Acceleration of Ureteral Peristalsis by Adrenal Compression

Abstract. *Unilateral adrenal compression resulted in bilateral acceleration of ureteral peristalsis which could be reversed by an injection of alpha-adrenergic inhibitor, providing additional evidence for the presence of adrenergic receptors in the ureter.*

The ureters of dogs and patients contain significant quantities of adrenaline and noradrenaline. Uptake of tritiated noradrenaline and depletion of endogenous noradrenaline by reserpine have also been demonstrated (*1*). Infusions of adrenaline, noradrenaline, and phenylephrine stimulate ureteral peristaltic frequency in dogs.

We have now succeeded in stimulating ureteral peristalsis by adrenal compression, presumably by the release of endogenous adrenaline or noradrenaline. Hence, the ureter has adrenergic receptors that respond to endogenous sympathetic neurohumor. Ureteral peristalsis has been believed to be myogenic and completely devoid of nervous or humoral control.

Ureteral peristalsis in dogs anesthetized with sodium pentobarbital was monitored by indwelling ended, fluid-filled ureteral catheters (No. 5 French open-ended) in series with pressure transducers. Recordings were made on a twin-channel, direct-writing electronic recorder. The catheters were passed through the bladder after cystotomy, or through the ureteral orifices exposed on the surface of the abdomen by previous bladder explantation (*2*). After suitable control tracings of several minutes to an hour in order to allow the peristaltic rate, amplitude, rhythm, and pattern to be recorded continuously, the adrenal gland was exposed surgically by retraction of the bowel and squeezed between the thumb and fingers or by a surgical sponge forceps, continuously or intermittently (*3*).

In all nine experiments on five dogs, after 2 to 3 minutes of adrenal compression, the ureteral peristaltic rate doubled or tripled. The interperistaltic interval disappeared entirely, and the baseline pressure rose (Figs. 1 and 2). Bradycardia and hypertension accompanied the ureteral response. The effect persisted for 1 to 3 minutes after the cessation of massage and was shortened by the injection of phentolamine (5 mg intravenously), an alpha-adrenergic inhibitor. When the experiment was repeated after the first injection of phentolamine, the effect was much less; and a second injection succeeded in abolishing the second effect. Both the ipsilateral and the contralateral ureters responded, ruling out mechanical stimulation as the cause. The same type of response follows the injection of adrenaline, noradrenaline, phenylephrine, or other ureteral stimulants such as histamine (*4*).

Tanagho (*5*) has demonstrated that the ureteral orifice and trigone are under sympathetic nervous control. Durand and Descotes (*6*) and Learmonth (*7*) have reported that splanchnic or hypogastric nerve stimulation stimulates ureteral peristalsis.

We now present further evidence for the presence of adrenergic receptors that are of physiologic significance in the ureter. The ureter is known to respond to catecholamines in vitro.

Clinical syndromes of uncertain etiology such as ureteral spasticity, ureteral dyskinesia, neurogenic megaloureter, ureteral spasm, and others, now seem to us to have a possible pathophysiologic basis. Sudden stress or strong emotion may cause secretion of sufficient catecholamines into the blood stream to excite ureteral peristalsis. If a patient should have a low-grade obstruction, ureteral kink, partial obstruction, stone, muscular deficiency, or other anatomic abnormality, this stimulation could conceivably result in a peristaltic overload or block due to decompensation if the increased peristaltic rate failed to empty the lumen. The production of an acute hydronephrosis by sudden diuresis in the presence of a low-grade ureteral obstruction is well known to the clinical urologist.

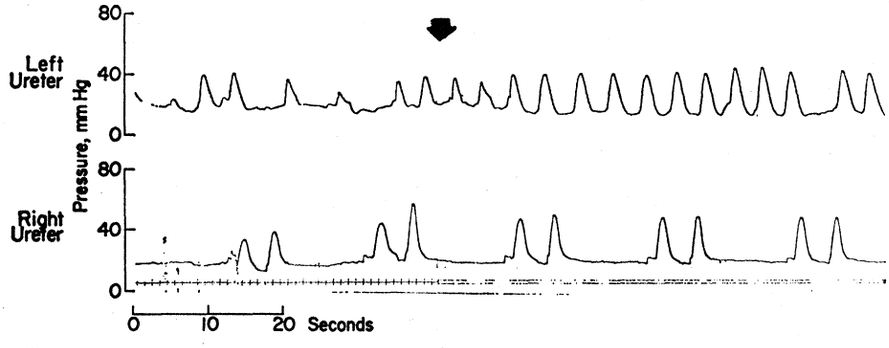

Fig. 1. Start of adrenal compression. Note more rapid onset of acceleration of ureteral peristalsis on left, the side of compression. Vertical height represents intraluminal pressure; horizontal distance, time.

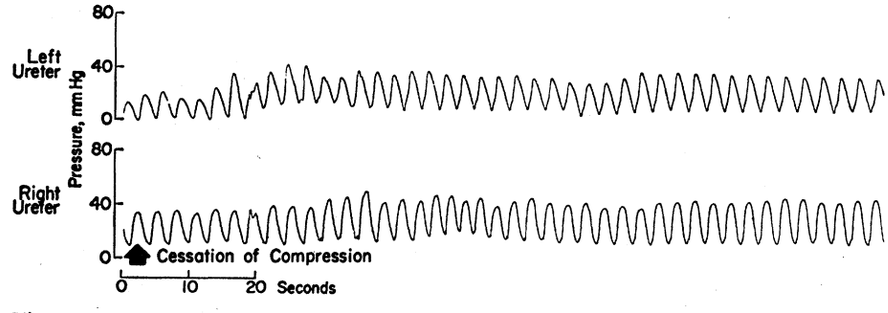

Fig. 2. After 3 minutes, ureteral peristaltic rates are 21 per minute on the right and 24 per minute on the left. Effect persists beyond cessation of massage.

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References and Notes

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3. In four experiments during antidiuresis, urine flows (measured with a probable error of 0.05 cm^3/min per ureter) showed no significant change from control levels during 3 minutes' compression nor for 5 minutes thereafter.
4. S. Boyarsky, N. Kirshner, P. Catacutan-Labay, unpublished data.
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8. Acknowledgment is made to the National Institutes of Health, Eli Lilly and Company, the Mary Duke Biddle Foundation and the Veterans Administration Hospital.

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