Antidromic Inhibition of Presumed Fusimotor Neurones by Repetitive Stimulation of the Ventral Root in the Decerebrate Cat

Inhibition of α-motoneurone discharge by antidromic stimulation of ventral root or muscle nerve was first described by RENSHAW[1,2]. The absence of a similar inhibition acting on fusimotor neurones has been noted on several occasions[3–6]. However, ECCLES et al.[5] recorded a small amount of antidromic inhibition in a soleus motoneurone in which the axon had a high threshold to excitation and a conduction velocity of 35 m/sec. They considered this a 'doubtful instance of a γ-motoneurone receiving recurrent inhibition'.

Very recently ELLAWAY[7] found that antidromic stimulation of the ventral root produced weak inhibition in 6 out of 20 fusimotor fibres of measured conduction velocity, isolated from muscle nerve. Single shocks, subthreshold for the fusimotor fibre, were applied to the corresponding ventral root. The effect was seen as a prolongation of the interspike interval immediately following the arrival of the antidromic volley at the spinal cord.

In the course of experiments undertaken to determine the reflex effects of vibration of muscle on the efferent discharge in ventral root filaments[8], we were led to reexamine the possibility that there is antidromic inhibition of fusimotor neurones. Longitudinal small-amplitude vibration of a muscle selectively excites the primary endings of the muscle spindles[9]. Vibration of the triceps surae muscle in decerebrate cats was found to produce contrasting effects on 2 types of unit recorded in ventral root filaments. 1 type of unit was excited to discharge by the vibration. These units had large amplitude spikes and were not spontaneously active, although some of them could be made to discharge by maintained stretch of the muscle. These were presumed to be α-motoneurones. The other type of unit was spontaneously active and had small amplitude spikes. None of these units could be excited to discharge by either stretch or vibration of the muscle. Indeed some of them were inhibited by the vibration. These units were presumed to be fusimotor neurones. It was found that antidromic stimulation of the ventral root inhibited the discharge of some of these presumed fusimotor neurones.

Materials and methods. The experiments were performed on 3 unanaesthetized, decerebrate cats in which the right hind limb was denervated, except for the triceps surae (Figure 1). The tendons of this muscle were attached to an electro-magnetic stretcher[10] by which, in addition to stretching the muscle, longitudinal, small-amplitude vibration could be delivered to the muscle[9]. The dorsal roots were left intact but the L_7 and S_1 ventral roots were cut close to their exit from the spinal canal. A portion of 1 of the ventral roots was split into fine filaments containing 1–3 spontaneously active units; the remainder, approximately $^2/_3$ of the same root, was placed on silver wire electrodes for stimulation. The amplified action potentials of the spontaneously discharging units were recorded by external electrodes and led through a 'window circuit' which allowed selection of 1 of the spikes. This spike triggered a short pulse which was used to activate a reciprocal pulse interval display[11].

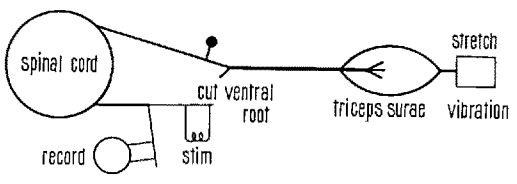

Fig. 1. Experimental arrangement.

Results and discussion. A total of 17 spontaneously active units were tested for the effect of antidromic stimulation of the ventral root. The units had a spontaneous discharge frequency of 10–70 imp./sec; they were all selected for study because they were reflexly inhibited by vibration of the triceps surae. In 8 of the units, repetitive antidromic stimulation (30–100/sec) of the central end of the cut ventral root caused an inhibition of their firing rate. The drop in frequency ranged from 5–35 imp./sec. An example of the effect is shown in Figure 2. The unit was initially discharging at 55/sec, but with the onset of the antidromic stimulation the firing rate fell to 30/sec. On cessation of stimulation the discharge returned approximately to its previous level. Figure 3 shows the effect on the same unit of varying the

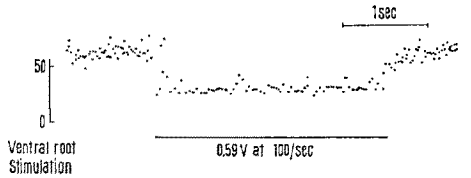

Fig. 2. Frequency (reciprocal pulse interval) display of the response of a unit to antidromic stimulation of the ventral root at 100/sec.

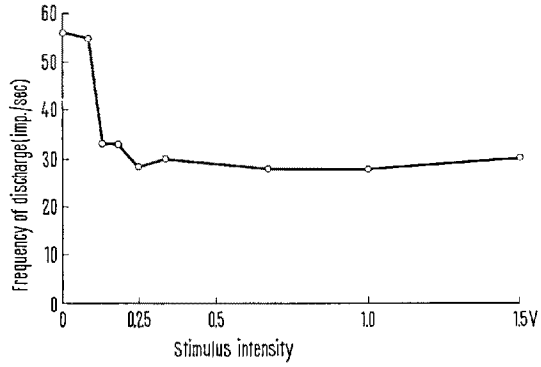

Fig. 3. The effect of increasing stimulus intensity at 100/sec to the ventral root upon the discharge of the same unit as shown in Figure 1.

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strength of the stimulus applied to the ventral root. As the stimulus was increased the inhibition appeared abruptly at approximately 0.1 V, and increased little further with further increases in stimulus strength. The threshold of α-motor fibres with the present stimulating arrangements is 50–150 mV[12] so that most if not all the inhibition was due to stimulation of α-motor fibres rather than of fusimotor fibres, which have a higher threshold. All 5 units studied with graded stimuli were inhibited by shocks around the presumed α-threshold and no definite evidence was obtained of increasing inhibition on further increasing the stimulus intensity. However, the variability of the responses was such that a slight, or variable, superadded inhibition on exciting fusimotor fibres has not been excluded. In this respect, it is interesting that ECCLES, FATT and KOKETSU[13] failed to find any additional effect on RENSHAW cells with stimuli strong enough to excite 'small motor axons'. Thus it may be concluded, in agreement with ELLAWAY, that antidromic inhibition of fusimotor neurones may occur on occasion and that it is primarily due to stimulation of α-motor fibres.

Résumé. On a enregistré des décharges de 17 fibres fusimotrices dont on suppose l'existence dans les filaments de racines ventrales des chats décérébrés. 8 fibres ont été inhibées par stimulation répétée de la portion centrale isolée du reste de la racine ventrale. Ces résultats montrent qu'il existe une inhibition antidromique dans les neurones fusimoteurs.

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University Laboratory of Physiology, Oxford (England), 8 July 1968.

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Swelling in Tissues of Nephrectomized Rats

It is known that the water content of different tissues becomes modified after the inhibition of their metabolism[1], or when disturbed by distemper or thirst[2–4], the process being partly governed by the osmotic concentration present in the cells in vivo[5]. In a previous study into the electrolyte composition of nephrectomized rats' tissues, we made some calculations on cellular osmolarity[6]. It appeared that, depending on various tissues and conditions, the cellular concentration of particles with restricted diffusibility was increased. Accordingly it may be expected that the water content of cells should increase if (1) the above-mentioned cell osmolarity has augmented (as for example in case of a higher uremic catabolism), (2) membrane permeability has not essentially altered and (3) metabolism has been inhibited. To check this suggestion we examined the swelling of muscles of nephrectomized and control rats.

Material and method. After having nephrectomized rats bilaterally, a group of them was water deprived and another group drank ad libitum. Cube sugar was offered as food. Sham-operated and untreated animals kept under similar circumstances served as controls. 2–3 days later, using ethyl ether as narcotic, rats were decapitated and both the soleus and the extensor dig. l. muscles were carefully prepared off the bones and soaked at 5 °C for different periods in Ringer's saline. In the following, an account will be given on the changes in the water content deduced from the difference of muscle weights before and after soaking.

Results and discussion. After 4.5 h soaking the weight increase of M. soleus of the nephrectomized rats was

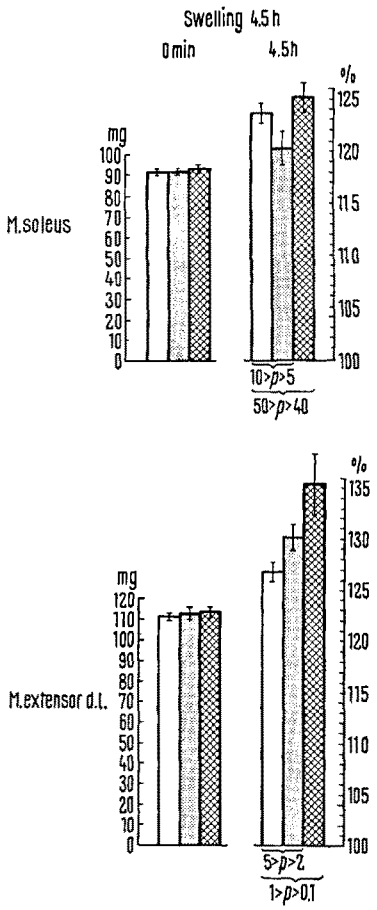

Fig. 1. Left side, mg muscle weight before soaking. Right side, changes of muscle weight in % on soaking. White columns: Muscles of sham-operated and untreated rats. Stippled columns: Muscles of nephrectomized rats drinking ad libitum. Columns with oblique squares: Muscles of thirsting nephrectomized rats.

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