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HAEMODYNAMIC RESPONSES AND RENIN RELEASE DURING STIMULATION OF AFFERENT RENAL NERVES IN THE CAT

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SUMMARY

1. Experiments were done on anaesthetized cats to study the effect of electrical stimulation of afferent renal nerves on the circulatory system and on the release of renin from the kidney.

2. Stimulation of afferent renal nerves over a wide range of parameters consistently elicited an increase in arterial pressure and heart rate. This response was still present in paralysed animals and was not accompanied by changes in respiration or in sympathetic autonomic activity usually associated with painful stimulation. Mesenteric and iliac vasoconstriction was observed concomitantly with the increase in arterial pressure.

3. Release of renin from the contralateral innervated kidney was not significantly changed by stimulation of afferent renal nerves.

4. The existence of renal vascular mechanoreceptors was investigated by altering renal circulation. Stenosis of the renal artery or a marked reduction in renal perfusion pressure elicited an increase in arterial pressure while stenosis of the renal vein elicited a decrease in arterial pressure. These responses, however, were not affected by denervation of the kidney and were therefore interpreted as not being due to neural mechanisms.

5. The precise nature, location and physiological role of renal receptors involved in the cardiovascular responses observed during electrical stimulation of afferent renal nerves remain to be determined.

INTRODUCTION

The role of physiological signals originating in the kidney and carried in afferent renal nerves in the regulation of the cardiovascular system is unknown. Electrical activity has been recorded from renal afferent fibres

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in rats (Aström & Crafoord, 1967), cats (Aström & Crafoord, 1968; Beacham & Kunze, 1969; Kady, 1974), dogs (Ueda, Uchida & Kamisaka, 1967; Uchida, Kamisaka & Ueda, 1971; Kady, 1974), and rabbits (Niijima, 1971, 1972, 1975) and the consensus is that the kidney contains mechanoreceptors whose precise location, anatomical connexions and functional significance are poorly defined.

With regard to the functional role of afferent renal nerves in the regulation of arterial pressure there are two reports demonstrating a decrease in systemic pressure during electrical stimulation of these nerves (Ueda *et al.* 1967; Aars & Akre, 1970) and one report of no change in arterial pressure during stimulation (Aström & Crafoord, 1968). The suggestion has been made (Aars & Akre, 1970; Niijima, 1971) that an important role of afferent renal nerves is to carry information from baroreceptors with activity similar to that of sino-aortic baroreceptors.

If renal baroreceptors do indeed exist it may then be suggested that they are also involved in a reflex arc leading to release of renin, as supported by the finding that there is a neural component in the release of renin when renal perfusion pressure is decreased by suprarenal aortic stenosis (Zanchetti & Stella, 1975). The experiments reported here were done to investigate the effect of electrical stimulation of afferent renal nerves on systemic arterial pressure and on the release of renin in the cat. In addition, the possibility of the existence of renal mechanoreceptors was investigated by altering renal circulatory conditions. A preliminary note of the results has appeared (Cafiero, Calaresu, Stella & Zanchetti, 1975).

METHODS

Results were obtained from thirty-three adult (2·5–4·3 kg) cats of either sex, of which thirty were anaesthetized with sodium pentobarbitone (Nembutal Abbott, 40 mg/kg I.P. initially and additional maintenance dose I.V.) and three with α-chloralose (E. Merck, Darmstadt, 60 mg/kg I.V. after ether induction). Rectal temperature was maintained at $38 \pm 0{\cdot}5°$ C. In all animals heart rate was recorded on the polygraph using a tachograph triggered by an e.c.g. signal; arterial pressure was monitored from a catheter in the descending aorta connected to a Statham P23Db pressure transducer, and recorded with other haemodynamic variables on a model 7 Grass polygraph.

Electrical stimulation of afferent renal nerves

Haemodynamic studies. Renal nerves on either side were exposed through a mid line abdominal incision and isolated from surrounding tissues as far laterally as the kidney hilum. They were then placed on stainless bipolar sleeve electrodes and positively identified as renal nerves by demonstrating a decrease in blood flow in the ipsilateral kidney during electrical stimulation at 5 V, 0·2 msec, 20 Hz. After identification renal nerves were ligated and crushed distal to the electrodes to allow selective stimulation of afferent fibres as was demonstrated by the disappearance of ipsilateral renal vasoconstriction during stimulation after crushing. Rectangular

pulses were delivered through a SIU4678 Grass isolation unit from a Grass S4K stimulator. In the experiments concentrated on haemodynamic changes, the train duration was rather short (20–30 sec) and parameters of stimulation widely varied (duration 0·05–5 msec, frequency 5–50 Hz, amplitude 3–30 V). Instantaneous velocity of blood flow was measured in different vascular beds (renal, mesenteric and iliac) by a Statham model 4001 electromagnetic flowmeter and MDQ probes (1·5–2 mm i.d.), integrated by a Grass 7P10A integrator reset at 2 sec intervals and recorded on a Grass 7 polygraph. In some experiments respiration was monitored as a change in pressure of an oesophageal balloon filled with water, connected to a Statham P23De transducer and recorded on the polygraph. To test the possibility that some of the haemodynamic responses might have been secondary to muscle activity, in a few experiments the animal was paralysed with gallamine triethiodide (Sincurarina Farmitalia, 5 mg/kg I.V.) and artificially ventilated with a Harvard model 607 pump.

Renin release. When testing effects on renin release, stimulation of afferent renal nerves was prolonged for 5 min, and other parameters selected among those found most effective haemodynamically (5 msec, 20 Hz, 15 V). Polyethylene catheters (1 mm o.d.) were placed in both renal veins, on the right via the femoral vein through the inferior vena cava and on the left via the gonadal vein. The abdominal incision was then closed and the animal was allowed a rest period of 1 hr. A first set (control) of blood samples for determination of plasma renin activity from each renal vein and from a peripheral artery was then collected. Soon after this collection afferent renal nerves were stimulated for 5 min, and during the last minute of the stimulated period a second set of samples (response) was obtained. Plasma renin activity was measured by radioimmunoassay for angiotensin I (AI) and renin release was calculated according to methods previously described (Richardson, Stella, Leonetti, Bartorelli & Zanchetti, 1974).

In all the experiments in which renal circulatory or renin-releasing responses were investigated, the integrity of renal nerves to the contralateral kidney was tested at the beginning and end of each experiment by brief pontine stimulation to induce a conspicuous vasoconstriction in the innervated kidney (Richardson *et al.* 1974).

Manoeuvres to alter circulatory conditions of the kidney

Obstruction of the renal vein on either side was effected by placing a no. 4 surgical silk thread around the vein approximately one cm from the inferior vena cava and crossing it manually. The extent of the stenosis was controlled by monitoring on a Grass polygraph pressure in the renal vein from a catheter placed in the vein and connected to a Statham P23De transducer. Obstruction of the renal artery was obtained using a similar method. In addition, in four animals, the kidney was perfused at different pressures by the use of a Holter pump model RE161. The perfusing blood was drawn from a cannula inserted into the carotid artery and pushed by the pump into a cannula inserted into the femoral artery, the descending aorta and finally placed and ligated in the renal artery. With this method the kidney was deprived of its blood supply for very short periods of time. Blood flow to the kidney could be changed by varying the speed of the pump, and changes in renal perfusion pressure measured by a Statham P23Db transducer connected with the kidney inflow.

In these experiments, in which the stimulus was a change in circulatory conditions of a kidney, integrity of innervation to the stimulated kidney was tested as well as the effectiveness of subsequent denervation. Before denervation pontine stimulation elicited renal vasoconstriction, while after isolating and cutting renal nerves vasoconstriction entirely disappeared.

Analysis of data. Results of all experiments were compared statistically using analysis of variance with double classification (Snedecor & Cochran, 1967).

RESULTS

Stimulation of renal nerves

Changes in arterial pressure and heart rate. The results obtained in animals under chloralose anaesthesia were essentially similar to those obtained under barbiturate and are therefore presented together. These results are shown in Table 1 and records of typical experiments are shown in Fig. 1. Whenever effective, stimulation of afferent renal nerves at all parameters used always produced an increase in systemic arterial pressure and a small but consistent increase in heart rate. On no occasion and with none of the stimulating parameters employed was a depressor effect observed, though a decrease in blood pressure occurred when the central stump of a femoral nerve was stimulated with suitable parameters.

TABLE 1. Means and % changes (± S.E.) of haemodynamic responses to electrical stimulation of afferent renal nerves compared to control values. *P* values refer to differences between means, *n* is number of experimental runs

	n	Control	Response	% changes from control	*P*
Arterial pressure (mmHg)	40	120·2 ± 2·7	139·2 ± 2·9	16·6 ± 1·6	< 0·001
Heart rate (beats/min)	20	205·5 ± 7·1	211·0 ± 7·2	2·7 ± 0·4	< 0·001
Sup. mesenteric bed					
Flow (ml./min)	18	22·0 ± 0·9	15·8 ± 1·2	− 29·1 ± 3·6	< 0·001
Conductance (ml. min^{-1}/mmHg)		0·194 ± 0·046	0·118 ± 0·009	− 39·7 ± 3·6	< 0·001
Iliac bed					
Flow (ml./min)	22	14·2 ± 1·0	11·6 ± 0·8	− 19·0 ± 1·3	< 0·001
Conductance (ml. min^{-1}/mmHg)		0·108 ± 0·009	0·071 ± 0·008	− 34·6 ± 5·9	< 0·001
Contralateral renal bed					
Flow (ml./min)	22	22·5 ± 1·3	23·2 ± 1·3	3·4 ± 1·1	n.s.
Conductance (ml. min^{-1}/mmHg)		0·177 ± 0·008	0·160 ± 0·010	− 10·0 ± 1·6	< 0·001
Ipsilateral renal bed					
Flow (ml./min)	15	26·31 ± 2·54	26·53 ± 2·67	0·9 ± 3·0	n.s.
Conductance (ml. min^{-1}/mmHg)		0·216 ± 0·020	0·192 ± 0·019	− 11·2 ± 3·9	< 0·01

To investigate the possibility that the pressor response observed might have been due to a non-specific response to pain, in three animals frequency of respiration was recorded and possible signs of activation of the sympathetic nervous system (increased pupillary diameter, contraction of the

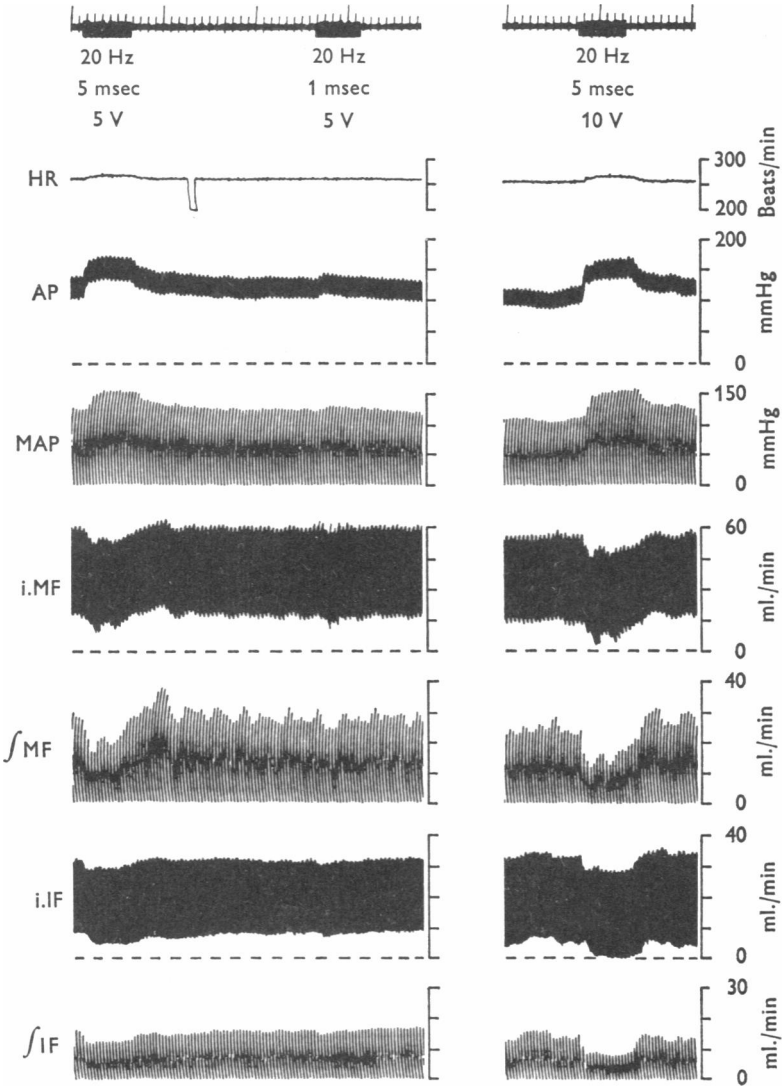

Fig. 1. Stimulation of afferent fibres in the left renal nerve with different parameters as indicated on the top of the figure. From above downward: time in 5 sec and 1 min intervals, and (thick line) stimulus marker; HR, heart rate; AP, aortic pressure; MAP, mean (integrated) aortic pressure; i.MF, instantaneous superior mesenteric blood flow; ∫MF, mean (integrated) superior mesenteric blood flow; i.IF, instantaneous left external iliac blood flow; ∫IF, mean (integrated) left external iliac blood flow.

nictitating membrane, pilo-erection) were monitored. During stimulation of renal nerves eliciting a typical cardiovascular response frequency of respiration did not change and signs of increased sympathetic activity outside the cardiovascular system were never observed. In contrast, in the same animals, stimulation of the cut central end of the femoral nerve with the same parameters elicited increased arterial pressure and heart rate, a greater frequency of respiration, increased pupillary diameter and contraction of the nictitating membrane.

An experiment demonstrating the different responses to stimulation of the renal and femoral nerves is shown in Fig. 2.

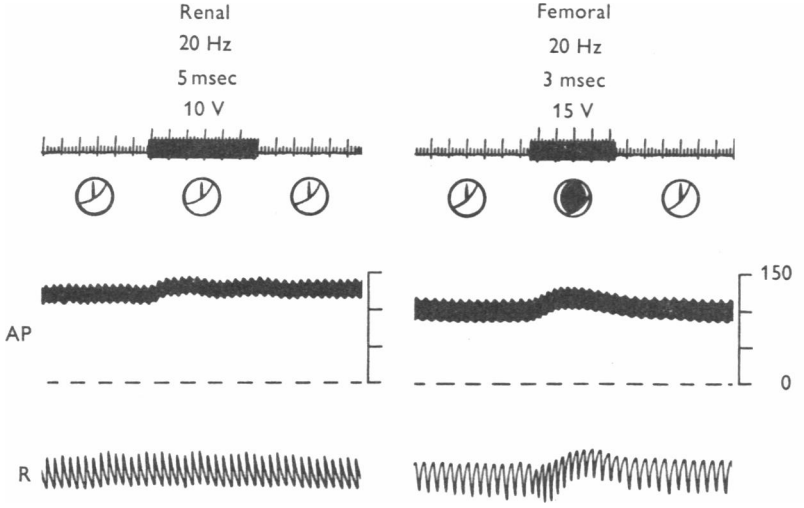

Fig. 2. Stimulation of afferent fibres in the left renal nerve (left) and in the left femoral nerve (right). From above downwards: time in 1 and 5 sec intervals, and (thick line) stimulus marker; sketch of pupillary diameter and nictitating membrane; AP, aortic pressure; R, respiratory movements.

The possibility that the observed response might have been secondary to changes in muscular tone or activity was investigated in six animals by comparing the response to electrical stimulation of renal nerves before and during muscle paralysis. The response was not significantly ($P > 0{\cdot}05$) altered by the administration of gallamine: the increase in arterial pressure was from 129 ± 3 to 156 ± 4 mmHg (21 %) before and from 98 ± 4 to 124 ± 7 mmHg (26 %) after gallamine.

Changes in blood flow in different vascular beds. During stimulation of afferent renal nerves a decrease in blood flow and a conspicuous reduction in vascular conductance was shown to occur in the mesenteric and iliac beds. In contrast blood flow to the contralateral kidney remained

unchanged, but a significant decrease in conductance in the renal bed could be calculated because of the increase in arterial pressure. Similarly blood flow to the ipsilateral denervated kidney did not change and the decrease in local vascular conductance was comparable to that calculated for the innervated kidney. These results are shown in Table 1 and typical records are shown in Fig. 1.

TABLE 2. Plasma renin activity and renin release before and after unilateral stimulation of afferent renal nerves for 5 min. Control and response mean arterial pressures are also shown. *P* values refer to differences between means. $n = 8$ experiments in six cats

	Control	Response	*P*
Mean arterial pressure (mmHg)	126 ± 4·1	144 ± 1·5	< 0·001
Arterial renin activity (AI ng/ml. hr^{-1})	9·6 ± 0·5	10·6 ± 1	n.s.
Ipsilateral kidney			
Venous renin activity (AI ng/ml. hr^{-1})	10·9 ± 0·9	12·8 ± 1·2	< 0·01
Venous arterial difference (AI ng/ml. hr^{-1})	1·4 ± 0·9	2·3 ± 1·0	n.s.
Renin release (AI ng/min)	22 ± 15	36 ± 6 1	n.s.
Contralateral kidney			
Venous renin activity (AI ng/ml. hr^{-1})	13·7 ± 0·9	15·2 ± 1·7	n.s.
Venous arterial difference (AI ng/ml. hr^{-1})	4·1 ± 0·8	4·7 ± 1·0	n.s.
Renin release (AI ng/min)	58 ± 12	61 ± 13	n.s.

Renin release. Stimulation of afferent renal nerves on either side for periods of 5 min was found to increase significantly mean arterial pressure but not to alter significantly the release of renin from the contralateral innervated kidney. More variable changes in renin release from the ipsilateral kidney, which were statistically non-significant, were probably due to incomplete peripheral crushing of the stimulated nerve in two cats. The results on renin release are shown in Table 2.

Manoeuvres to alter renal circulation

Occlusion of renal artery. Complete occlusion of the renal artery on either side in eight animals consistently elicited a moderate increase in systemic arterial pressure. This response was not greater in those experiments like the one illustrated in Fig. 3*A*, in which the contralateral kidney was denervated in order to abolish a possible buffering action exerted by contralateral influences. Furthermore the response was not statistically different after denervation of the manipulated (i.e. ipsilateral) kidney (mean % change 15% before, 16% after denervation). Results are summarized in the first row of Table 3.

Perfusion of the renal artery. In four animals the possible existence of renal vascular receptors involved in the pressor response observed was investigated further by altering experimentally the perfusion pressure to

TABLE 3. Peak changes in systemic mean arterial pressure during manoeuvres to alter renal circulation before and after denervation. *P* values refer to differences between the two preceding columns; *n* is the number of trials. More than one trial was generally performed in each cat. Only ten of fourteen trials of occlusion of renal vein (with venous pressure greater than 30 mmHg) were used for calculating the depressor response: the four trials with no change in arterial pressure were excluded

		Innervated kidney			Denervated kidney			% changes		
	n	Control	Response	*P*	Control	Response	*P*	Innervated	Denervated	*P*
Occlusion of renal artery	8	121·8 ± 9·2	136·8 ± 6·2	< 0·01	106·2 ± 12·2	119·7 ± 11·0	< 0·01	15·0 ± 5·8	16·0 ± 5·8	n.s.
Perfusion of renal artery										
Decreases in perfusion pressure greater than 40 mmHg	9	143·7 ± 4·4	155·8 ± 5·1	< 0·01	108·3 ± 10·2	120·0 ± 11·2	< 0·001	8·5 ± 2·4	10·9 ± 1·6	n.s.
Decreases in perfusion pressure smaller than 40 mmHg	8	148·0 ± 2·0	148·4 ± 2·2	n.s.	120·6 ± 6·4	120·9 ± 6·4	n.s.	0·2 ± 0·2	0·2 ± 0·2	n.s.
Occlusion of renal vein										
Renal vein pressure greater than 30 mmHg	10	118·0 ± 6·7	108·8 ± 5·9	< 0·001	119·6 ± 4·1	108·5 ± 5·5	< 0·001	−7·7 ± 0·7	−9·7 ± 1·7	n.s.
Renal vein pressure smaller than 30 mmHg	9	146·7 ± 6·3	145·0 ± 6·6	n.s.	125·5 ± 4·6	123·9 ± 4·9	n.s.	−1·2 ± 0·9	−1·3 ± 1·0	n.s.

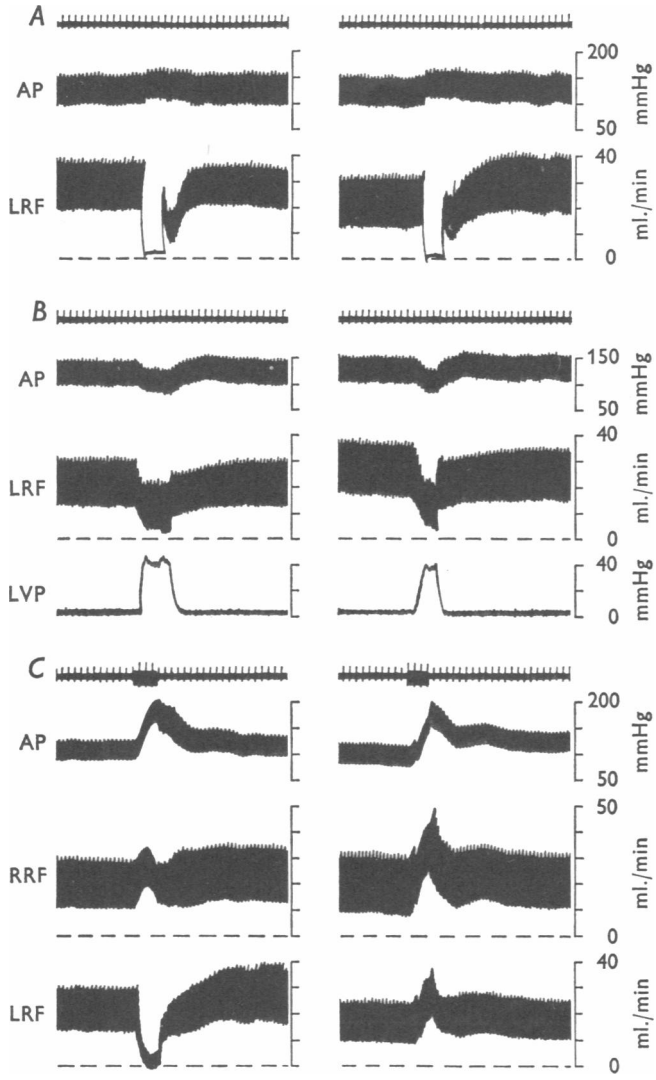

Fig. 3. Occlusion of the left renal artery (*A*), or of the left renal vein (*B*) before (left column) and after denervation of the left kidney (right column). *C*, electrical stimulation of the pontine vasomotor centre (during the period marked by the signal) to show intact innervation of the left kidney (left column) and its denervation (right column). Right kidney was denervated from the beginning of the experiment. In *A*: time in 5 sec intervals; AP, aortic pressure; LRF, left renal blood flow. In *B*: time, AP and LRF as in *A*, LVP, left renal vein pressure. In *C*: time, AP and LRF as in *A*, RRF, right renal blood flow.

the kidney. It was shown that small changes in perfusion pressure and flow (less than 40 mmHg, corresponding to changes in flow of approximately 10 ml./min) did not alter systemic pressure appreciably (Table 3, third row). However, a step change in perfusion pressure greater than 40 mmHg consistently elicited an increase in arterial pressure (mean % change 8%) which was not changed significantly by denervation (10%) (Table 3, second row; and Fig. 4).

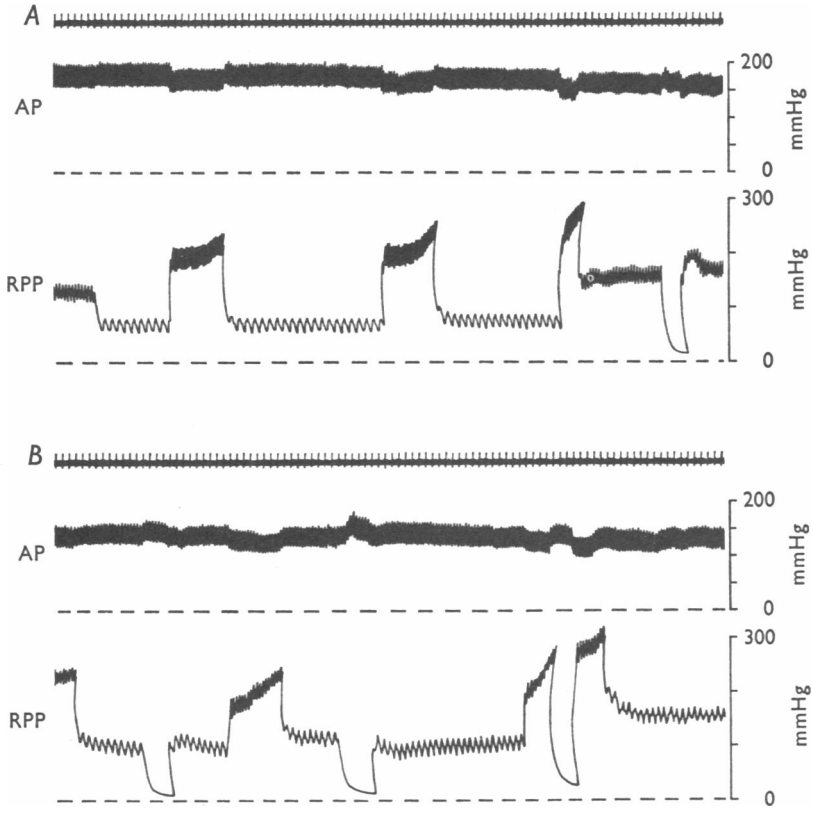

Fig. 4. Effects of changes in renal perfusion pressure (RPP) on aortic pressure (AP). Pump-perfused kidney *in situ*. Upper part (*A*), perfused kidney normally innervated; lower part (*B*), after denervation of perfused kidney. Time in 5 sec intervals.

Occlusion of renal vein. Occlusion of the renal vein on either side was produced in ten animals so as to elicit changes in pressure in the renal vein of different magnitudes. Changes in renal vein pressure less than 30 mmHg did not elicit any significant changes in systemic arterial pressure. However, in most cases (10/14) arterial hypotension was observed when renal vein

pressure was raised above 30 mmHg. Here again, the hypotensive response was not altered by denervation of the contralateral kidney, and survived also ipsilateral renal denervation: arterial pressure decreased by 8% before and by 10% after denervation (Table 3, fourth and fifth rows; Fig. 3*B*).

DISCUSSION

Stimulation of afferent renal nerves consistently elicited an increase in arterial pressure and heart rate which was accompanied by widespread vasoconstriction in both visceral (mesenteric) and musculocutaneous (iliac) beds. A remarkable finding was that although the response was greater with stimuli of longer duration and greater magnitude, results qualitatively similar were observed with pulse durations as short as 0·05 msec, voltages as low as 3 V and frequencies in the range of 5–50 Hz, and under no circumstances of stimulation was a decrease in arterial pressure observed, though a depressor effect could be elicited in our animals by suitable stimulation of a somatic afferent nerve. The characteristic haemodynamic response observed in these experiments was unexpected in view of the results of previous workers describing either a decrease (Ueda *et al.* 1967, in dogs; Aars & Akre, 1970, in rabbits) or no change (Aström & Crafoord, 1968, in cats) in systemic arterial pressure. Although the species in which hypotension was elicited by stimulation were different from that used in these experiments it does not appear probable that a species difference may account for the different results. An alternative explanation may be that different anaesthetics were used which may have produced preparations with different responsiveness. This possibility is not unlikely in view of the demonstration that opposite changes in arterial pressure may be elicited by stimulation of central sites under different anaesthetic agents (Calaresu & Mogenson, 1972). It has to be remarked, however, that we observed a pressor response both in cats under barbiturate anaesthesia and in cats under chloralose.

The pressor response is very unlikely to have been due to liberation of catecholamines as the latency of the response after application of the stimulus was of 2–5 sec and the pressure returned to control levels 10–40 sec after the stimulus was discontinued. The possibility that the response might have been secondary to muscular activity can also be excluded as the increase in pressure was still present in paralysed animals.

It is remarkable that, although the vasoconstrictor response was widespread involving beds with different functional significance, it appeared not to include the vascular bed of the organ from which the afferent stimuli originate, i.e. the kidney. Indeed, no changes in renal blood flow were observed and the small decrease in renal conductance was entirely

due to the rise in perfusion pressure and was identical to the decrease in conductance calculated for the ipsilateral denervated kidney. It is reasonable to assume that it was due to vascular autoregulation.

Lack of involvement of the kidney in this reflex of renal origin is supported by the observation that stimulation of afferent renal nerves did not affect in any significant way renin release from either the contralateral innervated or the ipsilateral denervated kidney. Although our results have failed to demonstrate the existence of a reno-renal reflex, they do not exclude the possibility that it may exist. First of all, if autoregulation can easily account for the resistance changes in the denervated kidney, similar changes might be produced in the innervated kidney by neurally mediated constriction. Secondly, our experimental approach which required crushing of the renal nerves distal to the stimulating electrodes obviously eliminated the possibility of investigating a reflex whose efferent limb is ipsilateral. Finally, the change in renin release induced by activation of afferent renal fibres may be minor and would be demonstrable only in a larger series of animals, and possibly after preventing the renin-suppressive influence of the reflex increase in renal perfusion pressure.

If it is accepted that electrical stimulation of presumably all afferent fibres in the renal nerves in animals under the experimental conditions used in this study elicits an increase in arterial pressure it is tempting to speculate on the nature and location of renal receptors which mediate this reflex response and their functional significance. Of all the possible renal receptors (mechanoreceptors, thermoreceptors, chemoreceptors, osmoreceptors and pain receptors) renal mechanoreceptors have been suggested because of the existence of afferent fibres whose frequency of firing is augmented by increases in renal vein pressure and in ureteral pressure (Aström & Crafoord, 1967; Beacham & Kunze, 1969), by changes in pressure in the interlobar arteries (Niijima, 1971), by stimulation of the renal parenchyma with a mechanical probe (Niijima, 1975), and by increased pressure within the kidney (Kady, 1974). From these results it seems reasonable to assume that there are probably different sorts of mechanoreceptors located in different parts of the vasculature and parenchyma of the kidney, but that the precise function of these receptors has not been studied with selective stimuli.

We have tried to activate some of these presumptive receptors by various manoeuvres altering renal circulation and to study whether these alterations could cause reflex changes in the systemic circulation similar to those observed during electrical stimulation of afferent renal nerves. However, both the increase in arterial pressure elicited by occlusion of one renal artery or by reducing pressure and flow to a perfused kidney, and the systemic hypotension caused by occlusion of a renal vein, were not affected

by denervation. This clearly demonstrates that both responses are not of reflex origin, and probably are mechanical phenomena. Arterial occlusion is likely to cause a sudden increase in peripheral resistance, and the hypotension elicited by venous occlusion can be accounted for by a decrease in venous return and a resultant reduction in cardiac output.

Systemic arterial hypotension during occlusion of the renal vein is worth further comment, as this response had already been described (Ueda *et al.* 1967; Beacham & Kunze, 1969; Kady, 1974), and interpreted as a reflex response (Beacham & Kunze, 1969). Evidence supporting this interpretation was quite indirect, being based on the observation that a similar hypotension is elicited by increasing ureteral pressure in the innervated but not in the denervated kidney. Our experiments clearly show that this is not the case for the effects of increased venous pressure. As we carefully checked the integrity of the innervation of the stimulated kidney in control trials by measuring the renal vasomotor effects of brain stem stimulation (see Fig. 3*C*), we can safely rule out that renal mechanoreceptors influenced by the manoeuvres we performed can induce reflex changes in systemic circulation under the conditions of our experiments.

The possibility that the reflex pressor response we observed during stimulation of afferent renal nerves might have been due to activation of pain fibres has now to be carefully considered. Afferent fibres of the A-delta and C types, the fibres traditionally associated with pain sensation, have been described in renal nerves (Niijima, 1975), although the presence of these fibres does not necessarily imply that pain receptors exist within the kidney. Our results, in fact, would appear to suggest that pain fibres are unlikely to originate from the kidney as shown by the absence in our experiments of the characteristic autonomic and respiratory responses usually associated with painful stimulation (Woodworth & Sherrington, 1904); these responses, however, were easily observed during stimulation of the central end of a somatic nerve (see Fig. 2). An alternative explanation is that activation of pain fibres in the renal nerves may have been masked by the simultaneous activation of other sensory fibres. Finally, we cannot rule out that electrical stimulation of afferent renal nerves might have simulated activation of chemo-, osmo- or thermoreceptors possibly present in the kidney, but in the absence of any direct demonstration that these receptors exist it seems fruitless to engage in any further speculation.

In summary, these experiments have demonstrated the existence of a renal neural input to the central nervous system which, when activated under the experimental conditions described, brings about an increase in systemic arterial pressure, which in turn appears to be due to widespread vasoconstriction. This response is unlikely to be mediated by renal vascular mechanoreceptors or by pain fibres, but is likely to be a

homoeostatic response concerned with the control of arterial pressure triggered by undetermined physiological or pathological signals from the kidney. In addition, it does not appear likely that there is a reno-renal reflex regulating renin release as stimulation of afferent renal nerves did not significantly affect renin release from the contralateral innervated kidney.

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