Journal of Physiology (1992), **445**, *pp.* 345–354
With 4 figures
Printed in Great Britain

DEPOLARIZATION OF Ib AFFERENT AXONS IN THE CAT SPINAL CORD DURING HOMONYMOUS MUSCLE CONTRACTION

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(*Received 8 May 1991*)

SUMMARY

1. Intra-axonal records from the intraspinal course of Ib and Ia afferent fibres innervating the gastrocnemius medialis muscle were obtained in chloralose or Nembutal-anaesthetized cats during submaximal contractions of the muscle.

2. Afferent fibres in continuity with their muscle of origin were functionally identified by their responses to muscle stretch or contraction.

3. In six out of eight Ib afferents, primary depolarizations (PADs) were recorded during contraction. They were independent of the presence of orthodromic impulses fired by tendon organs.

4. These observations support the assumption that the reduction of Ib autogenetic inhibition in homonymous and synergic motoneurones during GM contractions is due to presynaptic inhibition of transmission in Ib pathways.

INTRODUCTION

In a previous study, we observed the effects of sustained contractions of an ankle extensor muscle, gastrocnemius medialis (GM), on homonymous and synergic α-motoneurones (Horcholle-Bossavit, Jami, Lafleur, Lamy & Zytnicki, 1988; Zytnicki, Lafleur, Horcholle-Bossavit, Lamy & Jami, 1990*b*). Inhibitory postsynaptic potentials (IPSPs) appeared at the onset of a series of GM twitches, or of an unfused tetanus, and were ascribed to Ib autogenetic inhibition evoked by input arising from Golgi tendon organs during contraction (Laporte & Lloyd, 1952; Eccles, Eccles & Lundberg, 1957; Granit, Kellerth & Szumski, 1966). But an unexpected finding was that the amplitude of contraction-induced inhibitory potentials rapidly subsided even though the contraction maintained a constant force level. This observation raised the question of the mechanism responsible for the decline in Ib inhibition of homonymous and synergic motoneurones. As it was verified that GM tendon organ discharges persist throughout the contraction (Zytnicki *et al.* 1990*b*), the decline was likely to depend on a central mechanism.

One possible assumption was that presynaptic inhibition of Ib terminals occurred in the spinal cord during sustained contractions, resulting in a decrease of synaptic

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transmission between these terminals and first-order interneurones. In support of this assumption, it is well known that Ib impulses from extensor muscles elicit a depolarization of Ib terminals from various muscles. This primary afferent depolarization (PAD) is the electrophysiological correlate of presynaptic inhibition

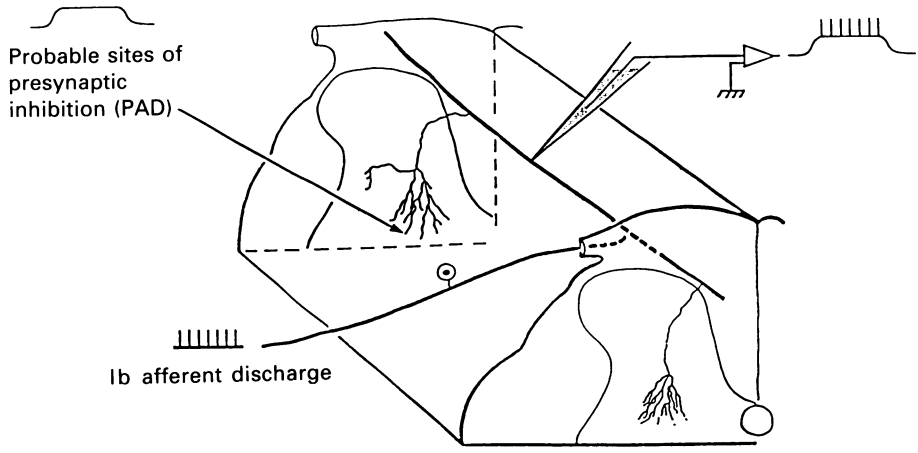

Fig. 1. Diagrammatic three-dimensional representation of a dorsal quadrant of the spinal cord with the trajectory of a Ib fibre (adapted from Brown, 1981) to show the site of microelectrode impalements of afferent fibres in dorsal column. In the diagrammatic records two signals are superimposed: the contraction-induced PAD and the discharge of tendon organ impulses.

(see Eccles, Schmidt & Willis, 1963*a*). Devanandan, Eccles & Stenhouse (1966) observed an increased excitability, indicating PAD, of Ia and Ib afferent terminals from ankle extensors on contraction of heteronymous muscles. They considered that the discharges from contraction-activated tendon organs were responsible for this effect. However, evidence that afferent impulses evoked by muscle contraction produce presynaptic inhibition of *homonymous* Ib terminals had never been provided.

The aim of the present work was to find whether PAD occurred within identified Ib afferent fibres from GM during contractions of this muscle. The method of intra-axonal recording from afferent fibres in their intraspinal course was chosen because (i) it is the most direct approach, and (ii) the fibres remain in continuity with their receptors allowing physiological identification of fibre group (see also Jimenez, Rudomin & Solodkin, 1988). Other methods, such as recording of dorsal root potentials or testing of intraspinal excitability changes in group I fibre populations (see Schmidt, 1973), do not allow distinction between Ia and Ib fibres.

The diagram in Fig. 1 shows the positions, in the spinal cord, of the terminals where presynaptic inhibition is thought to take place (Eccles, Magni & Willis, 1962) and of the site where Ib fibres are most likely to be impaled by microelectrodes. Contraction is known to cause tendon organ discharges travelling to the central nervous system in Ib fibres. If, in addition, it induces presynaptic inhibition of homonymous Ib terminals, the intra-axonal record obtained at the impalement site

may be expected to show two superimposed signals: (i) a contraction-induced PAD electrotonically spreading to the recording site, and (ii) orthodromic impulses fired by the innervated tendon organ during the contraction (Fig. 1). Such records were actually obtained, and the demonstration of contraction-induced PAD in homonymous Ib afferents supports our assumption that the decline of autogenetic inhibition during sustained contractions may be due to presynaptic inhibition of Ib terminals. In the course of the study, Ia afferent fibres from GM were also recorded and, with a single exception, were found to lack contraction-induced PAD.

A preliminary report of this work has been published in abstract form (Zytnicki, Lafleur, Horcholle-Bossavit & Jami, 1990*a*).

METHODS

The experiments were carried out on adult cats (1·9–3 kg). In seven experiments, the dissection was done under halothane anaesthesia (Fluotan, Coopers, 1·5–2·5 % in a mixture of 50 % air and 50 % oxygen flowing at 4 l min^{-1}), followed by chloralose anaesthesia (50–70 mg kg^{-1}, I.V., initial dose, supplemented by doses of 20 mg kg^{-1} given at 3–5 h intervals). One additional experiment was done under sodium pentobarbitone anaesthesia (Sanofi, initial dose of 45 mg kg^{-1}, I.P., supplemented whenever necessary by additional I.V. doses of 4 mg kg^{-1}). The results obtained with both preparations were similar and have therefore been pooled. The experimental set-up has already been described in detail elsewhere (Zytnicki *et al.* 1990*b*) and only the specific procedures used in the present work will be described.

The gastrocnemius medialis (GM) muscle was dissected without disturbing its blood supply and its tendon was firmly attached to a force transducer (Celaster, compliance 60 μm per 5 kgf, i.e. the full range of the transducer) connected to an amplifier. Partial isometric GM contractions were elicited by stimulating either the distal end of a cut portion of the muscle nerve (in five experiments), or the muscle directly (in three experiments). In the latter experiments, the animals were curarized (Flaxedil, Spécia, 8 mg kg^{-1} h^{-1}) and artificially ventilated, end-tidal CO_2 being maintained around 4 %. In curarized animals, the adequacy of anaesthesia was assessed on: (i) miotic pupils, (ii) regularity of heart rate, and (iii) stability of blood pressure.

Submaximal contractions were produced by stimulation at 20 s^{-1} for 0·5 s. In curarized preparations, the strength of direct muscle stimulation was limited to elicit relatively weak contractions (less than 10 % of the total muscle force, see Figs 2–4). Care was taken to verify that direct stimulation did not directly excite afferent fibres. This was done by averaging large numbers of records from GM nerve or/and spinal cord surface near the entry of L7 dorsal root. Action potentials at short constant latencies had to be absent from such records during muscle stimulation.

Conventional glass micropipettes filled with either 3 M-sodium chloride (1·5–3 MΩ) or 2 M-potassium acetate (2–4 MΩ) were used for intra-axonal recordings of afferent fibres. The microelectrode was driven in the ventromedial direction into the dorsal columns, close to the entry of rostral S1 or caudal L7 dorsal roots in the spinal cord, and intra-axonal impalements occurred at depths in a range of 0·2–2 mm.

Orthodromic activation following electrical stimulation of GM nerve ascertained the origin of the impaled fibre, whose functional identification rested on two criteria: (i) axonal conduction velocity above 70 m s^{-1} indicated a fibre belonging to group I; (ii) responses of the innervated receptor to either muscle stretch or contraction allowed distinction of Ia from Ib fibres. Tendon organs, innervated by Ib fibres, were silent at rest for physiological muscle lengths, and insensitive to small muscle lengthenings, but responded during contractions. Spindle primary endings, innervated by Ia fibres, usually had a resting discharge and paused during contractions, while responding with a high dynamic sensitivity to small muscle lengthenings. In experiments where a portion of the GM nerve had been cut, identification was possible only for the fibres remaining in continuity with the muscle.

Simultaneous records of intra-axonal membrane potential, cord dorsum potential led from the entry zone of L7 dorsal root, and muscle contractile force were amplified (using a conventional AC

amplifier for cord dorsum potential, and DC amplifiers for intra-axonal potentials and muscle force) and fed into a 4-channel Nicolet 4094A digital oscilloscope performing on-line response averaging. From five to ten responses were averaged, stored on a floppy disc (Nicolet CF-44 unit) and subsequently displayed on a HP 7550A digital plotter. After withdrawal of the microelectrode from the axon by a few micrometres, extra-axonal records were taken to verify that contraction-induced depolarizations were not recorded outside the axon (see Fig. 2).

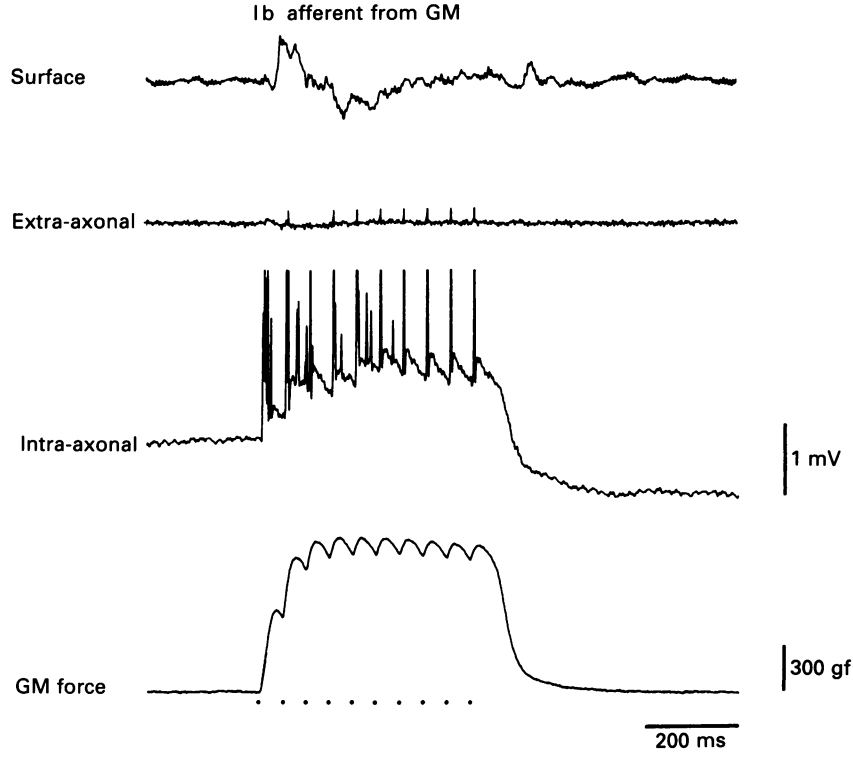

Fig. 2. Response of a Ib afferent fibre from GM (axonal conduction velocity, 87 m s^{-1}) to unfused GM tetanus. From top to bottom: cord dorsum potential (positivity downward); extra-axonal record; intra-axonal membrane potential, with superimposed impulses fired by the innervated tendon organ; simultaneous record of contractile force expressed in gram force units (gf). Stimulation of the distal portion of a cut nerve branch represented by points under force trace. Five averaged responses in each trace. Note the variable amplitudes of tendon organ action potentials due to averaging of several responses. The largest potentials had to be cut because their amplitudes were over 40 mV. The extra-axonal record was obtained during a different series of contractions with identical force profiles.

RESULTS

Among the forty-seven afferent fibres from GM that were recorded, only seventeen group I fibres (conduction velocities. 73–90 m s^{-1}) could be completely tested for occurrence of PAD during unfused GM contractions. The sample included eight Ib fibres (four from curarized and four from non-curarized preparations), and five Ia fibres (respectively, one and four from curarized and non-curarized preparations),

plus four fibres, all from non-curarized preparations, that could not be identified because their axons ran in the cut nerve branch (see Methods). The low yield of these experiments had two causes. First, the relatively small size of the explored Ib fibre population. The dorsal columns of the spinal cord contain at most forty-four Ib fibres from GM (Eldred, Bridgman, Swett & Eldred, 1962) and the number of fibres remaining in continuity with receptors was reduced in experiments where stimulation of a cut nerve branch was used to produce contraction. Second, tiny displacements of the spinal cord during contraction were not always controllable, resulting in unsteady recordings and/or expulsions of the microelectrode tip from the fibre before completion of the series of averaged tetani. The reported results nevertheless provided an answer to the question raised.

Effects of unfused tetanus on Ib afferent fibres

GM contractions elicited PAD in six (four from curarized and two from non-curarized preparations) out of the eight homonymous Ib afferents. In the example illustrated in Fig. 2, the muscle developed about 1 kg force and afferent input evoked by this contraction induced a depolarization of the homonymous Ib fibre terminals (compare the intra-axonal record in Fig. 2 with the expected record shown in Fig. 1). The depolarization displayed small oscillations of axonal membrane potential, in phase with oscillations of contractile force, superimposed on a steady level. This level reached a maximal amplitude of about 1 mV, approximately 230 ms after the onset of contraction. The PAD lasted as long as the tetanus and rapidly fell down during relaxation, after which a long-lasting hyperpolarization took place. The origin of this after-hyperpolarization is not clear; possibly, it was a consequence of repetitive firing of the fibre during the tetanus (Eccles & Krnjevic, 1959). However, this was not a consistent finding since after-hyperpolarization was not observed in other Ib afferents (see Fig. 3). As expected, the tendon organ impulses discharged during the contraction were superimposed on the PAD, which indicates that impulses propagating down the fibre to its central terminals do not hinder development of PAD (see Eccles, Schmidt & Willis, 1963*b*). In the extra-axonal record, PAD was absent whereas some of the tendon organ impulses were still visible as small action potentials. The fact that no depolarization was recorded extra-axonally provided evidence that the observed PAD was not an artifact due to some extracellular field potential. A contribution of movement artifact to the intra-axonal depolarization was not believed to be present because, in the same experiment, the same contraction (entailing similar movement) elicited depolarization in some but not all of the recorded afferent fibres.

The surface potential was most likely a composite recording of interneuronal activity and of the positive wave correlated with the development of PAD (Eccles *et al.* 1962). One cannot, however, speculate on the difference in time course between PAD and surface potential since our cord dorsum records were probably distorted by the AC amplifier which cut off low frequencies.

Contractions developing relatively small forces (Fig. 3) also elicited PADs in Ib fibres. In the example of Fig. 3*B*, the innervated tendon organ discharged a single impulse during the rising phase of a partial muscle contraction and this impulse did not appear consistently, suggesting that the tendon organ was not in series with the

contracting fibres (Stuart, Mosher, Gerlach & Reinking, 1972). However, the fact that a contraction-induced PAD was recorded in this Ib fibre indicates that depolarization is not dependent on the repetitive discharge of action potentials in the fibre.

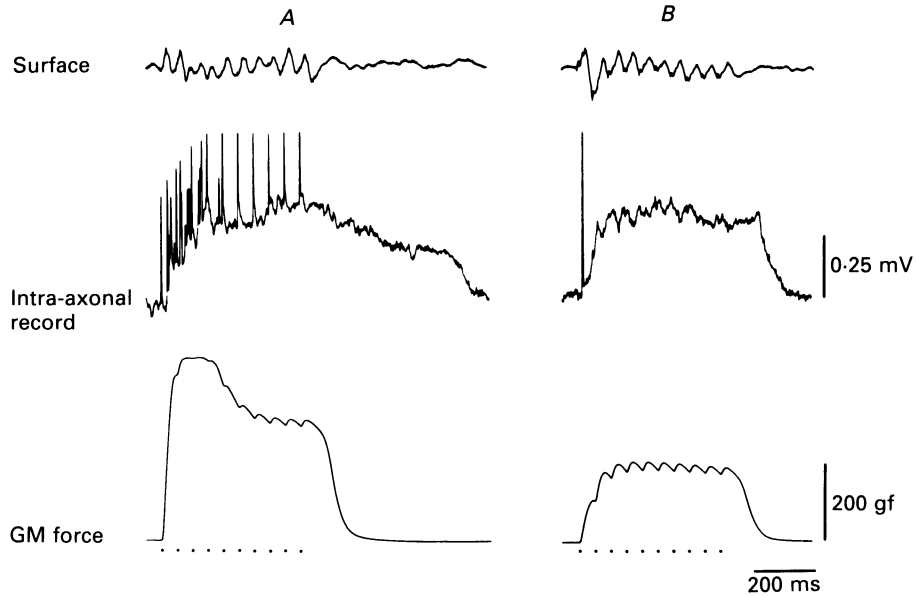

Fig. 3. Effects of GM contractions elicited by direct muscle stimulation on two Ib afferents from GM recorded in different experiments (axonal conduction velocities, 85 m s^{-1} for *A*, 87 m s^{-1} for *B*). Upper traces, cord dorsum potential (positivity downward). Middle traces, axonal membrane potential, with superimposed impulses fired by the innervated tendon organ. Extra-axonal records have been omitted for simplicity. In *A*, the discharge persisted throughout contraction; in *B*, a single impulse was fired at the onset of tetanus. Lower traces, contractile force. Ten averaged responses in each trace. Same comment as for Fig. 1 concerning the amplitudes of Ib action potentials.

The amplitudes of contraction-induced PADs were in the range of 0·1–1 mV. Their time course differed from one fibre to another. In the example of Fig. 2, depolarization and contraction had similar time courses, whereas in Fig. 3*A* the PAD decreased slowly, outlasting the contraction by about 300 ms. The contraction-induced PAD shown in Fig. 3*B* was maintained for 100 ms after onset of relaxation, and subsequently declined quite abruptly. Other PADs (not illustrated) were seen to occur only at the beginning of a tetanic contraction and were not sustained during the plateau.

Effects of contraction on Ia afferent fibres

In our sample, contraction-induced PAD occurred in only one of the five recorded Ia fibres (this was in a curarized preparation). This single instance is shown in Fig. 4. While the innervated primary ending paused during muscle contraction, a

depolarization slowly developed in the Ia fibre. This PAD did not last as long as the contraction; it started to decline during the force plateau, and was followed by a hyperpolarization (note that in this case, the hyperpolarization was not preceded by a discharge).

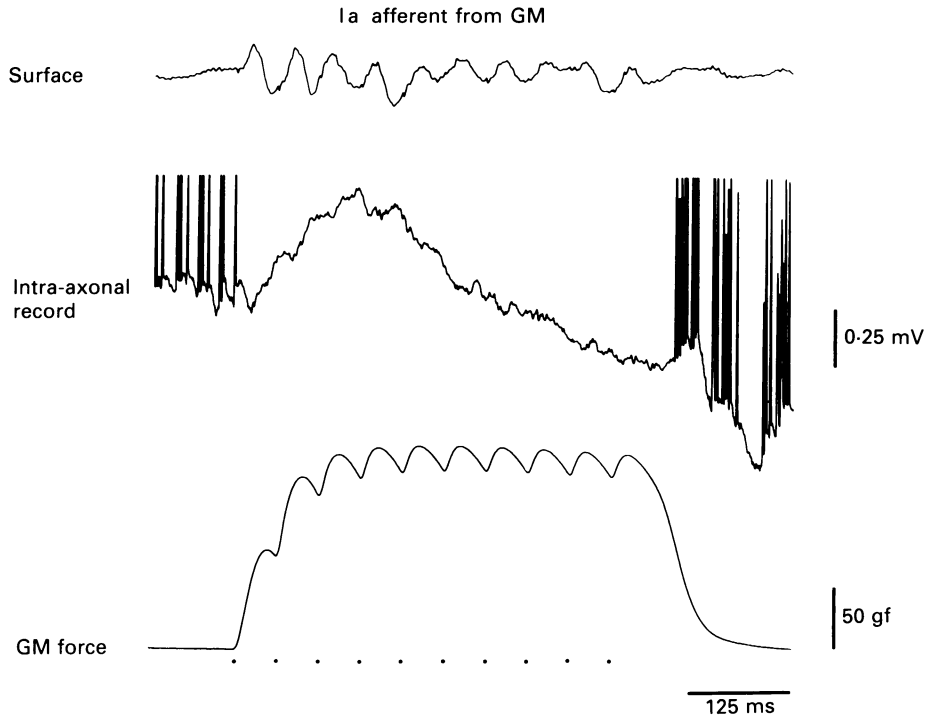

Fig. 4. Effects of a GM contraction elicited by direct muscle stimulation on a Ia afferent fibre from GM (axonal conduction velocity, 90 m s^{-1}). Same arrangement as in Fig. 3. Note the pause in the discharge of the innervated primary ending during contraction. Ten averaged responses in each trace.

Finally, two out of the four group I fibres for which functional identification was not possible displayed PADs in response to GM muscle contraction.

DISCUSSION

The main observation made in this study was that six out of eight Ib fibres from GM were depolarized by input from this muscle during contraction. Notwithstanding the limited number of observations, these results strongly suggest that GM contraction produces presynaptic inhibition in a significant proportion of homonymous Ib fibres.

The presumed sites of presynaptic inhibition (Eccles *et al.* 1962; Eccles *et al.* 1963*a*, *b*; see also Schmidt, 1973, and Rudomin, 1990) are close to Ib central terminals whose locations are known (Hongo, Ishizuka, Mannen & Sasaki, 1978; Brown & Fyffe, 1979; see Fig. 1), although axo-axonal synapses on these terminals have not been demonstrated so far. In the present experiments, the intra-axonal

impalements were made at some distance from these sites and, consequently, electrotonic attenuation could be expected to smooth out the recorded PADs. The actual amount of attenuation, depending on the length constant of the Ib fibre and on the distance between sites of PAD initiation and recording, was not known. At any rate, the amplitudes and time courses of recorded depolarizations were unlikely to exactly reflect their original shapes. This might explain the variability of PAD amplitudes and durations in our sample. However, the upper end of our range of PAD amplitudes compares with values of about 1 mV reported in previous studies (see e.g. Fig. 2 in Eccles & Krnjevic, 1959, Fig. 14 in Eccles *et al.* 1962, Fig. 14 in Eccles *et al.* 1963*a*). Such PAD sizes were considered to account for the excitability changes in group Ib fibres observed by Eccles *et al.* (1963*a*). It is therefore likely that they correspond to a functionally significant reduction of Ib inhibition during contractions.

A particularly effective attenuation in last-order branches of Ia fibres might account for the scarcity of contraction-induced PAD observed in these fibres. But an alternative explanation could be that, in our experimental conditions, impulses from GM afferents are not very efficient in producing PAD in homonymous Ia fibres during contractions.

The demonstration of contraction-induced PADs in Ib fibres supports the assumption that presynaptic inhibition is responsible for the reduction of autogenetic inhibition in motoneurones during GM contractions. The Ib IPSPs appearing in homonymous and synergic motoneurones at the onset of a contraction (and also upon abrupt force augmentation; see Zytnicki *et al.* 1990*b*) are quickly filtered out by presynaptic inhibition. The effect of this action is that the feedback information transmitted by Ib afferents to homonymous and synergic motoneurones mainly refers to increases in contractile force.

Experiments with electrical stimulation of afferent fibres have established that the main segmental sources of depolarization in flexor and extensor Ib terminals are the Ib fibres of both extensor and flexor muscles (Eccles *et al.* 1963*a*). In addition, it is well known that tendon organs are specifically activated by contraction (Houk & Henneman, 1967). It is therefore possible that the PAD in homonymous Ib terminals was caused by impulses from GM tendon organs. However, impulses arising from other contraction-sensitive receptors (see Bessou & Laporte, 1960; Mense & Meyer, 1985; Cleland, Hayward & Rymer, 1990) innervated by group II and III fibres could also reinforce the PAD of Ib fibres (Eccles *et al.* 1963*a*).

The pattern of presynaptic inhibition of group I fibres is not rigid. Observations on cyclic changes in the excitability of Ia and Ib fibres during fictive locomotion suggest that central pattern generators can modulate presynaptic input to group I fibres (Duenas & Rudomin, 1988; see also Dubuc, Cabelguen & Rossignol, 1988). In man, the level of presynaptic inhibition of homonymous Ib fibres has been shown to change during a voluntary ramp-and-hold plantar flexion (Meunier & Pierrot-Deseilligny, 1989). The complex organization of pathways mediating presynaptic inhibition accounts for this flexibility. In the cat spinal cord, the shortest pathway is trisynaptic (see Schmidt, 1973; Jankowska, McCrea, Rudomin & Sykova, 1981), and there are several distinct populations of interneurones mediating PAD of group I fibres (Brink, Jankowska & Skoog, 1984). Various sources of segmental and

descending inputs converge onto interneurones mediating presynaptic inhibition (Schmidt, 1973; Baldissera, Hultborn & Illert, 1981; Rudomin, 1990), and it is likely that alternative subgroups of interneurones may be activated or inhibited depending on the type of movement. It is very difficult to predict which pattern of presynaptic inhibition will prevail under natural conditions, in execution of automatic or voluntary movements. At any rate, presynaptic filtering of information from Ib fibres during a sustained contraction might help to maintain firing of motoneurones and to recruit new motor units when the effort has to be increased.

The authors wish to thank Professor Y. Laporte for constant support and interest in their work. The financial support of INSERM (CRE-886008) and AFM-IRME (CS 17 05 89) is gratefully acknowledged.

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