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Evidence of rhythmic inhibitory synaptic influences in hindlimb motoneurons during fictive locomotion in the thalamic cat

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Summary. 1. Intracellular recordings of various motoneurons of proximal hindlimb muscles were performed on thalamic paralyzed cats, during fictive locomotion that was either spontaneous or evoked by stimulation of the subthalamic region. 2. In motoneurons innervating sartorius (medialis and lateralis), vasti (intermedius, medialis and lateralis) and anterior biceps-semimembranous, one depolarization occurred in each locomotor cycle, alternating with a phase of repolarization that was synchronous with the activation of the antagonistic muscle nerve. This latter phase could be decreased or reversed by intracellular injection of chloride ions or current, revealing the presence of inhibitory inputs onto motoneurons. 3. The pattern of membrane potential variations was more complex in motoneurons of rectus femoris and posterior biceps-semitendinosus muscles, but phases of chloride dependent inhibition were nevertheless identified, mainly during the sartorius nerve activation in the case of rectus femoris, and during the vasti and anterior biceps-semimembranosus nerve activations in the case of posterior biceps-semitendinosus. These inhibitory influences were shown to be controlled by the level of activity in exteroceptive afferents. 4. The characteristics of the excitatory and inhibitory inputs to the hindlimb motoneurons identified here are discussed in relation with the organization of the central pattern generator for locomotion.

Key words: Locomotion – Cat – Hindlimb motoneurons – Inhibition – Excitation

Introduction

Several authors have recorded periodic oscillating membrane potentials from cat hindlimb motoneurons during fictive locomotion (Edgerton et al. 1976; Jordan et al. 1981; Perret and Cabelguen 1980; Schomburg et al. 1977). It has been shown that, in both flexor and extensor motoneurons, these oscillations resulted from rhythmic alternating excitatory and inhibitory synaptic inputs (Edgerton et al. 1976; Jordan 1983). However, the precise time relations of these synaptic events with the phases of the fictive locomotor cycle remained to be established, especially in the case of motoneurons innervating muscles with a complex pattern of activation during each locomotor cycle (Perret and Cabelguen 1980).

Therefore, the aim of the present study was to further investigate the synaptic inputs to motoneurons innervating various hindlimb muscle groups during fictive locomotion in the thalamic cat. In an attempt to assess the relative contribution of excitatory and inhibitory synaptic inputs to the periodic changes in motoneuron membrane potential, the method of intracellular chloride ion injection (Coombs et al. 1955; Araki et al. 1961; Ito et al. 1962) was employed. The results confirm the existence of excitatory and inhibitory pathways to motoneurons from the central pattern generator network responsible for locomotion. A hypothesis about the organization of these pathways for the different muscle groups of the thigh will be proposed.

A preliminary report of some of the data included in this study has previously been published (Perret 1983).

Methods

The experiments were performed on 23 adult thalamic cats (2.5–3 kg). Under short lasting general methohexital anaesthesia (Brietal 10 mg/kg i.v.), a tracheotomy was performed, a cannula inserted into the left jugular vein and the cat rigidly mounted in a frame that fixed the head and the lumbar spine. After a bilateral craniotomy, a complete ablation of the cerebral cortex and all

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tissue rostral to A 10 was performed by suction. The animal was then paralyzed with gallamine triethiodide (Flaxedil 20 mg/kg per hour i.v.) and artificially ventilated. Thoracic movements due to ventilation were abolished by a bilateral pneumothorax.

The right hindlimb was held semi-flexed in an abducted position. Through a lateral opening, the insertion of rectus femoris on the ilium was cut and the muscle was drawn laterally to expose the branches of the femoral nerve innervating the anterior thigh muscles. The innervation of the hamstring muscles was exposed by reflexion of the antero-lateral border of biceps femoris. The nerves to sartorius (lateral and medial parts), rectus femoris, vasti (intermedius, medialis and lateralis) or anterior biceps-semimembranosus, and posterior biceps-semitendinosus were dissected free and cut. Their central stumps were mounted on silver bipolar electrodes for simultaneous recording or for stimulation. No attempt was made to record separately from the nerve branches supplying a given muscle (such as sartorius) or a given muscle group (such as posterior biceps and semitendinosus) since it was previously shown that they exhibit the same efferent pattern during fictive locomotion in the thalamic cat (Perret and Cabelguen 1980, see Discussion).

After recovery from anaesthesia, spontaneous locomotor-like activities could be recorded from the muscle nerves ("fictive locomotion", cf. Perret 1976). In some experiments, fictive locomotion was induced by electrical stimulation (rectangular pulses of 0.6–0.8 ms duration, 50 Hz, 3–6 mA) of the subthalamic region, using bipolar electrodes positioned on the right or left side at stereotaxic coordinates A9 L2 H3 (see e.g. Dimarco et al. 1983).

Following a laminectomy exposing L5 to S1 spinal segments, the dura was opened and the pia was partially removed in the zones of penetration of the microelectrode. Intracellular d.c. recordings were made using glass micropipettes, filled with 3 mol/l potassium chloride (tip diameter 2 micrometers, impedance 5–10 MΩ), connected to an electronic device allowing simultaneous recording and d.c. current injection through the microelectrode. Impaled cells were identified as alpha motoneurons by the latency of their antidromic responses. Only motoneurons with membrane potentials of at least 60 mV during the whole recording period were accepted for study. A total of 10 to 15 alpha motoneurons of each motor nuclei were successfully recorded before, during and after intracellular injection of chloride ions, which was made by passing a hyperpolarizing d.c. current (10 to 30 nA, occasionally up to 50 nA) through the recording microelectrode during 1 to 5 min.

Paraffin oil pools were formed at the spinal cord and hindlimb levels by drawing up skin flaps. Body and oil temperatures were kept near 38° C by means of a radiant heat lamp.

The nerve activities and the intracellular potentials were displayed on a Siemens Oscillomink paper recorder (linear response: 0–1200 Hz). They were also stored on FM magnetic tape for subsequent analysis.

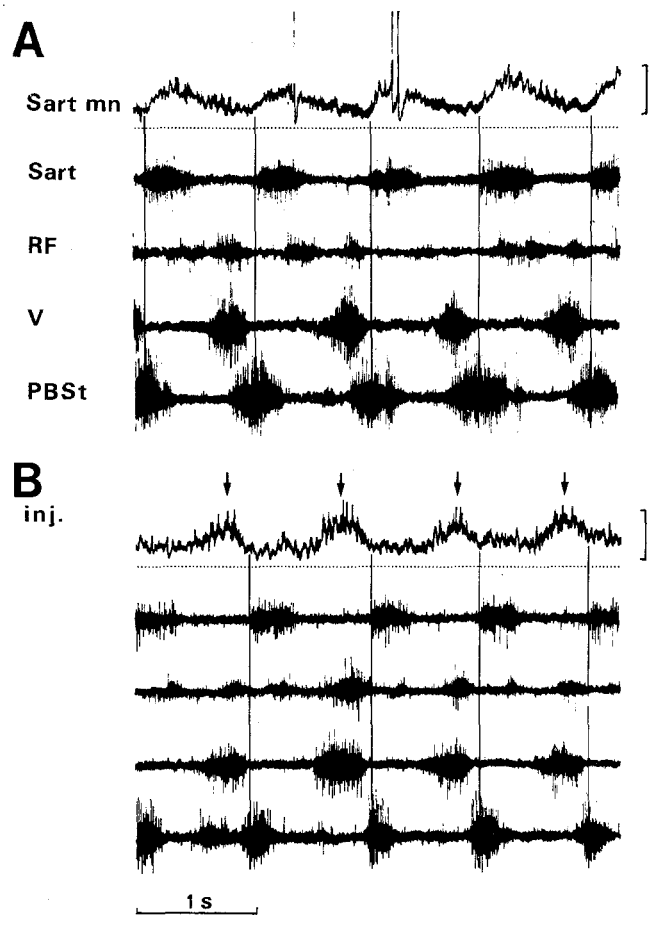

Fig. 1A, B. Effect of intracellular injection of chloride ions on the membrane potential oscillations of a sartorius motoneuron during fictive locomotion. In **A, B** upper trace: intracellular d.c. recording from a sartorius motoneuron (Sart mn); lower traces, electroneurograms of nerves to sartorius (Sart), rectus femoris (RF), vasti (V) and posterior biceps-semitendinosus (PBSt). **A** Control recording before chloride injection. **B** Recording after injection of chloride ions by passing a 30 nA hyperpolarizing current for 3 mn. Note large depolarizations during V nerve bursts (arrowed) and small depolarizations during Sart nerve bursts. Spikes have been truncated and retouched for good reproduction. In this and subsequent figures, vertical calibration (10 mV) is only for intracellular records. Vertical lines have been drawn at onset of Sart nerve bursts in order to facilitate observation of temporal relations. Dotted lines beneath the intracellular recordings have been added in order to facilitate observation of membrane potential variations

Results

Intracellular recordings revealed that, during fictive locomotion, all the impaled motoneurons, without exception, displayed membrane potential oscillations with a locomotory periodicity. Only some of the recorded motoneurons fired action potentials, the others did not, either because they remained subthreshold or because the microelectrode impalement injured the spike generating mechanism.

The time course of the cyclic changes in membrane potential of a given motoneuron closely paralleled that of the activity in the related nerve. The amplitude of these oscillations (range 5 to 15 mV peak to peak) increased with the amplitude of the discharge in the related nerve.

It was impossible to relate such membrane potential variations with excitatory or inhibitory synaptic events mainly because the reference level of the resting potential was unknown. Nevertheless, since postsynaptic inhibition in motoneurons involves

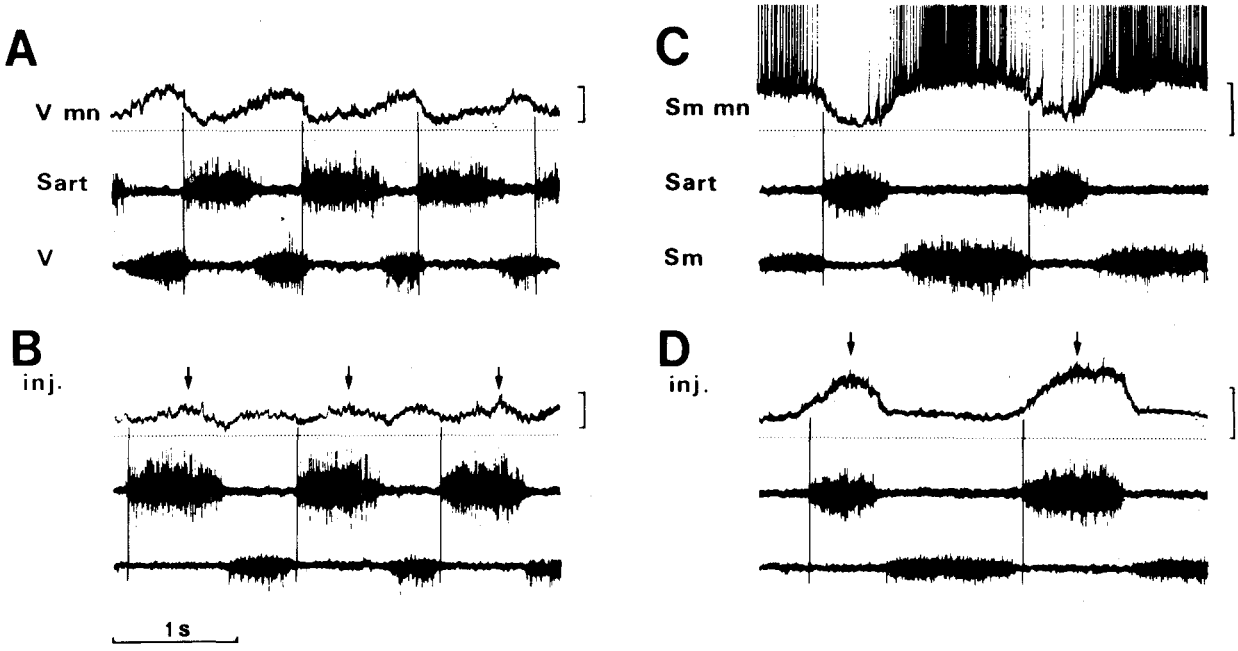

Fig. 2A–D. Effect of intracellular injection of chloride ions or hyperpolarizing current on the membrane potential oscillations of vasti (**A, B**) and anterior biceps-semimembranosus (**C, D**) motoneurons during fictive locomotion. Upper trace: intracellular d.c. recording from a vasti motoneuron (V mn) or from an anterior biceps-semimembranosus motoneurons (Sm mn); lower traces, electroneurograms of nerves to sartorius (Sart) and vasti (V) or anterior biceps-semimembranosus (Sm). **A, C** Control recordings. **B** Recording of V motoneuron after injection of chloride ions by passing a 30 nA hyperpolarizing current for 2 mn. Note depolarizations during the Sart nerve bursts (arrowed) and during the V nerve bursts. **D** Recording of Sm motoneuron 5 s after switching on hyperpolarizing current. Note the simultaneous variations in the Sart nerve burst and in the Sm motoneuron depolarization (arrowed)

chloride ions (Coombs et al. 1955), we have observed the effects of chloride ion injection in motoneurons of the different muscle groups during fictive locomotion. The membrane potential variations will be described in relation to the phases of the locomotor cycle defined by the periods of activity of the muscle nerves selected for study.

During fictive locomotion, motoneurons of the sartorius (Fig. 1A), vasti and anterior biceps-semimembranosus muscles (Fig. 2A, C) displayed one depolarizing wave during each related nerve burst, followed by one repolarizing wave during the subsequent antagonistic nerve burst. Intracellular injection of hyperpolarizing current induced changes mainly in the membrane potential variations occurring during the periods of activity of the antagonistic muscle nerve (Figs. 1B, 2B, D): the synaptic noise increased and the repolarizing wave first became flattened and was then progressively replaced by a depolarizing wave with an opposite time course (arrowed in Figs. 1B, 2B, D). Thus, during injection, the motoneurons could show two successive depolarizing waves within each fictive step cycle (see for example the vasti motoneuron illustrated in Fig. 2B). One was still temporally related to the burst in the corresponding nerve, and usually had a shorter duration, while the other was temporally related to the antagonistic nerve burst, and was typically longer in duration. Changes in the amplitude of this additional depolarizing wave were associated with changes in the amplitude of the antagonistic nerve burst (see anterior biceps-semimembranosus motoneuron in Fig. 2D) and more generally, to changes in the fictive locomotor pattern.

If the injection lasted for long enough, the new depolarization could be so large that the original one was no longer visible. In that case, the variations of the membrane potential were opposite to the variations of activity of the corresponding nerve (Figs. 1B, 2D).

These modifications could persist several minutes after removing the hyperpolarizing current. Then, progressively, the original pattern of membrane potential variations became reestablished. These effects of hyperpolarizing current can mainly be accounted for by an increase of intracellular chloride ion activity. However, a direct effect of current which may shift the value of the membrane potential could not be excluded since, in some cases (Fig. 2D), the use of currents with higher intensity (higher than 30 nA) led to reversals of repolarizing waves which occurred immediately when the current injection was

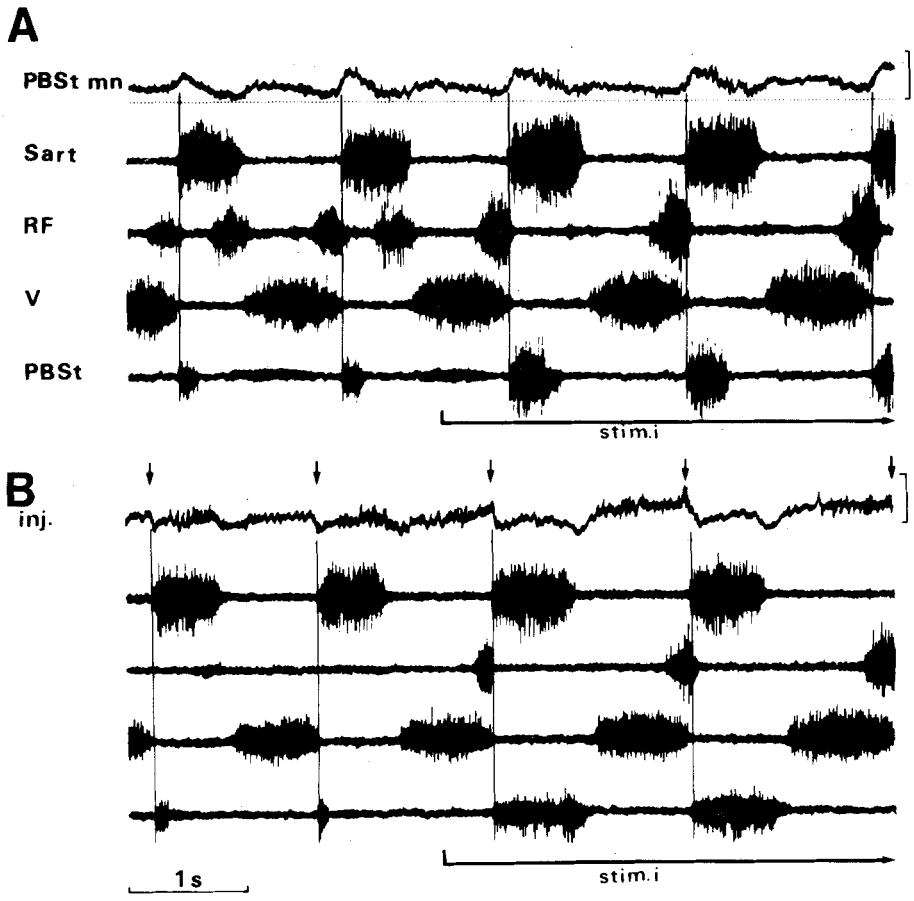

Fig. 3A, B. Effect of intracellular injection of chloride ions on the membrane potential oscillations of a posterior biceps-semitendinosus motoneuron during fictive locomotion evoked by stimulation of the subthalamic region. In **A**, **B**, upper trace: intracellular d.c. recording from a posterior biceps-semitendinosus motoneuron (PBSt mn); lower traces, electroneurograms of nerves to sartorius (Sart), rectus femoris (RF), vasti (V) and posterior biceps-semitendinosus (PBSt). Periods of light tonic pinching applied to ipsilateral toes (stim. i) are indicated by horizontal arrows. **A** Control recording. Note the small repolarizations during the later part of the V nerve bursts. **B** Recording after injection of chloride ions by passing a 30 nA hyperpolarizing current for 2 mn. Note the depolarizations (arrowed) enhanced by the stimulation, during the later part of the V nerve bursts

turned on and disappeared when it was turned off. The time course of these effects suggested that they did not result from the release of chloride ions from the microelectrode.

An example of the membrane potential oscillations of posterior biceps-semitendinosus motoneurons during fictive locomotion is shown in Fig. 3A. Two depolarizing waves could be seen within each fictive locomotor cycle (Fig. 3A: cycles on the left): the first one peaked at the beginning of the sartorius nerve burst and was related to the flexor-like burst of the posterior biceps-semitendinosus nerve: the other one peaked at the beginning of the vasti nerve burst and was related to the extensor-like burst of the posterior biceps-semitendinosus nerve when present. Note that in the experiment illustrated in Fig. 3A, this second depolarizing wave remained subthreshold in most of the posterior biceps-semitendinosus motoneurons as suggested by the lack of a clear corresponding nerve burst. Between the depolarizing waves, the membrane potential reached maximum polarities which were in close temporal relation with bursts of activity in the rectus femoris nerve, occurring respectively during the last part of sartorius and vasti nerve bursts.

Peripheral stimulation such as light tonic pinching of the ipsilateral toes induced an increase of the amplitude and duration of the depolarizing wave during the sartorius nerve burst (Fig. 3A: cycles on the right), associated with an increase of the amplitude and duration of the corresponding peripheral nerve burst.

The clearest effect observed during and after intracellular injection of hyperpolarizing current was the occurrence of an additional depolarizing wave peaking at the end of the vasti nerve burst, (arrowed in Fig. 3B: cycles on the left), instead of the maximum of polarity previously observed at this moment of the locomotor cycle. As described earlier for the other motoneurons, a large synaptic noise was superimposed on the new depolarizing wave, and the reversal effect persisted for a few minutes after removing the hyperpolarizing current. Tonic stimulation of the ipsilateral toes induced an increase of the amplitude of this new depolarizing wave in association with a facilitation of the simultaneous burst of activity in the antagonistic (rectus femoris) nerve (Fig. 3B: cycles on the right). Removal of the stimulation made the additional depolarizing wave recover its control amplitude, meaning that the

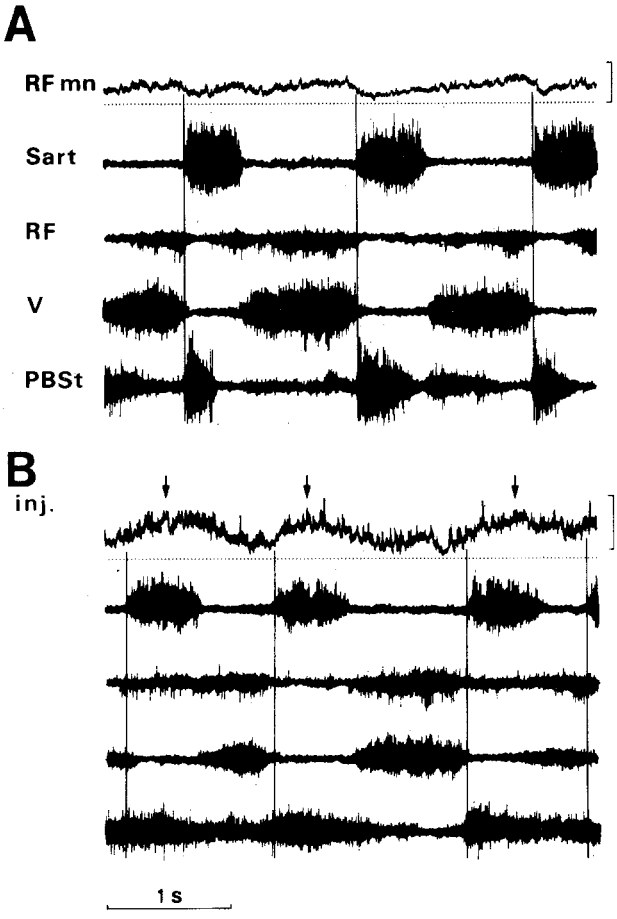

Fig. 4A, B. Effect of intracellular injection of chloride ions on the membrane potential oscillations of a rectus femoris motoneuron during fictive locomotion. In **A**, **B**, upper trace: intracellular d.c. recording from a rectus femoris motoneuron (RF mn); lower traces, electroneurograms of nerves to sartorius (Sart), rectus femoris (RF), vasti (V) and posterior biceps-semitendinosus (PBSt). **A** Control recording. Note the large repolarization in the motoneuron during the first part of the Sart nerve bursts. **B** Recording after injection of chloride ions by passing a 30 nA hyperpolarizing current for 3 mn. Note the depolarization (arrowed) during the first part of the Sart nerve bursts

observed effects cannot be due to additional chloride ions coming out of the microelectrode during the period of peripheral stimulation.

There was also a modification of the membrane potential variations of posterior biceps-semitendinosus motoneurons due to chloride injection during the periods of sartorius nerve activity, where a prolonged depolarizing wave developed (Fig. 3B, cycles on the left). Some experiments suggested that this prolonged depolarizing wave resulted from an overlap of the original flexor-like one with a new depolarizing wave, accompanied by an intense synaptic noise, occurring during the second part of the sartorius nerve burst.

Rectus femoris motoneurons could usually exhibit two depolarizing waves per fictive locomotor cycle: one peaking during the end of sartorius nerve activation and the other during the last part of the vasti nerve burst (first cycle of Fig. 4A). The constant feature of the periodic changes in the membrane potential of rectus femoris motoneurons was the existence of a maximum of membrane polarity during the flexor-like burst of the posterior biceps-semitendinosus nerve.

Intracellular injection of hyperpolarizing current into rectus femoris motoneurons made this repolarizing wave change into a depolarizing one (arrowed in Fig. 4B) more or less fused to the preceding and following original depolarizations and accompanied by increased synaptic noise.

Discussion

The average time courses of the membrane potential oscillations of the hindlimb motoneurons studied here during fictive locomotion are summarized in Fig. 5A. An alternation of depolarization/repolarization occurred once a cycle for flexor (sartorius) and extensor (vasti and anterior biceps-semimembranosus) motoneurons (Fig. 5A, F and E), but twice a cycle for posterior biceps-semitendinosus and rectus femoris motoneurons (Fig. 5A: PBSt and RF). Moreover, in each pair of antagonistic motoneurons (F and E, PBSt and RF), membrane potential oscillations (black fillings) were opposite, in that the membrane depolarization of a given motoneuron corresponded with a repolarizing wave in the antagonist.

These results confirm previous studies (Perret and Cabelguen 1980; Perret 1983). However, Loeb et al. (1985) demonstrated differences between the patterns of e.m.g. activity of sartorius medialis (active in swing phase) and sartorius lateralis (active in swing and stance phases) during unrestrained locomotion in normal cats (see also Engberg and Lundberg 1969). Our present finding that all the impaled sartorius motoneurons displayed a single flexor depolarization during the fictive locomotor cycle (i.e. with no phasic afferent inputs) suggests that the additional e.m.g. burst in sartorius lateralis during the stance phase has a reflex origin, but this remains to be tested in further experiments. On the other hand, differences between discharge patterns of sartorius motoneurons in thalamic and intact preparations cannot be excluded (see however Perret 1983).

Injection of hyperpolarizing current changed the repolarizing waves into additional depolarizing ones

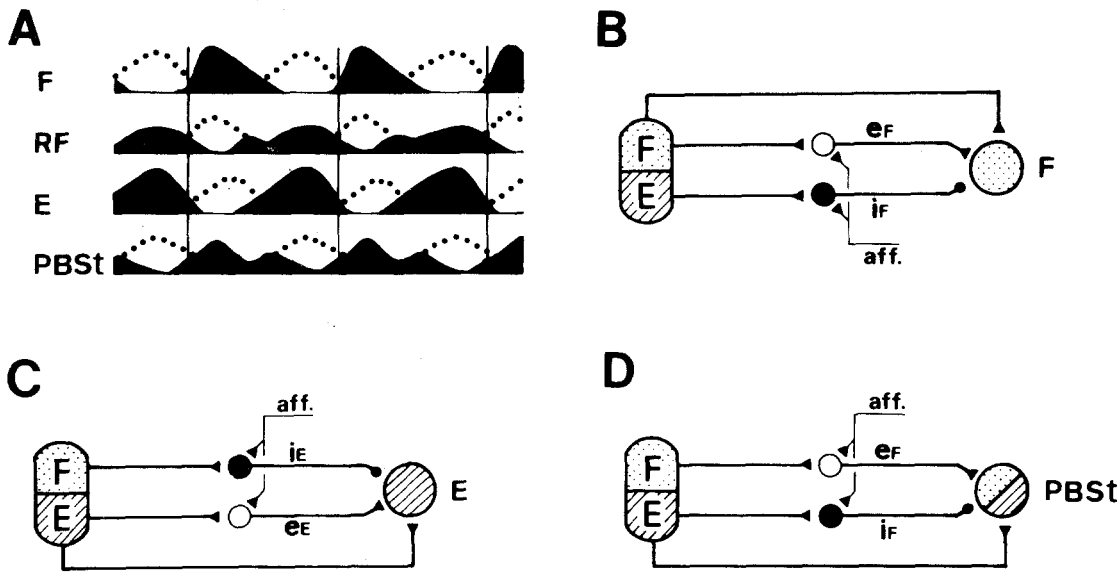

Fig. 5A–D. Interpretation of the effects of current and/or chloride injection in hindlimb motoneurons during fictive locomotion. **A** Schematic representation of the membrane potential oscillations of motoneurons. Four cases are illustrated: flexors (F), rectus femoris (RF), extensors (E) and posterior biceps-semitendinosus (PBSt). Control time courses are drawn in solid black. Dotted lines indicate the modified time course of membrane potential oscillations after chloride injection. **B–D** Speculative wiring diagrams of the origin of synaptic influences exerted upon F, E and PBSt motoneurons, respectively. See text for further explanations. Inhibitory terminals are represented by filled circles, excitatory ones by triangles. Each interneuron (eF, iF, eE and iE) represents a chain. aff.: ipsilateral afferent inputs

(Fig. 5A, dotted lines). These reversed waves corresponded with the original depolarizing waves in antagonistic motoneurons. These effects seem to be a consequence of an increase of intracellular chloride ions activity (Coombs et al. 1955; Araki et al. 1961; Ito et al. 1962) since, in most cases: i) these effects were observed only with KCl microelectrodes; ii) currents up to 30 nA, had no direct effects, but within 2–5 min led to a progressive flattening and reversal of the repolarizing waves (Sears 1964); iii) the observed effects persisted a few minutes after turning off the injecting current. Furthermore, since the additional depolarizing waves had a shape which closely mirrored the original repolarizing waves and were associated with intense synaptic noise, they can be interpreted as being due to the reversal of inhibitory potentials. The use of higher intensities (40–50 nA) sometimes induced a reversal of the repolarizing waves which appeared immediately after turning on the current injection and ceased when it was turned off. Thus, since the microelectrode tip is likely to penetrate the soma in most cases, the immediate effect of currents with high intensity and the delayed effect of current with low intensity suggest that the corresponding inhibitory synapses are located far from the soma (Richter et al. 1975; 1979). Recent work by Shefchyk and Jordan (Shefchyk and Jordan 1984) verifies more directly this interpretation. However, the geometrical characteristics of the microelectrodes should be taken into account in interpreting the efficiency of the injection.

During intracellular chloride injection, repolarizing waves were reversed but the original depolarizing events remained present. The difference of the effects of current injection on the repolarizing and depolarizing waves indicates that the latter correspond to true excitatory influences (Wallen 1982).

Thus the modulation of the membrane potential in motoneurons during fictive locomotion, appears to be a consequence of a succession of excitatory and inhibitory synaptic influences overlapping each other throughout the locomotor cycle.

These results confirm the inhibitory nature of the motoneuron repolarization found in premammillary or mesencephalic cats during fictive locomotion induced by stimulation of the mesencephalic locomotor region (Shefchyk and Jordan 1985) and in spinal cats (Edgerton et al. 1976; Chandler et al. 1984).

A model premotoneuronal network for the generation of the locomotor pattern, including inhibitory influences, can be proposed (see Perret 1983). This model is based on the following assumptions. Firstly, the time course and amplitude of the inhibition of a given motoneuron are closely linked to the time course and the amplitude of excitatory waves displayed simultaneously in antagonistic motoneurons. This indicates that the activation of interneurons

inhibiting a given pool of motoneurons and the activation of antagonistic pools of motoneurons originate from the same part of the central pattern generator for locomotion (CPG). Secondly, since a modulation of efferent responses (Forssberg et al. 1977; Perret and Cabelguen 1980; Duysens et al. 1980; Andersson and Grillner 1981; Forssberg 1981; Rossignol et al. 1981), and motoneuron EPSPs and IPSPs (Andersson et al. 1978; Schomburg et al. 1978; Schomburg et al. 1981) to phasic peripheral stimulation has been demonstrated during the locomotor cycle, it can be postulated that reflex pathways like those activated by flexor reflex afferents, belong to the premotoneuronal part of the CPG. Thirdly, since Ia inhibitory interneurons have been shown to be activated synchronously with locomotor rhythmic activities (Feldman and Orlovsky 1975; McCrea et al. 1980), they are likely to be involved in the inhibitory influences on motoneurons (see however Chandler et al. 1984). Thus, flexor motoneurons receive their excitatory flexor influences not only through a connexion from a central generator (labelled F–E in Fig. 5B), but also through interneurons belonging to segmental reflex pathways (eF in Fig. 5B). The inhibition of flexor motoneurons observed during the extensor phase of the generator activity would be the consequence of the excitation of interneurons belonging to reciprocal inhibitory reflex pathways (iF in Fig. 5B). A similar interpretation can be given for extensor motoneurons (Fig. 5C). In contrast, posterior biceps-semitendinosus motoneurons receive excitatory influences during both flexor and extensor phases (Fig. 5D): the extensor one from the E–F central generator through "private" pathways, and the flexor one through interneurons of excitatory reflex pathways (eF in Fig. 5D) (Perret and Cabelguen 1980). Furthermore, during the extensor phase, a period of inhibition, facilitated by afferent stimulation, and superimposed on an excitation, is developed by the F–E central generator, through activation of inhibitory reflex pathways (iF in Fig. 5D). This variable inhibitory command superimposed on the excitatory one, leads to the depolarization and firing of posterior biceps-semitendinosus motoneurons mainly at the beginning and sometimes at the end of the extensor burst, and is able to explain the variability of the activity of this muscle during this phase. In rectus femoris motoneurons, a symmetrical scheme accounts for the inhibition superimposed on the excitation during the flexor burst, leading to depolarization (and firing) only at the end and sometimes also at the beginning of the flexor burst.

The role of motoneuron inhibition could be to prevent perturbations of the locomotor pattern by direct inputs in phases of the cycle where their activation is inadequate. It appears that they also have a direct role in the elaboration of the final efferent pattern.

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References

Andersson O, Forssberg H, Grillner S, Lindquist M (1978) Phasic gain control of the transmission in cutaneous reflex pathways to motoneurones during "fictive" locomotion. Brain Res 149: 503–507

Andersson O, Grillner S (1981) Peripheral control of the cat's step cycle. I. Phase dependent effects of ramp-movements of the hip during "fictive locomotion". Acta Physiol Scand 113: 89–101

Araki T, Ito M, Oscarsson O (1961) Anion permeability of the synaptic and non-synpatic motoneurone membrane. J Physiol (Lond) 159: 410–435

Chandler SH, Baker LL, Golberg LJ (1984) Characterisation of synaptic potentials in hindlimb extensor motoneurons during L-DOPA-induced fictive locomotion in acute and chronic spinal cats. Brain Res 303: 91–100

Coombs JS, Eccles JC, Fatt P (1955) The specific ionic conductances and the ionic movements across the motoneuronal membrane that produce the inhibitory postsynaptic potential. J Physiol (Lond) 130: 326–373

Dimarco AF, Romaniuk JR, Von Euler C, Yamamoto Y (1983) Immediate changes in ventilation and respiratory pattern associated with onset and cessation of locomotion in the cat. J Physiol (Lond) 343: 1–16

Duysens J, Loeb GE (1980) Modulation of ipsi- and contralateral reflex responses in unrestrained walking cats. J Neurophysiol 44: 1024–1037

Edgerton VR, Grillner S, Sjöström A, Zangger P (1976) Central generation of locomotion in vertebrates. In: Herman RM, Grillner S, Stein P, Stuart DG (eds) Neural control of locomotion. Plenum Press, New York, pp 439–464

Engberg I, Lundberg A (1969) An electromyographic analysis of muscular activity in the hindlimb of the cat during unrestrained locomotion. Acta Physiol Scand 75: 614–630

Feldman AG, Orlovsky GN (1975) Activity of interneurons mediating reciprocal Ia inhibition during locomotion. Brain Res 84: 181–194

Forssberg H (1981) Phasic gating of cutaneous reflexes during locomotion. In: Taylor A, Prochazka A (eds) Muscle receptors and movement. Macmillan, London, pp 403–412

Forssberg H, Grillner S, Rossignol S (1977) Phasic gain control of reflexes from the dorsum of the paw during spinal locomotion. Brain Res 132: 121–139

Ito M, Kostyuk PG, Oshima T (1962) Further study on anion permeability in cat spinal motoneurones. J Physiol (Lond) 164: 150–156

Jordan LM (1983) Factors determining motoneuron rhythmicity during fictive locomotion. Soc Exp Biol Symp 37: 423–444

Jordan LM, Pratt CA, Menzies JE (1981) Intraspinal mechanisms for the control of locomotion. In: Szentagothai J, Palkovits M, Hamori J (eds) Advances in physiological sciences, Vol 1. Pergamon Press/Akademiai Kiado, Budapest, pp 183–186

Loeb GE, Hoffer JA, Pratt CA (1985) Activity of spindle afferents from cat anterior thigh muscles. I. Identification and patterns during normal locomotion. J Neurophysiol 54: 549–564

McCrea DA, Pratt CA, Jordan LM (1980) Renshaw cell activity and recurrent effects on motoneurons during fictive locomotion. J Neurophysiol 44: 475–488

Perret C (1976) Neural control of locomotion in the decorticate cat. In: Herman RM, Grillner S, Stein PSG, Stuart DG (eds) Neural control of locomotion. Plenum Press, New York, pp 178–188

Perret C (1983) Centrally generated pattern of motoneuron activity during locomotion in the cat. In: Roberts A, Roberts B (eds) Neural origin of rhythmic movements. Cambridge University Press, Cambridge, pp 405–422

Perret C, Cabelguen JM (1980) Main characteristics of the hindlimb locomotor cycle in the decorticate cat with special reference to bifunctional muscles. Brain Res 187: 333–352

Richter DW, Heyde F, Gabriel M (1975) Intracellular recordings from different types of medullary respiratory neurons of the cat. J Neurophysiol 38: 1162–1171

Richter DW, Camerer H, Meesmann M, Röhrig N (1979) Studies on the synaptic interconnection between bulbar respiratory neurones of cats. Pflügers Arch 380: 245–257

Rossignol S, Julien C, Gauthier L, Lund JP (1981) State-dependent responses during locomotion. In: Taylor A, Prochazka A (eds) Muscle receptors and movement. Macmillan, London, pp 389–402

Schomburg ED, Roesler J, Meinck HM (1977) Phase dependent transmission in the excitatory propriospinal reflex pathway from forelimb afferents to lumbar motoneurones during fictive locomotion. Neurosci Lett 4: 249–252

Schomburg ED, Behrends HB (1978) Phasic control of the transmission in the excitatory and inhibitory reflex pathways from cutaneous afferents to alpha motoneurones during fictive locomotion in cats. Neurosci Lett 8: 277–282

Schomburg ED, Behrends HB, Steffens H (1981) Changes in segmental and propriospinal reflex pathways during spinal locomotion. In: Taylor A, Prochazka A (eds) Muscle receptors and movement. Macmillan, London, pp 413–425

Sears TA (1964) The slow potentials of thoracic respiratory motoneurones and their relation to breathing. J Physiol (Lond) 175: 404–424

Shefchyk SJ, Jordan LM (1984) Spatial segregation of excitatory and inhibitory synaptic terminals producing locomotor drive potentials in alpha motoneurons. Soc Neurosci Abstr 10: 629

Shefchyk SJ, Jordan LM (1985) Excitatory and inhibitory postsynaptic potentials in α-motoneurons produced during fictive locomotion by stimulation of the mesencephalic locomotor region. J Neurophysiol 53: 1345–1355

Wallen P (1982) Spinal mechanisms controlling locomotion in dogfish and lamprey. Acta Physiol Scand Suppl 503: pp 1–45

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