CONTRIBUTION OF THE SPINAL CORD DORSAL COLUMNS TO POSTURAL CONTROL IN THE DOG

by

Patrick J. Reynolds

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INTRODUCTION

The Dog Postural Control System

Since the following discussion is intended to provide some basis for experiments analyzing the postural control system in the dog, a definition of a system is first in order.

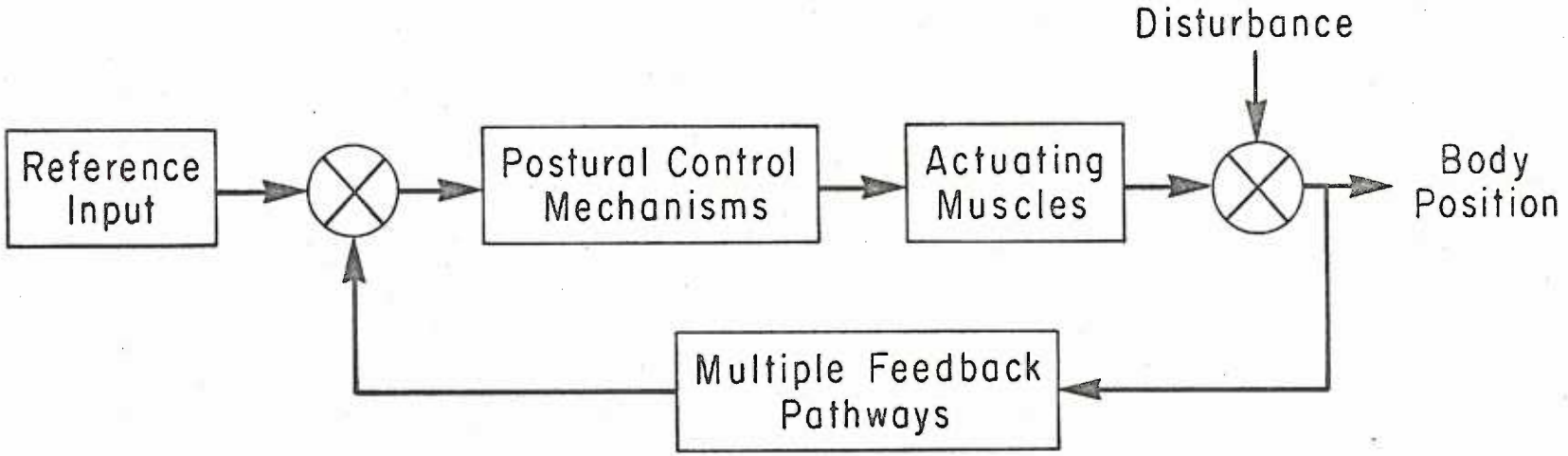
Paraphrasing Milsum (78) a system is defined as any collection of interacting materials and processes which together perform some function and whose behavior is determined by: (1) the characteristics of the components, (2) the structure of the interaction pathways (usually involving feedback pathways), and (3) the input signals. The function of a system is usually defined in terms of some identifiable kind of output and the system may be thought of as an input-output device.

A rigorous science of systems analysis has developed over the past 20 years, and the terminology and technique of the systems engineer have been applied to many different biological systems over the past 10 years (78). Detailed description of the analytical technique of the systems engineer is really not appropriate here; at the present stage of investigation the applicability of engineering technique to study of the dog postural control system is simply assumed. Only after some basic identification of system components is accomplished will it be possible to devise a model of the system and

subject it to intensive analysis.

The maintenance of an erect, quiet stance has been assumed to be the output of a biological system in the dog, and investigation of the system has been underway (15, 16, 17, 79, 80, 86, 87, 96). A block diagram illustrating a simplified concept of the interacting components of the system is shown in Fig. 1. The reference input is a completely mysterious entity; it functions in the awake, alert nervous system of the dog and commands the system into action. It can be activated by training an animal, and fortunately its nature seems to be reproducible from dog to dog and from day to day within dogs (17). Activation of the reference input somehow affects somatic behavior so that the animal assumes the quietly standing body position in place of the wide variety of activities he might engage in. The reference input feeds to a summing point, the outflow of which impinges on another poorly understood block of the system. The postural control mechanisms constitute the sum total of integrative activities of the central nervous system that affect the outflow of activity over motor fibers to skeletal muscles. The role of actuating muscles in the maintenance of orientation of skeletal parts is reasonable to assume. The system output, the appropriately maintained body position, can be continually assessed by sensory mechanisms which send information over multiple feedback pathways (104). This sensory information is compared with the reference signal to appropriately adjust postural integrative

Figure 1. Simplified postural control system block diagram. See text for description of component interaction.


Disturbance
Reference Input
Postural Control Mechanisms
Actuating Muscles
Body Position
Multiple Feedback Pathways

activity in a closed loop system. The skeletal parts to which actuating muscles attach may also be affected by outside influences in the form of postural disturbance. The simplified system blocks will eventually be split into well defined subsystems. The experiment to be described herein represents an initial attempt in evaluating the contribution to control provided by one of the possible feedback pathways in the dog spinal cord.

Afferent Activity Appropriate to Postural Control

The exhaustive experiments of de Kleijn, Magnus, and Rademaker demonstrating the importance of organs of the special senses as well as somatic receptors in the organization of "postural reflexes" are summarized by Roberts (104). Nakao and Brookhart (86, 87) have presented evidence that somatic receptors have a predominant role in the organization of postural activity in the dog during undisturbed quiet stance, and a wide variety of receptors whose activation might reasonably be expected to affect postural organization project to the spinal cord and brain from the periphery.

The first of these is the muscle spindle whose behavior was first quantitatively investigated by Adrian in 1926 (1, 2). Matthews (74) used electrophysiological techniques to study isolated afferents from mammalian muscle and disclosed activity which he attributed to four distinct types of muscle nerve afferents. He recognized that

there were two distinct types of afferent activity arising from the muscle spindle itself, and that the other two types arose from receptors in muscle but distinct from the spindle. The nature of afferent activity from the mammalian muscle spindle has undergone extensive observation (8, 58, 59, 62, 63, 71) since the initial study by Matthews, and it is well summarized by Roberts (104):

> The features of the mammalian muscle spindle which should be borne in mind may be summarized as follows: there are two types of intrafusal muscle-fiber -- "nuclear bag" and "nuclear chain". Both of these differ from extrafusal muscle-fibres, and each has its own independent motor innervation. There are two types of sensory receptor, both located in the central, lymph-space, region of the spindle. Each spindle has one, and only one, primary (annulospiral) receptor, and may have from zero to five secondary receptors. The adequate stimulus is extension of that part of the intrafusal muscle-fibres that carries the receptors. Normally this local extension is related to the tension in the polar parts of the spindle and may be affected either by changing the overall length of the spindle, e.g., by stretch, or by changing the mechanical activity of the intrafusal muscle-fibres, e.g. by impulses in the gamma motor axons. The response contains static and dynamic components which may be independently influenced by different fusimotor axons.

The remaining two types of afferent discharge in muscle nerves arise from free nerve endings in muscle and from the well defined Golgi tendon organ (57, 60, 64). The tendon organ discharge is initiated by actively or passively induced tension in the muscle which is transmitted to the muscle connective tissue (either tendon or its extension into the muscle belly) in which the receptor lies. Various kinds of

mechanical manipulation can produce discharge over the fibers which arise from the unspecialized endings in the muscle.

Another class of somatic receptor which has been studied extensively since Goldscheider (51) suggested its importance is that of the joint capsule and ligament receptors. Anatomical studies preceded and guided electrophysiological studies, but it is remarkable to note that the powerful single-unit analysis techniques were not even attempted on articular receptors until after 1950. A review by Gardner (41) emphasizes the confused state of knowledge of articular receptor physiology prior to electrophysiological investigation. The existence of specialized nerve endings in and around joint capsules giving rise to large diameter myelinated afferents was well recognized.

Several investigators (10, 26, 30, 38, 42, 75, 109) studied the effects of articular afferent activity, by electrical or mechanical stimulation, on segmental reflex activity. The results, especially with electrical stimulation, were highly variable and yielded little knowledge pertinent to understanding the role of articular afferents in segmental reflex organization. An excellent study by Skoglund (109) clearly defines the anatomy and physiology of the knee joint innervation in the cat. Certainly other investigators (4, 12, 13, 85) should be given credit for disclosing properties of joint receptor discharge, but the organization and amplitude of Skoglund's investigation give it an overshadowing position.

In the cat knee joint, nerve fibers, ranging from 1 to 18 microns in diameter, arise from superficial joint capsule and ligament regions and form two distinct (posterior and medial) nerves, which then travel over various peripheral nerve routes (including several muscle nerves) to enter the spinal cord at several segmental levels. The fibers arise from three types of nerve endings, each of which has a particular distribution in the joint structure. Ruffini endings, as many as 5 per axon, are seen primarily in the joint capsule and occasionally in the medial collateral ligament. Golgi endings, very similar in appearance to the tendon organs of Golgi, occur in several joint ligaments, but not in the capsule. A small number of Vater-Pacini endings are seen in the capsule. Many free nerve endings are seen, giving rise to small diameter fibers, and unmyelinated fibers of the sympathetic efferent type are seen with blood vessels

When single unit discharges from dissected articular nerves are examined during natural stimulation of the knee joint (movement in the directions of flexion and extension) two types of discharge are seen to occur: slowly adapting and rapidly adapting. Some slowly adapting units, characterized by smaller spikes, are seen to have a "critical angle" (position of joint) through which they discharge, and are silent when the joint is outside this angle. Their discharge rate is seen to rise rapidly as the joint moves into the critical angle, to peak somewhere within (often at the middle) the critical angle, and to drop off

rapidly as the joint moves out of the excitatory position. Critical angles observed for several receptors have a great deal of overlap, and individual receptors discharge over remarkably restricted angles in the range of 10 to 30 degrees. Changes in discharge rate could be superimposed on these critical angle patterns of discharge by additional manipulation of the joint, such as twisting, compression, or probing. Localization of the responsible receptors by probing, excision of the tissue and histological examination shows that Ruffini endings in the joint capsule give the slowly adapting critical angle type of discharge. Other slowly adapting units, giving larger spikes, have maximal discharge rates at extreme positions of the joint and can be localized in the joint ligaments, which have only the Golgi endings. The rapidly adapting units, activated by any and every type of joint manipulation, give maximal bursts of activity with the most rapid movements of the joint, and are localized to the capsular Vater-Pacini endings.

The interpretations of these patterns of activity are as follows: (1) the slowly adapting capsular units can signal direction and rate of joint movement, but are not good steady state position indicators because muscle activity may influence their output, (2) the slowly adapting ligament units can signal direction of movement and steady state position since they are not affected by compression or twisting, and (3) the rapidly adapting units can signal acceleration of bones about

the joint. The dependence of joint afferent activity on muscle activity makes it appear that here is an extra-muscular monitor of resistance to movement. The possibility that articular afferent activity contributes to the function of postural control has been recognized (20, 25, 102, 104) and demonstrated experimentally by Freeman and Wyke (39).

Although the most intensive joint innervation studies have been confined to the cat knee joint, anatomical and electrophysiological studies mentioned above suggest that similar types of information may be signalled from nearly all axial and appendicular articulations in vertebrates.

Another class of deep somatic receptor consists of a host of specialized and free nerve endings distributed throughout such tissues as periosteum, interosseous fascia, mesentery, and other connective tissues (60, 82). The specialized endings are often of the Vater-Pacini type as described in joint capsules. Little information is available pertinent to the functional significance of such receptors; they are extremely sensitive to mechanical stimuli and can signal a variety of things including muscle tension, passive movement, arterial pulsation, distension of hollow viscera, and many types of externally applied stimuli.

A final class of receptor that may possibly be involved in postural control is that of the cutaneous mechanoreceptor (6, 19, 25, 99, 110, 116). Large diameter fibers arise from a wide variety of

specialized and free cutaneous nerve endings, and respond to many discrete types of stimuli, such as hair movement, claw movement, localized light probing or vibration and steadily applied pressure on foot pads.

Cutaneous and deep receptors signalling itch, pain, tickle, diffuse pressure, or temperature certainly have powerful segmental reflex connections as well as projections into higher CNS structures, but their functional significance in postural organization is minimized in this report simply because the experiment is designed in such a way as to avoid adequate stimuli for this class of receptors.

Somatic Afferent Projection Pathways in the Dog Spinal Cord

Afferents arriving over dorsal roots terminate somewhere in the central nervous system in synaptic contact with other neurons; the courses followed before synaptic termination are complex, divergent, and highly variable in pattern depending on the type of afferent (115). Probably all afferents have branches terminating in the spinal segment where they enter, and these terminations can bring about both localized and distant-projecting activity in the second order neurons upon which they impinge. Large numbers of afferents may send branches as well into both higher and lower spinal segments, and these terminals again may induce both localized and projecting second order activity. And fewer, but still very numerous afferents, project

entirely through the spinal cord to terminate in the brainstem in addition to issuing segmental branches. The functional significance of the arrival of afferent activity, or input to central processing centers, is in some cases clearly understood, as in simple reflex activity, and in some cases very difficult to comprehend, likely subserving "higher" functions such as conscious awareness, learning, memory, and formation of ideas. Terminations of afferent activity at various levels of the neuraxis thus subserve a wide variety of functions and included among these functions is the organization of postural reflexes (104).

A great deal of the experimental evidence discussed below concerning somatic afferent projections is derived from work on cats. It seems reasonable to assume at least gross similarity between dog and cat. It should be kept in mind that the apparently direct courses of the primary and higher order projection pathways described below are in fact divergent and the descriptions are not intended to imply singularity of function. Bilateral symmetry throughout the CNS is assumed to be understood in the following discussion.

A pair of second order projection pathways in the lateral and ventral funicles ascend to the ventrobasal thalamus. These spinothalamic pathways travel contralateral to the location of the primary afferent fibers and constitute a link in a pathway to somatosensory cortex (115).

Another pathway to somatosensory cortex via ventrobasal thalamus has been described by Morin (81). A synaptically activated spinal tract in the dorsolateral spinal funiculus, the spinocervical tract, terminates in the high cervical spinal cord on the cells of the lateral cervical nucleus. The spinocervical tract units are activated by primary afferents and travel on the same side of the cord as the afferents are located (43, 66, 90, 91, 112). The third order fibers from lateral cervical nucleus cross in the high spinal cord and ascend with medial lemniscal fibers to the thalamus.

Long ascending tracts activated by somatic mechanoreceptors of all types that ultimately terminate in the cerebellum are numerous and have been the subject of exhaustive investigation (68, 69, 72, 94). Two separate pathways (the dorsal and ventral spinocerebellar tracts) arise from hindlimb afferents, and another pair (the rostral spinocerebellar and cuneocerebellar tracts) arise from forelimb receptors. The spinocerebellar pathways travel in the lateral or ventral funicles; there is gross forelimb-hindlimb similarity in the organization of these tracts, but with notable specific differences. Collaterals of the hindlimb spinocerebellar tracts apparently activate lateral cervical nucleus units (43).

Other tracts ascending in the lateral or ventral columns have been described and include the spino-olivary and spinoreticular pathways (43, 55). It seems possible that some lesser magnitude tracts

that have been described may actually be constituted of terminations of collaterals of the larger tracts described above (112).

The remaining important ascending spinal fiber tracts are located in the dorsal funicles of the cord. A large portion of posterior quadrant fibers are primary afferents which project through to the brainstem ipsilateral to the receptors from which they arise. In the upper spinal cord, for example, the cervical portion, these fibers can be identified as bilateral pairs of distinct bundles: the fasciculi gracili, projecting from the hindlimbs and lower trunk, and the fasciculi cuneati, projecting from receptors of the forelimbs, thoracic trunk, and neck. Fibers of the fasciculus cuneatus and fasciculus gracilis synapse with cells of the dorsal column nuclei (nucleus cuneatus and nucleus gracilis) which give rise to the fibers of the medial lemniscus that cross and ascend to the contralateral ventrobasal thalamus (115). This dorsal column nucleus-medial lemniscal system carrying afferent activity to cerebral cortex is the system conventionally brought to mind by the terminology "dorsal columns".

Also located in the cervical posterior quadrant are the primary afferents activating the cuneocerebellar tract (94). These primary afferents synapse in and near the lateral cuneate nucleus to give rise to the tract which travels via the restiform body to the cerebellum. A less clearly understood gracilocerebellar tract appears to arise from a similar set of afferents from hindlimbs.

There is evidence (116, 117) for the existence of a group of second order fibers in the cervical posterior funiculi. These fibers, whose central termination is unknown, travel deep in the dorsal quadrant and have been denoted the ascending dorsal funiculus tracts (ADFT).

A final component of posterior quadrant fibers, of unknown magnitude in the cervical cord of the dog, might be those of the type called propriospinal, consisting of either ascending or descending groups of primary or higher order fibers (88, 115). The human cervical posterior quadrant, for example, carries the well defined descending primary afferent bundle called the fasciculus interfascicularis (115). Such fibers may well exist and be of importance in the dog.

Afferent Populations Projecting into the Spinal Pathways

The spinothalamic pathways are classically believed to be activated by other receptors than those mechanoreceptors with large diameter, rapidly conducting fibers mentioned above (105). Some exceptions may exist; Perl and Whitlock (98) report that ventrobasal thalamic units in the cat and monkey are responsive to contralateral joint rotation after spinal cord lesions sparing only the ventrolateral quadrant of the spinal cord. Such joint receptor activity would apparently have to traverse the ventral spinothalamic tract.

The spinocerebellar tracts are activated in large part by fibers arising from primary (annulospiral) endings of the muscle spindles. Secondary spindle afferents and large-fiber cutaneous afferents, including hair, claw, and pad-pressure sensitive units are also represented in the spinocerebellar tracts (94). Oscarsson does not believe that large fiber joint receptors are represented in the spino- or cuneocerebellar tracts, although some doubt exists (55).

The spinocervical tracts are very prominently activated by specific cutaneous mechanoreceptors so that their role in tactile sensibility is emphasized (64, 88, 90, 112). These tracts are apparently also activated by the whole spectrum of large-afferent somatic mechanoreceptors (43).

Confusion reigns over the subject of afferent contributions to the dorsal column nucleus-medial lemniscal system. It is not apparent from the literature if secondary muscle spindle afferents giving rise to fibers of the group II classification project into the DCN-medial lemniscal system. Some group II afferents in muscle nerves do enter the system but might very well arise from joint receptors (109). The primary spindle afferent and tendon organ give rise to fibers of the group I classification. Mountcastle et al. (82) reported that neither stretch of muscle nor stimulation of group I muscle afferents evoke responses in the cat sensorimotor cortex, to which the DCN-medial lemniscal system projects. The observations were limited to hindlimb

afferents, however, and Oscarsson and Rosen (95) have demonstrated that forelimb group I muscle afferents definitely project through the DCN-medial lemniscal system.

Skoglund (109) reported evoked sensorimotor cortical potentials in response to just suprathreshold joint nerve stimulation which could be abolished by the transection of the dorsal columns. However, Burgess and Clark (21, 22) report that the cat knee joint receptors giving the slowly-adapting responses to joint manipulation do not project centrally via the DCN-medial lemniscal system. Rapidly adapting receptors do. The apparent discrepancy with the findings of Skoglund might be explained by a threshold difference between the fibers of the two receptor types. Skoglund's interpretation that the several types of joint receptors send fibers over the DCN-medial lemniscal system is not tenable; Burgess and Clark offer no alternate projection pathway for the slowly adapting receptors.

General agreement exists on the contribution of rapidly conducting cutaneous afferents to the DCN-medial lemniscal system; hair, claw, pad pressure, touch, and other cutaneous receptors are known to contribute to the system (18, 92).

The extent of contribution of the rapidly conducting afferents from receptors in mesentery, interosseous fascia, periosteum, and other connective tissue to the dorsal column nucleus-medial lemniscal system is not clear. Rapidly conducting visceral afferents from the

splanchnic nerve as well as fibers arising from Pacinian corpuscles in the crural interosseous membrane of the cat do travel in the dorsal columns (3, 76). Many inferences in the literature give the impression that this class of receptors provides a large contribution to the dorsal column system.

Functional Loss in Subjects with Posterior Column Lesions

The overwhelming impression from the literature is that the dorsal column nucleus-medial lemniscal system subserves the function of sensation. The production of an evoked cortical potential in the primary sensory receiving area is a common result of synchronous activation of dorsal column fibers in animals and man (113), and it is reasonable to assume that this phenomenon underlies the production of sensation in man (85). Reported clinical signs of dorsal column damage in human patients include a loss of kinesthetic sense, or awareness of position and motion of body parts, and a loss of some aspects of tactile sensation that have been termed epicritic, or discriminative (105, 115). The anatomical arrangement of the system provides a possible basis for precisely organized sensory function; laminar organization of fibers in the spinal cord is the first evidence of the "somatotopic" organization seen through the ascending stages (24, 46, 106, 115). Orderly synaptic termination of primary afferents in the dorsal column nuclei give rise to medial lemniscal fibers with

somatotopic organization still intact (35, 37, 50, 99, 122). The lemniscal fibers cross in the brainstem as internal arcuate fibers and ascend to the ventrobasal thalamus to terminate in a precisely organized arrangement (83, 101). And the third order thalamocortical fibers from the ventrobasal complex activate neurons of the cortical receiving areas (SI and SII) in such a way that a map of the body surface is represented on the cortical surface (82, 84, 85, 113).

Observations on experimental animals, however, cast doubt on the singular importance of the dorsal column nucleus-medial lemniscal system in kinesthetic and discriminative tactile sensation. Cats with surgical transection of dorsal columns retain 80-100% of normal tactile discrimination capability (66). Dorsal column transection in dogs does not interfere with conditioned reflex response to hindlimb light tactile (air puff) stimulus (90). And monkeys retain the capability to respond to vibratory or limb position stimuli after dorsal column transection (108, 118).

Wall (120) has criticized much of the classical clinical inference of dorsal column function in human patients on the basis of the nature of the lesions; many reports were apparently based on cases in which lesions extended beyond dorsal columns. With carefully controlled dorsal column lesions in human patients the position and discriminative tactile sensations remain after a transient impairment (120).

Ferraro and Barrera (36) describe the effects of thoracic and cervical dorsal column transection in monkey. Their observations do not provide a quantitative basis for estimating tactile and position sensation, but describe motor deficits, especially in control of digits, and more profound in forelimbs than hindlimbs following cervical lesions. They observed "no postural defects" in the chronic animal, but their observations were strictly qualitative. They simply infer that monkeys are subject to a loss of position and tactile sensation on the basis of gross observations of motor function.

An important concomitant observation in the majority of recent experiments mentioned above on the tactile and position sensation function of the dorsal column nucleus-medial lemniscal system is that by extending lesions to include dorsolateral funiculi, especially in the region of the spinocervical tracts, severe and lasting sensory deficits occur.

To date, then, the most intensive studies of dorsal column function have focused on the position and tactile discriminative sensation function. And the dorsal column nucleus-medial lemniscal system seems to definitely participate in this function, but not uniquely.

Motor deficits possibly related to this type of sensory input exist in the chronically dorsal column sectioned monkey. Qualitative postural defects are not observed in monkeys recovered from dorsal column transection.

The effect of dorsal column transection on any of the aspects of output of the postural control system of the dog is not known.

Sensation, Reflex Organization, and the Dorsal Columns

The throughput of information to the primary sensory area of the cerebral cortex via the dorsal column nucleus-medial lemniscal system is strongly emphasized in the discussion above. Not strongly emphasized, however, is the fact that the sensory projection areas of cerebral cortex in carnivores and primates are intimately associated with motor function, so that these areas in experimental animals are usually referred to as the sensorimotor cortical areas (113). Inferring a sensory function in a region upon which afferent activity impinges is often carelessly done; afferent activity need not produce central activity related to sensory perception or conscious awareness at all (105). For example, the primary spindle afferents of skeletal muscle directly produce motor outflow over ventral roots in the monosynaptic reflex. A large proportion of afferent activity ascending in spinal tracts terminates in the cerebellum and apparently does not give rise to any sort of sensation (104). It is even likely that some afferent activity arriving at sensorimotor cortex in the cat does not produce sensation: using EEG arousal as an index, Giaquinto, Pompeiano and Swett suggested that cat forelimb group I muscle afferents do not produce sensation (44). The primary afferents travelling in the dorsal

columns synapse with brainstem neurons to give rise to activity coursing over pathways additional to the well recognized medial lemniscus-ventrobasal thalamus projection (92). Gordon and Seed (48, 49) reported units in rostral portions of nucleus gracilis of the cat that could be activated antidromically (by their criteria) by electrical stimulation of either the contralateral medial lemniscus or the anterior lobe of the cerebellum. Later investigation of this pathway to cerebellum by Gordon and Horrobin (47) indicated, by use of the impulse collision technique, that the antidromic activation criteria were too lax, and that the magnitude of the direct gracilocerebellar projection was actually overestimated. Apparently, a large proportion of rostral gracilis units activated by cerebellar stimulation are monosynaptically activated by fibers from cerebellum to brainstem; some direct gracilocerebellar fibers were encountered, however. The nature of this pathway is difficult to derive from the literature; no anatomical studies seem to show retrograde degeneration in the dorsal column nuclei after cerebellar lesions, with exception of degeneration in the external cuneate nucleus whose direct projection to cerebellum has been described (4, 65, 94). Lack of evidence of the projection to cerebellum by retrograde degeneration studies may be due to a dual projection of such fibers. In the raccoon a group of cells located within a transition zone between the cuneate-gracile complex and the external cuneate nucleus are seen to degenerate only when lesions are made in both

contralateral brainstem (including medial lemniscus) and ipsilateral cerebellar peduncles (65). Orthograde degeneration of a pathway directly from cuneate-gracile nuclei to cerebellum is not described in the literature, although no studies have been done according to a protocol which might possibly disclose such a projection.

The course and termination of fibers arising from the main cuneate and gracile nuclei are apparently not subject to general agreement among investigators today. No doubt exists concerning the important connection to the ventrobasal thalamus via the medial lemniscus; a host of conflicting information can be found alluding to either additional fibers projecting to brainstem nuclei or collateral projections to brainstem nuclei of the fibers of the medial lemniscus. Medial lemniscal collaterals have been described, anatomically terminating the substantia nigra, arcuate nuclei, and diffuse medial and ventral portions of the thalamic complex by Ariens-Kappers, Huber, and Crosby (5). Bowsher (11) produced thermal lesions in an undefined rostro-caudal position within monkey cuneate and gracile nuclei and traced degenerating fibers using the Nauta-Gygax technique. He specifically excluded reticular, centromedian or other thalamic nuclei, globus pallidus, hypothalamus, zona incerta, subthalamus, substantia nigra, or red nucleus as regions of terminal degeneration. He found complete decussation of degenerating fibers terminating almost wholly in the nucleus ventralis posterolateralis thalami with a small amount

of terminal degeneration in nucleus paralemniscalis of the mesencephalon. It may well be that the lesion was too restricted in rostrocaudal extent to affect cells of origin of fibers previously described as terminating in regions other than ventrobasal thalamus, but an illustration shows very large numbers of degenerated lemniscal fibers in a mid-pontine section. Ebbesson (29) aspirated portions of cat dorsal column nuclei and traced degeneration using the Nauta Technique. A lesion placed in the rostral portion of nucleus gracilis and middle of cuneatus produced a small amount of terminal degeneration in the medial accessory olive and a massive region of terminal degeneration in the dorsal accessory olive. He did not trace degeneration to any other regions. Rostro-caudal cytoarchitectonic and functional differentiation within gracile and cuneate nuclei may be very critical in consideration of origin of projection pathways from the nuclei (29, 56, 65, 70). Hand and Liu (56) present an abstract, with apparently no following publication, describing results of unilateral lesions in middle and rostral nucleus gracilis of cats. Cells of rostral gracilis are said to send projections to ventrobasal thalamus, medial regions of ipsilateral spinal trigeminal nucleus, ventral portions of dorsal accessory olive, basal and lateral regions of corpora quadrigemina, mesencephalic reticular formation, magnocellular portions of medial geniculate and red nucleus, an area ventral to red nucleus and caudal to fields of Forel, zona incerta, H1 field of Forel, and posterior thalamic complex.

The data are not available, nor is a discussion of the points of conflict with previous investigators presented. The question of collateral projections of medial lemniscal fibers seems to be open; however, it does seem likely that fibers arising from dorsal column nuclei, activated by afferents ascending in spinal cord dorsal columns, project to structures not related to sensation but still appropriate for postural control involvement. The possibility that major differences in functional organization of dorsal column nucleus projections between bipeds and quadrupeds account for some of the above conflicting reports seems possible. Lund and Webster (70) report terminal degeneration in some, but not all, extrathalamic brainstem regions of the rat as reported by Hand and Liu for the cat. In the case of the dog it appears that there is currently no available evidence relating to extralemniscal or lemniscal collateral projections of dorsal column nucleus cells.

The implication of neural activity arising from somatic receptors and terminating in cerebellum or brainstem structures below the thalamus is profound with respect to postural control reactions. Many physiologists must be remembered for establishing the descriptive terminology and investigating the patterns of interaction of the integrative brainstem functions classed as "postural reactions" or "postural reflexes". Fulton, Landau, Liddell, Magnus, Rademaker, Sherrington, and others provided framework to which subsequent electrophysiological study has primarily furnished confirmation or

extension. Magnus (73) describes various remnants of postural reactions remaining in animals with transections of the brainstem at various levels. He makes it clear that postural behavior is very near to normal in an animal that is decorticate, thus deprived of sensory perception, but has a nervous system intact up to the level of the thalamus. The dependence of righting reflexes (reflex orientation of the head and body with respect to gravity and the environment) on proprioceptive and exteroceptive somatic neural activity and an intact brainstem is clearly established.

Not clearly established to date is the degree of participation of the classical postural reflexes in the maintenance of undisturbed quiet stance in the intact dog. It seems reasonable to assume that they play some role and to design experiments testing the assumption. And among many questions that can be asked is included a question about which spinal cord afferent pathways carry information involved in the organization of postural activity. At this point, then, a physiological problem has been identified and a rationale for seeking a solution has been suggested:

1. Receptors signalling information appropriate to postural control project in the dog spinal cord dorsal columns.
2. Second order activity generated by these projections is involved in production of sensation, but

3. Additional second order activity projects to structures not related to sensation but appropriate to postural control involvement.

4. Some postural reactions are known to function in the absence of sensation.

5. The question arises: do fibers projecting in the dorsal columns contribute importantly to the postural reactions not involving sensation?

The Experiment

The possibility that some of the large rapidly conducting afferents traveling in the spinal cord dorsal columns participate in organization of postural activity might be tested by observing the postural behavior of dogs before and after transection of the dorsal columns. With the intent of gathering information suitable for evaluation by the technique of the systems engineer, observations will be made on the steady-state operation of the postural control system as well as on the dynamic response of the system to a controlled disturbance. Normal dogs will be trained to the task of maintaining as quiet and laterally symmetric a stance as possible. Once the limit of their capability in this respect is evaluated, they will be subjected to observation of the nature of response to a postural disturbance in the form of a brief longitudinal displacement of their supporting platform during task

performance. After quantitative evaluation of these observations on steady-state and dynamic postural system behavior, the dogs will be subjected to surgical transection of the spinal cord dorsal columns above the zone of entry of afferents from the brachial plexus, thus blocking long projecting fibers from all limbs and from the trunk below the neck. Clinical observations will be noted and the quantitative observations will be resumed as soon as possible and continued until recovery has stabilized. Immediate and long term effects of the lesion will be examined in the hope of evaluating the contribution of the spinal cord dorsal columns to postural control in the dog.

FACILITIES AND APPARATUS

Animals and Housing

The dogs used in this study were selected only according to weight and body conformation, and were not of uniform breed. They were supplied by and housed in the facilities of the University of Oregon Medical School Department of Animal Care. They were all of approximately 25 kg body weight on ad lib diet at the time they were accepted for the study. Differences in size and weight of the animals were not given any consideration in the physical setup of the experimental apparatus. The dogs were all trained to stand on the same base of support on the platform in the experimental laboratory.

A total of six animals was used. All survived the surgical procedure and were observed through the complete control and experimental protocol. A well-equipped experimental surgery suite was used to perform the laminectomy and spinal cord lesion and a veterinarian supervised the anesthesia. The animals were housed in individual runs for about 30 recovery days after surgery. The regular dry food diet was supplemented with specially formulated high protein canned food for 2 to 3 weeks after surgery. Minor complications in wound healing and two possible cases of meningitis from surgical sepsis were treated according to the advice of the veterinarian. The immediate post-operative recovery period before experiments were

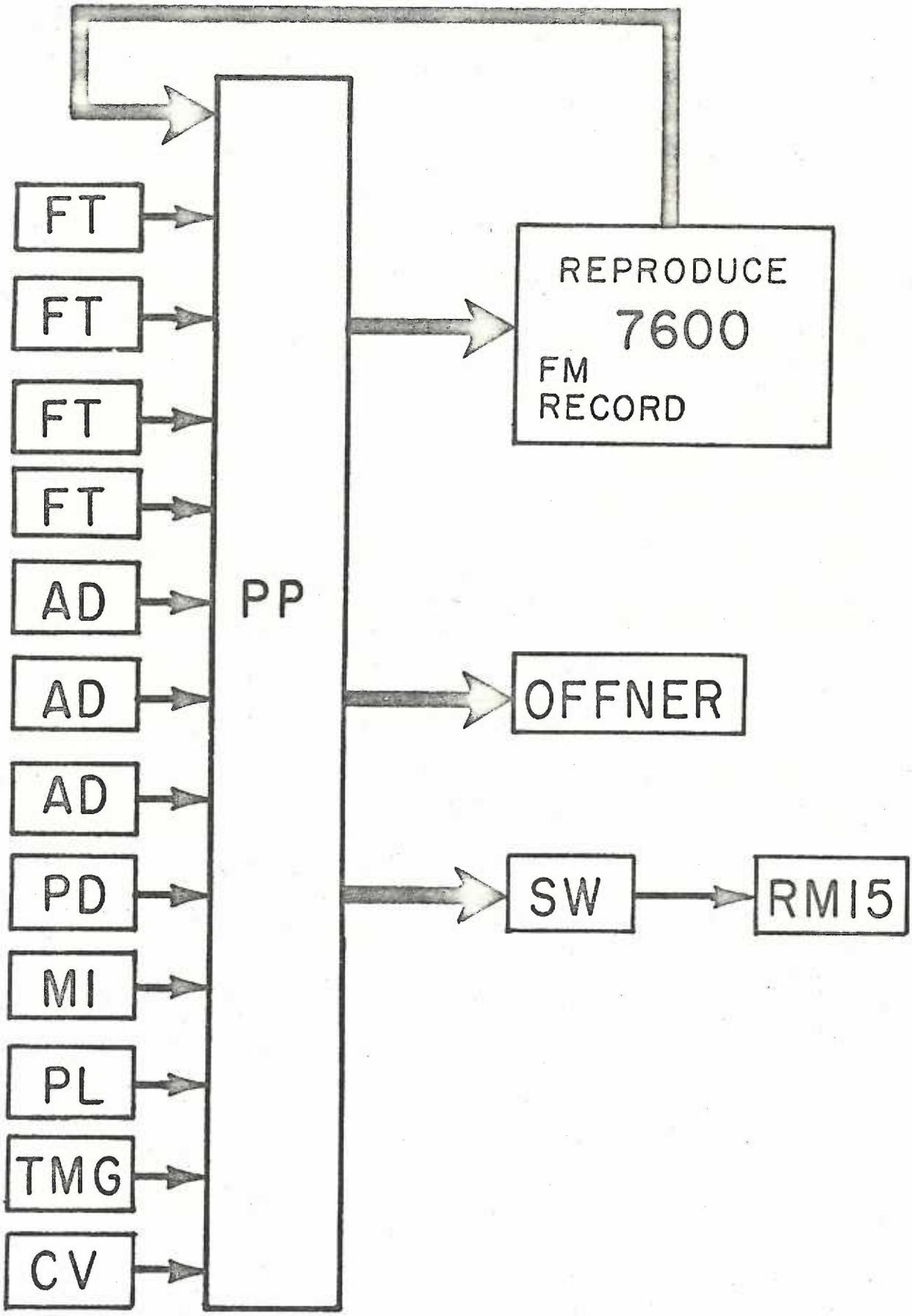
begun ranged from 1 to 10 days.

Laboratory and Data Acquisition System

The basic apparatus of the laboratory in which the experiments were conducted has been described previously (17, 79). The hydraulically powered, electronically controlled movable platform supported an array of four force transducers upon which the animals were trained to stand. A position detector (16) could be fixed to the skin overlying the lumbar spine of the animals. Hindlimb joint angles could be detected by placing sensors on the shaved skin overlying the long bones of the leg (103).

A block diagram of the data acquisition system is shown in Fig. 2. Signals from the processing amplifiers for the force, position, and angle detectors as well as the mode indicator signal (see below), platform position signal, and a time mark generator output were led to a patch panel at the rear of a card enclosure. By selecting appropriately wired patch cards, any desired combination of signals could be led to the 7 FM record inputs of a Honeywell 7600 magnetic tape recording system, to the inputs of an eight channel Offner inkwriting oscillograph, and, via a selecting switch, to the input of a Tektronix RM15 monitoring oscilloscope. The recording and monitoring systems could be operated independently or simultaneously.

Figure 2. Block diagram of the data acquisition system. Recorded data were derived from signal processing amplifiers for the force transducers (FT), angle detectors (AD) and position detector (PD). These signals, as well as signals from the mode indicator (MI; see Fig. 4), platform position feedback potentiometer (PL), a Tektronix type 180 time mark generator (TMG), and a calibration voltage source (CV) were led to patch panel (PP) at the rear of a card enclosure. When appropriately wired patch cards were placed in the patch bay any desired combination of signals could be led over trunks to the seven FM record inputs of the magnetic tape recording system (7600), to the eight inputs of the inkwriting oscillograph (OFFNER), and to a selecting switch (SW) leading to a monitor oscilloscope (RM15). Recording devices could be operated independently and/or simultaneously, and the playback of the FM tape recorder could be monitored on the inkwriter and oscilloscope.


FT
FT
FT
FT
AD
AD
AD
PD
MI
PL
TMG
CV
PP
REPRODUCE
7600
FM
RECORD
OFFNER
SW
RM15

The 7600 magnetic tape recording system was operated in the standard FM mode at 7 1/2 ips (center frequency 6.75 KHz). The specified data bandwidth (within 1.0 db) is 0-1250 Hz. Linearity is specified to be $\pm$ 0.5% full deviation, with 10 volts RMS the maximum input level. The linearity was checked using a -5V to +5V ramp generated by an EAI 680 analog computer and was within specifications for all FM channels after appropriate modifications were made to the reproduce amplifiers.

Signal processing amplifiers were adjusted to keep maximum expected data signals within the $\pm$ 5V linear range of the recording system. Typical recorded signal amplitudes were of the order of 4 volts peak to peak. Data system calibration was done by recording transducer signals or voltage sources on the FM tape immediately prior to conducting experiments each day and converting these records to digital form at a low sampling frequency. Digital averages of about 25 samples (at 1 Hz) at calibration zero level and at calibration step level were then used to normalize the amplitude of all disk data files prior to dumping to cards or performing calculations. Once normalized, no calibration factors were further required for any data files and all programs could treat all data files equivalently.

Calibration zero values for each analog data channel depended on the transducer. The force plate channels were connected to their respective carrier amplifier outputs, and the amplifiers were

balanced to zero volts (± 10mV) with 5 kg preloads on the transducers. The angle and position detector channels were connected to the supply ground of a regulated (± 1mV) DC power supply. The platform position channel was connected to the position feedback signal in the platform control amplifier with the platform held stationary at the "zero" position.

The calibration step was +1.00 kg on the force transducers, +1.000 volt at the input of the angle and position channels, and +20.0 mm in the platform position. The linearity of the data handling system was dependent primarily on the linearity of the FM recording system, which was measured to be ± 0.5% full scale. The system-throughput method of calibration offered many advantages in simplicity and accuracy, especially in not depending on a fixed zero-reference for each recording channel. The standard deviations of the computed individual data channel calibration vectors remained nearly constant from day to day and ranged from 5 to 15 mV with a mean of about 7 mV. This was well within the resolution limits of the transducers. The force transducers had a resolution of about 0.01 kg, represented by 10 mV at the carrier amplifier output at the sensitivity used. Each angle detector had a resolution of about 0.1°, represented by 20 mV at the output. The position detector had a resolution of about 0.3 mm, represented by 15 mV at the output at the sensitivity used. The power supply for the angle and position detector

amplifiers was the same one used for the calibration voltage source. The schematic for the position detector amplifier is located in Appendix A.

Experiment Controller

A system was designed to control automatically the course of individual experiments, each of which could be initiated by the experimenter. The purpose of the controlling system was to assure as much as possible that the experimental observations were subject only to animal variability from trial to trial and that the animals were performing to the limit of their capability (encouraged by attainment of a food reward). A dog standing on the platform and performing for reward is depicted in Fig. 3.

The system block diagram is shown in Fig. 4. Dashed lines enclose closely related sets of components. The apparatus presented to the animal and intended to draw his keen attention is the light panel, speaker, and feeder. This set of components is physically located immediately in front of the platform on which the animal stands with his feet placed on the force transducers (see Fig. 3). The system works by presenting a visual and auditory cue to the animal of the passage of time toward the attainment of a food reward. Attainment of the reward is contingent on the maintenance of symmetry, within adjustable limits, of the weight distribution beneath the front feet for

Figure 3. Sketch of dog standing on platform and performing task for reward. Dog stands on the array of vertical force transducers. Visual field is dominated by the panel with vertical array of 16 indicator lamps. Feeder, when triggered by control electronics, drops individual meatballs through funnel into dish, from which dog can obtain reward with minimum disturbance of stance.

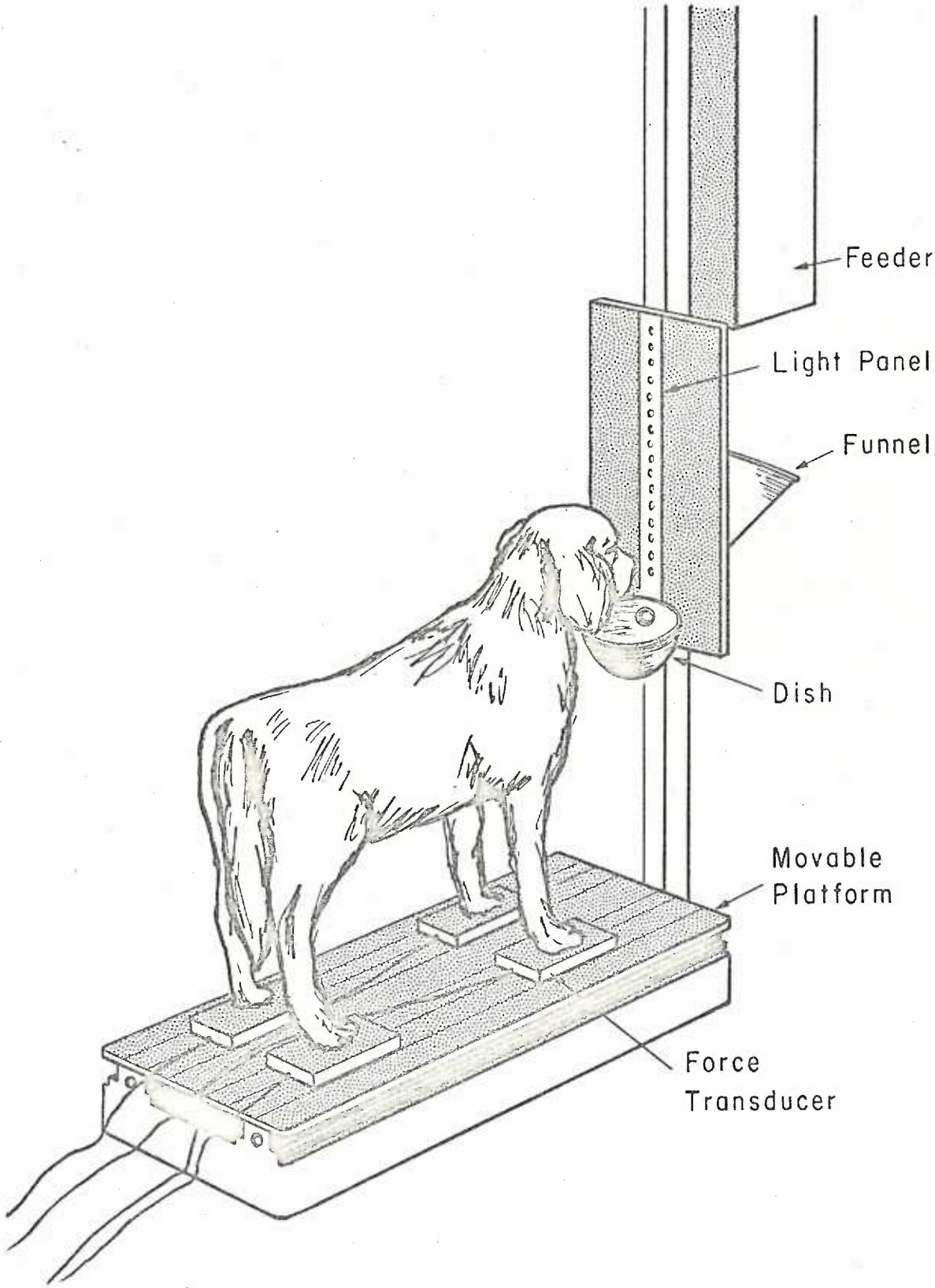
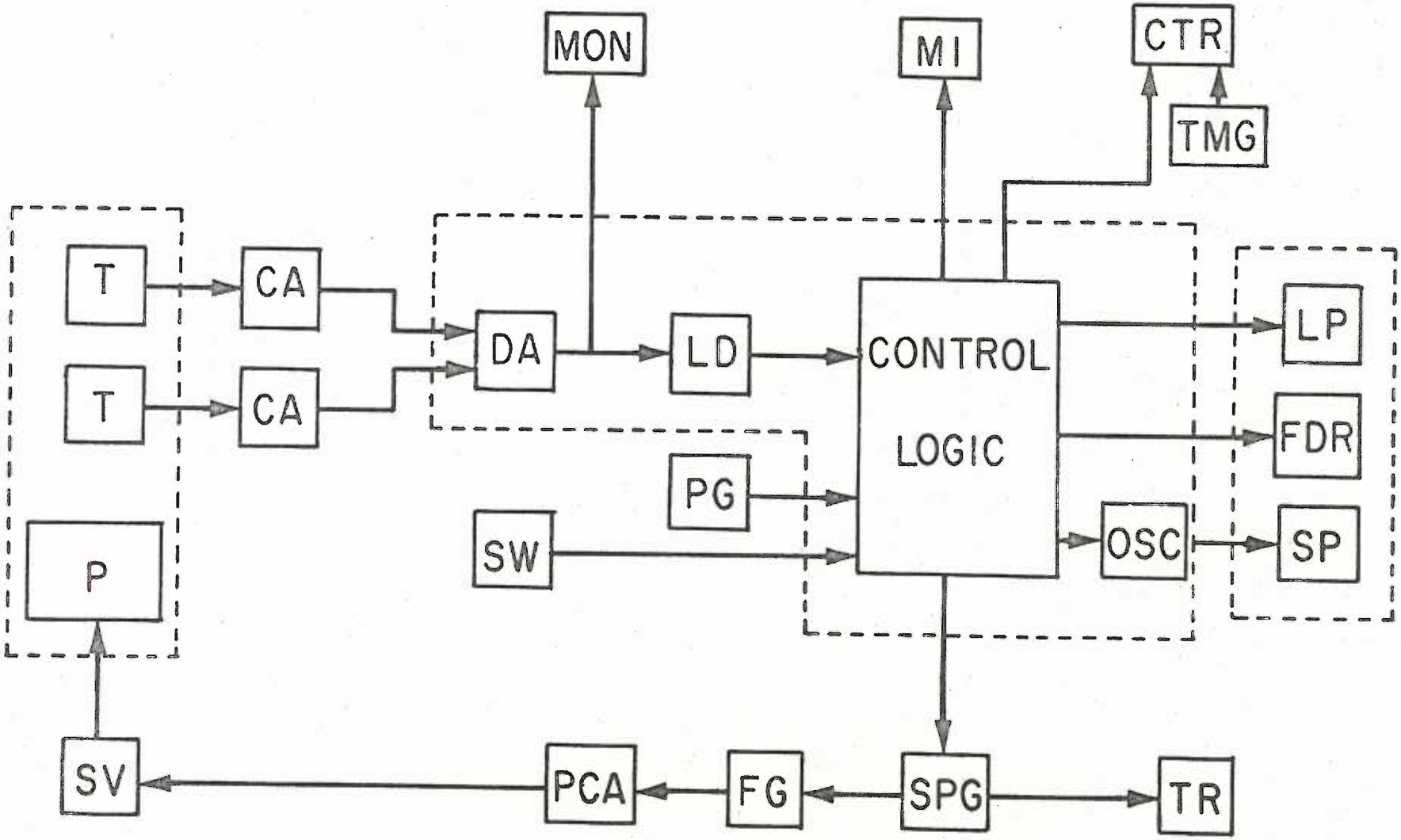

Feeder
Light Panel
Funnel
Dish
Movable
Platform
Force
Transducer

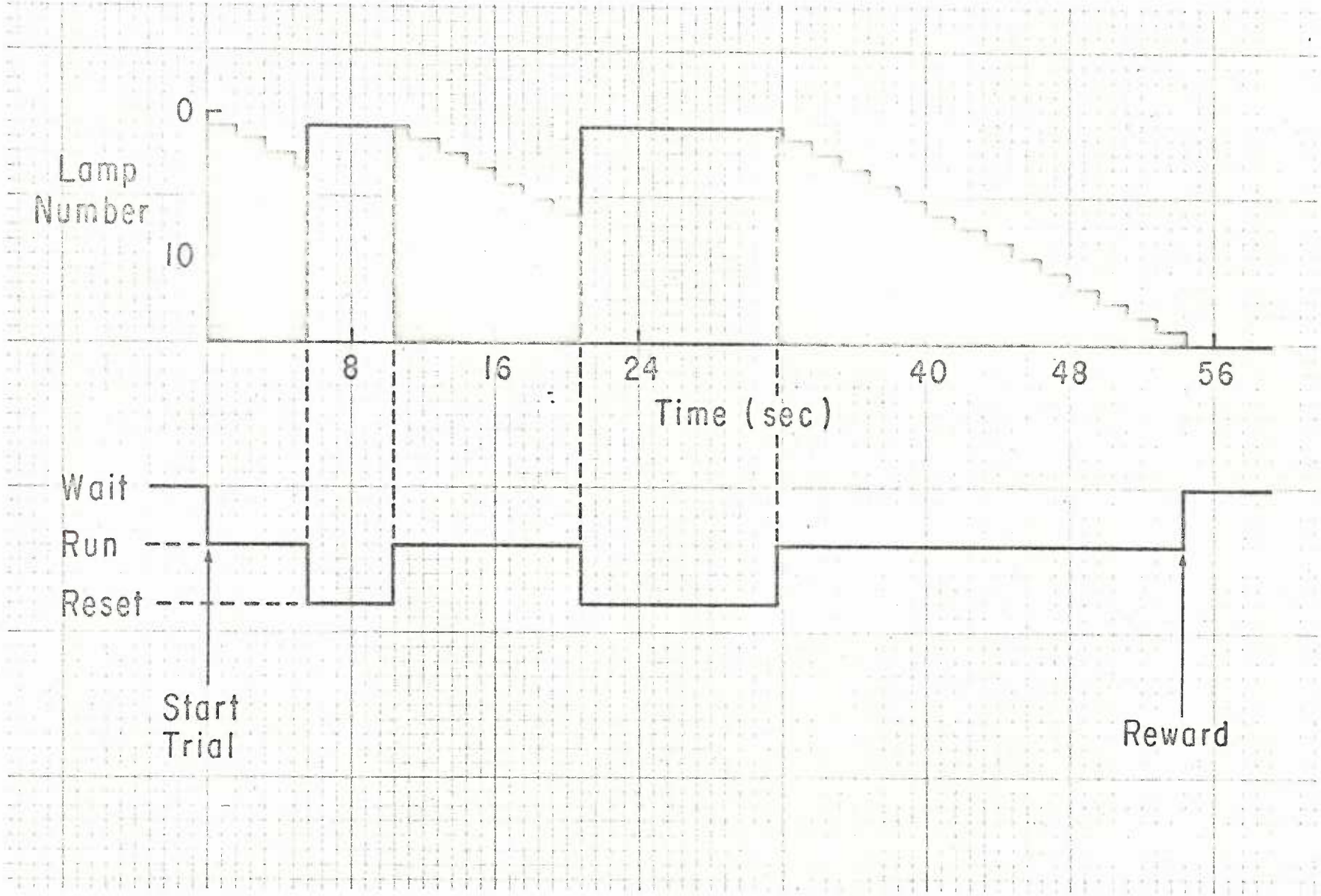
Figure 4. Block diagram of experiment controller system. Component identification: CA-carrier amplifier; CTR-counter; DA-difference amplifier; FDR-feeder; FG-function generator; LD-limit detector; LP-light panel; MI-mode indicator; MON-monitor meter; OSC-oscillator; P-platform; PCA-platform control amplifier; PG-pulse generator; SP-speaker; SPG-sampling pulse generator; SV-servo valve; SW-start switch; T-force transducer; TMG-time mark generator; TR-FM magnetic tape recorder. Dashed lines enclose physically apposed sets of elements. See text for description of component interaction.


MON
MI
CTR
TMG
T
T
CA
CA
DA
LD
CONTROL
LOGIC
LP
FDR
SP
OSC
PG
SW
P
SV
PCA
FG
SPG
TR

a period of time which can be set by the investigator. Signals from the control logic successively light the 16 panel indicator lamps on the light panel. These lamps are located in a vertical array, approximately one inch apart, immediately above the dish into which the feeder dispenses the reward. The adjustable limit detector, driven by signals from the force transducers, via carrier amplifiers and the difference amplifier, signals the control logic to reset and begin the lamp countdown again whenever front foot weight difference exceeds the set level. The length of time (duration of one trial) required to attain reward, provided no reset occurs, is set by fixing the output interval of the pulse generator which drives the control logic. The reward is dispensed by triggering the feeder with the same signal that turns on lamp number 16. Fig. 5 illustrates the course of events during an hypothetical trial in which two resets occurred. The reward is dispensed coincident with the lighting of lamp number 16. Interval between lamps is 1.6 seconds, and a trial without reset would have been completed in 24 sec.

In a preliminary design stage of the apparatus an analog of the projection onto the horizontal plane of the position of the center of weight distribution of an animal was generated from the signals from the force transducers beneath all four feet. A double-ended limit detecting circuit was used to constrain this center of weight distribution within a diamond shaped region, the position and dimensions of which

Figure 5. Illustration of events during hypothetical trial in which two resets have occurred. Trial is initiated by pressing start switch. Sequence of lamp lighting is indicated by the step diagram above. Mode indicator record sketched below: inter-trial interval mode is high (wait); operating mode is middle (run); reset mode is low (reset). Since pulse generator runs free, countdown initiates only at first pulse following termination of reset mode. Reward is dispensed coincident with lighting of lamp 16.


0
Lamp
Number
10
8
16
24
40
48
56
Time (sec)
Wait
Run
Reset
Start
Trial
Reward

could be adjusted. Experiments were conducted using trained animals from studies described previously (16) and the analog of the center of weight distribution was monitored on a storage oscilloscope. Two major problems were encountered. Shifts of weight distribution into patterns with diagonal asymmetry were often observed which failed to trigger the limit detector. It was also necessary to readjust the limit detecting circuit to accommodate for differences in the longitudinal position of center of weight distribution of the "characteristic" stance of each dog. For these two reasons the simplified limit detecting scheme of the present apparatus was adopted. A low-pass filter (3 db down at 3 Hz) between the difference amplifier and limit detector minimized the occurrence of resets at panting frequency.

There are three modes of operation of the apparatus, and these are all signaled by the output level of the mode indicator: (1) inter-trial interval mode, control logic not activated; (2) operating mode, initiated by depressing the switch and terminated by the "last lamp" pulse (returning the system to the inter-trial interval mode, (3) reset mode, initiated by the output of the limit detector and terminated when the limit detector turns off. The control logic returns to the beginning of the operating mode sequence each time the reset mode terminates. Trials may last any length of time then, depending on the occurrence of resets; the duration is 16 pulse intervals past the depressing of the switch or termination of the last reset. The counter (Beckman EPUT),

driven by the time mark generator (TRIGGER output of a Tektronix type 180 @ 100 Hz) and gated by the control logic, provides a display of trial duration at the termination of each trial. The auditory cue, delivered via the speaker located behind the light panel, is turned on only when the system is in the operating mode. The remaining components at the bottom of the figure will be discussed in the section on the data processing system. Schematics for the controller system electronics are located in Appendix B.

Data Processing System

Two separate types of observations were made on the animals and data handling was completely different for the two. The first type of observation was made as the animals stood quietly undisturbed and was intended to detect the limit of their capability to maintain a quiet stance. In this type of experiment the data, consisting of force plate and mode indicator output as well as counter display, were recorded on the inkwriting oscillograph and by hand and provided information only about the success or failure of the dog in completing individual trials. These data were not subjected to any kind of automatic processing.

The second type of observation was made as the animals were subjected to a controlled displacement of the platform on which they stood. In this case transducers signalling hind limb joint angles and

body position were attached. The controlling system was again used and the animals were still behaving in effort to attain reward for maintaining a quiet symmetric stance. The outputs of the force, angle, and position detectors were recorded on magnetic tape in such a way that responses to several successive displacements could be subjected to digital processing and derivation of appropriate statistics.

The experimental apparatus involved in the data processing system is seen in the lower portion of Fig. 4. Trials, initiated again under control of the experimenter by depressing the switch, were conducted so that the platform was triggered to move in either the headward or tailward direction at precisely 2 seconds after the trial began. The control logic triggered a sampling pulse generator whose output was recorded on the direct-record channel of the Honeywell tape recorder. The sampling pulse generator delivered pulses at the rate of 5 Hz for 2.0 seconds, 320 Hz for the succeeding 500 msec , and 5 Hz for 20.0 seconds thereafter. Thus during every trial 270 pulses (10 @ 5 Hz, 160 @ 320 Hz, and 100 @ 5 Hz) were recorded in 22.5 seconds and these were exactly registered on the same time base (magnetic tape) as the transducer signals from the animal. The sampling pulse generator also delivered a trigger pulse to an Exact model 255 function generator coincident with the 10th sampling pulse. The function generator output drove the platform control amplifier which in turn actuated the servo valve in the hydraulic system impelling the

platform. The platform servo system worked by nulling a platform position feedback signal against the function generator output and thus reproduced the function generator waveform in mechanical displacement of the platform in one (longitudinal axis of the standing dog) direction.

Inkwriter records of the transducer signals, platform position, and mode indicator were kept as the magnetic tape records were generated and these were used for purposes of editing data during the automatic processing. Magnetic tape records consisted of six FM channels of transducer outputs from the dog, one FM channel for platform position, and the direct record channel with sampling pulses.

The analog to digital data conversion system included a Raytheon Multiverter (externally triggerable), the conversion control logic (converter), a buffer memory, and a Kennedy digital tape recorder. The analog tape outputs went to the inputs of the multiverter and the sampling pulses, appropriately shaped by a digital read amplifier (Appendix C) went to the converter. Digital tapes generated from the seven channels of analog data consisted of serially written binary coded digits representing essentially simultaneous (28μ S slew) measurements on the data at points in time determined by the sampling pulse rate. Manual start and stop buttons on the converter made it possible to run the analog transport without converting data; data was converted to digital form on sampling pulse command only after a

manual start of the conversion system. Thus was provided the opportunity to edit the data. Schematics for the sampling pulse generator and digital read amplifier are located in Appendix C.

The digital tape recorder was interfaced to an IBM 1130 computing system. Converted data could thus be read from tape and stored as files on the 1130 disk cartridge. Disk data files were stored in permanent form on punched cards and were available in this form for later processing with the 1130 computer. The nature of the data processing programs will be discussed in the methods section.

METHODS

Shaping

Not all the animals were unacquainted with the laboratory at the time the present study was undertaken; however, the observations reported here begin at the point where each animal was totally naive to the automatic reward dispensing apparatus.

Each animal was subjected to a series of shaping experiments the number of which varied from animal to animal. The general pattern of the shaping experiments was similar for all animals. Initially a dog was simply presented with the hand-held feeding dish and meatballs (12-15 mm in diameter, weighing approximately 2 grams) were dropped into the dish. As soon as the dog learned to seek meatballs from the dish, the dish was placed on the light panel and the animal was lifted onto the platform with his feet placed on the force transducers. The experiment controller was set for a very wide reset limit (4 kg difference between front feet) and the time to reward was set very short (4.8 seconds from first light to last). The dog was simply manipulated by hand in and out of the reset condition and his attention was directed to the light panel and auditory cue. Each shaping session lasted about five minutes and an animal received 20 to 40 meatballs. Gradually, over several days' time course and depending on how well the animal seemed to be learning, the time to reward was

extended and the intervention of the experimenter was withdrawn. It was often possible to get successful performance with limits set more restrictive than 4 kg. For each dog the limits were then set to a value at which he could easily attain a reward but at which he definitely reset the countdown when he turned his head away from the light panel or otherwise grossly shifted his weight distribution. This accounts for the differences in the limit settings seen from dog to dog in the reported results.

Quiet Standing Experiments

When a dog became capable of completing two or three trials without reset with a 24 second trial duration during a given shaping session experiments were begun and results were recorded. The number of shaping sessions per dog ranged from 2 to 11. Experiments consisted of sessions of 15 trials. A trial was the event initiated by depressing the switch (under control of the experimenter) and terminated by dispensing of the meatball reward. A trial without reset was 24 seconds in duration, and the duration could be extended indefinitely depending on the occurrence of resets. The 15-trial sessions, with ten-second inter-trial intervals, would last about 8 1/2 minutes when no resets occurred. It became evident that some dogs tired of the task after about ten minutes in any given session, so the number of trials per session was reduced to 11. This provided for sessions of about

five minutes but left sufficient time for extended duration trials without exceeding ten minutes. It also provided for a simple derivation of the median trial duration since it was an odd number. On the basis of the evidence of fatigue in long-lasting sessions, the elapsed clock time was watched and experiments were aborted if the ninth trial was not completed by the end of nine minutes. Many aborted experiments are interspersed with those reported in the results, and no provision has been made for considering any of the trials of those sessions in the results.

As will be discussed in the results section, the experiments were conducted in such a way as to attempt to disclose evidence for a "learning curve" or increase in quiet standing capability over time. Attention was focused on the median trial duration of each experiment as an index of performance, and the limit for reset was set to a more restrictive level after each dog showed consistent "successful" performance according to the criterion of achieving a median trial duration of 24 seconds in several successive experiments. It was sometimes necessary to abort sessions immediately following a transition in the limit setting; in this event, experiments were simply repeated and the new limit was maintained so that the animals were presented only with an increasing step pattern of change in difficulty of the task. Not all the dogs were presented with an 0.5 kg limit, but two that were (8692 and 7611) were never able to finish a single trial. Thus no

complete experiment was ever done at a limit setting more restrictive than 1 kg. The attempted experiments at the 0.5 kg limit were undertaken well after the animals had been introduced to the 1 kg limit. Examination of results for all dogs indicated that the 1 kg limit provided a distinct challenge to the capability of the animals, so all dogs were subjected to observations at this limit over an extended time period while preparations were being made for conducting the step displacement type of experiment.

As the experiments were begun, attempt was made to record enough data that behavior during each and every trial could be reliably reconstructed. An inkwriter was used to record the output of the mode indicator and the four force transducers beneath the feet. Thereby a permanent record was made of the point of beginning of each trial, the shifts from operating to reset mode, the force changes that caused the resets, and the point of completion of each trial. The counter display also indicated individual trial durations and these were posted at the completion of each trial. Information in the mode indicator record included the number of resets which occurred and the amount of time spent in the reset mode.

Statistics were then compiled and tabulated or plotted in order to discover if, in fact, a sensitive indicator of performance was being recorded. Some of the earliest experiments were conducted as 15-trial sessions but in these cases statistics were compiled from just

the first 11 trials so that all results reported are on 11-trial experiments. The examined statistics included: mean trial duration, median trial duration, mean duration of the five trials above the median, difference between median and mean trial duration, total experiment duration, time spent in the reset condition, ratio of reset time to total experiment duration, variability (standard deviation) of trial duration within one experiment, total number of resets, and number of trials with one or more resets.

It was recognized that the trial durations were from a population with a highly skewed distribution (minimum value 24.0 seconds) and thus did not provide a very satisfactory substrate for the above-mentioned normal statistical analyses. Another serious drawback to using any of the time measurements as indices of performance was that it was never possible to conduct any of the experiments without some degree of operator intervention. All of the dogs required some sort of signal or manipulation in order to keep them attentive to their task. It was usually necessary to guide them back to a symmetric stance when they drifted outside the limit. If they were left completely unattended, they would invariably lose interest and fail to continue working for reward. The rapidity and success of the operator in guiding the animals back to symmetry once a reset occurred was a strong determinant of trial duration and reset time duration.

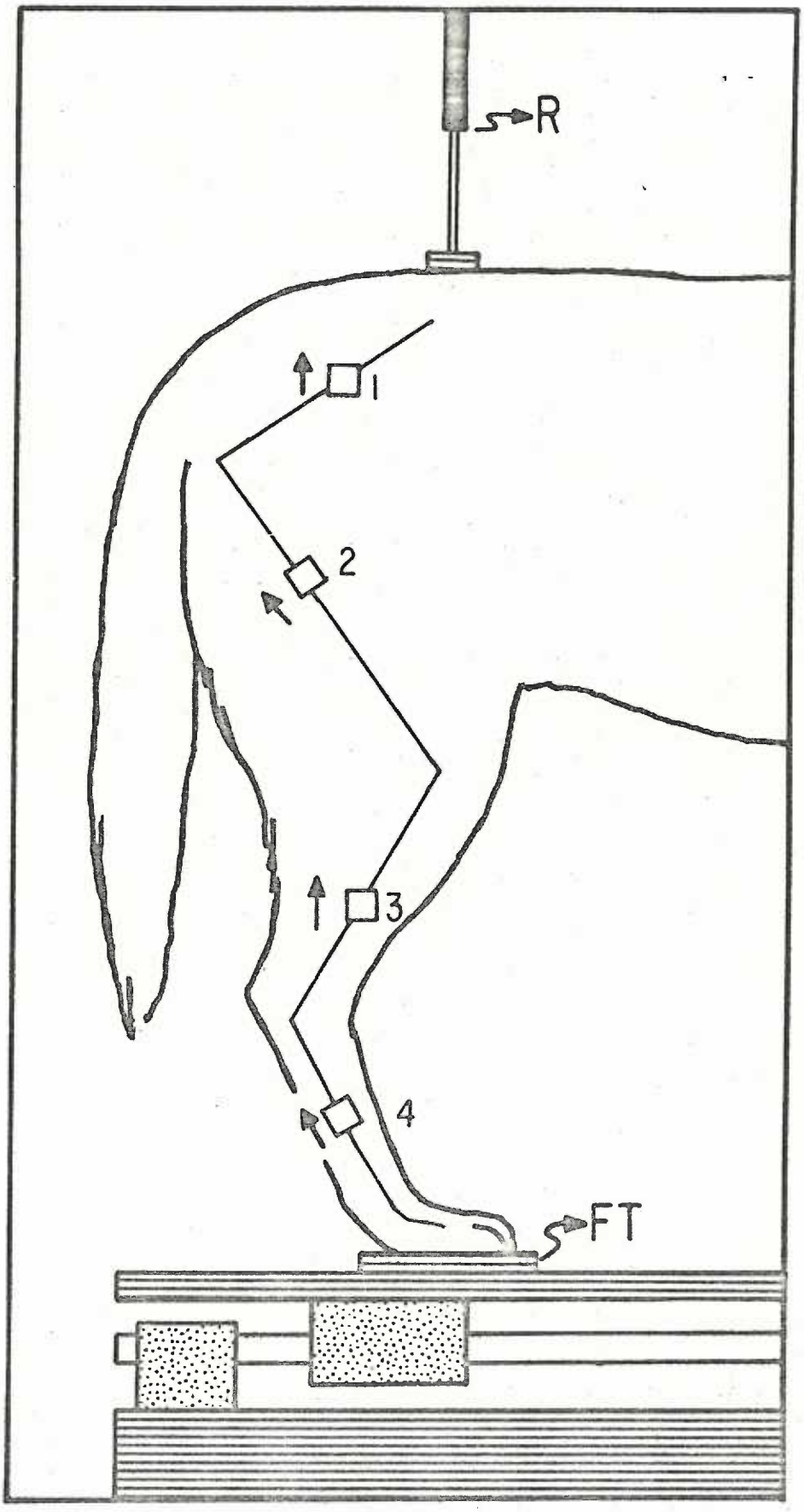
Examination of the above-mentioned statistics and consideration of some of the inherent limitations of their value led to the conclusion in the early stages of the experimental series that the most useful and sensitive indices of performance were the median trial duration and the number of trials per experiment in which one or more resets occurred. Trends in these observations usually matched the qualitative affect of "goodness" or "badness" of performance impressed on the experimenter by gross observation of the animals as the experiments were conducted.

Step Displacement Experiments

Factors other than the actual movement of the platform that differed in the displacement experiment from the quiet standing experiment included the shaving of the skin and attachment of transducer sensors and the additional noise of the hydraulic system and recording system. Quiet standing experiments were done on each dog with the sensors attached and additional apparatus turned on, and no difference in behavior was evident other than a minor "first time" distraction of attention which disappeared after a few trials. The sketch of Fig. 6 shows the placement of transducer sensors on the hindquarters of a dog.

Reactions of each animal to the displacement of the platform were highly varied and different tactics were employed in training

Figure 6. Sketch of hindquarters of a dog on the movable platform showing attachment of transducers. The feet are placed on vertical force transducers (FT). The four angle sensors are attached with Dow Medical Adhesive to the skin overlying the long bones of the hindlimb. The directions of the planes of polarization of the material covering the solar cells is indicated by the arrows. The hip joint angle is signalled by the sensor pair 1 and 2; the knee joint by 2 and 3, and the ankle joint by 3 and 4. The orientation is such that a positive output indicates a change in the direction of flexion (or closure) at each angle. The position detector (a rotary potentiometer fixed firmly at a point 50 cm above the dog's pelvis) is attached with adhesive via a telescoping steel rod (R) to the skin overlying the pelvis.


R
1
2
3
4
FT

each to stand still in the face of the displacement. The first presentation of the displacement to each dog was simply a slow ramp (20 mm amplitude, 1 to 5 seconds in duration) triggered by the sampling pulse generator at precisely two seconds after the initiation of a normal trial. With some dogs it was possible to simply decrease the ramp duration down to the desired 60 msec over the course of a few trials. With other dogs it was necessary to completely change the nature of the experiment and subject the platform to sinusoidal oscillations of varied amplitude and frequency without rewarding the dog. It was necessary with every dog to set the reset limit to 2 or 3 kg in order to avoid resets during the displacement in the training period. Eventually every animal reached the point where he could respond to the 20 mm, 60 msec displacement in a smooth repeatable manner without apparently being distracted from the task of behaving for reward. The 2 kg limit was chosen for conducting experiments since it was moderately restrictive but usually resulted in trials without reset. It was desired to record entire trials (22.5 seconds) in which reset did not occur in order to avoid possible gross shifts of weight as responses to the occurrence of reset. The training sessions for the displacement experiment were not recorded and were interspersed with regular quiet standing experiments at the 1 kg limit.

After the dogs were "well trained" they were subjected to a series of five displacement experiments of ten trials each (five

headward and five tailward displacements per experiment) over a few days' time course. Records of these experiments were made on the inkwriter as well as analog tape.

The front foot weight difference was monitored during the displacement experiments. When resets occurred the monitor was checked to determine the magnitude of the weight shift. A given trial was considered acceptable if the weight difference did not exceed 2.2 kg (10% in excess of the reset trigger level). The sampling pulse generator ran independently of the control logic after it was triggered; thus the sampling pulse train was completed in 22.5 seconds regardless of how long a trial might last. Therefore, to conserve magnetic tape and facilitate data conversion, trials with reset were always terminated soon after 22.5 seconds by switching the lamp countdown pulse interval ten times fast and manipulating the dog back to symmetry. Trials without reset were simply allowed to run free. If gross weight shifts made given trials within an experiment unacceptable, additional trials were done. In case additional trials were needed, they were always done in contiguous headward-tailward pairs so that the intervals between displacements always remained the same. Intertrial intervals were always ten seconds as in the quiet standing experiments.

The experiments always included 11 trials, only 10 of which were recorded; the platform was manually switched to the +20 mm

(headward) position, the dog was placed on the platform, the sensors appropriately attached, and an initial tailward trial was done to "set up" the dog for the experiment. If the initial response appeared smooth and normal, the FM recorder was turned on and the five pairs of trials were done. As much as two extra pairs of trials were recorded in some experiments. Notations made on the accompanying inkwriter record indicated which trials were unacceptable by the front foot weight difference criterion; abnormalities that showed up in some other fashion were also noted on the ink record and provided basis for the decision to perform extra trials. In any case, a "good" experiment had to consist of at least five headward-tailward pairs of trials with satisfactory quiet standing performance throughout each trial before and after displacement.

Data Processing

Data recorded in the step displacement experiments were converted and written in BCD format on digital magnetic tape. Appropriate annotation was written with a Teletype onto the digital tape to accompany each experiment. Visual editing of the data was done on the inkwriter records prior to conversion so that the digital tapes contained only the "good" experiments consisting of the five headward-tailward pairs of trials sampled in the formatted scheme.

The digital tapes were then read by the IBM 1130 system. Raw data files (each trial became an 1130 disk file with converted values from seven channels of analog data each sampled at 270 points) were adjusted with the appropriate calibration factor so that all files from all experiments were equivalent in amplitude scaling. Equivalent time base scaling was provided by the sampling format. Each file was named, logged in a record book, and dumped to punched cards.

Responses to displacement were examined in several ways. Individual trials could be plotted on a Calcomp model 500 incremental plotter and each converted analog data channel thus displayed. This was seldom done other than in error checking.

Averaged responses over the five individual headward and five tailward trials of one experiment could be computed along with their standard deviation at each sampling point (a rough estimate of the variability of the response). This was done on all experiments and the results plotted on the Calcomp. The same treatment was applied to all of the 25 headward and 25 tailward trials of the five pre-operative experiments. In the case of dog 8692 a machine failure in punching cards was not detected until after the original data were destroyed, and the data from one pre-operative experiment were thus lost. The pre-operative series for this dog was averaged over 20 headward and 20 tailward trials.

A variability not due to animal behavior but due simply to the experimental apparatus was removed from the data prior to averaging. This variability arose from the fact that set-up of the sensors for angle and position recording was done by hand and it was not possible to precisely set outputs to a repeatable null initial condition from one experiment to the next. If data from separate experiments were averaged, this set-up variability would, of course, be superimposed on the true animal variability. The data from each individual trial were therefore nulled to equivalent initial conditions digitally. This was done by computing the average of the ten data values at the sampling points just preceding displacement for each trial and then subtracting this value at each of the 270 data points. This treatment seemed reasonable since it was the dynamic nature of behavior that was being tested. A steady-state variability from trial to trial within an experiment is also removed by this treatment, but steady-state behavior is more effectively evaluated in the quiet standing experiment.

In addition to plotting the averaged responses, parameters of the response waveform were derived and listed by the computer for the angle and position responses. These parameters included response amplitudes, rise times, fall times, overshoot, drift, times of occurrence of overshoot and peak, and steady-state errors. They were derived by making measurements on individual trial responses. Statistics such as means, standard deviations, medians, and ranges

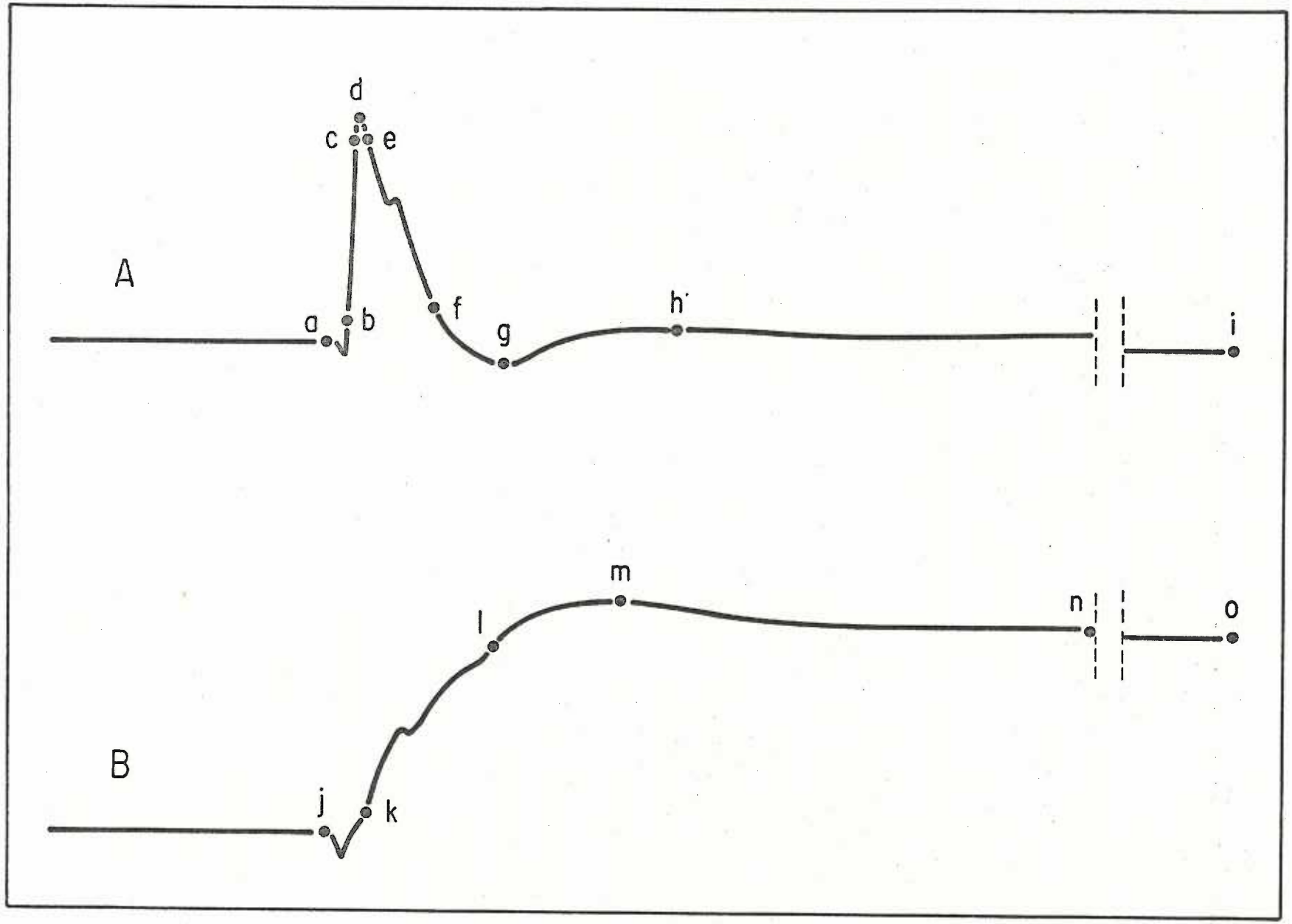
were calculated from the measurements. Fig. 7 illustrates the method of parameter determination on "typical" joint angle and position responses to displacement. The parameters were rather arbitrarily defined by making the assumption that the displacement responses were essentially complete at a point in time 2.5 seconds after the platform was triggered for angle responses and 5.5 seconds after the platform was triggered for pelvis position responses. Measurements were then made on the data at 2 seconds past the beginning of each trial (immediately prior to platform displacement), at 4.5 seconds (2.5 seconds after the platform was triggered) for angle responses, and at 7.5 seconds (5.5 seconds after the platform was triggered) for position responses. The response amplitude for angle changes was defined as the difference between the 2 sec measurement and the peak value. The position early response amplitude was defined as the difference between the 2 sec measurement and the 7.5 sec measurement. A late response amplitude for position was defined as the difference between the 2 sec and the 22.5 sec measurement.

Rise times and fall times were defined in terms of the 10 percent and 90 percent points of a rising or falling waveform. For all angle changes except the hip joint response to tailward displacement, the waveforms rose to a positive peak and then fell, more slowly and with or without overshoot, back to the pre-displacement value or nearly so. The one exception was essentially an inversion of the

Figure 7. (cont.

Amplitude value detected at m (maximum value between j and n). Amplitude values calculated for k and l (10% and 90%, respectively, of [n-j]). Points in time at k, l, m derived to nearest msec by linear interpolation between values at nearest data points. Amplitude parameters calculated as the following differences: EARLY RESPONSE AMPLITUDE = n-j; LATE RESPONSE AMPLITUDE = o-j; DRIFT = n-o; OVERSHOOT = m-j. Time parameters: RISE TIME = (time at l)-(time at k); TIME AT OVERSHOOT = time at m. In cases where actual overshoot did not occur, OVERSHOOT = n-j and TIME AT OVERSHOOT = 7.5 secs.

Figure 7. Illustration of the method of determination of displacement response wave form parameters. Each response first vertically adjusted by deriving the mean value from the first ten data points and then subtracting this value at all 270 data points, thus estimating dynamic rather than steady-state nature of behavior. A: typical angle response. Measurements made at a (data point number 10, @ 2 secs), h (data point number 180, @ 4.5 secs) and i (data point number 270, @ 22.5 secs). Amplitude values detected at d (maximum value between a and h) and g (minimum value between d and h). Amplitude values calculated for b and c (10% and 90%, respectively of [d-h]). Points in time at b, c, d, e, f, g derived to nearest msec by linear interpolation between values at nearest data points. Amplitude parameters calculated as the following differences: EARLY ERROR = a-h; LATE ERROR = a-i; DRIFT = h-i; RESPONSE AMPLITUDE = d-a; OVERSHOOT = a-g. Time parameters: TIME AT OVERSHOOT = time at g; TIME AT PEAK = time at d; RISE TIME = (time at c) - (time at b); FALL TIME = (time at f)-(time at e). In cases where actual overshoot did not occur, OVERSHOOT = a-h, and TIME AT OVERSHOOT = 4.5 secs. B: typical position response (headward; tailward response treated with appropriate sign inversion). Measurements made at j (data point number 10, @ 2 secs), n (data point number 195, @ 7.5 secs) and o (data point number 270, @ 22.5 secs).


A
a
b
c
d
e
f
g
h
i
B
j
k
l
m
n
o

pattern and was subjected to analogous measurement with appropriate parameter definition. Rise time was then defined as the time required for the response to rise from 10% to 90% of the value defined as the response amplitude. Fall time for angle responses was defined as the time required for the waveform to fall from 10% to 90% of the distance from the peak to the value measured at 4.5 seconds. For position responses, the rise time for the headward response and fall time for the tailward response were defined as the times required for the waveform to go from the 10% to the 90% points on the above defined response amplitude curves. In case of oscillation the rise and fall times were determined on the first crossing of the 10% level and last crossing of the 90% level for all waveforms.

Overshoot was determined as the maximum (for a rising waveform) or minimum (for a falling waveform) value reached after the completion of the rise (or fall) and before the 4.5 sec or 7.5 sec observation.

Drift was defined as the difference between the measurement at 4.5 or 7.5 seconds and the measurement at 22.5 seconds. For all of the above defined waveform parameter determinations, the data were first corrected for set-up variability by the subtraction of the mean of the ten pre-displacement values in each trial as described above.

For angle measurements, steady state errors were calculated at 4.5 seconds (early error) and at 22.5 seconds (late error) and

were defined as the differences between the 2 sec measurement and the measurements at these times.

Lesion and Post-Operative Observations

It happened that the dogs reached equivalent stages of step displacement training in pairs, so that a pair at a time was subjected to the pre-operative step displacement experiment series, the data processed, and the lesion inflicted, the schedule for pairs following at approximately two-week intervals. The schedule of protocol for each dog is shown in Table I.

Table I. Protocol schedule for individual dogs.

Dog	Shaping	Pre-op Displacement Series	Operation	Post-op Displacement Series	Sacrifice
	(1970)	(1970)	(1970)	(1970)	
7611	1/20	6/1-6/5	6/12	9/21-10/1	12/31/70
7624	2/18	6/16-6/19	6/29	9/22-10/6	2/5/71
8692	1/6	6/1-6/5	6/12	9/23-10/6	1/22/71
8751	1/21	6/16-6/19	6/29	9/22-10/6	2/5/71
9064	3/12	7/2-7/10	7/13	9/21-10/1	1/11/71
9073	1/6	7/3-7/10	7/13-7/16	9/23-10/6	1/29/71

All six dogs were subjected to laminectomy and dorsal column transection using the same facilities and technique. Anesthesia was induced with sodium thiamylal (Surital) and maintained with halothane administered via an endotracheal tube. A field was prepared for aseptic surgical approach to the laminae of the cervical vertebrae. The dorsal spinous process of vertebra C_2 was exposed and the nuchal

ligament was retracted to the right side, the operator standing on the left side. Exposure was deepened to the dorsal spinous process of C_3 and the lamina was exposed by scraping with a periosteal elevator, avoiding either superior or inferior articulations. Laminectomy was then produced with a medium rongeur and was made 5 to 8 mm long and wide enough to visualize each dorsal root entry line. Spinal nerve C_4 exits in the C_3-C_4 intervertebral foramen and thus the center of the exposed area was in spinal segment C_4 about 10 to 15 mm rostral to the exit of nerve C_4.

An H incision in the dura was made and the dorsal root entry line of each side was visualized with the aid of fine forceps and a loupe. A pair of cam-operated iridectomy scissors, selected because the opened blades nicely span the posterior quadrant of a cord of the size encountered, were oriented perpendicular to the cord and each blade was punched down through the pia at the root entry line to a depth estimated to reach the central canal. The scissors were then closed and withdrawn. The damage was expected to include both dorsal gray horns through which the blades penetrated as well as the entire posterior quadrant, transected upon closure of the blades.

The dura was laid back in place, the exposure packed with Gelfoam, and closure made using chromic gut or silk. Superficial seromas appeared in five out of six dogs at five to ten days postoperative, but seemed to be smaller and shorter lasting in two dogs closed

without chromic.

Two dogs (7611 and 9064) showed signs of weakness, loss of appetite, and elevated temperature within a day or two from surgery, but responded well to antibiotic therapy under the supervision of a veterinarian. All dogs were given a preventive dose of Bicillin (an intramuscular repository form of penicillin) but the dose was increased and maintained for the two dogs with the possible signs of meningitis.

The clinical observations made on dog 9073 in the first two postoperative days strongly suggested that the left posterior column was spared, so on the third day another exposure was made and a new transection of the dorsal columns was produced about 2 mm caudal to the first site.

After the lesions were produced, the animals were observed carefully for clinical signs, every few hours for the first few days and less often thereafter. As soon as each animal was capable, quiet standing experiments were performed and the course of recovery was carefully observed. Step-displacement experiments were also done during the recovery period, although the results of these observations are not reported here. Reset limits were kept as restrictive as possible without completely frustrating the animals with an impossible task. All animals were eventually capable of performing at the 1 kg limit, and a series of 15 quiet standing experiments were done on

each dog in the period about 4 to 8 weeks postoperatively. Following these series of experiments the animals all exhibited apparent clinical recovery and were subjected to a post-operative series of five step-displacement experiments each, all done in an identical manner to the pre-operative series. These experiments were done in the period of about 8 to 12 weeks postoperatively.

After the data were processed and examined with statistical tests the animals were sacrificed and the spinal cords prepared for histological identification of the extent of the lesions.

RESULTS

Histological Examination of Spinal Cord Lesions

After recovery and completion of the experimental observations, the animals were sacrificed and the spinal cords were examined to determine the extent of the lesion. An anesthetic dose of sodium pentobarbital (Nembutal) was given, and each animal was bled from the femoral artery. All animals but one (dog 7611) were perfused by intracardiac injection of normal saline followed by 10% formalin, with the descending aorta clamped. The spinal cord from below exit of nerve C_5 to and including the first 1 cm of the brainstem above the obex was removed and placed in formalin. Blocks were then cut for histological preparation, five blocks from each spinal cord taken from approximately the same location. The block containing the lesion from each cord was serially sectioned and stained at 450 micron intervals, and single samples were prepared from each of the remaining blocks. The locations of the blocks were: rostral segment C_5 (below exit of nerve C_4); the portion of segment C_4 containing the lesion (the adherent scar tissue covered an extent of 10 to 12 mm); caudal segment C_3 (above exit of nerve C_3); caudal segment C_2 (below exit of nerve C_2); and brainstem, 2 to 3 mm above the obex. Gross examination of the tissue and inspection for signs of Wallerian degeneration, cysts, or other abnormalities was done and the blocks were then

imbedded in paraffin and cut to 15 micron sections and stained with a combination of Luxol fast blue (for myelin) and Holme's axon stain.

Microscopic examination of the stained sections was then done to determine the nature and extent of the lesions. The illustrations of Fig. 8, in two parts, show cross sections at levels estimated to nearly coincide with the actual scissors penetration and approximately one spinal segment rostral to the lesion for each animal. The drawings were made by tracing over the projected tissue sections under a 35 mm photographic enlarger. The tracks of the penetrating scissors blades can be detected in the sections at the site of the lesion.

In two dogs (7611 and 9064) cysts, approximately 1 mm in diameter, were present in the formalin treated tissue, stained brown, possibly with hematin, and extending longitudinally into segments above and below the lesion. They appeared to dissect through the dorsolateral funiculus (left in dog 7611 and right in dog 9064) without doing damage to neural tissue. These may have been related to the signs of weakness, elevated temperature, and delayed recovery seen in these animals. The extent of the cysts in the region of the lesion was difficult to detect.

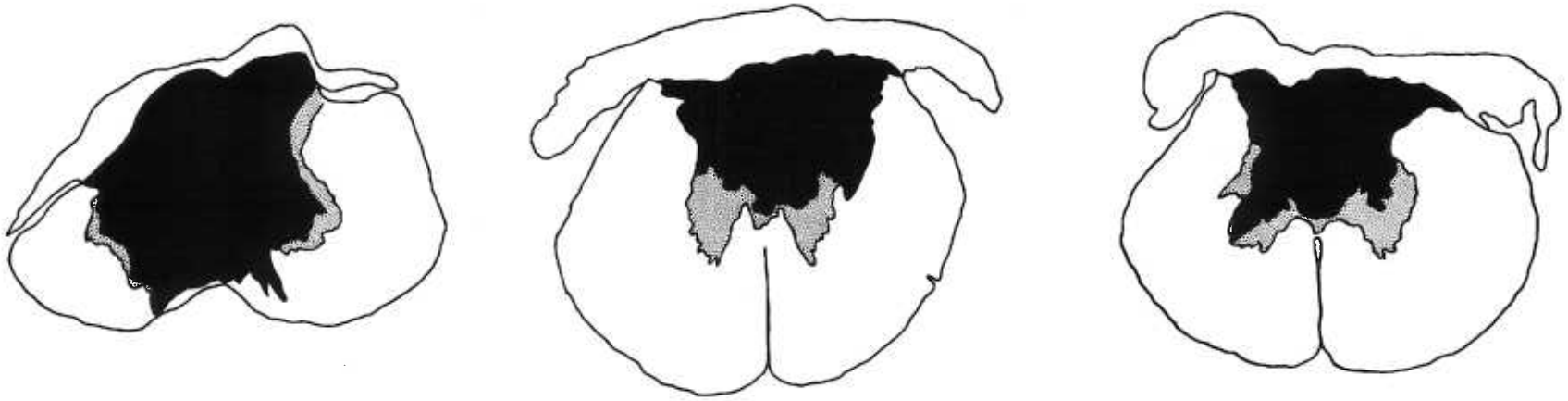
In the two dogs in which the lesion spared the central canal of the cord (8751 and 9064) some remnant of the posterior column fibers was seen, the largest remnant being in dog 8751. For the remaining animals, posterior column transection was complete, the destroyed

Figure 8. Sketches of spinal cord cross sections at two levels from each animal. Sketches were prepared by tracing projected sections under 35 mm photo enlarger. Sections at level of lesion were selected from the 450 μ skip serial sections and estimated to most closely coincide with the actual scissors penetration. Regions of complete fiber tract or cell body degeneration are blacked out; partial degeneration in dorsal portions of lateral quadrants appear as mottled. Gray matter of spinal cord is indicated by fine stippling.

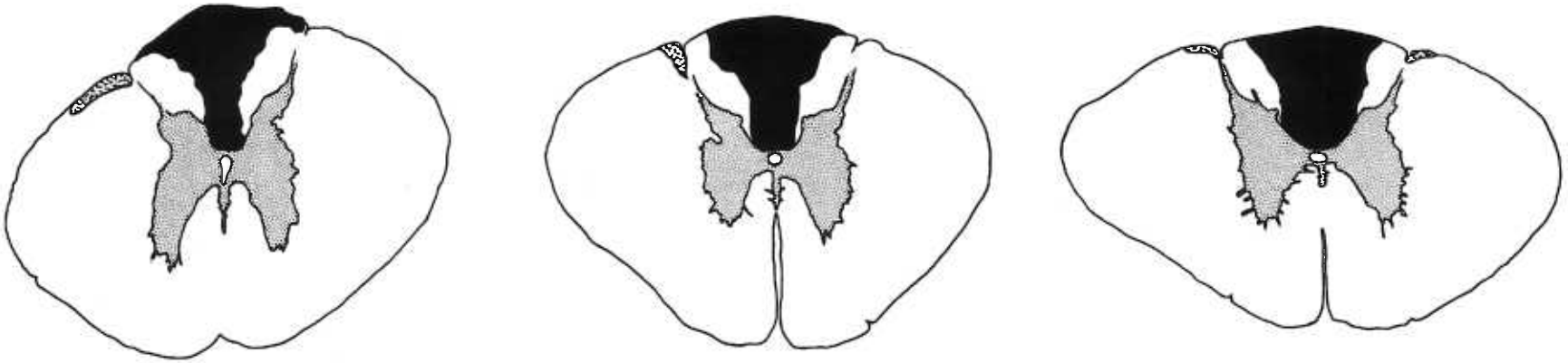
DOG 7611

DOG 7624

DOG 8692



SECTIONS AT LEVEL OF LESION

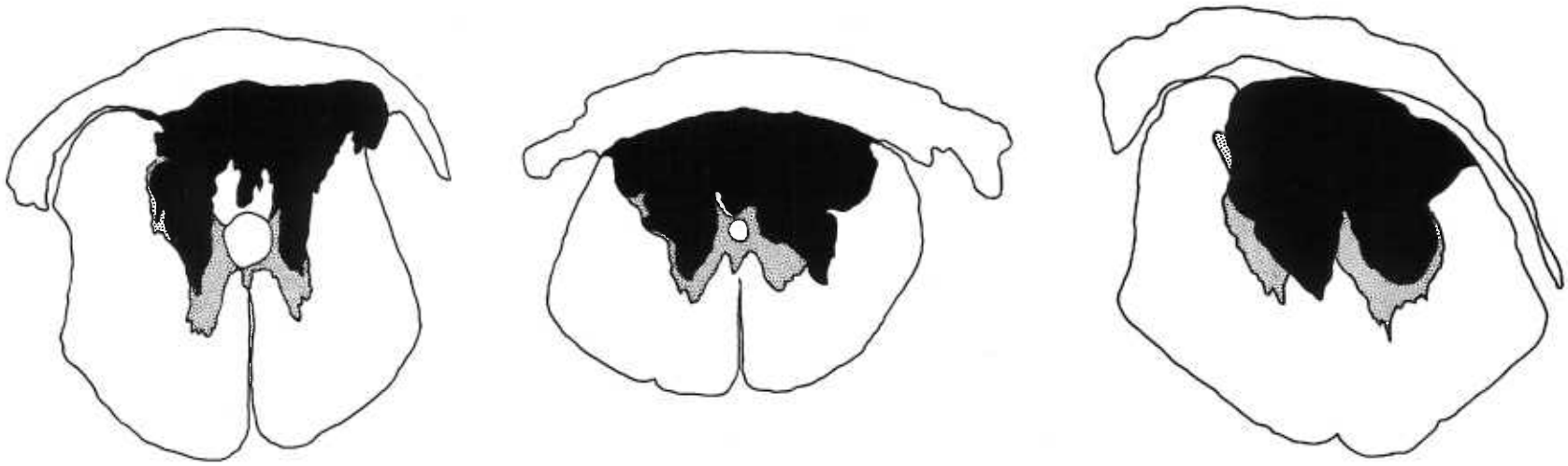


SECTIONS ONE SEGMENT ROSTRAL TO LESION

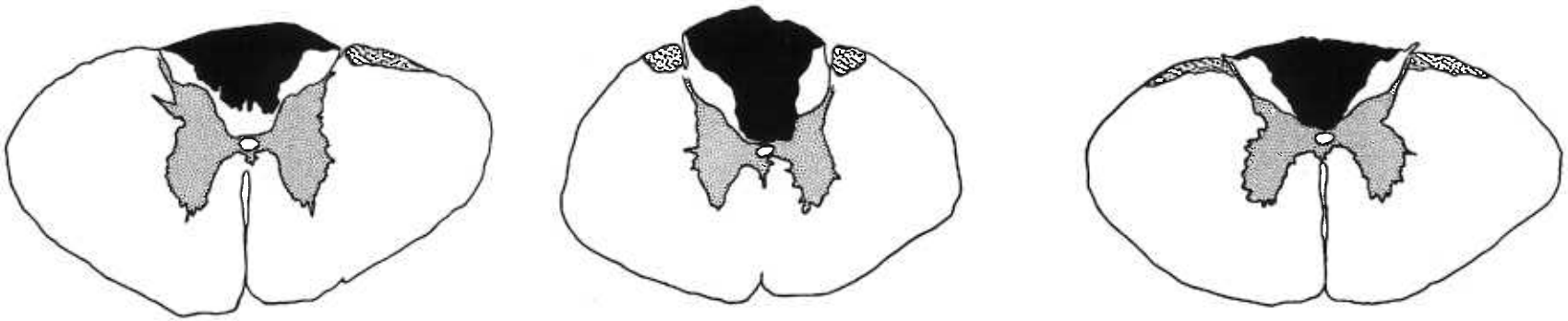
DOG 8751

DOG 9064

DOG 9073



SECTIONS AT LEVEL OF LESION



SECTIONS ONE SEGMENT ROSTRAL TO LESION

area including some or all of the dorsal gray horns and the medial gray to the level of the ventral white commissure.

Extension of degeneration into some ascending and descending fibers of the dorsolateral columns was seen, and the origin of this additional degeneration did not appear to be direct trauma to fibers. Ascending or descending branches of small arteries were apparently cut so that evidence of ischemia, especially in the gray, showed up in several serial sections above and below the ones illustrated. The degeneration of ascending fibers in the extreme dorsal dorsolateral columns was very prominent at a level 2 to 3 mm above the site of the lesion and continued largely unchanged in intensity or location through spinal segment C_2. These fibers may well have arisen from cells in the ischemic gray. They might also have been long tract fibers indirectly traumatized by compression as the scissors blades were punched through the pia-arachnoid.

A slightly more diffuse region of descending degeneration was seen, to a lesser or greater degree in extent, but in the same region for all dogs except 7611. The region was in the medial portion of the posterior half of the right lateral quadrant. The extent ranged from about 0.5 to 1 mm in diameter and the intensity ranged from partial to complete at the center. A similar pattern of descending degeneration was seen in dog 7611, but on the left rather than right side. In addition to the posterolateral degeneration, however, some diffuse

degeneration in this dog appeared to sweep down into the ventral quadrant and was most prominent in a band on both sides of the ventral sulcus. The origin of all descending degeneration was difficult to localize but appeared to be related in magnitude to the extent of the ischemic degeneration in the gray horns below the lesion.

From the apparent extent of damage at the site of lesion in dog 7611 it is surprising that so much white matter was intact above and below the lesion. An extensive cyst apparently contributed to the distortion of the section at lesion level. The magnitude of the damage corresponded with the clinical observation that the most profound and long-lasting signs were seen in this dog. No clearly recognizable clinical signs of asymmetry were observed in any animal.

The stain used did not facilitate tracing of degeneration into the brainstem. The degeneration of dorsal column fibers in the C_3 samples appeared very complete and looked entirely different from the diffuse ascending and descending degeneration seen in lateral and ventral quadrants as described above. A very few widely scattered large diameter intact fibers were occasionally seen in the degenerated area, but were most likely projections of afferents entering over roots above the site of the lesion. The prominent layers of intact afferents from neck region are seen on the lateral borders of the posterior quadrant in C_3 and demonstrate the large numbers of afferents arriving over dorsal roots in this region of the cord. It was not

possible to detect descending degeneration in the interfascicular region in the C_5 sections by simple microscopic observation. This does not rule out the existence of, but seems to minimize the dimension of, the fasciculus interfascicularis in this portion of the dog spinal cord. The absence of any intact fibers in the C_3 sections of the dogs suffering complete C_4 posterior column transection indicates that any propriospinal fibers in the cervical cord at least do not descend through the C_3 dorsal quadrant.

Quiet Standing Experiments

Introduction

The task of evaluating the quiet standing performance of the dog has gone through several gross changes in method over the past few years although the goal remains unchanged. At the outset it was desired to ascertain precisely how "still" the dog could stand as he assumed a trained "command" posture (17). It was also deemed valuable to estimate the time course of approach to the stable trained condition. Hopefully, the base of recorded information from the normal dog would provide control against which to test the effect of procedures such as peripheral nerve block or central nervous system lesion on the animal's capability to stand still.

It should be noted that the basic assumption in recording from the quiet standing animal is that he is standing still to the very limit of his capability. The validity of this assumption is crucial to the conduct of the nerve block and lesion experiments. Evolution of the experimental technique centered around this point, and the method used in these studies seems to assure that it is the dog's capability and not his willingness or attentiveness to the task that is being measured.

Establishment of a Sensitive Test of Performance

As discussed previously, several statistics derived from time measurements were examined but were considered to be inutile as the experiment progressed primarily due to the necessity of intervention by the experimenter during the course of individual trials. The only remaining information that could be extracted from the records and would seem to have a significant bearing on the quality of performance was that pertaining to the occurrence of resets. Such information included the total number of resets, the average number of resets per trial, and the number of trials per experiment in which reset occurred. A definite weakness in use of numbers of resets as bases for statistical testing was also apparent. Depending on whether or not an animal was panting (a type of behavior that was accepted and not discouraged during the experiments) the total number of

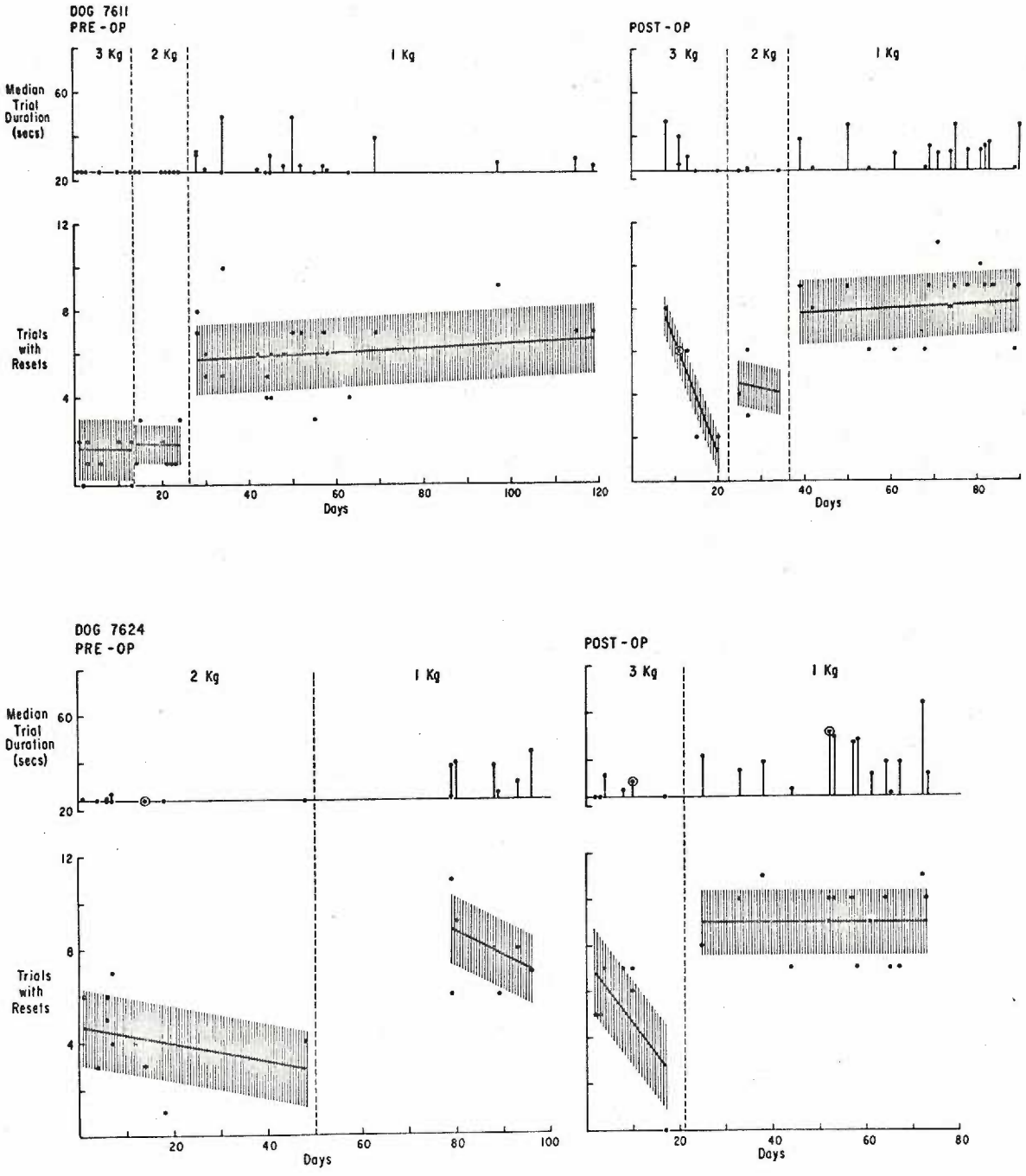
resets that would occur in a single drift toward the reset limit might be only one (without panting) or as many as 20 (with panting), yet there would be no readily discernible difference in the "quiet standing" behavior between the two cases.

Although data of all types mentioned above were kept throughout the course of the experiments, the impression evolved that a satisfactory method of testing might be to examine individual trials simply on the basis of a reset-no reset dichotomy. The criterion for an additional stepwise restriction of limits as the animals progressed in performance was the occurrence of 24-second median trial durations in several successive experiments. This could also be considered a dichotomy-type criterion, that is the occurrence of six or more trials without reset in several successive experiments.

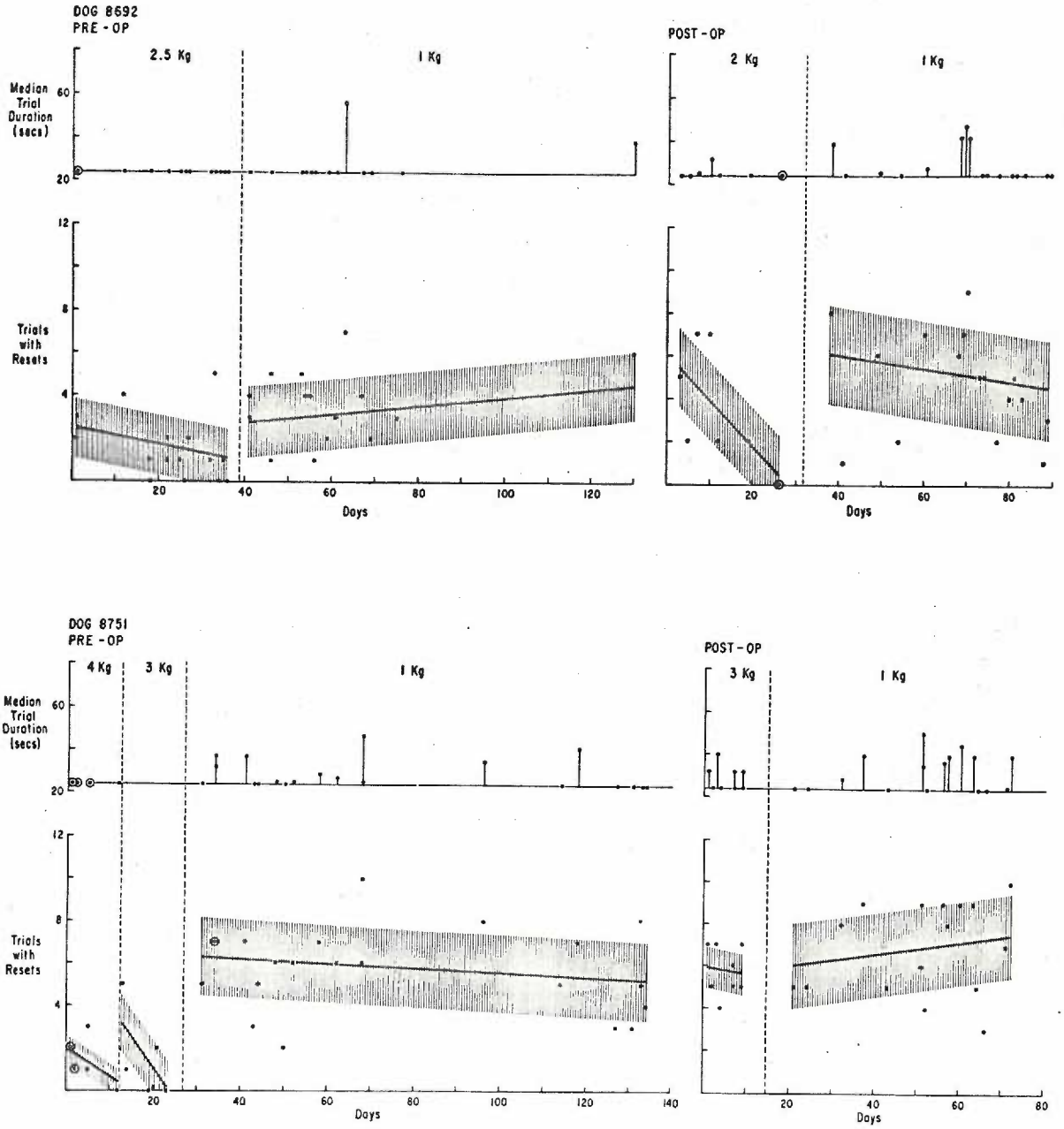
The observations plotted in Fig. 9, which appears in three parts, include the median trial durations and number of trials per experiment with one or more resets for all dogs. The data collected from the normal dogs were subjected to a test intending to determine the efficacy of the dichotomy treatment in estimating the level of performance. For some dogs there was little or no difference in the number of trials per experiment with one or more resets as the limits were shifted between values less restrictive than 1 kg limits. So the data were lumped into two sets: those collected at limits less restrictive than 1 kg (i.e., 4, 3, and 2 kg) and those collected at the

Figure 9. Summary of quiet standing experiment data, in three parts. Observations of median trial duration from each 11-trial quiet standing experiment (upper portion) and number of trials per experiment with one or more resets (lower portion) plotted across time for each dog. Circled points indicate identical observations in two experiments done on the same day. Line of regression of trials with resets against time is drawn for each set of observations at each limit setting. Vertical dashed lines separate the sets of observations into groups done at the limit settings indicated at the top. Hatched areas indicate regions within one standard error of estimate of regression of resets on time.

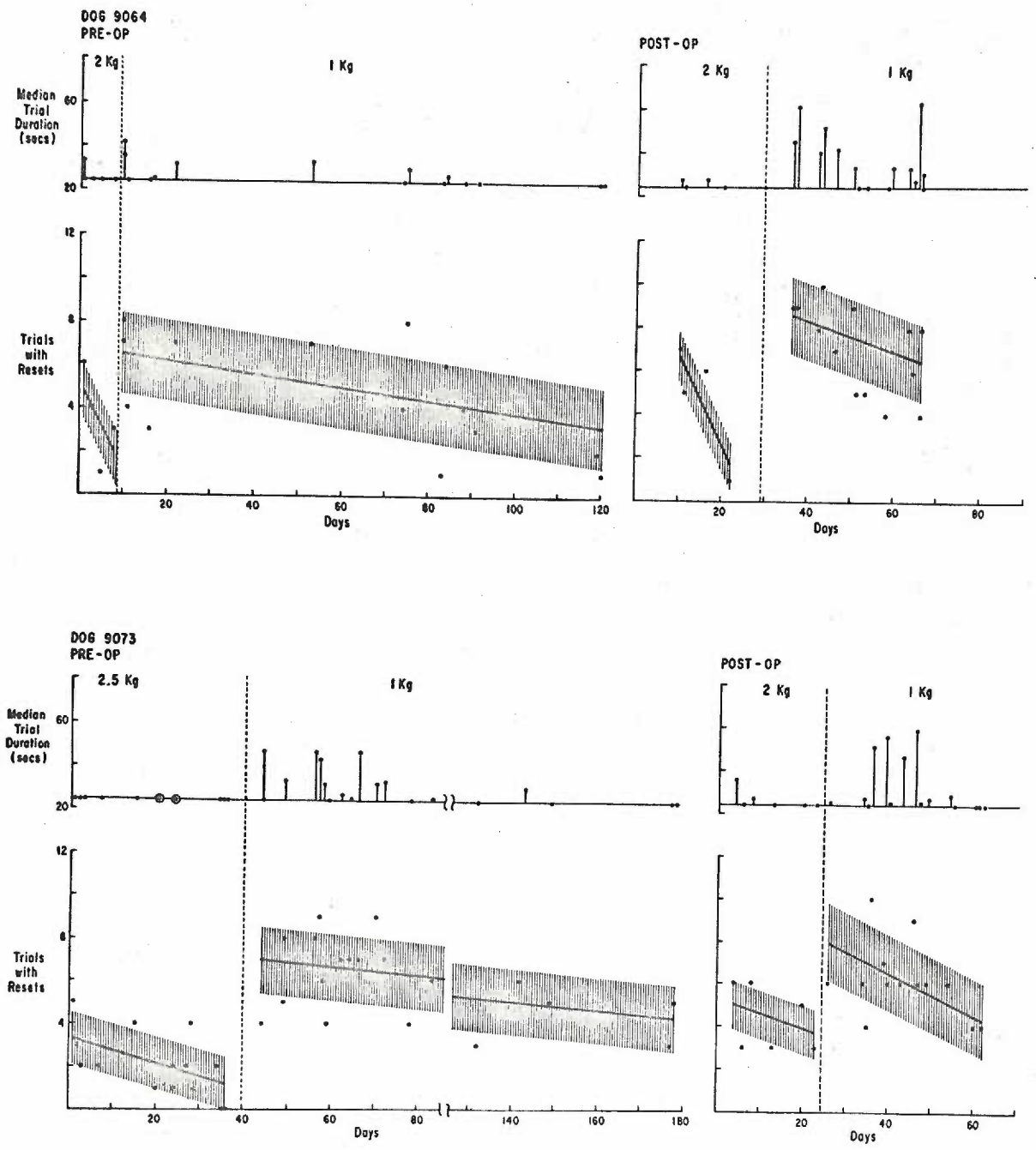
A


DOG 7611
PRE - OP
POST - OP
3 Kg
2 Kg
1 Kg
Median Trial Duration (secs)
Trials with Resets
Days
DOG 7624
PRE - OP
POST - OP
2 Kg
1 Kg
3 Kg
1 Kg
Median Trial Duration (secs)
Trials with Resets
Days

B


DOG 8692
PRE - OP
2.5 Kg
1 Kg
POST - OP
2 Kg
1 Kg
Median Trial Duration (secs)
Trials with Resets
Days
DOG 8751
PRE - OP
4 Kg
3 Kg
1 Kg
POST - OP
3 Kg
1 Kg
Median Trial Duration (secs)
Trials with Resets
Days

C


DOG 9064
PRE-OP
2 Kg
1 Kg
POST-OP
2 Kg
1 Kg
Median Trial Duration (secs)
60
20
Trials with Resets
12
8
4
20
40
60
80
100
120
Days
20
40
60
80
Days
DOG 9073
PRE-OP
2.5 Kg
1 Kg
POST-OP
2 Kg
1 Kg
Median Trial Duration (secs)
60
20
Trials with Resets
12
8
4
20
40
60
80
140
160
180
Days
20
40
60
Days

1 kg limit. The test used was the Wilcoxon signed-rank test taken from Bradley (14). This is a distribution-free test and thus calls for no restrictive assumptions about normality of data distribution. Since different numbers of trials were done on each dog, the data were normalized to a ratio of trials with resets to total trials. The data in Table II are tested to detect a difference, if any, in these ratios under the two performance conditions.

The null hypothesis is that there is no difference between the observations under the conditions of limits less restrictive than 1 kg and the condition of 1 kg limits. The test statistic W_+ is found to be zero, and from the tabulated distribution of the statistic for N = 6, the probability of zero is less than 0.0156. The null hypothesis is thus rejected with $p < .0156$, and it is inferred that the experimental scheme provides a sensitive test of the capability of the normal dog to maintain a quiet stance with front foot weight distribution maintained within a certain limit. The "threshold" for each animal was not detected, but rather a "suprathreshold" limit for all dogs was established. Only two of the dogs were ever subjected to trials at a limit more restrictive than 1 kg (0.5 kg). Neither was ever capable of completing a single trial at the 0.5 kg limit, so that no experiments could be conducted nor statistical tests performed on behavior at such a limit. It seemed that the test at 1 kg limit was sensitive enough and provided a satisfactory control against which to test operated

Table II. Wilcoxon signed-rank test on quiet standing performance of normal dogs.

	Greater than 1 kg limit			1 kg limit			(A)-(B)	Signed rank (SR)
	Trials with resets	Total trials	Ratio @ 1 kg (A)	Trials with resets	Total trials	Ratio @ 1 kg (B)		
Dog 7611	25	187	.133	140	242	.578	-.455	-6
Dog 7624	39	110	.355	55	77	.714	-.359	-4
Dog 8692	23	165	.139	54	165	.188	-.188	-2
Dog 8751	18	154	.117	112	220	.509	-.392	-5
Dog 9064	18	55	.327	67	165	.406	-.079	-1
Dog 9073	36	187	.193	121	220	.550	-.357	-3

$W_- = \sum_{S_i=-} S_i R_i = -21$ $\qquad$ $W_+ = \sum_{S_i=+} S_i R_i = 0$

for $N = 6$, $p(W_+ = 0) < 0.0156$

animals.

Qualitative Aspects of Normal Dog Behavior

All of the dogs included in the present study became very familiar with the experimental apparatus and learned to inspect the feeding dish for meatballs upon entering the laboratory. They usually would walk directly to the platform, stand on it (never in the command posture with feet properly placed), and gaze intently at the feeding apparatus as if expecting a meatball to be dispensed. In order to begin an experiment, however, it was always necessary to place the dogs in the command posture by hand and this might involve placing and replacing feet on the force transducers up to half a dozen times. Once these manipulations were complete and the first trial started, the auditory cue nearly always brought about an "alerting" response where the dogs perked up ears, fixed their gaze either on the light panel or the feeding dish, and generally stopped all gross body movements.

After the initiation of the first trial of each experiment the behavior of each animal became more individualized and less subject to general description. There was never any convincing evidence that any dog intently followed the progress of the light countdown. The auditory cue did not seem to produce any overt response when the animals shifted in and out of the reset mode. It was usually necessary to

give hand signals or physically manipulate the dogs back to the symmetric stance when they drifted out, otherwise they might stand very still, in the reset mode, for five minutes or more without attaining a reward.

Nonetheless, all of the dogs did show evidence of expecting the reward to be dispensed as the light countdown neared completion in each trial. Regardless of what sort of individual behavior pattern might have developed during the early portions of each trial, all of the dogs perked up their ears and fixed their gaze on the feeding dish when the countdown reached the fourteenth or fifteenth light. They all apparently learned to stand still in order to attain reward, but not one dog seemed to grasp the necessity of maintaining front foot symmetry in order to avoid reset. Slow, unidirectional drift in some dogs might cause repeated resets in a given trial and the reward would ultimately be dispensed only because the drift happened to be slow enough to barely fail to cause reset during one 24-second period. A non-drifting stance set well toward the reset limit in some instances might result in repeated resets as the dog panted, but the dog would make no shift in stance without a signal from the experimenter. These evidences of apparent inadequacy of the experimental design are offset by the observation that all dogs were successful in completing large numbers of trials without reset, even at the most restrictive limit setting (see Table I).

Qualitative Aspects of Post-Operative Dog Behavior

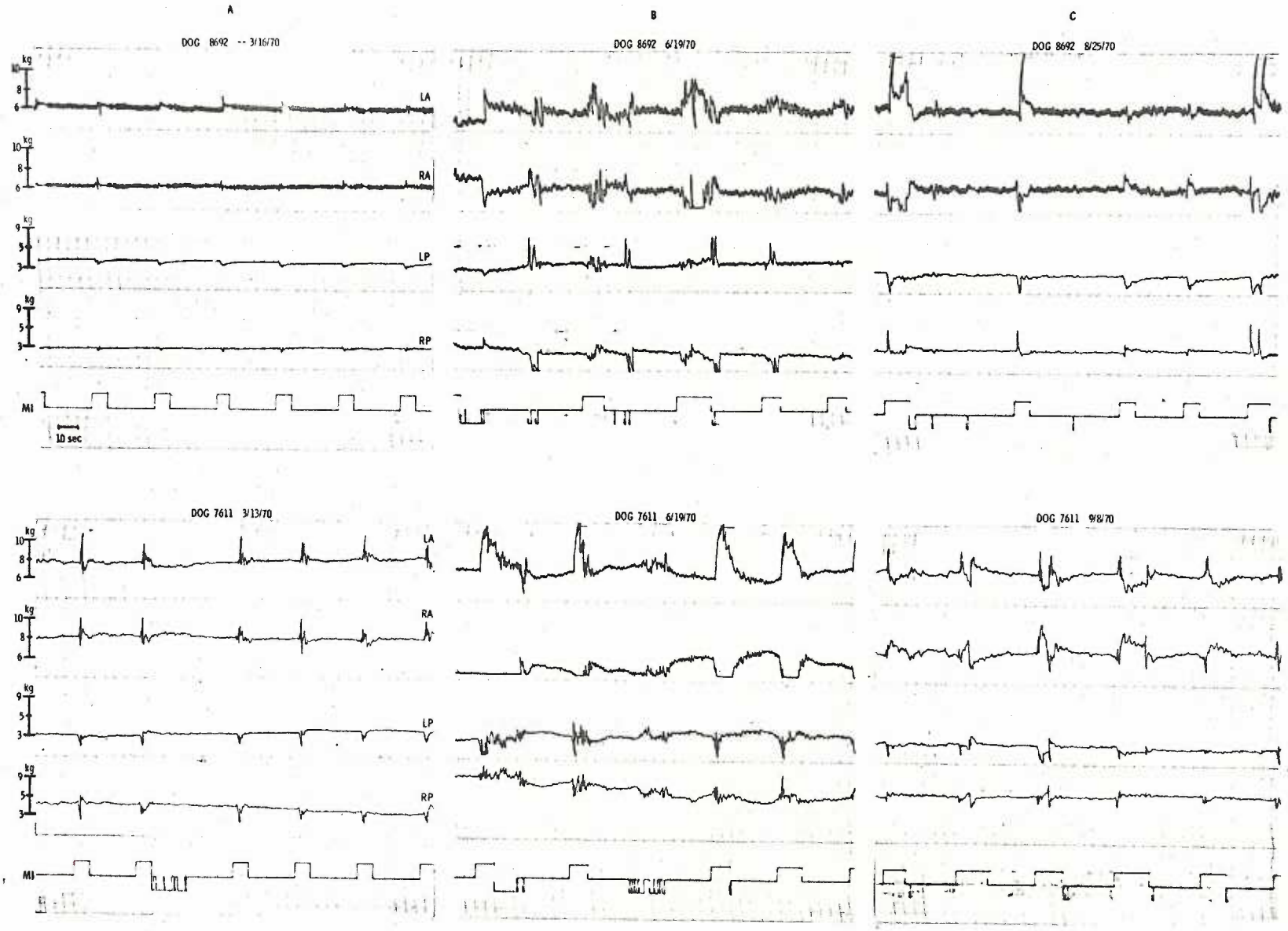
After the laminectomy and dorsal column transection the dogs showed a variable time course of recovery. Two dogs (7611 and 9064) showed signs possibly related to post-surgical meningitis with fever, loss of appetite, and grossly depressed physical activity, but responded to antibiotic treatment.

Several stereotyped behavioral traits were evident in all the animals during recovery. On the first post-operative day all of the dogs were able to stand and begin walking with the exception of dog 7611. The gait was slow and extremely ataxic, and they usually took very few steps without falling. Forelimb tonus appeared to be very poor, and the forelimbs would collapse often, even when the animals were standing still, so that they could only stand for brief periods of time, usually less than a minute. They would very often step or support weight on the dorsum of their forepaws, and this was often the cause of falling as they attempted to walk. The placing, hopping, and supporting reactions of all four limbs seemed to be absent; the limbs could be placed in awkward postures without being replaced by the dog. The early post-operative vigor of gait and duration of brief periods of stance varied from dog to dog. Dog 7611 was unable to stand on his own until the fourth post-operative day, while dog 8692 actually ran out of his recovery cage into the hallway (awkwardly)

only three hours after the surgery was completed. The recovery pattern from dog to dog varied primarily in time course; all of the described traits were observed in all of the dogs in the same general sequence. Within two or three days the hindlimb placing, hopping, and supporting reactions began to return to normal, and the evidences of the lesion seemed to be confined to the forelimbs. Very gradually over a few days time the overall activity of the dogs increased toward normal, the collapsing of the forelimbs diminished, the stumbling and falling diminished and the gait became more coordinated. Between 7 and 14 days a characteristic gait began to show up which persisted to some small degree until the animals were sacrificed. During ambulation the forelimbs were lifted abnormally high with each step as if to overcome the problems of stepping on the forepaw dorsum and stumbling. This high stepping gait was a characteristic phenomenon in all the animals. There were varying degrees of long-term persistence of signs of the lesion, with the most rapid and complete return toward normal behavior exhibited by dog 8692 and the slowest and least complete seen in dog 7611. Six months postoperatively, dog 7611 still showed signs of awkward gait with occasional stumbling. For all other dogs it was difficult to detect any gross sign other than a faint residue of the high stepping gait after about two months of recovery.

In the experimental condition, however, distinct signs of the lesion were readily apparent in records of forces beneath feet and position of the body. Fine oscillations in vertical force, about 0.5 to 1 kg in amplitude and 0.5 to 1 Hz in frequency showed up in the front foot force records. These oscillatory force records were highly recognizable and consistently observed in all dogs and persisted for the duration of the post-operative observation time. Larger oscillations, slower in time course, were apparent during early recovery and actually represented instability in the position of the center of weight distribution. These larger oscillations correspond to grossly observable sway in the stance of the animals. Even after the sway in the stance disappeared (within two to three weeks) the superimposed fine oscillations persisted. Hindlimb forces and body position also exhibited traces of this fine oscillatory pattern, but in the digitized and averaged data displays no evidence of it remained. Examples of the records of forces beneath feet kept during quiet standing experiments for two dogs are shown in Fig. 10. The records are intended to display typical performance at three stages during the course of the experiments: stage A, during the pre-operative series of observations at 1 kg limits; stage B, during the early post-operative recovery period, and stage C, during the recovered post-operative series of observations at 1 kg limits. The examples are taken from the clinically "best" (dog 8692) and "worst") (dog 7611) subjects judged on an overall impression of the rapidity and

Figure 10. Examples of force records made during quiet standing experiments from two dogs at three stages. A. Pre-operative, during the series of observations at 1 kg limits. B. 7 days post-operative. Limits 2 kg for dog 8692 and 3 kg for dog 7611. C. Recovered post-operative, during the series of observations at 1 kg limits. See Fig. 5 for explanation of mode indicator record (MI). Force records: LA, left anterior; RA, right anterior; LP, left posterior; RP, right posterior.


A
B
C
DOG 8692 -- 3/16/70
DOG 8692 6/19/70
DOG 8692 8/25/70
DOG 7611 3/13/70
DOG 7611 6/19/70
DOG 7611 9/8/70
kg
LA
RA
LP
RP
MI
10 sec

completeness of recovery. Note the lateral symmetry in both anterior and posterior foot forces in the pre-operative condition, with an approximately 60% anterior-40% posterior total weight distribution. This condition is severely disturbed in the early post-operative stage, with two separate types of instability in evidence. The first type is a gross disturbance in the mean pattern of weight distribution with large, low frequency shifts about the normal mean (whole body sway). The second is a finer, occasionally periodic oscillatory pattern superimposed on the total force and more prominent in the front foot records. The wide band of the front foot records from dog 8692 is due to panting, a behavior he engaged in almost 100% of the time during observation, and it can be seen that the postoperative fine oscillations are at a much lower frequency than panting. In the recovered post-operative stage, the large oscillations are greatly diminished; the mean weight distribution seems to be very nearly as stable as in the normal dog, with the mean distribution pattern very similar to normal. The hind foot asymmetry in the record from dog 7611 was not a consistently observed phenomenon. The long-term persistence of the fine force oscillations is apparent in the records made during the recovered post-operative stage.

Quiet standing experiments conducted in the first few days post-operatively were often subject to termination without completion when the animals simply lost the support of their forelimbs and collapsed.

The collapse occurred usually due to a forward buckling of the wrist joint. Even in these terminated experiments, however, all of the dogs were capable of completing a few trials without reset at the wide limit (usually 3 kg). They all appeared to be eager to work for the reward and were apparently attempting to maintain a quiet stance. Their stances appeared to be very awkward and uncomfortable and they exhibited large degrees of body sway which resulted in many resets at the wide limits. Gradual recovery of capability is evidenced in the plotted regression of resets on time in Fig. 9. Recovery of a grossly normal and comfortable appearing stance followed the same two to three week time course as the first post-operative segments of the plotted reset results.

During the period of about four to eight weeks post-operative the quiet standing performance seemed to be stabilized with little evidence of continued recovery. Experiments were conducted at the 1 kg limit during this time to be used to compare to the pre-operative experiments at 1 kg.

Quantitative Comparison of Behavior of Normal and Post-Operative Animals

The data from quiet standing observations on the normal and post-operative dogs were assembled in order to detect possible differences in performance between these conditions: (1) normal dogs,

limits less restrictive than 1 kg, (2) normal dogs, 1 kg limit, (3) post-operative dogs, limits less restrictive than 1 kg, and (4) post-operative dogs, 1 kg limit. The test used is the Friedman test taken from Bradley (14), and like the Wilcoxon test used above is sensitive to differences in quantitative observations of similar types made on groups of individuals under different conditions. Systematic tendencies across conditions register a positive result (rejection of the null hypothesis) even in the face of large differences in the behavior of individuals within a condition.

The observations are entered in Table III and the conditions are ordered 1, 3, 2, 4 (less restrictive limits before the 1 kg limits). Rejection of the null hypothesis implies that the performances under the different conditions are different (not identical). The test statistic S is found to be 150, and from the tabulated critical values of S for six subjects and four conditions, $p(S \leq 128) = 0.001$. Therefore, the null hypothesis is rejected, with $p < 0.001$, that the performance across conditions does not change. The inference is that the conditions represent tasks of increasing difficulty (in the order 1, 3, 2, 4), or that the animals diminish in capability of performing without reset across conditions; a significantly higher ratio of trials with resets to total trials is seen in the whole group of animals across conditions.

Table III. Friedman multi-sample test on quiet standing performance of normal and operated dogs.

	Condition 1 Limit less restrictive than 1 kg, pre-op				Condition 3 Limit less restrictive than 1 kg, post-op				Condition 2 1 kg limit, pre-op				Condition 4 1 kg limit, post-op			
	Trials with reset	Total trials	Ratio	Rank	Trials with reset	Total trials	Ratio	Rank	Trials with reset	Total trials	Ratio	Rank	Trials with reset	Total trials	Ratio	Rank
Dog 7611	25	187	.133	1	47	110	.427	2	140	242	.578	3	133	176	.756	4
Dog 7624	39	110	.355	1	41	88	.466	2	55	77	.714	3	136	165	.824	4
Dog 8692	23	165	.139	1	25	88	.284	2	54	165	.327	3	75	176	.426	4
Dog 8751	18	154	.117	1	51	99	.515	3	112	220	.509	2	111	176	.631	4
Dog 9064	18	55	.327	1	19	44	.432	3	67	165	.406	2	109	165	.661	4
Dog 9073	36	187	.193	1	26	66	.394	2	121	220	.550	4	84	165	.509	3
Ti				6				14				17				23

$$\left.\begin{matrix} R = 6 \\ C = 4 \end{matrix}\right\} \quad \frac{R(C+1)}{2} = 15$$

$$S = \sum_{i=1}^{C} \left[T_i - \frac{R(C+1)}{2} \right]^2 = \sum_{i=1}^{4} \left[T_i - 15 \right]^2 = 81 + 1 + 4 + 64 = 150$$

$$p\left[s \geq 128 \right] = 0.001$$

A second aspect of behavior of the animals that was quantitatively examined was a possible trend toward increased quality of performance over time (i.e., an evidence of "learning" the task or becoming more proficient upon repeated exposure). Many types of "moving average" tests were examined but found to be not applicable; most tests for trend require continuity, and the reset data of Fig. 9 are seen to have large discontinuities at the points where limits are changed. A simple linear regression of resets on time with a test for independence (28) was used, and the regression lines calculated over observations at each separate limit setting. Many of the regression lines show a negative slope, which can be interpreted as a tendency toward fewer resets with repeated exposure to the task. Only in one case, however, did the test for independence show a significant ($p < 0.05$) relationship between performance and time, and that was for dog 7611 at the 3 kg limit postoperatively. Increased numbers of observations may have provided a more clear basis for statistical testing, but the plan for conducting the observations was to keep moving on to the most restrictive limit possible in order to test the capability of the animals.

Summary

In summary, these results are based on a technique of proven sensitivity for evaluating the capability of a dog to maintain a quiet stance with lateral symmetry of front foot weight distribution and

describe the effect of bilateral transection of the cervical spinal cord dorsal columns on this capability. The spinal cord lesion causes a severe transient impairment of performance, with a marked daily improvement over a three week time course. A slight degradation of performance persists in recovered animals. The mean pattern of weight distribution is normal and seems to be nearly as stable in recovered animals as in normal animals. A persistent instability, evident primarily in front foot force records, can be seen in fine oscillations of force, roughly periodic in some instances, that are superimposed on a normal mean. These oscillations are reminiscent of a pattern previously described in dogs with anesthetic blockade of digital afferents (80).

Step Displacement Experiments

Introduction

Dynamic aspects of control system behavior are conveniently characterized by observing responses at the output occurring when controlled inputs are applied (78). Among the analytically useful types of controlled input to a system is included the input step, and reasons have been given for considering a brief ramp of displacement to be a virtual step input to the dog postural control system (79). The nature of the postural control system response to displacement has been

observed only in rather gross aspect and the new type of observation reported in this study, that of the changes of hindlimb joint angles occurring in response to a "step displacement", is again of a gross nature. In the final analysis precise quantitative information regarding transfer functions of neuronal relays and mechanical function of posture-maintaining muscles will have to be obtained, but in the meanwhile the more gross aspects of system behavior are expected to be qualitatively useful in gaining insight into system behavior.

Qualitative Aspects of Normal Dog Response

Examples of individual responses to displacement of the platform during task performance are shown in Figs. 11 and 12. These examples were not selected for any particular feature and are intended to convey the impression that each response pattern, i.e., knee, headward; hip, tailward, etc., is recognizable in such features as direction of initial deflection and gross shape of the response pattern. In these as well as following figures the event producing the response was the displacement of the platform over a distance of 20 mm in 60 msec as seen in the lowermost plotted variable. Time zero marks the beginning of the trial, initiated by the experimenter by depressing the start switch. Coincident with the tenth data sampling point, at 2 sec past the beginning of the trial, the platform was triggered to move. The trial was initiated at a time when the stance of the dog was quiet and

Figure 11. Records from an individual pre-operative headward displacement. These plotted responses were prepared only for illustrative purposes and such plots were not usually generated unless needed for error-checking. The digitized and amplitude-calibrated outputs of each transducer during the course of a single trial during which a step displacement of the platform occurred were plotted by the Calcomp plotter. The data were plotted from a single digital file, one of the 25 used to derive the appropriate averaged responses plotted in following figures.

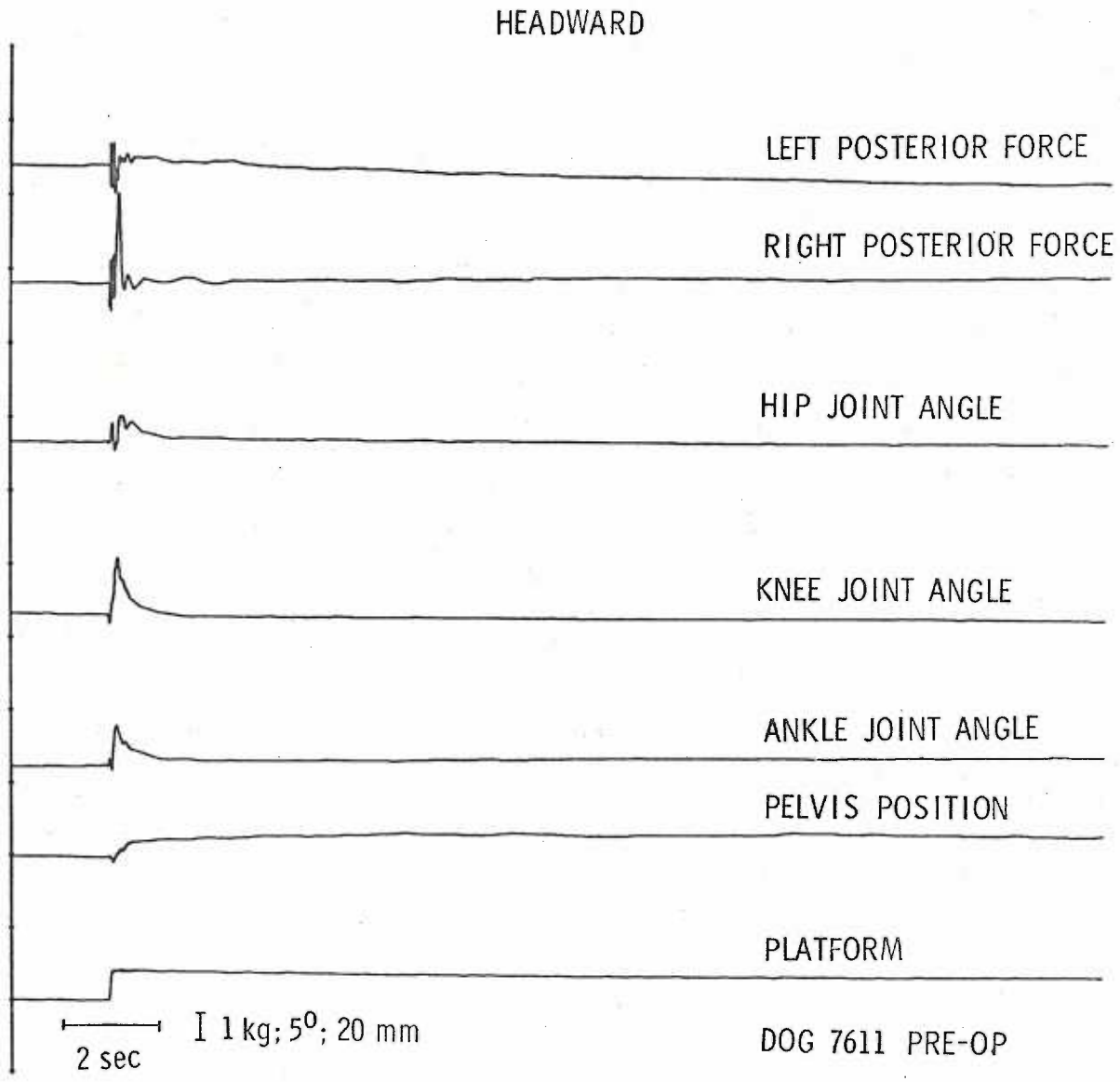
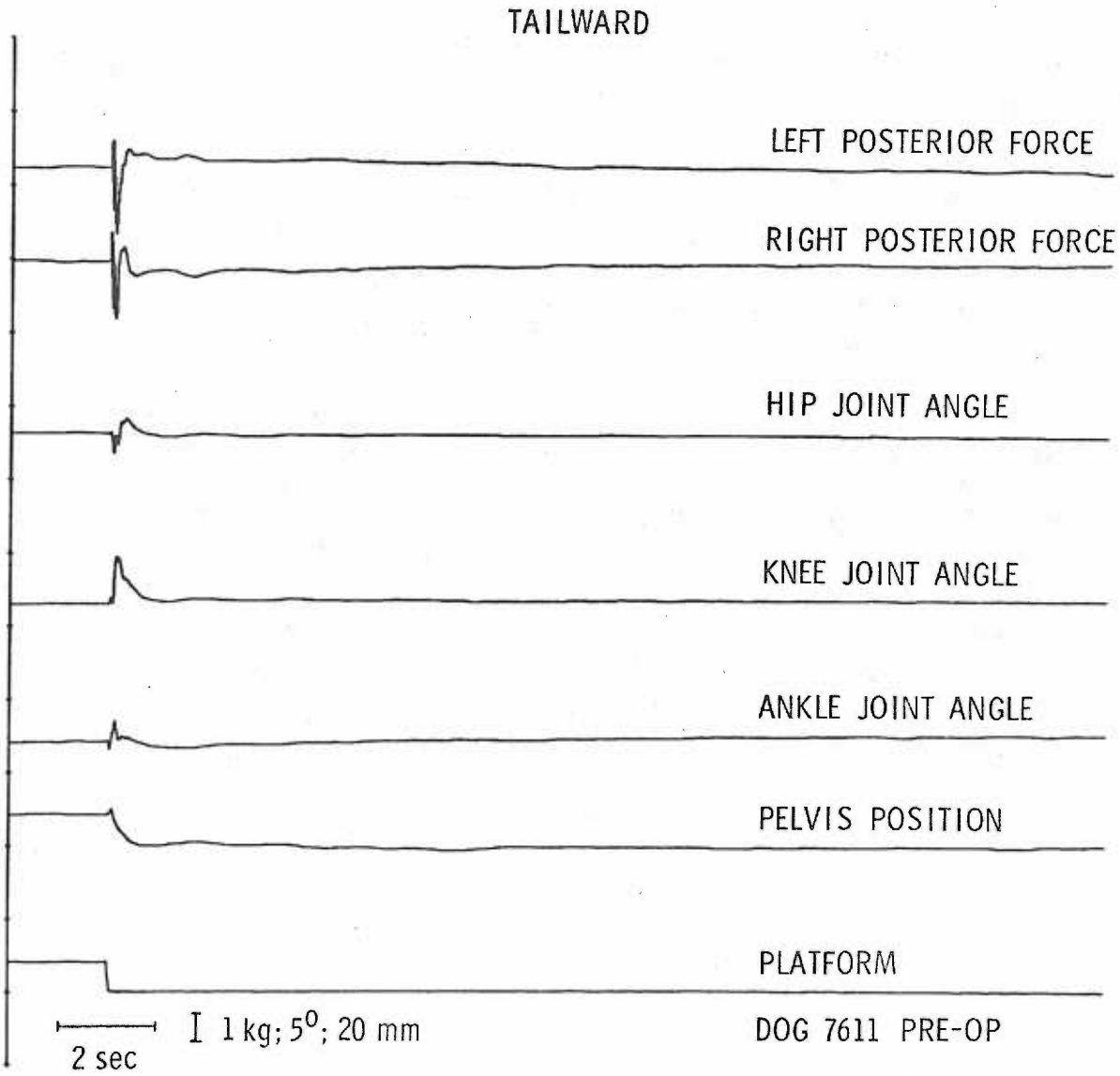

HEADWARD
LEFT POSTERIOR FORCE
RIGHT POSTERIOR FORCE
HIP JOINT ANGLE
KNEE JOINT ANGLE
ANKLE JOINT ANGLE
PELVIS POSITION
PLATFORM
2 sec
1 kg; 5°; 20 mm
DOG 7611 PRE-OP

Figure 12. Records from an individual pre-operative tailward displacement. See Fig. 11 for explanation.


TAILWARD
LEFT POSTERIOR FORCE
RIGHT POSTERIOR FORCE
HIP JOINT ANGLE
KNEE JOINT ANGLE
ANKLE JOINT ANGLE
PELVIS POSITION
PLATFORM
I 1 kg; 5°; 20 mm
2 sec
DOG 7611 PRE-OP

symmetric, as in the quiet standing experiments, so that the displacement responses represent the behavior of the dog postural control system to a disturbance occurring during steady-state behavior. Since points in time past the beginning of each trial are marked by the data sampling pulse train, the data from individual responses are thus available for digital processing to derive statistics from large numbers of responses.

Gross observation of behaving animals during platform displacement has given the impression that the disturbance was well-tolerated in that it evoked no sort of distraction of attention and presented no serious obstacle to completion of the task for reward. Front foot force oscillations, as much as 4 or 5 kg in amplitude, were symmetrical and did not usually cause a reset. After an initial period of becoming accustomed to the abrupt displacement, each animal reached a state where he could respond to the displacement in a smooth, coordinated manner. The nature of the response was similar across dogs, as will be seen.

The averaged angle and position responses to the 25 headward and 25 tailward displacements of the five pre-operative experiments on the same dog as in the previous figures are shown in Figs. 13 and 14. The magnitude of the standard deviation about the mean for each response is plotted immediately below. Comparison of individual with averaged responses reveals the preservation of distinctive features of

Figure 13. Averaged responses to 25 pre-operative headward displacements from one animal. Mean values (and their standard deviations) of the data at each sampling point from the appropriate set of trials were calculated and then plotted by the Calcomp plotter. Such plots were generated and inspected for each animal and from them the method of deriving characterizing parameters was developed. The beginning points of each trace (angle, position, and their accompanying standard deviations plotted immediately below) were digitally adjusted to zero, as described in the methods section.

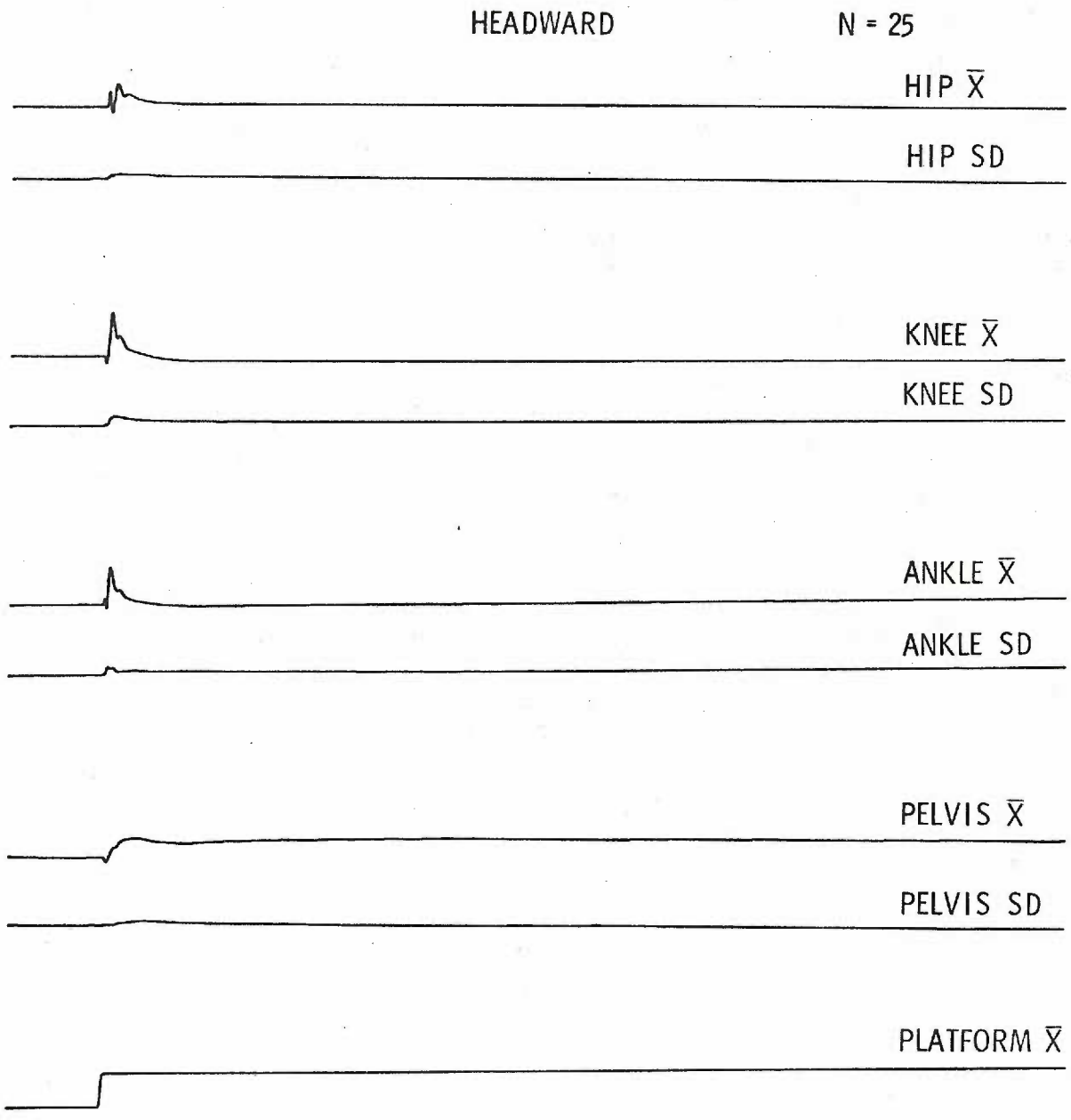
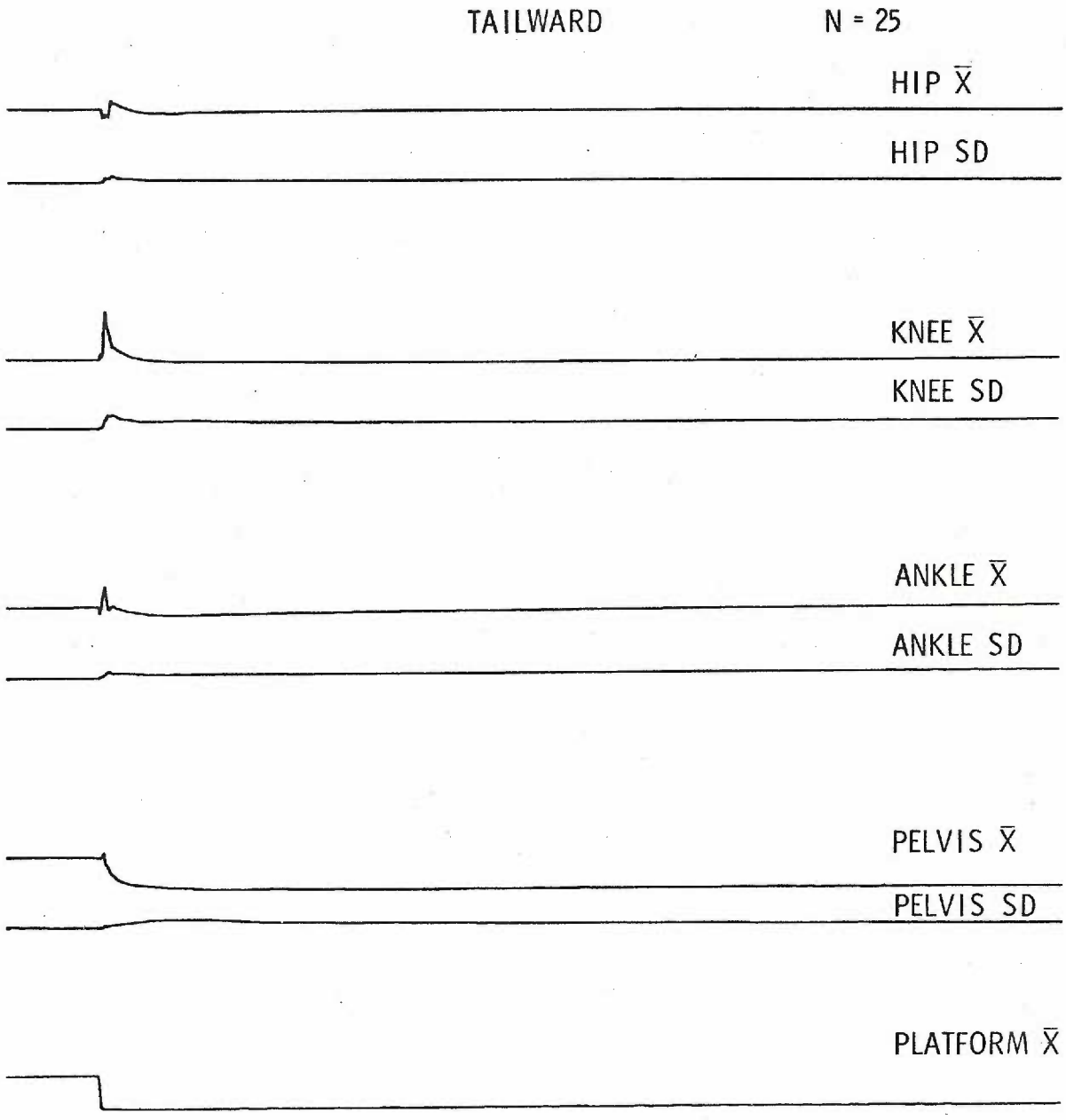

HEADWARD
N = 25
HIP X̄
HIP SD
KNEE X̄
KNEE SD
ANKLE X̄
ANKLE SD
PELVIS X̄
PELVIS SD
PLATFORM X̄
I 5°; 20 mm
2 sec
DOG 7611
PRE-OP

Figure 14. Averaged responses to 25 pre-operative tailward displacements from one animal. See Fig. 13 for explanation.


TAILWARD
N = 25
HIP $\bar{X}$
HIP SD
KNEE $\bar{X}$
KNEE SD
ANKLE $\bar{X}$
ANKLE SD
PELVIS $\bar{X}$
PELVIS SD
PLATFORM $\bar{X}$
I 5°; 20 mm
2 sec
DOG 7611
PRE-OP

the individual responses in the average: the initial deflection is in the same direction in averaged responses, and as can be seen from the standard deviation plotted below, it has little variability in magnitude. This initial component of all the responses is direction sensitive, since it reverses with opposite direction platform displacement, and it is also very constant in magnitude from dog to dog. The later components of angle and position responses are also seen to be repeatable from trial to trial, but major distinctions between angle and position responses are evident. The position responses are appropriately direction sensitive, since the animal follows the platform after it moves. The position change occurs over a much longer time course than any angle changes, and there is no sharp peak in the variability, the maximum usually being reached about 1.5 to 2 sec after the onset of displacement. The angle changes, however, are seen not to be direction sensitive in their major components, with the exception of the hip. Upward deflection represents closure (flexion) at each joint, and simultaneous upward deflections of records from all joints thus indicates a slight collapse or lowering of the hindquarters. And the visual impression of the response of the dog to two directions of displacement is that of a slight collapse and immediate recovery. The variability in angle response usually has a recognizable peak and this occurs at the same time as the major peak of the mean response. The angle changes are all small, typically on the order of 5 degrees. The

maximum individual response was a 21.1 degree knee joint response to tailward displacement, not from the dog illustrated here.

Records from the force transducers were averaged and plotted, but are recognized to contain response components of both the animal and the transducer. They were kept for comparison with previous studies in which electromyograms of hindlimb muscles were recorded and will be analyzed further when a technique of separating transducer response from animal response is developed. They are not reported or discussed here.

The platform position signals were averaged. Variability in platform position was near to zero before, during, and after displacement and its magnitude (representing about 0.1 mm) was the same as zero-signal data system variability for that channel.

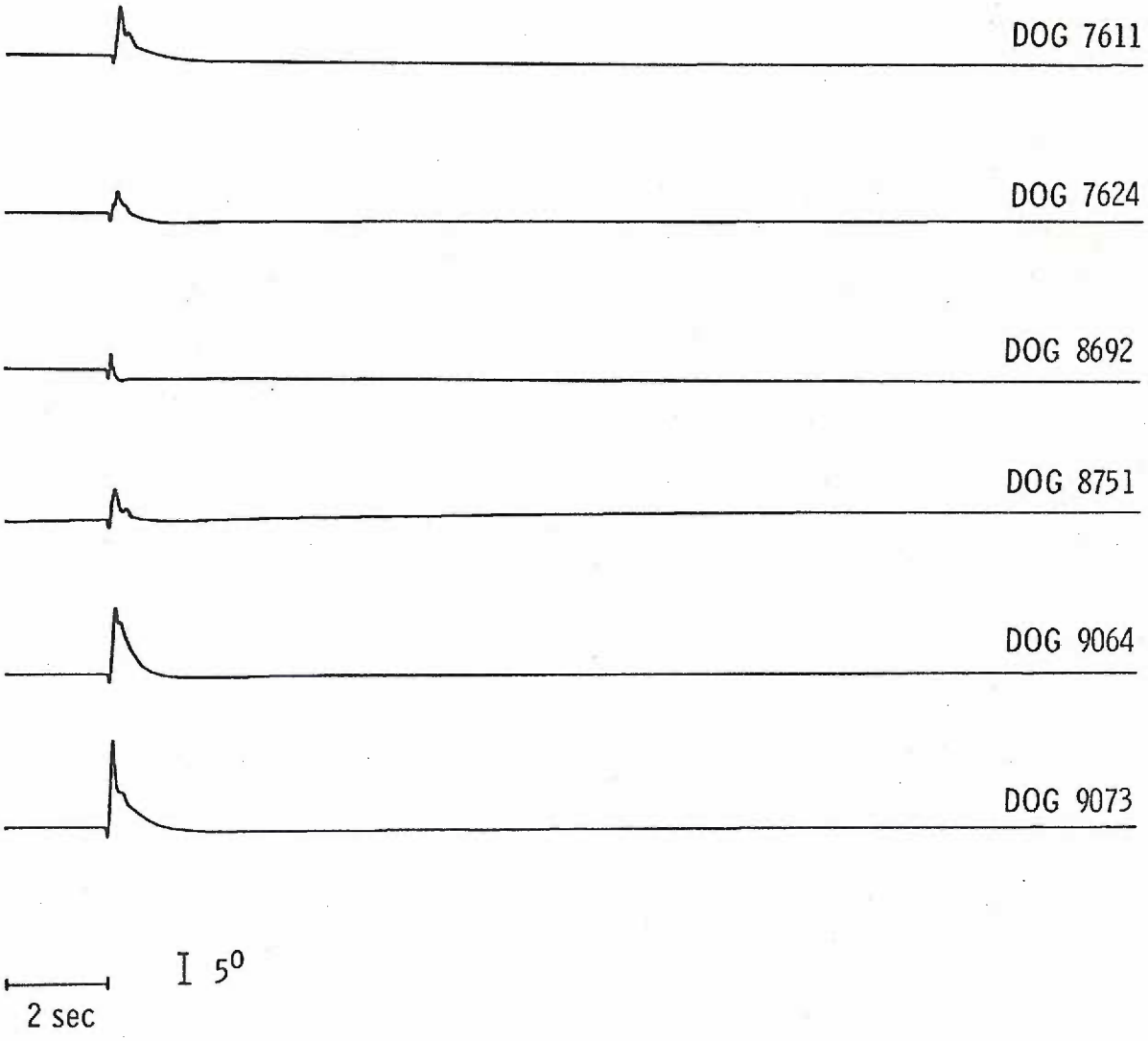
A sample comparison, across dogs, of response to displacement is shown in Fig. 15 where the averaged knee joint angle response to headward displacement from the pre-operative series for each dog is displayed. This figure is intended to convey the impression that the nature of the response to displacement is similar from dog to dog. The initial deflections are nearly identical across dogs, the direction of the major component is the same, the time of occurrence of the peak varies little from dog to dog; it appears that the problem of recovering from the postural disturbance is handled in very much the same way in all of the dogs in this study. This is not intended to

Figure 15. Averaged knee joint responses to 25 pre-operative headward displacements from six animals. Responses as plotted in Fig. 13 were derived for all six animals. The plotted knee joint responses are assembled here for purposes of providing an example of across-dog comparison.

AVERAGED KNEE JOINT RESPONSES

HEADWARD DISPLACEMENT

PRE-OP



disclaim any sort of individual characteristic in response pattern between dogs. The patterns of response in Fig. 15 are individually distinguishable, but it is possible to subject any and all of them to measurement of the parameters described in Methods to characterize them quantitatively.

Qualitative Aspects of Post-Operative Dog Response

During the immediate post-operative recovery period it was usually not possible to perform experiments at the same limit settings used during the pre-operative series of displacement experiments. The displacement appeared to be a very disturbing event to the animals and it was not always possible to complete a full ten-trial experiment without the animal collapsing. None of the experiments done during the rapidly changing phase of recovery are reported here.

One of the signs of stabilization of recovery was an increase in the repeatability of the response to displacement. The post-operative series of displacement experiments was done in the period of 8 to 12 weeks after surgery, and at this time the animals were capable of responding to displacement without distraction and were successful in attaining reward with the same reset limit used in the pre-operative series. There was at this time, to the experienced observer's eye, a gross characteristic of the response that distinguished it from normal. Each animal responded to displacement in a slightly stiffer,

brisker, less coordinated manner than he did in pre-operative experiments. This slight difference was in no way a compromise of capability to recover from disturbance; all dogs were successful in achieving task performance in the face of platform displacement, and a casual observer might interpret the response as normal dog behavior. The gross characteristic seemed to be reflected in some quantitative changes described below.

Samples of individual post-operative displacement responses from the same animal as illustrated above are seen in Figs. 16 and 17. And the averaged responses from the five-experiment post-operative series on this dog are shown in Figs. 18 and 19. A quick appraisal of the individual post-operative responses as compared to the pre-operative ones of Figs. 11 and 12 might lead to the impression that post-operative responses were aberrant and likely to show much increased variability. The initial deflections in each response are seen to be virtually identical from pre-operative to post-operative condition, but the later components of both angle and position responses are considerably different in shape and time course. If the averaged responses of Fig. 18 and 19 are compared with the individual post-operative responses, however, it can be seen that the post-operative responses did have characteristic and highly replicable features; there was not a marked increase in the standard deviation about the mean as compared to pre-operative responses.

Figure 16. Records from an individual post-operative headward displacement. See Fig. 11 for explanation.

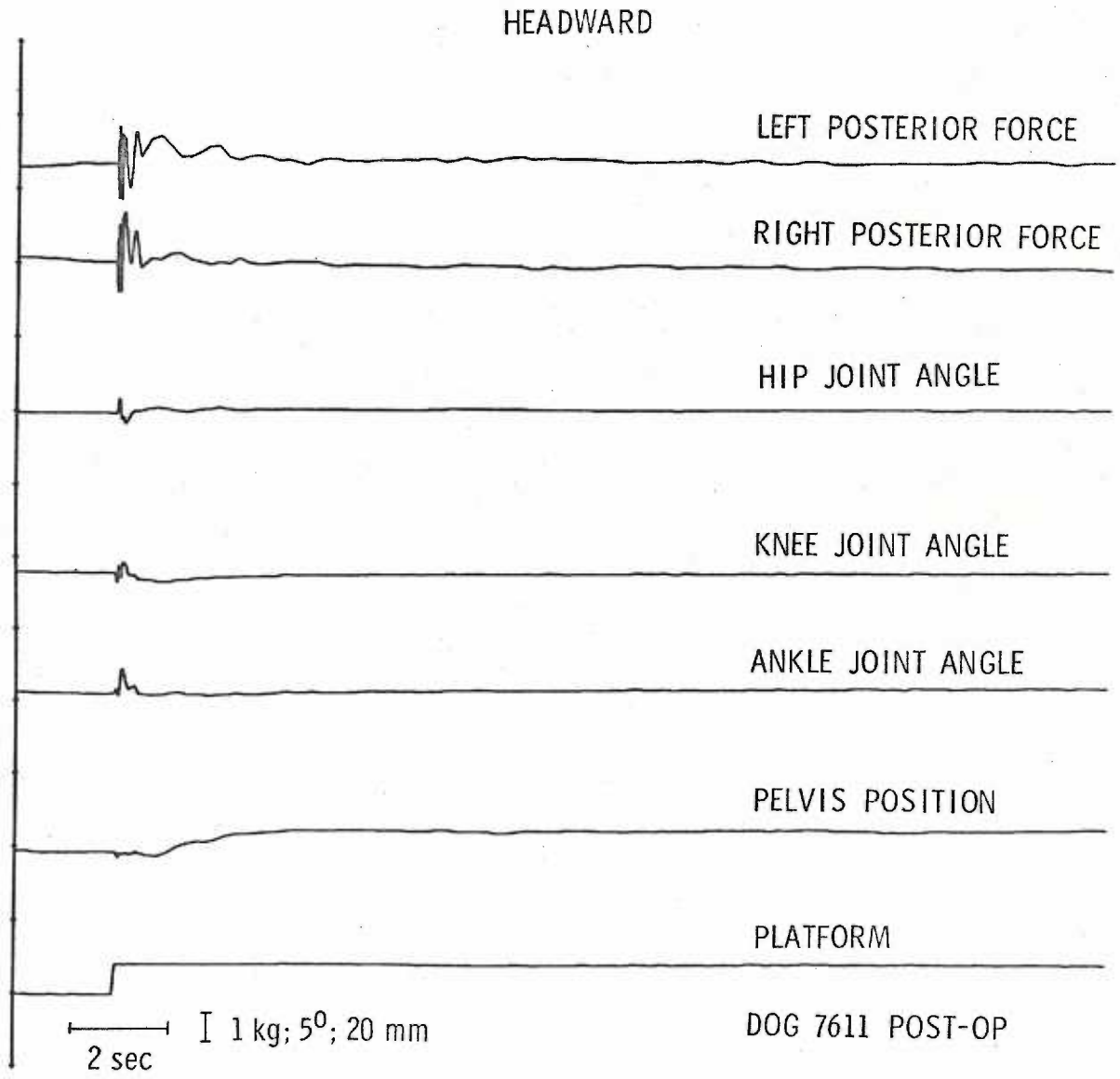

HEADWARD
LEFT POSTERIOR FORCE
RIGHT POSTERIOR FORCE
HIP JOINT ANGLE
KNEE JOINT ANGLE
ANKLE JOINT ANGLE
PELVIS POSITION
PLATFORM
I 1 kg; 5°; 20 mm
2 sec
DOG 7611 POST-OP

Figure 17. Records from an individual post-operative tailward displacement. See Fig. 11 for explanation.

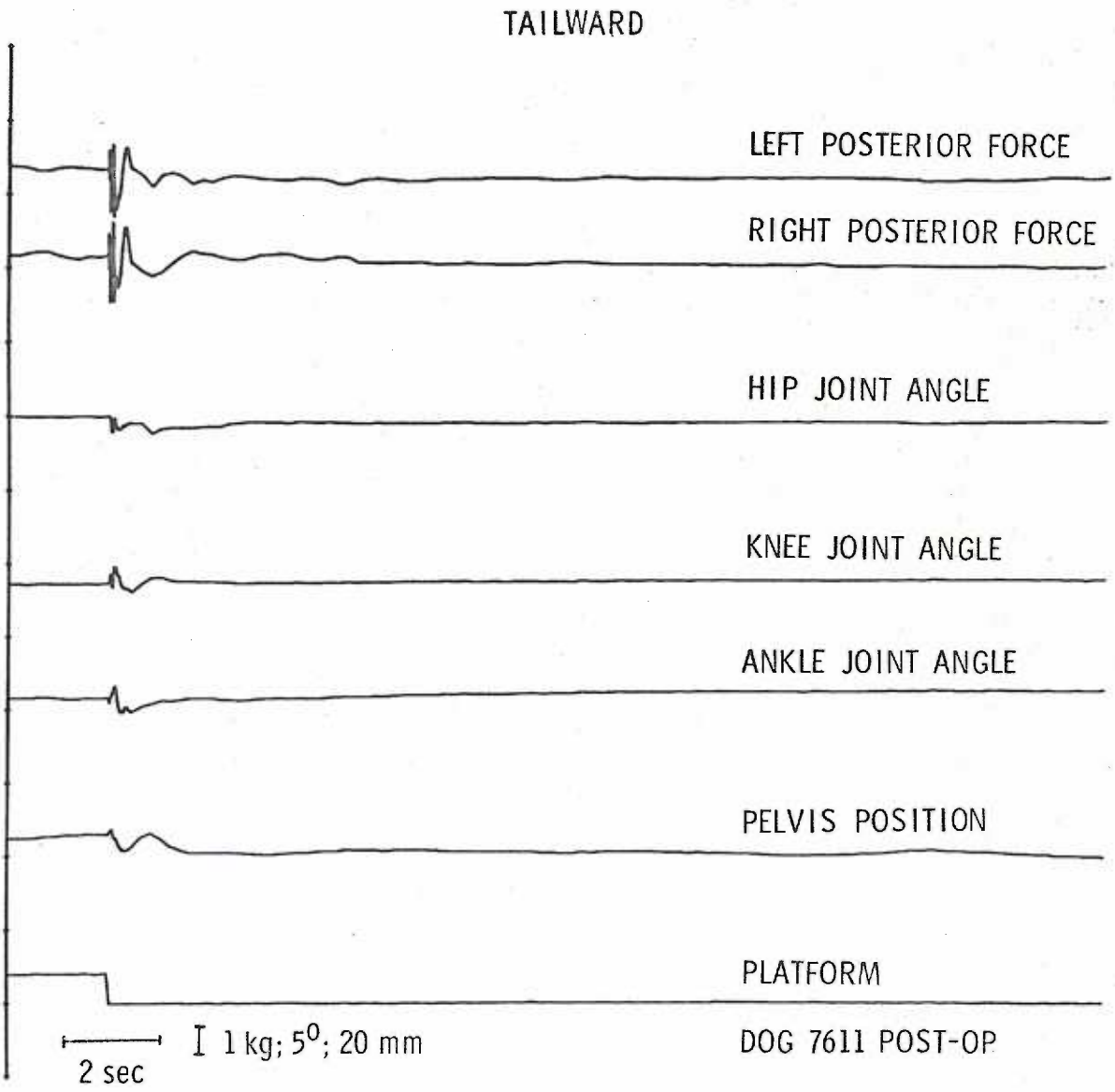

TAILWARD
LEFT POSTERIOR FORCE
RIGHT POSTERIOR FORCE
HIP JOINT ANGLE
KNEE JOINT ANGLE
ANKLE JOINT ANGLE
PELVIS POSITION
PLATFORM
2 sec
1 kg; 5°; 20 mm
DOG 7611 POST-OP

Figure 18. Averaged responses to 25 post-operative headward displacements from one animal. See Fig. 13 for explanation.

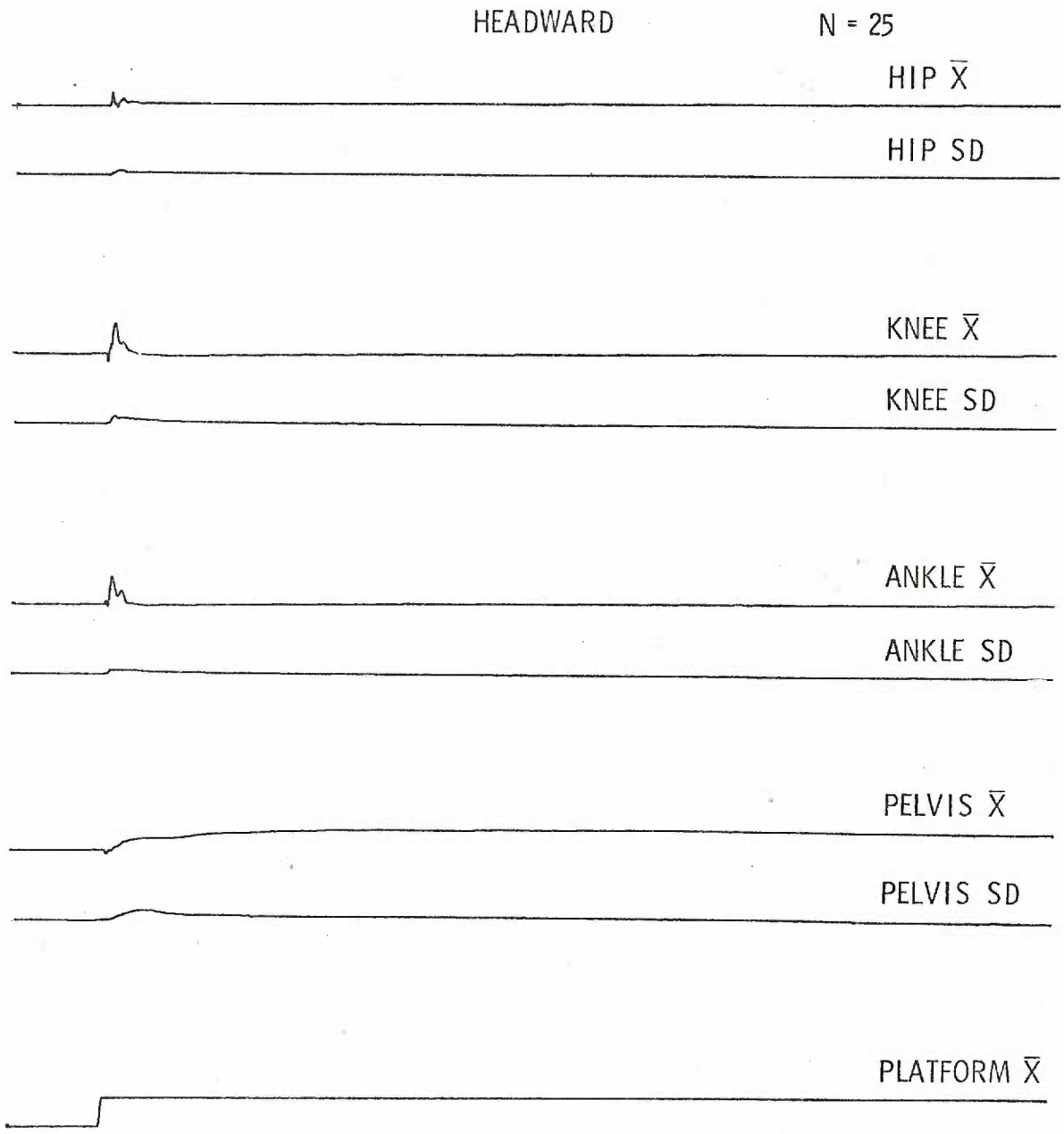
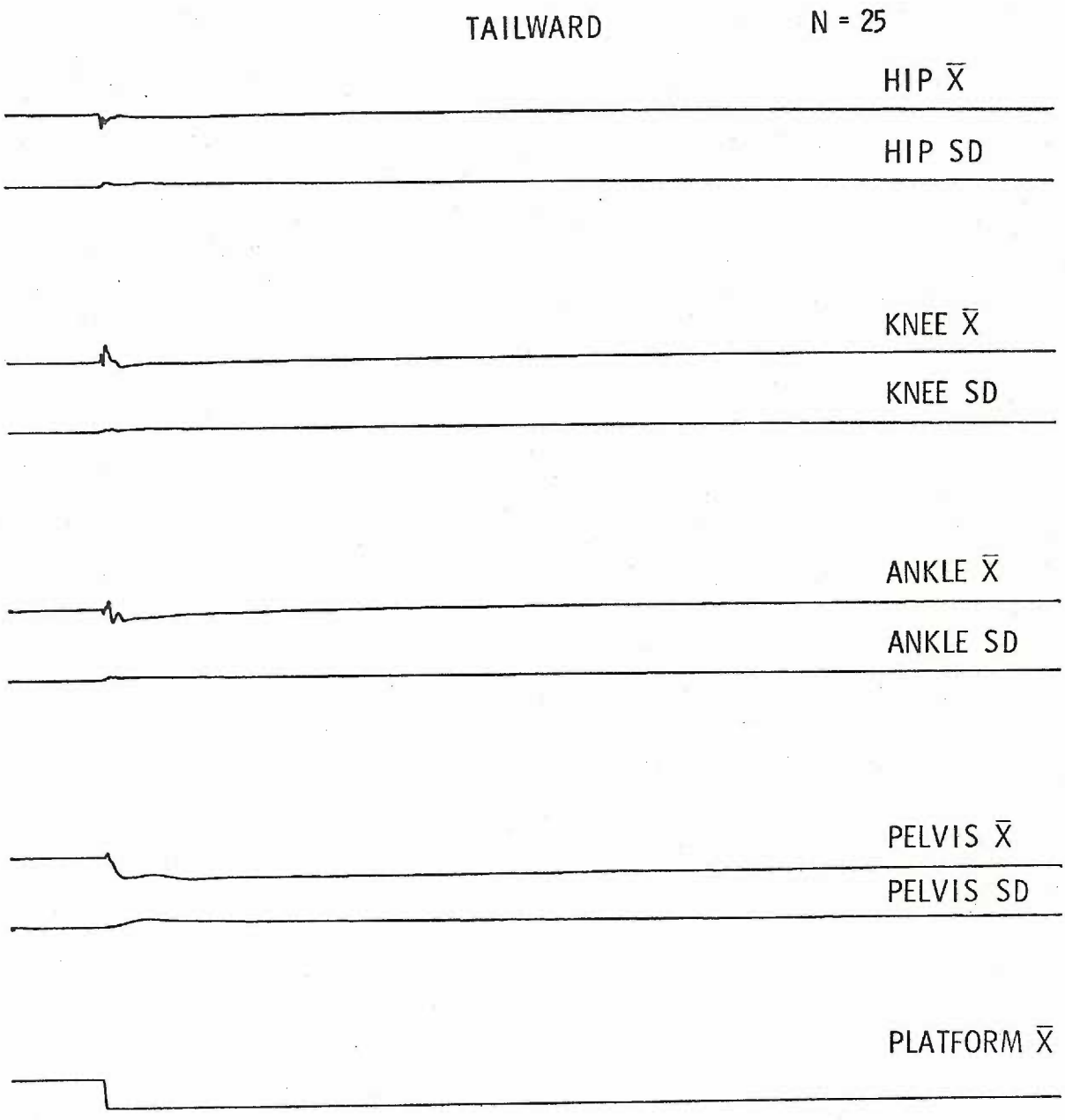

HEADWARD
N = 25
HIP $\bar{X}$
HIP SD
KNEE $\bar{X}$
KNEE SD
ANKLE $\bar{X}$
ANKLE SD
PELVIS $\bar{X}$
PELVIS SD
PLATFORM $\bar{X}$
I 5°; 20 mm
2 sec
DOG 7611
POST-OP

Figure 19. Averaged responses to 25 post-operative tailward displacements. See Fig. 13 for explanation.


TAILWARD
N = 25
HIP X̄
HIP SD
KNEE X̄
KNEE SD
ANKLE X̄
ANKLE SD
PELVIS X̄
PELVIS SD
PLATFORM X̄
I 5°; 20 mm
2 sec
DOG 7611
POST-OP

Quantitative Results

For the purpose of providing a typical example to accompany the following discussion, the averaged ankle joint angle responses to headward displacement from Fig. 13 (pre-operative) and Fig. 18 (post-operative) are juxtaposed in Fig. 20. The reader should refer to Fig. 7 for definitions of the waveform parameters discussed below.

Angle and position responses were averaged and then plotted (as in Fig. 20) for all dogs for both directions of displacement for both pre- and post-operative sets of experiments. These plotted waveforms were all inspected for any gross errors in digital processing, and errors were corrected when any appeared. Then the individual responses were subjected to measurement of the values from which the parameters were derived. These time and amplitude values are listed in section A of the Supplement under separate cover. They include measurements on all responses to both headward and tailward displacement from both pre- and post-operative experiments for all dogs.

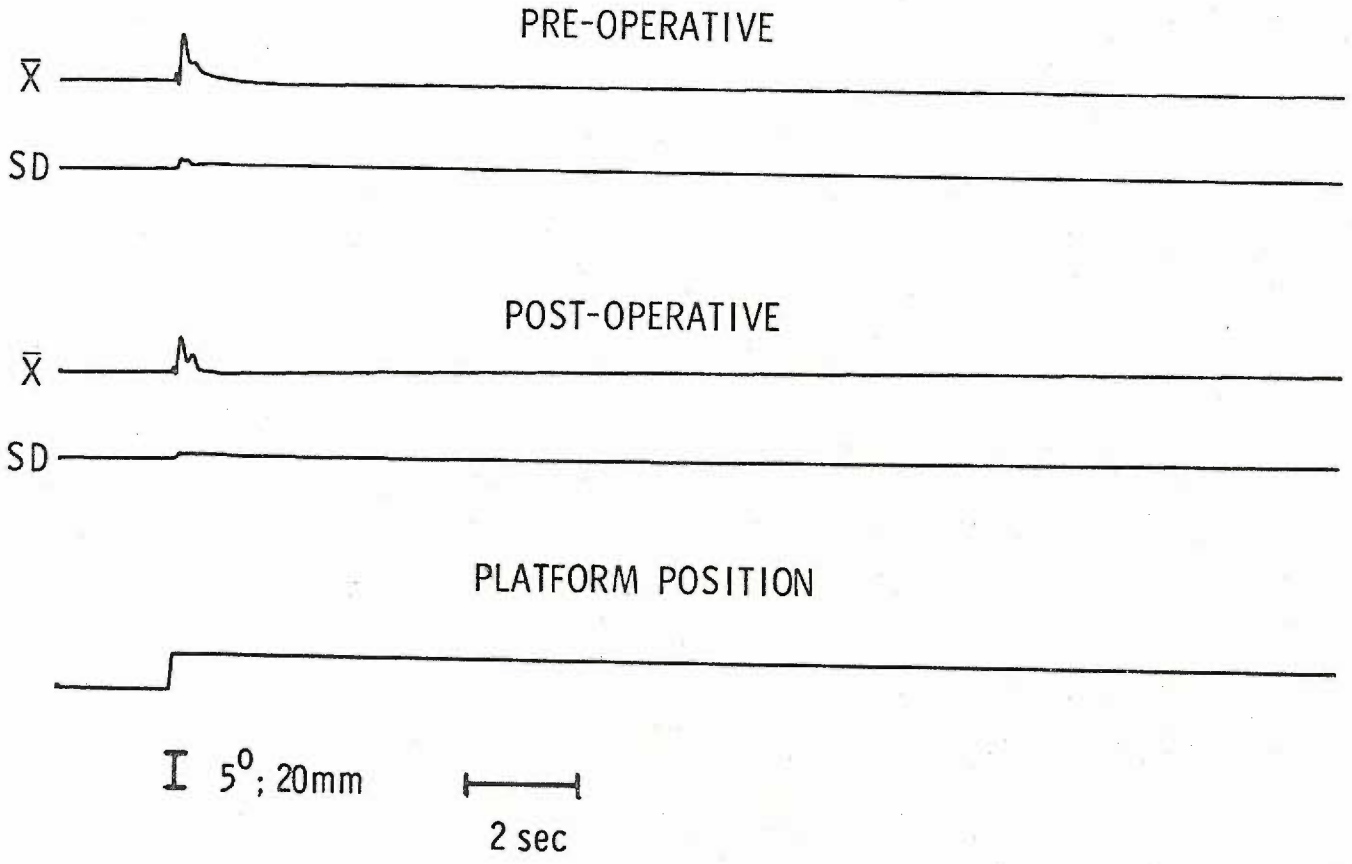
The characterizing waveform parameters were derived from the data measurements and subjected to several kinds of statistical tests. In an initial attempt to simply show up gross pre-operative-post-operative differences, means and standard deviations were derived from the parameters in each response category. These statistics are listed in section B of the Supplement. Bar graphs not shown here were

Figure 20. Averaged pre-operative and post-operative ankle joint angle responses to headward displacement from one animal. The responses plotted here are the same as those in Figs. 13 and 18 and are juxtaposed here to facilitate visual comparison.

HEADWARD DISPLACEMENT

ANKLE JOINT ANGLE RESPONSE

DOG 7611 N = 25

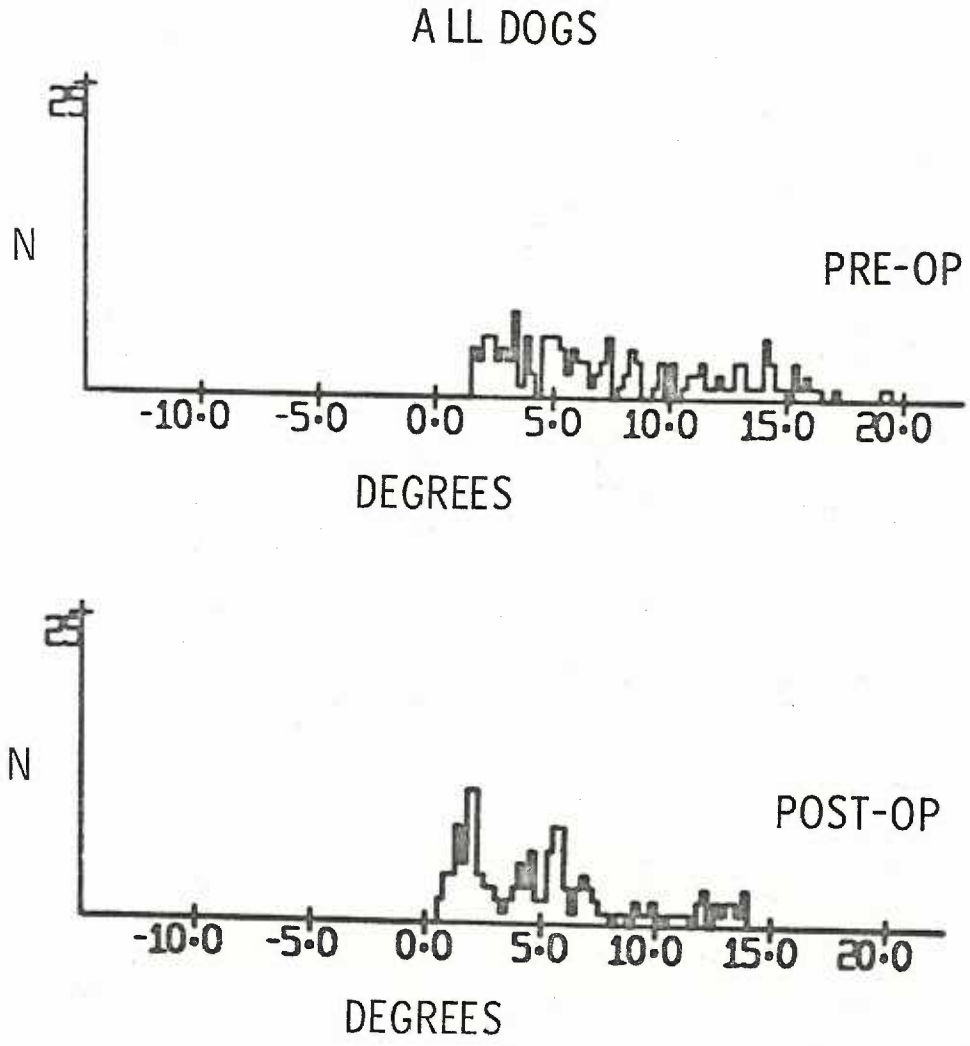


constructed from the listed means, and it immediately became apparent that there was not a consistent trend across dogs for a change in a given direction of parameter means or for a clear increase in variability following the spinal cord lesion. Parameter means changed in opposite directions across dogs or did not change at all. The patterns of variability across dogs and changes in variability within dogs following the lesion suggested that a close look at the distributions of parameter values was in order.

The sets of parameters were thus subjected to derivation of median, range, and interquartile range, and distribution histograms were generated. The listed values pertaining to distribution are located in Supplement section C for individual dogs and for all dogs combined. As the histograms were generated, it became apparent that normal statistics would be entirely unsatisfactory for testing many of the parameters. Some parameters, such as the TIME AT PEAK for angle changes, fell into tight normal distributions for individual dogs and for the whole population. But many others, such as RESPONSE AMPLITUDE from knee joints, shown in Fig. 21, are from very broadly spread distributions. The data of Fig. 21 illustrate one of the few phenomena that was consistently observed across dogs, and that is a tendency toward decreased amplitude of response in postoperative dogs. The distribution of knee joint RESPONSE AMPLITUDE to headward displacement shifted to the left postoperatively,

Figure 21. Histograms of pre-operative and post-operative knee joint angle RESPONSE AMPLITUDE to headward displacement from six animals. Such histograms, for individual dogs and for all dogs combined, were generated for these and other angle and position responses parameters. The disclosure of many such non-normally distributed populations of parameters provided the reason for seeking a distribution-free test to disclose pre-op-post-op differences. Displays were made on a Tektronix T4005 Graphic Display Controller and visually inspected. Only a few such histograms were photographed. Distribution statistics were simultaneously printed out and are listed in Supplement section C.

KNEE JOINT RESPONSE AMPLITUDE DISTRIBUTION



and the median value decreased from 6.45° to 4.69°. This observation was accompanied by observations of similar shifts of RESPONSE AMPLITUDE from hip and ankle joint responses to both directions of displacement for all dogs. The statistical significance of this phenomenon is discussed below. The listed medians and ranges can be scanned to reveal many cases where a given parameter was changed a great deal in one direction following the lesion in one dog and the opposite direction in another dog, and there are also many parameters showing little or no change following the lesion. Observation of these haphazard and disorganized sorts of changes made it apparent that a test lumping data from all dogs would not be satisfactory and that a test assuming normality of distribution would also not be satisfactory for disclosing significant effects of the lesion on dog behavior.

Turning to the question of a possible increase in variability in post-operative responses, the maximum variability usually occurred at or near the response peak, so a coefficient of variability at this point was calculated for all dogs. The coefficient is the ratio of standard deviation to RESPONSE AMPLITUDE in percent taken from the list of parameter means and standard deviations. The coefficients are tabulated in Table IV. It can be seen that if means or ranges of coefficient values are used, there is no evidence for an increase in variability, and there is even a case (hip, headward) where the variability shows a decrease postoperatively. If, however, the individual

Table IV. Coefficients of variability of angle responses at peak.

Dog	Headward		Tailward	
	Pre-op	Post-op	Pre-op	Post-op
		A. Hip angle		
7611	18.7	15.2	9.8	9.0
7624	26.6	13.4	25.5	12.0
8692	16.1	19.8	15.7	22.9
8751	20.2	17.3	14.7	20.7
9064	32.3	25.9	14.3	10.1
9073	39.3	17.6	11.6	20.1
Mean	25.5	18.2	15.3	15.8
Range	16.1-39.3	13.4-25.9	9.8-25.5	9.0-22.9
		B. Knee angle		
7611	20.7	27.0	18.5	12.2
7624	35.4	42.3	26.1	15.1
8692	47.4	31.1	20.1	29.8
8751	23.9	30.3	32.2	30.3
9064	24.2	51.3	25.0	24.8
9073	16.6	14.0	17.6	26.5
Mean	28.0	32.7	23.3	23.1
Range	16.6-47.4	14.0-51.3	17.6-32.2	12.2-30.3
		C. Ankle angle		
7611	23.4	17.9	17.3	41.0
7624	18.9	15.6	55.7	51.7
8692	29.2	16.4	95.6	33.2
8751	25.4	26.0	32.4	76.9
9064	35.4	40.1	42.6	32.8
9073	14.9	21.1	20.4	33.7
Mean	24.5	22.9	44.0	44.9
Range	14.9-35.4	15.6-40.1	17.3-95.6	32.8-76.9

dog values are examined, it is evident that some large changes in variability occurred, and in opposite directions from dog to dog within conditions. Within dogs, variability changes can be opposite in direction from angle to angle or from headward to tailward direction of displacement. In brief, there is no evidence for a consistent effect of the lesion on the variability of amplitude of animal response to displacement. The same conclusion results from examining variability of other aspects of the responses.

A final statistical test was used; this one selected because of the nature of the parameter distributions. It was the Wilcoxon rank-sum test taken from Bradley (14), and is a distribution-free test. The test is designed to compare populations, not necessarily matched or paired, of observations, and its null hypothesis is that there is no difference between the populations. The inference on rejection of the null hypothesis must be tailored by the user since the test does not specify a parameter of the tested distributions that differs.

Individual dog data were tested in attempt to disclose an effect of the lesion on any of the response waveform parameters for both headward and tailward displacement. The calculated sums of ranks used to enter the table of critical values are listed in section D of the accompanying Supplement. The test results are summarized in Table V, in three parts, where significant differences, using an 0.05 cutoff, are indicated with a symbol to distinguish them from unchanged values.

Table V. Results of the Wilcoxon rank-sum tests on individual dog displacement responses to detect an effect of the lesion. The significance cutoff level was chosen as .05. The symbols entered in the table indicate significance of change, if any, as well as direction. The symbol 0 indicates no change; the symbol + means a significant increase in the value following the lesion; the symbol - means a significant decrease in the value following the lesion. Dog identification: A, dog 7611; B, dog 7624; C, dog 8692; D, dog 8751; E, dog 9064; F, dog 9073.

Table V. Results of Wilcoxon rank-sum test on displacement response parameters.

	Hip						Knee						Ankle					
	A	B	C	D	E	F	A	B	C	D	E	F	A	B	C	D	E	F
A. Headward displacement, angle parameters																		
Early error	0	-	-	-	-	0	+	+	-	0	+	0	+	+	+	0	+	0
Late error	+	+	0	0	+	+	+	0	-	-	0	+	+	0	-	-	+	+
Drift	0	-	-	-	-	0	+	+	-	0	+	0	+	+	+	0	+	0
Response amplitude	-	0	0	+	-	0	-	-	-	0	-	-	-	0	-	0	-	0
Overshoot	-	0	-	0	0	-	-	0	0	-	-	-	-	0	0	+	-	-
Time at overshoot	-	+	-	+	-	-	-	-	0	0	0	-	-	0	0	+	-	-
Time at peak	-	-	-	-	-	-	+	0	-	-	0	-	0	0	0	0	0	0
Rise time	-	-	-	0	-	-	+	+	-	+	0	+	+	0	0	-	-	-
Fall time	0	0	-	0	-	-	-	0	0	0	-	-	-	0	0	+	-	0
B. Tailward displacement, angle parameters																		
Early error	0	-	-	-	0	0	0	+	0	-	-	-	+	+	0	0	0	-
Late error	+	+	0	0	+	0	+	0	-	-	0	+	0	0	-	-	0	+
Drift	0	-	-	-	0	0	0	+	0	-	-	-	+	+	0	0	0	-
Response amplitude	+	-	0	0	+	+	-	-	-	-	-	-	-	-	-	0	-	+
Overshoot	-	-	-	0	-	0	-	0	0	-	-	-	-	0	0	+	-	0
Time at overshoot	+	+	0	0	0	0	-	0	-	-	-	-	-	0	0	+	-	+
Time at peak	-	+	0	+	-	0	-	-	-	0	+	+	0	-	0	0	+	0
Rise time	-	+	0	+	-	0	0	0	0	0	-	-	+	-	0	0	+	0
Fall time	0	0	0	0	+	+	-	+	0	0	0	-	-	0	0	0	-	+

C. Headward and tailward displacement, position parameters

	Headward						Tailward					
	A	B	C	D	E	F	A	B	C	D	E	F
Early response amplitude	0	0	-	+	0	0	+	0	0	0	0	+
Late response amplitude	0	-	0	-	-	0	0	-	-	-	-	0
Drift	0	0	-	+	0	0	+	0	0	0	0	+
Rise time	0	0	0	-	0	0	0	+	0	0	0	0
Overshoot	+	0	0	0	0	0	0	0	-	0	0	-
Time at overshoot	0	-	-	-	0	0	0	+	0	0	0	0

Since ranks were assigned simply on the basis of magnitudes of parameters, the inference on rejection of the null hypothesis is only that populations differ in magnitude. This inference should reasonably be expected to be reflected in some measure of central tendency, and as a matter of fact, the results of this test agree strongly with the magnitudes and directions of changes seen in the listed means of parameters. The results in Table V show a confusing array of significant changes, opposite in direction from dog to dog, accompanied by many cases of no significant change. It should be mentioned that many of the significant values were well below the 0.001 cutoff value.

An apparently more appropriate statistical test, then, fails to yield any additional information. Differences in characteristics of individual response patterns can be reconstructed by laboriously selecting out the differences indicated in Table V, but they invariably correspond to the visually discernible differences seen in the plotted mean response patterns as in Fig. 20. One response parameter that appeared to show a consistent trend across dogs was that of joint angle RESPONSE AMPLITUDE. If the directions of the non-significant changes are entered in the place of zeros in Table V, and it is recalled that the tailward hip joint angle response was an inverted waveform, then it is seen that the amplitudes of angle responses to both headward and tailward displacement were decreased following the lesion. The significant changes were usually small, and there are three instances

of significant differences of opposite sign. The clinical impression of a briskness or stiffness in the response of the recovered dogs correlates well with the observation of diminished response amplitudes.

Summary

Some dynamic characteristics of the behavior of the dog postural control system have been observed using the technique of imposing a postural disturbance to animals during the steady-state condition of maintaining an undisturbed quiet stance. And the possibility that the afferents traveling in the spinal cord dorsal columns constitute a feedback loop of importance to the operation of the postural control system has been tested by observing the dynamic response after transection of the dorsal columns. Distinct phases of transient impairment of behavior and of recovered behavior followed the spinal cord lesion. In the earliest portions of the transient impairment of behavior, the animals were unable to stand and then unable to respond to disturbance without collapsing. Over a two to three week time course they regained ability to respond to displacement during quiet stance but did not behave in a replicable manner. The phase of recovered behavior followed, during which they responded to displacement in replicable fashion. Quantitative aspects of the dynamic responses were seen to differ in the recovered post-operative dog from the normal dog. The differences were slight, subtle, and in no way indicative of a loss of

capability to respond to disturbance. The gross nature of the recorded responses did not make it possible to estimate in any way what functional loss might account for the differences seen in response patterns. The changes were extremely difficult to define, in that no clear pattern or trend was apparent; highly significant differences in response characteristics were seen to occur with opposite sign from dog to dog and accompanied by instances of zero change in some dogs. No clear pattern of large changes or small changes within dogs was seen. The clinical signs following the lesion were consistent across dogs, with a possible gradation in "severity" of the signs. No quantitative distinctions were apparently correlated with this gradation. It eventually became apparent that some subtle, long-term effects of dorsal column transection on postural control system behavior did exist, but were not well described by the method used.

DISCUSSION

Some Considerations of the Experimental Method

A factor that was expected, at the outset, to have a strong bearing on the "level of performance" aspect of the quiet standing experiment was that of total weight of the animals. It would be expected that a heavier animal would be presented with a task of a greater degree of difficulty than a lighter animal would experience when required to constrain front-foot weight difference within a set limit. The basis for this distinction is disclosed in Tables VI and VII. The dogs used in this study ranged in weight from about 20 to 23 kg on their restricted diets, and with 60-40% anterior-posterior weight distributions had total front-foot weights in the range of 12 to 14 kg. The actual lateral distances (either to the left or right of midline) that an animal would have to shift the center of his front-foot weight distribution in order to reset the apparatus at various limit settings is tabulated for the weight range extremes in Table VI. These calculations are based on a distance between feet of 150 mm, with the force plates set in a 150 by 500 mm rectangular array and with identical sensitivities for all dogs regardless of weight. In Table VII the percentage changes in force beneath individual feet at different reset limits are shown. The numbers represent amounts of force change, in percent, from the symmetrically distributed values at which reset

limits would be reached. For example, a 23 kg dog would have about 7 kg of weight on each front foot at lateral symmetry. To reach reset limit at the 1 kg setting, he would have to shift 7.2% of the total weight from one foot to the other, provided the shift was a pure lateral front-foot weight shift and did not involve any anterior-posterior redistribution. The 20 kg dog would need to alter individual foot weights by 8.4% to reach the reset limit. The principle would apply as well if there were anterior-posterior shifts, but the numbers would differ. These calculations seem to indicate about a 17% lesser restriction for the lighter dog. However, this could only be effective if position or weight signalling receptors had nearly infinite resolution so that the difference between 7.2 and 8.4% represented a continuum rather than at best a few integral multiples of just noticeable differences. The results displayed in Table VIII may possibly indicate differences between dogs on the basis of the effect of total weight difference. The lightest dog definitely performed at the highest level, showing the lowest ratio of trials with resets to total trials at the 1 kg limit. However, the results of the study are not affected by this difference between animals. The statistical tests used sets of observations matched across conditions. They were not affected by individual differences within conditions but rather disclosed population differences across conditions.

Table VI. Lateral distances from midline of center of front-foot weight distribution at various reset limits depending on total weight.

Reset Limit	Total Front Foot Weight 12 kg	14 kg
4 kg	25 mm	21.5 mm
3 kg	18.8 mm	16 mm
2 kg	12.5 mm	10.7 mm
1 kg	6.5 mm	5.5 mm

Table VII. Percentage force change beneath one front foot from symmetry at various reset limits depending on total weight.

Reset Limit	Individual Front Foot Weight at Symmetry 6 kg	7 kg
4 kg	33%	28.6%
3 kg	25%	21.2%
2 kg	16.7%	14.3%
1 kg	8.4%	7.2%

Table VIII. Quiet standing performance ratios tabulated according to total weight.

Dog	Weight (kg)	Pre-Op Ratio at 1 kg Limit	Post-Op Ratio at 1 kg Limit
7611	23.0	.578	.756
8751	22.6	.509	.631
9064	22.6	.406	.661
7624	21.8	.714	.824
9073	20.9	.550	.509
8692	20.4	.327	.426

(i.e., 2, 3, or 4 kg) provide little challenge to most dogs, whereas the 1 kg limit provides a task of some degree of difficulty. The 24 second observation period was arbitrarily chosen, and the results might have been quite different if the duration of performance to reward was different. For example, the two dogs tested at 0.5 kg limits could have obtained many rewards with a 5 second trial duration. Trials of longer duration than 24 seconds might not have been completed by any of these animals at 1 kg limits. At this point it can only be assumed that a rough magnitude estimate of the limits of steady-state behavior of the dog postural control system has been made on the basis of the 24 second observation period. The "limit of capability" of a dog to maintain a quiet stance must be recognized to represent a range of the variability of the output of a dynamic system. Further insight into steady-state postural control system behavior will probably be gained by studying the steady-state frequency response during sinusoidal forcing.

A distinct weakness of the present experimental method for use in comparing displacement responses to those of previous studies can be seen in the records of pelvis position response. None of the animals actually followed the platform by moving 20 mm when the platform moved 20 mm. It is suggested that the attenuated mean position responses (sometimes as little as 9 mm) resulted from the fixation in space of the light panel and feeding dish; the animals may have

tried to maintain a fixed position of nose relative to dish visually. This problem might be alleviated by coupling the apparatus to the platform. The effect of this "position fixing" behavior on the joint angle responses to displacement will have to be estimated in future studies. There is no reason to believe that this might in any way alter the conclusions of the present study. The gross nature of the displacement response did not appear to the observer's eye to differ in this study from previous studies.

Factors Affecting Early Post-Operative Behavior

Non-Specific Effects of Surgical Trauma

Trauma to the spinal cord in the form of concussion, compression, contusion, or laceration has long been recognized to produce signs of fiber tract transection when, in fact, no transection has occurred (7, 111, 119). The mechanisms underlying these often rapidly reversible signs are not understood, and little or no information is available concerning the time course of such phenomena in experimental animals. Spinal tractotomy, especially for the relief of intractable pain, is a common operation in human patients and neurological examination of cordotomy patients can give an estimate of the extent and time course of such non-specific effects of spinal cord surgery (7, 40, 121). There is apparently considerable variability in such

effects depending on the operative technique and the level of the spinal cord operated. Some estimates of duration and extent of these effects in patients subjected to open anterior cordotomy have been given by Gallo (40). A rise in the initial level of anesthesia, as tested with a pin, is usually observed over the 72 hours following the surgery. This peak level of anesthesia then begins to fall, and usually stabilizes in 48 hours, either at or below the initial level. The changes can be reduced or even abolished by administration of steroid hormones and would thus appear to be due in large part to the inflammatory tissue changes associated with wound healing. Although ischemia due to transection of small arterial branches may affect the total result of the surgery, it probably does not play a role in these reversible changes over the five day time course. The signs of tract interruption may often spread posteriorly in the cord so that a patient shows signs of a hemisection at the end of three days. If the tractotomy is done in the high cervical cord, signs of spinocerebellar tract interruption (ataxic gait) may sometimes be seen. The method of fiber tract transection also has a great influence on the degree of spreading of these reversible changes, a knife cut inducing the greatest spread and RF current delivered via a small penetrating needle inducing the least.

Another factor which may bring about an extension of functional loss is ischemia caused by transection of intraspinal arteries. The

onset of such an effect should be rapid, and any reversible components would certainly have to run their course in a very few hours; irreversible ischemic depression of function would bring about degenerative changes which could be seen accompanying the degeneration due to direct trauma in histological preparations.

A final non-specific factor which might affect the early post-operative signs is that of gross cord compression. The penetration of a blade tip through the pia-arachnoid requires some force and the underlying tissue is certainly subjected to compression. It is probable that the reversible effects of compression of this nature run their course in a very few hours (111), and again the irreversible effects would appear along with the effects of direct trauma as histological degeneration. It must thus be recognized that some of the acute signs of functional loss in a subject following the production of a specific spinal cord lesion are not related to the interruption of specific fiber tracts but rather to a gross non-specific tissue response affecting intact non-traumatized fibers and possibly cell bodies. Such effects may have a gradual onset over 72 hours, but must certainly run a more variable time course in a case complicated by surgical sepsis or hemmorhage.

The Role of Pathway Interruption

Examination of the histologically prepared material has indicated that all but one dog suffered complete transection of essentially all posterior quadrant fibers at about the middle of spinal segment C_4. In the one exception a substantial number of deep posterior quadrant fibers remained intact and showed no signs of degeneration (see Fig. 8). This indication was supported by corresponding patterns of complete posterior quadrant fiber degeneration in spinal segment C_3 except in the lateral margins, where primary afferent fibers arriving over dorsal roots above the level of the lesion contribute to the posterior quadrant population. It appears that all of the transected fibers were projecting rostrally since no degenerated fibers could be seen in the posterior quadrant of the C_5 sections from any animal.

In addition to the posterior quadrant fiber degeneration resulting from surgical transection, there were regions of diffuse, less complete fiber tract degeneration which seemed to arise from indirect factors rather than direct surgical trauma. A consistently observed region of degeneration of descending fibers was seen in the right lateral cortico-spinal tract region in the C_5 sections from each dog except 7611. The origin of this degeneration was difficult to detect from the serial sections of segment C_4. It may have resulted from direct trauma, as the descending blade pushed through the dorsal gray horn

and swept through a portion of the lateral quadrant, or from compression of lateral quadrant fibers as the blade was pushed down through the gray horn, or from ischemia following direct trauma to branches of the anterior spinal artery. There was evidence of extensive ischemic degeneration in the gray columns, extending as much as 2 or 3 mm rostral and caudal from the site of the lesion, and this might reasonably be expected to have consequences in the white matter as well. The most extensive ischemic degeneration of gray matter was seen in dog 7611, and in this dog the diffuse descending fiber degeneration was seen in the left, rather than right, dorsomedial lateral quadrant as well as in the medial portion of the ventral quadrant. Whatever the origin of the descending degeneration, it was in all cases less complete than that seen in transected posterior columns, so that degenerated fibers were distributed among intact fibers. The asymmetry of this pattern of degeneration was not accompanied by <u>any</u> sign of asymmetry in any reflex or voluntary motor behavior pattern observed in any dog during the post-operative series of experiments. This is not to say that it might not have been detected by appropriate neurological examination, but simply that no gross observations gave a hint of its presence. The most pronounced clinical signs in dog 7611 were persistent ataxia, throughout the post-operative observation period, and persistence of the high-stepping front foot gait, which we have assumed to be a compensatory behavior pattern following a sensory

deficit. Thus it seems that no motor deficit directly related to the descending fiber degeneration was observed in any animal. All recovered animals appeared to have normal strength and vigor and engaged in the full repertoire of normal dog activities.

A diffuse region of ascending fiber tract degeneration was seen in the extreme dorsal part of the left, right, or both lateral quadrants. Exactly what projecting pathway these fibers belong to is difficult to determine. In a low cervical segment, one might immediately identfy them as spino-cervical tract fibers on an anatomical basis. However, they project through segment C_3 and C_2 on the extreme surface of the cord and largely unchanged in location between the segments. The anatomical location of spinocervical tract fibers in this region of the cord is not clearly pictured in the literature, but one would assume that these fibers would begin to plunge from the surface of the cord in toward the lateral cervical nucleus, at least in segment C_2 if not in C_3. If the fibers are passing fibers of such a long tract as the dorsal spinocerebellar tract, either direct or compression trauma would have been the cause of the degeneration. However, on examination of the serial sections above the lesion, it appears that the degenerating fibers are layered onto the cord surface from within over a distance of 2 or 3 mm. This would lead one to believe they might be rostral spinocerebellar tract fibers arising from the ischemically degenerated gray matter. The degeneration of these ascending fibers is much more

intense than that seen in the above mentioned descending tracts, but there are still many intact fibers intermingled with degenerated ones.

Because of lack of any experimental evidence to correspond to the observed descending fiber degeneration, no further discussion of it will follow. The ascending fiber degeneration in the dorsal portions of lateral quadrants will simply be lumped with that of the posterior quadrant in the following discussion. Again, no signs of asymmetry were noted in any observations that might correspond to the asymmetry of the degeneration patterns.

The posterior quadrant fibers were cut at a level well above the zone of entry of afferents from the brachial plexus (77). Thus the posterior quadrant projections of somatic afferents from hindlimbs, trunk, forelimbs, and low neck regions were transected. Most important among these fibers are numerous primary afferents projecting to the dorsal column nucleus complex (gracile, cuneate, and external cuneate nuclei). Also included among the cut fibers are the second order fibers of the ADFT of Uddenberg (116, 117), whose central terminations remain unknown. A completely unknown number of ascending or descending propriospinal fibers may have been cut, but the histology does not show that they are either numerous or project great distances.

Since all of the cut primary afferents terminate in the low brainstem, it is the second or higher order projections that are of interest

in a discussion of their contribution to postural control.

The large cerebellar projection, at least of the cuneocerebellar tract from the external cuneate nucleus, and possibly of other fibers from main cuneate and gracile nuclei, has a profound implication in postural control, since the output of the cerebellum very prominently impinges on all supraspinal structures involved in control of skeletal muscle motor neurons. Included among these structures under cerebellar influence are the vestibular nuclei, oculomotor nuclei, midbrain reticulum, sensorimotor cortex (via thalamus), red nucleus, basal ganglia, and certainly more. Both the pyramidal and extrapyramidal motor pathways, including such important projections as corticospinal, reticulospinal, rubrospinal, and vestibulospinal pathways are under constant influence of cerebellar activity.

The medial lemniscal system might well be expected to be involved in postural control. If only its projection to sensorimotor cortex via thalamus were considered, there is ample reason to believe that it might influence the involuntary control of skeletal muscles occurring during postural adjustments. Some postural reflexes such as the hopping reflex described by Magnus depend on an intact nervous system including the cortex in cats (73). The pyramidal tract, arising from the pyramidal cells of the sensorimotor cortex, is usually considered to be involved in the guidance of voluntary movements, but it is possible that many aspects of reflex control of skeletal muscle are

organized at the cortical level. Evarts (32, 33, 34) has demonstrated that pyramidal tract units related to precise distal forelimb movements in the monkey discharge with a frequency proportional to the force required to achieve those movements. The movements studied were produced by contraction of muscles whose ventral horn motor neuron pools are known to be under strong pyramidal tract influence (100). Cortical control of analogous forelimb muscles in the cat has been studied by Asanuma et al. (6) who have developed the capability of stimulating and recording from precisely localized small groups of pyramidal tract units. Cells whose motor outflow influences such forelimb muscles have been seen to be under excitatory influence of cutaneous and deep (joint) receptor projection pathways. Cutaneous receptive fields feeding back excitatory influences on such cortical neurons lie on the surfaces toward which those neurons tend to move the limb. Joint units excitatory to such neurons lie in joints distal to the activated muscles. Thus it seems that receptor activity capable of signaling resistance to movement (an index of the force required to execute the movement) feeds back to motor cortex in a fashion appropriate to insuring the effectiveness of the movement. The importance of the sensorimotor cortex and the role of such reflexes as the hopping reflex in the maintenance of a quiet standing posture have not been quantitatively estimated. It would seem that the cortex plays a minimal role since qualitatively normal postural behavior has been

described by Magnus in decorticate or thalamic animals (73). It is the musculature controlling distal portions of the limbs of dogs that is under the strongest cortical pyramidal tract influence, and it has been observed that some deficiencies of very fine postural adjustment (not grossly observable) follow local anesthetic blockade of the afferents from the paws of dogs (80).

Since it is known that a great deal of postural organization takes place in supraspinal but infracortical structures, it is likely that some of the most important terminations of afferents contributing to postural control might be those influencing the extrapyramidal motor system. The terminology "extrapyramidal" probably has a different meaning every time it is used, but we will use it here to include all supraspinal structures (including cortex) influencing behavior of skeletal muscle over pathways other than the corticobulbar and corticospinal paths. It should be well recognized that no portions of the nervous system in the intact condition are in fact independent of or isolated from any other portions; divergence of neuronal projections makes it possible for activity from any neuron to reach any neuron within the CNS. Thus there exists anatomical framework for many possible interconnecting loops to control behavior of skeletal muscle. It is highly unlikely, then, that pyramidal tract activity can bring about some sort of skeletal muscle activity without simultaneously influencing or being influenced by extrapyramidal activity. For example, suppose some

pyramidal tract outflow produced some hindlimb muscle tension alteration in a quietly standing dog that resulted in a slight movement of the animal. Immediately visual, labyrinthine, and somatic receptors would send afferent signals that would reach spinal reflex centers, brainstem reticulum, vestibular and olivary nuclei, thalamus, cerebellum, and cerebrum over pathways already described. Activity could then reflect back from cerebrum and cerebellum to basal ganglia, red nucleus, thalamus, brainstem reticulum, vestibular and olivary nuclei, and spinal cord to produce alterations in activity over reticulospinal, rubrospinal, and vestibulospinal (all extrapyramidal) pathways. And at the same time continuous loops of activity might be able to go on from cerebellum to thalamus to cerebrum to brainstem to cerebellum, for example, or over an almost infinite array of smaller or larger loops. The point is that the possible interconnections exist and rather than chaos resulting, well organized CNS activity brings about well organized skeletal muscle behavior so that simultaneous voluntary activity and involuntary reflex adjustment of muscles not directly involved in the voluntary activity can take place. And further, many of the reflexly organized patterns of behavior result from activity in structures that <u>can</u>, but not necessarily <u>do</u>, function independently of the cerebral cortex.

Therefore, since the basal ganglia, substantia nigra, red nucleus, thalamus, midbrain and medullary reticulum, olivary nuclear complex,

vestibular nuclear complex, and cerebellum are all importantly involved in adjustment of the extrapyramidal motor outflow to skeletal muscle, and since postural behavior is nearly normal in decorticate animals, one must conclude that afferent activity reaching these structures and no higher must contribute to postural control. And evidence mentioned above indicates that primary afferents in the dorsal columns project activity to most or all of these structures in addition to the well recognized medial lemniscal projection to sensorimotor cortex (4, 5, 29, 47, 48, 49, 56, 65, 70, 92, 94).

The physical transection of nerve fibers within the central nervous system brings about an immediate and irreversible interruption of impulse traffic on those fibers. In the six dogs used in this study, somatic sensory information of the type suitable to reflex organization of all skeletal muscle below the neck was abruptly interrupted at the primary afferent stage by surgical transection. The effects of this procedure on postural behavior provide a basis for conclusion about the role of such afferent activity in postural integration in the normal dog. The conclusion remains tentative until the anatomical extent and functional significance of the above outlined central terminations of "postural afferent" activity are disclosed for the dog.

Tentative Conclusions

All six dogs were immediately and profoundly incapacitated by the surgery, so that for several hours after recovery from surgical anesthesia they were not able to stand, with the exception of dog 8692 who could stand and walk but in a severely abnormal manner. They all showed, in their initial attempts to stand and walk, a severe ataxia, signs of skeletal muscle weakness, with collapsing of limbs, especially forelimbs, signs of loss of position sensation, with awkward positioning of limbs and stepping on dorsum of paws, and loss of the tactile placing and hopping reactions. Two dogs showed progressing signs of weakness, loss of appetite, and elevated temperature, but responded to antibiotic therapy and eventually followed a recovery pattern similar to the remaining four. The four that recovered without complication showed only day by day improvement, with no worsening of any of the observed signs, so that the post-traumatic inflammatory response seemed to play a very minor role in the observed effects of surgery. No unilateral motor deficits were seen to correspond to the histologically observed diffuse descending fiber tract degeneration; this was a minor consequence overwhelmed in extent by the effect of ascending fiber transection.

That all of the postural deficits resulted from interrupting the afferents to the cuneocerebellar tract is unlikely. The ataxia and

possibly the weakness may be explained by the removal of normal input to the cerebellum. The loss of placing and hopping reactions and the abnormal awkward positioning of limbs depended on the interruption of afferent activity bound elsewhere than cerebellum. The additional ascending degeneration seen in the dorsal poles of the lateral columns affected somatic mechanoreceptor activity traveling primarily to the cerebellum, but lack of any detectable signs unique to this additional fiber tract interruption obviates any conclusion from this experiment alone.

It is tentatively concluded that the primary afferents projecting in the spinal cord dorsal columns give rise to second and higher order activity that is essential to the normal function of the dog postural control system. An atraumatic and reversible technique of dorsal column fiber blockade would provide a much more satisfactory basis for such conclusion but at present we are limited to the ablative lesion technique.

Factors Affecting the Long Term Post-Operative Behavior

Recovery of Function Following Central Nervous System Lesions

For many years neurophysiologists have realized that the severe impairment of function following some central nervous sytem injury or experimental extirpation is gradually replaced by a partial or

complete recovery of that function (27). In mammalian organisms it is not the regeneration of destroyed cell bodies or their axonal projections that underlies such recovery, for although a great deal of new growth of neural tissue can occur after injury, there is some mysterious factor which prevents its successful re-establishment of the normal condition (54). The compensation that takes place in an injured nervous system is little understood, but involves, rather than regeneration, reorganization of remaining intact structures (93). The process may involve mechanisms that occur in normal nervous systems so that many aspects of compensation might appropriately be called re-learning and rely on the same kinds of anatomical and functional plasticity of interneuronal connections that underlie expansion of behavioral repertoire, learning and memory (31, 61, 89, 107, 114). Since the whole subject of information storage and retrieval in the nervous system is so vaguely comprehended at present, only a few pertinent conjectures concerning mechanisms of recovery of function can be made here. An important first principle to keep in mind is the anatomical substrate for the possibility of divergence of activity from any neuron within the CNS to any other neuron offset by the physiological evidence for well organized and repeatable neural function rather than chaos. In the normal condition there must be aspects of neural interrelations that facilitate information flow through specific pathways and that provide for continuing expansion of the number of such

facilitated pathways. Since neural activity provides the only basis for provision of information to the CNS and the information usually passes through several interneuronal relays, we can look on each post-synaptic element as a decoder of the information train impinging on it and as an encoder of the transmitted information (97). Many factors can affect the nature of the transmitted information in a post-synaptic unit and among these are the anatomical arrangements of synapses on the cell body and dendritic tree and the responsiveness of the post-synaptic membrane to release of transmitter from the pre-synaptic terminal (31, 61, 89, 97, 107). It is known that neurons exert "trophic" influences on other cells, that is influences which initiate or control molecular modifications in the other cell (53). Trophic influences of nerve on nerve are less understood than those of nerve on effector; however, chemical changes in neurons definitely can take place when other neurons terminating synaptically on them are destroyed (67). Such molecular rearrangements in neurons must certainly be expected to alter the information processing or transmitting characteristics of those neurons. It is clearly possible, then, that anatomical and molecular rearrangements in structures surviving trauma in the CNS may replace the function of the ablated structures, much like growth of collaterals re-establishes blood supply following trauma to the circulatory system. The effectiveness of such neural compensatory mechanisms is not necessarily great enough to

totally reproduce the normal condition, and is especially limited in the case that portions of the nervous system are extirpated or that lesions are massive. The discrete transection of a fiber tract provides the optimum condition for possible recovery of function; the regions to which activity in that tract normally travels might very well be served by alternate pathways from which the same information might be extracted after compensation takes place. Cells upon which projections of the dorsal column nuclei normally terminate, for example, might regain normal function following dorsal column transection if they are also served by collaterals from other afferent paths (spinocerebellar, for example) and the information transmitting character of the collateral terminations is appropriately altered. Possibilities for compensation become limited as the lesion becomes less discrete, and when structures are extirpated their function obviously cannot be restored. Such capability for functional reorganization complicates the interpretation of results of lesions, then, because the condition in the recovered nervous system does not necessarily represent absence of the function of the ablated structure.

There is at present no specific information that sheds light on the precise patterns of reorganization that underlie some known recovery of function following a nervous system lesion. Glees and Cole (45) report experiments in which they localized the cortical motor area producing a precise movement such as thumb movement in a

monkey. They undercut the localized area, producing a transient paralysis of the movement, but observed recovery in a few days' time. In a subsequent surgery they found that stimulation of cortex surrounding the undercut area could produce movement of the originally paralyzed structure. Such an observation indicates that new interneuronal relations have become functional, but does not hint at the nature of the new connecting pathway. Many examples might be drawn from the classical or recent literature, but few investigations concerning recovery of function are as eloquently reported as the study of cerebellar release phenomena by Batini, Moruzzi and Pompeiano (9). They dealt with four acute phenomena that can be observed in cats and attempted to evaluate their interaction in normal postural control of forelimb muscles:

1. Brainstem transection at about mid-collicular level brings about a marked extensor hypertonus
2. cutting the dorsal roots C_5 through T_2 abolishes this forelimb tonus in the decerebrate cat, indicating that the rigidity depends on the spindle reflex and thus arises in part from increased excitation of gamma neurons.
3. extensor hypertonus can be markedly augmented in the decerebrate cat or restored in the decerebrate deafferented cat by cerebellectomy or bilateral ablation of the fastigial nuclei; this rigidity arises from increased alpha neuron excitation and is

accompanied by opisthotonus (dorsiflexion of the head)

4. extensor hypertonus can be increased in the decerebrate cat or restored in the decerebrate deafferented cat by transection of the spinal cord below the segments giving rise to the brachial plexus; this Schiff-Sherrington phenomenon apparently involves a normal spino-bulbo-spinal inhibitory mechanism on alpha neurons and is not accompanied by opisthotonus.

They found that either cerebellectomy or bilateral fastigial nucleus destruction in the intact cat produced an opisthotonus and forelimb extensor rigidity that gradually faded in about a week. The same effects were present in deafferented cats, with an increase in rapidity of compensation of the forelimb hypertonus. When the compensated cerebellectomized or cerebellectomized plus deafferented cats were subjected to postbrachial spinal transection, the hypertonus and opisthotonus reappeared. The cerebellar and Schiff-Sherrington phenomena apparently result from withdrawal of inhibitory influences on alpha motor neurons. The compensated cerebellectomized animal apparently is under the influence of Schiff-Sherrington inhibition. When the postbrachial transection is done on normal cats, mild hypertonus without opisthotonus results, but fades in a few hours. Bilateral fastigial nucleus destruction done at this time results in hypertonus plus an extremely marked and persistent opisthotonus. Now if chronically cerebellectomized animals are decerebrated, extensor

hypertonus ensues and can be abolished by deafferentation. At this time, postbrachial spinal transection again establishes the extensor hypertonus.

The evidence upholds the view that some tonic inhibitory influences on alpha motor neurons operate in the normal animal via two independent pathways, one involving outflow from the cerebellum and the other involving a spino-bulbo-spinal pathway. The compensation following cerebellectomy apparently depends on reorganization of the Schiff-Sherrington inhibitory mechanisms.

A great deal is yet to be learned regarding mechanisms of compensation, but it is true that interruption of information flow over given sensory or motor paths ultimately results in the establishment of functional connections from other regions of the nervous system that partially or completely substitute for the lost function.

Some Possible Parallel Afferent Pathways

The transected dorsal column fibers are primarily afferents arising from an extensive array of somatic mechanoreceptors. They give rise to neural activity projecting to the cerebellum, cerebral sensorimotor cortex, and probably a large number of brainstem nuclei very intimately involved in reflex control of skeletal muscle activity. The total complement of collateral projections of other somatic afferent pathways to these same structures is not fully known. Recent

electrophysiological evidence shows that collaterals of dorsal spinocerebellar and cuneocerebellar tracts can activate lateral reticular nucleus (23). Dorsal, ventral, and rostral spinocerebellar tracts as well as the cuneocerebellar tract all carry activity from some and possibly all of the somatic mechanoreceptor types that project in the dorsal columns to some and possibly more than is presently known of the supraspinal structures that dorsal column afferents project toward. The spinocervical pathway is activated by many of the somatic mechanoreceptors, and in turn activates lateral cervical nucleus units which project activity to the thalamus; a cerebellar projection of these units is decisively ruled out (52), but experiments excluding other brainstem projections than that to the ventrobasal thalamus have not been done. Somatic mechanoreceptors even project to the thalamus in spinothalamic paths (98) and the full extent and/or existence of collateral projections of this mechanoreceptor path is not fully investigated.

Forelimb mechanoreceptor projections are more extensively interfered with than those of hindlimbs by the dorsal column transection in that a major set of primary afferents projecting to the cerebellum in the cuneo-cerebellar tract are cut in addition to the afferents bound toward the gracile and main cuneate nuclei. The intact alternate projection paths for somatic mechanoreceptor activity from hindlimbs, trunk, forelimbs, and low neck following dorsal column

transection include: dorsal spinocerebellar, ventral spinocerebellar, rostral spinocerebellar, spinocervical, and spinothalamic.

The Role of Dorsal Columns in Normal Behavior

Some chronic deficiencies in postural behavior remain in the compensated dog following bilateral cervical dorsal column transection. The deficiencies are very slight and apparently involve only some of the fine, rapid adjustments of distal limb musculature involved in the dynamic operation of the postural control system. It is interesting to note that the fine patterns of force variation seen beneath the feet of compensated dorsal column sectioned dogs is similar to that seen in dogs subjected to anesthetic blockade of afferents from the distal portions of the limbs, especially forepaws (80). The existence of uncompensated signs of the lesion would suggest that either some postural control afferents travel uniquely in the dorsal columns or that some central termination of dorsal column afferents is not served well by alternate projecting pathways. The profound acute effects of dorsal column transection that disappear over a time course consistent with the many reported compensatory phenomena in the literature suggest that the afferents traveling in the dorsal columns are very importantly involved in the normal mechanisms whereby a dog adjusts skeletal muscle activity to maintain an erect quiet stance.

Conclusions

The experiments reported here indicate that the afferents traveling in the dog spinal cord dorsal columns provide input to the nervous system that is very important in the normal function of maintaining a quiet stance. However, after compensation, probably by establishment of new functional connections by afferents traveling in paths parallel to the dorsal columns, dogs with transected dorsal columns can stand in a nearly normal fashion with postural deficiencies that are slight and not clearly defined by the methods of examination used. The study provides a baseline against which the degrees of dispensability of alternate pathways can be evaluated in future experiments. Further anatomical and physiological studies of the dog central nervous system with emphasis on the differential analysis of peripheral receptor spectrum and central projection terminations of the postural afferent projection pathways must be done before further understanding can be achieved.

SUMMARY AND CONCLUSIONS

The maintenance of an erect quiet stance by a dog has been viewed as the dynamic output of a feedback operated control system involving sensory receptors, central nervous system processing centers, and output regulating tension in appropriate skeletal muscles. A test has been devised to estimate an aspect of steady-state system behavior (the limit within which front-foot weight distribution may be maintained) and the dynamic response to a virtual step input has been characterized.

Sensory afferents projecting in the spinal cord dorsal columns signal information about muscle tension and position of body parts with respect to each other and their surroundings. Such information projects to supraspinal structures known to be important to the organization of postural behavior. The possible contribution of this information to normal operation of the postural control system has been tested by observing steady-state and dynamic system behavior before and after transection of the dorsal columns at a level blocking hindlimb, trunk, forelimb, and low neck receptor projections.

Dogs are severely incapacitated by cervical dorsal column transection so that acutely no shred of normal postural behavior is displayed. A period of recovery follows, with dramatic change during the first few days and more gradual change over the course of

about four weeks. After stabilization of clinical recovery, they show a slight but significant deterioration in steady-state performance. Dynamic response to step displacement, measured as change in body position or angles at hindlimb joints, is affected by the lesion, but in a way that is not clearly understood on the basis of the observations reported here; the changes are slight, subtle, and in no way indicative of a loss of capability to respond to displacement.

Compensation following the lesion probably involves a great deal of anatomical and functional reorganization at central terminals of any or all of the remaining spinal cord projection pathways. The relative importance of the dorsal column projections in postural organization may be tested in further experiments by observing acute and chronic effects of lesions in other projecting spinal cord paths on postural behavior. The profound acute postural deficiencies following dorsal column transection indicate that the afferents projecting in the dorsal columns are very importantly involved in operation of the normal dog postural control system.

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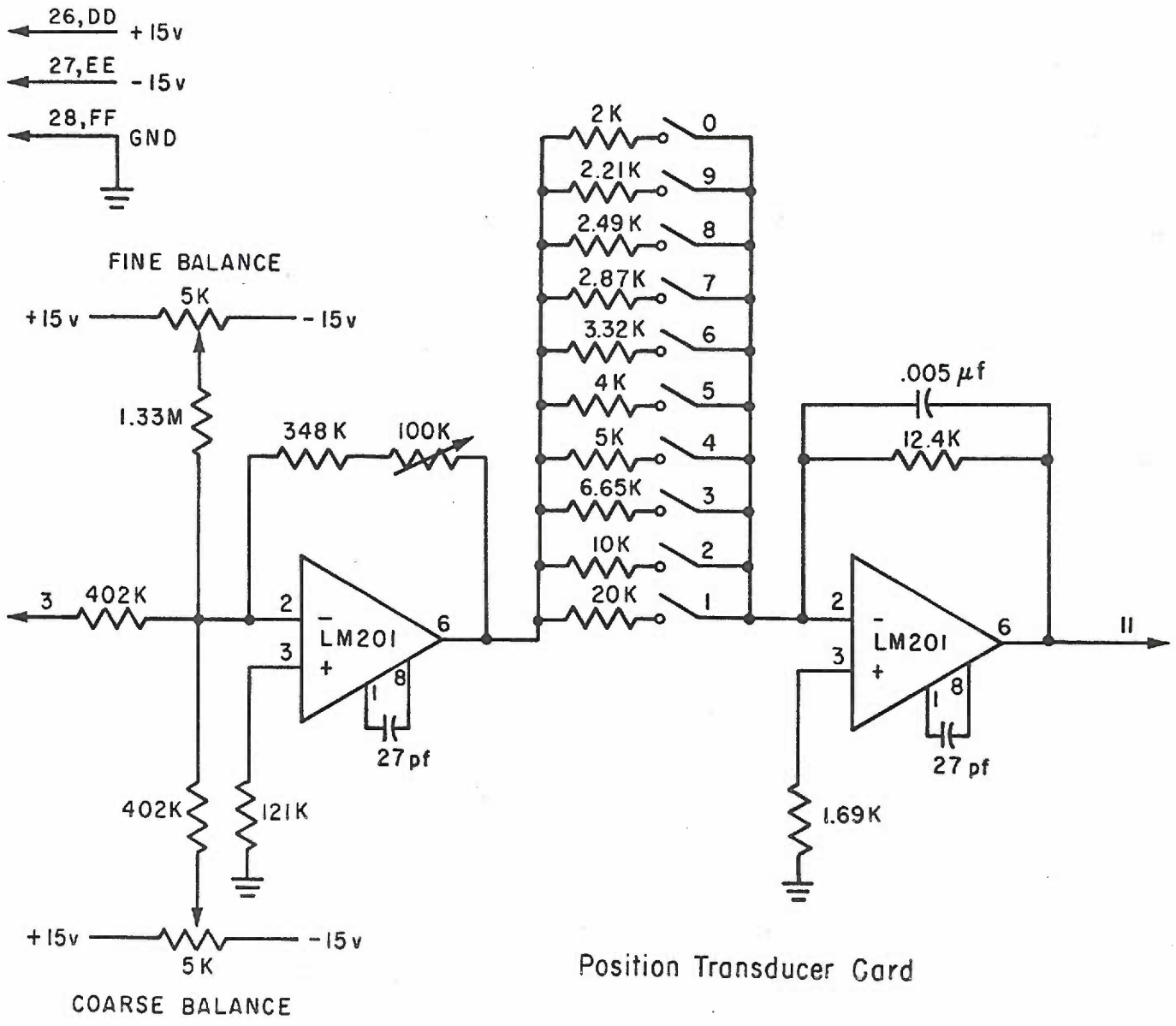
APPENDICES

APPENDIX A

Schematic of the Position Detector Output Amplifier

The position detector is comprised of a lever-actuated high resolution potentiometer (New England Instruments ECONOPOT, 1K, ± 10%, linearity 0.25%) operated with the ± 15V supply, with its wiper connected to the input (pin 3) of the amplifier shown here. The decade switch provides gain adjust in steps of 1 through 10 times first stage gain. With overall gain adjusted to provide a nominal sensitivity of 50 mV per mm deflection of the lever tip, the system provided resolution of ± 0.3 mm over a ± 100 mm range. All resistors are 1/4 watt, ± 1% with exception of the potentiometers.



Position Transducer Card

APPENDIX B

Schematics of Control Electronics

The components of the following four schematics are all located on the same card, so that the component identification and pin connections refer to all four schematics. The control section, limit detector, oscillator, and mode indicator all function interdependently, and they are connected at the indicated points A, B, C, and D. Control function and component interaction are discussed in methods.

Components:

U1: TI SN7420N dual 4-input positive NAND gate

U2: TI SN7400N Quadruple 2-input positive NAND gate

U3: TI SN7474N Dual D-type edge-triggered flip-flop

U4, 5: TI SN7476N Dual J-K master-slave flip-flops

U6: Fairchild MSI 9311 one-of-sixteen decoder

U7, 8, 9: TI SN7404N Hex inverters

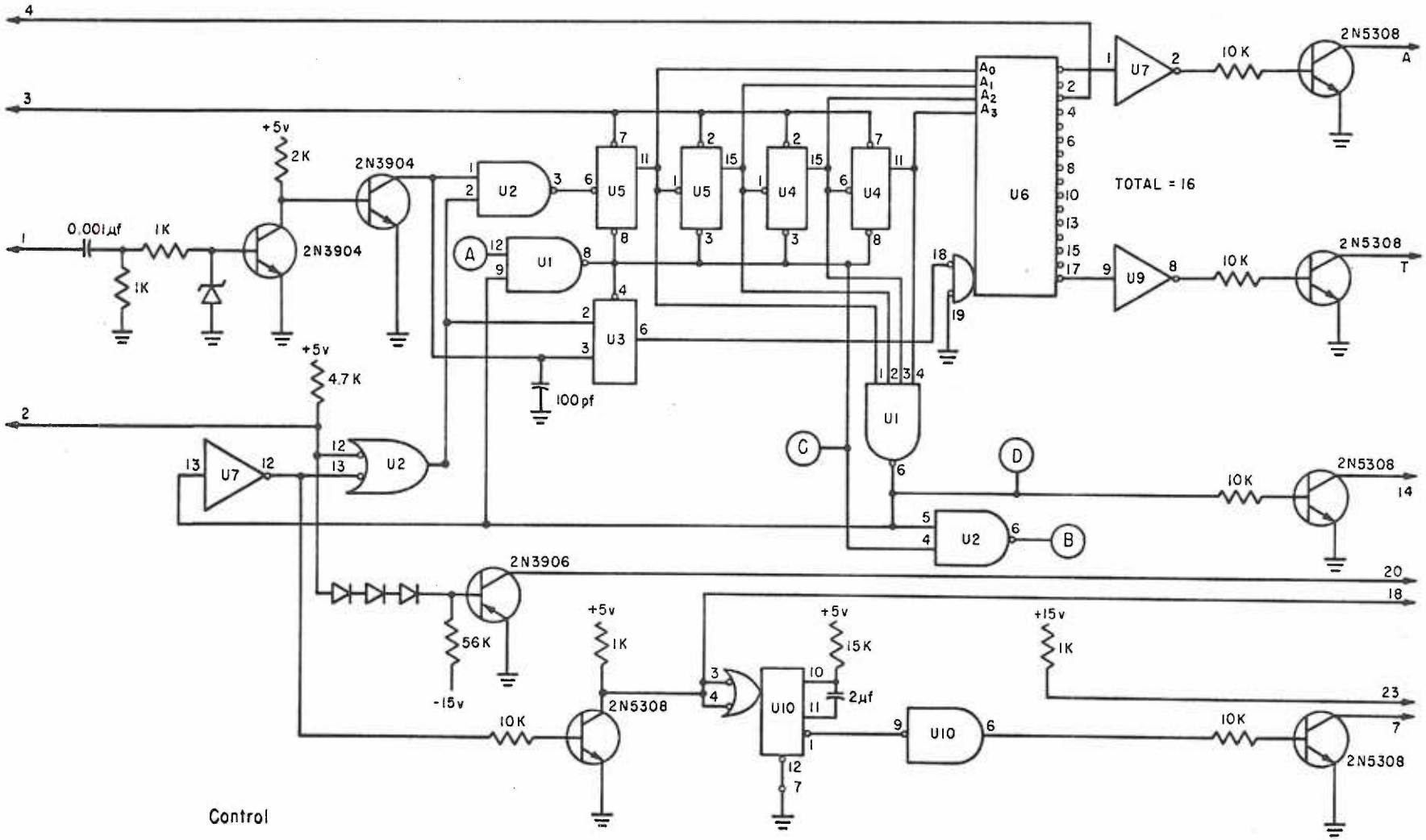
U10: Raytheon RM988 monostable multivibrator

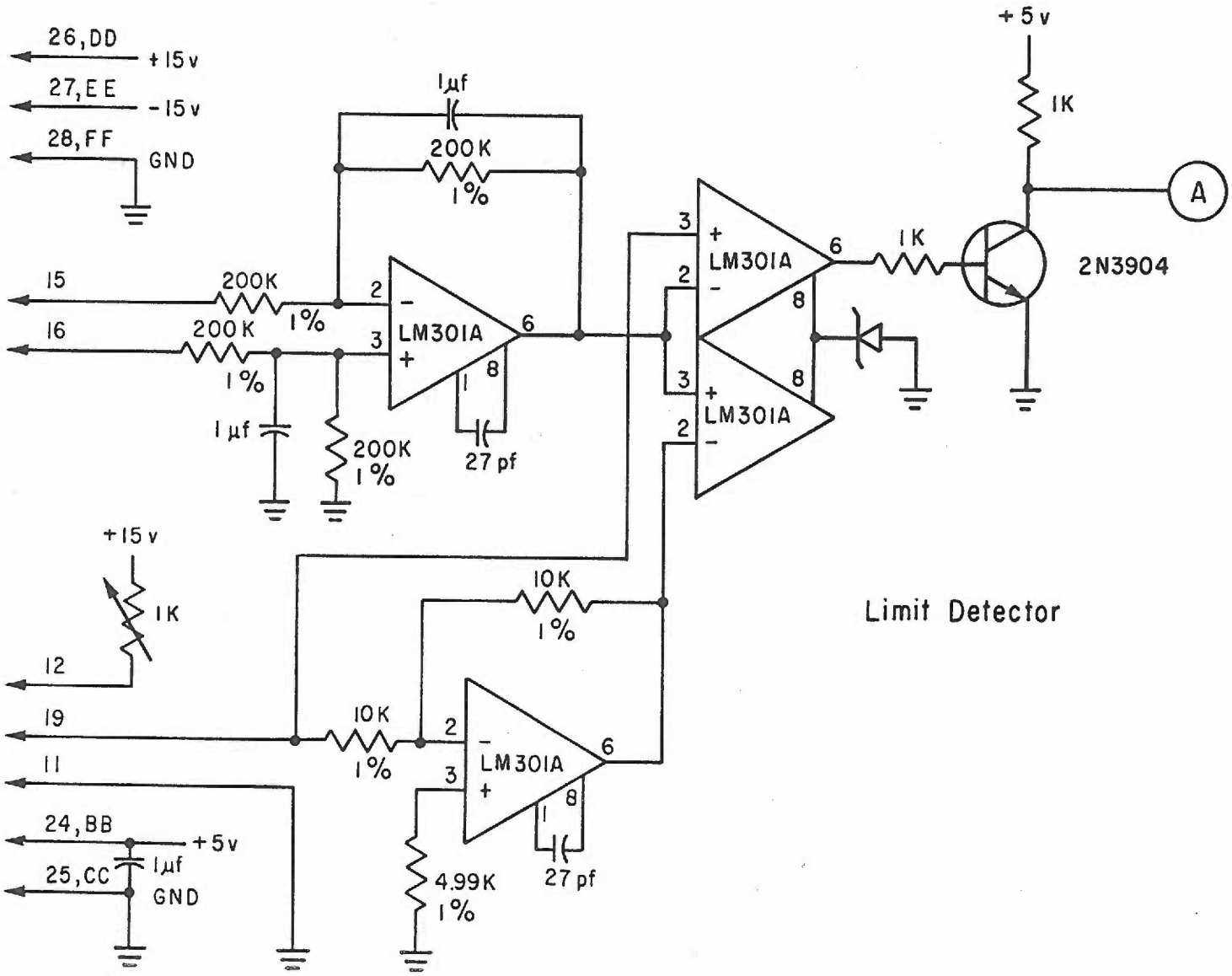
(TI-Texas Instruments)

All resistors are 1/4 watt, 5% except as noted.

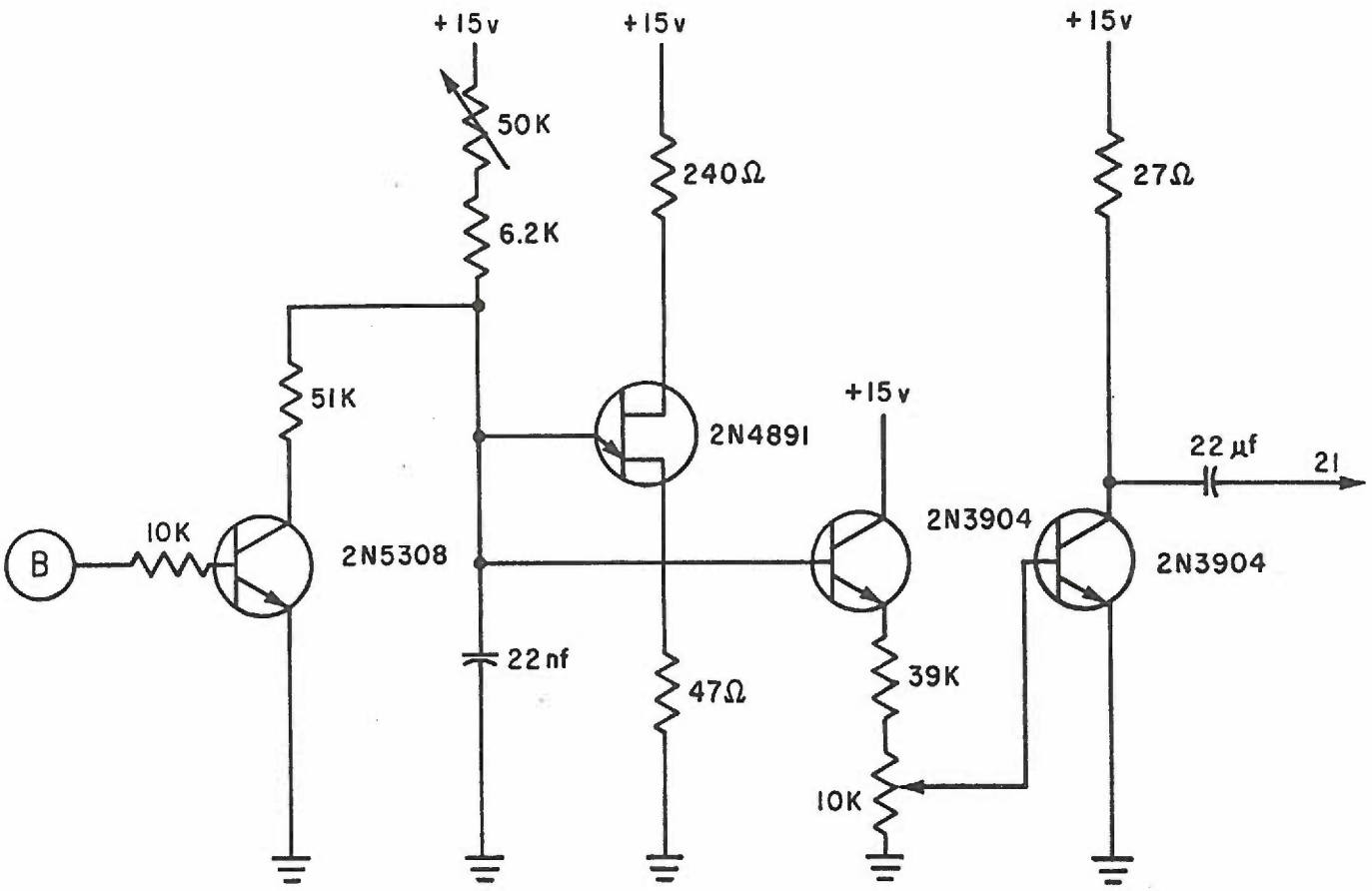
Pin connections:

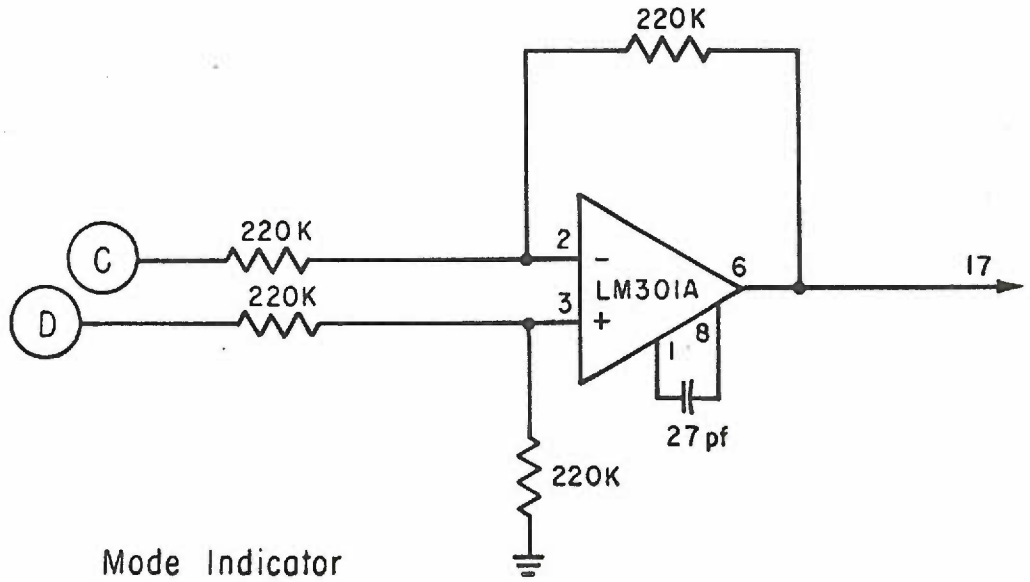
1	Input clock	A	To lamp 1
2	Start	B	To lamp 2
3	J-K flip-flop resets	C	To lamp 3


4
3
2
1
+5v
2K
2N3904
0.001μf
1K
1K
2N3904
+5v
4.7K
100pf
U1
U2
U3
U5
U4
U6
U7
U9
A
B
C
D
TOTAL = 16
10K
2N5308
A
T
14
2N3906
56K
-15v
+5v
1K
2N5308
+5v
15K
2μf
U10
+15v
1K
23
7
Control


26, DD
+15v
27, EE
-15v
28, FF
GND
1µf
200K
1%
+5v
1K
A
15
200K
1%
16
200K
1%
LM301A
2N3904
1K
1µf
200K
1%
27 pf
+15v
1K
12
10K
1%
19
10K
1%
11
LM301A
24, BB
+5v
1µf
25, CC
GND
4.99K
1%
27 pf
Limit Detector

Oscillator


+15v
+15v
+15v
50K
6.2K
240Ω
27Ω
51K
2N4891
+15v
22 µf
21
B
10K
2N5308
2N3904
2N3904
22 nf
47Ω
39K
10K



Mode Indicator

4	Level for quick-feed option	D	To lamp 4
5		E	To lamp 5
6		F	To lamp 6
7	Trigger to feeder relay	H	To lamp 7
8		J	To lamp 8
9		K	To lamp 9
10		L	To lamp 10
11	GND	M	To lamp 11
12	+ To limit trigger level pot	N	To lamp 12
13		P	To lamp 13
14	Start Counter	R	To lamp 14
15	Signal in (left front foot)	S	To lamp 15
16	Signal in (right front foot)	T	To lamp 16
17	Mode signal out	U	+ 5V to all lamps
18	Trigger out	V	
19	Limit trigger level	W	
20	Reset counter	X	
21	OSC signal to speaker	Y	
22		Z	
23	+15V to feeder relay	AA	
24	+5V supply	BB	+5V supply
25	GND	CC	GND
26	+15V supply	DD	+15V supply
27	-15V supply	EE	-15V supply
28	Supply return	FF	Supply return

APPENDIX C

Schematics of Digital Sampling Format Electronics

The sampling pulse generator output drives the direct record channel record head of the Honeywell 7600. The trigger out, coincident with the tenth sampling pulse, triggers the Exact model 255 function generator. The trigger in is provided by the starting switch of the control electronics (Appendix B).

The digital read amplifier replaces the direct reproduce amplifier of the Honeywell 7600 and its output provides properly shaped input pulses to the Raytheon Multiverter for data sampling (see Methods).

For both cards, resistors are 1/4 watt, 5% except as noted.

Sampling pulse generator components:

U1: Fairchild TT μ L 9601 monostable multivibrator

U2, 4, 10, 11: TI SN7400N Quad 2-input NAND gate

U3, 8, 9: TI SN7490N Decade counter

U5, 6, 12: TI SN7476N Dual J-K master-slave flip-flops

U7: Raytheon RC 988D monostable multivibrator

Q1, 2: 2N5308

Q3, 5, 6, 8: 2N3906

Q4, 7: 2N3904

U13: Fairchild DTμ l 9951 Monostable multivibrator (T0-100 case)

(TI-Texas Instruments)

Units 1, 2, and 4 (part) make up the clock. The monostable multivibrator is in the astable mode. The frequency is tuned to 320.0 Hz with R1. The output pulses are about 70 nanoseconds wide.

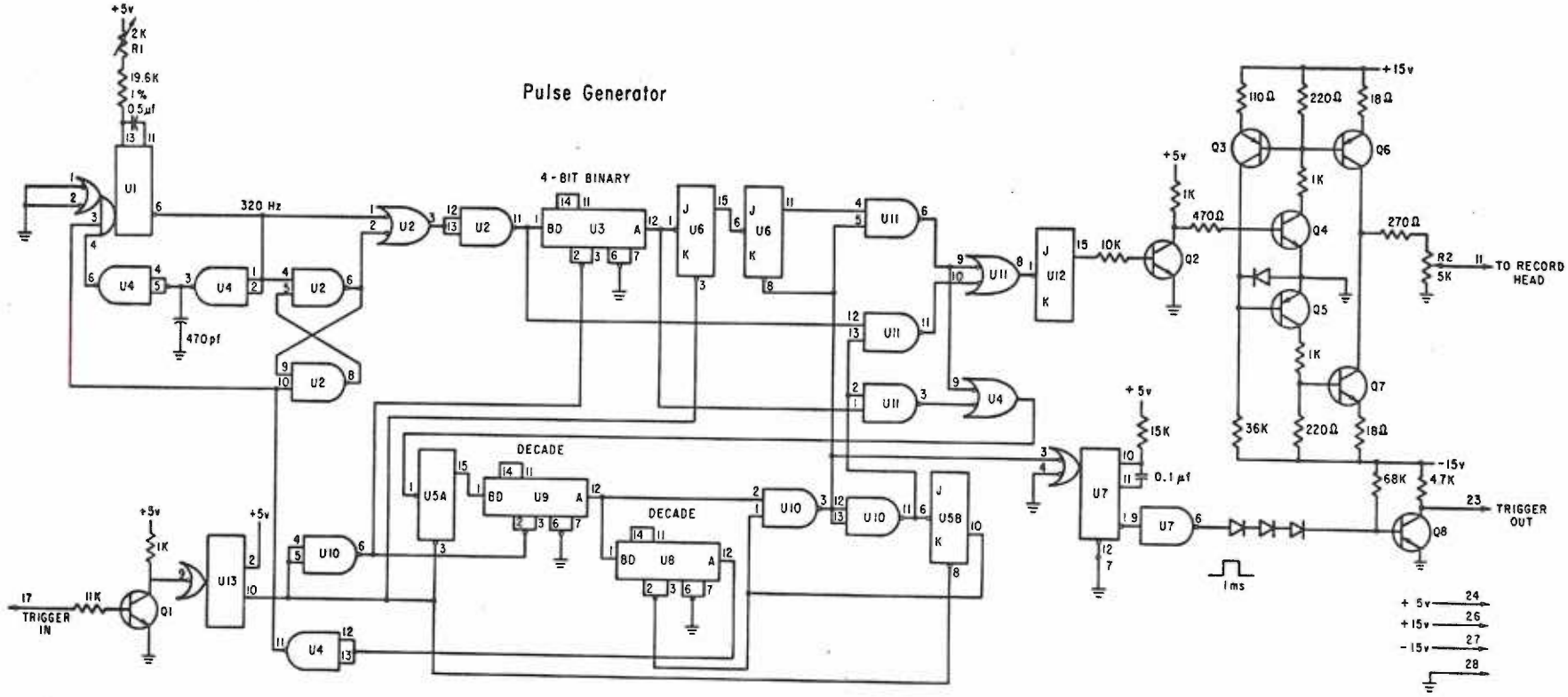
Units 3 and 6 drop the clock frequency to a 5Hz square wave.

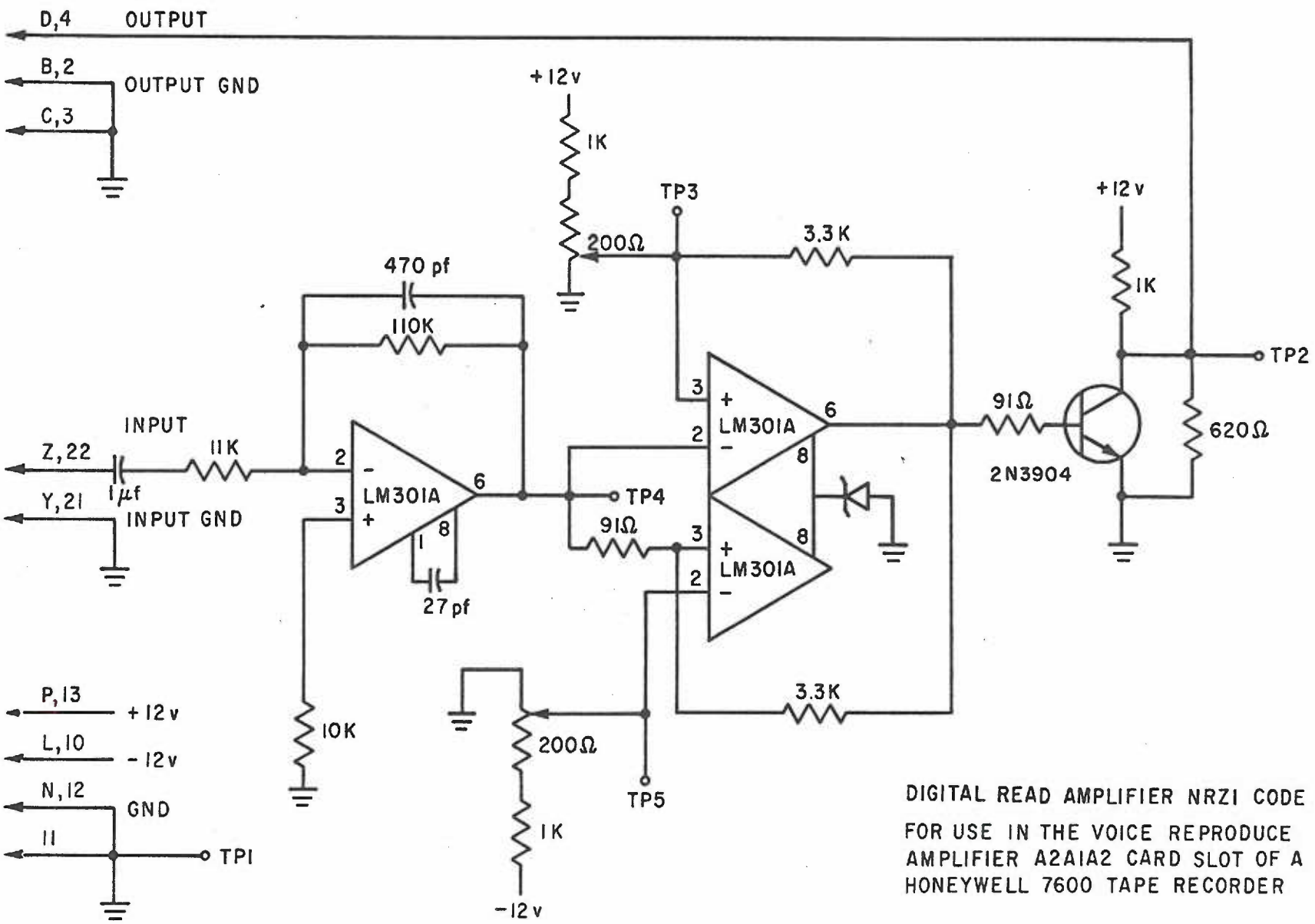
Units 4 (part), 5, 8, 9, 10 and 11 control the output frequency and number of pulses: a trigger in pulse sets decade counters 3 and 9 to zero, flip-flops 5a and 6 to zero, and flip-flop 5b to one thus setting and holding counter 8 at zero. This enables the clock. Ten pulses @ 5Hz, followed by 160 pulses @ 320 Hz, followed by 100 pulses @ 5Hz are generated. The trigger out (-15V, 1 msec) is generated at the end of the tenth output pulse. The clock is disabled at the end of the 270th output pulse.

Unit 7 provides the 1 msec trigger out pulse.

Unit 12 transforms its input pulses so that each transition at its output provides a sampling pulse to the head driver amplifier.

Transistors Q2 through Q7 make up the head driver amplifier.


Pulse Generator
+5v
2K
R1
19.6K
1%
0.5μf
U1
320 Hz
U4
U4
U2
470pf
U2
U2
U2
4-BIT BINARY
BD U3 A
J U6 K
J U6 K
U11
U11
U11
U4
U11
J U12 K
15 10K
+5v
1K
470Ω
Q2
+15v
110Ω
220Ω
18Ω
Q3
Q6
1K
Q4
270Ω
R2 5K
TO RECORD HEAD
Q5
1K
Q7
36K
220Ω
18Ω
-15v
68K
4.7K
23
TRIGGER OUT
Q8
+5v
15K
0.1 μf
U7
U7
1ms
DECADE
U5A
BD U9 A
DECADE
BD U8 A
U10
U10
U10
U5B
+5v
1K
+5v
17
TRIGGER IN
11K
Q1
U13
U10
U4
+ 5v 24
+15v 26
-15v 27
28



DIGITAL READ AMPLIFIER NRZI CODE FOR USE IN THE VOICE REPRODUCE AMPLIFIER A2A1A2 CARD SLOT OF A HONEYWELL 7600 TAPE RECORDER