Response of the Renal Pacemaker to $\beta_1$-Adrenoceptor Agonist: Difference from the Cardiac Pacemaker

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MORITA, T., SUZUKI, T., KONDO, S., SAEKI, H. and TSUCHIDA, S. Response of the Renal Pacemaker to $\beta_1$-Adrenoceptor Agonist: Difference from the Cardiac Pacemaker. Tohoku J. exp. Med., 1984, 143 (4), 409-412 — Electromyographic responses of the canine-pyeloureter to dobutamine, a $\beta_1$-adrenoceptor agonist were investigated in vitro. Dobutamine produced a marked enhancement of potential in the pelvicalyceal (PC-) border, but failed to evoke noticeable responses in the renal pelvis and ureter. Dobutamine, however, made the pacemaker rhythm a little slow. This finding shows that the renal pacemaker is somewhat different from the cardiac pacemaker, pace of which is quickened by dobutamine.

Electromyogram; pacemaker of ureteral peristalsis; beta-one agonist; in vitro experiment

Gosling et al. (1974) in their anatomical study reported the occurrence of pacemaker cells containing an abundance of cytoplasmic glycogen granules in the regions of the upper part of renal pelvis. It has also been demonstrated electrophysiologically that the ureteral peristaltic pacemaker exists in the PC-border; so-called slow rising potentials were initiated in this region and gradual increase of urine volume led to an elevation of the rate of propagation of the potential to the pelvis and ureter (Tsuchida et al. 1981). It is well known that $\beta_1$-adrenoceptor agonists enhance cardiac pacemaker activity. The objective of this study is to investigate the effect of a $\beta_1$-adrenoceptor agonist on the pacemaker of ureteral peristalsis, in isolated pelvicalyceal preparation.

MATERIALS AND METHODS

Ten canine kidney and ureters were removed. The parenchyme was resected to expose the pelvis up to the PC-border. A 5 Fr. double-lumen polyethylene catheter was then inserted into the pelvis through the parenchyme. One end was connected to a infusion pump for continuous intrapelvic infusion of Krebs-Ringer solution at a rate of 0.60 ml/min, and the other end was attached to a Statham P-50 pressure transducer for recording of the intrapelvic pressure. The organ preparation was maintained in the bath of oxygenated Krebs-Ringer solution at 37°C, as shown in Fig. 1, and electromyograms were recorded from

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the PC-border, PUJ and ureter via glass microelectrodes 200 μ in diameter (time constant: 0.01 sec). Pelvic pressure was also recorded. Dobutamine (10⁻⁵ to 10⁻³ g/ml) was used as β₁-adrenoceptor agonist.

RESULTS AND DISCUSSION

Typical responses in No. 3 preparation are shown in Fig. 2. Minute potentials of about 15 μV with constant intervals of 3.80 ± 0.25 sec (n = 50) were recorded from the PC-border prior to dobutamine. In response to dobutamine, the potentials in the PC-border increased in amplitude two- to three-fold (30–50 μV) at a dose of 10⁻⁴ g/ml and four- to seven-fold (70–100 μV) at 10⁻³ g/ml, as compared to the predosing level. Furthermore the wave became similar to that of ureteral potential in a dose of 10⁻³ g/ml. The frequency became somewhat unstable with slight prolongation of discharge intervals. In contrast, the potentials from PUJ increased slightly and no appreciable change occurred in ureteral potential, following the administration of dobutamine. The pelvic contraction pressure little changed at all doses.

The changes of the discharge interval and amplitude of action potential in the pacemaker, PUJ and ureter were illustrated in Fig. 3. Exactly the same tendency of responses as No. 3 preparation was seen in other 9 pyeloureteres.

The present data have demonstrated that the β₁-adrenoceptor agonist caused a distinct increase in action potential of pacemaker cells in the PC-border. The drug, however, produced a little slowing of the rhythm of the renal pacemaker. These findings suggested clearly the presence of pacemaker cells in canine PC-border and that the renal pacemaker is different from the cardiac pacemaker, pace of which is quickened by β₁-adrenoceptor agonist.
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Fig. 2. Representative tracings following administration of dobutamine (Organ preparation No. 3). The intrapelvic pressure became slightly inconstant but the contraction pressure remained virtually unaltered at a dose of $10^{-3}$ g/ml. Pacemaker potential increased markedly and its discharge intervals was slightly prolonged with increasing dose of dobutamine from $10^{-5}$ to $10^{-4}$ and to $10^{-3}$ g/ml. PUJ and ureter showed no significant change in action potential.

Fig. 3. Diagrammatic representation of discharge intervals and amplitudes of action-potentials at before and after administration of dobutamine.
References
