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TOPOLOGIC AND TEMPORAL CHARACTERISTICS OF
LATERAL GENICULATE PROJECTIONS
IN THE CAT

by

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INTRODUCTION

A "point-for-point" or highly localized projection of the retina upon striate cortex is the neural mechanism most often cited to explain visual localization of objects in space. (Polyak, 1957). In brief, the retinotopic theory proposes that for every point on the retina, there is a corresponding point in the lateral geniculate body and at visual cortex. Accordingly, since a retinal stimulus excites a discrete cortical locus (the "cortical retina"), the spatial position of the object is known to the observer (Henschen, 1906).

A number of anatomical studies describing the spatial organization of visual fibers are cited to support this point-for-point concept. The topologic relationship between the retinal surface and visual cortex is traced in a variety of mammals, especially in cat and monkey (Brouwer, 1927; Polyak, 1957), and a strictly punctate, retinostriate projection system is often reported. More recent studies, however, particularly of the cat visual fibers, describe marked branching of collateral visual fibers and their lateral spread, not only in the retina, but also within the geniculate and across visual cortex itself (Colonnier, 1964). These findings suggest that multiple cerebral regions may be excited by a single retinal stimulus, and that a widespread distribution of visual projections may underly visual function, rather

than a strictly point-for-point system.

Clinico-pathological data are also used to evaluate the "point-for-point" concept of visual localization. Perimetric assessment of visual deficits following localized cerebral lesion in man frequently discloses a high degree of spatial specificity between the location of blindness in the visual field, and the locus of damage in the geniculo-calcarine system (Holmes, 1919; Spalding, 1952). A number of clinical studies (e.g. Battersby, 1951; Bay, 1953) show however, that visual deficits after brain damage may be widespread within the visual field if special testing techniques are utilized. In general, such findings argue against strict point-for-point function of the visual projections.

A third, perhaps more direct, approach to studying the spatial organization of the visual system is electrophysiological: to evaluate the response patterns of the potentials evoked in visual cortex by photic or electrical stimuli. The classic studies of Marshall and Talbot (1941, 1942) for example, varied the location of a small photic stimulus on the retina, and determined for each location, the cortical locus which gave the largest amplitude and shortest latency response. In this fashion, the cortex was mapped in terms of its retinal input, a topological relationship between retina and cortex was described, and deductions were drawn regarding spatial discrimination between two stimuli ("cortical acuity"). All of these experiments were conducted on deeply anes-

thetized cats and monkeys, and only single light flashes were used, despite the generalizations to two discrete stimuli. Subsequent electrophysiological studies confirming the topological representation reported by Marshall and Talbot again use only deeply barbiturized preparations, and single stimuli. Other electrophysiological studies, however, describe a diffuse responsiveness of cat visual cortex rather than a highly localized response, and question the functional significance of the widespread distribution of evoked responses (Doty, 1961).

The Present Study

Previous electrophysiological investigations have used only retinal stimuli to study the topological relationships of the visual system. In order to achieve maximum stimulus control for the present study, a punctate electrical stimulus was delivered to the lateral geniculate body (LGB), and this shock stimulus served to activate cerebral visual pathways synchronously. In addition, retinal and peripheral visual fibers were bypassed, eliminating a retinal photochemical response, and avoiding entopic light spread initiated by a photic stimulus.

The geniculo-cortical topologic relationship was investigated by varying the site of the stimulus in LGB. Evoked activity at visuo-cortical loci responding to any one LGB stimulus was studied, and that cortical locus which gave the maximum amplitude and shortest latency re-

sponse was noted (the "peak locus"). Since many cortical loci responded to any one LGB stimulus, other experiments were designed to investigate whether or not the shock stimulus was restricted to a single geniculate position, what neural mechanisms are responsible for the diffuseness of the cortical response, and how the widespread responses might influence evoked activity at the peak locus.

More than one stimulus could be delivered to separate sites in the LGB, and the neural mechanism subserving spatial acuity could be studied by presenting two LGB stimuli simultaneously, and recording cortical responses to this dual stimulation. In addition, the temporal delay between two geniculate shocks was varied to determine the excitability cycles of visual cortex using two spatially-discrete stimuli.

The geniculo-cortical topologic relationship has not previously been studied by stimulating the LGB itself. In addition to exciting geniculo-cortical projections directly, this stimulation method provides a uniquely reliable shock-evoked potential in the cat. The cortical response clearly comprises pre-synaptic afferent components as well as post-synaptic cortical components, and can be used to analyze afferent versus intracortical topology separately. Curiously, earlier studies did not make this distinction, in spite of the fact that the intracortical components may display a different topologic organization than the afferent activity. The cat was chosen for this study

because there is already abundant literature on the anatomy and physiology of its visual system, especially analyses of the shock-evoked cortical response. Also, within the past few years, there have been a number of neuro-anatomical reports on the cat's retino-cortical structure, but few electrophysiological studies of topologic relations. The anatomical evidence suggests a widespread distribution of visual fibers, yet behaviorally, the cat demonstrates good spatial discrimination (Baden et al. 1965; Held and Hein, 1963). Primate and human visual systems may display similar physiological and behavior characteristics.

Previous electrophysiological studies of topological relations of the cat's visual system have used only retinal stimuli in the deeply anesthetized preparation. Since barbiturates are known to depress cortical activity (Curtis, 1940; Marshall et al., 1943; Wagman and Battersby, 1964), the present study chose to investigate non-barbiturized cortical responses. Cortical responsivity was assumed to be near-normal, and the topologic system under study, to be functioning as in the alert animal.

LITERATURE SURVEY

Anatomical Studies

Whether the topologic organization of the cerebral visual pathways is highly localized or less circumscribed and more widespread has fueled an historical argument dating back to the studies of von Monakow and his students (see Polyak, 1957). Although von Monakow was one of the first to describe discrete projections from retina to cortex, he suggested that visual function depended upon more diffuse processes in the cerebrum. His students Minkowski and Henschen, on the other hand, argued for a strict "point-for-point" functional geniculocalcarine system, and this latter point of view is generally accepted today. The anatomical data described below pertain only to the necessary structural conditions for visual behavior. The sufficient physiological conditions, to be discussed later, are derived from studies of response patterns in the functioning cerebrum.

In order to conform to primate studies, the anatomical literature dealing with cat visual system limits its descriptions to the topological arrangement of area 17 (Brodman, 1906) or so-called "striate" cortex. The striate area in the cat brain, however, (Otsuka and Hassler, 1962) is less identifiable under gross and microscopic examination than the clearly striped area in primate cortex. It comprises

much of the suprasplenial and marginal gyri, dorsally along their anterior-posterior extent. Since visual areas of cat cortex include at least this "striate region" but also include adjacent extrastriate cortex, it would seem important to study the topologic organization across striate and extrastriate areas of the cat brain.

Anatomical projections from the retina to the thalamus and to visual cortex are traced by making discrete lesions in one of these three visual structures, and then studying the resultant prograde or retrograde degeneration in the remaining two areas. After a lesion in one of the retinal quadrants for instance, an area of fiber or cellular degeneration (depending on the staining procedure) is seen in the LGB, and also in a number of other thalamic and midbrain structures, including the superior colliculus, the pretectal nuclei, the ventral nucleus of the LGB, and occasionally in numerous other nuclei (Brouwer, Zeeman, and Houwer, 1923; Clark and Penman, 1934; Garey and Powell, 1968; Laties and Sprague, 1966; Minkowski, 1913; Moore, Karapas, and Frenkel, 1964; Overbosch, 1927; Polyak, 1927, Stone and Hansen, 1966, von Monakow, 1880's according to Polyak, 1957). Similarly, after localized cortical lesions, areas of retrograde degeneration are readily identified in LGB and can be spatially related to the locus of the cortical lesion (Beresford, 1960; Garey and Powell, 1967; Minkowski, 1920; Polyak, 1927). Retino-cortical projections are deduced by comparing the maps of LGB degeneration, one after retinal

lesions producing prograde degeneration, the other after cortical lesion producing retrograde degeneration. Geniculo-cortical pathways have thus been mapped backwards (i.e. from cortex to LGB), although there is one study describing lesions at a number of geniculate sites in order to study prograde geniculo-cortical degeneration (Wilson and Cragg, 1967). In this way, anatomists have described which parts of the retina project via various portions of the LGB to spatially-predictable areas of visual cortex (Fig. 1 schematically illustrates the topologic projections in the cat).

The later studies, using finer lesion-making and staining procedures, have only added details concerning macular and meridional representation in LGB or on cortex. As mapped by these degeneration studies, the retino-geniculo-cortical topologic organization in the cat can be summarized as follows:

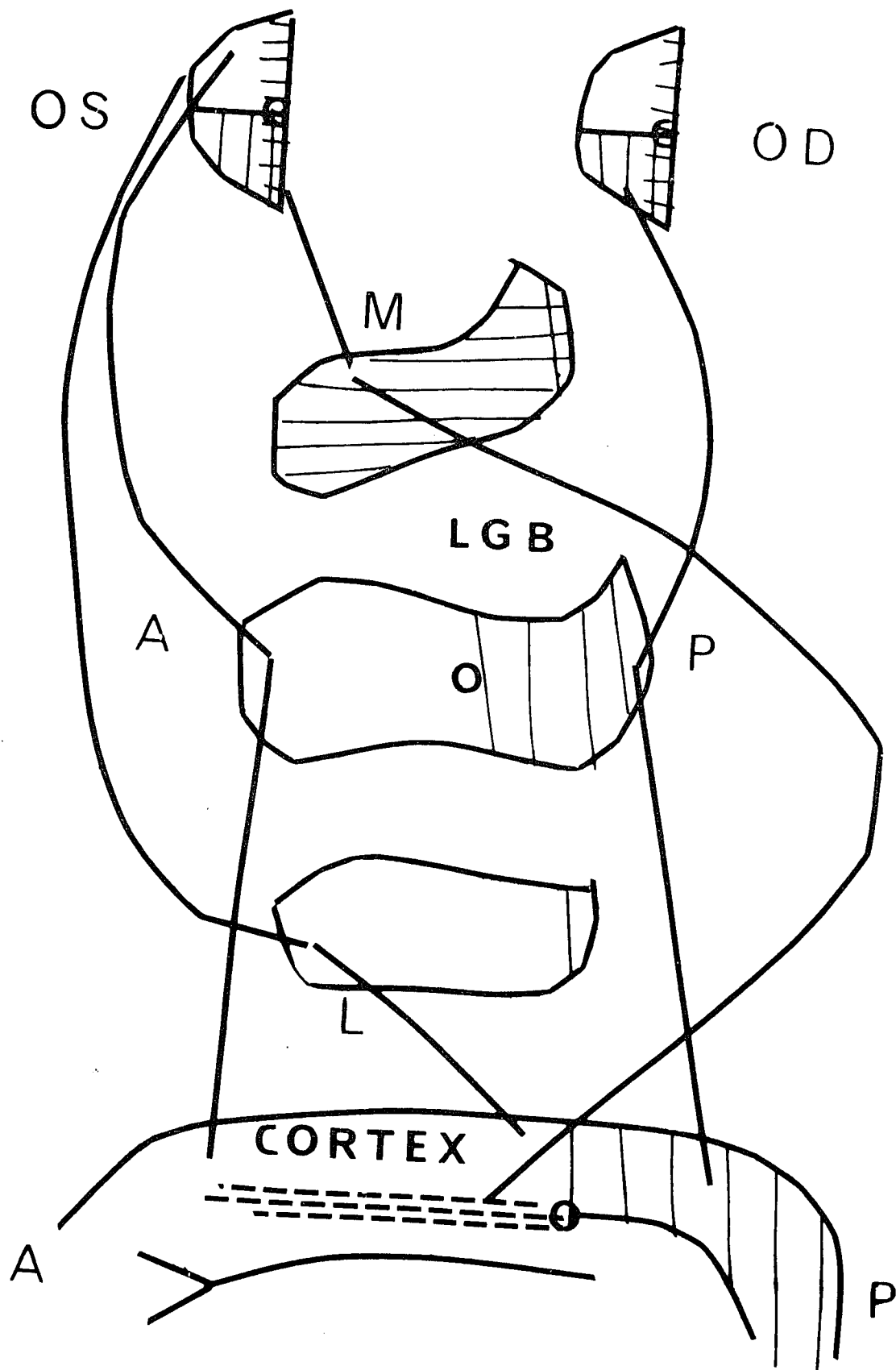
(a) Dorsal retinal quadrants send optic nerve fibers to anterior regions of the LGB, which in turn send radiation fibers to anterior visual cortical loci;

(b) Ventral retinal quadrants send fibers to posterior geniculate positions, which send fibers to posterior visual cortical loci;

(c) Peripheral retinal areas project to lateral LGB regions, which project to lateral loci on the marginal gyrus, and probably to mesial loci on the suprasplenic gyrus;

(d) Central (toward the vertical meridian) retinal

Fig. 1. Schematic illustration of the retino-geniculo-cortical pathways in the cat. The topology is traced from the left temporal and right nasal retina, to the left lateral geniculate body (LGB), to visual cortex of the left hemisphere.



areas project toward medial LGB regions, which may project two fiber groups to more medial loci on the marginal gyrus.

e) The area centralis is represented just posteriorly to the mid anterior-posterior length in the LGB, on its medial edge, and cortically, about where the marginal gyrus meets the posterolateral gyrus (at about AO to A+4 in the A-P plane, using the atlas of Jasper and Ajmone-Marsan, 1954).

It is important to mention some general qualifications that should be appended to these anatomical reports of topology in the visual system. First, degeneration methods vary in terms of size of the lesion, time allowed for degeneration, and staining technique (for example, a fiber or a cell stain may reveal larger or smaller degeneration areas). Second, there is the possibility that "sustaining collaterals" (Lorente de No., 1949) exist and may function to alter retinostriate maps in the following manner. A localized lesion in the retina or on cortex may produce only localized geniculate atrophy; however, that same retinal or cortical site may also send fibers to other geniculate areas whose cellular structure is "sustained" by fibers from intact retinal or cortical regions. Thus the pattern of degeneration may not reflect the true pattern of functional projections. Similarly, if a single cortical locus shows atrophy after a retinal or geniculate lesion, other cortical loci may receive projections from that same retinal or geniculate area, yet be sustained by collateral fibers. It is likely, in fact, that collaterals do operate

to sustain structure, since there is anatomical evidence for tangentially oriented processes in visual cortex of the cat (Coleman and Riesen, 1968; Colonnier, 1964, 1966; O'Leary, 1941; Ramon y Cajal, 1955; Sholl, 1955), and also for bifurcating and collateral fibers within the LGB (Glees, 1941; Hayhow, 1958; O'Leary, 1940; Peters and Palay, 1966; Smith et al., 1964). Lateral spreading of dendritic and axonal material in cortex, for instance, extends to at least a distance of three millimeters (Coleman and Riesen, 1968; Sholl, 1955). Similar branching in the LGB may be as extensive (O'Leary, 1940). In general, one could hypothesize that the lateral or horizontal spread of visual projections may interact with the vertical system of retinotopic projection so as to alter the precision of a "cortical retina." If this is true, then visuo-spatial localization depends not only upon discrete input from specific retinal and geniculate positions to visual cortex, but also upon the degree of interaction between these positions, or upon widespread sensory cortical activity.

Anatomical studies have also suggested that the primary visual pathway activates many cortical regions outside the classical sensory receiving area (Glickstein et al., 1965; 1967), even as far removed as posterior cingulate gyrus (Harmon and Berry, 1965). Radiation fibers in the cat directly innervate at least suprasylvian cortex (Vastola, 1961).

In addition to relaying in the LGB, optic tract fibers, activate multiple thalamic and midbrain regions (Meikle and Sprague, 1964) which in turn may influence cortical topological relationships via diffuse projection systems (Jasper et al., 1955; Magoun, 1952). Electrophysiological investigations confirm the diffuse cortical response to visual input. (see below)

In summary, the anatomical literature includes reports of a relatively precise topology within the visual projections, so that a point-for-point cortical map is easily described. However, other anatomical reports reveal a lateral spread of fibers throughout the cat's visual system: in the retina, in geniculate, and in visual cortex. Since these fibers may sustain cerebral regions after one of the afferent paths is severed, and may distribute the afferent signal to widespread cerebral regions, there may exist a more widespread or diffuse anatomical projection system than has heretofore been recognized. It should be noted that the anatomical studies are restricted to descriptions of single retinostriate pathways, and cannot report on the interaction among spatially-separate pathways. The studies to be reviewed below, of behavioral and electrophysiological visual responses, do discuss this problem of functional interaction among topologically-distinct pathways.

Clinico-Pathological Studies

Studies of residual visual capacity after localized cerebral damage in man provide additional material with which to evaluate the point-for-point concept of visual function. The clinical reports of Henschen and Minkowski around the early part of this century (see Polyak, 1957) presented the classical data in support of a precise and stable topologic arrangement of the retina on visual cortex. The clinical sign of localized cerebral damage was a delimited "blind" area in the visual field (a scotoma), and its location varied systematically with the site of cerebral damage. Accordingly, the clinicians proposed that each part of man's retina projected to a localized cerebral area, so that there exists a spatial specificity between the visual field and the cerebrum. These conclusions were based on perimetric evaluation of the extent of the visual field, and on locus and site of injury as described at surgery or upon radiography. Post-mortem confirmation was rare. G. Holmes and J. Spalding were among the prominent proponents of a strict point-for-point visual system (Holmes, 1918; Holmes and Lister, 1916; Spalding, 1952). Other clinicians (von Monakow, Goldstein and Gelb, as cited by Polyak, 1957) recognized that there was an orderly projection from retina to cortex, but cited the complexity of visual disturbances to support the concept of a more

widespread or functionally plastic cerebral system underlying visual sensations.

Recent analyses of visual dysfunction after cerebral damage reveal that there are a number of perceptual abnormalities which are difficult to explain in terms of a strict point-for-point retinocortical projection. For example, an early clinical sign of visual damage is an amblyopia (an alteration but not a complete loss of vision) which is not well localized in the visual field. In such amblyopic areas, other signs of early visual defect include abnormal color perception and spatial orientation (Bay, 1953; Bender 1952; Critchley, 1953) Goldstein, 1942; Teuber et al., 1960), micro- and macropsias, spatial inversion of seen objects, inaccurate perception of motion, defects in dark adaptation (Krieger, 1952), and abnormal flicker fusion frequency thresholds (Battersby, 1951). Portions of visual space are frequently ignored by the patient, indicating that cognitive as well as sensory deficits may accompany damage to visual cortex (Battersby et al., 1956). Upon double stimulation, visual thresholds are raised in portions of the field which appear normal during routine perimetry (Wagman and Battersby, 1959). Furthermore, the areas of anopia, one in each eye's field, are rarely exactly congruent, as a point-for-point retinocortical system might predict. It is important to note that these clinical tests of visual malfunction are carried out on pathological material, and the conclusions

may or may not apply to the normal visual system.

In brief, clinical tests of visual function following damage to the visual lobe in man reveal impaired vision throughout large areas of visual space, outside of a scotoma. Also, residual visual function within the scotoma itself is demonstrated under proper testing conditions (Bay, 1953). Therefore, a rigid point-for-point visual projection does not adequately explain the above visual dysfunctions, because seemingly delimited areas of brain damage do not reliably result in circumscribed visual field amblyopias. Instead of a spatially-punctate cortical response, a more widespread activation of visual cortex and a diffuseness of the cortical response may be the necessary neural mechanism for normal visual abilities, in man as well as in cat.

Electrophysiological Studies

As noted above, both anatomical and clinico-pathological studies have contributed to our understanding of the mechanism subserving the visual perception of space. From a purely functional point of view, however, the spatial organization of the cerebral visual system can only be understood by studying the pattern of electrical activity which is initiated in the brain by stimulation. Although electrophysiological studies of the cat's visuo-spatial functions are needed to accomplish this, compared to the growing number of anatomical studies, there have been few such physio-

logical reports.

The earliest and best known electrophysiological studies of the topology of the cat's visual system were by Talbot and Marshall (1941, 1942). They recorded the responses at selected loci on visual cortex of cat, under chloralose and barbiturate anesthesia, evoked by a small target flashed at varying locations in the visual field. For any given cortical locus, the field position of the light flash which evoked a response of maximum amplitude and minimum latency was determined by moving the stimulus light. Another cortical locus was then chosen and the process repeated until "critical" field locations for a number of cortical loci had been determined. Using this mapping method, they found that dorsal field positions projected to posterolateral and posterior splenial gyri, ventral field positions to anterior marginal and splenial gyri, and the area centralis to a cortical locus around AO¹ (Marshall and Talbot, 1940, 1942; Talbot, 1940, 1942; Talbot and Marshall, 1941). Their data, which they only described rather than presenting illustrations or quantitative measures of the evoked responses, agreed in general with the anatomical reports. In a later study, Talbot (1942) provided tentative electrophysiological confirmation of the cortical vertical meridian, lying approximately along the antero-posterior extent of the marginal gyrus at the middle of its medio-lateral extent.

From the retino-cortical maps Marshall and Talbot

¹AO or A-zero: the stereotaxic coordinate determined by the anterior position of the cat's ear canals, on the dorsal surface of visual cortex, approximately at the anterior edge of the posterolateral sulcus.

constructed, they were able to estimate the amount of spatial resolution at cortex ("cortical discrimination") by determining how many degrees of visual angle in the field projected to how many millimeters of visual cortex. They estimated that "cortical discrimination" averaged five degrees of field per millimeter of cortex at 30 degrees below gaze in the cat, and one degree of field per millimeter in the area centralis. Considering this cortical "magnification" of the area centralis, plus the known divergence of optic fibers in the LGB and in visual cortex, Talbot and Marshall proposed that "...the retinal mosaic supplied a much finer-grained cortical mosaic, and (that) the distribution of excitation is highly peaked by the overlap...(the peak) may be even narrower than the initial excitation at the retina." (Talbot and Marshall, 1941, p. 1261). Thus they proposed that the cortical image could be more important to the observer than the retinal image for visual discrimination, although they did not investigate this problem directly by using two visual stimuli to study possible interactions between them. Furthermore, they were the first to stress the importance of spontaneous cortical activity and oscillations of the visual pattern caused by optic nystagmus in visual discrimination; yet their studies were carried out on deeply anesthetized animals whose spontaneous activity was minimal and whose eye movements were virtually absent. The present study was specifically designed to investigate all three

of these problems: the topology of cat visual cortex as determined by single stimuli; in the unanesthetized, alert animal; and to study the cortical response to two spatially-different stimuli delivered simultaneously or separated in time.

The only other systematic electrophysiological mapping of cat visual cortex was reported by Bilge and his coworkers (1967). They studied the gross evoked potential and, in addition, the extracellular unit activity elicited by small photic stimuli at varying locations in the field. The field-cortex topology was described for the splenial and marginal gyri. This study, in general, confirms Marshall and Talbot's earlier work, in that dorsal retina was found to project to anterior cortical loci, and ventral retina to posterior loci; but in addition, Bilge et al. drew their map of the cortical retina for every ten degrees of the cat's gaze (Marshall and Talbot had simply indicated the general trend of the topologic projection, as determined by gross evoked responses only). There are other relevant studies by Doty (Doty, 1958, 1961; Doty and Grimm, 1962) who used both photic and electric shock stimuli to the retina, the latter to avoid the possible contamination of entopic stray light. He found, in both instances, that stimuli presumably limited to one retinal location elicited responses over widespread portions of striate, extrastriate, and suprasylvian cortex. Even when the supposed striate areas were removed, responses over nonvisual cortex remained

intact. Doty has raised the question of how these diffuse cortical responses may affect the precision of the topological system.

There are two studies of photically-elicited unit potentials in the LGB which deal with the spatial organization of the retina onto the lateral geniculate body (P.O. Bishop et al., 1962; Daniel et al., 1961). Both confirm the reported retino-geniculate organization: light flashes in dorsal retina activated units primarily (but not exclusively) in anterior LGB, while ventral retinal flashes usually elicited activity in posterior LGB. In addition, peripheral flashes evoked unit firing in lateral regions of the LGB, whereas flashes closer to the retinal vertical meridian evoked activity more medially in the LGB.

In common with most other investigations of visuo-cortical activity, Talbot and Marshall used deeply anesthetized preparations only (Marshall and Talbot, 1940; 1942; Talbot, 1940, 1942). Barbiturate anesthesia is known to depress activity of the diffuse cerebral systems (in order to anesthetize the animal) and even to alter and depress the specific cortical response (Curtis, 1940; Marshall et. al., 1943: Wagman and Battersby, 1964). Therefore, most electrophysiological mapping studies have been carried out in preparations whose cortices have been relatively unresponsive. Briefly, one might conclude that such studies have dealt with the response of visual radiations, but not with the normal cortical

organization which underlies visual localization and discrimination. If visual topology were studied in the unanesthetized preparation, then presumably the cortical mechanisms for spatial vision would be more like those in the normal state.

Some electrophysiological studies report that large cortical areas respond to a flash stimulus, larger cortical areas than implied by the anatomical literature. Regions of suprasylvian cortex, for instance, are quite responsive to visual stimuli (Clare and Bishop 1955; Landau et al., 1961; Marshall and Talbot, 1940; Woolsey, 1962). The region of cat cortex that is most responsive to photic and shock stimuli to the optic pathway lies outside the structurally-defined striate area, more laterally on the marginal gyrus (Battersby, 1963; Berkeley et al., 1967; Doty, 1958; 1961; Malis, 1964). Therefore studies of visuo-cortical physiology, especially those investigating the neural processes underlying spatial localization, should record activity of extrastriate as well as of striate cortex.

Electrophysiological studies which define receptive fields of units in visual cortex are relevant to the problem of visual topology in the cat. Stimulation of selected visual field positions activates units within topologically-predictable cortical areas (Hubel and Wiesel, 1959, 1962, 1963; Baumgartner et al., 1965; Creutzfeldt, 1969; Jung, 1961); these findings can be cited in support of the

retinotopic organization on visual cortex. However, the size of the receptive fields of cortical units (defined as the total visual field area which can activate a selected unit) is typically five to ten degrees of visual angle, and occasionally, as large as 40 degrees. Since single cells and fibers in the visual cortex respond to stimuli over widespread retinal areas, it seems difficult to reconcile a precise and circumscribed point-for-point visual projection with these large receptive fields. Moreover, it raises the question of how visuo-cortical activity could subserve accurate visual localization and precise discrimination. If a "lateral interaction" among cortical units exists, then the widespread activity might produce a localized cortical response. Such a circumscribed response would depend upon mutual excitation and inhibition across many units or regions of cortex, as reported by Hartline (1940) for the peripheral visual cell, and by Hubel and Wiesel (1962) for the cat geniculate and cortical cells. It should be noted here that cortical neurons are only one terminus for visual fibers from the LGB. The response of one unit may influence the response of another, and also be modulated by activity in diffuse multi-sensory and efferent pathways. In recent years, the functional importance of mesencephalo- and thalamo-cortical "activating" systems for sensation has been stressed. Specifically, the cortical response to photic or shock stimuli can be influenced by activity in

other cortical or subcortical systems: by the reticular activating system of the brain stem (Jung, 1958), by the medial and intralaminar thalamic nuclei (Jasper, 1952, 1955), by lateral intrinsic thalamic nuclei (Battersby and Oesterreich, 1962; Morillo, 1961), and by input from or to the optic tectum and pretectal nuclei. In short, normal visual perception is mediated not only via a specific retinotopic projection pathway which may be more widespread, anatomically and physiologically, than is commonly assumed, but also through interactions among many neural mechanisms outside the classical visual pathway.

In spite of the multiple and diverse afferent supply to visual cortex, the visual system permits the accurate localization and discrimination of spatial stimuli. In addition to analyzing space, visual cortex must respond to the temporal sequence of incoming visuo-spatial events (Bartley, 1968). The intrinsic spontaneous rhythms of the brain (Bishop, 1933; Talbot and Marshall, 1941), as well as the interval between responses to two stimuli are both temporal patterns which influence cerebral visual function. For example, the visual system may or may not respond to a stimulus which follows another stimulus by a very brief interval, although it would normally respond to that second stimulus alone.

Some electrophysiological studies of "temporal resolution" at visual cortex have presented two flashes or

shocks to a single spatial position on the retina or in the LGB (Marshall, 1949; Marshall and Talbot, 1940; Sturr and Battersby, 1966). In general, the evoked response to the second stimulus (the "test" stimulus) is briefly enhanced for one to five milliseconds (according to some investigators), and then becomes markedly depressed at intervals following the first stimulus (the "conditioning" stimulus) of about 50 to 100 milliseconds. Thereafter, at longer delays, the evoked potential to the test stimulus begins to recover, to reach full amplitude at delays of approximately one-half to one second (Bartley, 1933; Bishop and Clare, 1953; Landau and Clare, 1956; Malis and Kruger, 1956; Schoolman and Evarts, 1959; Sturr and Battersby, 1964, 1966). In the cat, this "recovery" or "excitability" cycle probably depends upon activity in multisynaptic or competitive neural groups, which require about one full second to recover normal function.

The long-lasting influence of one response on another has special significance for studies of the cortical mechanisms subserving spatial localization and discrimination. Previous electrophysiological studies of visual topology have used only single stimuli, yet have generalized their results to theories of discrimination which discuss responses to at least two stimuli. Since a second visual stimulus is known to alter the response to another stimulus, it appears that the relative spatial positions of the two stimuli should be specifically investigated to determine whether the topologic arrangement of visual cortex is altered by

prior stimulation. The present study was designed to investigate effects of dual stimulation on the topologic cortical response.

The electrophysiological design to be described below for the study of the cat geniculo-cortical projection system delivered single stimuli to various positions in the LGB, and recorded responses at the maximally responsive cortical locus and at several other loci as well. In addition to determining visual topology with single stimuli, the study was designed to measure cortical responses to two spatially-distinct stimuli which were presented either simultaneously to the LGB (cortical discrimination), or were separated by varying temporal delays (cortical excitability).

METHODS AND MATERIALS

Preparations - Surgery and Histology

Observations were obtained on a total of 47 adult cats, weighing between 2 to 3 kilograms. Ether induction was accomplished in an airtight box, the trachea cannulated, and the animal thereafter maintained on an ether bottle so that it showed minimal withdrawal to acute pressure between the toes. The animal was aligned in the stereotaxic instrument, and the left saphenous vein cannulated for later infusion of gallamine triethiodide. A midline skin incision was made, and the left temporal muscle was reflected laterally to expose the entire left side of the calvarium. Two trephine openings were made and enlarged with rongeurs, one dorsal and the other lateral. The dura was reflected to expose the left marginal ("lateral") gyrus from about A+16 to A-5 beneath the dorsal opening, and the left ectosylvian cortex beneath the lateral opening. (see Fig. 2, a drawing of the exposures). Bone wax and gelfoam were used during surgery to control bone and tissue bleeding respectively, and a cottonoid and plastic-wrap "dam" was constructed around the cranial defects to retain the moisture from continual saline irrigation of the exposed visual cortex. The comb of four stimulating electrodes (Fig. 3) was calibrated for each experiment and mounted on the stereotaxic instrument to penetrate the lateral exposure and impale, horizontally, the left LGB (at

Fig. 2. Illustration of preparation in stereotaxic instrument. Dorsal exposure of left visual cortex. Stimulating electrodes impale left LGB horizontally.

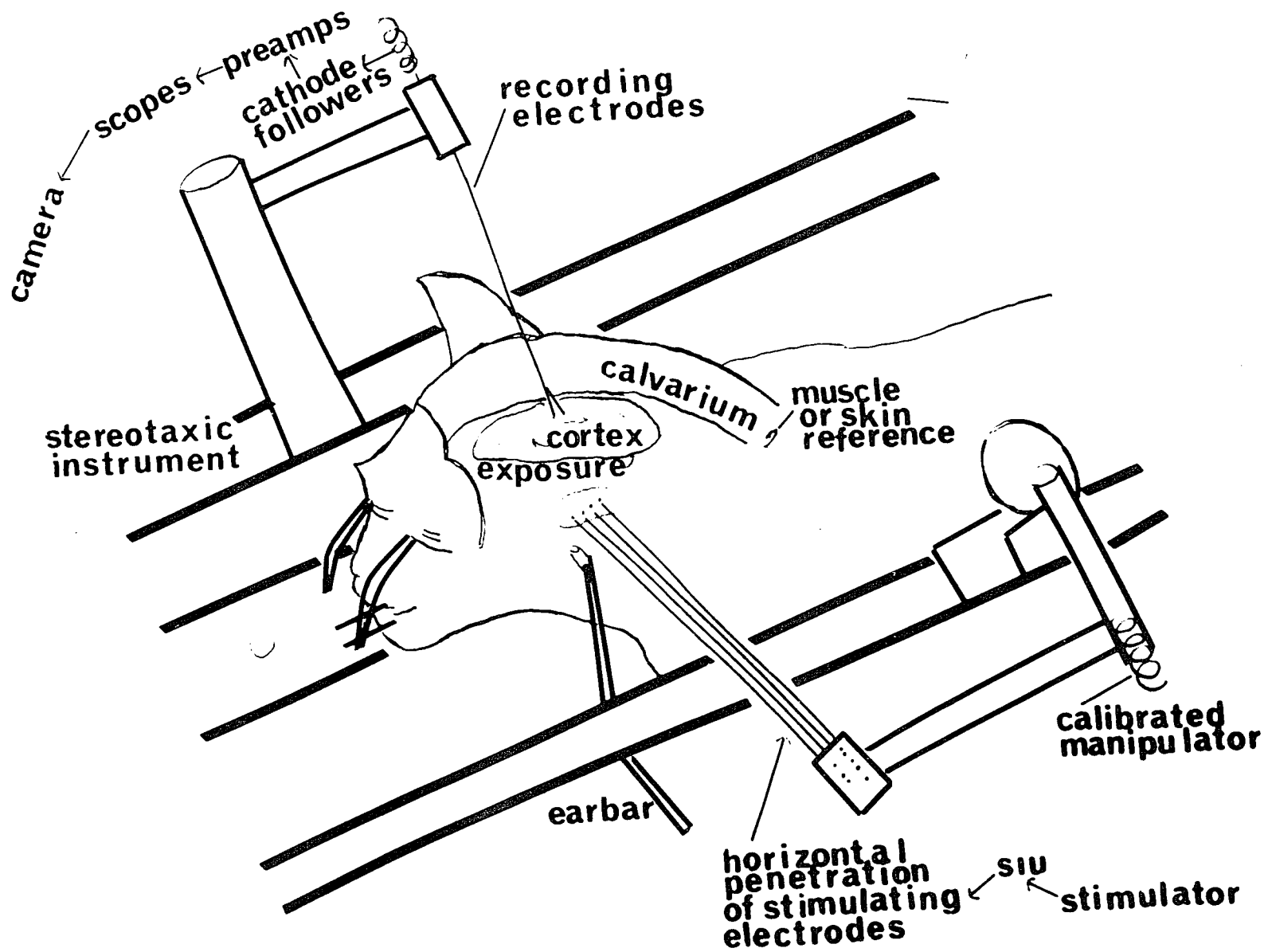
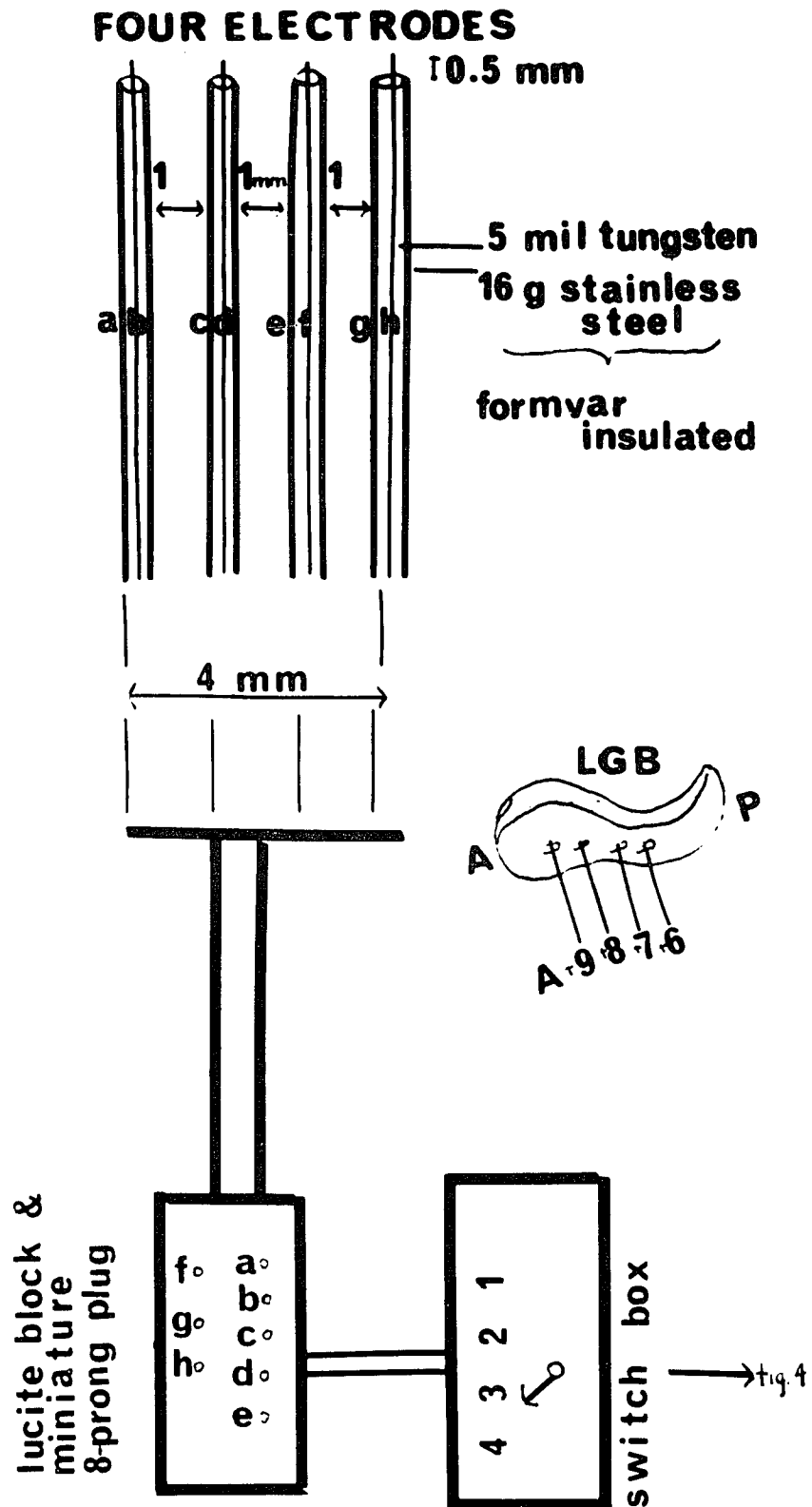


Fig. 3. Schematic drawing of comb of four concentric stimulating electrodes, their configuration and dimensions.



A+9 to A+5; L9.5; and H+3, according to the atlas of Jasper and Ajmone-Marsan, 1954). In this way, the stimulating electrodes did not penetrate visual cortex, and lateral-to-medial positions in LGB could be stimulated sequentially (by varying depth of penetration) in addition to the four anterior-to-posterior positions. All wounds and pressure points were infiltrated with propitocaine. At this point in the procedure, a 1:10 dilute solution of Flaxedil (gallamine triethiodide) was infused slowly into the saphenous vein to immobilize the cat. Ether narcosis was discontinued, and the respirator attached to maintain the animal on artificial respiration at about 35 cc per stroke, and 26 strokes per minute. A heating blanket at body temperature was wrapped around the cat's abdomen, and the rear of the animal was raised to prevent stasis.

At the conclusion of each experiment, selected stimulating positions in LGB were either marked or fulgurated for subsequent histological identification. Marking was accomplished by passing current between the stainless steel tubing of the electrode and the stereotaxic earbars to deposit iron ions for later staining with Hutchison's iron reaction (1953). More frequently, a geniculate position was fulgurated by passing a D.C. current of 0.5 milliamperes between the electrode tip and the earbars of the stereotaxic instrument for 15 to 20 seconds. Two electrodes were usually used to mark geniculate positions at various depths of penetration: the first and third, or the second and fourth electrodes,

although all four tracks could be seen clearly in the sagittal section of the LGB. The animal was then sacrificed with an overdose of Nembutal (intraperitoneally administered), and the brain was marked with a surgical cut made parallel or perpendicular to the angle of the penetrating electrodes. The brain was removed, rinsed, and placed in a 10% buffered Formalin solution for 10 to 20 days. Frozen frontal or sagittal sections, each 48 micra thick, were cut through the left LGB and left visual cortex, and every fifth section was stained and examined under the microscope in order to reconstruct the LGB stimulating positions for each cat (see Fig. 4 which presents LGB sections for five different cats). Most brain sections were stained with the Klüver and Barrera stain (1953) for fibers and cells, except those stained by Hutchison's iron reaction as described above.

Electrodes and Equipment

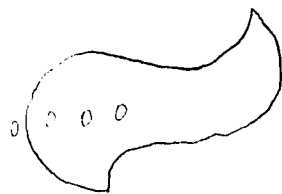
The recording electrodes were monopolar silver balled-tip surface electrodes, each tip about 0.5 millimeter in diameter. Their position on the cortical surface was controlled by a calibrated manipulator and all locations were expressed in terms of the Jasper and Ajmone-Marsan coordinates for the dorsal view of the cat cortex. Each electrode recorded cortical activity monopolarly against a muscle referent. Two electrodes were used simultaneously, separated by about two millimeters in

the medio-lateral direction on the pial surface. Responses from cortex were fed through two Grass cathode followers and two differential AC preamplifiers (Grass), each with a band pass of 7 cps to 10 kc, and then to a two-channel oscilloscope (Tektronix Number 561) and to a slave storage oscilloscope (Tektronix Number 564). Five or more responses were superimposed and photographed for later analyses. Occasionally, bipolar tungsten recording electrodes were used, both tips resting on the pial surface, or tips separated in depth by one or two millimeters. After recording the responses to all four LGB shocks at two adjacent cortical loci, the two recording electrodes were moved to two other cortical loci, as described below.

The comb of four stimulating concentric electrodes was especially designed for this study (Fig. 3). Each electrode consisted of a five mil tungsten wire insulated with Formvar to the tip, placed through the center of a 16 gauge stainless steel tube, also insulated with Formvar to the edge. The center wire projected about 0.5 millimeter beyond the edge of the surrounding tubing. Each electrode was wired independently to a miniature plug mounted on a lucite block on the electrode holder. The plug led to a switch box which was used to select one of the four electrodes through which to pass the stimulating current. Electrode impedances varied between 10 to 50 kohms, as measured in vivo and also in normal saline with an ohmmeter. This comb of four electrodes was used

Fig. 4 Histological sections cut sagittally through left LGB, for five cats, illustrating the anterior-posterior extent of the nucleus and of the electrode tracks. Used to identify stimulating sites in LGB for all cats.

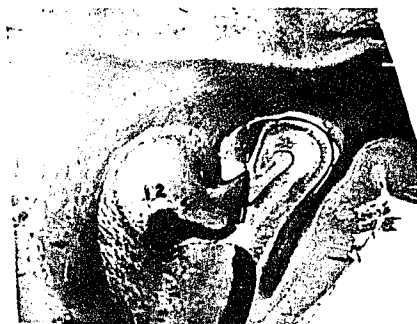
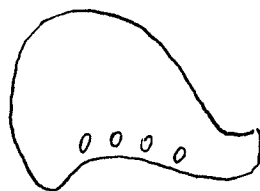
CAT Q158



CAT Q150



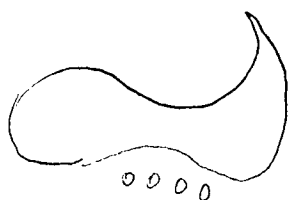
CAT Q171



CAT Q145



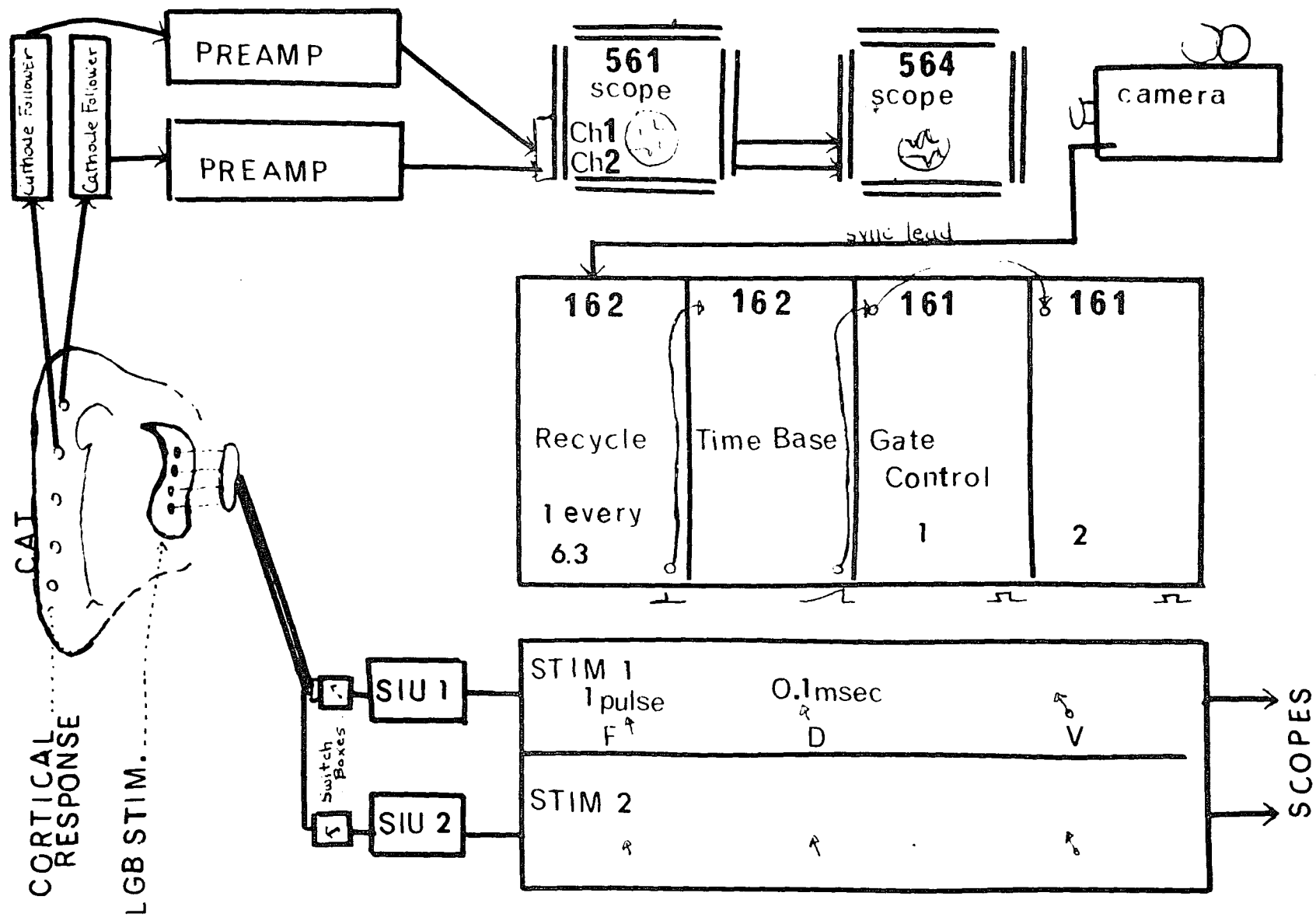
CAT Q179



for the last 36 preparations. For the first 11 cats, three or four concentric bipolar electrodes were aligned for vertical ($n=3$) or horizontal ($n=8$) penetration into the left LGB. Quantitative data were collected for 42 preparations, in which 2, 3, or all 4 electrodes impaled LGB. Only qualitative observations were made in the other five cats (either because only one electrode impaled LGB, or because the film records were overexposed).

The overall timing for stimulation of selected LGB positions, for recording cortical responses, and for photographing the faces of oscilloscopes was controlled by a combination of the Kymograph camera and Tektronix Waveform (Model 162) and Pulse (Model 161) generators which programmed one or both Grass S-4 stimulators and triggered the tracings on the oscilloscopes (see Fig. 5). One Tektronix pulse generator recycled the stimulator to deliver a square wave pulse of 0.5 millisecond duration once every 6.3 seconds. The frequency, duration and voltage settings for a single supramaximal pulse were set on the stimulator, which delivered the stimulating current through an RF isolation unit to the electrode. The current used throughout was set individually for each of the four electrodes and ranged from 0.5 to 1.0 milliamperes, as measured in vivo in terms of the voltage drop across a known in series resistance. When two stimuli were used to study the excitability cycle of the cortical response, the second Grass stimulator was triggered at variable delays after

Fig. 5. Schematic of electrophysiological stimulating and recording system.



the first, using the second pulse generator unit.

Procedures and Data Analysis

All experiments were performed in a shielded, dark, and quiet room. While monitoring electrical activity of visual cortex, the stimulating electrodes were advanced toward the LGB in 0.5 millimeter steps, and each of the four electrodes stimulated in turn until definite responses were noted. The evoked responses to each of the four electrodes were then recorded at the two cortical loci, and the recording electrodes were then moved from these loci to the next. If the anterior-posterior topologic organization was to be studied, the electrodes were moved anteriorly or posteriorly, two to three millimeters at a time, along the marginal and posterolateral gyri, so that at least five A-P loci were studied. If the medio-lateral geniculocortical topology was under investigation, the electrodes were moved one or two millimeters at a time across the width of the marginal and posterolateral gyri from the midline to the lateral sulcus, and at least three A-P loci were studied.

The filmed data for each cat, of five superimposed tracings in response to each stimulus, were analyzed. Measurements were made of the amplitudes and latencies of each component and the total shock-evoked response (see Fig. 6). Latency was defined as the time between the stimulus and the peak of the component, i.e. the component's

maximum deflection from the baseline cortical activity. Since most records were filmed at a sweep speed of two milliseconds per centimeter on the oscilloscope, baseline activity was essentially a flat line, and signal to noise ratios were excellent. Amplitude of the total response was expressed, in microvolts, from the maximum positive-going deflection to the maximum negative-going deflection. The amplitude of each component was measured, also in microvolts, between the initial deviation from baseline or between the preceding component and the maximum (peak) deflection from baseline (Fig. 6 illustrates the response).

For any stimulating position in LGB, that cortical locus which yielded the largest amplitude response was designated the "peak locus" and the magnitude of responses at all other cortical locations was expressed in percent of maximum. By disregarding absolute magnitude of the response, the cortical distribution could be studied apart from varying electrode resistances at different stimulating sites within LGB. It should be noted that the peak cortical locus for one LGB position may have shown a larger amplitude response to stimulation of another LGB position in absolute terms. The response to this second stimulating position would have peaked, however, at a different cortical locus.

When the experiment dealt with cortical recovery cycles, two stimulating electrodes were selected, one to deliver the "conditioning" or first stimulus, and the second to deliver the "test" stimulus; and responses at

several loci on cortex were filmed. The temporal interval between the two stimulators was varied between 0 milliseconds and 400 milliseconds in eight to ten steps. Each pulse had a duration of 0.1 millisecond, and was adjusted for supramaximal strength as described above for the single stimulus. In some preparations, the conditioning and test stimuli were delivered through the same concentric electrode to contrast this excitability cycle with those obtained when two spatially-different electrodes were used.

The amplitudes of the responses to double stimulation were graphed differently from the data gathered using single stimulation. For each temporal delay studied, the amplitude of the response to the "test" stimulus alone was set at 100 per cent (the control value), and the amplitude of this response when it followed a "conditioning" stimulus was expressed in percentage terms of the control. Thus if the amplitude to a conditioned response was less (or more) than 100%, it was concluded that the conditioning stimulus served to depress (or enhance) the test response.

Three different methods were used to depress the activity of a selected cortical locus. In three preparations a two millimeter square of KCl-soaked filter paper (of 10% to almost saturated concentrations) was placed on the cortical locus, surrounded by an annulus of MgCl_2 -soaked filter paper. The latter was intended to restrict the spread of KCl so as to depress activity of cortex only at the center. In two other cats a chip of dry ice was applied

topically to a two to three square millimeter area of exposed cortex in order to depress its activity (via hypothermia). In seven instances, the cortical locus was aspirated using an 18 gauge needle. After piercing the pia, a circular area about three to four millimeters in depth down to the underlying white matter could easily be aspirated with little if any bleeding.

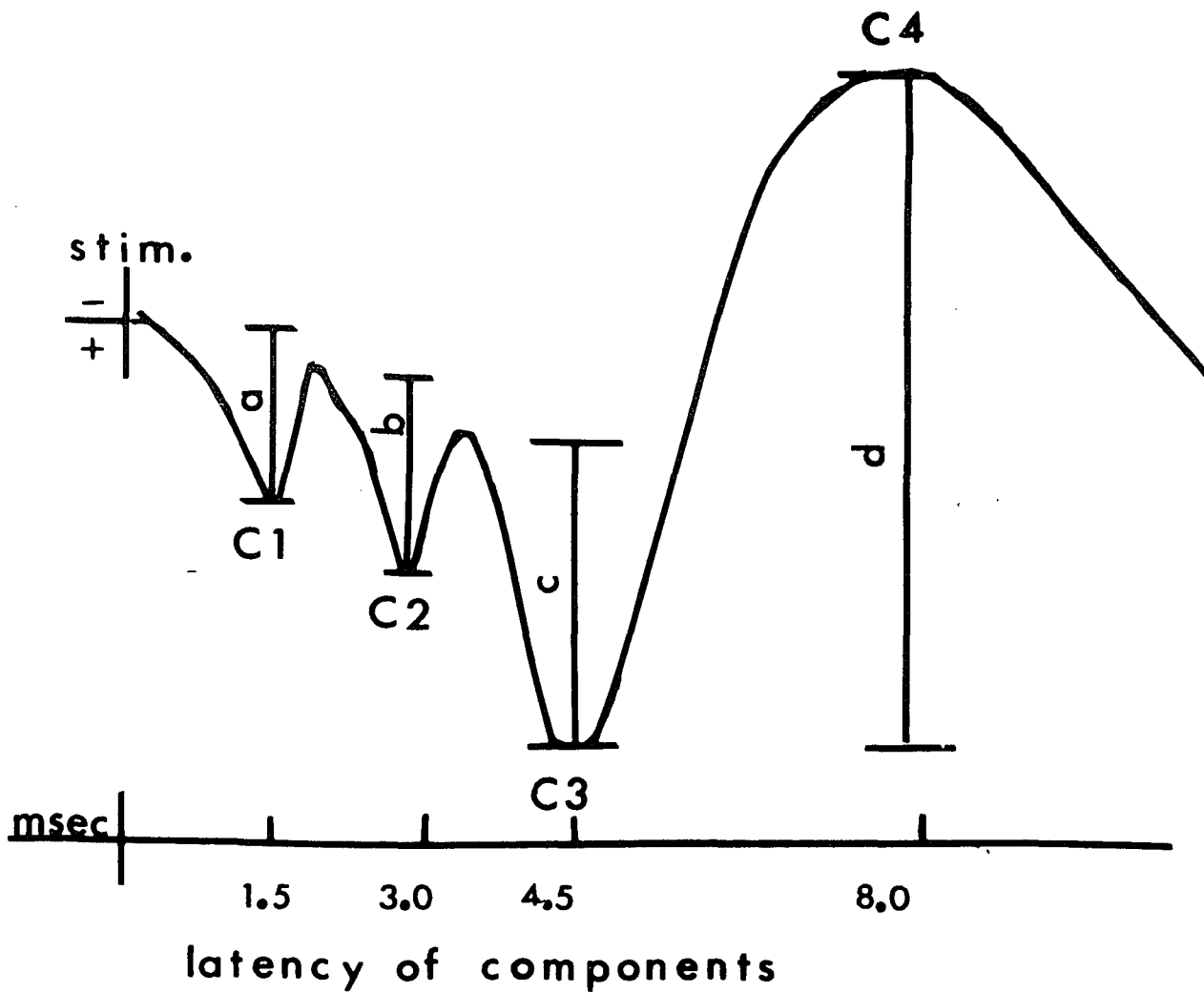
RESULTS

The Potential Evoked in Visual Cortex by Geniculate Shock: General Characteristics

The present study utilized the potential evoked in visual cortex by geniculate shock because the response is reliable, and because so much prior work has been devoted to the analysis of its origin (see Bartley, 1959; and Landau, 1967 for recent summaries). Usually the various components of this response are numbered sequentially, either four or five or six components being described, depending upon the author. The four major (largest) components were analyzed in the present study and are illustrated along with the method of their analysis in Fig. 6. The response consists of a series of three (or more) surface positive deflections followed by a final surface negative deflection. The amplitude of the total evoked potential is measured as the separation between the largest surface positive component (usually but not always component three, C3) and the peak of the surface negative deflection. For the response illustrated in Fig. 6, the total amplitude measure was the same as the measure of C4 alone; when the largest positive deflection was C1 or C2, instead of C3 as illustrated, the amplitude of the total response differed from the measure of the amplitude of C4 alone. Typical latency values for each of the four components are shown in Fig. 6.

Fig. 6. A shematic drawing of the typical shock-evoked response in visual cortex of the cat. The measures of amplitude and latency for each component are illustrated. (Surface positive activity with respect to the referent is indicated as a downward deflection, and negativity as an upward deflection.)

- a - amplitude of C1
- b - amplitude of C2
- c - amplitude of C3
- d - amplitude of C4 & of ER



Previous studies have used only barbiturized preparations to study the characteristics of the shock-evoked cortical response. The present study used unanesthetized cats so that the spatial and temporal aspects of the visuo-cortical response would be as normal as possible. It was initially necessary, therefore, to evaluate the general form of the evoked response to the unanesthetized preparation. In comparison to two deeply barbiturized (Nembutal) preparations studied in the present series of experiments, the waveforms evoked by geniculate stimulation in the immobilized (unanesthetized) preparations were essentially similar, but the amplitudes and latencies of the response (especially of C3 and C4) seemed to be more variable than those of the barbiturized cat. It was assumed that the cortical activity in the unanesthetized preparation closely resembled the normal cortical responsivity.

Prior studies on anesthetized animals have shown that the various components of the shock-evoked response can be selectively altered when cortical excitability is either enhanced or depressed by drugs, mechanical pressure, anoxia, or surgical procedures. Most investigators agree that the earliest component (C1) does not react to these changes in cortical excitability (Bishop and Clare, 1952; Clare and Bishop, 1957; Bremer and Stoupe, 1956; Landau et al., 1964; Malis and Kruger, 1956). Some also report that C2 as well as C1 is not markedly affected (Chang, 1952; Chang and Kaada, 1950); therefore, C1 and C2 are often described as

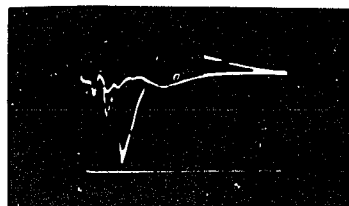
indicating afferent activity, i.e. the "radiation response." On the other hand, C3 and C4 show a marked response to these manipulations of cortical excitability, and are usually judged to be more dependent upon intracortical processes. Fig. 7 illustrates the effect produced by topical application of KCl in the unanesthetized cat used in the present study. The shock-evoked potential is attenuated, especially C3 and C4, which appear to be absent when the cortex is depressed. Similarly, Fig. 7 illustrates the effect on the evoked response produced by inducing hypothermia at the cortex in the unanesthetized cat. The response amplitude is diminished by about 67% after the cortex is cooled. Component C3 and C4 again are more vulnerable to alteration of cortical excitability than are C1 and C2. The results illustrated concur with data reported by previous investigators in the anesthetized preparation. The prior studies (as listed above), however, did not delimit the cortical area to be manipulated, i.e. they applied KCl or cooling techniques to all of visual cortex. Since the data are similar when only a delimited cortical area (two to three millimeters) is depressed in the unanesthetized cat, it may be that the unanesthetized preparation is more sensitive to cortical manipulations of the kind described above, possibly because its spontaneous activity level is higher than in the barbiturized cat.

Fig. 7 presents results when a circumscribed area of cortical tissue was aspirated and the response of under-

Fig. 7. Cortical Potential Evoked by Single Shock to LGB, before and after cortex was depressed by topical application of KCl (Cat Q166), by hypothermic cooling (Cat Q175), and by circumscribed aspiration of cortical tissue (Cat Q179). Note that the gain on the "after" response for Cat Q179 is twice the gain as the "before" response.

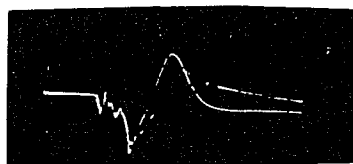
**CORTICAL
R**

**before KCl
2500 μ v**

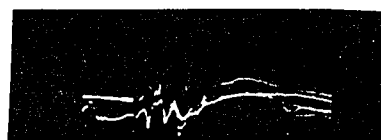


**DEPRESSED
R**

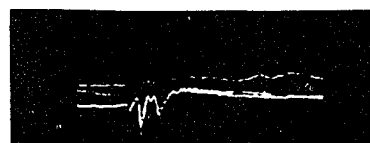
**after KCl
500 μ v**



**hypothermia
1500 μ v 450 μ v**



**a spiration
300 μ v 150 μ v**



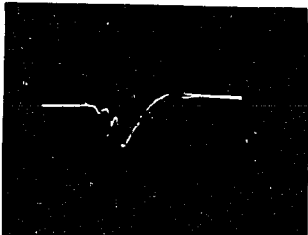
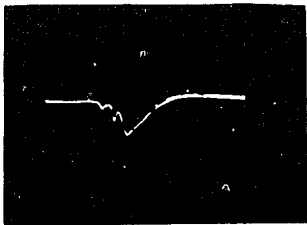
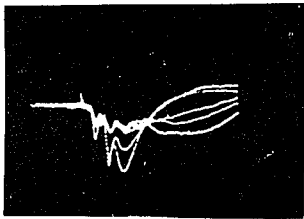
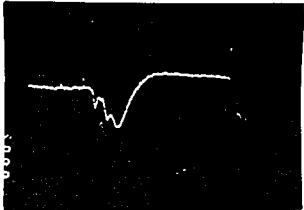
lying white matter was recorded, in the immobilized preparation. The response of the radiations is diminished by about 50% compared to the normal cortical response, but C3 and C4 are still evident, probably because they are recorded from adjacent cortical tissue.

This contrasts to previous studies in which the entire visual cortex was removed and C3 and C4 were shown to be absent from the evoked response.

The components of the shock response are also differentially affected by varying stimulus strength (Chang and Kaada, 1950). When the stimulus intensity is high, a supramaximal evoked potential results (see Fig. 8, top trace). Stimuli at lower intensities evoke responses of submaximal amplitude, as illustrated in the lower tracings of Fig. 8. Interestingly, C3 and C4, the intracortical responses, exhibit higher thresholds than the earlier components, since stronger stimuli are required to elicit them. Chang and Kaada (1950) who reported a more detailed analysis of the effect of stimulus strength on the evoked potential (i.e. thresholds and rate of growth of the components) concluded that C3 and C4 in their deeply anesthetized preparations also showed higher thresholds than the earlier components.

The four components analyzed in the present study were also shown to be differentially affected by prior conditioning stimuli (Bishop and O'Leary, 1938; Bremer and Stoupe, 1956; Clare and Bishop, 1952; Malis and

Fig. 8. Tracings of the Evoked Response at cortical locus AO after stimulating LGB at A+7 (Cat Q156). The supramaximal response (top picture) illustrates the typical shock-evoked potential measured in this study. Lower stimulus intensities, down to a threshold intensity (T), evoked small responses. (Response amplitudes, peak-to-peak, are indicated to the right). Five tracings are superimposed for these and all pictures; surface positivity is downward.

Stimulus I	CAT Q156	Amplitude
Tx3		1000 μ V
Tx2.5		900 μ V
Tx2		225 μ V
T		150 μ V

Kruger, 1956). The general paradigm for these studies is to deliver a "conditioning" shock to the geniculate (or to the tract or radiation fibers) and to measure its effect on the amplitude of a second response to a "test" shock presented at various intervals ranging from 0 to 500 milliseconds following the conditioning shock: the "recovery cycle" of the second cortical response. A typical recovery cycle is illustrated and graphed in Fig. 9 for the immobilized cats used in the present study. As in the previous reports, the conditioning and test shocks were delivered to the same spatial position in LGB. Since the amplitudes of C3 and C4 are more depressed relative to the amplitudes of C1 and C2 (seen especially at intervals of 25, 50, and 80 milliseconds), C3 and C4 may be interpreted as due to multisynaptic or long lasting dendritic cortical activity. The previous studies have presented similar data gathered from the barbiturized preparation, showing that the amplitudes of the later components are more depressed than the amplitudes of the earlier components, although the relative effect of a conditioning shock on the amplitude of C2 is still open to question.

The foregoing data¹ indicated no marked difference between the responses in our unanesthetized cat and the

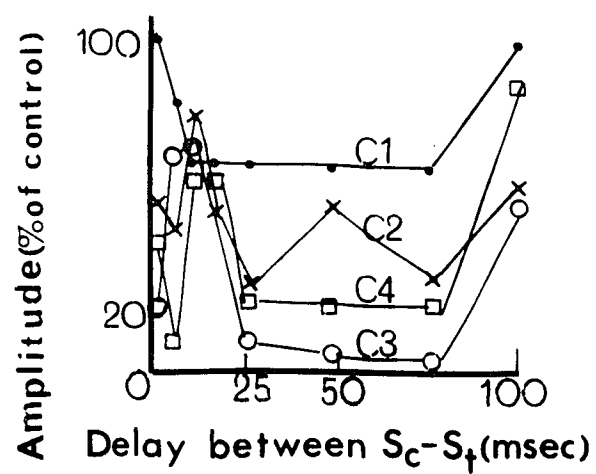
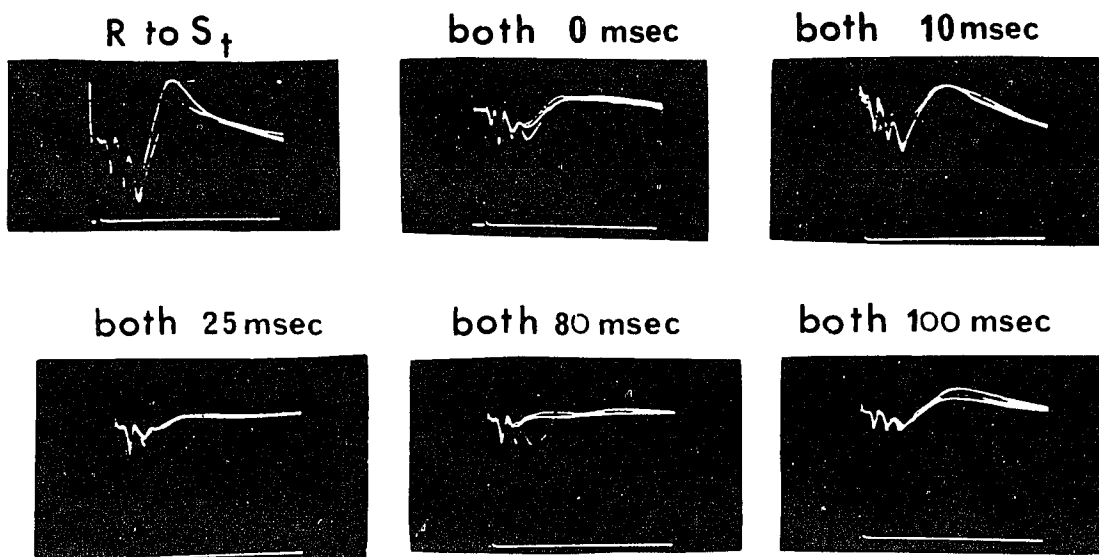
¹Laminar stimulation and recording have been used to investigate at which cortical depth each component originates (Bishop and Clare, 1951, 1952, 1953a, 1953b; Bishop and O'Leary, 1938; Bremer and Stoupe, 1956; Malis and Kruger, 1956). Different investigators have reported variant results, C1 is always and C2 is sometimes seen in the response of the white matter underlying visual cortex (i.e. the radiation response); C3 and C4 are recorded only within the cortical lamina. This procedure was not utilized in the present study.

Fig. 9. Excitability cycle for cortical locus A+3 in Cat Q166. Both S_c (conditioning shock and S_t (test shock) were delivered to A+7 in LGB. The response to a single shock (control response) is depicted in the first picture (top left), and the conditioned responses at five delays (0, 10, 25, 80, 100 msec.) follow. The bottom figure graphs amplitudes (per cent of control value) of the four components of the response (C_1, C_2, C_3, C_4) across eight temporal delays.

CAT Q166

R at A+3

Shock at A+7



general characteristics of the shock-evoked potential on visual cortex of the heretofore exclusively used anesthetized preparation. Therefore, the "normal" or unanesthetized cat was used for the studies to be described, and the shock-evoked response and its four components were measured. The following sections describe studies on spatial and temporal aspects of the geniculo-cortical system. The topology of the cerebral visual system is investigated by delivering shocks to varying spatial positions in LGB, and recording cortical responses across several visual loci. The influence of temporal parameters of stimulation on the topologic projection is also described. Other studies below use the above procedures to manipulate cortical excitability in order to distinguish the afferent activity and its spatial and temporal characteristics (C1 and C2) from the cortical response (C3 and C4) and its distribution across cortex. Additional studies below investigate the degree of current spread at the stimulating position in LGB, and others describe the mechanisms underlying the diffuse response at cortex.

Spatial Aspects of Geniculo-Cortical Projection

The topologic relationship between the lateral geniculate body and visual cortex was studied in 47 preparations. Emphasis was placed upon the analysis of the anterior-posterior projection, but four preparations were devoted exclusively to studying the topologic arrangement of medial-lateral projections. Responses at all cortical loci to any one LGB stimulating position were analyzed in order to establish the "cortical distribution" of evoked responses for each LGB position. Fig. 10 presents data which are typical of the cortical distributions of evoked responses which were obtained. Photographs of the evoked responses illustrate how the amplitude of the responses at A+6¹ and at A-3 on visual cortex varied with the position of stimulation in LGB. At the anterior cortical locus of A+6 (to the left), for example, stimulation at anterior LGB (A+9) evoked the largest amplitude response. The more posterior the stimulating electrode in LGB (at A+8, A+7, or A+6), the smaller were the responses at this anterior cortical locus. At the posterior cortical locus of A-3 (to the right), the most posterior stimulating position in LGB (A+6) evoked the largest amplitude response, and the more anterior the shock in LGB (A+7, A+8, or A+9), the smaller the evoked potential at this posterior cortical locus. These tracings

¹The coordinate of the cortical locus is italicized throughout the report; the LGB coordinate position is not.

also illustrate how the waveform of the evoked response changes across cortex. For example, stimulating LGB at A+6 evoked a large response at A-3 on cortex, and a small response at A+6 on cortex. At A+6, the later components (C3 and C4) occur at slightly longer latencies than at A-3, and in fact, C3 and C4 are inverted in sign (C3 appears as a surface negative and C4 as a surface positive wave). Because the later portion of the waveform of the evoked response at the cortical locus (A+6) distant from the peak locus (i.e. the locus of the largest amplitude response, A-3) was inverted, it is possible that the later portion of the smaller potential results from propagation of the larger response away from its peak locus intracortically. Therefore, C3 and C4 of the smaller potential cannot be attributed to electrotonic spread of the response within cortical tissue, since electrotonic potentials display no latency differences.

For any electrode site in LGB, that cortical locus which gave the largest amplitude response was designated the "peak locus," and the amplitude of responses at all other cortical loci were expressed relative to it in percentage terms. By dealing with relative rather than absolute response magnitudes, the cortical distribution could be studied apart from varying electrode resistances at different stimulating and recording sites, and could also be independent of the specific geometry of underlying fiber inputs at various geniculate or cortical regions. It should be

noted, however, that the peak cortical locus for one LGB position could show an increase in response amplitude (in absolute terms) when another LGB position was stimulated. The responses to this second stimulating position would have peaked at still another cortical locus however. In the center of Fig. 10, the relative response amplitudes (per cent of maximum) for each of four stimulating positions in LGB (A+9, A+8, A+7, A+6) are plotted as functions of location on visual cortex. These data show that stimulating an anterior portion of LGB (at A+9) produced evoked potentials across 10 to 12 millimeters of cortex, but peak amplitude occurred anteriorly, at A+6 on cortex. As more posterior portions of LGB were stimulated, the distribution of evoked potentials also shifted posteriorly so that the maximum amplitude response appeared at more posterior peak loci. Fig. 11 presents similar cortical distributions of evoked responses for four other preparations (Cats Q150, 145, 171 and 179). In each case, in agreement with known anatomy, stimulating anteriorly in LGB evoked responses which peaked at anterior cortical loci, while posterior stimulation evoked peak responses more posteriorly on cortex. There is definite variability in the data, however, from one preparation to another. For example, in all of the five cats illustrated in Fig. 10 and 11, LGB was stimulated at about A+7. Yet the peak cortical locus varied from anterior cortex (A+13 and A+1) to posterior loci (A0, A-4, A-5). For each cat though, an anterior-posterior

Fig. 10. Column on left gives tracings of evoked responses at cortical locus A+6 to stimulation at four sites in LGB as indicated (A+9, A+8, A+7, or A+6). Column on right gives same data for cortical locus A-3. Center graph portrays relative amplitude of evoked responses on the ordinate (per cent of maximum) as a function of cortical locus (on abscissa in millimeters), for four LGB stimulating sites. As stimulating position in LGB shifts posteriorly, there is a corresponding shift in the cortical locus of the maximum amplitude response, the "peak" response. The vertical bar adjacent to each tracing indicates the extent of $600\mu\text{v}$ in the response. All tracings are at the same sweep speed, as indicated in bottom (center) of figure.

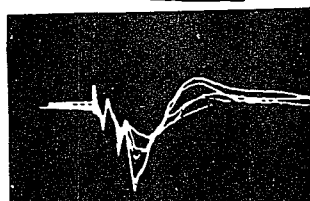
CAT 158

CORTICAL RESPONSES

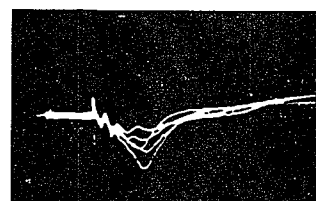
AT A+6

LGB
STIM

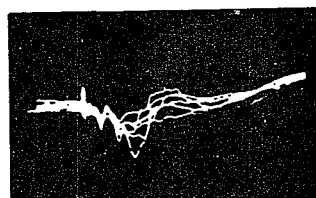
A+9



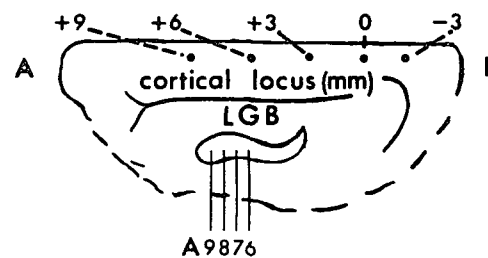
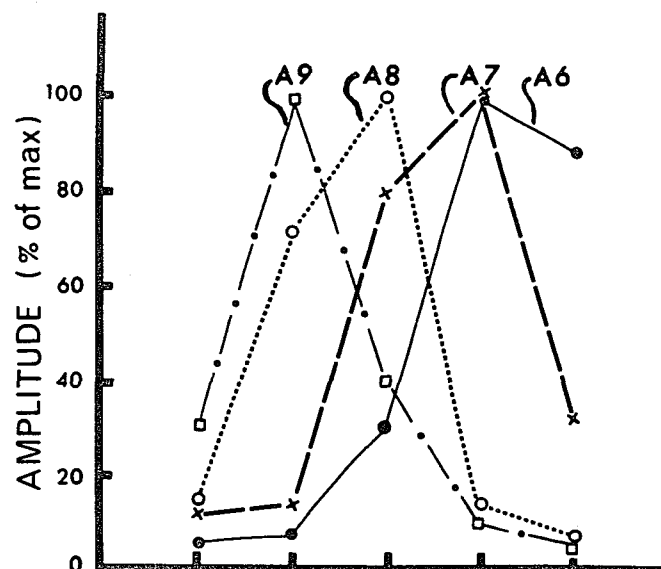
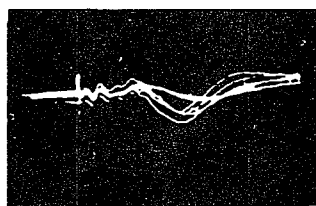
A+8



A+7



A+6



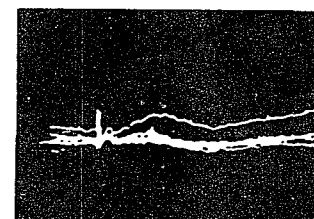
600 μ V
8 msec

CORTICAL RESPONSES

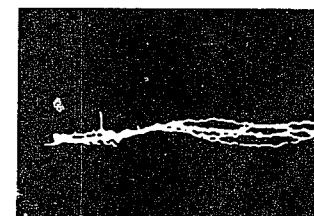
AT A-3

LGB
STIM

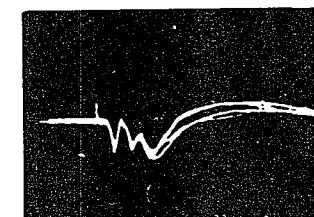
A+9



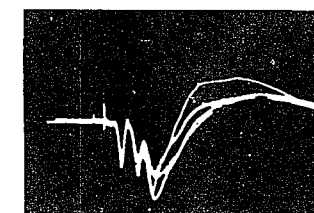
A+8



A+7



A+6

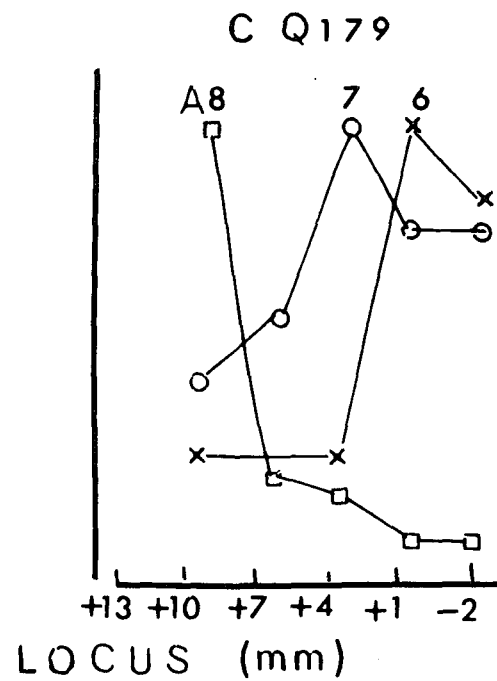
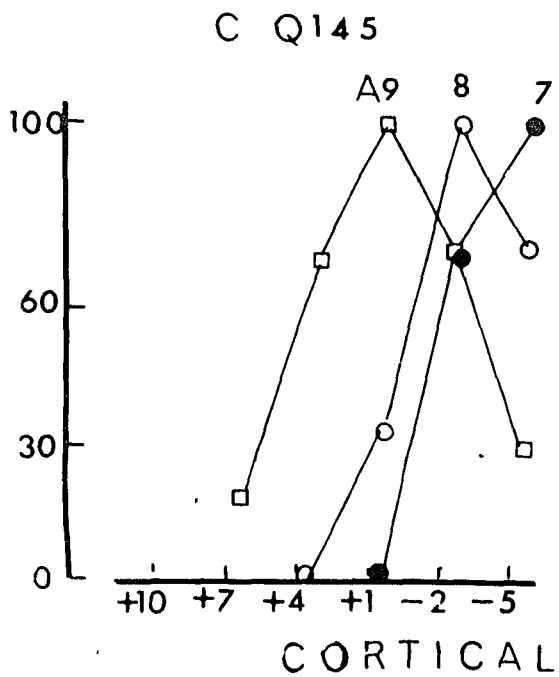
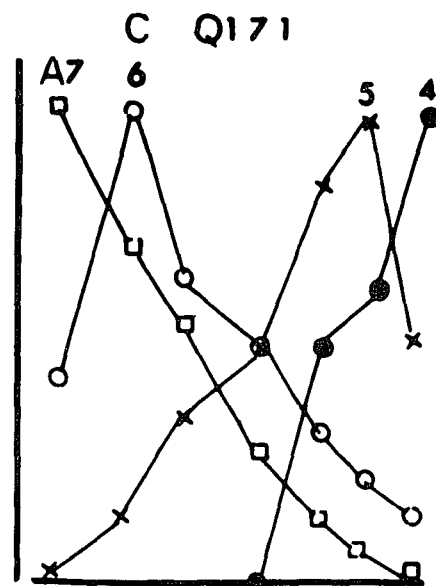
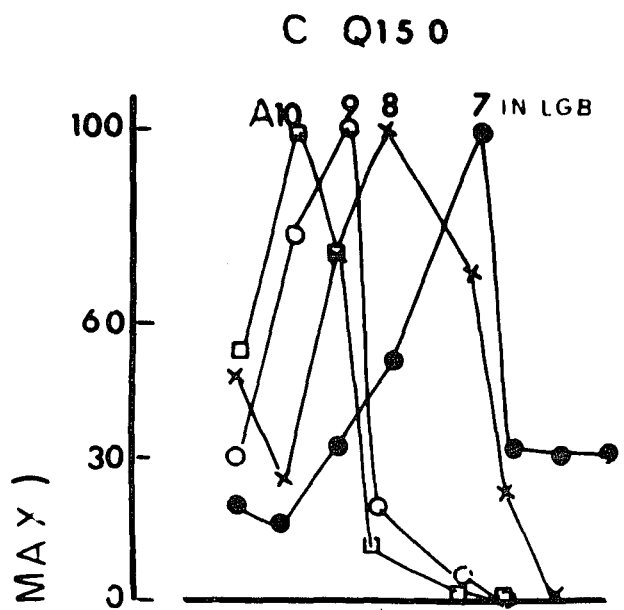


gradient of peak responses was obtained which shifted systematically with the locus of LGB activated. (The histological sections of LGB for these five cats are presented in Fig. 4).

The spread of evoked responses around the peak locus was generally quite broad, most of visual cortex being activated by a single LGB shock. A strict point-for-point topologic organization might hold for peak loci, but would not explain the presence of the smaller amplitude potentials which might functionally interact with the peak responses in the spatial organization of the cat's visual system (see below).

Four preparations were devoted exclusively to the study of cortical distributions of evoked responses as the LGB shock was varied from lateral to medial sites. Here, the distribution of responses across three or four lateralities on visual cortex was studied, instead of the anterior-posterior distribution. Since the exposed area of visual cortex extended from the midline laterally only four or five millimeters, the resolution among medio-lateral projections could not be studied as accurately as when the distribution of responses in the A-P dimension was analyzed. All four cats, however, gave essentially similar data, which are illustrated in Fig. 12 and 13, for one of the cats, Cat Q211. Amplitude of evoked response (per cent of maximum) as a function of the medial-lateral displacement (in millimeters) on visual cortex (L2.0, L3.5, L5.0) are shown

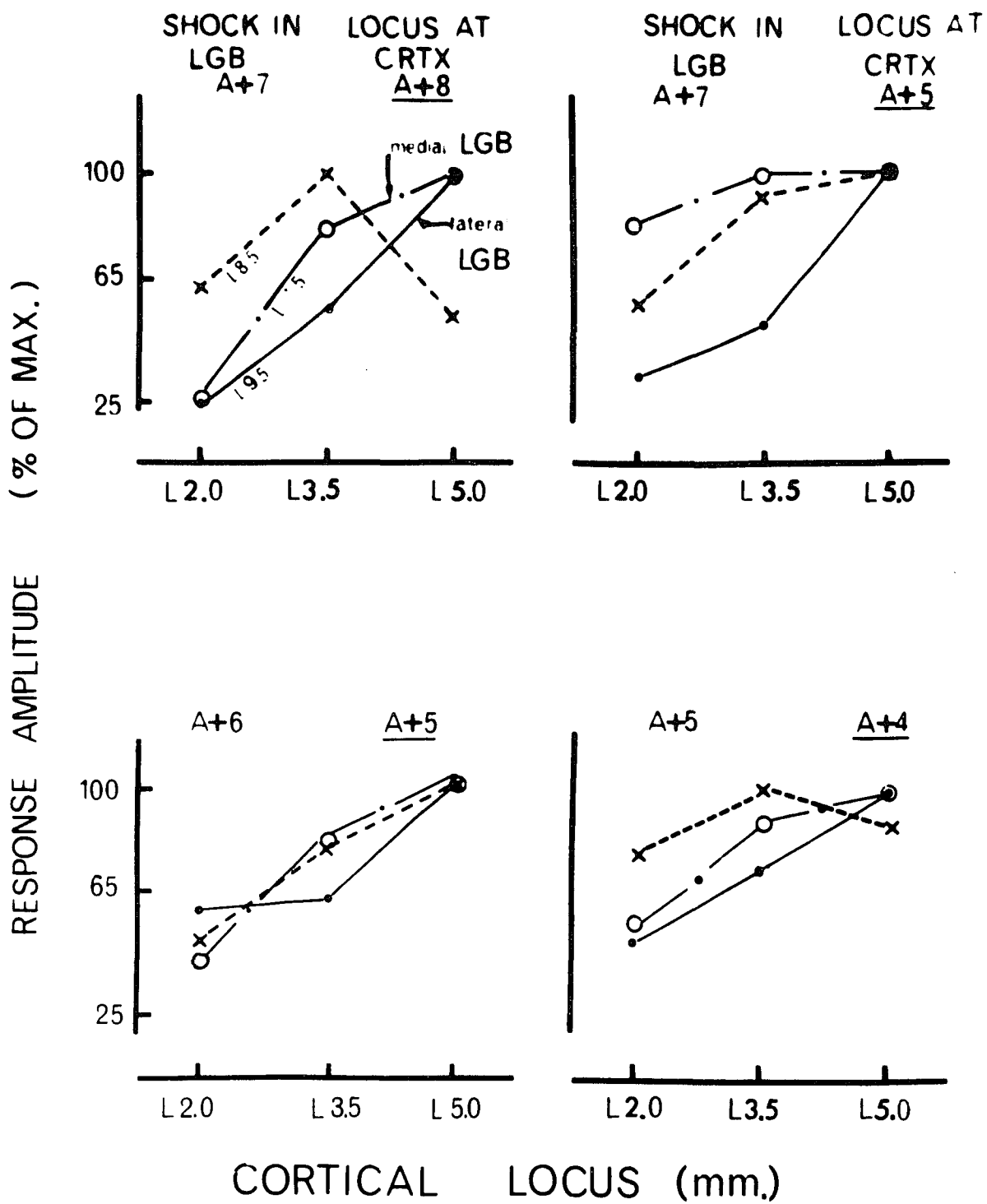
Fig. 11. The A-P topologic relation for four additional cats: Cat Q150, Q171, Q145, and Q179. Amplitude (percent of maximum) is graphed across cortical loci (in millimeters) for either four or three LGB stimulating positions. (See Fig. 4 for histological sections in these cats.)



in each of the four graphs in Fig. 12. The three functions within each graph represent the three stimulation lateralities (in millimeters from midline) within LGB (L9.5, L8.5, and L7.5 in LGB). For example, the top left graph in Fig. 12 is described as follows: when the LGB was stimulated at A+7 in the A-P plane and responses were measured at A+8 on visual cortex, the evoked response peaked at the most lateral cortical locus (L5.0) when the lateral portion of LGB was stimulated (L9.5). When the middle of the LGB was stimulated (at L8.5), the response peaked one and one-half millimeters more medially on visual cortex (on L3.5). However, the most medial LGB stimulus (at L7.5) evoked responses which peaked again at L5.0, the most lateral cortical locus. The same results are seen in the lower right graph for an LGB shock at A+5 and responses at cortical locus A-4. In general, all four graphs show evoked responses of largest amplitude about three to five millimeters from the midline, independent of the A-P location or site stimulated in LGB. Other investigators have noted the same result (Doty, 1958), although some anatomical studies (see above literature review) describe separate geniculo-cortical pathways for medial versus lateral portions of areas 17 and 18 on visual cortex. No clear-cut evidence for these two geniculo-cortical pathways was found in the present study. The spatial resolution of the recording method may not have been sufficiently accurate to measure small changes in response amplitude as the medio-lateral recording locus was varied on visual cortex. (Bi-

Fig. 12. The Medial-Lateral Topologic relation in Cat Q211. Each graph shows the amplitude of the response (percent of maximum) across three lateralities of a cortical locus (L2.0, L3.5, and L5.0), and as a function of the LGB laterality stimulated L9.5, L8.5, or L7.5 going more medially in LGB). In all, three different A-P positions in LGB were studied: A+7, A+6, and A+5. Responses are shown at three A-P cortical loci: A+8, A+5, and A-4, as depicted in the four graphs.

CAT Q211

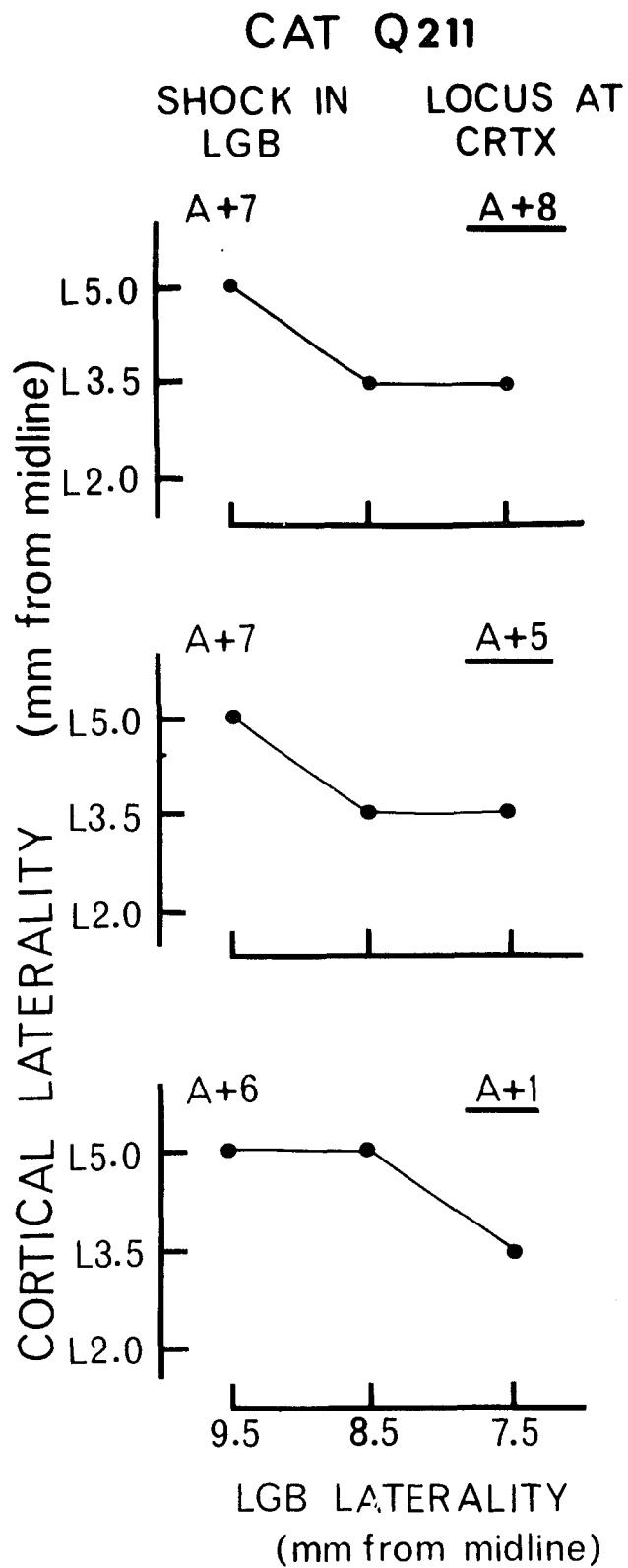


polar derivation of cortical responses was used in one preparation in an attempt to clarify the medio-lateral topology of the cat's cerebral visual system, but no obvious differences between these data and the results above were seen.)

In general, the results of the investigation of the topologic organization of medial-lateral projections can be summarized: (a) At the most medial cortical locus from which it was possible to record in these preparations (i.e. one or two millimeters from midline), the responses were always smaller than at more lateral loci. This was evident even when the stimulus was delivered to medial sites within LGB. (b) The peak response, no matter what the laterality of the LGB shock, was always three to five millimeters lateral to the midline. (c) There was some trend for the locus of the maximum amplitude response to shift medially on visual cortex as the LGB shock moved medially in the thalamus; this trend can be seen in Fig. 13 which graphs the cortical laterality of the maximum amplitude evoked response as a function of the shock laterality in LGB. As the position of the shock moved medially in LGB (from L9.5 in to L7.5), the cortical locus exhibiting the maximum amplitude response also shifted medially toward L3.5.

The cortical distributions describing anterior-posterior and medial-lateral relationships in the cerebral visual system were derived from peak-to-peak amplitude

Fig. 13. The Medial-Lateral Topologic relation: the cortical laterality of the largest amplitude response (L2.0, L3.5, or L5.0) is plotted as a function of the shock laterality in LGB (at L9.5, L8.5 or L7.5). The three graphs illustrate trends at cortical loci A+8, A+5, and A+1 when the shock was delivered to A+7 or A+6 in LGB. (Same cat as in Figure 12, but repeated measures of response amplitudes)



measures of the total evoked potentials at cortex. The various components of the responses were also studied, in order to differentiate between the topology of the presumed radiation input (C1 and C2) as compared to the topologic arrangement of the cortical response itself (C3 and C4).

In 15 preparations, the evoked potentials across visual cortex were of sufficiently large amplitude to analyze each component separately. In each of these cats, the earlier components, C1 and C2, displayed different distributions on cortex than did the later components, C3 and C4. To illustrate, the data for Cat Q171 are presented in Fig. 14. The relative amplitudes (per cent of maximum) of the four major components of the response are graphed across the eight cortical loci (A+16 to A-2) after an LGB shock at anterior LGB (at A+8), or at posterior LGB (at A+6). To the left, the cortical distributions of the components, after LGB shock at A+8, all peak around A+13 on cortex. To the right, the cortical distributions of the components, after LGB shock at A+6, all peak around A0 on cortex. In both graphs, however, the distributions of C1 and of C2 are wider and less restricted around the peak locus than are the distributions of C3 and C4. The earlier components seem to activate a widespread area on visual cortex, while the later components are present across a more narrow cortical area.

In the center of Fig. 14, four tracings illustrate amplitude changes of the four components of the total

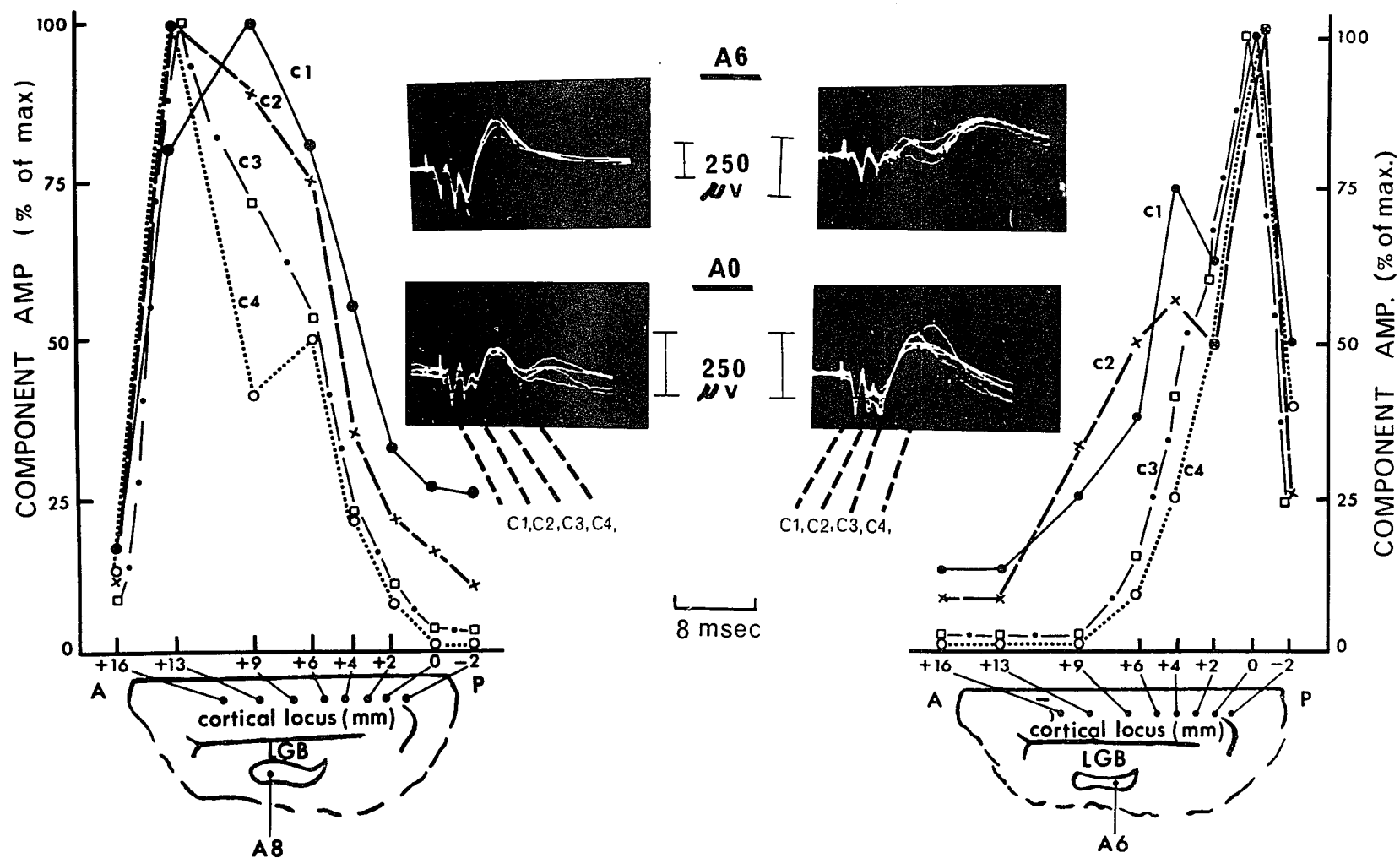
response. When the shock was delivered to A+6 in LGB (the two pictures on the right), the response at anterior cortex (A+6) was about 160 microvolts. The response on posterior cortex (AO) to the same stimulus is larger, about 250 microvolts. Component C1 and C2 remain intact in the smaller response at A+6, while the later components, C3 and C4 are attenuated. The tracings on the left illustrate analogous data at the same two cortical loci (A+6 and AO) after stimulation at A+8 in LGB.

The spatial organization of geniculo-cortical projection, both for afferent input and for the cortical response itself may be contaminated by the fact that all responses were derived using the monopolar recording technique. To investigate if this was so, closely-spaced bipolar electrodes were used in two additional preparations, to record the cortical response. In one, the wire tips were separated by approximately 0.25 to 0.50 millimeter in the anterior-posterior direction, and both tips rested on the pial surface. The cortical distributions obtained from this preparation are illustrated in Fig. 15 for Cat Q149. After a shock was delivered to A+8 or A+7 in the LGB, the response and its components were measured at five cortical loci (at A+9, A+6, A+3, AO, and A-3.) Component C1 and C2 again show distributions in which their relative amplitudes at most loci

Fig. 14. Graph on left gives amplitude on the abscissa (in per cent of maximum), of Components C1, C2, C3 and C4, recorded at eight cortical loci (on abscissa in mm) in response to stimulation of anterior LGB (at A+8). Graph on right gives same data for stimulation of posterior LGB (at A+6). The distribution of later cortical components (C3 and C4) is more restricted around a peak locus (A+13 or A0 respectively) than the distribution of the C1 and C2. The center tracings illustrate the relative amplitudes of the different components at two cortical loci: A+6 and A0, e.g. in response to posterior LGB stimulation, C1 and C2 appeared at both cortical loci, but C3 and C4 were attenuated at A+6.

CAT 171

STIM. POSITION IN LGB
ANT. (A8) POST. (A6)
CORTICAL LOCUS:



across cortex are larger than the amplitudes of C3 and C4 (expressed in per cent of maximum). The width of the distributions for all four components is more difficult to interpret, perhaps because the recording electrode was now showing potential differences only between two closely-spaced cortical sites at one locus, instead of showing any potential change between that locus and a more distant (inactive) referent.

In summary, several characteristics of the functional topologic system within the cat's geniculo-cortical pathways were revealed in these studies. In agreement with the anatomical literature, stimulating a discrete LGB position evoked a response of maximum amplitude at a topologically-specific cortical locus. The geniculo-cortical topology was such that anterior portions of LGB projected to anterior cortical loci although the same LGB position across several cats did not always activate the same cortical locus, identified in terms of its stereotaxic coordinates. In general, the most anterior locus activated by an LGB stimulus was about A+13 on the marginal gyrus. More posterior LGB stimulating positions activated more posterior visual cortex, usually back at about A-4 on the posterolateral gyrus. The A-P geniculate topologic arrangement on cortex is summarized in Fig. 16 for eleven preparations which are discussed in detail in the Results. One can see that geniculate positions from A+9 to A+6 (histologically determined) usually activated peak cortical loci from A+9

to A-1. The line (of best fit) drawn through the points can be thought to represent a scale for geniculo-cortical projection, i.e. which site in LGB projects to which cortical locus maximally, and also, how many millimeters of LGB activate any given area of visual cortex in the cat. For example, one millimeter in the center of LGB, around A+7 can be thought of activating about four millimeters of visual cortex, from about A0 to A+4.

The distribution of evoked responses across visual cortex was analyzed further by comparing the separate distributions of the four components of the total response. Each components also peaked at a topologically-specific cortical locus following a single LGB shock to anterior or posterior LGB. The later components, C3 and C4, which presumably result from activation of intracortical processes, exhibited narrower or more restricted cortical distribution than the earlier components, even when the spatial characteristics of the recording electrode were varied from monopolar to bipolar derivation of cortical activity. The more diffuse cortical distribution of the earlier components, C1 and C2, may reflect diffuse geniculo-cortical afferent input to cortex, as compared to the response of the cortex itself.

Various medial to lateral positions in LGB were stimulated in order to study the cortical distribution across lateralities of cortical loci, i.e. from the mid-line laterally to the lateral and posterolateral sulcus.

Fig. 15. Amplitudes of four components of response recorded with bipolar surface electrode, in Cat Q149, after stimulating LGB at A+8 (top graph) or at A+7 (bottom graph). Note that C1 has an unusually widespread distribution across all cortical loci.

CAT Q149

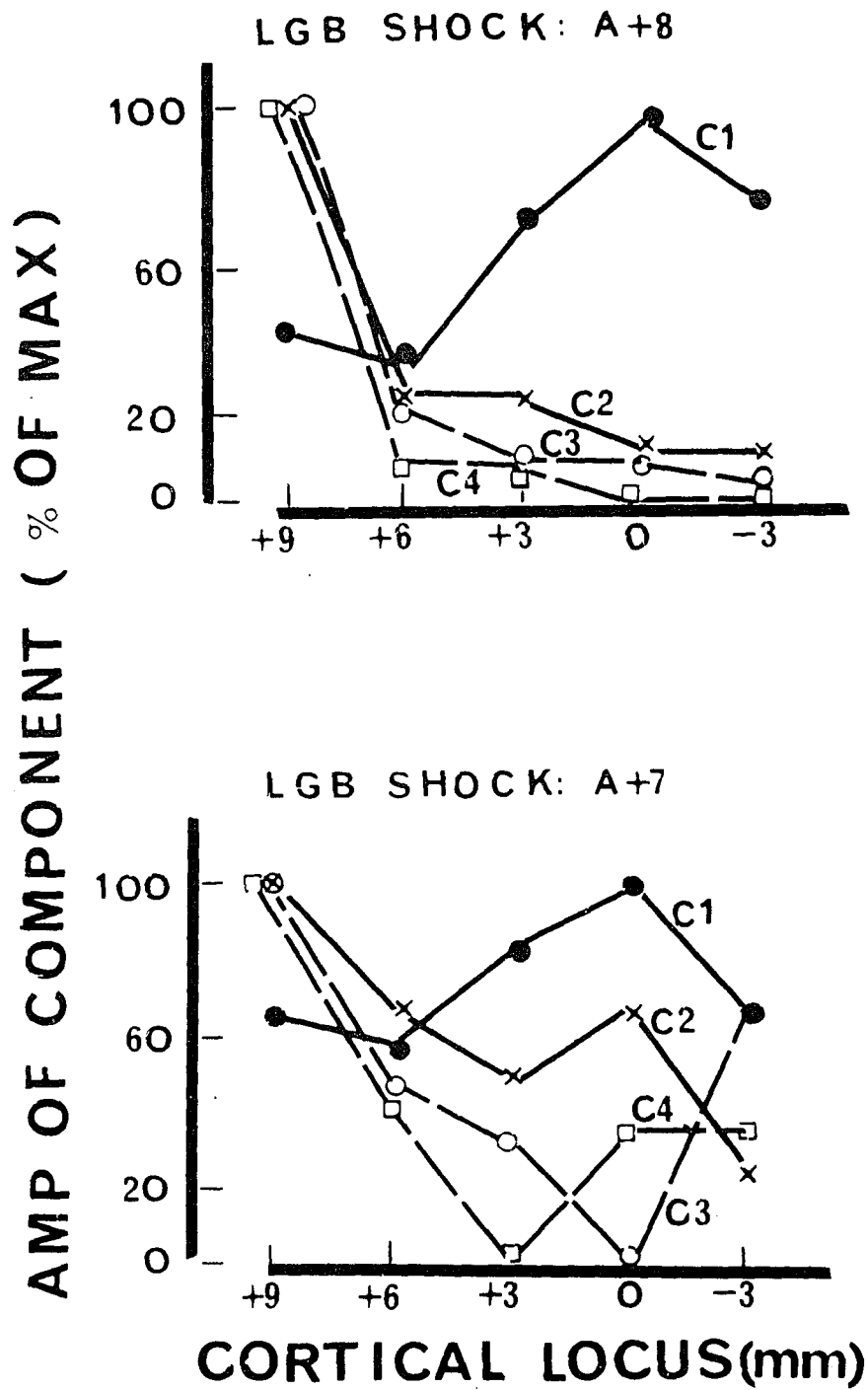
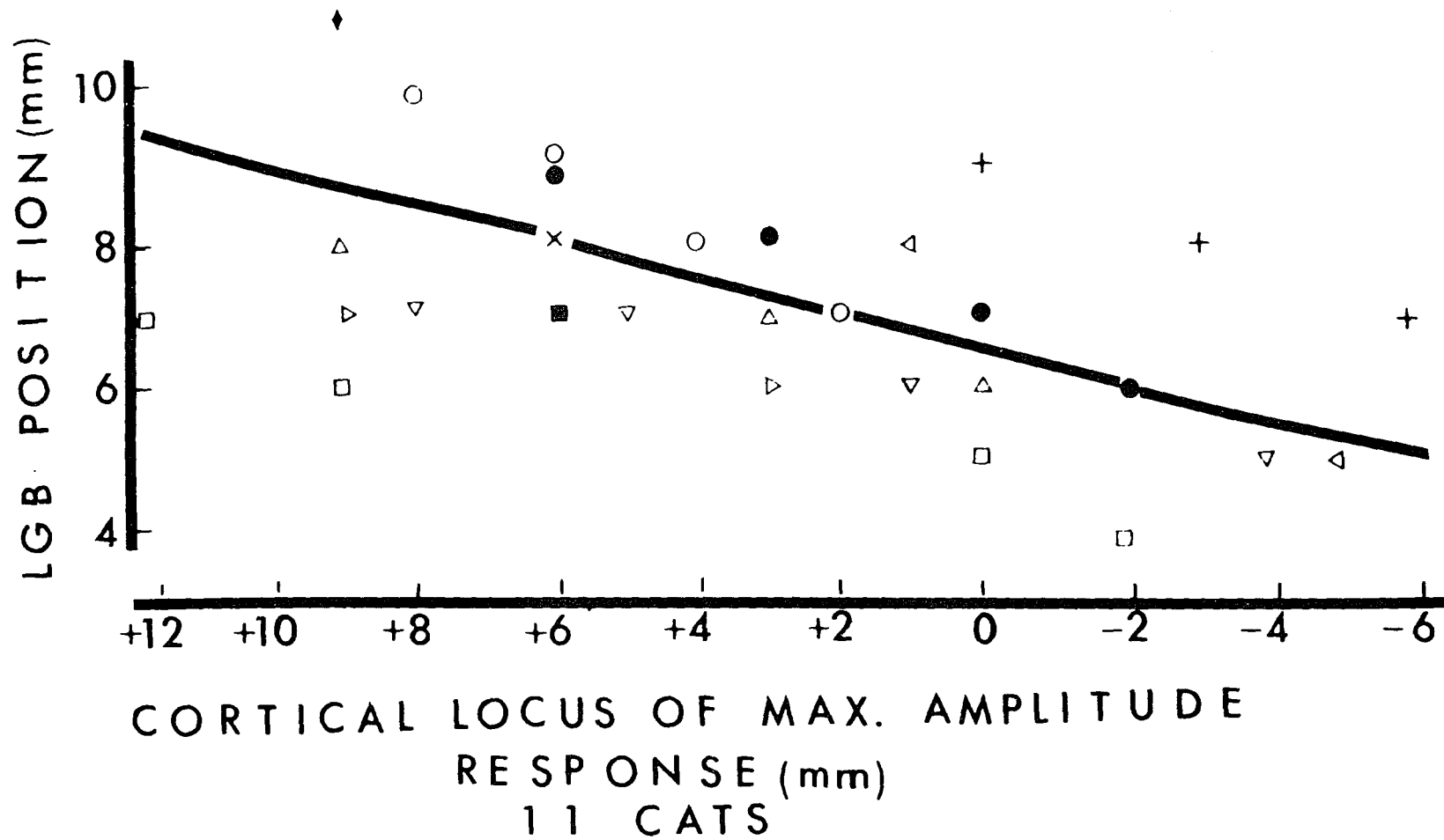


Fig. 16. Summary of anterior-posterior topologic organization in 11 cats as determined by correlating histological examination of LGB with cortical locus of peak response. On ordinate, A-P position of stimulating electrode in LGB (in mm). On abscissa, A-P location of cortical response of maximum amplitude (in mm), using Jasper and Ajmone-Marsan's atlas of cat brain. The estimated line of best fit indicates that about one mm. of LGB activates roughly four mm. of visual cortex (across all stimulating and recording loci).



There was some trend for the cortical locus of the maximum amplitude response to shift medially as the stimulus excited more medial portions of the LGB, but in general, the largest response was usually seen between three and five millimeters lateral to the midline, independent of which LGB laterality was activated.

Temporal Aspects of Geniculo-Cortical Projection

The present research was designed to investigate some of the temporal aspects of geniculo-cortical projection, in addition to its spatial projection as described above. Specifically, two different positions were stimulated in LGB, either simultaneously or at varying temporal delays ranging from five to 200 milliseconds, and the degree of their interaction studied by cortical records. In this way, it was possible to evaluate the significance of the lower amplitude responses elicited at non-peak loci by an LGB stimulus, i.e. the significance of widespread activity within geniculo-cortical projections.

In previous studies, two LGB shocks have been delivered to a single LGB position only, in order to study the recovery cycle of one cortical response. No prior studies have tested how the topologically-specific cortical response to one LGB shock is affected by activation of a spatially different geniculo-cortical pathway. Since no others have used the present approach, the results could only be predicted as follows: If the amplitude of a peak or maximal response to one geniculate shock could be shown to be altered by a response, at that same locus, to a different LGB shock, then one could assume that activity along one geniculo-cortical pathway influences the activity in a spatially-different projection. For example, if the non-peak potentials at any given cortical locus serve a

functional role in the visual system's spatial organization, then these small responses may alter the amplitude of a peak response elicited by a second stimulus. Accordingly, the studies below used the dual stimulation procedure to activate two different LGB positions to evaluate the cortical mechanisms which may underly visual resolution. Also, the interval between the two shocks was varied, and the excitability cycle of the cortical response was studied as a function of the amplitude of the response to the conditioning shock and the cortical locus chosen. Thirdly, it was assumed that if the small non-peak response affected another response occurring up to 50 or 100 milliseconds later in time, that the first small response indicated long-lasting cortical activation.

In all, ten cats were devoted exclusively to studying the effects of double stimulation. Fig. 17 presents the data for three preparations, Cats Q196, 207, and 166. The peak locus of the "conditioning shock" (the first shock of the pair) was, in all three cats, posterior on visual cortex (at A-5, A-3, or A+3 respectively) compared to the peak locus for the "test" or second shock, which was at anterior cortex (at A+1, A+3, and A+9 respectively). Since the conditioning shock evoked small potentials at these anterior cortical loci, the interaction between the small response and the peak response to the test shock could be evaluated. The amplitude of the response to the test shock alone was set at 100% (the control value), and

the response to the test shock preceded by the conditioning shock was expressed relative to the control value. For example, for Cat Q196, the conditioning shock was delivered at A+5 in LGB, and the test shock to A+8 in LGB. At the peak locus of the conditioning shock, at A-5 on cortex, the response to the test shock, when preceded by the conditioning shock was diminished in amplitude relative to the control value, at all nine temporal delays (0 to 200 msec) between the two shocks. This response was maximally depressed (to 20% of control value) at the delay of 80 milliseconds. An entirely different excitability cycle is seen at the peak locus of the test stimulus, at A+1: the small response here to the conditioning shock enhances the size of the test response at its own peak locus. The test response is enhanced (from 10% to 30% of control value) at all temporal delays.

The other two cats illustrated in Fig. 17 show similar trends: at the peak locus of the conditioning shock, the size of the response to the test shock is usually diminished when it is preceded by the conditioning shock. Also, at the peak locus of the test shock, a prior conditioning shock usually enhances the size of the test response by up to 40% of control value. Cat Q166 shows only a short-lasting enhancement of the test response (from 16 to 25 msec. delays), but the preceeding and ensuing depression of the test response is minimal (75% of control), compared to the

responses at A+3 (reduced to about 20% of control amplitude).

Similar results were obtained in all nine preparations used to study the cortical response to dual stimulation. It was evident that the diminished excitability cycle was of longer duration than the enhanced cycle, and that the diminished test response was often reduced to 10 to 20% of control amplitude. A tabulation (Table One) of the data from nine cats shows that the response was diminished at intervals of 50 msec. in all cats, at 10, 25, and 100 msec. intervals in eight cats, and at 5 and 80 msec. intervals in seven cats. In four cats, the test response was still diminished at the 200 msec. interval.

The data on the enhanced test response, also from nine preparations, was tabulated for comparison. In eight cats, the test response was enhanced at the 25 msec. interval, and in seven cats, at the 100 msec. interval. Five cats showed an enhanced test response at the 5 msec. delay. There was also an enhanced response at the 10 msec. delay (four cats), and at the 50 and 200 msec intervals (three cats). The enhanced excitability effect, especially the long lasting influence of a brief conditioning shock, was judged to be a significant finding, qualified by the inter-subject differences above. The magnitude of both the enhanced and diminished excitability effect depended upon the relative amplitudes of the cortical responses to both geniculate shocks, as discussed below.

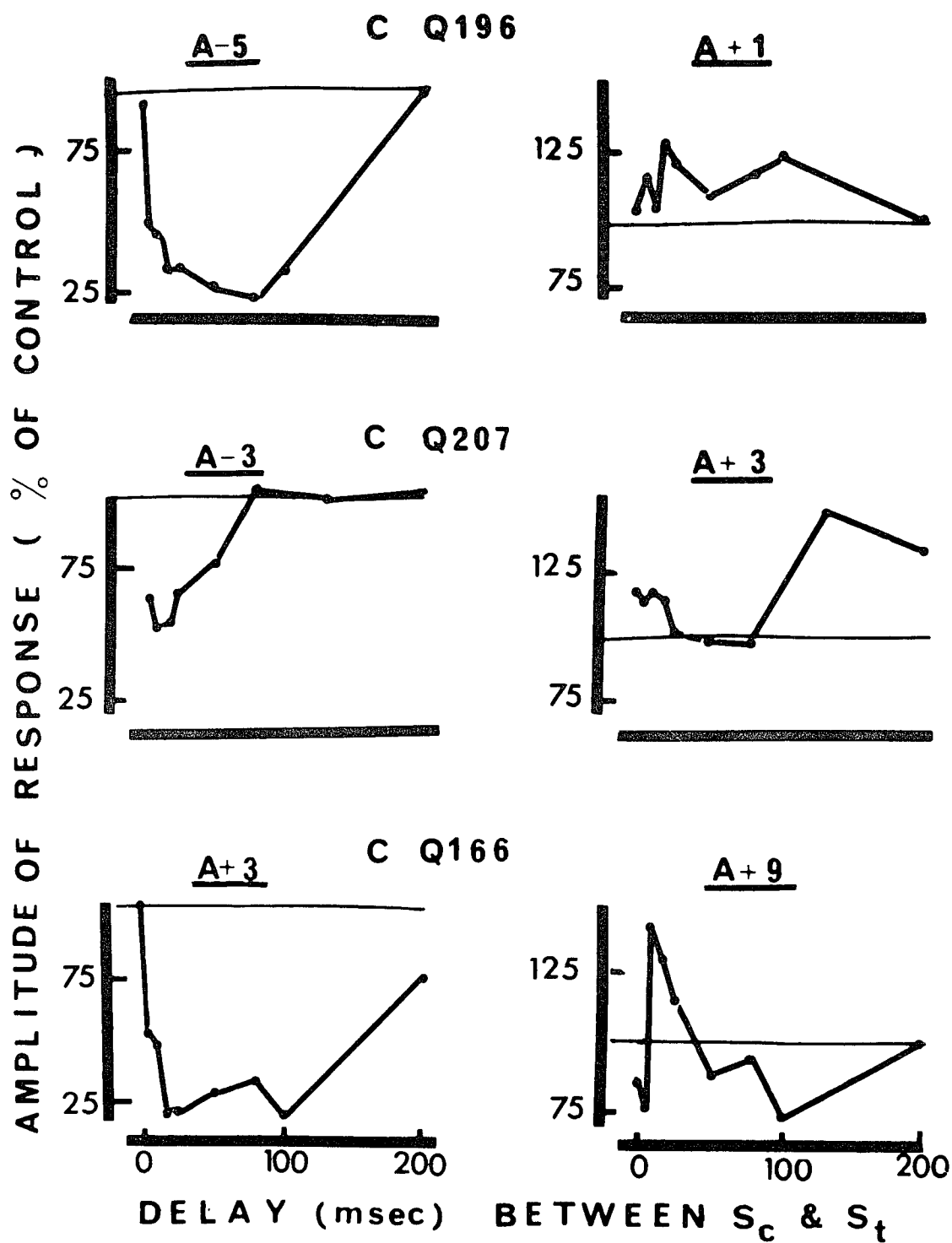
Both temporal phenomena (depression and enhancement of the test response) are seen when the four components recovery cycles are analyzed. Fig. 18 presents the re-

TABLE ONE
Results of Nine Preparations

Enhanced Test Response at following intervals:							Diminished Test Response at following intervals:						
-	-	25	50	100	200	(1)	5	10	25	50	-	100	200
-	10	25	-	100	-	(2)	5	10	25	50	80	100	200
5	-	25	-	-	200	(3)	5	10	25	50	80	100	-
-	10	25	-	-	-	(4)	-	10	25	50	80	100	200
5	-	25	50	100	-	(5)	-	10	-	50	80	100	200
5	10	25	50	100	-	(6)	5	10	25	50	80	100	-
-	-	-	-	100	-	(7)	5	10	25	50	80	100	-
5	-	25	-	100	-	(8)	5	-	25	50	80	100	-
5	10	25	-	100	200	(9)	5	10	25	50	-	-	-

sults for Cat. Q166, where the conditioning shock was delivered to A+6 in LGB, and the test shock to A+7 (one millimeter more anteriorly in LGB). The top graph illustrates the recovery cycle of the components at A+3 on cortex (the peak locus of the conditioning stimulus). Component C1 and C2 here are maximally depressed, to about 20% to 50% of their control value, at intervals between 16 and 100 msec. (Note also that only C1 shows some enhancement or summation potential at the 10 msec. delay.) At the longest interval, 200 msec., the two shocks elicit C1 and C2 of normal amplitude. The amplitudes of the later components, C3 and C4, are more depressed by the conditioning shock than were C1 and C2: at delays between 16 and 50 msec., they are barely visible in the response, because their amplitude has diminished to 10% or less of control value. Even at the delay of 200 msec., they have not fully recovered, and are only about 70% of control value. The lower graph illustrates the converse effect obtained at the peak locus of the test response: Components C3 and C4 are now enhanced (40% or 60% above control amplitude) at delays of 10 to 25 msec, and this effect dissipates more quickly than the opposite (depression) effect. The earlier components here are again less influenced by the conditioning shock since C2 shows a minimal enhancement at the 10 msec. delay, while C1 shows no change in amplitude at any delay. Illustrative tracings of the control and conditioned responses at A+3 and at A+9 are shown for the temporal delay of 25 msec.

Fig. 17. Excitability cycles for double stimulation to LGB, in three cats: Q196, Q207, and Q166. Column to left: response amplitudes (Percent of response to St alone) at peak cortical locus of conditioning shock (on the ordinate) after two LGB shocks at various temporal delays from 0 to 200 milliseconds (on abscissa). Column to right: response amplitudes at peak cortical locus of test shock across same temporal delays.

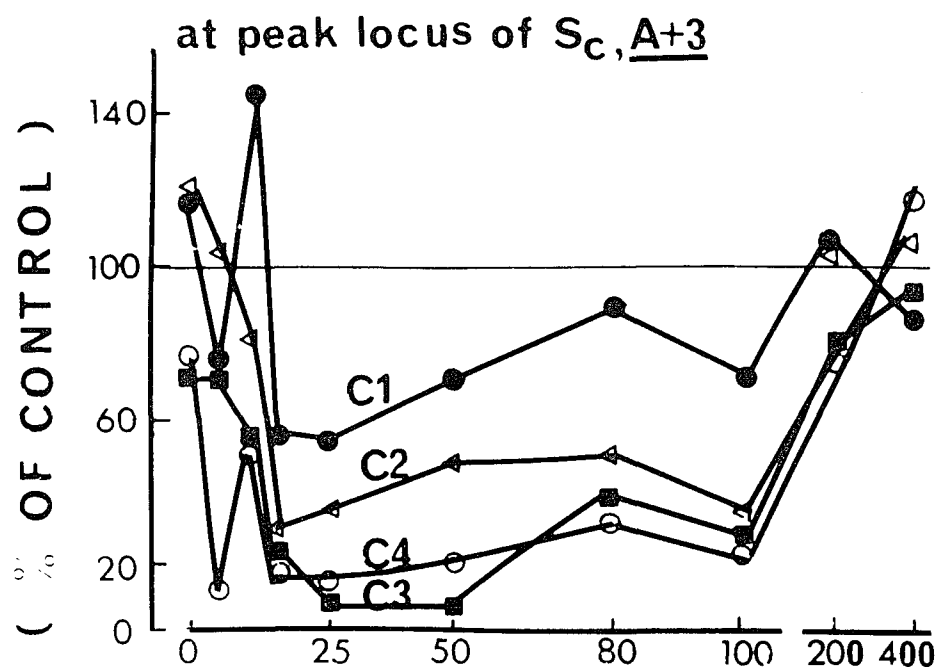
AT PEAK LOCUS OF S_c AT PEAK LOCUS OF S_t 

For the above three preparations in Fig. 17 and 18, the response to the test shock at its peak locus was of larger absolute amplitude than the response at this locus to the conditioning shock. Under this condition, where a smaller response preceded a larger response, the larger response was enhanced by stimulating an adjacent geniculocortical pathway. This sort of physiological interaction could play a role in increasing resolution between two spatially discrete stimuli. In some preparations, the absolute size of the response to the conditioning shock was larger than that of the test response, even at the peak locus of the test response. Under this condition, the excitability cycle of the response at the peak locus of the test shock shows a diminished test response. Fig. 19 illustrates this for Cat Q181 in graphical and pictorial form. These data are analogous to the data to the left of Fig. 17, however, because, in all cases, the test response is diminished in amplitude at any given cortical locus.

It is apparent that the absolute magnitude of the responses to the conditioning shock alone, and to the test shock alone, determines the type of excitability cycle which one obtains in the geniculocortical system. If the response to the conditioning shock is larger than the response to the test shock, then the response to double stimulation is depressed. If the response to the conditioning shock is of smaller amplitude than the response to

Fig. 18. Excitability cycle in Cat Q166. Conditioning shock delivered to A+6 in LGB, test shock to A+7 in LGB. Top graph: response amplitudes of four components (C1,C2,C3,C4) at A+3 on visual cortex (expressed as percent of amplitude of response to test shock alone), across various delays from 0 to 400 milliseconds. Bottom graph: response amplitudes of components at A+9 on visual cortex, to same two shocks in LGB, at same temporal delays. Tracings in column to right illustrate the evoked responses to S_c alone, to S_t alone, and to both at 25 msec. delay, at A+3 and at A+9 on visual cortex.

CAT Q166



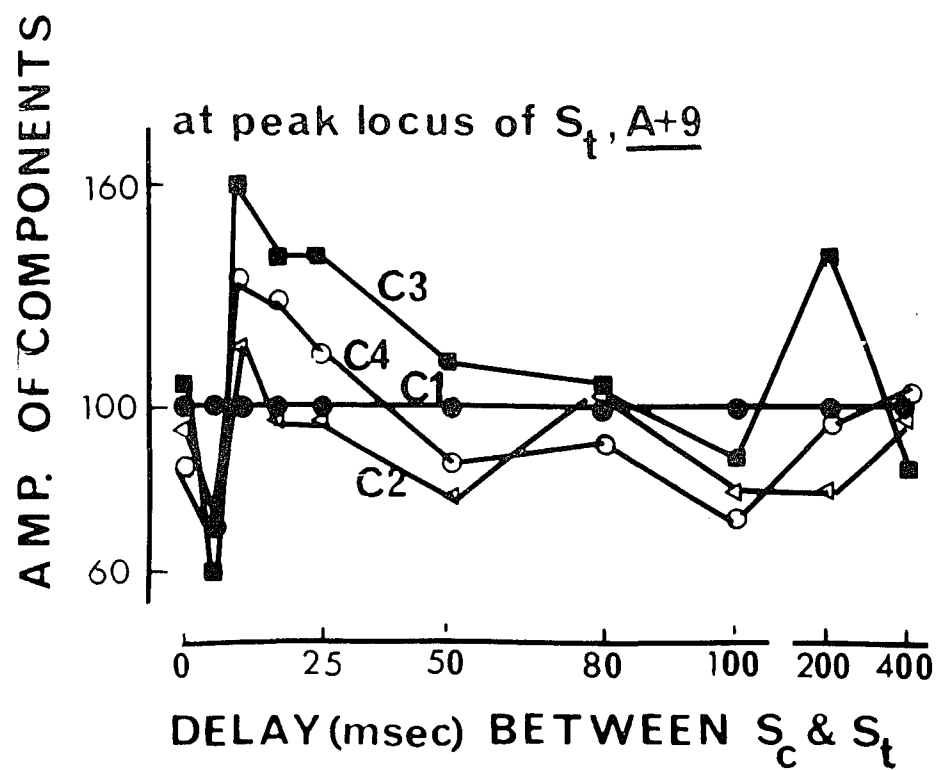
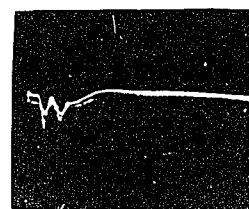
response to
 S_c 3000 μ v



S_t 2700 μ v



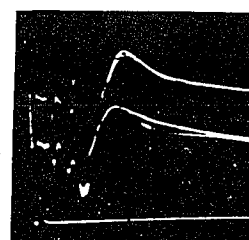
both 500 μ v



S_c 450 μ v



S_t 3000 μ v
both 4000 μ v

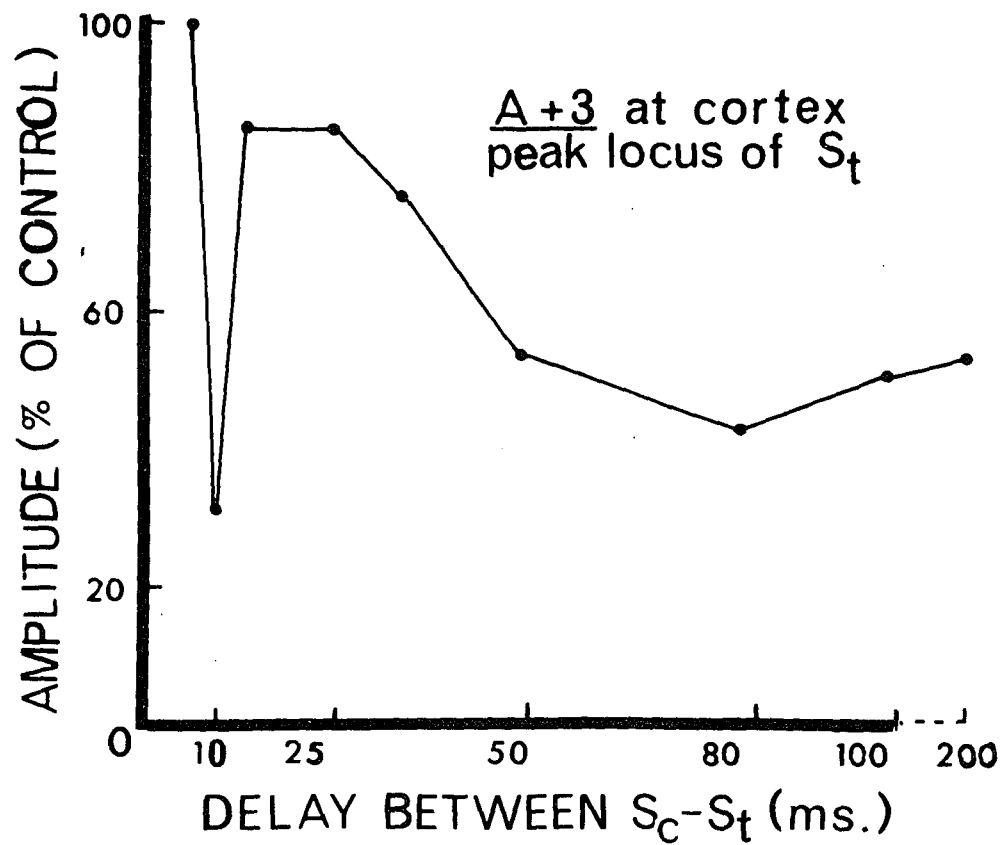


the test shock, then the excitability cycle is enhanced. For this generalization to hold, one should be able to vary the intensity of the conditioning stimulus, and obtain either depression or enhancement of the test response at the same cortical locus, depending upon response amplitudes. Fig. 20 illustrates this result for Cat Q223 in graphical and pictorial form. On the ordinate are the amplitudes of the responses at the cortical locus of A+5, expressed in per cent of the control value. When the response to the conditioning stimulus was smaller than the response to the test stimulus (because the intensity of the conditioning shock was set below the supramaximal level—top graph, the test response was slightly enhanced: for instance at a delay of 16 msec. between the two shocks, the response was about 27% larger than the control value. When the response to the conditioning shock was augmented by increasing shock intensity, the response to the test shock at this same cortical locus (bottom graph) diminished, especially at delays from 25 to 100 msec. The tracings illustrate responses to the test shock when 25 msec. separated the two shocks.

In order to study whether this interactive effect between two cortical potentials was dependent upon the peak response to the first shock, the peak locus of the conditioning response was aspirated. Even when the peak locus of the conditioning shock was depressed in this fashion, thus eliminating the peak response, the smaller potentials

Fig. 19. Excitability cycle for Cat Q181 at A+3 on visual cortex, where response to S_c (A+5 in LGB) was of larger amplitude than response to S_t (A+4 in LGB). On ordinate: response amplitudes expressed as percent of response to S_t alone. On abscissa: temporal delay between two stimuli from 0 to 200 milliseconds. The tracings illustrate responses to S_c alone, to S_t alone, and to both shocks at 50 and 100 msec. delays, with peak-to-peak amplitudes as indicated.

CAT Q181

R
to
 S_C 

2000 uv

100 uv

both
50msR
to
 S_t 

250 uv

125 uv

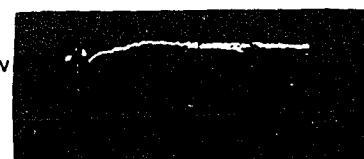
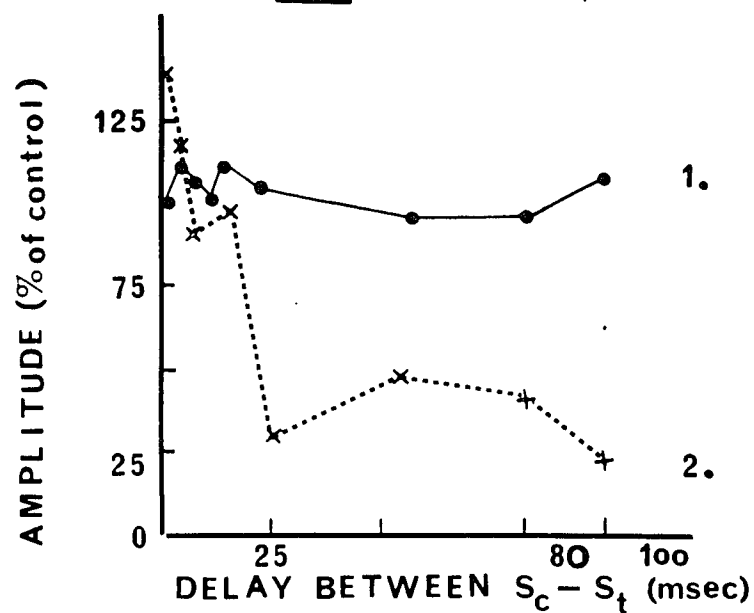
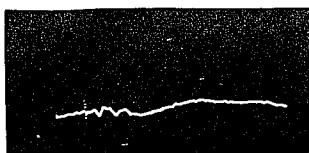
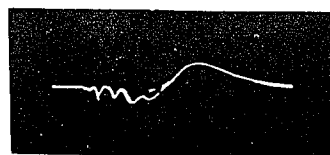
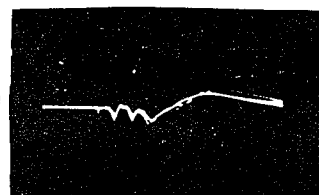
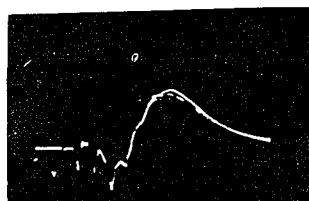
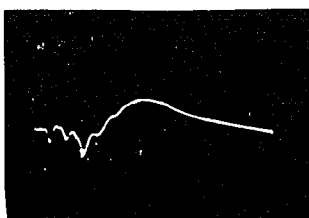
both
100ms

Fig. 20. Excitability cycles for Cat Q223, at A+5 on visual cortex. When intensity of S_c (to A+11 in LGB) was low and its response of small amplitude (top function and tracings to the left), the conditioned response was enhanced. When the intensity of S_c was high and its response amplitude large, the response to S_t was diminished in amplitude (bottom function and tracings to the right).

CAT Q223

at A+5:1. R to $S_c < R$ to S_t R to S_c 2. R to $S_c > R$ to S_t R to S_t 

both, 25msec



500uv
20msec

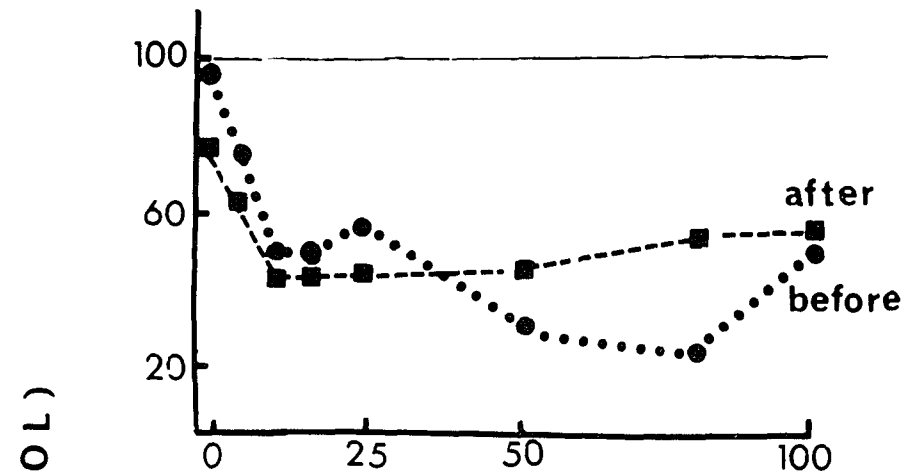


in neighboring cortical regions remained intact (see below), and more importantly, continued to interact with the test response during double stimulation. The data in Fig. 21 (top graph) illustrate the excitability cycle at A-5 on cortex for Cat Q196. The conditioning shock was delivered to A+8 in LGB, the test shock to A+5 in LGB. The solid line represents the excitability cycle before aspiration, and the dashed line represents the excitability cycle after aspiration (of about two cubic millimeters) of the peak locus for the conditioning shock (A+1 on cortex). After aspiration, the excitability cycle is unchanged, indicating that the response to the conditioning shock at A-5 on cortex influenced the response to the test shock, independent of whether or not the peak locus of the conditioning shock was intact. Similarly, the data in the bottom graph of Fig. 21 (For Cat Q223) illustrate that an enhanced excitability cycle also remains essentially unchanged after local aspiration of the peak locus (A+9) of the conditioning shock.

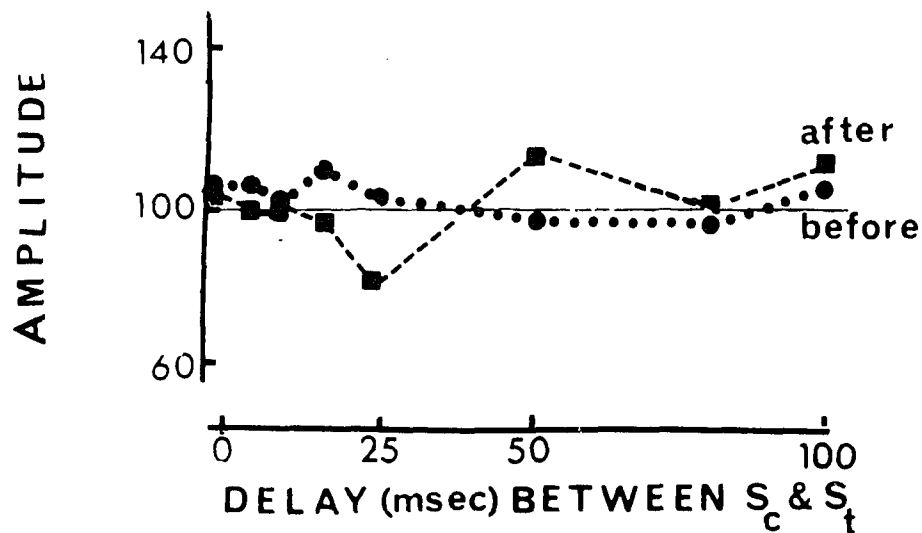
In summary, the foregoing excitability studies revealed several interesting temporal aspects of the functioning visual system. Spatially separate but adjacent geniculo-cortical projections interact over time to alter the amplitudes of evoked potentials at several cortical loci. If a response at one locus is larger than a subsequent response, the second is diminished, often to 15% or 20% of control

Fig. 21. Excitability cycles for Cat Q196 (top graph) and for Cat Q223 (bottom graph). On top: response amplitude (percent of control) to both shocks, at A-5 on visual cortex, both "before" and "after" aspiration of peak cortical locus of S_C at A+1 on cortex. Depressed excitability cycle remains intact. On bottom: response amplitude (percent of control) to both shocks at A+5 on visual cortex, "before" and "after" aspiration of peak cortical locus of S_C at A+9 on cortex. Enhanced excitability cycle remains intact.

CAT Q196 AT A-5 ON CORTEX
LGB SHOCKS A8→A5



CAT Q223 AT A+5 ON CORTEX
LGB SHOCKS A11→A10



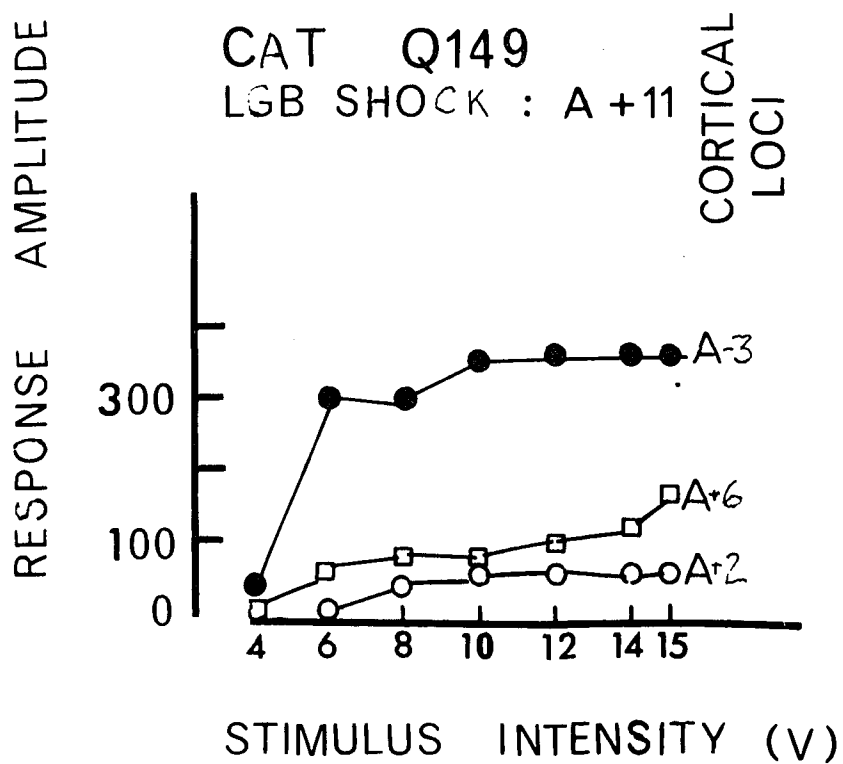
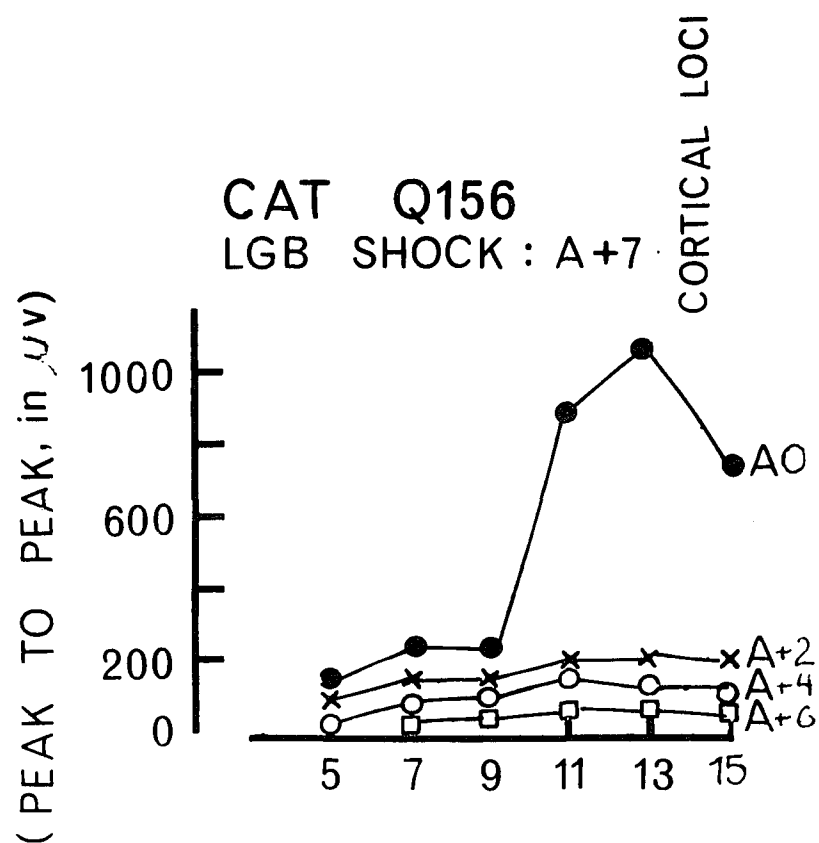
value. The later cortical components, C3 and C4 are especially influenced by the conditioning shock, since their amplitudes are more markedly diminished than are the amplitudes of C1 and C2. When the response to the first shock is smaller than the response to the second, however, a significantly enhanced evoked response occurs to the second shock. The effects of a conditioning shock were shown to be dependent upon the absolute size of its cortical response as varied by changing the intensity of the first shock; and these effects were not dependent upon the presence of the peak response to the conditioning shock at some distant cortical locus (see below for further discussion of this cortical procedure and its significance). The most intriguing result was illustrated in Fig. 17, where the response at a topologically-specific cortical locus was enhanced if it occurred at its own peak locus, but was depressed if it occurred at the peak locus of a spatially different stimulus.

Studies on the Spread of Stimulating Current in the Lateral Geniculate Body

Further generalizations from the results described above depend upon the presumed restriction of the stimulating current (about 0.5 milliampere on the average) to one location in LGB. Therefore two sorts of control experiments were completed to assess the significance of current spread from one geniculate location to another: (a) systematic variation of the stimulus intensity (from sub- to suprathreshold values) and its effect on the cortical distribution of evoked responses; (b) reversal of polarity between the stimulating electrodes to determine if excitation in LGB occurred primarily around the cathode.

The cortical evoked potential is known to change amplitude when shock intensity is varied, but prior studies have looked only at the response of a single cortical locus. For the present control experiment, the strength of the LGB shock was varied to study response changes at the peak locus and, in addition, at other responsive visuo-cortical loci. Fig. 22 illustrates the resultant changes in the cortical distribution for Cats Q156 and 149 (similar trends were seen in eight preparations). Amplitude of the evoked response is graphed as a function of increasing stimulus strength. Only at the peak locus of the stimulating electrode (at AO and at A-3 respectively) did the response grow in amplitude as stimulus strength reached and exceeded the supramaximal intensity. The smaller responses at non-peak

Fig. 22. Response amplitudes at several cortical loci as stimulus intensity increases from threshold to above supra-maximal levels, for Cats Q156 and Q149. Peak-to-peak amplitude (in microvolts) is on ordinate; increasing stimulus strength on abscissa. Peak locus for LGB shock at A+7 in Cat Q156 was at AO; peak locus for LGB shock at A+6 in Cat Q149 was A-3 at cortex.



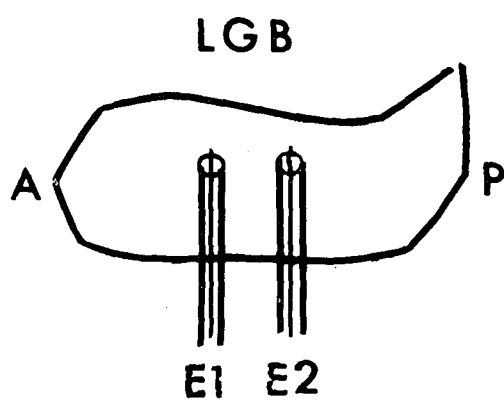
loci did not increase significantly in amplitude. If the stimulating current had spread to adjacent LGB positions, then strong stimuli should have evoked larger responses all across visual cortex, particularly when supramaximal shocks were used. Since this did not occur, the lower level potentials may be attributed to activity in cerebral visual pathways which are already supramaximally excited by the weakest stimulus strength used, or which do not depend on stimulus strength.

For the second control procedure, to investigate if the cathodal current was in fact the selected site of LGB stimulation, the experimenter placed two adjacent concentric electrodes into LGB in the usual manner, electrode one (E1) being about two millimeters anterior to electrode two (E2). The shock current was impressed either between the centers of these two electrodes, or through one of these electrodes with respect to its concentric shield. Fig. 23 for Cat Q221 illustrates the results obtained (typical for three preparations). The four different stimulating conditions were (1) a shock delivered between E1 and E2, E1 as the cathode; (2) a shock delivered between E2 and E1, E2 as the cathode; (3) a shock through E1 alone (tip cathodal to shield; and (4) a shock through E2 alone (tip cathodal to shield).

When the shock was delivered between E1 (cathode) and E2, the distribution of responses peaked at A+9 on visual cortex (distribution number one). When the cathodal site was reversed so that excitation occurred between E2 (cathode) and E1, the distribution then peaked at A0 on

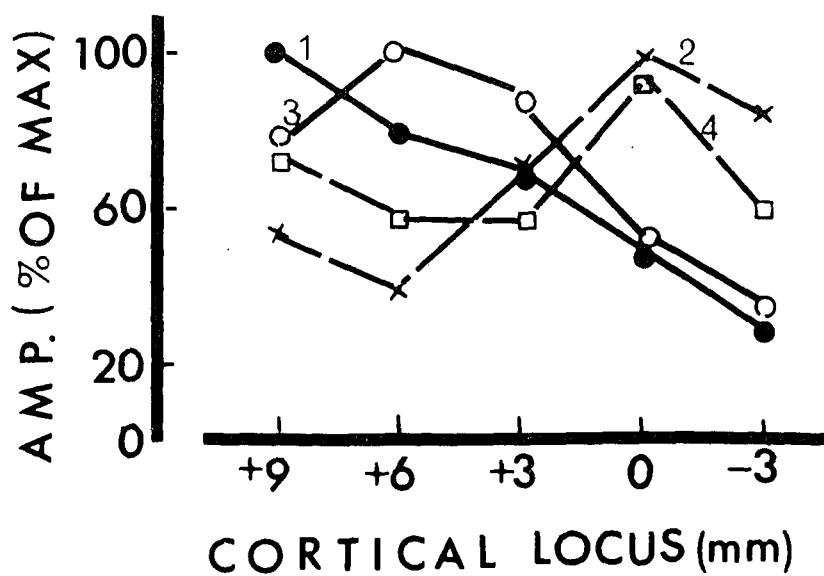
Fig. 23. Effect of reversing polarity of stimulating electrodes in LGB. Four stimulating conditions are outlined to the top right (see text for further explanation). On ordinate of graph: response amplitude (percent of maximum); on abscissa: five cortical loci from A-3 to A+9.

C A T Q 2 2 1



STIM. CONDITION

- | | |
|--------------------|---|
| 1 E1(cathode) - E2 | ● |
| 2 E2(cathode) - E1 | × |
| 3 E1 alone | ○ |
| 4 E2 alone | □ |



cortex (distribution number two). When the shock current was delivered through E1 alone, the distribution of evoked responses closely approximated that of distribution number one. Finally, when the shock current was delivered through E2 alone, the distribution of evoked responses closely approximated distribution number two. These findings demonstrate that the site of the cathodal current was the actual site of LGB activation, and that the stimulating electrodes provided a relatively restricted source of stimulation. These results, in combination with the data obtained from the manipulation of stimulus intensity, were sufficient to conclude that the shock was relatively delimited to any one selected LGB position.

Studies on Possible Mechanisms Underlying the Diffuse Response at Cortex

Since the above results showed that the LGB shock was relatively restricted, the most interesting finding to be explained was the widespread cortical responsivity to a single stimulus. A number of possible mechanisms can be advanced. The diffuse cortical response may depend upon some sort of branching within the geniculate itself, in the projection pathways to cortex, in the cortex, or any combination of all three mechanisms. In the studies described below, attempts were made to isolate one or another of these mechanisms as the source of the widespread distribution of cortical responses.

The first series of experiments involved a careful analysis of latencies of the evoked responses across all cortical loci, in eight preparations. A single LGB shock evoked responses which were recorded with varying latencies across several cortical loci. Although the fast-occurring early components of the response, C1 and C2, seemed to exhibit equal latencies at all loci, clear latency differences were observed in the later components, C3 and C4. To measure latency changes in the later part of the waveform most accurately, response latency was derived from the delay (in milliseconds) between C1 and C3, or between C1 and C4. Fig. 24 illustrates response latencies for three preparations (Cats Q166, 161, and 171). The delay between C1 and C3 of the response to one LGB shock is

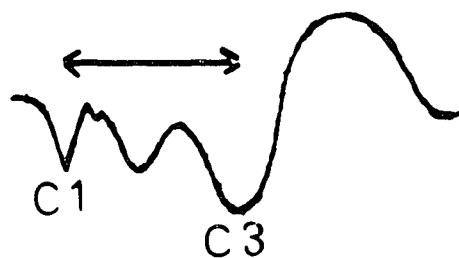
plotted across various cortical loci. For example, stimulating LGB at A+6 in Cat Q166 evoked its largest amplitude response at A+3 on cortex. This response also showed the shortest latency (delay) between C1 and C3. Other responses at surrounding cortical loci showed systematically longer latencies: the further the locus from the peak locus, the longer the latency of the later components. Cats Q161 and 171 show the same trends.

Fig. 25 again illustrates, in graphical and pictorial form, latency changes for Cat Q190, derived from three measures. The peak locus for stimulating LGB at A+7 was A+6 at cortex. The three measures of latency graphed are: (1) delays between C1 and C3, which show only a slight trend toward longer delays at the non-peak loci; (2) delays between C1 and C4, which are better indicators of latency change in this preparation than (1) since the non-peak loci clearly show longer latencies; and (3) the latency of C4 (i.e. the interval between the shock and the peak of the negative deflection) which matches well the previous plot of delay between C1 and C4, indicating that the latency of C1 was relatively constant at all loci.

Components C1 and C2 did not exhibit latency changes across various cortical loci, and might be considered, therefore, as reflecting simultaneous activation of multiple-geniculo-cortical pathways. Fiber branching in the LGB or in cortex itself would require a synaptic delay of at least 0.3 or 0.5 millisecond. If the source of the

Fig. 24. Latency of evoked responses across several cortical loci, to a single LGB shock, in three cats, Q171, Q161, and Q166. Latency derived from the delay between C1 and C3 (as illustrated) is graphed on ordinate (in msec.). The arrows on abscissa indicate peak loci for the three cats, at A+9.5, A+6, and A+1 respectively. See next figure for illustrative tracings.

LATENCY



DELAY (msec) BETWEEN C1 & C3

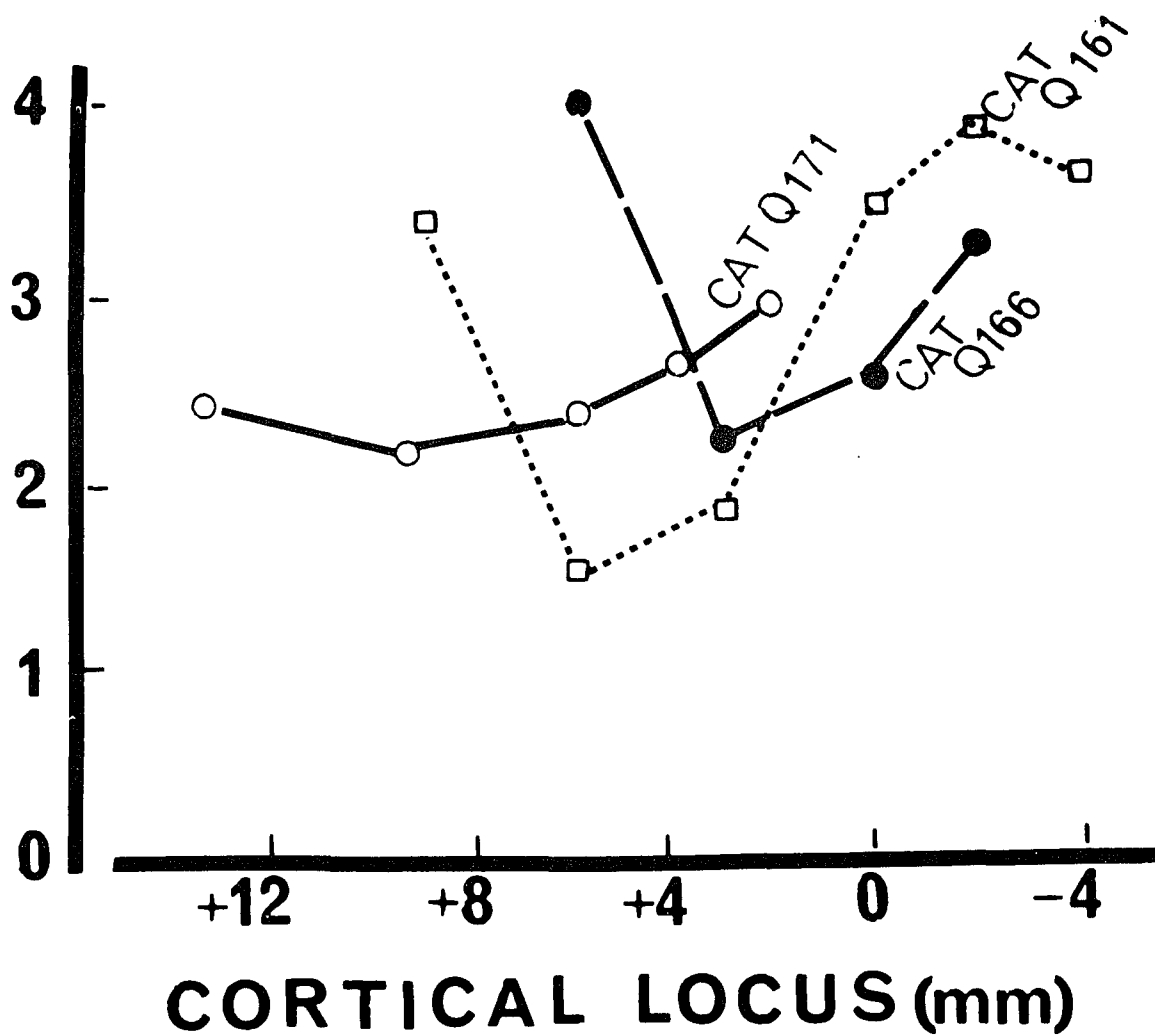


Fig. 25. Latency of evoked response across five cortical loci to LGB shock at A+7 in Cat Q190. Delay between stimulus and C4, between C1 and C3, and between C1 and C4 (in msec.) is on ordinate. Peak locus was A+6 at visual cortex. Tracings illustrate peak response and smaller amplitude and longer latency responses at A+3 and at AO.

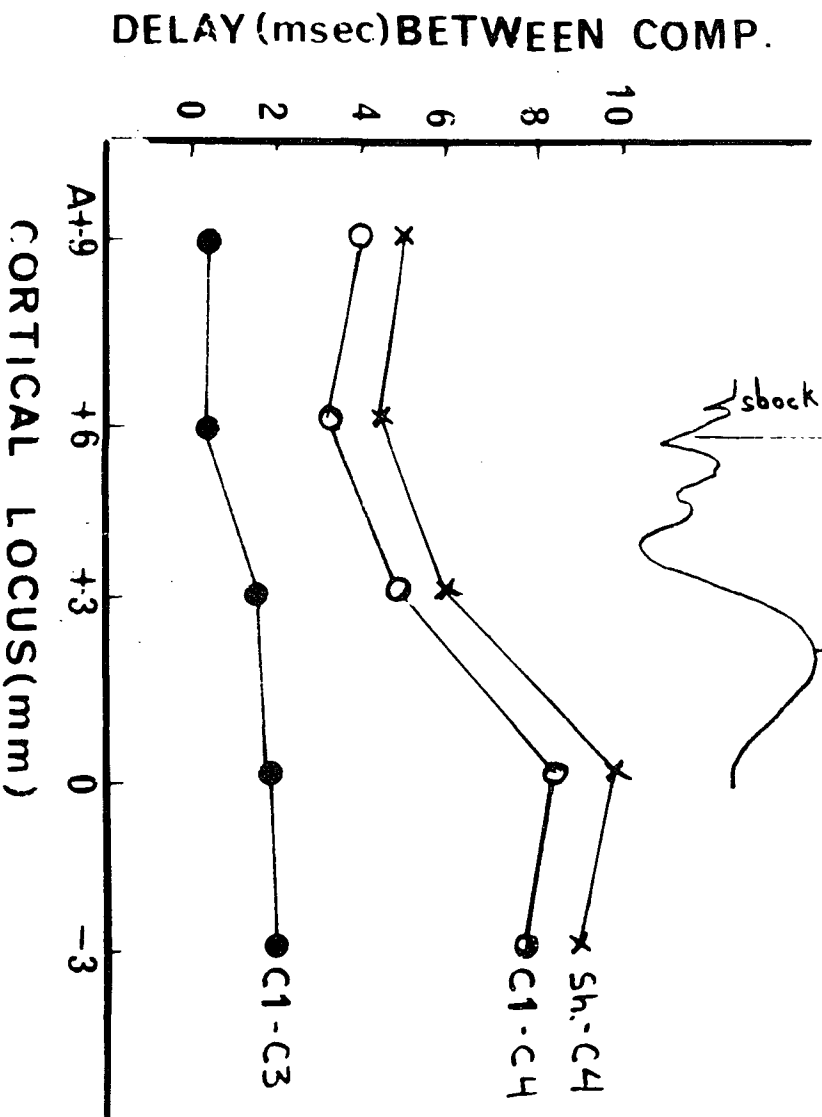
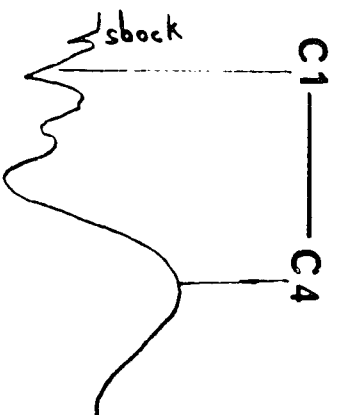
CAT Q190

R at A+6A+3A+0

5 msec

6 msec

11 msec



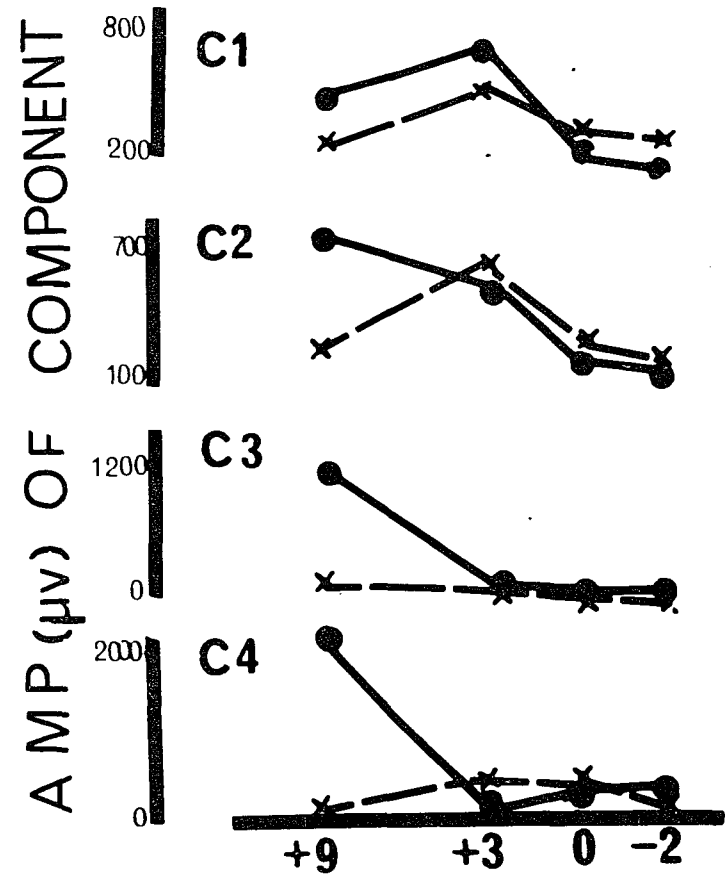
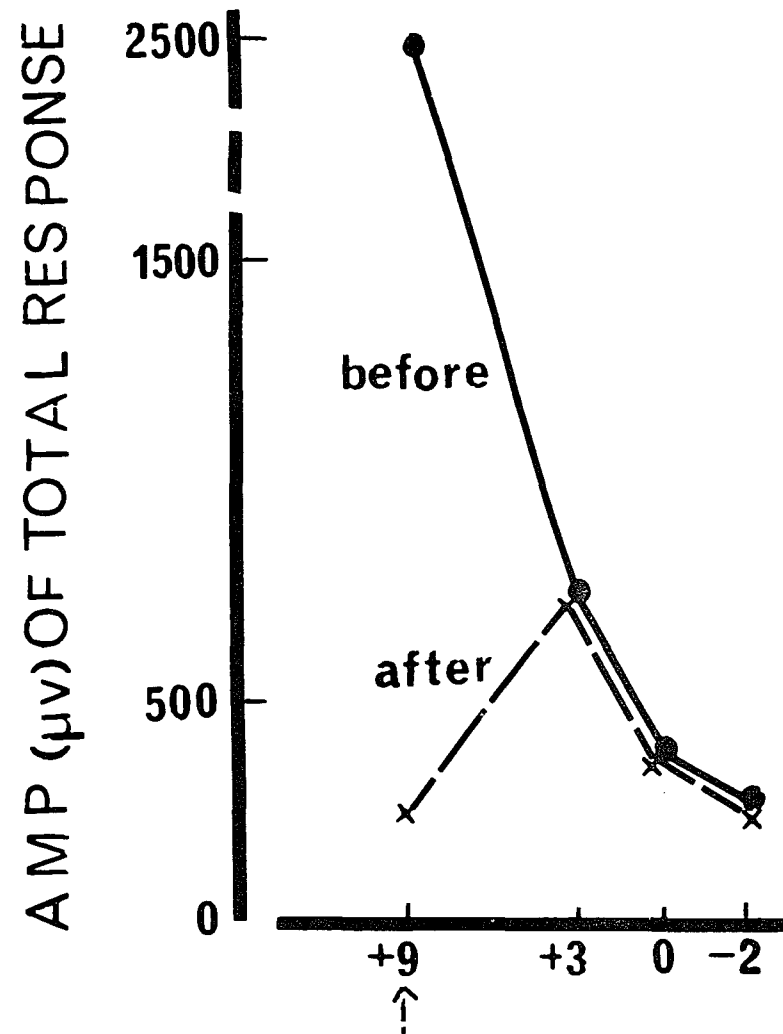
widespread presence of C1 and C2 across loci were along branching of radiation fibers, however, then the radiation activity could presumably arrive at multiple cortical loci with equal latencies. The possibility remains that the early components are distributed across cortex electrotonically from the peak locus. It should be noted that this explanation could not account for the diffusivity of C3 and C4, which both exhibit systematic latency changes across cortical loci. The magnitude of latency differences reaches six milliseconds in Cat Q190, time enough for multiple synaptic transmission in LGB, in cortex, or at both cerebral levels, and also for slow dendritic processes to transmit this portion of the response intracortically.

In order to evaluate the significance of intracortical mechanisms, another group of procedures was used. The peak cortical locus for one LGB shock was either depressed with topical KCl, or aspirated by subpial suction, or cooled to induce hypothermic depression, and then the potentials at surrounding loci were charted to see if they changed in amplitude. Fig. 26 for Cat Q166 illustrates data obtained before and after the activity of the peak locus was depressed by circumscribed topical application of KCl to the pial surface. Prior to the manipulation of cortical excitability by drug application, the LGB shock at A+8 evoked its peak response at A+9 on cortex, but also evoked smaller potentials posteriorly to A-2. After local depression of about three millimeters around A+9, the

.....*

Fig. 26. Response amplitudes at peak locus (A+9) and at three other loci when LGB shock at A+8, for Cat Q166. To left: amplitude of total peak-to-peak response (in microvolts) before and after depression of A+9 with KCl. To right: amplitude of each component before and after depression of peak locus.

CAT Q166



CORTICAL LOCUS (mm)

evoked response at A+9 was functionally depressed, but amplitudes of responses at the other loci remained the same as before. This "before" and "after" comparison was made for each component, to the right of Fig. 26. Components at non-peak loci were not markedly altered by depression of the components at A+9.

In other preparations, the peak cortical locus was aspirated down to the underlying white matter. After removing A+9 for instance in Cat Q179, the responses at distant non-peak loci (at A+3, AO, and A-2) remained as before. Fig. 27 shows the tracings at A+9 and at AO. In two preparations, the size of the aspirated area was enlarged further surgically (by suction) to include roughly five to eight millimeters of cortex in the A-P dimension. Under this condition, it was noted that the responses at remaining cortical loci tended to be somewhat depressed, especially their later components.

Local freezing of the peak locus in Cat Q175 yielded comparable data (see Fig. 28 and also Fig 7). The amplitude of the evoked response at the peak locus (at A+6) to LGB shock at A+8 was depressed, whereas the amplitudes of responses at distant loci were relatively unchanged.

In summary, the procedures which depressed or removed the peak locus for the shock-evoked response demonstrated that the smaller potentials at non-peak loci, especially the first two components of the response, do not depend solely upon intracortical processes. Since the

Fig. 27. Amplitude of total response (in microvolts) to LGB shock at A+8, for Cat Q179, before and after aspiration of peak cortical locus at A+9.

CAT Q179
LGB SHOCK: A+7

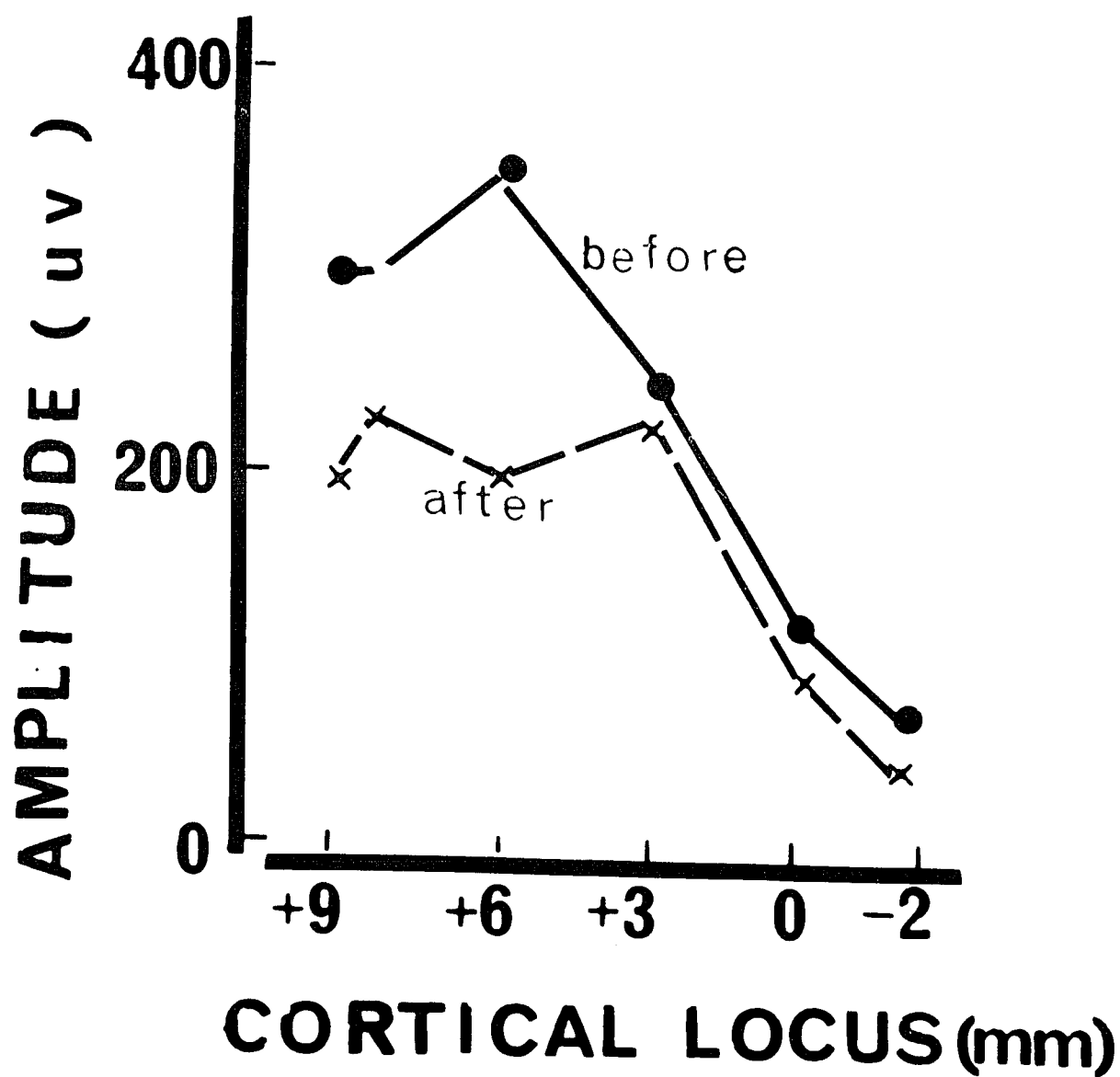
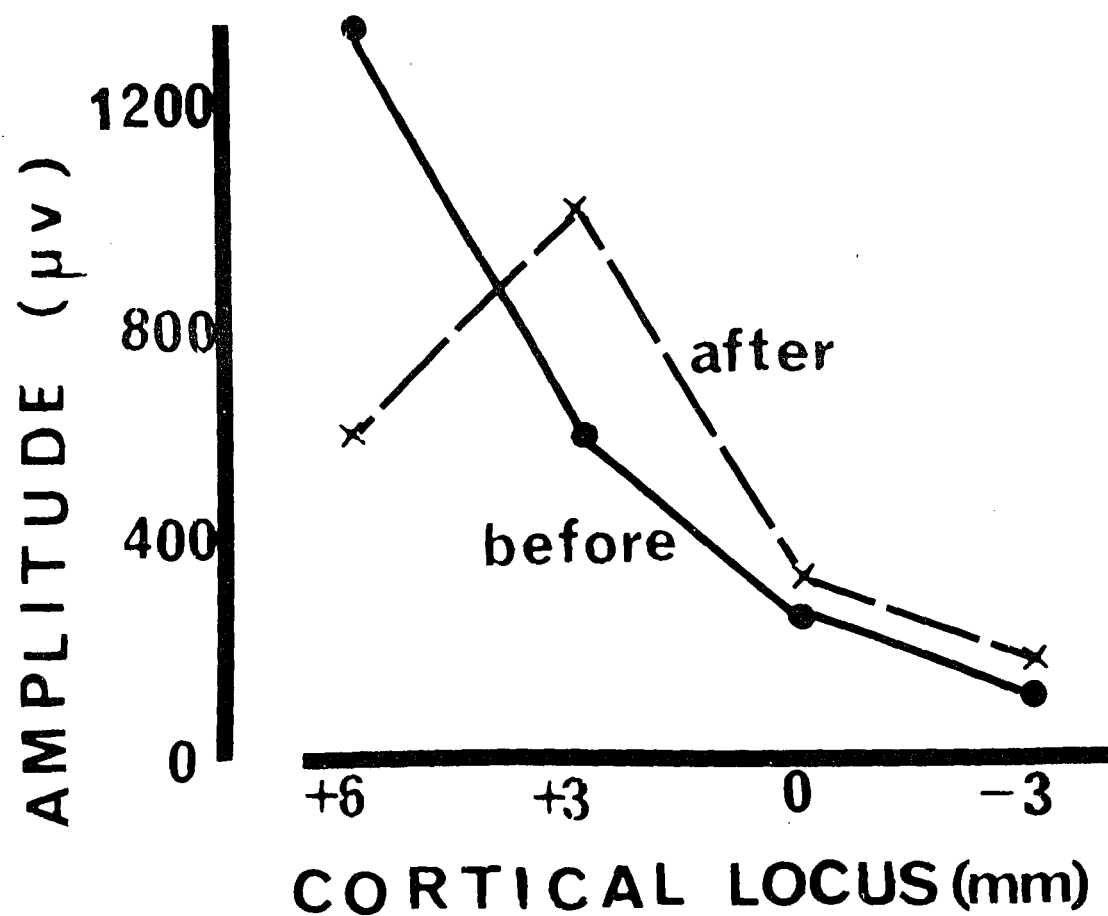


Fig. 28. Amplitude of total response (in microvolts) to LGB shock at A+8, for Cat Q175, before and after depression of peak cortical locus (at A+6) with chip of dry ice to induce hypothermia.

CAT Q175
LGB SHOCK: A +8



peak locus was removed and the non-peak responses remained intact, the diffuse cortical response to a single LGB shock may depend upon the integrity of subcortical processes: intralaminar fibers in the LGB, or branching of radiation fibers. It was noted, however, that non-peak responses did diminish in amplitude if a larger area of cortex was aspirated, indicating that some intracortical process probably contributes to the diffuse distribution of evoked responses. The studies of latency changes described in this section suggested that the earlier components might reflect branching in radiation fibers, but that the later components might indicate intra-geniculate or intracortical processes.

DISCUSSION

The results of the present research indicate that temporal as well as spatial processes must be considered when evaluating geniculo-cortical projection in the cat. The spatial characteristics of this physiological system were consistent with known anatomy of the cat's visual pathways as long as one considered only the cortical location of the maximum amplitude response; i.e. the peak locus. It is significant, however, that the early components of the shock-evoked response (the "radiation" components) displayed a wider distribution on cortex than the later ("intracortical") components. In any event, a single LGB shock produced low amplitude potentials over a wide area of cortex, a finding which the control studies suggest may be attributable to branching of afferents in the radiation fibers, or in the cortex itself.

The temporal sequence of geniculate stimuli was shown to be functionally significant for electrophysiologically recorded cortical activity, since the widespread low amplitude potentials described above for a single shock affected the cortical response to a spatially different LGB shock, as a function of the temporal delay between the two stimuli. Further studies of the excitability phenomena showed that the form of the recovery function depended upon the relative amplitudes of the cortical responses to the two shocks.

In brief, the major and unique findings in the

present study were that the geniculo-cortical topology of the cat visual system has diffuse or widespread aspects in addition to the point-for-point peak response, and that the topologic system is functionally dependent upon the temporal sequence of incoming geniculate stimuli.

The topologic arrangement of the cat's geniculo-cortical system was determined electrophysiologically: shocks which excited anterior LGB positions evoked cortical potentials which peaked at anterior visual loci; posterior LGB shocks evoked potentials which peaked at posterior loci. As stimulation in LGB moved medially, the cortical laterality of the maximum amplitude response usually remained constant at about three to five millimeters from the cortical midline. However, in some instances, the locus of the maximum cortical response did move medially when the shock excited medial LGB sites. Since these medial-lateral results are variable, only the data on anterior-posterior topology are discussed in detail below.

The topologic relationship found between LGB and visual cortex was summarized in Fig. 16. Using the data reported in prior electrophysiological studies of retino-cortical organization (Bilge et al, 1967; Talbot and Marshall, 1941), one can estimate roughly that one degree of the cat's visual field projects to from 0.3 to 0.6 millimeter at visual cortex. Similarly, according to the prior electrophysiological study by P.O. Bishop et al. (1962), one degree of the field activates about 0.1

millimeter in LGB. Consequently, one can predict that one millimeter in LGB will project to from three to six millimeters at visual cortex. The present study indicates that any one millimeter of LGB sends fibers to activate at least four millimeters of cat visual cortex, and suggests (see Fig. 16) that LGB regions subserving peripheral and central field positions project to quantitatively similar cortical areas.

The earlier components of the shock-evoked cortical response were widespread across many cortical loci, and one possible neural mechanism subserving this diffusivity of C1 and C2 is that the visual radiations themselves branch en route to cortex and activate widespread regions. The radiation fibers are buried beneath the cortical mantle and since they project to many cerebral regions from LGB it is difficult to study their route to cortex by direct anatomical procedures. The present electrophysiological study examined the potentials across visual cortex and it was shown that the latency of C1 and C2 was the same value at all loci responsive to one LGB shock. This finding suggests that one LGB position simultaneously activates a widespread area of visual cortex, and the simultaneity of C1 and C2 indicates that no graded or synaptic process is necessary to activate all cortical loci. Multiple radiation fibers, on the other hand, which branch from a single LGB site could activate several cortical loci at once since no synapse or dendritic process is involved.

Further evidence in support of some branching within the visual radiations was derived from the studies which varied stimulus strength. Since increasing stimulus intensity did not increase response amplitudes at non-peak loci, one can postulate that the branching which may underly the diffusity of C1 and C2 is within fibers which reach (a low) threshold simultaneously and have nearly equivalent thresholds. Radiation fibers, branching from one geniculate site, could activate the non-peak loci in this way. In addition, if an electrotonic potential from LGB was being picked up at widespread cortical loci and was responsible for the non-peak responses, or if there was branching within the geniculate or within cortex itself, then the stronger stimuli should have increased the excitation area in LGB and the amplitudes of the non-peak responses should have increased proportionately. This did not occur; therefore the diffusity of C1 and C2 is probably due to branching of radiations which activate multiple cortical loci when a single shock is delivered to LGB. Finally, since the shock was shown to be relatively restricted at a single LGB site, the widespread distribution of C1 and C2 could not be attributed to the activation of many LGB positions.

The literature on the origins of C1 and C2 of this shock-evoked response reports disparate results for C2, although all investigators agree that C1 reflects the response of the visual radiation. Component 2 has been variously described as radiation activity, as presynaptic

cortical activity, and as a postsynaptic cortical response (as described in the results, section one). The present study showed that C2 and C1 presented at least four similar functional characteristics: (1) both components remained in the response recorded after cortical depression; (2) their thresholds were similar; (3) their distributions across cortical loci were similar in extent; and (4) their amplitudes were similarly affected by conditioning shocks (to be discussed below). Therefore, C2 was judged to represent activity in the radiation, or to be early presynaptic intracortical activity. These four observations reinforce the conclusion that the diffusivity of C1 and C2 across visual cortex depends upon branching in the radiation fibers.

The later components of the shock-evoked response, C3 and C4, are generally agreed to represent cortical activity, although the exact neural process responsible for their presence is not yet known. Component C3 and C4 may represent activity in dendritic processes (in the apical dendrites of the pyramidal cells of cortex), the summated firing of groups of cortical units, the synaptic activation of adjacent or distant cortical regions via intracortical or subcortical fiber processes, or may be corticofugal excitation of subcortical cerebral systems. In any event, the present study showed that C3 and C4, in response to one LGB shock, were of maximal size and minimal latency at a peak cortical locus, but that they were also present across widespread cortical regions. Since C3 and

C4 at non-peak loci showed significantly longer latencies than the latency at the peak locus, their diffuseness across visual cortex can only be explained by postulating that C3 and C4 are propagated dendritically or synaptically within visual cortex from one cortical area to another, and require roughly two to ten milliseconds to reach cortical regions distant from the peak locus. Components C3 and C4 continued to appear at non-peak loci and to affect the amplitudes of later-occurring responses, even when their peak locus was functionally depressed or completely aspirated, although they were occasionally of smaller amplitude than in the normal or unmanipulated cortex. This might indicate that the propagation of C3 and C4 from the peak locus to a distant one occurs via subcortical processes. However, when a large cortical region encompassing three to four loci (i.e. six to eight millimeters of cortex including the peak locus) was aspirated, C3 and C4 diminished in amplitude at the cortical loci remaining intact. Therefore, the diffuseness of C3 and C4 was shown to be dependent upon intracortical activity emanating from the peak locus, as well as being partially due to subcortical fiber branching which activated widespread regions of visual cortex.

Temporal Aspects of Geniculo-Cortical Projection

More than a strictly topologic projection to cortex must be assumed in order to explain all the present find-

ings, particularly those dealing with excitability effects. It appears that any geniculate stimulus activates a widespread area of visual cortex, and that the activation is focused at a maximally responsive zone (the peak locus) which conforms to the topologic arrangement between LGB and cortex. Smaller potentials are generated at non-peak loci which reflect intracortical and/or radiation fiber activity, and are functionally significant to the spatial characteristics of the cat's visual pathways. The small potentials at non-peak loci were shown to alter the amplitude of later-occurring responses to a second spatial stimulus, and the direction of this effect was predictable based on the relative amplitudes of the responses to the two shocks.

When the excitability cycle of the test response was studied at the peak locus for the test shock, the response was enhanced by a prior conditioning shock delivered to a different site in LGB. This enhancement effect was shown to depend upon the amplitudes of the conditioning response and the test responses at any given cortical locus. In the above instance, when the response to the conditioning shock was of smaller absolute amplitude than the response to the test shock alone, the second response was enhanced. Conversely, if the excitability cycle was studied at the peak locus for the conditioning shock (where the response to the conditioning shock was usually of larger absolute amplitude than the response to the test shock), a diminished response resulted after dual stimulation. Thus the topologic

response at any given cortical locus was shown to be markedly altered by prior stimulation of another LGB site, and the effect was such that it "sharpened" the response at its own peak locus, and "depressed" the response at its non-peak loci. This sort of "lateral interaction" in mammalian visual cortex has not (to the author's knowledge) previously been demonstrated using such a technique.

The temporal characteristics of the individual components of the shock-evoked response reveal further instances of interactive effects between the radiation pathways and the cortical regions activated by two shocks. Components C1 and C2 of the first response served to enhance or depress the amplitudes of the early components of a second response at the same cortical locus, depending upon the relative amplitudes of the responses as described above. Since the early components of one response influenced the amplitudes of the same components in a second response, even after delays of from 50 to 100 milliseconds, the radiation activity must initiate long-lasting (intracortical?) effects which serve to alter a later-occurring

response. Interestingly, C3 and C4 were even more influential than the earlier components in altering a second response at any given cortical locus. As described above, if C3 and C4 were of smaller or larger amplitude to one shock in LGB, they acted to enhance or depress, respectively, the amplitudes of the responses to the second spatially-different shock in LGB. The magnitude of the excitability effect was much greater for the later components, i.e. C3 and C4 were relatively more enhanced (or depressed) than were C1 and C2, indicating that the intracortical response is more sensitive to prior stimuli delivered to different spatial sites in the visual system. Therefore, the temporal characteristics of the intracortical components may exert a stronger influence than the radiation components over the spatial characteristics of the geniculo-cortical projection.

Implications for Visual Behavior

These several spatial and temporal characteristics of the cerebral visual system are directly relevant for understanding the neurophysiological mechanisms underlying visual localization of objects, the ability to resolve spatial differences (visual acuity), and the system's capacity for handling temporal sequences of visual stimulation.

Although the observer probably utilizes several cerebral systems to localize an object in his visual field, certainly the assumed point-for-point retinotopic system

is a necessary visual mechanism. The present study demonstrated that the peak response at cortex following geniculate shock coincides with what the retinotopic theory predicts, but that lower level potentials across many cortical loci are indicative of a diffuseness within the sensory pathways. Aside from the possibility that the non-peak loci may maintain the cerebral pattern of activity necessary to localize the visual stimulus if the peak locus is made inactive or damaged (Lashley's theory of "equipotentiality"), the responses at non-peak loci have been shown to affect the normal topologic response of the intact and alert system in three important ways:

(1) The cortical distributions of the early and late components to a single LGB shock are dissimilar, since the distributions of the radiation response (C1 and C2) are more widespread across cortical loci than are the distributions of the cortical components (C3 and C4). The latter response of visual cortex itself appears to "sharpen" or restrict the cortical distribution, and may be one mechanism underlying a sharp sensation of the spatial locus of a visual stimulus.

(2) It was demonstrated that two cortical potentials evoked by two spatially-different stimuli, at one cortical locus, interact, whether they occur nearly simultaneously or whether several milliseconds separate their occurrence. Furthermore, the interactive effect was such that the larger response (usually the topologic response specific to that cortical locus) is enhanced, while the smaller re-

sponse (whose corresponding peak response occurs at a distant locus) is diminished in amplitude. This cortical mechanism, which was shown for all components of the shock-evoked response, but was especially marked for the cortical response (C3 and C4), could presumably subserve both the spatial localization of a single stimulus, and the spatial discrimination between two or more such stimuli.

(3) Accurate spatial resolution between two points in space may also depend upon the two cortical gradients of responses evoked by the two stimuli, as Marshall and Talbot had postulated (1941, 1942) from their data on responses to single flash stimuli. For example, cortical activity underlying visual acuity may depend upon the distance between the two peaks of the two cortical distributions, and upon the relative amplitudes of the low level responses in one distribution as compared to the low level responses in the second distribution. Since it was shown that two responses at any one locus interact to modify the second response, this sort of interaction at all loci of visual cortex may be necessary for the precise discrimination between two closely spaced points in the visual field.

The cortical gradients described above in response to two stimuli can be conceived as the physiological response subserving perceptual "contrast," in the following way: the perceptual report of simultaneous contrast is that a light (or white) visual area immediately bordering upon a darker (or black) area seems even lighter than it actu-

ally is. If the cortical gradients evoked by the lighter and by the darker areas overlap so as to effect an interaction between them, then the amount of overlap and the relative amplitudes of the responses in that cortical area could result in an apparent accentuation of the contrast. Perception of contrast between two temporally-separate stimuli could be subserved by the same cortical mechanism, depending upon the relative response amplitudes to the two stimuli, as described above in the studies of cortical excitability.

The excitability studies, in addition to showing some temporal influences over activity in the topologic system, may be viewed as studies of cerebral mechanisms underlying the visual system's temporal resolution. Specifically, the cortical response, when diminished by prior activity in the visual projection, was shown to require at least 100 to 200 milliseconds to recover normal amplitude. Presumably, the visuocortical system of the cat is not maximally responsive to a second stimulus until 200 milliseconds have elapsed, although a submaximal response can be evoked, as well as responses to flickering light. Conversely, if the first of two stimuli is the weaker of the pair, then the visuocortical response to the second stimulus is enhanced for varying delays, sometimes as long as 100 to 200 milliseconds. The enhanced response here is due solely to dual stimulation of the specific relay nucleus (the LGB) without any direct stimulation of so-called diffuse or activating cerebral

systems. Consequently, the sensory system alone has the capacity to enhance the amplitude of the specific cortical response, and to respond to various temporal intervals of stimulation; it was also shown to display two opposite response cycles, depending upon the relative intensities of the two stimuli.

In conclusion, the electrophysiological studies described above have demonstrated some visuocortical response patterns evoked by direct stimulation of several spatial sites in the LGB. Previous reports have described anatomical, clinico-pathological, or electrophysiological procedures used to trace the visual projection from retina to cerebrum. The present study has investigated the topologic relationship between LGB and visual cortex by direct stimulation of LGB, and has studied cortical responsivity in the non-barbiturized preparation. The present approach also provided a means of studying temporal aspects of geniculo-cortical function. Specifically, excitability cycles of shock-evoked cortical responses were determined as a function of the interval separating two spatially-different stimuli. These latter studies demonstrated that the cortical response could be enhanced or diminished in amplitude for long-lasting intervals, depending upon the strength and spatial position of the first stimulus of the pair. In general, the results of all experiments showed that the "peak" cortical response to geniculate

shock supports a point-for-point relationship between LGB and visual cortex, but that one shock evokes additional responses across widespread cortical areas, due to activation of multiple pathways in addition to the peak projection. Furthermore, these lower level diffuse cortical responses alter the topologic system so as to enhance or to reduce the peak amplitude, and, presumably, direct subjective localization and resolution of objects in the visual field.

SUMMARY

The functional topology of the cat's geniculocortical system was studied by delivering discrete shocks to different spatial sites in the lateral geniculate body, and then recording the resultant distribution of responses across visual cortex, to each shock. An orderly anterior-posterior projection system was found if one used the peak response to map geniculocortical pathways. Each stimulus, however, evoked smaller amplitude potentials at numerous cortical loci, in addition to the maximum amplitude response at the peak locus, and the distribution of the afferent activity was more widespread at cortex than the distribution of the intracortical activity.

Since the stimulus was shown to be restricted to a single geniculate site, the widespread cortical responses were judged to be dependent upon activity in radiation collaterals and intracortical fiber processes, which served to activate large regions of the nonbarbiturized visual cortex. Most importantly, the small potentials exerted a long-lasting influence over subsequent cortical activity in response to spatially-different stimuli. These spatial and temporal characteristics of the cat's cerebral visual system were discussed in terms of their implications for spatial localization of visual objects, and for visual resolution between objects.

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