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ORIGINALS

The Effect of Autonomic Drugs on Ureteric Peristalsis: A Canine in vivo Study

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Summary. An in vivo canine model was developed in which the renal pelvis was perfused by a cannula nephrostomy and ureteric activity assessed by monitoring bolus volume and interperistaltic interval. The effect of autonomic drugs showed that the ureter contained α-adrenergic receptors which on stimulation caused an increase in ureteric contraction rate and a decrease in bolus volume. With β-adrenergic receptor stimulation, there was complete inhibition of ureteric peristalsis for a variable period and evidence is presented that the β-adrenergic receptors may be β1 rather than β2. Cholinergic stimulation of ureteric rate was observed, but seemed to be mediated indirectly via α-adrenergic receptors. No significant change was seen in ureteric activity with adrenergic blocking agents alone, suggesting that the physiological importance of these receptors in normal activity is questionable.

Key words: Autonomic receptor, Bolus volume, Interperistaltic interval.

INTRODUCTION

The presence of adrenergic receptors (α stimulatory and β inhibitory) have been demonstrated in the ureter (14, 15, 19, 23) and functional innervation has been shown to be present for the α-adrenergic receptors in in vitro ureteric (24) and caliceal (13) preparations. Cholinergic agents have been shown to stimulate peristalsis (2, 8), but Rose and Gillenwater found that this was secondary to catecholamine release (19). Although the ureter can function independently of nerve supply after transplantation (16), denervation (28) and reversal of ureteric segments (26, 27), the autonomic adrenergic nerve supply is thought to have a modulating effect (25). However, this has not yet been clearly demonstrated.

Most in vivo methods used previously to investigate the effects of drugs on ureteric activity have been relatively invasive. Direct cannulation and perfusion of the ureter (18, 20), although separating the indirect effects of urine flow from the direct effect of the drug on peristalsis, excluded the site of the normal pacemaker in the pelvis or calices (4, 6, 7). Measurement of intraluminal pressure with a fluid-filled catheter or other measuring device has been widely used for many years, but Weinberg (21, 22) and Dale et al. (5) clearly demonstrated the problems associated with intraluminal devices. It was found that the size of an intraureteric catheter influenced the amplitude of both the basal and contractile pressures and the presence of a catheter altered the interperistaltic interval which normally had a modal distribution. More recently, less invasive methods have been developed in which the activity of the ureter was monitored in terms of peristaltic rate (or interperistaltic interval) and bolus volume without using intraluminal measuring devices or direct ureteric perfusion (34, 29).

In this study, using similar less invasive techniques, the effects of autonomic drugs on the interperistaltic interval and bolus volume have been determined. Natural urine flow was kept to a minimum and the renal pelvis was perfused via a cannula nephrostomy. Any change in natural urine flow was monitored so that its effects could be separated from those due to the effect of the drug on the ureter.

MATERIALS AND METHODS

Nineteen female adult mongrel dogs, mean weight 19.75 (SE 0.31) kg, were anesthetised with intra-

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venous pentobarbitol using an initial dose of 25 mg/kg and anaesthesia was maintained with 60 mg increments. Endotracheal intubation and ventilation with room air were performed when necessary. A mid-line laparotomy incision was made, the bladder opened and a 5 F ureteric catheter inserted transvesically into the lower 2 to 3 cm of each ureter and secured in place with a silk ligature (Fig. 1).

Ureteric flow rate was measured by using an optical reflectance method to count the number of drops from the ureteric catheters per unit time. Each drop was detected by light reflecting from its surface and causing an increase in the voltage of a photosensor. This was converted to a digital signal that produced a step-wise increase in voltage for 9 drops, and with the 10th drop the voltage returned to 0. In this way the pen of the chart recorder traversed the paper once and returned to baseline every 10 drops (Fig. 2). Calibration with various flow rates showed that with a 25-gauge needle drop volume was 0.0105 ml and that flow rates of up to 4 ml/min could be recorded. Drop volume was constant up to rates of 1.5 ml/min, but tended to decrease above this. The sensitivity of this recording method was sufficient to allow the determination of urine bolus volume as well as ureteric flow rates below 1.5 ml/min.

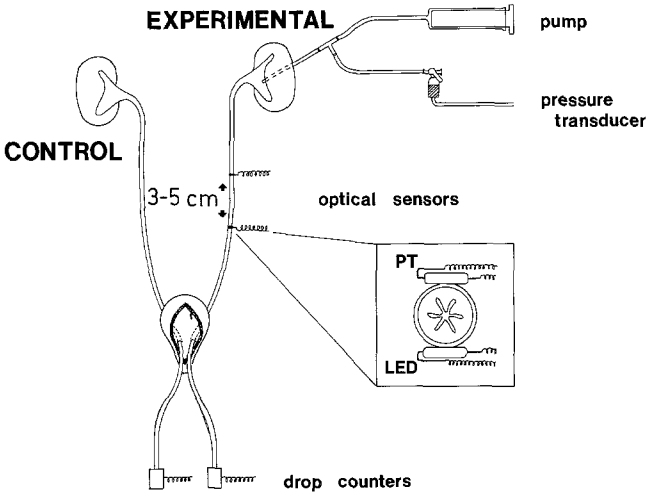

Fig. 1. Schematic diagram illustrating the position of the sensors and catheters in the experimental preparation

Ureteric peristalsis was monitored with extraluminal optoelectronic sensors. These were originally developed by Halbert et al. (9) for use on the oviducts of experimental animals. For the study of ureteric peristalsis the original configuration of a rigid cuff was modified by separating, physically, the light emitting diode (LED) and phototransistor (PT) thereby permitting the ureter to expand without restriction. The larger LED was placed posteriorly and the smaller PT, weighing approximately 15 mg, was placed anteriorly. Both halves were fixed to the ureteric wall with cyanoacrylate tissue adhesive after making a small incision through the posterior peritoneum. Two sensors were placed at a separation of 2.5 to 5 cm in order to record peristaltic direction.

The optical sensor signal is related to mechanical events in the ureter according to a complex mechanico-optical transfer function. Since this relationship cannot be precisely defined, the device was used as a muscle activity "event" sensor rather than a true mechanical transducer. It is especially sensitive to change in geometry of the wall and lumen, and it was found that with small boluses at low flow rates the bolus was associated usually with a decrease in light transmission while the contraction wave behind the bolus produced an increase in light transmission. Respiration and other artefacts could readily be distinguished from the signal associated with the peristaltic event as with correct positioning of the sensors the signal from the latter was two or three times greater than that produced by respiratory or mechanical artefacts. Cinematographic studies showed that the circumferential and longitudinal wall movements did not appear to be affected significantly by these measuring devices.

A 16-gauge cannula (Angiocath) was inserted into the renal pelvis for the purpose of perfusing the renal pelvis and recording intrapelvic pressure. This was performed by incising the renal capsule and then advancing the cannula without the needle in place until the tip lay in the renal pelvis. It was found that with a 20 kg dog the tip

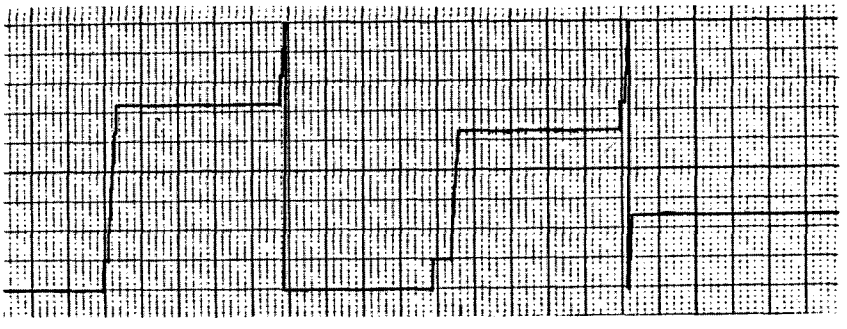

Fig. 2. The experimental ureter output, as measured by the drop counter, with natural urine flow. It can be seen that the number of steps per bolus varies from 3 to 7. (There are 9 steps from baseline to near the top of the chart and one to return the pen to baseline)

lay in the renal pelvis when it was inserted to a depth of 3 cm from the capsule. Also, the resistance to insertion of the cannula would suddenly decrease as the tip of the papilla was punctured and the renal pelvis entered. The cannula was secured to the renal capsule with a silk ligature and the proximal end passed through a small, separate flank incision. The cannula was attached to a Harvard pump filled with Ringer's lactate solution and the usual perfusion rate was 0.475 ml/min. Pressure in the renal pelvis was measured via a side arm in the system by a standard pressure transducer and amplifier. The retroperitoneum was bathed in Ringer's lactate, warmed to 37°C and the abdomen covered to prevent evaporation from the exposed peritoneal surfaces.

The left femoral artery and vein were cannulated for measuring blood pressure and for infusing fluids and drugs. A 6-channel recorder produced a continuous recording of arterial pressure, renal pelvic pressure, the output from each ureter measured by the drop counters and the activity of the experimental ureter from the 2 optical sensors. Ringer's lactate was infused intravenously at a rate of 1 ml/min throughout the experiment, and all drugs were given over a 1 min period. When antagonists were given prior to an agonist, 3 min were allowed to elapse between the start of each drug. A total of 19 dogs were used in this study and the total number of animals in each group and the doses given are shown in the tables. The dose of phentolamine was 500 μg/kg in all except two of the experiments in which it was used. Seven of the 8 animals receiving propranolol were given 100 μg/kg or greater and 6 of the 7 animals receiving terbutaline were given 50 μg/kg or greater. The 5.0 μg/kg dose of methacholine was found to produce very marked cardiovascular effects and was decreased to 2.5 μg/kg in 5 of 7 experiments. A control period of 3-5 min was obtained before the drugs were given and effects were usually observed within one minute of the start of administration and continued for a variable period. Blood pressure, renal pressure, flow rate, bolus volume and interperistaltic interval were compared before and after each drug and statistical analysis was performed using the paired t-test on the raw data.

RESULTS

Norepinephrine decreased the interperistaltic interval, indicating an increase in ureteric contraction rate, and decreased the bolus volume (Fig. 3, Table 1). The effects of norepinephrine on the ureters were completely blocked by pretreatment with phentolamine, an α-adrenergic antagonist, but the hypertensive effects were only partly blocked. In four experiments, there was a short initial period [1.49 ± (0.33) min] of complete inhibition of ureteric peristalsis in

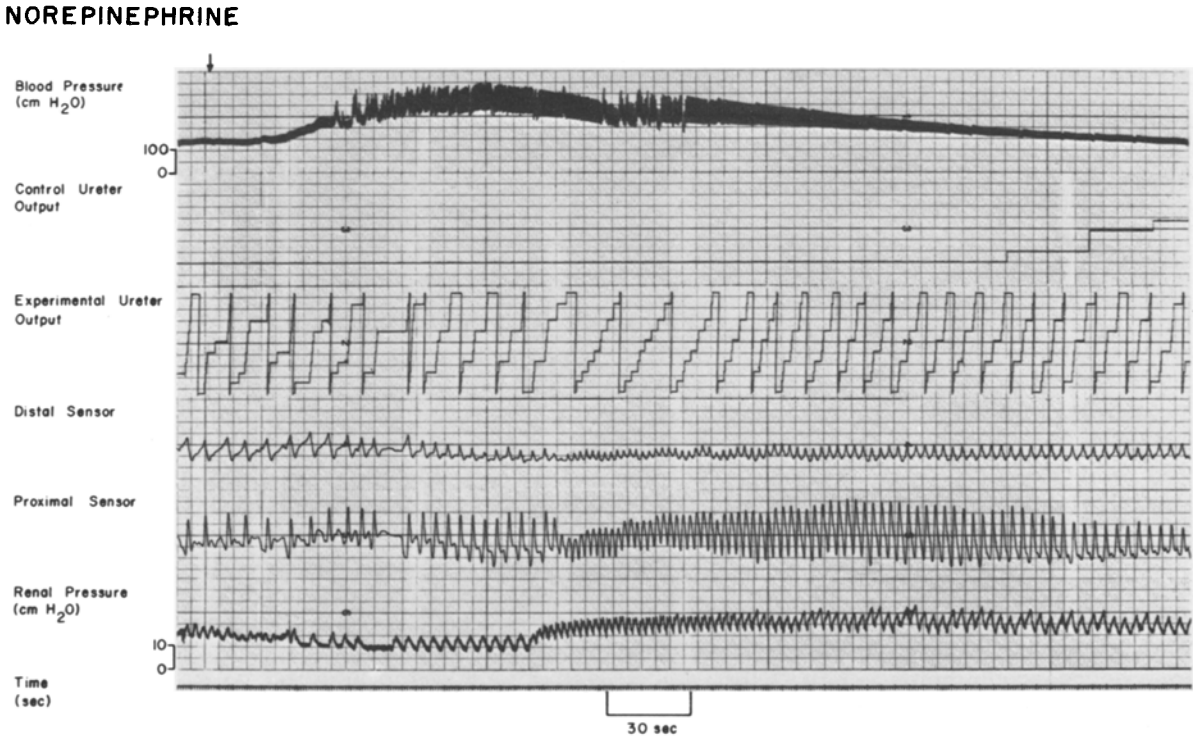

Fig. 3. The effect of norepinephrine is to increase the ureteric and renal pelvic contraction rates, decrease the bolus volume and increase the blood pressure

Table 1. Effect of norepinephrine and antagonists on ureteric activity

		(Drug doses in μg/kg) [Raw data in means (± SE)]		
		Norepinephrine (5.0)	Norepinephrine (0.5) after Phentolamine (250-500)	Phentolamine (250-500) alone
N		7	7	7
BP ($cm H_2O$)	Before	178 (±8.6)	150 (± 7.2)	173 (±8.2)
	After	393 (±8.2)	220 (±11.2)	150 (±7.2)
	% change	↑121***	↑47***	↓13***
Renal pressure ($cm H_2O$)	Before	16.5 (±1.5)	15.1 (± 1.3)	15.8 (±1.5)
	After	21.2 (±3.8)	15.0 (± 1.0)	15.1 (±1.3)
	% change	↑28	↓1	↓4
Flow rate (ml/min)	Before	0.64(±0.04)	0.74(±0.12)	0.63(±0.06)
	After	0.38(±0.11)	0.84(±0.10)	0.74(±0.12)
	% change	↓41	↑14*	↑18
Bolus volume (ml)	Before	0.09(±0.01)	0.14(±0.02)	0.12(±0.02)
	After	0.02(±0)	0.16(±0.01)	0.14(±0.02)
	% change	↓78*	↑14	↑17
Interperistaltic interval (min)	Before	0.13(±0.02)	0.18(±0.02)	0.18(±0.02)
	After	0.05(±0.01)	0.21(±0.03)	0.18(±0.02)
	% change	↓62**	↑17	0
Period of drug effect (min)		2.18(±0.38)	3.72(±0.56)	3.00(±0)

where: *p = 0.05-0.01
**p = 0.01-0.001
***p ≤ 0.001

response to norepinephrine following pretreatment with phentolamine. Of all the drugs tested, norepinephrine produced the greatest change in renal pressure (an increase of 28%), but this was not statistically significant. Phentolamine alone produced a significant drop in blood pressure, but no significant change in ureteric function.

Isoproterenol, a β-receptor agonist, completely inhibited ureteric activity for a short period of time (Fig. 4, Table 2). After pretreatment with propranolol, the inhibitory effects of isoproterenol were decreased in that the interperistaltic interval and bolus volume were increased but activity was not abolished (Table 2). Propranolol alone not only failed to produce a significant change in blood pressure but also caused no significant change in renal pressure, bolus volume or interperistaltic interval.

Methacholine appeared to stimulate the rate of peristalsis and decrease the bolus volume without altering flow rate (Table 3). These effects were blocked by both atropine and phentolamine, suggesting that the cholinergic stimulation occurred via α- adrenergic receptors. Atropine alone produced no significant change in any of the parameters measured. Serotonin produced no obvious effect either alone or after pretreatment with methylsergide and after methylsergide alone, there was no significant change in activity (Table 4).

DISCUSSION

In previous studies the presence of a catheter in the ureter has been shown to alter the normal modal distribution of the interperistaltic intervals (5) probably by producing retrograde contractions from the site of the catheter. In the studies described here, using extraluminal sensors, retrograde contractions were observed occasionally, particularly in the initial period of monitoring and were often related to partial obstruction by blood clots in the catheter or in the drop counter needle. However, after an initial period of stabilisation, and when fluid flow was unimpeded, spontaneous retrograde activity was observed infrequently. When used

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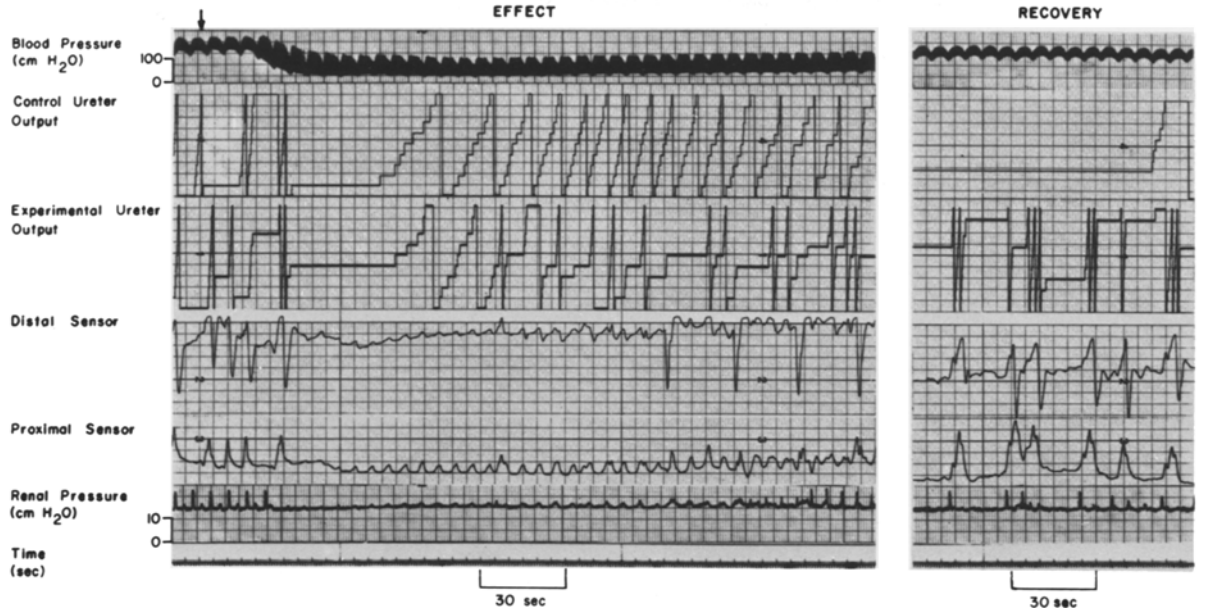

Fig. 4. Isoproterenol produces an inactive period with a marked fall in blood pressure

Table 2. Effect of isoproterenol (and antagonists) and terbutaline on ureteric activity

		(Drug doses in µg/kg) [Raw data in means (± SE)]			
		Isoproterenol (15)	Isoproternol (15) after Propranolol (50-200)	Propranolol (50-200) alone	Terbutaline (25-100)
N		8	5	8	7
BP (cm H_2O)	Before	160 (±34.0)	156 (±12.8)	154 (±12.5)	191 (±12.9)
	After	86 (±20.5)	116 (±12.5)	157 (±12.8)	166 (± 5.7)
	% change	↓46***	↓26***	↑1	↓13
Renal pressure (cm H_2O)	Before	18.3 (± 1.9)	18.3 (± 5.6)	19.1 (± 2.1)	18.3 (± 1.7)
	After	18.2 (± 1.4)	16.8 (± 4.6)	18.3 (± 2.0)	18.8 (± 2.0)
	% change	↓1	↓8	↓4	↑3
Flow rate (ml/min)	Before		0.77(±0.05)	0.64(±0.08)	0.70 (±0.05)
	After		0.66(±0.11)	0.69(±0.05)	0.59 (±0.05)
	% change		↓14	↑8	↓16
Bolus volume (ml)	Before		0.16(±0.02)	0.16(±0.03)	0.14 (±0.02)
	After		0.20(±0.02)	0.16(±0.03)	0.12 (±0.01)
	% change		↑25*	0	↓14
Interperistaltic interval (min)	Before		0.19(±0.04)	0.23(±0.05)	0.20 (±0.03)
	After		0.33(±0.10)	0.23(±0.05)	0.19 (±0.03)
	% change		↑74*	0	↓5
Period of drug effect (min)		Inactive period for 2.41(±1.80) recovery in (5.39(±3.14)	4.02(±1.36)	3.00(±0)	2.88 (±0.40)

where: *p = 0.05 - 0.01
**p = 0.01 - 0.001
***p ≤ 0.001

Table 3. Effect of methacholine and atropine on ureteric activity

		(Drug doses in µg/kg) [Raw data in means (± SE)]			
		Methacholine (2.5-5.0)	Methacholine (2.5-5.0) after Atropine (20)	Methacholine (2.5-5.0) after Phentolamine (250-500)	Atropine (20) alone
N		7	6	5	6
BP ($cm H_2O$)	Before	183 (±10.3)	187 (± 8.9)	166 (±39.0)	180 (±8.3)
	After	138 (±19.4)	174 (±10.8)	121 (±33.0)	187 (±8.9)
	% change	↓25*	↓7	↓27	↑3
Renal pressure ($cm H_2O$)	Before	14.1 (± 1.7)	14.4 (± 1.4)	13.0 (± 2.6)	14.4 (±1.4)
	After	15.4 (± 1.1)	15.9 (± 2.0)	12.3 (± 3.0)	14.4 (±1.4)
	% change	↑9	↑10	↓5	0
Flow rate (ml/min)	Before	0.63(±0.03)	0.69(±0.03)	0.68(±0.06)	0.67(±0.03)
	After	0.62(±0.05)	0.60(±0.05)	0.68(±0.05)	0.69(±0.03)
	% change	↓2	↓13	0	↑3
Bolus volume (ml)	Before	0.11(±0.02)	0.11(±0.02)	0.12(±0.05)	0.11(±0.02)
	After	0.07(±0.02)	0.10(±0.02)	0.12(±0.04)	0.11(±0.02)
	% change	↓36**	↓9	0	0
Interperistaltic interval (min)	Before	0.17(±0.02)	0.15(±0.03)	0.19(±0.10)	0.16(±0.03)
	After	0.10(±0.02)	0.16(±0.04)	0.17(±0.07)	0.15(±0.03)
	% change	↓41**	↑6	↓11	↑6
Period of drug effect (min)		2.09(±0.34)	4.62(±1.47)	2.41(±0.41)	3.00(±0)

where: *p = 0.05 - 0.01
**p = 0.01 - 0.001
***p ≤ 0.001

on the oviduct in rabbits and monkeys (9) these sensors did not alter organ function, and the current studies suggest that this extraluminal monitoring device has less effect on ureteric function than intraluminal devices. With inhibition of peristalsis, and occasionally with increase in peristaltic rate, the flow from the ureteric catheter was decreased temporarily. During the periods of decreased flow, perfusion fluid accumulated in the renal pelvis and ureter, and was eliminated subsequently when peristalsis returned to normal. The presence of constant flow at 0.475 ml/min meant that activity did not decrease merely because of a sudden reduction in urine output.

Norepinephrine produced a significant decrease in interperistaltic interval and a decrease in bolus volume. These changes were blocked by pretreatment with phentolamine; however, phentolamine alone did not alter ureteric activity. It is possible that the primary effect of α-adrenergic stimulation is to produce a change in the ureteric contraction rate so that the decrease in bolus volume is secondary. The fact that no significant change was observed with α-adrenergic antagonists alone indicates that in this model the α-adrenergic receptors may have little physiological importance in regulating normal, baseline ureteric activity.

Isoproterenol has both β1 and β2 receptor activity, but there was no significant response to terbutaline (a β2 agonist) which is in contrast to the findings of Reid et al. (17) who showed that terbutaline had an inhibitory effect on the ureter.

Also, in 4 of the 7 experiments in which norepinephrine was given after pretreatment with phentolamine, there was a period when contrations were completely inhibited. It is postulated that this inhibition was due to the unopposed effect of norepinephrine on the β receptors. Norepinephrine stimulates both α and β1 adrenergic receptors and is supposed to have feeble β2 effects (10) which also suggests that the ureter may possess β1 receptors like the small intestine (11). However, the β1 receptors probably have little

Table 4. Effect of serotonin and antagonist on ureteric activity

		(Drug doses in μg/kg) [Raw data in means (± SE)]		
		Serotonin (15)	Serotonin (15) after Methylsergide (50)	Methylsergide (50) alone
N		6	5	6
BP ($cm H_2O$)	Before	177 (± 4.3)	176 (±8.22)	176 (±7.5)
	After	184 (±11.4)	172 (±12.1)	176 (±8.2)
	% change	↑4	↓2	0
Renal pressure ($cm H_2O$)	Before	18.2 (± 2.0)	15.6 (± 1.8)	15.0 (±1.6)
	After	21.1 (± 5.6)	14.3 (± 4.2)	15.6 (±1.8)
	% change	↑16	↓8	↑4
Flow rate (ml/min)	Before	0.71(±0.06)	0.74(±0.09)	0.82(±0.07)
	After	0.82(±0.12)	0.61(±0.15)	0.74(±0.09)
	% change	↑16	↓18	↓10
Bolus volume (ml)	Before	0.15(±0.03)	0.16(±0.04)	0.17(±0.05)
	After	0.16(±0.04)	0.15(±0.06)	0.16(±0.04)
	% change	↑7	↓6	↓6
Interperistaltic interval	Before	0.17(±0.05)	0.21(±0.07)	0.18(±0.05)
	After	0.18(±0.07)	0.17(±0.03)	0.21(±0.07)
	% change	↑6	↓19	↑17
Period of drug effect (min)		1.95(±0.58)	1.95(±0.79)	3.00(±0)

where: $*p = 0.05\text{-}0.01$, $**p = 0.01\text{-}0.001$, $***p \leq 0.001$

physiological importance in controlling normal baseline activity in that β blockade alone did not alter ureteral activity.

The stimulating effect of methacholine on ureteric contraction rate was blocked by atropine and phentolamine and this effect is probably mediated indirectly via muscarinic receptors in sympathetic ganglia (1). Long and Nergardh (12) found that with serotonin a constant dose-dependent stimulatory effect was seen in human ureteric muscle strips in vitro, but in our experiments serotonin did not produce any constant effect and no change was observed with methylsergide alone.

The effect of anaesthesia on this model can be judged by comparing our results with those of Reid et al. (17) who altered ureteric activity by administering various drugs to conditioned unanaesthetised dogs while monitoring ureteric pressure with intraluminal catheters. Their results were similar to ours except that they demonstrated inhibitory activity with β2 agonists.

In conclusion, although the ureter seems to have α and β2 adrenergic receptors and the α receptors have been shown by other workers to be functionally innervated, they are probably of little physiological significance. The possibility of pharmacological manipulation of the diseased ureter with autonomic agents seems unlikely in that the effects are short-lived and usually associated with marked cardiovascular side effects.

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