Recurrent inhibition of the monosynaptic reflex in the upper caudal cord of the cat

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Summary. In spinal cats both ipsi- and contralateral antidromic volleys in the 3rd sacral ventral roots produce recurrent inhibition of the monosynaptic reflex evoked by stimulation of the 1st caudal dorsal root and recorded from the corresponding ventral root.

Antidromic stimulation of the motor axons inhibits all motoneurones in proximity to the motor cells from which these axons originate. This inhibitory action was first described by Renshaw[1] and was then named recurrent inhibition. Antidromic volleys through motor axon collaterals excite the Renshaw cells which in turn inhibit the motoneurones. In the spinal cord recurrent inhibition has mainly been studied in the lumbar segments, where transmitter substances at the synaptic junctions were identified and the time course of inhibition was determined[2-4]. The lower sacral and caudal segments differ in many respects from the lumbar cord. This is exemplified by the distribution of the dorsal roots terminating on the motor cells, and by the properties of the dorsal root potentials spreading to the contralateral side of the cord[5-7]. These data suggest that the pattern of recurrent inhibition in the lowermost parts of the cord may also exhibit features distinct from those found in the lumbar segments. In the present investigation recurrent inhibition of the monosynaptic reflex was studied in the first caudal (Ca 1) segment of the spinal cord.

Methods. The experiments were performed on adult cats anaesthetized with pentobarbital sodium (35 mg/kg, i.p.). The monosynaptic reflex was evoked by single pulses lasting 0.5 msec applied to Ca 1 dorsal root and recorded from the ventral root of the same segment. The strength of stimulation was 3 times the threshold. The monosynaptic reflex was conditioned by antidromic stimulation of the central end of the 3rd sacral (S 3) ventral root. Ventral roots from both sides of the cord were used for conditioning. The strength of stimulation of the ipsi- and contralateral ventral root was adjusted separately to reduce the monosynaptic reflex to 45-50% of the control value. To exclude the possibility of the stimulus current spread the dorsal and ventral roots were cut most distally near their exits through the dura and stimulating electrodes were placed close to their cut ends. In all preparations the distance between electrodes and the entrance of the roots into the cord was not less than 22 mm.

Results and discussion. The amplitude of the testing monosynaptic reflex is reduced by a preceding antidromic volley in the ventral root of the same side of the cord. The inhibition depends on the interval between the conditioning and testing volleys. The records of figure 1A and the curve of figure 2 show that the initial reduction of the reflex occurs rapidly. At the testing interval of 8 msec the monosynaptic reflex is lowered to 63% of the control while at the interval of 20 msec depression is at its maximum and the reflex discharge only reaches 45% of the initial value. At longer intervals between the conditioning and testing stimuli a slow increase in the size of the monosynaptic reflex is observed. A full recovery of the reflex has a time course of about 100 msec.

The records of figure 1B demonstrate that also contralateral ventral root volleys inhibit the monosynaptic reflex. From the curve of figure 2 it may be seen that the decrease in the size of the discharge is more rapid than during conditioning with ipsilateral volleys, and its largest reduction is observed at the testing interval of 8 msec. With lengthening of the testing interval the size of the monosynaptic reflex commences to increase and it regains the control value at the interval of about 64 msec.

Similar results were obtained in 16 experiments. The mean testing interval at which recurrent inhibition evoked by ipsilateral ventral root volleys was maximal amounted to 16 ± 2.45 msec. After contralateral volleys the maximal recurrent inhibition occurred at the mean interval of 8.5 ± 0.87 msec. The asymptotic course of the inhibitory curve made it sometimes difficult to estimate the recovery of the monosynaptic reflex from recurrent inhibition. It was found that the mean time course of recovery of the reflex preceded by an ipsilateral conditioning volley was 92.5 ± 23.56 msec and that preceded by a contralateral antidromic volley amounted to 53.8 ± 11.4 msec.

Our experiments show that recurrent inhibition in the Ca 1 segment is bilateral. The time course of reduction of the monosynaptic reflex evoked by ipsilateral conditioning is similar in the lumbar, sacral and caudal cord[3,8]. It may be thus surmised that in these segments ipsilateral recurrent

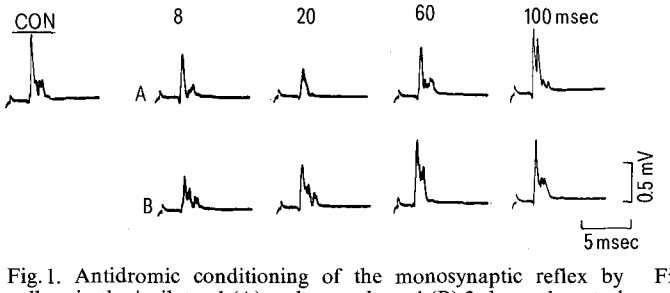

Fig. 1. Antidromic conditioning of the monosynaptic reflex by volleys in the ipsilateral (A) and contralateral (B) 3rd sacral ventral root. The monosynaptic reflex was produced by stimulation of the 1st caudal dorsal root and recorded from corresponding ventral root. The first record (CON) represents control reflex not preceded by antidromic volley. The next records illustrate conditioning of the monosynaptic reflex. Numbers above records indicate intervals between conditioning and testing volleys.

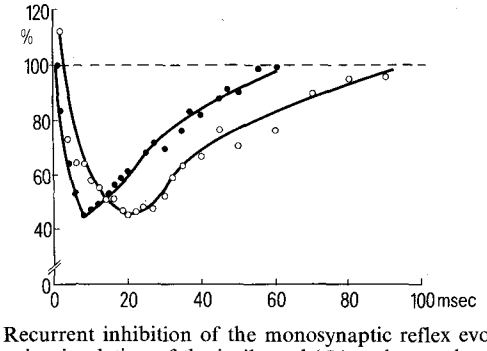

Fig. 2. Recurrent inhibition of the monosynaptic reflex evoked by antidromic stimulation of the ipsilateral (○) and contralateral (●) ventral roots. Monosynaptic reflex was produced by stimulation of the 1st caudal dorsal root and recorded from the ventral root of the same segment. It was conditioned by stimulation of the 3rd sacral ventral roots. Abscissa, interval between conditioning and testing volleys. Ordinate, size of the monosynaptic reflex calculated as percentages of the mean control value. Each point is the arithmetic mean of 9-12 records.

inhibition is subserved by analogous neuronal pathways. An important difference between these segments consists in the occurrence of recurrent inhibition produced by contralateral antidromic volleys. In the lumbar cord motoneurones are only inhibited by ipsilateral conditioning[2,9]. However, in the sacral cord both somatic and parasympathetic neurones are affected by contralateral volleys. Our preliminary experiments on the effect of 5-hydroxytryptophan on somatic recurrent inhibition in the S 3 segment suggest that it may be bilateral[8]. De Groat and Ryall[10] found that antidromic stimulation of the ventral roots containing parasympathetic preganglionic fibres depressed spontaneous contractions of the bladder. Although this depression was observed in preparations in which only crossed effects could be studied, it is very probable that autonomic recurrent inhibition is bilateral.

It remains to be elucidated whether the shorter time course of recurrent inhibition evoked in the Ca 1 segment by contralateral conditioning depends on specific properties of the pathway transmitting antidromic inhibitory action to the opposite side of the cord. Since the caudal segments together with the lower sacral segments innervate the midline structures of the body, bilateral recurrent inhibition provides the best coordination of the motor function of symmetrical halves of the cord.

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Cellularity and composition of epididymal adipose tissue from cold-acclimatized rats[1]

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Summary. Cold acclimatization induces morphological and compositional modifications of rat epididymal adipose tissue: a decrease in fat cell size, an increase of fat cell number per g of tissue, but no significant increase in total fat cell number in the tissue; finally, an increase in protein content and a decrease in triglyceride content.

The epididymal adipose tissue from cold-acclimatized rats (CA) shows an increased capacity of synthesizing fatty acids from acetate[2] and an increased lipolytic potential in response to norepinephrine[3]. In agreement with these observations, in this laboratory it was found[4] that cold acclimatization induced a twofold increase in the turnover rate of fatty acids in the triglycerides of the rat epididymal adipose tissue. This result led us to investigate the effects of 4 weeks of cold-exposure upon the cellularity and the chemical composition of rat epididymal adipose tissue in order to determine if morphological and compositional alterations were associated with the metabolic ones previously described.

Materials and methods. Experiments were performed on Long Evans male rats. The animals were acclimatized to different thermic conditions when 7 week old: the control group was maintained at 28 °C (thermal neutrality) for 3 weeks; the cold-acclimatized group (CA) was exposed to a constant temperature of 5 °C for 4 weeks. This schedule resulted in 2 groups of mean b.wt between 280 and 305 g. The animals were maintained on a daily 12-h dark-light cycle and fed on a standard laboratory diet with water ad libitum until sacrifice.

Isolated fat cells were prepared according to the procedure of Rodbell et al.[5], with minor modifications: the absence of glucose, a lowering of the collagenase concentration (5 mg/g adipose tissue), a decrease in the dissociation period (45 min) and 2 cell washing procedures instead of 3.

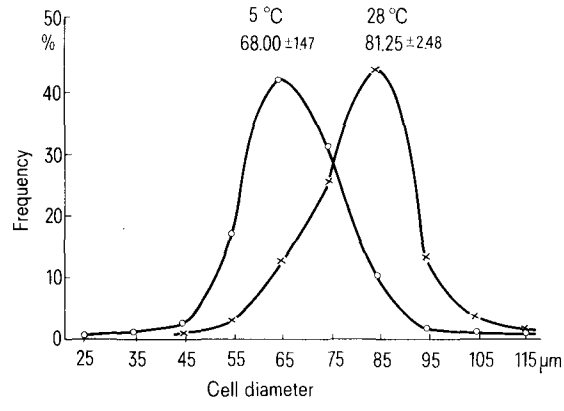
Fat cell size was determined according to the photomicrographic method described by Lavau et al.[6]. The fat cell number was determined using the method of Di Girolamo et al.[7]. Fat cell yields (approx. 80%) did not differ significantly between the 2 groups.

Total lipids were extracted from tissue samples by the procedure of Folch et al.[8]. Aliquots of lipid extracts were analysed for phospholipid phosphorus using Bartlett's method[9]. After hydrolysis and selective precipitation by digitonin, as described by Sperry and Webb[10], additional aliquots were analyzed for cholesterol content by ZAK's technique[11].

Triglyceride (TG) content (percent wet weight of tissue) was calculated as follows: TG content = total lipid content (percent wet weight of tissue) − [phospholipid content (percent wet weight of tissue) + cholesterol content (percent wet weight of tissue)].

Protein content was determined by the method of Lowry et al.[12] after precipitation by 100% perchloric acid.

Results and discussion. Effects of cold acclimatization on the diameter distribution pattern of epididymal fat cells. A diameter distribution pattern of epididymal fat cells from CA rats and controls is shown in the figure. Cold acclimat-



Epididymal fat cell diameter distribution patterns of CA and control rats. The values shown at $\bar{X} \pm$ SEM represent the mean fat cell diameter for 4 rats in each group.