THE ROLE OF CANINE VISUAL CORTEX IN VISUAL FEEDBACK FOR POSTURAL CONTROL

By

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF FREQUENTLY USED ABBREVIATIONS

CNS	- central nervous system.
LGN_d	- dorsal lateral geniculate nucleus.
LGN_d(lam)	- laminar portion of LGN_d.
LS	- lateral suprasylvian cortex (equivalent to Clare-Bishop area)
NP	- normal platform experiment, involving platform perturbation during normal vision.
BP	- blindfolded platform experiment, involving platform perturbation during blindfolding.
COP	- central-only, platform experiment, involving platform perturbation during restriction to central visual field.
POP	- peripheral-only, platform experiment, involving platform perturbation during restriction to peripheral visual field.
NB	- normal both experiment, involving simultaneous platform and roof perturbation during normal vision.
POB	- peripheral-only, both experiment, involving simultaneous platform and roof perturbation during restriction to peripheral visual field.
BB	- blindfolded both experiment, involving simultaneous platform and roof perturbation during blindfolding.
COB'	- central-only, both experiment, involving simultaneous perturbation of platform and roof with an attached front panel, during restriction to central visual field.

INTRODUCTION

The Postural Control System

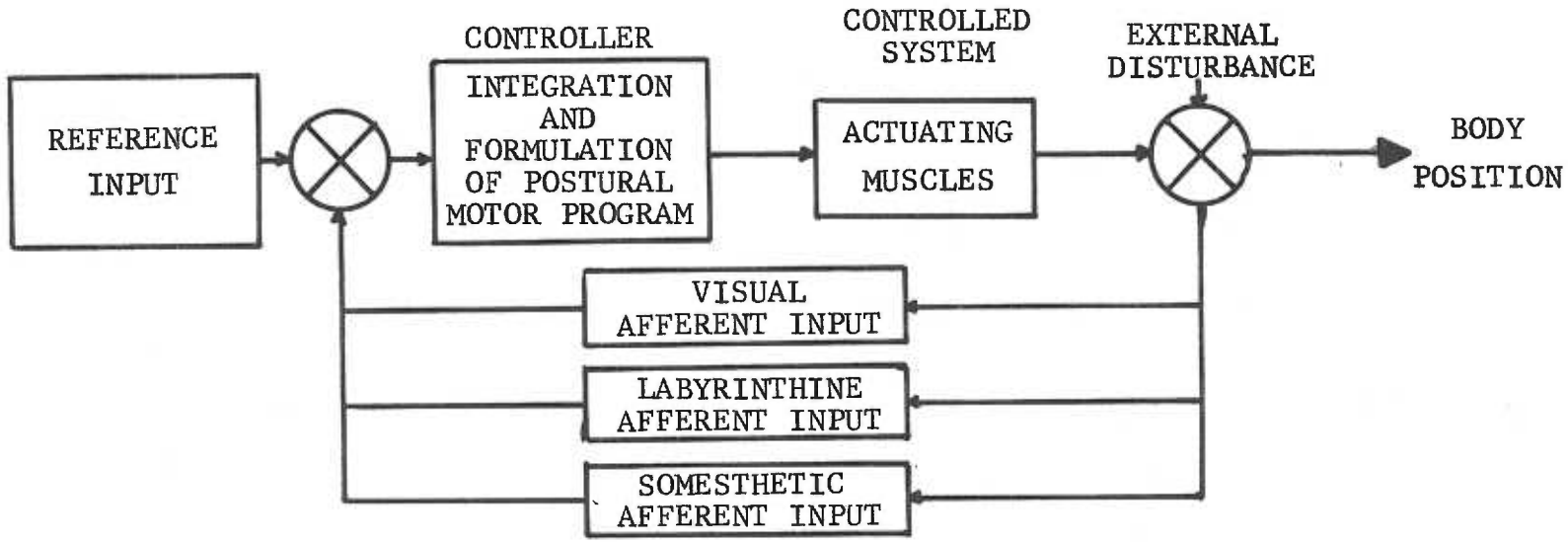
The intricate motor patterns which humans and animals generate daily are accompanied normally by the maintenance of balance. This control of a stable posture is an operation that accompanies all motor behavior and is intimately incorporated into the motor orders continuously being formulated by the central nervous system (CNS). Postural control can also operate more-or-less independently such as during quiet standing; the CNS motor orders then deal predominantly with postural musculature. As reflected in the precision and reproducibility of the maintenance of balance, a part of the postural control system can be considered stable and apparently "hard-wired" (18). The above identification of two operational modes of this control system, i.e. as part of ongoing motor behavior and as a separate defined goal, is an indication of the modulation capability that higher nervous system function has acquired over such hard-wired, feedback dependent control systems.

The postural control system includes the afferents, efferents and central nervous system components that are required to maintain normal posture. Appropriate sensory input includes labyrinthine -- both macular and ampullar types; proprioceptive -- from muscles, tendons and joints; exteroceptive -- from cutaneous nerve endings of the body surface; and, teleceptive -- mainly visual, but including auditory and olfactory inputs (60). Motor output follows common spinal and peripheral pathways that transmit cortical and subcortical activity cortico-

fugally. The CNS activity referred to as "integration" remains largely a mystery with regard to specific location and mechanism. A conceptual model of this postural control system is illustrated in Fig. 1 (75, 109). The modulating influence of CNS higher centers provides the "reference input" to the control system. During motor behavior, this reference input is apparently hard-wired to ongoing CNS motor output, whereas in quiet standing, it defines a stable and repeatable goal stance (18). The reference input is compared with appropriate afferent information to direct the integration of error signals from the various sensory modalities into the formulation of proper neural orders to appropriate postural muscles. The CNS mechanisms that integrate these error signals and formulate appropriate postural orders will be referred to as the controller. The postural musculature and musculoskeletal properties will be called the controlled system. Three major sensory modalities (labyrinthine, somesthetic and visual) are shown feeding back information concerning the changes in body position.

The feedback of visual information has been shown to be an important input for postural control (54). The necessary role of vision is demonstrated clinically by the labyrinthless patient's total loss of body orientation due to the impairment of visual feedback that accompanies immersion in water (60). Experimentally in humans, it has been shown that removal of vision increases spontaneous postural oscillations by up to 50% (27). In addition, subjects presented with tilted, stationary visual scenes demonstrate changes in the description of subjective vertical (22). Hence, visual input for humans is important for evaluation of body orientation and, consequently, for postural control.

Figure 1. Simplified model of the postural control system. The "reference input" represents that higher nervous system function which defines the appropriate body orientation at any given time. See text for details. (75,109)


CONTROLLER
CONTROLLED SYSTEM
EXTERNAL DISTURBANCE
REFERENCE INPUT
INTEGRATION AND FORMULATION OF POSTURAL MOTOR PROGRAM
ACTUATING MUSCLES
BODY POSITION
VISUAL AFFERENT INPUT
LABYRINTHINE AFFERENT INPUT
SOMESTHETIC AFFERENT INPUT

The role of visual feedback in quiet standing dogs has been quantitatively investigated (70). Based on the absence of any significant increase in longitudinal and transverse movements of weight distribution during blindfolding, it was concluded that either visual input is of minimal importance in the operation of the postural control system or that quiet standing does not adequately test the system to reveal any existent deficits. The latter is apparently correct, since a more demanding postural problem (e.g. sinusoidal perturbation) revealed significant changes in the dog's postural stability as a result of blindfolding (110). Experiments on human subjects also demonstrated that vision is a more potent sensory feedback channel under "dynamic" conditions (e.g. a sinusoidally swaying wall in front of the subject) than under "static" circumstances, when visual input is utilized less than proprioceptive and vestibular feedback for postural control (12).

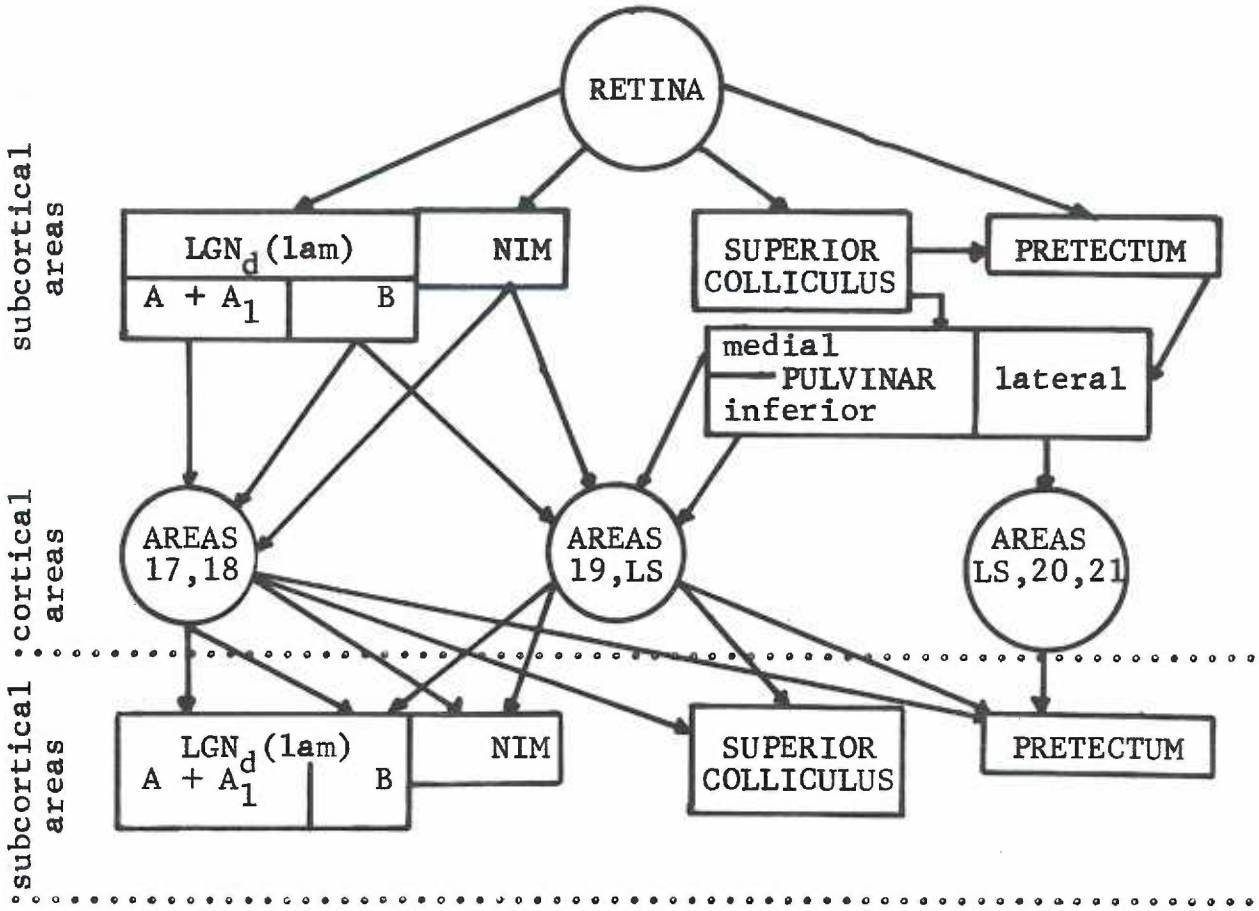
The objective of this investigation takes origin directly from work by Talbott (108,110), which demonstrated that blindfolding modifies the dog's postural response to longitudinal, sinusoidal perturbation. This effect was quantified by changes in the dog's body motion and various joint angle changes that together describe the animal's overall response. These results suggest that visual information is necessarily and predictably utilized in generating a central motor program for stabilization of posture. The objective of this investigation is to identify the role of the dog's visual cortex in integrating appropriate visual information into postural control motor programs.

Anatomy of the Visual System

The projection of visual input to various cortical and subcortical visual areas has been well established by both anatomical and physiological studies. In the cat, four parallel pathways transmit visual information from retina to cerebral cortex, as shown schematically in Fig. 2A (100). The laminar portion of the dorsal lateral geniculate nucleus (LGN_d(lam)) projects almost exclusively to visual cortical areas 17 and 18, while the medial interlaminar portion (NIM) projects predominantly to areas 18, 19 and lateral suprasylvian cortex (LS or Clare-Bishop area) (47,79). Thus, the LGN_d is not striate-specific (i.e. projecting only to area 17) in the cat, as is believed for primates (71). The superior colliculus projects to cortical areas via at least one synapse in a thalamic nucleus, primarily pulvinar (36,47,99). The cortical projection sites include areas 18, 19 and lateral suprasylvian cortex. Corticopetal projections from the pretectum (e.g. nucleus of optic tract, NOT) are primarily to association cortex (i.e. areas 20, 21) (100). Extensive reciprocal connections from visual cortex to subcortical nuclei are illustrated in Fig. 2B. The LGN_d(lam) receives corticofugal feedback primarily from areas 17 and 18, while the superior colliculus receives projections from all areas of visual cortex in a different fashion in the separate laminae (50,63,64,98). Thus, in the cat, visual information can reach cortex via at least four separate subcortical pathways. The LGN_d(lam) and superior colliculus receive the largest proportion of visual afferents, although all four of the primary subcortical projection areas are to some degree retinotopically organized (30,82,113). The overlap of different corticopetal pathways of

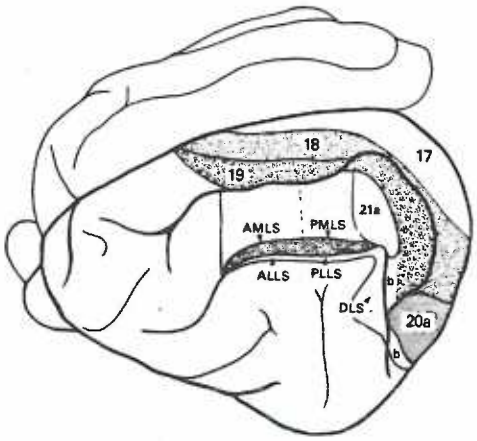
Figure 2. Schematic summary of (A) corticopetal projections of retinal afferents, (B) corticofugal projections onto subcortical visual areas, and (C) cortical visual areas in the cat. LGN_d(lam) = laminar portion of dorsal lateral geniculate nucleus, NIM = medial interlaminar nucleus, LS = lateral suprasylvian cortex, AMLS = anteromedial LS, PMLS = posteromedial LS, ALLS = anterolateral LS, PLLS = posterolateral LS, and DLS = descending LS (A and B from 47,50,64,79,98,99,100; C from 100).

(A)


RETINA
subcortical areas
LGN$_d$ (1am)
A + A$_1$
B
NIM
SUPERIOR COLLICULUS
PRETECTUM
medial
PULVINAR
inferior
lateral
cortical areas
AREAS 17,18
AREAS 19,LS
AREAS LS,20,21
subcortical areas
LGN$_d$ (1am)
A + A$_1$
B
NIM
SUPERIOR COLLICULUS
PRETECTUM

(B)

(C)

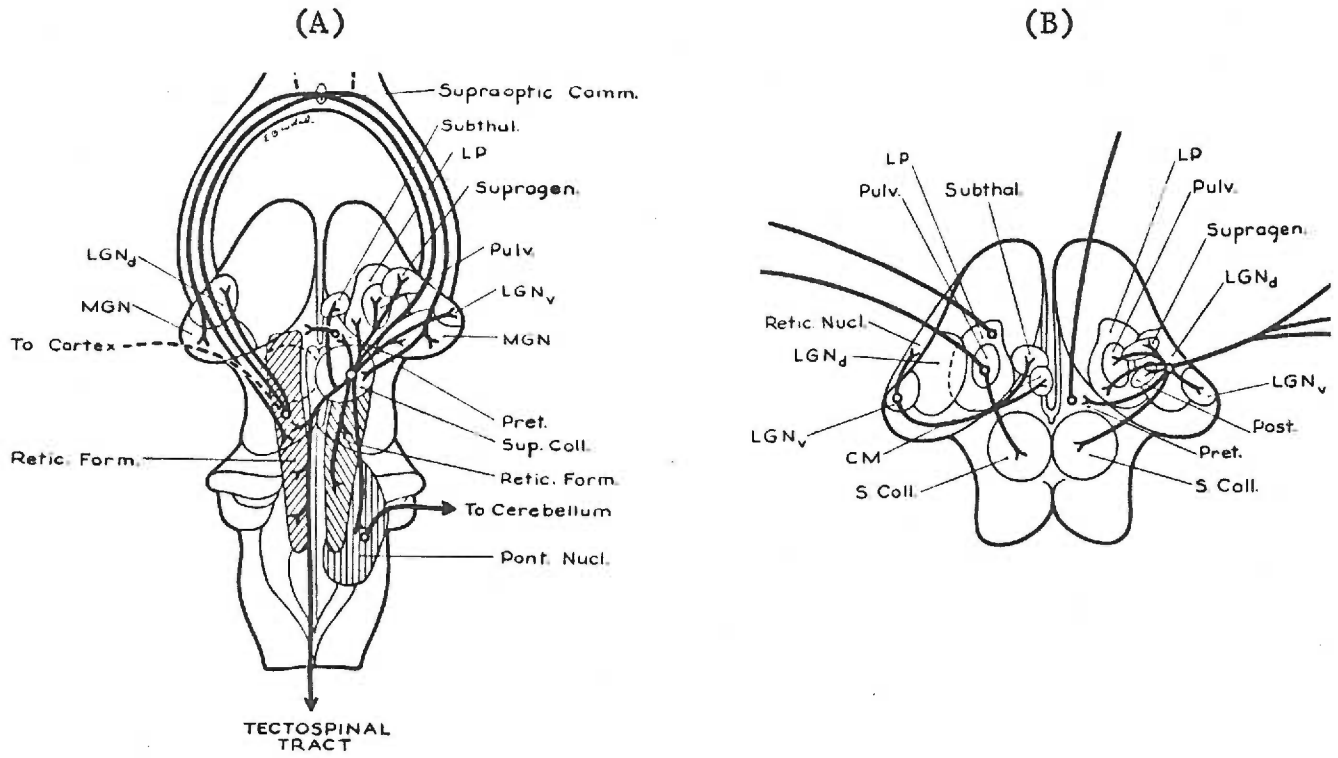

18
17
19
21a
AMLS
PMLS
ALLS
PLLS
DLS
20a
b

retinal afferents and the reciprocal relationships between subcortical nuclei and visual cortex support a view of visual processing that describes a high degree of interaction between the parallel pathways and their cortical and subcortical projection sites. The selective projection of laminae A and A_1 of the LGN_d to areas 17 and 18, and the equally specific corticofugal feedback onto A and A_1 suggests either a segregated visual processing by areas 17 and 18 serving a specialized function, or a redundant, parallel processing of similar visual information by various cortical areas.

In addition to some overlap of corticopetal projections from superior colliculus and LGN_d, great numbers of local interconnections exist between these subcortical nuclei which should not be ignored. Fig. 3 (A and B) illustrates schematically the local efferent projections from superior colliculus and LGN_d, respectively (64). Besides having many common thalamic nuclei and pretectal sites of termination, the superior colliculus and LGN_d also possess direct reciprocal connections with one another (36,64). In addition to these local interconnections, the cortical termini of LGN_d, pulvinar and lateral posterior nucleus of the thalamus all project retinotopically onto the superior colliculus, matching the direct retinocollicular input (63,99).

In summary, the salient points of this brief anatomic survey of the CNS visual system are the following: (1) the superior colliculus projects via thalamic relays to widespread areas of visual cortex that are partly outside the geniculocortical projection from laminae A and A_1 to areas 17 and 18; (2) the LGN_d laminae A and A_1 receive projections from primary visual cortex (i.e. areas 17 and 18), while the superior

Figure 3. Summary of local subcortical interconnections in the cat. (A) superior colliculus projections, (B) LGN_d projections. (64)



LGN$_d$: lateral geniculate nucleus, pars dorsalis. LGN$_v$: lateral geniculate nucleus, pars ventralis. LP: lateralis posterior nucleus. MGN: medial geniculate nucleus. Pont. Nucl.: pontine nucleus. Pret: pretectum. Pulv: pulvinar. Retic. Form.: reticular formation. Subthal.: subthalamus. Sup. Coll.: superior colliculus. Supragen: supragenicular nucleus. Supraoptic Comm.: Supraoptic Commissures.

colliculus receives projections from all areas of the visual cortex; and, (3) the geniculostriate and midbrain portions of the visual system are richly interconnected, often in retinotopic fashion, both locally and through cortical loops. It seems apparent that the potential pathways for extraction and integration of visual input into postural control responses are numerous -- whether involving either midbrain or thalamocortical processing exclusively, or requiring interaction between the cortical and subcortical parts of the visual system.

Physiology of the Visual System

No direct investigations on CNS visual function as it pertains to postural control have been performed. A large literature exists, however, on CNS processing of visual input during spontaneous or evoked eye and head movements in animals and during visually evoked motion in humans. The common experimental approaches have been of three designs: (1) electrophysiological, single-unit characterization of neuron populations from different visual areas; (2) selective lesioning of cortical and subcortical visual areas followed by more-or-less qualitative evaluation of visually-guided behaviors; and (3) systematic movement of parts of the visual field to reveal their subjective effect on body orientation and movement. In more recent investigations, a combination of the first two techniques has been utilized, allowing inquiry into the dependence of the functional properties of a given visual area on the integrity of other specific CNS sites.

Electrophysiological Evidence

The proper control of eye, head and body movement or posture

requires the detection of movement, whether real or self-induced, in the visual surround. The neuronal equivalent of the visual field is the receptive field. This refers to that region of visual field within which some stimulus can influence the firing of a specific neuron. The receptive field properties of neurons have been studied from all major visual areas of the CNS. The specific receptive field properties relevant to postural control are essential stimulus movement, direction selectivity and a wide range of velocity preferences. Theoretically, neurons involved in processing visual information for integration into movement or postural control would be expected to show a wide range of direction and velocity selectivity.

The neurons within the primary visual cortex were first characterized by Hubel and Wiesel in the cat (43,44). They reported that some neurons in area 17 responded differently to two diametrically opposite directions of movement or only to one direction of movement -- i.e. the phenomenon of direction selectivity. Also, a range of optimal stimulus velocities was found. Neurons of area 18 in the cat have since been well characterized and found to possess similar response properties to direction of stimulus movement, but different receptive field sizes and velocity preferences, as compared to area 17 (see Table I) (93,111). These differences in receptive field properties of neurons in areas 17 and 18 apparently contradict the hypothesis of serial processing of visual input in the visual cortex suggested by Hubel and Wiesel (44). Additionally, as illustrated in Fig. 2, the LGN_d(lam) projects directly to both these areas in the cat (99). The dependence of the receptive field properties of area 18 neurons on input from area 17 has been

directly investigated by selectively ablating area 17 in cats. Most properties of area 18 neurons were unchanged except for velocity preference, which increased slightly toward faster target speeds (26,87). Therefore, the neuronal populations in areas 17 and 18 receive direct LGN_d(lam) visual projections and demonstrate independent direction and velocity selectivity. In addition, the receptive field size and velocity preferences vary between the two cortical areas, possibly a manifestation of the superior colliculus-pulvinar projection received by area 18 along with the direct geniculocortical projection.

The lateral suprasylvian visual cortex (LS) receives visual inputs monosynaptically from midbrain visual areas (see Fig. 2), as well as crossed and uncrossed corticocortical projections from areas 17, 18 and 19 (30,49). Thus, the LS area is a cortical point of convergence of midbrain and geniculocortical projections, much like the superior colliculus (i.e. a midbrain point of convergence). Most cells in the LS cortex are specialized for response to moving stimuli, demonstrating direction selectivity and a wide range of effective stimulus velocities (see Table II) (95). In contrast to neurons of areas 17 and 18, the LS cells possess much larger receptive fields, respond to a more broad range of stimulus velocities and are less influenced by stimulus orientation. These receptive field properties suggest that the neurons of LS cortex may lie outside the proposed hierarchy of serial steps in visual processing. Instead, they may carry out some function unrelated to visual analysis, based on convergent cortical and subcortical inputs.

The receptive fields of cells in the superior colliculus may be characterized as well; but, the anatomical fact that a convergence of direct retinal and indirect retinocortical input occurs retinotopically onto most superior colliculus visual neurons must not be forgotten. Most collicular visual neurons require a moving stimulus to evoke consistent responses throughout their receptive fields (34). The receptive field size is substantially larger for superior colliculus neurons than neurons of visual cortex, with a range from 1° X 1° to the entire contralateral hemifield (102). The proportion of collicular cells directionally selective also varies depending on which study one reads, but the median percentage centers around 75% - 80% of those cells located in the superficial laminae which receive only visual input (34,104). This figure is very similar to that reported for all visual cortical neurons (Tables I and II). The velocity preference of collicular units is very broad, ranging from 2.5°/sec to over 100°/sec (102). However, the distribution is bimodal, with optimal velocities for the population of 10°/sec and 50°/sec. Recalling the difference in velocity preference reported for areas 17 and 18 (Table I), it would appear that the neurons in superior colliculus either receive convergent input from both cortical areas or, if independent of visual cortex for velocity preference, process retinal input in a parallel fashion to the visual cortical areas.

The question of superior colliculus dependence on cortical input for specific receptive field properties has been examined directly. Anatomical degeneration experiments have established that cortical input to superior colliculus is predominantly ipsilateral (29,98),

and that retinocollicular projections are largely contralateral (29, 52). When lesions are placed in visual cortical areas 17, 18 and 19, the receptive field properties of ipsilateral collicular units are drastically changed when tested from 1 to 4 weeks later (115). The approximately 75% of units normally directionally selective is reduced to near 0%, and the normally 80% of superior colliculus cells binocularly driven is reduced to near 30%. Other investigators have ablated only area 17 and found similar results (e.g. 75% directionally selective reduced to 12%, and 80% binocularly driven to 29%). In addition, when areas 18, 19 and LS were ablated, leaving area 17 intact, no change occurred in any superior colliculus receptive field property (80). At least one study has found no evidence of collicular dependence on visual cortex for receptive field properties of movement or directional selectivity (41). Overall, the weight of data is supportive of visual neurons in the cat's superior colliculus being dependent on retinal and cortical convergence (i.e. area 17 primarily) for receptive field properties of directional selectivity and binocularity.

For a full appreciation of the potential functions of the superior colliculus, it is necessary to briefly review the variety of sensory modalities which converge on different laminae of this midbrain nucleus. Visual input is not the only afferent input to the superior colliculus. The dorsal, superficial laminae (zonal, superficial grey and optic layers) consist of neurons that predominantly receive exclusively visual input, while the intermediate laminae (intermediate grey and intermediate white layers) and ventralmost, deep laminae (deep grey and deep white layers) contain neurons receiving a variety of convergent input (34). Neurons

in the intermediate laminae have been identified which respond to stimuli from visual, somesthetic and auditory sensory modalities (34, 104). Further, certain characteristics of the receptive fields for the various modalities are quite similar -- receptive field sizes, requirements for moving stimuli and direction selectivity (35,114). A tactile representation in the superior colliculus has revealed a somatotopic organization which is similar to the visuotopic representation (35,103). Specifically, neurons in the rostral superior colliculus possess visual receptive fields near the area centralis, while somatic receptive fields represent the face. Moving caudally in the superior colliculus, neurons are encountered with visual receptive fields more toward the periphery, while the somatic receptive fields move to forelimbs and then to shoulders and trunk (35). As a result of this extravagant convergence of multimodal afferents onto collicular neurons, and the receptive field similarities between modalities represented in the superior colliculus, an hypothesis has been proposed that the superior colliculus integrates and organizes the control of head and eye "orienting" and "following" responses on the basis of multimodal cues (34). Thus, the use of visual processing occurring at this midbrain visual center for feedback into the postural control system is a definite possibility.

Assuming that receptive field properties of (1) essential stimulus movement, (2) direction selectivity, and (3) wide range of velocity preferences are basic in the processing of visual information for postural control, a pontocerebellar pathway that receives secondary visual projections from both superior colliculus and visual cortex has

been identified as possessing these appropriate receptive field properties (32). The receptive field properties of the neurons in the rostral pontine nuclei that receive visual input and project to cerebellum include: 96% of a population of 232 recorded units direction selective; a requirement for moving stimuli; no velocity preference with normal direction selectivity up to velocities of 1000°/sec; large receptive fields ranging from 3° X 4° up to entire hemifields; 98% of neurons binocularly driven; and no convergence with other sensory modalities (i.e. auditory and somatosensory cells are located in nearby nuclei) (2). The input to these pontine nuclei arises from both superior colliculus and visual cortex, and is segregated -- superior colliculus projects to dorsolateral nucleus in ipsilateral pons, while areas 17, 18 and 19 plus LS cortex project to ventral and peduncular pontine nuclei (49). Selective cortical lesioning experiments have demonstrated that although all parts of area 17 project onto ventral pontine nuclei, the projection from that portion representing approximately central visual field is relatively "sparse," while it is "heavy" from parts representing more peripheral visual field (2,17). The projection of the pontine nuclei is predominantly via mossy fibers to vermal lobules VI, VII and VIII (39,94). A visual projection has also been identified from midbrain (i.e. accessory optic tract and nucleus of optic tract, AOT and NOT) to the inferior olive and then, via climbing fibers to the flocculonodular lobe (59,92). The latter pathway is considered an intricate part of the vestibulo-ocular interactions controlling eye movements (4). Overall, the properties of the pontine visual cells suggest a

visual pontocerebellar pathway that is sensitive to specific directions of moving stimuli predominantly from the peripheral visual field, not sensitive to precise form, and responsive to a broad range of velocities. Therefore, realizing the role of the cerebellum in all phases of motor control (for postural, locomotive and skilled movements), the pontocerebellar pathway, whether involving projections from visual cortex, superior colliculus and/or midbrain tectum (NOT), is a likely route for integration of visual feedback into postural control motor responses.

Another potential route for incorporation of visual information into postural control is a more direct pathway from visual areas to vestibular nuclei in the medulla and thence, to cerebellum. Neurons whose activity is strongly coupled to eye movements and presumably involved in their programming have been identified in the vestibular nuclei of monkeys (51,65). However, some neurons have been reported to show activity not correlated with eye movements, but still visually modulated - suggesting an involvement not in oculomotor programming but in programming of compensatory head and possibly body movements (65). When rabbits were subjected to pure vestibular, pure visual or simultaneous vestibular-visual rotatory movements, vestibular units were identified that discharged similarly to corresponding directions of visual or vestibular motion. They also demonstrated facilitatory or inhibitory modulation when simultaneous visual-vestibular motion was appropriately matched or incompatible, respectively (38). Therefore, it seems that vestibular neurons can relay information concerning body motion based on <u>both</u> vestibular and visual signals. The involvement of

this pathway for visual input into postural responses is another possibility.

Electrophysiological experiments provide inferential information on the postural control function of various visual areas based on receptive field properties that would be expected for neuron populations processing visual input into postural motor responses. The visual cortex and superior colliculus both qualify as potential sites. Striate cortex (area 17) receives no midbrain projections and possesses selective reciprocal connections with the laminar portion of the LGN_d. Yet, it also possesses neuronal receptive field properties that do not preclude a postural control function. The extrastriate (areas 18, 19 and LS) cortex possesses receptive field properties theoretically more suggestive of a postural function, but receives tectocortical projections via pulvinar, suggesting a great deal of convergent interaction in visual processing. The midbrain (superior colliculus and pretectum) receives a great deal of convergent input from all areas of visual cortex and possesses receptive field properties similar to those found in LS cortical neurons. Likewise, the midbrain is theoretically a plausible site for visual processing used in postural control. The identification of a pontocerebellar pathway that relays visual input to the cerebellum provides no means of identifying a specific visual area that may function in postural motor control, since all of the visual cortical areas and midbrain centers project to pontine nuclei and are relayed to the cerebellum. Thus, the electrophysiologic evidence on the physiology of the various visual centers provides only suggestive information on possible CNS candidates for the

postural controller. None of the major cortical and midbrain visual centers are precluded, although some are more likely than others. Importantly, the striate cortex is the only visual area which receives a relatively homogeneous input (i.e. retino-geniculostriate projection) and yet contains a subpopulation of neurons whose receptive field properties might support some postural control function.

Effects of Selective Lesions

The ablation of cortical and subcortical visual areas and the evaluation of subsequent deficits in function or behavior is a technique which has been used for more than a century, but has suffered from a basic inadequacy for equally as long. The post-operative evaluation consists customarily of the clinical description of visual acuity, or visually-guided movements, or of flux or pattern discrimination, all of which involve more-or-less qualitative judgements on the part of the investigator. In addition, the desired lesion is practically never complete in its extent, or else impinges upon adjacent structures. The lesion must always be verified post mortem by the investigator using histologic methods and then be evaluated by the individual reader. Conclusions based on such data are often difficult to reproduce exactly, so that conflicting interpretations abound. However, if one realizes the qualitative nature of such data and concentrates on the conformable findings, not the discrepant ones, then such investigations can be useful in identifying CNS visual areas that participate in certain visually-guided behaviors.

Early experiments on the cat and dog in which unilateral or bilateral occipital ablation was performed, consistently showed

resultant contralateral hemianopsia or total anopsia, respectively, i.e. complete blindness (28,62,66). This loss of vision was generally attributed to the removal of striate cortex (area 17). However, close inspection of the described lesions reveals them to involve most of areas 17, 18 and 19, i.e. large lesions extending beyond the striate cortex. More recent, rigorous lesion studies have been performed extensively on cats. Selective ablation of area 17 reportedly produces "no discernible deficits" in visually-guided behavior (e.g. visual following, visual orienting responses, visual neglect or perceptual rivalry, eye blink to visual threat, visual placing, general visually-guided avoidance, jumping and climbing) (24,97,99,100). Extension of the cortical lesion to include both areas 17 and 18 has no effect -- visually-guided behavior remains essentially normal (100). However, when areas 17, 18 and 19 are ablated, "mild deficits" result: visual following is depressed, especially to fast velocity movements; eye blinks to visual threat become irregular or absent; mild changes occur in visual placing, i.e. descent from a raised platform is performed "cautiously"; and, visual orienting responses remain uneffected (24,97). These deficits are most noticeable immediately post-operatively and ameliorate partially in the course of one month. Only when cortical ablations include all of the occipitotemporal cortex (i.e. areas 17, 18, 19, LS and parts of areas 7 and 5) does a near complete contralateral hemianopsia or total anopsia result (77,88,97, 99). Orienting responses, visual following, visual placing, visual avoidance and blink to visual threat are all lost permanently.

Recent studies investigating flux and pattern discrimination in cats after selective lesions of various cortical and subcortical visual areas contradict the view of separate visual functions mediated at cortical and subcortical levels. Hubel and Wiesel (43,44) originated the concept that visual form discrimination derives from a serial or hierarchical processing of visual information along chains of neurons with increasingly complex receptive field properties, beginning with the geniculostriate pathway and spreading to extrastriate cortex. Subcortical visual centers other than the LGN_d have been thought to mediate purely reflex visuomotor behaviors. After ablation of area 17 alone or along with area 18, cats show both normal learning of various complex pattern discriminations and of flux discrimination, and normal retention of learned pattern discriminations (24,100). Extension of the lesion to include area 19 has no effect on the ability to relearn a discrimination between horizontally and vertically striped patterns that have been equated for total luminous flux (5). However, a large, bilateral occipitotemporal ablation alters visual discrimination ability -- striped pattern discriminations are not retained nor relearned, while light/dark discrimination is partially retained and normally relearned (58). Finally, a lesion of areas 19 and LS, sparing most of 17 and 18, has little effect on flux or striped pattern discrimination, but eliminates the ability to discriminate complex shape patterns (100). Thus, it is apparent that visual areas outside of areas 17 and 18 can mediate visual form discrimination.

Studies on the effects of selective lesions of subcortical areas on visual form and luminous flux discrimination have dealt primarily with

superior colliculus and pretectum. Unilateral ablation of pretectum and superior colliculus in split-brain cats or bilateral ablation of both structures, has no effect on learning or retention of brightness discriminations and retention of shape pattern discriminations; however, the combined lesion impairs the learning of new shape pattern discriminations (7,34,99). Lesions restricted chiefly to either the superior colliculus or pretectum produce effects similar to the combined lesion, except for the deficit in learning pattern discriminations, which is less marked (7). Therefore, the midbrain apparently plays an important role in integrating and mediating pattern discriminations, but is not essential for luminous flux discrimination in the cat.

The ablation of midbrain visual areas results in visuomotor deficits, as well. Bilateral lesions of the superior colliculus in the cat produce serious deficits in visually-guided behavior (i.e. absent visual following, avoidance responses, placing and orienting), most of which disappear somewhat with time, except for the orienting response to laterally introduced stimuli (34,99,101). Visual placing returns to a near normal state during the second post-operative month, visual avoidance is vastly improved, visual following improves for slow stimulus speeds, but lateral orienting responses remain defective. Thus, visually-guided behavior in the cat (except for lateral orienting responses) can apparently be mediated near-normally by visual centers other than superior colliculus. This conclusion is confirmed by other investigators as well -- normal detection of moving and stationary stimuli in the cat's central visual field does not require superior colliculus, while the superior colliculus is essential for normal

visual orienting to peripheral stimuli (72).

The results of the effects of selective cortical and subcortical lesions on visually-guided behavior and visual discrimination ability reviewed above are summarized in Table III. It is apparent that the ablation of visual cortex produces serious deficits in visually-guided behavior only with massive lesions that include all striate and extrastriate visual areas. These large occipitotemporal lesions permanently disrupt all visually-guided behaviors. On the other hand, ablation of superior colliculus produces a complete deficit only in orienting behavior. It appears, then, that all visually-guided behaviors require the integrity of some visual cortex, whereas only the orienting response absolutely requires the superior colliculus. The ability to learn patterned visual discriminations depends on the complexity of the problem -- striped patterns, but not shape patterns, can be learned after ablation of areas 17, 18 and 19. And, if the tectocortical connections between midbrain and LS cortex are interrupted in any way, shape patterns cannot be discriminated, suggesting necessary cortical-midbrain interaction. The ability to learn a light/dark brightness discrimination does not seem to depend upon any specific visual area, but requires only that some CNS visual area be intact.

A functional interaction between the cortical and subcortical visual centers has been directly demonstrated by combined lesions of visual cortex and midbrain (58,88,90,96,98). Complete ablation of occipitotemporal cortex unilaterally produces a contralateral hemianopsia with almost no compensation over time. If a contralateral ablation of the superior colliculus is then performed, some visually-

guided behavior using the originally hemianopic visual field returns, i.e. visual orienting, partial following, blink to threat and placing (96). Thus, one hemifield is apparently being served by its contralateral visual cortex, while the other is projecting to its contralateral superior colliculus. From these combined lesion studies, it is concluded that cortex and midbrain can interact in mediating visuomotor behavior. This interaction is apparently essential to balance reciprocal inhibitory influences between colliculi (i.e. any small area of striate, extrastriate or LS cortex is apparently adequate), since the overall cortical influence onto the superior colliculus is considered to be facilitatory (96,97). This hypothesis is supported by the ability of collicular commissure transection to reinstitute bilateral visual following and orienting responses that had been lost after bilateral occipitotemporal cortex ablation (90). Therefore, it appears that normal visually-guided behavior remains intact as long as some interaction between visual cortex and superior colliculus is possible. Otherwise, the superior colliculus alone may permit near-normal visual orienting and following responses once the commissure is cut, while the cortex alone may allow near-normal visual avoidance, placing and following, but only poor orienting behavior.

The effects of combined cortical and midbrain lesions on visual discrimination have also been investigated. Whereas a transection of the collicular commissure after bilateral occipitotemporal ablation results in the return of some visually-guided behavior, learned pattern discrimination is not retained nor can it be relearned, even though a brightness discrimination is retained throughout (58). Thus,

although the midbrain visual centers are essential for learning a "shape pattern" discrimination (perhaps through the tecto-pulvinar-cortical pathways described earlier - see Fig. 2), in the absence of all visual cortex they cannot mediate pattern discrimination as they do some visually-guided orienting behaviors.

Some investigators have studied the effects of selective lesions of either the superior colliculus or visual cortex on an animal's ability to make a complete "discriminative motor response" (e.g. proper locomotion toward and selection between two doors with different patterns at the end of a runway). Experiments on hamsters, rats, tree shrews, squirrel monkeys and cats appear to support a segregation of cortical and midbrain visual function, which is not obvious from the previously described clinical evaluations of selective lesions (i.e. visually-guided behavior and discrimination learning). Studies on hamsters reinforce the unique role of the superior colliculus in orienting responses of the eyes, head and body to visual stimuli, i.e. "localization" (85,86). The visual cortex is reported to be necessary for "identification" and thus discrimination of objects and patterns, and for initiation of locomotor actions directed toward the object (85). Similar experiments on rats evaluating the visuo-spatial guidance of locomotor movements reinforce the idea that visual cortex is necessary to mediate these responses -- removal of superior colliculus has no effect on the normal coordinated motor response (33). Studies on tree shrews, squirrel monkeys and cats assessing their visually-guided locomotive behavior further support this dichotomy of visual function (46,61). It is concluded that normal visual guidance

of locomotion (i.e. ambulatory localization) in a goal alley is dependent upon cortical mechanisms, while appropriate orienting behavior (i.e. stationary localization) is a midbrain function. In these experiments, cats and monkeys were trained to emerge from a starting gate, move into an open field and enter a goal box with a light turned on at its terminus (another goal box being at right angles to the first, at the other end of the open field and without a light on). Superior colliculus ablation resulted in inattentiveness to visual stimulus discrimination when the animal remained stationary, but demonstrated no effect when ambulation toward the goal was taking place. Ablation of areas 17, 18 and 19, however, resulted in the cat or monkey emerging from the starting gate, following the wall of the enclosure until it reached the goal box, peering inside, and then, responding correctly to enter the lighted box. This is interpreted as essentially converting the ambulatory localization task, involving "readjustment and coordination of the visual field with each movement toward the goal", into a stationary localization problem, solvable by the superior colliculus (61).

It is not clear whether visual processing in postural control is similar to that in either visually-guided locomotive behavior (ambulatory localization) or visually-guided lateral orienting responses (stationary localization). In normal daily activities, the visual surround is most commonly stationary as the animal's body is in motion (i.e. self-induced movement of the visual surround) -- an ambulatory localization task. At other times, however, the animal may be stationary and a movement occur in some part of its visual

field (i.e. real object movement) -- a stationary localization task. A likely possibility for postural control is that both types of visual information are integrated into the postural control response, thus requiring cortical and tectal interaction. Therefore, depending on the immediate behavior, either superior colliculus or visual cortex, or both, may be necessary for proper visual processing in the postural control system.

Human Psychophysical Studies

During the early stages of development of the postural control system in human infants, vision functions powerfully in standing (53). At this time, the creation of a conflicting relationship between visual and proprioceptive feedback is usually dominated by the visual, suggesting that for infants visual input is more potent than proprioceptive input. It is hypothesized that when infants are learning to stand they rely mainly on visual feedback, and only later after some practice does proprioception acquire its normal efficiency. Similar studies repeated on adults suggest that visual feedback plays a lead role in tuning up proprioception, both in learning new postural stances and in general (55).

First evaluated in the infant, the potency of visual feedback in evoking changes in body orientation has also been investigated in adult subjects, by a different approach. The rotation of a drum about a seated subject (7), or the projection of film strips in the peripheral visual field (9), can cause the subjective sense of self-rotation (i.e. circularvection) or longitudinal self-motion (i.e. linearvection), respectively. Any such optokinetic stimulus can be

interpreted by the subject in two ways: as exocentric motion perception -- i.e. an illusion of self-motion, where the moving surround appears stationary, or as egocentric motion perception -- i.e. perceived motion about a stationary self (14). The investigation of those visual field characteristics essential for producing each of these perceptions provides some useful information for speculations about visual processing for postural control.

Experiments on circularvection demonstrate that exocentric motion perception is mediated by peripheral visual field stimuli, while egocentric perception is served by the central visual field (14,23). This dichotomy of visual field processing was uncovered in part by the following experiments: (1) masking the central field (up to 120° in diameter) and stimulating the peripheral field by circular motion of the surround; (2) stimulating only the central field (up to 30° in diameter) with circular motion of the surround. Experiment 1 resulted in perceived circularvection similar in magnitude to that evoked by stimulation of the entire visual field; while experiment 2 induced exclusive egocentric motion perception. Input from the central visual field can, in certain situations, modify the perception of circularvection evoked by peripheral motion (14). When the exposure of central visual field was enlarged to a diameter of about 100° during rotation of peripheral visual field, circularvection began to be suppressed and replaced by egocentric motion perception. This apparent specialization in processing of peripheral visual field movement as exocentric motion would be a useful adaptation for accurate postural control during locomotion.

The dynamic frequency range within which peripheral visual feedback induces a perception of self-motion is lower than for vestibular feedback; both the amplitude and phase of linearvection decrease as the frequency of peripheral motion increases from 0.01 to 1 Hz (9). However, the relative potency of the visual input in perception of body orientation in adults can be qualitatively assessed by studying the interaction between visual and vestibular sensory input (9,57). For these experiments, subjects were seated on a cart which was moved in a forward-backward direction at pseudorandom frequencies and amplitudes, while a scene, moving only backwards at a constant velocity, was projected onto peripheral visual fields. The subjective direction and velocity of movement were described by the subject's movement of a lever, both with the eyes open and closed. With the eyes closed, the subjects reproduced the changes in direction and relative velocity of the cart quite accurately, but with the eyes open a constant forward linearvection was reported. These results reflect the powerful effects of information from the periphery of the visual field in the interpretation of self-motion. The quantitative comparison of visual and vestibular involvement is not valid, however, since it is uncertain whether both visual and vestibular stimuli were of comparable strength, or whether, for instance, vestibular stimulation might have been near threshold, while visual was nearly saturated. However, this visual-vestibular interaction, reflected by the dominance of peripheral visual input over conflicting vestibular cues, is modifiable, since a reduction in the visual dominance occurs with repeated trials (9).

The pertinent implications of these human psychophysical experiments in determining what visual processing occurs in postural control are the following: that the peripheral visual field appears to be processed more-or-less selectively for exocentric motion perception; that the central visual field is very little involved in exocentric perception, is utilized predominantly for egocentric motion information, and can inhibit exocentric perception evoked from the periphery; and that peripheral visual input, evoking exocentric linearvection can in certain instances override vestibular input when the two are in conflict -- although visual feedback for postural control operates more effectively at lower frequencies of motion than vestibular feedback. This apparent specialization for exocentric perception of peripheral visual field movement suggests a postural role for peripheral visual field input and the CNS areas processing it.

EXPERIMENTAL OBJECTIVES

The general objective described earlier expressed the aim of identifying the role of the dog's visual cortex in integration of appropriate visual information into postural control motor programs. The preceding review of literature dealt with visual processing in various visually-guided behaviors and provided some data on which to formulate a more detailed experimental objective in terms of specific, testable hypotheses.

The anatomical evidence concerning connections of superior colliculus, LGN_d and visual cortex emphasized the great number of interconnections between these visual centers. However, the midbrain projection to visual cortex is partly outside the primary geniculostriate projection to areas 17 and 18. The many interconnections between cortical and subcortical visual areas suggests extensive interactive functioning, while the homogeneity of geniculocortical connections with areas 17 and 18 hints at some uniqueness in function. Studies on the receptive fields of neuron populations in cortical and midbrain visual centers revealed superior colliculus dependence on connections with the visual cortex (primarily area 17) for direction selectivity and binocularity. These findings support a mutual dependence between the two main visual centers for some of their normal functions. Certain neuron populations of the superior colliculus receive convergent input from a variety of sensory modalities, supporting the contention that superior colliculus integrates multimodal input, directing orienting responses of eyes and head. This was confirmed for visual input by the effects of superior colliculus

ablation. The normal orienting response to lateral stimuli is apparently a unique function of superior colliculus in the cat. However, some visual cortex appears to be necessary for normal mediation of visually-guided behavior by the midbrain, possibly because of the necessity of cortical facilitatory tone. For visual guidance of locomotion, the visual cortex appears to be essential. Thus, both unique and parallel visual processing apparently takes place in cortical and midbrain sites to direct different types of motor behaviors. The investigations on the role of visual input in subjective kinesthesis and balance in human subjects indicates that the peripheral visual field plays a potent role in postural control, while the central field is involved in egocentric motion perception of real object movement. The apparent specialization for exocentric perception of peripheral visual field movement, besides suggesting a likely postural role for peripheral retinal input, indirectly intimates that the superior colliculus may be similarly specialized. Experiments described earlier involving bilateral occipitotemporal ablation followed by transection of the collicular commissure suggest that the retinotectal pathway processes peripheral retinal vision to a greater degree than central vision, whereas the visual cortex processes central retina more extensively than peripheral retina (16,88). The inference from combined human and cat data assigns the superior colliculus a likely role in specialized visual processing of peripheral visual input for postural control, but must be cautiously considered due to the unknown extent of interspecies variability. The central visual field, besides mediating egocentric

motion perception, can interfere with peripheral visually-induced motion perception (14). This implies an integration of central and peripheral retinal input, whether locally at the midbrain level or involving cortical-subcortical interconnections.

Therefore, to elucidate the role of primary visual cortex (areas 17 and 18) in the integration of central versus peripheral visual input into postural control motor commands, the following hypotheses were tested:

(1) If primary visual cortex is not essential in processing necessary visual input for normal postural control motor responses, then, the chronic ablation of visual cortex should produce no change in the postural response, and blindfolding should continue to change the postural response as it did pre-operatively.

(2) If primary visual cortex is essential for processing peripheral visual field input, then, the chronic ablation of areas 17 and 18 should abolish visually-induced postural corrections evoked by peripheral visual field movement.

(3) If primary visual cortex is essential for processing of central visual field input, then, the chronic ablation of area 17 and 18 should result in the inability of central visual field movement to inhibit the visually-induced postural response evoked by peripheral field movement.

(4) If peripheral visual field input is used exclusively for visual processing in postural control, then, blocking peripheral visual field feedback should produce a deficit

equivalent to blindfolding in the postural response.

The above hypotheses require the quantitative evaluation of a postural response so that the effects of chronic ablation and visual field masking can be determined reliably. In addition, visually-induced postural responses must be quantifiable and repeatable. The experimental design and methods for this type of quantitative testing of the postural control system are described in the following sections.

MATERIALS

Experimental Animal

Although most visual system research is performed on the cat or monkey, the dog was the experimental animal in this investigation for two reasons. A great deal of background information on the dog's postural control system has been accumulated through the investigations of Brookhart, Talbott and various associates. This isolated bit of information on the role of visual feedback in postural control becomes more meaningful when fit into an increasingly complete picture of a CNS control system. Also, the ability of the dog to be trained and to establish a stable, reproducible response to repeated testing both across trials and between individual animals permits statistical treatment of the data (19). This is necessary to permit quantitative evaluation of the effects of CNS lesions on the dog's ability to respond to postural disturbances and visual stimuli.

The anatomy of the dog's visual system has been little studied in comparison to that of the cat, but what has been described differs only slightly. The horizontal extent of the cat's visual field ascertained by perimetric testing is about 130° for each eye -- 90°- 100° ipsilateral and 30°- 45° contralateral (88,96). The dog's monocular visual field is 135°- 150° wide, extending approximately 120° ipsilaterally and 15°- 30° contralaterally, the contralateral boundary being vague and difficult to define accurately (91). Thus, although the dog's eyes are slightly more laterally placed than the cat's, the monocular and binocular segments of visual field are not too dissimilar

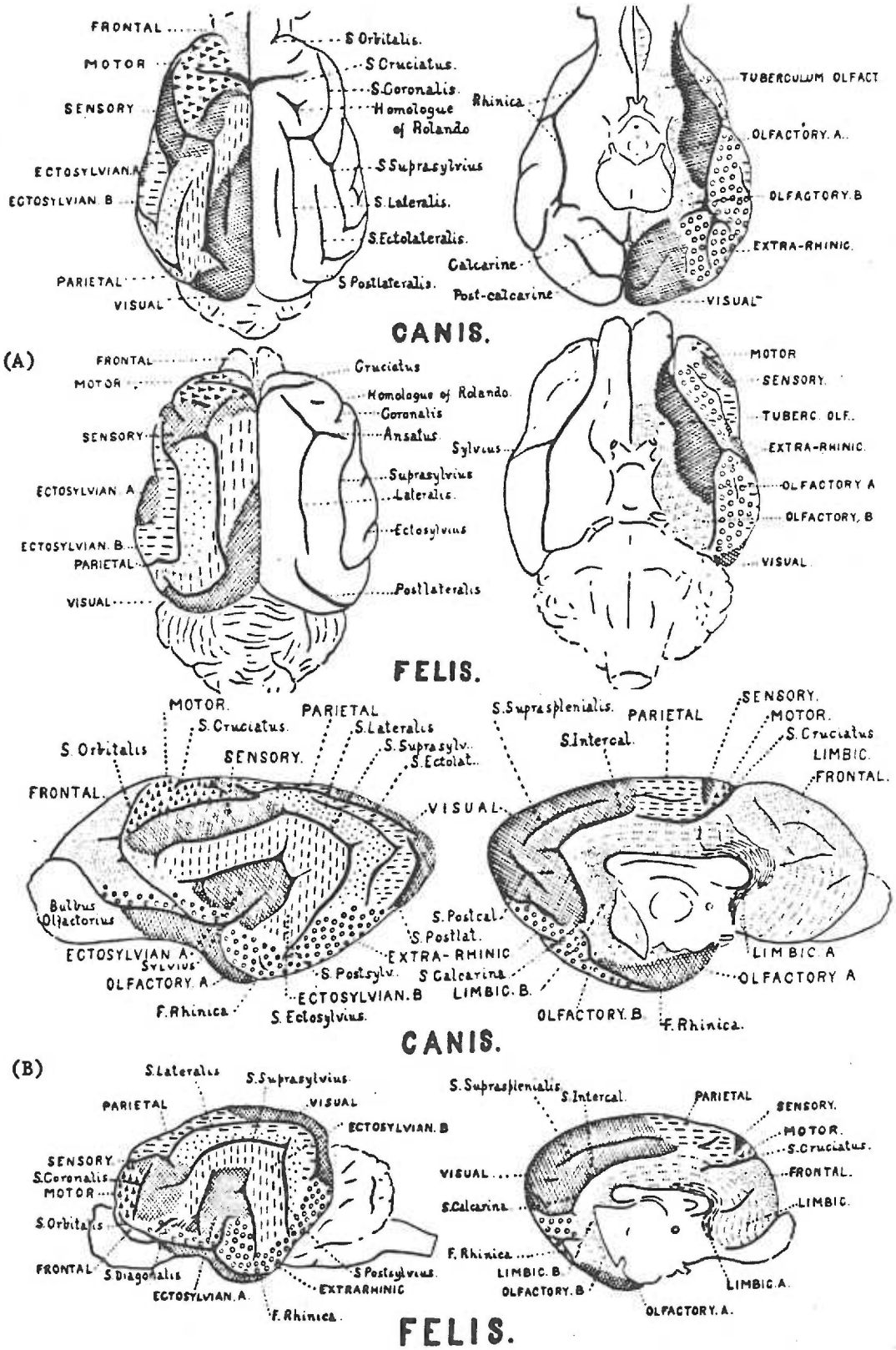
in the two animals. The degree of decussation of optic nerve fibers in the chiasm also varies somewhat. In the dog, 25% of the fibers do not cross (74), supporting a report in 1890, that "each ipsilateral visual cortex receives fibers from the lateral 1/4 of the ipsilateral retina and the medial 3/4 of the contralateral retina" (69). In the cat, however, 65 - 70% of the optic nerve fibers cross and 30 - 35% remain uncrossed (74,96). Whether this represents an increased capability for binocular vision by the cat or an increased use of central visual field (i.e. foveation) for heightened acuity is not certain. Interspecies and experimental variability challenge the significance of this small intergenus difference.

Retinal histology, however, supports the assertion of better developed central, high-acuity vision in the cat. The dog's retina contains predominantly rod receptors (about 95%) and no rod-free region, nor area of increased cone density (73). However, a horizontally elliptical area centralis of greater sensitivity to light is identifiable on the basis of a reduced density of large blood vessels (73,74). The cat, on the other hand, has a definite area centralis or macula composed largely of cone receptors that decrease in density toward the periphery (74). This retinal structure supports the hypothesis that the cat has a central retina more highly developed for high acuity vision. Overall, the slightly greater binocularity and possibly enhanced acuity suggest a phylogenetically higher developed visual sense in the cat than in the dog.

Histologic and electrophysiologic evidence indicate similar boundaries and cellular architecture in the cat and dog LGN_d, superior

colliculus and primary visual cortex. Campbell in 1905 localized the visual cortex in both animals based on cell and fiber-stained material. Fig. 4 shows Campbell's description of the visual cortex. For the cat, this description is supported by many recent anatomic and electrophysiologic investigations (26,64,96,106). For the dog, it is supported by a recent histologic description (1) and an evoked potential study performed by this investigator (Fig. 5). The visual area identified in the evoked potential study and delineated on the dorsal, posterior surface of the cortex by Campbell refers to primary visual cortex, i.e. areas 17 and 18, and includes lateral, postlateral, splenial and suprasplenial gyri. Secondary visual cortex, i.e. area 19 and LS area, lies in the ectolateral and suprasylvian gyri, respectively. The LGN_d appears histologically comparable between cat and dog (Fig. 6). The superior colliculus has also been histologically characterized and shows no marked differences between these two animals (76). The histologic similarity of subcortical visual centers in the two animals cannot be regarded as evidence of identical functional properties, but does suggest the absence of any major differences in function. Based on these similarities in structure of the visual centers in the cat and dog, and accepting the small difference in binocularity and the apparently more highly developed foveal region in the cat, the data on cat's visual processing reviewed in the preceding sections are assumed to be more-or-less applicable to the dog. Therefore, the hypotheses formulated from the background literature on cat and human studies were tested on the dog.

Figure 4. Functional areas of the cerebral cortex in dog and cat delineated on the basis of histologic cell and fiber staining. (A) Dorsal and ventral views, (B) lateral and medial views. From Campbell, 1905 (20).


FRONTAL
MOTOR
SENSORY
ECTOSYLVIAN A
ECTOSYLVIAN B
PARIETAL
VISUAL
S Orbitalis.
S Cruciatus.
S.Coronalis.
Homologue of Rolando
S Suprasylvius
S. Lateralis.
S.Ectolateralis.
S Postlateralis.
Rhinica
TUBERCULUM OLFACT
OLFACTORY. A.
OLFACTORY. B
EXTRA-RHINIC.
Calcarine
Post-calcarine
VISUAL
CANIS.
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FRONTAL
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ECTOSYLVIAN A
ECTOSYLVIAN. B
PARIETAL
VISUAL
Cruciatus
Homologue of Rolando.
Coronalis
Ansatus
Suprasylvius
Lateralis.
Ectosylvius
Postlateralis
Sylvius
MOTOR
SENSORY.
TUBERC. OLF.
EXTRA-RHINIC.
OLFACTORY A
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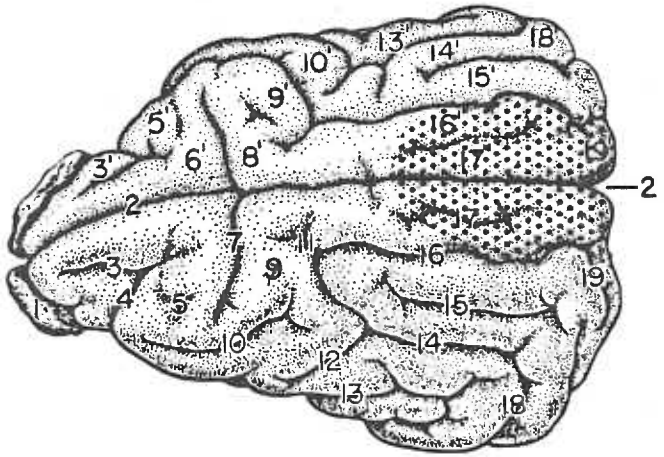
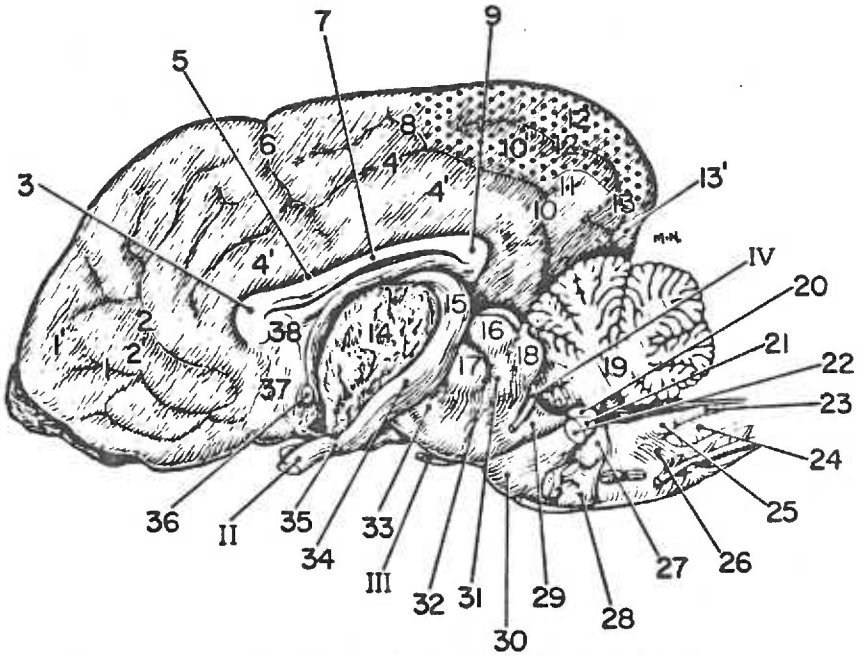
VISUAL
FELIS.
MOTOR.
S. Cruciatus.
PARIETAL
S.Lateralis
S. Orbitalis
SENSORY.
S.Suprasylv.
S.Ectolat.
FRONTAL.
VISUAL
Bulbus Olfactorius
ECTOSYLVIAN A.
Sylvius
OLFACTORY. A.
F.Rhinica
S. Ectosylvius.
S. Postsylv.
ECTOSYLVIAN. B
EXTRA- RHINIC
S Calcarina
LIMBIC. B.
S. Postcal.
S. Postlat.
OLFACTORY. B
S.Suprasplenialis.
PARIETAL
SENSORY.
S.Intercal.
MOTOR.
S. Cruciatus
LIMBIC.
FRONTAL.
LIMBIC A
OLFACTORY A
F. Rhinica.
CANIS.
(B)
S.Lateralis
S.Suprasylvius
PARIETAL
VISUAL
ECTOSYLVIAN. B
SENSORY
S.Coronalis
MOTOR
S.Orbitalis
FRONTAL
S.Diagonalis
ECTOSYLVIAN. A.
F. Rhinica.
S Postsylvius.
EXTRARHINIC
S.Suprasplenialis
S.Intercal.
PARIETAL
SENSORY.
MOTOR.
S.Cruciatus.
FRONTAL.
LIMBIC.
VISUAL
S.Calcarina
F. Rhinica
LIMBIC. B
OLFACTORY. B
LIMBIC.A.
OLFACTORY. A.
FELIS.

Figure 5. Area of canine cerebral cortex from which surface potentials to gross photic stimulation could be evoked. Criterion response was a 20 microvolt initial positive-going waveform with a latency to peak within 15 msec. The midline and posterior boundaries are poorly defined due to their inaccessibility with the surface electrode. The dorsal and anterior limits of this primary visual projection area are well defined. Four dogs were studied.



Sulci and gyri of the cerebrum (left dorsolateral view).

1. Olfactory bulb
2. Longitudinal fissure
3. Prorean sulcus
3'. Prorean gyrus
4. Presylvian sulcus
5. Precruciate sulcus
5'. Precruciate gyrus
6'. Anterior sigmoid gyrus
7. Cruciate sulcus
8'. Posterior sigmoid gyrus
9. Postcruciate sulcus
9'. Postcruciate gyrus
10. Coronal sulcus
10'. Coronal gyrus
11. Ansate sulcus
12. Anterior suprasylvian sulcus
13. Ectosylvian sulcus
13'. Ectosylvian gyrus
14. Middle suprasylvian sulcus
14'. Middle suprasylvian gyrus
15. Ectolateral sulcus
15'. Ectolateral gyrus
16. Lateral sulcus
16'. Lateral gyrus
17. Entolateral sulcus
17'. Entolateral gyrus
18. Posterior suprasylvian sulcus
18'. Posterior suprasylvian gyrus
19. Postlateral sulcus
19'. Postlateral gyrus

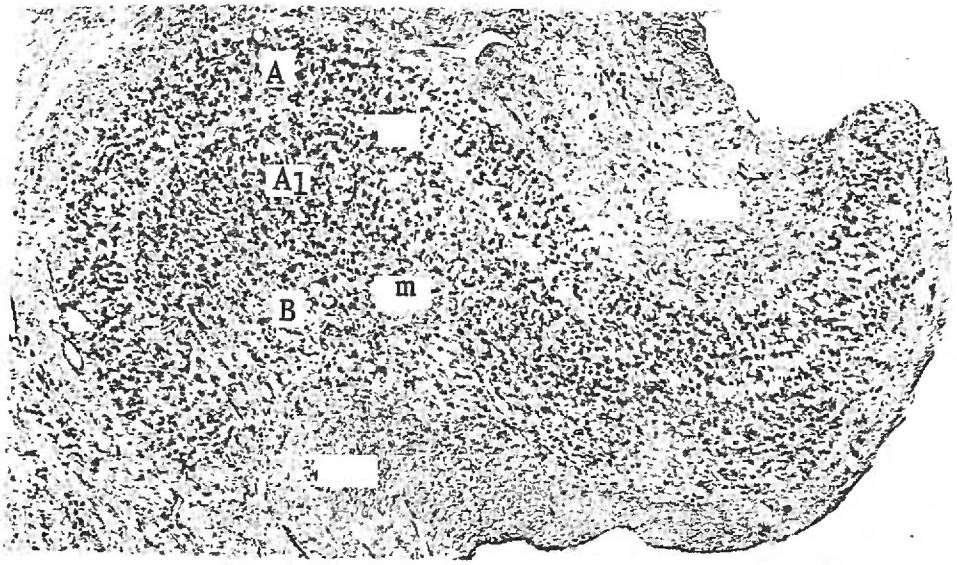


Medial surface of the right cerebral hemisphere and lateral surface of the brain stem.

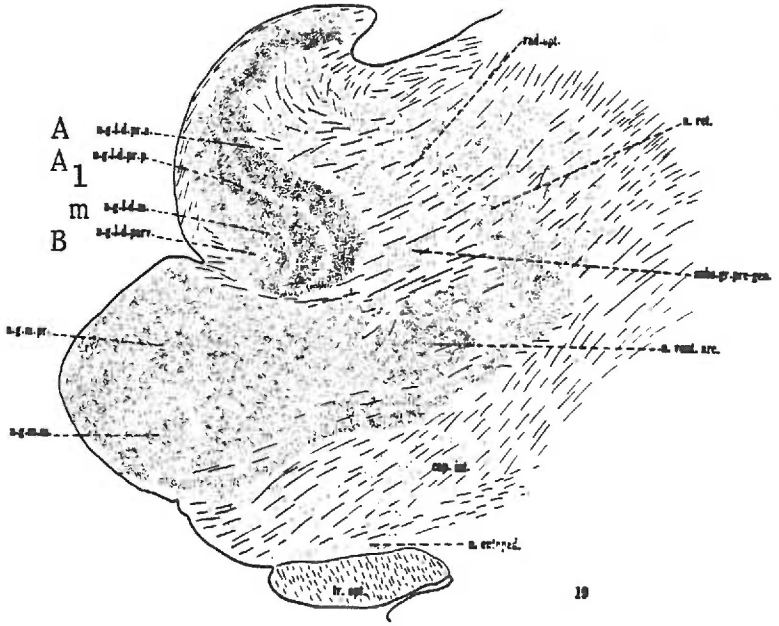
1. Ectogenual sulcus
1'. Ectogenual gyrus
2. Genual sulcus
2'. Genual gyrus
3. Genu of corpus callosum
4. Cingulate sulcus
4'. Cingulate gyrus
5. Callosal sulcus
6. Cruciate sulcus
7. Body of corpus callosum
8. Lesser cruciate sulcus
9. Splenium of corpus callosum
10. Splenial sulcus
10'. Splenial gyrus
11. Posterior horizontal ramus of splenial sulcus
12. Suprasplenial sulcus
12'. Suprasplenial gyrus
13. Postsplenial sulcus
13'. Postsplenial gyrus
14. Cut surface between cerebrum and brain stem
15. Lateral geniculate body
16. Superior colliculus
17. Medial geniculate body
18. Inferior colliculus
19. Arbor vitae cerebelli
20. Superior cerebellar peduncle
21. Inferior cerebellar peduncle
22. Middle cerebellar peduncle
23. Fasciculus cuneatus
24. Spinal tract of trigeminal nerve
25. Accessory cuneate nucleus
26. External arcuate fibers
27. Cochlear nuclei
28. Trapezoid body
29. Lateral lemniscus
30. Transverse fibers of pons
31. Brachium of inferior colliculus
32. Transverse peduncular tract
33. Crus cerebri
34. Left optic tract
35. Optic chiasm
36. Anterior commissure
37. Paraterminal gyrus
38. Septum pellucidum
II. Optic nerve
III. Oculomotor nerve
IV. Trochlear nerve

Figure 6. Cell-stained, parasagittal sections of (A) LGN_d of the cat (76) and (B) LGN_d of the dog (76). Different laminae are labelled: A, A_1, B and lamina magnocellularis. The histology of the dog LGN_d has been confirmed by this investigator in Nissl stained coronal and parasagittal sections.

(A)



(B)



Six dogs were used in this investigation, five experimental animals and one sham control animal. The dogs were selected on the basis of the following characteristics: a body weight between 20 - 25 kg; a mesocephalic head type; freedom from hip dysplasia and other defects; and a behavior pattern suggesting intelligence, lack of fear and trainability. Differences in breed were tolerated, although most of the dogs were in some part collie or shepherd. The dogs were purchased through, and housed in, the Department of Animal Care at the University of Oregon Health Sciences Center.

Postural Test System

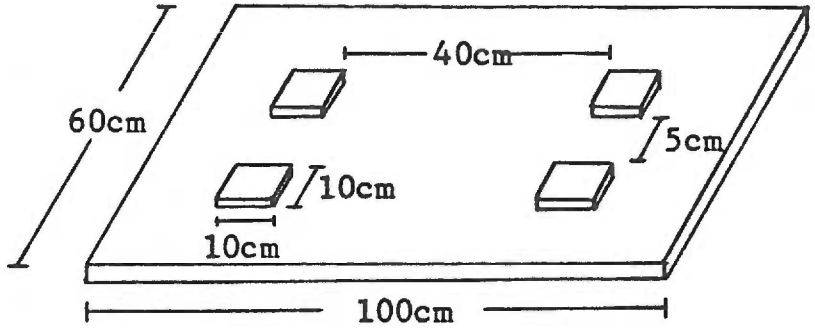
The experimental equipment on which the dogs were trained and tested was designed and assembled during previous research on the dog's postural control system. Descriptions of the techniques have been published in part elsewhere (18,19,109).

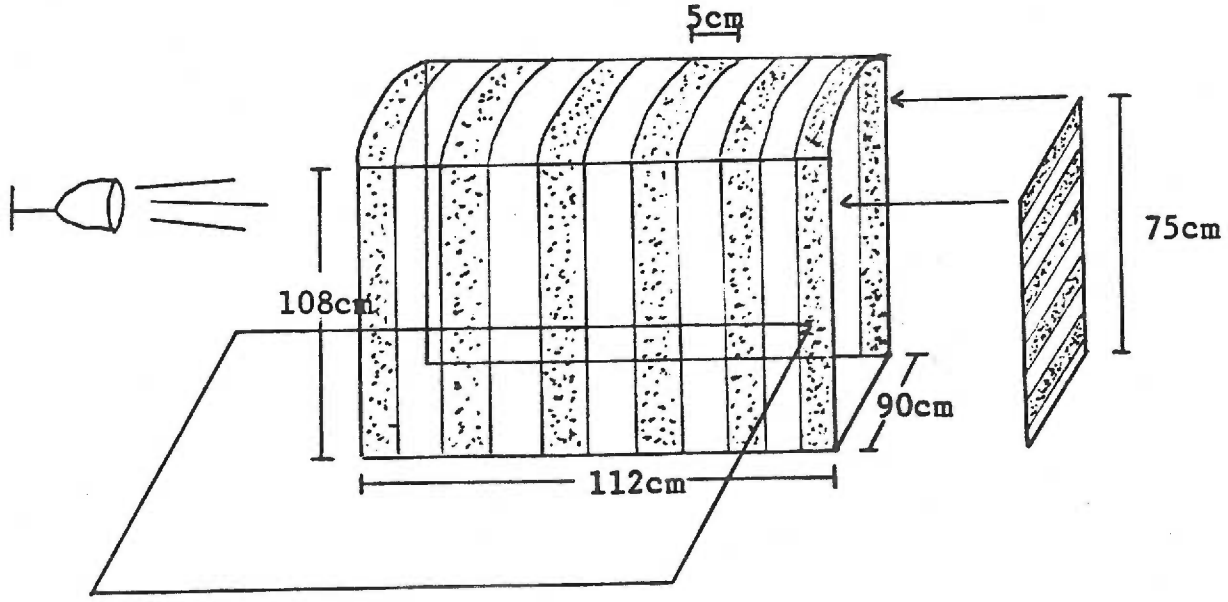
The platform on which dogs were trained to stand was hydraulically powered, electronically controlled and capable of reproducing sine wave input waveforms as sinusoidally oscillating headward-tailward movement (Fig. 7). All experiments utilized a fixed amplitude of 8 cm. peak-to-peak platform motion and covered a frequency range of platform oscillation from 0.20 Hz. to 1.73 Hz.

Vertically striped panels surrounded the dog on both sides and overhead (the "roof") and could be moved sinusoidally in a longitudinal direction, independently of the platform (Superior Electric Slow-Syn® Synchronous/Stepping Motor). The roof was easily converted into an open-ended room by the addition of a horizontally striped panel to the front of the roof (Fig. 8). The small portion of central visual field

Figure 7. Dimensions of moveable platform on which dogs were trained to stand.

Figure 8. Dimensions of moveable roof which surrounded dog on both sides and overhead. Note the front panel which converted the roof into an open-ended room when attached.


40cm
60cm
5cm
10cm
10cm
100cm


5cm
75cm
108cm
90cm
112cm

not occupied by the roof or front panel consisted of a black curtain. The only source of light was a 100 W, incandescent bulb situated 50 cm. behind the dog (Fig. 8). An assistant, whose purpose was to reward and reprimand the dog, was seated directly in front of the dog. Roof movement was fixed at an amplitude of 8 cm. peak-to-peak and varied over a frequency range from 0.15 Hz. to 0.60 Hz.

The positions of the dog, platform and roof were continuously monitored by appropriately located potentiometers. All three potentiometers were calibrated and checked weekly to maintain their output around 0 volts ($\pm$ 5 V. range). This insured linear operation of the potentiometers and linearity in the digitizing process. The platform and roof positions were derived from the feedback signals to their control amplifiers. A high-resolution potentiometer (NEJ Econopot), actuated by a lever attached to the skin midway between the cranial dorsal spines of the ilium, detected the dog's pelvis position. This parameter is a reasonably good measure of the displacement of the dog's center of gravity in the horizontal plane and is exceptionally stable and reproducible across trials in a given animal and even between different animals (19). Therefore, the pelvis position was used to quantitate the dog's behavioral response to both mechanically and visually-evoked postural perturbations.

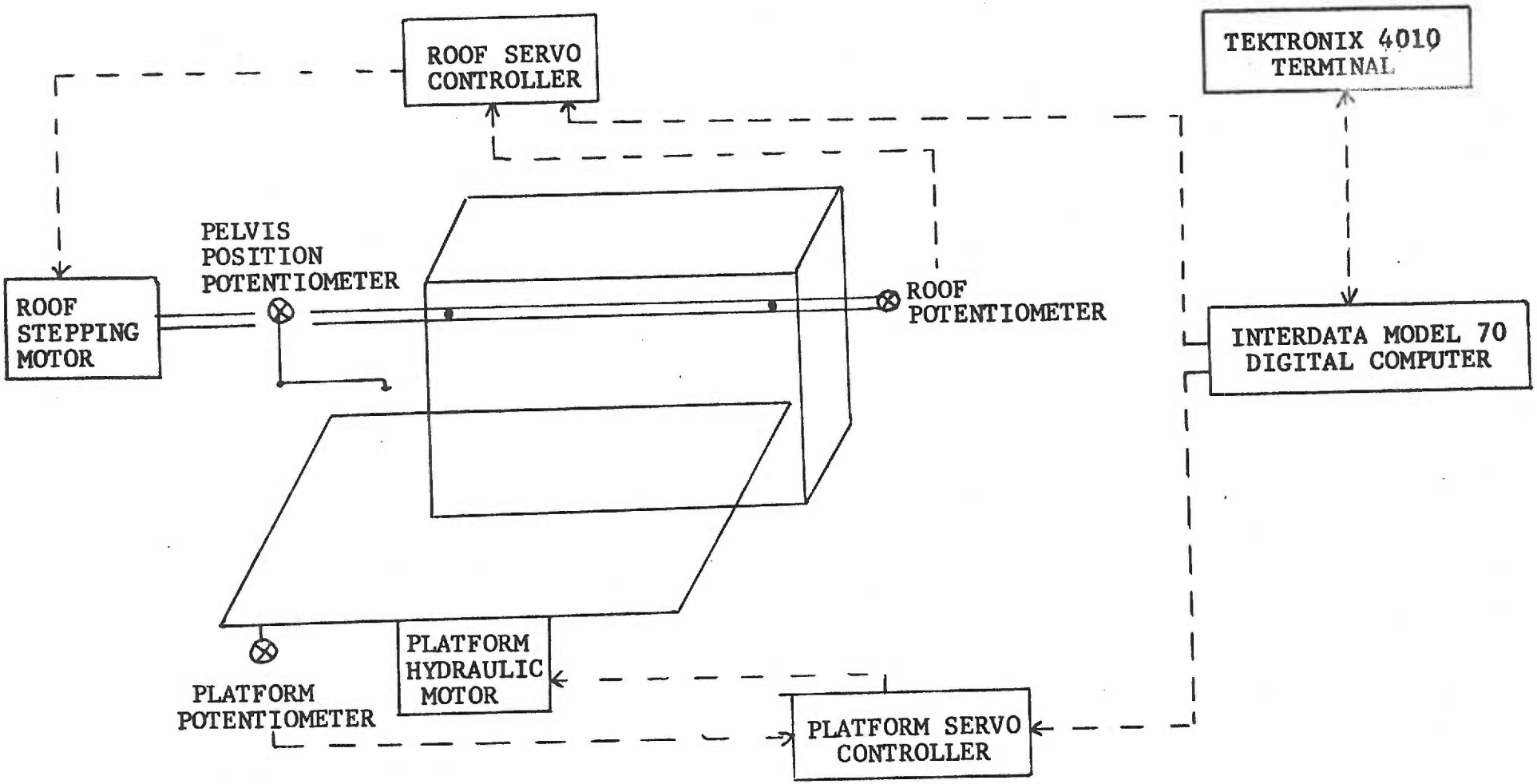
The control of platform and roof oscillation and the sampling of pelvis, roof and platform position were automated using a digital computer (Interdata Model 70). A control program permitted the experimenter to initiate either platform or roof oscillation alone, or simultaneous roof and platform oscillations, at specified frequencies.

Commands were entered by the experimenter at a computer display terminal (Tektronix 4010). Once platform and/or roof oscillation had begun, the sampling of data (i.e. platform, roof and dog's pelvis position) could be initiated by command at the terminal. Data were automatically sampled for eight cycles of platform oscillation. When simultaneous roof and platform oscillation was tested, sampling continued for eight cycles of roof oscillation, which was always at a slower frequency than platform oscillation. The experimenter had the choice of saving the data from a run (i.e. the eight cycles which were sampled), or repeating that run. The control program was then ready to initiate a second run at the command of the experimenter. Fig. 9 schematically summarizes the components of the postural test system.

Data Processing System

The continuous analog voltages from platform, roof and pelvis potentiometers were digitized by an analog-to-digital converter (Raytheon Multiverter). The eight cycles of position data were sampled 128 times per cycle, providing a phase resolution of 2.8125°. The total 1024 data points for each of the three channels were then subjected to a fast Fourier transform that yielded the first 64 sine and cosine Fourier coefficients (6). If the data from a given run were saved, the sine and cosine coefficients were written to digital tape (Kennedy 3110 digital magnetic tape recorder) from computer core. In addition, the run from which data was saved was indicated on a printout (Tally 2000 line printer) by run number, frequency and amplitude of platform and/or roof oscillation, date and dog number. The analog signals

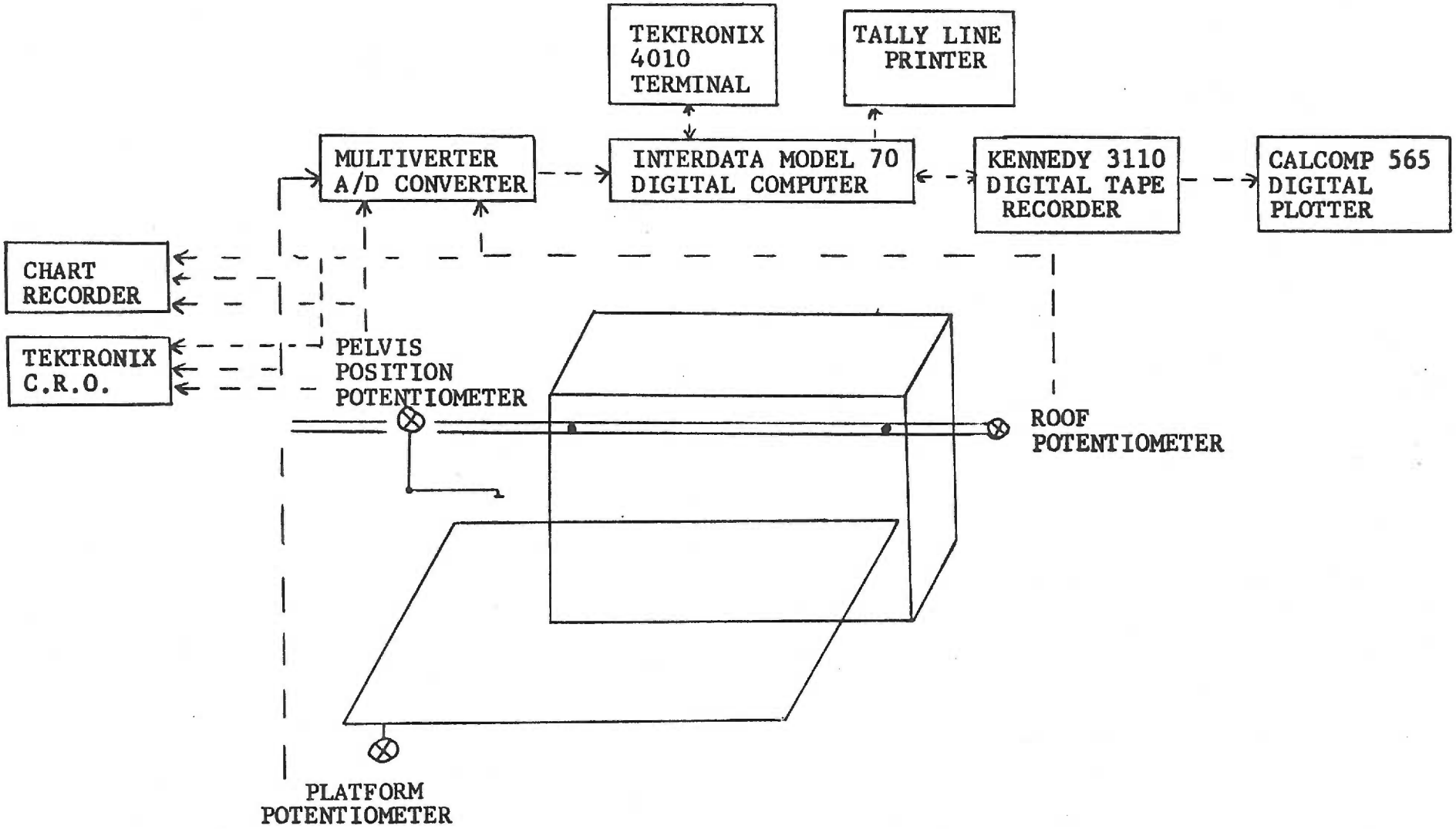
Figure 9. Block diagram summarizing postural test system. Refer to text for description of individual components.


ROOF SERVO CONTROLLER
TEKTRONIX 4010 TERMINAL
PELVIS POSITION POTENTIOMETER
ROOF STEPPING MOTOR
ROOF POTENTIOMETER
INTERDATA MODEL 70 DIGITAL COMPUTER
PLATFORM HYDRAULIC MOTOR
PLATFORM POTENTIOMETER
PLATFORM SERVO CONTROLLER

from platform, roof and dog's pelvis position potentiometers were also output to a chart recorder (Dynograph Type 504) and to an oscilloscope (Tektronix Type RM 565 dual beam). These allowed for visualization of the dog's response and the operation of platform and/or roof during the course of the experiment, and for later referral if necessary.

At the termination of an experiment, the postural test system provided on digital tape a data file consisting of the first 64 sine and cosine Fourier coefficients of pelvis position, platform position and roof position for as many runs as were saved. Several statistical computer programs were written to process and evaluate the data in a number of ways. The results of these programs were both printed out and plotted (Calcomp 565 Digital incremental plotter). Fig. 10 illustrates the components of the data processing system.

Figure 10. Block diagram summarizing data processing system. Refer to text for description of separate components.


TEKTRONIX
4010
TERMINAL
TALLY LINE
PRINTER
MULTIVERTER
A/D CONVERTER
INTERDATA MODEL 70
DIGITAL COMPUTER
KENNEDY 3110
DIGITAL TAPE
RECORDER
CALCOMP 565
DIGITAL
PLOTTER
CHART
RECORDER
TEKTRONIX
C.R.O.
PELVIS
POSITION
POTENTIOMETER
ROOF
POTENTIOMETER
PLATFORM
POTENTIOMETER

METHODS

The Training Phase

At the onset of training, all but one of the six dogs were naive regarding the laboratory and experiments. The dogs were trained utilizing predominantly positive reinforcement in the form of food reward and verbal praise. The first objective was to develop reliable maintenance of the desired position -- one of attention, with head and neck held high and a symmetric weight distribution (Fig. 11). When this was mastered, platform oscillation was begun at a full 8 cm. peak-to-peak excursion, but at a frequency of 0.10 Hz. The dogs tolerated the platform movement well, and in the course of a week were usually able to maintain the desired position at the maximal frequency of 1.73 Hz. Subsequently, roof oscillation was introduced at the full 8 cm. peak-to-peak excursion and at a frequency of 0.10 Hz. This movement in the peripheral visual field was initially disturbing to most of the dogs, but was tolerated and outwardly ignored after a week or two of repetition. The frequency of roof movement was increased to the maximal 0.60 Hz. with maintenance of trained posture after approximately two weeks of repetitive training. Having already become familiar with the disturbing movement of the roof, the dogs adapted to simultaneous platform and roof oscillation after another week of training.

Three restrictions were placed on the dogs' vision -- blindfolding, central visual field exposure only and peripheral visual field exposure only (Fig. 12). Generally, the "central-only" mask was introduced first, since it allowed the animal to see the person seated directly in front

Figure 11. Stance of a trained dog on the moveable platform showing location of the pelvis-position sensor and arrangement of independently moveable roof and platform (17).

POSTURAL TESTING APPARATUS

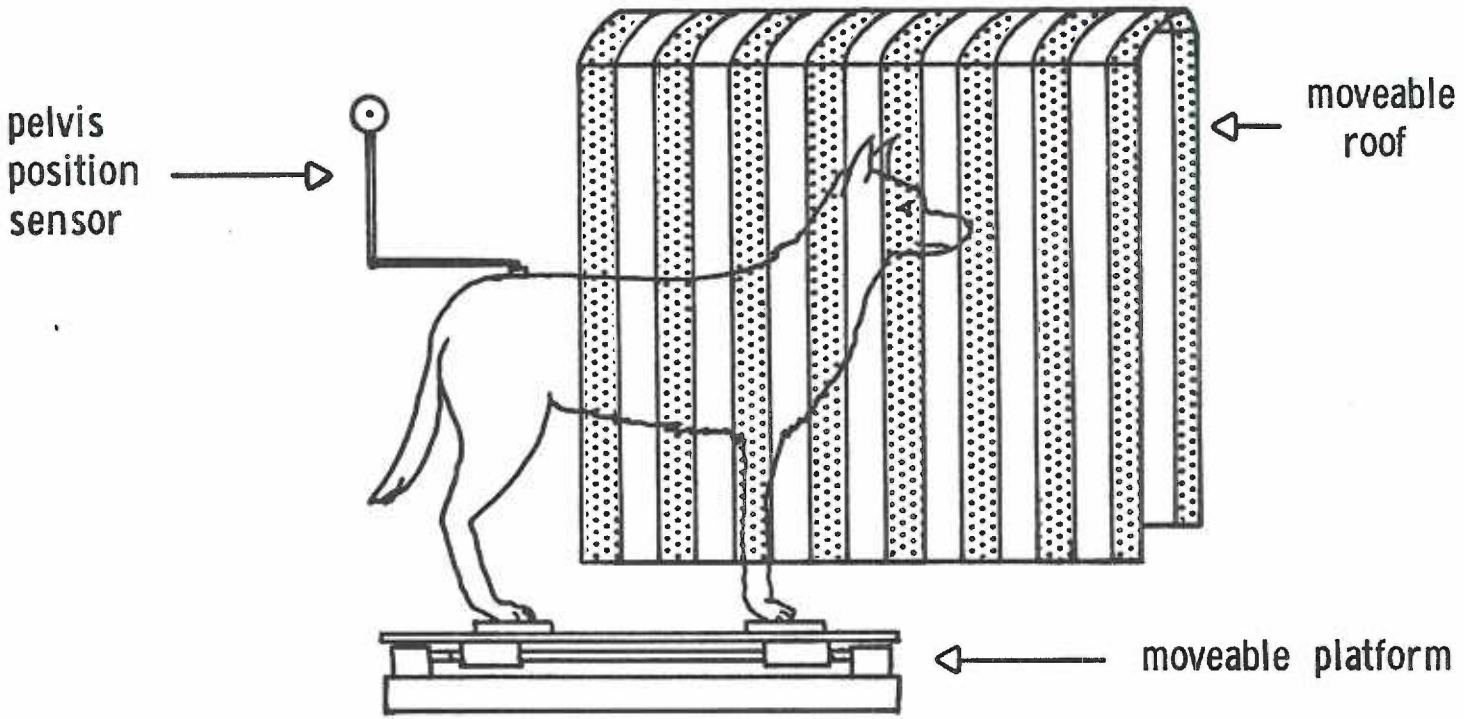
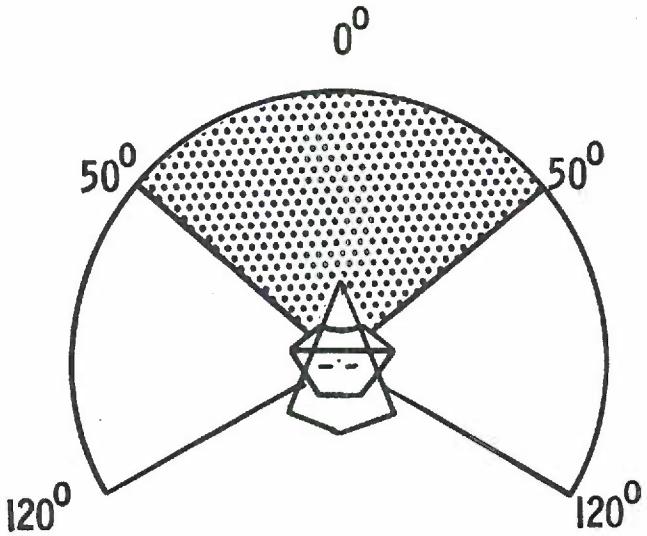
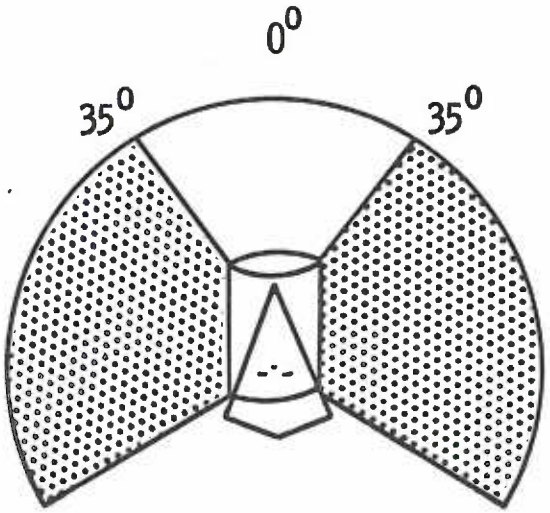


Figure 12. Schematic of three types of masks which were secured to the dogs' heads in order to restrict visual field exposure.

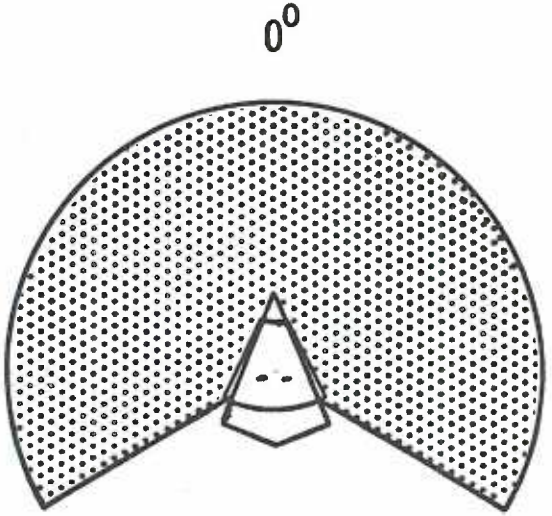
RESTRICTION OF VISUAL FEEDBACK



peripheral
visual field
only



central
visual field
only



blindfold

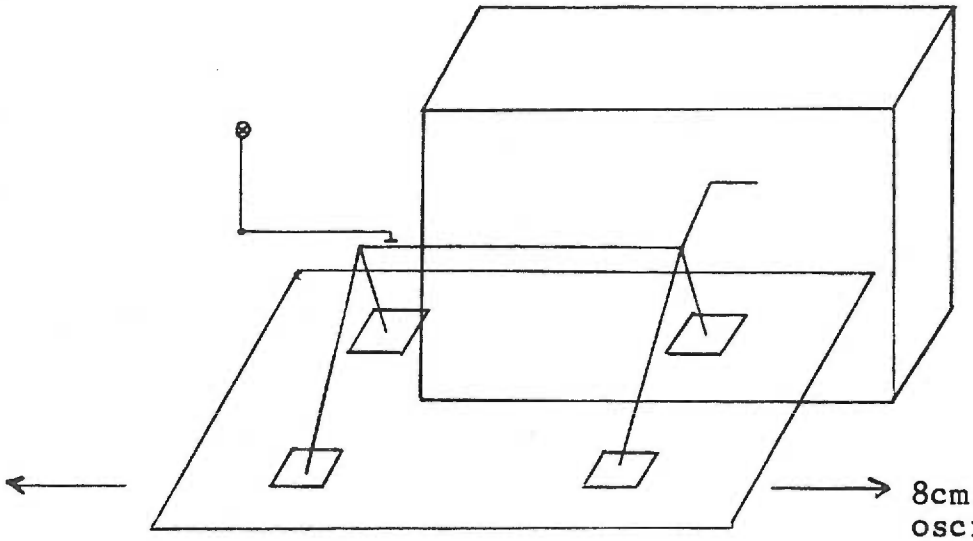
of him and produced the least anxiety. Stable performance during platform oscillation was achieved quickly. The "peripheral-only mask" was presented next. During blocking of the central visual field, the dog's only visual input consisted of the striped roof in the peripheral visual field. This evoked some anxiety, but after one to two weeks of repetitive training the desired position was properly maintained during both platform oscillation alone and simultaneous platform and roof oscillation. With the dog already trained relatively well to all of the above conditions, the "blindfold" was quickly accepted. The entire training phase varied in duration for different dogs, ranging from one to three months for its satisfactory completion.

Quantitative Evaluation of Postural Control During Platform Perturbation -- Platform Experiments

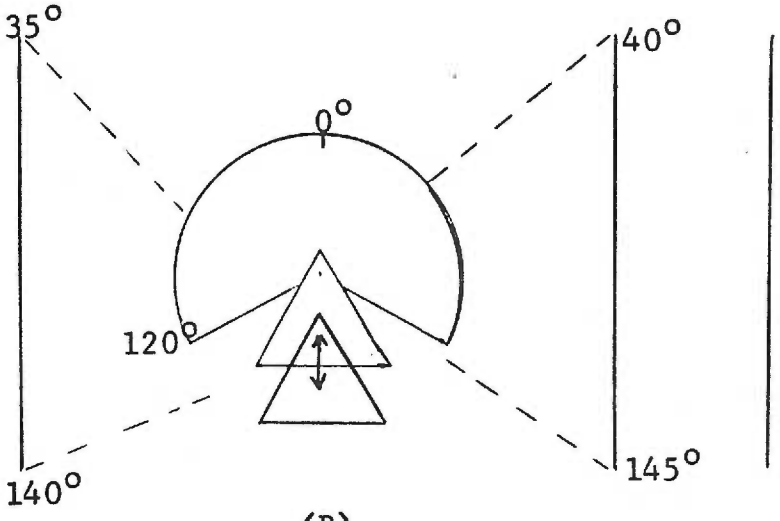
This first group of experiments involved perturbation of the dog's posture by oscillation of the supporting platform. The visual surround remained stationary and, therefore, provided accurate visual feedback concerning the postural problem. Fig. 13A illustrates the postural task presented to the dog.

The experimental procedure involved platform oscillation at seven frequencies -- 0.20, 0.30, 0.43, 0.58, 0.86, 1.15 and 1.73 Hz. Data sampling was begun between three to ten cycles after the onset of platform oscillation, depending on platform frequency. During this interval the dog's body position normally reflected an initial startle response, followed by a period of some instability for less than two cycles, before tightly phase-locked, cyclic changes in body position ensued. At that point it was assumed that the postural control system was

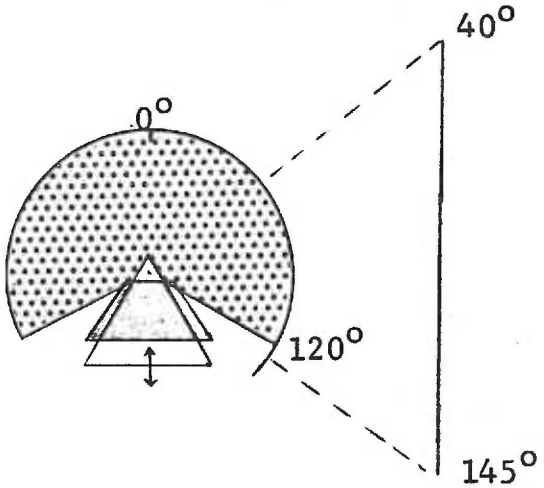
Figure 13. Schematic description of four different types of platform experiments. (A) Illustration of postural control problem posed to the dog, (B) NP experiment (Note, the angles on the far right side indicate angles in the visual field occupied by the edges of the roof when the platform is in the full headward position; the angles on the far left side indicate the position of the roof edges after the platform has moved to the full tailward position. The portion of visual field occupied by the roof fluctuates between these two sets of angles during sinusoidal oscillation of the platform in all four experiments.), (C) BP experiment (Note, the complete masking of visual field is indicated by shading.), (D) POP experiment (Note, vision is restricted to lateral visual fields beyond 50°.), (E) COP experiment (Note, only the central 70° of visual field is visible.). See text for details.


8cm. sinusoidal
oscillation at 7
different frequencies

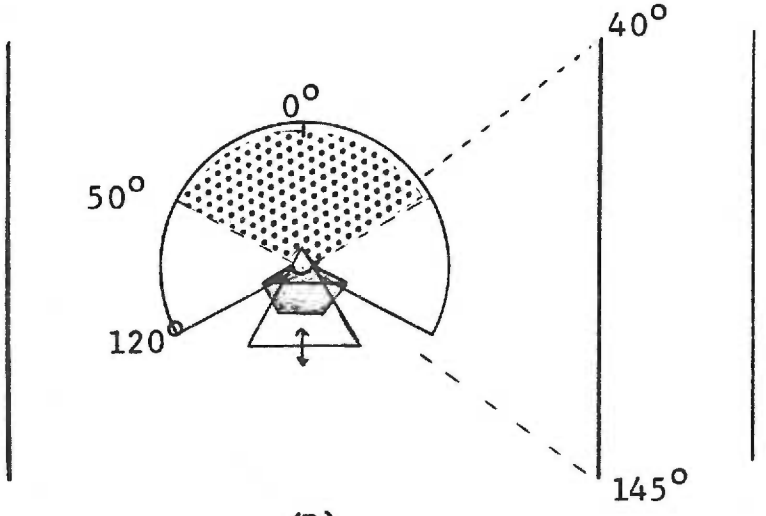
(A)


35°
40°
0°
120°
140°
145°

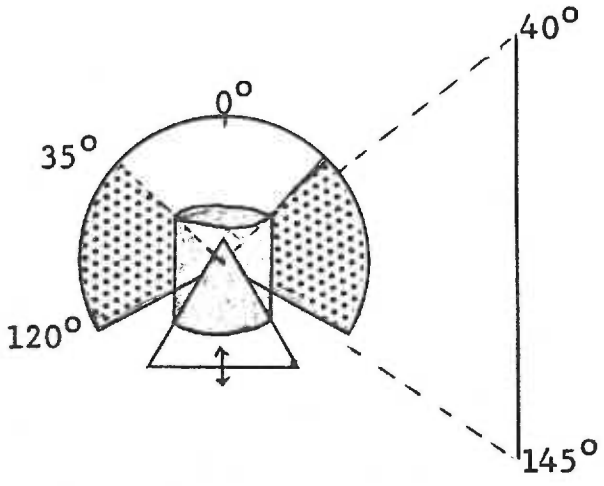
(B)


40°
0°
120°
145°

(C)


40°
0°
50°
120°
145°

(D)


40°
0°
35°
120°
145°

(E)

operating in a stationary state, evidenced by the fact that over eight cycles of platform motion cyclic changes in body position varied little in amplitude and phase and demonstrated no transient changes. Five runs were repeated at each frequency so that the data file for the experiment contained the first 64 harmonic Fourier sine and cosine coefficients of platform, roof and pelvis position for 35 runs. After each run, the dog was rewarded verbally and with a food reward. If the dog made any spurious movements (e.g. head turning, tail wagging, coughing or weight redistribution), detected either by the assistant directly or the experimenter monitoring the oscilloscope and polygraph records, the run was immediately aborted and the dog verbally reprimanded and denied food reward. The typical platform experiment required approximately 30 minutes for completion. All of the platform experiments were repeated at least biweekly, both pre- and postoperatively, until stable interday response patterns resulted. The data from five days of stable responses to platform experiments were then combined to reflect both inter-run and interday variability of the output of the postural control system.

This experimental plan was repeated under four visual conditions:

(1) Normal visual field exposure, i.e. normal platform, NP, experiment;

(2) Blindfolded, i.e. blindfolded platform, BP, experiment;

(3) Only central visual field exposure, i.e. central-only platform, COP experiment; and

(4) Only peripheral visual field exposure, i.e. peripheral-only platform, POP, experiment.

Fig. 13B-E schematically defines the four types of platform experiments and illustrates the masks used to accomplish each visual restriction.

The portion of visual field occupied by the roof during platform experiments varies regularly as platform oscillation is occurring. At rest, the roof extends from 40° to 145° bilaterally. As the platform moves tailward 8 cm., the area of visual field occupied by the striped roof moves centrally to occupy from 35° to 140° bilaterally. The leading edge of the roof, then, varies sinusoidally from 35° to 40° during ongoing platform oscillation. The overhead leading edge of the roof occurs at 40° to 50° in the visual field, depending on the height of the individual dog. This angle would likewise fluctuate 5° during platform oscillation. The cyclic change in exposure of the roof in the visual field is important as it relates to effectiveness of the masks in restricting visual field exposure. The NP and BP experiments are not influenced, but the COP and POP experiments could potentially be affected. The mask restricting vision to only peripheral visual field blocks out the central 100° and all visual field overhead. Thus, the fluctuation from 35° to 40° of the leading roof edge is of no consequence, and visual feedback is restricted completely to the striped roof in the periphery. The mask which restricts vision to only central visual field permits vision only in the central 70° of visual field. As a result, when the platform moves tailward, the leading roof edge approaches the 35° limit of visual field bilaterally. Exposure of the leading roof edge within the field of vision would, however, depend on some turning of the head on the part of the dog, would be unilateral if occurring at all, and would, therefore, result in discard of that run.

Fig. 13 diagrams the visual field dimensions, showing the areas restricted and the changing angles resulting from platform movement.

Quantitative Evaluation of Visually-induced Postural Responses -- Platform-roof Experiments

Visually-induced postural responses were studied by oscillating the peripheral visual field alone or the central and peripheral visual fieldssimultaneously.

Three conditions were tested in the platform-roof experiments without the attachment of the front panel:

(1) Normal visual field exposure, i.e. normal both platform and roof, NB, experiment;

(2) Peripheral visual field exposure only, i.e. peripheral only, both, POB, experiment; and

(3) Blindfolded, i.e. blindfolded, both, BB, experiment.

Initially, peripheral visual field movement produced by sinusoidal oscillation of the vertically-striped roof was presented with the platform stationary. However, the adjustments in body position that were evoked averaged less than a millimeter in magnitude and demonstrated large inter-run variability and intolerable interday variability. For these reasons, a different experimental procedure was adopted based on observations made by Talbott. It was observed that amplitude of postural responses evoked by roof movement was increased nearly ten-fold by simultaneous oscillation of the platform at some greater, unrelated frequency (109a). Asynchronous sinusoidal roof and platform movements were, therefore, paired. The frequency ratio of platform to roof oscillation was fixed at 2.875, with the platform frequency being higher

in all cases; both platform and roof moved 8 cm. peak-to-peak (in the course of the experiment) at five different frequency combinations:

Roof	0.15	0.20	0.30	0.40	0.60	Hz.
Platform	0.43	0.58	0.86	1.15	1.73	Hz.

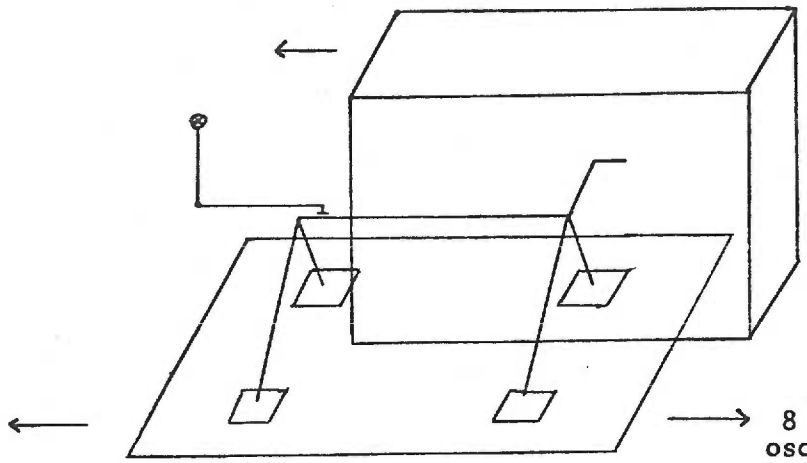
Each frequency combination was repeated for five runs, resulting in a data file that contained the first 64 harmonic Fourier sine and cosine coefficients of platform, roof and pelvis position for a total of 25 runs. After each successful run, the dog was rewarded both verbally and by presentation of a food reward by the assistant seated in front of the dog. Fig. 14 defines the three platform-roof experiments and illustrates the experimental apparatus. As was the case in the platform experiments, the platform-roof experiments were repeated daily until stable response patterns occurred. Subsequently, five days' data were collected and combined to reflect the normal variability present in the postural control system.

One visual condition was tested when the front panel was attached to the roof:

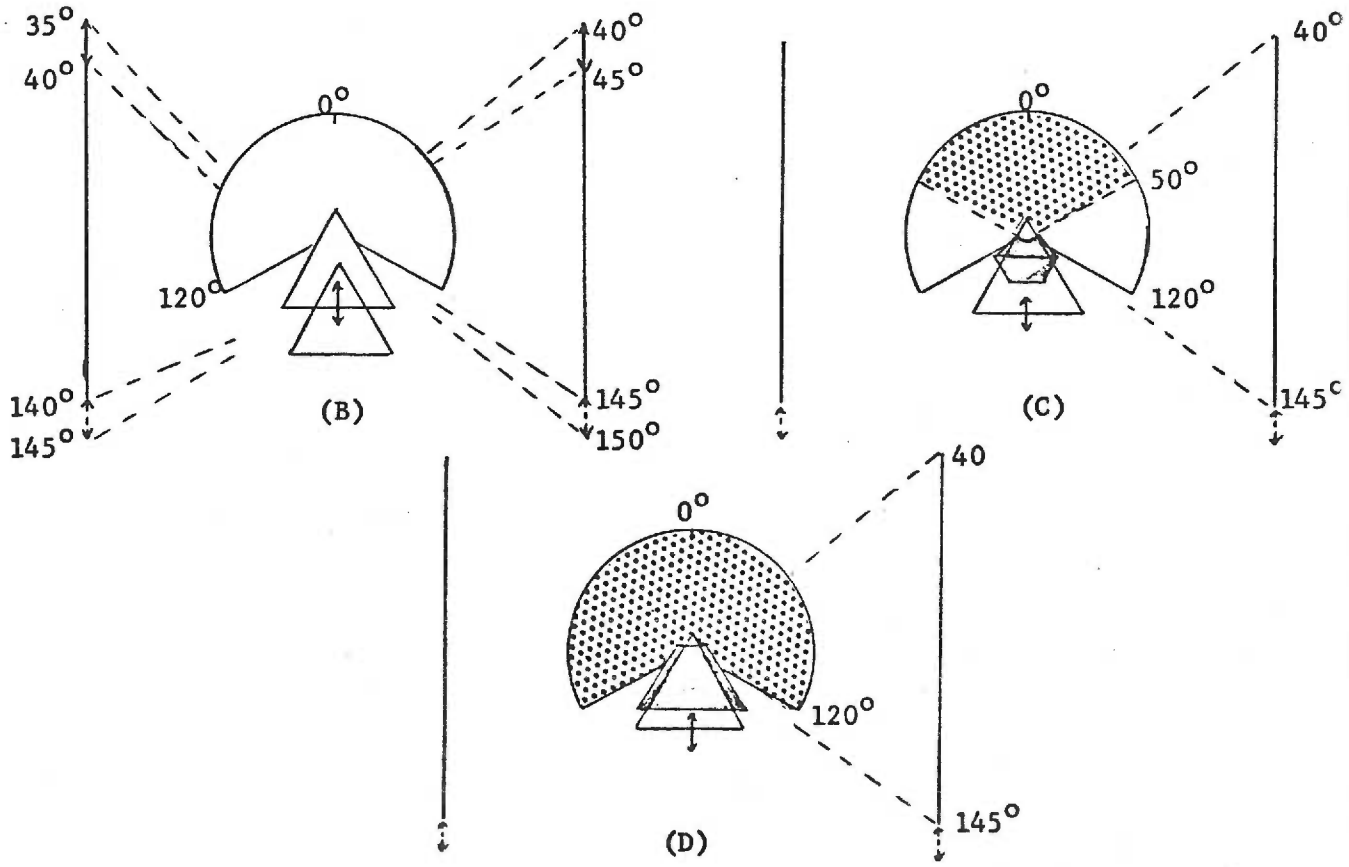
(1) Central visual field exposure only, i.e. central only, both platform and roof with front panel, COB', experiment.

It should be noted that this experiment with front panel attached was neither repeated prior to testing to familiarize the dog with its conditions, nor multiply tested, as were the platform and the other platform-roof experiments. Instead, the COB' experiment was performed just once pre-operatively and once post-operatively (one to three months later) and, therefore, represented novel conditions to the animal. The conditions of the other two groups of experiments became very familiar

Figure 14. Schematic description of three different types of platform-roof experiments. (A) Illustration of experimental apparatus, (B) NB experiment (Note, the position of the leading and trailing edges of the roof in the visual field is given for four extreme positions. When the platform is full forward, the roof oscillates between the visual angles given on the right side; while, when the platform is full backward, the roof oscillates over the angles given on the left side. Therefore, in the combined roof-platform motion, the headward edge of the roof fluctuates between 35°-45°, while the tailward edge varies between 140° and 150°. These limits apply to all three platform-roof experiments.), (C) POB experiment (Note, visual field exposure is limited to greater than 50° bilaterally. Therefore, the leading edge of the roof is never visible to the dog.), (D) BB experiment (Note, although all vision is masked, the roof continues to be oscillated to evaluate use of auditory cues.).


8 cm. sinusoidal oscillation (8th harmonic) at 5 frequencies
8 cm. sinusoidal oscillation (23rd harmonic) at 5 frequencies

(A)


35°
40°
0°
120°
140°
145°
(B)
40°
45°
145°
150°
0°
50°
120°
40°
145°
(C)
0°
120°
40
145°
(D)

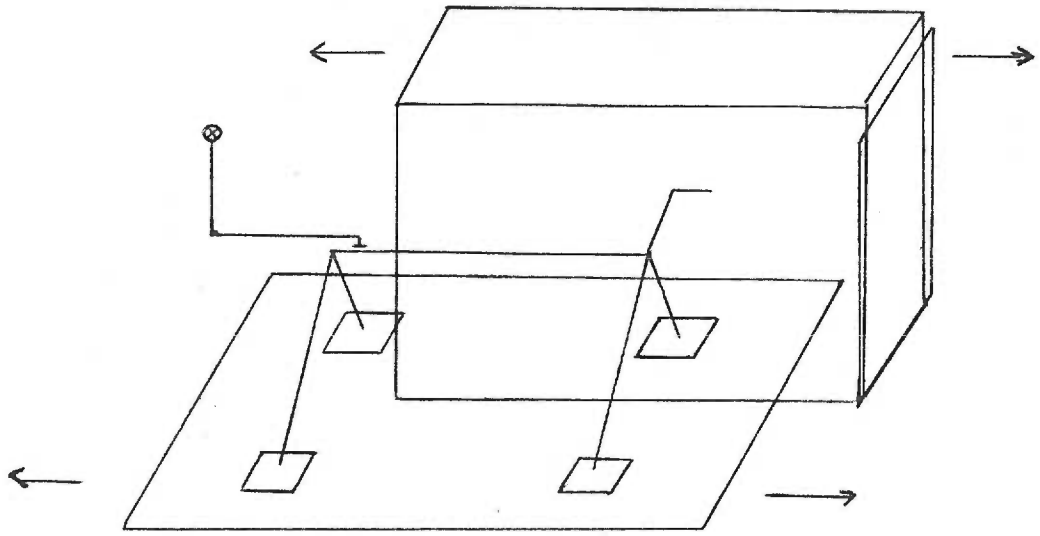
and predictable to the animal after repeated training and experimental trials.

Otherwise, the COB' experiment followed the same procedure as other platform-roof experiments: 8 cm. sinusoidal perturbation of both the roof and platform at the same five frequency combinations; the roof always at the lower frequency and the platform at the higher frequency; and, five successful runs repeated at each frequency. Fig. 15 schematizes the COB' experiment and the experimental apparatus.

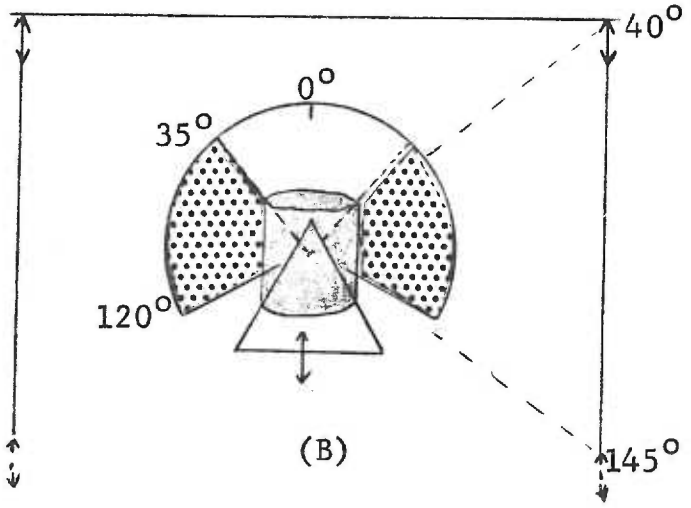
Qualitative Evaluation of Visually-guided Behaviors

Two qualitative approaches were taken to assess the dogs' visually-guided behaviors pre- and post-operatively. Observations and various clinical tests of visually-guided behavior were made throughout the study. In addition, the limits of the visual field were tested both pre- and post-operatively. Clinical evaluation of visually-guided behavior was performed twice pre-operatively and every day post-operatively until stable behaviors occurred. The observations included the following: visual following of a food stimulus by movement of eyes, head and body, tested horizontally, vertically and in a circle around the animal; gross visual orienting responses, tested by moving a second food stimulus in the visual field while the dog fixates a stationary food stimulus centrally; visual placing as manifested by "step and stair" ascent and descent performance; general visually-guided behavior including avoidance of obstacles, jumping, reaching for food with paws and head, and catching tossed food; and, eye blink to a visual threat versus tactile stimulation of eye lash or cornea.

Figure 15. Schematic description of the COB' experiment illustrating (A) experimental apparatus modified for testing central visual field, (B) COB' experiment (Note, the exposure of visual field is restricted to the central 70°; also, the leading edge of the roof is located between 35°- 45° in the visual field as simultaneous platform and roof oscillation occurs. Therefore, any visual input from the vertically-striped roof would necessitate the dog turning its head, which normally was grounds for discarding that particular run).



(A)



(B)

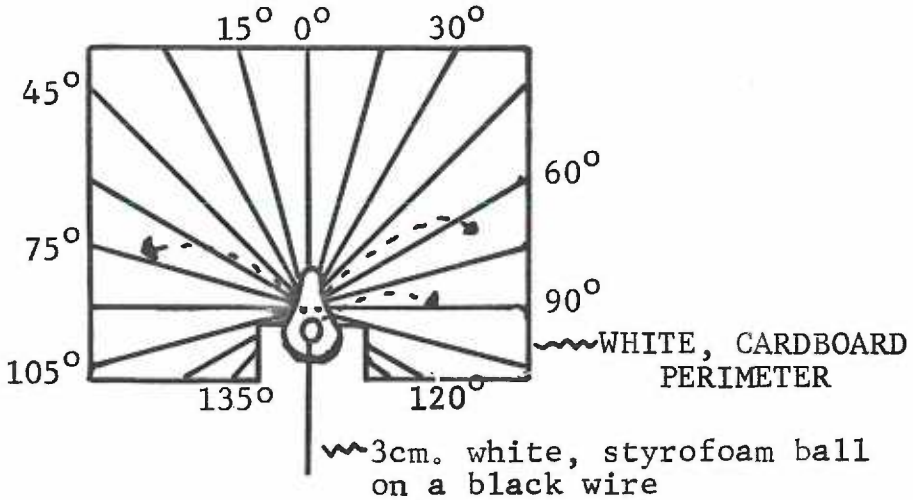
The orienting response to a 3 cm. white ball was tested bilaterally at 15° increments from 0° to 135° in the horizontal visual field, using a perimeter marked off with intersecting guidelines every 15°. The surround was draped with a black curtain, and the perimeter located under the dog's head was lighted from overhead. As the dog was fixating at 0° on a piece of food (the "fixation object"), the white ball (the "novel stimulus") was introduced from directly behind the dog's head and moved along a particular guideline to that prescribed angle at the dog's eye level (Fig. 16). A movement of the eyes and/or head toward the novel stimulus was considered a positive response. No eye or head movement during approximately a three-second presentation of the novel stimulus was recorded as a negative response. A movement of the eyes or head to the side opposite the ball was also recorded as a negative response. Twenty trials were repeated at each angle, and forty control trials were interspersed throughout, both pre- and post-operatively. The control trials consisted of a random three-second interval when no ball was presented, but any eye or head movements away from 0° were noted. Thus, the percentage of positive responses at each angle was calculated and compared with the background percentage of random positive responses in order to determine the lateral limits of the dog's visual field. In addition, comparison of pre- and post-operative responses reflected possible selective visual field deficits. Fig. 16 schematizes the method of testing.

Ablation of Visual Cortex

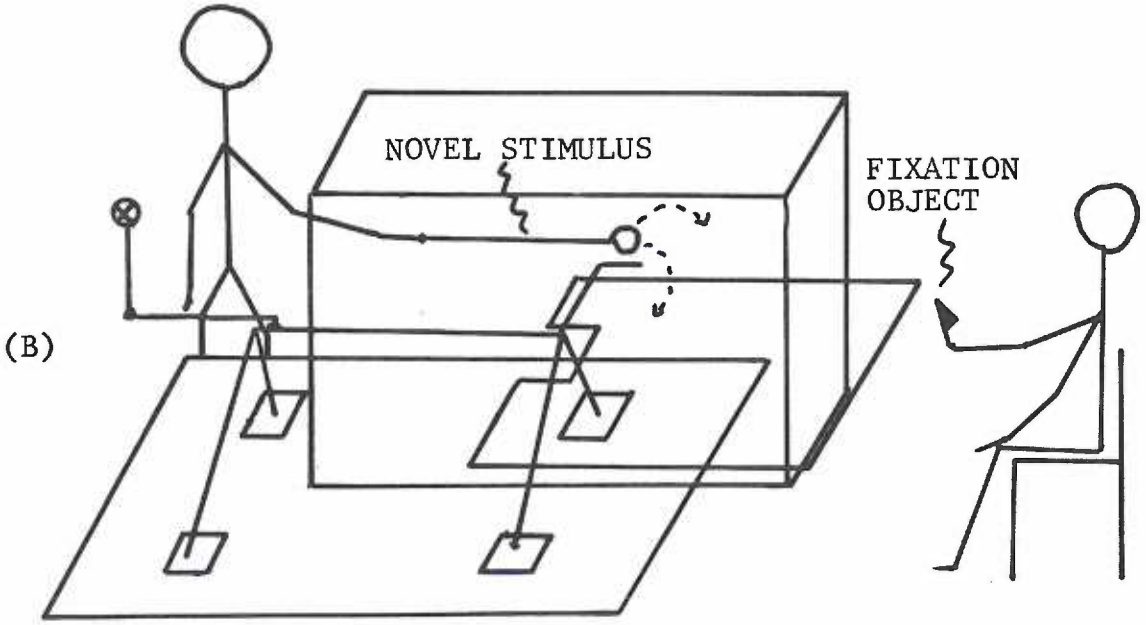
Upon completion of pre-operative experiments quantifying the dog's

Figure 16. The testing of orienting responses to all areas of visual field and of the limits of lateral visual field. (A) Overhead view of dog's head and perimetry board, (B) overall view of dog on platform surrounded by the roof, which is draped in black. See text for description of testing procedure.

(A)


15° 0° 30°
45°
60°
75°
90°
105°
135°
120°
WHITE, CARDBOARD PERIMETER
3cm. white, styrofoam ball on a black wire

(B)


NOVEL STIMULUS
FIXATION OBJECT

postural control responses and describing visually-guided behaviors, sterile surgery to chronically ablate all of areas 17 and 18 was performed. Evoked potential experiments mentioned earlier (Fig. 5) that were performed by this investigator provide approximate limits to the primary cortical projection area. These data on dogs, plus histologic evidence in the literature on dog and cat visual cortex (Fig. 2 and 4), delineate the dog's primary visual cortex (i.e. areas 17 and 18) as the middle and posterior entolateral, lateral, postlateral, suprasplenial and splenial gyri. It appears that the primary visual cortex extends posteriorly, ventrally and medially along the tentorium cerebelli to the splenial sulcus. The surgical objective was to remove all of the primary visual cortex.

Common aseptic procedures were followed in preparing for a surgical approach to the dorsal surface of cerebral cortex. Sodium pentobarbital (30 mg/kg) was administered intravenously to induce deep, long-lasting anesthesia. The aseptic field extended to postorbital processes anteriorly, to the base of the neck posteriorly, and to the ears bilaterally. A midline skin incision extending from postorbital processes to the base of the skull was, initially in the study, made with an electrocautery (first three dogs) and then, with a scalpel (last three dogs). Epidermal necrosis and subcutaneous infections were common post-operatively when electrocautery was used for the incision, thereby prompting the change to the scalpel. Platysma and auricularis muscles were divided along the midline using electrocautery to minimize bleeding. The tendon of the temporalis muscle was incised also along the midline, and the temporalis muscle scraped free

from the parietal bone, external sagittal crest and interparietal process. All of the muscles and skin were then laterally retracted, exposing the full extent of the parietal bone bilaterally. A trephine was used bilaterally to remove a 1.5 cm. button of parietal bone located approximately 1 cm. lateral to the midline and 1 cm. anterior to the occipital crests. Bone wax was utilized to stop bleeding from between the two tables of parietal bone, as the cortical exposure was extended anteriorly and medially with bone rongeurs. The final bilateral brain exposures approximated 4 cm. anteroposteriorly and 3 cm. laterally. Dura and arachnoid mater were incised longitudinally to expose the pia mater. The one sham-operated control dog then had the dura mater flaps approximated and the muscles and skin sutured. There was no deliberate intrusion into pia mater. The purpose of the sham operation was to determine if the trauma of surgery, excluding only the ablation of brain tissue, had any quantitative effects on the trained postural responses.

In the five experimental dogs, a long nose, narrow bore (1-2 mm. diameter) glass pipette was used subpially under high suction (10-15 inches of water) to withdraw brain tissue. The degree of hemorrhage varied widely as a function both of the extent of pial trauma and of individual variations in cortical vasculature. Gelfoam and direct pressure were used to induce hemostasis. The two flaps of dura mater were approximated with gut suture. The temporalis and superficial muscles were sutured with a synthetic, absorbable suture (Vicryl®). The skin incision was closed with nylon suture. Penicillin was administered intramuscularly immediately upon completion, and repeated

for two to three days.

After awakening, the animals were observed every few hours for clinical signs of any visual deficits. Post-operative experiments were begun as early as the dogs were capable, which varied from two to six days post-operatively. The same quantitative and qualitative tests described previously were repeated post-operatively.

Evaluation of Surgical Lesions

All six dogs were sacrificed by an intravenously administered overdose of anesthetic (Beuthanasia@). Immediately thereafter, the dogs were prepared for transcardiac perfusion of the brain. The sternum was split longitudinally, the rib cage spread and the pericardium exposed. The aortic arch, descending aorta, right atrium and left ventricle were isolated, and the following steps quickly performed: descending aorta clamped, aortic arch palpated, left ventricle incised at the apex, right atrium incised widely, and a cannula inserted through the left ventricle into the aortic arch. First, two liters of normal saline were infused, followed by two liters of formalin (10% formaldehyde). Within four hours the brains were removed from the skull and stored in formalin.

After the brain tissue was well fixed, the approximate extent of the lesions was determined by gross observation and by photographing the brains both before and after blocking for histologic examination. Coronal sections (40 micron thickness) of the posterior half of the cerebral cortex were prepared, to identify more precisely the extent of the lesions. Staining of every 25th section with cresylecht violet

(Nissl stain), permitted serial reconstruction of the cortical ablation by tracing of the projected sections.

Coronal sections (40 micron thickness) of the midbrain and diencephalon were also prepared and stained with cresylecht violet. Microscopic examination of every 10th section allowed for evaluation of the extent of retrograde atrophy in the LGN_d(lam) resulting from anatomical injury to the axons of geniculate neurons in the visual cortex. Based on this information, comparisons could be made between dogs as to the absolute and relative extent of removal of areas 17 and 18.

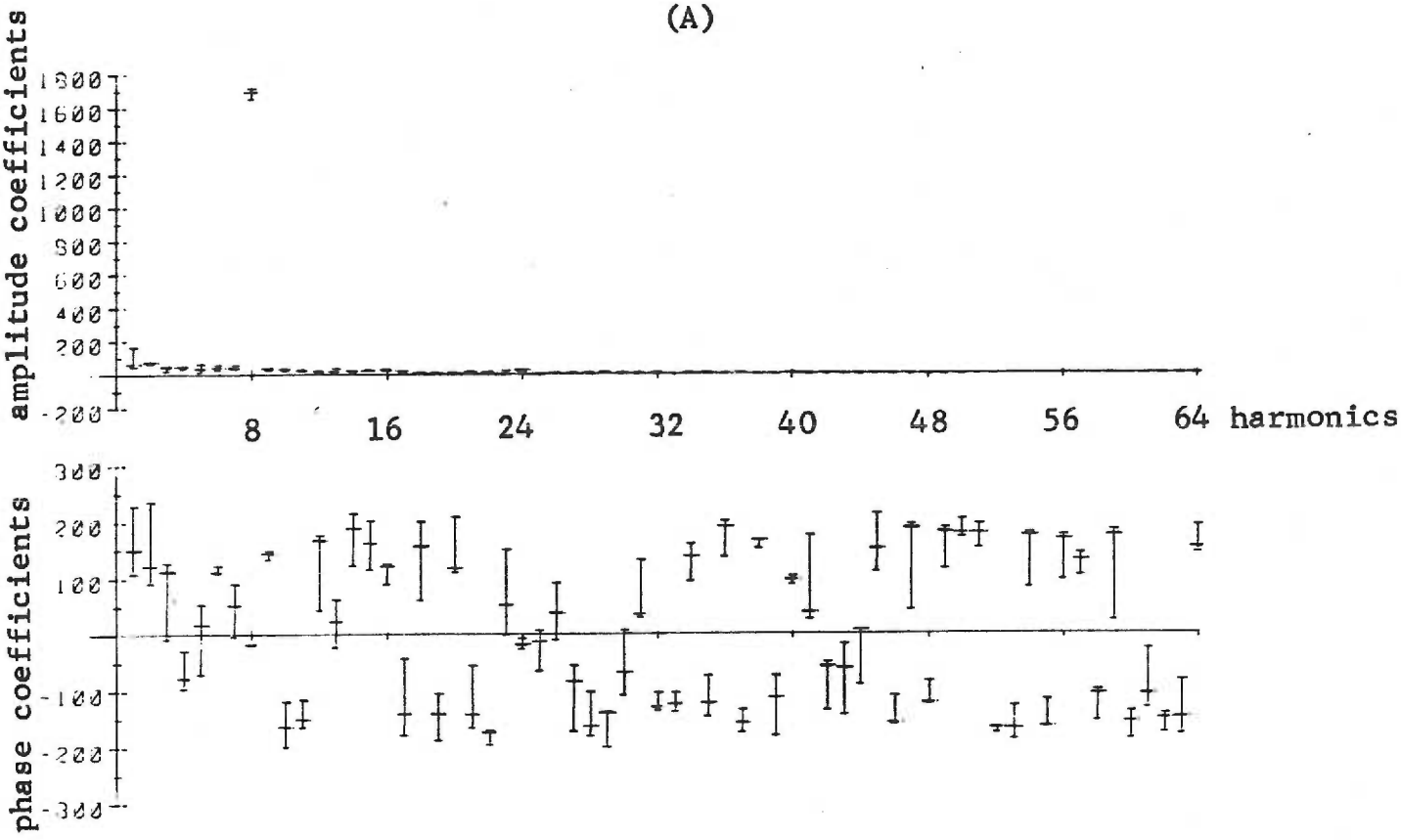
Evaluation of Data

The final data files consisted of the first 64 harmonic Fourier sine and cosine coefficients of platform, roof and pelvis position that were collected from the quantitative testing of the postural response to platform perturbation or combined platform-roof perturbation described above. Several methods were used in summarizing and testing these data.

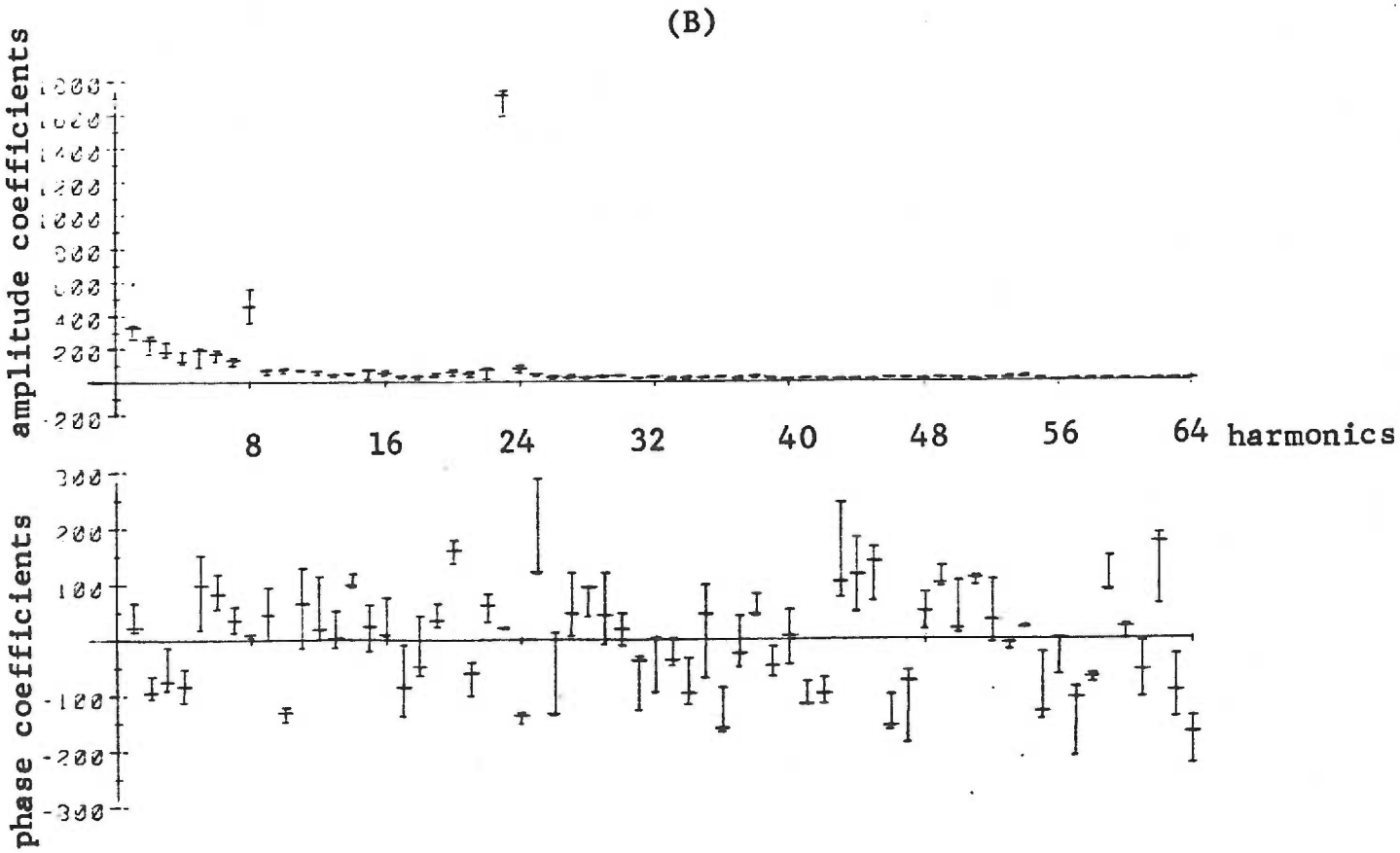
First, Fourier amplitude and phase coefficients were calculated from the sine and cosine coefficients for each of the 64 harmonics. Fig. 17 plots these first 64 amplitude and phase coefficients for five runs repeated at two frequencies or frequency combinations from a platform, NP, and platform-roof, POB, experiment. In the platform-roof experiments, the eighth Fourier coefficients represent the changes in body position synchronized to the roof input frequency, while the 23rd Fourier coefficients describe the changes in body position synchronized to the platform input frequency. For the platform-roof experiments,

Figure 17. Relative values of the first 64 amplitude and phase Fourier coefficients from an (A) NP experiment (0.20 Hz), and (B) POB experiment (0.15/0.43 Hz).

(A)


amplitude coefficients
1800
1600
1400
1200
1000
800
600
400
200
-200
8
16
24
32
40
48
56
64 harmonics
phase coefficients
300
200
100
-100
-200
-300

(B)

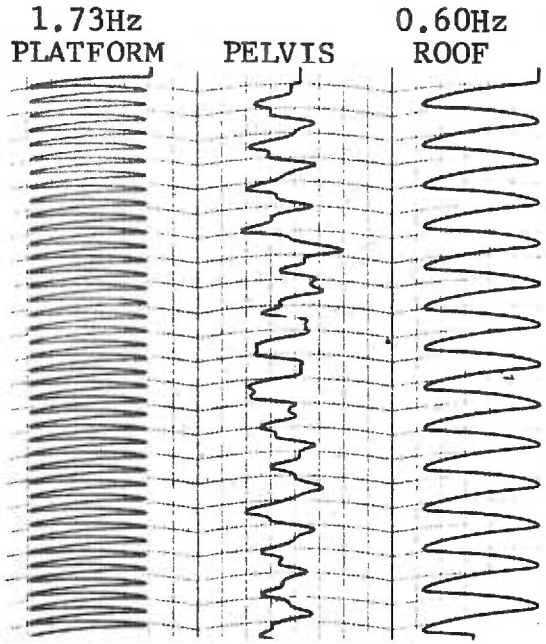

amplitude coefficients
8
16
24
32
40
48
56
64 harmonics
phase coefficients
300
200
100
-100
-200
-300

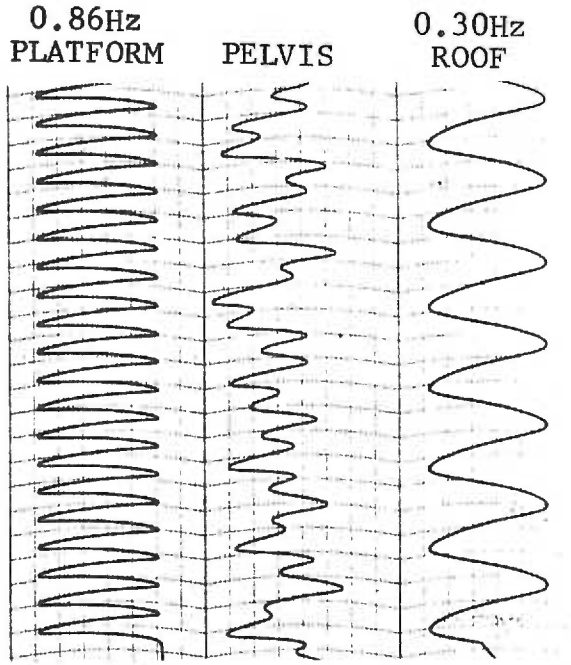
the eighth harmonic will be termed the "roof forcing frequency" and the 23rd harmonic the "platform forcing frequency." The effects of platform and roof forcing frequencies at these two harmonics was predictable since the sampling period of body position data was always eight cycles of roof oscillation, during which time the platform traversed eight times 2.875 or 23 cycles.

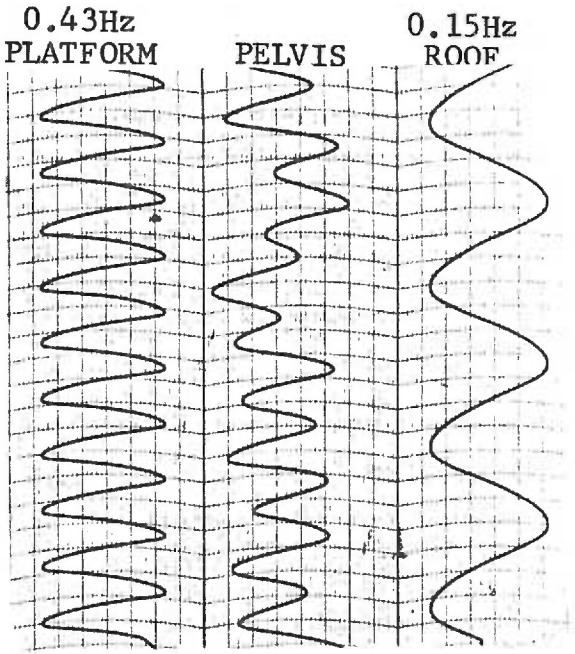
It is readily apparent from Fig. 17B that the dog's response to this combined platform-roof perturbation occurred largely at the platform and roof forcing frequencies, with some contribution from the amplitude coefficients of subharmonics one to eight, but almost no response at higher harmonics (i.e. second and third harmonics of the forcing frequencies = 16th and 24th, or 46th). The phase coefficients demonstrate the least variability at the two forcing frequencies, suggesting a stable, phase-locked response pattern. Non-forcing frequency phase coefficients, both subharmonics and higher harmonics, possess much larger variability and reflect small shifts in body position occurring randomly with respect to platform and roof movement ("noise").

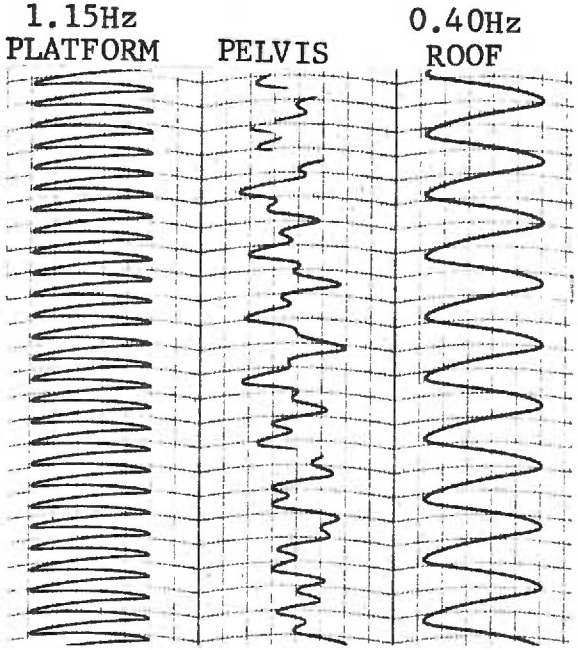
The above conclusions are supported by observations of the raw analog records, Fig. 18. It is apparent that the dog's movement was essentially sinusoidal and frequency-related to both the platform oscillation and the slower roof oscillation. The first 64 harmonic Fourier amplitude and phase coefficients for the platform experiment are similar to those for the platform-roof experiment, except that only the platform's forcing frequency is present in the dog's response, represented by the eighth harmonic (Fig. 17A). This is also evident from the raw data reproduced in Fig. 19. Therefore, based on the

Figure 18. Analog voltage records from roof, pelvis and platform position potentiometers in a POB experiment. Frequencies of platform and roof oscillation are given to the left of each record.


1.73Hz
PLATFORM
PELVIS
0.60Hz
ROOF


0.86Hz
PLATFORM
PELVIS
0.30Hz
ROOF


0.43Hz
PLATFORM
PELVIS
0.15Hz
ROOF


1.15Hz
PLATFORM
PELVIS
0.40Hz
ROOF

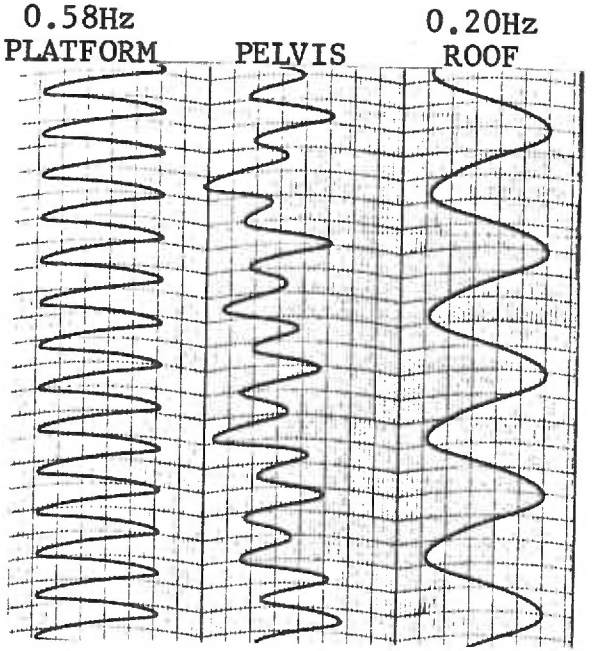
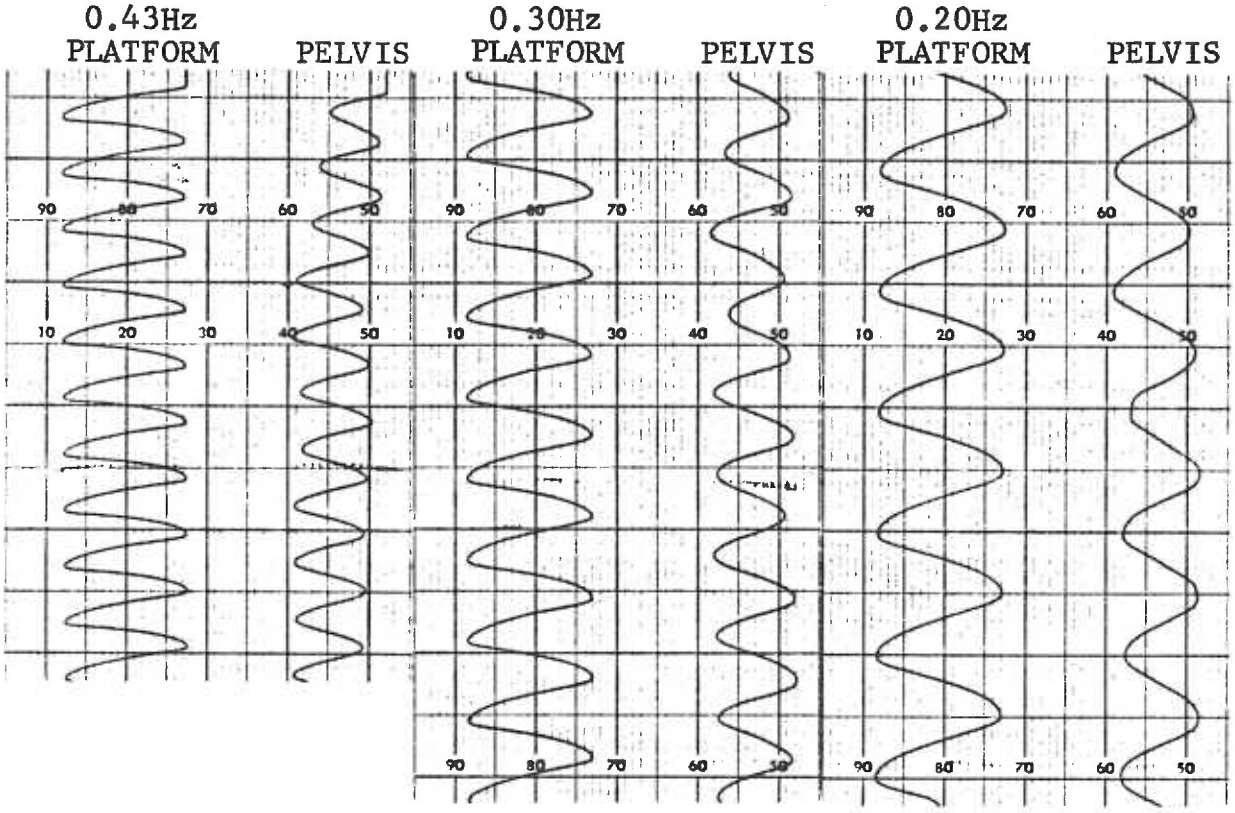
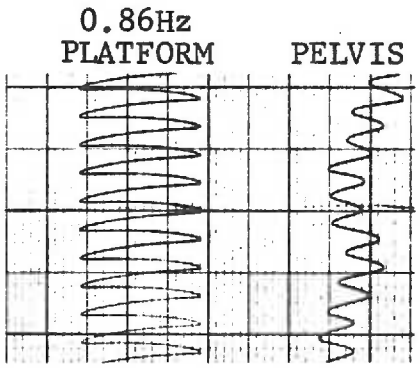
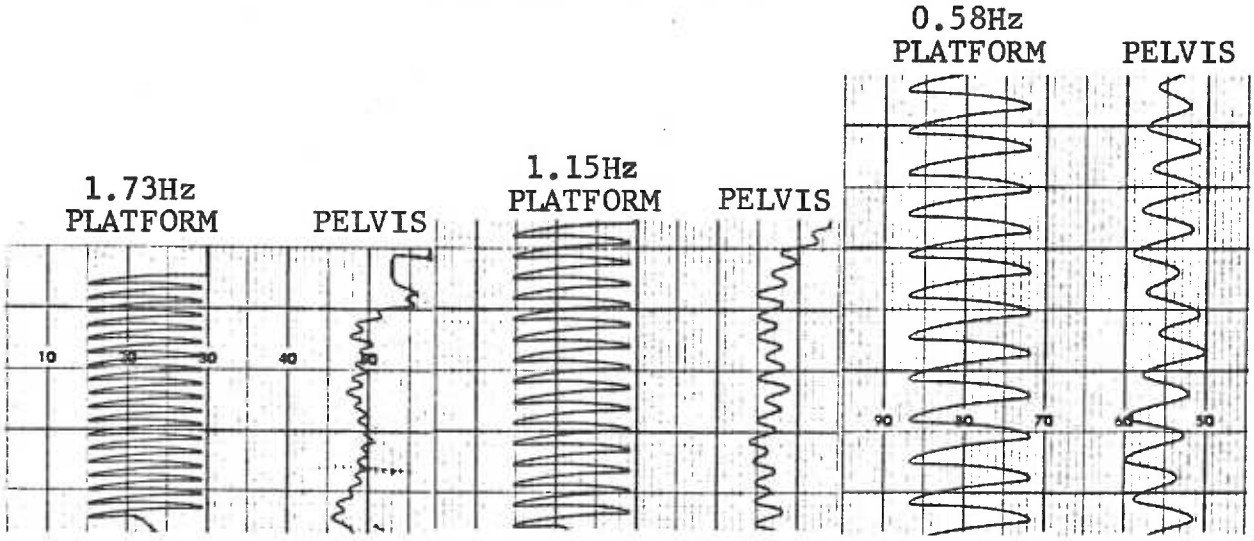

0.58Hz
PLATFORM
PELVIS
0.20Hz
ROOF

Figure 19. Analog voltage records from pelvis and platform position potentiometers in a NP experiment. Frequencies of platform oscillation are given to the left of each record.


0.43Hz
PLATFORM
PELVIS
0.30Hz
PLATFORM
PELVIS
0.20Hz
PLATFORM
PELVIS


0.86Hz
PLATFORM
PELVIS


0.58Hz
PLATFORM
PELVIS
1.73Hz
PLATFORM
PELVIS
1.15Hz
PLATFORM
PELVIS

predominance of the forcing frequencies in the dog's pelvis position response in both groups of experiments, the dog's postural responses were analyzed using only the forcing frequency Fourier coefficients for platform and roof motion. Harmonic distortion was not systematically evaluated.

Next, the forcing frequency amplitude and phase coefficients were normalized for platform and/or roof motion. Dividing the appropriate forcing frequency amplitude coefficient of pelvis position by that of the platform or roof, for each run, yielded a value, denoted gain. Also, the appropriate forcing frequency phase coefficient of the pelvis position had the platform or roof fundamental phase coefficient subtracted from it, for each run, to obtain the value denoted relative phase. Thus, the data for one experimental condition for each dog consist of the gain and relative phase values for five runs on each of five days at each frequency used. The exception to this general pattern applies to the COB' experiment, in which the data files include only five runs for each condition.

The forcing frequency gain and relative phase were summarized so that for each frequency in each experiment the mean, median, standard deviation, interquartile range and absolute range were calculated from the 25 (or five) runs in the experimental data files. Two statistical tests were utilized to compare various pairs of experiments (e,g. pre-operative versus post-operative NP, or NP versus BP) for any difference in the gain of the pelvis position response. A factorial design analysis of variance test was used to determine whether the dog's gain response across all tested frequencies was significantly different in

two specific experiments (20a). If a significant change was present at the 0.01 level, a Student's t-test was performed on the individual frequencies to identify specifically how many, and which, frequencies showed a changed gain response (20A). The analysis of variance and Student's t-test methods are summarized in Fig. 20 and 21, respectively.

The entire procedure for processing the data is summarized in Fig. 22.

Interpretation of Data

Once the dog was reliably able to maintain a quiet, erect stance after the training phase, it was assumed that the "reference input" to the dog's postural "controller" (see Fig. 1) remained relatively stable during the period of the experiment. According to the model of a postural control system, an "external disturbance," such as platform or combined platform-roof oscillation perturbs the dog's trained stance and results in operation of the postural control system aimed at regaining the goal posture. After initial transients, the predictable, sinusoidal perturbation elicits a relatively stable pattern of changes in body position that is assumed to reflect a stationary state of optimal output from the control system, given the experimental conditions.

The gain of the postural response was more sensitive than the relative phase in reflecting the changes in the dog's postural control behavior evoked by visual restriction or cortical ablation. Therefore, to test the hypotheses set forth in the Experimental Objectives section, the statistical methods described in the previous section were used to compare shifts in the gain of postural responses effected by the

Figure 20. Factorial design analysis of variance test used to determine if either the operation or a particular visual restriction had any effect on the gain of the dog's response across all frequencies (20a).

	FACTOR B = frequency				
FACTOR A	0.20	0.30	1.15	1.73	
Pre-op or NP	X_{111}	X_{121}	X_{161}	X_{171}	$T_{1..}$
	$X_{11(25)}$	$X_{12(25)}$	$X_{16(25)}$	$X_{17(25)}$	
Post-op or BP	X_{211}	X_{221}	X_{261}	X_{271}	$T_{2..}$
	$X_{21(25)}$	$X_{22(25)}$	$X_{26(25)}$	$X_{27(25)}$	
	$T_{.1.}$	$T_{.2.}$	$T_{.6.}$	$T_{.7.}$	

$$C = \left(\sum_{a=1}^{2} \sum_{b=1}^{7} \sum_{n=1}^{25} X_{abn} \right)^2 / 2*7*25$$

$$SS_{total} = \left(\sum_{a=1}^{2} \sum_{b=1}^{7} \sum_{n=1}^{25} X_{abn}^2 \right) - C$$

$$SS_{treatments} = \left(\sum_{a=1}^{2} \sum_{b=1}^{7} T_{ab}^2 / 25 \right) - C$$

$$SS_A = \sum_{a=1}^{2} T_{a..}^2 / 7*25 - C$$

$$SS_B = \sum_{b=1}^{7} T_{.b.}^2 / 2*25 - C$$

$$SS_{AB} = SS_{treatments} - SS_A - SS_B$$

$$SS_{residual} = SS_{total} - SS_{treatments}$$

Analysis of Variance Table

Source	SS	df	MS	V.R.
A	SS_A	1	SS_A/df	$MS_A/MS_{residual}$
B	SS_B	6	SS_B/df	$MS_B/MS_{residual}$
AB	SS_{AB}	1*6	SS_{AB}/df	$MS_{AB}/MS_{residual}$
Treatments	SS_{tr}	1*7-1		
Residual	SS_{res}	1*7*24	SS_{res}/df	

Each of the Variance Ratios (V.R.) follows an F-distribution with the calculated degrees of freedom (df). A significance level of 0.01 was chosen to test the null hypothesis that Factor A is not a source of variability.

Figure 21. Student's t-test for the difference between two sample means was used to test for significant changes in the gain values of the dog's response at a given frequency (20a).

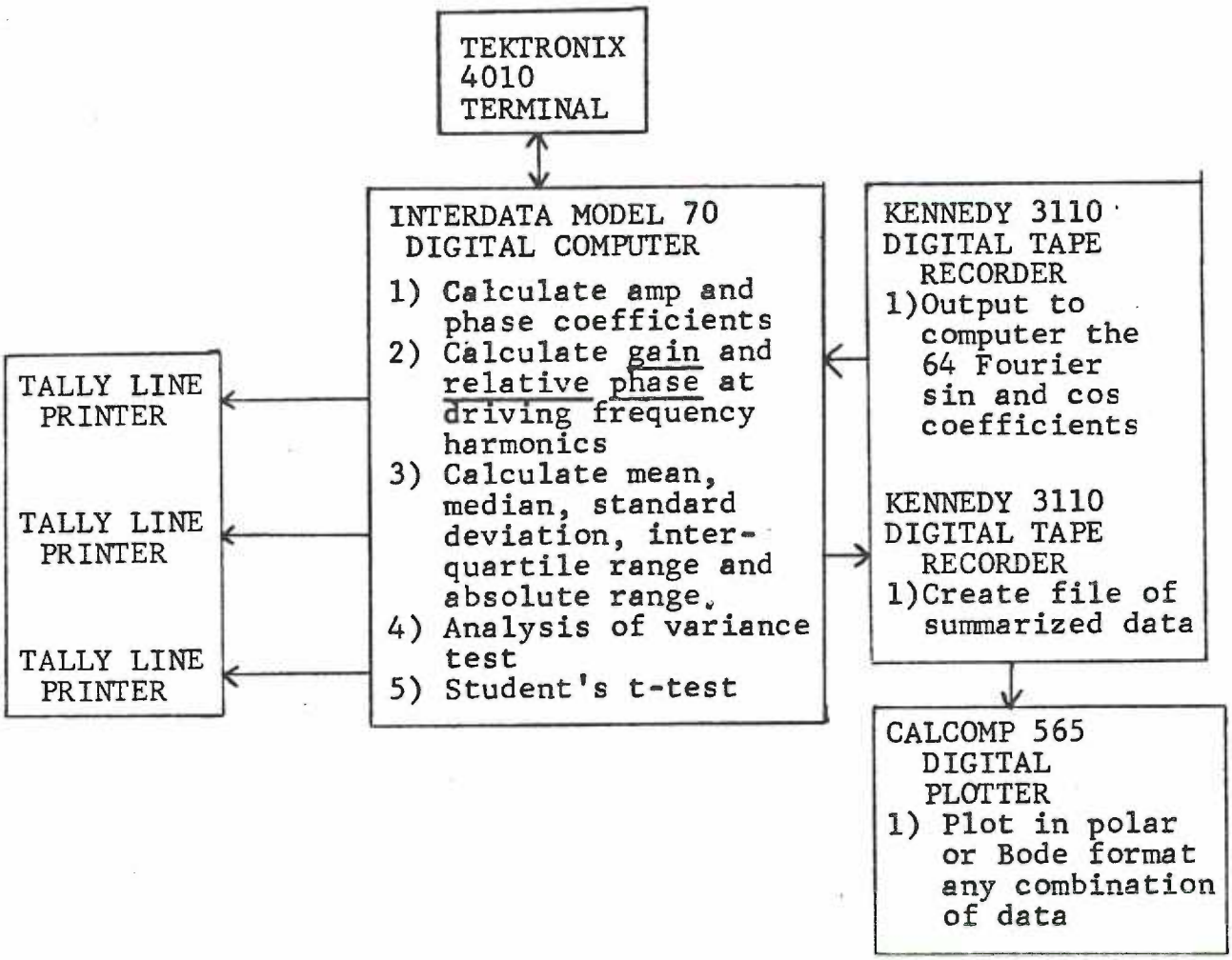
$$t' = \frac{(\bar{X}_1 - \bar{X}_2) - (u_1 - u_2)}{(s_1^2/n_1 + s_2^2/n_2)^{\frac{1}{2}}}$$

$$df' = \frac{(s_1^2/n_1 + s_2^2/n_2)^2}{(s_1^2/n_1)^2/n_1 + (s_2^2/n_2)^2/n_2}$$

where, X = sample mean
u = population mean
s = sample standard deviation
n = sample size

The null hypothesis that the population means for the two experiments' corresponding frequency responses being tested are equal ($u_1 = u_2$) was tested at 0.005 and 0.001 significance levels. A modified formula for df was used to take into account unequal population variances, which are normally assumed equal in the Student's t-test.

Figure 22. Summary of procedure used for processing and testing data. See text for details.


TEKTRONIX
4010
TERMINAL
INTERDATA MODEL 70
DIGITAL COMPUTER
1) Calculate amp and phase coefficients
2) Calculate gain and relative phase at driving frequency harmonics
3) Calculate mean, median, standard deviation, inter-quartile range and absolute range.
4) Analysis of variance test
5) Student's t-test
KENNEDY 3110
DIGITAL TAPE
RECORDER
1)Output to computer the 64 Fourier sin and cos coefficients
KENNEDY 3110
DIGITAL TAPE
RECORDER
1)Create file of summarized data
TALLY LINE
PRINTER
TALLY LINE
PRINTER
TALLY LINE
PRINTER
CALCOMP 565
DIGITAL
PLOTTER
1) Plot in polar or Bode format any combination of data

experimental conditions. However, the interpretation of changes in gain differs for the two groups of experiments.

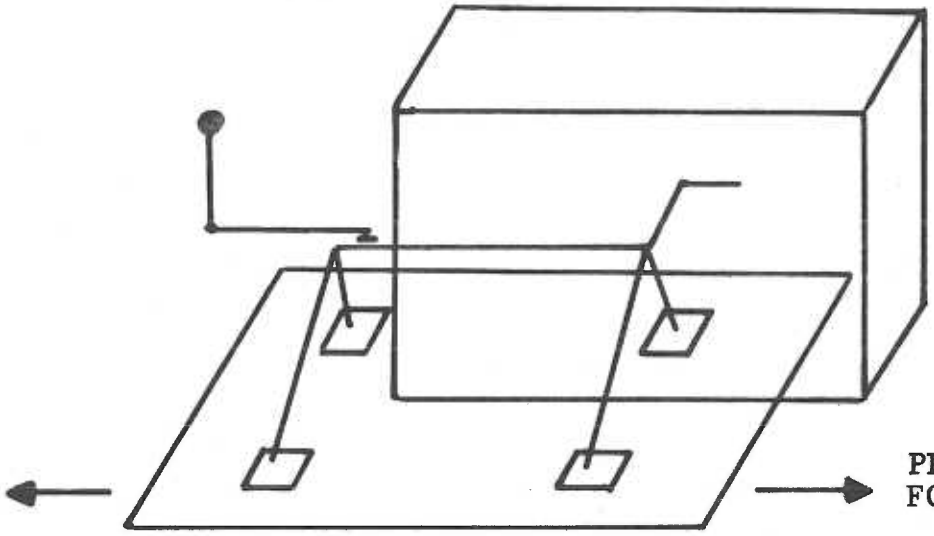
In the platform experiments, the dog was presented with a predictable postural control problem. The gain of the subsequent postural response reflected the optimal stabilization of body position achieved by the postural control system. Therefore, changes in pelvis gain for platform experiments were interpreted as being inversely proportional to optimal postural control, and directly proportional to some deficit in postural control.

In the platform-roof experiments, the gain of postural responses evoked purely by an optokinetic stimulus was quantified, reflecting the effectiveness of visual feedback in the postural control system given the experimental conditions. Thus, changes in pelvis gain at roof driving frequencies were interpreted as being directly related to the potency of visual feedback in evoking postural responses.

Fig. 23 summarizes the critical differences in interpretation of changes in pelvis gain between the two groups of experiments.

Figure 23. Interpretation of changes in gain of the pelvis position evoked during (A) platform experiments and (B) platform-roof experiments.

(A) PLATFORM EXPERIMENTS



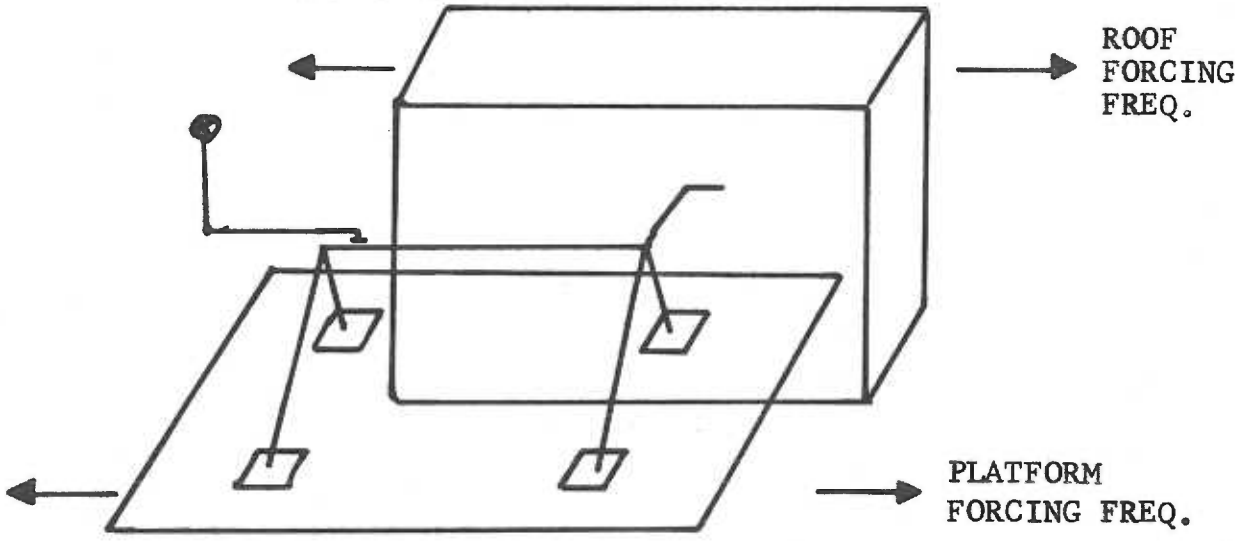
GAIN (at platform forcing frequency)

1.0 = pelvis position is perturbed same distance as platform (about 8 cm.)

↓ = decreased perturbation of pelvis -- interpreted as more optimal postural control

↑ = increased perturbation of pelvis -- interpreted as a deficit in postural control

(B) PLATFORM-ROOF EXPERIMENTS



GAIN (at roof forcing frequency)

↑ = larger amplitude of postural perturbation evoked by roof motion -- interpreted as a greater potency of visual feedback in postural control

↓ = smaller amplitude postural perturbation evoked by roof motion -- interpreted as less potent visual feedback in postural control

RESULTS

Histological Evaluation of Visual Cortex Lesions

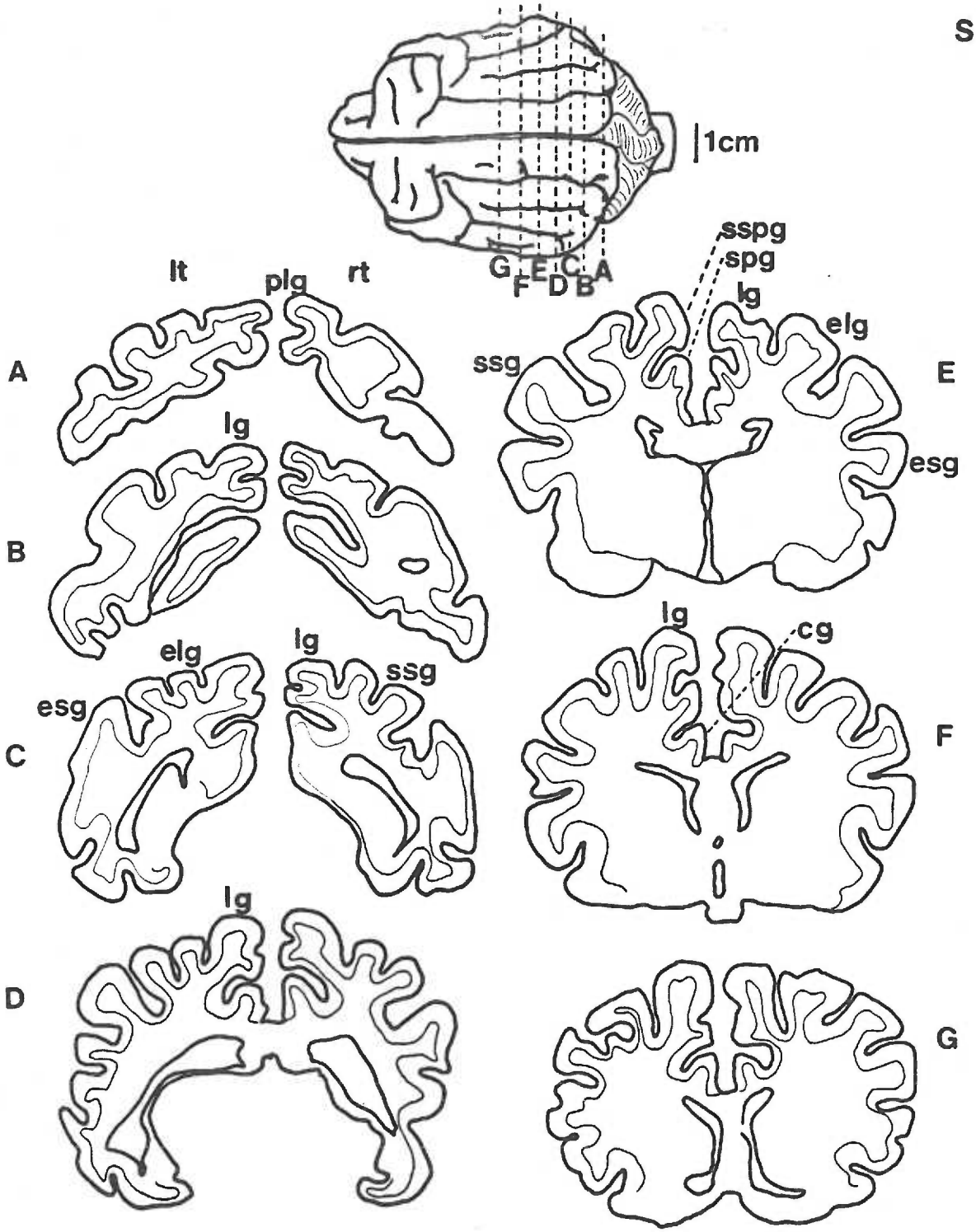
Drawings of dorsal brain topography from photographs and serial reconstructions of histological sections of cortex permit a detailed description of each lesion. In addition, photographs of serial sections through the dorsal lateral geniculate nucleus (LGN_d) provide an index of the completeness of ablation of primary visual cortex, based on the degree of retrograde degeneration present.

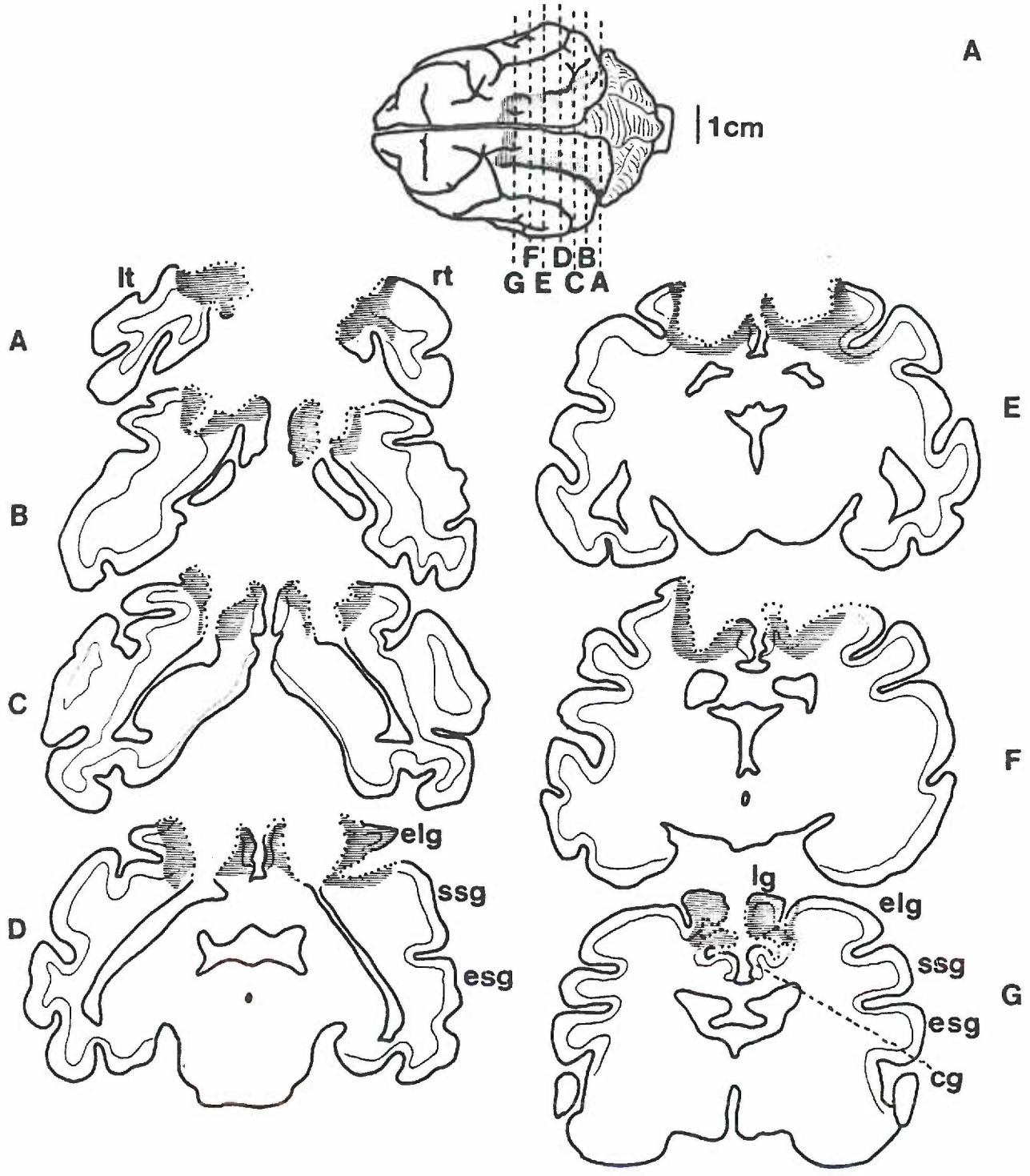
Fig. 24 displays the dorsal topography of the cerebral cortex and the selective coronal sections of the occipital pole of cortex for the sham-operated dog as well as the five lesioned dogs. The drawings from the sham-operated dog (Fig. 24S) demonstrate the normal gyral pattern. Note the lateral gyrus (lg) which includes visual areas 17, 18 and 19, the postlateral gyrus (plg) which houses area 17, and the suprasplenial (sspg) and splenial (spg) gyri which predominantly represent area 17 on the medial aspect of the hemispheres (1,20,100).

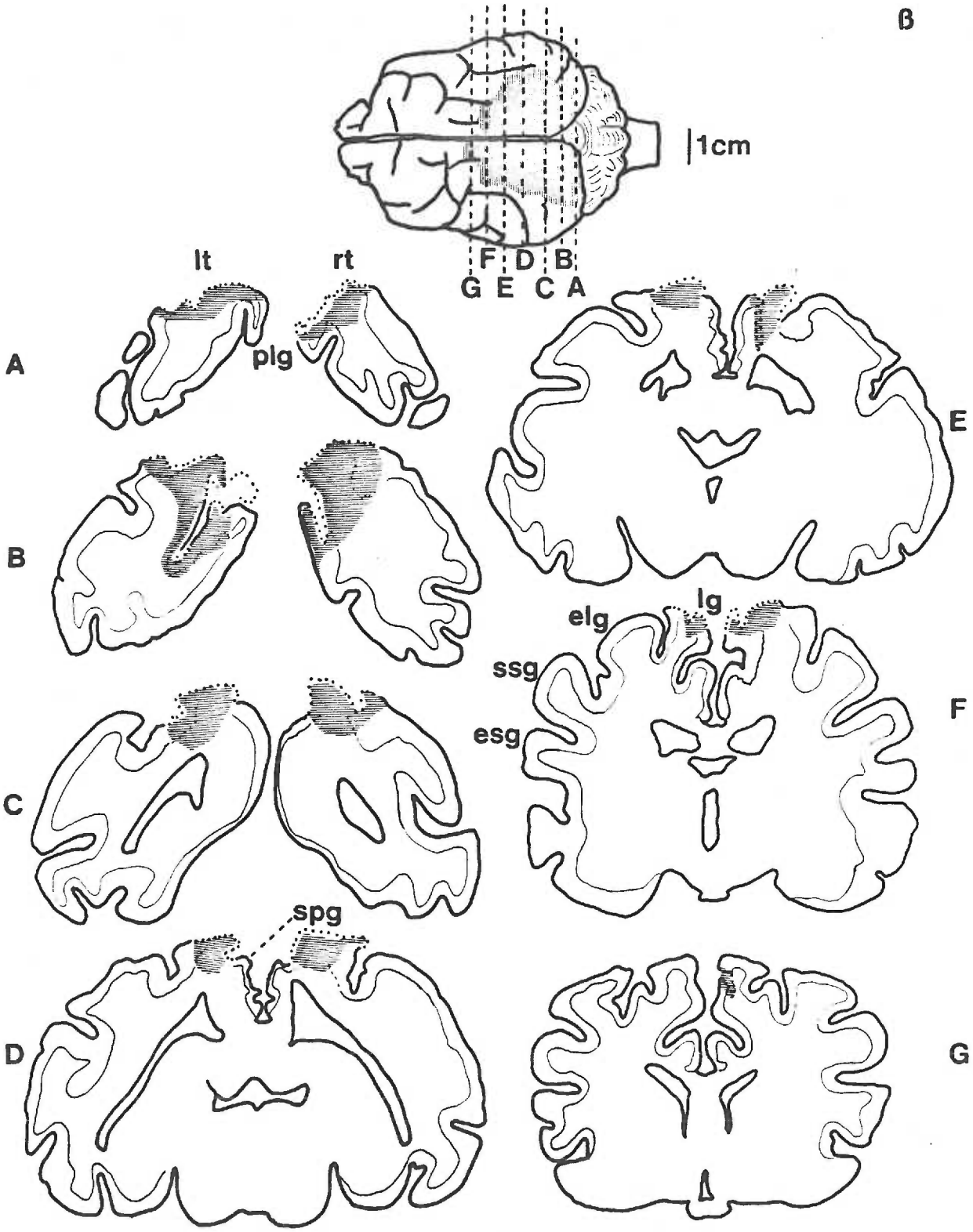
In dog A (Fig. 24A), all of the cortex on the medial surface of the hemispheres comprising suprasplenial and splenial gyri was ablated, with variable extension into the cingulate gyrus; on the dorsal surface, the postlateral gyrus and posterior three-fourths of the lateral gyrus were completely removed with undercutting of the anterior portion of the lateral gyrus and variable damage to the adjacent ectolateral gyrus. From the serial reconstruction of occipital cortex, the lesion of primary visual cortex in dog A appears complete.

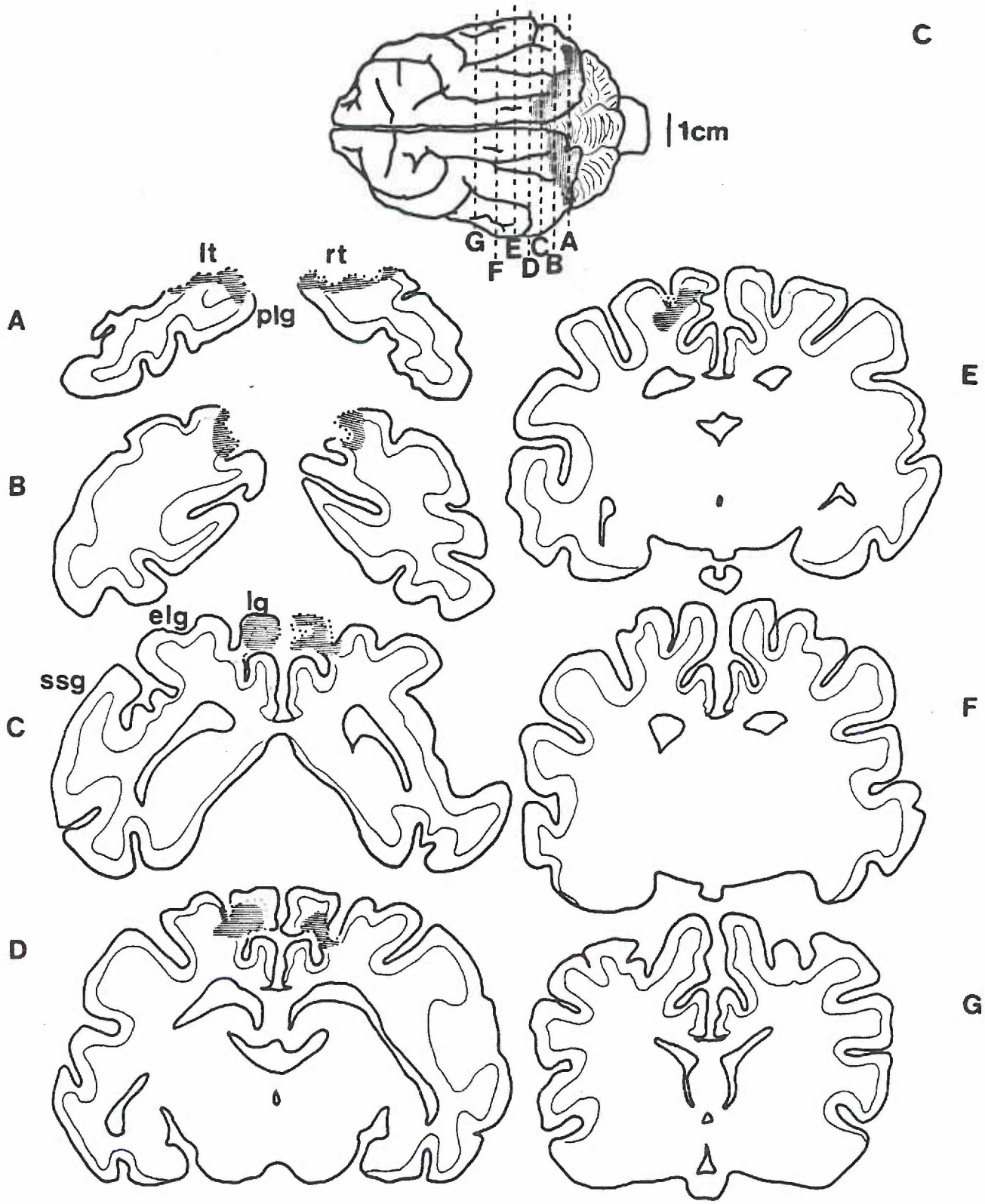
Dog B (Fig. 24B), on the other hand, retains some areas of intact visual cortex. The drawing of the dorsal view of the hemispheres

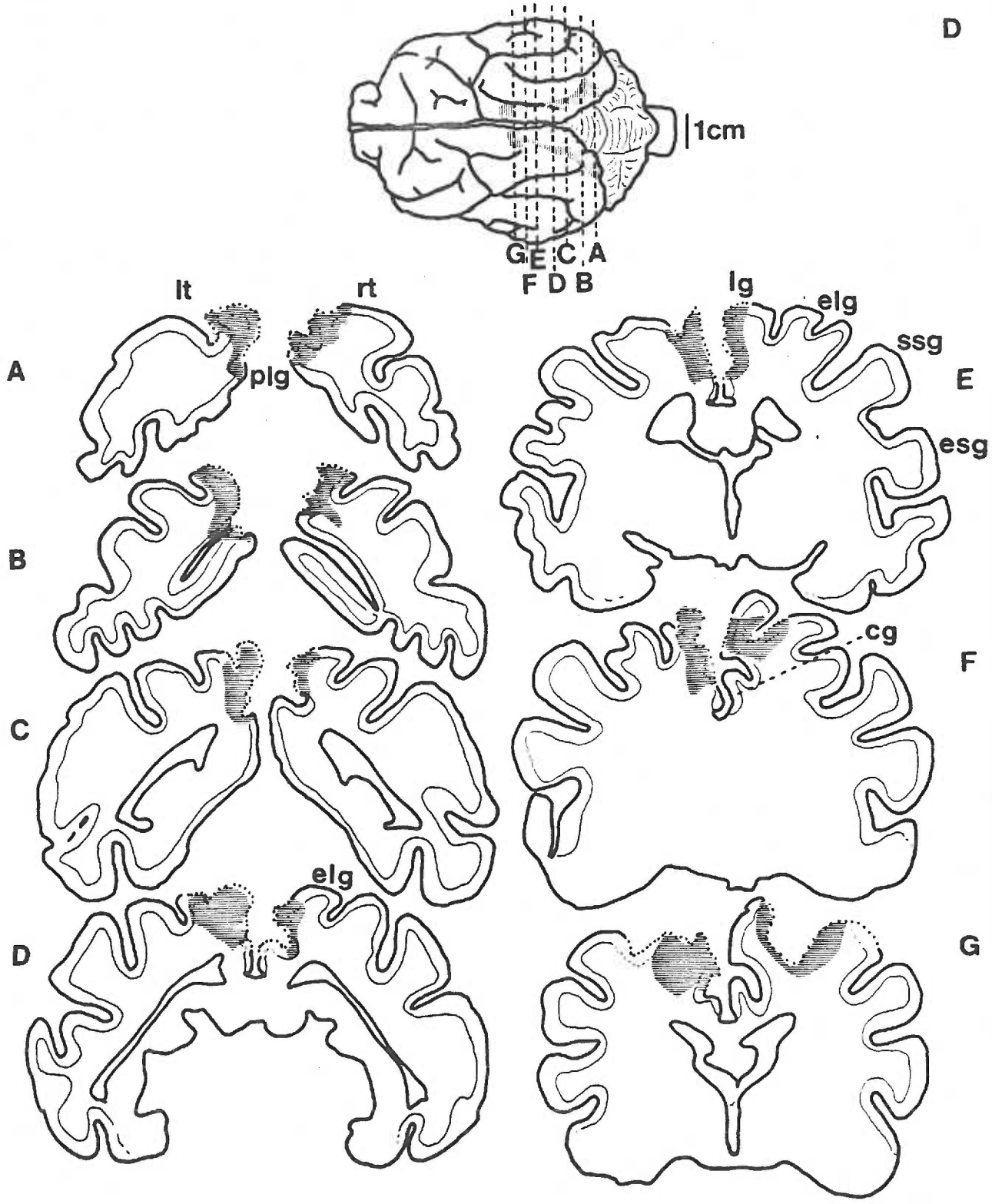
Figure 24. (S, A-E) Drawings of dorsal topography of the cerebral hemispheres and of selected coronal sections through the posterior pole of cortex in dog S and dogs A through E. Note that each coronal section is labelled A through G, with its level indicated by transverse lines on the dorsal view of the cerebral hemispheres. Dotted lines indicate areas where cortex had been removed. Shaded areas of cortex delimit disruption of normal columnar organization of neurons, and obvious scarring. (lt = left, rt = right, lg = lateral gyrus, plg = postlateral gyrus, elg = ectolateral gyrus, ssg = suprasylvian gyrus, esg = ectosylvian gyrus, sspg = suprasplenial gyrus, spg = splenial gyrus, cg = cingulate gyrus)

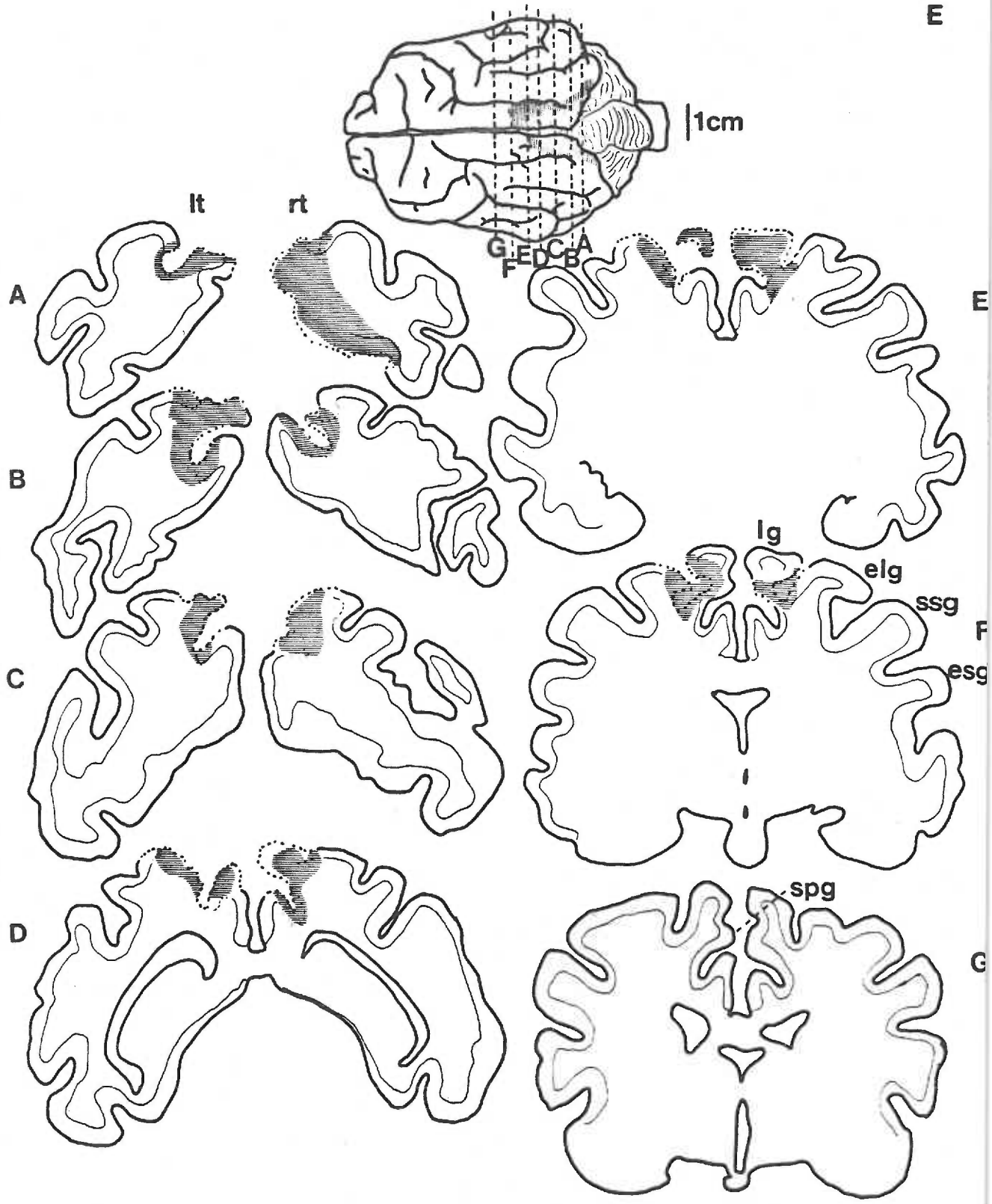

S
1cm
G
F
E
D
C
B
A
lt
plg
rt
lg
elg
esg
ssg
sspg
spg
cg


A
1cm
F D B
G E C A
lt
rt
A
B
C
D
E
F
G
elg
ssg
esg
lg
elg
ssg
esg
cg


ß
1cm
F D B
G E C A
lt
rt
A
plg
B
C
spg
D
E
elg
lg
ssg
esg
F
G


C
1cm
G
E
C
A
F
D
B
lt
rt
plg
A
B
C
D
E
F
G
elg
lg
ssg


D
1cm
G E C A
F D B
lt
rt
lg
elg
ssg
plg
A
E
esg
B
cg
F
C
elg
D
G


E
1cm
lt
rt
G
F
E
D
C
B
A
A
B
C
D
E
lg
elg
ssg
esg
F
spg
G

appears to depict a large cortical ablation; however, the widely shaded area represents scar tissue that was adherent to the cortical surface, obscuring the true extent of the lesion. The serial sections point out that large parts of the splenial gyrus and some postlateral gyrus were both spared bilaterally, but the lateral and suprasplenial gyri were almost completely ablated.

In dog C (Fig. 24C), as well, the lesion is incomplete. The surface drawing of the hemispheres shows bilateral sparing of the lateral gyrus except for the posterior one-fourth. This is verified by the serial sections, although some undercutting occurred primarily in the left middle lateral gyrus. All but the posterior one-fourth of the splenial and suprasplenial gyri were intact. Overall, approximately the posterior one-half of the primary visual cortex had been ablated, except for some sparing of the medialmost parts of both postlateral gyri.

A much more complete lesion was present in dog D (Fig. 24D). The suprasplenial and splenial gyri were ablated completely, with the exception of some sparing in the very anterior extent of the gyri on the right side, possibly outside the visual area. Only small remnants of lateral gyrus were spared -- on the right side in the anterior one-fourth and on the left side only slightly in the anterior portion. Postlateral gyrus was ablated except for a small remnant located medially on the right side. Based on the serial reconstruction, dog D appeared to have a nearly complete lesion.

Finally, in dog E (Fig. 24E), some sparing of the anterior one-fourth of lateral gyrus occurred bilaterally. The splenial and supra-

splenial gyri were partly intact anteriorly and at the posterior pole. The postlateral gyrus was spared on the left side.

The dorsal topography of the cerebral hemispheres and the serial reconstructions of the posterior half of both hemispheres show an apparently complete ablation of primary visual cortex in dog A and a nearly complete lesion in dog D; significant areas of visual cortex were spared in dogs B and E, and a great deal of sparing occurred in dog C.

A second, more sensitive method of assessing the limits and completeness of the lesions is based upon identification of retrograde atrophy in the LGN_d of the thalamus (16). The neurons in the LGN_d (i.e. laminae A and A_1) whose axons project exclusively to ablated parts of areas 17 and 18 demonstrate late retrograde changes that result in cell atrophy and loss. Therefore, when studying Nissl stained sections of the LGN_d, areas of decreased cell density are clearly identifiable (Fig. 25). The retrograde atrophy is restricted to large (type II) and medium-sized neurons, with sparing of the small (type III) cells (89). Fig. 26 consists of photographs of coronal sections through anterior, middle and posterior levels of both dorsal lateral geniculate nuclei for each dog.

Fig. 26S compares the left LGN_d of a control dog that was sacrificed strictly for histologic control with the left LGN_d from the sham-operated control dog (dog S). No obvious differences in cytoarchitecture are present, indicating the absence of any damage to the visual cortex in dog S. Note the identification of various laminae on the photographs for dog S. Lamina A_1 consists of neurons receiving purely

Figure 25. Photomicrographs of low and high power views of the left dorsal lateral geniculate nucleus. (A) Dog S, the sham-operated dog. (B) Dog A, an ablated dog. Note the complete atrophy of primarily the large (L) and medium (M) sized neurons, with sparing of the small (S) sized neurons in dog A. See text for details.

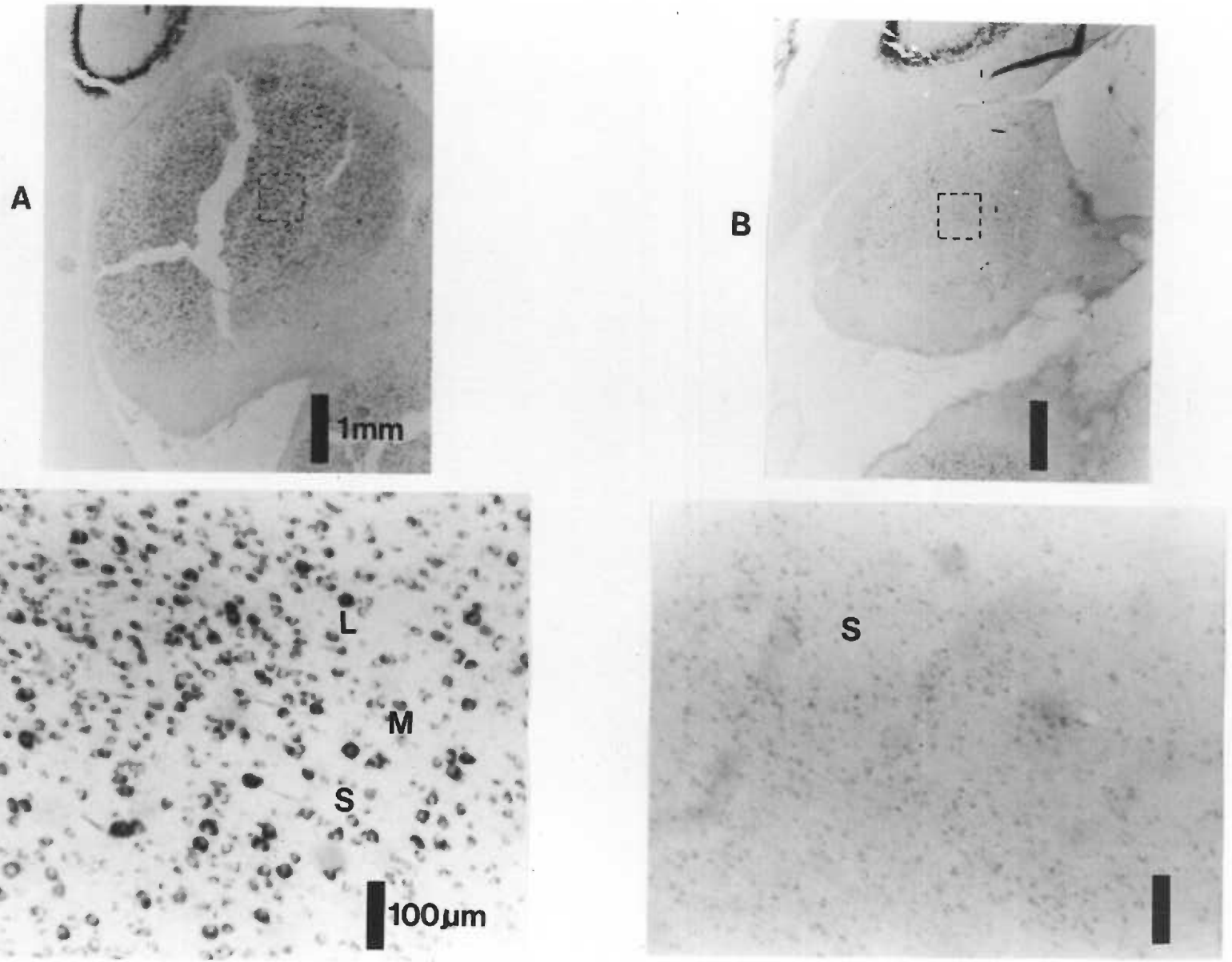
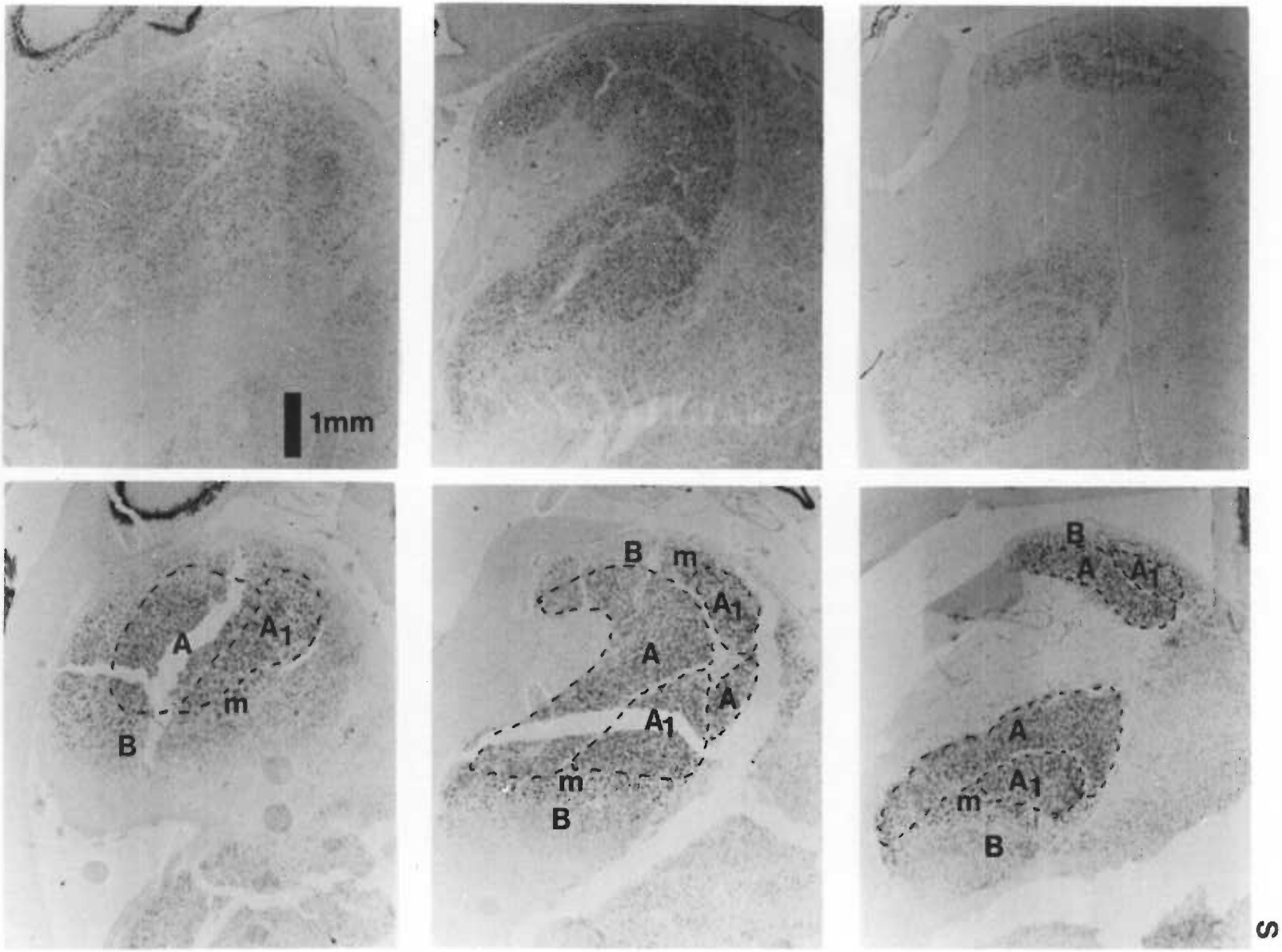
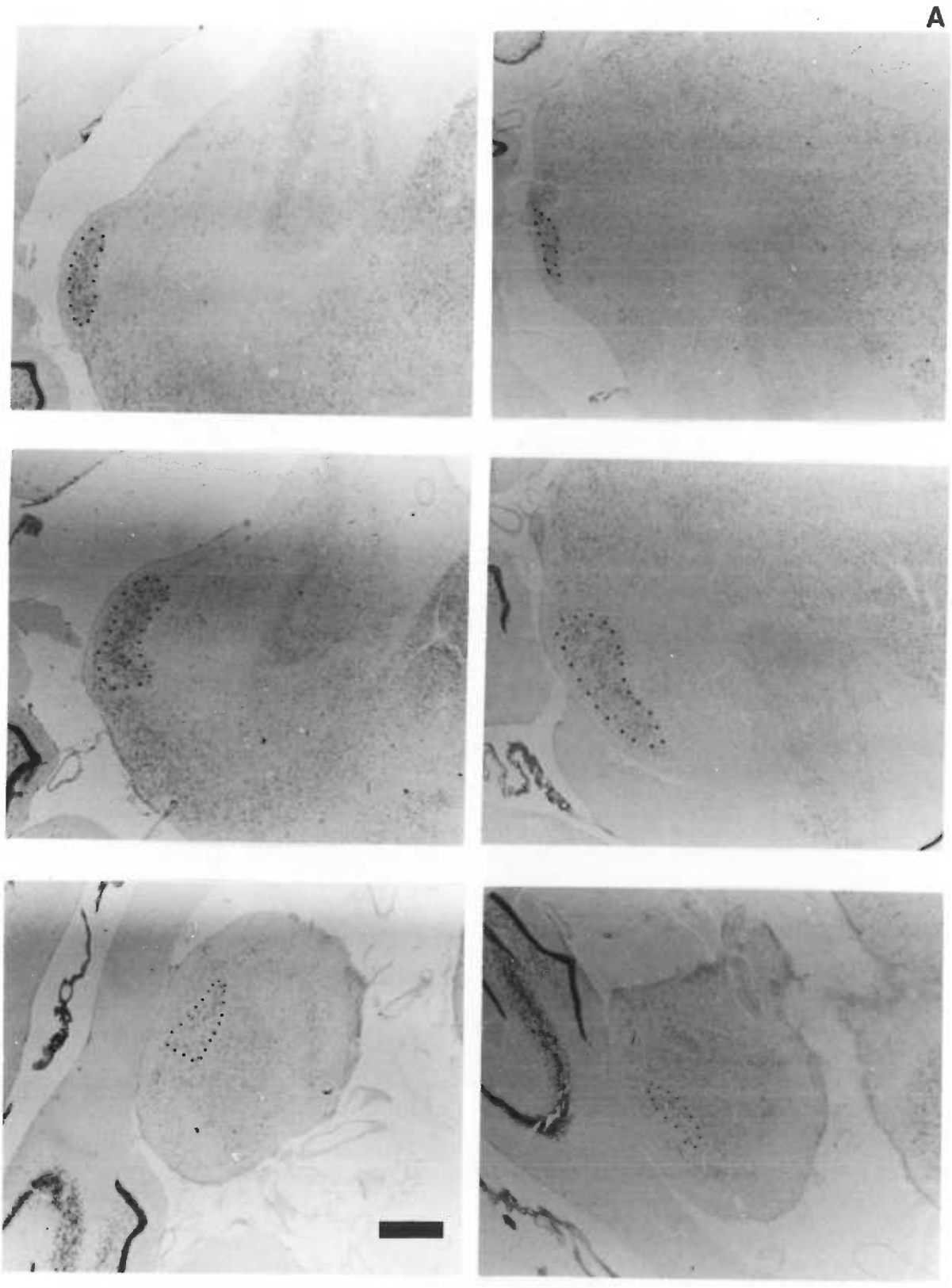
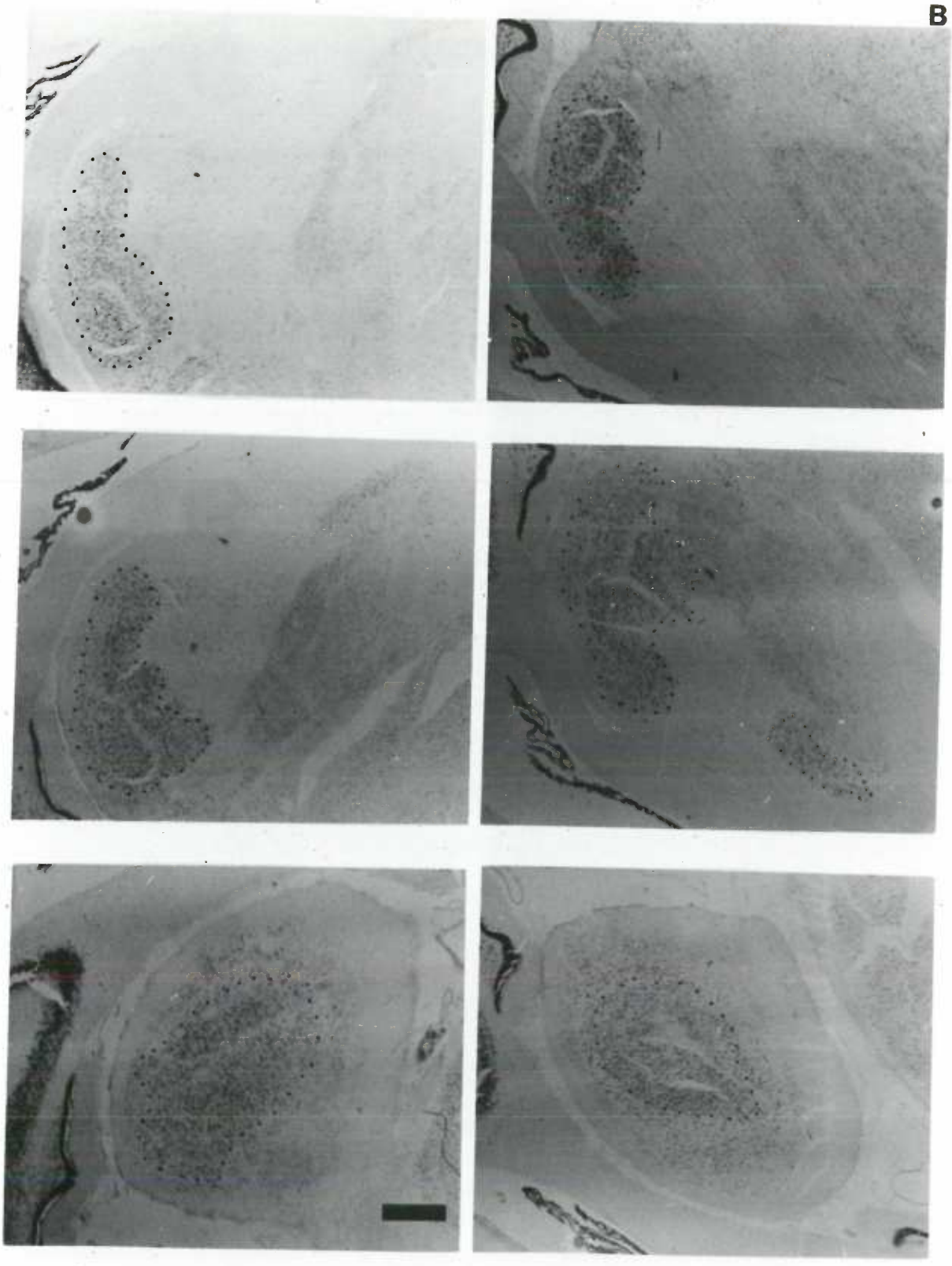
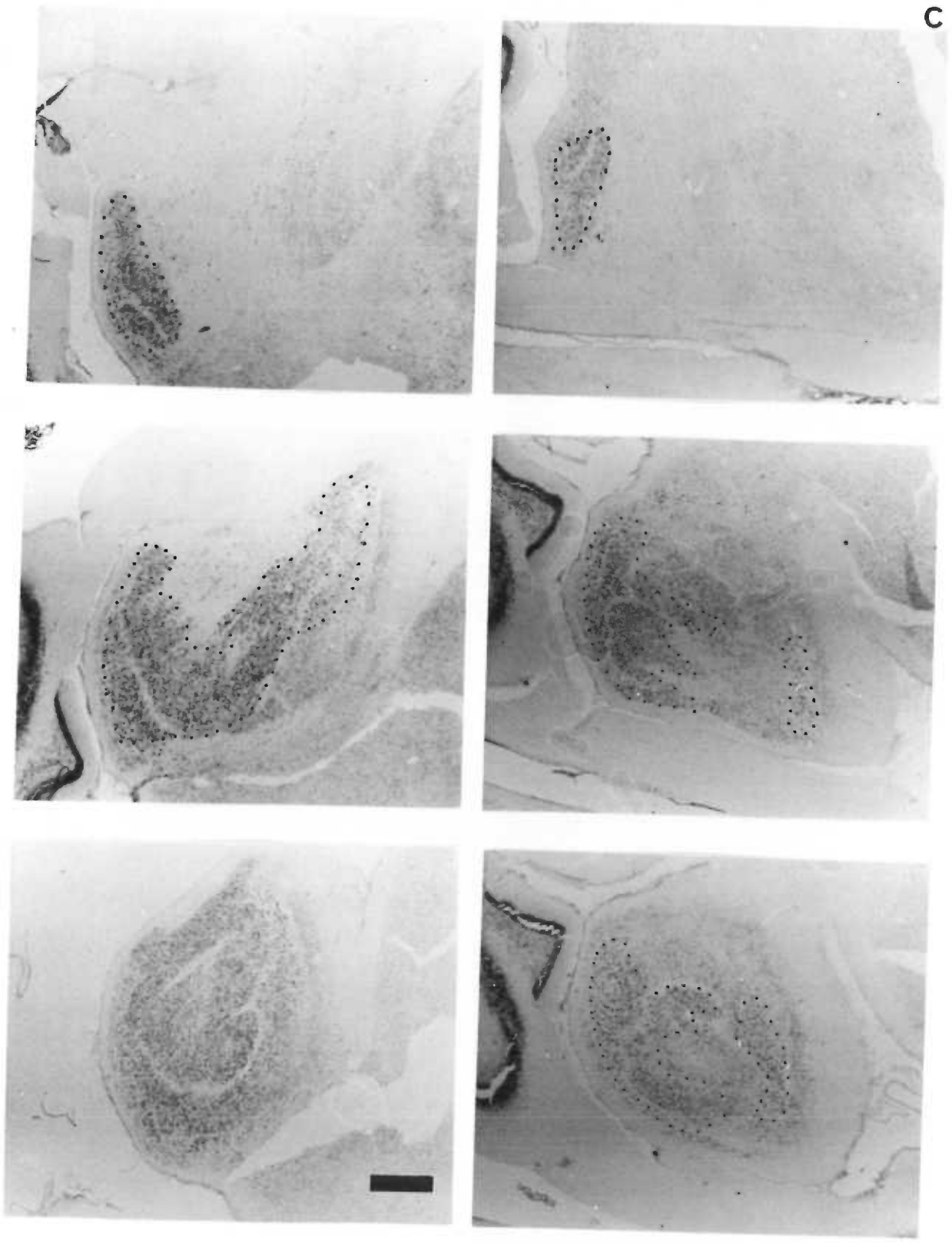

A
1mm
B
L
M
S
100µm
S

Figure 26. (S, A-E) Photomicrographs of transverse sections of the dorsal lateral geniculate nucleus. (S) Comparison of the left LGN_d from a normal dog (upper) with that from dog S (lower) at three levels; posterior, middle and anterior levels are displayed left to right. Note that the separate laminae (A, A_1, B and m) of the LGN_d are labelled in dog S. (A) Dog A. Right LGN_d = top row of photographs, left LGN_d = bottom row of photographs. Posterior, middle, and anterior transverse levels displayed left to right. Areas of intact neurons are encircled by dots. (B) Dog B. (C) Dog C. (D) Dog D. (E) Dog E.


1mm
A
A1
m
B
B
m
A1
A
A
A1
m
B
B
A
A1
A
m
B
S


A


B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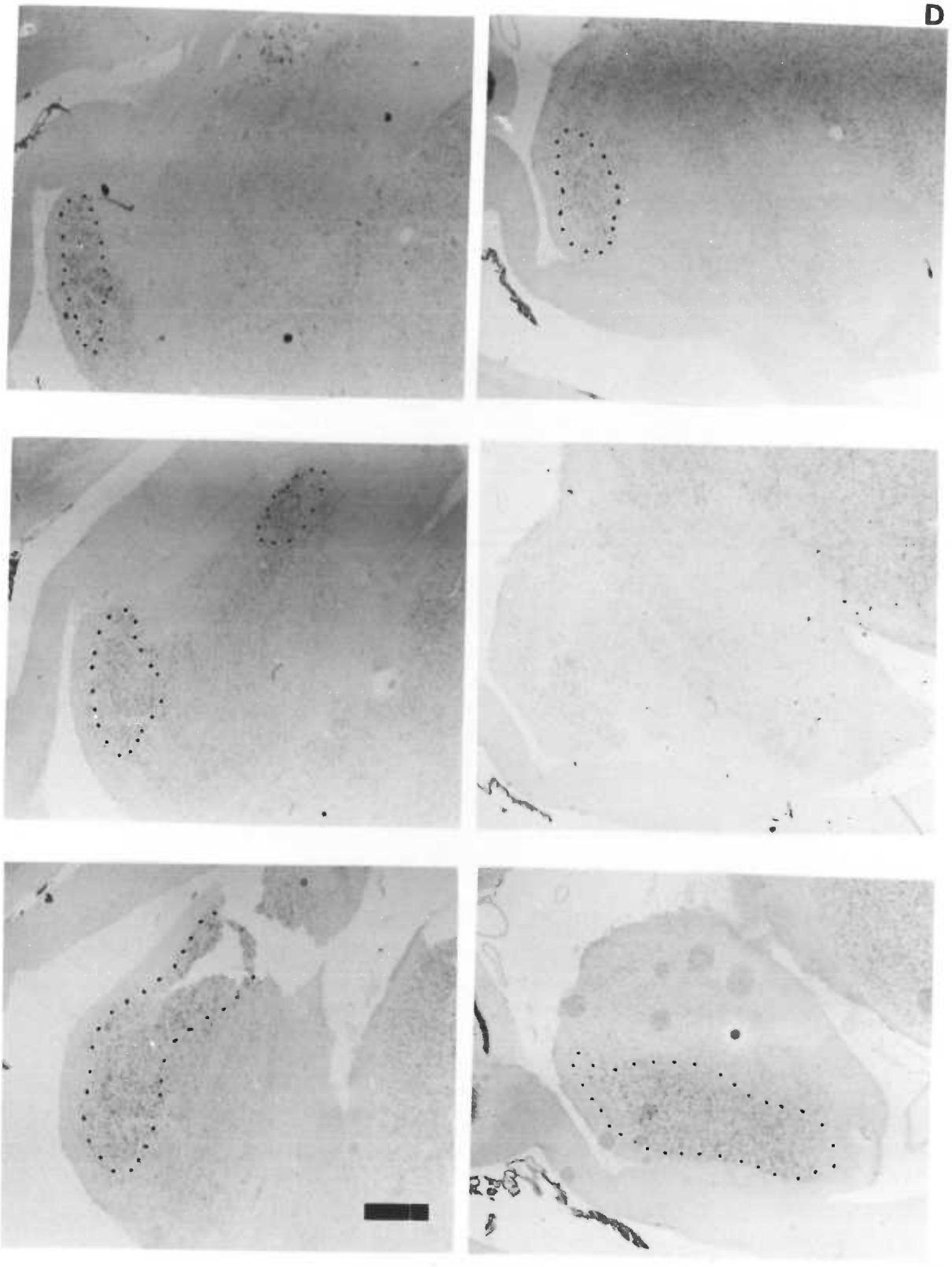
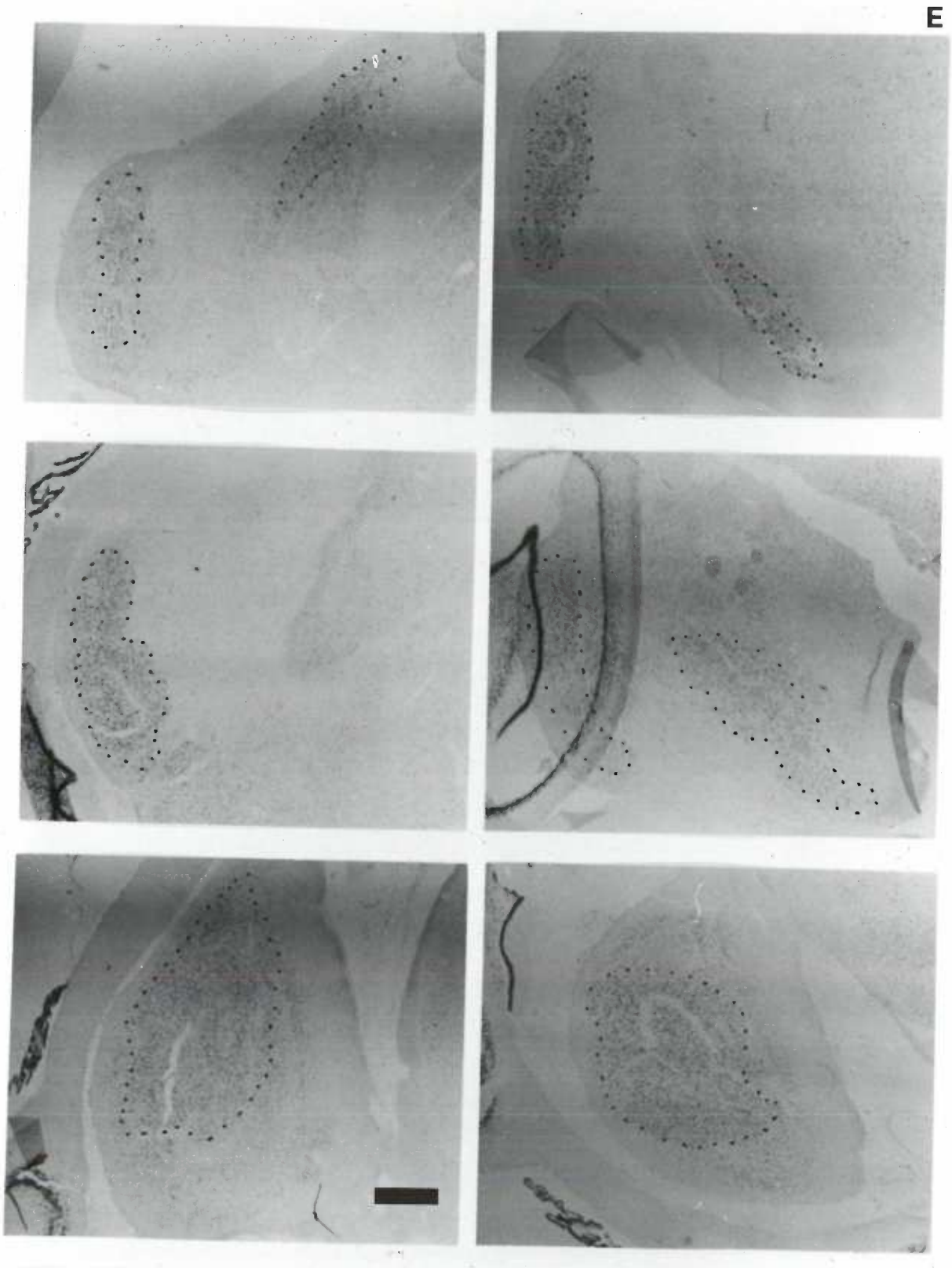

D


E

ipsilateral retinal input, while laminae A and B receive contralateral, nasal retinal input (64). However, as shown in Fig. 2, lamina B does not project exclusively to areas 17 and 18 as do laminae A and A_1. The magnocellular lamina (m) is an interlaminar nuclear area receiving convergent inputs from both retinae and projecting to multiple sites (52,82). Therefore, for purposes of analysis of retrograde atrophy in the LGN_d, laminae A and A_1 are of primary concern.

In dog A (Fig. 26A), the LGN_d was severely atrophied. A small area in the very dorsal portion of the nucleus had much less cell loss, but may represent laminae B and m. Dog B (Fig. 26B) demonstrated only partial atrophy of the LGN_d. Severe atrophy occurred in the ventral parts of both anterior and middle levels of the nucleus, while both posterior levels and dorsal parts of the middle and anterior levels showed almost no degeneration. The pattern of atrophy in dog C (Fig. 26C) was somewhat similar to dog B. Severe atrophy had occurred in the ventral part of both anterior levels of the nucleus and focally in ventral areas of the middle and posterior levels on the left side. Very little atrophy, however, had taken place in the dorsal part of both anterior levels, in the posterior and middle levels on the right, and in the dorsal parts of the posterior and middle levels on the left. In dog D (Fig. 26D), severe atrophy had taken place throughout large parts of the LGN_d. Areas of incomplete atrophy were identifiable in the lateral part of both posterior levels and in the dorsal part of both anterior levels of the nucleus. Finally, dog E (Fig. 26E) showed little atrophy at both posterior levels, in the dorsal part of both middle and anterior levels, and very slightly in the lateral-ventral

portion of both anterior levels. Severe cell loss was present in the ventral part of the right middle level of the nucleus and in the medial-ventral parts of the left middle and both anterior levels. Overall, the relative completeness of the lesions determined by retrograde atrophy in the LGN_d corroborates the differences in the serially reconstructed extent of the lesions. Dog A possessed the most complete ablation, while dog D's was nearly complete. The results of serial reconstruction of the lesions and the analysis of retrograde degeneration in the dorsal lateral geniculate nucleus are summarized in Table IV.

Qualitative Evaluation of Visually-guided Behavior

Clinical Observations

The post-operative observations and clinical testing of visually-guided behaviors provide some general information about the visual deficits resulting from the visual cortex ablation. The degree and time course of compensation are presumed to be manifested by progressive improvements in the visually-guided behaviors. Additionally, a relative stability in visual function can be assumed from the eventual stability in visually-guided behavior.

As a basis for comparison, the pre-operative results of clinical tests of visually-guided behavior are described below:

VISUAL FOLLOWING: Dogs accurately tracked a moving food stimulus by rapid turning of eyes, head, neck and body in all directions, including a full circle. The stimulus was never lost during following.

VISUAL ORIENTING: Rapid turning of eyes, head and neck were made to fixate a moving food stimulus presented in any area of the visual field.

VISUAL PLACING: This was directly tested by assessing the dog's performance in ascending and descending a flight of stairs and in stepping off of and onto a single step. These visual placing behaviors were accomplished rapidly, accurately and smoothly -- the dog often bounding up the stairs two or three steps at a time.

BLINK REFLEX: Eye blink was repeatedly elicited by a threatening movement of a food stimulus or finger toward either eye from any area of the visual field. The absence of eye blink in response to threat occurred infrequently. Obvious, consistent blinks were made to corneal or lash tactile stimulation.

AVOIDANCE BEHAVIOR: Dogs avoided all obstacles scattered throughout the room as they moved rapidly in pursuit of the "fleetfooted" experimenter who occasionally did not. Novel environments provided no difficulty in avoidance of obstacles. Quick turns and choices were accomplished in any area cluttered with assorted obstacles.

LOCALIZATION AND IDENTIFICATION: If a food stimulus was either moved or held stationary, the dogs immediately localized the food by orienting to it and fixating on it until fed. Identification of the stimulus as food was evident from the dogs' salivation and anticipatory behavior.

The post-operative visually-guided behavior varied but demonstrated the largest deficit on day 1 after surgery and showed subsequent improvement. Different visual behaviors compensated to varying

degrees and at various times post-operatively. The post-operative observations and clinical tests are summarized for all five operated dogs in Table V. Three dogs (A, B and D) demonstrated a complete lack of visually-guided behavior for almost one week after surgery, while two dogs (C and E) showed residual avoidance behavior and occasional visual orienting and following as early as the first post-operative day. During the second post-operative week, all dogs demonstrated some compensation, primarily in visual orienting behavior to moving peripheral stimuli. Dogs C and E continued to show the greatest residual visual function. Compensation in visual following and avoidance behavior occurred at varying rates for individual dogs between the third and sixth weeks. By day 40, these visually-guided behaviors were stable and demonstrated little further improvement. Dogs C and E possessed the most normal visually-guided behavior: visual following, visual orienting and avoidance behavior approached pre-operative performance; however, visual placing and localization were slow, cautious and, often, inaccurate. Dogs A, B and D all exhibited similar permanent deficits: short duration and infrequent following responses and diminished orienting behavior; no visual placing; and poor visual localization, identification and avoidance behavior. All five dogs lost the blink reflex to visual threat post-operatively, while corneal or lash tactile stimulation continued to elicit eye blink. The pupillary light reflex was unchanged throughout the study for all dogs.

Dog S, the sham-operated control dog, showed no changes in visually-guided behaviors at anytime post-operatively.

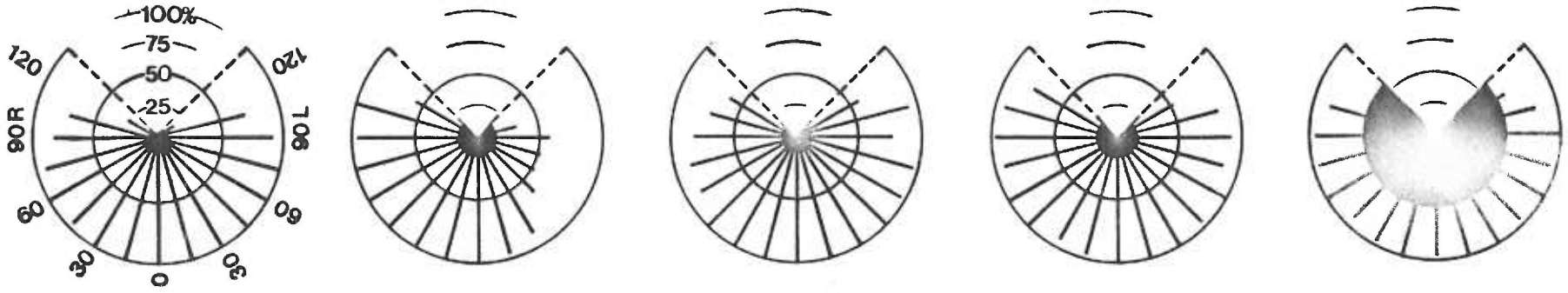
Perimetry Experiments

The systematic testing of the dogs' orienting responses to a three centimeter white ball ("novel stimulus") introduced at various angles of the visual field (see Fig. 17) provided information about the normal limits of the dogs' visual fields and defined quantitatively any residual post-operative visual orienting behavior.

Pre-operatively, the dogs made appropriate orienting responses bilaterally to almost 100% of the test trials from 0° through 90°. As the test stimulus was presented at 105°, 120° and 135°, the dogs responded less regularly. The lateral limit of visual field from which the novel stimulus could not evoke orienting responses more frequently than spontaneous responses (the "critical angle"), averaged 120° bilaterally, while ranging from 105° to 135° for the five experimental dogs. Complete pre-operative data for all five lesioned dogs are presented in Fig. 27A. The data were similar for dogs A, C and D. Dog B demonstrated a curious asymmetry in its orienting behavior, i.e. the novel stimulus evoked fewer orienting responses from the left hemifield as compared to the right hemifield. The basis for the asymmetric response pattern in this particular dog was probably a result of the training that all dogs received in maintenance of an erect, forward-directed stance. Dog B was the best trained dog of the group due to its use in previous experiments by other investigators in the laboratory. It had become well-trained not to turn to its left, where the food reward was kept. As a result, the dog's orienting responses to the left hemifield were fewer, more subtle, but still present out to 105° laterally at a greater rate than spontaneous

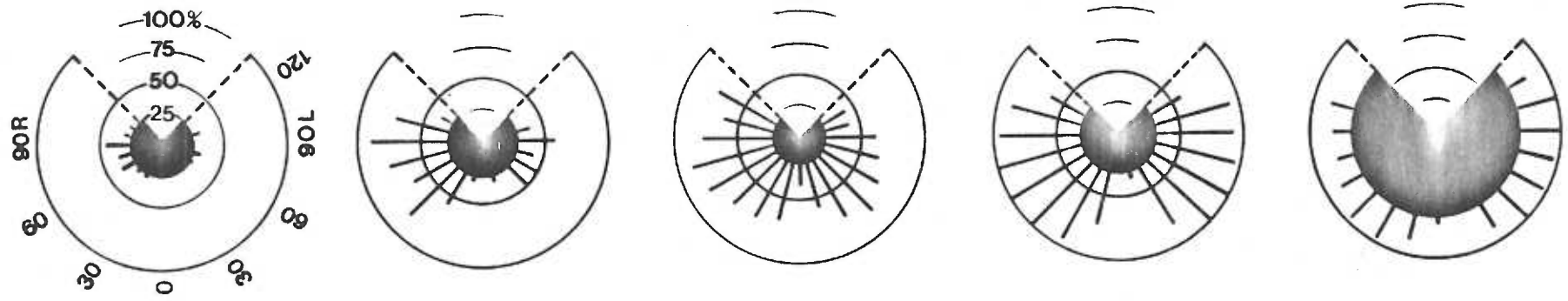
Figure 27. Pre- and post-operative orienting responses in the five ablated dogs. Polar coordinate plot represents the response levels, for 20 trials, at 15° increments (from 0° through 135° bilaterally). The darkened area around the origin represents the level of spontaneous orienting (when no stimulus was presented) over 40 trials. (A) Pre-operative orienting behavior. (B) Post-operative orienting behavior. See text for details.

(A)



DOGS : A B C D E

(B)



responses. Dog E demonstrated a different peculiarity in its orienting behavior. This dog's control level of spontaneous orienting responses was more than four times that of the other four dogs. This may have been attributable to the very hyperactive temperament of this dog. Although the dog was adequately trained to maintain a relatively stable, erect stance during the experiments, its overall behavior remained hyperkinetic. However, the limits of lateral visual field were similar to the other dogs, i.e. 120° left and 105° to the right.

Post-operatively, all dogs manifested some alteration in visual function, demonstrating varying degrees of decreased visual orienting behavior in all cases. Similar post-operative changes occurred in all dogs, except dog A. The orienting behavior after ablation of primary visual cortex in the four dogs was characterized by: no change in the lateral limits of visual field, i.e. 120° bilaterally; an overall decrease in the average percentage of elicitable orienting responses from the central area of the visual field, out to 15° - 45° ipsilaterally in different dogs; and an increase in the percentage of spontaneous orienting responses by 10-15%. Each of these changes in visual orienting behavior occurred to different degrees in the four dogs, as shown in Fig. 27B. However, the basic finding is that some visual orienting behavior remained intact in <u>all</u> dogs.

The data from dog A support the characteristic changes in orienting behavior described above for the other four dogs, but suggest a more severe interference in normal visual function. Orienting responses above the spontaneous level occurred out to 120° to the right and to 105° to the left; the overall percentage of orienting responses

was drastically reduced by 60-70%; stimuli presented in the central 60° to 90° of visual field did not elicit orienting responses above the spontaneous percentage; and the spontaneous level of responses increased by 20%.

Platform Experiments

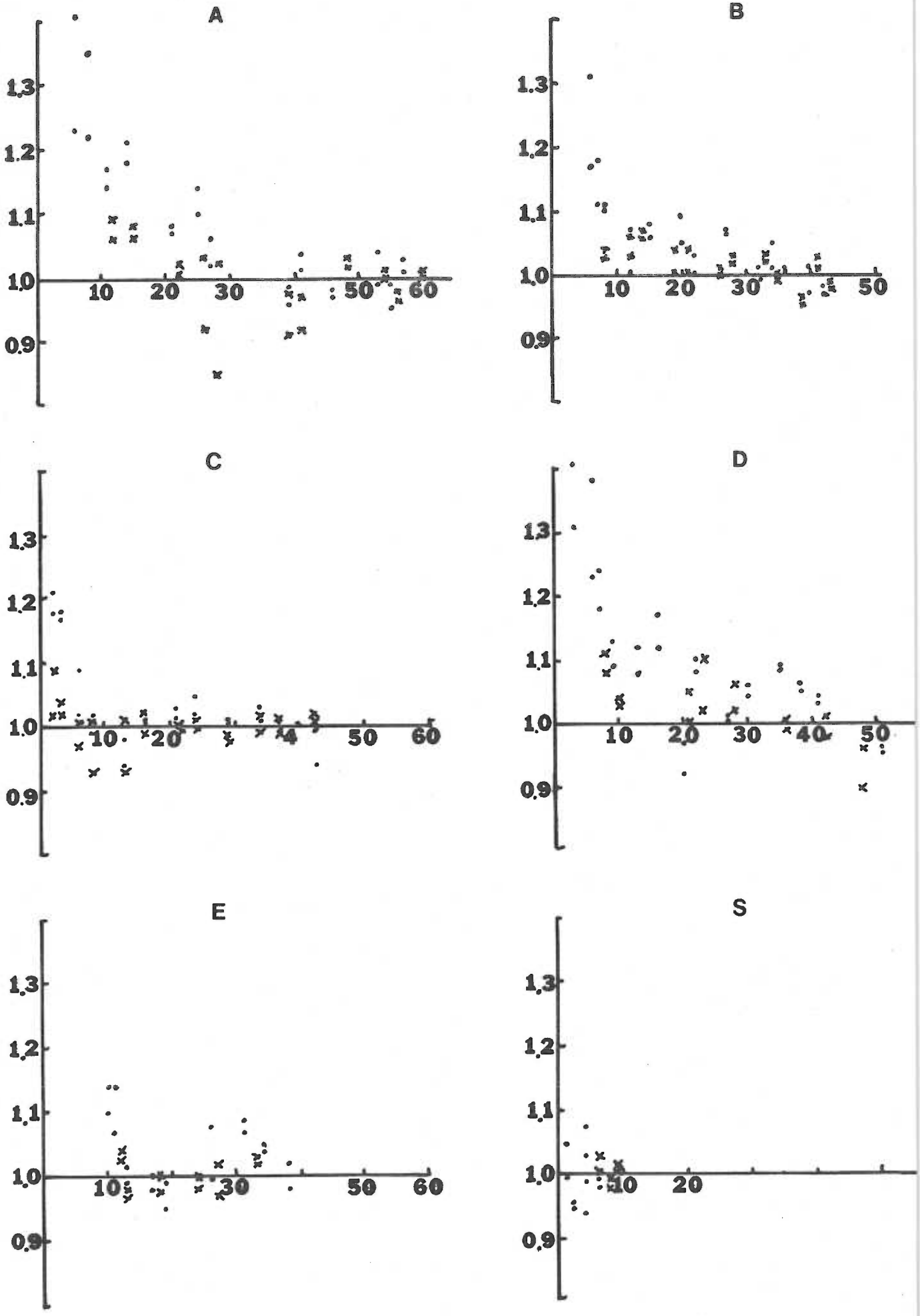
Postural Control Preceding Stable Response Patterns

The data from the platform experiments were collected during a relatively stable response period. Once stable performance had been attained and maintained, five consecutive days of data for each experiment were combined and the median plus first and third inter-quartile ranges calculated. These stable post-operative data, although reflecting the integrity of the postural control system without primary visual cortex, neglect the immediate effects of visual cortex ablation and the time course of any ongoing compensation.

The NP and BP experiments were the only platform experiments repeated as early as possible post-operatively on a regular basis. The COP and POP experiments were usually not performed until the dog's behavior was relatively stable. Fig. 28 plots the gain ratios (median gain of five runs from a single experiment divided by median gain of 25 runs during the stable performance period) of postural responses produced by 0.20 and 0.30 Hz platform oscillation from all NP and BP experiments repeated post-operatively. For all experimental dogs, the last five days' values were combined to represent stable response data. Only three days' data were used to describe the sham control dog's stable performance.

The NP gain ratios for all five experimental dogs were greatest the first day tested and then progressively approached the stable

Figure 28. Post-operative changes in the gain of postural responses for NP (•) and BP (×) experiments. The postural response gains from individual experiments are plotted as gain ratios, calculated by dividing the median gain from an individual experiment by the stable, five-day gain. Only the gains for two frequencies of perturbation (0.20 and 0.30 Hz) are plotted for each experiment. Number of days post-ablation are plotted on the abscissa. The data from the last five days constitute the assumed stable response period.


A
1.3
1.2
1.1
1.0
0.9
10
20
30
50
60
B
1.3
1.2
1.1
1.0
0.9
10
20
30
50
C
1.3
1.2
1.1
1.0
0.9
10
20
50
60
D
1.3
1.2
1.1
1.0
0.9
10
20
30
40
50
E
1.3
1.2
1.1
1.0
0.9
10
30
50
60
S
1.3
1.2
1.1
1.0
0.9
10
20

amplitude level and became less variable. As a measure of the amount of time needed for return of stable postural responses, the number of days necessary for the median gains at 0.20 and 0.30 Hz (from a single experiment) to fall within ± 10% of the stable five day median gain was calculated for each dog. Two dogs required 20 or more days for stable postural behavior; two other dogs needed an intermediate length of time; while one dog improved rapidly to a stable condition, as follows:

DOG:	A	B	C	D	E
	27 days	12 days	6 days	20 days	12 days

The sham-control dog demonstrated much more stable postural responses immediately after the sham operation. Since the beginning of post-operative testing varied slightly between dogs, a different measure of improvement was to tabulate the number of times the experiment was repeated before a stable level within ± 10% of the five day stable performance level was achieved. The same two dogs required the longest to achieve stable postural responses. The experiment beyond which each dog's gains remained within ± 10% of the stable gain is as follows:

DOG:	A	B	C	D	E	
	7th	3rd	3rd	7th	3rd	experiment

The gains of the sham-operated dog's postural responses were never greater than 10% of the stable value.

Therefore, post-operatively, the dogs demonstrated the greatest postural control deficit immediately and improved with repeated testing of the NP experiment. Dogs A and D required the longest length of time to attain stable postural control.

The gain ratios of BP experiments varied much less. In all experimental dogs, the first post-operative experiment produced postural responses which were already within 10% of the stable gain from the five day values. The response gains changed little with further testing. Thus, the postural responses in the BP condition remained relatively stable throughout the post-operative period. Since the BP experiments were begun a day or more later in most of the experimental dogs, it is possible that the dogs' postural control had already stabilized by the time of testing. To evaluate this possibility, the gain ratios of the first BP experiment for each dog can be compared with the actual or interpolated gain ratio for the NP experiment on that day. The gain ratios at the time of the first BP experiment compare as follows:

DOG:	A	B	C	D	E
BP:	1.07	1.04	1.05	1.09	1.03
NP:	1.17	1.10	1.19	1.17	1.05

Except for dog E, the NP median gains remain at least 10% larger than their respective five day stable gains, while the BP gains are all within 10% of their stable gain values. Thus, the postural responses during the BP condition were immediately more stable post-operatively than during the NP condition.

Postural Control With and Without Visual Feedback

The dogs' stable postural responses to the simple postural control problem presented by platform oscillation alone under restricted and blindfolded conditions were compared pre- and post-operatively.

In the figures for platform experiments, the median and the first to third interquartile range of the gain and relative phase of dog A's

postural responses are plotted for each of the seven frequencies of platform oscillation. When comparing two experiments, the data are presented on the same polar axis and consist of five days' experiments performed after the dog's performance had stabilized. The results of an analysis of variance (Fig. 20) on the gain of the dog's overall postural response for the two experimental conditions are presented above each plot in tabular form. If the analysis of variance indicates a significant difference at $p<0.01$, the results of a Student's t-test (Fig. 21) (comparing at individual frequencies the gains of the postural responses of two experimental conditions) are reported in the table. The two responses were considered significantly different if $p<0.005$ (noted by "$\pm$ .005"). Otherwise, an "NS" reflects no significant difference between the two postural responses.

Pre-operatively, the removal of all visual feedback resulted in a shift in dog A's postural control response ($p<0.01$) (Fig. 29). All five experimental dogs and the sham-control dog exhibited significant increases in the gain of their responses at almost all frequencies ($p<0.005$). Thus, pre-operatively the masking of all visual feedback resulted in an increased gain of the postural response evoked by platform oscillation.

Post-operatively, blindfolding continued to produce a shift in dog A's postural response ($p<0.01$), in the form of increased gains at all frequencies ($p<0.005$) (Fig. 30). Similar shifts occurred in all five experimental dogs and the sham-control dog, although at the highest frequencies dog S showed no significant increases in gain. Therefore, after lesions to the primary visual cortex, masking of all

Figure 29. Comparison of pre-operative postural responses from NP and BP experiments.

The table lists for all dogs the results of both an analysis of variance and a Student's t-test that compare the difference between mean gains of postural responses for the two experiments. If the analysis of variance indicates no significant difference at $p<0.01$, a single "NS" appears across from the dog letter; otherwise, the t-test is performed on the postural response gains at individual frequencies. A $p<0.005$ for the t-test is considered to be strongly suggestive of physiological significance. A "$\pm$" sign preceding "0.005" indicates the direction of any significant difference in (NP-BP) gains.

The polar plot illustrates the gain and phase of postural responses at all seven frequencies for the NP (open boxes) and BP (shaded boxes) experiments, in dog A. The median gain and phase are represented by the intersection of the cross hairs, while the first to third interquartile range is indicated by the ends of each cross hair.

	PLATFORM FREQUENCY						
DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	-.005	-.005	-.005	-.005	-.005	-.005	-.005
B	-.005	-.005	-.005	-.005	-.005	-.005	-.005
C	-.005	-.005	-.005	-.005	-.005	-.005	NS
D	-.005	-.005	-.005	-.005	-.005	-.005	-.005
E	-.005	-.005	-.005	-.005	-.005	-.005	-.005
S	-.005	-.005	-.005	-.005	-.005	-.005	NS

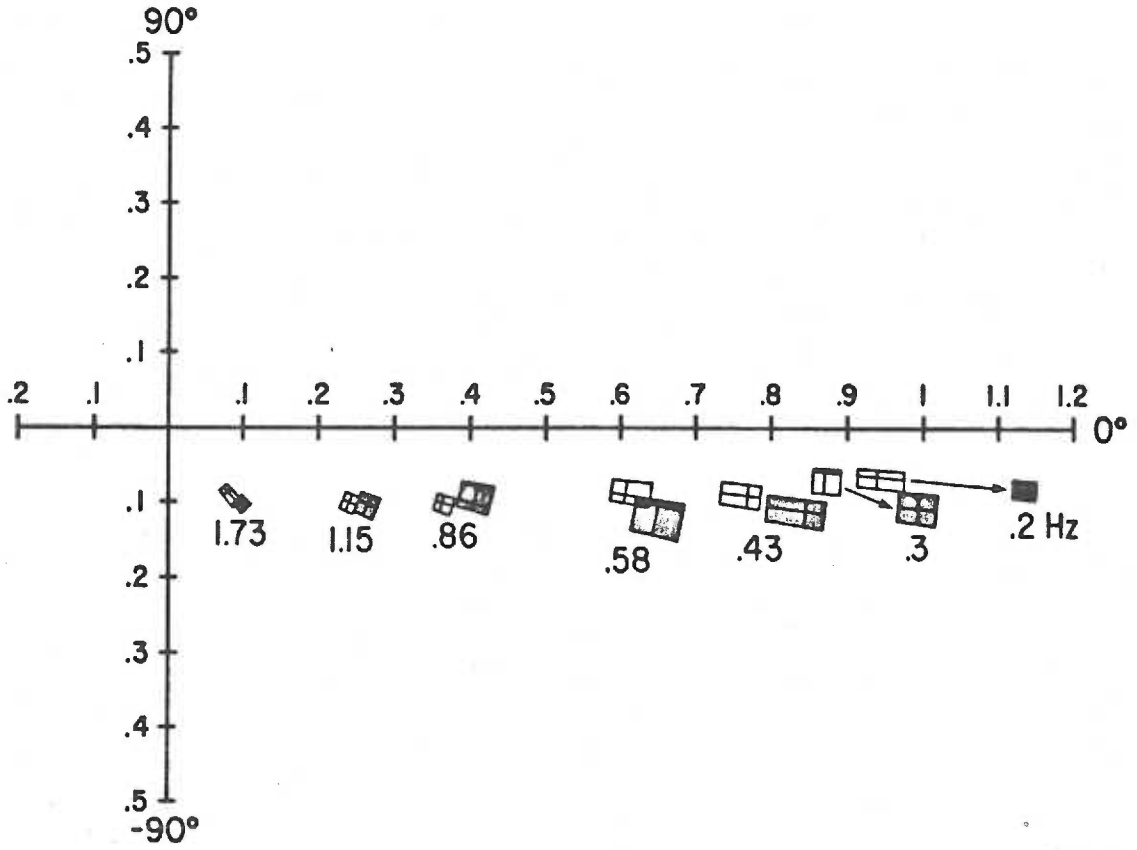


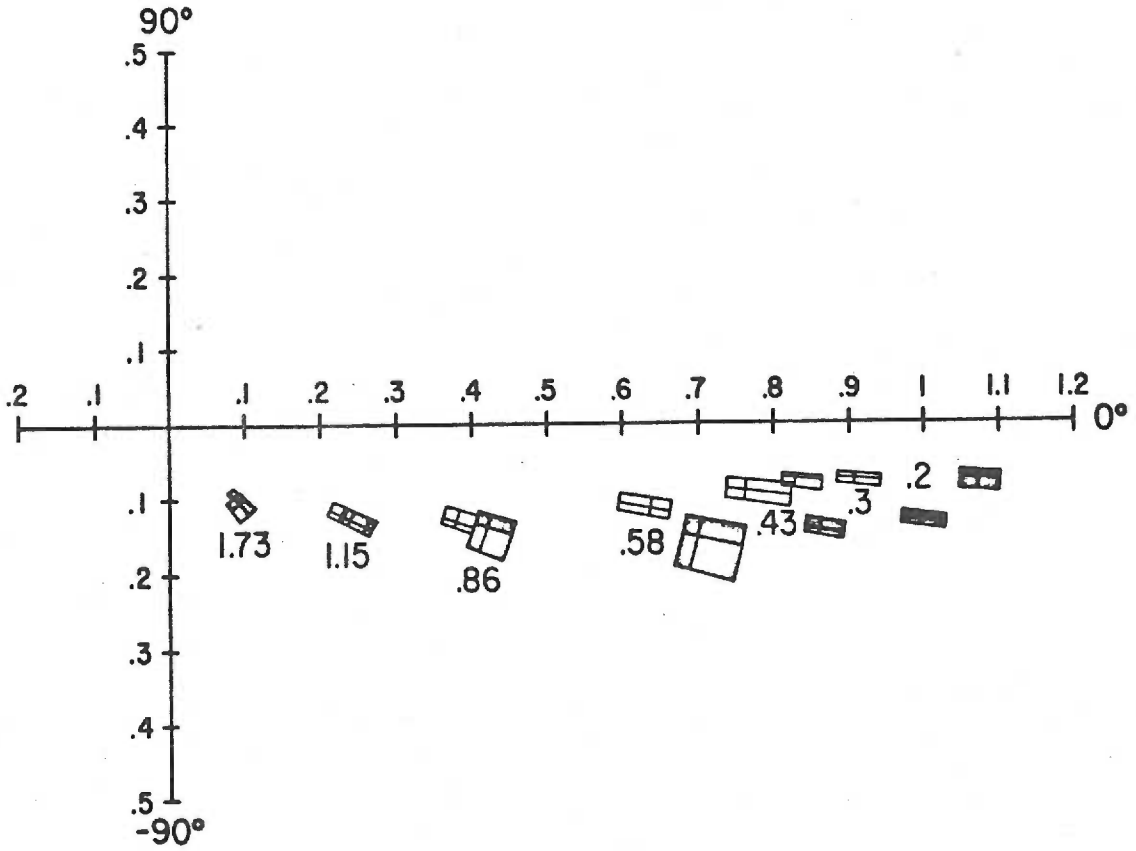
Figure 30. Comparison of post-operative postural responses from NP and BP experiments.

See Fig. 29 for an explanation of the results of the statistical tests summarized in the table. A "+" sign preceding "0.005" indicates the direction of a significant difference between experiments (NP - BP gains).

The polar plot illustrates the postural responses at all seven frequencies for the NP (open boxes) and BP (shaded boxes) experiments, in dog A. See Fig. 29 for an explanation of data points.

PLATFORM FREQUENCY

DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	-.005	-.005	-.005	-.005	-.005	-.005	-.005
B	-.005	-.005	-.005	-.005	-.005	-.005	-.005
C	-.005	-.005	-.005	-.005	-.005	-.005	NS
D	-.005	-.005	-.005	-.005	-.005	-.005	-.005
E	-.005	-.005	-.005	-.005	-.005	-.005	NS
S	-.005	-.005	-.005	NS	NS	NS	NS



visual feedback continued to effect the postural response to platform oscillation, reflected by increased gains of pelvis movement.

When pre- and post-operative postural control responses during unrestricted visual feedback were compared, no significant difference in the responses was present ($p<0.01$) (Fig. 31). However, the sham-control dog did change its postural control response at four of the seven frequencies. Thus, the stable postural control response to platform oscillation in the cortically ablated dogs is unchanged by lesions of visual cortex.

The postural control responses during blindfolding did show some change after the ablation of primary visual cortex, but not uniformly. Three dogs exhibited significant overall changes in the gain of their blindfolded postural control responses ($p<0.01$), while two dogs showed no significant change in overall postural control (Fig. 32). In the first three dogs, the direction of shift in the gains was uniformly toward smaller values post-operatively. The sham-operated control dog as well exhibited smaller gains post-operatively.

In summary, the effect on postural control of blindfolding was similar pre- and post-operatively; namely, a shift in the gains of postural control responses to larger values. Postural control capability during unrestricted visual feedback was unchanged by primary visual cortex removal. Postural control responses during blindfolding showed decreased gains post-operatively in three of five ablated dogs and no change in the others.

Postural Control During Selective Visual Restriction

The effects of central and peripheral visual field restriction

Figure 31. Comparison of pre- and post-operative postural responses from an NP experiment.

See Fig. 29 for explanation of the statistical tests whose results are summarized in the table. A "±" sign preceding "0.005" indicates the direction of any significant difference (Pre - Post) between the gain of responses from the two experiments.

The polar plot illustrates the postural responses at all frequencies for the NP experiment pre-operatively (open boxes) and post-operatively (shaded boxes), in dog A. Median and first to third interquartile range are represented by the intersection of cross hairs and the ends of cross hairs, respectively.

PLATFORM FREQUENCY

DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	----------			NS	----------		
B	----------			NS	----------		
C	----------			NS	----------		
D	----------			NS	----------		
E	----------			NS	----------		
S	NS	NS	-.005	NS	+.005	+.005	+.005

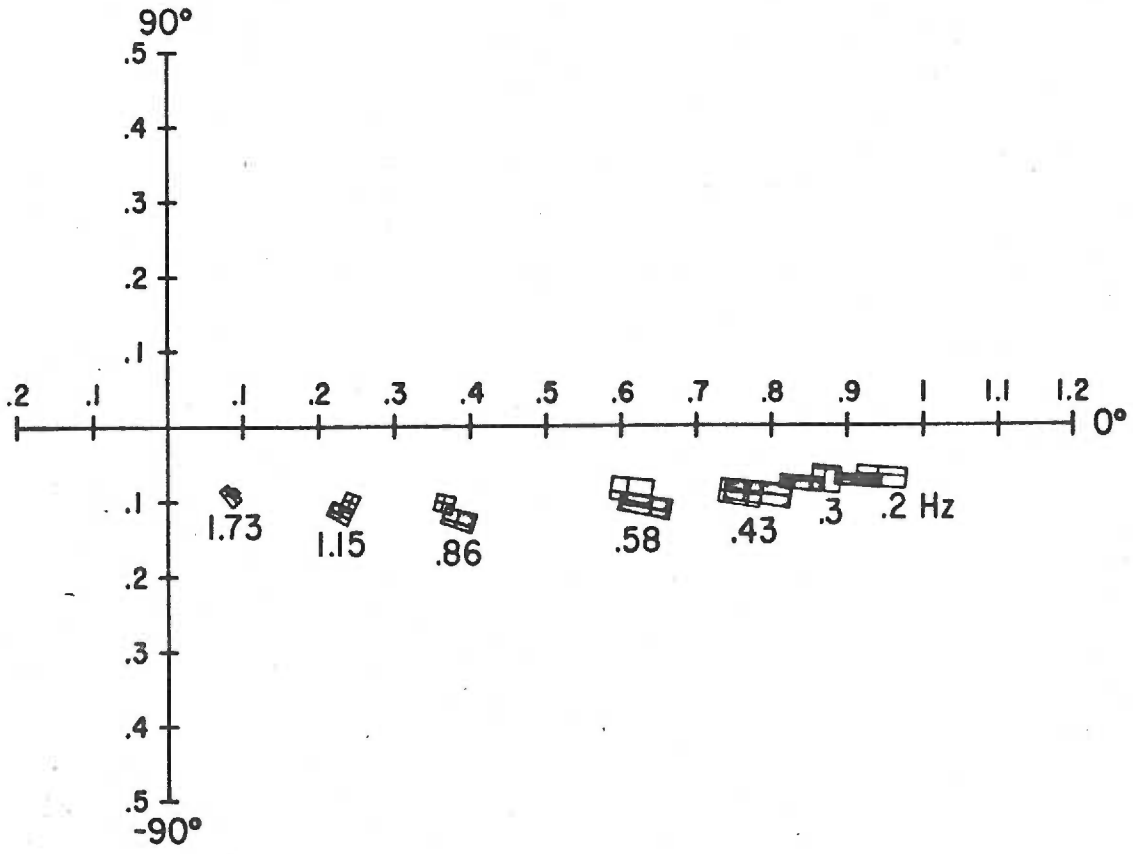


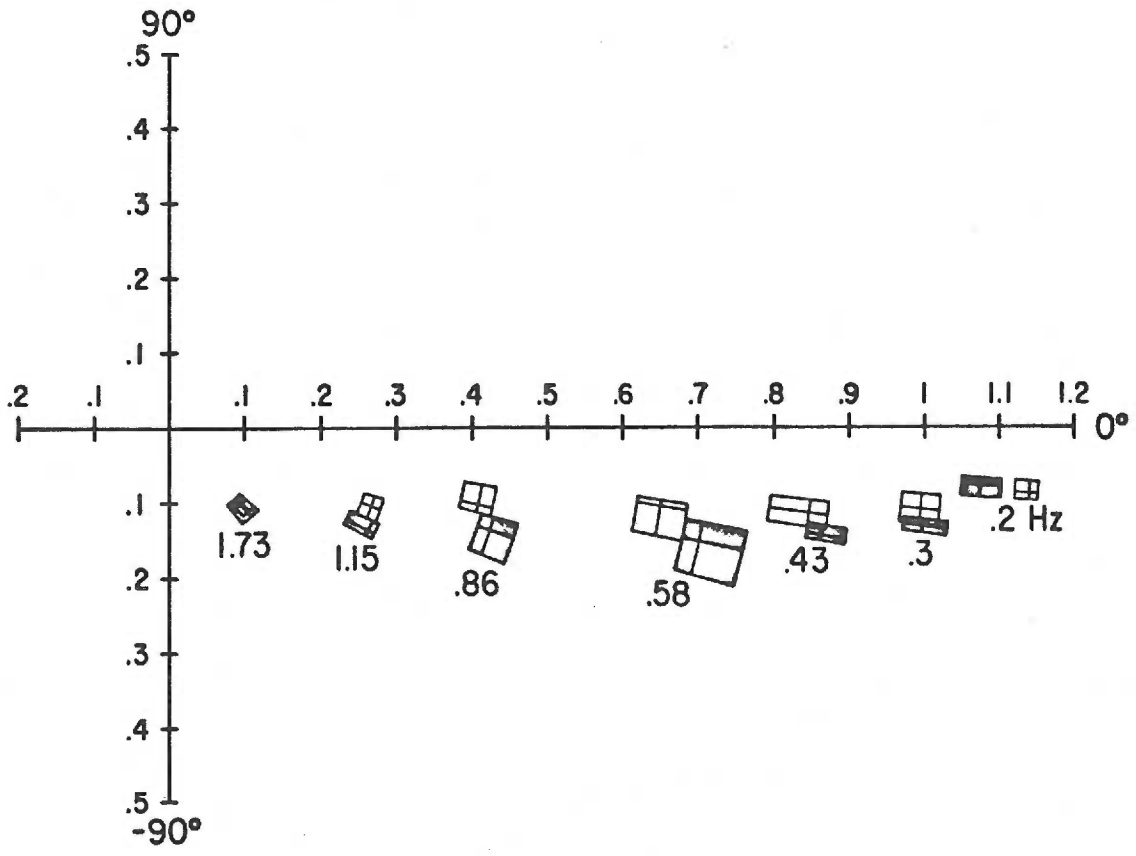
Figure 32. Comparison of pre- and post-operative postural responses from a BP experiment.

Fig. 29 explains the statistical test results summarized in the table. A "$\pm$" preceding "0.005" indicates the direction of a significant difference (Pre - Post) between the gain of responses from the two experiments.

The polar plot illustrates the postural responses at all frequencies for the BP experiment pre-operatively (open boxes) and post-operatively (shaded boxes), in dog A. Median and first to third interquartile range are represented by the intersection of cross hairs and the ends of cross hairs, respectively.

PLATFORM FREQUENCY

DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	----------	----------	----------	NS	----------	----------	----------
B	+.005	+.005	+.005	+.005	NS	+.005	+.005
C	+.005	NS	+.005	NS	NS	NS	NS
D	----------	----------	----------	NS	----------	----------	----------
E	+.005	+.005	+.005	NS	+.005	NS	+.005
S	NS	+.005	+.005	+.005	+.005	+.005	+.005



on postural control responses to platform oscillation were studied pre- and post-operatively.

Pre-operatively, the restriction of visual feedback to either the central visual field or peripheral visual field resulted in a shift of the gains of postural responses to a lesser degree than that produced by blindfolding. The relative effect of central versus peripheral visual restriction was not uniform for all dogs. In four of the five dogs, there was a significant difference in the overall postural response during peripheral versus central visual restriction ($p<0.01$) (Fig. 33). At the individual frequencies, two of the four dogs (B and C) exhibited uniformly larger gains when exposed to only central visual feedback, while the other two dogs (A and E) demonstrated larger gains with peripheral visual feedback alone. Thus, pre-operatively in four dogs, the removal of either central or peripheral visual field feedback produced an increase in the gain of postural control responses which was of smaller magnitude than blindfolding but bore no fixed relationship to one or the other area of visual field. Experimental dog D and the sham-control dog responded no differently to the platform oscillation whether centrally or peripherally restricted.

Post-operatively, postural responses were consistently different for central versus peripheral visual field restriction. Postural responses of all five lesioned dogs to platform oscillation were of larger gain during central visual feedback alone (Fig. 34). The sham-control dog showed no difference in postural response whether centrally or peripherally restricted. Thus, post-operatively, the blocking of peripheral visual field produced postural responses of greater gain than the

Figure 33. Comparison of pre-operative postural responses from COP and POP experiments.

See Fig. 29 for an explanation of the results of statistical tests summarized in the table. A "+" sign indicates the direction of a significant difference (COP - POP) between the gain of responses from the two conditions.

The polar plot illustrates the postural responses at all seven frequencies of the POP (open boxes) and COP (shaded boxes) experiments, in dog A. Median and first to third interquartile range are represented by intersection and ends of cross hairs, respectively.

PLATFORM FREQUENCY

DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	-.005	-.005	NS	NS	NS	NS	NS
B	+.005	NS	+.005	NS	NS	NS	NS
C	NS	+.005	NS	NS	NS	NS	NS
D	----------	----------	----------	NS	----------	----------	----------
E	-.005	-.005	-.005	NS	NS	NS	NS
S	NS	NS	NS	NS	NS	NS	NS

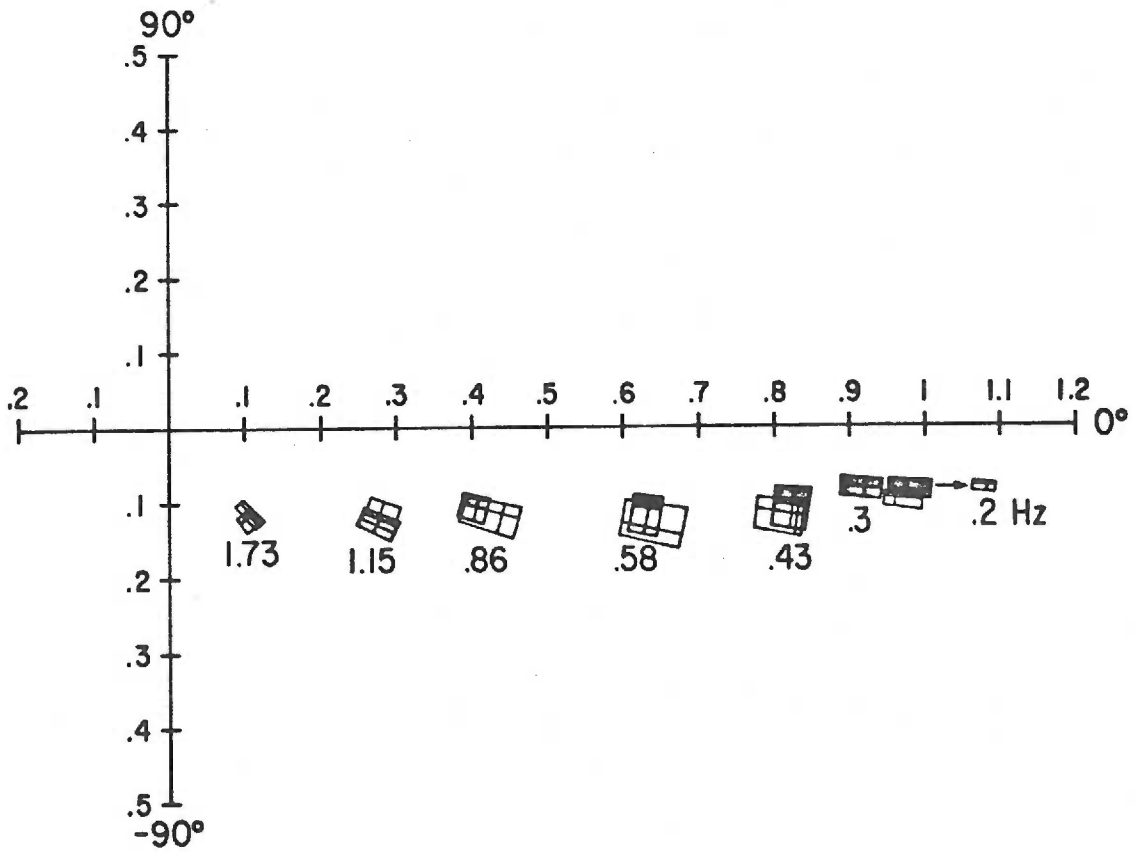


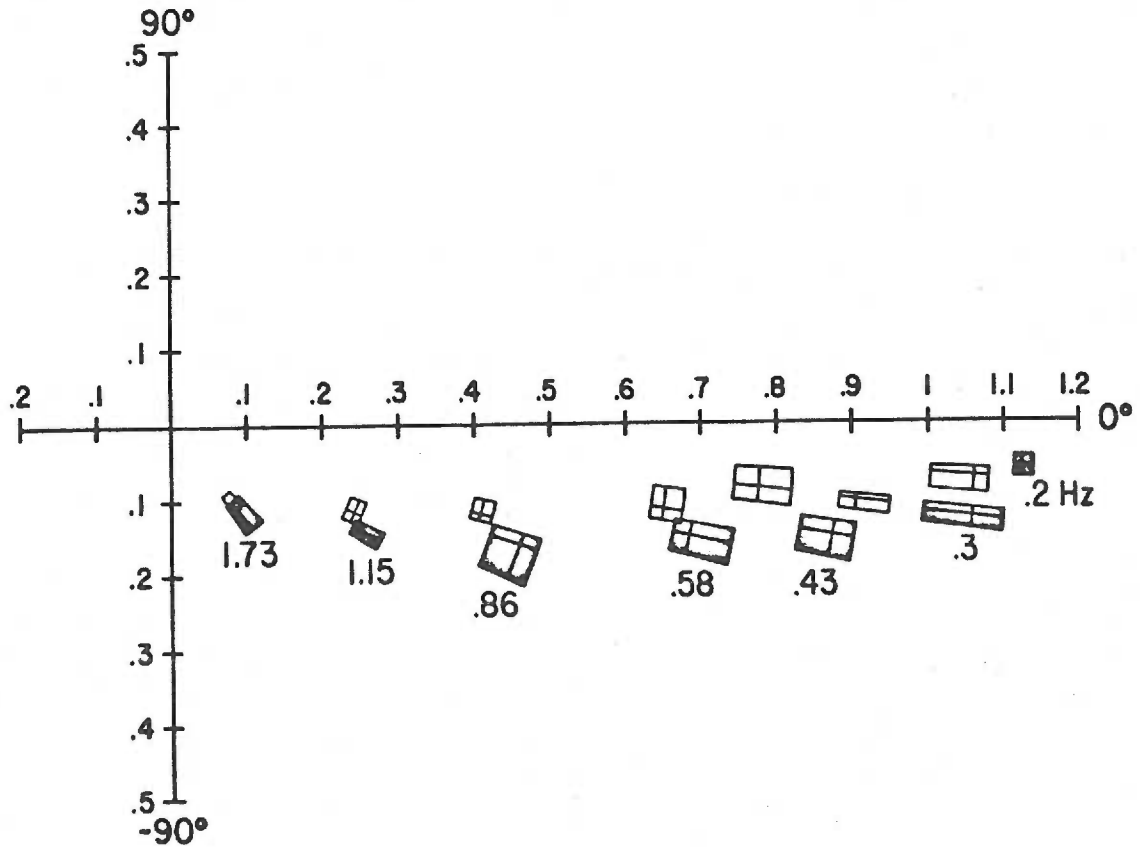
Figure 34. Comparison of post-operative postural responses from COP and POP experiments.

The results of statistical tests summarized in the table are explained in Fig. 29. A "+" indicates the direction of a significant difference (COP - POP) between the gain of responses from the two conditions.

The polar plot illustrates the postural responses at all frequencies of POP (open boxes) and COP (shaded boxes) experiments, of dog A. Median and first to third interquartile range are represented by intersection and ends of cross hairs, respectively.

PLATFORM FREQUENCY

DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	+.005	+.005	+.005	+.005	+.005	+.005	+.005
B	+.005	+.005	+.005	+.005	NS	NS	NS
C	+.005	+.005	+.005	+.005	+.005	+.005	+.005
D	+.005	+.005	+.005	+.005	+.005	+.005	+.005
E	+.005	+.005	NS	NS	+.005	NS	NS
S	----------			NS	----------		



blocking of central visual field. In fact, the gains of postural responses during central visual feedback alone post-operatively were similar or greater than during blindfolding in four of five dogs (Fig. 35). Only dog C and the sham-control dog continued to show smaller gains with central field restriction than with blindfolding, as occurred pre-operatively.

Therefore, the platform experiments evaluating postural control capability indicate that postural control responses during unrestricted visual feedback are unchanged by removal of primary visual cortex, and that blindfolding continues to produce a shift to larger gain postural responses. Pre-operatively, postural control responses during restricted central or peripheral visual field feedback are not uniformly different and both produce small shifts in gain as a result of the masking. Post-operatively, the blocking of peripheral visual field feedback produces uniformly greater increases in gain than the blocking of central visual field, approaching the increased gain that results from blindfolding. These results from the platform experiments are summarized in Table VI A.

Platform-roof Experiments

Visually-evoked Postural Responses Preceding Stable Response Patterns

The post-operative platform-roof experiments were not begun immediately after the surgery, as were the platform experiments. Following a recuperative period, each dog was repeatedly tested with the combined platform-roof motions. Once stable postural control behavior had been achieved, as indicated by the NP experimental data,

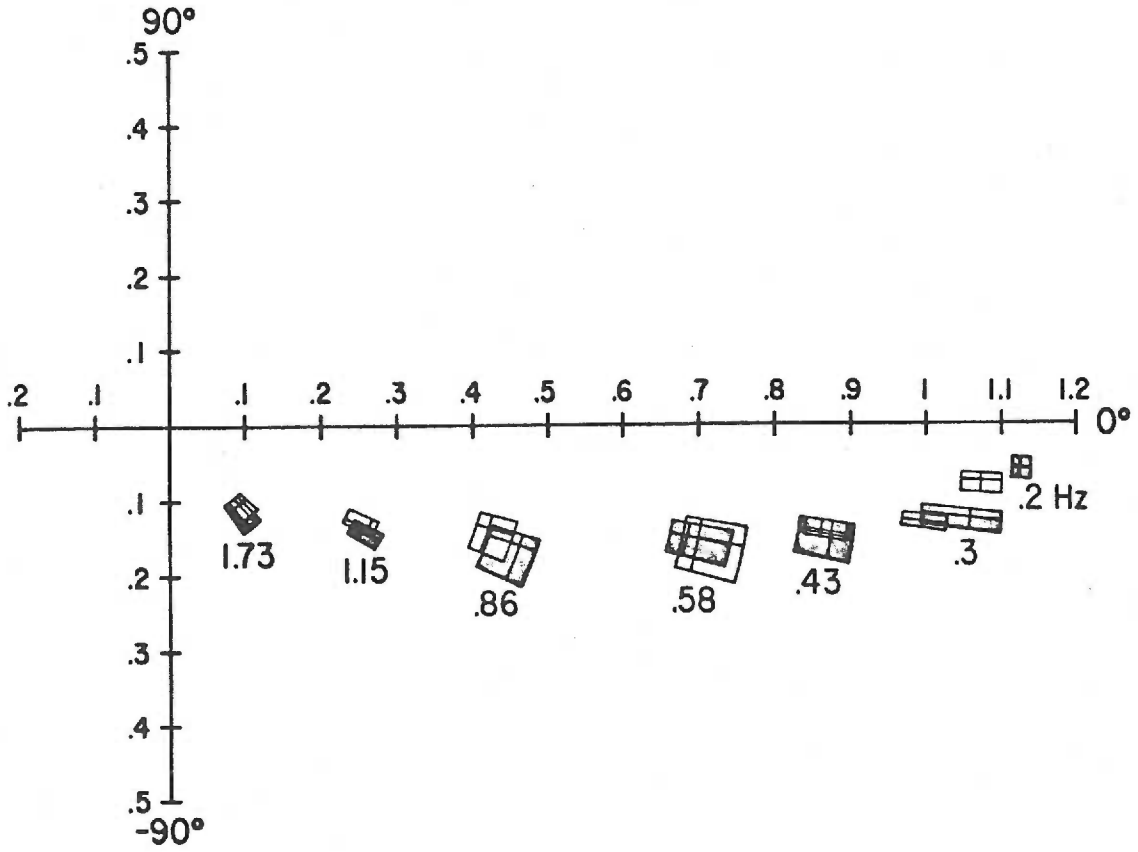
Figure 35. Comparison of post-operative postural responses from COP and BP experiments.

Fig. 29 explains the results of statistical tests summarized in the table. A "$\pm$" preceding "0.005" indicates the direction of a significant difference (COP - BP) between the gain of responses from the two conditions.

The polar plot illustrates the postural responses at all frequencies of the BP (open boxes) and COP (shaded boxes) experiments, in dog A. Median and first to third interquartile range are represented by intersection and ends of cross hairs, respectively.

PLATFORM FREQUENCY

DOG	0.20	0.30	0.43	0.58	0.86	1.15	1.73
A	+.005	NS	NS	NS	NS	NS	NS
B	+.005	+.005	NS	NS	NS	NS	NS
C	-.005	-.005	NS	NS	NS	NS	NS
D	NS	NS	NS	NS	NS	NS	NS
E	----------			NS	----------		
S	-.005	-.005	-.005	NS	-.005	NS	-.005



five days of data were collected from the different platform-roof experiments to describe stable post-operative visually-evoked postural behavior.

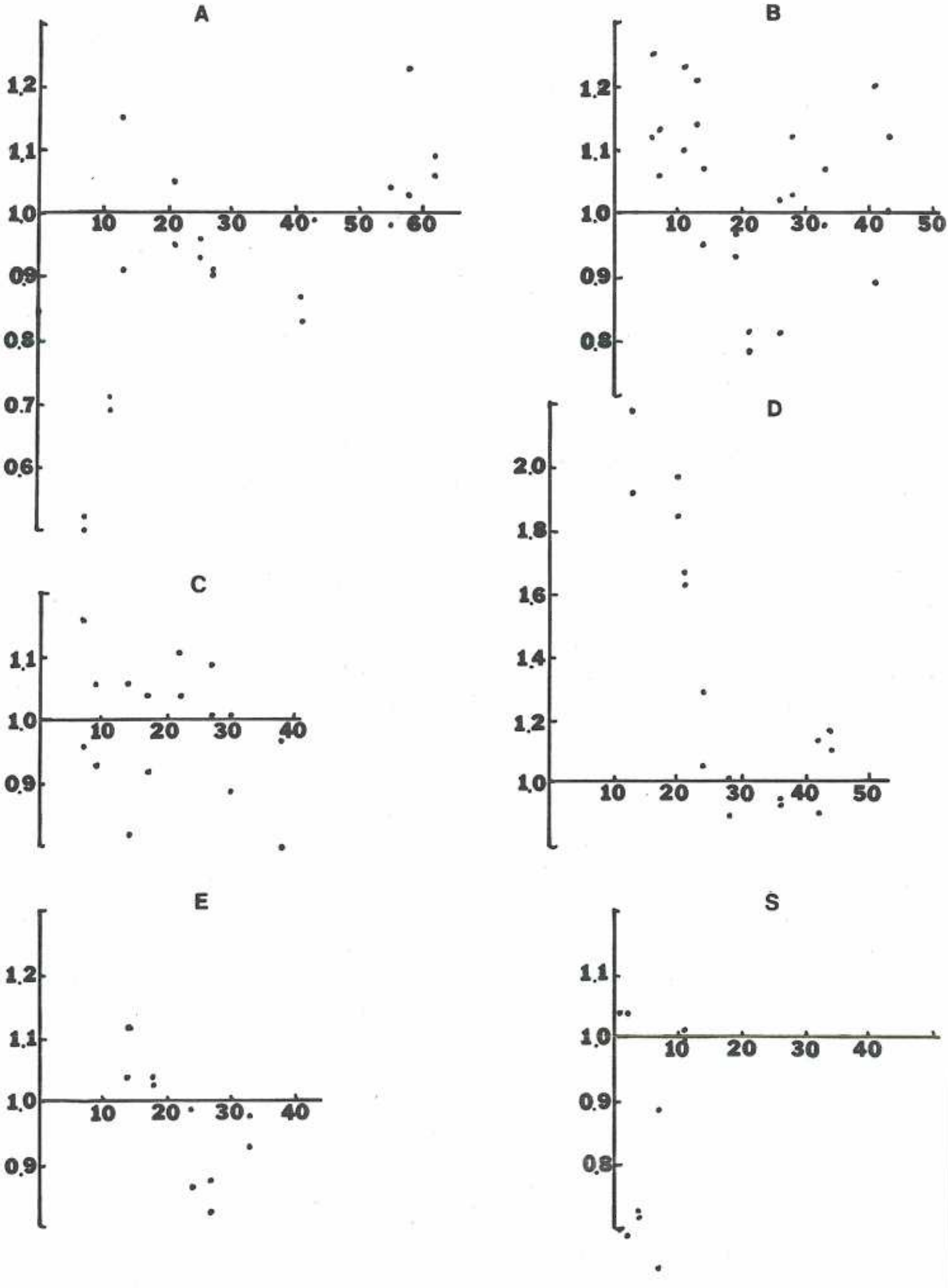
Fig. 36 displays for each dog the post-operative changes in gain ratios for postural responses that were evoked by 0.30 and 0.40 Hz roof oscillation during ongoing platform oscillation (i.e, NB experiment). The variability in the gain of visually-evoked postural responses even during stable postural control behavior was much greater than for the platform experiments -- approximately $\pm$ 20% compared to $\pm$ 10%. In addition, the progressive stabilization of the responses observed in the platform experiments was not obvious in the platform-roof experiments; rather, the visually-evoked responses remained more variable during the post-operative period. Dogs B, C and E showed essentially no change in the gain ratios, while dogs A and D stabilized somewhat at the onset but in opposite directions.

Overall, the dogs showed no definite trend throughout the post-operative period in the gains of their visually-evoked postural responses.

Peripheral Visually-evoked Postural Responses

The platform-roof experiments were designed to describe the postural motor responses selectively evoked by visual feedback on a background of platform movement. The gain of the postural response at the driving frequency of roof oscillation was utilized as a measure of the potency of visual input. In all following figures, the gains (i.e. median plus first to third interquartile range) for dog A are plotted for each of the five frequencies of roof oscillation in a Bode

Figure 36. Post-operative changes in the gain of postural responses evoked in NB experiments (by 0.30 and 0.40 Hz roof motion). The postural response gains from individual experiments are plotted as gain ratios, calculated by dividing the median gain from an individual experiment by the stable, five-day gain. Number of days post-ablation are plotted on the abscissa.


A
1.2
1.1
1.0
0.9
0.8
0.7
0.6
10
20
30
40
50
60
B
1.2
1.1
1.0
0.9
0.8
10
20
30
40
50
C
1.1
1.0
0.9
10
20
30
40
D
2.0
1.8
1.6
1.4
1.2
1.0
10
20
30
40
50
E
1.2
1.1
1.0
0.9
10
20
30
40
S
1.1
1.0
0.9
0.8
10
20
30
40

format. To facilitate comparison, the stable, five day data from two experiments are presented on the same plot. Again, the results of the analysis of variance (Fig. 20) and the Student's t-test (Fig. 21) for all dogs are given above each plot in tabular form. If the analysis of variance indicates a significant difference between the two overall responses ($p<0.01$), then the t-test is performed on comparisons of individual frequencies. Otherwise, a single "NS" is present next to the dog number.

The relative magnitude of the postural responses selectively elicited from the peripheral visual field was reduced after primary visual cortex ablation in four of five dogs ($p<0.01$), while the sham-control dog showed no change in its evoked response (Fig. 37). Inter-animal variability existed in the number of frequencies at which a post-operatively smaller response was evoked. Dog A showed unchanged gains at four of five frequencies and a greater gain at one frequency, post-operatively. Dogs B, C and D demonstrated almost identical decreases in gain at most frequencies, while dog E showed decreased gains at only two frequencies.

Although four of the five dogs showed decreased gains in postural responses evoked post-operatively from the peripheral field, these motor responses were all larger than visually-undriven postural corrections elicited by primarily auditory stimuli. Fig. 38 compares the gains of post-operative postural responses evoked by peripheral visual field oscillation with and without a blindfold. In dogs A, C and S, visually-evoked response gains at the lowest frequency were not significantly different from the blindfolded response gains.

Figure 37. Comparison of pre- and post-operative postural responses evoked in POB experiments.

The table lists for all dogs the results of both an analysis of variance and a Student's t-test that compare the difference between mean gains of visually-evoked postural responses for the two sets of experiments. If the analysis of variance indicates no significant difference at $p<0.01$, a single "NS" appears across from the dog letter (as for dog S); otherwise, the t-test is performed on the postural response gains at individual frequencies. A $p<0.005$ for the t-test is considered to be strongly suggestive of physiological significance. A "$\pm$" sign preceding the "0.005" indicates the direction of any significant difference in (Pre - Post) gains.

The Bode plot for gain of postural responses illustrates the pre-operative (solid circles) and post-operative (open circles) results for dog A. The median gains for the two conditions are represented by the open and solid circles, while the respective first to third inter-quartile ranges are indicated by the dashed lines with wider crossbars and solid lines with narrower crossbars.

ROOF FREQUENCY

DOG	0.15	0.20	0.30	0.40	0.60
A	NS	-.005	NS	NS	NS
B	+.005	+.005	+.005	+.005	+.005
C	+.005	+.005	+.005	+.005	+.005
D	NS	+.005	+.005	+.005	+.005
E	NS	NS	NS	+.005	+.005
S	----------	----------	NS	----------	----------

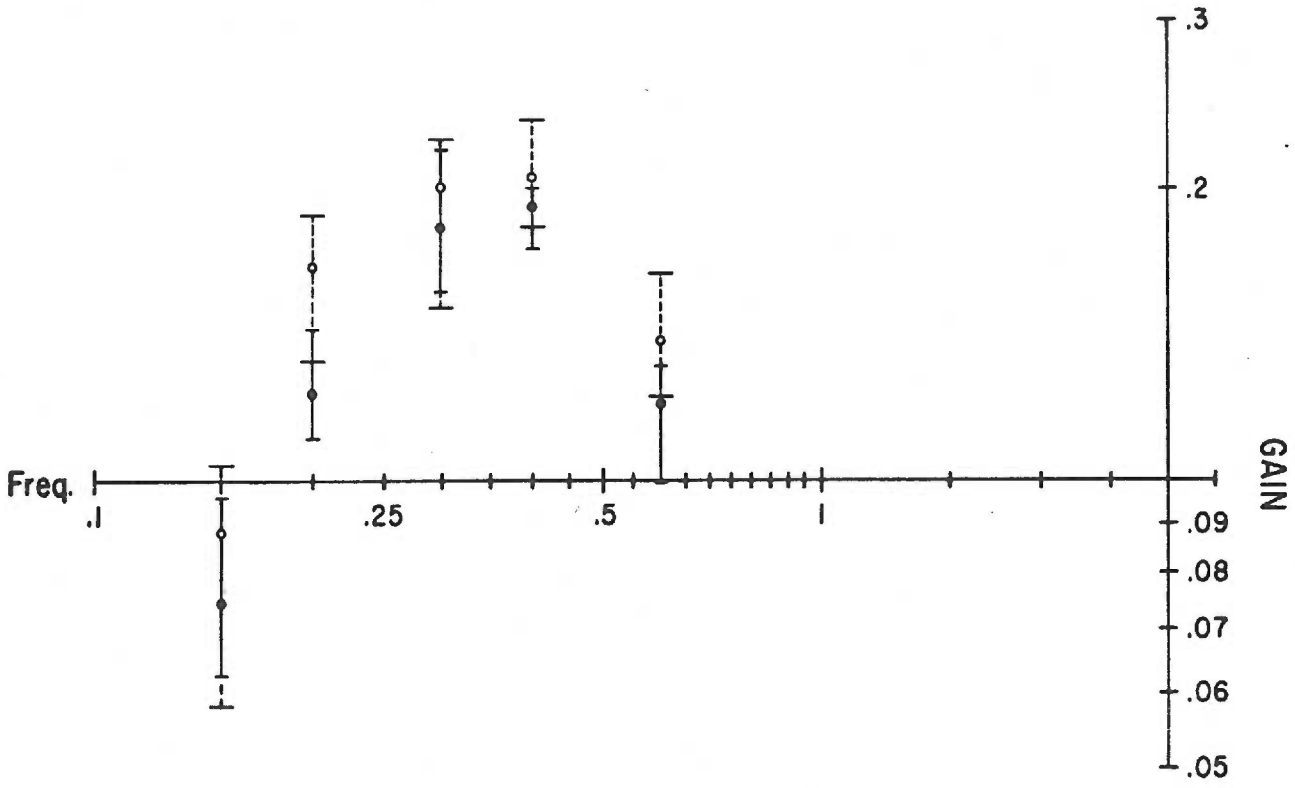
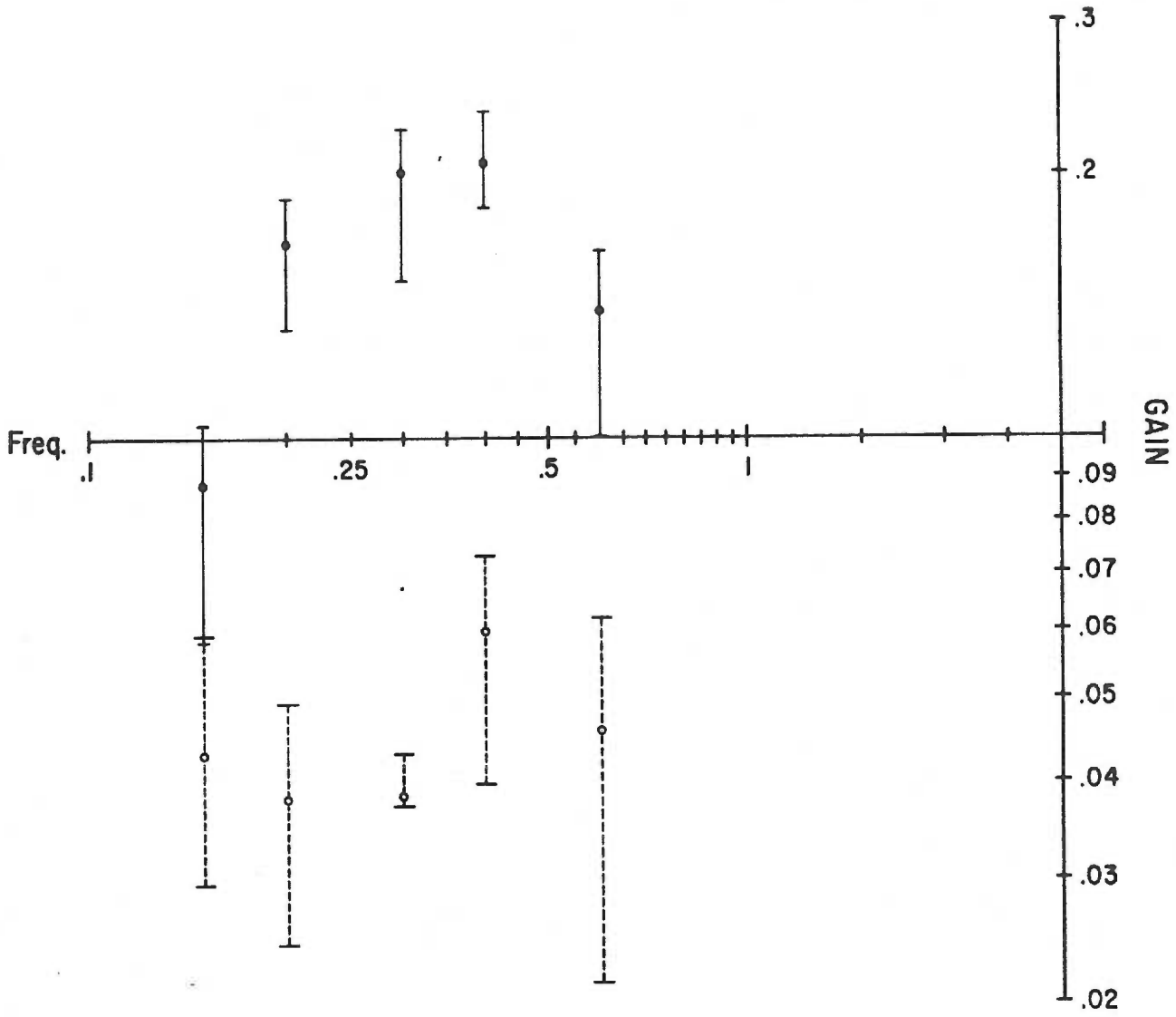


Figure 38. Comparison of post-operative postural responses evoked in POB and BB experiments.

See Fig. 37 for explanation of the statistical tests summarized in the table. A "±" indicates the direction of a significant difference (POB - BB) between the gain of responses evoked by the two conditions.

The Bode plot illustrates the POB (solid points) and BB (open, dashed points) results for dog A. Median gain is represented by a point, while first to third interquartile range is indicated by crossbars.

	ROOF FREQUENCY				
DOG	0.15	0.20	0.30	0.40	0.60
A	NS	+.005	+.005	+.005	+.005
B	+.005	+.005	+.005	+.005	+.005
C	NS	+.005	+.005	+.005	+.005
D	+.005	+.005	+.005	+.005	+.005
E	+.005	+.005	+.005	+.005	+.005
S	NS	+.005	+.005	+.005	+.005



However, all other frequencies of peripheral visual field motion evoked significantly larger postural responses.

Thus, although the gain of visually-evoked postural responses from peripheral visual field oscillation decreased somewhat without primary visual cortex in four of five dogs, the post-operative visually-evoked responses were not abolished.

Effects of Conflicting Central Visual Field Feedback

The previous experiments dealing with peripheral visually-evoked postural responses quantified the dog's response at the roof's driving frequency, when visual feedback was restricted to only the peripheral visual field. When the dog was not restricted in its visual feedback during a platform-roof experiment, the feedback it received was non-homogeneous. Under these conditions, the peripheral visual field information included a component of movement at the roof frequency, while the central visual field (i.e, stationary, seated assistant surrounded by a black curtain) contained visual movement at the platform frequency alone. As a result, central visual field feedback was in conflict with peripheral visual field feedback that evoked the postural responses.

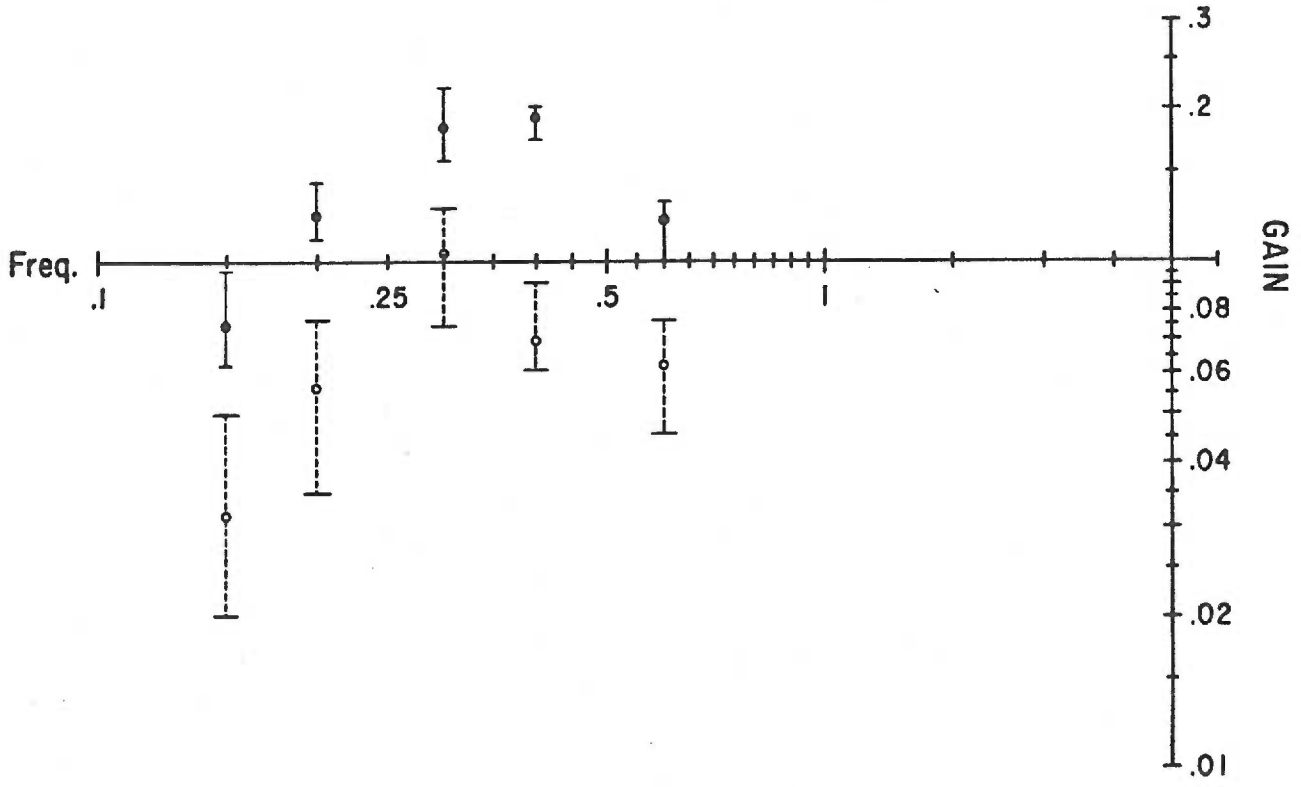
Pre-operatively, the gains of responses evoked by peripheral field movement were depressed when motion information from the central visual field was included (Fig, 39). This inhibitory influence of conflicting central field input on the gain of visually-evoked postural responses was present at all frequencies tested ($p < 0.005$) and in all five experimental (plus sham control) dogs.

Figure 39. Comparison of pre-operative postural responses evoked in NB and POB experiments.

Fig. 37 explains the statistical tests summarized in the table. A "$\pm$" preceding the "0.005" indicates the direction of a significant difference (NB - POB) between the gain of responses evoked by the two conditions.

The Bode plot illustrates the POB (solid points) and NB (open, dashed points) results for dog A. Median gain is represented by a point, while first to third interquartile range is indicated by crossbars.

	ROOF FREQUENCY				
DOG	0.15	0.20	0.30	0.40	0.60
A	-.005	-.005	-.005	-.005	-.005
B	-.005	-.005	-.005	-.005	-.005
C	-.005	-.005	-.005	-.005	-.005
D	-.005	-.005	-.005	-.005	-.005
E	-.005	-.005	-.005	-.005	-.005
S	NS	NS	NS	-.005	-.005



Post-operatively, however, this effect was completely abolished. Two of the five operated dogs showed no overall difference in the gains of peripheral visually-evoked responses with or without inclusion of the conflicting central field input; the other three dogs showed no difference at some frequencies, and actually greater responses at other frequencies when the conflicting central field information was present (Fig. 40). The control dog continued to exhibit a decrease in gain, when the conflicting central input was included.

Thus, the removal of primary visual cortex interfered with the normal integration of central and peripheral visual field feedback in postural control. Specifically, central visual field information no longer affected the visually-evoked response from the peripheral visual field.

Central Visually-evoked Postural Responses

Direct testing of postural responses evoked from the central visual field required modification of the roof by the addition of a front panel to create an open-ended room surrounding the dog (see Fig. 8). The top edge of the room was open to facilitate rewarding of the dog, but provided no visual cues due to the black curtain surrounding the front of the roof. Restriction of the dog's vision to the central field allowed selective study of visually-evoked postural responses resulting from central field motion. Motion in the central visual field differed from that in the peripheral visual field in that it involved changes in depth.

The gains of centrally-elicited postural responses changed dramatically after visual cortex ablation. Fig. 41 shows the large

Figure 40. Comparison of post-operative postural responses evoked in NB and POB experiments.

Fig. 37 explains the statistical tests summarized in the table. A "$\pm$" indicates the direction of a significant difference (NB - POB) between the gains of responses evoked by the two conditions.

The Bode plot illustrates the POB (solid points) and NB (open, dashed points) results, for dog A. Median gain is represented by the point, while first to third interquartile range is indicated by cross-bars.

	ROOF FREQUENCY				
DOG	0.15	0.20	0.30	0.40	0.60
A	----------	----------	NS	----------	----------
B	NS	NS	+.005	+.005	NS
C	----------	----------	NS	----------	----------
D	NS	+.005	NS	NS	NS
E	NS	+.005	+.005	NS	NS
S	NS	NS	-.005	-.005	-.005

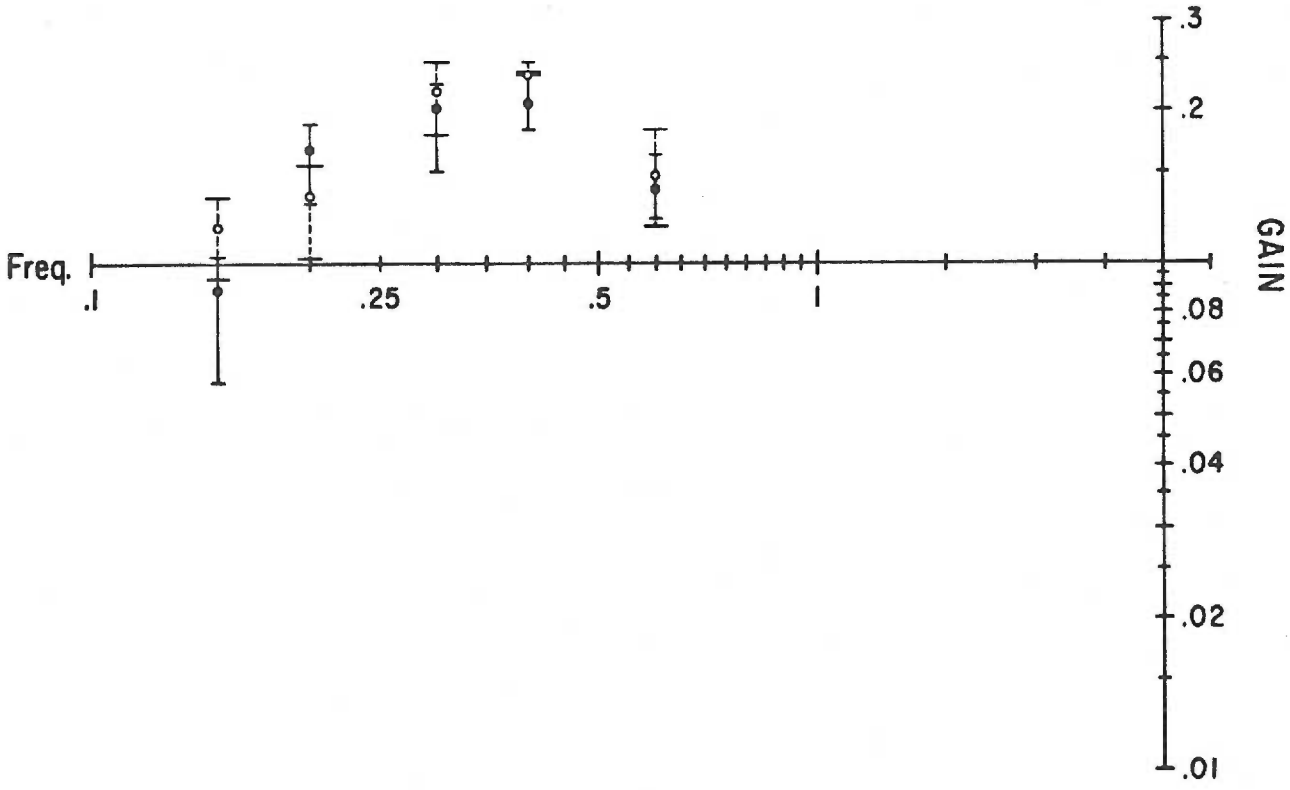
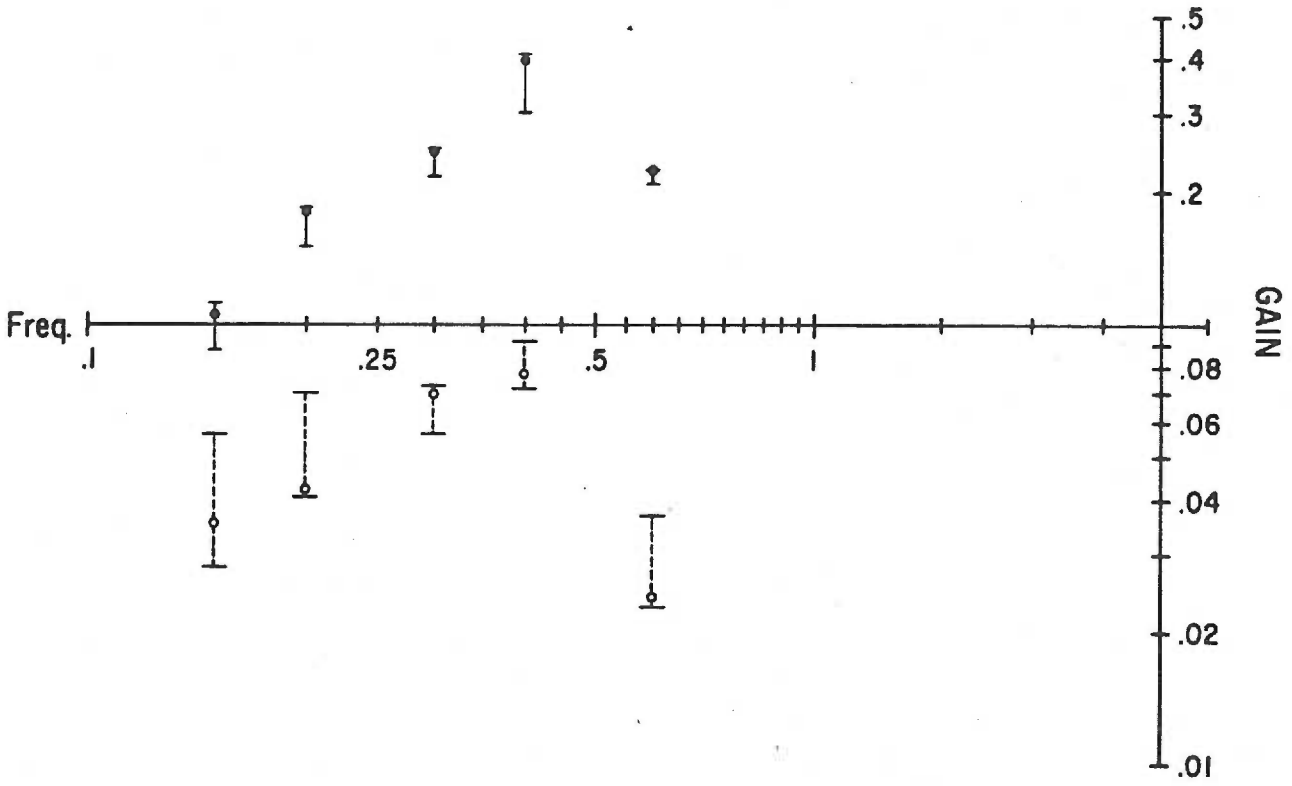


Figure 41. Comparison of pre- and post-operative postural responses evoked in COB' experiments.

See Fig. 37 for explanation of the statistical tests summarized in the table. A "±" indicates the direction of a significant difference (Pre - Post) between the gain of responses evoked in the two sets of experiments.

The Bode plot illustrates the pre-operative (solid points) and post-operative (open, dashed points) COB' results, for dog A. Median gain is represented by a point, while first to third interquartile range is indicated by crossbars.

	ROOF FREQUENCY				
DOG	0.15	0.20	0.30	0.40	0.60
A	NS	+.005	+.005	+.005	+.005
B	+.005	+.005	+.005	+.005	+.005
C	+.005	+.005	+.005	+.005	+.005
D	+.005	+.005	+.005	+.005	+.005
E	+.005	NS	+.005	+.005	+.005
S	----------	----------	NS	----------	----------



decrease in gain of central visually-evoked postural responses that resulted from removal of primary visual cortex in all five experimental dogs. The sham-control dog, on the other hand, did not change the gain of its postural responses to the central field motion. Comparison of the post-operative gains of the postural responses elicited by central field motion with and without the blindfold reveals no difference in the responses of four of the five operated dogs (Fig. 42). One experimental dog (C) and the sham-control dog responded with an overall larger postural response to the central visual field movement when not blindfolded ($p<0.01$).

Therefore, following primary visual cortex ablation, the gain of postural responses elicited by longitudinal oscillation of the central visual field is decreased to values no different from the gain of visually undriven postural responses.

Summary of Results

The effect of removal of primary visual cortex on the postural control system was approached by studying specific effects on visually-guided behaviors, on orienting responses tested perimetrically, on postural control capability testing by platform experiments, and on visually-evoked motor responses tested in platform-roof experiments.

Post-operative changes in visually-guided behaviors occurred to a similar degree in three of five experimental dogs. Dogs A, B and D exhibited the following post-ablative changes: loss of both blink reflex to threatening visual stimuli and visual placing response to a step or ledge; infrequent following and diminished orienting responses; poor localization and identification of stationary objects; and markedly

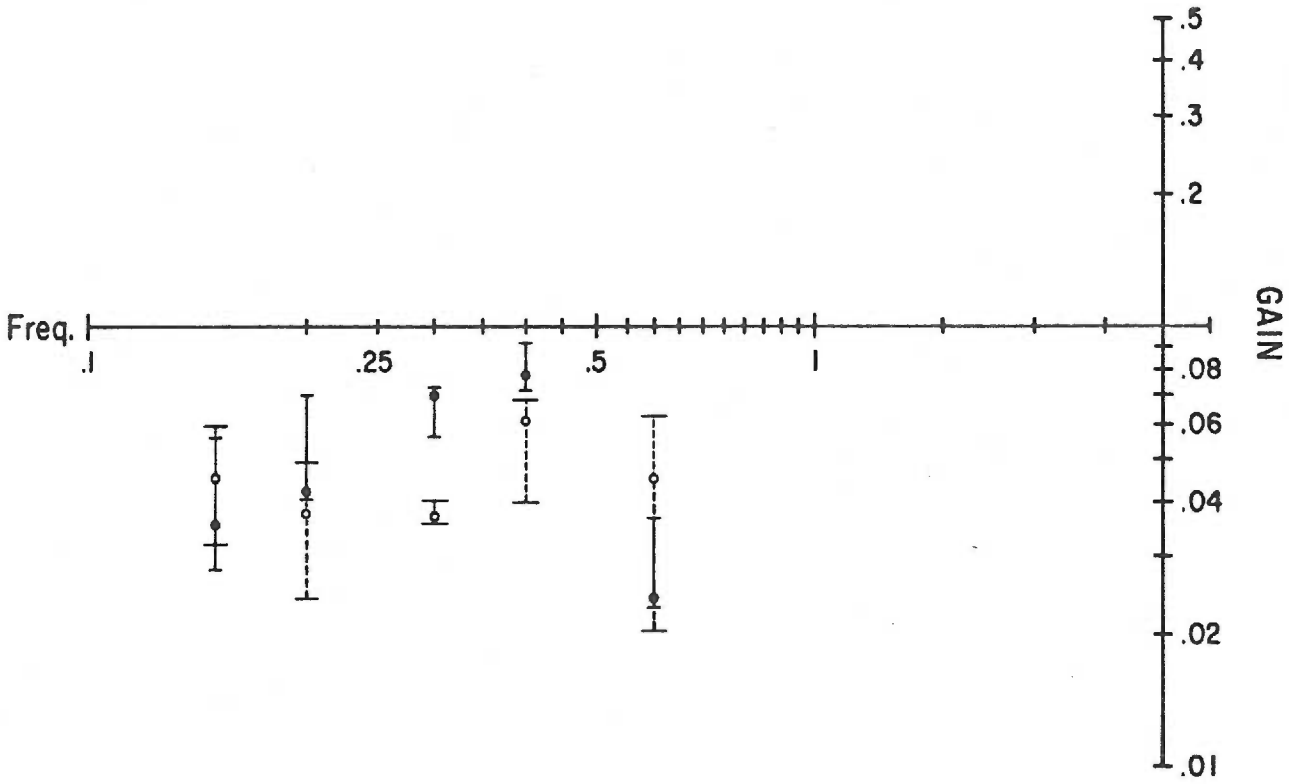
Figure 42. Comparison of post-operative postural responses evoked in COB' and BB experiments.

Fig. 37 explains the statistical tests summarized in the table. A "+" indicates the direction of a significant difference (COB' - BB) between the gains of responses evoked by the two conditions.

The Bode plot illustrates the COB' (solid points) and BB (open, dashed points) results, for dog A. Median gain is represented by the point, while first to third interquartile range is indicated by cross-bars.

ROOF FREQUENCY

DOG	0.15	0.20	0.30	0.40	0.60
A	----------	----------	NS	----------	----------
B	----------	----------	NS	----------	----------
C	NS	NS	NS	NS	+.005
D	----------	----------	NS	----------	----------
E	----------	----------	NS	----------	----------
S	NS	NS	NS	NS	NS



diminished avoidance behavior. Dogs C and E also lost the blink reflex to visual threat, but retained slowed visual placing responses and stimulus localization ability, with visual following, orienting and avoidance behaviors approaching normal values.

The use of perimetry in quantifying orienting responses, while uncovering a general pattern of deficit, supported the observation that orienting was not abolished by visual cortex ablation. The general pattern of deficit in orienting behavior was manifested to varying degrees by the five lesioned dogs. The lateral limits of visual field were unchanged and averaged 120° to each side. The average percentage of elicitable orienting responses decreased overall by between 5% and 70%, with a greater decrease for the central visual field in all dogs. The spontaneous level of orienting responses increased by 10% to 20%.

Platform experiments revealed that: gains of postural control responses were unchanged by cortical ablation when visual feedback was unrestricted; blindfolding produced an increase in the gains of postural responses both pre-and post-operatively; restriction of visual feedback to either the central or peripheral visual field had a similar effect on postural responses pre-operatively (i.e. producing an increase in gain, though less marked than that produced by blindfolding), but produced dissimilar deficits post-operatively (i.e. a greater gain occurred with central field feedback alone than with peripheral field feedback alone); and that post-operatively, the effect of restriction to central field feedback alone approached the increase in gain produced by blindfolding. These results are summarized in Table VI A.

Visually-evoked postural responses were elicitable by longitudinal, peripheral field movement both pre- and post-operatively, although the post-operative responses were of smaller amplitude. The varying degree to which longitudinal movement in the central visual field was able to elicit motor responses pre- and post-operatively was clear-cut: pre-operatively, the responses were of similar gain to those of peripherally-evoked responses; post-operatively, however the centrally-evoked responses were no different from blindfolded, noise-level responses. This clear-cut difference pre- and post-operatively was also manifested by the inability of central visual field feedback to depress the gain of peripheral visual field evoked postural responses after visual cortex ablation. The results of platform-roof experiments are summarized in Table VI B.

These four different approaches have provided a great deal of data concerning the role played by primary visual cortex in visual feedback for postural control.

DISCUSSION

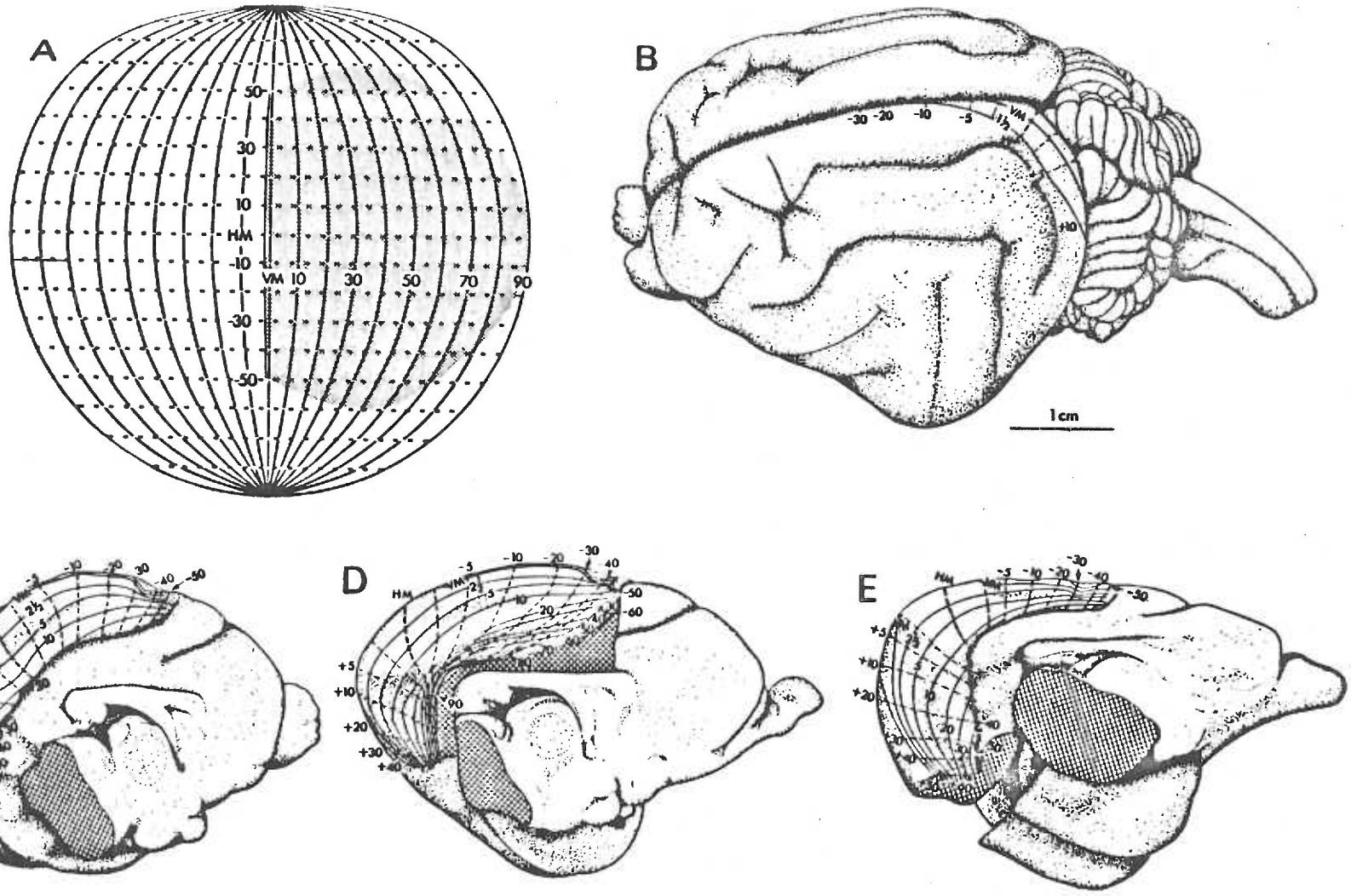
Completeness of Visual Cortical Removal

Drawings of the dorsal topography of the cerebral hemispheres and serial reconstructions of the occipital pole provide a detailed illustration of the ablation produced in each dog (summarized in Table IV). The visuotopic representation of any spared visual cortical areas must be defined in order to validly determine post-operative utilization of peripheral versus central visual field input. Information on the dog's visuotopic projection to both dorsal lateral geniculate nucleus (LGN_d) and primary visual cortex (areas 17 and 18) has not been reported in the literature; therefore, the general patterns reported for the cat will be utilized, based on the known similarities between cat and dog visual cortex and LGN_d, described earlier (see Fig. 4 and 6).

The specific visuotopic projection onto primary visual cortex has been repeatedly confirmed since the introduction of modern electrophysiologic recording techniques (106,107,112,116). The contralateral upper visual field is represented in the caudal part of visual areas 17 and 18; the contralateral lower visual field is represented rostrally; the fovea projects onto the dorsal surface of lateral gyrus in its posterior part; and, the lateral visual field is represented in the depths of the midline suprasplenial and splenial gyri. Fig. 43 illustrates the representation of the contralateral visual field on cat's visual cortex (112).

The retinotopic organization of the LGN_d was originally identified using Marchi degeneration techniques (64). Recently, electrophysiologic

Figure 43. Representation of visual field in area 17 of cat. (A) Perimeter chart showing the extent of visual field represented in area 17. Isoelevations are drawn as dashed lines and isoazimuths are drawn as solid lines. Location of visual field in area 17 is illustrated in drawings of a dorsolateral view (B), a posteromedial view (C), a medial view with cingulate gyrus removed to exposure superior bank of splenial gyrus (D), and a ventromedial view with hippocampal formation removed to expose posterior part of superior bank of splenial gyrus (E) (112).

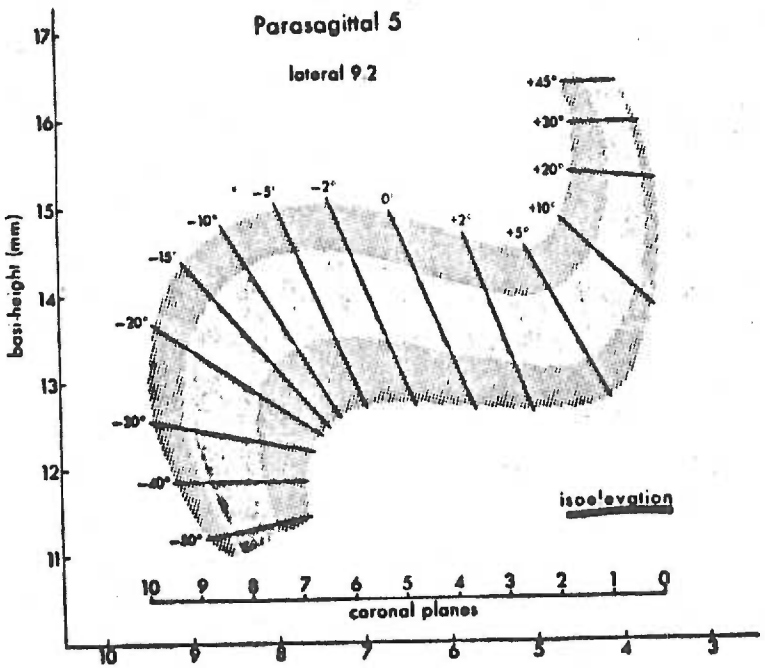

A
50
30
10
HM
-10
VM 10
30
50
70
90
-30
-50
B
-30 -20 -10 -5
VM
+10
1 cm
C
D
HM
VM
+5
+10
+20
+30
+40
-50
-60
E
HM
VM

sampling of the LGN_d has provided a more precise map of the visuotopic representation in cat's LGN_d (82). Fig. 44 presents three coronal sections through the cat's LGN_d equatable with the levels presented for the dog in this investigation (Fig. 26). Although in sagittal section the S-shaped LGN_d appears to be more vertically oriented in the dog, the following general visuotopic relationships are considered valid: the upper visual field is represented dorsally and posteriorly in the nucleus; the lower visual field is represented ventrally and anteriorly; and, the lateral visual field projects onto lateral parts of the nucleus. Accordingly, the rostral part of primary visual cortex connects with the anterior part of the LGN_d, and the caudal part of visual cortex with the posterior part of the nucleus (66,71). Medial parts of laminae A, A_1 and B project to the dorsal surface of the lateral and postlateral gyri; while progressively more lateral parts of the LGN_d relate to increasingly more ventral parts of the medial surface of the postlateral, suprasplenial and, ultimately, splenial gyri (71).

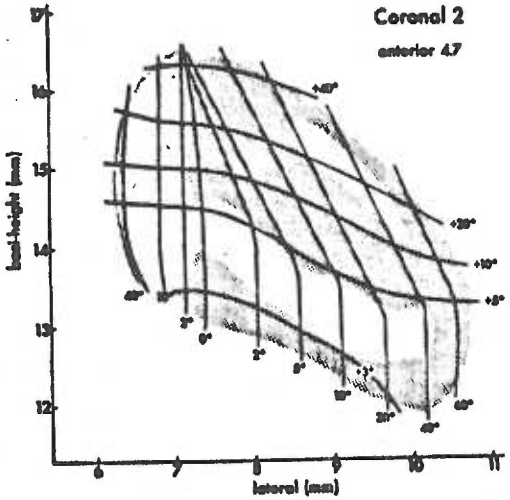
Interpretation of the pattern of retrograde atrophy in the LGN_d following visual cortex ablation is complicated by several factors. Complete ablation of area 17 in the cat does not result in total degeneration of the dorsal part of the nucleus (64,66). The spared dorsal area corresponds largely to lamina B, which projects also to cortical areas outside of primary visual cortex and to other thalamic nuclei (10). Additionally, only large and medium sized neurons are considered to project directly to primary visual cortex (89). The small diameter cells of laminae A and A_1 in the cat are considered to

Figure 44. Projection of visual field onto cat's dorsal lateral geniculate nucleus. (A) Pattern of isoelevations in a parasagittal section through the middle of the LGN_d. (B) Pattern of visual field isoelevations (given in signed degrees) and isoazimuths (given in unsigned degrees) in a coronal section through the posterior part of the LGN_d. (C) Pattern of isoelevations and isoazimuths in a coronal section through the middle part of the LGN_d. (D) Pattern of isoelevations and isoazimuths in a coronal section through the anterior part of the LGN_d (82).

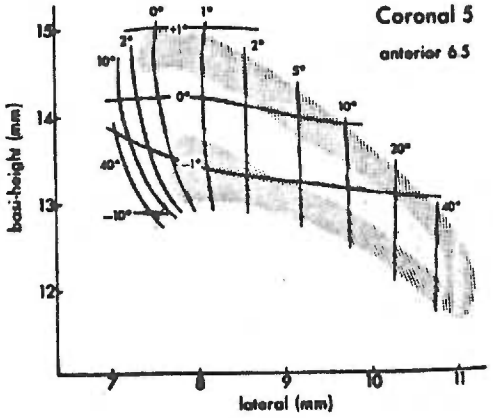
(A)


Parasagittal 5
lateral 9.2
basi-height (mm)
isoelevation
coronal planes

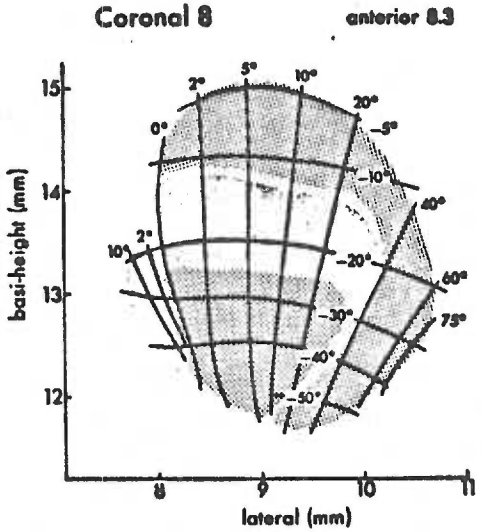
(B)


Coronal 2
anterior 4.7
lateral (mm)

(C)


Coronal 5
anterior 6.5
basi-height (mm)
lateral (mm)

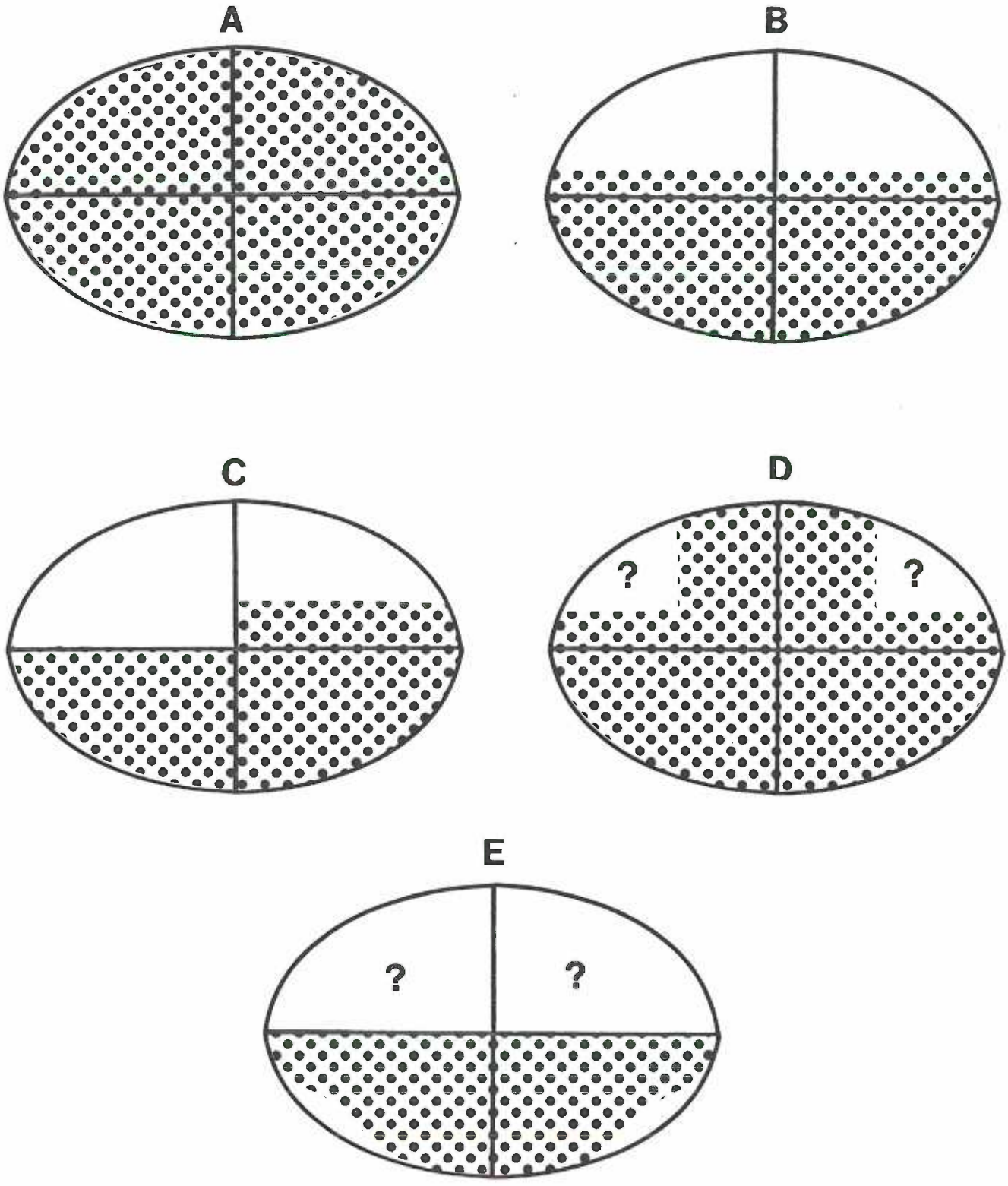
(D)


Coronal 8
anterior 8.3
basi-height (mm)
lateral (mm)

provide intrageniculate and intrathalamic connections, since the geniculostriate fibers are generally fast-conducting large and medium size neurons (10). Therefore, these small, short-axon cells are postulated to serve as interneurons (11) and should remain intact because their axons are not damaged. Large diameter neurons project to both area 17 and area 18 and require the ablation of both cortical areas to undergo complete retrograde atrophy; medium size neurons degenerate in response to striate cortex (area 17) ablation alone (31).

Dogs A and D received the most extensive ablations of primary visual cortex (see Table IV). Dog A possessed no intact representation of any part of the visual field. Specifically, serial reconstruction revealed no well-defined remnants of visual cortical tissue. This was supported by the complete retrograde atrophy of the LGN_d, except for the dorsalmost part of lamina B which projects to areas outside of primary visual cortex. Dog D may have retained some intact representation of the very lateral parts of the upper visual field, but the evidence is unclear. The uncertainty arises from the <u>partial</u> sparing of a cluster of medium and small sized neurons in the lateral half of the posterior level of the LGN_d, bilaterally. However, since atrophy of the large size neurons in this area did occur, the degree of functional significance ascribable to the visual representation of this area is questionable. Thus, dog D did not retain a <u>completely</u> intact representation of any area of visual field, although <u>partially</u> intact cortical representation of very lateral parts of upper visual field is probable. Fig. 45 schematically summarizes the areas of

Figure 45. Summary of approximate areas of intact visual field based on the analysis of retrograde atrophy in the LGN_d and on the serial reconstruction of each dog's lesion.


A
B
C
D
?
?
E
?
?
ablated

visual field that may have been partially spared in dog D.

Dogs B, C and E showed considerably more substantial anatomical evidence for sparing. In each of these three dogs some part of laminae A or A_1 exhibited no atrophy whatsoever. This represents presumptive evidence for functional integrity of corresponding parts of primary visual cortex and of related areas of visual field.

Dog B displayed an intact cortical representation for almost all of the upper visual field above 5°. No atrophy was evident bilaterally in the posterior levels or in the dorsal part of the middle levels of LGN_d. Serial reconstructions of the lesion (see Table IV) displayed evidence of sparing in corresponding areas of the primary visual cortex.

Dog C was left with significant sparing of almost all of its upper visual field, except for some deficit found up to about 20° in the right quadrant. Complete sparing of neurons occurred throughout the posterior and middle levels of the right LGN_d, in the dorsal and middle parts of the posterior and middle levels of the left LGN_d, and bilaterally in the dorsal part of the anterior level. The corresponding cortical areas that were spared included the postlateral gyri and posterior parts of splenial and suprasplenial gyri, while the middle parts of lateral, suprasplenial and splenial gyri were ablated. The areas of visual field presumably spared are schematically described in Fig. 45.

Finally, dog E may have retained a partially intact cortical representation for the upper visual hemifield and for some peripheral parts of the lower visual field. The complete loss of large neurons

throughout the LGN_d indicated widespread removal of areas 17 and 18. However, although their numbers were reduced, the presence of medium size neurons implied partial sparing of certain areas. The occurrence of incomplete atrophy can be attributed to the overlap of the topographic geniculostriate projections. Focal lesions of the striate cortex produced a well-defined area of severe degeneration in the LGN_d; a less affected surround remained where reduced numbers of medium-sized cells were present (71). Hence, diffuse foci of spared cortex could produce incompletely atrophic areas of LGN_d. Therefore, the atrophy of large neurons throughout most of the LGN_d suggested diffuse deafferentation of widespread parts of primary visual cortex of dog E.

In summary, it appears that only dog A definitely received a complete ablation of primary visual cortex. Dog D received a nearly complete lesion, with only partial sparing of the very lateralmost parts of upper visual field. Dog E possessed a great deal of partial sparing that comprised the entire upper visual field, along with peripheral parts of the lower visual field. In both dogs D and E the extent of functional sparing that accompanied the anatomical evidence of some intact neurons in the LGN_d was unclear. Both dogs B and C possessed definite sparing of the peripheral parts of the upper visual field.

Implications of Post-operative Visually-guided Behavior

The stable post-operative visually-guided behaviors were affected to varying degrees in the five ablated dogs. Dogs A and D demonstrated the most extensive long-term deficits, dogs C and E only slight deficits, and dog B intermediate deficits. This pattern correlates well with the

extent of the cortical ablations discussed in the previous section. Also, based on the aforementioned studies in cats, loss of particular visually-guided behavior provides an indication of the relative damage to areas 17, 18 and 19.

Two visually-guided behaviors were consistently affected, in all dogs, by cortical ablation. Eye blinks were not elicited post-operatively by a threatening stimulus moved directly at the eye from any part of the visual field. The eye blink reflex to visual threat is dependent on visual cortex and is independent of superior colliculus (see Table III). In light of the variations in degree of cortical ablation described in the previous section, the loss of the eye blink reflex to threat suggests that functional damage to primary visual cortex, severe enough to impair this cortical reflex, was present in every case. The second visually-guided behavior that presented uniformly in the ablated dogs was localization and identification of visual stimuli. These visual behaviors require integrity of visual cortex, since they depend upon fixation and high-acuity central vision to provide pattern discrimination (24a,100). The dog was presumed to have identified a food stimulus when persistent fixation of the stimulus and accompanying anticipatory behavior toward the food were exhibited. Post-operatively, visual localization and identification were almost completely absent and showed essentially no compensation (see Table V). Once again, the loss of a visual behavior dependent upon visual cortex is evidence for a significant degree of functional impairment in primary visual cortex of all five ablated dogs.

Overall, the stable, long-term deficits in visually-guided behavior were greatest in dogs A and D. Visual placing (a visual behavior dependent upon visual cortex) was absent frontally; orienting responses to moving stimuli in the lateral visual fields (mediated by superior colliculus) were present but decreased; visual following (dependent upon visual cortex) was erratic and present only to very slowly moving stimuli; and visual avoidance (largely mediated by visual cortex, but also by superior colliculus) was present only for large obstacles, approached tangentially. This pattern of deficits most closely approximated the effects of ablation of areas 17, 18 and 19 in the cat (see Table III). The only dissimilarity was in visual orienting which, post-operatively, was normal in cats but slightly reduced in the dogs in this study. Thus, dogs A and D, who possessed anatomical evidence of having most complete ablations, showed deficits in visually-guided behavior similar to those reported for the cat after removal of areas 17, 18 and 19.

Dogs C and E showed only mild deficits in visually-guided behavior. After some compensation occurred during the first 40 post-operative days, the visual following and orienting responses were almost normal, as was avoidance behavior to obstacles and behavior in novel environments. Visual placing improved within four weeks so that errors in negotiating stairs and single steps were infrequent. For the cat, the same pattern of compensation in visually-guided behavior was observed following ablation of area 17 either alone, or in combination with area 18 (see Table III). The only discrepancy was in the blink reflex to visual threat, which remained intact in the cat. The loss of the blink

reflex in the dogs may have occurred due to the small size of the threatening stimulus, creating a higher acuity task which could not be accomplished because of the decreased acuity accompanying partial lesions of areas 17 and 18. The anatomical evaluation of the cortical ablations in dogs C and E demonstrated sparing of most of the upper hemifield. Thus, the very mild degree of deficits in visually-guided behavior is most likely due to the significant amount of intact visual cortex representing this area of visual field.

The visually-guided behavior demonstrated post-operatively by dog B was midway between the relative extremes exhibited by the four other dogs. As in dogs A and D, visual following responses, frontal placing responses, and avoidance behavior were very poor or not elicitable. However, like dogs C and E, the visual orienting responses to lateral, moving stimuli were near-normal. The pattern of deficits in visually-guided behavior indicate the integrity of that visual behavior mediated by superior colliculus and the impairment of all visual behaviors dependent on visual cortex. From the anatomical analysis of the extent of ablation, it was determined that a significant representation of the peripheral upper hemifield was spared. Therefore, while the functional deficits in visually-guided behavior in dog B suggest a more complete lesion, similar to those incurred by dogs A and D, the anatomical evidence paradoxically indicates only a partial lesion.

The patterns of post-operative visually-guided behaviors support the anatomical determinations of the extent of visual cortical removal: dogs A and D, who received the most complete ablations, demonstrated

the greatest deficits to those visually-guided behaviors dependent on cortical mediation; dogs C and E, who revealed a great deal of sparing in upper and in parts of lower visual field, had the smallest deficits in the visual behaviors mediated by visual cortex; only dog B demonstrated greater functional deficits than predicted by the anatomical representation of the lesion.

Perimetric Studies

The perimetry experiments revealed that orienting responses to moving stimuli introduced into the lateral 30° to 120° of the visual field were present in all dogs, while orienting responses to central visual field stimuli between 0° to 45° bilaterally were decreased to different degrees in the five dogs. This was presumably due to one of the following: disproportionate removal of areas of primary visual cortex representing central, as opposed to peripheral, visual field; or preferential processing of peripheral visual field input for lateral orienting responses at midbrain visual centers.

The disproportionate sparing of peripheral parts of the visual field might account for the data in dogs B, C and E, all of whom retained some intact upper peripheral visual field, this was not the case, however, in dogs A and D, where ablation of primary visual cortex was complete or nearly complete. Considering the variable extent of visual cortical ablations in the five dogs, the presence of similar, inter-animal, post-operative orienting responses to lateral parts of the visual field suggests that a similar mechanism was intact across all. This supports the conclusion that the intact midbrain visual

centers in all these dogs were processing the peripheral visual field input for mediation of the lateral orienting responses.

Cats with large occipitotemporal lesions (who are thereby dependent upon midbrain visual processing) demonstrate orienting responses only for the nasal retina of each eye and not for temporal retina (88,90). This same situation in the dog would explain the persistence of orienting responses from areas of visual field as far central as 15° to 30°. The presence of some orienting to 0° by dog C may have resulted from slight deviation from 0° at the time the novel stimulus was introduced.

An additional source of variability in the orienting responses, post-operatively, was the motivation or drive of the animal to perform. Dog A was somewhat withdrawn and reluctant to perform in the perimetry experiments. This variable may explain the greater overall decrease in orienting responses present in dog A, and the slightly different degrees of diminution between the other dogs, post-operatively.

Therefore, the orienting response to a lateral moving stimulus, which persisted post-operatively in all the dogs, appears to be mediated by midbrain visual centers, most likely the superior colliculus. Visual cortical involvement is not likely, since the different dogs possessed visual cortical ablations of varying degrees of completeness, yet demonstrated similar persistence of the orienting response. Dogs A, B and D did not significantly regain any of the visually-guided behaviors mediated by the visual cortex, suggesting that functionally their primary visual cortex was completely ablated.

Compensation of Visual Deficits

The pattern of post-operative improvement of visually-guided behaviors and postural control provides some important implications concerning the CNS visual centers that are involved in compensation.

The degree of improvement in visually-guided behaviors varied, depending on the particular behavior and the specific dog. However, dogs A and D, who received the most complete ablations, showed some compensation in visual orienting behavior which stabilized between days 7 to 14 (see table V). Slight compensation in visual following and in avoidance behavior did not stabilize until between days 26 to 40. Thus, the superior colliculus-mediated visual behavior stabilized much earlier than the behavior dependent on visual cortex. Dogs C and E showed a similar pattern in compensation, although their visually-guided behaviors improved to a greater extent.

The progressive improvement of visual function was apparent in postural control as well. Immediately post-operatively, the dogs' performance in the NP experiment began to reflect compensation for initial deficits in postural control, improving with repeated sessions (see Fig. 28). Dogs A and D required the greatest number of experiments before stable postural control responses ensued, after 27 and 20 days, respectively. The pattern of compensation in visually-guided behaviors indicates that by the time stabilization of postural control occurred, the superior colliculus had already stabilized, while the primary visual cortex had not. This implies that improvement of postural control post-operatively is mediated by superior colliculus. Dogs B, C and E attained stable postural responses earlier than dogs A

and D, between days 7 and 14. This provides additional evidence that superior colliculus may be critical for visual processing in postural control.

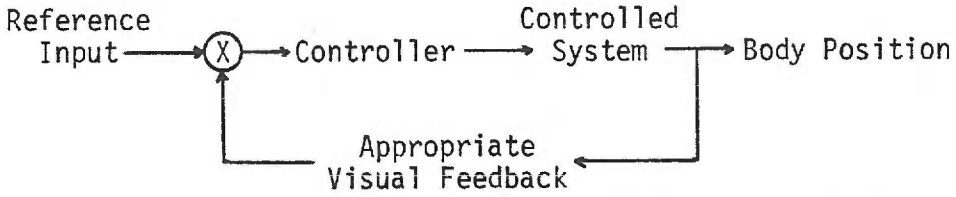
However, the improvement in postural control during the NP experiment could have more than one source: the CNS visual system may have readjusted to regain visual processing which was temporarily lost, or other sensory feedback loops may have increased their gains to correct for the absence of visual feedback.

To distinguish between these two alternatives, postural control was tested with visual feedback completely blocked. There was no progressive decrease in the gains (i.e. improvement) of the postural responses, suggesting that the labyrinthine and somesthetic feedback channels were not being used increasingly to compensate for a deficit in visual feedback. Therefore, the compensation in postural control behavior observed following primary visual cortex ablation appears to involve the readjustment of intact visual processing, for proper utilization by the postural control system. Based on the time course of compensation and relative degrees of stabilization in visually-guided behaviors and postural control, this intact visual processing must include the superior colliculus.

Postural Control

The specific roles played by different areas of the visual field and by various CNS visual centers in postural control will be discussed in the ensuing sections. Comparison of the results of different groups of experiments tests each of the four hypotheses posed originally (see

Experimental Objectives, p. 32). The simplified model of the components of the postural control system (Fig. 1) depicted visual feedback of body position as a single black box, as follows:



Testing of each of the four hypotheses should provide information permitting further elaboration of this part of the model.

Changes in Postural Control Capability -- Hypothesis I

The initial hypothesis deals with the necessity of primary visual cortex for normal postural control. It states that if primary visual cortex is not essential for processing of necessary visual input for normal postural control motor responses, then chronic ablation of visual cortex should produce no change in the postural response; yet, blindfolding should continue to change the postural response, as it did pre-operatively.

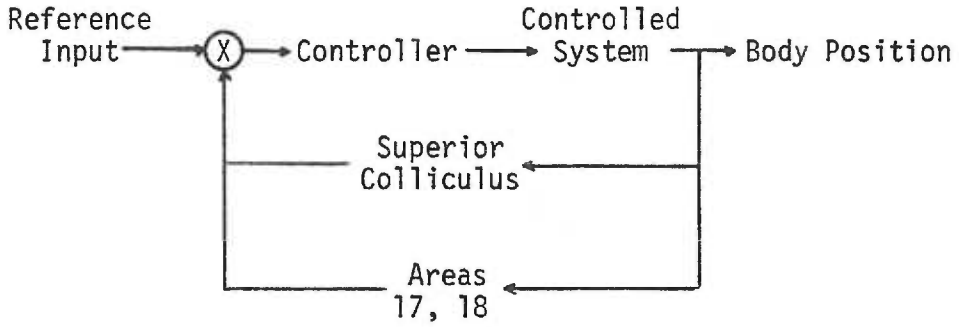
After the visual cortex was ablated in the five dogs, the stable, postural control response to platform motion did not change significantly in any of these dogs (Fig. 31). This finding suggests that the dog's postural control capability was not altered by complete removal or partial damage to the primary visual cortex. However, since this reflects only the stable post-operative performance, the question of increased weighting of other sensory modalities, as compensation, is a possibility. When the dog's visual fields were completely blocked during the platform perturbation a deficit in postural control resulted

at virtually all frequencies of platform oscillation, both pre-operatively (Fig. 29) and post-operatively (Fig. 30). Therefore, visual feedback continued to be an effective sensory feedback modality for postural control, following complete and partial lesions of primary visual cortex.

The comparison of the dog's pre- and post-operative postural control response when blindfolded permits an assessment of any change in relative utilization of the other sensory modalities. In dogs A and D, there was no significant difference in the overall postural response during blindfinding pre- and post-operatively. The unchanged postural response in the two most completely ablated dogs implies an intact visual feedback pathway into the postural control system, with no increased utilization of other sensory inputs. The other three, less completely ablated, dogs did exhibit increased postural control capability in the blindfolded condition, post-operatively, suggesting some increase in the utilization of the labyrinthine and somesthetic feedback channels in these dogs.

Therefore, in all dogs, the ablation of primary visual cortex was without effect on postural control capability. Furthermore, dogs A and D clearly appeared to be utilizing intact visual pathways, which did not involve primary visual cortex, for incorporation of visual input into the postural control system. The intact visual pathway processing visual feedback into postural control is most likely the superior colliculus, based on the rapid stabilization of the orienting response mediated by this midbrain nucleus, and on the permanent deficits in visually-guided behavior mediated by visual cortex. These results

provide a basis for remodelling the visual feedback black box as follows:



The implication of this modification in the model of the control system is that while both cortical and midbrain visual centers may process visual feedback for postural control, the feedback loop does not require essential passage through the primary visual cortex.

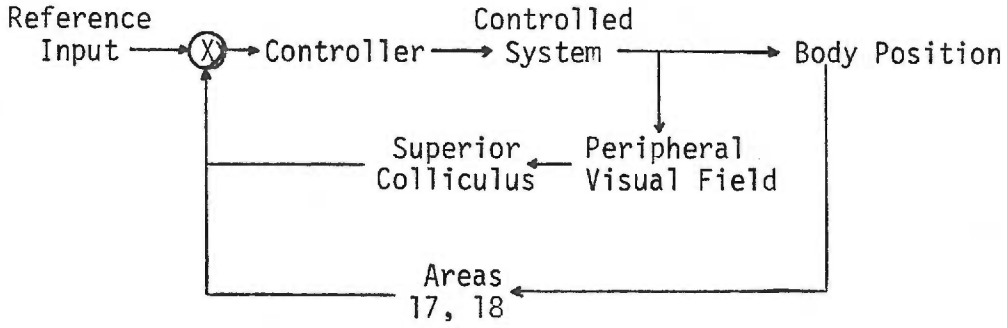
Peripheral Visual Field Utilization -- Hypothesis II

The second hypothesis deals with primary visual cortex involvement in processing of peripheral visual field input for postural control. It predicts that if primary visual cortex is essential for processing peripheral visual field input, then chronic ablation of areas 17 and 18 should abolish visually-induced postural corrections normally evoked by peripheral visual field movement.

When central visual field input was blocked, the ability of purely peripheral visual field motion to elicit postural responses was determined by oscillation of the peripheral roof. The amplitude of such visually-evoked postural responses decreased post-operatively except in dog A, who exhibited no significant change at four of five frequencies (Fig. 37). It is remarkable that in the most completely lesioned dog, the visual potency of peripheral motion is unchanged, whereas in the

less completely ablated dogs, some decrease in potency of peripheral field motion occurred. This decrease in potency in four of the five dogs does not represent abolition of peripheral visual field input into postural control, since the post-operative peripherally-evoked postural responses are significantly larger than the responses evoked during blindfolding (Fig. 38).

Therefore, the peripheral visual field remains capable of influencing the postural control system after complete or partial ablation of primary visual cortex. This finding is supported by the known role superior colliculus plays in lateral orienting responses to peripheral parts of the visual field (88,90,99). Thus, the model of visual feedback in the postural control system can be further modified as follows:



This modification in the model merely shows that primary visual cortex is not essential in the processing of peripheral visual field input.

Central Visual Field Usage -- Hypothesis III

Human psychophysical experiments yielded the observation that central visual field input could interfere with the perception of peripheral visual field motion (14). The third hypothesis deals with the ability of central visual field input to influence the postural

control system, pre- and post-operatively. It states that if primary visual cortex is essential for processing central visual field input, the chronic ablation of areas 17 and 18 should result in the inability of central visual field movement to interfere with the visually-induced postural response evoked by peripheral field movement.

The ability of peripheral visual field motion to evoke postural responses, pre-operatively, was decreased when conflicting motion information was presented simultaneously into the central visual field (Fig. 39). This supports the conclusions from human studies, i.e. that peripheral visual field motion is perceived as exocentric motion and evokes a compensatory postural response to counter the perceived self-motion, while central visual field motion is processed as egocentric motion and can interfere or detract from exocentric motion perception.

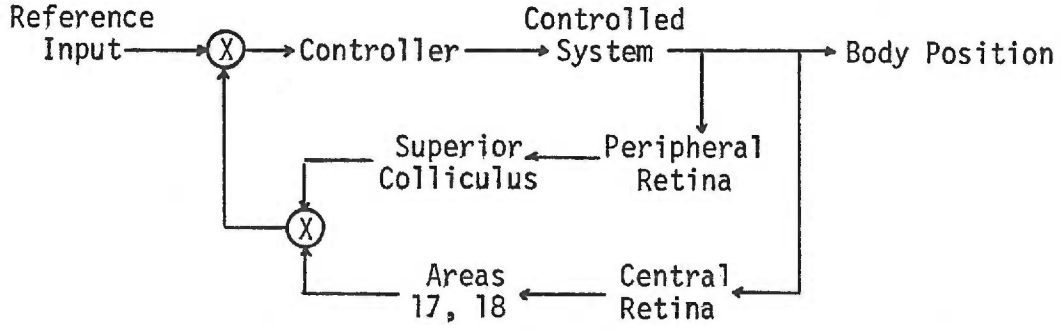
Post-operatively, conflicting motion information in central visual field does not have an inhibitory effect on the amplitude of the peripherally-evoked postural response (Fig. 40). Dogs A and C demonstrated no significant difference in the peripherally-evoked responses either with or without conflicting central visual field information while dogs B, D and E actually showed some increase in gain of the response when conflicting central input was included. Dog S, however, continued to exhibit a decrement in gain in response to the conflicting input.

Thus, after complete or partial removal of primary visual cortex, central visual field feedback cannot be integrated into the postural control system. This indirectly observed phenomenon was more directly

verified by studying the changes in amplitude of postural responses evoked directly by central visual field motion. After ablation of primary visual cortex, the amplitudes of evoked postural responses were dramatically diminished in all five dogs while the sham control dog showed no change (Fig. 41). Thus, the potency of central visual field motion is abolished by the complete or partial ablation of primary visual cortex.

The gains of post-operative, centrally-evoked postural responses are not significantly different overall from blindfolded responses for dogs A, B, D and E; dogs C and S responded to the central visual field motion with significantly larger overall responses (Fig. 42).

Therefore, in complete absence or with partial destruction of primary visual cortex, the central visual field is no longer capable of affecting postural control, whether indirectly (by integrating with peripheral visual feedback), or directly (by evoking postural responses). The modified model thus becomes even more complex:



The visual input from central visual field must be processed by areas 17 and 18 in order to be incorporated into the postural control system. Also, since the central visual field input can inhibit the postural responses evoked from peripheral visual field, integration of these

two inputs must take place.

Relative Use of Peripheral and Central Retinal Input for Postural Control -- Hypothesis IV

The fourth hypothesis considers the exclusive use of peripheral visual field feedback by the postural control system. It predicts that if peripheral visual field input is used exclusively for visual processing in postural control, then blocking peripheral visual field feedback should produce a deficit equivalent to blindfolding in the postural response.

Pre-operatively, the effect of restricting visual feedback to either the central or peripheral visual field was not uniform across all dogs (Fig. 33). Since postural control capability was unaffected in two dogs, diminished somewhat in two other dogs, and enhanced in two other dogs, it appeared that either central or peripheral visual field could provide appropriate visual feedback that could be incorporated into postural control.

Post-operatively, there existed an inequality in the ability to utilize central versus peripheral visual input. In all dogs, the postural control capability was consistently decreased at most frequencies of platform oscillation, during restriction of vision to the central visual fields (Fig. 34). However, dog S continued to show similar postural control whether restricted to either part of the visual field.

The decrement in post-operative postural control capability, during restriction to central vision, was greater or no different than blindfolding in dogs A, B, D and E. Dog C exhibited somewhat greater deficits

in postural control capability with blindfolding than with central vision alone, suggesting some intact central visual field, while dog S showed much more postural control capability with central vision alone than when blindfolded.

Therefore, in the modified model of visual feedback in postural control both central and peripheral retinal inputs contribute visual feedback for postural control, with similar deficits resulting from their separate masking. However, complete or partial ablation of primary visual cortex removes this equipotentiality, resulting in the effective postural utilization of peripheral visual field alone. Although only peripheral visual field feedback is being processed, the overall postural control capability is unchanged from the pre-operative state. Thus, although normally peripheral visual field input is not used exclusively in postural control, it apparently has the capability of providing support for normal postural control in the absence of control-relevant input from the central visual field.

PERSPECTIVE

The proper perspective for defining the role primary visual cortex plays in processing visual feedback for postural control depends upon accurate knowledge of the anatomical extent of the visual cortical lesions, the effects of these lesions on assorted visual behaviors, the changes in postural control resulting from the lesions, and the alternate routes available for visual processing which bypass the lesioned areas.

The extent of visual cortical ablation demonstrated substantial inter-dog variability. The removal of primary visual cortex was nearly complete in two dogs. For a third dog, the indirect evidence from the lateral geniculate suggested that diffusely spared areas of visual cortex may have retained some unknown degree of functional integrity. Sparing of a significant portion of areas 17 and 18 occurred in two other dogs.

Lesions of primary visual cortex produced changes in visually-guided behaviors in all dogs. The lateral orienting response, which is mediated by superior colliculus, was not abolished in any dog, while the visually-guided behaviors dependent upon visual cortex were lost to a degree correlating with the anatomical extent of the ablation. Therefore, the greatest deficits in such visual cortically-mediated behaviors as avoidance and localization, visual following and visual placing occurred in the two dogs receiving the most complete lesions.

The results of perimetric testing of the orienting response provided further evidence for the integrity of lateral orienting responses, independent of the extent of the cortical ablation. This evidence

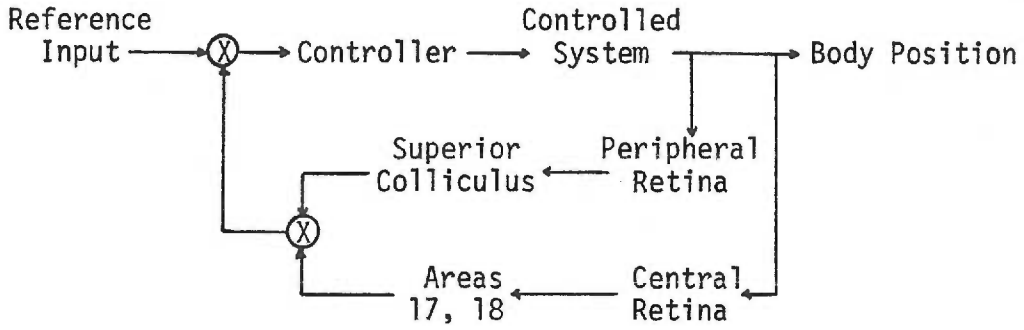
suggests that the superior colliculus continued to function normally following ablation of primary visual cortex.

The dog's visually-guided behaviors and postural control responses improved during early post-operative testing. The time course of compensation in visual processing differed between visual cortex and superior colliculus. This was reflected by earlier stabilization of the visually-guided behavior mediated by superior colliculus. Stabilization of postural control responses occurred during this same time. Thus, the compensation in postural control behavior seemed to parallel the compensation in visual processing by the superior colliculus.

Platform experiments, testing postural control capability, revealed that primary visual cortex is not essential for normal postural control. In its absence, peripheral visual field information continues to be incorporated into postural control, while central visual field input is prevented from influencing the control system. As a result, post-operatively the postural control system is able to provide normal postural control while utilizing only feedback from the peripheral visual field.

Platform-roof experiments, testing the ability of visual motion to evoke postural control responses, revealed that although primary visual cortex is not essential for peripheral visual field-evoked responses, it is essential for both central visual field-evoked responses and integration of inputs from the two areas of visual field.

Based on all of the above results, the following modification of the visual feedback loop in the model of a postural control system (Fig. 1) is suggested.



The processing of visual feedback from peripheral retina is shown to take place in the superior colliculus. Besides reaching superior colliculus over a direct retinocollicular pathway, peripheral retinal input might reach lateral suprasylvian (LS) cortex over either retino-tectal-pulvinar pathways or retinogeniculate-area 19 pathways (see Fig. 2). Based on the known role of superior colliculus in mediation of orienting responses, and the observed persistence of these responses in the absence of primary visual cortex, the superior colliculus remains the most likely site for processing of peripheral retinal input for postural control.

In the model, visual information from central retina is diagrammed as a feedback pathway that traverses areas 17 and 18. This visual information is then transferred into the postural controller. That information transfer by areas 17 and 18 is separate from postural control processing, was demonstrated by the operational integrity of the controller after the removal of areas 17 and 18 had interrupted information transfer. However, the available evidence does not permit speculation on the degree of information processing carried out in areas 17 and 18.

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Table I. Summarized properties of receptive fields in areas 17 and 18 of cat visual cortex. (modified from ref. 111)

Receptive field property	Area 17	Area 18
A. Mean diameter	2.2° ± 1.4°	6.5° ± 3.0°
B. Direction selectivity	76%	74%
C. Velocity preference		
0-2° /sec	11%	0%
3-10°/sec	41%	16%
11-20°/sec	31%	31%
>20°/sec	17%	53%

Table II. Receptive field properties of neurons in lateral suprasylvian (LS) cortex. (modified from ref. 95)

Receptive field property	LS area
A. Mean diameter	17° (1.75 - 47° range)
B. Direction selectivity	81%
C. Velocity preference	
0-20° /sec	7%
21-40° /sec	21%
41-100°/sec	7%
>100°/sec	10%
No preference (0 to >200°/sec)	55%

Table III. Summary of cat's stable visually-guided behavior after chronic, selective lesions to cortical and subcortical visual areas. + represents normal behavior, ± represents a partial deficit in the behavior, and - represents a complete deficit. (5,7,24,97,99,100,101)

	Visual following	Visual placing	Visual orienting to lateral stimulus	Eye blink to visual threat	Visual avoidance	Learning a flux discrimination	Learning a striped pattern discrimination	Learning a shape pattern discrimination
I. Cortical								
A. Area 17	+	+	+	+	+	+	+	+
B. Areas 17,18	+	+	+	+	+	+	+	+
C. Areas 17,18,19	±	±	+	±	±	+	+	-
D. Areas 17, 18,19 and LS	-	-	-	-	-	+	-	-
E. Areas 19,LS	±	±	+	+		+	+	-
II. Subcortical								
A. SC	±	+	-	+	±	+		±
B. Pretectum						+		±
C. SC, Pretectum						+		-

Table IV. Summary of the extents of visual cortical lesions in all dogs based on serial reconstructions of the occipital pole of cortex and on analyses of retrograde atrophy in the LGN_d.

	Right side							Left side						
	CORTEX				LGN d			CORTEX				LGN d		
DOG	PLG	LG	SPG	SSPG	P	M	A	PLG	LG	SPG	SSPG	P	M	A
A	A	A	A	A	SA	SA MA_d	SA MA_d	A	A	A	A	SA	SA MA_d	SA MA_d
B	I	A	I	A	NA	SA_v NA_d	SA_v NA_d	I	A	I	A	NA	SA_v NA_d	SA_v NA_d
C	I	A $I^m_{a,p}$	A $I^m_{a,p}$	A $I^m_{a,p}$	NA	NA	NA_d SA^d_v	I	A $I^m_{a,p}$	A $I^m_{a,p}$	A $I^m_{a,p}$	NA_d MA^d_v	NA_d SA^d_v	NA_d SA^d_v
D	A I_m	A I_a	A	A	SA MA_l	SA	SA MA_d	A	A I_a	A	A	SA MA_l	SA	SA MA_d
E	A I_m	A I_a	A_m $I_{a,p}$	A_m $I_{a,p}$	MA	MA_d SA_v	$MA_{d,lv}$ SA_{mv}	A	A I_a	A $I_{a,p}$	A $I_{a,p}$	MA	MA_d SA_v	$MA_{d,lv}$ SA_{mv}

Cortex: A=ablated, I=intact, a=anterior, p=posterior, m=middle

LGN_d: SA=severe atrophy, MA=mild atrophy, NA=no atrophy, d=dorsal, v=ventral, l=lateral, lv=lateroventral, mv=medioventral

TABLE V. Summary of post-operative observations of visually-guided behaviors.

	VISUALLY-GUIDED BEHAVIORS					
	Visual following	Visual orienting	Visual placing	Blink reflex	Avoidance behavior	Localization & identification
POST-OP DAYS 1 - 6	None (A, B,C,D) At slow speeds (E)	None (A,D) Diminished (B,C,E)	None (A, B,C,D,E)	None to visual threat (all dogs)	None(A,B,D) To large obstacles (C,E)	Poor (A,B,C,D,E)
POST-OP DAYS 7 - 14	None (A, D) At slow speeds (B) At slow and medium speed (C,E)	Diminished (A,B,D) Near normal (C,E)	None (A,B,D) Some (C,E)	"	None(A,B,D) To large obstacles (E) Near normal (C)	Poor (A,B,D) Slow(C,E)
POST-OP DAYS 15 - 25	None (A, D) At slow speeds (B) Near normal (C,E)	"	None (A,B,D) Fair (C,E)	"	None (B,D) To large obstacles (A,E) Near normal (C)	"
POST-OP DAYS 26 - 40	At slow speeds (A,B,D) Near normal (C,E)	Diminished (A,D) Near normal (B,C,E)	"	"	To large obstacles (A,B,D) Near normal (C,E)	"
POST-OP DAYS 40-end	NO CHANGES ---- stable visually-guided behaviors					

Table VI. Summary of results from platform and platform-roof experiments.

(A) Platform experiments

EXPERIMENTAL CONDITION	COMPARISON	RESULT	FIGURE
Pre-op	Normal vision/ blindfolded	Blindfolding increased gain in all dogs	29
Post-op	Normal vision/ blindfolded	Blindfolding increased gain in all dogs	30
Normal vision	Pre-op/post-op	No change, except dog S	31
Blindfolded	Pre-op/post op	No change - dogs A,D; post-op decreased gain in dogs B,C,E,S	32
Pre-op	Central vision/ peripheral vision	Central increased gain - dogs B,C; peripheral increased gain - dogs A,E; no change - dogs D,S	33
Post-op	Central vision/ peripheral vision	Central increased gain in all dogs, except dog S	34
Post-op	Central vision/ blindfolded	No change - dogs D,E; central increased gain - dogs A,B; central decreased gain - dogs C,S	35

(B) Platform-roof experiments

Peripheral vision	Pre-op/post-op	Post-op decreased gain - dogs B,C,D,E; no change - dog S; post-op slightly increased gain - dog A	37
Post-op	Peripheral vision/blindfolded	Peripheral vision increased gains in all dogs	38
Pre-op	Normal vision/ peripheral vision	Normal vision decreased gain in all dogs	39

(continued on next page)

EXPERIMENTAL CONDITION	COMPARISON	RESULT	FIGURE
Post-op	Normal vision/ peripheral vision	No change - dogs A,C; normal vision increased gain - dogs B,D,E; normal vision decreased gain - dog S	40
Central vision	Pre-op/post-op	Post-op decreased gain in all dogs, except dog S	41
Post-op	Central vision/ blindfolded	No change - dogs A,B,D,E; blindfolding decreased gain - dogs C,S	42