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Mechanical simulation of renal pelvic wall peristalsis in the rat[1]

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Summary. A urinary concentrating defect was induced in Munich-Wistar rats by removing the renal pelvis from 1 kidney. This defect was partially corrected by crudely simulating the actions of pelvic wall peristalsis with a mechanical system that cyclically compressed the exposed renal papilla.

It has been shown, in rodents, that the upper portion of the ureter (i.e. the renal pelvic wall) physically 'milks' the renal papilla[3–5]. The peristaltic contractions of the pelvic wall cause discontinuous fluid flow in the papillary collecting ducts and vasa recta[4] and appear to alter fluid distributions within the loops of Henle, medullary interstitium, vasa recta and collecting ducts[6]. These observations have led to a variety of observations concerning the role of the upper ureter in the renal concentrating process[4–7].

Methods. A concentrating defect was created in Munich-Wistar female rats (Simonsen Laboratories, Gilroy, CA, USA) by removing the upper ureter from the right kidney and then, intermittency of fluid flow was simulated by a mechanical system that periodically compressed the exposed renal papilla. The changes in urine osmotic concentrations were compared during each of these periods.

On the evening before experimentation food and, in some cases, water were witheld from the animals. Surgical procedures, which have been described in detail elsewhere[4,8], were performed to expose the right kidney. Approximately 90 min after the administration of anesthesia (Inactin, i.p., 150 mg/kg) the right renal pelvic wall was removed with iridectomy scissors and the ureteral stump ligated; the exposed papillae ranged from 1250–1500 µm in length. Urine samples were then taken during the next 45 min from the openings of the ducts of Bellini with a micropipette. The osmotic concentrations of the uncontaminated samples (i.e. erythrocyte free) were determined on a Clifton nanoliter osmometer. During the next 45 min the action of the renal pelvic wall was crudely simulated with a 'mechanical pump' and urine samples continued to be collected for analysis. This pump consisted of a fiber optic probe (tip diameter = 1 mm) that was pushed against the side of the papilla 10 times per min by a motorized Zeiss micromanipulator. A stationary glass rod (diameter = 1 mm) was also positioned to prevent lateral movement of the papilla so that during each cycle there was sufficient compression to temporarily occlude blood flow in the vasa recta (about 20% of the 1000 µm papilla width).

Major drawbacks of this technique were bleeding of the vasa recta, loss of vasa recta blood flow and loss of urine flow during pumping. Of 25 attempts only 6 experiments produced enough uncontaminated urine samples for analysis (these rats weighed between 79 and 94 g).

In order to correct for variations in urine concentration from animal to animal, the results were expressed as a ratio of the osmolalities of urines from the exposed papillae and the bladder (i.e. urine from the contralateral control kidney) collected at the same time. The rate of change of this ratio was compared before and during pumping with a paired Student t-test.

Results and discussion. Exposure of the renal papilla produced a concentrating defect that was more pronounced with increasing control urine osmolality (range of control urine osmotic concentrations = 0.4–2 Osm/kg; ratio of the exposed:control urines = 1.1–0.4 × control urine Osm/kg; $N = 6$; $r^2 = 0.75$). Extrapolation of these data suggests that the pelvic wall has no effect when urine is isotonic to blood (i.e. exposed:control = 1; osmotic concentration = 0.3 Osm/kg). Since the presence of the upper ureter is of lesser importance at lower urine osmolalities, only those animals having control kidney osmotic concentrations greater than about 1 Osm/kg were used for determining the effects of mechanical compression.

Mechanical compression of the renal papilla reversed the falling trend in urine osmolality caused by papillary exposure (table). The actual increases in urine osmolality during the 45 min of pumping were modest (about 0.2 Osm/kg) and indicate that our mechanical system is a poor substitute for the complex peristaltic actions of the upper urinary tract. In addition, the pumping rate used (10 cycles/min) was about 50% slower than the observed contraction rate of the Munich-Wistar rat ureter.

These findings imply that the physiological 'milking' of the renal papilla may result in increased urine concentration. One

Rate of change of exposed : control urine osmolalities (ΔE/C per h)

Control kidney urine osmolality (mOsm/kg H_2O)	Rate of change following exposure	Rate of change during compression
1386	− 0.151	0.106
1924	− 0.052	0.097
1546	− 0.041	0.068
978	− 0.164	− 0.076
Mean ± SEM	− 0.102 ± 0.032	0.049 ± 0.042

The means shown are significantly different ($p = 0.028$).

effect of this milking action might be to decrease effective papillary plasma flow which in turn would decrease solute washout by the ascending vasa recta. In fact, Chuang et al.[9] have shown that removal of the upper ureter is accompanied by an increased papillary blood flow.

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Epidermal sensitivity to hypoxia in the lugworm[1]

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Summary. Physiological and anatomical data support the idea that in the caudal epidermis of the lugworm, *Arenicola marina* (L.), there are chemoreceptors detecting variations of oxygen partial pressure in the ambient water.

Numerous aquatic organisms live in tubes or in more or less consolidated burrows through which they actively pump ambient water. Usually the main purpose of this irrigation is to provide the animal with oxygen. It thus corresponds to a true ventilation, and has been extensively analyzed in several species of polychaete annelids[2-4]. The lugworm, *Arenicola marina* (L.), forces sea water through its L-shaped burrow in tail-to-head waves running along its dorsal surface. This ventilatory behavior is part of a recurrent sequence of activities lasting 30–40 min and controlled by pacemakers[5], but the cycle's normal pattern can be altered. For instance, the ventilatory activity is inhibited when the salinity of the sea water overlying the burrow is decreased[6], when the burrow contains anoxic water during low tide[7], or when, in experimental conditions, the partial pressure of oxygen (P_{O_2}) in the inspired sea water is lower than 40 Torr[8]. We suggest that, contrary to a generally accepted idea[9], the reduced oxygenation of the respiratory medium could be directly involved in the ventilation inhibition. From data in the literature[10] and from preliminary observations, we also suspected that the tail of the lugworm could be especially sensitive to the level of oxygen. Here, we report physiological and anatomical data which support these two hypotheses.

Material and methods. Lugworms were collected in the vicinity of the Station Biologique, Roscoff, and kept unfed in air-saturated running sea water (14–16 °C). An unrestrained lugworm, 15–20 g fresh weight, was placed in an artificial burrow, a straight glass tube 30 cm long, inner diameter 1 cm. The tail end of the tube was connected to a system of 2 tonometers in which sea water, chlorinity ~19.5‰, was equilibrated against an anoxic ($P_{O_2} \sim 0$ Torr) or a normoxic ($P_{O_2} \sim 155$ Torr) gas phase. Identical values for sea water P_{CO_2}, ~0.25 Torr, and pH, ~8.25, were maintained in the 2 tonometers. The glass tube could be instantaneously switched to either tonometer through a 3-way stopcock. An additional device allowed a permanent flow (3 ml per min) of sea water from the tonometer to be applied directly to the lugworm's tail even when the animal was not ventilating. The head end of the tube was connected to a Gilson flow meter and to a Statham P23BB pressure transducer. The ventilated sea water volumes and the ventilation-induced hydrostatic pressure changes in the glass tube were simultaneously recorded on a Sefram Servotrace recorder. All experiments were carried out at 15 °C.

For examination by scannning and transmission electron microscopy, small samples of tail epidermis in several normoxic animals were prepared by standard techniques.

Results. Shift from normoxic to anoxic water (fig. 1, A and B). When the tube was connected to the normoxic water the lugworm ventilated steadily and the pressure inside the artificial burrow varied with a period (13.4 sec, SEM = 0.4 sec, N = 41) corresponding to the ventilatory period, i.e. to the time lapse between the onset of 2 successive ventilatory waves. A control shift from normoxic to normoxic water had no effect on the ventilatory rhythm (fig. 1A, arrow). Conversely, after a switch from normoxic to anoxic water the ventilatory peristaltism slowed down, then stopped, and the hydrostatic pressure and ventilated water flow dropped to zero (fig. 1A). Figure 1B illustrates the mean effects of 41 normoxic-to-anoxic switches in 17 animals. 63% of the responses occurred within 27 sec, or 2 ventilatory periods. The highest response frequency occurred during the 2nd ventilatory period following the application of the stimulus to the tail of the animal.

Shift from anoxic to normoxic water (fig. 1, C and D): when the tube was connected to the tonometer of anoxic water, the lugworm did not ventilate and the difference of hydrostatic pressure inside and outside the experimental device was almost zero. A control switch from anoxic to anoxic water had no effect. Conversely, after a switch from anoxic to normoxic water (fig. 1C), the lugworm quickly resumed ventilating: pressure and ventilated volumes increased sharply. Figure 1D illustrates the mean effects of 41 anoxic-to-normoxic switches on the 17 animals of figure 1B. 56% of the responses to the normoxic stimulus occurred within about 40 sec, or 3 ventilatory periods. As with the normoxic-to-anoxic switch, the highest response frequency occurred during the time interval corresponding to the 2nd ventilatory cycle after the start of the normoxic stimulus.

Ultrastructure of the caudal epidermis. Epidermal papillae scattered all over the lugworm body are more conspicuous on the tail as rings of prominent structures. They are formed by a