voltage was set at a value just sufficient to produce a maximum amplitude response. This criterion usually meant that the LGB shock intensity was at or near the upper limit of the dynamic range and hence the proportion of enhancement due to subcortical activation was consistently smaller or completely absent at the maximal locus, while being proportionately larger at submaximal locations.

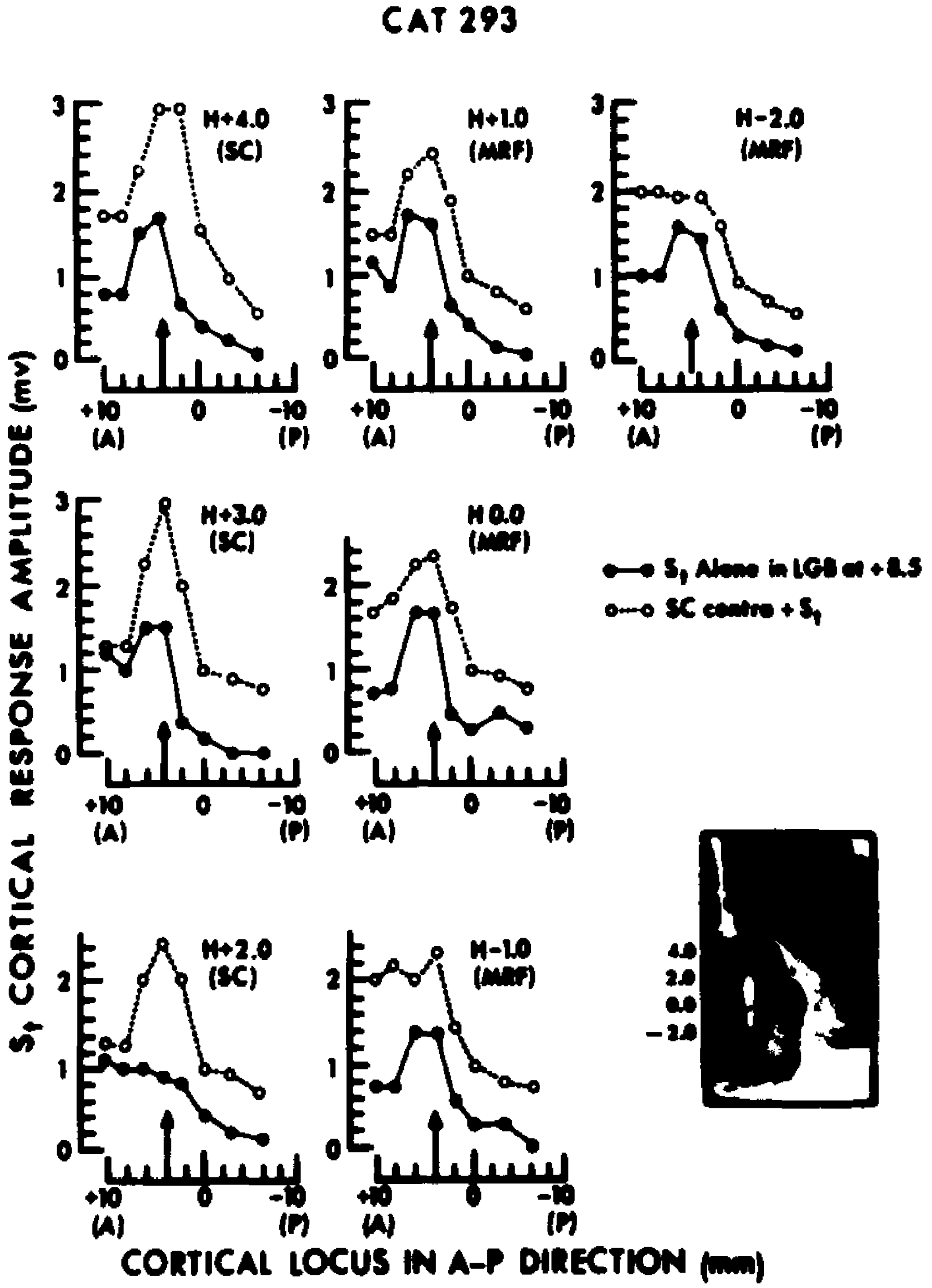
Fig. 7 Effects of SC, MRF, and Pul activation on a maximal (+5) and submaximal (-2) cortical response evoked by four S_t shock intensities ranging from 0.5 to 8.0 volts.


CAT 324
S_t INTENSITY IN LGB (v)
0.5
2
6
8
S_t Alone
SC + S_t
MRF + S_t
PUL + S_t
CORTICAL LOCUS (mm)
+5
-2
1mv
10 ms
S_t RESPONSE AMPL. (mv)
Maximal Cortical Response Locus +5
Submaximal Cortical Response Locus -2
0
0.5
1.0
1.5
0
2
4
6
8
10
MRF + S_t
PUL + S_t
SC + S_t
S_t Alone
S_t INTENSITY IN LGB (v)

Midbrain levels influencing the spatial distribution of shock-evoked responses. In the present study the influence of MRF activation on the spatial distribution of cortical responses evoked by LGB shock was examined in 21 animals. Activation of either the ipsilateral (N=19) or contralateral (N=13) MRF was found to influence the spatial distribution of evoked responses in a manner similar to that produced by SC activation. That is, for peak distributions elicited by near maximal intensity shocks to LGB, the proportion of enhancement at submaximal locations in cortex was greater than at the maximal location. Conversely, for flat distributions the proportion of enhancement was greater at the maximal location in cortex.

The most dorsal portion of the MRF just ventral to SC is known to carry fibers which originate in ventral layers of SC and terminate in different levels of the MRF, i.e., tectoreticular and tectospinal collateral fibers (Altman and Carpenter, 1961). In 8 animals, the effects of activation of this area on the cortical gradient was compared with SC or MRF activation. The data in Fig. 8 for Cat 293 is representative of the results obtained for all animals. The left LGB was stimulated to produce a distribution of responses across the left marginal gyrus, and the right midbrain area was activated electrically to produce enhancement. The midbrain electrode was lowered in 1 millimeter steps from the level of the superior colliculus to the MRF, and the spatial distribution of test responses alone and when preceded by midbrain activation

Fig. 8 Effects of activation of different levels of the contralateral midbrain extending from H+4.0 in SC to H-2.0 in MRF. Arrow on abscissa indicates location of maximal cortical response. Dorsal MRF levels: H+1.0 and H0.0; Ventral MRF levels: H-1.0 and H-2.0. Microphotograph at right indicates the tract of the electrode which is unusually large due to the tubing surrounding the electrode, electrolytic markings, and additional damage in withdrawing the electrode.


CAT 293
S1 CORTICAL RESPONSE AMPLITUDE (mv)
CORTICAL LOCUS IN A–P DIRECTION (mm)
H+4.0 (SC)
H+1.0 (MRF)
H-2.0 (MRF)
H+3.0 (SC)
H 0.0 (MRF)
H+2.0 (SC)
H-1.0 (MRF)
0
1
2
3
+10 (A)
0
-10 (P)
S1 Alone in LGB at +8.5
SC contra + S1
4.0
2.0
0.0
-2.0

were examined; intensity of activation was constant for all conditions. As indicated in the graphs for each level of activation (from H=4.0 to H-2.0), enhancement of the test response occurred at all cortical locations when the contralateral midbrain was activated at all levels indicated above. Moreover, the magnitude of this enhancement effect did not materially differ between SC and dorsal and ventral portions of MRF. Tetanization of SC rarely produced any pupillomotor responses, however, but such responses occurred in many instances as the midbrain electrode was lowered to approach H0.0, i.e., the MRF. Comparing the effects of SC and MRF across a group of 37 cats, it was noted that slightly higher stimulating voltages were usually necessary to produce SC enhancement as compared to MRF. The mean stimulating intensity for SC (N=37) was 4.3 volts (range 2.0 to 6.0), while that for MRF (N=21) was 3.2 volts (range 1.0 to 6.0). These differences were considered insignificant.

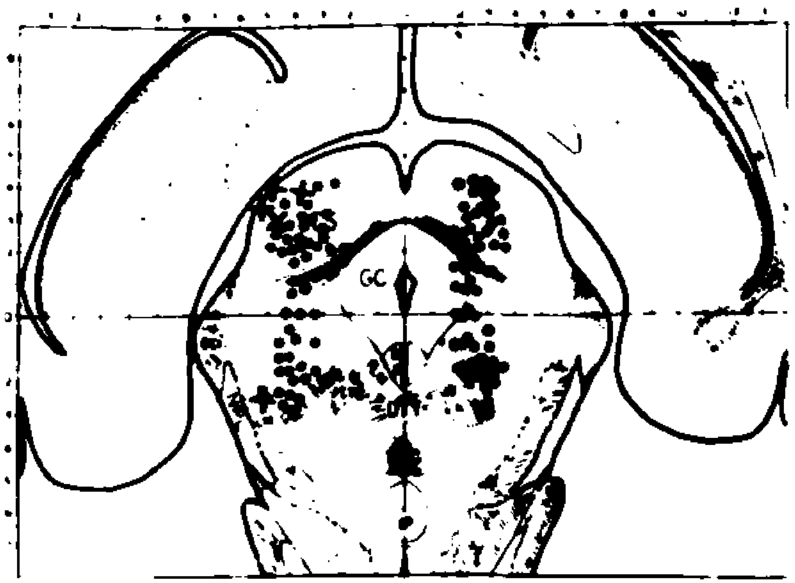
Figure 9 (left side) shows the histologically verified electrode locations in the midbrain used in the present study. Note that positive loci within the superior colliculus are distributed throughout the intermediate and ventral layers, meaning that these levels of SC were usually found to be the most effective sites for enhancing the LGB shock response, when the criterion used was the minimal intensity of tetanization necessary to produce maximal enhancement. Negative loci in SC, where they were verified histologically, were found to be more laterally and dorsally situated. However, no concerted

Fig. 9 Coronal sections from the cat brain atlas of Jasper and Ajmone-Marsan (1954) at Fr. 2.0 (left) and Fr. 6.0 (right). Stimulating at positive loci (closed circles) resulted in enhancement of the evoked potential while stimulating at negative loci (x's) failed to enhance the evoked potential.

EVOKED POTENTIAL ENHANCEMENT

SC and MRF

N=36
Fr. 2.0

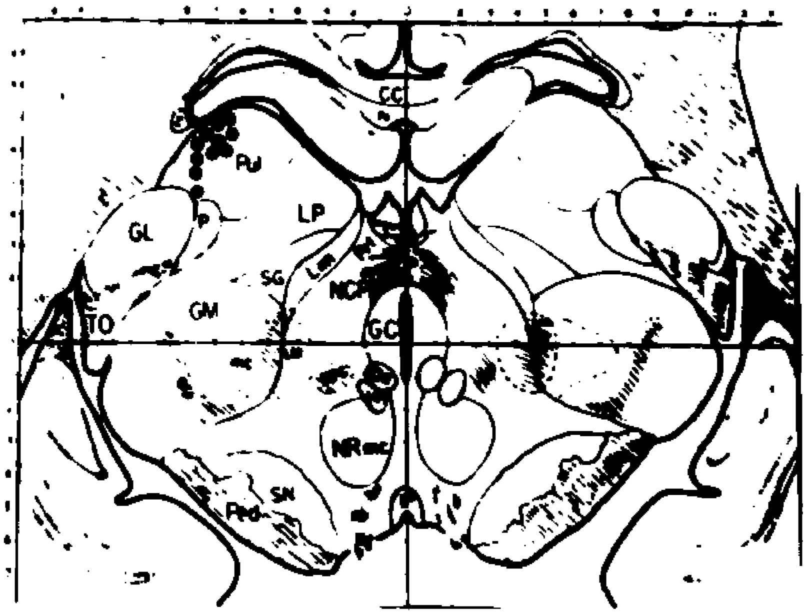


99+Sites = ●

5 −Sites = x

Pulvinar

N=9
Fr. 6.0

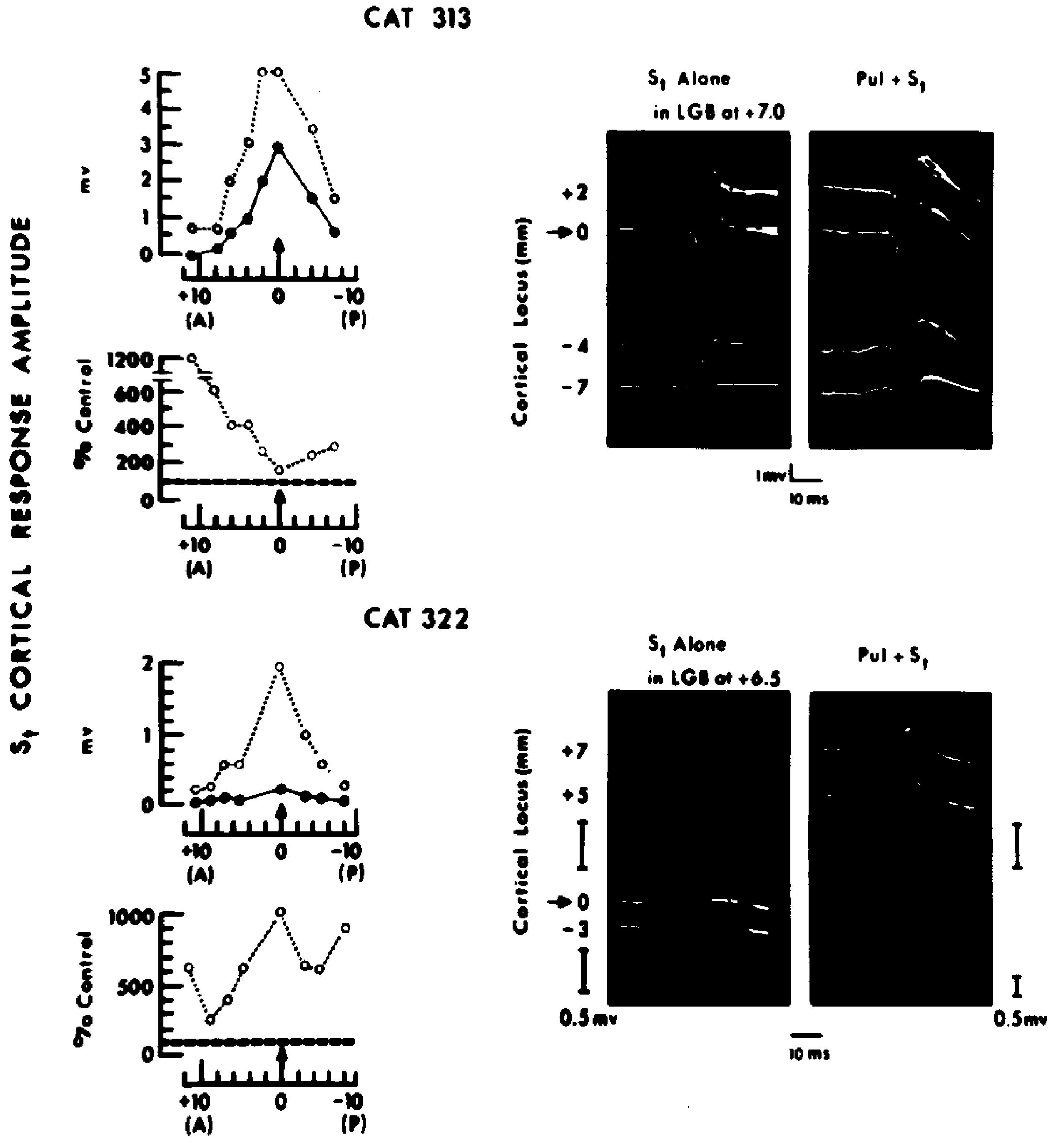


11+Sites = ●

effort was made to histologically verify all negative loci. In one animal, however, tetanization of the inferior colliculus and the area ventral to it (not shown in Fig. 9) failed to produce any enhancement of the LGB shock response.

Influence of Pulvinar activation on the cortical gradient of responses elicited by LGB shock. In 10 cats the influence of stimulating the ipsilateral pulvinar on the spatial distribution of shock evoked potentials was examined. Since similar findings were obtained in all animals, the results for only two of them are shown in Fig. 10. For Cat 313 (top) stimulation of LGB at Fr. 7.0 produced a steep spatial gradient in cortex with a maximal locus at 0. When preceded by Pul activation S_t response amplitude was increased at all cortical locations, with the greatest proportionate enhancement occurring at submaximal locations. For Cat 322, stimulation at Fr. 6.5 in LGB produced a flat distribution of low amplitude, longer latency responses in cortex. When preceded by Pul activation, however, a steep spatial gradient with a maximal locus at 0 was produced, with the maximal enhancement occurring at this location. These effects are illustrated in the tracings on the right. In general, activation of the pulvinar influences the spatial distribution of the shock evoked potential in the same manner as that observed for SC or MRF. In addition, it was found that the mean stimulating voltage necessary for Pul enhancement was slightly higher (mean at 4.7 volts, range 3.0 to 6.0) than for SC (4.3 volts) and MRF (3.2 volts). These differences were considered insignificant. Figure 9

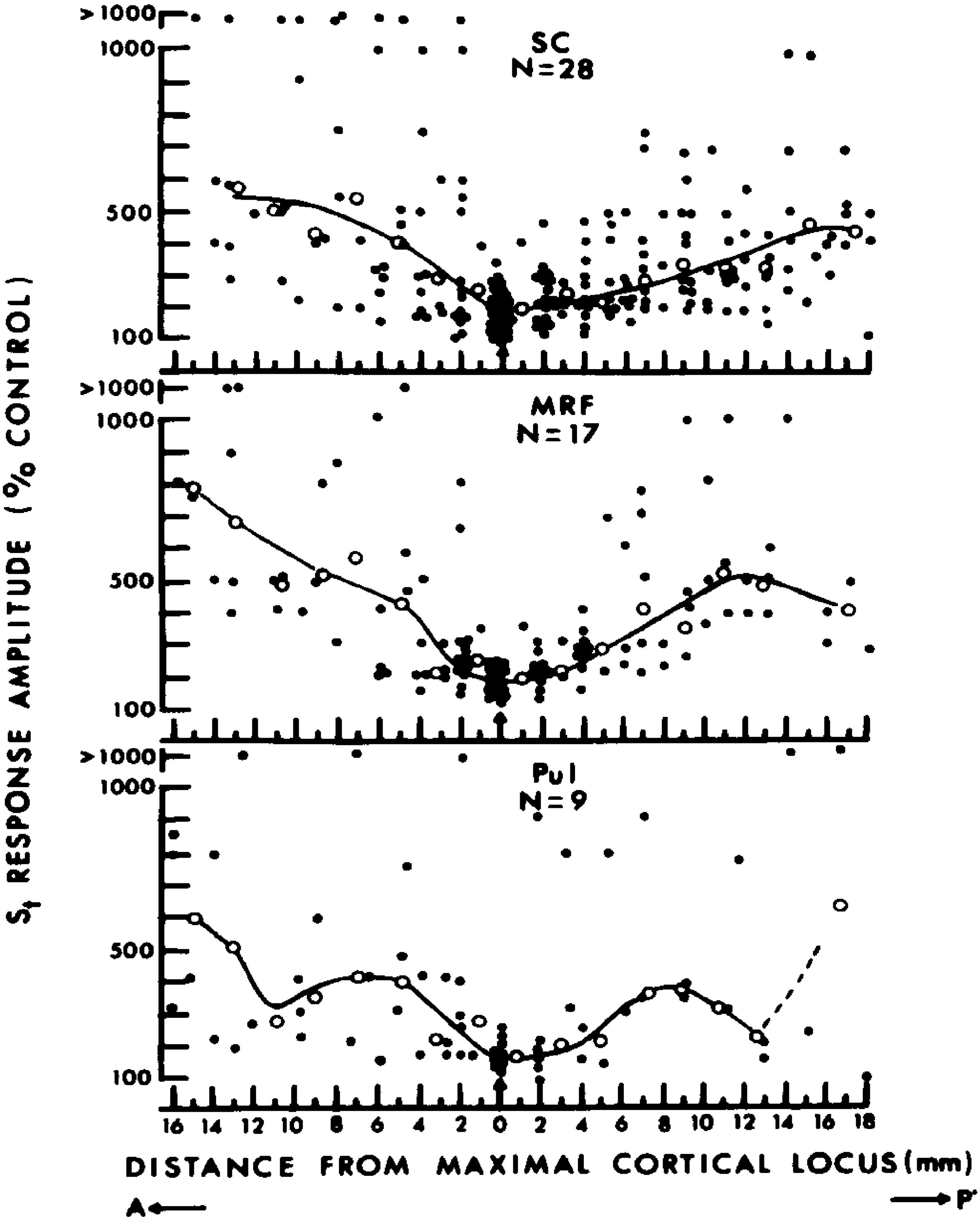
Fig. 10 Comparison of the effects of Pul activation on a peak distribution (Cat 313) and a flat distribution (Cat 322). For each cat, upper graph shows response amplitude to S_t alone (closed circles) and to SC plus S_t (open circles) as a function of cortical location and lower graph (open circles) is the same data normalized (horizontal dashed line indicates 100 per cent control level). Arrow in each graph indicates location of maximal response. Sample tracings are given for each cat (maximal) locus indicated by arrow). Note change in gain calibration in lower tracings.


S_1 CORTICAL RESPONSE AMPLITUDE
CAT 313
mv
5
4
3
2
1
0
+10
(A)
0
-10
(P)
% Control
1200
600
400
200
0
+10
(A)
0
-10
(P)
S_1 Alone
in LGB at +7.0
Pul + S_1
Cortical Locus (mm)
+2
0
-4
-7
1mv
10 ms
CAT 322
mv
2
1
0
+10
(A)
0
-10
(P)
% Control
1000
500
0
+10
(A)
0
-10
(P)
S_1 Alone
in LGB at +6.5
Pul + S_1
Cortical Locus (mm)
+7
+5
0
-3
0.5mv
10 ms
0.5mv

(right side) shows the histologically verified positive electrode tetanization sites within the Pul that were found effective in producing enhancement in the present study.

In an effort to present a summary of all the data, the three scattergrams shown in Fig. 11 were constructed from the individual data of all animals (number of animals indicated by N), in which the influence of SC (top), MRF (middle) or Pul (bottom) activation on peak cortical gradients was examined. The relative magnitude of response enhancement is plotted on the ordinate (in per cent of control) and cortical location is on the abscissa in terms of distance in millimeters from the location of maximal response (indicated by arrow at 0). Each closed circle on the scattergram indicates for each animal the proportionate enhancement obtained at any given cortical location. The median of all these points (within a class interval of 2 millimeters along the abscissa) was then picked by inspection (open circles), and a best fitting curve hand fit to the medians. A comparison of these curves indicates a similar trend in the effects of SC, MRF, or Pul activation on cortical gradients. In each case, the proportion of enhancement at submaximal response locations is always greater than at the maximal location. In addition, for SC and MRF activation the proportionate enhancement increases with distance from the maximal locus. The irregular nature of some of these curves, particularly the Pul data at the extremes, may

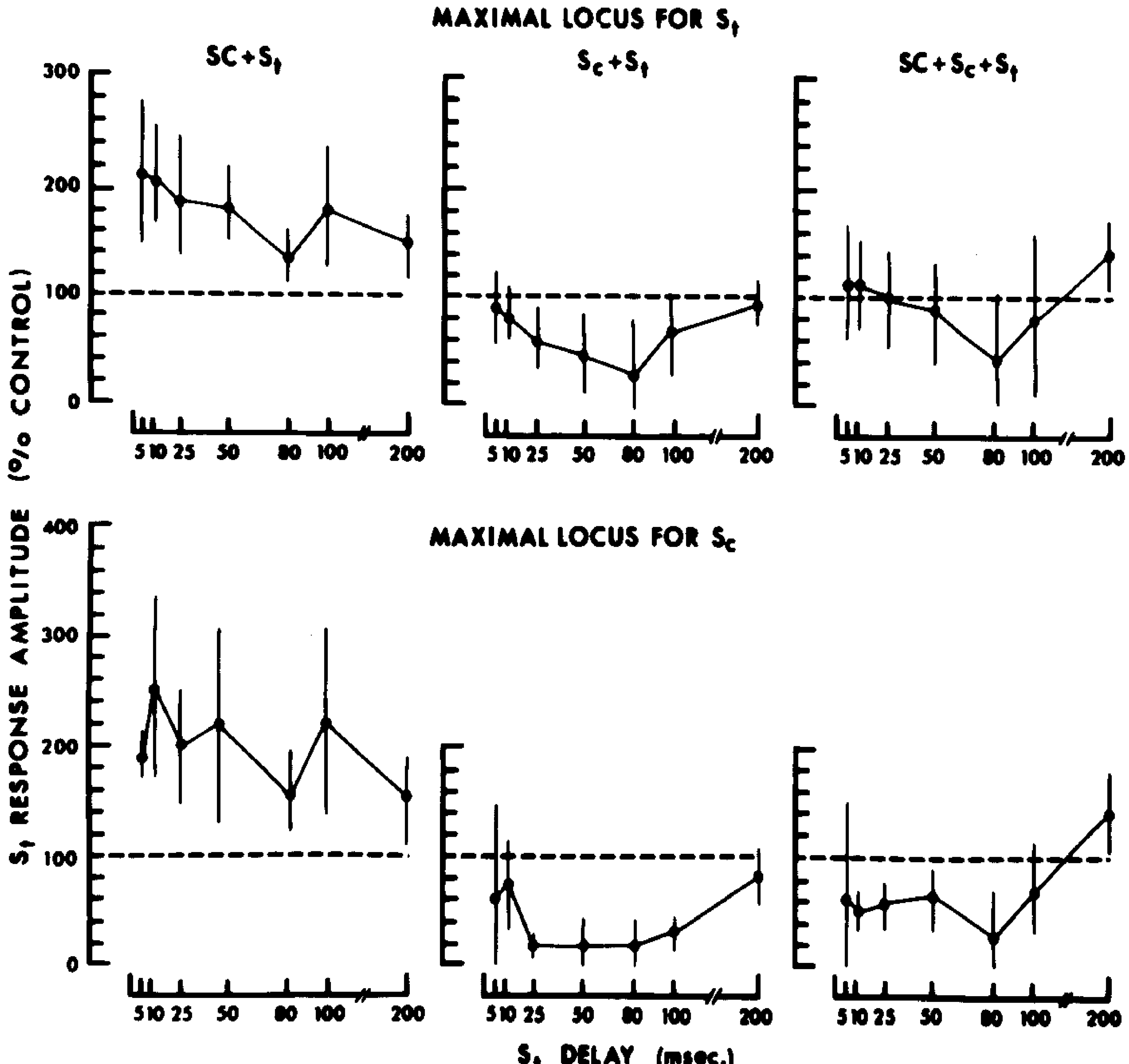
Fig. 11 Group data normalized to compare the effects of SC, MRF, and Pul activation on peak distributions. See text for details.


SC
N=28
MRF
N=17
Pul
N=9
>1000
1000
500
100
S_t RESPONSE AMPLITUDE (% CONTROL)
16 14 12 10 8 6 4 2 0 2 4 6 8 10 12 14 16 18
DISTANCE FROM MAXIMAL CORTICAL LOCUS (mm)
A
P

be attributed to the small number of observations or to other factors as yet unknown.

Collicular influences on the spatial interaction of responses in cortex elicited from different LGB sites. The results presented so far describe the effects of SC activation on the distribution of responses in cortex to a single LGB test shock. Recently, Storck et al (1972) have shown that the cortical gradient of response to a single LGB shock (S_t) was influenced by the prior response to a preceding conditioning shock (S_c) delivered at a neighboring locus in LGB. In brief, they found that at the maximal locus of S_c, the response to S_t was usually decreased while at the maximal locus for S_t, the response to S_t could be either increased or decreased depending upon the amplitude of the first (conditioning) response. Since SC activation has also been shown to influence the recovery cycle to two successive shocks at a single LGB locus (Chalupa et al, 1973), the influence of such activation on the cortical gradient of responses elicited from different LGB sites was examined in 11 animals. Specifically, we analyzed the responses in cortex to a LGB test shock (S_t) given alone, and when preceded by SC activation, or by a conditioning shock (S_c) to another LGB site, or to both of these stimuli. The graphs in Fig. 12 show the group data for 11 animals in terms of mean response amplitude expressed as percentage of control (solid circles) and associated fiducial limits ($p<.05$) as functions of S_c to S_t delay for the dual LGB shock condition. Control data was obtained with a single

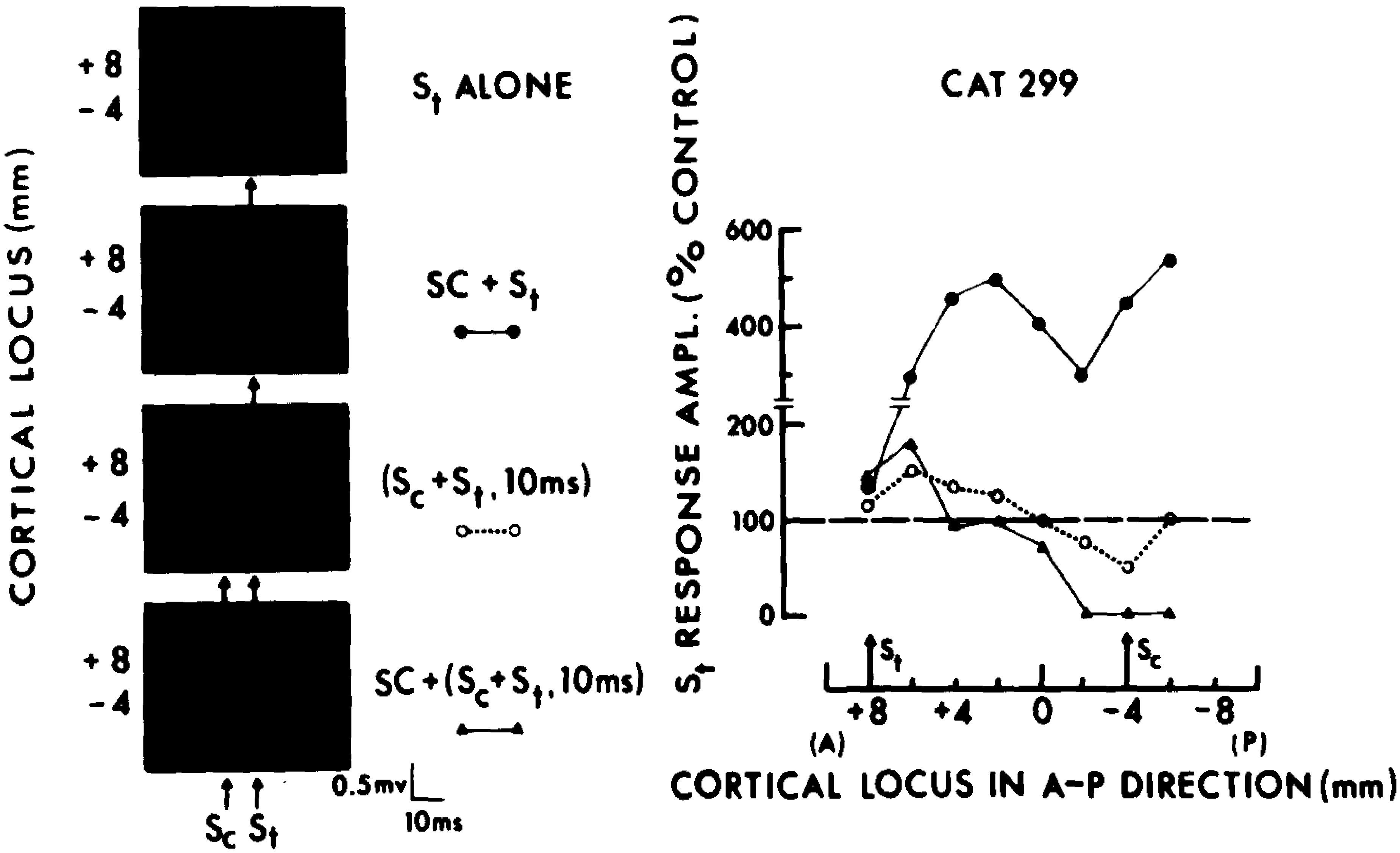
Fig. 12 Spatial Interaction Group Data: Means and fiducial limits ($p<.05$) for test response (per cent of control) when preceded by SC activation (left graphs) or by S_c (middle graphs) or both (right graphs) at the maximal locus for S_t (top graphs) and at the maximal locus for S_c (bottom graphs). Abscissa indicates delay between S_c and S_t. Data for one atypical animal was dropped in computing the bottom sets of graphs.


MAXIMAL LOCUS FOR S_t
$SC+S_t$
S_c+S_t
$SC+S_c+S_t$
MAXIMAL LOCUS FOR S_c
S_t RESPONSE AMPLITUDE (% CONTROL)
0
100
200
300
400
5 10 25 50 80 100 200
S_t DELAY (msec.)

LGB shock delayed a commensurate amount from the end of SC activation. Data on the left show that the single test response amplitude is increased following SC activation, the magnitude of this effect decreasing somewhat as the interval between SC activation and S_t increased at both the maximal locus for S_t (top graph) and the maximal locus for S_c (bottom graph). The middle set of graphs presents the control data for the normal recovery cycle (no SC activation) to S_c and S_t (S_c and S_t were always of equal intensity), and shows a depression of test response reaching a maximum effect at 50 to 80 milliseconds and returning to control levels by 200 milliseconds. Following SC activation (right graphs), the excitability cycle is slightly altered throughout its extent. Test amplitude is not depressed as markedly as in the normal recovery cycle, and by the 200 millisecond interval approaches the control value obtained at the commensurate delay for the SC plus S_t condition. In general, amplitude of response to S_t seemed more affected by the prior S_c response than by SC activation until the two shocks were separated by about 200 milliseconds.

In 5 of our 11 animals a prior conditioning shock increased rather than decreased the response to S_t at its maximal locus, confirming an observation previously noted by Storck et al,(1972). In these 5 animals it was noted that at short intervals only (5 to 10 milliseconds), prior SC activation produced an interesting effect which is illustrated in Fig. 13. On the left are shown 5 superimposed traces of responses to S_t alone (top pair), SC activation plus S_t (second pair), S_c and S_t at a 10 millisecond interval (third pair), and SC activation plus S_c and S_t (bottom pair);in all cases top tracings are from S_t maximal locus and bottom tracings from S_c maximal locus on cortex. As shown earlier, SC activation enhanced the response to S_t more at submaximal than at maximal locations (compare in the first two sets of tracings the amplitude changes at +8 and -4 locations). When S_c preceded S_t, the response to S_t was slightly enhanced at its maximal locus (A+8) but depressed at the maximal locus for S_c (P-4) (compare first and third sets of tracings at +8 and -4). When S_c and S_t shocks were preceded by SC activation, the response to S_t was enhanced slightly more at its maximal locus (A+8) but completely eliminated at the maximal locus for S_c (P-4) (compare third and fourth sets of tracings). The graph on the right portrays functions of the amplitude of response versus cortical location, as elicited by S_t, when preceded by SC activation (solid circles), by S_c at a fixed 10 millisecond interval (open circles), or to both SC and S_c (triangles). Eight cortical locations along the marginal gyrus were sampled varying in the A-P direction from A+8 (maximal locus for S_t) to P-6 (P-4 being the maximal locus for S_c). These data show that SC activation (triangles) tends to raise very slightly the test response amplitude near its maximal locus on cortex (around +8), and

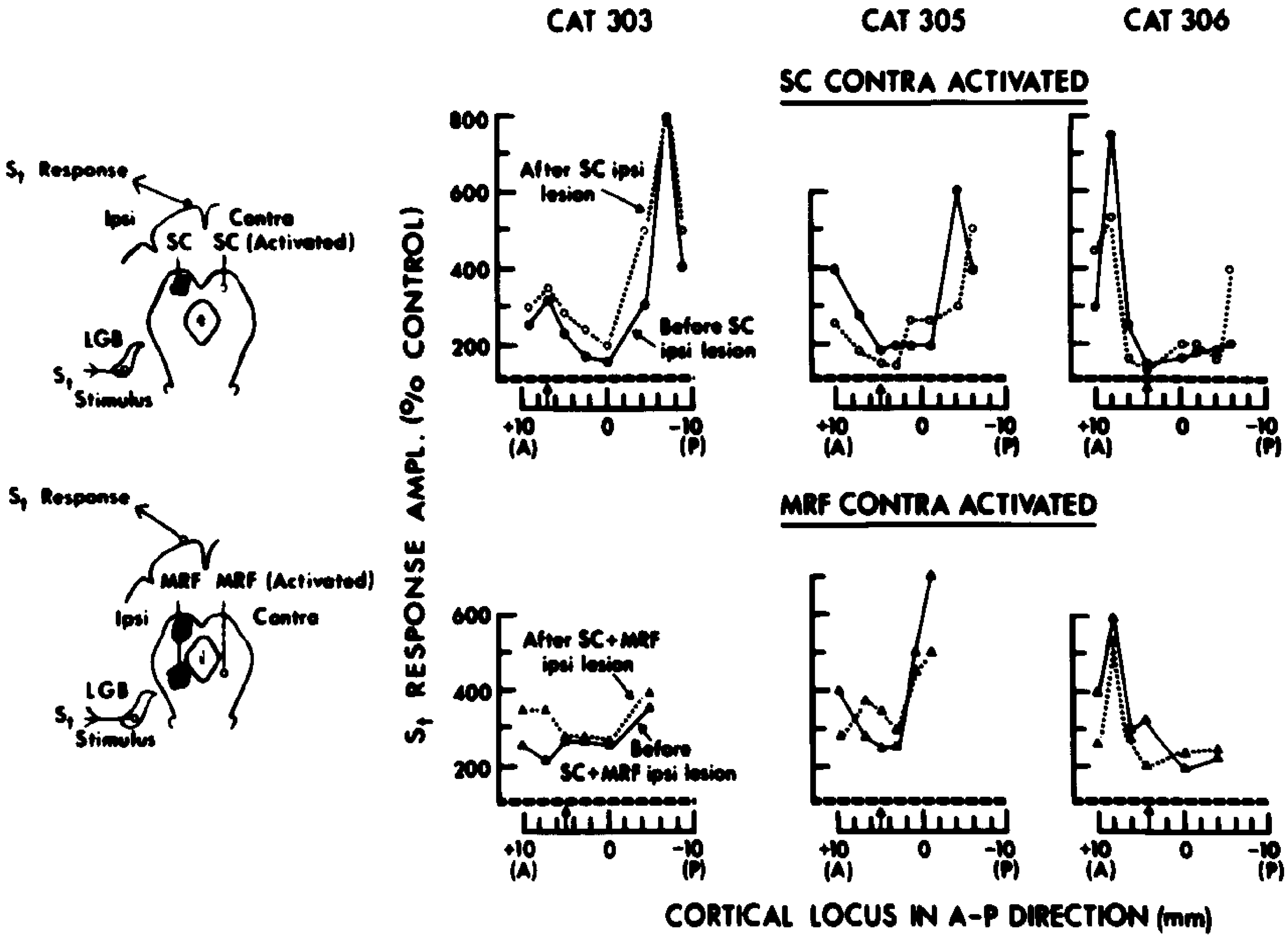
Fig. 13 Effects of SC activation on the spatial interaction of responses in cortex to two LGB shocks (10 milliseconds apart) for Cat 299. Tracings show cortical response at the maximal locus for S_t (+8) and at the maximal locus for S_c (-4). Graph shows S_t response (expressed as per cent of control) for different locations across cortex for the three experimental conditions indicated. Arrows on graph indicate cortical location of maximal response to S_t (+8) and to S_c (-4).


CORTICAL LOCUS (mm)
+ 8
- 4
S_t ALONE
SC + S_t
●—●
(S_c + S_t, 10ms)
○⋯○
SC + (S_c + S_t, 10ms)
▲—▲
0.5mv
10ms
S_c S_t
CAT 299
S_t RESPONSE AMPL. (% CONTROL)
600
400
200
100
0
S_t
S_c
+8
+4
0
-4
-8
(A)
(P)
CORTICAL LOCUS IN A-P DIRECTION (mm)

to depress more definitely the test amplitude in the vicinity of the S_c maximal locus (around P-4). The overall effect of SC activation, then, is to exaggerate somewhat the slope of the cortical gradient of the recovery function (open circles). Such a "sharpening" effect following SC activation was observed in all 5 animals.

<u>The effects of subcortical lesions on the enhancement effect</u>. In the present study it was shown that enhancement of shock evoked responses could be elicited in cortex after either ipsilateral or contralateral SC activation. It is possible that such a contralateral enhancement effect was due to indirect activation of the ipsilateral SC via known intercollicular connections (Altman and Carpenter, 1961). In 7 animals, therefore, the effects of contralateral SC activation on the spatial distribution of the shock response in cortex was examined, before and after a lesion was made in the ipsilateral superior colliculus. In all 7 experiments the results were the same and are illustrated in Fig. 14 for three of these animals. As indicated in the upper row of graphs the proportion of contralateral collicular enhancement is unchanged after ipsilateral collicular lesion. When the electrodes were lowered into the MRF bilaterally (bottom row of graphs), a similar enhancement was obtained from the contralateral MRF and this effect persisted following additional lesion of the ipsilateral MRF. In these experiments, topical KCl was applied to the contralateral cortex during the post-lesion stage to control for possible transcallosal effects.

Fig. 14 Top graphs: In all three cats lesion of the ipsilateral SC did not alter the enhancement effect produced by contralateral SC activation. Bottom graphs: Same results were obtained when contralateral MRF was activated following lesion of the ipsilateral MRF.

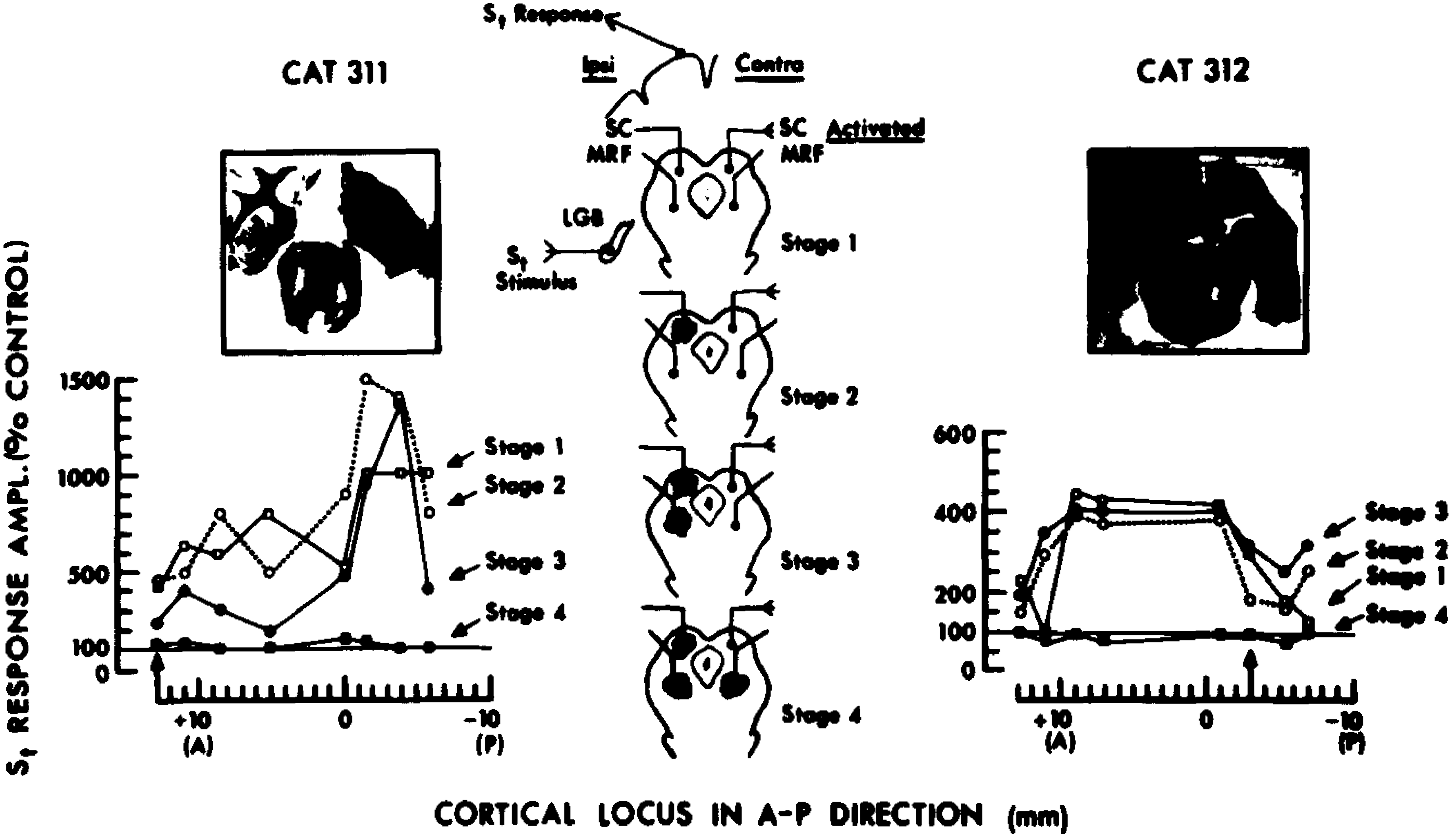


CAT 303
CAT 305
CAT 306
SC CONTRA ACTIVATED
MRF CONTRA ACTIVATED
S₁ Response
Ipsi
Contra
SC
SC (Activated)
LGB
S₁
Stimulus
MRF
MRF (Activated)
800
600
400
200
After SC ipsi lesion
Before SC ipsi lesion
After SC+MRF ipsi lesion
Before SC+MRF ipsi lesion
+10 (A)
0
-10 (P)
S₁ RESPONSE AMPL. (% CONTROL)
CORTICAL LOCUS IN A-P DIRECTION (mm)

No change was observed in the enhancement effect for either contralateral SC or MRF activation following KCl administration. In brief, the findings indicate that the enhancement effect obtainable for both SC and MRF is bilateral and not dependent upon commissural connections at the midbrain or cortical levels.

Since there are no known direct tectal inputs to the cortex, the superior colliculus may mediate its influence on cortex indirectly through the MRF, as suggested by Brown and Marco (1967), or through the pulvinar. Both structures have reciprocal connections with SC (Altman and Carpenter, 1961; Anderson and Berry, 1959) and as demonstrated in this, as well as in earlier electrophysiological studies (Chalupa et al, 1973) they appear to be functionally related. In 9 animals, therefore, a series of single or multiple-stage lesions were made to determine possible functional relations between SC and MRF. Figure 15 shows for two cats, 311 and 312, the relative enhancement of LGB elicited responses across cortex (graphs at bottom) due to contralateral SC activation, before and after a consecutive series of three midbrain lesions. In both cats, the relative amount of collicular-induced enhancement was unchanged following lesion of the ipsilateral colliculus (Stage 2), as noted above. A subsequent lesion of the ipsilateral MRF (Stage 3) produced a minimal reduction in the enhancement effect in Cat 311 and no effect in Cat 312. When an additional lesion was made in the contralateral MRF (Stage 4) collicular enhancement was

Fig. 15 Effects of serial midbrain lesions on contralateral SC activation: Ipsilateral SC lesion (Stage 2) produced no change; ipsilateral MRF lesion (Stage 3) produced some effect in Cat 311 and no change in Cat 312; additional lesion to the underlying MRF (Stage 4) abolished SC enhancement effect in both cats. Accompanying microphotographs identify lesion sites.


CAT 311
CAT 312
S_t Response
Ipsi
Contra
SC
MRF
SC
MRF
Activated
LGB
S_t
Stimulus
Stage 1
Stage 2
Stage 3
Stage 4
S_t RESPONSE AMPL. (% CONTROL)
1500
1000
500
100
0
600
400
200
100
0
Stage 1
Stage 2
Stage 3
Stage 4
Stage 3
Stage 2
Stage 1
Stage 4
+10
(A)
0
-10
(P)
+10
(A)
0
-10
(P)
CORTICAL LOCUS IN A-P DIRECTION (mm)

completely abolished and the curve drops to control level.

Figure 16 illustrates for two more cats, 314 and 318, the cortical gradient of the enhancement effect induced by either ipsilateral or contralateral SC activation. Prior to the MRF lesion, activation of either colliculus produced about the same effect across cortex (left hand graphs). When a lesion was made in the ipsilateral MRF (middle graphs), there was some reduction in the enhancement effect obtainable by activating the colliculus on the same side (ipsilateral SC). Essentially the same effects were obtained in two other animals (Cats 321 and 323) not shown here. After a bilateral MRF lesion (right hand graphs), however, the collicular enhancement obtainable from either side was abolished in the two cats shown in Fig. 16.

The influence of Pul lesions on collicular enhancement was examined in three animals, as shown in Fig. 17. As the top graphs show, the relative enhancement effect elicited by SC activation was unchanged after a Pul lesion on the same side as the activated colliculus. The same Pul lesion had little consistent effect on the enhancement produced by activation of the MRF on the same side (bottom graphs), except possibly in Cat 329. However, as the top graphs indicate, a subsequent ipsilateral lesion of the MRF, i.e., on the same side as the prior Pul lesion, completely abolished the enhancement effect obtainable from SC in all three animals. These findings suggest that efferents from SC to the Pul are not necessary for obtaining enhancement, whereas those to the

Fig. 16 Effects of serial bilateral MRF lesions: unilateral MRF lesion (Stage 2) reduced somewhat the enhancement obtained by activation of the overlying colliculus; bilateral MRF lesions (Stage 3) markedly reduced the enhancement effect obtained from either colliculus.

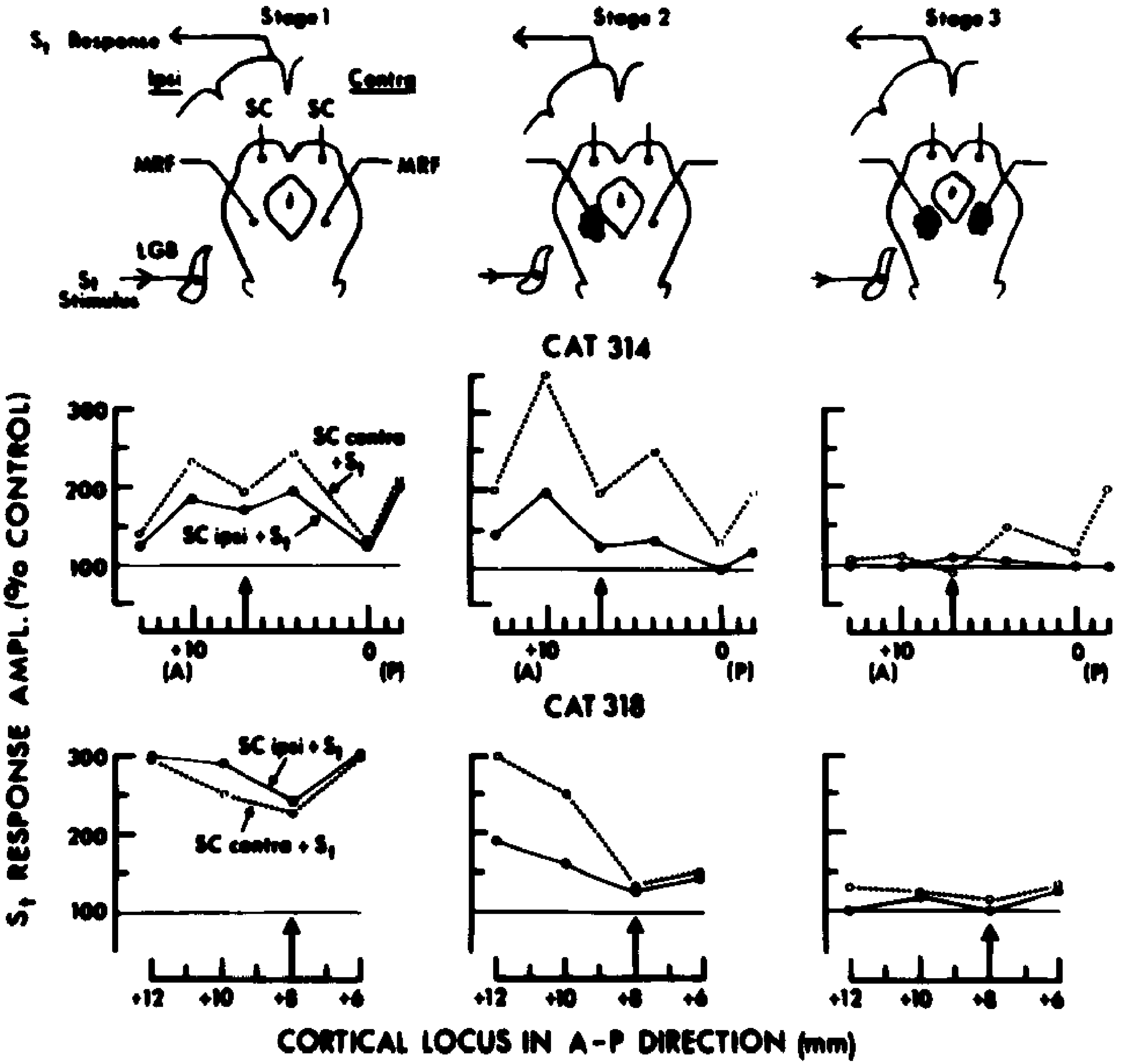
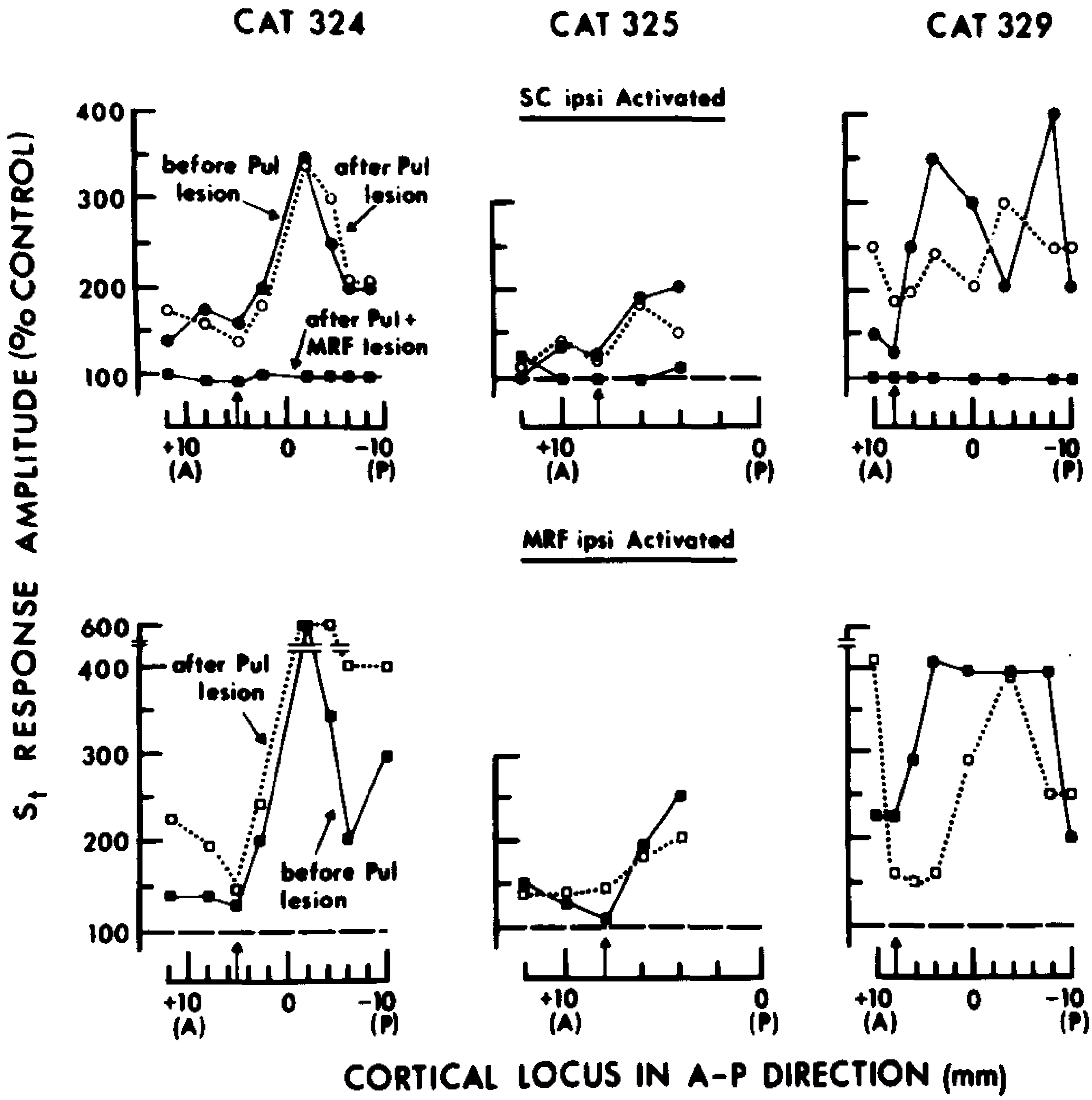

Stage 1
Stage 2
Stage 3
S1 Response
Ipsi
Contra
SC
SC
MRF
MRF
LGB
S1 Stimulus
CAT 314
CAT 318
S1 RESPONSE AMPL. (% CONTROL)
300
200
100
SC contra + S1
SC ipsi + S1
+10 (A)
0 (P)
+12
+10
+8
+6
CORTICAL LOCUS IN A-P DIRECTION (mm)

Fig. 17 In three cats Pul lesions produced no change in the enhancement effect obtained by activating SC (top graphs) or MRF (bottom graphs) on the same side but in all cases additional lesion of MRF (top graphs) reduced SC enhancement effect to control level.


CAT 324
CAT 325
CAT 329
SC ipsi Activated
MRF ipsi Activated
S_t RESPONSE AMPLITUDE (% CONTROL)
400
300
200
100
600
400
300
200
100
before Pul lesion
after Pul lesion
after Pul + MRF lesion
after Pul lesion
before Pul lesion
+10 (A)
0
-10 (P)
+10 (A)
0 (P)
CORTICAL LOCUS IN A-P DIRECTION (mm)

MRF appear to be essential. Yet Pul appears to be involved in some way since a combined Pul and MRF lesion on one side was as effective in abolishing collicular enhancement as a bilateral MRF lesion.

In summary, 15 animals were used to investigate the subcortical pathways involved in mediating collicular enhancement of the geniculate shock evoked cortical response. The findings indicate that: 1) the bilateral effects of collicular enhancement do not depend upon the integrity of intercollicular or transcallosal pathways; 2) the MRF underlying the activated colliculus appears to be involved in mediating collicular enhancement; 3) bilateral pathways through the MRF appear to be essential for this enhancement to occur; and, 4) ipsilateral pathways through the Pul do not appear to be essential for collicular enhancement, but appear to be involved in some, as yet, unknown manner.

DISCUSSION

The results of the present study show that activation of the ipsilateral or contralateral superior colliculus increases the response amplitude elicited by a single geniculate test shock, at both focal (maximal) and non-focal (submaximal) locations on cortex. Interaction between the cortical responses elicited by two shocks to different LGB loci was also affected by SC activation. In 11 animals, SC activation reliably shortened the normal recovery cycle in that it returned to control levels within 200 milliseconds. In 5 of these animals, at short S_c to S_t intervals, test response amplitude was slightly enhanced at the maximal location for the test shock, but more definitely depressed at the maximal cortical location for the conditioning shock. Enhancement of responses in visual cortex, obtained by activating one SC, survived lesion of the contralateral SC, even when KCl was applied topically to the contralateral cortex. In addition, SC enhancement survived ipsilateral Pul lesion, was reduced following ipsilateral MRF lesion, and essentially eliminated by bilateral MRF lesions or by ipsilateral Pul and MRF lesions. The import of these effects will be discussed below.

<u>The cortical gradient of collicular enhancement</u>. Prior studies have shown that activation of the superior colliculus enhances the cortical response to LGB shock (Bremer, 1966a, 1966b; Chalupa et al, 1973) or optic tract

stimulation (Brown and Marco, 1967), but collicular influences on the gradient of evoked responses across cortex have not been previously described. It was found in the present study that the relative amount of SC enhancement (per cent of control amplitude) between maximal and submaximal cortical locations depended upon the nature of the cortical gradient of response elicited by LGB shock. In those distributions where definite peak responses were elicited (usually with a maximal intensity test shock), the greatest proportion of enhancement occurred at submaximal locations (see Fig. 11). Statistically, these distributions were obtained by test shocks in lamina A or A' of LGB (see Table 1). On the other hand, even maximal intensity stimulation in posterior or ventral portions of LGB (lamina B) usually produced flat distributions across cortex of low amplitude, longer latency responses, usually 1 to 2 milliseconds longer than responses elicited from lamina A or A' (see Figs. 5 and 10 for details). In such distributions, prior activation of SC either made it possible to elicit a definite peak locus for the first time, or, if a slight peak locus existed, it was markedly enhanced; i.e., the greatest proportionate enhancement occurred at this maximal location.

The above findings suggest that an indirect pathway from lamina B of LGB to visual cortex exists. Such a pathway is possible in view of prior electrophysiological studies (Bishop and Clare, 1955; Chalupa et al, 1972) showing connections between lamina B and the Pul-LP complex. Since the

Pul is known to project to suprasylvian cortex (Waller and Barris, 1937; Graybiel, 1972), and since loop fibers are known to exist between this projection field and the visual cortex (Wilson, 1968), lamina B can project indirectly to visual cortex, albeit with slightly longer latency.

Other subcortical structures yielding cortical enhancement. It was shown in the present study that activation of the midbrain extending ventrally from SC into the MRF produced an enhancement of the spatial distribution of evoked responses in cortex (see Figs. 8 and 9), which was essentially the same as that elicited from SC. Previous studies have shown that MRF activation enhances the evoked response to LGB shock (Bremer, 1966a, 1966b; Chalupa et al, 1973), but the effects of tetanizing the tegmental area adjacent to the central gray have not been previously described. This latter area is known to carry reciprocal connections between SC and MRF (Altman and Carpenter, 1961; Anderson and Berry, 1959), and recent studies have shown that LGB activity can be influenced by activation of this area (Tatton and Crapper, 1972), as well as by activation of the ventral MRF (Ogawa, 1963; Satinsky, 1968; Singer and Drager, 1972). Our finding of a SC enhancement effect can be explained most simply in terms of fibers coursing from SC ventrally into MRF, so that SC activation essentially results in MRF activation. An alternate interpretation would stress the significance of spread of stimulating current from SC to MRF. While such a factor cannot be completely excluded, it could not be the critical mechanism, since SC tetanization rarely produced pupillary dilatation (and where present it was slight and of longer latency), whereas MRF tetanization always produced a pupillary response of large magnitude and short latency. Moreover, the postulation of current spread from SC to MRF could hardly explain the fact that an ipsilateral MRF lesion had only a slight depressing effect on SC enhancement, while bilateral MRF lesions were most effective (see discussion below).

It should be stressed, however, that efferents from SC are not limited to fibers coursing to MRF. A small but well defined number of SC efferents are known to project to Pul (Altman and Carpenter, 1961), and activation of the Pul was shown in the present study (see Fig. 10) to influence cortical responses in a manner similar to SC and MRF activation. In other studies, activation of the Pul has been shown to enhance the evoked response to light (Battersby and Oesterreich, 1963), optic tract (Brown and Marco, 1967) or lateral geniculate shock stimulation (Chalupa et al, 1973). Accordingly, on the basis of known anatomical and electrophysiological data as outlined above, it is possible that part of the ipsilateral SC enhancement effect was mediated through the Pul.

Fibers projecting from one SC to the other provide yet another possible mechanism for the SC enhancement effect. The present study showed that activation of either SC could produce similar enhancement effects on the cortical response to LGB shock; i.e., the effects of stimulating either colliculus were bilateral. It is possible, however, that the effect of SC tetanization in the ipsilateral hemisphere is mediated via mechanisms noted above, but that the effect in the contralateral hemisphere depends upon an intercollicular projection. In brief, activating one SC may have resulted in activation of the other. Recent concepts arrived at primarily from ablation studies (Sprague, 1966), however, have stressed the role of intercollicular inhibition, not activation, and there is some electrophysiological support for this concept (Hoffman and Straschill, 1971).

In general, the findings discussed above indicate that a variety of complex interactions can occur between the colliculi, as well as between SC and the Pul and MRF. The lesion experiments discussed below represent an attempt to test for the significance of some of these interactions.

Effects of subcortical lesions on cortical enhancement. The present study showed that SC enhancement effects were bilateral in cortex, a finding earlier reported by Bremer (1966a, 1966b). It was further shown, however, that a lesion of the ipsilateral colliculus produced no change in the effect of enhancement as elicited in that same hemisphere from the contralateral SC (see Fig. 14), indicating that collicular enhancement is essentially independent of direct intercollicular projection. It is possible that these negative results can be attributed to an incomplete lesion of the ipsilateral SC. This would not, however, account for the positive results obtained following large, yet incomplete ipsilateral or bilateral MRF lesions. Some authors (Sprague, 1966) have postulated that when a lesion is made in SC on one side, the remaining SC will be "released" from a normal intercollicular inhibition. Consequently, stimulation of the remaining SC should produce a greater cortical enhancement of the LGB elicited response than prior to lesion due to this "release" phenomenon. This did not occur in the present experiment, perhaps because intercollicular inhibition either does not exist, or does not operate in our preparations where both visual cortices were left intact. On the other hand, a more direct method would have been to section the intercollicular commissure. This was not done in the present study. However, the present study also showed that post-lesion application of KCl to the contralateral cortex did not alter the magnitude of contralateral SC enhancement. This finding raises questions for those concepts which postulate a functional alteration within one SC after ipsilateral visual cortex ablation plus SC lesion on the opposite side (Sprague, 1966), and, in fact, some negative data exists (Keane et al, 1968).

Like SC, MRF enhancement was similarly found to be bilateral in cortex, and the effects obtained from one MRF survived lesion of the other, even when KCl was applied topically to the overlying cortex. Collectively, these findings could be explained by postulating that SC and MRF influences on cortex are mediated through a diencephalic pathway, most likely the diffuse reticulo-thalamo-cortical system (Jasper and Ajmone-Marsan, 1952). Such a hypothesis could be tested by examining the effects of SC and MRF enhancement before and after lesions of such thalamic structures as the centre median, suprageniculate, and centralis lateralis nuclei.

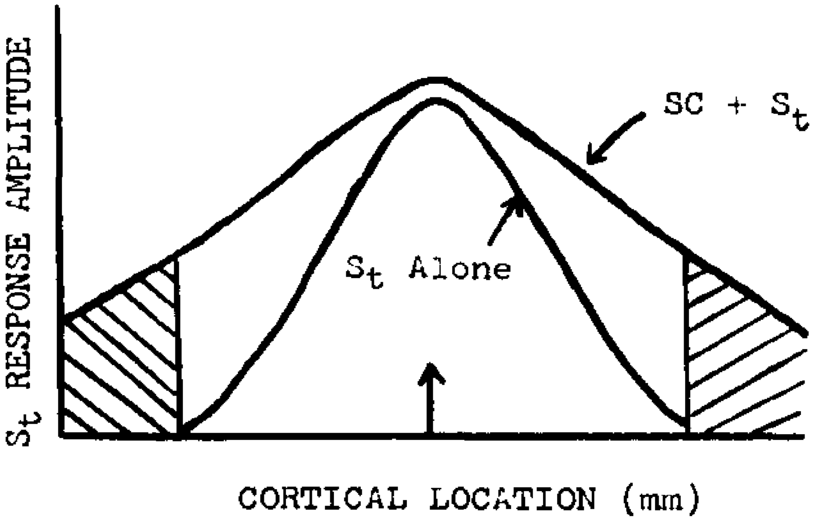
The possibility of bilateral enhancement effects from the Pul and their possible survival following unilateral Pul lesions were not examined in the present study, and must therefore await future investigation. However, the influence of unilateral Pul lesion on the enhancement effect elicited by SC and MRF activation was assessed (see Fig. 17), and no alteration from pre-lesion status was noted. When a MRF lesion was added on the same side as the Pul lesion, however, a complete loss of the SC elicited enhancement effect occurred. This finding suggests that the enhancement phenomenon depends upon alternate pathways from SC, one through MRF and the diffuse projection system, the other via the Pul and its parastriate cortical projection.

Extending an earlier observation, (Chalupa, 1970), the present study has shown that unilateral MRF lesions reduced somewhat the enhancement effect elicited from the ipsilateral (overlying) colliculus, but not from the contralateral colliculus. Bilateral MRF lesions essentially abolished collicular enhancement from either side. This finding indicates that the enhancement phenomenon depends upon a bilateral projection of each SC into both MRF structures. In fact, fibers from the deep layers of SC are known to project bilaterally into MRF, some crossing in SC and others in the dorsal tegmental decussation, distributing collaterals to the contralateral MRF (Altman and Carpenter, 1961). Collicular influences mediated through MRF could then ascend in the diffuse projections ending in midline and intralaminar nuclei (Nauta and Whitlock, 1954), and thence could affect cortex via the reticular nucleus (Jasper and Ajmone-Marsan, 1952). Unfortunately, this postulation would not by itself explain the positive effects of combined Pul and MRF lesion on the same side, as noted above (page 66). One possibility is that the Pul influence on cortical enhancement is dependent upon MRF integrity on the same side. There is some support for this hypothesis. Chalupa et al (1973) showed, for example, that the time course for Pul and MRF potentiation of a single LGB shock response was similar, but both of them differed somewhat from that elicited from SC. This suggests that Pul and MRF effects are mediated through some common pathway. Moreover, Chalupa (1970) found that MRF lesions markedly disrupted the effects of this Pul enhancement. In preliminary studies (not described in this work), we have obtained similar results on the cortical gradient of enhancement for 2 out of 3 cats. These observations require more systematic study but they tend to support the hypothesis of a critical pathway from Pul to MRF.

Collicular effects on spatial interaction. Recently, Storck et al (1972) have shown that the cortical response to an LGB test shock (S_t) can be influenced by the response to a preceding conditioning shock (S_c) delivered to a different site within LGB. Specifically, the response to the second ("test") shock increased at its maximal cortical location as compared to control (S_t alone) data, but decreased at the maximal cortical location for S_c. The authors postulated that a "lateral interaction" process, similar to that operating at retinal levels, exists also at the cortical level, such that the contrast between two incoming stimuli is increased; i.e., a "sharpening" mechanism is operative. Such a mechanism could be significant in such behavioral functions as visual acuity and spatial localization. The present study showed that SC activation could very definitely affect the degree of spatial interaction elicited by paired shocks at different locations in LGB (see Fig. 12). More importantly, perhaps, with such LGB shocks at short intervals (5 to 10 milliseconds), the response to S_t could be further enhanced (as compared to control excitability data) at its maximal location, and further depressed at the maximal location for S_c (see Fig. 13). This observation suggests that the superior colliculus can influence the interaction of cortical responses initiated by two incoming sources of activity, such that the contrast between two environmental stimuli is magnified. Our findings in conjunction with those of Storck et al (1972) suggest that interaction within the geniculo-striate system involves both excitatory and inhibitory mechanisms, and their influence on cortical responsiveness can be altered by activation of SC. As suggested by several earlier studies (Jung, 1958, 1961; Morillo, 1961), a "unit" approach would permit a more detailed examination of such mechanisms, and if intracellular in nature might allow for an ultimate explanation in terms of shifts in membrane potential (see Li et al, 1960; Watanabe et al, 1966; Creutzfeld and Ito, 1968).

Behavioral Implications. Bilateral SC lesions in cats have been said to result in problems related to orientation and object localization which cannot be attributed exclusively to either eye, head, and neck coordination or visual field dificits (Sprague and Meikle, 1965). In freely moving cats, stimulation of SC produces eye and head movements towards the contralateral side, which have been interpreted as some sort of "orienting behavior" (Syka and Radil-Weiss, 1971). The present study suggests that activation of the superior colliculus increases the spatial dispersion of the response to a single LGB shock. It is possible, for example, that areas of cortex not normally excited by radiation input do become excited upon collicular activation, thereby increasing the spatial capacity of the system. This is illustrated by the diagram in Fig. 18 where a theoretical test response amplitude is plotted as a function of cortical location for S_t alone (lower curve)

Fig. 18 Theoretical test response amplitude as a function of cortical location for S_t alone (lower curve) and SC + S_t (upper curve). Hatched areas indicate areas of increased spatial dispersion of responses to S_t when it is preceded by SC activation. Arrow on the abscissa indicates location of maximal response.


SC + S_t
S_t Alone
S_t RESPONSE AMPLITUDE
CORTICAL LOCATION (mm)

and for SC plus S_t (upper curve). The hatched areas indicate the extended distribution of responses to S_t when it is preceded by SC activation. The significance of such a postulate could be tested in chronic preparations by comparing the behavioral effects of SC activation, eg., on object localization, before and after cortical lesions which restrict spatial dispersion, such as in the hatched areas of Fig. 18. The superior colliculus may also exert a "sharpening" effect on the distribution of responses to a test shock which is preceded by a conditioning shock to another LGB site. As noted above, such an effect might be significant for enhancing the contrast between two environmental stimuli. In brief, both behavioral and electrophysiological data suggest that SC is involved in the spatial processing of information in the visual system.

While conceptually intriguing, the above hypothesis does not have direct support since the relationship between behavioral and evoked potential data has not been demonstrated. Nevertheless, there are some interesting correlated sets of data. For example, the present study has shown that bilateral MRF lesions abolish SC enhancement. Similarly, Myers (1964) has shown that pattern discrimination in cats is markedly disrupted following bilateral SC lesions, but only when these lesions also include the dorsal aspects of the MRF. More recently, Berlucchi et al (1972) have shown that the learning of monocular pattern discriminations in split brain cats was more markedly impaired if the ipsilateral

SC lesion was accompanied by pretectal lesion which apparently also extended into the subcollicular area (the MRF), as well as into the area rostral to the SC. In fact, an early behavioral study (Sprague et al, 1963) which attempted to compare the effects of midbrain lemniscal and reticular formation lesions, actually included extensive damage to the deeper layers of SC (including the tecto-spinal and tectoreticular fibers (see Sprague and Meikle, 1965, page 140). Accordingly, it is not surprising that the "lemniscal" lesions in the above study produced behavioral disturbances similar to those following colliculectomy.

Aside from SC and MRF interactions, some consideration has been given to the behavioral significance of SC and Pul interactions. Specifically, Sprague and Meikle (1965) showed that unilateral interruption of tectothalamic fibers in cats produced visual field defects ("visual neglect") with no accompanying motor disturbances whereas lesion of the tectospinal tract produced motor disturbances associated with the eyes, head, and neck and no visual deficits. The present study showed that unilateral lesion of Pul alone produced no alteration in SC enhancement whereas a combined MRF plus Pul lesion abolished SC enhancement. Collectively, these findings suggest that Pul is involved in mediating SC functions but more experimental data are required to accurately assess this involvement, particularly those where adequate stimulation is utilized. In this regard, the use of chronic preparations with cortical and subcortical implanted electrodes would be helpful. In such preparations the effects of activating SC, MRF, and Pul could be assessed behaviorally and electrophysiologically and these results compared with SC activation following Pul or MRF lesion.

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