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Positive Proprioceptive Feedback Elicited By Isometric Contractions of Ankle Flexors on Pretibial Motoneurons in Cats

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Brizzi, L., L. H. Ting, and D. Zytnicki. Positive proprioceptive feedback elicited by isometric contractions of ankle flexors on pretibial motoneurons in cats. *J Neurophysiol* 88: 2207–2214, 2002; 10.1152/jn.01051.2001. Pretibial flexor motoneurons were recorded intracellularly in anesthetized cats during unfused isometric contractions of a subpopulation of motor units from either tibialis anterior (TA) or extensor digitorum longus (EDL) muscles. The contractions elicited excitatory postsynaptic potentials in 23 of 28 pretibial flexor motoneurons. No effect was observed in the remaining motoneurons. In control experiments, the effects of electrical stimulation of afferents within the TA nerve were investigated to help identify afferents responsible for the contraction-induced positive feedback. This feedback was ascribed to actions of Ia fibers because the pattern of the contraction-induced excitatory potentials was consistent with the known pattern of Ia discharge; in control experiments, electrical stimulation of group I fibers elicited only monosynaptic excitatory potentials; and the distribution of both the contraction-induced positive feedback among motor nuclei as well as the electrically evoked Ia excitatory monosynaptic potentials were restricted to homonymous and synergic motoneurons. Observation of the Ia contraction-induced positive feedback was facilitated by the absence of Ib autogenic inhibition. This contraction-induced Ia excitatory feedback in ankle flexors might either reinforce Ia-induced reflexes when these muscles are lengthened or help to lift the leg over an obstacle.

INTRODUCTION

Many data suggest that the net effect of spinal pathways fed by proprioceptive impulses arising from leg muscle sensors during contraction is to help maintain or develop the contraction. In *anesthetized cats*, the sustained isometric contraction of peroneus brevis [which produces foot eversion and abduction with a slight contribution to ankle flexion (Lawrence et al. 1993)] elicited excitatory postsynaptic potentials (EPSPs) in peroneal motoneurons (partly due to input from β-activated spindles) while Ib inhibition was absent (Kouchtir et al. 1995). Peroneus brevis contractions therefore elicit positive feedback on peroneal motoneurons that reinforces the ongoing contraction. In similar experimental conditions, the contraction of triceps surae elicited only transient Ib inhibitory postsynaptic potentials (IPSPs). The quick decrease of this inhibition was found to be due to a central mechanism involving presynaptic inhibition of Ib terminals (Lafleur et al. 1992; Zytnicki et al. 1990). The rapid disappearance of autogenic inhibition allows rapid restoration of motoneuronal excitability and facilitates recruitment of new motoneurons. These results suggested that autogenic inhibition may smooth force development without restricting force amplitude. In *unanesthetized decerebrate cats*, sensory feedback from ankle extensors was found to elicit a substantial amount of force generation in synergist muscles during the stance phase of locomotion (Hiebert and Pearson 1999; Pearson and Collins 1993; Stein et al. 2000). This effect was partly due to the replacement of Ib autogenic inhibition in ankle extensor motoneurons by a disynaptic group I excitation during the extensor phase of the locomotor cycle (McCrea et al. 1995). Altogether, these data show that proprioceptive afferents from muscles can regulate motoneuronal activity by positive feedback. Theoretical work by Prochazka et al. (1997) suggested that such feedbacks would be stabilized by muscle intrinsic properties, length feedback, and delays in operating neuronal pathways. An important function of proprioceptive afferents may thus be to assist movement and weight support by increasing the force of contracting muscles.

In contrast, the classic work of Green and Kellerth (1967) reported that impulses arising from contraction-activated mechanoreceptors of ankle flexors elicited inhibitory potentials in pretibial flexor motoneurons. This was consistent with Laporte and Lloyd (1952), who showed that electrical stimulation of high-threshold group I (presumably Ib) afferents from extensor digitorum longus (EDL) could induce an inhibition of the monosynaptic reflex of tibialis anterior (TA) by a disynaptic pathway. However, Eccles et al. (1957b), using intracellular recording of motoneurons, found that electrical stimulation of group I fibers from pretibial flexors only rarely evoked IPSPs in homonymous and synergistic motoneurons. Generally, Ib inhibition is lacking in flexor motoneurons although it is known that tendon organs in flexor muscles are efficiently activated during contraction (see Jami 1992). In decerebrate cats, Nichols (1989) found that effects of proprioceptive afferents in ankle flexor motoneurons were activity dependent: under quiescent conditions, stretches of TA muscle excited EDL motoneurons (and reciprocally) as judged by the increase of electromyographic activity and force in the corresponding muscle, while mutual inhibition appeared when a background activity was present. Because there is no consensus on whether

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proprioceptive signals arising from ankle flexor contractions elicit negative or positive feedback onto pretibial flexor motoneurons, the present study re-examined this issue. We sought to answer the following questions: in anesthetized cats, what is the feedback elicited by contraction-activated afferents from TA and EDL muscles in pretibial flexor motoneurons and which afferents are responsible for the observed effects?

METHODS

Experiments were carried out on adult cats (2.5–3.5 kg) anesthetized with either sodium pentobarbitone (Sagatal, May and Baker, 8 cats) or α-chloralose (5 cats). The barbiturate anesthesia was induced with an intraperitoneal injection of Sagatal (45 mg/kg) supplemented whenever necessary by intravenous injections (3.6 mg/kg). In α-chloralose experiments, anesthesia was induced by inhalation of 4–5% halothane (Laboratoire Belamont) in air and continued during the surgery by inspiration through a tracheal canula of 1.5–2.5% halothane in a mixture of air (2 l/min) and O_2 (2 l/min). After the laminectomy, gas anesthesia was discontinued and replaced by chloralose anesthesia (initial dose of 50–70 mg/kg intravenous supplemented by additional doses of 15–20 mg/kg whenever necessary). Among the 13 cats, 4 (1 sodium pentobarbitone and 3 α-chloralose) were paralyzed (Flaxedil, 8 mg $\cdot$ kg^{-1} $\cdot$ h^{-1}, Specia) and artificially ventilated (end tidal PCO_2 maintained around 4%). In all cases, criteria for control of the adequacy of anesthesia were: myotic pupils associated with stability of blood pressure (measured in the carotid) and heart rate. Absence of movement in response to ear pinching was also checked in unparalyzed preparations. An antibiotic (Clamoxyl, Merieux, 500 mg) was given subcutaneously to prevent the risk of infection. The central temperature was maintained at 38°C. Blood pressure was maintained above 90 mmHg by infusion, at a rate of 3–12 ml/h, of a 4% glucose solution containing $NaHCO_3$ (1%) and gelatin (14% Plasmagel, Roger Bellon). A catheter allowed evacuation of urine from bladder.

Either the TA (8 experiments) or EDL (3 experiments) muscle was dissected without disturbing its blood supply. The corresponding tendon was detached from its insertion point and attached to a force transducer (Entran, compliance 5 μm per 100 N, i.e., the full range of the transducer) connected to an amplifier. The limb was rigidly fixed, and the muscle length was set near the length for which the twitch force was maximal, i.e., the "optimal" physiological length. The contraction was induced using one of the three following protocols. *1*) When two branches of the muscle nerve were present, the distal portion of one branch was dissected, cut, and mounted on a pair of silver hook stimulating electrodes (anode distal). This method, used in five experiments, produced contractions developing 20–50% of the total muscle force. Its drawback was that it reduced the number of afferents in continuity from the muscle to the spinal cord. *2*) In four experiments, a portion of L_6 or L_7 ventral root was cut as close as possible to the spinal cord, whereas all the hindlimb and hip muscles were denervated, except for the TA or EDL, to restrict contractions to the investigated muscle. The root was then split, and one filament, containing motor axons for the muscle under study, was mounted on stimulating electrodes. The size of the filament varied among preparations: single shock stimulations produced 10–25% of the total muscle twitch force. *3*) In two experiments, contractions were elicited by direct electrical stimulation of the muscle with a pair of stainless steel intramuscular electrodes. In these cases, the cats were paralyzed and only weak stimulations, eliciting less than 5% of the total force, were used to prevent direct electrical stimulation of afferent fibers within the muscle belly. Lack of an afferent volley at the entry of the spinal cord (see following text) was taken as an indication that the electrical stimulation of the muscle did not recruit afferent fibers. In all three protocols, the motor threshold (MT), i.e., the minimal intensity for which a contraction could be detected, was determined and stimulation intensities were expressed in multiple of MT (2–20 MT). Stimulation frequencies were kept between 10 and 40 Hz, that is, within the discharge rate of hindlimb motor units in freely moving cats (Hoffer et al. 1987). Stimulation sequences lasted 0.5–4 s and were separated by intervals of 1–5 s to avoid unwanted fatigue. The only difference among the three protocols is the number of activated muscle fibers and therefore the amplitude of the contractile force. Altogether, tetanic plateaus were in the range of 1.5–13.3 N.

Two control experiments were carried out in paralyzed animals. In these experiments, the TA and EDL muscles were also denervated and afferent fibers were stimulated in TA nerve mounted on a bipolar stimulating electrode. In this case, afferent stimulation was expressed in multiple of threshold (T), i.e., the intensity for which the most excitable group I afferents were recruited.

In all experiments the following nerves were cut, dissected, and mounted on a pair of stimulating electrodes: anterior branch of the biceps and semimembranosus taken together (ABSm), gastrocnemius medialis together with gastrocnemius lateralis and soleus (triceps surae, TS), and the nerve to the pretibial flexor (TA or EDL) that was not made to contract. The pretibial flexor nerve remaining in continuity with its muscle was stimulated with a unipolar electrode (cathode).

The lumbosacral spinal cord segments were exposed by a laminectomy. Conventional glass micropipettes filled with 2 M potassium acetate and 0.6 M potassium chloride were used for intracellular recordings of motoneurons in L_7–L_6 segments (mixing potassium chloride with acetate helped to keep micropipette impedance in the 4-to 6-MΩ range). Presence of chloride ions in the micropipette did not prevent detection of IPSPs in motoneurons: *1*) in the process of identifying motoneurons, muscle nerves were stimulated in turn and both group I TS afferents and group II PBSt afferents were found to elicit IPSPs in ankle flexor motoneurons. *2*) Group II afferents from ankle flexors could induce IPSPs in homonymous (see Fig. 3*A3*) and ABSm (see Fig. 4*B*) motoneurons. Similar electrodes were used in previous studies (Perrier et al. 2000a,b) in which cutaneous afferents frequently elicited IPSPs in peroneal motoneurons. Motoneurons were generally identified by their antidromic response to nerve stimulation, but in the second protocol, those motoneurons that had their axon in the cut ventral rootlet were identified on their pattern of Ia connections (Eccles et al. 1957a). Afferent volleys were recorded by a silver ball electrode on the surface of the spinal cord near the entry of L_7 dorsal root. All the exposed tissues in hindlimb and spinal cord were covered by pools of mineral oil kept at 38°C.

Simultaneous records of DC-coupled motoneuron membrane potential and muscle force were amplified and fed into a Nicolet PRO 20 digital oscilloscope performing on-line averaging of responses. Axograph software (Axon instruments) was subsequently used for off-line analysis.

RESULTS

This report is based on data from 50 motoneurons: 22 EDL, 6 TA, 11 TS, and 11 ABSm, in which effects of proprioceptive inputs arising from TA or EDL during sustained unfused isometric contractions were investigated. In two further experiments, effects of electrical stimulation of afferents from pretibial flexor nerves were studied in 11 EDL, 10 ABSm, and 2 TS motoneurons. All motoneurons had a resting potential between -50 and -80 mV that remained stable during the recording session.

Excitatory potentials induced by unfused isometric contractions of pretibial flexor muscles in EDL or TA motoneurons

The contraction of EDL induced excitatory potentials in homonymous motoneurons. In the example in Fig. 1*A*, 2-mV

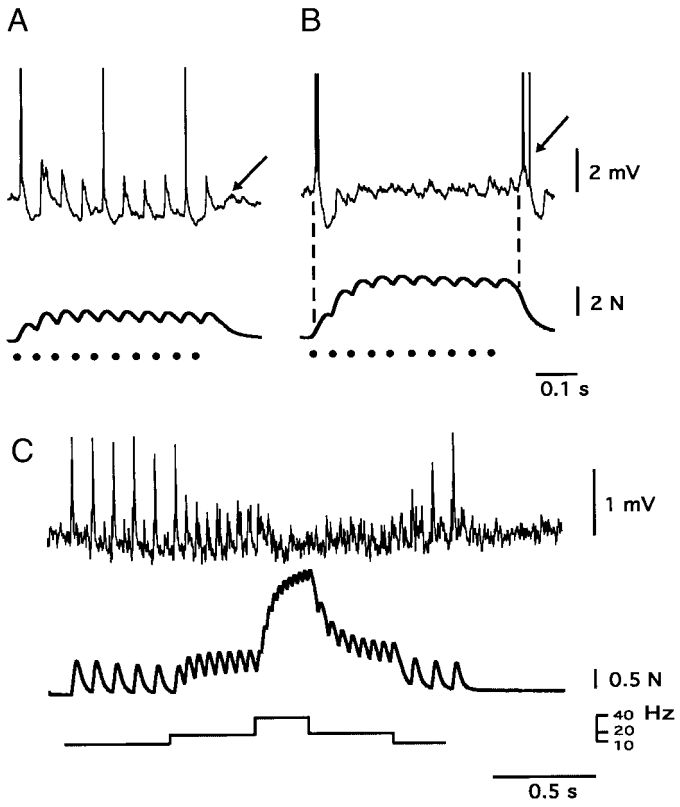

FIG. 1. Effects of unfused isometric extensor digitorum longus (EDL) contractions on 3 homonymous motoneurons. For each panel, the *top trace* is the DC-membrane potential and the *bottom trace* is the contractile force. In *A* and *B*, the dots below contractile force indicate electrical stimulation (20 Hz) of the distal portion the cut nerve branch. In *C*, the line represents the stimulation step pattern (stimulation sequence as follows: 10 Hz during 0.5 s, 20 Hz during 0.5 s, 40 Hz during 0.25 s, 20 Hz during 0.5 s, and 10 Hz during 0.5 s). In all cases, the stimulation strength was 2.5 MT. Each trace is the average of 10 successive records in *A* and 6 in *B* and *C*. In *B*, actions potentials did not appear with the same latency in each of the 6 successive traces. Action potentials with large amplitudes had to be cut to fit in the figure. Arrows point to the responses ascribed to spindle discharges during tetanus relaxation. *A–C* are 3 motoneurons from the same experiment.

excitatory potentials occurred in phase with 0.7-N oscillations of contractile force throughout an unfused tetanus (2.1-N peak amplitude and 500-ms duration). The excitation was sufficient to elicit action potentials. In another EDL motoneuron (Fig. 1*B*), a larger contraction (4 N) induced excitatory potentials reaching the discharge threshold at the onset and during the relaxation of the tetanus as indicated by dashed lines. Only very small excitatory potentials occurred in phase with oscillations throughout contraction. Contraction of pretibial flexors induced excitatory potentials not only in homonymous motoneurons but also in synergistic ones. Effects from EDL and TA were similar, and the results from these two muscles were therefore pooled together. Contraction of either of these two muscles was found to induce excitatory potentials in 23 of the 28 pretibial flexor motoneurons tested. The responses resembled the patterns shown in Fig. 1, *A* and *B*, in 14 and 9 cases, respectively. In the remaining five motoneurons (all from TA, recorded in 2 of the 4 experiments in which the contraction was induced by stimulation of a portion of L_6–L_7 ventral root), no effect was observed, but in these cases, the contraction was quite weak (0.8–1.5 N). Hyperpolarization of membrane potential failed to unmask inhibitory potentials. Observation of excitatory potentials was probably facilitated by this lack of inhibition.

The first component of excitatory potentials occurred early during the rising phase of the tetanus (Fig. 2*A*). Among the 23 motoneurons in which excitation was observed, the excitatory potentials appeared with latencies of 3.5 to 11 ms [7.9 ± 2.0 (SD) ms, Fig. 2*B*] after the onset of contraction. Only a single motoneuron was excited with a latency shorter than 5 ms. These latencies, measured with respect to the *onset* of contraction were too long to have been caused by *ephaptic* stimulation of Ia afferents, i.e., by the electrical field developed within the muscle by activation of numerous muscle fibers (Hunt and Kuffler 1951). Ephaptic stimulation would have occurred several milliseconds *before* any force was detected at the tendon

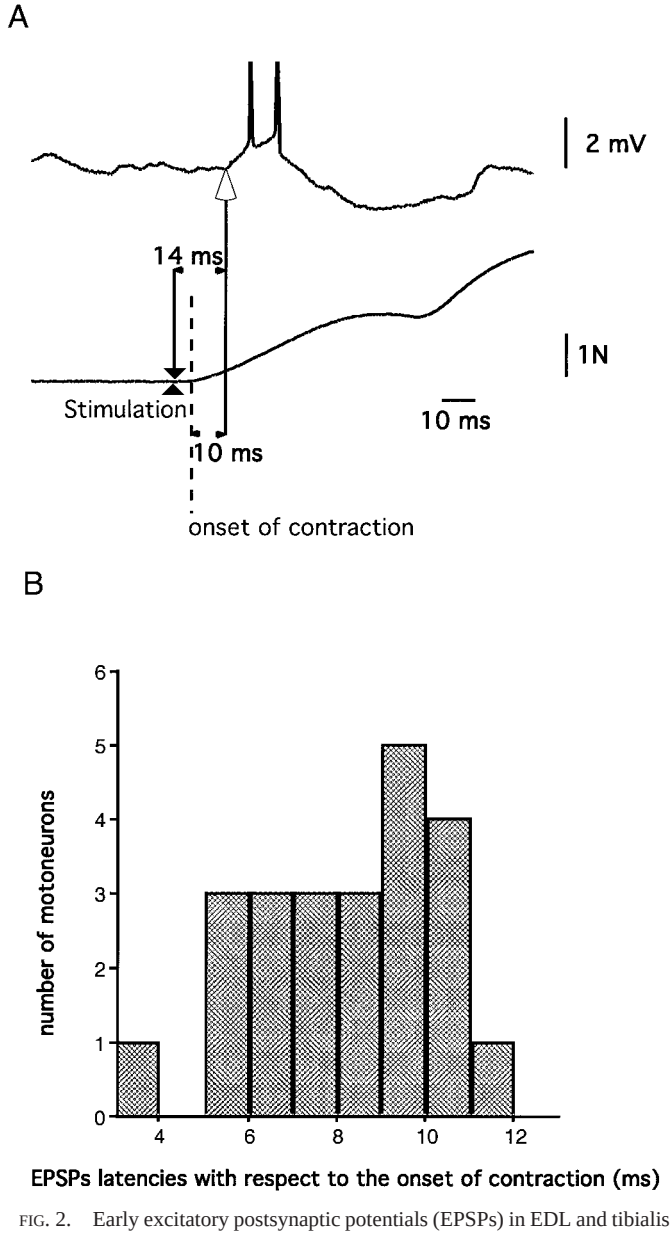

FIG. 2. Early excitatory postsynaptic potentials (EPSPs) in EDL and tibialis anterior (TA) motoneurons. *A*: expansion of the trace shown in Fig. 1*B*. The vertical dashed line is the onset of contraction. The white arrow points to the onset of the EPSP, and the black ones indicate the stimulation. *B*: histogram of EPSP latencies measured with respect to contraction onset for 23 EDL and TA motoneurons.

by the strain gauge and would have induced monosynaptic Ia excitatory potentials within a delay shorter than 4 ms (i.e., activation delay of Ia fibers in the muscle plus conduction time to and within the spinal cord plus synaptic transmission). Consequently, it would have induced excitatory potentials by the onset of contraction or even *before* (see Zytnicki et al. 1995). Observed excitatory potentials were thus ascribed to impulses arising from the mechanical activation of muscle proprioceptors during the contraction.

Ia afferents were responsible for the contraction-induced excitatory potentials

Contraction-induced excitatory potentials in ankle flexor motoneurons were caused by the mechanical activation of *spindle primary endings.* Several criteria were used to establish this relationship.

First, it is well-known that all spindle primary ending discharges do not pause during isometric contraction. Some spindles produce a spike train during the rising phase of either twitches (Hunt and Kuffler 1951; Kuffler et al. 1951; Matthews 1933) or oscillations throughout unfused tetanus (Jami et al. 1985). Such discharges can depend not only on activation of intrafusal fibers by fusimotor (γ) and/or skeletofusimotor (β) axons but also on *passive* mechanical "shaking" of spindles during contraction of extrafusal fibers (see DISCUSSION). In our experiments, motoneuron EPSPs that appeared during a step pattern stimulation of motor units (see an example in Fig. 1*C*) might be related to a passive activation of primary endings. During the 10-Hz stimulation, producing 0.9-N twitches (i.e., appreciably shaking a few spindles), EPSPs of 1.5-mV amplitude were recorded. Smaller EPSPs (0.6 mV) were elicited during unfused tetanus at 20 Hz with force oscillations of 0.5 N only that would produce a smaller passive activation of primary endings. During the 40-Hz step, when the tetanus was almost fused and unlikely to significantly shake the passive spindles, EPSPs almost disappeared. These contraction-induced EPSPs could not be ascribed to Ib input because, in similar contractions, the tendon organ ensemble discharge was found to be larger during the stimulation at 40 Hz than during a series of twitches at 10 Hz (Horcholle-Bossavit et al. 1990).

A second argument in favor of Ia actions on motoneurons is given by the fact that repetitive electrical stimulation of group I ankle flexor afferent induced *monosynaptic* excitatory potentials in EDL motoneurons as illustrated in Fig. 3*A1*. Trains of three shocks spaced by 5 ms and repeated every 50 ms were used to mimic the repetitive activation of mechanoreceptors during unfused contraction (see Zytnicki et al. 1990). In all cases (11 motoneurons), the amplitude of excitatory potentials grew when stimulation strength was increased within the group I range. Central latencies (measured from the onset of the earliest afferent volley entering the cord dorsum) of the first component of these excitatory potentials were in the 0.4- to 1-ms range (Fig. 3*B*), i.e., compatible with a monosynaptic linkage indicating that they were due to Ia fiber action. In agreement with the data of Eccles et al. (1957b), we did not observe group I inhibitory effects. In most cases, group I excitatory potentials reached the discharge threshold when stimulation strength was increased. However, in a few cases, as the one illustrated in Fig. 3*A2*, EPSPs did not reach the threshold, allowing us to observe the effects of group II fibers. At stimulation strengths above 2T, large inhibitory potentials superimposed on group I excitatory potentials but with longer apparent latencies (see Fig. 3*A2*).

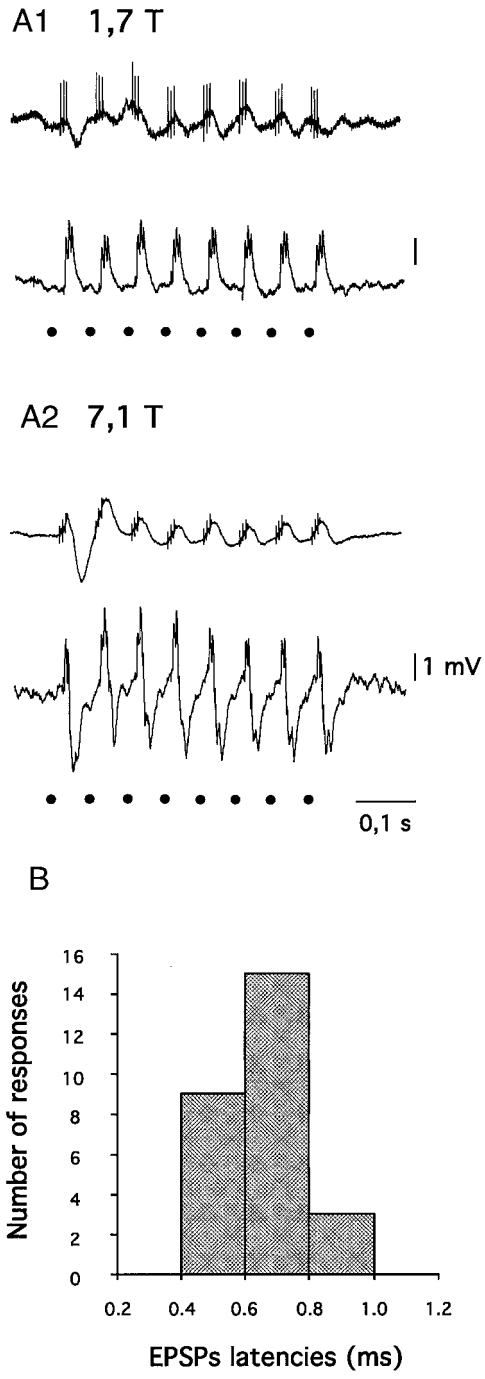

FIG. 3. Responses of EDL motoneuron to the repetitive electrical stimulation of TA afferent fibers. *A*: for each panel, the *top trace* is the cord dorsum potential and the *bottom trace* is DC-membrane potential. Each dot represents a train of 3 stimulation shocks spaced by 5 ms. Each train was repeated every 50 ms. The stimulation intensity is expressed in multiples of T. Note that only EPSPs were visible for stimulation intensity within the group I range (*A1*), whereas inhibitory postsynaptic potentials (IPSPs) appeared at longer latencies when group II fibers were recruited (*A2*). Each trace is the average of 5 succesive responses. *B*: histogram of group I EPSP latencies measured in 11 motoneurons. For each motoneuron, 1–4 responses were recorded at different stimulation intensities in the group I range (the maximal variation of latency was 0.4 ms for a given motoneuron).

Third, contraction-induced excitatory potentials were restricted to *homonymous* and *synergic* motoneurons. Excitatory potentials were not observed in antagonist motoneurons (TS). Ankle flexor contractions induced very small hyperpolarizations in six TS motoneurons and no effect at all in the remaining five. Contraction-induced excitatory potentials were not observed in ABSm motoneurons (innervating hip extensors), suggesting that they would not be observed in motoneurons innervating muscles acting on joints other than ankle. The restricted distribution to homonymous and synergic motor nuclei of the contraction-induced excitatory potentials is parallel to the distribution of monosynaptic connections from Ia ankle flexor afferents (Eccles et al. 1957a). Transient contraction-induced hyperpolarizations were observed in 7 of 11 ABSm motoneurons as illustrated in Fig. 4*A*. In the remaining four motoneurons, no effect was observed. These hyperpolarizations were likely to be due to inhibitory actions of group II afferents as suggested by Fig. 4*B*. Inhibitory potentials appeared in these motoneurons for stimulation intensities above 2T and increased when recruiting further group II afferents (amplitude of the initial inhibitory potential 2 mV at 2.8T and 4.4 mV at 6.4T). In addition, central latencies were in the 4- to 5.5-ms range, indicating that inhibitory potentials might be due to intraspinal oligosynaptic pathways or/and possibly to inputs from the slowest group II afferents running into shorter pathways.

We were not able to identify in motoneurons any clear effect that we could ascribe to activation of spindles by γ motor axons. Such effects were systematically searched for by increasing the stimulus intensity $\leq$15 MT (in experiments in which the contraction was induced either by stimulation of a cut nerve branch or by stimulation of a portion of L_7 ventral root) to recruit γ motor axons. This negative result was likely due to the fact that the low frequency (10–40 Hz) at which γ motor axons were stimulated only induced a weak activation of primary endings (Bessou et al. 1968).

DISCUSSION

Our results show that contraction of flexor ankle muscles induces a positive feedback in homonymous and synergic motoneurons. This is in sharp contrast with autogenic inhibition induced in ankle extensor motoneurons by triceps surae contraction (Zytnicki et al. 1990). The contraction-induced excitatory potentials were sometimes large enough to reach the discharge threshold. These effects could be ascribed to Ia action for three reasons: timing of excitatory potentials was consistent with the pattern of Ia discharge of passive spindles

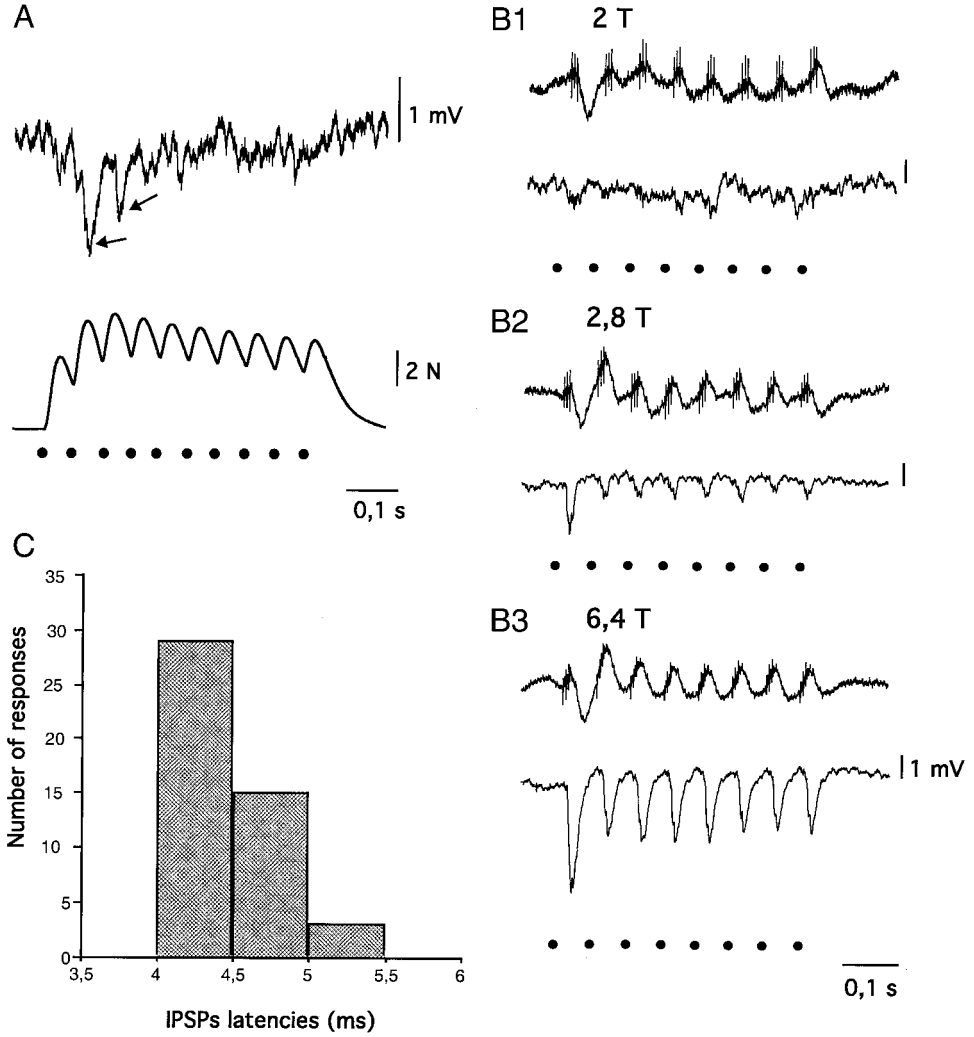

FIG. 4. Effects of inputs from ankle flexors on hip extensor motoneurons (ABSm). *A*: effects of TA contraction on an ABSm motoneuron. Same arrangement as in Fig. 1. The stimulation strength was 4 motor threshold (MT) and 6 successive responses were averaged. Arrows point to transient hyperpolarizations (resting membrane potential, −51 mV). *B*: effects of the electrical repetitive stimulation of TA afferents on an ABSm motoneuron. Same arrangement as in Fig. 3*A*. Each trace is the average of 5 successive responses. Stimulation below 2T did not evoke any effect in the motoneuron (not illustrated). *C*: histogram of group II IPSP latencies measured in 10 ABSm motoneurons. For each motoneuron, 2–7 responses were recorded at different stimulation intensities in the group II range (the maximal variation of latency was 1 ms for a given motoneuron).

(see following text), electrical stimulation of group I fibers only elicited monosynaptic excitatory potentials, and the distribution of positive contraction feedback as well as that of Ia excitatory monosynaptic potentials were restricted to homonymous and synergic motoneurons. In addition, Ia effects were not counteracted by Ib autogenic inhibition, which was lacking for these motor nuclei. Only group II fibers were found to elicit inhibitory actions.

These results are in disagreement with those of Green and Kellerth (1967), who observed that twitches of ankle flexors induced inhibitory potentials in homonymous motoneurons. Although they ascribed this negative feedback to the effects of afferents from tendon organs, inhibitory potentials were still observed when the muscle was slackened such that almost no force was recorded at the tendon. In contrast, when the muscle was extended, excitatory potentials were observed at the onset and the relaxation of contraction (see their Figs. 1 and 5). At physiological lengths, excitatory potentials increased and inhibitory potentials were hardly visible (see their Fig. 4); this is consistent with the present findings. In our experiments, the muscle was set to its optimal physiological length. Shortening of 2–3 mm did not significantly change the feedback pattern to motoneurons. Further shortening, well beyond the physiological range, was not attempted because of the risk of damaging the blood circulation within the muscle.

In the present study, observation of excitatory potentials was facilitated by the lack of contraction-induced Ib inhibition. In addition, repetitive electrical stimulation of ankle flexor group I fibers did not elicit, in our experimental conditions, any inhibitory potentials in homonymous and synergic motoneurons (in agreement with classic data from Eccles et al. 1957a) (but see following text). It is therefore unlikely that the contraction-induced inhibitory potentials observed by Green and Kellerth (1967), in similar experimental conditions, were due to the action of Ib fibers. The present work (see Fig. 3) rather suggests that actions of group II fibers might account for their observations. Inhibitory action of group II fibers onto flexor motoneurons is not in keeping with the classical FRA pattern (Eccles and Lundberg 1959), but the presence of alternative excitatory group II pathways was extensively demonstrated by Lundberg et al. (1987). In addition, it was shown that electrical stimulation of group II afferents from TA induces a resetting from flexion to extension during fictive locomotion in the cat (Perreault et al. 1995). A plausible scenario accounting for the Green and Kellerth results (1967) could be as follows: if spindle secondary endings elicited inhibitory potentials in pretibial flexor motoneurons, it is possible that their tonic discharge at physiological lengths held the resting membrane potential of motoneurons near to the inversion potential of inhibitory synapses. When the muscle was slackened, the tonic discharge of secondary endings stopped, the membrane potential of motoneurons was depolarized and the contraction-induced activation of secondaries could induce transient inhibitory potentials.

Why do ankle flexor motoneurons lack Ib inhibition even though their muscles have a "normal complement" of tendon organs? EDL muscle was found to contain about 10 tendon organs (J.J.A. Scott, personal communication), and although their exact number is unknown for TA, numerous tendon organs were regularly found in this muscle and efficiently activated during contractions (Jami and Petit 1976). However, the lack of Ib autogenic inhibition does not exclude that this pathway is present but not activated under our experimental conditions for different causes such as: contraction-induced presynaptic inhibition of Ib terminals, which was demonstrated to operate on ankle extensor Ib fibers (Devanandan et al. 1966; Lafleur et al. 1992; Lamotte d'Incamps et al. 1998), or low excitability of inhibitory interneurons projecting to flexor motoneurons. Whatever the case, the apparent lack of Ib autogenic inhibition in flexors motoneurons contrasts with observations made in extensor motoneurons under similar experimental conditions (Zytnicki et al. 1990). Recent works (McCrea et al. 1995; Quevedo et al. 2000) showed that pathways operating on motoneurons are very flexible and depend on experimental conditions. In decerebrate unanaesthetized cats, Ib inhibition of ankle extensor motoneurons is suppressed in the flexor phase of fictive locomotion and replaced by disynaptic excitation during the extension phase (McCrea et al. 1995). In flexor motoneurons, a disynaptic excitation, present during both phases, is larger during flexion than during extension (Quevedo et al. 2000). Ib afferents might contribute to these disynaptic excitations.

The positive feedback onto pretibial flexor motoneurons elicited by contraction-activated ankle flexor Ia afferents found in the present work is similar to the one previously observed in peroneal nuclei (Kouchtir et al. 1995). However, in peroneal nuclei, contraction-induced excitatory potentials had a tendency to partially fuse and summate, whereas in the present work, excitatory potentials did not fuse during contraction. Three main reasons might account for these differences. First, spindles are less numerous in ankle flexor than in peroneal muscles. EDL muscle has a complement of 18 spindles (J.J.A. Scott, personal communication), whereas peroneus brevis has about 40 spindles (Scott and Young 1987). Second, in peroneal motor nuclei, the positive feedback was partly due to the activation of intrafusal fibers by the skeleto-fusimotor axons (β innervation) (Kouchtir et al. 1995). More than 30% of motor units are innervated by β axons in peroneus brevis and tertius (Emonet-Dénand et al. 1992; Jami et al. 1982). The incidence of β innervation has never been quantitatively assessed in ankle flexors (but see Emonet-Dénand et al. 1975).

Third, in flexor muscles, activation of primary endings might be due to *passive mechanical activation* of spindles rather than *contraction* of their intrafusal fibers. Such passive activation was first demonstrated by Hunt and Kuffler (1951) who suggested several non-mutually exclusive mechanisms. *1*) Frictional forces that arise from unequal rates of contraction of surrounding extrafusal muscle fibers, which is consistent with the fact that in TA and EDL muscles, slow and fast contracting fibers are largely intermingled (see for instance Bodine et al. 1988). Frictional forces on spindles could also occur when some surrounding extrafusal muscle fibers are active while others are passive as in our experimental conditions where only a fraction of the motor units was stimulated. *2*) Compression forces that are exerted on the spindle capsule during contraction of extrafusal muscle fiber. *3*) Some spindles, by the peculiarity of their insertion on intramuscular connective tissue, may lie "in series" with some muscle fibers so that they are pulled on during contraction of these fibers. Whatever the case, primary endings are sensitive to the dynamic components of

the strain induced on spindles by the contraction of extrafusal fibers. Passive mechanical activation of spindles by the extrafusal portion of motor units during unfused contraction was frequently observed in the course of experiments in which the effects of skeletofusimotor axons (β) were analyzed (see Jami et al. 1985). In their physiological range of activation (10–40 Hz) (see Hoffer et al. 1987), motor units are either twitching or developing unfused contractions with significant force oscillations, so that contraction-induced passive activation of spindles is likely to be a quite common phenomenon even when motor units are contracting asynchronously. However, passive contraction-induced activation of spindles is less likely to occur when the muscle is shortened (Hunt and Kuffler 1951).

In awake behaving cats, tibialis anterior was found to be activated during flexion of the limb away from the ground for obstacle avoidance (McFayden et al. 1999), paw lift elicited by a cutaneous tap (Abraham and Loeb 1985), or stumbling corrective reactions (Forssberg 1979). The positive contraction-induced feedback observed in the present work might become critical in two cases. First, when an obstacle induces a lengthening of flexors, Ia-induced stretch reflexes, which help to counteract this lengthening, would be reinforced by the contraction-induced positive feedback. Second, when the limb is held back, producing a situation where flexor muscles are contracting without shortening, the positive feedback would cause the muscle activity to increase, producing a greater contraction to overcome the obstacle. Then during the flexion movement, when flexors are shortening, the positive feedback would decrease. Such reinforcing effects of flexor afferent feedback on the activity of a hip flexor muscle have been demonstrated in a decerebrate preparation (Lam and Pearson 2001) in which the limb was impeded during the flexion phase of locomotion causing increased flexor activity. Furthermore, static fusimotor actions were observed to be much greater in flexors than extensors in a decorticate preparation (Cabelguen 1981), thereby increasing the contraction-induced positive feedback.

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