J. Physiol. (1953) 121, 638–640

POTASSIUM EXCHANGE IN GANGLIA AFTER POSTGANGLIONIC NERVE SECTION

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(*Received* 20 *April* 1953)

Three weeks after section of axons issuing from sympathetic ganglia, transmission of impulses through the ganglion on maximal stimulation of the preganglionic fibres is abolished, although conduction in the postganglionic axons up to the point of section is unimpaired, and ganglionic transmission to other postganglionic nerves is also normal (Brown, McLennan & Pascoe, 1952). The failure of transmission might be due to a permanent depolarization of the ganglion cells as a result of the 'axotomy', and this in turn might be reflected in a change of the potassium turnover of these ganglion cells.

Potassium exchanges in axotomized ganglia have therefore been investigated using radioactive ^{42}K as a tracer, and the results compared with those obtained on normal ganglia described in the preceding paper (Harris & McLennan, 1953). It has been found that the potassium exchange in axotomized ganglia remains unaltered.

METHODS

The superior cervical ganglia of rats and rabbits have been used, since nearly all their postganglionic fibres can be readily identified and sectioned without damage to the tissue or its vascular supply. Unilateral axotomies were carried out with aseptic precautions, the nerves being cut close to the ganglion. The contralateral ganglion served as a control. After allowing a period of 3 weeks for complete degeneration, both ganglia were excised, the capsules removed, and the fresh weights determined with a torsion balance.

The determination of the rates of K influx and efflux has been described in detail in the preceding paper. All the incubations were carried out at room temperature, in a saline medium containing the appropriate bicarbonate concentration to give pH 7·4 when equilibrated with 5% CO_2 at 18° C. K analyses were again performed with a flame photometer.

RESULTS

The comparison between normal and axotomized sympathetic ganglia has failed to reveal any differences either in the total amount of K contained in the tissue or in the rate and extent of its turnover. One consideration must, however, be made: comparison of the fresh weights of eleven pairs of normal ganglia from both rats and rabbits has shown that the weights of two ganglia from the same animal agree within 3% of each other, whereas an axotomized ganglion may weigh as much as 40% more than the control. The discrepancy is due to the development of a neuroma of varying size at the point of section, and for this reason calculations have been made for the whole ganglion rather than per gram of tissue. Analyses of three pairs of fresh ganglia have given values of 0·16, 0·15 (rats) and 0·46 (rabbit) μequiv K per ganglion for the axotomized, and 0·10, 0·15 and 0·43 μequiv per ganglion for the corresponding controls.

TABLE 1. Potassium exchanges in normal and axotomized ganglia

Parameters for the equations for influx and efflux of ^{42}K.

Influx: $^*K = A[1-\exp(-at)] + B[1-\exp(-bt)]$.

Efflux: $^*K = A[1-\exp(-at)] + B[1-\exp(-bt)] - A\{1-\exp[-a(t-t_1)]\} - B\{1-\exp[-b(t-t_1)]\}$.

A and B as % of total K; 'a' and 'b' in hr^{-1}.

Animal	Temp. (° C)	Ganglion	A	a	B	b
Rat	16	Normal	14	9	46	0·56
		Axotomized	11	12	49	0·66
Rabbit	17	Normal	3	6	47	0·22
		Axotomized	10	6	47	0·31
Rabbit	17·5	Normal	—	—	35	0·31
		Axotomized	—	—	39	0·24

The time course of the influx and efflux of K in the axotomized ganglia, as in the normal, can best be described by an expression involving two exponential terms. Table 1 shows the parameters obtained from the experimental curves for three pairs of ganglia for the uptake and loss of ^{42}K when the tissue was incubated in saline medium. Although the agreement between the values for the control and the operated ganglion is not particularly good, especially in the values for 'b', there is no sign of a consistent shift in any direction which might indicate that an important alteration has taken place as a result of the axotomy.

The behaviour observed when the tissue was placed in isotonic K phosphate solution also did not differ from normal. There was a rapid uptake of labelled K until a steady state corresponding to complete exchange of the tissue K was reached.

DISCUSSION

These results show that in resting axotomized ganglia *in vitro* the degree and time course of the movement of K is the same as in normal ganglia. If it is true that a part at least of the K concentration in ganglia is maintained in a Donnan equilibrium, as is the case in nerve and muscle, then a reduction of the potential difference is likely to increase the rate of efflux of the intracellular K. It would seem, therefore, unlikely that an axotomized ganglion cell can be appreciably depolarized, for if this were so its total K content should be considerably diminished and the rate of K efflux increased.

In the preceding paper the question was discussed whether the measured rate constants for K turnover are in fact applicable to the penetration of K into the cells. If the slower rate constant '*b*' merely represented the limitation imposed by slow diffusion of K within or without the cells, then '*b*' would be unlikely to show change after axotomy unless great structural changes occurred in the tissue, either in the cells or in the extracellular space, and no such changes have been observed histologically (Sternschein, 1920).

SUMMARY

The K contents, and the rates of K exchange, have been determined in sympathetic ganglia after section of the postganglionic axons. No differences between normal and axotomized ganglia have been found.

The expenses of this work have been covered by a grant from the Government Grants Committee of the Royal Society.

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