Effects of Noradrenaline and Acetylcholine on Ureteral Peristalsis

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MORITA, T., SUZUKI, T., KONDO, S. and TSUCHIDA, S. Effects of Noradrenaline and Acetylcholine on Ureteral Peristalsis. Tohoku J. exp. Med., 1983, 141 (4), 489-490 — Experiments in vitro were performed to compare effects of noradrenaline and acetylcholine on the pyeloureter, both of which activate ureteral peristalsis. Noradrenaline elevated the baseline renal pressure to accelerate the ureteral peristaltic pace in a ratio of 1:1 with the pacemaker discharge at the pelvicalyceal border (PC-border). Acetylcholine also accelerated the ureteral pace for a short time, as compared with the effect of noradrenaline. The baseline renal pelvic pressure dropped after administration of acetylcholine. Noradrenaline a little quickened the rhythm of the pacemaker contraction at the PC-border, and acetylcholine also quickened the pacemaker rhythm. These data suggest that noradrenaline and acetylcholine, which both activate pelviureteral peristalsis, exert different effects on pelvic pressure.

In vivo study recording ureteral electromyograms and in vitro study using isolated ureters have shown that ureteral peristalsis is generally activated by not only noradrenaline but also acetylcholine. Recently it has been proved that ureteral peristalsis is controlled by the renal

NORADRENALINE

BEFORE 10sec 120sec AFTER

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Fig. 1. Simultaneous recording of the pelvic pressure and electromyograms at the PC-border (pacemaker), PUJ and ureter before and after application of $10^{-4}$ g/ml noradrenaline.

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pelvic pacemaker. The present experiments were performed to examine how these drugs, which
can activate the ureteral activity, influence the pacemaker at the PC-border and the pelvic
pressure.

Ten adult mongrel dogs were laparotomized and the kidneys were removed along with the
ureter. The kidney was immediately placed in a bath of Krebs-Ringer solution saturated with
95% oxygen and 5% carbon dioxide, in which the renal pelvis was widely exposed up to the
extent of calyces by removal of parenchyma. A 4 Fr. catheter was inserted into the renal pelvis.
The pelviureter was constantly infused with Krebs-Ringer solution containing noradrenaline
\((10^{-5} \text{ to } 10^{-3} \text{ g/ml})\), or acetylcholine \((10^{-5} \text{ to } 10^{-3} \text{ g/ml})\), through this catheter using an automatic
injector at a rate of 0.80 ml/min. Changes in pelvic pressure were recorded simultaneously with
the electromyograms at the PC-border, pelviureteric junction (PUJ) and the ureter. Eight
kidneys were prepared for noradrenaline and 12 kidneys for acetylcholine.

As shown in Fig. 1, after application of noradrenaline intervals of pacemaker discharges
were slightly shortened and the baseline pelvic pressure was elevated, and contraction pressure
became stronger. Furthermore, the electrical discharges at the PUJ and ureter tended to be
frequently coinciding the pacemaker discharge. With application of acetylcholine, as shown in
Fig. 2, pacemaker discharges were initially a little quickened, and subsequently, the discharge
intervals became longer than before drug application. The baseline pelvic pressure and systolic
pressure fell down. The ureteral peristalsis was also stimulated for short time after administra-
tion of the acetylcholine. These data suggest that noradrenaline can demonstrably stimulate the
ureteral peristalsis through the renal pelvic action which controls the ureter. On the other hand,
acetylcholine may slightly stimulate the pelviureteral activity, but it drops the renal pelvic
pressure and a little elongates the pacemaker rhythm subsequently.

References

![Fig. 2. Renal pelvic pressure and electromyograms at the PC-border, PUJ and ureter, before
and after application of \(10^{-4} \text{ g/ml} \) acetylcholine.](image)