ネコの体軸（体幹及び尾）の運動、姿勢の神経制御に関する研究

和田　直己

Neuronal control of posture and movements of trunk and tail in cats

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CONTENTS

1 GENERAL INTRODUCTION

The movements and posture of animals have always intrigued man. From prehistoric paintings depicting man's prey in action, to the dances of primitive people mimicking familiar animals, to current anatomical and physiological studies of specific biomechanical and energetic problems, interest in how animals move continues and keep posture. The animal body is obviously a complex system. Even its subsystems are complex. Actually, a single cell is already complex enough to be considered a whole world of its own. To understand the movements and posture, it is necessary to study the cells (i.e., neurons, muscle fibers), the connections among the cells, the various kinds of structures (i.e., the cerebral cortex, the cerebellum), connection among the structures, and behaviors (i.e., posture, locomotion,). Many investigators have been studying intrinsic behaviors of motoneurons and pattern generators, effects of various descending neuronal pathways to motoneurons, reflex pathways activated various kind of afferents innervating various part of the body (i.e.. group Ia, Ib, II, Aαβ). Electromyographic and kinematic study of movements were well done in cats and dogs. However, these studies are concentrated on the hindlimbs, forelimbs and necks. The studies of neuronal control of movements and posture of trunk and tail have been insufficient. Trunk represent a large part of the total body mass in all kind of animals and constitutes an integral part of the motor system. Adequate control of trunk movements and posture is

therefore a prerequisite for the maintenance of the body equilibrium during various motor tasks. Furthermore, in jumping and galloping of four legged animals, trunk movements produce proceeding powers. Most of animals has tail. In fish, amphibians and reptiles with tails, and marine mammals like whales and dolphins, the tail is the most important part of the body for swimming, and rhythmical lateral and vertical undulate movements of the tail produce the swimming power. The neuronal system producing undulate movements for swimming has been especially well studied in fish. The varieties in size, shape and function of the tail are especially significant in terrestrial mammals. For example, the cat has a long tail and shows various types of tail movements for locomotion, balance, defense, micturition, defecation, and sexual activities. Trunk and tail are continuos parts of each others and have the close anatomical relationships between tail and trunk, but the functions are very different.

In the present experiments, we studied neuronal control of tail and trunk movements in the cats. Especially, the present experiments were concentrated on neuronal pathways from peripheral afferent inputs on motoneurons innervating tail and trunk muscles.

2, Neuronal pathways for spinal reflexes activated by group I and group II muscle afferents in the spinal segment (Co1) innervating tail in the low spinalized cat

Summary

We studied neuronal pathways for spinal reflexes activated by group I and group II muscle afferents in the spinal segments innervating tail in the unanesthestized and spinalized (L1) cats. Experiments were performed on 25 adult cats of both sexes. The effects of stimulating nerves innervating 6 tail muscles on both sides were recorded from tail motoneurons in the first coccygeal spinal segment (Co1) using glass microelectrodes.

Stable recordings were obtained from 150 tail motoneurons. Stimulation of group I muscle afferents (stimulus intensity < 1.8T) often produced EPSPs (82/150) on stimulation of nerves innervating neighboring tail muscles. Motoneurons innervating the long tendoned muscles, m. extensor caudae lateralis and m. flexor caudae longus (ECL and FCL) , received heteronyms monosynaptic connections from group I muscle afferents innervating the ipsilateral tail muscles. The motoneurons innervating segmental muscles, m. extensor caudae medialis and m. flexor caudae brevis (ECM and FCB), receive heteronyms monosynaptic connections from group I muscle afferents innervating tail muscles on both sides. The motoneurons innervating tail muscles originated from the ossa coxae, m. abductor caudae externus and m. abductor caudae internus (ACE and ACI) received monosynaptic connections from group I muscle afferents innervating most of the tail muscles on both sides. Crossed disynaptic inhibitory pathways activated by group I muscle afferent inputs were observed in ECM, ACE, FCL and FCB motoneurons. The effects of group

II afferent inputs were not dependent on the kind of motoneuron and alternative excitatory and inhibitory pathways were not clearly observed in the tail motoneuron pool. It is suggested that variability of the neuronal pathways from group I and II muscle afferents to tail motoneurons correspnds to functional relationships among tail muscles involved in the tail movements.

Introduction

The size, shape and function of the tail varies with the kind of mammal. For example, the cat has a long tail and shows various types of tail movements for locomotion, balance, defense, micturition, defecation and sexual activities. The tail movements of cats are accomplished by systematic activity of the tail muscles, mainly consisting of the following 6 muscles: the extensor caudae medialis , extensor caudae lateralis, abductor caudae externus, abductor caudae internus, flexor caudae longus and flexor caudae brevis (Fig. 3A, Wada et al. 1994). Table 1 shows abbreviation, origin, insertion and function of each tail muscle. Studies on the neural control of tail movements in cats indicate that peripheral afferent inputs from other parts of the body as well as various descending pathways influence activities of the tail motoneuron (Wada et al. 1993; 1995a; 1995b; 1996a; 1996b). Information from various receptors such as muscle spindle, Golgi tendon organ, cutaneous receptors and joint receptors is important to control the movement and posture (Lundberg 1979; Prochazka et al. 1989; Rossignol et al. 1989; Schomburg 1990). Goldfinger and Fukami (1982) demonstrated the high density distribution of muscle spindle and Golgi tendon organs in tail muscles, suggesting that afferent inputs from tail muscles play an important role in controlling the movement

not only of tail itself but also of the limbs and trunk. Many investigators have studied neuronal pathways from muscle afferents to motoneurons (see review by Schomburg 1990). However, these studies were mainly concentrated on the neuronal pathways in the spinal cord innervating the legs and there are few studies of the neural pathways from tail muscle afferents to tail motoneurons. In the present experiments, we studied the neuronal pathways from group I and group II muscle afferents innervating tail muscles to tail motoneurons in low spinalized cats.

Materials and Methods

Experiments were performed on 25 long-tailed adult cats (2.4-3.8 Kg) of both sexes. Under anesthesia with halothane-nitrous oxide, the animals were decerebrated by passing a spatula rostroventrally from a line about 1 mm rostral to the superior colliculus and aspirating tissue rostral to the transection. Then, anesthesia was discontinued, and animals were further spinalized at the L1 spinal segment. Tail muscles are innervated by numerous fine nerve branches from several spinal segments. Since the nerve branches innervating tail muscles are very thin and short and it is very difficult to isolate and mount all tail muscle nerves on stimulating electrodes, the nerves to the tail muscles on both sides were stimulated using teflon-insulated stainless steel wires (5 _ m gauge) with 2-3 mm bared areas at the distal end of the wires, which were inserted into each muscle at the level of caudal vertebrae 2-3 (Ca2-3) innervated by spinal nerves from the third sacral and first coccygeal spinal segments (S3 and Co1) (unpublished data). The electrode position was carefully adjusted under an operating microscope so that stimulation through the electrodes produced a contraction only in the muscle in which the stimulating

electrodes were located as far as the stimulus intensity was equal to or below five times threshold. The threshold value (T) was determined by the appearance of afferent volley in the cord dorsum potential (CDP) recorded at the Co1 level, and ranged between 50-160 mV (0.3 ms duration). All ventral roots from the sacral and coccygeal spinal segments were cut to eliminate the effects of recurrent inhibition (Jankowska et al. 1978). The proximal end of the Co1 ventral root was mounted on bipolar electrodes for stimulation to identify motoneurons. The animals were paralyzed with pancuronium bromide (0.4 mg/kg per h) and artificially ventilated. End-tidal CO2 concentration was monitored and maintained at approximately 4.0% by adjusting the respiratory rate and tidal volume. The rectal temperature was monitored and maintained close to 37 °C with a heating mat. Arterial blood pressure was monitored through a catheter inserted into the right common carotid artery and maintained above 80 mmHg (mean ± SD: 107± 15.5 mmHg) during the experiments. Laminectomy was performed between L5 and the sacral vertebrae. The animal was fixed in a stereotaxic frame. The exposed spinal cord was covered with warmed mineral oil (36-38 °C), the temperature of which was kept constant by a coiled tube, with warm water flowing through. Intracellular recordings from motoneurons in Co1 were obtained using a glass microelectrode filled with 3M potassium citrate solution (input resistance in the spinal cord: 20-35MΩ). Tail motoneurons were identified by the existence of an antidromic spike after stimulation of the Co1 ventral root on the left side and monosynaptic EPSP after stimulation of group I muscle afferents in the tail muscle nerves on the left. When motoneurons showed monosynaptic EPSPs after stimulation of more than two muscle nerves, we determined the muscle innervated by motoneuron from the

size of maximal EPSPs and time course of the rising phase (Henneman and Mendell 1981). The stimulation of homonymous muscle nerve produced the largest monosynaptic EPSPs with the steepest rising phase. The tail muscle nerves were electrically stimulated at 1.1-10 times the threshold of each peripheral nerve. In 12 cats, EPSP and IPSP were proven by reversing the potentials by injecting depolarizing and hyperpolarizing current, respectively. The intracellular membrane potential and cord dorsum potential (CDP) were recorded on magnetic tape (TEAC, RD-135T). The central latency value was obtained by signal averaging of 10 consecutive sweeps (NEC Sanei, signal processor 7T17). Minimal central latencies were measured during ten single sweeps (NEC Sanei, signal processor 7T17).

Results

Figure 1 shows a typical example of the cord dorsum potentials (CDPs) recorded by a monopolar electrode put on the Co1 spinal segments when the intensity of stimulation of the ipsilateral ECL muscle nerves was gradually increased from 1.2T to 10T. At 1.2T, a downward peak (p) was observed in CDP and the size of p was gradually increased when the stimulus intensity was increased from 1.2T to 2.0T. At 2.0T, a second negative peak (s) following the first peak was also observed. We measures the nerve length (d) from stimulating electrode to recording electrode (CDP), and latencies of two waves from stimulating artifact on CDP (p: Tp, s: Ts). d/Tp and d/Ts were 88.4 ± 5.4 m/s and 52.3 ± 4.1 m/s, respectively. Increasing the stimulus intensity from 2.0T to 10T led to an increase in the size of s. Thus, stimulation at an intensity equal to or below 1.8T seems to activate only group I muscle afferents, and at 2T or stronger intensity, both

group I and group II muscle afferents were likely to be stimulated (Willis and Coggeshall 1991). At 10T, a third component of CDP (t) was detectable.

Stable recordings were obtained from 150 tail motoneurons, 24 ECM, 25 ECL, 21 ACE, 24 ACI, 31 FCL and 25 FCB motoneurons. The membrane potentials and spike amplitudes averaged approximately -62 (minimum - maximum values; -56 - -68 mV) mV and 72 mV (minimum - maximum values; 62 - 81 mV), respectively.

Figure 2-7 show examples of PSPs evoked by single pulse stimulation of tail muscle nerves at 1.5 and 5T. Upper, middle and lower columns of each figure show antidromic action potentials produced after stimulation of Co1 ventral root (Fig. 2-7a), and PSPs after stimulation of tail muscle nerves at 1.5T (Fig. 2-7b) and 5T (Fig. 2-8c), respectively. Fig. 2 shows PSPs recorded from an ECM motoneuron. At 1.5T, depolarizing PSPs (EPSPs) were observed after stimulation of ECM, ECL, ACI, FCL and FCB on the ipsilateral side (iECM, iECL, iACI, iFCL, iFCB) and FCB on the contralateral side (cFCB). At 5T, EPSPs, EPSP followed by hyperpolarizing PSPs (EPSP/IPSPs) or spike (stimulation of iEM) were observed after stimulation of all muscle nerves except for cACI and cFCL. Fig. 3 shows PSPs recorded from an ECL motoneuron. At 1.5T, EPSPs or EPSP/IPSPs were observed after stimulation of all muscle nerves of the ipsilateral side and cEM. EPSPs induced by stimulation of iECL and iACE showed a steep rising phase of EPSPs and monosynaptic segmental latencies (iECL: 0.5 ms, iACE: 0.7ms), while EPSPs produced by stimulation of iACI, iFCL and iFCB gradually increased in size and showed a long segmental latency. At 5T, EPSP/IPSPs were observed after stimulation of all tail muscle nerves on the ipsilateral side and

ECM, FCL and FCB on the contralateral side, while IPSPs followed by EPSP/IPSPs were observed after stimulation of cECL and cACI. Fig. 4 shows PSPs recorded from an ACE motoneuron. At 1.5T, EPSPs were observed after stimulation of iECL, iACE and cACI. The EPSPs produced by stimulation of iECL and iACE have short segmental latencies corresponding to monosynaptic connections (iECL: 0.8ms, iACE: 0.5ms). Stimulation of cECM produced IPSP. At 5T, EPSP/IPSPs were observed after stimulation of all tail muscle nerves except for iECM, cECL and cACE. Fig. 5 shows PSPs recorded from an ACI motoneuron. At 1.5T, EPSPs were observed after stimulation of iACE, iACI, iFCL, iFCB and cFCL. EPSPs were monosynaptically produced by stimulating iACE, iACI and iFCB (iACE: 0.8 ms, iACI: 0.7 ms, iFCB: 0.9 ms). At 5T, EPSP/IPSPs were observed by stimulating all tail muscle nerves except for iECL and cECM. Fig. 6 shows PSPs recorded from an FCL motoneuron. At 1.5T, EPSPs were observed after stimulation of iFCL, iFCB, cECM and cFCL. At 5.0T, EPSP/IPSPs were observed after stimulation of all tail muscle nerves except for iACE and iACI. Fig. 7 illustrates PSPs recorded from an FCB motoneuron. At 1.5T, EPSPs were observed after stimulation of all tail muscle nerves except for iACI and stimulation of cFCB produced EPSP/IPSP. At 5T, EPSPs followed by IPSPs were produced by stimulating all tail muscle nerves except for iECM and iFCL. In general, PSPs produced by stimulation of muscle nerves (1.2-1.8T) innervating neighboring muscles showed a steep rising phase of PSPs and short segmental latencies. Increasing the stimulus intensity from 1.2-1.8T to 5T increased the PSP size, changed monophasic PSPs into mixed PSPs and increased the number of tail muscle nerves that produced PSPs.

Twelve tail muscles in pairs are arranged around the caudal vertebrae (Ca1-4) as shown in Fig. 8A (Wada et al. 1994). We determined the incidences at which different kinds of PSPs (%) appeared in 12 regions of interest (Fig. 8B), EPSPs (dotted area), IPSPs (filled area) and mixed PSPs (EPSP/IPSPs, IPSP/EPSPs; hatched area), when the intensity of single pulse stimulation was 1.5T (upper column) and 5T (lower column). Stimulation at 1.5T produced EPSPs, IPSPs and EPSP/IPSPs. The incidence of EPSPs and EPSP/IPSPs was high when the muscle nerves innervating neighboring muscles were stimulated (82/150), while IPSPs were often observed after stimulation of nerves innervating contralateral and diagonal muscles. Stimulation at 5T produced EPSPs, IPSPs, EPSP/IPSPs and IPSP/EPSPs (including IPSP followed by EPSP/IPSP). The incidence of PSPs at 5T was remarkably high compared to that at 1.5T. Especially, the rate of mixed PSPs was remarkably high. Fig. 3 shows that there was no significant difference in the incidence of various PSP types at 5T depending on the stimulated muscle nerve.

Fig. 9 is a schematic diagram which indicates the monosynaptic connections from group I muscle afferents to tail muscle motoneurons. The girded and shaded areas indicate the homonymous and heteronymous monosynaptic connection, respectively, between group I muscle afferents and tail muscle motoneurons (< 1.0 ms: segmental latency, Lloyd and Wilson 1959). ECM motoneurons received heteronymous monosynaptic connections from muscle afferents innervating cECM, iECL, cECL, iACI, cFCL and cFCB. ECL motoneurons receive heteronymous monosynaptic connection from group I muscle afferents innervating ECM and ACE on the ipsilateral side. ACE motoneurons received monosynaptic connections from group I muscle afferents innervating all tail muscles on both sides.

ACI motoneurons received monosynaptic connection from group I muscle afferents innervating all tail muscles except for iECM, iECL and cACI. FCL motoneurons received heteronymous monosynaptic connection from group I muscle afferents innervating ACE, ACI and FCB on the ipsilateral side. FCB motoneurons received heteronymous monosynaptic connections from group I muscle afferents innervating iECL, cACE, cACE and cFCB. Fig. 9B indicates tail muscle nerves that produced disynaptic IPSPs (central latency < 1.8 ms, Eccles and Lundberg 1958: Lundberg 1975) in tail motoneurons. Disynaptic IPSPs in ECM motoneurons were observed after stimulating muscle nerves innervating iACE, iACI, iFCL, iFCB and cECL. Disynaptic IPSPs in ECL motoneurons were observed after stimulating muscle nerves innervating iFCL and iFCB. Disynaptic IPSPs in ACE motoneurons were observed after stimulating muscle nerves innervating ECM and ECL on both sides, cACE and iFCL. Disynaptic IPSPs in ACI motoneurons were produced only after stimulating muscle nerves innervating iECL. Disynaptic IPSPs in FCL and FCB motoneurons were observed after stimulating muscle nerves innervating cACE and cECM, respectively.

Discussion

Methodological aspect

In the present experiments, the potential problem is the contamination of adjacent muscle or non-muscle afferents, cutaneous and joint afferents. The electrical stimulation by paired wire electrodes at 1.2-5T produced contraction of only the inserted part of each muscle. CDP recorded by a monopolar electrode on Co1 differed depending on the stimulated muscle

nerve, and the stimulation of tail muscle nerves innervating adjacent muscles at 1.2-5T often showed different effects on tail motoneurons (Fig. 2-7). These facts indicate that the electrical stimulation below 5T did not stimulate other muscles or segments of muscles. Each tail muscle was covered by fasciae, and cutaneous nerves passed through gaps of adjacent tail muscles and outside of fasciae. The stimulation at 1.2-5T did not produce the contraction of adjacent muscles. These facts suggest that electrical stimulation at 1.2-5T did not pass through fasciae and did not stimulate cutaneous nerves. Afferent fibers innervating lumbar facet joints have been

demonstrated (Bogduk, 1983; Gillette et al. 1993), suggesting the existence of joint nerves innervating the caudal facet joint. We cannot disprove the existence of the effects of joint afferent inputs in PSPs produced after stimulation using wire electrodes. Especially stimulation at 5-10T, often produced late big waves in CDP, and effects of contaminating stimulation may exist in the late part of PSPs. However, it is most

reasonable that the effects of electrical stimulation at intensities below 5T were produced mainly by activation of group I and group II muscle afferents .

Monosynaptic excitatory pathways from group I afferents

The monosynaptic connection from group I muscle afferents to homonymous alpha-motoneurons was demonstrated by Lloyd (1943). Many investigators have studied the monosynaptic projection from muscle spindle afferents onto the homonymous motoneurons, and carried out its function in control of muscle length. It can

be considered that homonymous monosynaptic connections from the primary afferents to tail muscle motoneurons control muscle length and

maintain the tail in a stable position. Monosynaptic projections from Ia afferents have been demonstrated not only to homonymous alpha-motoneurons but also to alpha-motoneurons of various more or less synergistic muscles in the neck (Brink et al. 1981) , the hindlimb and forelimb (Baldissera et al. 1981; Eccles and Lundberg 1958; Edgley et al. 1986; Fournier at al. 1984; Hongo et al. 1984, Illert, 1996). Furthermore, some investigators showed the existence of monosynaptic projections to motoneurons innervating partly antagonistic muscles in the turtle (Steffens et al. 1985; Yamashita, 1986). However, monosynaptic projection to motoneurons innervating antagonistic muscles has not been shown. Some investigators (Edisen, 1963; Jankowska et al. 1978; Matsushita and Tanami, 1983; Ritz et al. 1989; 1991) suggested the existence of monosynaptic connection of group I muscle afferents to synergistic motoneurons and crossed monosynaptic connections in lower sacral segments. Motoneurons innervating the long tendoned muscles, ECL and FCL, received heteronymous monosynaptic connections from group I muscle afferents innervating the ipsilateral tail muscles. The motoneurons innervating segmental muscles, ECM and FCB receive heteronymous monosynaptic connections from group I muscle afferents innervating tail muscles on both sides. The motoneurons innervating tail muscles originated from the ossa coxae, ACE and ACI, received monosynaptic connection from group I muscle afferents innervating most tail muscles on both sides. Ia synergism induced by the multilateral monosynaptic Ia connections has been interpreted as a contribution to muscle synergy during locomotion (Conway et al. 1988; Lundberg 1969) and during the performance of skillful grasping movements of the forelimb (Clough et al. 1968; Fritz, 1981). During rhythmical movements of the foot, locomotion, foot shaking and scratching,

the cat shows rhythmic lateral tail movements. All tail muscles on one side are activated as synergistic muscles when cats perform rhythmic lateral tail movements (personal observation). Heteronymous monosynaptic connections were observed from group I muscles afferent in the ipsilateral tail muscles in all tail motoneurons. These facts suggest that monosynaptic connection in tail motoneurons can be interpreted as a contribution to muscle synergy during locomotion, foot-shaking and scratching. During vertical movements, reciprocal activities were observed between the dorsal (ECM, ECL) and ventral muscles (FCL, FCB) on both sides (Wada et al. 1994). The monosynaptic connections between ECM motoneurons and group I muscle afferents from ventral muscles, and FCB motoneurons and group I muscle afferents from dorsal muscles could be interpreted as a contribution to muscle synergy during vertical tail movements. In ACE and ACI motoneurons, monosynaptic EPSPs were produced by inputs from group I muscle afferents innervating all kinds of tail muscles on both sides. Activation of ACE and ACI produce vertical and rolling tail movements in addition to lateral tail movements (Table 1, and Wada et al. 1994). ACE and ACI show synergistic relationships with all tail muscles on both sides. This fact might be related to the pattern of multiple monosynaptic connections of group I muscle afferents in ACE and ACI motoneurons. An antagonistic relationship was observed between tail muscles (ECM, ECL, FCL and FCB) located in diagonal directions on transverse tail sections (ex, left ECM-right FCB or right FCL). The results of the present experiments show monosynaptic projection to motoneurons innervating antagonistic muscles in coccygeal spinal segments innervating tail.

Since we performed electrical stimulation of muscle nerves, we cannot determine whether the monosynaptic EPSPs are produced by activation of

group Ia, group Ib or both Ia and Ib afferents. In general, group Ib afferents perform di- or oligosynaptic inhibitory projection to synergistic motoneurons and an excitatory projection to antagonistic motoneurons (see review by Baldissera et al. 1981). It might be considered that monosynaptic excitatory effects of primary afferent activation are produced by activation of group Ia afferents. Multiple monosynaptic projection from group Ia and Ib muscle afferents still allows a variable synergy by presynaptic inhibition (see review by Baldissera et al. 1981).

Disynaptic inhibitory pathways from group I muscle afferents

Frank and Sprague (1959) and Jankowska et al. (1978) showed crossed disynaptic inhibitory pathways in lower sacral spinal segments. However, they did not perform selective stimulation of muscle afferents. Our results show the existence of crossed disynaptic inhibitory pathways activated by group I muscle afferent inputs in ECM, ACE, FCL and FCB motoneurons. Eccles et al. (1957) showed the disynaptic inhibitory pathways activated by group Ia afferents to motoneurons innervating muscles with a strictly antagonistic function. As described above, the antagonistic relationship was observed among tail muscles located in diagonal directions on transverse tail sections. Disynaptic IPSPs caused by activation of group I muscle afferents innervating antagonist were observed in FCL and FCB motoneurons in the present experiments. Investigations in low spinalized cats revealed a distinct asymmetry of the reciprocal effects of Ib afferents from flexors and extensors in the lumbar spinal cord innervating the hindlimb (Eccles et al. 1957; Eccles and Lundberg 1959; Lundberg et al. 1975; 1977; 1987). Ib afferents from extensors evoked distinct inhibition in extensor motoneurons and excitation in flexor motoneurons, while the reverse pattern from flexor Ib afferents was almost

completely missing in low spinalized cats. Further studies using selective stimulation of group Ia or Ib will be required to demonstrate whether the disynaptic inhibitory pathways are activated by afferent inputs from group Ia, group Ib or both groups Ia and Ib.

Pathways from group II muscle afferents

The additional effects on PSPs induced by increasing stimulus intensities from 1.8T to 2-5T may be effects of secondary muscle spindle afferents. By increasing stimulus intensities from 1.2-1.8T to 2-5T, PSP size and the incidence of PSPs were increased, especially those of mixed PSPs in all kinds of tail motoneurons (see Fig. 2-7, Fig. 9). Investigation of the interneuronal reflex effects on group II muscle afferents in the spinal cord innervating limb muscles demonstrated that group II muscle afferents from flexors and extensors facilitated the flexor and inhibited the extensors (Eccles and Lundberg 1959; Lundberg et al. 1975; 1987). The effects of group II afferent inputs were not dependent on the kind of motoneuron and alternative excitatory and inhibitory pathways were not clearly observed in the tail motoneuron pool. Further studies showed that both flexor and extensor motoneurons receive di- or polysynaptic excitatory and inhibitory group II connections in the lumbar spinal cord innervating hindlimb (see review by Schomburg 1990). Results of the present experiments indicate the existence of both excitatory and inhibitory pathways from group II spindle afferents to all kinds of tail motoneurons as demonstrated in the lumbar spinal cord.

Functional aspect of neuronal circuits from group I and group II muscle afferents to tail motoneurons.

The results of the present experiments indicate the existence of various types of neuronal pathways from group I and II muscle afferents to

tail motoneurons. Many investigators have shown stereotyped reflex pathways in the spinal cord innervating hindlimbs and forelimbs (see Reviews by Baldissera et al. 1981; Schomburg 1990). Effects of reflex pathways from group I and II muscle afferents which were similar to stereotyped reflex pathways in the lumbar spinal cord were observed in the coccygeal spinal cord. However, the characteristics of the reflex pathways in the coccygeal spinal cord showed variability in the effects of muscle afferent inputs.

Cats' tail movements are very complicated and varied. The functional relationship among tail muscles varies depending on the tail movement and is not fixed like muscles in limbs. It is suggested that variability of the neuronal pathways from group I and II muscle afferents to tail motoneurons relays to functional relationships among tail muscles involved in the tail movements.

Table

Table 1 The origin, insertion, and function of the tail muscles in the cat

Name (abbreviation)	Origin and insertion	Function
M. extensor caudae medialis (ECM)	ECM originates from the spinous process and is inserted on the caudal articular process of the sucessive caudal vertebrae	Dorsal and slight lateral bending of tail
M. extensor caudae lateralis (ECL)	ECL is the direct caudal continuation of the M. logissimus lumborum. ECL originates from the articular process of the sacral and caudal vertebrae. ECL has long tendons inserting on the mammilary process of the caudal vertebrae	Dorsolateral bending of tail
M. abductor caudae externus (ACE)	ACE originates from the medial side of the dorsal border of the ilium and sends an inserting tendon to the caudal transverse process of the caudal vertebrae as far as the 7–9th vertebrae	Lateral and slight dorsal bending of the proximal part of tail, clockwise twisting of the proximal part of tail
M. abductor caudae internus (ACI)	ACI originates from the spine of the ischium and inserts on the transverse process of the 2–5th caudal vertebrae	Slight ventrolateral bending and counter clockwise twisting of the proximal part of tail
M. flexor caudae longus (FCL)	FCL originates from the ventral surface of the lumbo, sacral, and caudal vertera. FCL has long tendons inserting on the cranial articular process of the caudal vertebrae	Ventrolateral bending of tail
M. flexor caudae brevis (FCB)	FCB originates from the ventral surfaces of the 3rd sacral and caudal vertebrae and terminates on the ventral surface of the caudal vertebrae	Ventral and slight lateral bending of tail

Legends for Figures

Fig. 1. Cord dorsum potentials (CDPs) recorded by monopolar recording electrode put on Co1 spinal segment after stimulation of ECL muscle nerves at various stimulus intensities. At 1.2T-1.8T, a downward peak (p) was observed in CDP. At 2.0T, a second downward peak (s) was observed in addition to p. Increasing stimulus intensity from 2T to 10T increased the size of s. At 10T, a third downward peak (t) was detected.

Fig. 2-7. Postsynaptic potentials (PSPs) after stimulating tail muscle nerves. Upper and lower column in each panel indicates PSP and CDP, respectively.

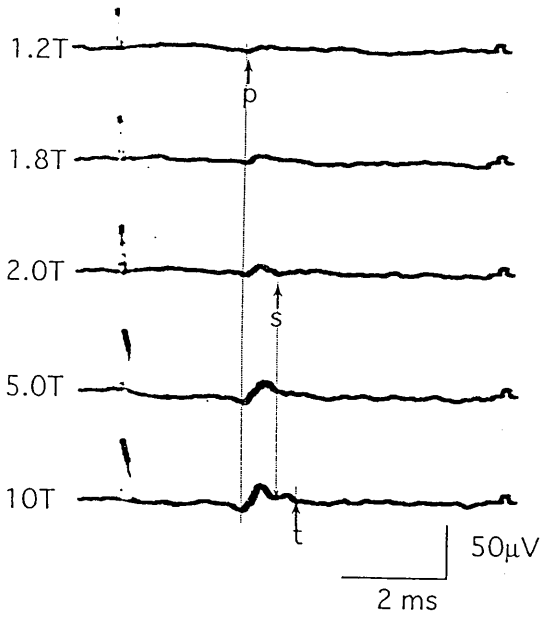
a: action potential after antidromic stimulation of Co1 ventral root, b: PSPs after stimulating tail muscle nerves at 1.5T, c: PSPs after stimulating tail muscle nerves at 5T. Figs. 2, 3, 4, 5, 6 and 7 show representative PSPs recorded from the M. extensor caudae medialis (ECM), M. extensor caudae lateralis (ECL), *M.* abductor caudae externus (ACE), *M.* abductor caudae internus (ACI), M. flexor caudae longus (FCL), *a*nd M. flexor caudae brevis (FCB) *mo*toneurons, respectively. See text for details. iECM--*iFCB Cor*responding muscles of the ipsilateral side, cECM--*cFCB cor*responding muscles of the contralateral side. See text for details.

Fig. 8, A: Schematic drawings of the tail muscles on transverse section of the tail at Ca2-4 vertebral levels, B: Incidences (%) of different type PSPs in tail muscle motoneurons (MN) after stimulating tail muscle nerves at 1.5T and 5T. The EPSP, IPSP and mixed PSPs (EPSP/IPSP, IPSP/EPSP) are indicated by dotted, filled and hatched areas. At 1.5T, the incidence of EPSP or EPSP/IPSP was high when stimulation was performed on muscle

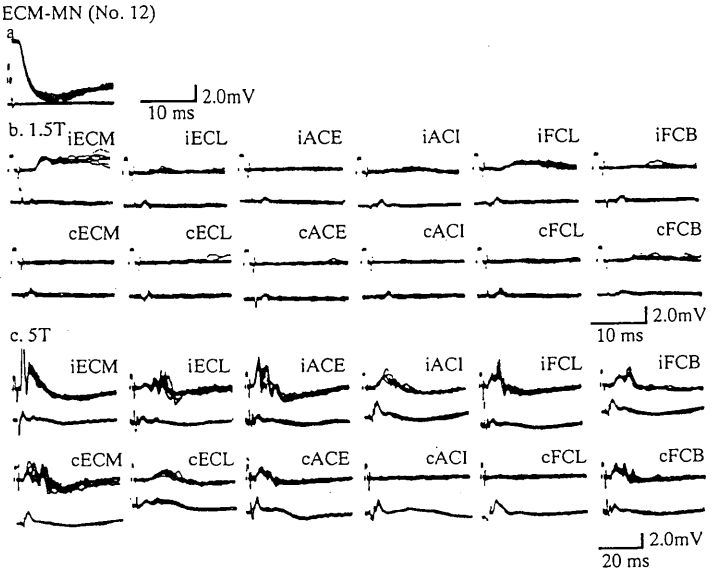
nerves innervating neighboring muscles. IPSPs were often observed when stimulation was performed on muscle nerves innervating contralateral tail muscles. At 5T, the rate of mixed PSPs was significantly increased.

Fig. 9, Patterns of monosynaptic excitation (A) and disynaptic inhibition (B) from group I muscle afferents. Experimental arrangement and neuronal circuits are as illustrated in the scheme in the lowest part. Homonymous connections are indicated by girded areas. Heteronymous monosynaptic excitatory connections are indicated by shaded areas. Disynaptic inhibitory connections are indicated by filled areas. MN; motoneuron.

Fig. 1



Figs 2-4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2.0mV
10 ms
b. 1.5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
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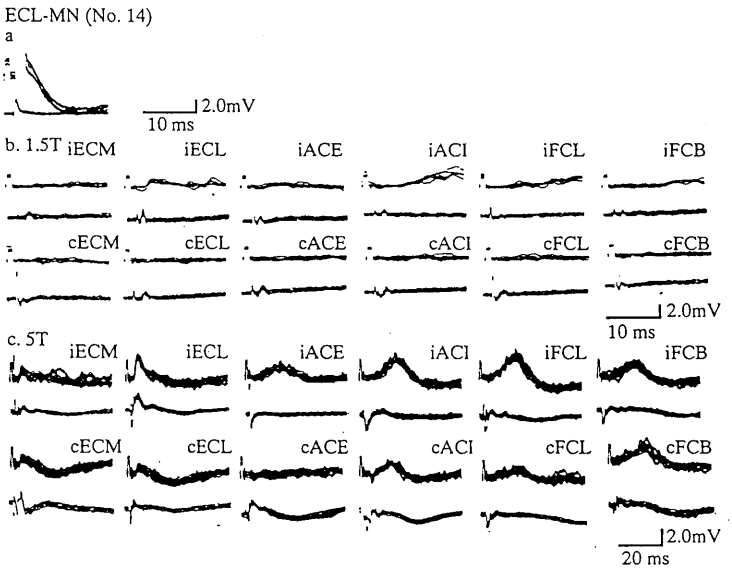
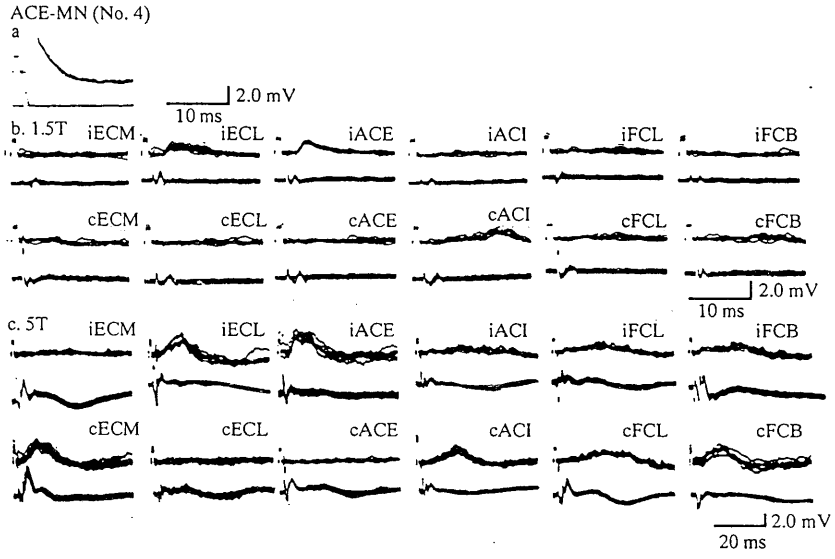
cACE
cACI
cFCL
cFCB
2.0mV
20 ms

2

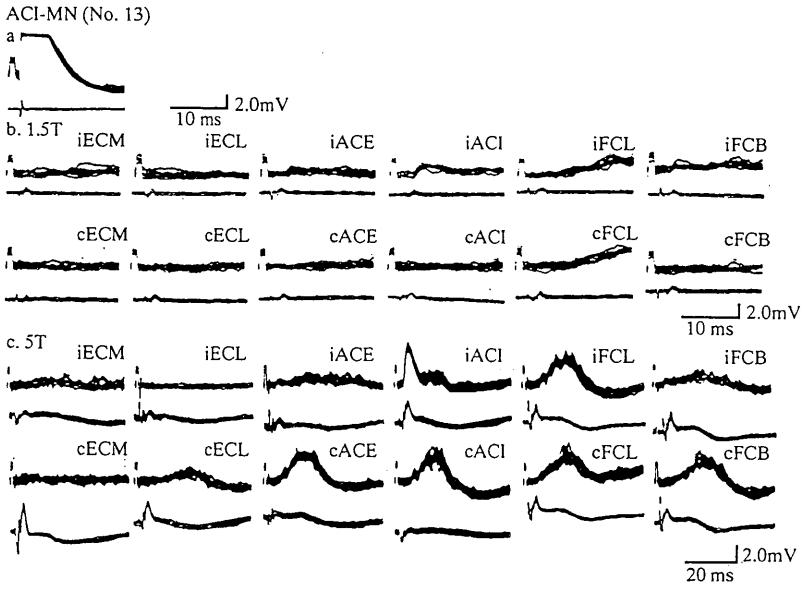

ECL-MN (No. 14)
a
2.0mV
10 ms
b. 1.5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
10 ms
c. 5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
20 ms

3

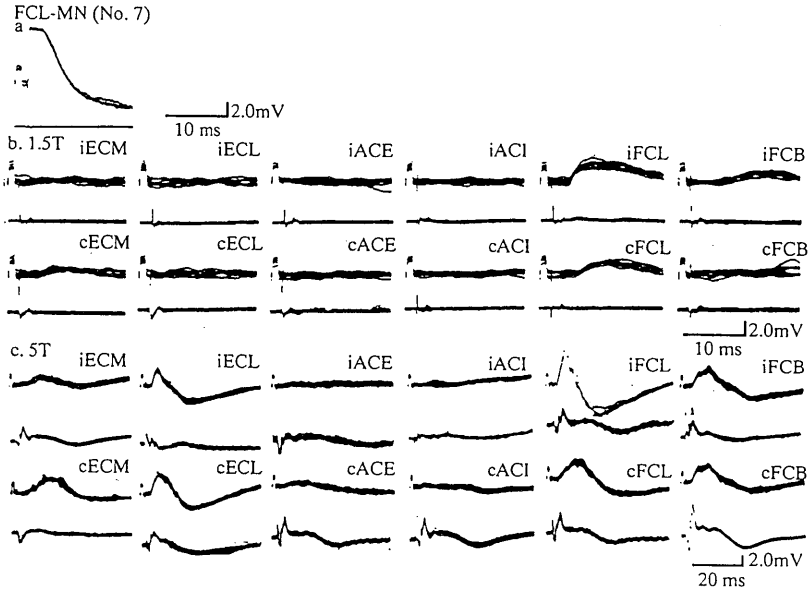

ACE-MN (No. 4)
a
2.0 mV
10 ms
b. 1.5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0 mV
10 ms
c. 5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0 mV
20 ms

4

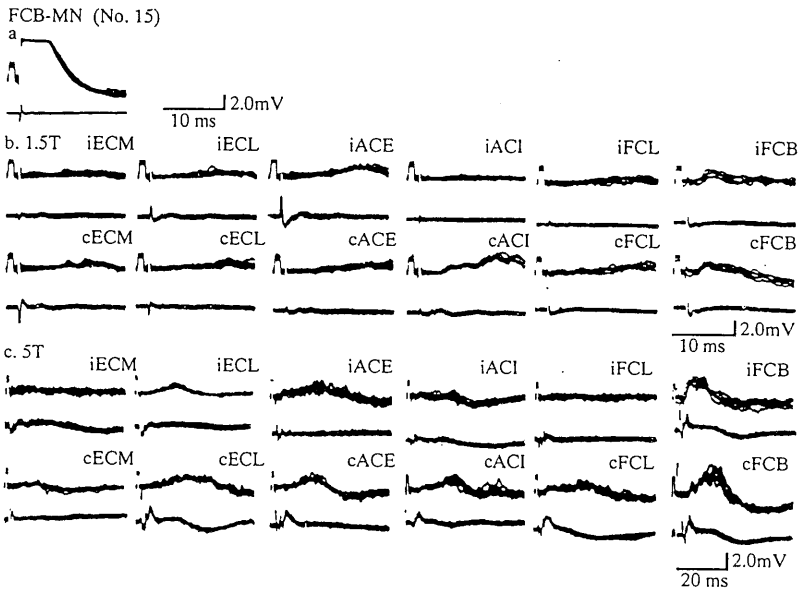
Figs. 5-7


ACI-MN (No. 13)
a
2.0mV
10 ms
b. 1.5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
10 ms
c. 5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
20 ms

5


FCL-MN (No. 7)
a
2.0mV
10 ms
b. 1.5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
10 ms
c. 5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
20 ms

6


FCB-MN (No. 15)
a
2.0mV
10 ms
b. 1.5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
10 ms
c. 5T
iECM
iECL
iACE
iACI
iFCL
iFCB
cECM
cECL
cACE
cACI
cFCL
cFCB
2.0mV
20 ms

7

Fig. 8

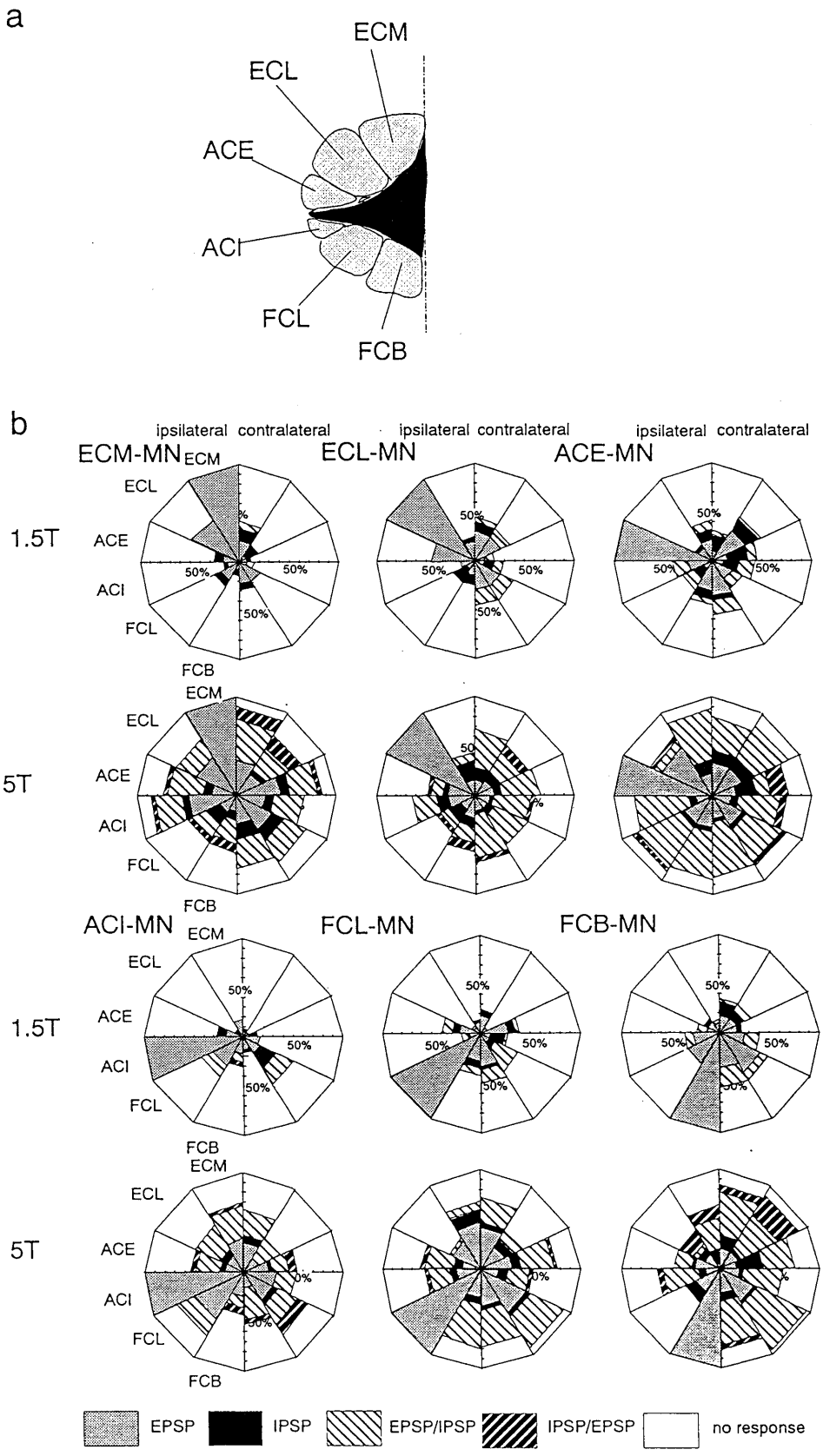
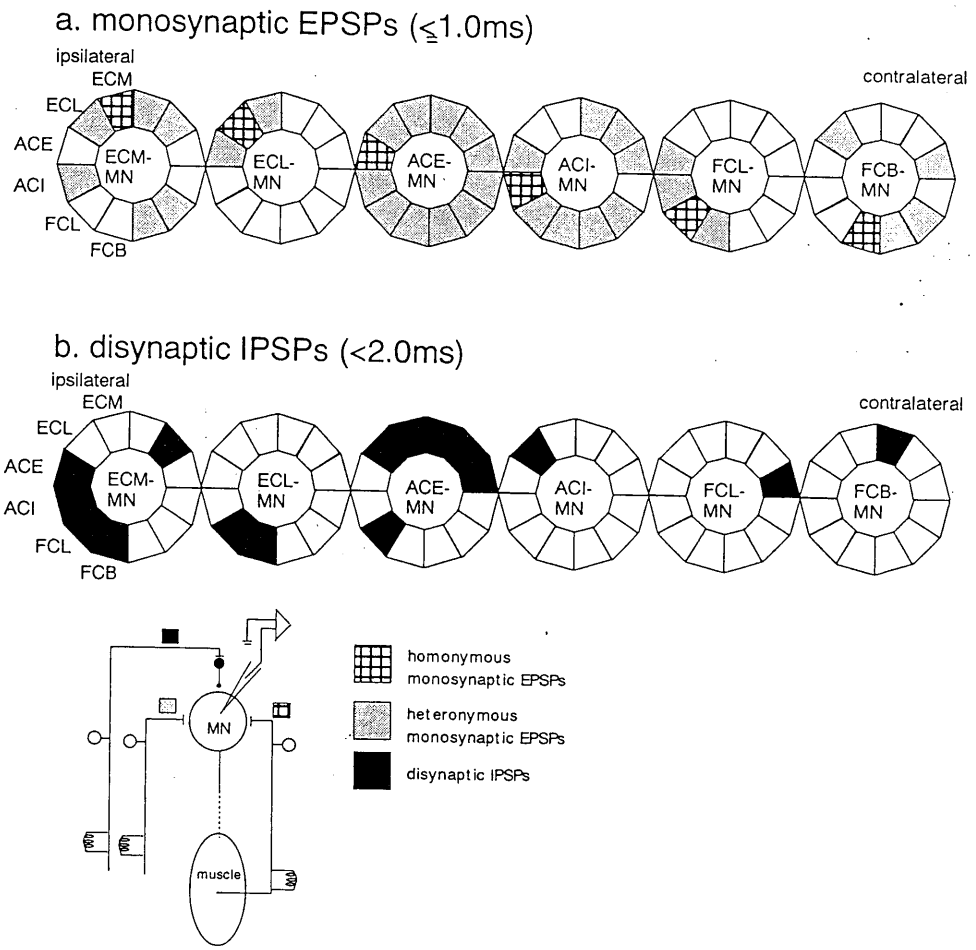

a
ECM
ECL
ACE
ACI
FCL
FCB
b
ipsilateral contralateral
ipsilateral contralateral
ipsilateral contralateral
ECM-MN
ECL-MN
ACE-MN
1.5T
5T
ACI-MN
FCL-MN
FCB-MN
50%
EPSP
IPSP
EPSP/IPSP
IPSP/EPSP
no response

Fig. 9


a. monosynaptic EPSPs (≦1.0ms)
ipsilateral
contralateral
ECM
ECL
ACE
ACI
FCL
FCB
ECM-MN
ECL-MN
ACE-MN
ACI-MN
FCL-MN
FCB-MN
b. disynaptic IPSPs (<2.0ms)
ipsilateral
contralateral
ECM
ECL
ACE
ACI
FCL
FCB
ECM-MN
ECL-MN
ACE-MN
ACI-MN
FCL-MN
FCB-MN
MN
muscle
homonymous monosynaptic EPSPs
heteronymous monosynaptic EPSPs
disynaptic IPSPs

3. POLYSYNAPTIC NEURONAL PATHWAYS FROM GROUP I AND GROUP II AFFERENTS INNERVATING TAIL MUSCLES TO HINDLIMB MOTONEURONS IN THE CAT

Summary

Postsynaptic potentials evoked in motoneurons innervating m. posterior biceps and semitendinosus (PBSt) and m. triceps surae (GS) by low threshold afferents from various tail muscles located at the level of the second-third caudal vertebrae were investigated in the non-anesthetized and spinalized cat. Afferent inputs from tail muscles on both sides predominantly evoked depolarizing potential in PBSt motoneurons and hyperpolarizing potential in GS motoneurons. The findings suggest that in general, tail muscle afferents facilitate flexor and inhibit extensor hindlimb motoneurons through polysynaptic pathways, so that the pelvic girdle is kept in a low position to maintain the stability of the body irrespective of different movements or posture of the tail.

Introduction

Information from various receptors such as muscle spindle, Golgi tendon organ, cutaneous receptors, and joint receptors is important for controlling movements and posture (Lundberg, 1979; Prochazka et al. 1989; Rossignol et al. 1989; Schomburg, 1991). We have been studying the neuronal control of tail movements in cats (Wada et al. et al. 1993; 1995a; 1995b; 1996a; 1996b). Our findings indicate that peripheral afferent inputs from other parts of the body as well as various descending pathways influence activities of the tail motoneurons. On the other hand, Goldfinger and Fukami (1982) demonstrated the high density of muscle spindles and Golgi

tendon organs in tail muscles, suggesting that afferent inputs from the tail muscles play an important role in controlling the movement not only of the tail itself but also of the limbs and trunk. In the present experiments, we studied the afferent inputs from the different tail muscles to hindlimb motoneurons in low spinalized cats.

Materials and Methods

The experiments were performed on 11 adult cats (2.5-5.0 kg) of either sex. Under anesthesia with halothane-nitrous oxide, the animal was decerebrated by passing a spatula rostroventrally from a line about 1 mm rostral to the superior colliculus, and the tissue rostral to the transection was suctioned with an aspirator. Then, anesthesia was discontinued, and the animal was further spinalized at the T13 spinal segment. A laminectomy was performed between L5 and the S1. The nerves innervating the m. posterior biceps and semitendinosus (PBSt, i.e., flexor muscle) and the m. triceps surae (GS, i.e., extensor muscle) of the left hindlimb were dissected out and severed near the entry to the muscle. Their central cut ends were mounted on the bipolar electrodes, and stimulated for identification of the PBSt or GS motoneurons. To stimulate afferents form the tail muscles, m. extensor caudae medialis (ECM), m. extensor caudae lateralis (ECL), m. abductor caudae externus (ACE), m. abductor caudae internus (ACI), m. flexor caudae longus (FCL), and m. flexor caudae brevis (FCB) , a pair of Teflon-insulated stainless steel wires (5mm gauge) with a 2-3 mm bared tip were inserted near the nerve in each muscle of both sides at the level of the second to third caudal vertebrae. The electrode position was carefully adjusted under an operating microscope so that stimulation through the electrodes produced a contraction only in the muscle in which the stimulating electrodes were located, as far as the stimulus intensity was

equal to or below five times threshold (5T). The threshold value was determined by an appearance of afferent volley in the cord dorsum potential (CDP) recorded at the S3-Co1 level, and ranged between 50-160 mV. When the stimulus intensity was raised as high as 10T, different levels of that muscle and/or other nearby muscles were also stimulated. Thus, stimulus intensity used in the present experiments ranged between 1.2 and 5T to avoid complication of current spread. All ventral roots from sacral and coccygeal spinal segments were sectioned. The animal was then fixed in a stereotaxic frame, paralyzed with pancronium bromide (0.4 mg/kg/h), and artificially ventilated. End-tidal CO2 concentration was monitored and maintained at approximately 4.0% by adjusting the respiratory rate or tidal volume. Rectal temperature was also monitored and maintained close to 37 °C with a heating mat. Mean arterial blood pressure was kept above 80 mmHg throughout the experiment. Using a glass microelectrode filled with 3 M potassium citrate solution (input resistance in the spinal cord: 25-40 MΩ), postsynaptic potentials (PSPs) produced by stimulation of tail muscle nerve were recorded from PBSt and GS motoneurons. The PBSt or GS motoneurons were identified by the presence of antidromic spikes after stimulation of the corresponding muscle nerve. The membrane potential of motoneurons and CDP recorded monopolarly at the L7 spinal segment were recorded on magnetic tape (TEAC, RD 135T) for later analysis. The segmental latency of PSPs was estimated from the difference between the onset times of the CDP and the PSP, which were averaged ten times by a signal processor (NEC, 7T17).

Stimulation of the muscle afferents at 1.2-1.8T evoked an incoming volley with simple shape in CDP. When the stimulus intensity was raised to 2-5T, a second deflection with longer latency appeared following the

first deflection. The conduction velocities of afferent fibers responsible for producing the first and the second deflections were estimated from the latency of these two waves and the conduction distance measured on the straight line that connected stimulating and recording electrodes, and were 71.3±7.5m/s and 43.7±5.4 m/s, respectively. Adal (1984) reported that the mean caliber was 8.5 mm for group I fibers and 5.0 mm for group II fibers in the tail muscle nerve. Thus, stimulation at an intensity equal to or below 1.8T seems to activate only group I muscle afferents, and at 2T or stronger intensity, both group I and group II muscle afferents were likely to be stimulated (Willis and Coggeshall, 1991).

Results

In the present experiments, PSPs recorded in 40 PBSt and 49 GS motoneurons were analyzed. The membrane potential was stable during recording, and the mean value for the total of 89 motoneurons was -68 mV. The mean amplitude of the antidromic spikes was 77 mV.

Figure 1 shows PSPs evoked in a PBSt motoneuron by stimulation of the nerves in different tail muscles. Stimulation of the ipsilateral ACE, ACI , FCL (iACE , iACI , iFCL) and contralateral ACI and FCL (cACI , cFCL) at 1.5T evoked EPSP (Fig. 1B). Other muscle nerves did not produce any apparent PSPs. At 5T, stimulation of afferents from all muscles except for iECM and cFCB (Fig. 1 C) evoked EPSP followed by IPSP (EPSP/IPSP). Records from a GS motoneuron are shown in Fig. 2. The EPSP was evoked by stimulation of iACE, iACI, iFCL, cACE and cFCL at 1.5T, but the amplitude of these potentials was relatively small compared to that of EPSPs in PBSt motoneurons (Fig. 2B). At 5T, stimulation of all tail muscle nerves except for iECM, iFCB and cFCB evoked EPSP followed by a large IPSP (Fig. 2C).

Table 1 shows the incidence (% of total) of different types of PSPs: EPSP, IPSP, EPSP/IPSP, and IPSP followed by EPSP (IPSP/EPSP). At 1.5T, 55% of PBSt motoneurons and 75% of GS motoneurons showed no PSPs. In PBSt motoneurons, EPSP or EPSP/IPSP was observed more frequently than IPSP or IPSP/EPSP, irrespective of the stimulated nerves. As the stimulus intensity was raised, the incidence of PSPs, especially of the EPSP/IPSP type, became higher. On the other hand, the incidence of IPSP was higher than that of EPSP in GS motoneurons, except in the cases of iACE and iACI stimulation. It could be considered that the effects of stimulation at 1.5T were due to the action of group I muscle afferents. It is not clear however, in the present experiments, whether these are mediated by Ia, Ib, or Ia+Ib, because we employed only electrical stimulation to activate muscle afferents. At 5T, PSP was evoked in 82% of PBSt motoneurons and 61% of GS motoneurons. The incidence of the EPSP/IPSP type of PSPs was greatly increased and most frequently seen in PBSt motoneurons. The difference between the effects of stimulation at 1.5T and 5T could be considered to be the result of additional inputs of group II muscle afferents. The divergence of group II muscle afferent inputs to various kinds of motoneurons and parallel excitatory and inhibitory pathways to each motoneuron pool in the lumbar spinal cord innervating hindlimb muscles has been reported (Schomburg, 1991). Group II muscle afferents innervating tail muscles seem to have similar divergent, parallel excitatory and inhibitory neuronal pathways to hindlimb motoneurons.

Central latencies of earliest PSPs are summarized in Table 2. The mean values of the averaged central latencies of PSP which were produced by group I muscle afferents ranged between 4.2 and 10.4 ms for PBSt

motoneurons, and 3.9 and 11.3 ms for GS motoneurons. In PBSt motoneurons, the minimal averaged central latencies of EPSP and IPSP, which were evoked by stimulation of iECM, were 3.1 and 2.2 ms, respectively. In GS motoneurons, the minimal averaged central latencies of EPSP and IPSP, which were produced by stimulation of cACI and iFCB, were 1.8 ms. In both PBSt and GS motoneurons, the average central latencies of PSPs after stimulation at 1.5T and 5T were independent of stimulated muscle nerves and varied greatly. These results suggest that neuronal pathways from tail muscle afferents, both group I and II afferents, to hindlimb motoneurons are probably polysynaptic via at least one interneuron (Lloyd and Wilson, 1995).

Discussion

It is very likely that low threshold (1.2-1.8T) group I afferents include those originating from the primary muscle spindle endings, and higher threshold (2-5T) group II afferents include those from the secondary muscle spindle endings. The present experiments demonstrated that both afferents evoked PSPs in the hindlimb motoneurons, indicating that hindlimb motoneurons are influenced by both dynamic movement and static position of tail. However, an increased incidence of PSPs at 5T stimulation compared to that at 1.5T suggests that the major inputs from tail to hindlimb motoneurons are mediated by group II afferents conveying static information. The activity of muscle spindles in each tail muscle seems to be influenced by direction, speed, and distance of dynamic movement, as well as static position of tail (Prochazka et al. 1989, and our unpublished data). In the present experiments, we found no specific relationship between the incidence of each type of PSP (i.e., EPSP, IPSP, EPSP/IPSP, or IPSP/EPSP) and the direction of tail movement which is

induced by activation of each tail muscle, or any combination of tail muscles examined. This suggests that hindlimb motoneurons are influenced by tail movements and posture, but not influenced by direction of tail movements and position. Furthermore, PSPs in PBSt motoneurons were predominantly depolarizing (EPSP or EPSP/IPSP) and those in GS motoneurons were hyperpolarizing in nature, indicating that, in general, muscle afferent inputs from tail facilitate flexor motoneurons and inhibit extensor motoneurons innervating the hindlimb. Thus, it might be considered that activation of neuronal pathways from low threshold muscle afferent innervating tail muscle keeps the pelvic girdle in a low position to maintain body balance during tail movements.

Table 1
Distribution of different types of PSPs evoked in PBSt and GS motoneurons by stimulation of low threshold afferents innervating different tail muscles

Nerves	Ipsilateral					Contralateral				
	EPSPs	IPSPs	EPSP/IPSP	IPSP/EPSP	No response	EPSPs	IPSPs	EPSP/IPSP	IPSP/EPSP	No response
PBSt-MN (n = 40)										
ECM	6 (12%)	0	2 (5%)	0	32 (80%)	10 (25%)	0	2 (5%)	0	27 (67%)
	2 (5%)	7 (17%)	15 (37%)	0	16 (40%)	10 (25%)	7 (17%)	14 (35%)	0	9 (22%)
ECL	5 (12%)	0	2 (5%)	0	33 (82%)	3 (7%)	4 (10%)	1 (2%)	0	32 (80%)
	4 (10%)	8 (20%)	16 (40%)	2 (5%)	10 (25%)	18 (45%)	9 (22%)	18 (45%)	0	13 (32%)
ACE	8 (20%)	4 (10%)	3 (7%)	0	25 (62%)	5 (12%)	0	2 (5%)	0	33 (82%)
	8 (20%)	4 (10%)	12 (30%)	1 (2%)	15 (37%)	6 (15%)	3 (7%)	20 (50%)	0	11 (27%)
ACI	7 (17%)	1 (2%)	2 (4%)	0	30 (75%)	10 (25%)	0	6 (15%)		24 (60%)
	10 (25%)	5 (12%)	12 (30%)	0	13 (32%)	3 (7%)	5 (12%)	21 (52%)		11 (27%)
FCL	3 (7%)	0	1 (2%)	0	36 (90%)	7 (17%)	3 (8%)	0	0	30 (75%)
	9 (22%)	4 (10%)	12 (30%)	0	15 (37%)	10 (25%)	8 (20%)	12 (30%)	1 (2%)	9 (22%)
FCB	0	0	0	0	40 (100%)	3 (7%)	0	1 (2%)	0	36 (90%)
	10 (25%)	3 (7%)	6 (12%)	1 (2%)	20 (50%)	5 (12%)	5 (12%)	7 (17%)	1 (2%)	22 (55%)
GS-MN (n = 49)										
ECM	0	6 (12%)	0	0	43 (87%)	2 (4%)	3 (6%)	0	0	44 (89%)
	0	10 (22%)	0	1 (2%)	38 (77%)	3 (6%)	7 (14%)	13 (26%)	0	26 (53%)
ECL	1 (2%)	3 (8%)	0	1 (2%)	44 (89%)	0	1 (2%)	0	0	48 (97%)
	0	10 (20%)	12 (24%)	2 (4%)	25 (51%)	1 (2%)	6 (12%)	9 (18%)	0	33 (67%)
ACE	4 (8%)	1 (2%)	1 (2%)	0	44 (89%)	1 (2%)	4 (8%)	1 (2%)	0	43 (87%)
	1 (2%)	6 (12%)	4 (8%)	0	38 (77%)	2 (4%)	8 (16%)	15 (30%)	0	24 (48%)
ACI	3 (6%)	0	0	0	46 (93%)	3 (6%)	5 (10%)	3 (6%)	5 (10%)	41 (83%)
	0	2 (4%)	3 (6%)	0	44 (89%)	0	8 (16%)	12 (24%)	0	29 (59%)
FCL	1 (2%)	3 (6%)	1 (2%)	0	44 (89%)	0	6 (12%)	0	0	43 (87%)
	0	5 (10%)	7 (14%)	0	37 (75%)	1 (2%)	8 (16%)	10 (20%)	1 (2%)	29 (59%)
FCB	2 (4%)	3 (6%)	0	0	44 (89%)	0	6 (12%)	0	0	43 (87%)
	0	6 (12%)	4 (8%)	0	39 (79%)	0	10 (20%)	2 (4%)	2 (4%)	35 (71%)

Upper columns: PSPs produced by the stimulation at 1.5 T.
Lower columns: PSPs produced by the stimulation at 5.0 T.

Table 2
Averaged central latencies of earliest PSPs produced by afferent inputs from different tail muscles

Nerves	Ipsilateral		Contralateral	
	EPSPs	IPSPs	EPSPs	IPSPs
PBSt-MN				
ECM	4.9 ± 0.7 (3.5)	–	4.5 ± 0.4 (3.7)	–
	7.0 ± 3.4 (3.1)	5.1 ± 2.8 (2.2)	6.8 ± 3.0 (3.5)	5.3 ± 2.6 (3.8)
ECL	4.7 ± 0.8 (3.5)	–	4.2 ± 0.5 (3.7)	4.7 ± 0.9 (3.7)
	7.1 ± 2.6 (3.2)	6.2 ± 3.3 (2.7)	7.1 ± 3.1 (3.8)	5.6 ± 1.6 (3.7)
ACE	4.7 ± 0.9 (4.0)	5.3 ± 1.2 (5.0)	4.5 ± 0.4 (4.0)	–
	6.2 ± 2.0 (3.9)	6.8 ± 1.4 (5.0)	6.5 ± 2.3 (4.0)	5.9 ± 0.9 (4.8)
ACI	4.3 ± 0.5 (3.7)	4.2	5.1 ± 0.6 (4.9)	–
	6.3 ± 1.8 (4.1)	5.7 ± 1.3 (4.1)	7.3 ± 2.4 (4.7)	5.6 ± 1.1 (4.4)
FCL	–	–	5.4 ± 0.9 (4.3)	4.1 ± 0.5 (3.8)
	6.1 ± 2.8 (4.2)	10.4 ± 3.0 (6.9)	7.1 ± 2.2 (4.5)	6.0 ± 1.9 (3.7)
FCB	–	–	4.3 ± 0.4 (3.8)	–
	6.1 ± 2.1 (4.1)	6.8 ± 2.6 (2.9)	7.3 ± 3.3 (3.8)	5.2 ± 1.0 (4.0)
GS-MN				
ECM	–	4.3 ± 0.5 (3.9)	4.5 ± 1.4 (3.5)	4.3 ± 0.4 (3.8)
	–	6.3 ± 2.1 (3.8)	7.0 ± 3.7 (3.2)	6.2 ± 2.2 (3.7)
ECL	1.9	2.6 ± 1.0 (1.9)	–	4.3
	4.9 ± 2.7 (1.8)	6.7 ± 2.3 (1.8)	7.5 ± 4.4 (3.1)	6.1 ± 1.6 (4.1)
ACE	5.1 ± 0.5 (4.7)	5.6	3.7 ± 0.7 (3.1)	5.0 ± 1.3 (3.2)
	5.8 ± 0.9 (4.5)	9.1 ± 2.9 (5.5)	5.8 ± 2.5 (3.0)	6.1 ± 3.5 (3.2)
ACI	8.6 ± 0.5 (8.3)	–	4.2 ± 0.6 (3.4)	4.7 ± 1.2 (3.1)
	11.3 ± 4.2 (8.8)	7.7 ± 1.1 (7.5)	8.8 ± 3.9 (3.2)	5.4 ± 2.1 (3.0)
FCL	4.8 ± 0.2 (4.6)	4.9 ± 0.4 (4.6)	–	5.6 ± 1.6 (4.5)
	7.0 ± 3.2 (4.5)	6.7 ± 2.2 (4.6)	7.8 ± 3.4 (3.1)	7.1 ± 2.4 (4.4)
FCB	4.0 ± 1.2 (3.1)	3.9 ± 0.9 (3.0)	–	4.6 ± 0.7 (3.8)
	7.6 ± 1.0 (2.8)	6.5 ± 2.2 (2.9)	5.5 ± 1.0 (4.4)	6.3 ± 2.0 (3.9)

Values are mean ± S.D. (minimal) in ms.
Upper columns: PSPs produced by the stimulation at 1.5 T.
Lower columns: PSPs produced by the stimulation at 5.0 T.

Figure legends

Fig. 1. Postsynaptic potentials (PSPs) evoked in a PBSt motoneuron by electrical stimulation of tail muscle nerves. Upper traces, intracellular records. Lower traces, afferent volleys recorded from the surface of the spinal cord close to the L7 dorsal root entry zone. A: antidromic action potential. Note that the spike is truncated. B: PSPs evoked by the stimulation of different tail muscle nerves. Stimulus intensity is 1.5 times threshold for the lowest threshold afferents in each nerve. C: Same as in B, but stimulus intensity is 5.0 times threshold. See text for details.

Fig. 2. Postsynaptic potentials evoked in a GS motoneurons. Figure arrangement and stimulating nerves are the same as in Fig. 1.

Fig. 1

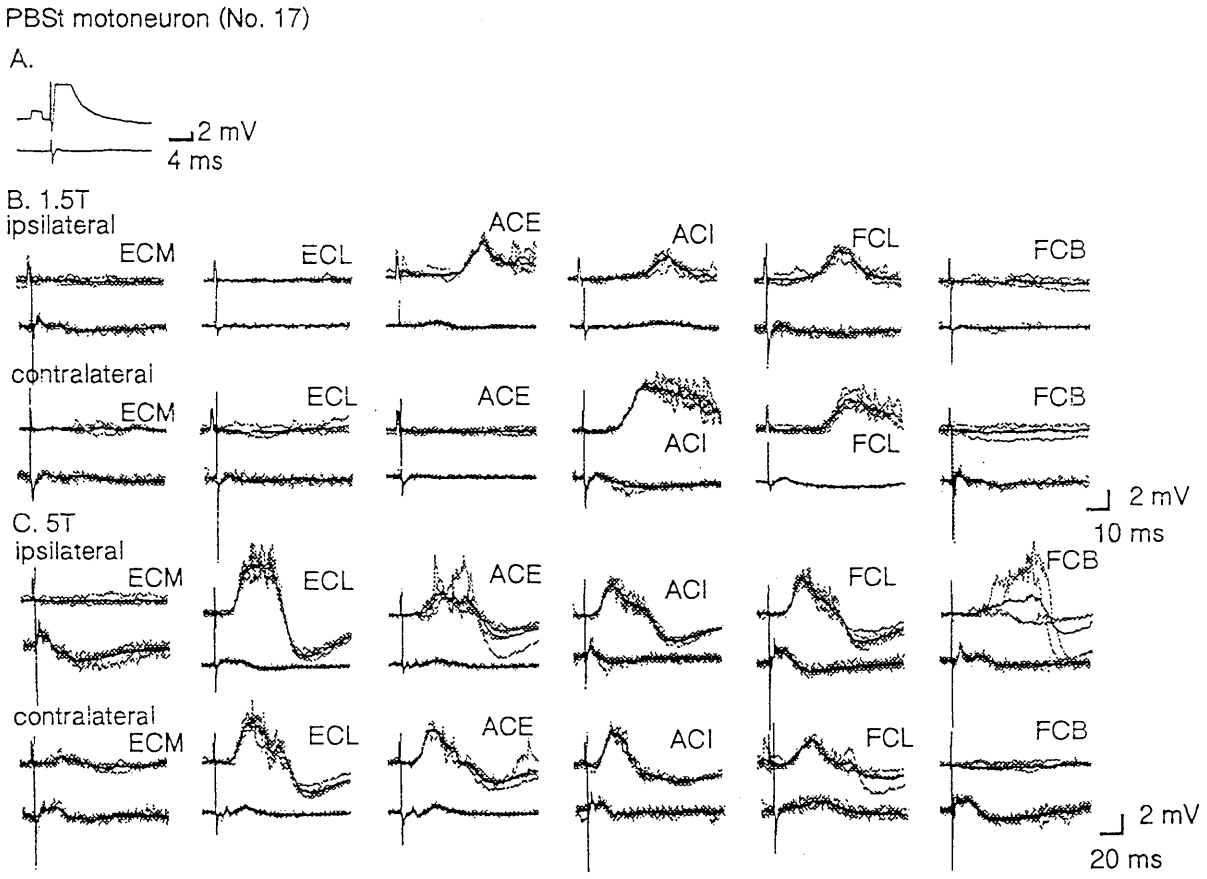
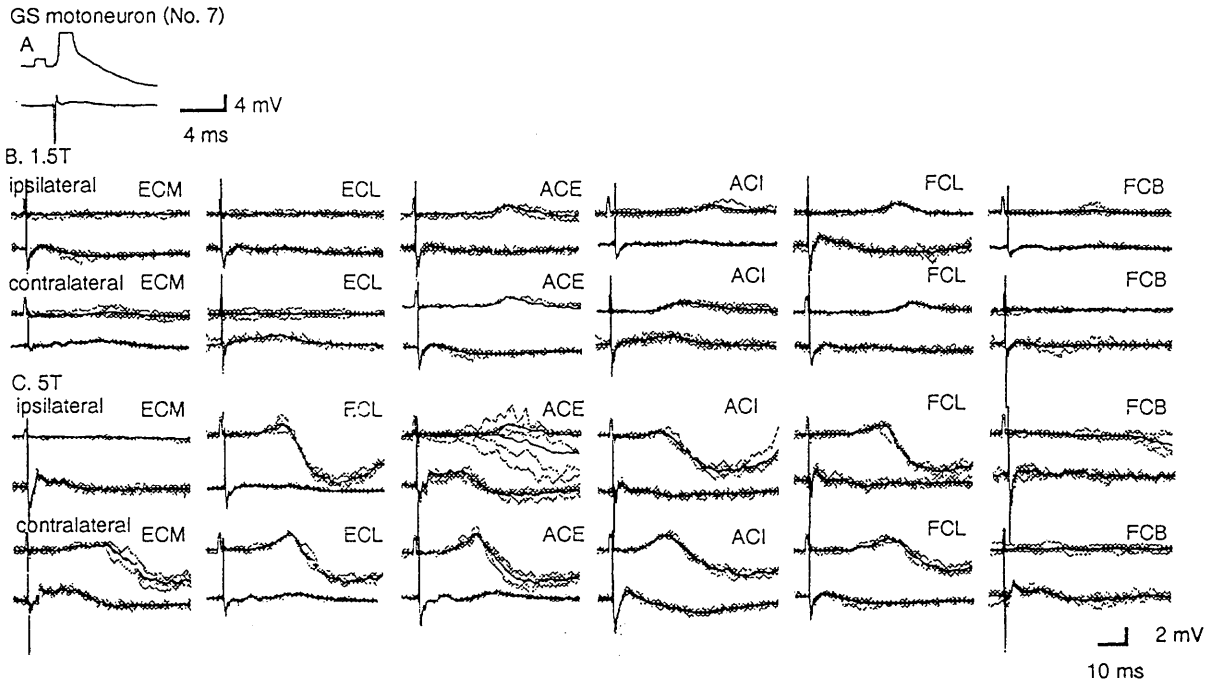

PBSt motoneuron (No. 17)
A.
2 mV
4 ms
B. 1.5T
ipsilateral
ECM
ECL
ACE
ACI
FCL
FCB
contralateral
ECM
ECL
ACE
ACI
FCL
FCB
2 mV
10 ms
C. 5T
ipsilateral
ECM
ECL
ACE
ACI
FCL
FCB
contralateral
ECM
ECL
ACE
ACI
FCL
FCB
2 mV
20 ms

Fig. 2


GS motoneuron (No. 7)
A
4 mV
4 ms
B. 1.5T
ipsilateral
ECM
ECL
ACE
ACI
FCL
FCB
contralateral
ECM
ECL
ACE
ACI
FCL
FCB
C. 5T
ipsilateral
ECM
ECL
ACE
ACI
FCL
FCB
contralateral
ECM
ECL
ACE
ACI
FCL
FCB
2 mV
10 ms

4. Neuronal pathways from low threshold hindlimb cutaneous afferents to motoneurons innervating trunk muscles in low spinalized cats.

Summary

Experiments were performed on 22 adult cats of both sexes. The animals were decerebrate non-anesthetized, spinalized at T13-L1 and maintained on artificial respiration.

Postsynaptic potentials (PSPs) evoked in motoneurons innervating the back (L5) and abdominal (L3) muscles in the lumbar part of the body by stimulating hindlimb cutaneous afferent nerves at 2 - 20 times the threshold (T) of each peripheral nerve were investigated. Electrical stimulation of hindlimb cutaneous nerves at 5T produced various types of PSPs: pure EPSPs, pure IPSPs and mixed PSPs (EPSP/IPSPs and IPSP/EPSPs) in 86% of motoneurons. Motoneurons were classified into 2 groups: motoneurons qualitatively showing the same effects and motoneurons showing different effects after stimulating ipsilateral and contralateral cutaneous nerves. In the former type, there were motoneurons showing PSPs of markedly different sizes and showing PSPs of similar sizes after stimulating ipsilateral and contralateral cutaneous nerves, respectively. It could be considered that the former type of motoneuron is related to vertical movements or stiffness of the trunk, while the latter type and the former type showing PSPs of markedly difference sizes are related to lateral bending of the trunk.

Introduction

The trunk represents a large part of the total body mass and constitutes an integral part of the motor system. Adequate control of trunk movements and posture is therefore a prerequisite for the maintenance of body equilibrium during various motor tasks. The trunk muscles of the lumbar part of the cat's body consist of the back muscles (m. multifidus lumborum, m. longissimus lumborum and m. iliocostalis lumborum) and the abdominal muscles (m. obliquus internus abdominus, m. obliquus externus abdominus, m. transversus abdominus and m. rectus abdominus) (Crouch 1969; Holstege et al. 1987). Activation of the back muscles produces extension of the dorsal column and lateral bending of the trunk and maintenance of the posture of the trunk (Crouch 1969). Some investigators suggested that the primary role of the back muscles during locomotion is to control the stiffness of the back in order to make the best use of propulsive force produced by hindlimb movements (Carlson et al. 1979; English 1980; Zomlefer et al. 1984). The function of the abdominal muscles is flexion and bending of the trunk, and increases abdominal pressure, which promotes defecation, urination, expiration and parturition (Floyd and Silver 1950; McCarthy and Borison 1974; Youmans et al. 1974). When an animal moves or maintains posture, afferent inputs from various mechanical receptors innervating various part of the body cause adjustment of the movements and the posture of the animal as needed (Baxendale et al. 1987; Boivie and Pearl 1975; Lundberg 1979; Prochazka et al. 1989; Rossignol et al. 1989).In particular, movements and posture of the lumbar part of the trunk are strongly influenced by movements of the hindlimb or placement of the hindfoot (personal observations). These facts suggest the importance of afferent inputs from the hindlimb for controlling movements and posture of

the lumbar part of the trunk. However, there have been very few studies of neural control of trunk muscle motoneurons(Carlson and Lindquvist 1976; Jankowska and Odutola 1980; Koehler et al. 1984). Koehler et al.(1984) demonstrated that rhythmic activities from trunk motoneurons were influenced by hindlimb cutaneous nerve stimulation during fictive locomotion. Carlson and Lindquist (1976) showed the effects of hindlimb cutaneous nerve stimulation on lumbar back muscle tone and reflex. However, they did not show any information about neuronal pathways from different kinds of hindlimb cutaneos afferens to motoneurons innervating different kind of trunk muscles. In the present report, We studied the effects of three cutaneous nerves from the hindlimb in motoneurons innervating caudal parts of 3 lumbar back muscles, m. multifidus lumborum (Multi), m. longissimus lumborum (Long) and m. iliocostalis lumborum (Ilio), and 2 abdominal muscles, m. obliquus externus abdominus (OEA) and m. obliquus internus abdominus (OIA), in spinalized cats using an intracellular recording technique.

Materials and methods

Experiments were performed on 22 adult cats (2.2-4.5kg) of either sex. Under anesthesia with halothane-nitrous oxide, the animals were decerebrated by passing a spatula rostroventrally from a line about 1 mm rostral to the superior colliculus and aspirating the tissue rostral to the transection. After decerebration, anesthesia was discontinued. The spinal cord was completely transected at T13-L1. In 12 cats (Group-A), the nerves innervating the m. multifidus (Multi), the m. longissimus (Long) and the m. iliocostalis (Ilio) from the L5 spinal segment on the left side were isolated from surrounding tissues. In 10 cats (Group-B), the nerves innervating the

m obliquus externus abdominus (OEA) and the m. obliquus internus abdominus (OIA) from the L3 spinal segments on the left side were isolated. The following hindlimb cutaneous nerves were isolated on both sides: the sural cutaneous nerve supplying the lateral hindlimb skin (Sur), the tibial nerve supplying the plantar part of the foot (Tib) and the superior peroneus cutaneous nerve supplying the instep of the foot (SPc). Laminectomy was performed between L4 and L7 in Group-A and between L2 and L7 in Group-B. The animals were fixed in a stereotaxic frame. The animals were paralyzed with pancuronium bromide (0.4 mg/kg per h) and artificially ventilated. End-tidal CO2 concentration was monitored and maintained at approximately 4.0% by adjusting the respiratory rate and tidal volume. The rectal temperature was monitored and maintained close to 37 °C with a heating mat. Arterial blood pressure was monitored and the mean blood pressure was maintained at above 80 mmHg during the experiments. The exposed spinal cord and isolated muscle nerves were covered with warm mineral oil (37-38 °C). The isolated muscle and cutaneous nerves were mounted on bipolar electrodes for stimulation. Intracellular recordings from motoneurons in the L3 (Group-B) or L5 (Group-A) spinal segments were obtained using glass microelectrodes filled with 3M potassium citrate solution (input resistance in the spinal cord, 20-45MΩ). The reported observations are from motoneurons in which recordings were stable for at least 30 min and action potentials were ≥ 60mV. The trunk muscle motoneurons were identified by the presence of antidromic action potentials after stimulating trunk muscle nerves. The hindlimb cutaneous nerves were electrically stimulated at 2- 20 times the threshold (T) of each peripheral nerve (0.1 ms duration), determined by monopolar recording of incoming volley at the L7 segmental level. In 58 motoneurons (3 Multi, 5 Long, 21

Ilio 19 OEA, 10 OIA motoneurons) in 16 cats (Group-A: 11, Group-B: 5), EPSPs and IPSPs were proved by changing of PSP size and/or reversing the potential by injecting depolarizing and hyperpolarizing current, respectively.

The intracellular membrane potential and cord dorsum potential (CDP) were recorded on magnetic tape (TEAC, RD-135T). The average central latency value and size of the earliest PSPs (resting level-peak amplitude) were obtained by signal averaging of 10 consecutive sweeps (MacLab/8s, ADInstruments). Minimal central latencies were measured during 10 signal sweeps (MacLab/8s, ADInstruments).

Results

Stable recordings were obtained from 159 motoneurons innervating trunk muscles (12 Multi, 21 Long, 53 Ilio, 51 OEA, 22 OIA motoneurons). The membrane potentials and spike amplitudes averaged approximately -65 (minimum - maximum values; -58 - -71 mV) mV and 72 mV (minimum - maximum values; 63 - 80 mV), respectively.

Figure 1 shows examples of PSPs recorded from Multi (Fig. 1A), Long (Fig.1B) and Ilio (Fig. 1C)、 OEA (Fig. 1D) and OIA (Fig. 1E) motoneurons evoked by single pulse stimulation of hindlimb cutaneous nerves at 2-10T. In the Multi and OEA motoneurons, EPSPs or EPSPs followed by IPSPs (EPSP/IPSPs) were produced by stimulation of Sur, SPc or Tib on both sides at 2-10T (PSPs after stimulating SPc and Tib at 5T are shown in Fig. 1A and 1D). The stimulation of contralateral Sur (cSur) at 10T produced spikes in this OEA motoneuron. In the Long and OIA motoneurons, EPSPs or EPSP/IPSPs were produced by stimulation of Sur on both sides at 2T-10T (Fig. 1B and 1D). In this OIA motoneuron, the

sizes of the earliest EPSPs and subsequent IPSPs (the resting level-peak values) after stimulating cSur were larger than those after stimulating iSur at the same intensity. In 12 out of 61 trunk motoneurons which showed EPSPs or EPSP/IPSPs after stimulating Sur on both sides at the same stimulus intensity (1 Multi, 2 Long, 3 Ilio, 4 OEA and 2 OIA motoneurons), the sizes of the earliest EPSP and subsequent IPSPs after stimulating cSur were larger than those after stimulating iSur. In the Ilio motoneuron, IPSPs were produced by stimulation of ipsilateral Sur (iSur) at 5T and 10T (Fig. 1C). In general, the earliest EPSPs appeared at 2-5T, and the size of the earliest EPSPs was increased by increasing the stimulus intensity from 2T or 5T to 5-10T, while the earliest IPSPs and subsequent IPSPs appeared at 2-5T, and the size of the IPSPs reached a maximum at around 10T.

Figure 2 shows the incidence (%) of different types of PSPs: EPSP, IPSP, EPSP/IPSP, IPSP followed by EPSP (IPSP/EPSP) at 2T (upper column) and 5T (lower column). The incidence of PSPs was markedly increased as stimulus intensity increased from 2T to 4-5T, while the effects on the PSP incidence of further increasing the stimulus intensity from 4-5T to 10T or 20T was very small. At 2T, PSPs were not observed in approximately 60% of motoneurons. EPSPs were the most frequently produced PSPs in all of the various trunk muscle motoneurons after stimulation of all hindlimb cutaneous nerves on both sides at 2T. At 5T, PSPs was evoked in 86% (137/159) of recorded motoneurons. Notably, the incidence of mixed PSPs (EPSP/IPSPs, IPSP/EPSPs) and IPSPs was greatly increased. In general, the incidence of PSPs was greater after stimulating the ipsilateral nerves than after stimulating the contralateral nerves at 2T and 5T. At 2T, the incidence of PSPs after stimulating Sur (45%) was greater than that after stimulating SPc (23%) or Tib (17%) in motoneurons innervating abdominal muscles

(OEA and OIA). At 5T, the difference in incidence depending on the kind of stimulated nerves was remarkable only in Multi motoneurons (Sur: 83%, SPc and Tib: 66%).

We classified motoneurons into 8 groups on the basis of the effects of stimulation of ipsilateral and contralateral Sur at 5T (Table 1). In trunk muscle motoneurons, motoneurons showing EPSP or EPSP/IPSP after stimulating Sur on both sides (EPSP(EPSP/IPSP)-EPSP(EPSP/IPSP)) were most frequently observed. In all Multi motoneurons showing any effects (83%), qualitatively the same effects (EPSP(EPSP/IPSP)-EPSP(EPSP/IPSP), IPSP-IPSP) were observed after iSur or cSur stimulation. In the remainding motoneurons, the incidences of motoneurons showing the same effects on both sides were 61% in Long, 44% in Ilio, 59% in OEA and 53% in OIA, while incidences of motoneurons showing bilaterally asymmetrical responses were 39% in Long, 56% in Ilio, 40% in OEA and 46% in OIA motoneurons. The percentage of motoneurons showing symmetrical responses was significant only in Multi motoneurons. We measured the size of the earliest PSPs in motoneurons which showed qualitatively the same effects after stimulation Sur on both sides (EPSP(EPSP/IPSP)-EPSP(EPSP/IPSP)). Remarkable differences in the size of PSPs in which the size of PSP evoked by stimulation of Sur on one side was less than 50% of the size of PSP evoked by stimulation of the contralateral Sur, were observed in 5 Multi, 6 Long, 11 Ilio, 9 OEA and 3 OIA motoneurons.

The average central latencies of the earliest PSPs and the minimum central latencies are summarized in Table 2. In back muscle motoneurons, the minimal central latencies of EPSP and IPSP, which were produced by stimulation of iSPc, were 1.2 (in the Long motoneuron) and 2.2 ms (in the

Ilio motoneuron), respectively. In abdominal muscle motoneurons, the minimal central latencies of EPSP and IPSP were 1.9 and 3.0 ms (in the OEA motoneuron), respectively.

Discussion

It is known that there are different types of afferent fibers, Aαβ and C fibers, in cutaneous nerves (Boivie and Pearl 1975; Willis and Coggeshall 1991). Aαβ fibers are stimulated at intensity below 5T, and it has been reported that the threshold of A δ fibers is approximately 5T when single square-pulse (0.1 ms duration) electrical stimuli are applied to cutaneous nerves (Boivie and Pearl 1975; Hori et al. 1986; Willis and Coggeshall 1991). C fibers are stimulated at 20-25T (Eccles and Lundberg 1959; Willis and Coggeshall 1991). Thus, it seems that the electrical stimulation at 2T in the present experiments activates Aαβ fibers, at 5T Aαβ + low threshold component of A δ fibers , and at 10T Aαβ+ A δ fibers.

It has been postulated that the shortest neuronal circuit from cutaneous afferent fibers innervating the hindlimb to hindlimb motoneurons have postulated a disynaptic pathway based on a segmental latency of 1.2 ms (Fleshman et al. 1988), 1.3 ms (Illert et al. 1975), or 1.5 ms (Hongo et al. 1969). The minimum central latency was 1.2ms in back muscle motoneurons in L5, and this fact suggests that the shortest neuronal pathways from hindlimb cutaneous fibers to back muscle motoneurons in L5 are disynaptic pathways. Since the branching patterns and the conduction velocity for afferent fibers in the lumbar spinal cord are not known, we cannot say whether the 1.7 ms latency in abdominal muscle motoneurons in L3 corresponds to a disynaptic pathway or not in the present experiments.

The results of the present experiments indicate that the inputs from cutaneous afferents supplying the lateral hindlimb skin (Sur), the plantar part of the foot (Tib) and the instep of the foot (SPc), influence trunk muscle activities. The purpose of the present experiments was to delineate the neuronal control of trunk muscle motoneurons by inputs from mechanical receptors activated during movement an posture. It has been reported that Aαβ fibers innervate mechanoreceptors, such as the Pacinian corpuscle, Merkel cells, Meissner's corpuscles and hair follicle endings (Willis and Coggeshall 1991). These receptors are activated by pressure, touch or vibrations which are produced by movements of the leg joints, foot landing, changing the position of the center of gravity, and so on. Concerning the physiological role of neuronal pathways from Aαβ fibers, we classified motoneurons into 8 groups on the basis of the effects of ipsilateral and contralateral Sur afferent inputs (Table 1). As described in the Introduction, the activation of trunk muscles produces vertical and lateral trunk movements and controls the stiffness of the trunk. It seems likely that motoneurons showing qualitatively the same effects after stimulation of cutaneous nerves on both sides are related to vertical movements and the stiffness of the trunk, while motoneurons showing different types of effects depending on the side of stimulation and marked differences in the

size of PSPs are related to lateral bending of the trunk. All Multi motoneurons showed the former type of effects after stimulating the Sur nerve on both sides and this fact suggests that the primary role of neuronal pathways from hindlimb cutaneous afferents to Multi motoneurons is to control the stiffness of the back (Crouch 1969). In the remainding kinds of motoneurons, various response patterns after stimulating ipsilateral and contralateral hindlimb cutaneous nerves were observed. This fact suggests that neuronal pathways from hindlimb cutaneous afferents to Long, Ilio, OEA and OIA motoneurons are to control vertical movements, the stiffness and lateral bending of the trunk. In the present experiments, we performed electrical stimulation on all branches of Sur. LaBella et al.(1989) reported that stimulation of the separate branches showed a completely different pattern of effects in hindlimb motoneurons (m. triceps surae). It could be considered that stimulation of a separate branch of Sur produces different effects on trunk motoneurons.

The incidence of PSPs after stimulating ipsilateral cutaneous nerves was greater than that after stimulating contralateral nerves. These facts suggest a close relationship between movements of the hindlimb and trunk on the same side.

The incidences of PSPs in and Multi and abdominal motoneurons (OEA and OIA) were greater after stimulating Sur than Tibs or SPc

at 5T and 2T, respectively. These facts suggest a close relationship between Multi & abdominal muscles and knee joint movements.

Koehler et al.(1984) demonstrated that rhythmic activities from motoneurons innervating Long and OEA in non-anesthetized and spinalized cats were influenced by hindlimb cutaneous nerve (Sur and SPc) stimulation during fictive locomotion. Especially, rhythmical discharge from OEA motoneurons was strongly enhanced by stimulation of cutaneous nerves. Carlson and Lindquist (1976) showed that the effects of hindlimb cutaneous nerve stimulation on lumbar back muscle tone and reflex were qualitatively same in decerebrated and spinalized cats. They observed that facilitation can be evoked from ipsilateral hindlimb while inhibition results from stimulation of other limbs. Our results showed that low threshold cutaneous afferent inputs from hindlimb cutaneous afferents on both sides produced both excitatory and inhibitory effects in motoneurons innervating the lumbar back and abdominal muscle. It might be considered that neuronal pathways shown in the present experiments partly correspond to effects of hindlimb cutaneous afferent inputs on trunk motoneurons demonstrated by Koehler et al and Carlson & Lindquist.

It has been reported that cutaneous interneurons are heavily controlled by supraspinal inputs (Baldissera et al. 1981) and our data

may not have functional significance in intact moving cats.

Table. 1, 2

Ipsilateral-contralateral	Multi	Long	Ilio	OEA	OIA
EPSP (EPSP/IPSP)-EPSP (EPSP/IPSP)	8	9	16	23	5
	66	42	30	46	22
IPSP-IPSP	2	2	5	5	3
	16	9	9	13	13
EPSP (EPSP/IPSP)-no response	0	2	10	6	5
	0	9	18	12	22
IPSP-no response	0	3	6	10	2
	0	14	11	21	9
EPSP (EPSP/IPSP)-IPSP	0	2	8	1	0
	0	9	15	2	0
IPSP-EPSP (EPSP/IPSP)	0	0	3	0	0
	0	0	5	0	0
No response-EPSP (EPSP/IPSP)	0	0	0	2	0
	0	0	0	4	0
No response-no response	2	3	5	4	7
	16	14	9	7	31
Total	12	21	53	51	22

Nerves	Ipsilateral		Contralateral	
	EPSP	IPSP	EPSP	IPSP
Multi motoneuron				
Sur	3.1±1.4	4.4±2.1	4.1±1.0	6.4±1.6
	1.6	2.2	2.7	5.1
SPc	4.7±2.2	4.7±1.2	4.5±3.1	
	1.4	3.4	1.4	
Tib	4.3±2.1		3.1±1.5	
	2.0		1.4	
Long motoneuron				
Sur61	5.3±1.1	4.4±1.0	6.1±1.5	7.3±1.3
	3.3	3.4	3.0	5.9
SPc61	7.2±6.0	5.9±2.7	6.2±3.8	11.3±4.9
	1.2	5.0	1.4	4.4
Tib	5.4±3.1	5.9±4.9	5.3±2.7	7.9±2.0
	1.4	2.8	1.3	6.0
Ilio motoneuron				
Sur61	5.6±1.2	6.5±2.0	6.1±3.5	6.9±1.4
	2.8	3.8	3.6	3.6
SPc	6.3±3.7	5.8±1.3	10.6±2.9	6.4±2.9
	1.6	4.0	4.8	4.9
Tib	4.3±1.3	5.3±2.2	5.0±3.6	7.8±4.0
	1.7	2.2	1.8	5.1
OEA motoneuron				
Sur	5.3±1.1	5.4±1.0	6.1±1.5	7.3±1.3
	3.6	3.5	3.2	5.7
SPc	7.2±5.0	5.9±2.7	6.1±2.8	8.6±4.9
	1.9	5.3	1.9	5.4
Tib	5.4±2.2	5.9±4.9	5.1±2.1	7.9±2.0
	2.5	3.0	2.1	6.0
OIA motoneuron				
Sur	5.4±1.8	6.2±1.7	6.3±1.5	8.1±1.4
	2.7	4.1	3.6	6.2
SPc	4.0±2.3	7.7±1.3	5.6±1.6	9.0±2.4
	3.1	6.1	3.2	6.3
Tib	5.9±2.1	4.5	5.1±2.7	8.3
	2.4		1.9	

Figure legends

Fig. 1. Postsynaptic potentials (PSPs) evoked in Multi (A), Long (B), Ilio (C), OEA (D) and OIA motoneurons (F) by electrical stimulation of hindlimb cutaneous nerves on the ipsilateral and contralateral sides at 2T-20T. Upper and lower traces in each panel show PSPs and CDPs. Each trace consists of 6 superimposed single sweeps after single pulse stimulation (dotted lines) and the average of these 6 sweeps (solid lines).In the Multi, Long, OEA and OIA motoneurons, EPSP or EPSP followed by IPSP (EPSP/IPSP) were produced by hindlimb cutaneous nerves on both sides at 2-10T. In the Ilio motoneuron, IPSPs were produced by ipsilateral Sur stimulation when stimulus intensity was above 5T.

Fig. 2. Incidence (%) of different types of PSPs in Multi, Long, Ilio, OEA and OIA motoneurons after stimulating hindlimb cutaneous nerves on both sides at 2T (upper column) and 5T (lower column). At 2T, EPSPs were predominantly produced. At 5T, IPSPs and EPSPs following IPSPs were significantly increased. See text for details.

Fig. 1

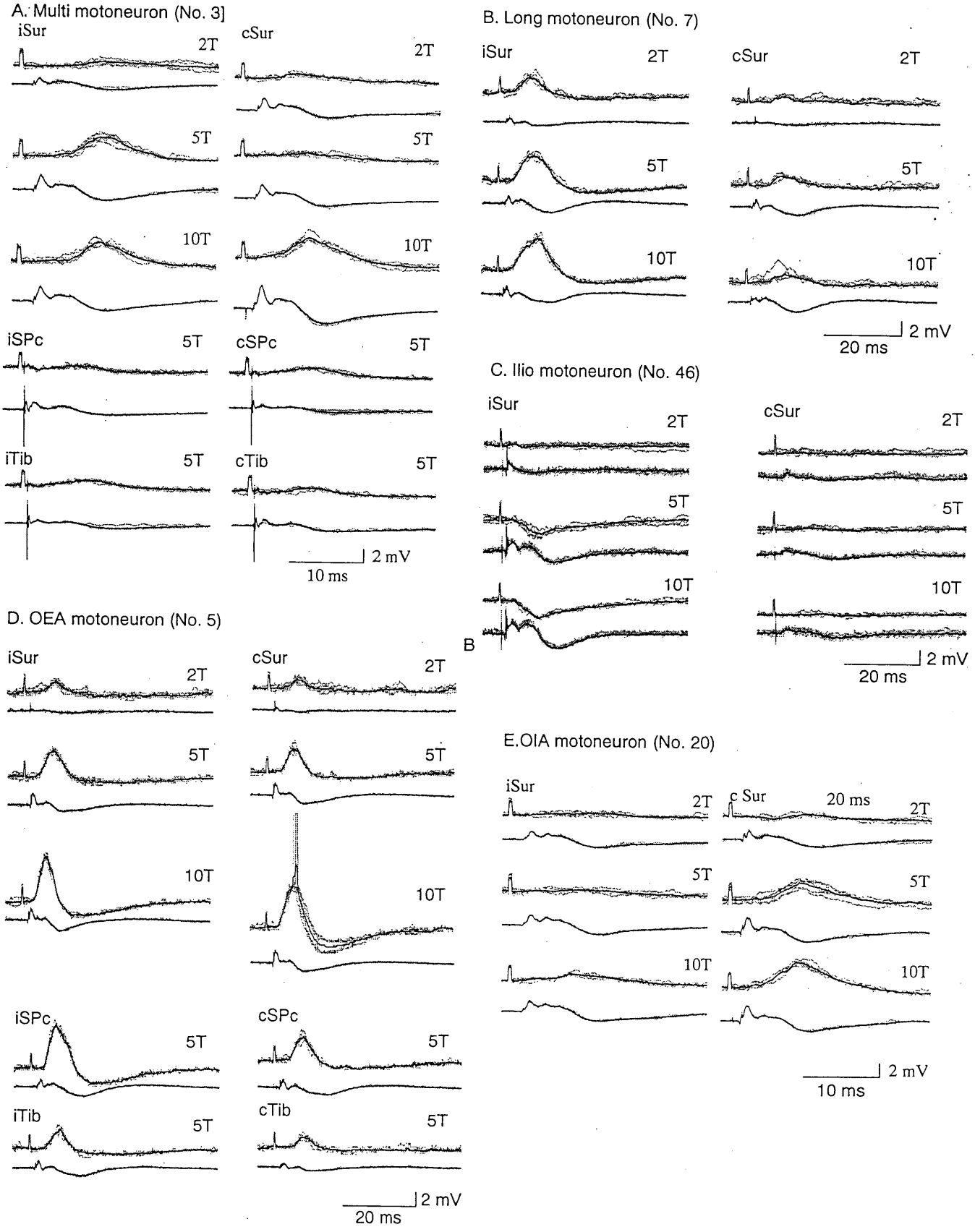
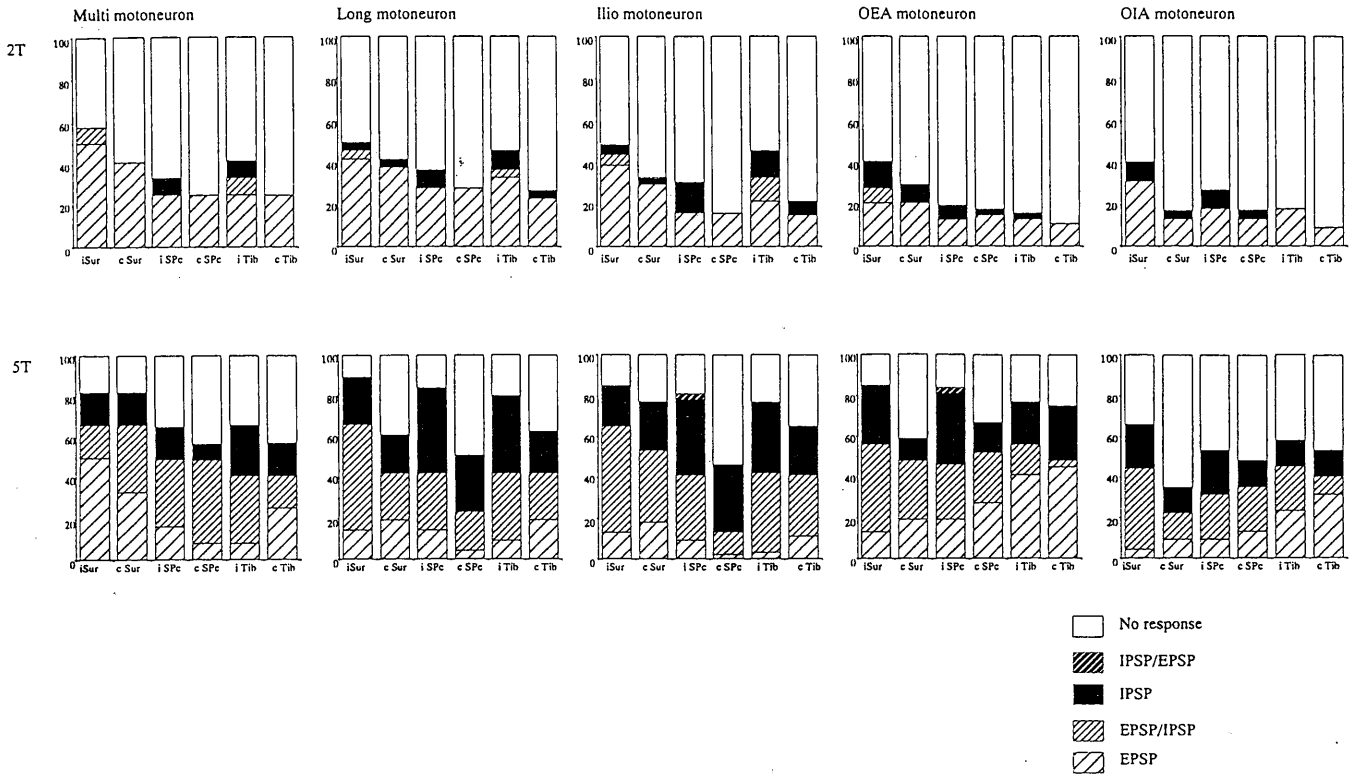

A. Multi motoneuron (No. 3]
iSur
2T
cSur
2T
5T
5T
10T
10T
iSPc
5T
cSPc
5T
iTib
5T
cTib
5T
2 mV
10 ms
B. Long motoneuron (No. 7)
iSur
2T
cSur
2T
5T
5T
10T
10T
2 mV
20 ms
C. Ilio motoneuron (No. 46)
iSur
2T
cSur
2T
5T
5T
10T
10T
B
2 mV
20 ms
D. OEA motoneuron (No. 5)
iSur
2T
cSur
2T
5T
5T
10T
10T
iSPc
5T
cSPc
5T
iTib
5T
cTib
5T
2 mV
20 ms
E.OIA motoneuron (No. 20)
iSur
2T
c Sur
20 ms
2T
5T
5T
10T
10T
2 mV
10 ms

Fig. 2


Multi motoneuron
Long motoneuron
Ilio motoneuron
OEA motoneuron
OIA motoneuron
2T
5T
100
80
60
40
20
0
iSur
c Sur
i SPc
c SPc
i Tib
c Tib
No response
IPSP/EPSP
IPSP
EPSP/IPSP
EPSP

5. Polysynaptic neuronal pathways from tail cutaneous afferents to hindlimb motoneurons in the spinalized cat

Summary

Postsynaptic potentials (PSPs) were recorded in motoneurons innervating the m. anterior bicepus and semimembranosus (ABSm), m. posterior biceps and semitendinosus (PBSt-MN) and m. triceps surae (GS-MN), m. plamtaris (Pl), m flexor digitorum longus and hollicis longus (FDHL), m. popliteus (Pop) and m. tibialis antrior (TA) in 15 spinalized adult cats, after electrical stimulation of muscle nerves innervating tail muscles on both sides following, the m. extensor caudae medialis (ECM), m. extensor caudae lateralis (ECL), m. abductor caudae externus (ACE), m. abductor caudae internus (ACI), m. flexor caudae longus (FCL) and m. flexor caudae brevis (FCB). With stimulation at 1.5-5 time threshold, inhibitory PSPs (IPSPs), excitatory PSPs (EPSPs), and mixed PSPs (IPSP/EPSPs, EPSP/IPSPs) were recorded in approximately 90% of PBSt-MN and 70% of GS-MN. IPSPs (IPSPs, IPSP/EPSPs) after stimulation of the contralateral TVC and TDC were observed in 54% and 52% of PBSt motoneurons, respectively. EPSPs (EPSP, EPSP/IPSPs) were recorded after stimulation of ipsilateral TVC in 56% of PBSt motoneurons. IPSPs produced after stimulation of ipsilateral TDC and TVC were observed in 65% and 63% of GS-MN showing PSPs after stimulation of tail cutaneous nerves, respectively. Measurement of segmental latencies of the earliest PSPs (PBSt-MN, IPSPs: 2.1-7.4 ms, EPSPs: 3.1-18.9 ms, GS-MN, IPSPs: 2.5-17.1 ms, EPSPs: 4.2-19.4 ms) suggested that most of the neural pathways from tail cutaneous afferents to hindlimb motoneurons are at least

trisynaptic in the L7 spinal segments. Hemisection of spinal cord at S1-S2 indicated that neural pathways from both the ipsilateral and contralateral low threshold cutaneous afferents pass through the ipsilateral spinal cord at S1-S2. It could be considered that the neural pathways from tail cutaneous afferents to hindlimb motoneurons maintain the balance of the pelvic girdle.

Introduction

We have been studying the neuronal control of tail movements. The motoneurons innervating tail muscles are influenced by various descending pathways and peripheral afferent inputs. The afferThe tail is a conspicuous part of an animal. The shape, size and function of tails vary according to the animal species and tails perform important tasks for each animal. Some investigators have suggested that four legged animals with long tails like cats, cheetahs, and so on use their tails as a rudder to maintain body balance (Kiley-Worthington 1975; Necker 1970). There have been few studies investigating neural control of tail movements, however. Our group has been studying neural control of tail movements in the cat (Wada 1991; Wada et al. 1990, 1993, 1994, 1995a, b, 1996a, b). Our results demonstrated that various peripheral afferent inputs influence the activity of tail motoneurons (Wada 1991, Wada et al. 1995a, b, 1996a). It is also necessary to investigate the neural pathways from peripheral afferents innervating the tail to understand the functional role of tail. In the present experiment, we studied the effects of cutaneous afferent inputs from the tail on the hindlimb motoneurons in spinalized cats.

Materials and Methods

The experiments were performed on 19 adult cats (2.5-5.0 kg) of either sex. Animals were anesthetized with pentobarbital-sodium (40-50 mg/kg, ip) and were decerebrated by passing a spatula rostroventrally from a line about 1 mm rostral to the superior colliculus and aspirating tissue rostral to the transection. Spinalization was performed at the T11-12 spinal segment. Laminectomy was performed between L5 and the sacral vertebrae. The nerves to flexor (m. posterior biceps and semitendinosus: PBSt) and extensor muscles (m. triceps surae: GS) of the left hindlimb were severed and mounted on bipolar stimulating electrodes. The cutaneous nerves innervating the dorsal (TDC) and ventral part of the tail (TVC) on both sides at the caudal vertebral 1-3 levels, were severed and mounted on bipolar stimulating electrodes. The ventral roots of spinal segments below L7 on both sides were cut and the proximal end of the L7 ventral root on the left side was mounted on bipolar electrodes for stimulation. The animals were then fixed in a stereotactic frame, paralyzed with pancuronium bromide (0.4 mg/kg per h) and artificially ventilated. End-tidal CO2 concentration was monitored and maintained at approximately 4.0% by adjusting the respiratory rate or tidal volume. Rectal temperature was monitored and maintained close to 37 C with a heating mat. Mean arterial blood pressure was usually above 80 mmHg during the experiments. The experiment was stopped when the mean blood pressure fell below 80 mmHg. Intracellular recordings from hindlimb motoneurons were performed with glass microelectrodes filled with 3 M potassium citrate solution (input resistance in the spinal cord: 8-20 MOhm. Motoneurons were identified by the presence of antidromic spikes and Ia excitatory postsynaptic potentials after stimulation of the muscle nerves. Tail

cutaneous nerves were electrically stimulated with 1.2-25 times the threshold (T) of each peripheral nerve (square pulse, duration: 0.1 ms), determined by monopolar recording of the incoming volley at the first or second coccygeal spinal segmental (Co 1-2) level. The intracellular membrane potential and cord dorsum potential (CDP) recorded by monopolar recording electrode placed on the L7 spinal segment, were recorded on magnetic tape (TEAC, RD-135T). Estimates of segmental latency were obtained by subtracting the onset time of the CDP, the first deflection in the CDP, from the onset time of the PSP. The average segmental latencies were measured on 20 time-averaged PSPs using a signal processor (NEC, 7T17). The shortest segmental latencies were measured on 10 consecutive responses as the minimal segmental latencies for a particular cutaneous nerve-motoneurons. We employed student-t test to confirm the difference of segmental latencies.

Results

Postsynaptic potentials after stimulation of tail cutaneous nerves

In the present experiment, stable recordings were performed from 49 PBSt and 42 GS motoneurons. The membrane potential and spike amplitude averaged approximately - 68 mV and 77 mV, respectively.

Figure 1 shows examples of PSPs recorded from a PBSt motoneuron after stimulating TDC and TVC on both sides at various stimulus intensities. The earliest PSPs evoked by stimulating TDC and TVC on both sides at low stimulus intensity (1.5-2T) were hyperpolarizing postsynaptic potentials (IPSPs). Increasing the stimulus intensity of

ipsilateral and contralateral TDC (iTDC and cTDC) from 1.5 T to 5T led to an increase in the amplitude of IPSPs, and depolarizing postsynaptic potentials (EPSPs) following IPSPs were detectable at 5-10T. Depolarizing potentials following IPSPs were evoked by stimulating iTVC and cTVC at 1.5T. Increasing stimulus intensity on tail cutaneous nerves from 1.5T to 5T increased the size of PSPs or produced EPSPs following IPSPs. Further increases in stimulus intensity from 5T to 10T-25T did not affect PSPs in most PBSt motoneurons.

Figure 2 shows the distribution of average segmental latencies of the earliest PSPs in PBSt motoneurons after stimulating TDC and TVC on both sides at 5.0T. The hatched and filled bars demonstrate the average segmental latencies of EPSPs and IPSPs, respectively. The histograms demonstrate that the average segmental latencies of EPSPs and IPSPs are distributed over a wide range. The mean values of the average segmental latencies of EPSPs and IPSPs are shown by arrow heads in the histograms. The mean values of average segmental latencies of IPSPs and EPSPs were observed at around 5 and 10 ms, respectively. The average segmental latencies of IPSPs were shorter than those of EPSPs in PBSt motoneurons ($P< 0.01$). The minimal values of the average segmental latencies of IPSPs and EPSPs in PBSt motoneurons were 2.1 and 3.1 ms and were observed in PSPs evoked by stimulating cTDC and iTVC, respectively. The minimal segmental latencies of these PSPs were 1.7 ms and 2.7 ms, respectively.

Figures 3A and B show examples of PSPs in GS motoneurons following stimulation of tail cutaneous nerves at 1.5-10T. The organization of these figures is similar to that in Fig. 1. In Fig. 3A, IPSPs followed by EPSPs were produced after stimulating all tail cutaneous

nerves at 1.5T. Increasing the stimulus intensity from 1.5T to 10T led to an increase in the size of PSPs. Further increases in stimulus intensity from 10T to 25T did not affect PSPs. In Fig. 3B, EPSPs were detectable after stimulating TDC on both sides at 5-10T, while EPSPs followed by hyperpolarization were evoked by stimulation of TVC on both sides at low stimulus intensity(1.5-2.0T). Increasing TVC stimulus intensity from 1.5 to 5T led to an increase in the size of PSPs, although further increases from 5 T to 10-25T did not influence the size of PSPs.

Figure 4 represents the distribution of average segmental latencies of the earliest PSPs in GS motoneurons after stimulating TDC and TVC on both sides at 5.0T. The organization of Fig. 4 is similar to that of Fig. 2. The average segmental latencies of EPSPs and IPSPs after tail cutaneous nerve stimulation were distributed over a wide range. The range of average segmental latencies of earliest PSPs in GS motoneurons was wider than that in PBSt motoneurons. The minimal values of the average segmental latencies of IPSPs and EPSPs were 2.5 and 4.2 ms, and were observed on PSPs evoked by stimulating cTDC and cTVC, respectively. The minimal segmental latencies of these PSPs were 2.1 and 3.9 ms, respectively.

Table 1 shows the PSPs evoked by stimulation of tail cutaneous nerves at 5T on PBSt motoneurons (upper column) and GS motoneurons (lower column). Stimulation of tail cutaneous nerve at 5T evoked PSPs in 96% of PBSt motoneurons (47/49) and in 72% of GS motoneurons (30/42). In PBSt motoneurons and GS motoneurons, stimulation of ipsilateral tail cutaneous nerves more effectively evoked PSPs than stimulation of contralateral cutaneous nerves. Stimulation of iTVC was most effective in evoking PSPs in both PBSt and GS motoneurons. Stimulation of tail cutaneous nerves evoked EPSPs, IPSPs and mixed PSPs (EPSP/IPSP,

IPSP/EPSP), but the rates at which EPSPs, IPSPs or mixed PSPs appeared, differed depending on the kind of cutaneous nerve stimulated and the kind of motoneuron. The earliest PSPs after stimulating cTDC and cTVC were IPSPs (IPSPs or IPSP/EPSPs) in 52% (26/49) and 54% (27/49) of PBSt motoneurons, respectively, while PSPs after stimulating iTVC were EPSPs (EPSPs or EPSP/IPSPs) in 56% of PBSt motoneurons. The rate of EPSP (33%, 21/49) was similar to that of IPSP (28 %, 22/49) when stimulation was performed on iTDC. PSPs after stimulating iTDC and iTVC were IPSPs (IPSP or IPSP/EPSP) in 65% (15/23) and 63% (19/30) of GS motoneurons showing PSPs. The rate of IPSPs was similar to that of EPSPs after stimulating the contralateral tail cutaneous nerves in GS motoneurons (iTDC, EPSPs: 11/49, IPSPs: 11/49. cTVC, EPSPs: 12, IPSPs: 11).

Effects of spinal transections and hemisections on postsynaptic potentials

The above results demonstrate the existence of neural pathways from tail cutaneous afferents to hindlimb motoneurons. To identify the neural pathways in the spinal cord from ipsilateral and contralateral tail cutaneous afferents to hindlimb motoneurons, we obtained recordings of PSPs from PBSt and GS motoneurons after spinal transection at the L6 and Co3 and hemisection between the S1 and S2 spinal segments in 6 cats. Figure 5 shows PSPs recorded from PBSt motoneurons after spinal transections and hemisections. The transeced or hemiseced sites on the spinal cord are indicated by the hatched square in the lower part of Fig. 5. After spinal transection at L5-6 and Co3-4 spinal segments (Fig. 5A), EPSPs were recorded after stimulating all tail cutaneous nerves at 1.5T. After

hemisection of the contralateral side of S1-S2 spinal segments in addition to spinal transection at L5-6 and Co3-4 spinal segments (Fig. 5B), EPSPs were evoked by stimulating tail cutaneous nerves at 1.5T. Stimulation of tail cutaneous nerves after the spinal transection at L6 and Co3 spinal segments or hemisection of the contralateral side at S1-S2 in addition to spinal transection at L6 and Co3 spinal segments, evoked IPSP, EPSP and mixed PSPs. After hemisection of the ipsilateral side of the S1-S2 spinal segments in addition to spinal transection at L6 and Co3 (Fig. 5C), PSPs were not induced by stimulation of tail cutaneous nerves at 1.2-5T in any of the recorded PBSt motoneurons (15 PBSt motoneurons: Fig. 5C-1, stimulus intensity: 10T), however, EPSPs with long segmental latencies (> 20 ms) and amplitudes larger than 3.4 mV (3.8 (0.44 mV) were observed in 3 of 15 PBSt motoneurons when the stimulus intensity was 10-25T (Fig. 5C-2). EPSPs with long segmental latencies (> 20 ms) and large PSPs amplitudes (> 3.4 mV) were not observed in either PBSt or GS motoneurons before hemisection of the ipsilateral side of S1-S2.

Discussion

Afferents responsible for PSPs in PBSt and GS motoneurons

PSPs were evoked by stimulating tail cutaneous nerves at 1.5T-2T and increasing stimulus intensity from 1.5T to 5-10T increased the size of PSPs or evoked PSPs following the earliest PSPs. Increasing the stimulus intensity from 5-10T to 20-25T did not induce additional effects on PSPs in most motoneurons. At low intensity stimulation (1.2-5T), Aab fibers with a lower threshold were activated (Boivie and Perl 1975; Willis and

Coggeshall 1991). It has also been reported that the threshold of Ad fibers is approximately 5T when a single square-pulse (0.1 ms in duration) electrical stimulus was applied to cutaneous nerves (Beall et al. 1977; Hori et al. 1986). Thus, PSPs evoked by low intensity stimulation at 1.5-5T may have been mainly attributable to activation of Aab fibers. Increasing stimulus intensity from 5 T to 10-25 T did not cause PSPs in most motoneurons. This fact suggests that afferent inputs from high threshold cutaneous afferent nerves (Ad and C fibers) do not affect hindlimb motoneuron activity. However, Behrends et al. (1983) demonstrated with the spatial facilitation technique in hindlimb motoneurons, that inputs activated by noxious radiant heat converge on interneurons activated by inputs from Aab fibers. This finding suggests that Aab or Ad, as well as thin afferent nerves share some common interneurons in the reflex circuit from hindlimb cutaneous afferents to hindlimb motoneurons. The results in the present experiments show that although the effects of low threshold cutaneous afferents are predominant, we cannot exclude the effects of inputs from thin cutaneous afferent nerves on hindlimb motoneurons. After hemisection of the ipsilateral spinal cord at S1-S2 (Fig. 5C), EPSPs with long latency and large amplitude were produced by stimulation of tail cutaneous nerves at 10-25T. These EPSPs were not observed when the stimulus intensity was 1.2-5T. This fact suggests that high threshold cutaneous afferents from the tail influence hindlimb motoneurons.

Pathway

The average segmental latencies in the L7 spinal segment of early PSPs in PBSt and GS motoneurons were distributed over a wide range. This finding demonstrates the existence of polysynaptic neural pathways

from tail cutaneous afferents to hindlimb motoneurons. Furthermore, segmental latencies of IPSPs were shorter than those of EPSP in both PBSt and GS motoneurons, and this finding suggests that EPSPs neural pathways have more interneurons than those of IPSPs or slower conducting fibers in the L7 spinal segment. Some investigators observed a short latency (<1.8 ms) EPSPs in the motoneurons and indicated that the minimal circuitry from hindlimb cutaneous afferents to hindlimb motoneuron was disynaptic (Edgley and Wallace 1989; Fleshman et al. 1984, 1988). In the present experiments, there were no EPSPs with a segmental latency shorter than 2.7 ms in either PBSt or GS motoneurons. These data suggest that neural pathways producing EPSPs from tail cutaneous afferents to hindlimb motoneurons are at least trisynaptic in the L7 spinal segment. Leahy and Durkovic (1991) demonstrated the existence of short-latency IPSPs (< 1.8 ms) and suggested the existence of a disynaptic cutaneous inhibitory circuit in hindlimb motoneurons. In the present experiments, the minimal segmental latencies of IPSPs were 1.7 ms in PBSt motoneurons and 2.5 ms in GS motoneurons. These findings suggest that most neural pathways producing IPSPs are at least trisynaptic in the L7 spinal segments, but we cannot exclude the possibility of disynaptic pathways to PBSt motoneurons in the L7 spinal segments.

PSPs after spinal transections at L6 and Co3 were similar to PSPs before spinal transections. This finding indicates that the PSPs seen in the present experiments were evoked by activation of neural circuits in the spinal segments between L7 and Co2. Hemisection of the contralateral side of the spinal cord at S1-S2 did not alter the PSPs pattern. Hemisection of the ipsilateral side of the spinal cord at S1-S2, however, abolished PSPs with a segmental latency shorter than 20 ms. These findings indicate that

the neural pathways from afferent fibers from the contralateral side cross at S3-Co2 and both neural pathways from the ipsilateral and contralateral tail cutaneous afferents pass through the ipsilateral side of the spinal cord at S1-S2. After hemisection of the ipsilateral side of the spinal cord at S1-S2, depolarizing potentials with latencies longer than 20 ms and large PSP amplitudes (> 3.7 mV) were observed when stimulation at 10-25T was applied to tail cutaneous nerves on both sides. It may be that these EPSPs were produced by actions of neural pathways from high threshold cutaneous afferents (Ad), through the contralateral side of the spinal cord. These large amplitude EPSPs with prolonged latencies were not observed before hemisection of the ipsilateral side at S1-S2. These data suggest that the neural pathways producing large amplitude EPSPs with prolonged latencies are inhibited by action of the neural pathways through the ipsilateral side of the spinal cord.

Function

Stimulation of cutaneous nerves produced different types of PSPs, EPSP, IPSP and mixed PSPs in PBSt and GS motoneurons. The effects of stimulating tail cutaneous afferents demonstrated in this experiment were varied and understanding the physiological functions of all these results is difficult. However, the rates at which EPSPs, IPSPs and mixed PSPs appeared differed depending on the kind of cutaneous nerve stimulated and the kind of motoneuron. In PBSt motoneurons, inhibitory effects were predominant after stimulating contralateral cutaneous nerves, while excitatory effects were often observed after stimulating ipsilateral cutaneous nerves. In GS motoneurons, inhibitory effects were predominant

after stimulation of ipsilateral cutaneous nerves. The rate at which EPSPs appeared was similar to that of IPSPs after stimulation of contralateral cutaneous nerves in GS motoneurons. We tried to understand the physiological role of the neural pathways which produced these predominant effects of tail cutaneous afferent inputs on PBSt and GS motoneurons. Figure 6 demonstrates the predominant actions of neural pathways from tail cutaneous afferents onto hindlimb motoneurons which are revealed in the present experiments (Fig. 6d) as well as those of hindlimb cutaneous nerves onto tail motoneurons shown in the previous paper (Fig. 6c; Wada et al. 1995b). Fig. 6 also includes the neural pathways mediating flexion reflex (Fig. 6a) and crossed extension reflex of the hindlimb (Fig. 6b; Baldissera et al. 1989). The filled and opened triangles indicate inhibitory and excitatory synapses, respectively. The hatched triangle on the neural pathways from tail cutaneous nerves to contralateral GS motoneurons indicates that the rate at which EPSPs appeared was similar to that of IPSPs. Activation of cutaneous afferents innervating the hindlimb evoked polysynaptic actions that were related to the pattern of the ipsilateral flexion reflex (Fig. 6a) and the crossed extension reflex (Fig. 6b), with appropriate reciprocal synaptic action on antagonist motoneurons (Eccles and Lundberg 1959). In a previous experiment, we studied the actions of neural pathways from low threshold cutaneous afferents innervating the hindlimb onto tail motoneurons (Wada et al. 1995b). The effects of neural pathways from hindlimb cutaneous afferents to tail motoneurons were excitatory and these excitatory effects were stronger in the ipsilateral motoneurons (large opened triangle) than the contralateral ones (small opened triangle). It may be that activation of these neural pathways (Fig. 6c) produces flexion of the tail toward the side of the

stimulated hindlimb. We previously showed that cutaneous receptors with low threshold afferent fibers innervating the tail skin were activated by stretching the skin during tail flexion (Wada et al. 1996b). The cutaneous afferent inputs from one side of the tail inhibited the contralateral flexor motoneurons (PBSt-MN) and the ipsilateral extensor motoneurons (GS-MN), and excited ipsilateral flexor motoneurons. The neural pathways from tail cutaneous afferents to hindlimb motoneurons inhibit the effects of neural pathways from hindlimb cutaneous nerves to hindlimb motoneurons. It seems as though the flexor reflex movement of the hindlimb shifts the center of gravity of the pelvic girdle to the side contralateral to the stimulated hindlimb, producing an imbalance. Tail reflex movement to the stimulated side produced by activation of the neural circuits from the hindlimb cutaneous nerves to tail motoneurones shifts the gravity center of the pelvic girdle toward the stimulated side. Furthermore, reflex movements of the hindlimb are inhibited by activation of the neural circuits from tail cutaneous afferents to hindlimb motoneurons. Thus, the neural circuit shown in Fig. 6 shows the neural circuit via tail cutaneous afferents (Aab) that may act to maintain body balance during flexor reflex movements of the hindlimb. Stimulation of tail cutaneous nerves, however, produced both EPSPs and IPSPs and often showed mixed effects (EPSP/IPSP and IPSP/EPSP) on hindlimb motoneurons, while increasing the stimulus intensity evoked changes in PSPs. Furthermore, we did not investigate presynaptic inhibition due to cutaneous afferent inputs from the tail in the present experiments. Further studies are required to explain all of the findings in the present experiment.

Table 1

Table I. - *Responses evoked by stimulating tail cutaneous nerves (intensity 5 T)*

Nerve	EPSP	EPSP/IPSP	IPSP	IPSP/EPSP	No response
PBSt motoneurons(n = 49)					
iTDC	16 (32%)	5 (14%)	14 (28%)	8 (16%)	6 (12%)
cTDC	11 (22%)	3 (6%)	18 (36%)	8 (16%)	9 (18%)
iTVC	21 (42%)	7 (14%)	8 (16%)	11 (22%)	2 (4%)
cTVC	12 (24%)	1 (2%)	17 (34%)	10 (20%)	9 (18%)
GS motoneurons(n = 42)					
iTDC	7 (16%)	1 (2%)	14 (33%)	1 (2%)	19 (45%)
cTDC	9 (21%)	2 (4%)	9 (21%)	2 (4%)	20 (47%)
iTVC	7 (16%)	4 (9%)	17 (40%)	2 (4%)	12 (28%)
cTVC	8 (19%)	4 (9%)	10 (23%)	1 (2%)	19 (45%)

Figure Legends

Fig. 1. PSPs in a PBSt motoneuron evoked by stimulating tail cutaneous nerves. Traces were superimposed from 8 consecutive trials. The upper and lower traces of each panel show PSPs and CDPs. At 1.5T, IPSPs were observed after stimulating all cutaneous nerves. SPs following IPSPs were significant after stimulating TVC on both sides at 2-10T. Increasing the stimulus intensity from 1.5 to 5T resulted in increased PSP size.

Fig. 2. Distribution of average segmental latencies of early PSPs after stimulating tail cutaneous nerves at 5T in PBSt motoneurons. Filled and hatched bars indicate IPSP and EPSP, respectively. 10< indicates that average segmental latencies were longer than 10 ms. The histograms demonstrate that average segmental latencies were distributed over a wide range. Arrow heads indicate the mean values of average segmental latencies of EPSPs and IPSPs. The average segmental latencies of IPSPs were shorter than those of EPSPs ($P < 0.01$).

Fig. 3. PSPs evoked by stimulating tail cutaneous nerves in two GS motoneurons. Organization of this figure is similar to that of Figure 1. A) IPSPs were produced after stimulating all tail cutaneous nerves at 1.5T. Increasing stimulus intensity led to increased PSP size. B) EPSPs were evoked by stimulating TVC at 1.5T and TDC at 5T on both sides.

Fig. 4. Distribution of averaged segmental latency of early PSPs after stimulating tail cutaneous nerves at 5T in GS motoneurons. Figure

arrangement is similar to that in Fig. 2. The distribution of average segmental latencies in GS motoneurons was significant. See text for details.

Fig. 5. PSPs in PBSt motoneurons after spinal transection at L6 and Co 3 spinal segments and hemisection between S1 and S2 spinal segments. Stimulus intensities were 1.5T in A and B,10T in C-1 and 20T in C-2. Sites of spinal sections are shown by hached square in the lower part of this figure. The PSPs in A, B and C were recorded after spinal section of A, B and C , respectively. A and B: EPSPs were observed after stimulation of all tail cutaneous nerves at 1.5T, C: PSPs were not observed at 1.2-5T(C-1:10T) and large amplitude EPSPs with long segmental latencies were observed in some motoneurons at 10-25T. See text for details.

Fig. 6 Schematic diagram of neural circuits between the tail and hindlimb motoneurons between L7 and Co2 spinal segement showing predominant action. The open and filled triangles indicate that excitatory and inhibitory synaptic effects were predominant. The hatched triangle indicates that the rate at which EPSPs appeared was similar to that of IPSPs. a, neural pathways of hindlimb flexor reflex, b, neural pathways of the crossed hindlimb extensor reflex, c, neural pathways from hindlimb cutaneous afferents to tail motoneurons. The magnitude of synaptic action is indicated by the size of the triangle. d, neural pathways from tail cutaneous afferents to hindlimb motoneurons. Arrow on the right side indicates the direction of tail flexion by activation of neural pathways from hindlimb cutaneous

afferents to tail motoneurons (d). GS-MN; GS motoneurons, PBSt-MN: PBSt motoneurons, Tail-MN, tail motoneurons.

Fig.1

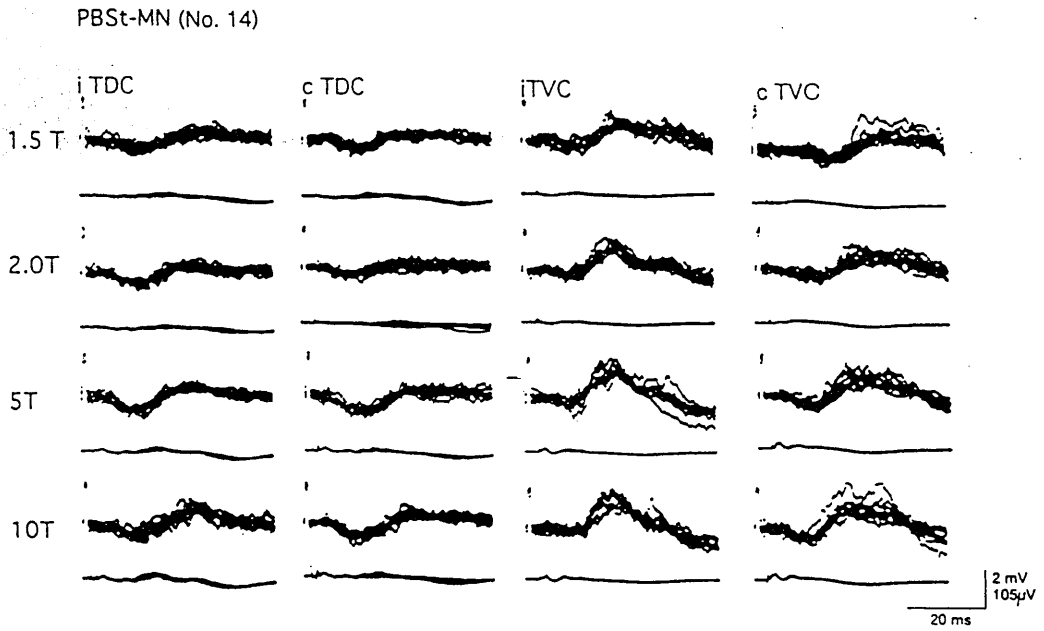
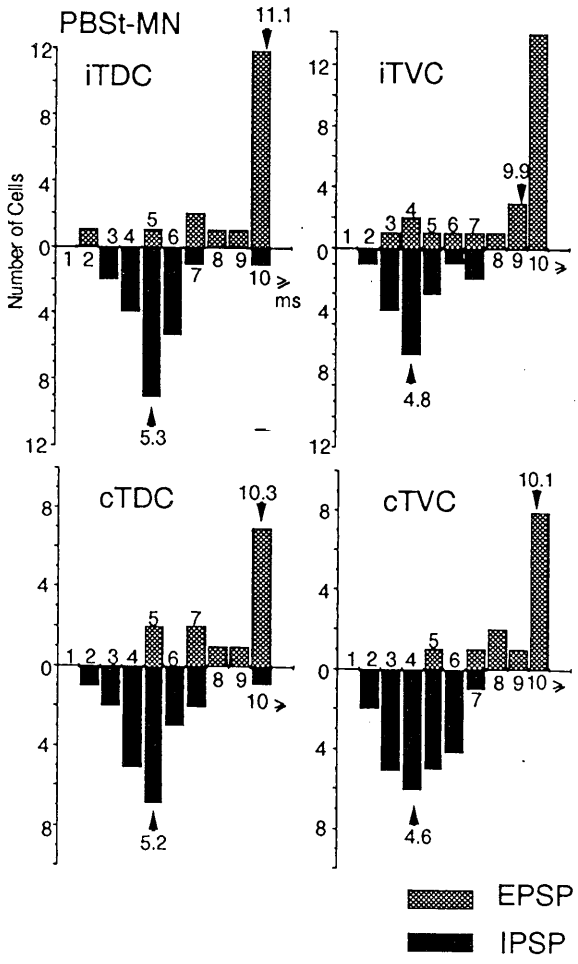


Fig. 2


PBSt-MN
iTDC
iTVC
cTDC
cTVC
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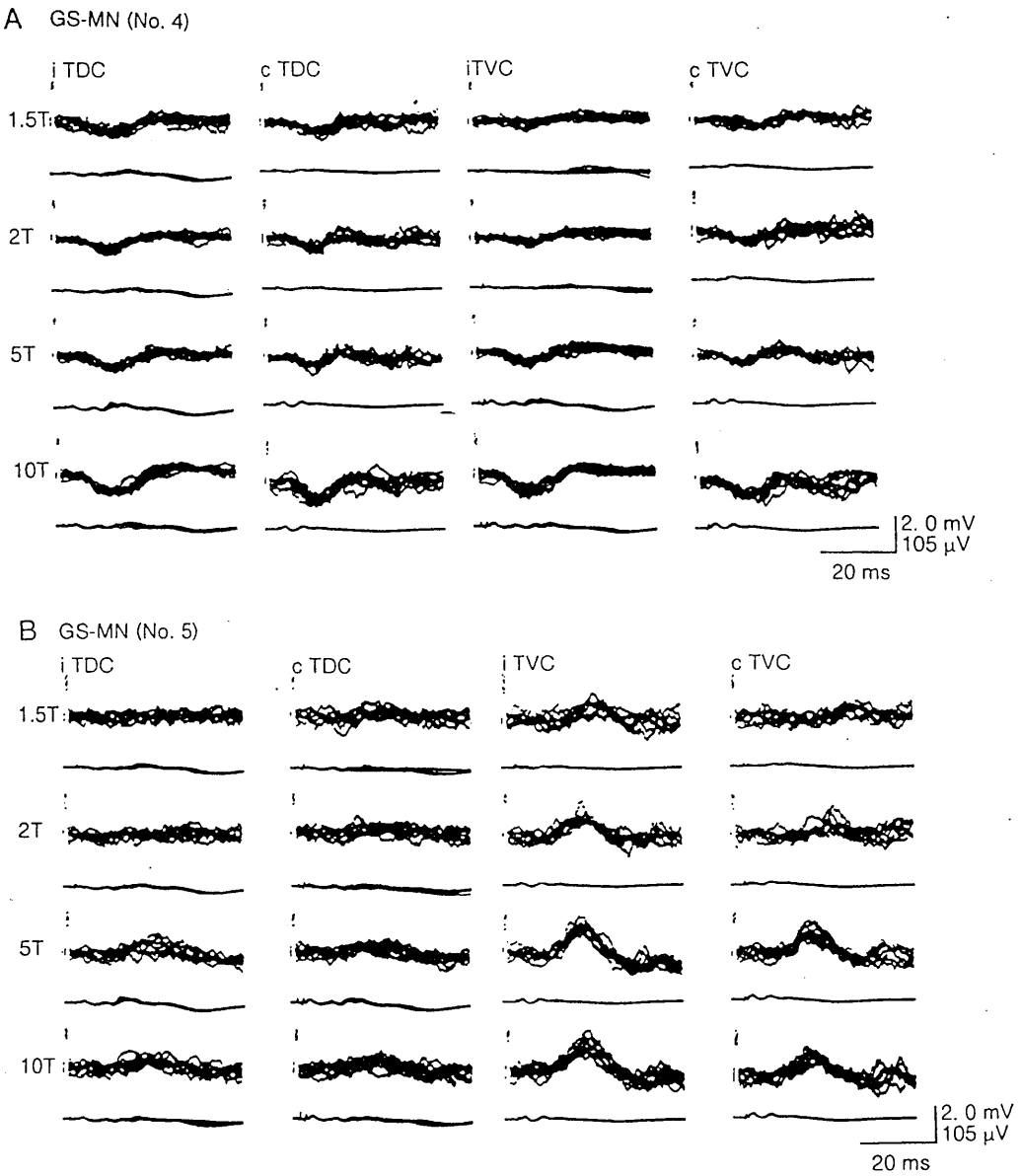
4.8
5.3
10.3
5.2
10.1
4.6
EPSP
IPSP

Fig. 3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c TDC
iTVC
c TVC
1.5T
2T
5T
10T
2. 0 mV
105 μV
20 ms
B GS-MN (No. 5)
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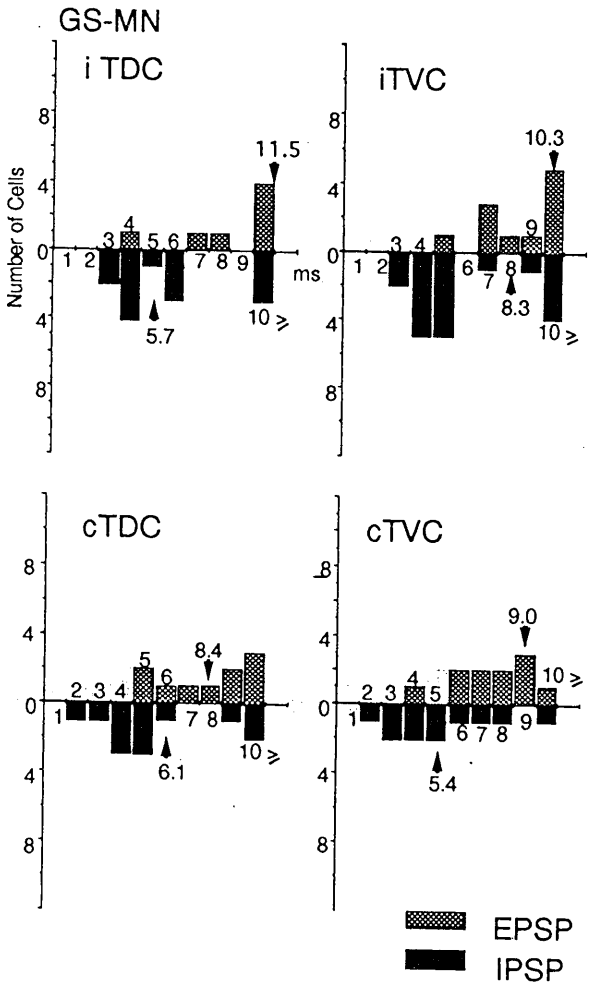
5T
10T
2. 0 mV
105 μV
20 ms

Fig. 4


GS-MN
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iTVC
cTDC
cTVC
Number of Cells
ms
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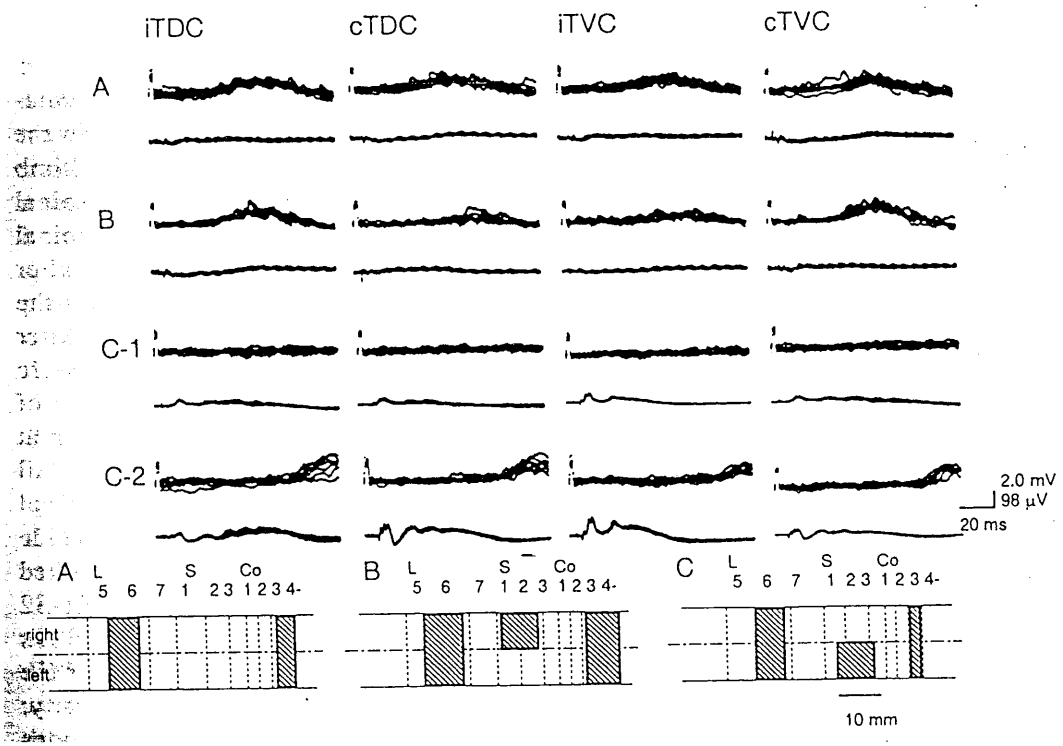
5.7
10.3
8.3
8.4
6.1
9.0
5.4
EPSP
IPSP

Fig. 5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cTDC
iTVC
cTVC
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B
C-1
C-2
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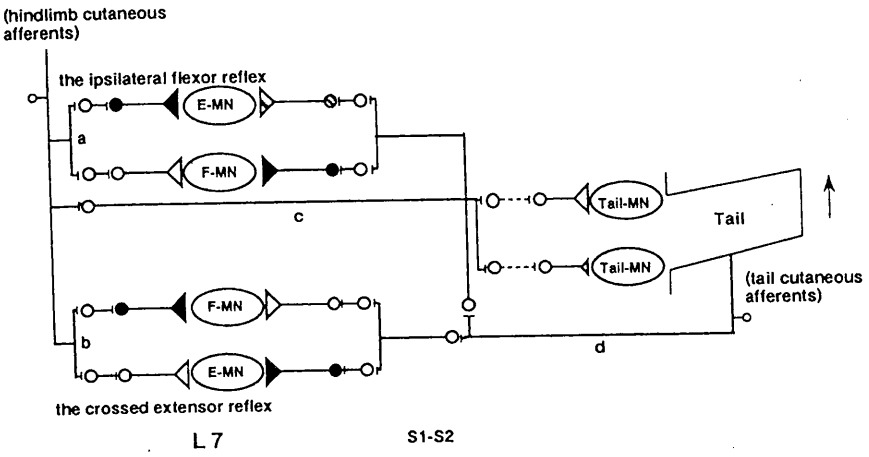
20 ms
A
B
C
L
S
Co
5 6 7 1 2 3 1 2 3 4-
right
left
10 mm

Fig. 6


(hindlimb cutaneous afferents)
the ipsilateral flexor reflex
E-MN
a
F-MN
c
Tail-MN
Tail
Tail-MN
(tail cutaneous afferents)
F-MN
b
E-MN
d
the crossed extensor reflex
L 7
S1-S2

6. RHYTHMIC DISCHARGES RECORDED FROM TAIL MUSCLE NERVES AFTER INJECTION OF NIALAMIDE AND L-DOPA SOLUTION IN SPINALIZED CATS

Summary

In 22 decapitated and high spinalized cats, rhythmic discharges were recorded from the nerves supplying the tail muscles, m. extensor caudae lateralis (ECL) and m. flexor caudae longus (FCL) after intravenous injection of Nialamide and L-DOPA solution. In 15 out of 22 cats, stable rhythmic discharges were recorded from tail muscle nerves. Two different discharge patterns were observed. The predominant pattern consisted of an alternating activation between left and right tail muscle nerves and a synchronous activation of ECL and FCL nerves on one side. The second pattern consisted of synchronous activity involving all four tail muscle nerves.

Introduction

We have been studying the neural control of tail movements in cats (15-20). During rhythmic movements such as locomotion. scratching and pawshake response, cats show rhythmic tail movements (personal observation). Some investigators have demonstrated that locomotion, scratching and paw-shake response are produced by pattern generators in the spinal cord (Deliagina et al. 1975; Grillner, 1981; Grillner and Zannger, 1974; 1979; Harris-Warrick, 1989; Jankowska et al. 1967; Kniffki et al. 1981; Miller and Van der Mechhe, 1975; Sabin and Smith, 1984; Smith et al. 1985). It could be considered that rhythmic tail movements are produced by pattern generators like those for rhythmic movement of the hindlimbs, forelimbs and trunk muscles. However, the existence of a pattern generator to produce rhythmic activities of tail muscles in cats has not yet been demonstrated. In the present study, we recorded the discharge from tail muscle nerves evoked by injection of Nialamide and L-DOPA solution in spinalized cats.

Materials and Methods

The experiments were performed on 22 cats (1 .0-2.5 kg) of either sex, each with a long tail. The animals were anesthetized with halothane-nitrous oxide and nerves to hindlimb muscles, m. posterior biceps and semitendinosus (PBSt) and tail muscles, m. extensor caudae lateralis (ECL) and flexor caudae longus (FCL) on both sides were severed and mounted on bipolar electrodes for recording. The cats were decapitated by the method described by Kniffki et al. (1981) and spinalized at C I . Animals were paralyzed with pancuronium bromide (0.4 mg/h) and artificially ventilated. End-tidal C02 concentration was monitored and maintained at approximately 4% by adjusting respiratory rate or tidal volume. Rectal temperature was maintained close to 37 'C with a heating mat. Arterial blood pressure was maintained at above 80 mmHg. Rhythmic discharge of muscle nerves was evoked by intravenous injection of Nialamide (Sigma, 70 mg/kg) followed by injection of L-DOPA (Sigma, 100 mg/kg). The discharge of muscle nerves was recorded by using differential amplifier (NEC, Sanei, bioelectrical amplifier-125.3) and stored on magnetic tape (band width: d.c.-2.5 kHz, TEAC, RD-135T).

Results

Within about 15-50 min after the injection of L-DOPA, rhythmic activities of PBSt nerves and tail muscle nerves started in 21 and 15 out of 22 cats, respectively. In general, an unstable discharge pattern was observed at the beginning, and a stable discharge pattern developed after about 50 min. Fig. I shows the basic patterns of rhythmic discharge of ECL and FCL' nerves. Fig. 1A shows the alternating discharge pattern between left and right tail muscle nerves, and synchronized discharge of ECL and FCL nerves on one side. Fig. 1B shows the synchronized discharge pattern of all 4 tail muscle nerves. The first and second discharge pattern were observed in 12 and 3 cats, respectively. The rhythmic discharge of muscle nerves was modulated by stimulation of various areas of the cat's body. Fig. 2 shows the effects of mechanical stimulation of cutaneous afferents using forceps on the rhythmic discharges of the PBSt and ECL nerves on both

sides. The rhythmic discharges of the PBSt nerve were enhanced, as shown by decreased intervals or increased amplitudes, by stimulation of cutaneous afferents innervating the forelimb (Fig. 2A), hindlimb (Fig. 2B), dorsal (Fig. 2C) and ventral part of trunk at T 10- L3 (Fig. 2D) of the right side and tail (Fig. 2E). The rhythmic discharges of ECL were enhanced by activation of cutaneous afferents innervating the hindlimb and tail, while stimulation of cutaneous afferents innervating the trunk decreased both the intervals and amplitudes of the discharge. The effects of stimulation of the forelimb on the rhythmic discharge of ECL was not significant.

Discussion

In conclusion, the present experiments confirm the existence of a pattern generator for rhythmic tail movements in the spinal cord. Our findings demonstrate that the rhythmic tail movements are produced from pattern generators in the spinal cord. Furthermore. the results (Fig. 1) show that the pattern generator produces different types of rhythmic discharge of tail muscle nerves. The contraction of ECL and FCL produces dorsolateral and ventrolateral flexion of the tail (Wada et al. 1994). Thus alternating the discharge pattern between' Ieft and right tail muscle nerves (Fig. 1A) might produce rhythmic lateral movements of the tail, while a synchronized pattern (Fig. 1 B) might produce rhythmic stretching of the tail. Rhythmic tail movements were observed during centrally generated rhythmic movements such as locomotion, scratching and paw-shake response and the relationship between hindlimb and tail movements were significant (personal observation). Furthermore, cats often show wave-like tail movements when sitting or maintaining a standing posture (personal observation). The rhythmic discharge of tail muscle nerves evoked by injection of Nialamide and L-DOPA solution often show a close relation to rhythmic discharge of PBSt, as shown in Fig. 2. Thus the rhythmic discharge of tail muscle nerves observed after injection of Nialamide and L-DOPA may be involved in tail movements during locomotion, scratching and paw-shake response. Other investigators have indicated the role of sensory feedback in the regulation of centrally generated rhythmic

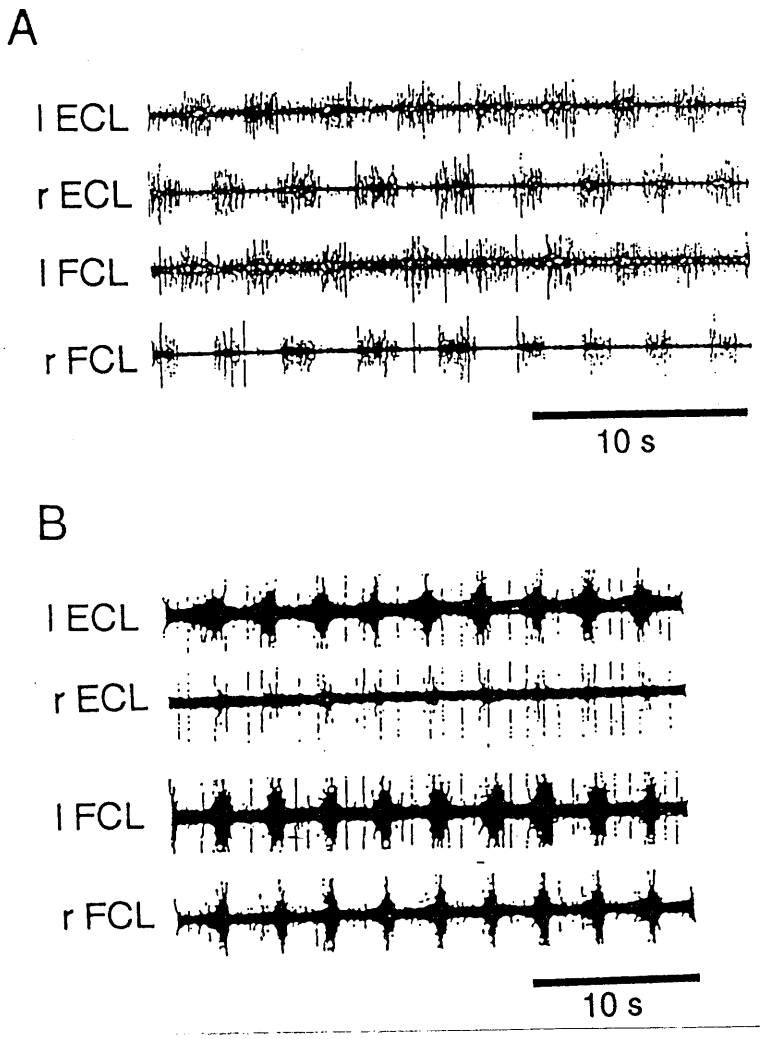
movements, locomotion, respiration and mastication (Rossignol et al. 1989). Fig. 2 demonstrates that rhythmic tail movements are strongly influenced by cutaneous afferent inputs from various areas of the cat's body and indicates that the neuronal pathways from cutaneous afferents to the nervous system enhance the rhythmic discharge of tail muscle nerves. Other investigators have suggested that cats use their tail as a rudder to maintain body balance during locomotion (Kiley-Worthington, 1975; Necker, 1970). It is likely that the neuronal pathways from the cutaneous afferents to the nervous structures responsible for rhythmic tail movements adjust these motor activities to maintain body balance during movement.

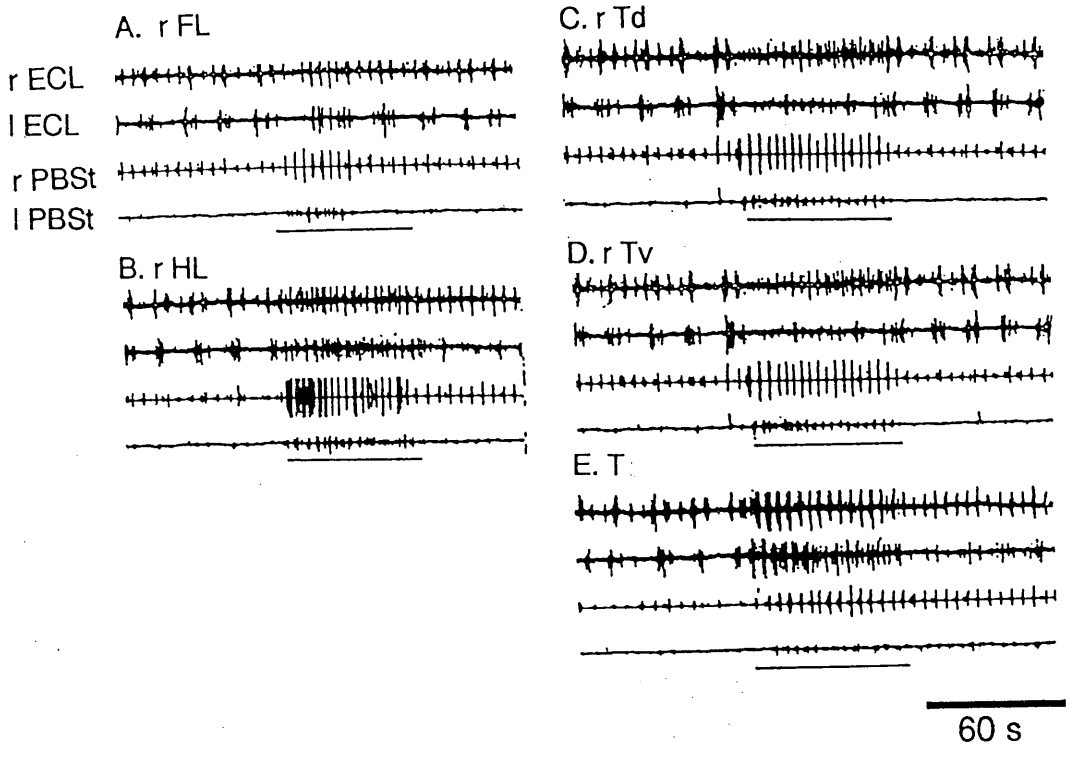
Legends to Figures

Fig. I - Basic patterns of rhythmic discharges of tail muscle nerves evoked by intravenous injection of Nialamide and L-DOPA solution. A: alternating pattern between left and right tail muscle nerves, B: synchronized pattern of all 4 tail muscle nerves.

Fig. 2- Effects of mechanical stimulation of cutaneous afferents innervating forelimb (A, rFL), hindlimb (B, rHL), dorsal (C. rTd) and ventral part of trunk at T13-L3 (D, rTV) of right side and tail (E. T) on rhythmic discharges recorded from ECL and PBSt nerves on both sides.
The line below the recording indicates the stimulus duration. The rhythmic discharge of PBSt nerves on both sides was enhanced by stimulation of peripheral cutaneous afferents. The rhythmic discharge of ECL nerves was influenced by stimulation of HL, T, Td and TV. See text for details.

Fig.1, 2


A
l ECL
r ECL
l FCL
r FCL
10 s
B
l ECL
r ECL
l FCL
r FCL
10 s


A. r FL
r ECL
l ECL
r PBSt
l PBSt
B. r HL
C. r Td
D. r Tv
E. T
60 s

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