BRAINSTEM AND SPINAL CORD PATHWAYS INVOLVED IN THE CONTROL OF AVIAN LOCOMOTION

By

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ABSTRACT

This study examined several aspects of the neural control of locomotion in birds. Initially, it was necessary to define an index of normal locomotor functions. This was accomplished for both flying and walking using electromyographic analysis of forelimb and hindlimb musculature to determine which muscles best define the flight and walking patterns respectively. Secondly, in chronic surviving birds, a series of subtotal spinal lesioning experiments were performed to determine which descending pathways were responsible for the initiation of hindlimb locomotion. Thirdly, results were recorded from brainstem electrical stimulation studies designed to determine the location of locomotor areas in the avian brainstem which effected the initiation and descending control of locomotion in these animals.

Results indicated the iliotibialis cranialis (ITC) and flexor cruris lateralis (FCL) muscles best define the swing and stance phases of hindlimb locomotion, respectively. Muscles which best defined the elevator and depressor phases of flying were deltoideus major (DM) and pectoralis (Pect), respectively.

Results of the low thoracic selective lesioning experiments support the hypothesis that the medullary reticulospinal pathway is necessary to the initiation and control of volitional hindlimb locomotion. Further, descending input to spinal cord pattern generators via the vestibulospinal pathways may play some adjunctive role or be necessary for the descending control of locomotion.

Electrical stimulation of the brainstem in acute decerebrate birds elicited locomotor behaviours in both hindlimbs and forelimbs. Four areas, including; an area near the lateral/medial spiriform nucleus; nucleus et tractus descendens trigemini; and central nucleus of the medulla, pars ventralis and dorsalis; and the lateral reticular nucleus produced varying locomotor behaviours when stimulated. Acute dorsal cord transection did not affect the electrically stimulated behaviour, indicating that descending pathways from supraspinal centres which travel in the dorsal cord do not affect the descending control of locomotion.

A strong parallel exists between the results of this study in two avian species and those found in the mammalian literature.

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GENERAL INTRODUCTION

The neural control of locomotion in vertebrates is achieved by the integration of several levels of organization. At its most simple level, the monosynaptic reflex arc utilizes only two neurons to produce a relatively simple motor behaviour in response to a simple mechanical stimulus (eg. patellar tendon reflex) at the spinal cord level. More complex levels of organization also occur within the spinal cord, where groups of intrinsic neurons interact to produce localized rhythmical movements.

Historically, Freusberg (1874), Freusberg and Goltz (1874,a,b) and Sherrington (1910) were the first to study the performance of cats and dogs following removal of supraspinal influences. After spinal transection or surgical decapitation, they found that the animals continued to step in a rhythmic alternating fashion. This "spinal stepping" provided the first direct evidence that higher brain structures (supraspinal) were not necessary for the maintenance of the basic pattern of locomotion. Sherrington (1910) also suggested that this post-transection locomotion was dependent on a series of reflex actions which were activated by peripheral afferent (sensory) input.

Graham-Brown (1911) disproved this hypothesis by demonstrating that previously deafferented animals could perform stepping movements following spinal cord transection. Graham-Brown (1914) postulated the Half-Centre hypothesis of spinal cord pattern generators as the mechanism underlying spinal stepping. The term pattern generator has been assigned to these neuronal

networks and implies that the motor pattern of each limb may be intrinsically generated within the isolated spinal cord. Each generator is thought to interact with the pattern generators of other limbs to produce the rhythmic coordinated movements of locomotor progression (Grillner, 1975). This rhythm was postulated to be an intrinsic property of the interconnections of antagonistic nerve cells (ie a central pattern generator). Grillner and Zangger (1975), supporting Graham-Brown's hypothesis, demonstrated that spinal-transected, paralysed animals devoid of any rhythmical peripheral feedback produce the patterns of alternating motor activity, as recorded from peripheral nerve efferents. The electroneurograms (ENGs) recorded from this procedure constitute "fictive" locomotion.

One difficulty with a phylogenetic theory of locomotor pattern generators occurs at the level of the primates. It has yet to be demonstrated that "spinal stepping" can occur in any primate species. Several investigators have hypothesized that this lack of "spinal stepping" in humans and other primates results from an increased dependency on supraspinal influences for the activation of spinal stepping mechanisms (Eidelberg, 1981a,b; Grillner, 1975). Eidelberg (1981a) postulates that tonic descending facilitatory influences are necessary for the pattern generators to access output motoneurons.

The highest level of motor integration occurs within supraspinal structures. These areas are responsible for the initiation and control of volitional locomotion.

Telencephalic structures are known to be directly involved in motivational commands eliciting locomotor responses (Wetzel and

Stuart, 1976; Eccles, 1980). In mammals, the premotor and motor cortex of the precentral gyrus give rise to descending corticospinal neurons of the pyramidal system (Kuypers, 1982). Stimulation of various regions of the motor cortex elicits discharge in motorneurons innervating both distal and proximal extremity muscles (Marsden et al., 1981, Kuypers, 1964). However, these direct corticospinal connections are thought to be considerably more important to highly fractionated movements of the distal extremity muscles than to the production of basic locomotor patterns (eg. walking) (Kuypers, 1982). Indeed, a major component of the corticospinal (pyramidal) tract arises from the somatosensory cortex and not from motor cortex (Kuypers, 1982). Stimulation of pyramidal tract neurons has only been shown to disrupt locomotion when high current strengths are used. Stimulation at lower stimulus strengths only increases flexor or extensor activity during the appropriate portion of the step cycle (Shik et al., 1966; Orlovsky, 1972a). Bilateral pyramidotomy within the caudal brainstem does not inhibit the initiation of locomotion by stimulation of extrapyramidal motor areas such as the mesencephalic locomotor region (MLR) (Shik et al., 1968). This would tend to indicate that corticofugal initiation and control of voluntary locomotion, even in primates, may be mediated via the phylogenetically older brainstem structures of the extrapyramidal motor system. In fact, decerebrate fishes, amphibians, birds, and all sub-primate mammals exhibit little locomotor difference from intact animals (Wetzel and Stuart, 1976; Grillner, 1975; Gabbott and Jones, personal communication).

One predominant extrapyramidal motor area is the basal ganglia (caudate nucleus, putamen, globus pallidus, and amygdaloid nuclear complex) which has been shown to be severely compromised in movement disorders such as Parkinsonianism, hemiballismus, and Huntingtons chorea (Carpenter 1978). Although these disorders constitute severe impairments to motor performance, their major effect appears to be of a postural nature (Wetzel and Stuart, 1976). Stimulation of the globus pallidus produces inconsistent locomotor responses in a lightly anaesthetized cat (Waller, 1940). Monkeys, dogs and cats with bilateral ablation of various portions of the basal ganglia do not show severe impairment of coordinated progression (Waller, 1940; Denny-Brown, 1966; Hinsey et al., 1930).

Diencephalic structures such as the epithalamus, thalamus, and hypothalamus (ventral thalamus) also effect control of locomotion. Hinsey et al. (1930) reported that thalamic and hypothalamic cats and dogs walk following surgery. If the rostral portion of the thalamus is left intact, the animals will then walk spontaneously and display behaviours which resemble motivated activities. Brainstem transection leaving only the caudal third of the thalamus intact produces animals which will locomote only under strong exteroceptive stimulation (Laughton, 1924). Thalamic cats with midline rostrocaudal division of the brainstem, however, lose the ability of coordinated progression indicating the possibility that thalamic locomotor neurons require bilateral connections to more caudal brain structures (Laughton, 1924).

The subthalamus and subthalamic nucleus (including the posterior and lateral hypothalamus) also appear to be involved in

motor control. Although destruction of the subthalamus in an intact cat does not prevent locomotion (Haertig and Masserman, 1940), electrical stimulation of this region is known to elicit alternating stepping movements in lightly anaesthetized animals (Waller, 1940; Haertig and Masserman, 1940). Further, Orlovsky (1969), found that stimulation of the subthalamic region in an acute thalamic cat evoked locomotion on a treadmill. Nevertheless, cats with bilateral destruction of this Subthalamic Locomotor Region (SLR) are able to run and walk during electrical stimulation of a more caudal brainstem motor area, the mesencephalic locomotor region (MLR) (Sirota and Shik, 1973). Shik and Orlovsky (1976) postulated that the SLR effects the initiation of locomotion in goal directed behaviours and therefore cannot be described in strictly motor terms. Shik, Severin, and Orlovsky (1966), discovered that electrical stimulation of an area in the midbrain, which they termed the Mesencephalic Locomotor Region (MLR), could elicit what appeared to be normal locomotion in a decerebrate cat walking on a treadmill. These findings have allowed an initial characterization of some of the brainstem areas which form the presumptive relay pathways between higher brain structures responsible for volitional control and the final common pathways of the spinal central pattern generators.

Since the discovery of the MLR, several investigators have enlarged the information available by delineating other areas which will evoke locomotion using electrical stimulation. These include the lateral parabrachial nucleus (Garcia-Rill et al., 1983a), the periaquaductal gray (Garcia-Rill et al., 1983a), the

nucleus tegmenti pedunculopontis (Garcia-Rill et al., 1983a), the ponto-medullary locomotor strip (PLS) (Budakova and Shik, 1980), Probst's tract (Garcia-Rill et al., 1983d), and the ventro-medial gigantocellular and magnocellular reticular formations (Shik and Yagodnitsyn, 1977; Mori et al., 1978; Steeves and Jordan, 1984). Presently, the functional pathways underlying these electrically stimulated responses are not well understood. It is presently unknown whether locomotion was evoked by stimulation of some of these proposed locomotor regions are simply the result of activating axonal fibres of passage from higher brain areas or, alternatively, that the stimulated areas elicit locomotion indirectly via other brainstem areas which also descend to spinal cord central pattern generators.

Areas in the hindbrain which are also thought to play a role in locomotion include the vestibular and red nuclei, both of which have direct spinal cord connections via the vestibulospinal and rubrospinal tracts (Kuypers, 1982). Orlovsky (1972b) found that vestibulospinal neurons arising predominantly from Dieters nucleus exerts a facilitatory effect on extensor muscles. The rubrospinal neurons primarily exert their effects on flexor muscles, facilitating flexor activity during the swing phase of locomotion (Orlovsky, 1972c). Stimulation of either of these nuclei or their descending pathways yeilded the same facilitatory results during both rest and locomotion (Orlovsky, 1972a). However, destruction of these pathways and their nuclei in chronic animals does not severely impair rhythmic locomotor movements (Yu and Eidelberg, 1981; Shik et al., 1968; Ingram and Ranson, 1932).

Reticular formation neurons descending directly to the spinal

cord are thought to play an important role in the control of spinal cord pattern generators (Eidelberg, 1981b; Steeves and Jordan, 1980; Afelt, 1974). Lesion studies in cats and monkeys in both acute decerebrate brainstem stimulated and chronic spinal preparations, have demonstrated that intact ventrolateral quadrants of the spinal cord are required for locomotion (Eidelberg, 1981b; Steeves and Jordan, 1980; Afelt, 1974). This portion of the cord is known to carry reticulospinal neurons descending from the mid and hindbrain in mammals (Kuypers, 1982).

To date, the initiation and control of spinal locomotor mechanisms by supraspinal brainstem centers has been studied in many vertebrate species (for review see Grillner, 1975; Eidelberg, 1981). "Spinal stepping" or its equivalent has been shown to occur in lower chordates such as lamprey (Grillner, 1975; Grillner, McClellan and Sigvardt, 1982); fish (Grillner and Wallen, 1977; Williams et al., 1984); reptiles (Ten Cate, 1965); birds (Tarchanoff, 1895; Ten Cate, 1960); and mammals excluding primates (Freusberg, 1874; Sherrington , 1910; Phillippson, 1905; Eidelberg, 1981b). The predominant experimental animal for locomotion studies has been the cat. Its easy aquisition, well defined quadrapedal gait patterns and mammalian character has made it a good experimental animal for studies of locomotion. Nevertheless, the complexities of the mammalian system have made it difficult to define the centrally programmed "step generators" in this animal. The quadrapedal gait and its' requisite neuronal interconnections between hindlimb and forelimb introduces a level of complexity which makes characterization of the pattern

generators considerably more difficult than in an animal with a less complex nervous system.

The bird appears to be an interesting animal for the study of locomotion. Its two completely different forms of locomotion require in-phase central pattern generation for flying, during which the wings beat synchronously, and out of phase generation for walking and swimming, where the legs alternate activity. Behaviourally, the interconnection of these two sets of generators appears to occur only during take-off and landing, when the modes of locomotion overlap. Jacobson and Hollyday (1982b) have suggested that two different supraspinal pathways might mediate the initiation of walking and flying in birds. Except for a few studies (Ten Cate 1960,1962; Tarchanoff, 1895), in which the phenomenon of spinal stepping was documented, very little research has been attempted to isolate and study the central control of movements in birds.

Supraspinal mechanisms responsible for the initiation and descending control of avian locomotion have not been examined. The previous findings in other vertebrates has led this experimenter to examine descending control systems in the avian central nervous system. While the cat and monkey have a well developed cerebral cortex, the bird does not possess a highly organized corticospinal (pyramidal) system (Cabot et al., 1982;) and no avian equivalent of motor cortex has been described in birds (Reiner et al., 1982). This deficiency strongly implicates more caudal brainstem structures in the control of locomotion and allows one to study a complex motor system that is devoid of corticomotor influences, even in the intact state.

The review of the literature indicates that very little is known about the physiology and anatomy of structures controlling locomotion in birds (Eidelberg, 1981b). Phylogenetically, birds occupy a "middle" position in vertebrate evolution yet display "advanced" qualities such as bipedal walking similar to human locomotion. All of which makes them very interesting from the standpoint of comparative physiology.

The Canada goose, Branta canadensis, was chosen as the experimental animal, as it displayed the qualities of being an excellent walking bird as well as a remarkable long distance flyer. Further, it is readily available for study, is easy to handle in captivity, and is not an endangered species. The domesticated non-flying, Pekin duck, Anas platyrhynchos and the Pekin/Mallard cross duck Anas platyrhynchos was also utilized in some of the acute brainstem stimulation experiments to determine the applicability of the experimental findings with the goose to other avian species.

Initially, it was necessary to define an index of normal locomotor functions. This was accomplished for both flying and walking using electromyographic analysis of forelimb and hindlimb musculature to determine which muscles best define the flight and walking patterns respectively. Secondly, in chronic birds, a series of subtotal spinal lesioning experiments were performed to determine which descending pathways were responsible for the initiation of hindlimb locomotion. Thirdly, preliminary results were recorded from brainstem electrical stimulation studies designed to determine the location of locomotor areas in the avian

brainstem which effected the initiation and descending control of locomotion in these animals.

CHAPTER I

FUNCTIONAL CHARACTERIZATION OF LIMB MUSCLES INVOLVED IN LOCOMOTION IN THE CANADA GOOSE, *Branta canadensis*

INTRODUCTION

The utilization of the Canada Goose as an experimental animal for its qualities of being both a good bipedal walker and an excellent flyer requires that the musculature be examined to determine which muscles could be readily utilized as indicators of normal locomotor patterns. Consequently, it is necessary to find wing (forelimb) muscles which are both active either during the elevator or depressor phases of flight. Hindlimb (leg) muscles which are active and necessary for defining normal walking (a toe extensor may be active during walking, but is not essential for "normal" appearing locomotion) are most useful if they are only active during either the swing or stance phases of locomotion. The bimodal or bifunctional character of many muscles (ie. active during both swing and stance phases or active across two joints) makes it necessary to define all muscles physiologically. Each muscle with potential value for functionally defining the step cycle was studied with electromyographic (EMG) recording techniques. The anatomical approach used by many experimenters in naming muscle groups does not always take into account the functional aspects of muscle activity. For example, the flexor cruris lateralis muscle of the leg is both a hip extensor and knee flexor (Nickel et al., 1977; Vanden Berge, 1975), yet, on the basis of anatomical examination, it is not possible to determine whether it is active during the swing phase or stance phase of walking.

As yet, no anatomical or physiological examination has been

undertaken on the musculature of the Canada Goose. Therefore, this formed the first part of this investigation into the neural control of avian locomotion.

Materials and Methods

Wild adult Canada geese were obtained under license and maintained in a large outdoor enclosure with adequate food and water.

All feathers overlying the muscle groups of interest were removed to facilitate the correct identification of each muscle and the proper placement of EMG electrodes. Initially, EMG electrodes were implanted after injection of local anaesthetic (xylocaine hydrochloride 2%) and reflection of the overlying skin. In subsequent trials, EMG electrodes were implanted percutaneously using a 22 gauge needle (Basmajian, 1962). Each electrode was made from laquer coated 28 gauge copper wire which was bared at the tip (approximately 3 mm). Two EMG electrode wires were positioned in each muscle examined (see Table 1) for differential records. A common ground electrode was implanted subcutaneously in the back. All electrodes wires were gathered together and supported by a flexible shielded cable harness sutured to the skin of the back. Local anaesthetic infiltration at all skin penetration points was used throughout each trial to minimize irritation. None of the animals showed any signs of discomfort from the recording electrodes.

To record hindlimb muscle activity during walking, each animal was placed on a treadmill enclosed by a box with a clear plexiglas front. The birds were permitted to walk unrestrained on the moving treadmill belt. Normal locomotion was defined by the animal walking with its head in a normal upright posture. This

usually occurred following a short training period (5-15 min). There were no observable differences between birds walking on a treadmill and those walking unrestrained in an open environment. EMG recordings were made at treadmill speeds up to 2.0 m/sec with an average speed being 0.4 m/sec.

Flight muscle EMGs were recorded by placing the animal in a relatively normal flying position while supporting the body and feet. Support of the feet was then removed and the bird was allowed to beat its wings in an unrestrained manner while tethered in midair.

All EMG signals were amplified and filtered prior to monitoring on a 4 channel oscilloscope and recording on magnetic tape. Permanent records were obtained on tape playback into a 4 channel strip chart recorder.

Hindlimb and forelimb anatomy was determined in both perfused (10% formalin) and unperfused sacrificed animals. Each EMG recording site was confirmed by intramuscular examination of the electrode placement. Superficial muscle groups and tendons were diagrammed, then removed to facilitate observation and documentation of inner muscle groups. To insure correct anatomical definition, the origin and insertion points for each muscle were also examined.

The observations were compared with previous studies (eg. Cracraft, 1971; George and Berger, 1966; Kaupp, 1918; Vanden Berge, 1975,1979; see Table 1). The nomenclature I have employed is that of Vanden Berge (1979) as approved by the International Committee on Avian Anatomical Nomenclature. In cases where no anatomical correlates were found in Vanden Berge (1979), a muscle

was named on the basis of a consensus from other sources.

RESULTS

Hindlimb and forelimb muscles, documented in this study, with alternate names in common use, are listed in Tables 1 and 2, respectively.

HINDLIMB:

Superficial hindlimb muscles that were examined for EMG activity included: Muscle (M.) iliotibialis cranialis (ITC), M. flexor cruris lateralis (FCL), M. flexor cruris medialis (FCM), M. caudo-ilio-femoralis (CFM), M. iliotibialis lateralis (ITL) and M. iliofibularis (IFB).

ITC (commonly named the sartorius muscle or extensor iliotibialis anterior, see Table 1), a muscle that lies anterior to the femur, attaches proximally on the anterior iliac crest and distally on the patella, (Figs. 1,2,3). Electrical activity of this muscle (Fig. 4) indicates a periodic monophasic EMG burst during the swing phase of the step cycle. Contractions of ipsilateral ITC alternate out of phase with activity in the contralateral ITC.

FCL (commonly named the semitendinosus muscle, see Table 1), forms a thin band of muscle overlying M. flexor cruris medialis (FCM). FCL arises proximally from the crista iliaca dorsalis posterior (posterior dorsal iliac crest) and inserts on the femur (Figs. 1,3). Percutaneous implantation of FCL yielded EMG recordings (Fig.4) that demonstrate prolonged activity during the

stance phase of walking. The reciprocal relationship between FCL and its contralateral homolog reveals a strict alternating, though overlapping pattern of activity at slow speeds. The temporal relationship of FCL with its ipsilateral antagonist, ITC, is also one of strict alternation. Even though ITC contraction on one side is almost simultaneous with that of the contralateral FCL, the duration of ITC activity is much shorter (Fig.4).

The following muscles of the hindlimb provided less reliable or totally equivocal EMG recordings during walking. In addition, some of the muscles were very difficult to penetrate percutaneously with EMG electrodes (eg.ITL and CFM, Figs. 1,2).

FCM (or semimembranosus) lies medial to FCL in the secondary layer of hindlimb muscles (Figs.2,5). FCM arises more medially than FCL from the ischium and inserts on the tibiotarsus. Physiologically, FCM has the same action as FCL (hip extension, weak knee flexion). Functionally, however, EMG recordings indicate that FCM is only active during the latter part of the stance phase maintaining its activity into the early stages of flexion (Fig.6). Therefore, it is not an appropriate marker to demonstrate the transition between extension and flexion phases of the step cycle.

CFM (or caudo-ilio-femoralis, piriformis, or femorocaudal, Table 1) lies medial to FCL and FCM and is partially exposed to the superficial layer (Fig.1). However, it is more properly classified as a muscle of the secondary layer (Fig.2). It arises proximally from the posterior dorsal iliac crest and distally terminates on the femur. EMG results (Fig 7) indicate that this muscle is primarily a hip extensor, active during the stance phase of the step cycle. It is possible that CFM was included

periodically in results from FCL; however, since both CFM and FCL are hip extensor muscles active during the same phase of the step cycle, it should not significantly distort FCL recordings during walking. Surgically, CFM was a difficult muscle to implant without reflection of the overlying dermis and was therefore abandoned as a useful marker for the stance phase of locomotion.

ITL (iliotibialis or gluteus primus, Table 1), is a large muscle that attaches proximally to the dorsal iliac crest and distally to the patella (Fig.1). Its period of activity is simultaneous, but of shorter duration than that of ITC. Although easily accessible however, in the goose, ITL produced equivocal EMGs during locomotion (ie. active throughout all phases of the step cycle, with major activity corresponding to that of ITC) (Fig.8). Therefore, it was not effective as an indicator of the alternating phases of stepping.

IFB (biceps femoris, Table 1) lies posterior to the femur (Figs. 1 and 2). It attaches proximally to the dorsal iliac crest, loops its tendon through the Ansa M. iliofibularis (Fig.5) and inserts on the fibula (Fig.3). In the goose it appears to be active during both stance and swing phases of hindlimb stepping (Fig.8).

FORELIMB:

Muscles of the forelimb (wing) that were implanted for EMGs included: M. pectoralis, M. deltoideus major (DM), M. latissimus dorsi, pars cranialis (LDCr) and pars caudalis (LDCa), and M.

scapulotriceps (TSC). The depressor and elevator phases of the flight cycle are best illustrated by the pectoralis and deltoideus musceles respectively (Fig. 9). LDCa, LDCr and TSC were either difficult to access by percutaneous implantation of EMG electrodes or were continuously active throughout the entire flight cycle.

Pectoralis is anatomically the largest muscle found in the goose (Fig.10). This muscle arises from the sternum and inserts on the humerus. It is the primary depressor of the wing, as indicated by EMG recordings during tethered (Fig.10) and unrestrained flight. (Butler et al., 1977). Tethered flight in the preparations of this study appeared to be comparable to actual flight, at least with regards to the activity pattern of pectoralis.

Deltoideus major arises from several bones of the shoulder joint, is anchored by means of the retinaculum and inserts ventrally midway along the humerus (Fig.10). Recordings only show activity during the elevation phase of the flight cycle (Fig.9)

LDCr arises from the neural spines of the most caudal cervical and most rostral thoracic vertebrae and inserts on the humerus (Fig.10). LDCa arises from the dorsal iliac crest caudal to and separated from LDCr. The point of insertion of LDCa is similar to that of LDCr. Both LDCa and LDCr were found to be physiologically weak elevators of the wing, active throughout the elevator phase of the flight cycle (Fig.11). However, it is difficult to distinguish LDCa and LDCr from the rhomboideus superficialis muscle on the basis of topographical criteria. This makes it very difficult to accurately implant percutaneously with EMG electrodes.

TSC (or triceps scapularis, Table 1) arises from the proximal

humerus, passes caudally along the humerus and inserts onto the ulna (Fig.10). Electromyographic results (Fig.11) show tonic muscle activity during wing extension, demonstrating that TSC is active throughout the entire flight cycle.

DISCUSSION

The characterization of muscles defining normal hindlimb and forelimb locomotor behaviours in the goose arose as a necessary prerequisite to determining the locomotor capabilities of chronic spinal cord lesioned and acute decerebrate animals. Due to the lack of information regarding the musculature of the Canada goose, it was necessary to anatomically characterize the musculature to allow topographical implantation of EMG electrodes which would define the step cycle flexor and extensor muscles necessary to locomotion. It was also necessary to physiologically characterize the musculature as to its efficacy in defining the avian step and flight cycles, for an anatomically defined muscle does not necessarily reflect its physiological function.

Hindlimb and forelimb muscles defining rhythmic locomotor patterns need to meet several criteria. These include: 1) the trauma to the animal must be minimized during electrode implantation; 2) the muscles must be essential to the production of normal locomotion; and 3) the muscle must produce an EMG trace that is clearly distinguishable, phasic, and reliably replicable.

The hindlimb muscles which met the above criteria were the iliotibialis cranialis (ITC) and the flexor cruris lateralis (FCL). ITC is a physiological flexor which is active during the majority of the swing phase of walking. It is located slightly anterior to the femur and is the easiest hindlimb muscle to implant percutaneously (Figs.1,2,3). Results show a periodic

monophasic burst of activity from this muscle during both swing extension and swing flexion (Fig.4) which are in agreement with previous findings of Jacobson and Hollyday (1982a) for the chick. EMG results from FCL indicate that it is active during the extension phase of the step cycle despite its anatomical nomenclature (Fig.4). A small component of this muscle's activity begins in the early swing extension phase of the step cycle. However, it defines the end of the extension phase of stepping precisely and as such is a good indicator of this portion of the cycle. This finding is also in agreement with that found for the chick (Jacobson and Hollyday, 1982a).

The possibility exists that FCM and CFM were implanted percutaneously along with FCL, as FCL form only a thin flat layer directly apposing the skin. The actions of FCM and CFM are similar to that of the overlying muscle (FCL) and therefore would not significantly alter EMG recordings from FCL. These muscles were not, however, easily penetrated with EMG electrodes, and as such could not be reliably used to define avian walking. Regardless, care was taken to implant only FCL by introduction of the recording electrodes along a flat plane parallel to the overlying skin. This precaution provided reliable EMG results identifying the entire extension phase of the step cycle.

Jacobson and Hollyday (1982a) describe ITL as a physiological flexor of the hindlimb and IFB as a biphasic muscle with activity during the stance phase of locomotion. However, in the goose, these two muscles did not prove to be usable indicators of locomotion, as they were active during both phases of the step cycle.

The forelimb musculature which provided the most reliable EMGs defining the flight cycle were the pectoralis and deltoideus major muscles. George and Berger (1966) used an anatomical basis for designation of the muscles controlling the elevator and depressor phases of the flight cycle. They determined that pectoralis and supracoracoideus were the major muscles responsible for the elevator and depressor phases, respectively. The results of this study indicate that the pectoralis muscle is the best muscle for defining the depressor phase of locomotion. However, the supracoracoideus muscle, which lies medial to the pectoralis muscle was not tested in this study, since it is very difficult to accurately percutaneously implant with electrodes. Deltoideus major, however, produces EMGs which alternate out of phase with pectoralis and is therefore considered to be a good indicator of the elevator phase of flight (Fig.9). LDCa, LDCr, and TSC were either difficult to implant percutaneouly or were tonically active throughout wing extension. Therefore they could not be utilized as indicators of the avian flight cycle.

The muscles which best exemplify the hindlimb locomotion and flying locomotion in the Canada goose for the purposes of future experiments have been identified as being ITC/FCL and Pect/DM respectively. In an attempt to clarify the avian muscle nomenclature, a list of synonyms is provided for the limb musculature in Tables I and II.

TABLE I

Nomenclature for hindlimb muscles in the Canada goose

ABBREVIATION	NAME & (SYNONYMS)	FUNCTION
caudofem. (CFM)	-M. caudo-ilio-femoralis (D- caudofemoralis) (I- piriformis) (K- femorocaudal)	L- hip extensor, tail depressor *J- hip extensor, knee flexor (weak)* *Q- hip extensor*
ext. dig. comm.	-M. extensor digitorum communis	L,P- digit extensor
fem. tib. ext.	-M. femorotibialis externus (D,G,H- vastus lateralis) (F,I,R- femoritibialis externus) (L- quadriceps femoris) (M- vastus externus component of M. extensor femoris)	L,P- knee extensor
fem. tib. med.	-M. femorotibialis medius (D,H- vastus medialis) (L- quadriceps femoris) (N- cruraeus component of extensor femoris)	L,P- knee extensor *J- knee extensor*
fib. brev.	-M. fibularis (peroneus) brevis	L- internal rotator tarsometatarsus (ankle) P- internal ankle rotation, ankle flexor
fib. long	-M. fibularis (peroneus) longus	P- ankle extensor *J- ankle extensor*

flex. cru. lat. (FCL)	-M. flexor cruris lateralis (I,K- semitendinosus) (G- caudilioflexorius)	L,P- hip extensor, knee flexor *J,Q- hip extensor knee flexor (weak)*
flex. cru. med. (FCM)	-M. flexor cruris medialis (I,K- semimembranosus) (F,J- ischioflexorius)	P- hip extensor, knee flexor *J,Q- hip extensor, knee flexor(weak)*
flex. p. dig. IV	-M. flexor perforans digiti IV (L- flexor perforatus dig. IV)	P- extension of tarsometatarsal joint (ankle) L,P- toe flexor
flex. p. dig. III	-M. flexor perforans digiti III (L- flexor perforatus dig. III)	L,P- toe flexor P- ankle extensor
flex. p. et p. dig. III	-M. flexor perforans et perforatus digitii III (K- flexor perforatus medius secundus pedis)	L.P- toe flexor P- ankle extensor
flex. p. et p. dig. II	-M. flexor perforans et perforatus digitii II (K- flexor perforatus indicis secundus pedis)	L,P- toe flexor P- ankle extensor
gastroc. lat.	-M. gastrocnemius pars lateralis (externa)	L,P- ankle extensor *J- ankle extensor*
gastroc. med.	-M. gastrocnemius pars medialis (interna)	L,P- ankle extensor *J- ankle extensor*
iliofib. (IFB)	-M. iliofibularis (I,F,G- biceps femoris or biceps femoralis) (D,H- extensor ilio-fibularis) (K,N- biceps flexor cruris)	L- hip extensor, knee flexor, leg abductor P- knee flexor *J,Q- knee flexor hip extensor*

iliotib. cran. (ITC)	-M. iliotibialis cranialis (I,J,K- sartorius) (D,J- extensor ilio-tibialis anterior)	L- hip flexor P- hip flexor, knee extensor *J,Q- hip flexor, knee extensor*
iliotib. lat. (ITL)	-M. iliotibialis lateralis (K,N- gluteus primus) (G- iliotibialis) (F,J- i) iliotibialis anterior ii) iliotibialis medius or tensor fasciae iii) iliotibialis posterior or gluteus posterior)	L- hip extensor P- hip extensor or hip flexor, hip abductor *J- iliotib. ant. = hip flexor iliotib. post. = hip extensor, knee extensor, external rotator of hip.*
iliotroch. caud.	-M. iliotrochantericus caudalis (D- gluteus profundus)	L,P- hip extensor *J- internal rotator of hip*
iliotroch. cran.	-M. iliotrochantericus cranialis (D- iliacus)	L- hip flexor P- internal rotator of hip *J- hip flexor, internal rotator of hip*
ischiofem.	-M. ischiofemoralis (D- flexor ischiofem-oralis externus) (N- obturator externus)	L,P- external hip rotator, hip extensor *J- external rotator of hip*
lev. caud.	-M. levator caudae (H,M)	P- extends and raises tail
pubo-ischio-fem.	-M. pubo-ischio-femoralis (C,D- adductor longus et brevis) (L- adductor femoris)	L,P- hip extensor, hip adductor *J- hip extensor*

tib. cran.	-M. tibialis cranialis	L,P- ankle flexor *J- ankle flexor*

Note: Italics denotes function identified on a physiological basis.

Source:

A) Vanden Berge (1979)- All names given except where designated.
B) Butler et al. (1977)
C) Cracaft (1971)
D) Fisher (1946) and Fisher and Goodman (1955)
E) Fujioka (1959)
F) Gadow and Selenka (1891)
G) George and Berger (1966)
H) Howell (1938)
I) Hudson (1937)
J) Jacobson and Hollyday (1982)
K) Kaupp (1918)
L) Nickel et al. (1977)
M) Romer (1927)
N) Shufeldt (1890)
O) Sullivan (1962)
P) Vanden Berge (1975)
Q) Weinstein et al., present study (see text)
R) Wilcox (1952)

TABLE II

Nomenclature for forelimb muscles in the Canada goose

ABBREVIATION	NAME & (SYNONYMS)	FUNCTION
add. alu.	-Muscle (M.) adductor alulae (P- adductor pollicis) (P- adductor alae digiti II)	L,P- digit adductor
bic.	-M. biceps brachii	L,P- elbow flexor P- shoulder extensor
delt. maj.	-M. deltoideus major	L- shoulder flexor P- wing elevator, wing flexor *Q- wing elevator*
delt. min.	-M. deltoideus minor	L,P- wing elevator, wing adductor
ectepi.	-M. ectepicondylo-ulnaris (G- anconeus)	L- elbow extensor P- elbow flexor, wing supinator
ext. long. dig. maj.	-M. extensor longus digiti majoris (D- extensor longus digiti III) (G- extensor indicis longus) (F,I- extensor medius longus) (D,G- extensor indicis longus and M. flexor metacarpi brevis)	L- digit extensor
ext. meta. rad.	-M. extensor metacarpi radialis (L- extensor carpi radialis)	L,P- extension of carpelmeta-carpal joints (wrist) P- elbow flexor

ext. meta. uln.	-M. extensor metacarpi ulnaris (L- extensor carpi ulnaris)	L- wrist extensor P- wrist flexor, elbow flexor
flex. dig. min.	-M. flexor digiti minoris (D,F- flexor digiti III or IV) (P- flexor digiti quarti)	P- metacarpal flexor
inteross. dor.	-M. interosseus dorsalis	L,P- extension of second digit
inteross. ventr.	-M. interosseus ventralis (D,F,G,L- interosseus palmaris) (O- interosseus volaris)	L,P- flexion of second digit
lat. dors. caud. (LDCa)	-M. latissimus dorsi pars caudalis	L,P- draws wing caudally, wing elevator and flexor *Q- wing elevator (weak)*
lat. dors. cran. (LDCr)	-M. latissimus dorsi pars cranialis	L,P- draws wing caudally, wing elevator and flexor *Q- wing elevator (weak) during wing extension*
pect.	-M. pectoralis	G,P- wing depressor *B,Q- wing depressor*
pect. propatag.	-M. pectoralis pars propatagialis	P- wing depressor
rhom. prof.	-M. rhomboideus profundus	L- elevate scapula P- stabilizes scapula

rhom. superf.	-M. rhomboideus superficialis (E- trapezius)	L- respiratory expiration P- stabilizes the scapula
scap. hum. caud.	-M. scapulohumeralis caudalis (G- proscapulohumeralis)	L- wing elevator P- stabilization of humerus during wing beat
serr. superf. caud.	-M. serratus superficialis pars caudalis (G- serratus posterior) (N- thoraco-scapularis)	L- wing depressor, respiratory expiration P- stabilizes scapula, respiration
serr. superf. metapatag.	-M. serratus superficialis pars metapatagialis	L- stretches the patagium P- stabilizes the scapula
sup.	-M. supinator	L,P- supination of the wing
T. of flex. dig. pro.	-Tendon of flexor digitorum profundus	
tens. propatag.	-M. tensor propatagialis (G- tensor patagii longus) (F,I- propatagialis longus)	L- wing adductor P- digit extensor, forearm flexor
tri. scap. (TSC)	-M. scapulotriceps (G- triceps scapularis or anconaeus scapularis or anconaeus longus) (L- anconaeus) (P- triceps brachii pars scapularis)	L- elbow extensor P- elbow extensor, shoulder flexor

Note: Italics denotes function identified on a physiological basis.
Source: as listed for Table I.

FIGURE 1

Lateral view of the superficial hindlimb musculature. Abbreviations: T= tendon, for muscles see Table 1.

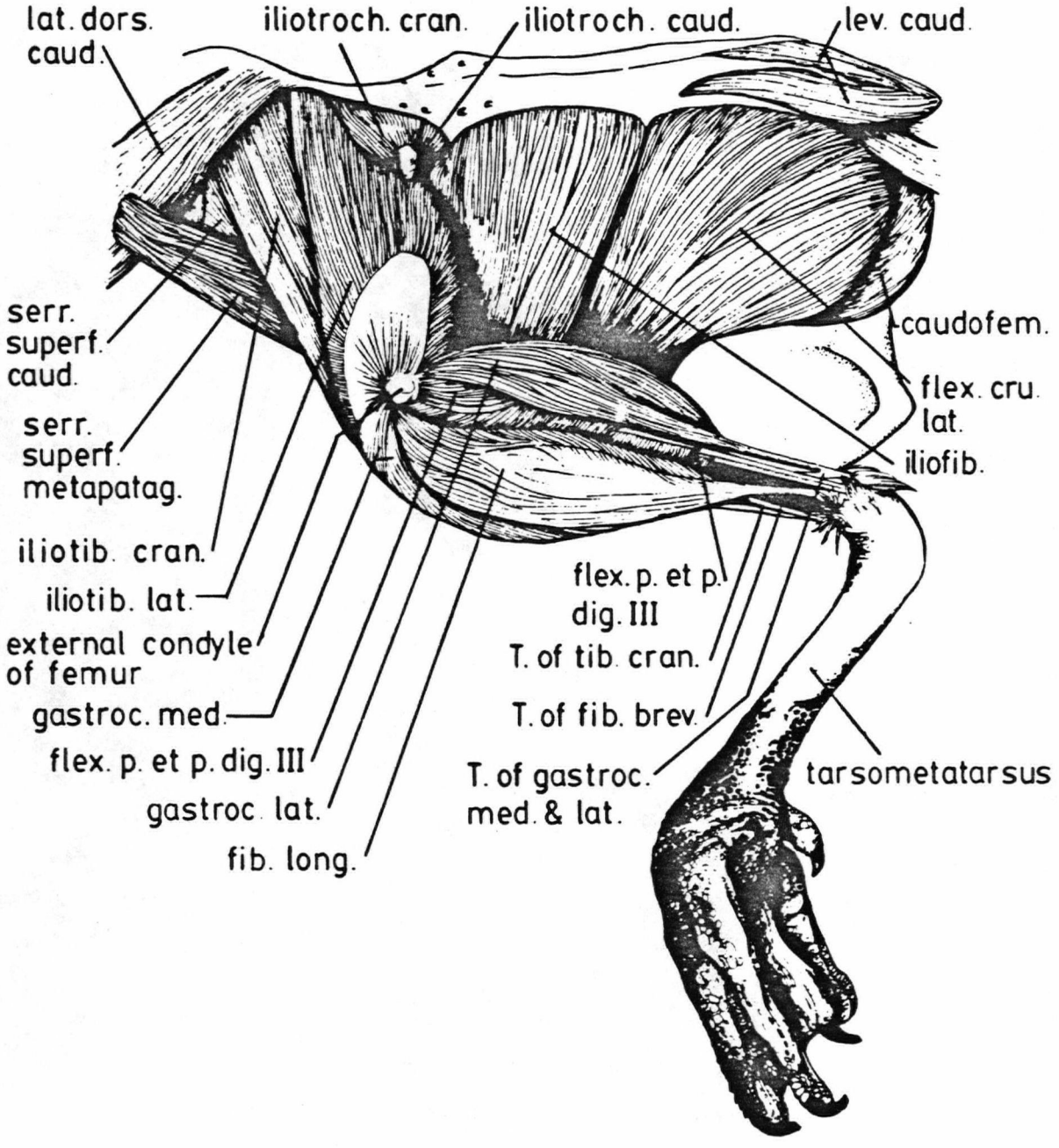

lat. dors. caud.
iliotroch. cran.
iliotroch. caud.
lev. caud.
serr. superf. caud.
serr. superf. metapatag.
iliotib. cran.
iliotib. lat.
external condyle of femur
gastroc. med.
flex. p. et p. dig. III
gastroc. lat.
fib. long.
caudofem.
flex. cru. lat.
iliofib.
flex. p. et p. dig. III
T. of tib. cran.
T. of fib. brev.
T. of gastroc. med. & lat.
tarsometatarsus

FIGURE 2

Lateral view of the second layer of hindlimb muscles underlying the superficial layer. To show the second layer, the following superficial muscles have been removed: fib. long., flex. cru. lat., gastroc. lat., iliotib. lat. and lat. dors. caud. Abbreviations: T= tendon, for muscles see Table 1.

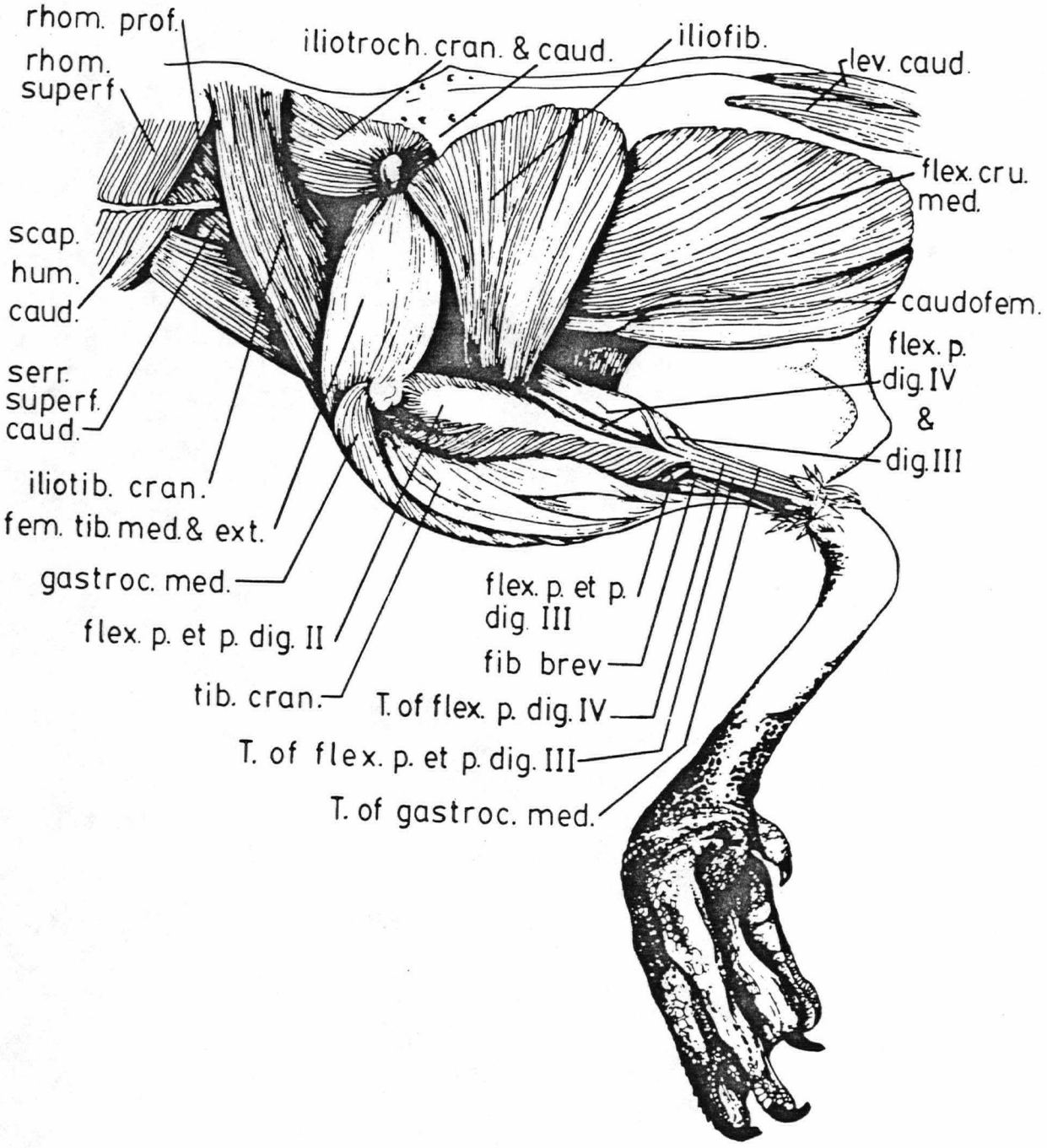

rhom. prof.
rhom. superf
iliotroch. cran. & caud.
iliofib.
lev. caud.
flex. cru. med.
scap.
hum.
caud.
caudofem.
flex. p. dig. IV
&
dig. III
serr. superf. caud.
iliotib. cran.
fem. tib. med. & ext.
gastroc. med.
flex. p. et p. dig. II
tib. cran.
flex. p. et p. dig. III
fib brev
T. of flex. p. dig. IV
T. of flex. p. et p. dig. III
T. of gastroc. med.

FIGURE 3

Dorsal view of hindlimb musculature. To reveal the second layer of muscle, the tendon of gastroc. lat. has been reflected. Abbreviations: T= tendon, R= rostral, C= caudal, for muscles see Table 1.

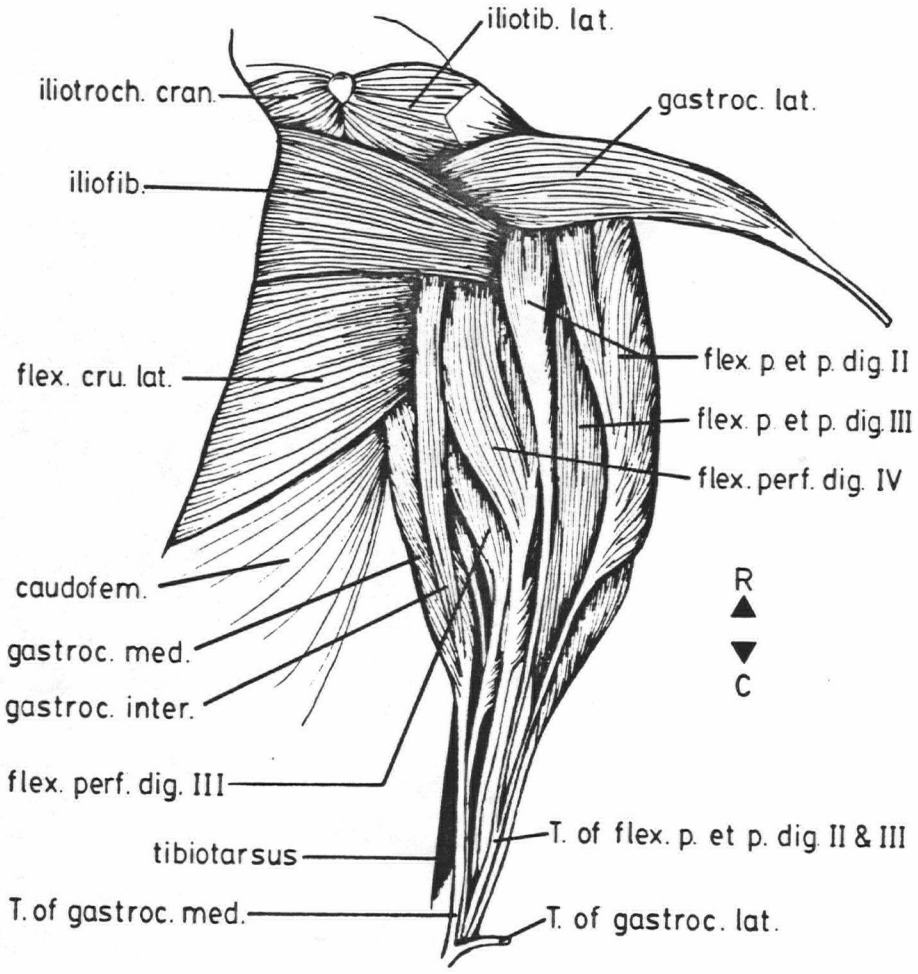

iliotib. lat.
iliotroch. cran.
gastroc. lat.
iliofib.
flex. cru. lat.
flex. p. et p. dig. II
flex. p. et p. dig. III
flex. perf. dig. IV
caudofem.
gastroc. med.
gastroc. inter.
flex. perf. dig. III
tibiotarsus
T. of gastroc. med.
T. of flex. p. et p. dig. II & III
T. of gastroc. lat.
R
C

FIGURE 4

Electromyographic (EMG) records from left and right hindlimb ITC and FCL muscles during treadmill walking (belt speed= 0.3m/sec). ITC is a flexor active throughout the stance phases (ie. foot contact with the substrate) are indicated by the bars beneath the EMG recordings. Abbreviations: ITC= iliotibialis cranialis, FCL= flexor cruris lateralis.

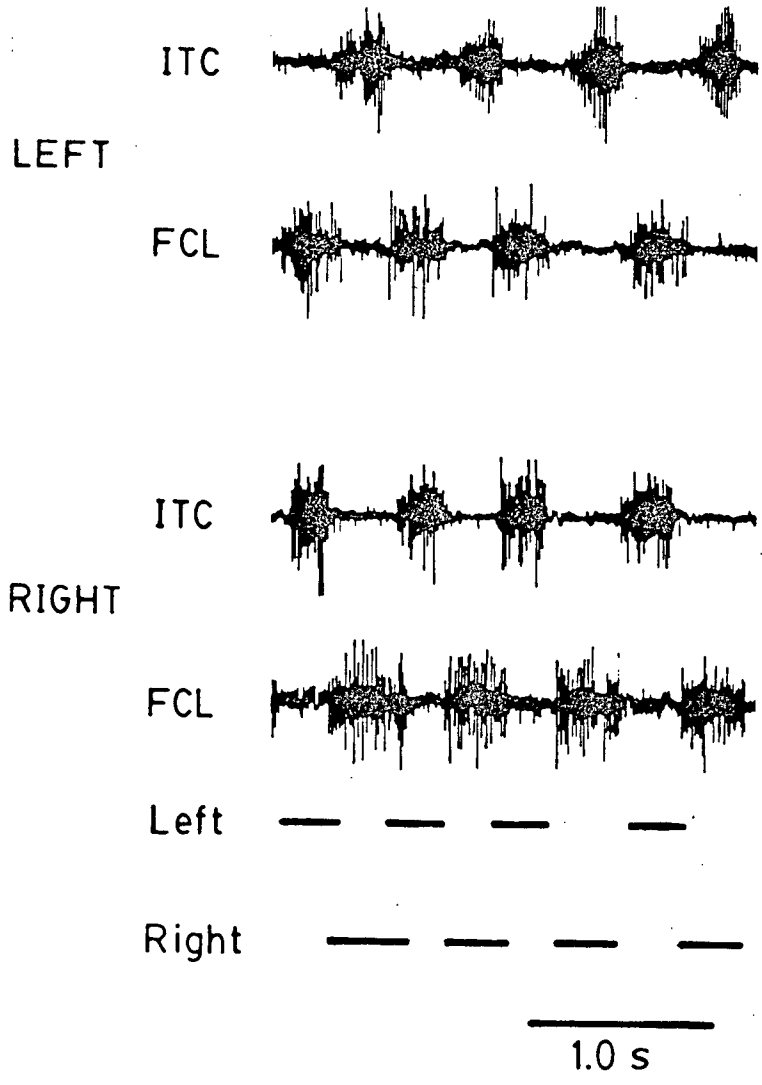

ITC
LEFT
FCL
ITC
RIGHT
FCL
Left
Right
1.0 s

FIGURE 5

Lateral view of the third layer of hindlimb musculature underlying the second layer. Reflection of the primary layer of the flex. cru. med., in addition to cutting the flex. cru. lat., and iliofib., reveals pubo-ishio-fem. and ischiofem.. Flex. p. et p. dig. II has been removed to reveal flex. p. et p. dig. III and the ansa (loop-like structure) iliofib.. Abbreviations: T= tendon, for muscles see Table 1.

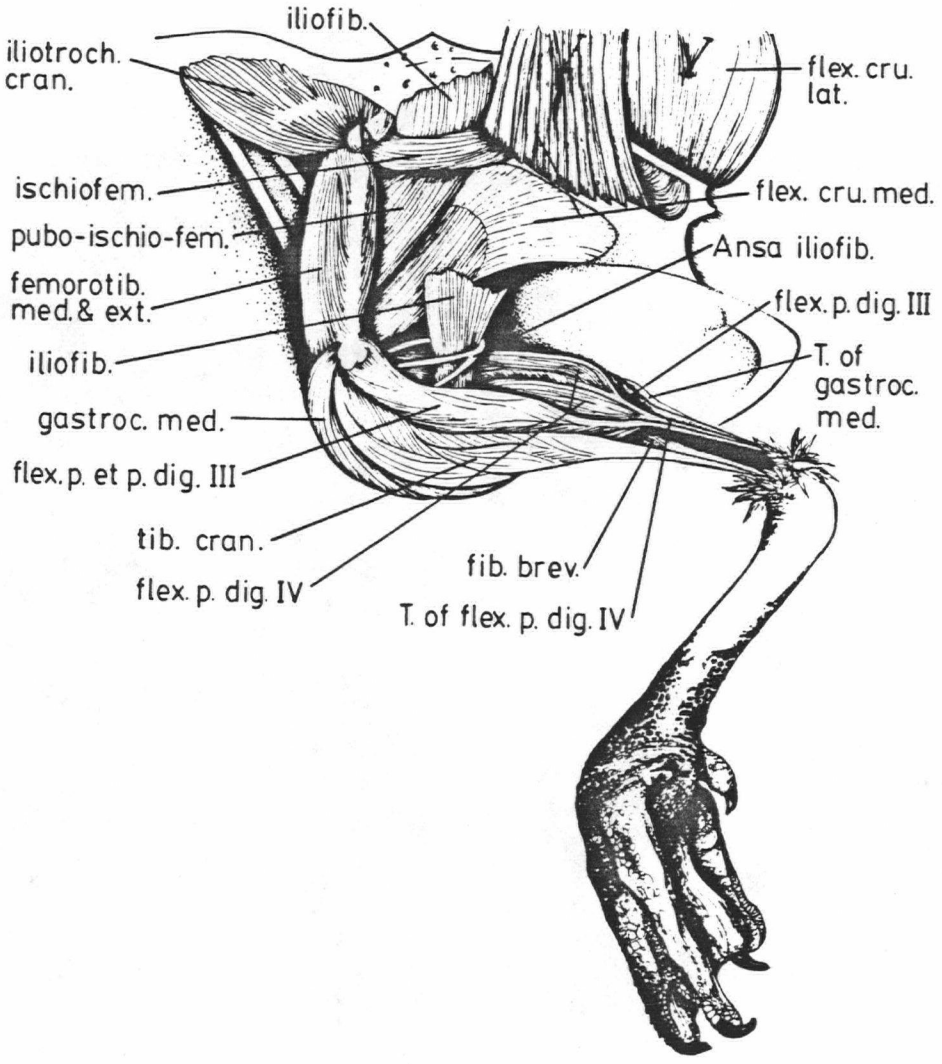

iliofib.
iliotroch. cran.
flex. cru. lat.
ischiofem.
flex. cru. med.
pubo-ischio-fem.
Ansa iliofib.
femorotib. med. & ext.
flex. p. dig. III
iliofib.
T. of gastroc. med.
gastroc. med.
flex. p. et p. dig. III
tib. cran.
fib. brev.
flex. p. dig. IV
T. of flex. p. dig. IV

FIGURE 6

Electromyographic recording of the hindlimb ITC and FCM during treadmill walking (belt speed= 0.4 m/sec). ITC is a flexor active throughout the swing phase. FCM is a hip extensor and knee flexor (weak) active during the stance phase with a component overlapping the swing phase during walking. Abbreviations: ITC= iliotibialis cranialis, FCM= flexor cruris medialis.

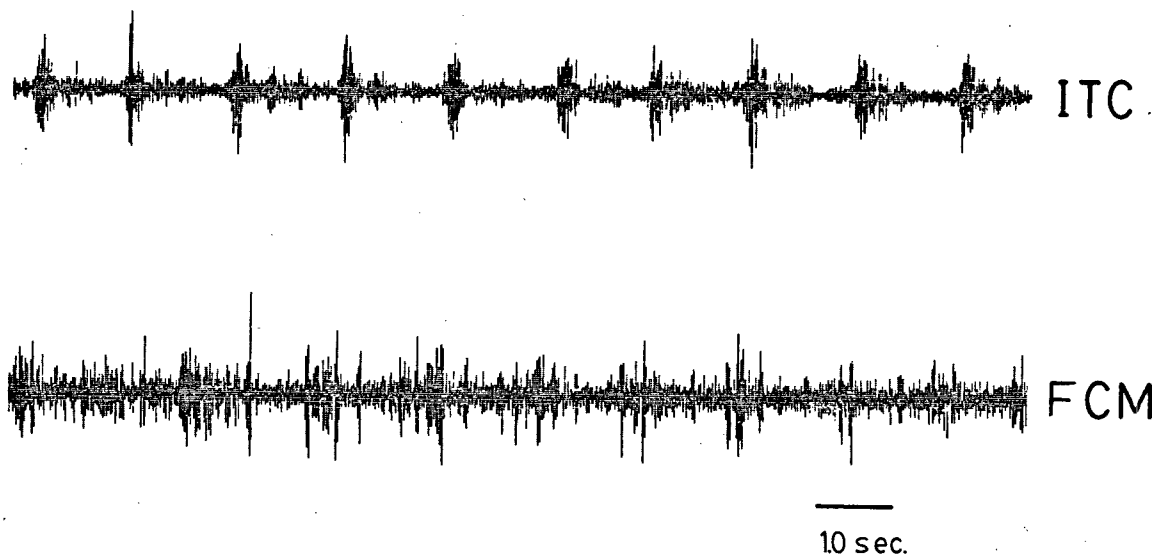

ITC
FCM
1.0 sec.

FIGURE 7

Electromyographic recordings of the hindlimb ITC and CFM muscles during treadmill walking. ITC is a flexor active throughout the swing phase. CFM is an hip extensor and knee flexor (weak) active throughout the stance phase of walking. Abbreviations: ITC= iliotibialis cranialis, CFM= caudo-ilio-femoralis.

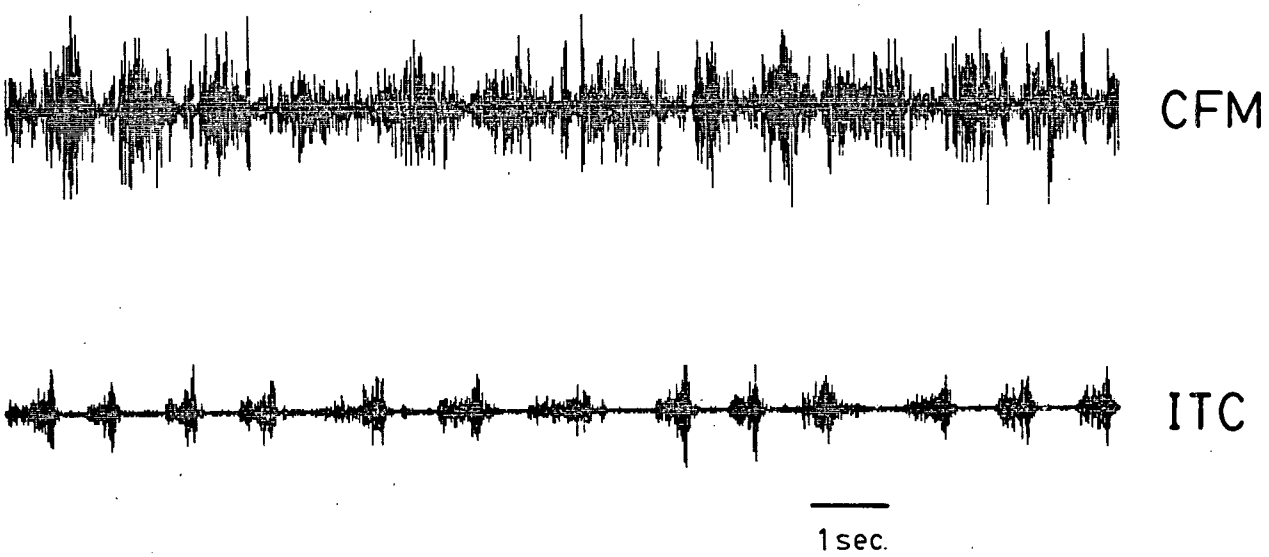

CFM
ITC
1 sec.

FIGURE 8

Electromyographic records from left and right ITC and left IFB muscles of the hindlimb during treadmill walking (belt speed= 0.3m/sec). ITC is a flexor active throughout the entire swing phase. IFB is active both during the stance phase and swing phase of walking. Abbreviations: ITC= iliotibialis cranialis, IFB= iliofibularis.

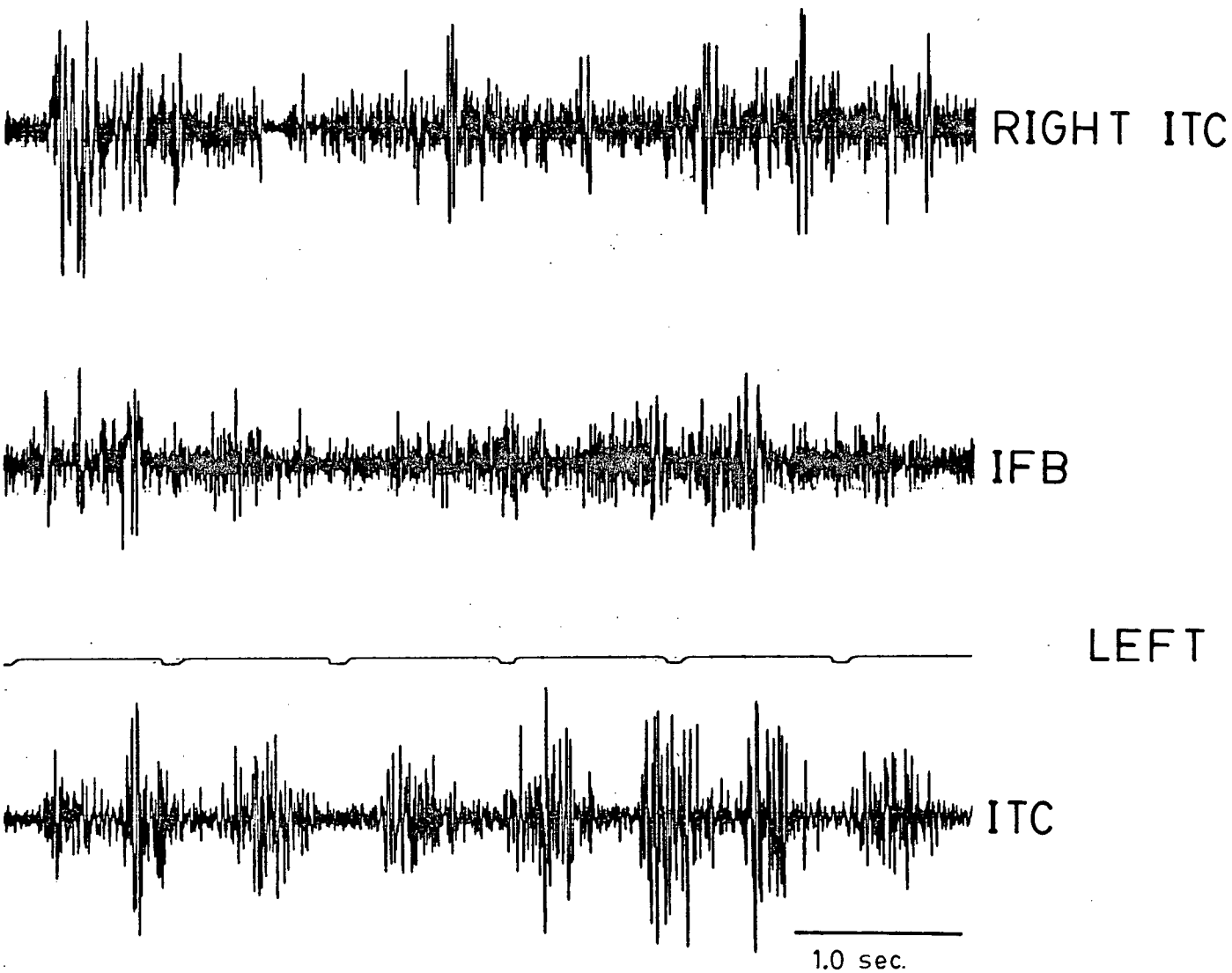

RIGHT ITC
IFB
LEFT
ITC
1.0 sec.

FIGURE 9

Electromyographic recordings of the forelimb Pect and DM muscles during tethered flapping of the wings. Pect is a wing depressor and DM is a wing elevator. Abbreviations: Pect= Pectoralis, DM= deltoideus major.

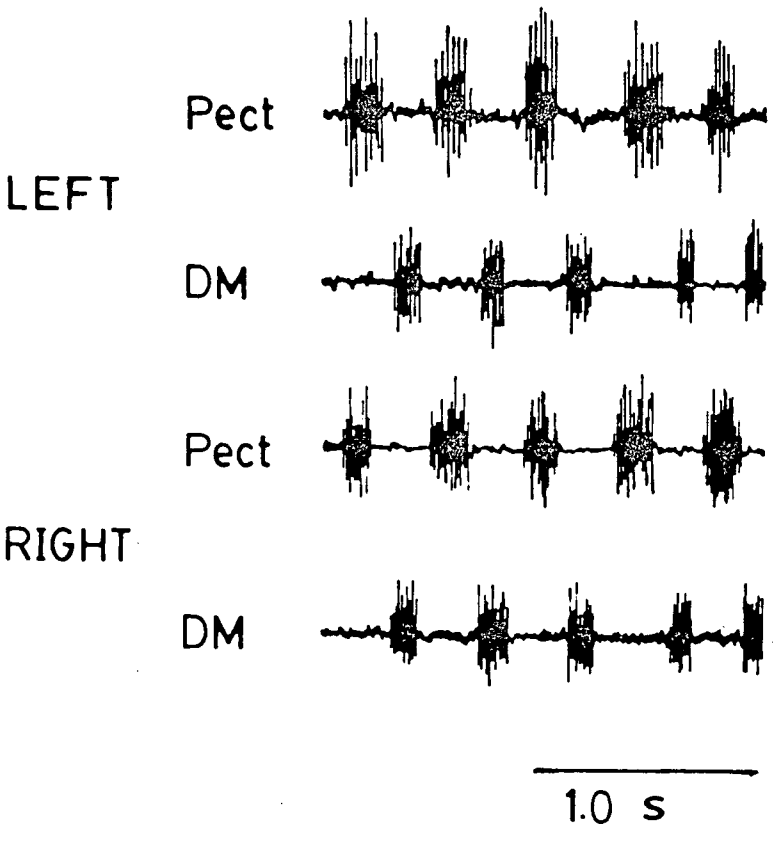

Pect
LEFT
DM
Pect
RIGHT
DM
1.0 s

FIGURE 10

Lateral view of the wing and associated axial trunk musculature. Abbreviations: T= tendon, for muscles see Table 2.

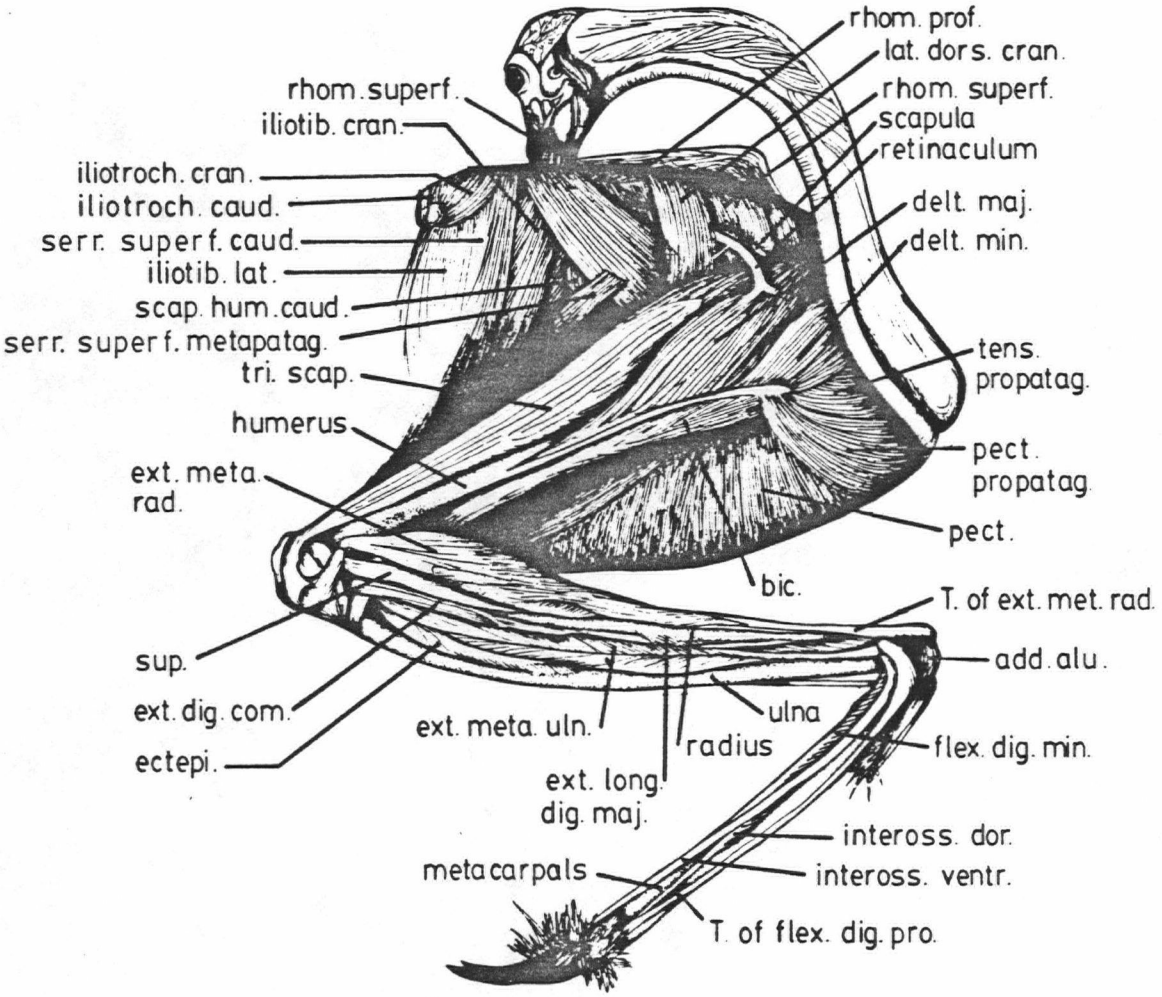
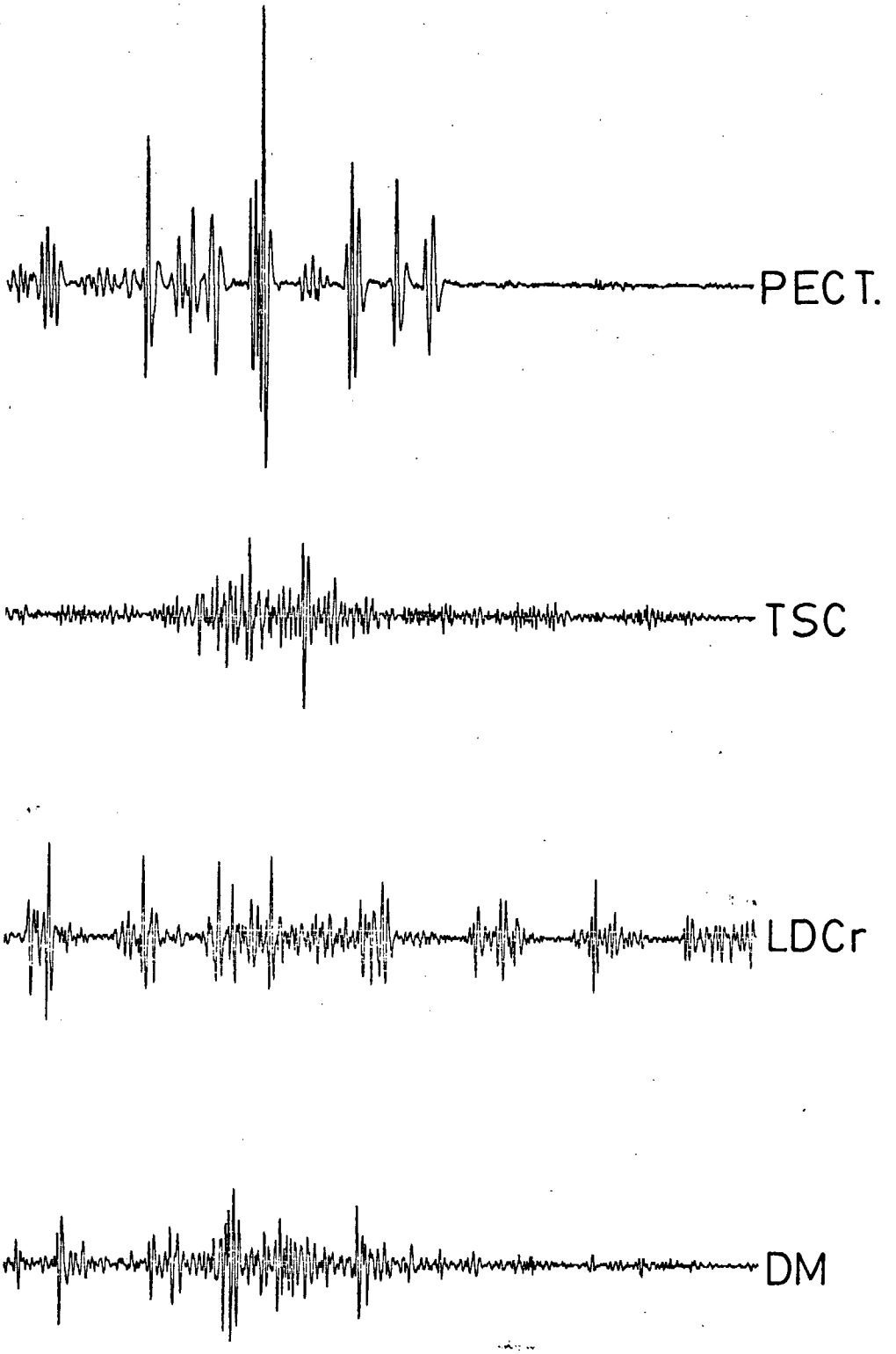

rhom. prof.
lat. dors. cran.
rhom. superf.
scapula
retinaculum
delt. maj.
delt. min.
tens. propatag.
pect. propatag.
pect.
bic.
T. of ext. met. rad.
add. alu.
flex. dig. min.
inteross. dor.
inteross. ventr.
T. of flex. dig. pro.
rhom. superf.
iliotib. cran.
iliotroch. cran.
iliotroch. caud.
serr. superf. caud.
iliotib. lat.
scap. hum. caud.
serr. superf. metapatag.
tri. scap.
humerus
ext. meta. rad.
sup.
ext. dig. com.
ectepi.
ext. meta. uln.
ulna
radius
ext. long. dig. maj.
metacarpals

FIGURE 11

Electromyographic records from the left forelimb LDCr, DM, TSC, and Pect muscles. Pect is active during the depressor phase of wing flapping. LDCr is active during the elevator phase of wing movement coincident with DM. DM alternates out of phase with Pect.. TSC shows tonic activity during wing extension and is active throughout the entire flight cycle. Abbreviations: DM= deltoideus major, LDCa= latissimus dorsi, pars caudalis, TSC= scapulotriceps.


PECT.
TSC
LDCr
DM

CHAPTER II

CHARACTERIZATION OF DESCENDING SPINAL CORD PATHWAYS NECESSARY FOR LOCOMOTION IN THE HINDLIMBS OF THE CANADA GOOSE, *Branta canadensis*

INTRODUCTION

Motor control is thought to be the result of descending input from centres in the hind, mid and forebrain. The pathways carrying this information travel in the spinal cord white matter before synapsing at the appropriate spinal level where they activate locomotor pattern generators (LPGs) (Grillner, 1975). Selective lesioning of descending spinal cord pathways at both cervical and thoracic levels has yielded information regarding the spinal trajectories of these locomotor pathways and provided valuable clues as to the supraspinal areas which effect the control of locomotion (Afelt, 1974; Steeves and Jordan, 1980; Yu and Eidelberg, 1981; Williams et al., 1984).

The literature suggests that descending reticulospinal pathways play an important role in the initiation and control of walking in cats and monkeys. The maintenance of both ventral quadrants of the spinal cord which have been shown to contain descending reticulospinal pathways (Kuypers, 1982), allows bilateral walking in acutely stimulated (MLR) mesencephalic cats (Steeves and Jordan, 1980), non-decerebrate chronic lesioned cats (Afelt, 1974; Eidelberg et al., 1981c) and non-decerebrate chronic lesioned monkeys (Eidelberg et al., 1981a).

The selective chronic lesioning experiments in this study with the the goose extend the work which has been done in mammals by delimiting the pathways in the avian spinal cord effecting descending control. A comparison between avian and mammalian studies may determine whether phylogenetic uniformity exists

between these groups, thereby allowing one to correlate the results from the avian central nervous system (CNS) with those of the mammalian system. It is also a necessary prerequisite in determining one or more of the essential supraspinal centres which control hindlimb locomotion in the avian nervous system. Finally, it is prerequisite to distinguishing any differences which may exist between supraspinal pathways controlling walking from those influencing flying (Jacobson and Hollyday, 1982).

MATERIALS AND METHODS

Adult Canada Geese from an outdoor enclosure were placed in a preoperative/postoperative holding room at least one day before electromyographic (EMG) characterization of locomotion as described in Chapter I. Food and water were supplied ad libidum. No more than three animals were placed in the pen at any one time. Following preoperative characterization of locomotion, the experimental animal was deprived of food for twenty four hours prior to surgery. This reduced or eliminated operative and postoperative regurgitation of food, thereby reducing the risk of asphyxiation.

The surgery was performed under Pentobarbitol (Somnitol) anaesthesia which was injected via a cannula (PE100) inserted into the ulnar or basilica veins of either wing. A basal dose of 20 mg/kg was injected in three aliquots with phosphate buffered saline (PBS) (pH 7.4) administered between each aliquot. Additional pentobarbitol was injected as needed. Total dosage depended on the individual animal and varied from approximately 20 mg/kg to 45 mg/kg. Xylocaine hydrochloride (2%) was utilized as a local anaesthetic and injected liberally surrounding all incisions. The criteria used for depth of anaesthesia were the eye nictitating membrane reflex response to touch (Fedde,1978) (it slows down as the level of anaesthesia increases) and the reflex withdrawal to foot web pinch (it is eliminated when the animal is anaesthetized). Ventilation was unnecessary as a sufficiently deep level of anaesthesia could be attained without a cessation of

breathing.

A laminectomy was performed at the thoracolumbar level through the fused vertebrae of the Crista iliaca dorsalis (dorsal iliac crest) taking care to remove and store the crest bone as a single unit. Trabeculae which join the crest and spinal cord bony covering were removed as necessary to allow exposure of the cord underlying the thoracolumbar vertebrae. The cord was exposed by removing the final thin layer of bone which covers it. As much bone as possible was left intact to limit the amount of postoperative movement-induced injury to the cord. Lesions were made with either fine dissecting micro-scissors, scalpel or modified dental instruments with the aid of a stereo dissecting microscope. Ventromedial lesions were produced by inserting a modified dental tool through the midline dorsoventrally. Lesion extent at the time of surgery was determined by visual observation. The cavity surrounding the lesion was continuous with the air sacs. To prevent blood from flowing into the air sacs and lesion site, the cavity was filled with sterile gauze impregnated with physiological saline. The iliac crest bone was replaced in its original position in an attempt to restabilize the iliac crest.

The wing cannula was removed and the vein tied off with #000 silk. The skin was then sutured. Post-mortem examination of the wound site revealed: 1) functional recovery of the sutured musculature; 2) adhesion of the replaced iliac crest bone to the surrounding crest; and 3) in most cases complete absence of scar tissue in the overlying skin.

Postoperative care required that the animal be kept warm

(infrared heat lamp) until such time as it was capable of maintaining its head in an upright position thus indicating recovery from anaesthesia (mean time of recovery from anaethesia was 3.5 hours). Each animal received Meperidine Hydrochloride (Demerol) analgaesic intramuscularly (1.8 mg/kg) every six hours for a period of two to three days, depending on its state of arousal. Ampicillin Sodium (Penbritin-250) antibiotic (250 mg injectable in 2ml. PBS) was administered intramuscularly daily for seven days.

The recovery period depended on the severity of the lesion and each animal was assessed daily as to its ability to stand, walk, respond to reflex inputs, drink, and feed. Animals which did not eat within three days postoperatively were force fed with liquid food (Buckerfields 16% Layer Pellets in water) twice daily until self-feeding resumed. This amount was sufficient to maintain the body mass for up to 45 days (the animals weight was monitored on a regular basis). Animals capable of walking usually began eating quite quickly after surgery.

Environmental conditions were observed to have importance to the return of locomotor function in lesioned animals. Maintenance of an animal in an isolated area appeared to be detrimental to return of function even though, in some cases, the lesion severity was minimal. Conditions conducive to return of eating and locomotion occurred when the animal was placed in the same area with another lesioned bird or in the same room, but separated from, an intact animal. Placement of an operate in the same enclosure with an intact animal produced detrimental effects on

locomotor capability in the operate perhaps due to aggression on the part of the intact animal. Restoration of self-feeding correlated well with a return of locomotor capability following the lesion. Animals force fed following a maximum of three postoperative days recovered function more quickly than animals which were not force fed for a lengthier postoperative period. Therefore, all animals lesioned subsequent to these findings were force fed within three days after the surgery until such time as self-feeding was apparent.

Each postoperative animal was held thirty days or longer (maximum 49 days) to allow for maximal recovery of function. The stabilization of locomotor ability was determined by visual observation, testing of reflexes, and assessment of muscle tonus.

Postoperative EMG recordings were undertaken utilizing the same procedures as for the preoperative EMG recordings. In cases where the animal was incapable of self-supporting locomotion on the treadmill apparatus, EMGs of hindlimb stepping were recorded by supporting it over the treadmill belt so that the feet rested on the moving belt. Animals incapable of any voluntary hindlimb movement were tested for muscle activity both in their normal resting postion and in mid-air.

Birds were then sacrificed under pentobarbitol anaesthesia. The sub-total or totally lesioned portion of the spinal cord was excised in situ with the surrounding vertebra and fixed in 10% formalin PBS. After a few days fixation, the bone surrounding the cord was removed. The cord was washed in water, dehydrated in alcohols, and embedded in wax (Paraplast or Paraplast +) for sectioning on a microtome.

Serial longitudinal or cross sections of cord were cut at thicknesses of 8 to 12 micrometers and floated on a warm water bath before mounting onto chrom-alum double subbed glass microscope slides. The sections were dried twenty four hours before staining for myelin (Luxol Fast Blue G) and cell bodies (Neutral Red). Stained slides were coverslipped and examined to determine the extent of the spinal cord lesion under a compound microscope.

RESULTS

A total of ten different lesion types were produced in order to delimit the areas of the spinal cord which carry pathways necessary for the control of hindlimb locomotion (Fig.12).

The sham operated animals, with no spinal cord lesion (Fig.12a) (n=2), showed no observable deficits in locomotor performance. Postoperative observation and EMG results (Fig.13) indicated a performance level comparable to preoperative levels with recovery of function within the first day following surgery. Histological examination verified that sham animals had intact spinal cords (Fig.14).

Complete transection of the cord (n=2) (Fig.15) produced locomotor deficits which left the animal totally incapable of self-support. In these birds, recovery periods of up to thirty days showed no indication for the return of any normal locomotion. Typical immediate postoperative posture was caudal extension of both legs and a lack of muscle tone (atonia). Spinal reflexes were depressed for a period of 4 to 9 days. Reflex responsiveness to foot web pinch reappeared between 4 and 9 days following the lesion. "Spinal stepping" (supported alternating stepping movements) occurred nine to ten days postoperatively. EMGs of "spinal stepping" demonstrated alternating flexor (ITC)(swing phase) and extensor (FCL)(extensor phase) activity in both legs (Fig. 16). Although alternate stepping movements occurred in most cases, sometimes these completely spinal birds demonstrated a simultaneous bilateral flexor/extensor action resulting in a

"hopping" or "pushing off" motion when the birds were placed on their sides. This action, as well as the bilateral alternating stepping occurred both spontaneously and as the result of perianal stimulation or pinching the webbing of the feet. Extension and flexion manipulation of one limb would also occasionally result in a period of "spinal stepping". In no case did the stepping resemble a motivated behaviour. Animals floating on water demonstrated no ability to paddle with their hindlegs. Placing the birds in water also alleviated the difficulty associated with the maintenance of lateral stability which is important for overground locomotion. Transected animals which demonstrated "spinal stepping" did not even produce a moderate degree of force during hindlimb extension and at no time could the animals produce enough extensor force to support themselves even when placed in an upright standing posture. Muscle wasting progressed with severe wasting apparent by the end of the experimental period (30 days postoperative). Histological examination of the lesions demonstrated a complete transection of the thoracolumbar spinal cord.

Hemisection of the spinal cord (n=3) (Fig.17) produced transient deficits in locomotor capability of the hindlimb. Reflex withdrawal to foot web pinch reappeared five to nine days postoperatively in the leg ipsilateral to the lesion and in but one day on the intact side. Caudal hyperextension of the ipsilateral leg was eventually replaced (9 to 26 days) by extension of the leg in a more normal stance position, parallel with the intact contralateral hindlimb. All animals were able to

stand (5-21 days postoperative) and walk (5-49 days), although locomotor capability (eg. flexion) across the distal joints in the ipsilateral leg remained somewhat restricted. The deficits appeared to result from an inability to fully flex/extend muscles across the distal (tibio-metatarsal) joints and a certain degree of extensor hypertonicity (rigidity) was apparent. EMG (Fig.18) results reveal the ability to flex and extend the femur across the hip joint in a relatively normal manner. Histological verification of a representative lesion site indicates a slightly incomplete hemisection with some sparing of the dorsal columns (Fig.17). All hemisected birds demonstrated similar characteristic recovery of function in their locomotor capabilities.

Transection of the dorsal half of the spinal cord, leaving the ventral cord intact (Fig.12d) (n=3), produced an initial deficit in standing ability characterized by an inability of the animals to maintain lateral and rostrocaudal stability. Maintained, balanced standing occurred after three to fourteen days, with self-supported walking reappearing between four and eighteen days postoperatively. Typically, the birds displayed an inability to step over uneven terrain without tripping, although this deficit disappeared rapidly with time. Walking improved to the point where it was impossible to discern any difference between normal unoperated animals and animals with the dorsal cord transected. EMG data demonstrate no difference between lesioned and normals (Fig.20). Verification of the lesion site shows dorsal cord section to the level of the central canal (Fig.19).

Unilateral ventral cord lesions (n=3) (Fig.12e) produced minor locomotor deficits in the hindlimb ipsilateral to the

lesion. Reflex response to foot web pinch appeared within one day postoperatively. Standing occurred between one and twelve days, with walking reappearing from one to thirteen days. Deficits were restricted to a mild hyperextension (rigidity) of the hindlimb which produced a walking gait having a limp on the lesioned side. EMG results indicate a near normal pattern of activity during treadmill locomotion (Fig.21) of flexor and extensor muscles within each hindlimb, with alternating activity of homologous muscles between limbs. Fig.22 illustrates a representative lesion for this type of subtotal transection.

Bilateral sectioning of the lateral margins of the spinal cord (Figs.12f,23) (n=3) produced little in the way of locomotor deficits. Foot web pinch reflex withdrawal reappeared within four days postoperatively. Standing occurred within one to two days and walking began within fourteen days. No motor deficits could be detected at the end of the thirty day postoperative period, as exemplified by the EMG results (Fig.24). Normal intralimb and interlimb timing for FCL/ITC muscles was apparent during treadmill locomotion.

A bilateral ventrolateral lesion (Fig.12g)(n=1) produced transient deficits in locomotor ability. Initial deficits included an inability to stand upright. Reflex withdrawal to foot web pinch reappeared at eleven days. Eventually, (after 20 days) the animal regained the ability to stand normally. Walking occurred at 22 days but the animal did not recover sufficiently during the experimental period to walk without falling. However, the locomotor pattern was near normal when the animal was tethered

in a sling. Histological verification of the lesion site (Fig.25) reveals that the damage included the entire ventral quadrant on one side, with only partial lesioning of the contralateral cord.

A ventromedial lesion (Figs.12h,26) (n=1) produced no locomotor deficits. Recovery of function occurred within one day for both standing and walking with no observable deficits. EMG results show normal activity for both hindlimbs (Fig.27) in FCL/ITC.

Animals in which only the ventromedial cord was left intact (Fig.12i)(n=6) demonstrated variable locomotor recovery depending on the extent of the lesioned area and the amount of ventromedial cord left intact. Two of the animals exhibited a histologically verified lesion (Figs.28a,b) which closely resembled the attempted lesion (Fig.12i). These birds recovered the ability to stand within five days postoperatively, with walking occurring between seven and nineteen days. Observations indicated normal walking, with representitive EMG results (Fig.29) demonstrating the relatively normal pattern of alternating flexion and extension for the hindlimb musculature.

Lesions maintaining the integrity of the ventrolateral cord bilaterally (Figs.12j,30) (n=4) allowed the birds to stand between five and fourteen days postoperatively. Walking began from seventeen to eighteen days. Postoperative EMGs of one experimental bird (Fig.31) clearly demonstrate alternating activity of FCL and ITC in each leg and rhythmic alternation of the two hindlimbs characterizing normal walking. No deficits could be observed at the end of the thirty day experimental period with the exception of one animal which demonstrated short steps and some lateral

instability.

If a single ventral quadrant (Figs.12k,32)(n=3) of the spinal cord remained intact, the bird was able to stand and take several steps, but was not capable of normal, self-supporting locomotion. Initial postoperative deficits included absence of reflex responsiveness to foot web pinch bilaterally. This reflex reappeared between 10 and 12 days following the lesion and was seen first in the leg ipsilateral to the intact portion of cord. Animals placed in water, allowing them to maintain lateral stability, were capable of producing voluntary swimming behaviour (alternating leg paddling). Postoperative hyperextension and rigidity occurred in the hindlimb contralateral to the intact portion of spinal cord. This was eventually replaced by movement of this limb into a more natural resting position under the animals body. Attempts to stand and support the body with the leg ipsilateral to the intact portion of cord preceded standing movements with the contralateral limb. The contralateral limb maintained a degree of rigidity which did not totally disappear during the experimental period, although a certain degree of flexibility was recovered in all of the joints of that limb. A reduction from normal force production was found in both hindlimbs with the deficit being more apparent on the transected side. Distal extremities (ie foot) muscle tonus appeared to be severely reduced and conseqently impeded normal locomotor activity. When the bird was standing unaided, however, the distal extremities were forced into a more natural position with the feet placed flatly on the ground. EMG data (Fig.33) supports the observation

that alternating hindlimb activity did occur. FCL/ITC activity within a hindlimb alternated out of phase with the contralateral hindlimb. However, the animals were not capable of normal self-supporting locomotion.

DISCUSSION

Supraspinal centres which control locomotion exert their influence on spinal cord pattern generators via descending pathways in the spinal cord (Steeves and Jordan, 1980) (Shik, Orlovsky, Severin, 1966) (Shik and Orlovsky, 1976) (Eidelberg, 1981). Evidence available at the present time indicates that this holds true for primates such as monkeys (Eidelberg et al., 1981a), cats (Eidelberg et al., 1981c) (Afelt, 1974) (Steeves and Jordan, 1980, 1984), and stingrays (Williams et al., 1984). The experiments upon which this information is based include both acute and chronic lesions of the spinal cord in acute brainstem stimulated and intact chronic animals. Disruption of particular descending pathways by selective lesioning of the spinal cord often leads to specific deficits in locomotor capability. This experimental procedure is one approach for defining those descending pathways which are essential to the initiation and modulation of spinal locomotor mechanisms. The approach requires a knowledge of the funicular trajectories of descending spinal pathways. Cabot et al. (1982), using anterograde and retrograde anatomical tracing techniques, described the trajectories of some of the major descending pathways in the pigeon (Fig. 34) which will be used in this study to define the pathways necessary to locomotion.

Complete transection of the goose spinal cord at the low thoracic level produces a period of "spinal shock" similar to that found in primates, cats, dogs, and lower vertebrates. This

"spinal shock" is characterized by somatic muscle flaccidity, absence of segmental reflex responses (hyporeflexia), and cloacal flaccidity. This period of "spinal shock" is shorter in duration than that for mammals and usually disappears within four days after the transection. These results support research findings that the period of "spinal shock" tends to shorten with a decrease in central nervous system complexity (Eidelberg, 1981).

The gradual return of hindlimb muscle tone in the geese allowed for the production of "spinal stepping" similar to that found following transection in many vertebrate species, excluding primates (Eidelberg et al., 1981a). This observation agrees with previous investigators of avian species (Tarchanoff, 1895) (Ten Cate, 1960), non-primate mammals (Phillippson, 1905-dogs) (Grillner, 1973-cats) (Stelzner et al., 1975-rats) (Graham Brown, 1911-guinea pig), and lower vertebrates (Cohen and Wallen, 1980-lamprey) (Grillner, 1974-dogfish) (Gray and Lissman, 1946-amphibians). Although some investigators have reported that completely transected animals were capable of self-support (Phillippson, 1905), no indication of this ability was found in any of the birds used in this study. "Spinal stepping" was restricted to alternating or simultaneous movements of the hindlimbs with a degree of force insufficient to support the body of these animals. One might postulate that this lack of force could be attributed to excessive muscle wasting which occurred prior to restitution of locomotor function. However, if on considers the short time period between the lesion and the appearance of "spinal stepping" coupled with the apparent lack of

any detectable muscle wasting in that time, then the postulate is highly unlikely. Alternatively, on might hypothesize that the generators are unable to, in the absence of descending influence, recruit a sufficient number of motor units for anti-gravity support.

The results of this study support the hypothesis of Graham Brown (1911) that a mechanism intrinsic to the spinal cord is responsible for the production of stepping (step generators) in the absence of descending supraspinal influences. However, adequate production of muscle force sufficient for self-supporting locomotion may require the integrity of some descending supraspinal input.

Spinal cord hemisection failed to produce major chronic deficits in locomotion in the goose. The birds displayed symptoms very similar to those described by Brown-Sequard (1870) in humans. The Brown-Sequard syndrome in humans is characterized by loss of postural sense, transitory ipsilateral spastic paralysis or severe paresis and contralateral loss of pain as well as thermal sense caudal to the lesion site. Later experimenters confirmed these results in non-human primates such as monkeys (Lassek and Anderson, 1961) and non-primate mammals such as cats (Marshall, 1895) and dogs (Weiss, 1879). Little research exists with respect to hemisection in lower vertebrates. The results of this study indicate that this type of lesion in the goose produces a short term deficit in motor function, with recovery of function in a period (5-21 days) that is usually more rapid than for mammalian species (Afelt, 1974; Eidelberg, 1981c). Motor recovery following hemisection was originally thought to be subserved by the

corticospinal system which sends both crossed and uncrossed projections to the spinal cord in mammals. It has recently been demonstrated however, that lesioning of the pyramids produces little disruption to primary locomotor patterns (eg. walking), only disrupting distal musculature responsible for fine control of movement (eg.Kuypers, 1982). The absence of a large, well defined corticospinal projection in the goose (Cabot et al., 1982; Reiner et al., 1982) supports the hypothesis that lower supraspinal centres are responsible for this recovery of function in the hemisected animal. However, due to the possible presence of crossed connections caudal to the lesion site, it is difficult, on the basis of hemisection locomotor deficits, to determine which pathways control and initiate locomotion.

Similarly, the dorsal cord lesion (Fig.12d) did not produce any long lasting effects on goose locomotor patterns. The initial inability of postoperative animals to navigate uneven terrain may reflect the loss of afferent sensory input via the ascending dorsal columns pathways responsible for tactile and kinesthetic information to supraspinal areas (Fasciculus gracilis, cf. Carpenter, 1978) . Lesion of the dorsal column pathways in humans and monkeys results in a loss of position sense resulting in dorsal column ataxia (Ferraro and Barrera, 1934; Gilman and Denny-Brown, 1966). However, Eidelberg et al. (1966) found that these deficits could be overcome by the production of specific motivational drive in pretrained animals.

By comparing the extent of the spinal cord lesions which do not significantly alter avian locomotion with the funicular

trajectories of known avian descending spinal pathways, it is possible to eliminate the role of certain neural pathways from the initiation and control of locomotion. Dorsal cord interruption of the descending hypothalamo-spinal, dorsolateral pontine-spinal, dorsal column nuclei-spinal, rubrospinal, N. raphe magnus-spinal, N. raphe pallidus-spinal, and portions of the N. raphe pallidus/obscurus-spinal tracts (Fig. 34) does not appear to produce any long lasting deficits in motor function.

Of the above lesioned pathways, only the rubrospinal tract has been definitely shown to be involved in the control of flexor motorneurons (Grillner and Lund, 1968) and in the initiation of locomotion. However, Lawrence and Kuypers (1968b) showed that bilateral lesion of the Red nuclei in monkeys had little effect on locomotion, only disrupting fine motor control. The observations obtained in this study tend to support the findings of Lawrence and Kuypers (1966) in that bilateral lesion of the rubrospinal tracts at the low thoracic level had little effect on the walking ability of the goose.

Bilateral ventromedial lesion (Fig. 26) of the spinal cord which disrupts the descending periaquaductal grey-spinal, the ipsilateral pontine reticulospinal, and portions of both vestibulospinal and raphe-spinal pathways (Fig. 34) was not effective in limiting locomotion in the goose. The rapid recovery and lack of any apparent deficit demonstrated that the periaquaductal grey-spinal and ipsilateral pontine reticulospinal former pathways are not essential for the production of locomotion in these animals, even though electrical stimulation of these areas in acute decerebrate cats evokes locomotion (Garcia-Rill et

al., 1983; Budakova and Shik, 1980). The partial destruction of continuity in the avian vestibulospinal and raphe-spinal pathways also did not seem to affect hindlimb walking.

Previous lesion studies have hypothesized the possibility that the vestibulospinal tract has some role in the initiation and control of locomotion in the cat (Yu and Eidelberg, 1981). Bilateral lesion of the vestibular nuclei produced severe deficits in posture and locomotion, which were manifested as reduction in extensor drive to the limbs and loss of exitability in neck motorneurones. The deficits diminished over time. Eidelberg postulated, however, that the vestibular nuclei, and in particular, the lateral vestibular nucleus (Dieters nucleus), (Orlovsky, 1972b) plays a major adjunctive role in the initiation and control of locomotion by enhancing the extensor elements of the step cycle. This is corroborated by acute experiments in which an increase in extensor activity resulted from stimulation of Dieters nucleus. Also, increased phasic activity of the vestibular neurons occurred during acute stimulation (MLR) in decerebrate freely performing animals and "fictive" preparations (Orlovsky, 1972a,b). The return of function which occurs after the initial deficits in chronic animals has yet to be explained.

Lesions which maintained the integrity of the ventomedial cord (Fig. 28) resulted in animals which recovered locomotor capability rather quickly. Descending pathways which travel in the lesioned area include the periaquaductal grey-spinal, pontine reticulospinal, portions of the vestibulospinal, and part of the raphe-spinal (Fig. 34). This lesion leaves intact the funiculi

which were lesioned by the ventromedial lesion and destroyed the material left intact by the ventromedial lesion. The periaquaductal grey-spinal and pontine reticulospinal pathways left intact in this lesion do not appear to be necessary for the production of locomotion. However, maintenance of parts of the vestibulospinal and the raphe pallidus/obscurus / reticulospinal pathways carried in the ventromedial cord appears to allow return of function following lesion of the rest of the cord.

Lesioning of the spinal cord leaving both ventrolateral quadrants intact also produced animals capable of normal locomotion. Therefore, it appears that maintenance of the ventral intermediate cord allows the standing and the initiation of locomotion in the goose. Descending pathways carried in this area include the ipsilateral projection from the medial reticular formation Nucleus reticularis gigantocellularis, the pathway from the nucleus raphe pallidus/obscurus, and the vestibulospinal projections (Fig. 34).

Removal of all descending projections except those travelling in one ventral quadrant (Fig. 32) also allowed bilateral locomotion, although the quality of movement was somewhat reduced owing to an apparent paresis and lack of strength in these preparations. The hindlimb ipsilateral to the spared white matter produced stronger stepping than in the contralateral hindlimb. These animals were capable of bilateral standing and stepping (Fig. 33) but locomotion did not return to normal ability. The recovery period was also considerably longer (35 days) for these geese than for the animals wherein a larger area of ventral cord remained intact.

The results of selective lesions to the spinal cord demonstrate that the ventral spinal cord is essential for the initiation, modulation and continuity of activation of locomotion. Further, unilateral sparing of a single ventral quadrant provides sufficient descending information to promote relatively normal locomotor patterns. Localization of specific pathways utilizing this method has demonstrated that several known descending pathways may be involved. The vestibulospinal pathway has previously been implicated in control of extensor elements of the step cycle (Orlovsky, 1972a). However, Yu and Eidelberg (1981) have shown that bilateral lesion of the vestibular nuclei in the cat did not abolish return of locomotor function. This restitution of function remained stable in the cats even when a dorsal cord lesion was performed, thus eliminating the possibility that compensation was occurring via the mammalian corticospinal and/or rubrospinal descending pathways (Yu and Eidelberg, 1981). Due to the phylogenetic differences between mammals and avian species, it is difficult to separate the role played by the vestibular pathways in the goose from that of other pathways present in the ventral cord. Lesioning of the dorsal spinal cord and vestibular nuclei would be necessary to determine the importance of the vestibulospinal pathway for the initiation of locomotion.

The raphe-spinal pathway, the major component of which in the ventral cord arises from the caudal portions of the raphe pallidus and obscurus nuclei, has not yet been implicated in any locomotor pathways but is thought to play a major role in the innervation of preganglionic sympathetic components of the avian nervous system

(Cabot et al., 1982).

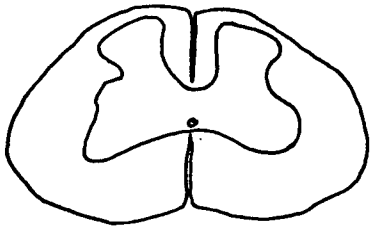
The ipsilateral reticulospinal pathways which travel in the ventral funiculus include the pontine reticulospinal pathway originating from the nucleus reticularis pontis caudalis, pars gigantocellularis and nucleus reticularis pontis oralis, as well as a projection originating from the medial medullary reticular nucleus reticularis gigantocellularis (Fig.34). Complete bilateral lesion of the pontine reticulospinal pathway produced no observable change in locomotion. Therefore it is unlikely that this pathway plays a predominant role in the initiation of avian walking. Bilateral partial lesions in the medial medullary reticular nucleus reticulospinal pathway also produced little observable difference. However, unilateral lesion of both pathways appeared to increase the length of the recovery period and decrease the quality of locomotion. One might hypothesize that the medullary reticulospinal pathway originating from N. reticularis gigantocellularis travelling as a diffuse projection in the ventral spinal cord plays an important role in the volitional control of locomotion. Lesioning of the spinal cord and vestibular nuclei interrupting the flow of information in the ventromedial cord and dorsal cord would help to establish the veracity of this latter hypothesis.

The results of this study support those found in monkey (Eidelberg et al., 1981a), cat (Steeves and Jordan, 1980) (Eidelberg et al., 1981c) and stingray (Williams et al., 1984) namely, that restitutution of function occurs following spinal cord lesions in both acute and chronic animals if a single ventral quadrant of spinal cord remains intact. Although the

stepping/swimming seen in these animals initially appears unilaterally, after a period of time, the stepping becomes bilateral. It is postulated that the descending pathway(s) controlling locomotion either innervates the locomotor generators bilaterally at the appropriate level or that it innervates unilaterally and the neuronal circuits of the pattern generators provide the necessary contralateral interconnections. The results support the hypothesis that the medullary reticulospinal pathway plays an important role in the initiation of locomotion and does not negate the hypothesis that the vestibulospinal tract plays some adjunctive role (Yu and Eidelberg, 1981) in the supraspinal control of locomotion.

FIGURE 12

Diagrammatic representation of attempted low thoracic spinal cord lesions used to delineate descending spinal cord pathways responsible for the supraspinal control of locomotion in the hindlimb. Lined areas indicate lesion extent. A= Sham Operate, B= Complete Transection, C= Hemisection, D= Dorsal Cord Transection, E= Unilateral Ventral Quadrant Lesion, F= Bilateral Lateral Margins Lesion, G= Bilateral Ventrolateral Lesion, H= Ventromedial Lesion, I= Ventromedial Intact, J= Bilateral Ventrolateral Intact, K= Ventral Quadrant Intact.



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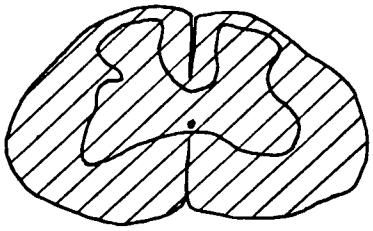

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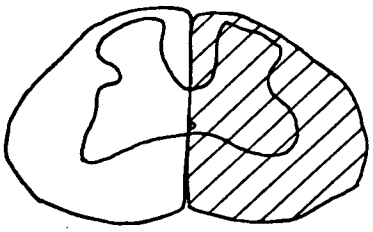

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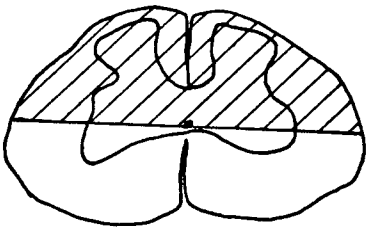

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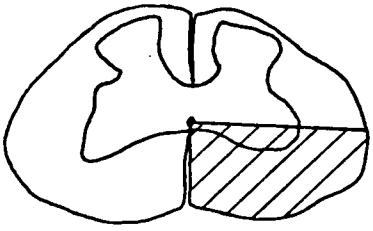

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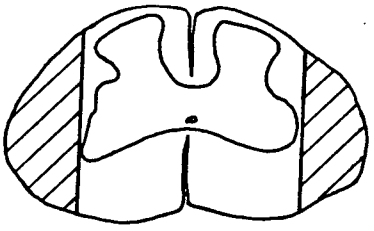

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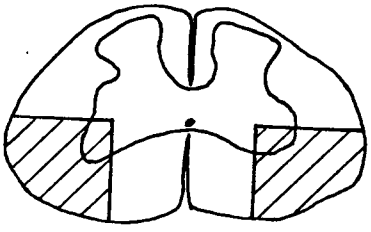

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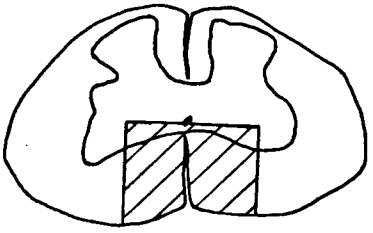

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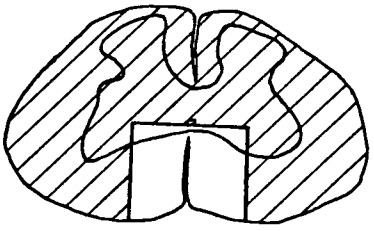

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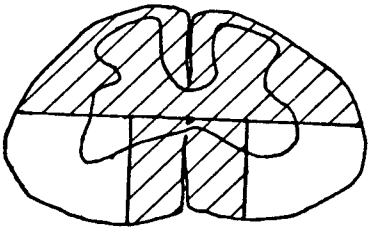

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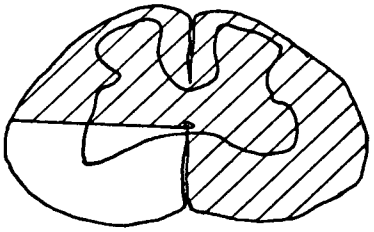


K

FIGURE 13

Postoperative electromyographic records from left and right hindlimb ITC and FCL muscles during treadmill locomotion in a sham operated goose during treadmill walking. ITC is a flexor active during the swing phase. FCL is an extensor active during the stance phase of walking. The records show alternating activity of homologous hindlimb muscles during walking (belt speed= 0.3m/sec). Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

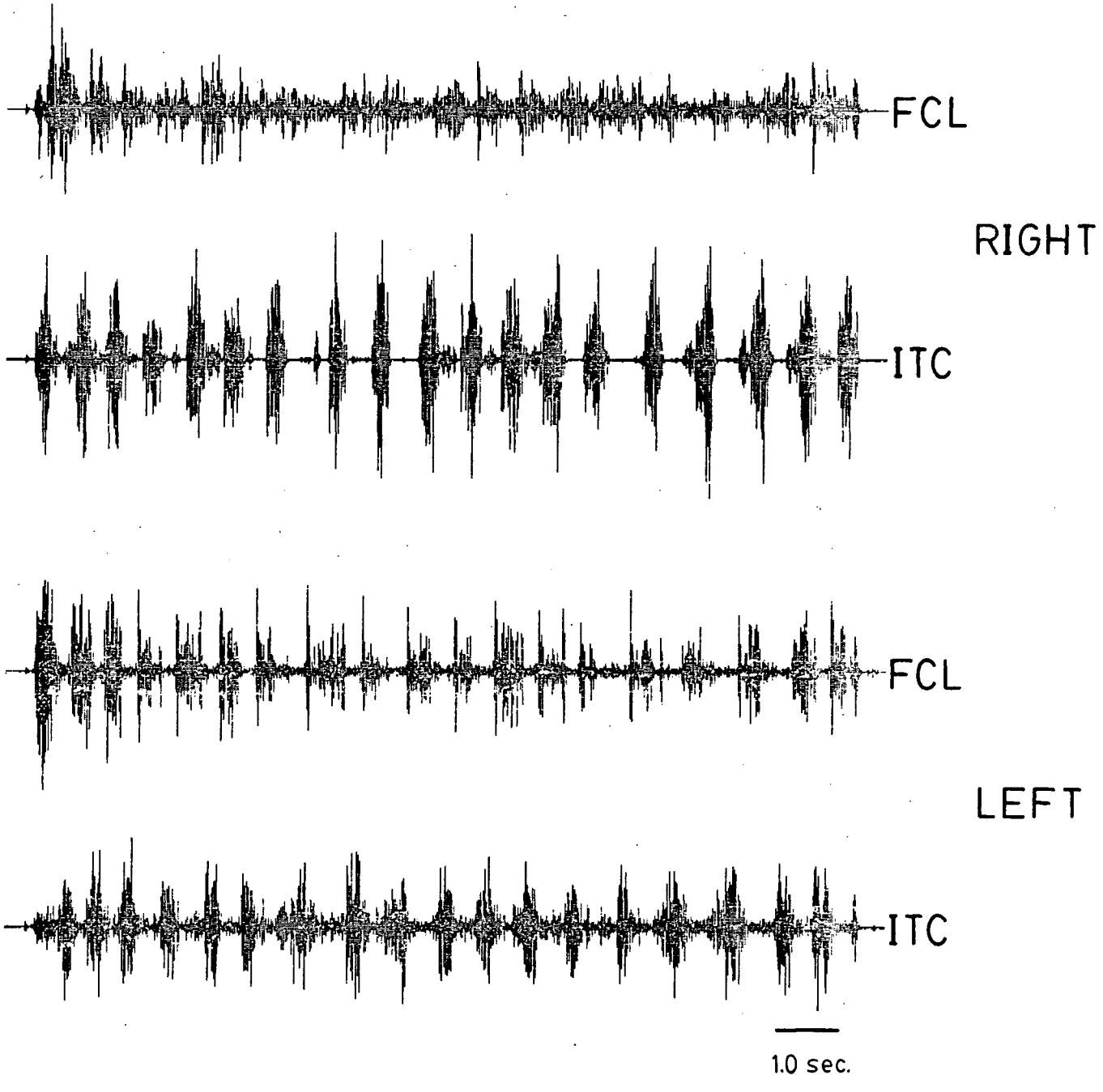
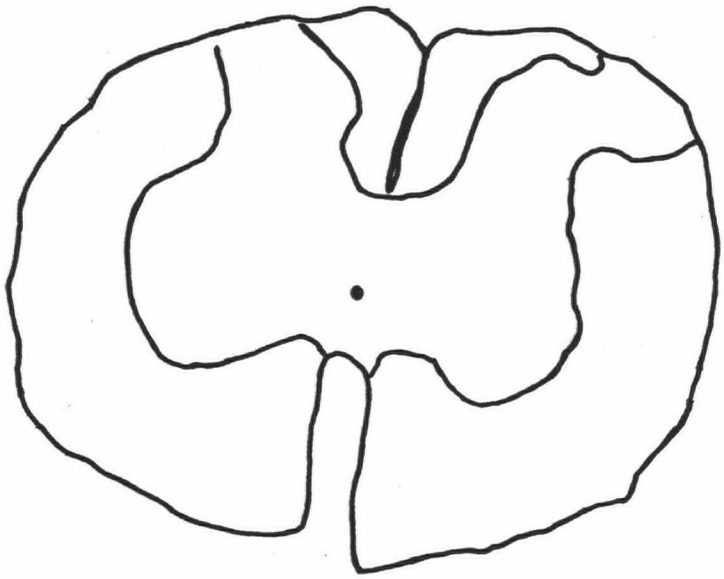

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 14

Diagrammatic representation of a cross section through the low thoracic spinal cord of a Sham Operated goose showing the absence of any lesion is shown in A. B is a photograph of intact spinal cord from the Sham Operated goose.



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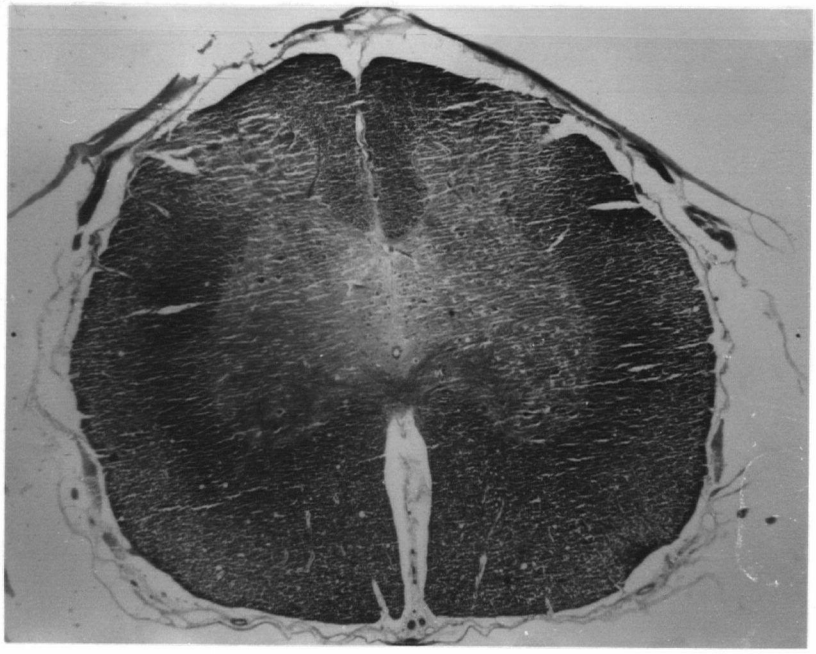


B

FIGURE 15

Diagrammatic representation of a cross section through the lesion site in a goose following a complete transection. The diagram is a composite compiled from serial sections through the lesion site. The lined area indicates the completeness of the attempted lesion.

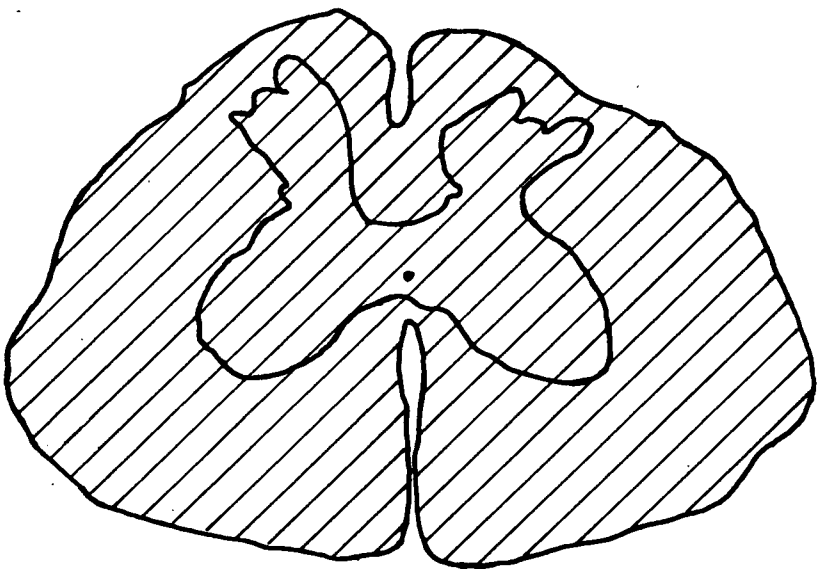


FIGURE 16

Electromyographic records from left and right hindlimb ITC and FCL muscles during "spinal" stepping in a completely transected animal. Records from the left leg show alternating activity of flexor (ITC) and extensor (FCL) muscles. Simultaneous activation of flexor bursts between left and right ITC muscles demonstrate the "pushing off" or "hopping" nature of the "spinal" locomotor behaviour. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

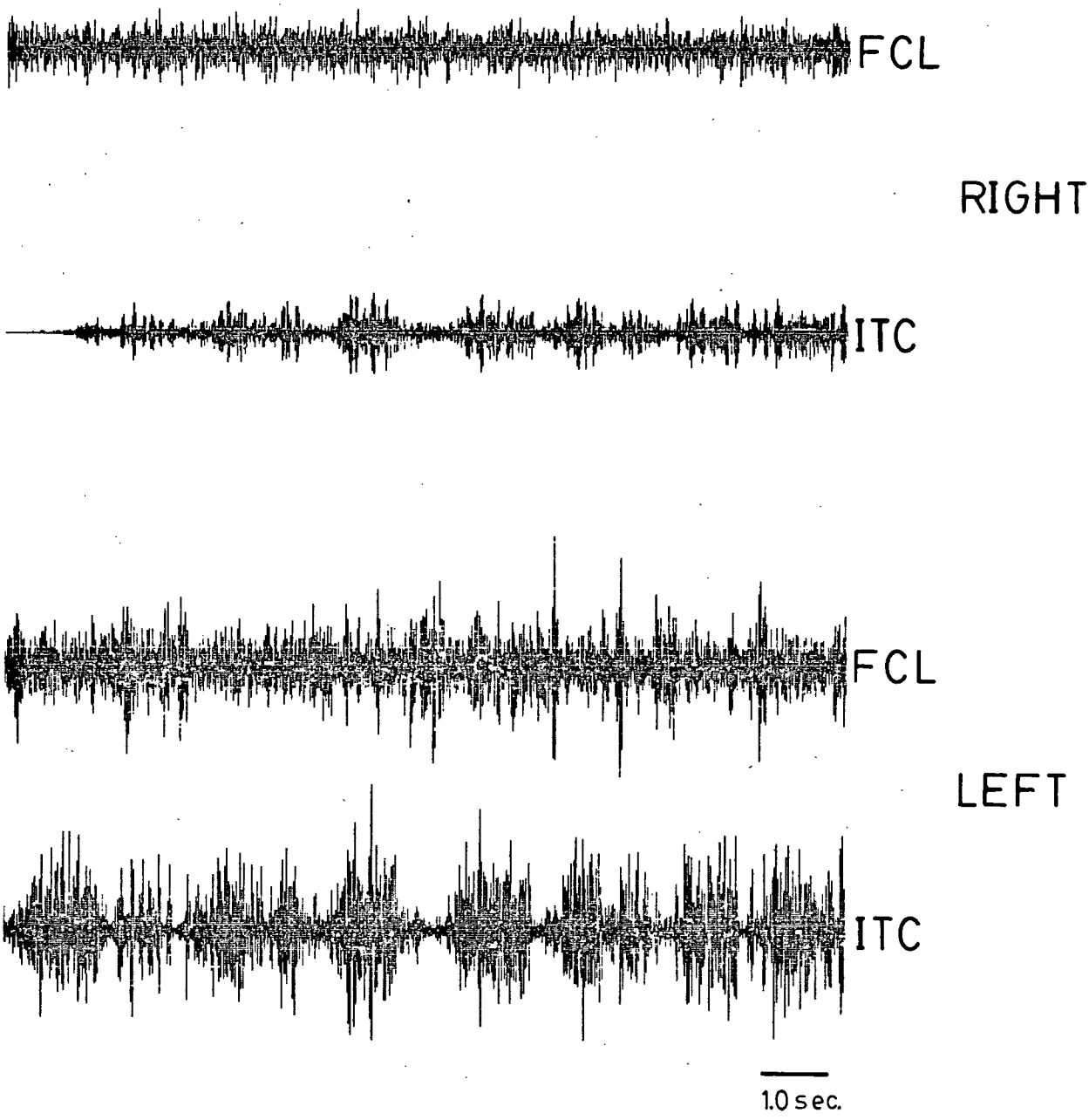

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 17

Diagrammatic representation of a cross section through the low thoracic spinal cord of a Hemisected bird. The diagram is a composite made from serial sections through the lesion site. The lined area shows the lesion extent, with sparing of a portion of the dorsal columns and dorsal horn gray matter.

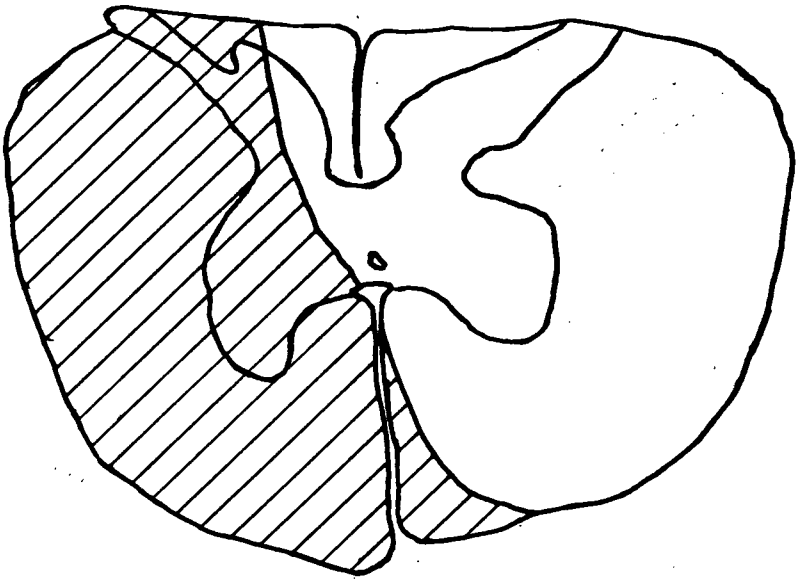


FIGURE 18

Postoperative electromyographic records of the goose hindlimb flexor (ITC) and extensor (FCL) muscles during treadmill walking (belt speed= 0.4 m/sec) following a chronic spinal cord hemisection. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

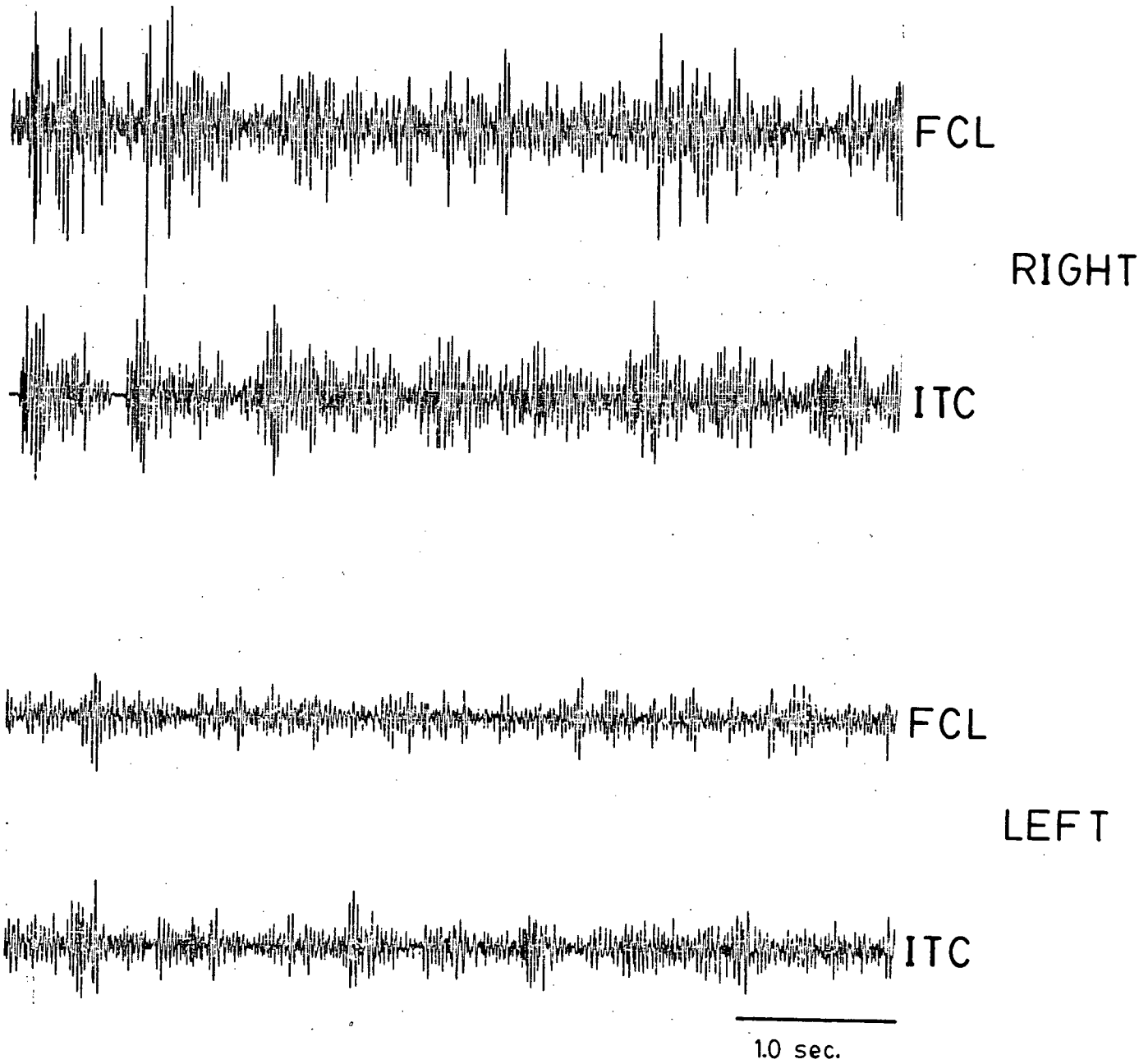
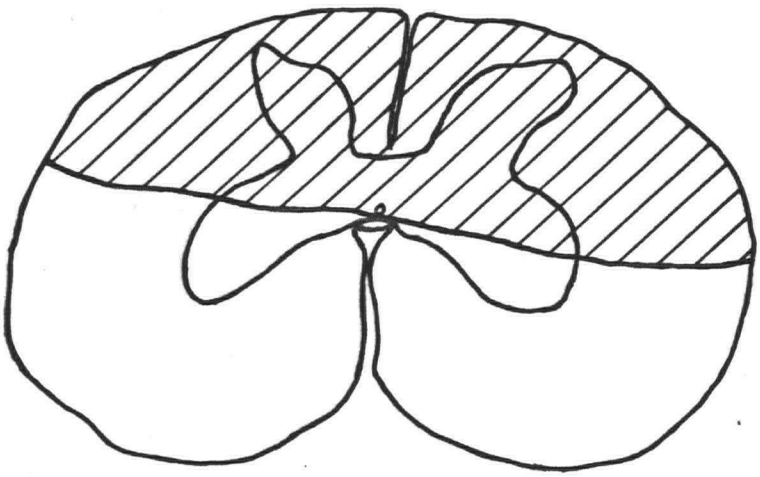
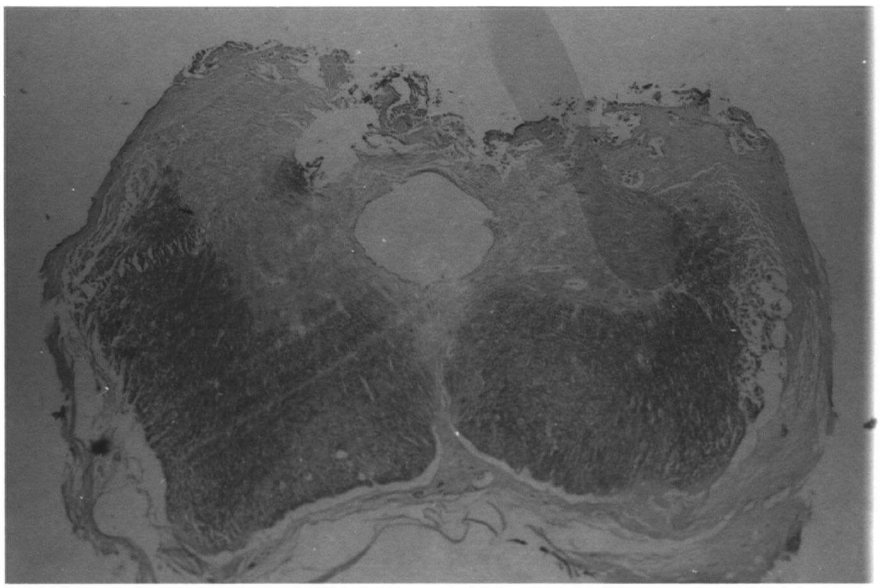

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 19

Diagrammatic representation of a cross section through the low thoracic spinal cord following a Dorsal Cord Transection (A). The diagram is a composite made from serial sections through the lesion site. The lined area shows the lesion extent, with sparing of the cord ventral to the central canal. B is a photograph of one section through the lesion site.



A



B

FIGURE 20

Postoperative electromyographic (EMG) records of the goose hindlimb flexor (ITC) and extensor (FCL) muscles during treadmill walking (belt speed= 0.3 m/sec) following a chronic Dorsal Cord Transection. The EMG records show normal alternating activity of antagonistic muscles of each limb and alternation of contralateral homologous muscles. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

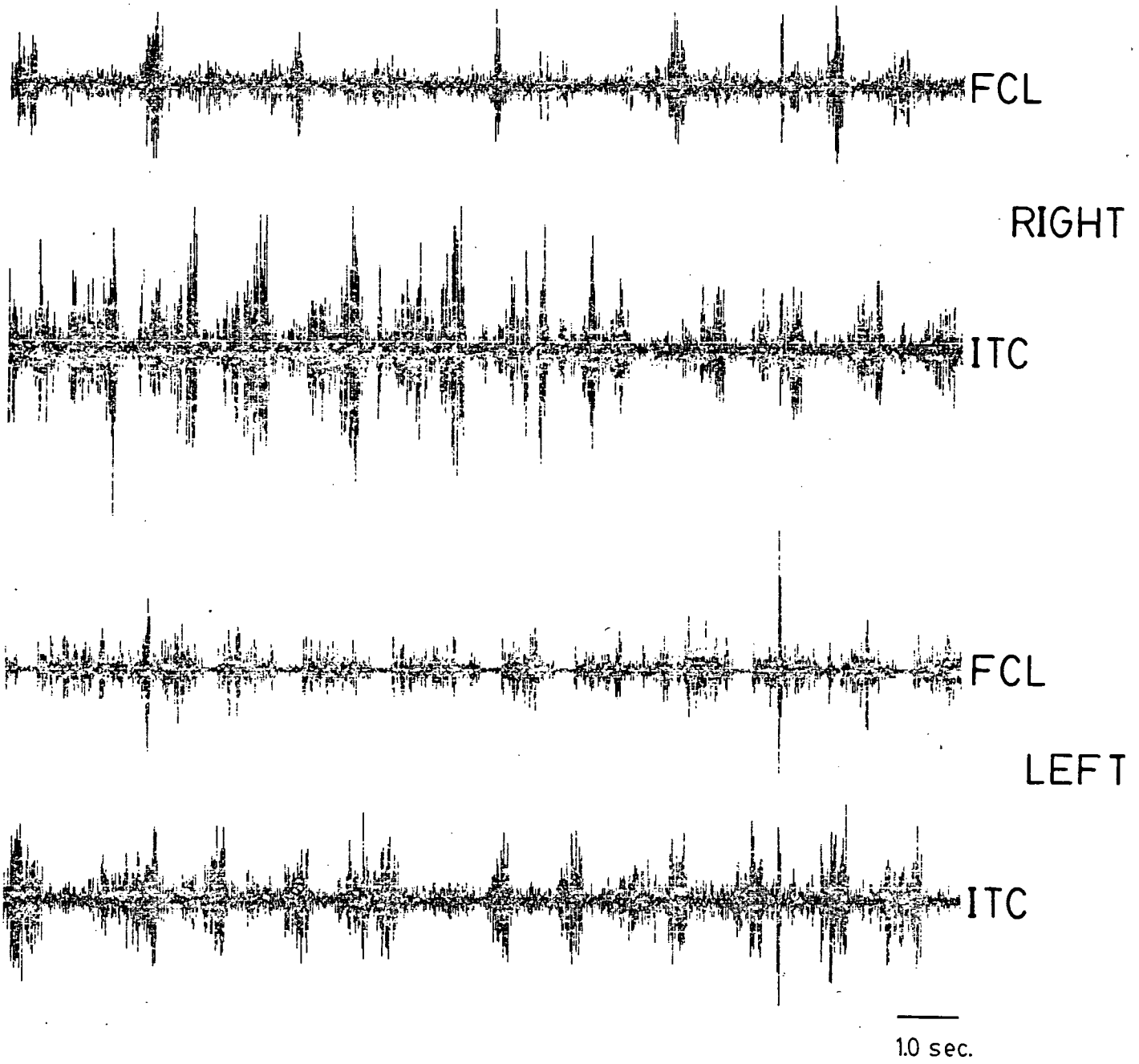

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 21

Postoperative electomyographic records of the goose hindlimb flexor (ITC) and extensor (FCL) muscles during treadmill walking following a chronic Unilateral Ventral Quadrant Lesion. ITC alternates out of phase with its ipsilateral antagonist (FCL) and contralateral homolog (ITC). Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

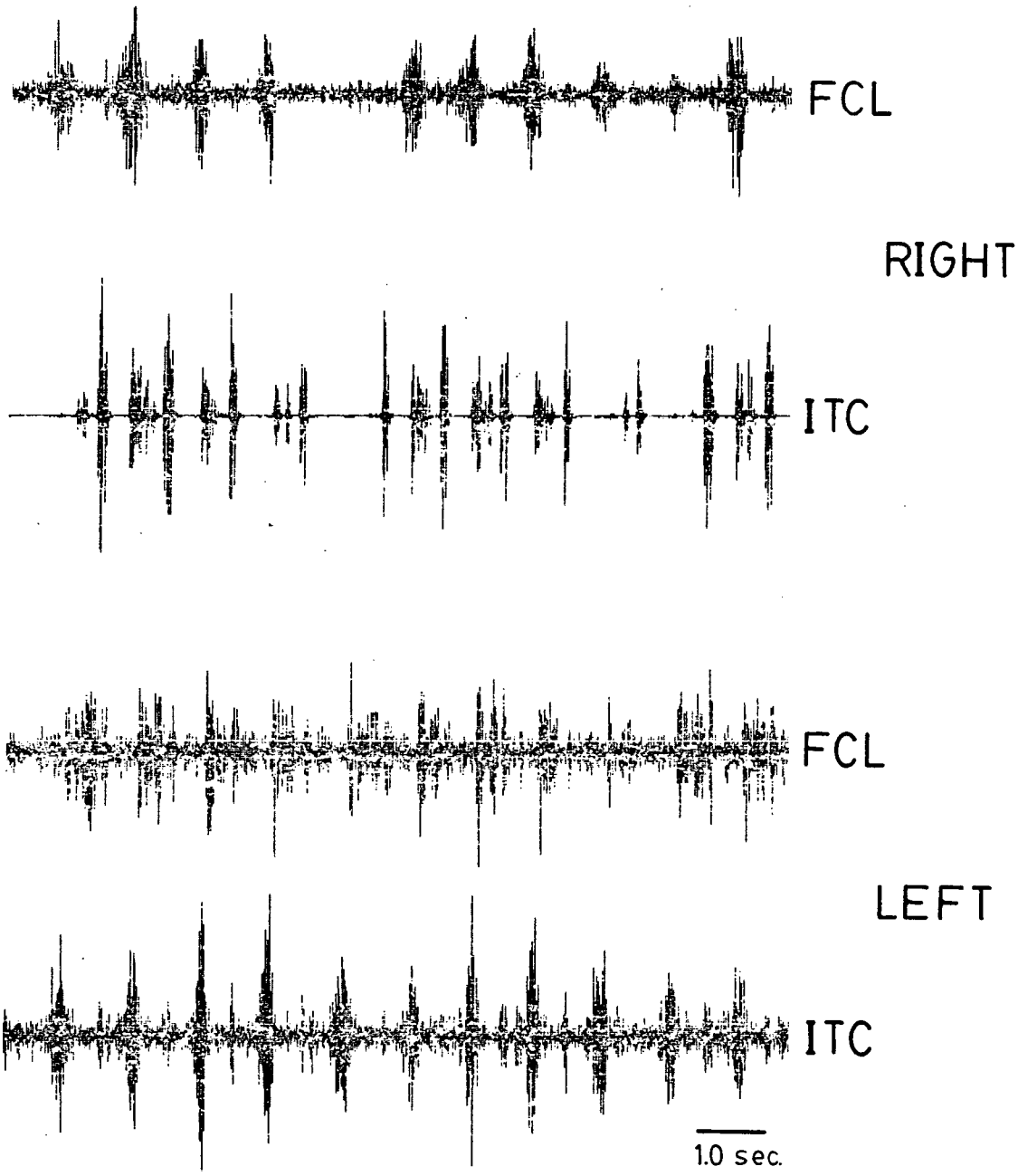
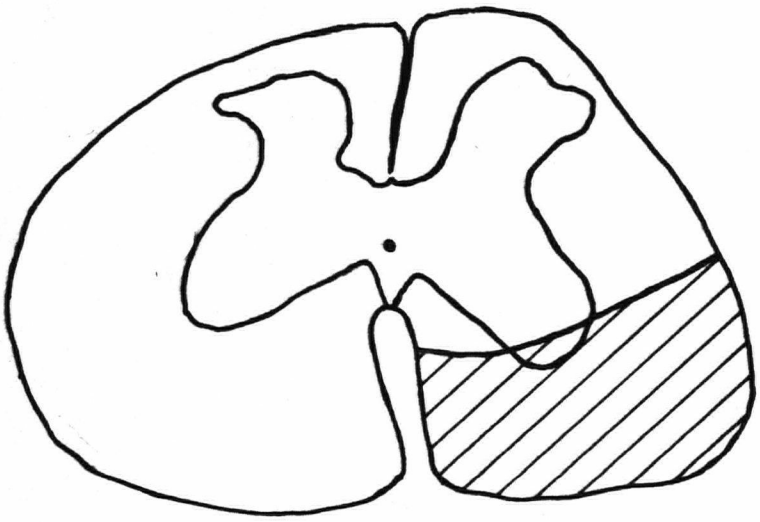
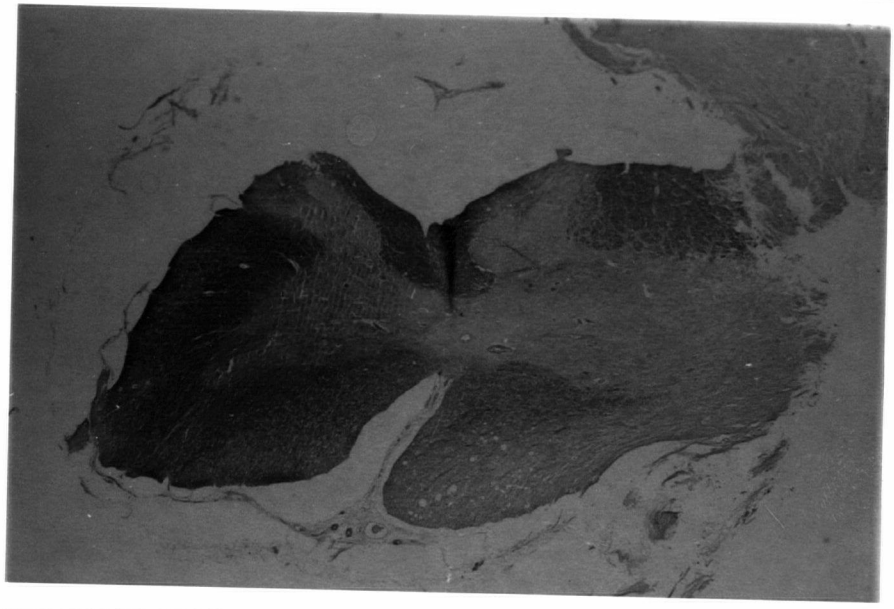

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 22

A Diagrammatic representation of a cross section through the low thoracic spinal cord following a Unilateral Ventral Quadrant lesion. The diagram is a composite made from serial sections though the lesion site. Lined areas indicate the lesion extent. The section of spinal cord in the diagram is slightly rostral to the lesioned area. B is a photograph of one transverse section through the lesion site.



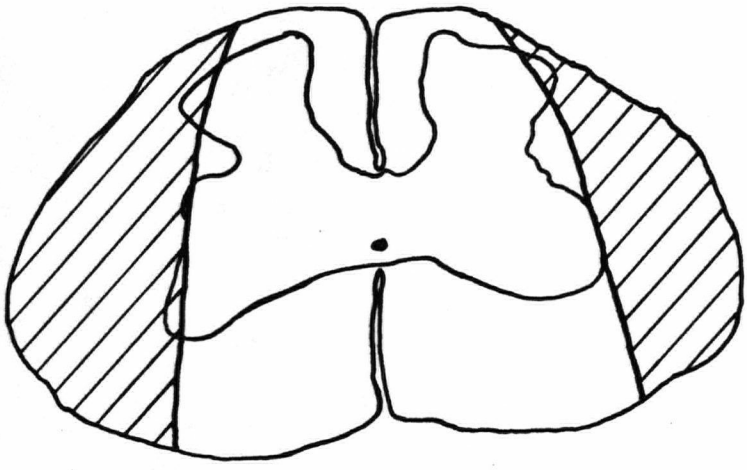
A



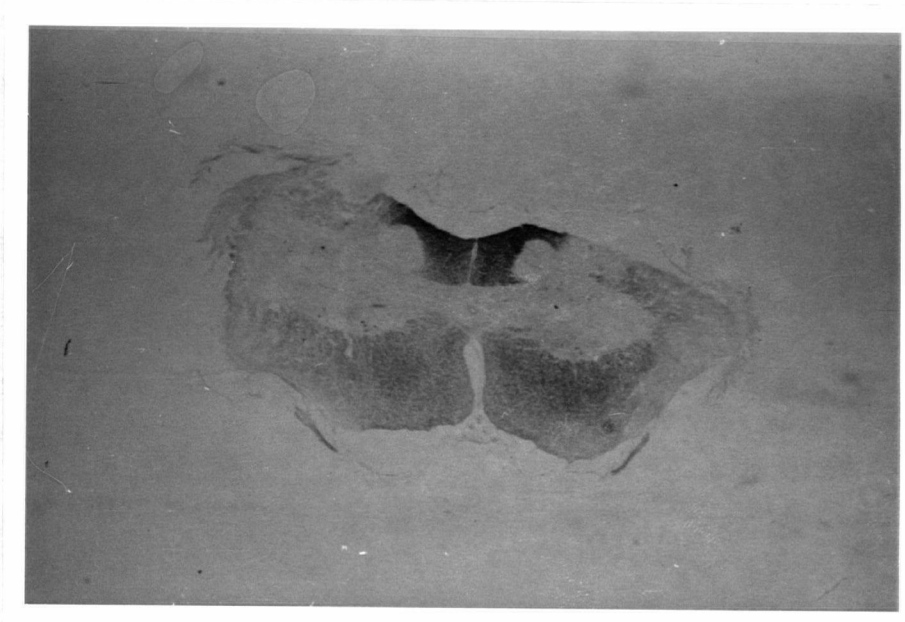
B

FIGURE 23

Cross section through the low thoracic spinal cord of a goose following a Bilateral Lateral Margins Lesion (B). The diagram (A) is a composite made from serial sections through the lesion site. Lined areas demonstrate the lesion extent, with sparing of the majority of medial spinal cord.



A



B

FIGURE 24

Postopertive electromyographic records of the flexor (ITC) and extensor (FCL) muscles during treadmill walking thirty days after a Bilateral Lateral Margins Lesion. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

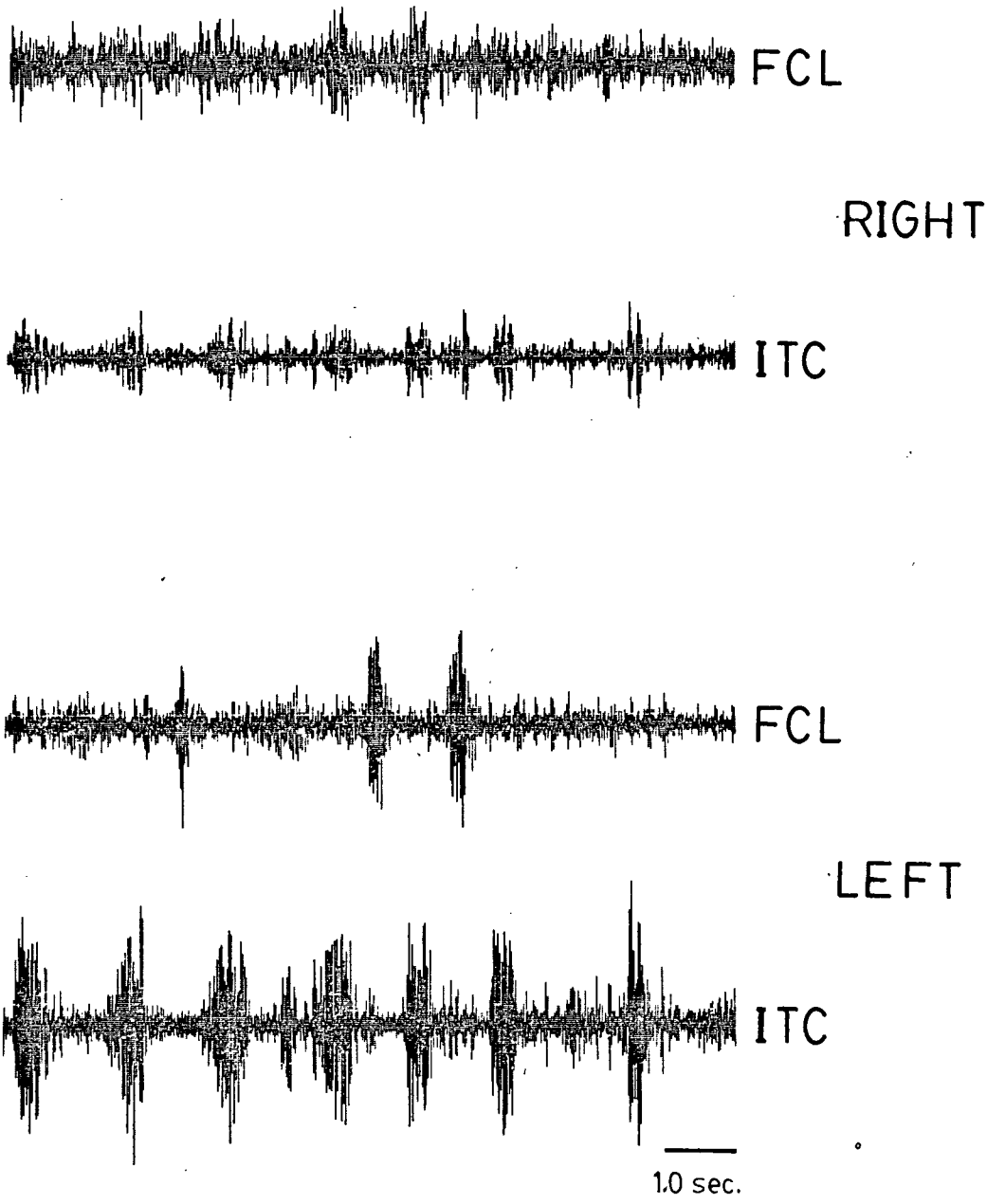

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 25

Diagrammatic representation of a cross section through the low thoracic spinal cord following a Bilateral Ventrolateral Lesion. The diagram is a composite made from serial cross sections through the lesion site. The lined area shows the lesion extent.

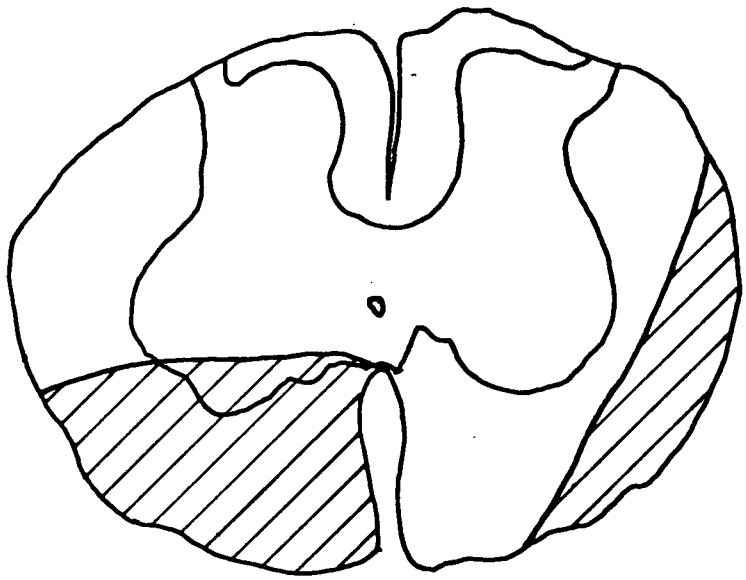
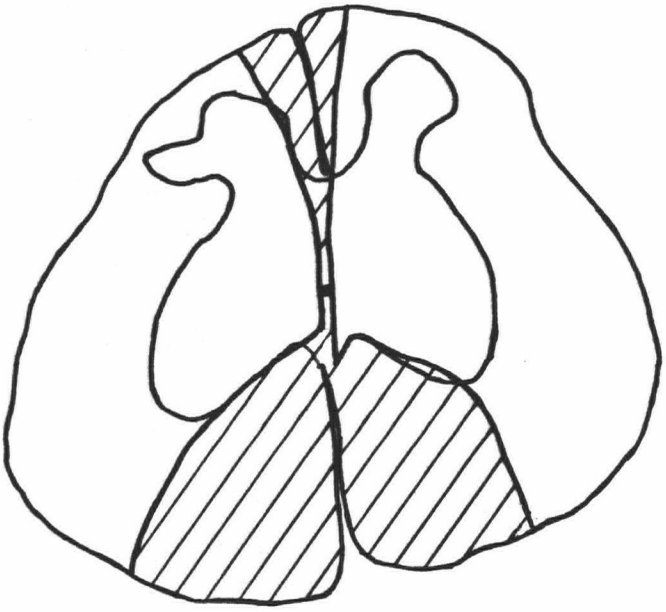
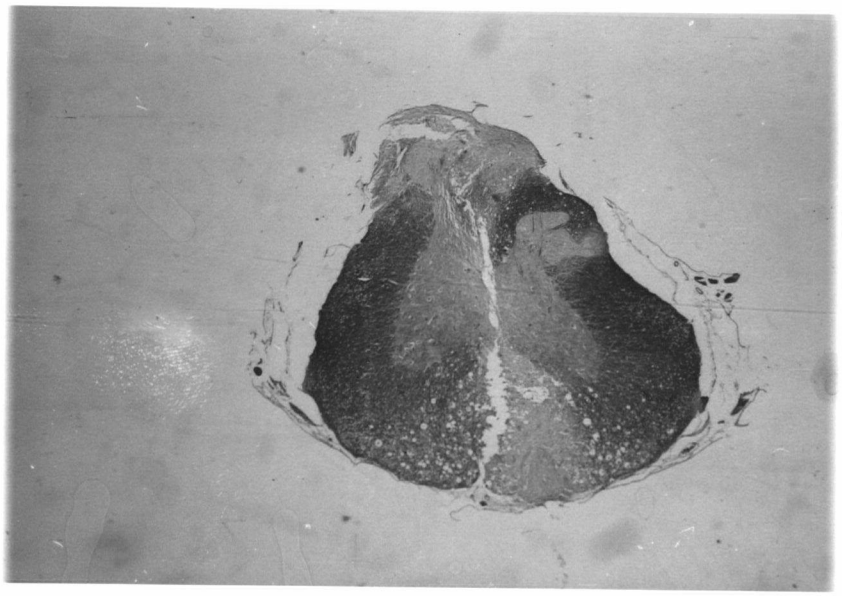


FIGURE 26

Diagram showing the lesion extent after a Ventromedial Lesion. The diagram is a composite drawn from serial sections through the lesion site (A). The lined area delimits the lesion extent, with partial lesioning of the dorsal columns and complete lesioning of the ventromedial cord. The photograph (B) shows a partial delineation of the lesion extent through one transverse section.



A



B

FIGURE 27

Postoperative electromyographic records of bilateral hindlimb flexor (ITC) and extensor (FCL) muscles during treadmill walking following a chronic Ventromedial Lesion. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

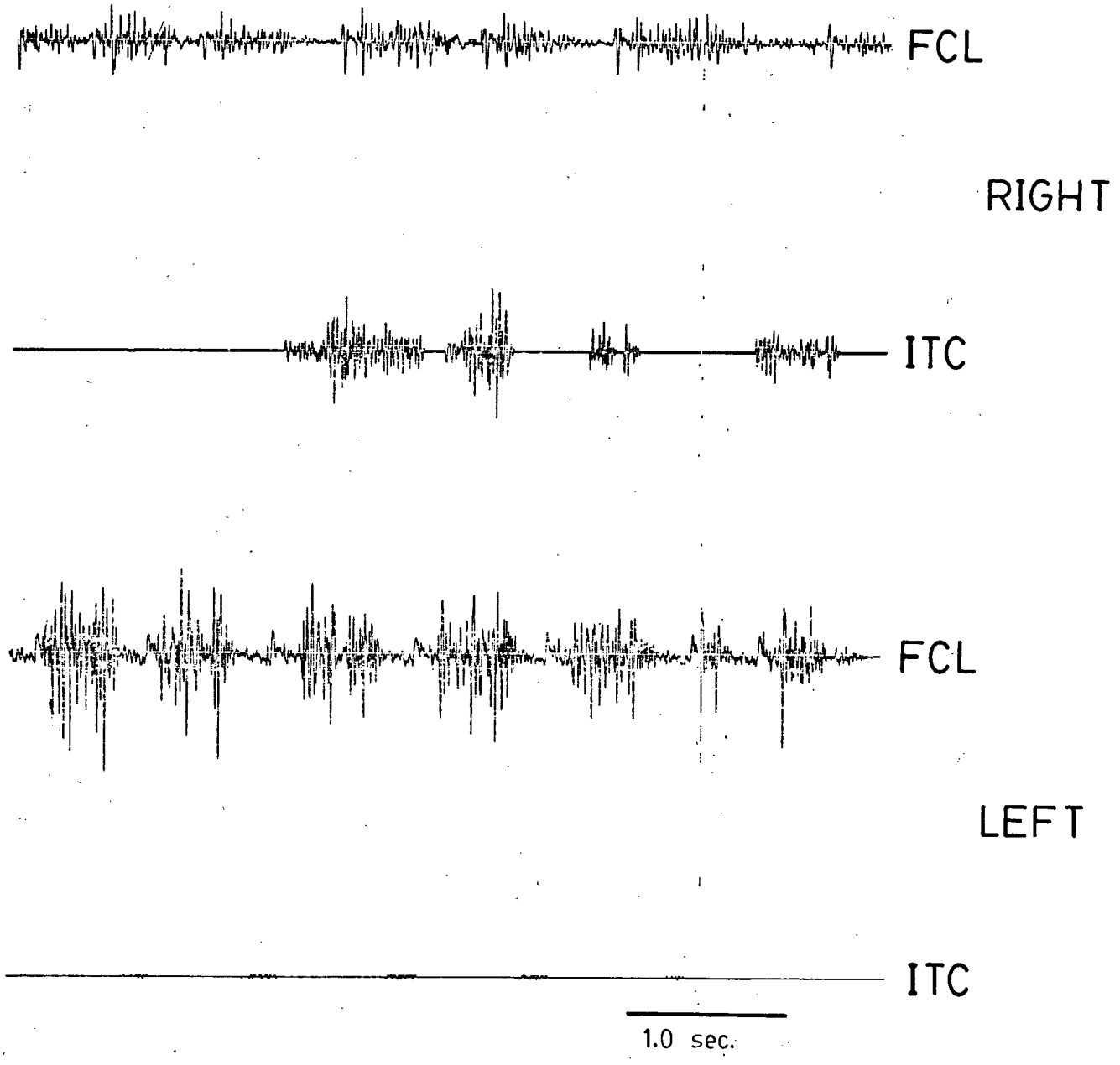
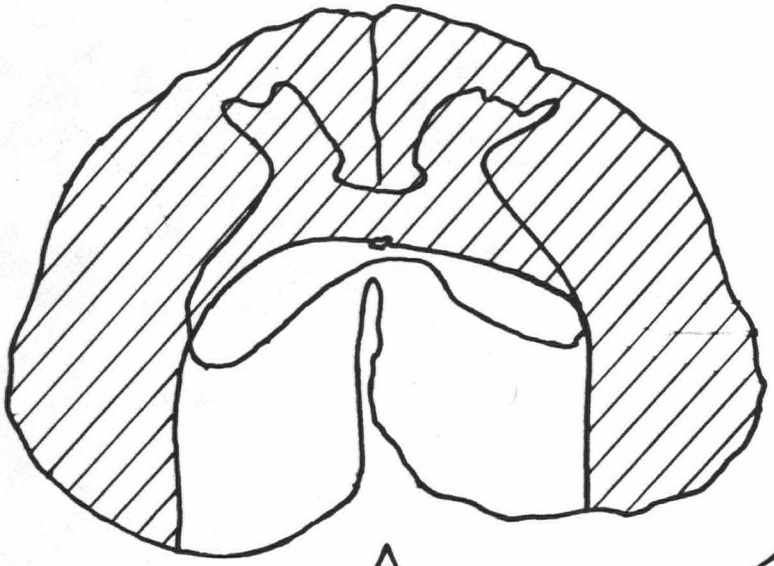
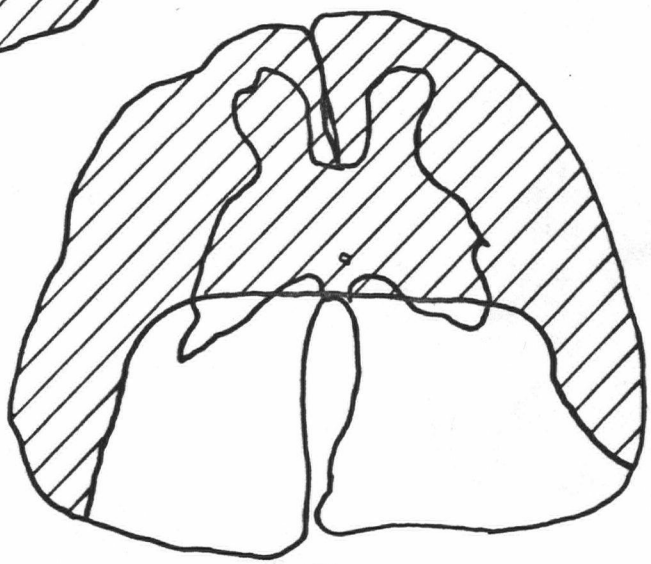

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 28

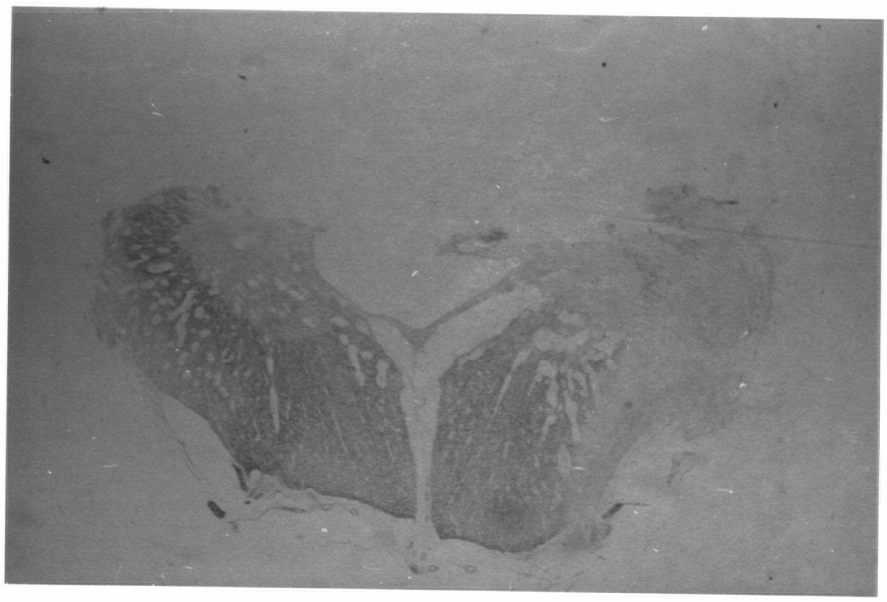
Diagrams showing the lesion extent resulting from Ventromedial Intact lesions in the low thoracic spinal cord. The diagrams are composites drawn from serial cross sections in A and serial longitudinal sections in B. The lined areas indicate lesion extent. The spinal cord outline utilized in Figure A is slightly rostral to the lesion, while that of Figure B is cord marginally caudal to the lesion site. The photograph (C) shows a portion of the lesion produced in A.



A



B



C

FIGURE 29

Postoperative electromyographic records of flexor (ITC) and extensor (FCL) muscle activity during treadmill walking following the chronic Ventromedial Intact spinal cord lesion. The EMG records indicate normal hindlimb walking by both legs. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

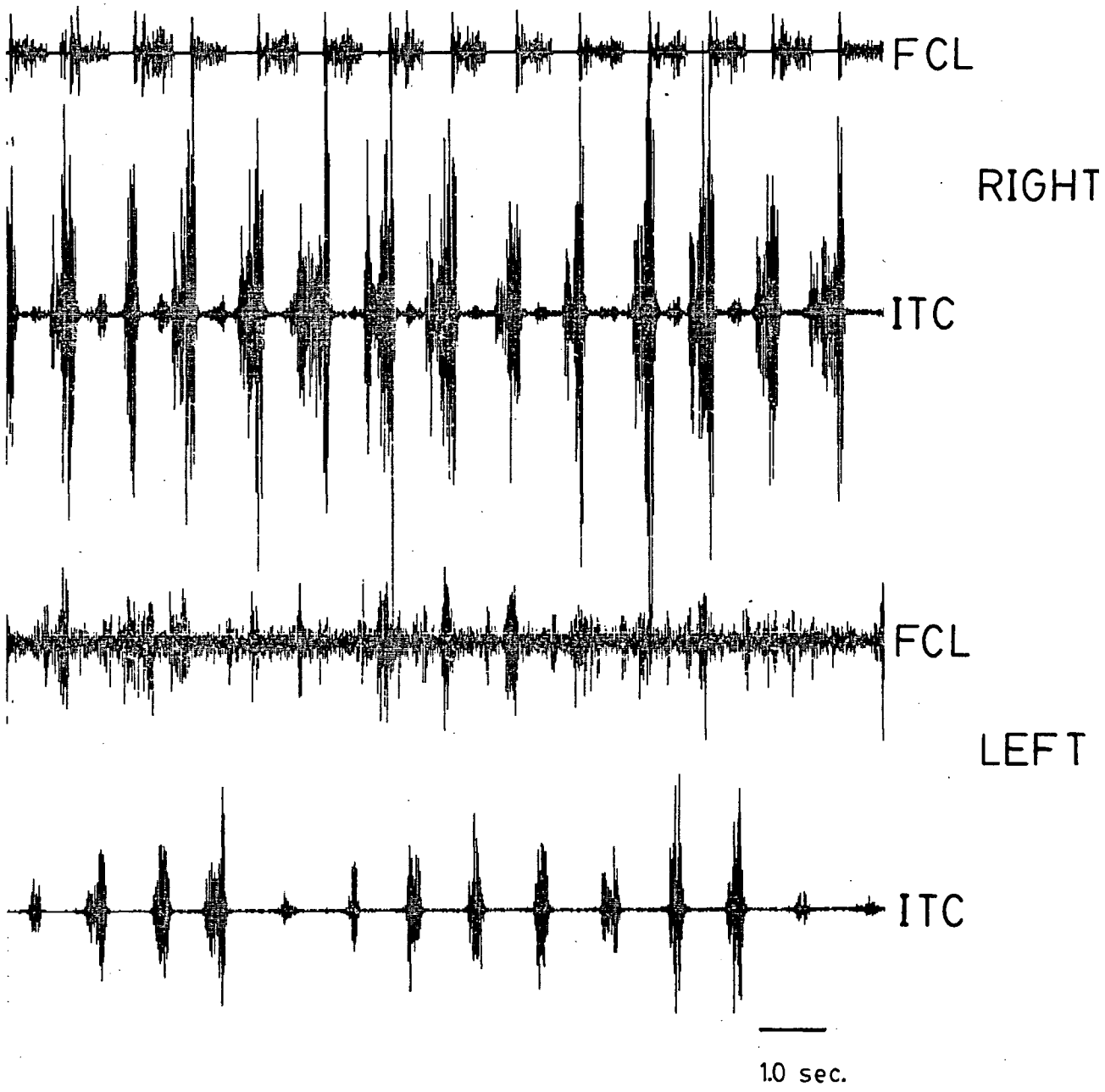
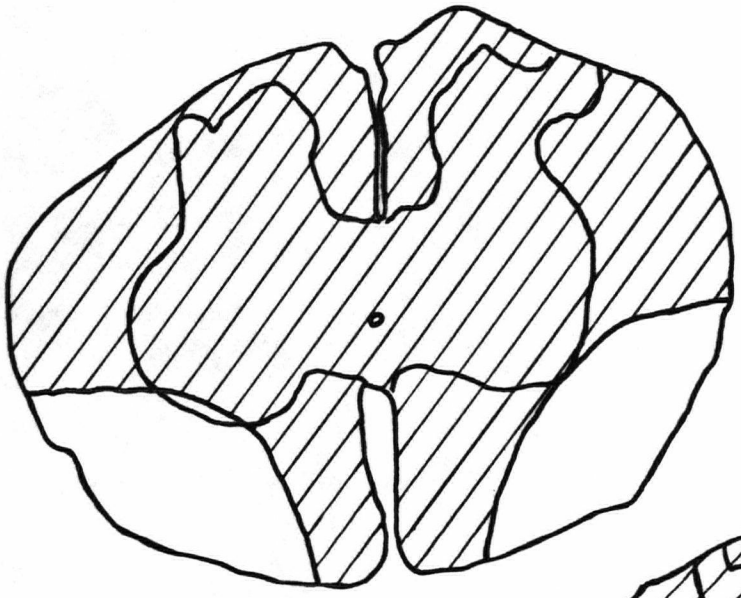
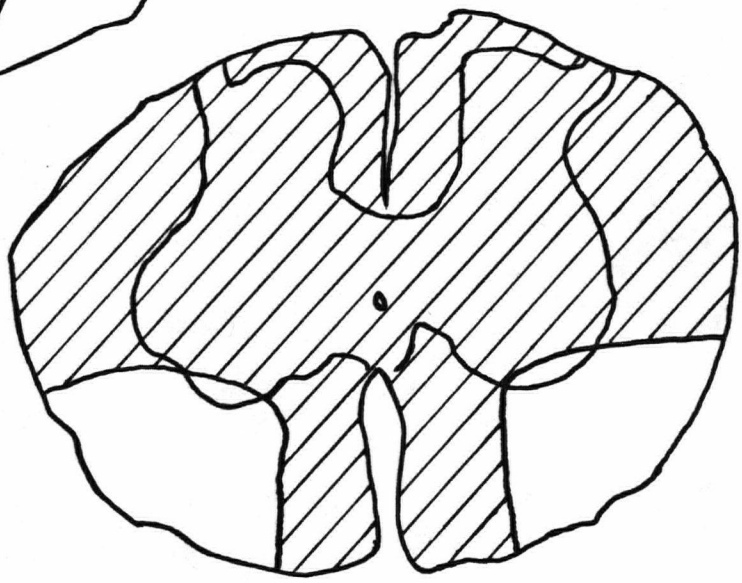

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 30

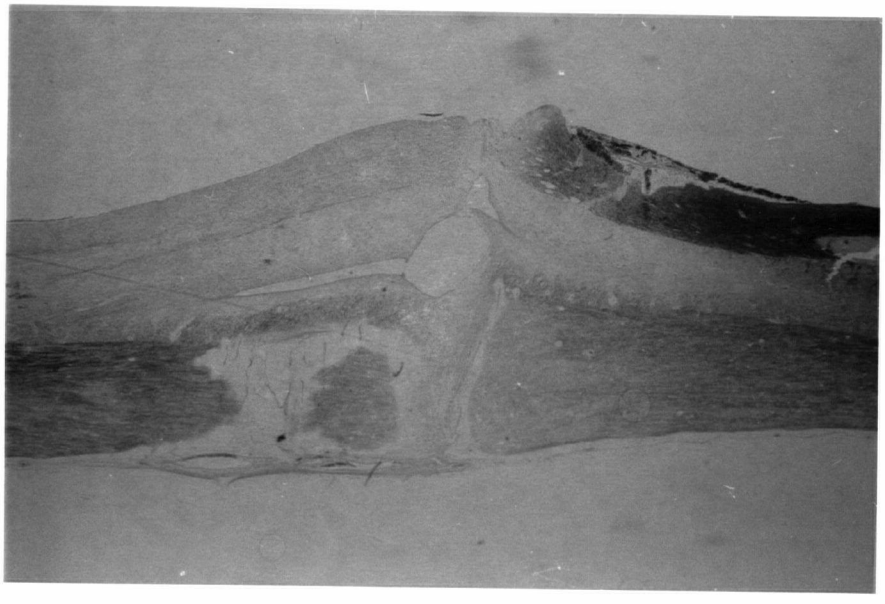
Diagrammatic representation of cross sections through the low thoracic spinal cord following the Bilateral Ventrolateral Intact lesion. The lesion extents in both A and B were determined by examination of either serial cross (A) or serial longitudinal (B) sections through the lesion sites. Lined areas indicate the extent of lesioned spinal cord. Figure C is a photograph of a longitudinal section through the lesion. Note the completeness of the dorsoventral lesion in this medial cord section. Abbreviations: cc= central canal, D= dorsal, V= ventral.



A



B



C

FIGURE 31

Electromyographic records of the goose hindlimb flexor (ITC) and extensor (FCL) muscles during treadmill walking. The locomotion EMG results demonstrate the normal alternating activity of antagonistic hindlimb and contralateral homologous muscles following a Bilateral Ventrolateral Intact lesion. Abbreviations: FCL= flexor cruris lateralis, ITC= iliotibialis cranialis.

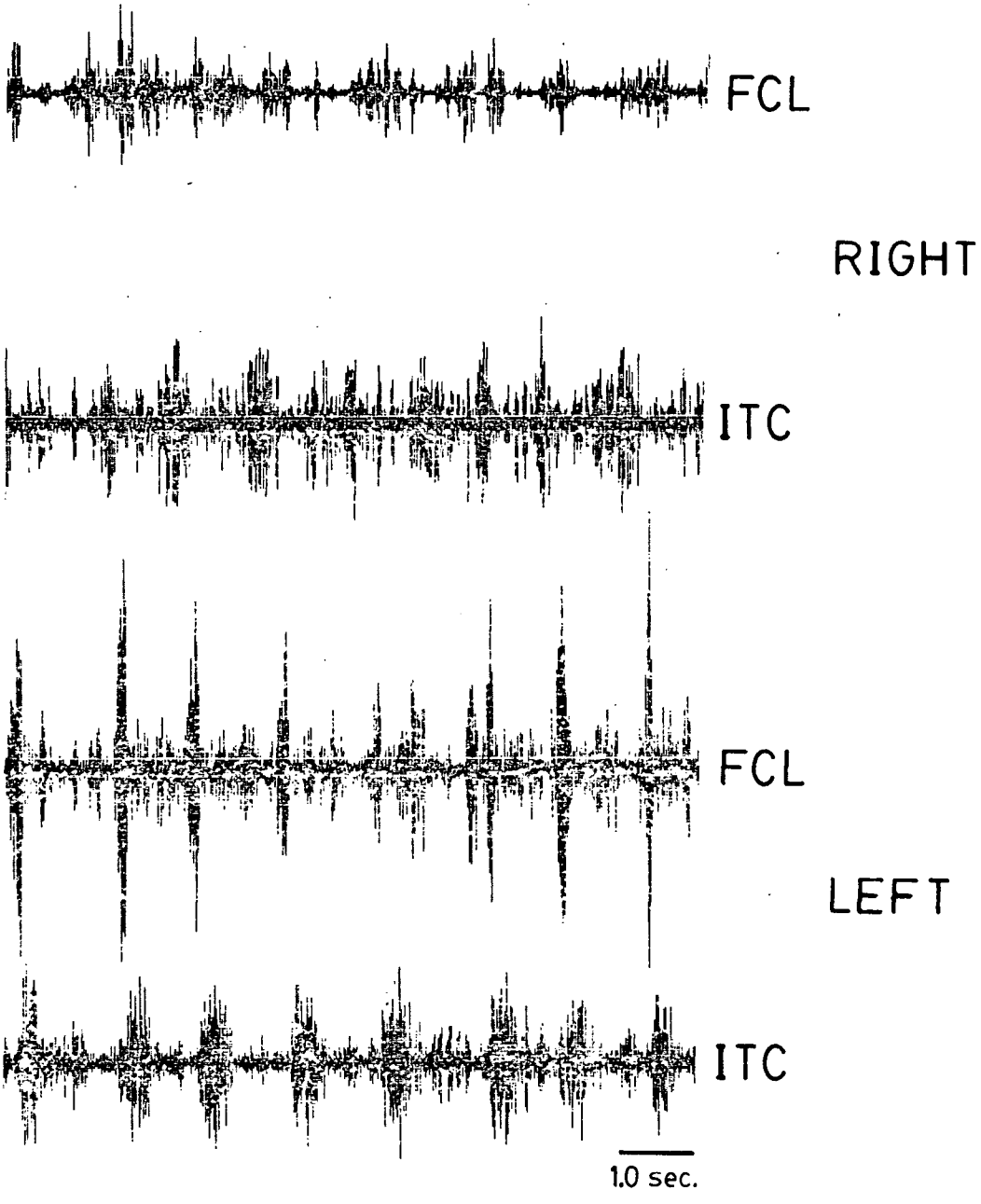

FCL
RIGHT
ITC
FCL
LEFT
ITC
1.0 sec.

FIGURE 32

Composite cross sectional diagram of the low thoracic spinal cord of a goose following a Ventral Quadrant Intact lesion. The lesion extent, which is marked by the lined area, demonstrates that only portions of the ventral quadrant remained intact.

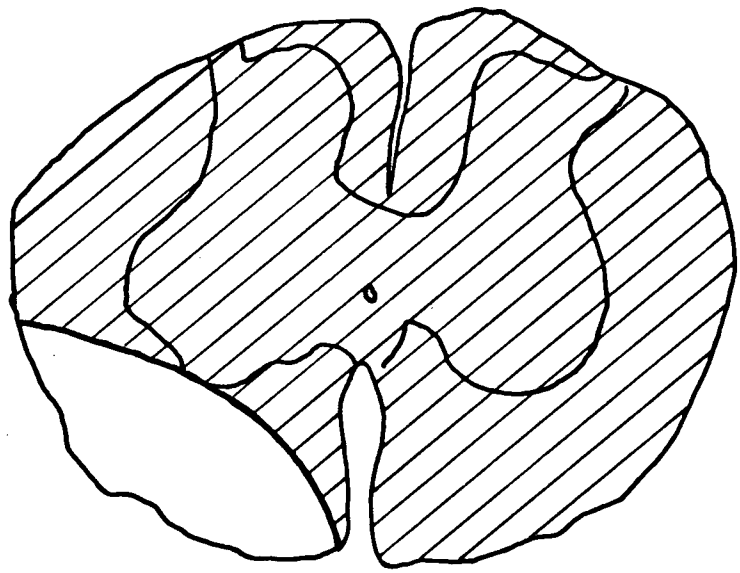


FIGURE 33

Postoperative electromyographic records of flexor (ITC) and extensor (FCL) muscles of the hindlimb during supported treadmill walking. The EMG records show alternating activity of the antagonistic muscles (ITC/FCL) for both hindlimbs. While brief periods of alternating hindlimb stepping were observed, no EMG data was obtained during supported treadmill walking.

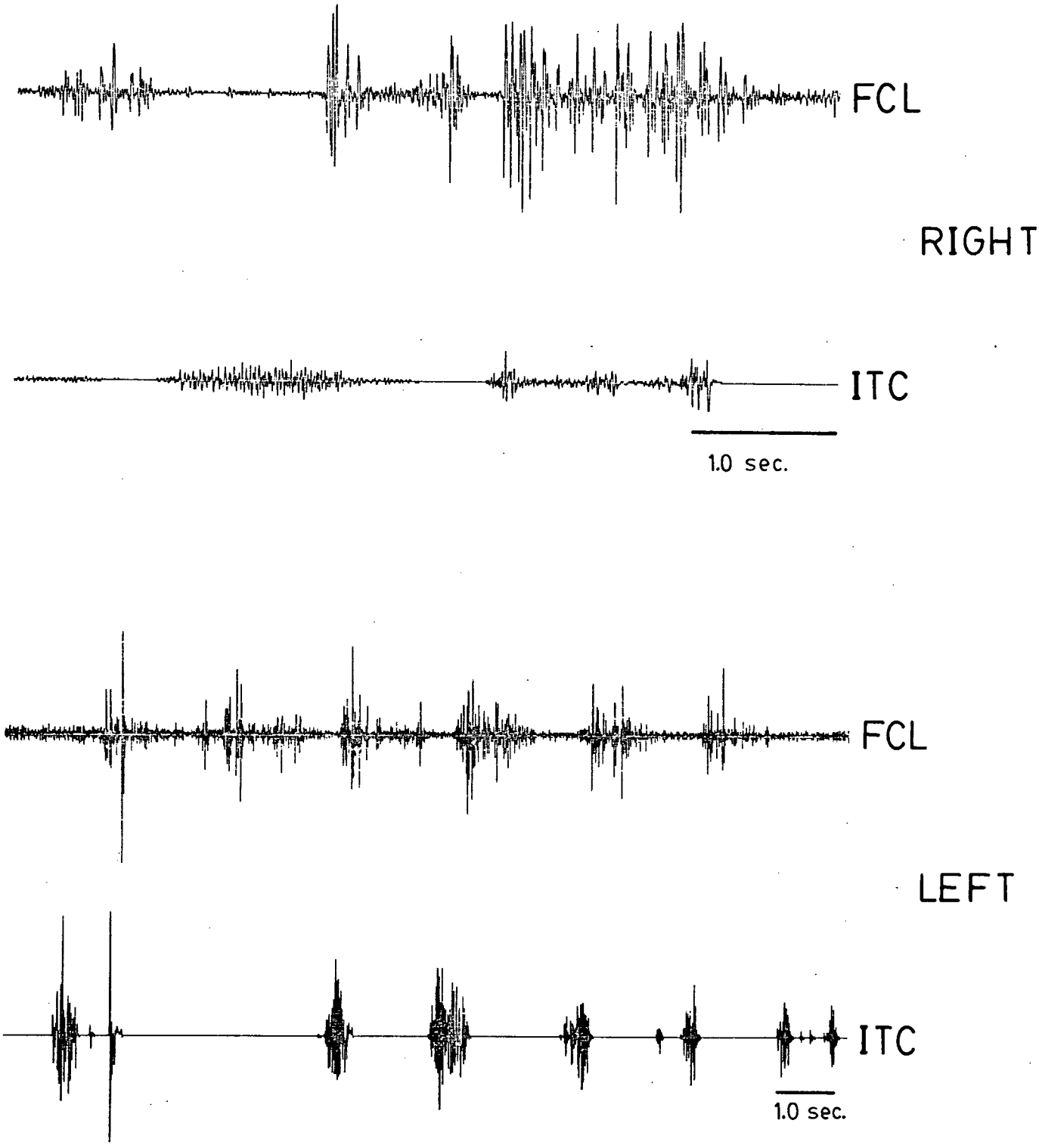

FCL
RIGHT
ITC
1.0 sec.
FCL
LEFT
ITC
1.0 sec.

FIGURE 34

Diagammatic representation of a cross section through the avian brachial spinal cord showing several trajectories of descending bulbospinal pathways. (Redrawn from Cabot et al., 1982)

Area 1= hypothalamo (PVM)-spinal

Area 2= periaquaductal gray (interstitio)-spinal

Area 3= rubrospinal (crossed projection)

Area 4= dorsolateral pontine-spinal

4a= not known

4b= N. subcoeruleus dorsalis (SCd)-spinal (ipsilateral)

Area 5= medial pontine (RPgc/RPO)-spinal (ipsilateral)

Area 6= vestibulospinal (VeD,VeLd,VeLv,VeM)

Area 7= raphe (medial medullary/caudal pontine)-spinal

7a) N. raphe magnus (alpha and beta)-spinal

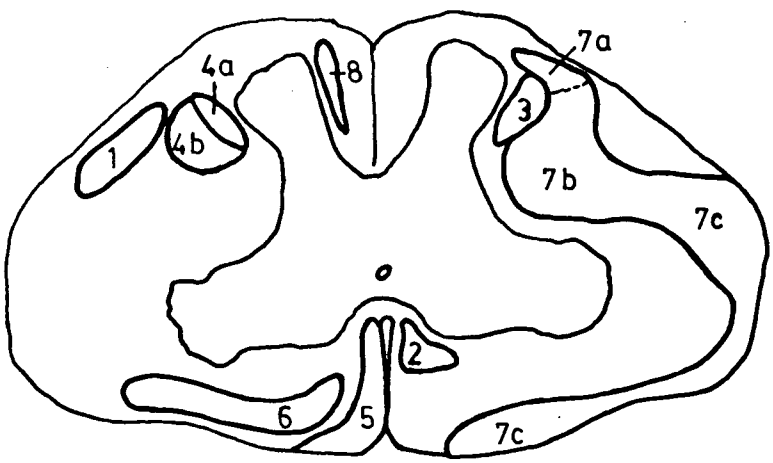
7b) N. raphe pallidus-spinal (rostral part)

7c) i) N. raphe pallidus and obscurus-spinal (caudal portion of nuclei)

ii) medial medullary reticulo-spinal (Rgc) (ipsilateral)

Area 8= dorsal columns nuclei-spinal

Abbreviations: PVM= nucleus (n.) periventricularis magnocellularis, Rgc= n. reticularis gigantocellularis, RPgc= n. reticularis pontis caudalis, pars gigantocellularis, RPO= n. reticularis pontis oralis, VeD= n. vestibularis descendens, VeLd= n. vestibularis lateralis, pars dorsalis, VeLv= n. vestibularis lateralis, pars ventralis, VeM= n. vestibularis medialis.


4a
8
7a
3
4b
1
7b
7c
2
6
5
7c

CHAPTER III

THE PRODUCTION OF LOCOMOTION RESULTING FROM ELECTRICAL STIMULATION OF THE BRAINSTEM IN THE ACUTE DECEREBRATE BIRD

INTRODUCTION

Electrical stimulation of regions in the diencephalon (Subthalamic Locomotor Region, periaquaductal gray (Garcia-Rill et al., 1983a)), mesencephalon (MLR (Shik et al., 1966)), hindbrain (ponto-medullary locomotor strip (Budakova and Shik, 1980)) and spinal cord (Jacobson and Hollyday, 1982b; Williams et al., 1984) produces locomotion in a variety of animals including stingray, chick and cat and in many different types of preparations such as spinalized, decerebrate, thalamic and mesencephalic. Physiological and anatomical tracing studies of spinal and supraspinal locomotion promoting structures indicate the possibility that two or more areas may independently effect this descending control (Garcia-Rill et al., 1983b: Steeves and Jordan, 1984). However, some controversy exists as to the relative importance of these areas, their supraspinal interconnections, and their interface with descending pathways controlling locomotion.

The present study was undertaken to locate and examine the regions in the avian brainstem which, when electrically stimulated, evoke locomotion. The avian brain provides a more simple system in which to study evoked locomotion. Utilization of this model system may alleviate some of the controversy surrounding mammalian "controlled" locomotion studies. The anatomical classification and physiological behavioural definition of locomotion-promoting stimulation sites in the avian brain facilitate further studies characterizing the neural control of locomotion.

MATERIALS AND METHODS

Adult Canada geese, adult and adolescent Pekin ducks and Pekin/Mallard cross ducks maintained in an outdoor enclosure were placed in a preoperative holding room without food twenty four hours prior to surgery. All surgery prior to halothane anaesthesia was performed under local anaesthetic (Xylocaine hydrochloride 2%). Both wing vein (brachial or ulnar) and artery (brachial or ulnar) were cannulated (PE 100 and PE 90 Intramedic tubing) after reflection of the overlying skin. Care was taken to prevent damage to the ulnar nerve which apposes the artery. The cannuli were sutured tightly into position before suturing of the skin (000 silk). Blood pressure was monitored from the arterial cannula. A tracheotomy tube was implanted at the mid-cervical level and the internal carotid arteries were surrounded with thread for later ligation. Care was taken to prevent blood and other fluids from infiltrating the air sacs located in this region. A second larger tube was sutured into the anterior air sac to allow for unidirectional ventilation (UDV) of the animal under halothane (Fluothane, Ayerst) anaesthesia (Fig.35). The unidirectional flow (95%O2/5%CO2)(flow rate 1.5-2.4 l/min.) was established through the tracheotomy tube and throughout the body before exhausting from the anterior air sac. Expelled gas was collected in an Ehrlenmeyer flask to monitor any ventilatory dehydration. The gas was then passed through a cold water jacket distillation chamber and flushed in running water.

The eye nictating membrane reflex, blood pressure, response

to foot web pinch and independent breathing were all criteria utilized to measure depth of anaesthesia. The two methods which proved most reliable were; 1)breathing, which stopped when the animal was deeply anaesthetized and 2) foot web pinch, which did not elicit a reflex response when depth of anaesthesia was sufficient. Blood pressure (BP) was not reliable as a measure of anaesthesia, for after an initial depression (in mammals, BP is depressed under anaesthesia) it returned to almost normal awake values. Heart rate was increased over normal values. The nictating membrane response was also unreliable with halothane, for its speed fluctuated widely in animals which by other criteria were deeply anaesthetized. The halothane concentration required to maintain anaesthesia varied considerably between and within animals. Induction levels were standardized at approximately 0.5% with maintenance levels varying between 0.05% and 0.1%.

Once anaesthetized, the animal was suspended in a stereotaxic apparatus (Narashige) over the treadmill with both legs and wings unrestrained. The ears were infiltrated with local anaesthetic and stereotaxic ear bars were fixed into the auditory canals. The beak was fixed to the stereotaxic nose piece with a clamp. This method produced a reliable, replicable method of insuring the relative uniformity of head and brain position in the stereotaxic apparatus for each species. Brain position appeared to be relatively independent of bird size. Within a species, size did not not vary substantially.

A craniotomy was performed to expose the rostral telencephalon to the root of the olfactory nerve. Enough bone was

removed to reveal the ventrolateral aspect of the telencephalon. The bone overlying the caudal cerebellum almost to the level of the foramen magnum was also removed. Great care was taken not to disturb the superior sagittal and transverse sinuses when one exposed the dorsal midline and rostral cerebellum, respectively, as puncture of these sinuses produced severe bleeding. Care was taken also in maintenance of the dura mater, for fragile surface vessels underlying this structure were prone to bleeding.

"Nipride" (Sodium Nitroferricyanide (0.144 ug/ml) in 5% dextrose), a vasodilator, was infused (Harvard Infusion Pump)(rate: 0.069-0.276 ml/min.) via venous cannula to reduce blood pressure and minimize bleeding during decerebration (G. Gabbott, personal communication). The internal carotids in the neck were ligated to reduce blood flow to the brain (J. Steeves, personal communication; Steeves and Jordan, 1980a).

The dura was reflected from the brain surface. The superior sagittal sinus was ligated, cut, and reflected rostrocaudally to expose the entire dorsal and lateral surfaces of telencephalon and cerebellum.

Two methods of decerebration were attempted. Initially, each telencephalic lobe was lifted and excised using a spatula inserted at the thalamic/telencephalic border. The instrument was directed rostrally with a horizontal orientation to the anterior cranium dorsal to the optic chiasm. With both lobes removed, the bleeding was controlled utilizing cotton swabs, Gelfoam (absorbable gelatin sponge, Upjohn) and cotton batting. The second procedure, which proved more successful, involved modifying the above procedure by cauterization of the attendent telencephalic vasculature before

excision of the lobes. This proved to be a reliable method for preventing subequent severe blood loss in the decerebrated animal.

After controlling the bleeding, the animal was removed from "nipride" allowing recovery of blood pressure to relatively normal levels. The animal was slowly removed from anaesthesia but ventilation was continued. In most cases, the bird began to breathe independently. Subsequently, the ventilation was removed after blockade of the anterior air sac ventilation tube. The blood pressure (BP) typically remained slightly below normal intact values and stroke volume was marginally damped. There was considerable variation in heart rate, BP, and stroke volume depending on the preparation.

Further surgical intervention, as in the case of the post-decerebrate thoracic cord lesion, was performed by replacing the animal under halothane as described above. The surgical procedure was performed in the acute animal, using the same method as described for the chronic animals in Chapter II.

Following a short recovery period, the bird was implanted with EMG electrodes into muscles of the hind and forelimb using the method outlined in Chapter I. A movement potentiometer was attached to each leg to monitor leg movement excursion and frequency. Leg movement potentiograms were recorded on magnetic tape and chart recorder.

The stimulating electrode (SNE 300, Kopf) was mounted in an electrode carrier (Narashige H1) placed on the stereotaxic apparatus. A ground electrode was clipped to the skin overlying the craniotomy. Single square wave stimulus pulses (Grass S88)

were modulated through a constant current unit (Grass CCU 1A) to ensure uniform current intensity. Stimulus intensity necessary to elicit locomotion and other behaviours varied from 25 microamps (uA) to 200 uA and stimulus frequency varied from 30-50 Hertz (Hz). Pulse duration of 0.3 milliseconds (msec) and delay of 0.01 msec remained uniform throughout all experiments.

Stimulation sites were initially defined by trial and error in areas which are known to evoke locomotion in cats. Subsequently, stereotaxic coordinates were established which allowed some measure of certainty in finding a locomotion promoting site (see Results). Stereotaxic zero was designated at the midline as the most rostral cerebellar border visible on the dorsal brain surface. A behaviour was deemed "evoked" if it initiated with stimulation onset and terminated when the stimulation was removed. Verification and monitoring of behaviours which were stimulation evoked was either by visual observation, EMGs, or movement potentiometers (MPs). In some experiments, walking movements were recorded with potentiometers which gave information concerning limb excursion frequency and pattern. Walking was also characterized utilizing EMG electrodes implanted into the ITC flexor muscles of the hindlimb as established in Chapters I and II. Flying movements were determined by EMG recording from the Pectoralis muscle of the wing and by visual observation.

A site which evoked locomotion was marked with an electrolytic lesion (lesion parameters: 1-5 milliamps (mA) DC - duration 5-8 sec or 30-50 uA DC - duration 30-50 sec) for future histological verification.

Each animal was sacrificed with pentobarbitol (i.v. bolus). The brain was removed and preserved in 10% formalin PBS before washing, dehydration in alcohols and infiltration with wax (Paraplast or Paraplast +). Thin cross or sagittal sections (12-20 um) were cut on a microtome (every tenth section in absence of lesion and serial sections through the lesion site). Sections were floated on water onto chrom alum double subbed glass slides, and stained with Cresyl Violet/Eosin or Luxol Fast Blue G/Neutral Red before coverslipping. Histological verification of lesion sites was done by visual observation in the stained sections using a stereo dissecting microscope. Anatomical definition of the sites evoking locomotion utilized the avian stereotaxic atlases of Zweers (1971) and Karten and Hodos (1967).

RESULTS

Transection Level

The level of decerebration for each animal is demonstrated in Fig. 36 (n=11). Excision of telencephalic structures rostral to the line shown in Fig. 36, Level 1, named the decerebrate preparation (Wetzel and Stuart, 1976), maintained the integrity of the optic chiasm and the thalamus, although a certain amount of damage to these underlying structures probably resulted from the surgical procedure. Characteristics of these preparations following anaesthetic removal, but prior to electrode implantation and electrical stimulation, included an elevated heart rate, decreased systolic/diastolic amplitude and slightly decreased blood pressure. Reflex withdrawal to foot web pinch was elicited but no stepping movements could be produced from tactile stimulation of the foot dorsum by the operating treadmill belt. With the exception of Expt. 11, a high decerebrate preparation (with probable retention of telencephalic structures rostral to Level 1 (Fig. 36)), no prestimulation locomotor activity was evident in these animals. Spontaneous prestimulation locomotor activity was recorded for the wings and legs in Expt.11 as shown in Fig. 37. Flying was represented by simultaneous Pectoralis muscle induced depression of the wings (EMGs). The absence of leg movements was recorded by movement potentiometers (MPs).

Animals with the transection shown in Fig.36, Level 2, (n=2),

named the "thalamic" preparation demonstrated short bouts of spontaneous stepping and wing flapping activity. Alternating stepping could be evoked by light tactile stimulation of the foot dorsum or anterior leg and by stimulation from the moving treadmill belt. Treadmill evoked walking tended to follow the velocity of the belt. Wing flapping (flying) usually accompanied the walking. Locomotor movements of both fore- and hindlimb could also be elicited by peri-anal stimulation.

A third level of transection (n=1) shown in Fig. 36, Level 3, the "mesencephalic" preparation, in which both thalamus and hypothalamus were excised, displayed no spontaneous post-operative movements and resembled the decerebrate preparation in response to foot web pinch and tactile stimulation.

Stimulation Sites

Introduction of the stimulating electrode into the brain tissue normally did not produce any visible effects. In several cases, however, translation of the electrode to the most ventral aspect of the brainstem produced a short duration movement which appeared to be more akin to discomfort than to any locomotor pattern. This phenomenon disappeared within a few seconds even when the electrode was left in position.

Electrical stimulation at a variety of sites produced several different behavioral responses. Table 3 lists the results from eleven different experiments utilizing Canada geese (Branta

canadensis), Pekin Ducks (Anas platyrhynchos), and Pekin/Mallard-cross Ducks (Anas platyrhynchos). Animals in Experiments 3 and 7 (Table 3) were "thalamic" preparations. The animal in Experiment 8 was a "mesencephalic" preparation. The remaining experiments demonstrate the "decerebrate" preparation shown in Fig 36, Level 1.

Brainstem stimulation of areas medial to or in the Nucleus et tractus descendens nervi trigemini (TTD)(Expts. 1b, 2a, 3b, 3c, 6b, 12b, 13a, and 13b) (Figs. 38,39) produced walking and flying movements at low (30-100uA/50Hz) stimulus intensities. Stimulation of caudal brainstem structures in or around TTD, with the exception of Expt. 13a, demonstrate both flying and walking or flying and running movements. However, stimulation occasionally resulted in flying movements alone (Expt 13a, Table 3). At higher stimulus intensities, this flying was accompanied by agitated foot movements which did not resemble walking. Foot movement was of higher intensity in the hindlimb contalateral to the stimulation site. Stimulation at the more rostral extent of TTD shows both walking alone and flying alone behaviours. Stimulation of the goose in Expt. 2a (Table 1) elicited flying behaviour with some initial running. This behaviour resembled the crossover point between running and takeoff during normal locomotion in flying birds. In one duck (Table 3, Expt. 12b), stimulation of TTD elicited walking only. Stimulation-induced walking is demonstrated in Fig. 40 with EMG and movement potentiometer records from both legs. The records show alternating stepping movements characterized by increased EMG activity in ITC bilaterally and potentiometer recordings of rostrocaudal excursions of both legs.

Pinioning of the wings during stimulation-induced walking and flying in Expt. 1b demonstrated the ability of the animal to continue its stepping movements in the absence of forelimb locomotion. Holding the legs during bimodal locomotion did not appear to inhibit wing flapping when stimulation occurred in the region of TTD.

Electrical stimulation of the trigeminal nerve principle sensory nucleus at a more rostral pontine level (Expt. 1a, Fig.41) also evoked both flying/running and flying motions at a more anterior site.

It must be noted that the area encompassing the lesion site and TTD in several experiments (Expts.3b,3c,13a) was in close proximity to the substantia gelatinosa Rolandi trigemini (SG). Owing to possible stimulus current spread through the tissue, this area cannot be excluded from putative locomotion producing sites.

A second major area which produced locomotion in the stimulated experimental animals was the Nucleus centralis medulla oblongata, pars dorsalis (Cnd) and pars ventralis (Cnv). This site, illustrated in several experiments (Expts. 2c, 3c, 4, 10a, 11a, 13b) (Fig. 42), produced walking and flying separately (Expt.2c), flying/running together (Expt.3c), flying/walking together (Expt.11a,13b,)(Figs.43,44), walking only (Expt.10a) and flying/leg flexion (Expt.4).

Histological examination of the lesion sites in these trials shows the majority of dorsal stimulation sites in close proximity to the major stimulation site TTD. This is further delineated in the results recorded in Table 3 (Expt.3c,13b). However, the site

shown in Expt.10a, (Fig. 42) is definitely not within the boundaries of TTD and lies more ventrally between the dorsal and ventral parts of the medullary central nucleus. It is possible that the stimulation site shown by the lesion in Fig. 42 borders the lateral reticular nucleus, for examination of the stereotaxic atlases of Karten and Hodos (1967) and Zweers (1971) do not clearly differentiate between the central medullary and lateral reticular nuclei. The flying/leg-flexion behaviour evidenced in Expt. 4 reflects the presence of a chronic low thoracic spinal cord lesion produced in the bird prior to this acute brainstem stimulation study. The animal, during the experimental period, displayed only rudimentary hindlimb locomotor capability prior to decerebration and was not able to support itself during overground locomotion. The wings of the animal were unaffected by the thoracic lesion.

A stimulation site (Fig. 45) in the lateral reticular nucleus (RL) (Expts. 2b,6a,10a) evoked flying and running movements at low stimulus intensity thresholds. Stimulation at two caudal areas (Expts.2b,6a) produced flying combined with running movements and at a third site produced walking only (Expt.10a) in the duck. Stimulus spread from the lesion site in RL to the more medial N. centralis medulla oblongata, pars ventralis (Cnv) was a possibility. Structures which were close to the stimulation site and therefore cannot be discounted as being possible areas which elicit locomotion include the ventral (VSCT) and dorsal (DSCT) spinocerebellar tracts (Expt.6a) and Cnd/Cnv (Expt.10a). While VSCT and DSCT are not repeatedly represented, stimulation of the area surrounding Cnd/Cnv has already been shown to produce

locomotion.

Stimulation of the midbrain structures near the lateral and medial spiriform nuclei and Ansa lenticularis (Table 1, Expt.12c,12d, Fig.46) produced walking at low and flying at higher stimulus intensities. Stimulation of the contralateral side (same coordinates) with no histological verification replicated this result. Other areas close to the stimulation site include the N. hypothalamicus posterior, pars lateralis and the lateral geniculate nucleus.

Electrical stimulation of an area encompassed by the medial lemniscus, Cnv, and close to N. raphe (Expt.8, Fig.47) in the medulla produced walking in the absence of any flying movements. An increase in current stimulus intensity produced increased force of stepping but no frequency change. Increase in treadmill velocity produced increases in footfall frequency. EMG and MP data (Fig.48) show rhythmic alternating stepping movements in both hindlimbs.

Stimulation Parameters

Changing stimulation parameters during evoked locomotion were examined in Expt.12. Increasing treadmill belt velocity, (Fig.49) holding electrical stimululation parameters constant, resulted in an increase in stepping frequency without an increase in excursion length. Increasing stimulation frequency did not produce a signifigant change to stepping behaviour when the belt was held at

constant speed. An increase in stimulation strength produced increases in the frequency of stepping (Fig. 50). An increase in current intensity was also observed to produce an increase in the force of stepping.

Acute Spinal Cord Lesions

Attempts to determine the location of spinal cord pathways carrying descending information during acute experiments was in part reflected by the results from Expts. 6 and 12. In Expt. 6, a prelesioned chronic animal (dorsal columns lesion extent Fig. 51) was decerebrated before stimulation. The hindlimb locomotor capability of this animal was severely restricted. Stimulation of the bird elicited normal wing flapping (flying) movements and some degree of hindlimb flexion. These observations reflected the pre-decerebrate locomotor capacity of the animal. In Expt. 12, following establishment of a stimulus site inducing a locomotor behaviour in a Pekin duck, an acute low thoracic Dorsal Cord Transection was performed (lesion extent Fig. 52). Acute post-transection stimulation at the same stimulus site resulted in normal alternating treadmill stepping initially at higher stimulation intensity and shortly thereafter walking at lower threshold (Table 1, Expt. 12b). This behaviour is documented utilizing EMG and MP records shown in Fig. 53.

DISCUSSION

Electrical stimulation of regions in the diencephalon, midbrain, hindbrain, and high cervical spinal cord has been shown to produce "controlled" locomotion in a variety of species (Kashin et al., 1974; Shik et al., 1966,; Jacobson and Hollyday, 1982; Williams et al., 1984; Eidelberg et al., 1981; McClellan, 1984). Stimulation in the area of the subthalamic nucleus (STN) (the Subthalamic Locomotor Region (SLR)) produces locomotion in the lightly anaesthetized cat (Waller,1940) and the "thalamic" cat (Orlovsky, 1969). Stimulation of the posterior substantia nigra elicits less normal walking in the precollicular postmammillary cat (Garcia-Rill, 1983). Stimulation of two nuclei in the mesencephalon, the pedunculopontine nucleus (PPN) and cuneiform nucleus (CFN), elicits locomotion in the precollicular-postmammillary cat (Shik, Severin, and Orlovsky, 1967). These two distinct nuclei are thought to be the anatomical correlates of the previously physiologically defined Mesencephalic Locomotor Region (MLR) (Garcia-Rill, 1983). Electrical stimulation of the pontobulbar locomotor strip (PLS) evokes locomotion in the precollicular postmamillary cat (Mori et al., 1977; Budakova and Shik, 1980) and high cervical cord stimulation will evoke bilateral locomotion in lower vertebrates (Jacobson and Hollyday, 1982b; Williams et al., 1984). The results of this study indicate that correlates of several of these supraspinal structures also exist in avians.

Transection Level

The level of transection affects the prestimulation locomotor capabilities of the experimental birds. Transection at the level shown in Fig. 36, Level 1, leaving portions of the thalamus and hypothalamus intact, produces an animal devoid of any spontaneous activity. A comparison of the exact level of transection between mammals and avians is difficult to discern due to the absence of the mammalian corpora quadrigemina and mammillary bodies in birds. However, the anatomical correlates between these animals indicate that this level of decerebration in birds is comparable to the cat thalamic preparation of Orlovsky and Shik (1976). Hinsey et al. (1930) reported that cats can walk almost normally following this transection if the caudal subthalamus is left intact. Owing to an evident paucity of anatomical information, this researcher cannot ensure the integrity of the subthalamus in the preparations in this study. However, Brauth et al. (1978) report that a possible homolog to the mammalian subthalamic nucleus may be the avian Ansa lenticularis, pars anterior. This structure was anatomically preserved in transections of this study, although there exists the possibility that it may have suffered damage by the surgical procedure. This may account for the absence of prestimulation locomotion in the majority of experiments and would make these transections comparable to the thalamic preparation of Orlovsky and Shik (1976). Support for this latter hypothesis was substantiated in Expt. 11, where more rostral transection of the ventral brain yielded spontaneous locomotion. Further studies are

required to define this problem.

Transection producing a "thalamic" (Fig. 36, Level 2) preparation allowed spontaneous locomotion to occur. Anatomical correlation between cat and avian species indicates that this spontaneously locomoting preparation is comparable to the precollicular-premamillary transection described by Grillner and Shik (1973).

The "mesencephalic" preparation (Fig. 36, Level 3) displayed no activity following the surgical procedure. This transection is similar to the mesencephalic preparation (precollicular-postmamillary transection) of Orlovsky and Shik (1976) and Grillner and Shik (1973) in which acute experimental cats displayed no spontaneous movements excepting when the mesencephalic locomotor region was electrically stimulated.

Presently, there is no substantive body of research to explain how varying transection levels effect locomotion. Possible connections between more rostral areas which are known to effect locomotion such as the entopeduncular nucleus of the basal ganglia and more caudal locomotor regions structures are currently being identified (Garcia-Rill et al. 1983; Steeves and Jordan, 1984). The physiological importance and significance of these connections on changes in spontaneous locomotor ability due to transection level has not yet been determined.

Stimulation Sites

The results indicate that a stimulus site in the region of the lateral/medial spiriform nucleus and the lateral part of the

posterior hypothalamic nucleus produces locomotion in avians. Reiner et al. (1982) and Brauth et al. (1978) have demonstrated a heavy projection from the lateral spiriform nucleus (SpL) to the tectum (layers 11-13). These tectal layers, which do not appear to descend directly to the spinal cord (Cabot et al.(1982), provide a major crossed descending input to the hindbrain reticular formation which gives rise to the reticulospinal pathways thought to effect the initiation and control of locomotion (Reiner et al., 1982). Further, SpL receives a significant input both from the N. ansa leticularis, pars anterior (ALa), which is suggested to be the avian equivalent of the mammalian subthalamic nucleus and the paleostriatum primitivum, the avian equivalent of the mammalian basal ganglia (Reiner et al., 1982). The subthalamic nucleus and hypothalamic nuclei (posterior and lateral) have previouly been implicated as a locomotor region (subthalamic locomotior region (SLR)) in stimulated cats (Orlovsky and Shik, 1976, Steeves and Jordan, 1984). The mammalian basal ganglia have long been believed to have importance to the initiation and control of locomotion in humans and cat (Garcia-Rill et al., 1983a; Neafsey et al., 1978). Bilateral lesions of SpL do not, however, produce deficits in locomotor behaviours (Bugbee, 1979). This indicates that, like the region of the SLR and MLR in cats (Mori et al. 1977; Garcia-Rill et al., 1983b), SpL may effect locomotion but is not essential to its production.

The results from stimulating in the area of the N. et Tractus descendens nervi trigemini (TTD) and the principle sensory nucleus of nerve V (PrV) showed that stimulation in decerebrates elicited

behaviours approaching normal locomotion in intact animals. Shik and Yagodnitsyn (1977) were the first to demonstrate that electrical stimulation of the pontobulbar "locomotor strip" (PLS), an area emanating from the MLR, and travelling in the dorsolateral medullary reticular formation, evoked walking in the cat. Garcia-Rill et al.(1983a) describe the PLS as being equivalent to Probst's tract, a pathway which is intimately associated with the trigeminospinal system. The findings in cat concur with those in the present study that stimulation of this area produces locomotion. The association of stimulus sites initiating hindlimb and forelimb locomotion correlates well with a long rostrocaudal strip which travels in the avian dorsolateral hindbrain reticular formation and which could be the equivalent of the cat PLS. However, the association of Probst's tract, which is not anatomically defined for avians, with the system eliciting locomotion in mammals appears anomalous. Truex and Carpenter (1969) describe Probst's tract as arising from cells of the noradrenergic nucleus Locus Coeruleus. Steeves et al. (1980) depleted both noradrenaline and 5-hydroxytryptamine in the cat without finding effect on MLR stimulation-induced locomotion. One can infer that either the anatomically poorly defined Probst's tract is not responsible for stimulation evoked locomotion or the cells of the Locus Coeruleus do not give rise to this descending bundle. However, evidence from depletion studies is seldom conclusive (Steeves, personal communication). Since MLR inactivation has little effect on PLS stimulation induced locomotion (Shefchyk et al., 1984), locomotion induced by stimulation of the trigeminal system is probably not mediated by

the MLR region and produces its effects via an alternate route. As the trigeminospinal system has not been shown to exist below a high cervical level in humans (Carpenter, 1978) and pigeon (Cabot et al., 1982), it is unlikely therefore that this system controls hindlimb locomotion directly via descending trigeminospinal pathways. The connections through which this area exerts its influence are still unknown.

Electrical stimulation of sites in the mediolateral pons and medulla produced locomotion in the decerebrate goose and duck. In some cases, it was possible that the stimulation included areas of the trigeminal system already discussed, although stimulus spread at the current levels used is probably insufficient to cause stimulation of this site (Bagshaw and Evans, 1976). Also, the location of several locomotion evoking stimulation sites which were more ventromedial, precludes the possibility that they were in the trigeminal area. Stimulation near the central nucleus of the medulla, pars dorsalis and ventralis (Cnd,Cnv) evoked locomotion in the decerebrate bird. This area is continuous with the reticular formation of the more rostral medulla and caudal pons but is cytoarchitectonically distinct from these structures (Cabot et al., 1982). Stimulation in the area of the lateral reticular nucleus at more rostral levels also led to locomotor activity. These results indicate that the above reticular formation structures may be equivalent to those found in the cat, where stimulation of the reticular formation gigantocellular and magnocellular nuclei (Steeves and Jordan, 1984; Budakova and Shik, 1980) produces locomotion.

Stimulation Parameters

The results of changing stimulation parameters correlates well with those found in mammals. Increases in treadmill belt velocity with the maintenance of stimulation strength produces increases in stepping frequency without changes in excursion length (Fig. 49). This corroborates results found in cat (Orlovsky and Shik, 1976), where increasing the velocity of the treadmill belt resulted in a temporal shortening of the step cycle in MLR stimulated animals. Modulation of the stimulation strength produced changes in the strength and frequency of stepping in both extensor and flexor muscles (Fig. 50). As in cats (Orlovsky and Shik, 1976), increasing stimulation strength produced increased force of stepping and a slight increase in step frequency. Orlovsky and Shik (1976) conclude that, at least in the case of MLR stimulation, the stimulation strength determines the strength of stepping and the frequency changes result indirectly from this increase in muscle activity. This hypothesis has not, to my knowledge, been tested in decerebrate paralysed animals where afferent sensory input ("fictive" preparation), which might modulate this result, has been eliminated. These results also correspond to those found in fish, where increasing stimulation in the midbrain tegementum resulted in increasing locomotor behaviours which resembled swimming (Kashin, Feldman, and Orlovsky, 1974). Changing the frequency of stimulation, holding other parameters constant, does not appear to significantly effect the speed or strength of stepping.

Acute Spinal Cord Lesions

Lesions of the spinal cord in acute decerebrate brainstem stimulated birds demonstrate that the dorsal half of the cord is not necessary to the production of locomotion. This is in agreement with previous findings of other experimenters in a variety of chronic and acute experimental animals (Steeves and Jordan, 1980; Eidelberg et al., 1981; Williams et al., 1984) in that sufficient pathways subserving locomotion are present in the ventral hemicord to allow locomotion to occur. It appears from the results in both chronic and acute animals that the reticulospinal pathways may play an important role in the initiation and control of locomotion in avians. However, in order to more precisely determine the importance and location of this and other possibly important information carrying pathways, it will be necessary to incorporate into the acute stimulation experiments a more complete study involving acute lesions of both thoracic and cervical spinal cord.

Conclusion

It is apparent from stimulation studies in the goose and duck that a strong parallel exists between stimulation induced locomotor behaviours in the avian and mammalian systems. In order to further compare these systems, it will be necessary to anatomically as well as physiologically define the locations of

sites evoking locomotor behaviours. Further, it will be necessary to determine anatomically both the origin of the pathway(s) descending to the spinal cord which subserve locomotor behaviours and the connection between these pathway(s) and more rostral structures which could initiate these behaviours.

TABLE III

EXPT.#	STIMULATION SITE	BEHAVIOUR	STIMULATION THRESHOLD
1a)	Nucleus (N.) sensorius principalis nervi trigemini (Anterior (A) 2, Lateral (L) 3)(K&H, A1.00)	-wing flapping, running -wing flapping alone	50uA/ 50Hz
b)	Between dorsal motor nuc. of vagus (solitary tract) and descending trigeminal nucleus and root (P7,L1) (K&H, P3.75) (Zweers, P3.48)	-walking and flapping -when wings held, continued stepping	50-100 uA/50Hz
2a)	Area of N. et tractus descendens trigemini (A1,L4)(K&H, P1.25) (Zweers, P2.32)	-flying with some initial running	75uA/ 50Hz
b)	N. reticularis lateralis (P5,L2)(K&H, P3.25) (Zweers, P3.48)	-running and flying	25uA/ 50Hz
c)	N. centralis medulla oblongata, pars dorsalis (P7,L1)(K&H, P4.0) (Zweers, P4.35)	-walking becomes flying as stimulation intensity increases	50-100 uA/50Hz
3a)	N. vagi dorsolateralis of Zweers, N. nervi glosso-dorsalis nervi vagi (P2,L1.5) (K&H, P1.75)(Zweers, P2.32)	-swallowing response	50uA/ 50Hz
b)	area between N. solitarius, substantia gelatinosa Rolandi (trigemini) and N. et tractus descendens nervi trigemini (P6,L1.5) (K&H, P3.50)(Zweers, P3.48)	-walking and flying	40uA/ 50Hz
c)	area of N. centralis medulla oblongata, pars dorsalis and medial to substantia gelatinosa Rolandi trigemini in area of N. et tractus descendens nervi	-walking and flying	30uA/ 50Hz

	trigemini (P8.0,L1.5)(K&H, P4.0) (Zweers, P5.22)		
4)	N. centralis medulla oblongata, pars dorsalis (P7,L2)(K&H, P3.75) (Zweers, P3.77) (Goose Chronic Expt. 39)	-wing flapping, slight flexion of right leg during flying, vocalization, decreased blood pressure and bradycardia	75uA/ 50Hz
6a)	lateral reticular formation (may also be ventral or dorsal spinocerebellar tracts)(P7,L2.0)(K&H, P3.50) (Zweers, P4.06)	-wing flapping & running	50uA/ 50Hz
b)	N. et tractus descendens nervi trigemini (P7,L1.5) (K&H, P3.50)(Zweers, P4.06)	-flying and running	50uA/ 50Hz
7)	no lesions (Duck)	-spontaneous response to treadmill. wing flapping and walking. Reflex withdrawal to foot web pinch and flying and running response to peri-anal stimulation.	
8)	medial lemniscus,central nucleus medulla oblongata pars ventralis, or raphe nucleus.(K&H, P2.5) (Zweers, P2.61)(Duck)	-walking only, increasing stimulus intensity causes increases in force of stepping but no increase in frequenc, changes in stepping frequency accompany treadmill velocity changes	75-150 uA/50Hz
10a)	N. centralis medulla oblongata, pars ventralis (dorsalis border) and lateral reticular nucleus (P7,L2)(K&H, P3.75) (Zweers, P3.48) (Duck)	-walking only	75uA/ 50Hz
11a)	N. centralis medulla oblongata, pars ventralis (P3.5 K&H)	-walking and flying	75uA/ 50Hz
b)	(P2,L2) no histological verification	-walking and flying	75uA/ 50Hz

12a)	area of lateral reticular nucleus, spinal lemniscus, and medial lemniscus. (P4,L2) (K&H, P3.00) (Zweers, 3.19) (Duck)	-swallowing	75uA/ 50Hz
b)	-between N. reticularis parvocellularis and N. et Tractus descendens nervi trigemini of K&H. -Lateral to N. subcoeruleus and medial to N. radicus descendens nervi trigemini of Zweers (P1,L1) (K&H, P0.25-AP0.00) (Zweers, P0.29)	-bilateral walking following spinal cord dorsal columns lesion walking only no wing movement	100-125 uA/50Hz went to 75uA/ 50Hz
c)	-area of lateral and medial spiriform nucleus and ansa lenticularis of K&H. -N. hypothalamicus posterior, pars lateralis or N. geniculatus lateralis of Zweers. (A3,L1) (K&H, A5.00) (Zweers, A6.38) (Right side)	-walking at low stimulus strength and flying at higher stimulus intensity.	75uA/ 50Hz
d)	(A3,L1) (Left side) no lesion	-walking and flying	75uA/ 50Hz
13a)	substantia gelatinosa Rolandi trigemini and N. et Tractus descendens trigemini (P6,L2) (K&H, P3.75) (Zweers, 4.06) (Left side) (Duck)	-flying only with legs tucked under body. An increase in stimulus intensity increases force of flapping. Agitated foot movements during flying show higher intensity in right leg than in left	60uA/ 50Hz
b)	N. centralis medulla oblongata, pars dorsalis and Plexus of Horsley; also close to N. et radix descendens nervi trigemini (P6,L1) (K&H, P3.75) (Zweers, P4.06) (Right side)	-flying and walking	100uA/ 50Hz

Abbreviations:

(P...,L...)= Stereotaxic coordinates of lesion site using rostral cerebellar border as anteroposterior (AP) stereotaxic zero.

(K&H,....)= Equivalent to stereotaxic atlas level of Karten and Hodos (1967)

(Zweers,..)= Equivalent to stereotaxic atlas level of Zweers (1971)

uA= microamperes

Hz= Hertz (cycles per second)

FIGURE 35

Experimental apparatus utilized during acute avian brainstem stimulation experiments. See text for further information. Abbreviations: b.p.= blood pressure, ccu= constant current unit (Grass), E.M.G.= electromyographic electrodes.

Brain Stimulation Experimental Apparatus

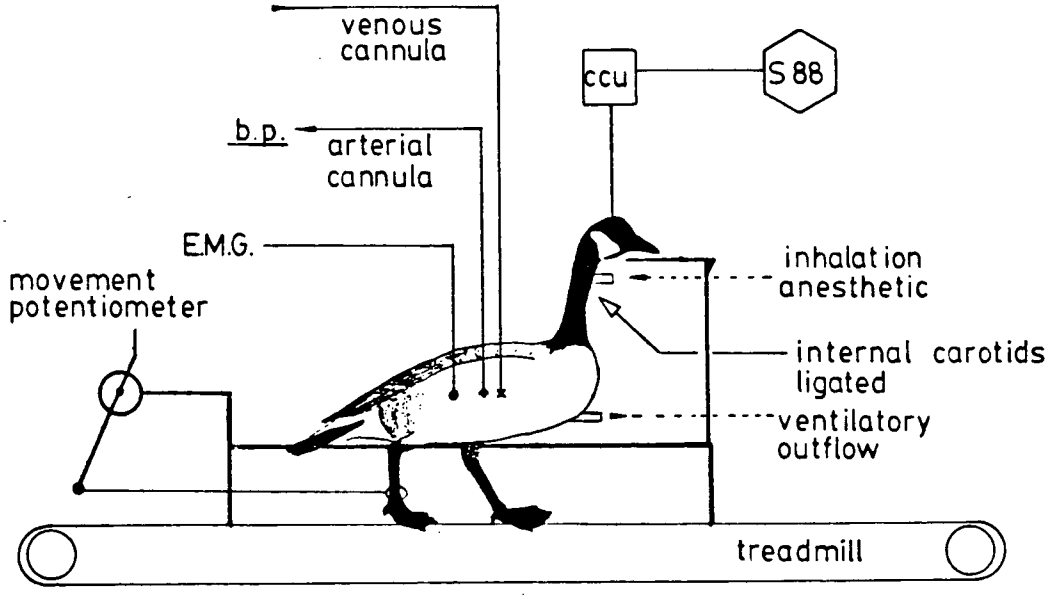


FIGURE 36

Parasaggital section (lateral 0.5) of the brain of the Canada goose (Branta canadensis) showing the levels of transection produced by the decerebration procedure. Level 1- decerebrate preparation, Level 2= "thalamic" preparation, Level 3= "mesencephalic" preparation (additional information is supplied in the text). Also shown are the positions of some anatomically identifiable structures in the avian brain. Lined areas indicate structures lateral to plane of section. Abbreviations: BO= olfactory bulb, Ca= anterior commissure, Cb= cerebellum, CO= optic chiasm, GCt= substantia grisea centralis, Hb= habenular nucleus, IO= inferior olivary nucleus, nIV= nucleus of trochlear nerve, nIX= nucleus of glossopharyngeal nerve, nX= nucleus of vagus nerve, NIII= occulomotor nerve root, Ov= ovoid nucleus, RPgc= nucleus reticularis pontis caudalis, pars gigantocellularis, Ru= red nucleus, SpL= lateral spiriform nucleus, TU= tuberal nucleus, V= ventricle, VeM= medial vestibular nucleus (nomenclature cited from Karten and Hodos, 1967)

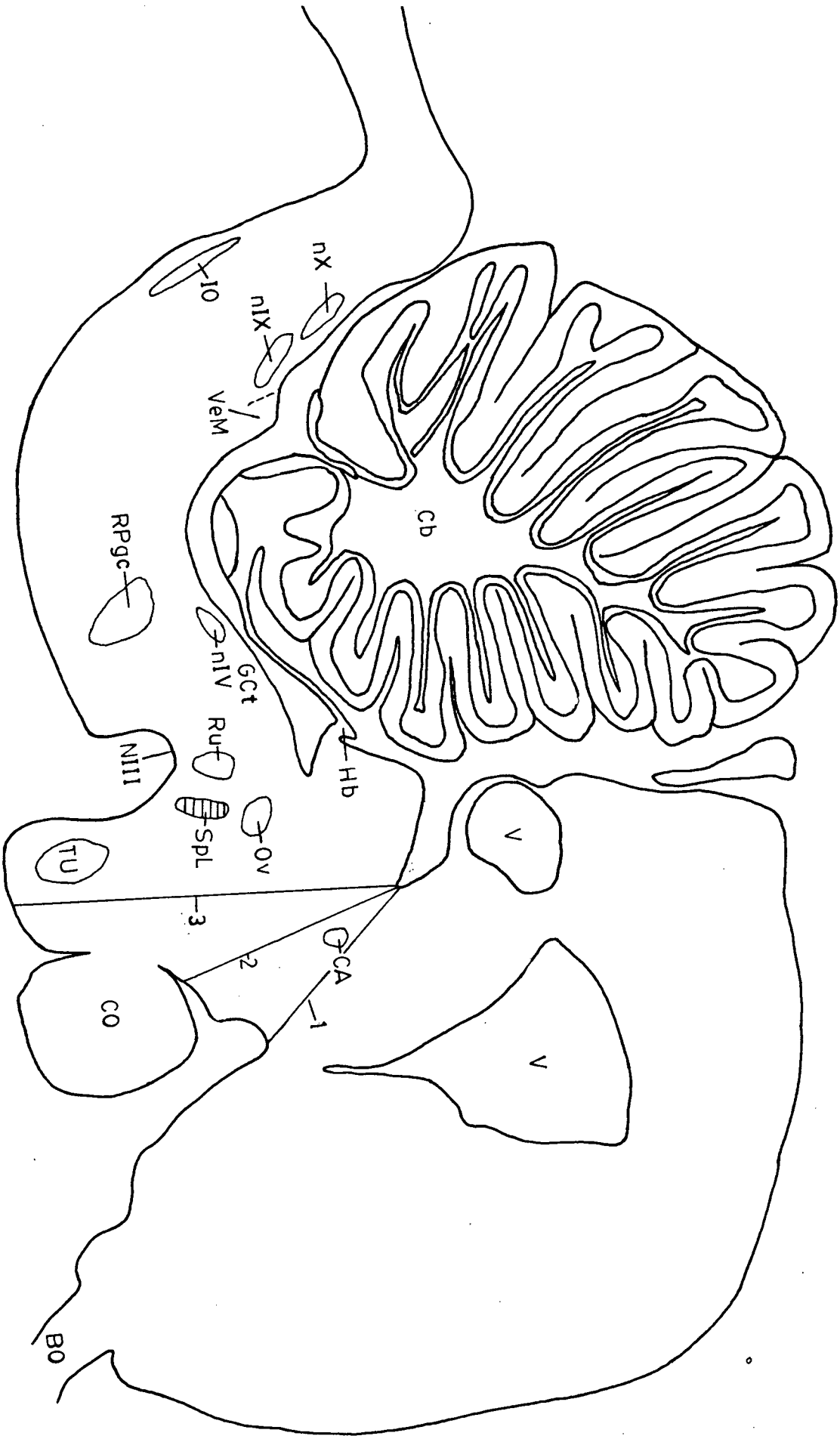

IO
nX
nIX
VeM
Cb
RPgc
GCt
nIV
Ru
NIII
Hb
SpL
Ov
TU
V
3
2
CA
1
CO
V
BO

FIGURE 37

Electromyographic records of prestimulation spontaneous locomotion with the wings (Pect) in the acute decerebrate bird. No hindlimb activity was apparent from the potentiometer records. Abbreviations: L= left, R= right, Hind.= hindlimb, Pect.= Pectoralis

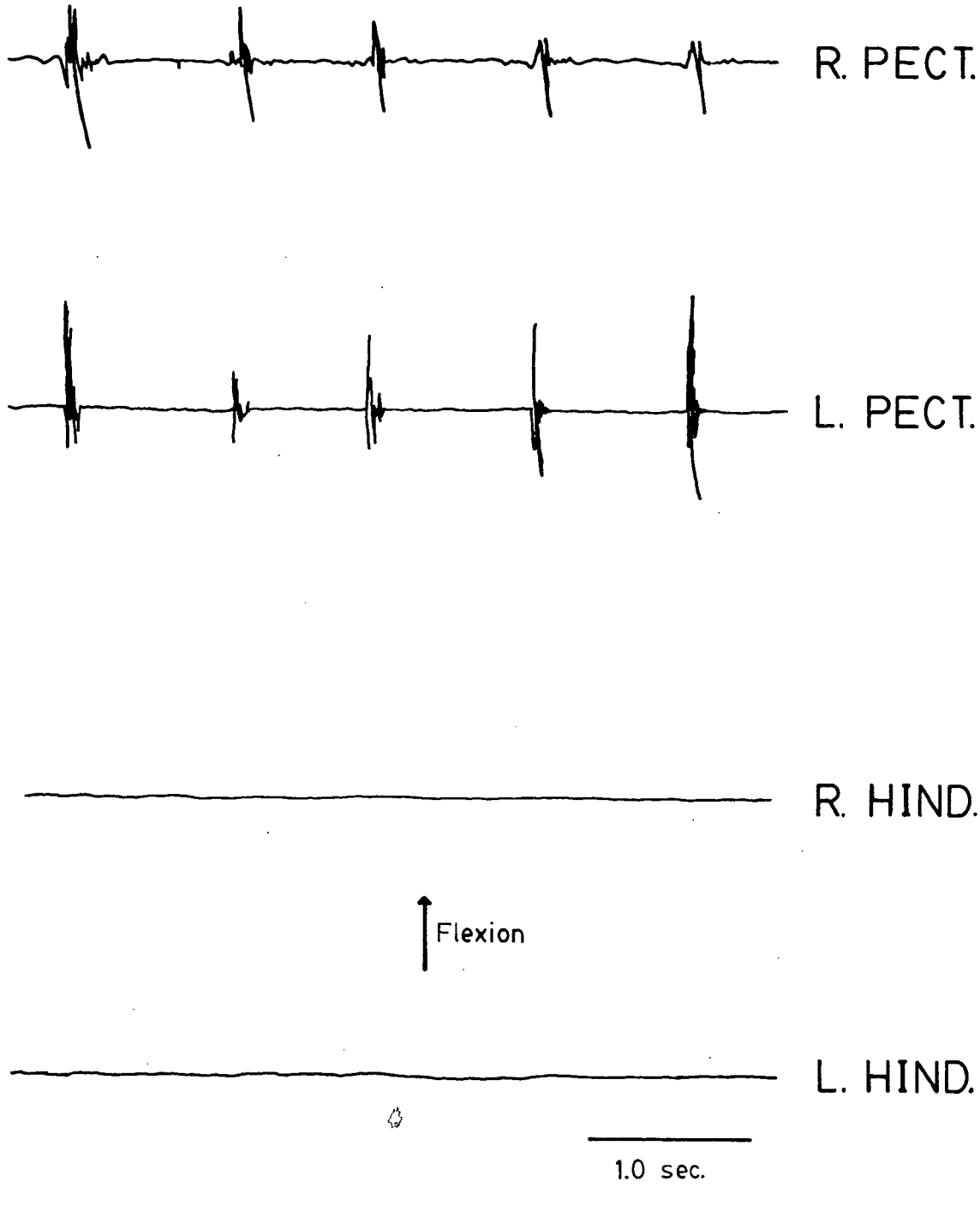
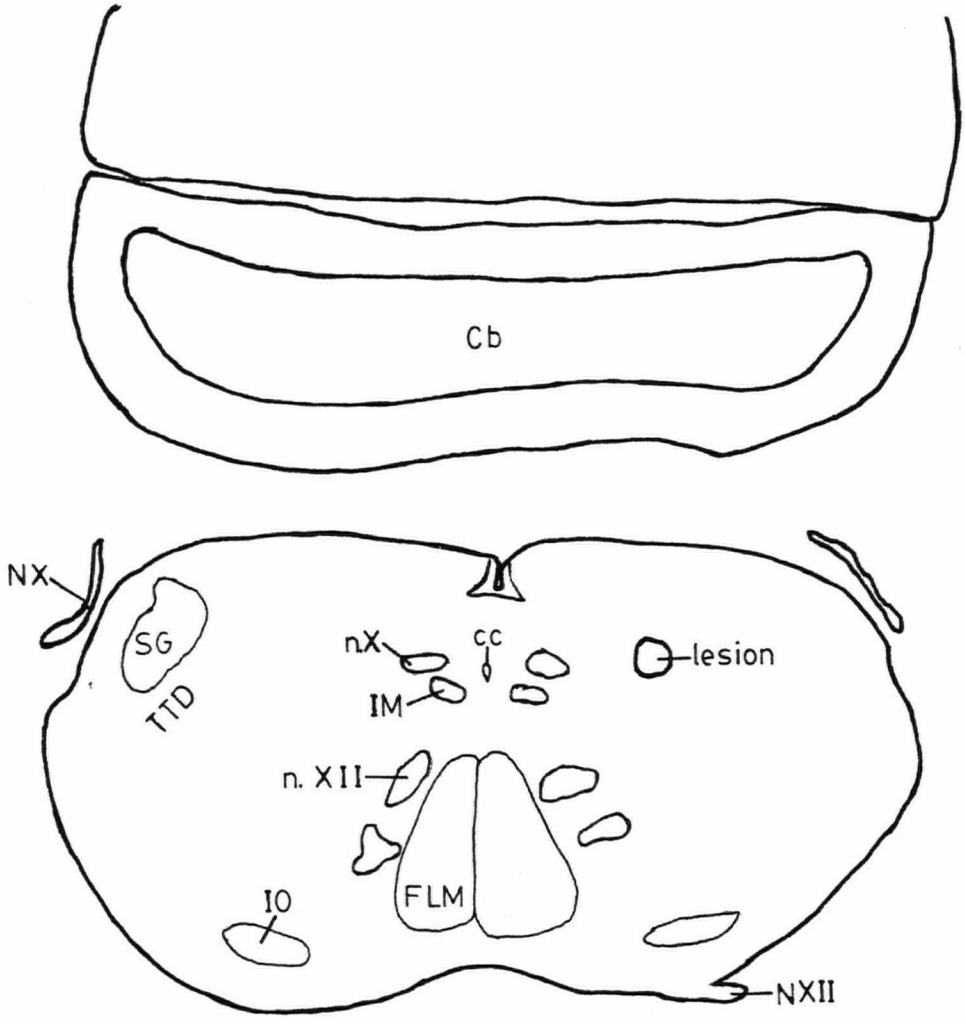
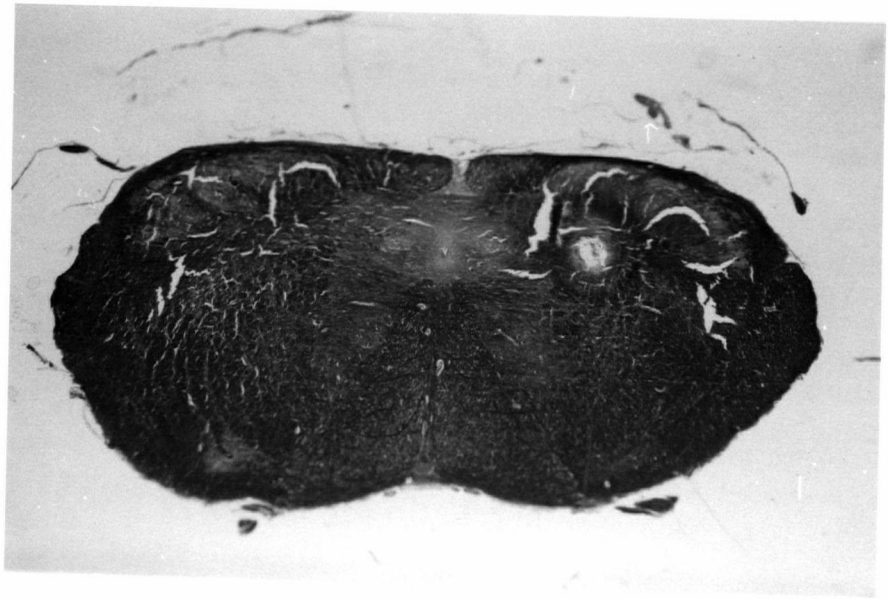

R. PECT.
L. PECT.
R. HIND.
Flexion
L. HIND.
1.0 sec.

FIGURE 38

Diagrammatic representation (A) of a transverse section through the rostral medulla indicating the site of electrical stimulation (lesion site) which evoked walking and flying movements in the acute decerebrate avian of Expt. 6b. B is a photograph of a section through the lesion site. The area of stimulation lies slightly medial to SG and is encompassed by TTD. The section level is comparable to that of stereotaxic coordinate Posterior (P) 3.5 of Karten and Hodos (1967). Abbreviations: Cb= cerebellum, cc= central canal, FLM= medial longitudinal fasciculus, IM= nucleus (n.) intermedius, IO= inferior olivary nucleus, n.X= nucleus of vagus nerve, n.XII= n. hypoglossal nerve, NX= vagus nerve, NXII= hypoglossal nerve, SG= substantia gelatinosa Rolandi (trigemini). (Magnification: 17.5x)



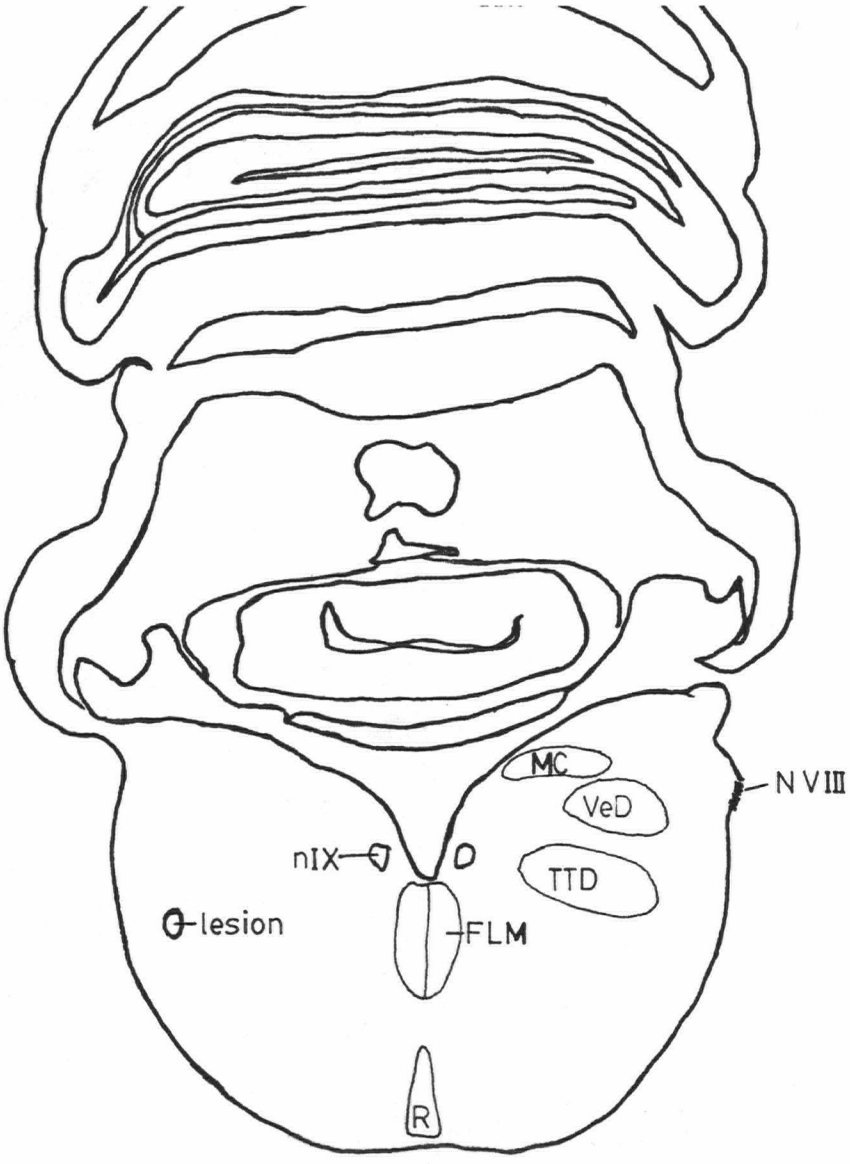
A



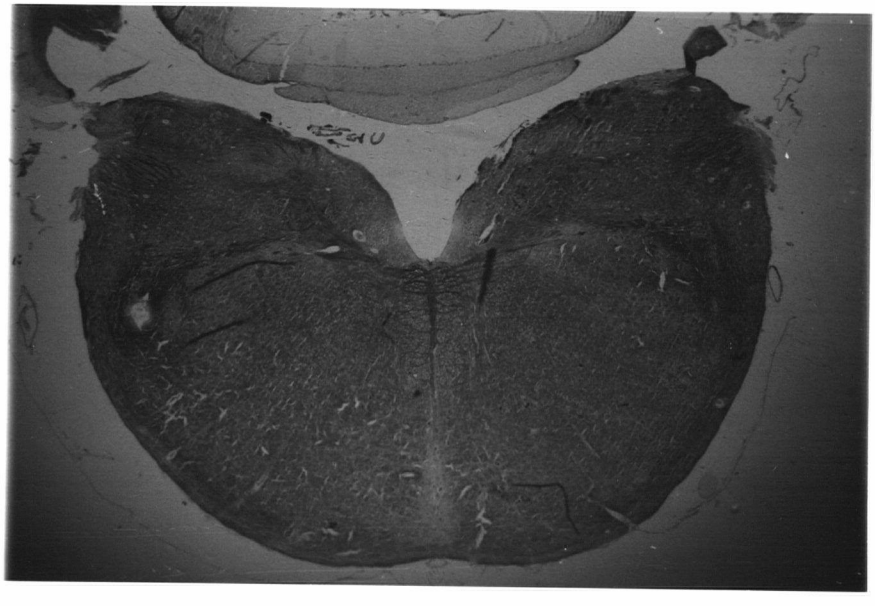
B

FIGURE 39

Diagrammatic representation (A) of a transverse section through the rostral medulla/caudal pons showing the site of electrical stimulation which evoked flying and running movements in the acute decerebrate avian of Expt. 2a. The area of stimulation is contained within TTD. Fig. B shows a photograph of a section through the lesion site. Section level is comparable to the section level P1.25 of Karten and Hodos (1967). Abbreviations: FLM= medial longitudinal fasciculus, MC= nucleus (n.) magnocellularis, nIX= n. of glossopharyngeal nerve, NVIII= vestibulocochlear nerve, R= raphe nucleus, TTD= descending tract and nucleus of the trigeminal nerve, VeD= n. vestibularis descendens. (Magnification: 9x).



A



B

FIGURE 40

Stimulation-induced walking in the acute decerebrate duck. Electromyographic records demonstrate alternating activity of the flexor muscles (ITC) of the hindlimb during treadmill walking. Potentiometer records show alternating flexion and extension excursions of both legs. The walking movements resulted from stimulation in the region of TTD (Expt. 12b). Abbreviations: ITC= iliotibialis cranialis, Hind.= hindlimb.

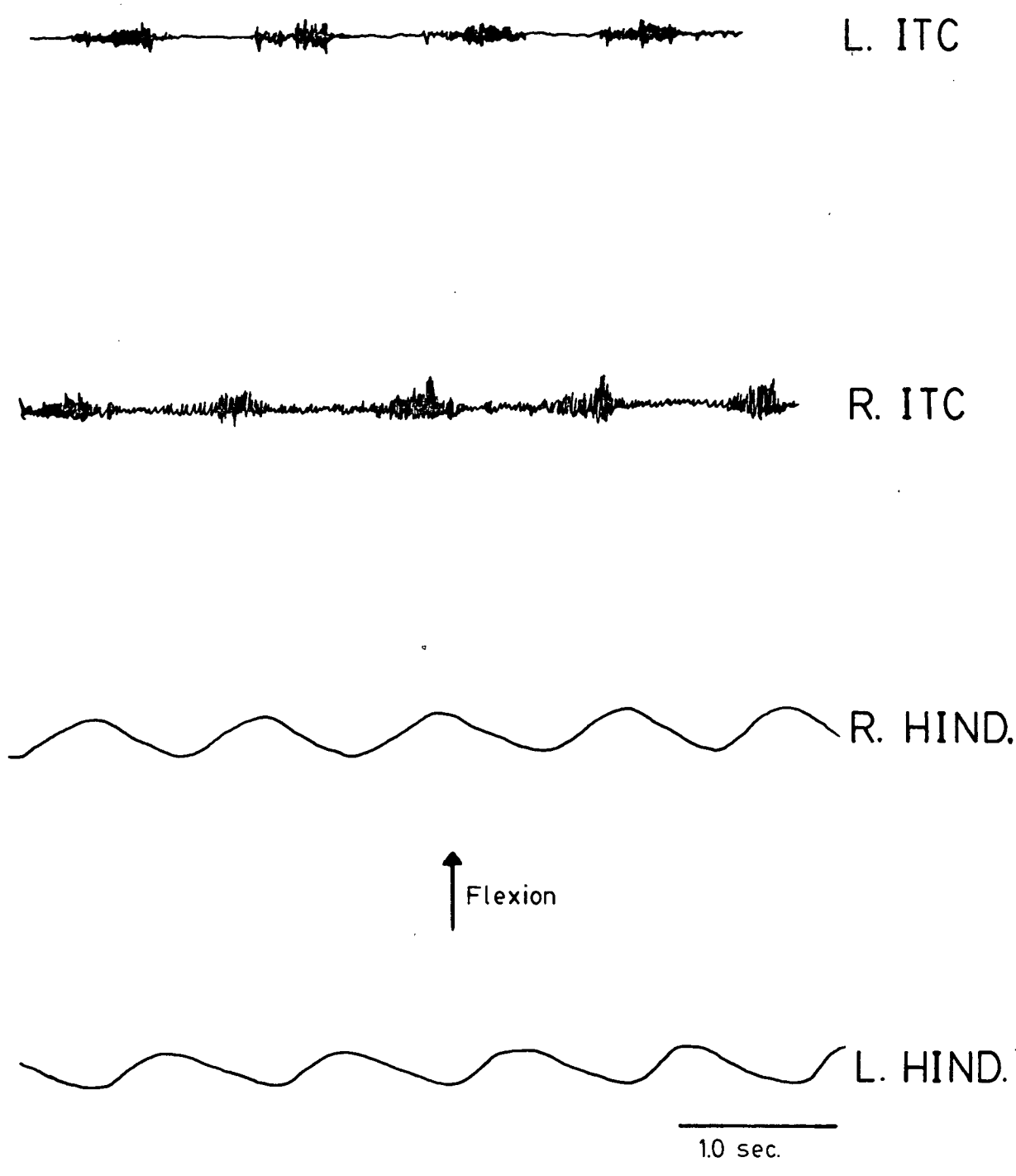
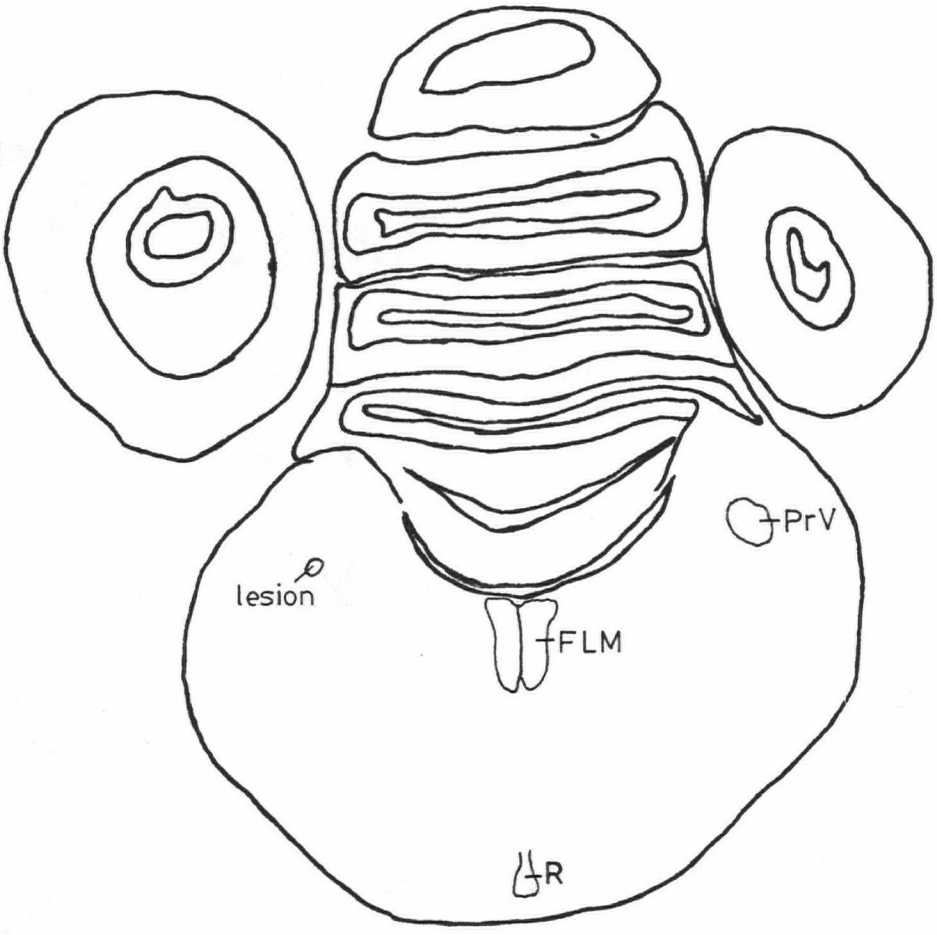
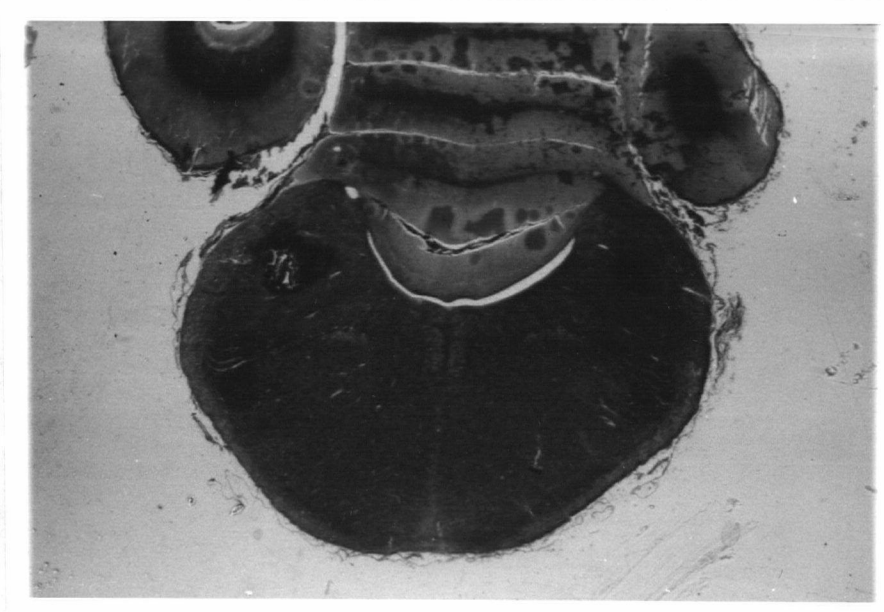

L. ITC
R. ITC
R. HIND.
Flexion
L. HIND.
1.0 sec.

FIGURE 41

Diagrammatic representation (A) of a transverse section through the mid-pontine region (A1.0 of Karten and Hodos (1967)) showing the site (lesion) which evoked flying and running movements when stimulated (Expt.1a). B is a photograph of a section through the lesion site. The lesion indicates the stimulation location to be in or near PrV. Abbreviations: FLM= medial longitudinal fasciculus, PrV= principle sensory nucleus of the trigeminal nerve, R= raphe nucleus. (Magnification: 9x)

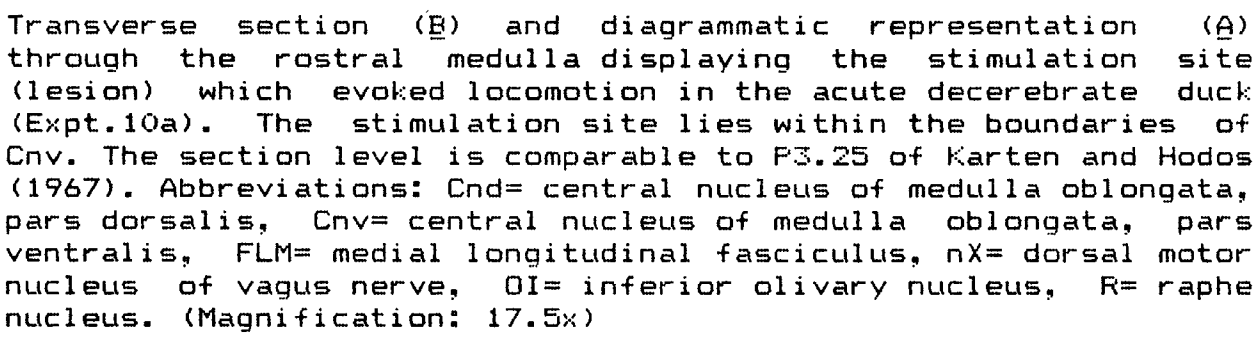


A

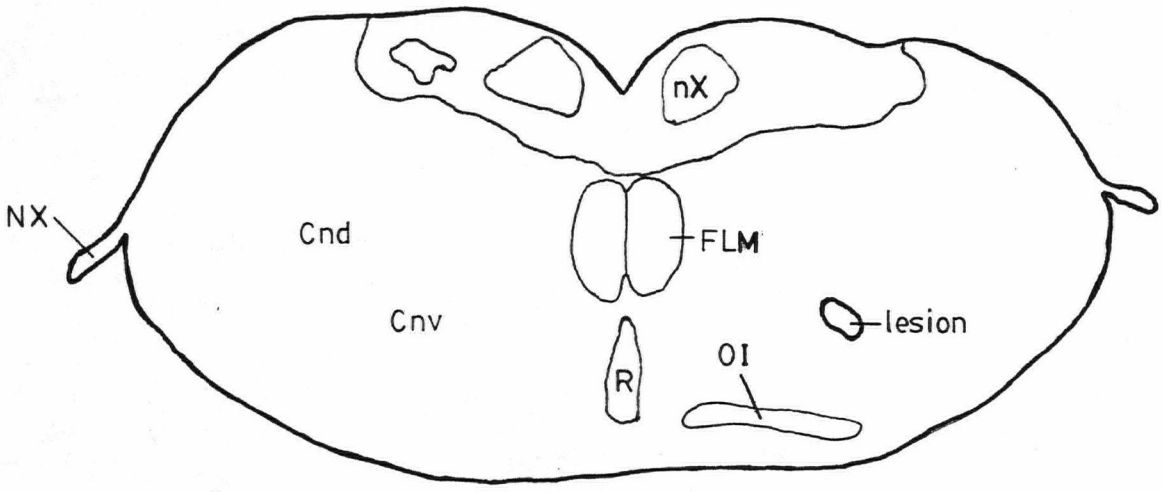


B

FIGURE 42



Transverse section (B) and diagrammatic representation (A) through the rostral medulla displaying the stimulation site (lesion) which evoked locomotion in the acute decerebrate duck (Expt.10a). The stimulation site lies within the boundaries of Cnv. The section level is comparable to P3.25 of Karten and Hodos (1967). Abbreviations: Cnd= central nucleus of medulla oblongata, pars dorsalis, Cnv= central nucleus of medulla oblongata, pars ventralis, FLM= medial longitudinal fasciculus, nX= dorsal motor nucleus of vagus nerve, OI= inferior olivary nucleus, R= raphe nucleus. (Magnification: 17.5x)



A

B

FIGURE 43

Simultaneous hindlimb and wing movements evoked by stimulation in the area of Cnv/Cnd are demonstrated by potentiometer (left and right hindlimbs) and electromyographic records (left pect)(Expt.11a). Although only a single pect was implanted with electrodes for the EMG records, rhythmic concurrent flapping movements occurred bilaterally. Abbreviations: Hind.= hindlimb, Pect.= Pectoralis

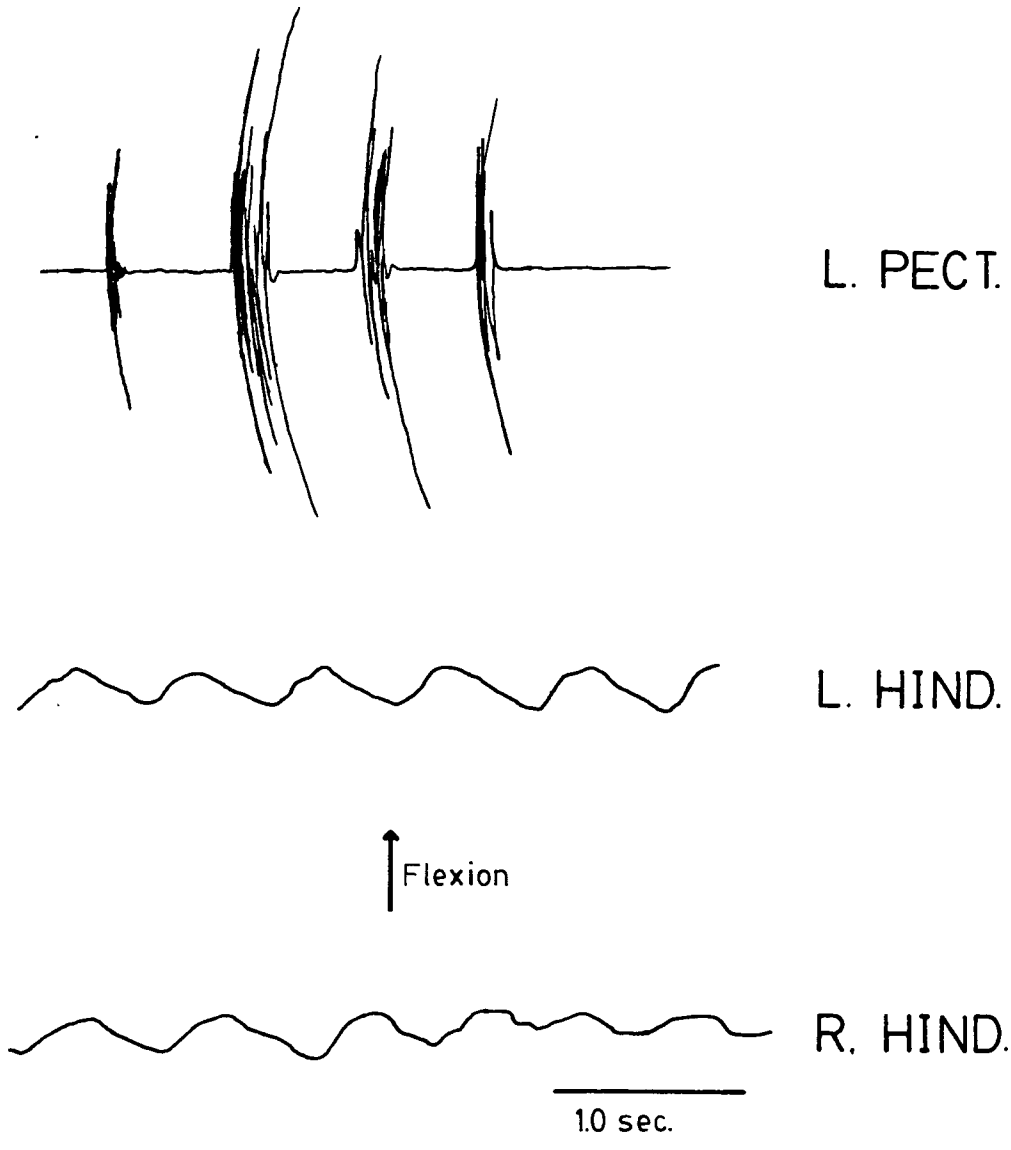

L. PECT.
L. HIND.
Flexion
R. HIND.
1.0 sec.

FIGURE 44

Electromyographic and potentiometer records of walking in the electrically stimulated avian. The EMG record of hindlimb stepping corroborates the potentiometer records showing alternating activity of hindlimb flexor (ITC) muscles during stimulation-evoked treadmill walking. The stimulation site was within Cnd (see Fig. 43, Expt. 11a). Abbreviations: ITC= iliotibialis cranialis, Hind.= hindlimb.

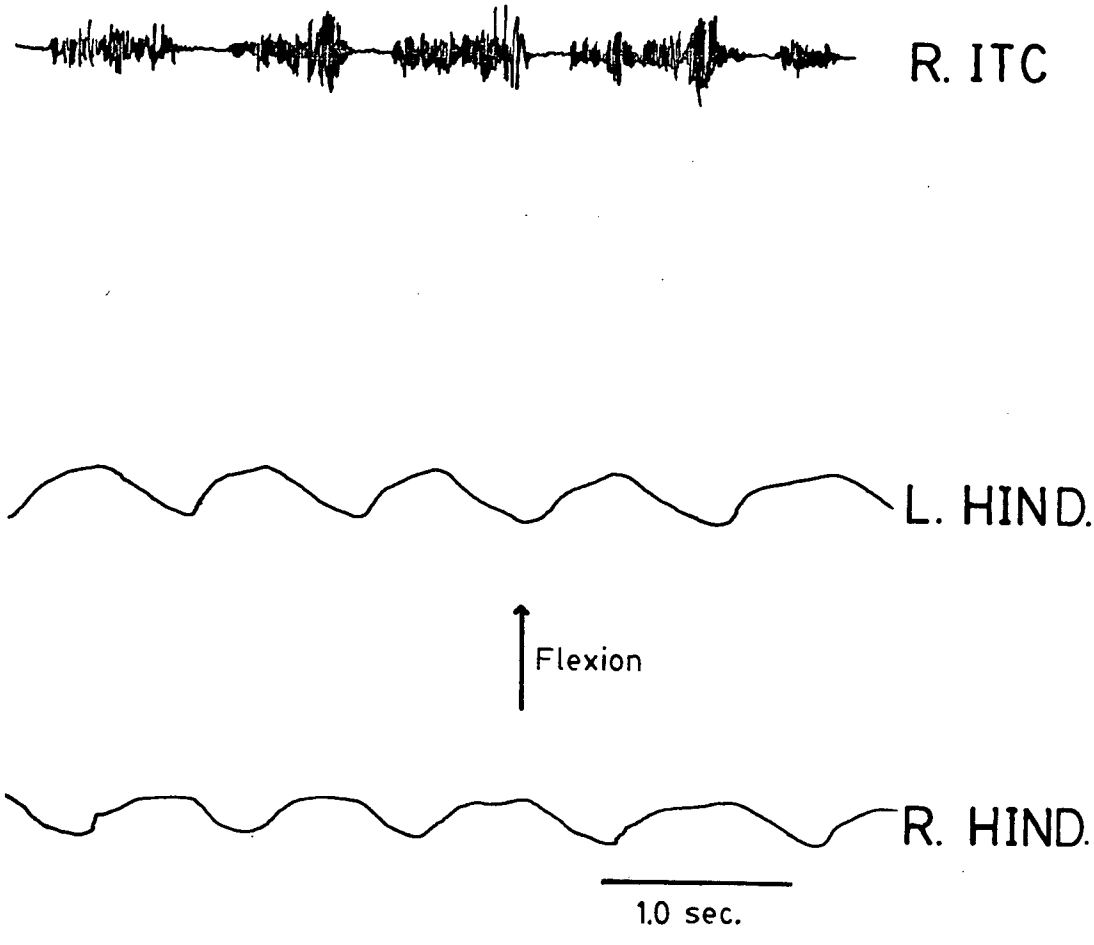
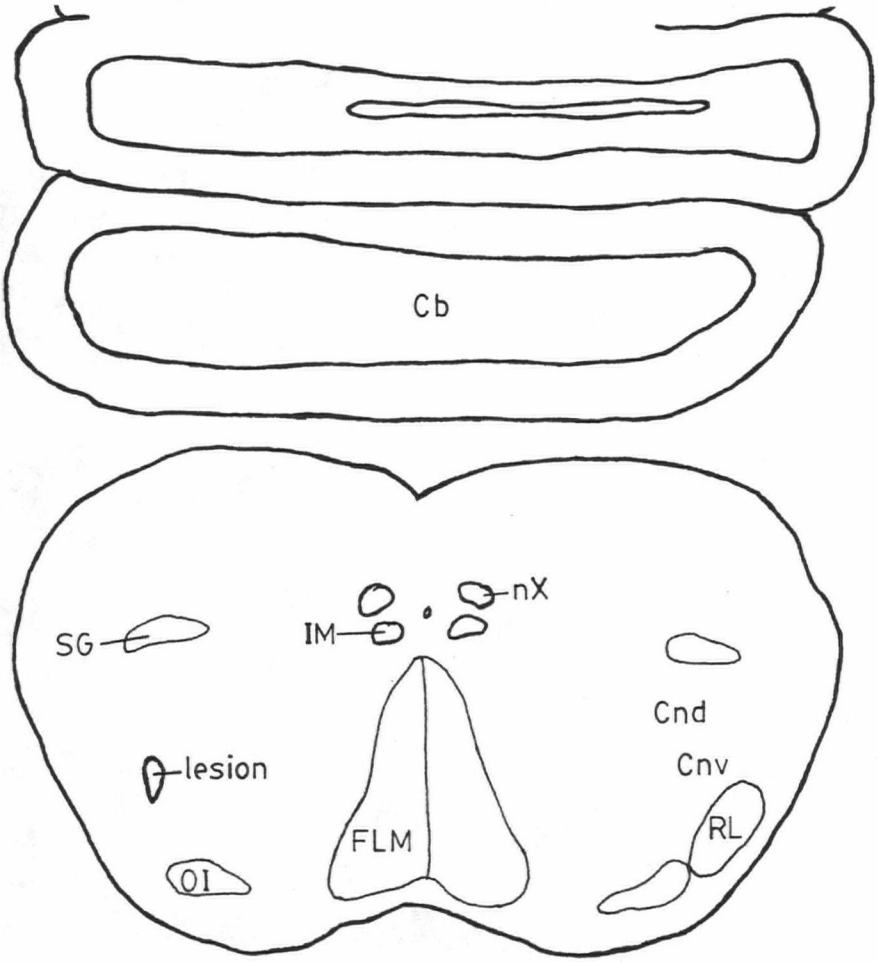
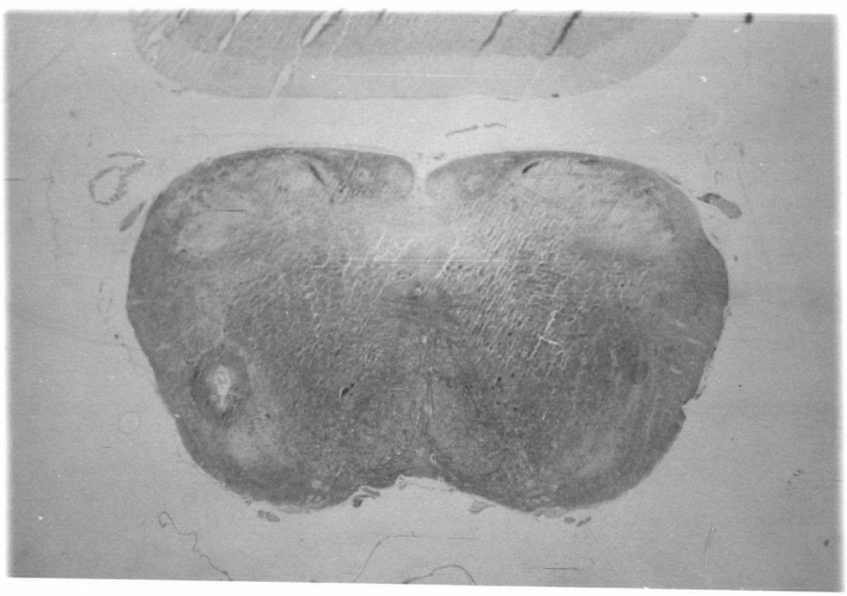

R. ITC
L. HIND.
Flexion
R. HIND.
1.0 sec.

FIGURE 45

Diagrammatic representation (A) of a transverse section through the medulla outlining the stimulation site (lesion) which evoked flying and treadmill running at low current stimulus intensity (25uA). The photograph in B shows the actual lesion. The stimulus area is bounded by RL and Cnv. The section level is comparable to P3.75 of Karten and Hodos (1967). Abbreviations: Cb= cerebellum, Cnd= central nucleus of medulla oblongata, pars dorsalis, Cnv= central nucleus of medulla oblongata, pars ventralis, FLM= medial longitudinal fasciculus, IM- intermediate nucleus, nX= dorsal motor nucleus of vagus nerve, OI= inferior olivary nucleus, RL= lateral reticular nucleus, SG= substantia gelatinosa Rolandi (trigemini). (Magnification: 17.5x).



A



B

FIGURE 46

Transverse section through the rostral mesencephalon displaying the stimulation site (lesion) which produced walking and wing flapping movements in an acute decerebrate duck (Expt.12c,d). The area of stimulation is centered near AL and SpL. Section level is comparable to A4.75 of Karten and Hodos (1967). Abbreviations: AL= ansa lenticularis, CF= campi Foreli, OM= occipitomesencephalic tract, PT= pretectal nucleus, QF= quintofrontal tract, SCE= external cellular stratum, SGF= stratum griseum et fibrosum superficiale, SpL= lateral spiriform nucleus, SpM= medial spiriform nucleus, VIII= ventricle III (Magnification: 13x)

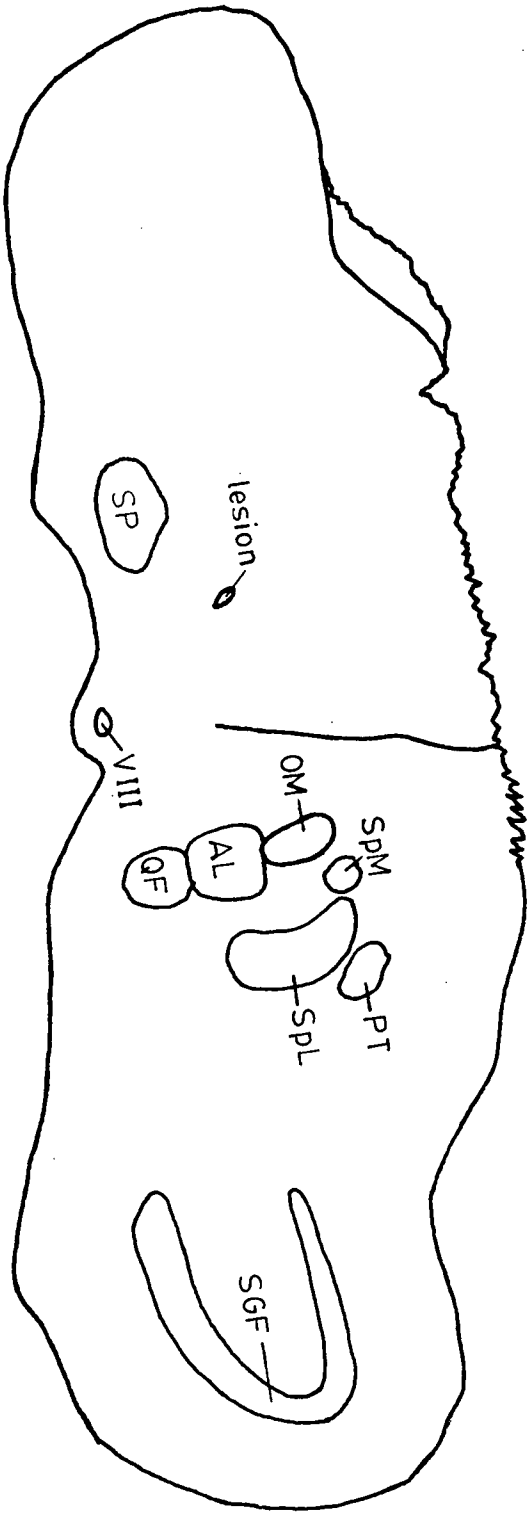
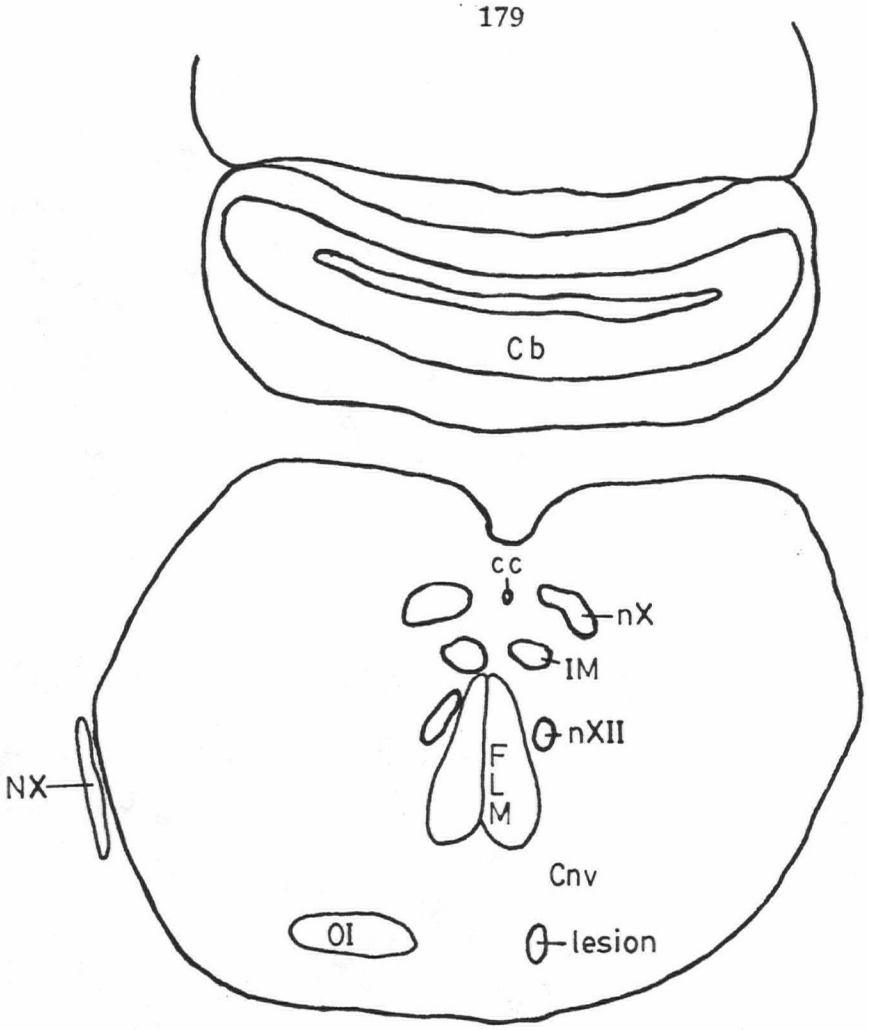
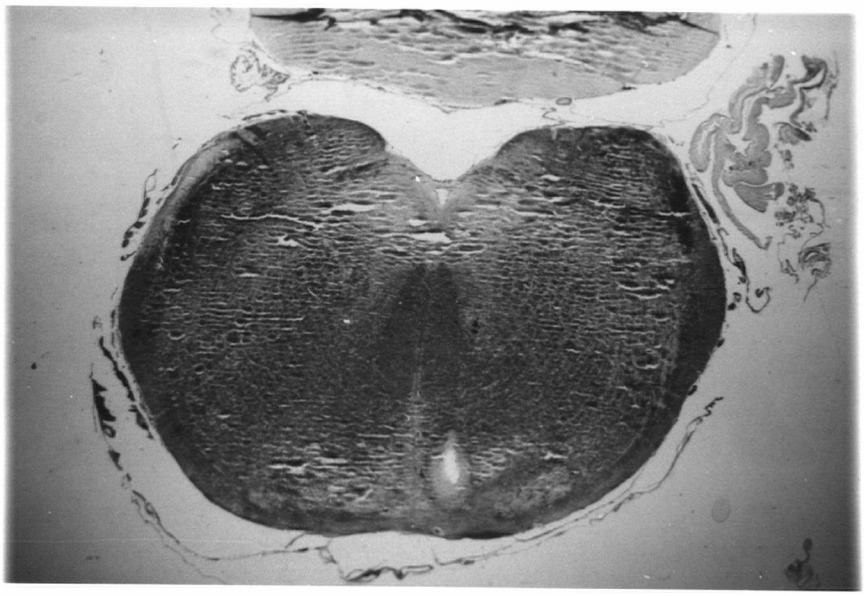

SP
lesion
VIII
OM
AL
QF
SpM
PT
SpL
SGF

FIGURE 47

Diagrammatic representation (A) and photograph (B) of a transverse section through the medulla (equivalent to Karten and Hodos P3.5) showing the stimulation site (lesion) which produced treadmill walking during electrical stimulation (Expt.8). The stimulation site lies medial to the inferior olivary nucleus in Cnv. Abbreviations: Cb= cerebellum, cc= central canal, Cnv= central nucleus of the medulla oblongata, pars ventralis, FLM= medial longitudinal fasciculus, IM= intermediate nucleus, nX= dorsal motor nucleus of vagus nerve, nXII= nucleus of hypoglossal nerve, NX= vagus nerve, OI= inferior olivary nucleus. (Magnification: 13x).



A



B

FIGURE 48

Electromyographic (ITC) and potentiometer (HIND.) records showing hindlimb activity during electrically evoked walking in the decerebrate avian (Expt.8). Abbreviations: ITC= iliotibialis cranialis, HIND.= hindlimb.

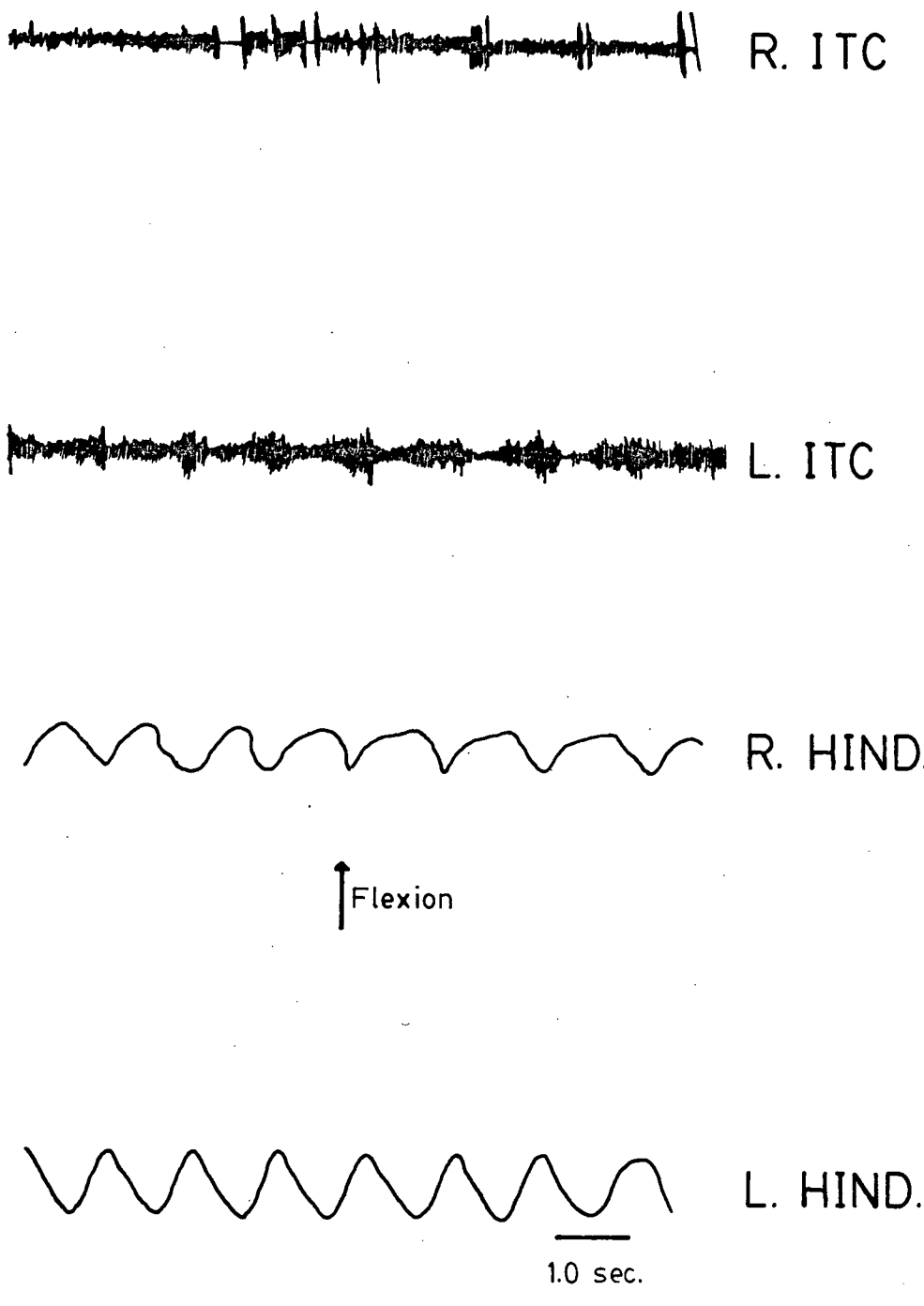

R. ITC
L. ITC
R. HIND.
Flexion
L. HIND.
1.0 sec.

FIGURE 49

Potentiometer records showing the effect of increasing treadmill belt velocity during evoked locomotion. The lines below the potentiometer records for the left hindlimb mark maximal rostral excursion of the limb and demonstrate that increased frequency of stepping occurs with relative increases in treadmill velocity. Abbreviation: HIND.= hindlimb

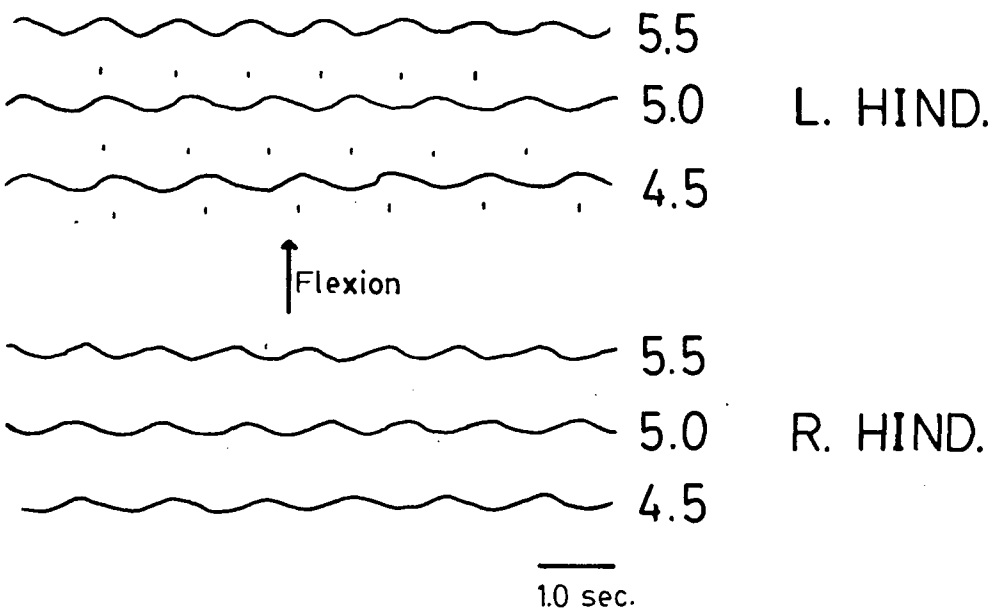

5.5
5.0
4.5
L. HIND.
Flexion
5.5
5.0
4.5
R. HIND.
1.0 sec.

FIGURE 50

Electromyographic and potentiometer records showing the effects of increasing stimulation current intensity during evoked locomotion. EMG records from left and right flexor (ITC) muscles show increased frequency of activity in response to augmented current strength. Potentiometer records demonstrate an increase in the frequency of stepping with increased stimulus intensity. The treadmill belt velocity and stimulation frequency were held constant. Abbreviations: ITC= iliotibialis cranialis, HIND.= hindlimb, uA= microamperes

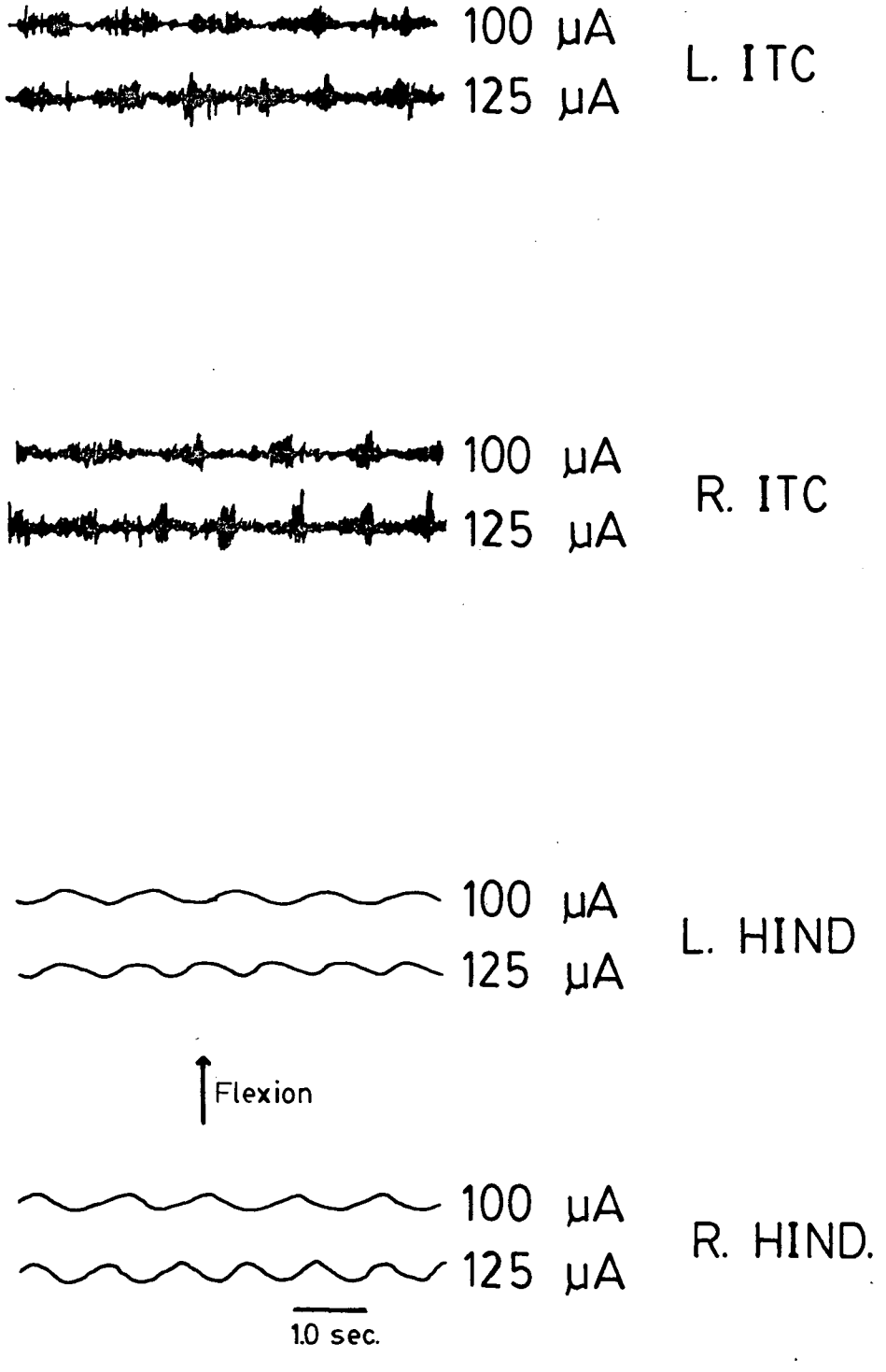
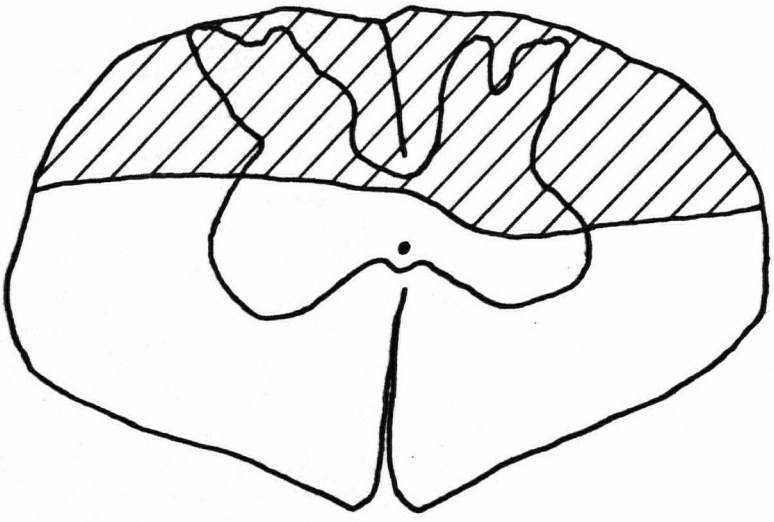
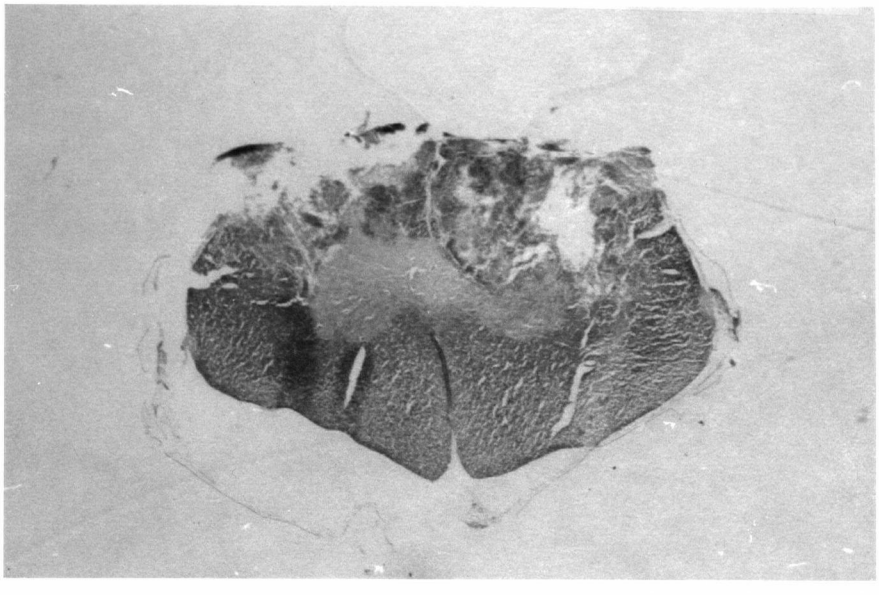

100 μA
125 μA
L. ITC
100 μA
125 μA
R. ITC
100 μA
125 μA
L. HIND
Flexion
100 μA
125 μA
R. HIND.
1.0 sec.

FIGURE 51

Diagrammatic representation (A) of a cross section through the low thoracic spinal cord following a chronic Dorsal Cord Transection (Expt.6). A portion of the total lesion is shown in B. The diagram is a composite from serial sections through the lesion site. The lined area demonstrates complete lesion extent.



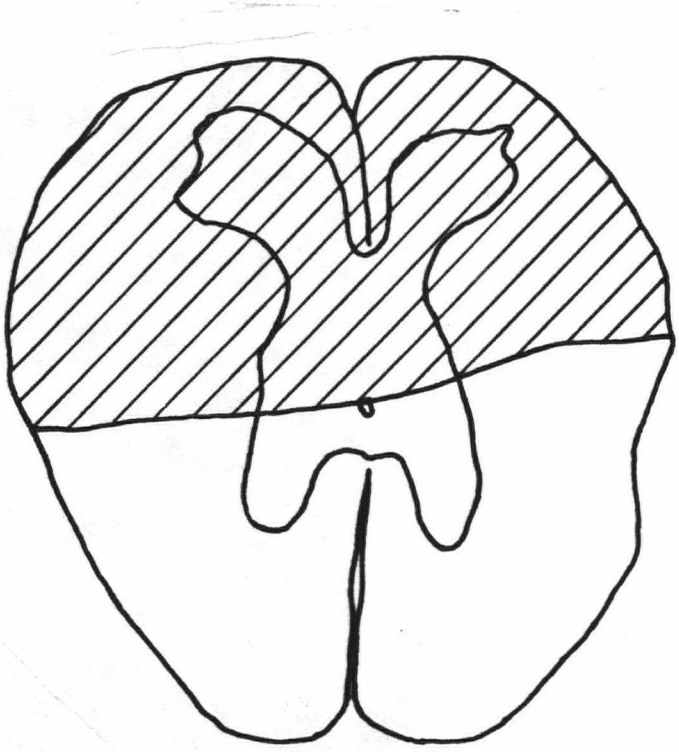
A



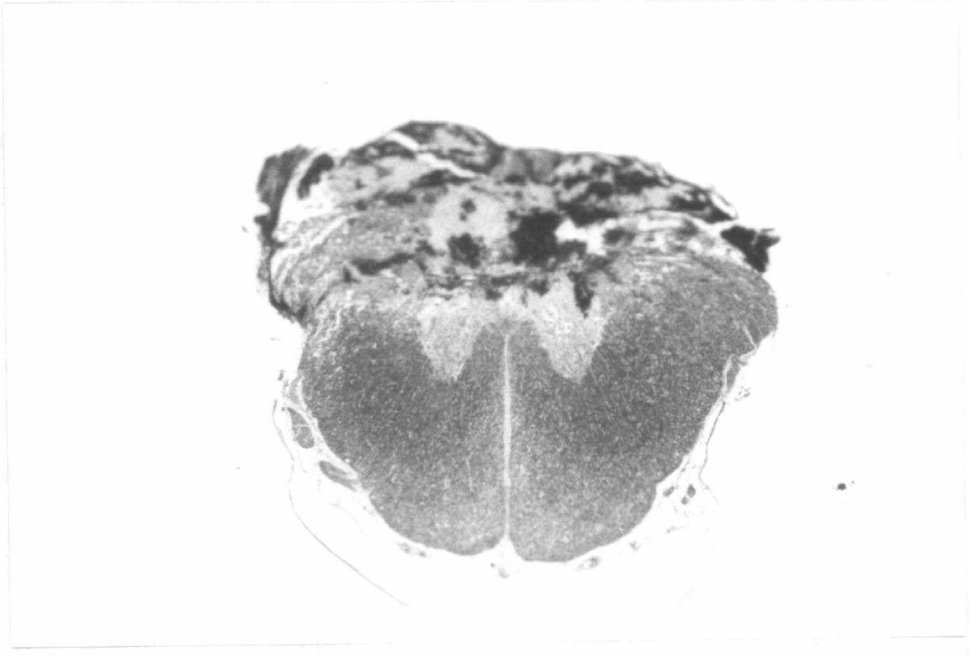
B

FIGURE 52

Transverse section (B) through the low thoracic spinal cord following an acute spinal cord transection (Expt.12). Histological verification of the lesion extent (lined area) was made using a composite of serial cross sections (a sample cross section is shown in B) through the lesion site .



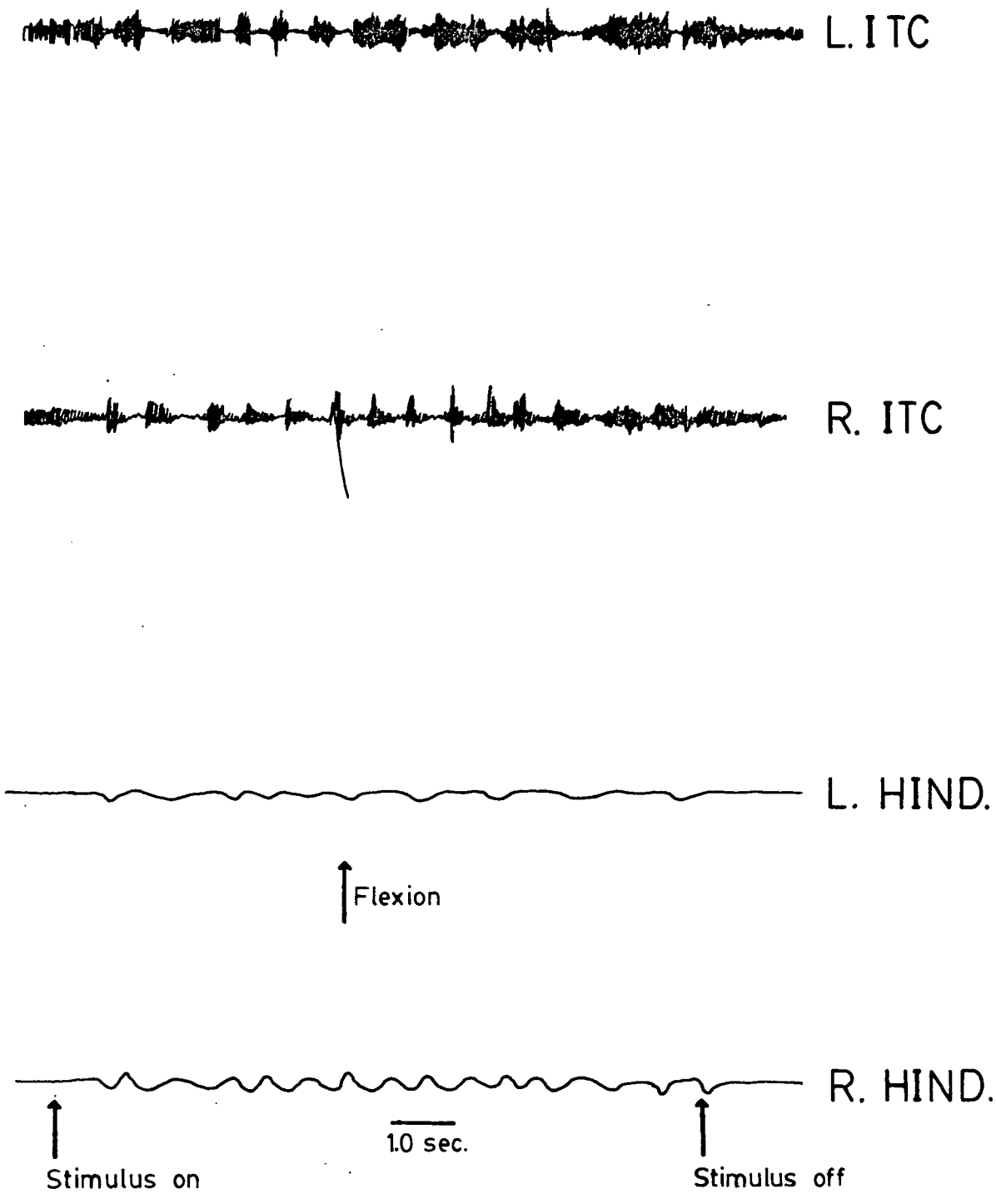
A



B

FIGURE 53

Electromyographic (EMG) and potentiometer records from hindlimb muscles (ITC) and legs following an acute low thoracic spinal cord Dorsal Cord Transection in a decerebrate brainstem stimulated animal (Expt.12). EMG records show alternating activity in homologous hindlimb flexors (ITC), with more clearly defined rhythmic flexion in the right ITC than the left ITC. Potentiometer records display the rostrocaudal excursions of both hindlimbs. They indicate that stepping of the right leg is rhythmic, with definitive excursions, while that of the left hindlimb is less pronounced. The stimulus on/off arrows indicate stimulus onset and termination. Walking commenced with onset and ceased at stimulus termination. Abbreviations: ITC= iliotibialis cranialis, HIND.= hindlimb.


L. ITC
R. ITC
L. HIND.
Flexion
R. HIND.
1.0 sec.
Stimulus on
Stimulus off

CONCLUSIONS

The findings of this study establish parallels between avian and mammalian descending systems controlling locomotion. These include: the locomotor program for stepping (ie. spinal stepping) is intrinsic to the spinal cord in both birds and mammals excluding primates; at least one intact spinal cord ventral quadrant is necessary for the production of voluntary locomotion in both bird and mammal; electrical stimulation of areas in the brainstem can evoke locomotion in acute decerebrate preparations of both groups; and the electrically stimulated locomotion evoking sites in the brainstem have anatomical correlation between birds and mammals.

"Spinal stepping" occurred following spinal cord transection at the low thoracic level in birds. Whether "spinal flying" can also be produced remains to be determined. However, cervical cord transection rostral to the cervical enlargement in chronic animals may show the existence of an intrinsic spinal cord locomotor flight generator. The different types of locomotion in birds may allow experimenters to define the neuronal circuits and interconnections of the two possibly different central pattern generators.

The reticulospinal pathways are implicated in the supraspinal descending control of locomotion in both birds and mammals. However, the results of this study cannot differentiate the role in locomotion of the reticulospinal pathway from that of the

vestibulospinal pathway which travels in the same spinal cord area. Bilateral lesioning of the vestibular nuclei, combined with selective spinal cord lesions in chronic animals would determine the requirement of this pathway for the production of voluntary hindlimb locomotion. As the results of chronic selective lesioning expereriments in this study apply only to hindlimb locomotion, it will be interesting to use the same technique to examine descending pathways controlling forelimb locomotion. Jacobson and Hollyday (1982b) have postulated the existence of two separate pathways in the cervical spinal cord controlling locomotion in the chick. These may also be examined using selective lesion studies to determine their necessity in locomotor behaviours. The origin of the pathway(s) responsible for descending control can be clarified by neuroanatomical tracing studies using retrograde horse radish peroxidase (HRP) and/or True Blue (TB) techniques combined with selective spinal cord lesions.

Results from the acute brainstem stimulation experiments have produced parallels between the avian and mammalian systems. Three areas known to evoke locomotion in the cat appear from this study to have correlates in birds. However, more analysis must be done to examine the interdependency of these areas, their interconnections, and the necessity of these areas to locomotion before a complete comparison can be made. Experiments utilizing selective bilateral lesions of locomotion evoking structures will determine the interdependence of these stuctures. Anatomical tracing studies using HRP or TB can be used in combination with electrical stimulation to delimit the interconnections of these structures. The dependence of locomotion on these supraspinal

structures may be determined by both selective bilateral lesions and by ablating these structures by changing the transection level in acute decerebrate preparations.

The bird has been relatively unexplored neurophysiologically with regard to its locomotor mechanisms yet its attributes provide an excellent substrate for locomotor research. The results of this study are the groundwork for future, potentially fruitful, studies into avian locomotion.

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