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THE EFFECT OF DISTENSION OF THE URINARY BLADDER ON ACTIVITY IN EFFERENT RENAL FIBRES IN ANAESTHETIZED DOGS

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SUMMARY

1. To test whether distension of the urinary bladder causes a consistent change in activity in efferent renal nerve fibres, bladder distension was performed in anaesthetized dogs. The carotid sinuses were vascularly isolated and perfused with blood at constant flow. Both ureters were cannulated and the urinary bladder was distended with warm Ringer solution at a steady intravesical pressure.

2. In a first series of experiments all twenty-six renal nerve fibres in eleven dogs which responded to changes in carotid sinus pressure and changes in the nature of the blood perfusing the carotid sinuses also responded to distension of the bladder.

3. In a second series of experiments, graded bladder distension over a range of pressure of 0–9·2 kPa led to a graded increase in activity in thirteen efferent renal fibres in six dogs. The magnitude of the renal nerve response was greater at low than at high carotid sinus pressure. Over a range of carotid sinus pressure of 9–30 kPa, the greatest renal nerve activity was obtained at the lower end of this range in the presence of bladder distension.

4. Thus distension of the urinary bladder resulted in the response of a consistent increase in efferent renal nerve activity, which could be graded according to intravesical pressure. The magnitude of the responses to bladder distension was affected by carotid sinus pressure.

INTRODUCTION

Distension of the urinary bladder has been shown to result in the reflexes of increases in heart rate and arterial blood pressure, a reduction in renal volume attributed to vasoconstriction and a decrease in renal blood flow (Guttmann & Whitteridge, 1947; Mukherjee, 1957*a*, *b*; Taylor, 1968; Tsuchida & Kumagai, 1978; Daly & Wood, 1982; Hassan, Hicks, Walters & Mary, 1987*a*). Reductions in renal blood flow and vasoconstriction may be achieved by activation of the renal nerves (e.g. Donald & Shepherd, 1980). Recently, however, in multifibre preparations in cats, bladder distension has been reported to result in both increases and decreases

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in efferent renal nerve activity, depending on the pressure used to distend the bladder (Weaver, 1985).

In previous studies, it has been shown that renal nerve fibres which responded to activation of baroreceptors and chemoreceptors in the carotid region also responded to stimulation of atrial receptors (Linden, Mary & Weatherill, 1981). However, changes in the level of activation of carotid baroreceptors did not affect the decrease in renal nerve activity in response to stimulation of atrial receptors (Linden *et al.* 1981).

The primary objective of the present investigation was to find out whether distension of the urinary bladder results in a consistent change in activity of efferent renal nerve fibres. In addition, the investigation examined whether this renal nerve response was affected by grading the pressure used to distend the bladder or the pressure in the perfused carotid sinuses. The methods used in this study have previously been demonstrated to the Physiological Society (Drinkhill, Hassan, Hicks, Mary, Ramadan & Vacca, 1987).

METHODS

Seventeen mongrel dogs of either sex, which weighed between 17·5 and 29·2 kg, were given a subcutaneous injection of morphine sulphate (0·5 mg kg^{-1}). Approximately 1 h later the animals were anaesthetized with an I.V. injection of α-chloralose (Vickers Laboratories Ltd, Yorks; 100 mg kg^{-1}) and artificially ventilated with oxygen-enriched air (approximately 40%) as described by Linden & Mary (1983). Throughout the subsequent surgical procedures, anaesthesia was maintained by a continuous I.V. infusion (0·5–1 mg kg^{-1} min^{-1}) of a 1% solution of chloralose in saline. During recording of activity in efferent renal nerve fibres, in some animals, suxamethonium chloride was administered (BDH Ltd, Dorset; 0·5 mg kg^{-1}). Cannulation of the femoral vessels was performed and the pressures in the right femoral artery, urinary bladder and carotid sinuses were recorded and monitored together with the electrocardiogram (ECG). The oesophageal temperature and the pH, P_{CO_2} and P_{O_2} of arterial blood were monitored and maintained within normal limits using techniques described by Linden & Mary (1983).

Both carotid sinuses were vascularly isolated and perfused with either arterial of venous blood from the same animal using a constant-flow roller pump, as described by Linden *et al.* (1981). Coagulation of the circulating blood was prevented by I.V injection of heparin (CP Pharmaceuticals Ltd, Wrexham; initial dose, 500 i.u. kg^{-1}; subsequent dose, 50 i.u. kg^{-1} every 30 min).

The urinary bladder and initially the left kidney were approached through a mid-line abdominal incision; the ureters were ligated proximal to their orifice in the bladder. Both ureters were cannulated proximal to the point of ligation to ensure free urine flow from the kidneys. A wide-bore double-lumen cannula was introduced into the urinary bladder retropubically through the urethra, to allow distension of the bladder with warm Ringer solution, and measurement of pressure inside it using a pressure transducer. The Ringer solution was kept in a reservoir maintained at 38 °C in a thermostatically controlled water-bath. For distension of the bladder this solution was driven by compressed air and the pressure exerted was measured using a mercury manometer connected to the reservoir and controlled using a Starling resistance fitted to the compressed air supply. This enabled the pressure inside the urinary bladder to be set to desired values and maintained at steady levels in all experiments.

Renal nerves running along the surface of the renal artery, close to the renal hilum, were identified. A binocular microscope was used to dissect fine strands from these nerves on a black Bakelite platform under warm liquid paraffin maintained at 38 °C by overhead heating lamps. Efferent activity from single or few-fibre preparations of the renal nerves was obtained using silver electrodes, a preamplifier (NL 104, Digitimer, Welwyn Garden City, London) and filter (NL 125, Digitimer). The action potentials were displayed on a digital storage oscilloscope (054020, Gould Ltd, Hainault, Essex) and passed on to a UV recorder (2112, SE Laboratories Ltd). In addition, the activity occurring in 5 s intervals was monitored using a spike processor (D130, Digitimer Ltd)

attached to a chart recorder (Phillips PM8041, Pye Unicam, Cambridge) and on-line computer (BBC, Model B, Acorn). At all times the activity was counted from single units which were assessed as previously reported (Linden *et al.* 1981). Renal nerve activity was displayed as described above along with pressures in the carotid sinuses, trachea, urinary bladder and the right femoral artery, and the ECG and was also recorded on electromagnetic tape (Teac R.71).

Experimental protocol

In all dogs, after a steady state had been attained with respect to heart rate, mean arterial blood pressure and anaesthetic dose, initially the following responses were obtained: an increase in heart rate and blood pressure to bladder distension, a decrease in heart rate and blood pressure to an increase in carotid sinus pressure, and a decrease in heart rate and an increase in blood pressure to perfusion of the carotid sinuses with venous blood. The activity in the efferent renal nerve fibres was then studied following the prevention of the reflex changes in heart rate and blood pressure by the administration of atropine sulphate (BDH Ltd, Dorset; 0·5 mg kg^{-1}) and bretylium tosylate (Wellcome Foundation, London; 10 mg kg^{-1}).

In a first series of experiments efferent renal nerve fibres which either did or did not respond to changes in carotid sinus pressure were identified. The fibres which responded were examined during each of five experimental procedures: an increase in carotid sinus pressure for 90 s, distension of the urinary bladder with warm Ringer solution for 90 s, perfusion of the carotid sinuses with venous blood for 120 s, another distension of the bladder for 90 s and finally a further increase in carotid sinus pressure for 90 s. The same protocol was used in studying some of the efferent renal nerve fibres which did not respond to an increase in carotid sinus pressure. The intervention was kept constant throughout its time period. The response to each intervention was calculated as the difference between the mean activity in impulses s^{-1} during the test period, which constituted the last 60 s of the intervention, and the average of the mean nerve activity during the two 60 s control periods immediately before and after the intervention period. As in previous studies (Hassan *et al.* 1987*a*, *b*), this protocol ensured a steady state of the measured variables during the test period of the intervention. The responses of heart rate and arterial blood pressure to the interventions of bladder distension, changes in carotid sinus pressure or nature of the perfusing blood were calculated in a similar manner.

In a second series of experiments the effect of renal nerve activity of increasing bladder pressure in four steps each of about 2 kPa was examined. Each of the steps lasted 90 s, their sequence was in a random order and they were repeated at two levels of carotid sinus pressure. Measurements were made during the last 60 s of each step. In the same animals, the effect on renal nerve activity of increasing carotid sinus pressure in steps of about 5 kPa with and without bladder distension was examined.

Student's *t* test for paired data was used for statistical analysis and group data are presented as mean ± S.E.M.

RESULTS

In seventeen dogs, recordings commenced approximately 4 h after the initial dose of anaesthetic had been given. The mean initial heart rate was 133·9 beats min^{-1} (range 78–200) and the mean arterial blood pressure was 19·2 kPa (range 13·2–25·2). The mean pH, P_{CO_2} and P_{O_2} of arterial blood respectively were 7·387 (range 7·346–7·44), 5·18 kPa (range 4·39–5·75) and 31·22 kPa (range 25·5–37·6). The haematocrit was 43·6% (mean; range 34–56).

In the seventeen dogs, the urinary bladder was distended to a mean intravesical pressure of 6·3 kPa (range 4·3–8·6). Pressure was increased at a rate of 1·0 kPa s^{-1} (range 0·2–2·0) so that this level of pressure was attained during the initial 5·9 s (mean; range 3–20) of the intervention. This distension resulted in an increase in arterial blood pressure and in heart rate; these increases (Fig. 1) were statistically significant ($P < 0·0005$). Incrementing the pressure in the carotid sinuses from a mean of 13·2 kPa (range 9·6–16·8) to a mean of 29·3 kPa (range 25·5–33·0) caused

significant ($P < 0.0005$) decreases in blood pressure and heart rate (Fig. 1). Changing the nature of the blood perfusing the carotid sinuses resulted in significant ($P < 0.0005$) increases and decreases respectively in blood pressure and heart rate (Fig. 1). In the same seventeen dogs, following the administration of bretylium tosylate and atropine sulphate, the above responses to the same levels of stimuli were drastically reduced or abolished (Fig. 1).

In two series of experiments in these seventeen dogs, a total of fifty-two efferent renal nerve fibre preparations were examined, following prevention of changes in heart rate and blood pressure as described above.

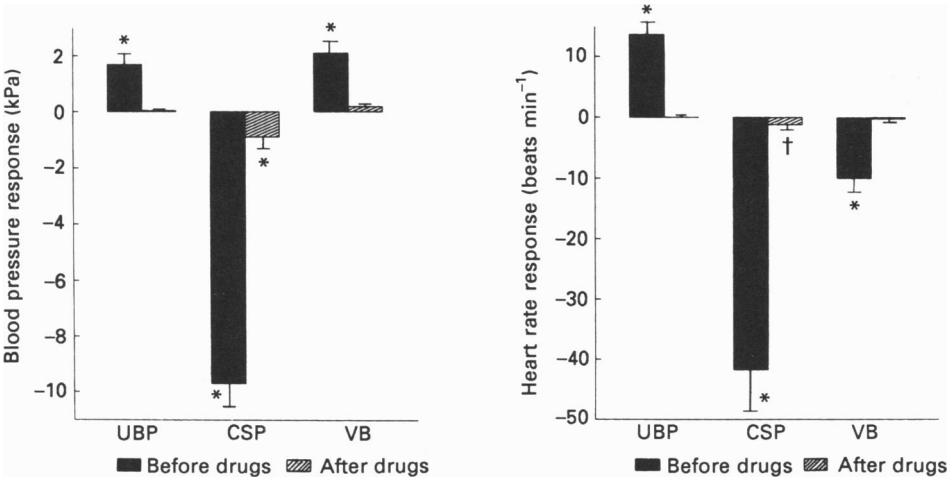

Fig. 1. Histograms showing responses of heart rate (right) and arterial blood pressure (left) to distension of the urinary bladder (UBP), or changes in carotid sinus pressure (CSP) or the nature of blood perfusing the sinuses (VB). The histograms represent the mean values of the responses, and the bars represent S.E.M. Statistically significant responses are labelled by * ($P < 0.0005$) and † ($P < 0.0125$).

First series of experiments

In eleven dogs a systematic search identified two groups of renal nerve fibres. Of a total of thirty-seven fibres, twenty-six responded to changes in carotid sinus pressure and eleven fibres did not respond. The latter group was not considered any further. In these fibres the mean activity during the control period was 1·27 impulses s^{-1} (range 0·20–2·58). This activity was not significantly altered during increases in carotid sinus pressure from a mean of 11·1 to 29·0 kPa. The measured alterations amounted to 0·01 impulses s^{-1} (mean; range −0·23 to 0·31; $P > 0.40$). Similarly no significant effect was found during bladder distension. The measured alteration during bladder distension to a mean pressure of 6·2 kPa (range 4·5–8·5) amounted to 0·03 impulses s^{-1} (mean; range −0·12 to 0·26; $P > 0.20$).

Responsive renal nerve fibres

Each of the twenty-six efferent renal nerve fibres, obtained in eleven dogs, which responded to changes in carotid sinus pressure always responded to distension of the

urinary bladder. An example is shown in Fig. 2. Distension of the bladder resulted in an increase in activity which was unaccompanied by any changes in femoral arterial pressure, carotid sinus pressure and heart rate. An example of the experimental protocol used and of responses obtained in one efferent renal nerve fibre is shown in Fig. 3. An increase in carotid sinus pressure from 12·8 to 29·1 kPa on the

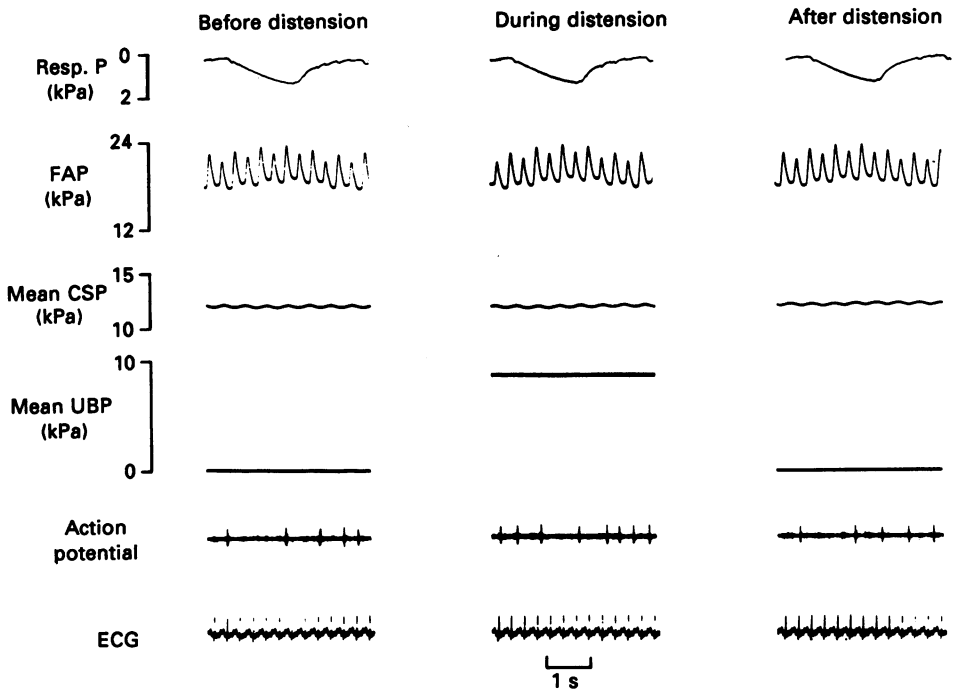

Fig. 2. Example of experimental records taken before, during and after distension of the urinary bladder following the administration of bretylium tosylate and atropine sulphate. From above down are records of tracheal pressure (Resp. P), femoral arterial pressure (FAP), mean carotid sinus pressure (CSP), mean bladder pressure (UBP), activity in an efferent renal nerve fibre (Action potential) and electrocardiogram (ECG). Distension of the urinary bladder, in this example, from 0·0 to 8·8 kPa caused an increase in activity in the efferent renal nerve fibre, and this increase was unaccompanied by changes in arterial blood pressure, carotid sinus pressure or in heart rate.

first occasion, and from 12·5 to 29·4 kPa on the second occasion resulted in a decrease in activity, whilst perfusion of the carotid sinuses with venous blood resulted in an increase in activity. Distension of the urinary bladder to 6·5 kPa on the two occasions resulted in an increase in activity. All these increases and decreases in activity were reversed by the removal of the stimulus.

The results in each of the twenty-six nerve fibres are shown in Fig. 4. A change in carotid sinus pressure from a mean of 11·1 kPa (range 7·6–13·5) to a mean of 28·9 kPa (range 23·3–32·2) resulted in a decrease in nerve activity in every fibre; in the whole group this response amounted to a mean of 0·99 impulses s^{-1} (range 0·13–2·53). In each of these nerve preparations distension of the urinary bladder to a mean intravesical pressure of 6·6 kPa (range 4·1–8·7) resulted in an increase in renal nerve

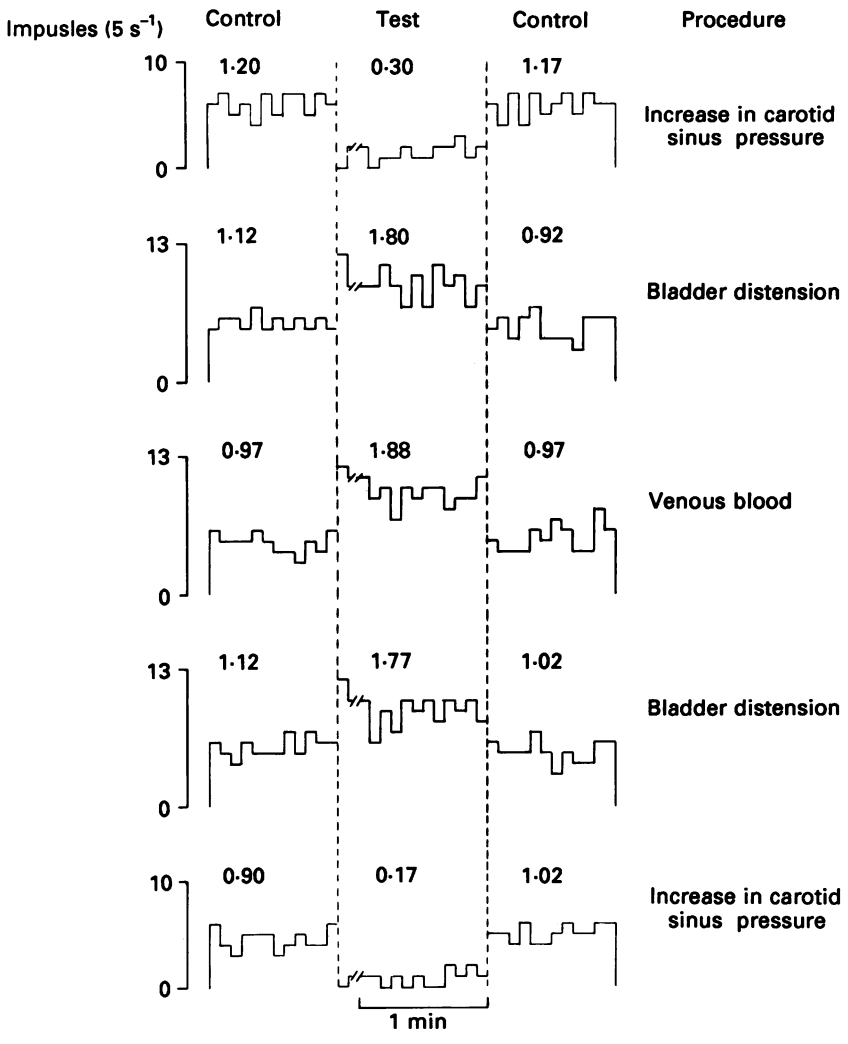

Fig. 3. Example of experimental protocol from the first series of experiments, showing results of changes in activity in an efferent renal nerve fibre during five procedures. On the ordinate is nerve activity measured over successive 5 s intervals and on the abscissa, the period of the experiment. The numbers refer to the mean nerve activity in impulses s^{-1} over 1 min of steady state.

activity; group responses were 1·12 impulses s^{-1} (mean; range 0·18–3·40). Perfusing the carotid sinuses with venous blood always resulted in an increase in activity; this response amounted to 1·23 impulses s^{-1} (mean; range 0·14–5·38). All these responses were statistically significant ($P < 0·0005$).

Second series of experiments

This second series examined the effects of grading intravesical and carotid sinus pressures. In six dogs, the activity in thirteen efferent renal nerve fibres was

measured before bladder distension and at four levels of distending intravesical pressure (Fig. 5); this procedure was completed at two levels of carotid sinus pressure, low and high, respectively averaging 9·9 and 25·4 kPa. The lowest carotid perfusion pressure was 9 kPa; pressures below 8 kPa are reported to excite carotid chemoreceptors (Biscoe, Bradley & Purves, 1970). At high and low carotid sinus

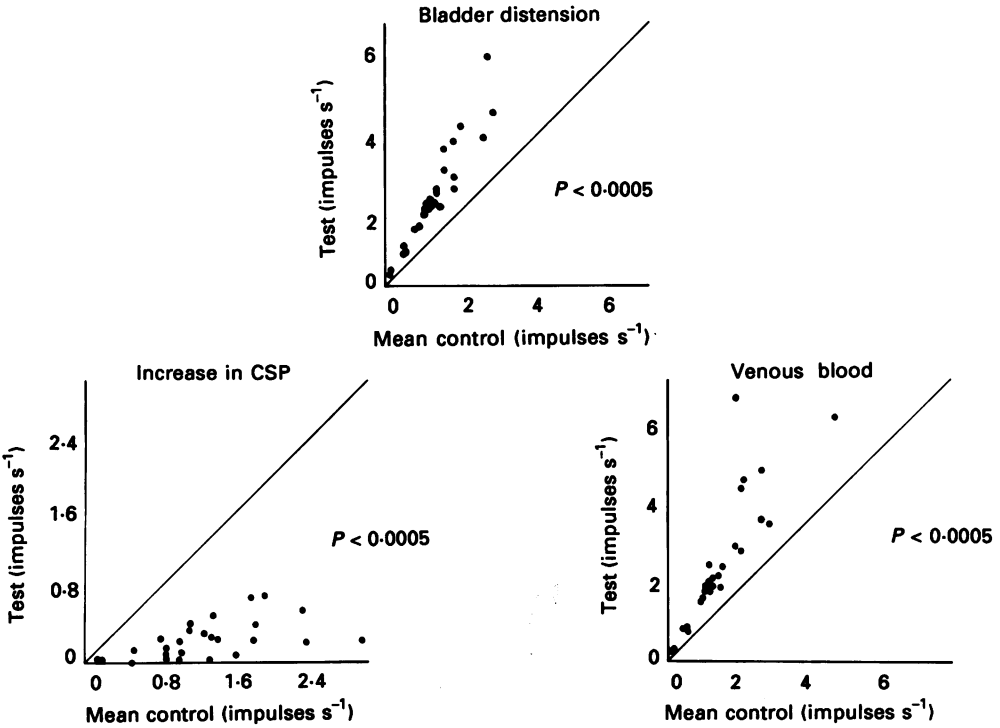

Fig. 4. Graphs showing the results obtained in each of the twenty-six efferent renal nerve fibres during distension of the urinary bladder (Bladder distension), increases in carotid sinus pressure (Increase in CSP) and perfusion of the carotid sinuses with venous blood (Venous blood). On the ordinate is nerve activity during the final 1 min of the test period and on the abscissa is the mean nerve activity during the two 1 min steady-state control periods; the mean of two responses is given for bladder distension and increase in CSP.

pressure, bladder distension resulted in increases in renal nerve activity which was graded according to the distending pressure (Fig. 5). At high carotid sinus pressure, the increases in nerve activity in response to increments in intravesical pressure attained statistical significance during the last three steps of bladder distension (at least $P < 0·05$). At low carotid sinus pressure, the increases in nerve activity mainly occurred during the first three steps of distension (see Fig. 5). With bladder distension at a mean of 8·1 kPa the difference in renal nerve activity between the two levels of carotid sinus pressure was $0·64 \pm 0·27$ impulses s^{-1} (mean $\pm$ S.E.M.), and was significantly greater ($P < 0·05$) than that of $0·38 \pm 0·10$ impulses s^{-1} (mean $\pm$ S.E.M.) obtained without bladder distension. The highest renal nerve activity could be

obtained only during the combination of low carotid sinus pressure and high intravesical pressure (Fig. 5). This suggested an influence of carotid sinus pressure on the effects of bladder distension. This suggestion was tested in fifteen efferent renal nerve fibres obtained in the same six dogs, which included the thirteen nerve fibres just described.

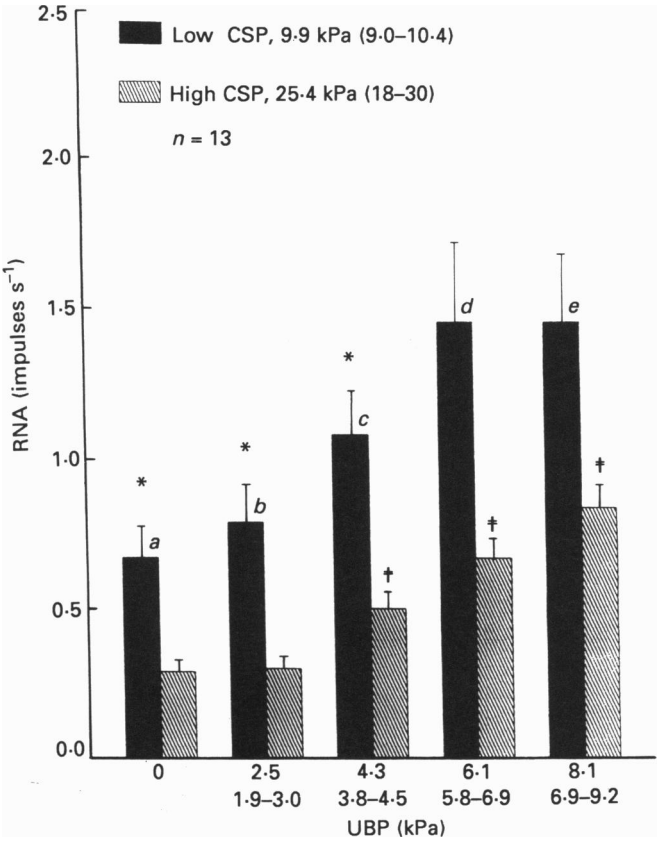

Fig. 5. The effect of high and low carotid sinus pressure (CSP), on the relation between renal nerve activity (RNA) and four step increases (*b–e*) in urinary bladder pressure (UBP) over the undistended state (*a*); UBP is shown as mean and range. The height of the columns depicts mean values, and S.E.M. is indicated by the bars. RNA was always greater at low than at high CSP (at least $P < 0{\cdot}0025$). * Indicates statistically significant difference in RNA at low CSP between *e*, the highest UBP, and the other steps (at least $P < 0{\cdot}025$). ‡ Indicates statistically significant difference in RNA at high CSP between *b* and other steps.

In a previous study which involved the relation between renal nerve activity and carotid sinus pressure over a range of 5–25 kPa, a 50 % reduction in activity occurred when the pressure was increased to 12 kPa (Linden *et al.* 1981). In the fifteen nerve fibres of the present investigation, the responses to bladder distension, obtained in the manner of the first series of experiments, were examined at levels of carotid sinus pressure of less than 12 kPa and at higher levels (Table 1). Lowering carotid sinus

pressure resulted in significant increases in control activity and in its response to bladder distension. To allow for the changes in control activity, the responses were normalized and expressed as a percentage of the highest control activity obtained at low carotid sinus pressure. These normalized responses were significantly greater at low levels of carotid sinus pressure than at high levels (Table 1). Similar results were

TABLE 1. Comparison of the increases in renal nerve activity in response to distension of the urinary bladder at levels of carotid sinus pressure less and greater than 12 kPa. The increases are expressed as absolute values in the third line and as normalized values (see text) in the fourth line

	Low CSP	High CSP	Difference	*P*
CSP (kPa)	9·9 ± 0·14 (8·1 – 10·9)	23·3 ± 1·6 (19·5 – 30·0)		
Mean control RNA (impulses s^{-1})	0·72 ± 0·08 (0·18 – 1·28)	0·11 ± 0·02 (0·03 – 0·30)	0·608 ± 0·07 (0·15 – 1·16)	< 0·0005
Increase (impulses s^{-1})	0·45 ± 0·09 (0·08 – 1·27)	0·14 ± 0·03 (0·04 – 0·36)	0·305 ± 0·08 (0·03 – 1·07)	< 0·0005
Increase (% normalized)	59·00 ± 8·80 (20·8 – 129·6)	20·70 ± 3·04 (5·00 – 44·4)	38·30 ± 8·00 (4·10 – 96·4)	< 0·0005

The values shown are mean ± S.E.M. (range). CSP, carotid sinus pressure; RNA, renal nerve activity. Number of fibres = 15.

obtained using normalization and expression of responses as percentage of the lowest control activity obtained at high carotid sinus pressure. Therefore, whilst bladder distension always resulted in the response of an increase in activity in the renal nerves, the magnitude of this response was greater at low than at high levels of carotid sinus pressure.

Finally, the same fifteen fibres were examined at five levels of carotid sinus pressure in the absence and presence of bladder distension (Fig. 6). There were significant decreases in renal nerve activity accompanying the increments in carotid sinus pressure, which occurred with or without bladder distension (at least $P < 0·05$). As shown in Fig. 6, the renal activity was always greater with bladder distension than without. At a mean carotid sinus pressure of 9·9 kPa, the difference in renal nerve activity attributed to bladder distension was 0·357 ± 0·084 impulses s^{-1} (mean ± S.E.M.), and was significantly greater ($P < 0·01$) than that of 0·02 ± 0·005 impulses s^{-1} (mean ± S.E.M.) obtained at a mean carotid sinus pressure of 29·5 kPa. The highest level of renal nerve activity was attained only during the combination of low carotid sinus pressure and bladder distension.

DISCUSSION

In the present study, efferent renal activity was examined in single nerve fibres. The bladder was distended to an intravesical pressure which can be kept constant to enable measurements in the steady state. The test period always began 30 s after attaining a steady intravesical pressure. The results have shown, in each of forty-one nerve fibres examined, that distension of the urinary bladder resulted in a consistent increase in activity, regardless of the value of distension pressure which was up to

9·2 kPa. The observed changes in renal nerve activity were directly attributable to bladder distension, since changes in haemodynamic variables with their modifying effects were prevented. The increase in activity could always be reversed by the release of bladder distension, and the level of renal nerve activity could be graded according to intravesical pressure. The levels of pressure used to distend the bladder

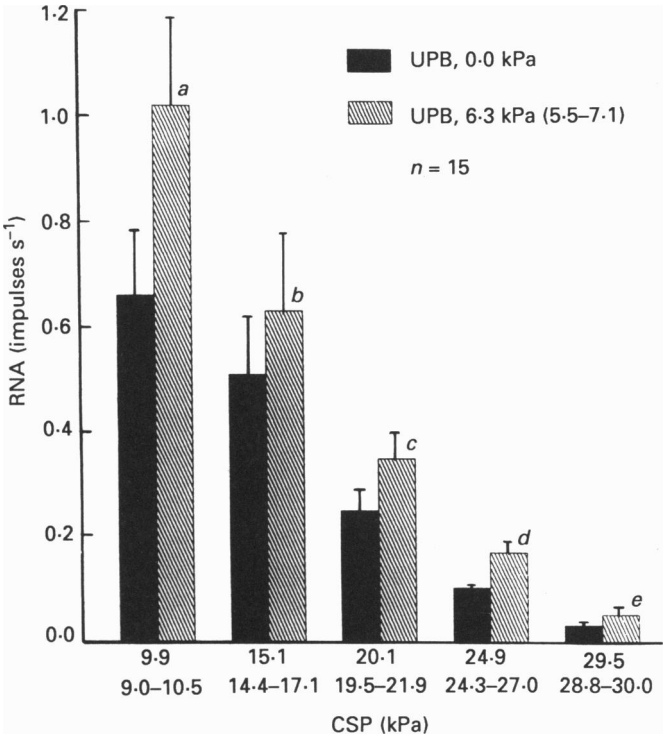

Fig. 6. Histograms showing the effect of the presence and absence of bladder distension (hatched and filled columns respectively), on the relation between renal nerve activity (RNA) and five step levels (*a–e*) of carotid sinus pressure (CSP); CSP is shown as mean and range. The height of the columns depicts mean values, and S.E.M. is indicated by the bars. RNA was always greater with bladder distension than without (at least $P < 0{\cdot}0025$). RNA with bladder distension in *a*, at the lowest CSP, was significantly greater than in *b–e* (at least $P < 0{\cdot}05$).

did not exceed 9·2 kPa; pressure higher than 10·5 kPa has been recorded to damage the bladder walls and impair its responses (Mukherjee, 1957*b*; Prabhakar, Nishith & Saxena, 1973). The levels include those previously reported in man under physiological conditions (Denny-Brown & Robertson, 1933; Tanagho & Miller, 1970; Walter, Olesen, Nordling & Hald, 1979) of spontaneously filled bladder at 1·8–2·5 kPa, and at micturition at 2·3–10·6 kPa.

The present results have also shown convergence of the efferent effects on the same renal nerve fibres of stimulating baroreceptors and chemoreceptors in the carotid region and of distension of the urinary bladder. This finding was established by measurement of activity in single efferent renal nerve fibres, with the prevention of

changes in heart rate and arterial blood pressure, during changes in pressure in the isolated perfused carotid sinuses and distension of the urinary bladder.

The second series showed that the magnitude of responses in the renal nerves to distension of the urinary bladder was significantly greater at low than at high carotid sinus pressure, and was previously reported by Linden *et al.* (1981). In addition, the effect of changes in carotid sinus pressure on the relation between intravesical pressure and renal nerve activity was examined as was the effect of changing intravesical pressure on the relation between carotid sinus pressure and renal nerve activity. The results have shown that the highest renal nerve activity could be obtained during the combination of bladder distension and low carotid sinus pressure.

The above results have important implications. The previously reported effect of bladder distension on the renal nerves has been controversial. For instance, Mukherjee (1957*a*, *b*) reported decreases in renal size in response to distension of the urinary bladder in anaesthetized cats. More recently, bladder distension in anaesthetized dogs has been shown to result in a reduction in renal blood flow and an increase in plasma renin activity (Tsuchida & Kumagai, 1978). Also, Prabhakar, Nishith & Saxena (1973) and Prabhakar & Marek (1979) in anaesthetized dogs, reported an increase in the level of angiotensin in the blood in response to bladder distension. These findings were attributed in part to the renal nerves, implying an increase in nerve activity in response to bladder distension. This interpretation is consistent with several studies on the effect of stimulation of renal nerves and reflex effects of changing arterial blood pressure which indicated that increases in plasma renin activity and decreases in renal blood flow were associated with activation of the renal nerves (e.g. Kirchheim & Gross, 1978; Gross & Kirchheim, 1980; Donald & Shephard, 1980; Di Bona, 1982; Funke, Prabhakar, Hertle, Runkel & Dahlheim, (1982). In contrast to these reports, Weaver (1985) found that bladder distension in anaesthetized cats resulted in a decrease or increase in efferent renal nerve activity recorded from multifibre preparations, depending on the level of distending pressure. A consistent increase in activity was obtained only at high distension pressure greater than 12 kPa. In the present study, using recordings from single nerve fibres, bladder distension was shown consistently to result in the response of an increase in renal nerve activity.

The present finding of convergence between the effects on efferent sympathetic fibres of activating receptors in the carotid region and distension of the bladder assumes further relevance. The effects of stimulating receptors in the atria and in the carotid region have also been shown to converge on to the same efferent renal nerves (Linden *et al.* 1981). However, in that case activation of the carotid receptors did not affect the response of the renal nerves to stimulation of the atrial receptors. In the present study, and unlike the situation with respect to the atrial receptors, activation of carotid baroreceptors was shown to affect the magnitude of the increase in renal nerve activity to distension of the urinary bladder. In particular, decreases in carotid sinus pressure were shown to enhance this renal nerve response.

There are further implications regarding the effect of the carotid baroreceptors. According to the definition of Kirchheim (1976) and Korner (1979), the results of the second series may be described as showing interaction between the responses to

bladder distension and to changes in carotid sinus pressure in the same renal nerve fibres. Interaction is said to occur when the net efferent response to combined stimuli is different from the algebraic sum of the responses to each. In the present study, the responses to bladder distension at a low carotid sinus pressure were three times greater than those at high carotid sinus pressure. Also, examination of renal nerve activity against a background of changing intravesical pressure of carotid sinus pressure showed that the greatest activity could be obtained during the combination of bladder distension and low carotid sinus pressure. For instance, the difference in renal nerve activity attributed to changing carotid sinus pressure from a mean of 9·9 kPa to 25·4 kPa during bladder distension, was about 1·6 times greater than that obtained without bladder distension. Conversely, the differences in renal nerve activity attributed to bladder distension by raising intravesical pressure to a mean of 6·3 kPa at low carotid sinus pressure, was about 18 times greater than that obtained at high carotid sinus pressure. It is notable that the differences in the magnitude of the response could be obtained despite higher levels of renal nerve activity at low than at high carotid sinus pressure. Any explanation for such differences in responses is not consistent with their attenuation by high values of activity on the stimulus–response curve (Heistad, Abboud & Ballard, 1971).

In conclusion, the present study on single efferent renal nerve fibres has shown consistent increases in nerve activity in response to distension of the urinary bladder. The efferent effects of bladder distension or stimulation of carotid baroreceptors or chemoreceptors were shown to converge on the same renal nerve fibre. The magnitude of this increase in activity could be graded according to the pressure distending the bladder, and was greater at low than at high carotid sinus pressure.

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