THE MECHANISM OF RENIN RELEASE FROM THE ISCHAEMIC KIDNEY

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The re-establishment of blood flow to an ischaemic kidney produced an elevation of blood pressure in the rat. This response did not occur in animals with a pithed spinal cord or in rats with low blood pressure produced by haemorrhage. When the blood pressure was raised in rats with pithed spinal cords, by the intravenous infusion of noradrenaline, the response was restored. Occlusion of the subclavian arteries did not prevent the response. It is considered that the increase in blood pressure, produced by renin release, after re-establishment of the blood flow in an ischaemic kidney is a 'washout' phenomenon independent of the integrity of the nervous system.

Introduction Taquini & Braun Menendez (1941) and Taquini, Blaquier & Taquini (1964) have demonstrated that the blood pressure elevation following re-establishment of blood flow to an ischaemic kidney was due to an increase of renin in the systemic circulation. Hayden & Targett (1971a, b) postulated that the nervous system is necessary for renin release and at least some of the pressor action of the renin is mediated by the nervous system. However, Long & Severs (1973) suggested that renin release from the ischaemic kidney is independent of the nervous system if adequate blood flow through the kidney is maintained and the pressor response obtained after re-establishing circulation to the ischaemic kidney is due to a 'washout' phenomenon.

Our purpose was to elucidate the importance of the nervous system in the elevation of blood pressure obtained after re-establishment of the blood flow to an ischaemic kidney.

Methods Wistar rats weighing about 200 g were used. The animals were anaesthetized with ether and the renal pedicle was exposed and occluded. About 3.5 h later the animals were anaesthetized with 1 g/kg urethane and respiration was controlled by means of a Harvard breathing pump (Model 680) using air at room temperature. The arterial pressure was recorded on a polygraph via a Statham P23AA pressure transducer connected to a cannula in the carotid artery. The animals were divided into five groups: (a) control; (b) rats with both subclavian arteries occluded before the origin of the vertebral artery; (c) rats with pithed spinal cord; (d) rats in which the spinal cord was pithed and in which noradrenaline (2.4 μg/min) was infused into the jugular vein with a Harvard pump

Table 1 Modification of the pressor response after re-establishing circulation to the ischaemic kidney

		Blood pressure (mmHg) ± s.e.		
		Basal	*Maximum increase*	n
Control rats	SP	137 ± 7	27 ± 6	10
	DP	98 ± 7	28 ± 5	
Rats with subclavian arteries occluded	SP	146 ± 8	18 ± 3	10
	DP	101 ± 7	20 ± 2	
Spinal pithed rats	SP	62 ± 5	4 ± 2*	10
	DP	34 ± 5	3 ± 2**	
Spinal pithed rats infused with noradrenaline	SP	165 ± 12	39 ± 7	9
	DP	116 ± 9	27 ± 5	
Haemorrhagic rats	SP	48 ± 4	1 ± 2**	10
	DP	21 ± 3	0 ± 2**	

SP, Systolic pressure; DP, Diastolic pressure.
* $P < 0.005$ compared with control.
** $P < 0.001$ compared with control.

until a stable blood pressure was obtained; (e) bled rats (3 ml/100 g). The spinal cord was pithed by the procedure of Long & Severs (1973).

Results The results obtained are summarized in Table 1. The increase of blood pressure after removal of the clamp on the renal pedicle had a rapid onset commencing in less than 1 min, reaching a peak in about 5 min and beginning to decrease in approximately 8 minutes.

Pithing the spinal cord prevented the increase of blood pressure obtained when the clamp was removed from the renal pedicle, but the response was restored when the arterial pressure was raised by an infusion of noradrenaline.

The bled rats had a low blood pressure similar to that of rats with a pithed spinal cord and like them they showed no rise in arterial pressure when the clamp was removed from the renal pedicle.

The occlusion of the subclavian arteries produced no modification of the ischaemic kidney pressor response.

Discussion The results obtained in control animals and in pithed rats are similar to those reported by Hayden & Targett (1971a) and Long & Severs (1973).

The fact that the response in rats with both subclavian arteries occluded was similar to that in control animals suggests that the increase in blood pressure was not due to a central action of angiotensin (Ferrario, Dickinson & McCubbin, 1970).

The results further suggest that it is the reduction of blood pressure consequent upon pithing rather than the removal of neural control which is responsible for the loss of the pressor response on restoring blood flow to the totally ischaemic kidney. This suggestion is supported by the fact that maintenance of blood pressure in the pithed rat restores the response, whilst reduction of blood pressure by means other than pithing (bleeding in this case) similarly abolishes it.

It is concluded that without adequate perfusion pressure when the renal artery is unclamped, renal cortical blood flow is not re-established and wash out of renin into the systemic circulation does not occur.

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