EFFECTS OF SALINE INFUSION AND ANTIDIURETIC HORMONE ON RENAL AND CARDIOVASCULAR FUNCTION IN CHICKS OF LEACH'S STORM PETREL (OCEANODROMA LEUCORHOA)

David L. Goldstein and Vicki R. Reed
Department of Biological Sciences
Wright State University
Dayton, OH 45435

Leach's storm petrel (Oceanodroma leucorhoa) can excrete ingested salt and water using either the salt glands or the kidneys. In a previous study, (Goldstein and Heflick, Bull. MDIBL 31: 57 and J. Comp. Physiol., in press), we examined the effect of saline infusion on renal function in chicks of Leach's storm petrel. During infusion of a relatively dilute saline (125 mM NaCl) all salt and water was eliminated by the kidneys; the rate of glomerular filtration (GFR) was high, as was the urine flow. A more concentrated infusion solution (250 mM) stimulated salt gland secretion and an 80% decrease in urine flow, the latter accomplished with no decrease in GFR. In contrast, the salt glands assumed full responsibility for osmoregulation during infusion of 550 mM NaCl; urine flow dropped to just 2% of the value during 125 mM infusion, and GFR decreased by more than 80%, primarily because of a cessation of filtration by nephrons located in superficial regions of the kidney.

In the course of the present studies we were able to replicate the previous finding of a reduced GFR (by 75%) in birds receiving 550 mM NaCl. However, we further noted that even among the birds infused with 125 mM NaCl there was a significant correlation between urine flow rate and GFR ($N = 29$, $r = 0.41$, $P < 0.02$) This provides additional evidence for the importance of renal blood flow regulation in the response of these birds to salt.

The mechanism of reduced filtration in the avian kidney is thought to involve constriction of afferent arterioles under the influence of the antidiuretic hormone arginine vasotocin (AVT) (Braun and Dantzler, Am. J. Physiol. 222: 617, 1972; Am. J. Physiol. 226: 1, 1974). AVT is elevated in at least some marine birds during salt infusion (Gray and Erasmus, Gen. Comp. Endocr. 74: 110, 1989), and in the present study we wished to evaluate whether AVT might regulate the renal response to salt loading in petrels. We were also interested in investigating a further question related to the reduction in GFR. The kidney receives approximately 20% of the cardiac output, and the renal vasculature represents an important component of the peripheral resistance. Constriction of renal arterioles would elevate peripheral resistance and, without some form of compensation, elevate blood pressure. We were interested in whether such a change in blood pressure actually does occur and, if not, whether we could detect changes in cardiac output or regional blood flow that might serve as compensatory mechanisms.

To evaluate cardiovascular compensations during saline infusion, we wished to measure blood pressure, heart rate, cardiac output, and regional blood flow distribution. To measure the first two of these we implanted arterial catheters into the brachial arteries of petrels. We could accomplish this in approximately 10 minutes with a minimum of restraint and without anaesthesia; birds subsequently secreted well from their salt glands, indicating that the procedure was minimally stressful to them (stress, like anesthesia, would inhibit salt gland secretion). We measured heart rate from the recorded pressure pulses.

To measure cardiac output and blood flow distribution we chose to use colored microspheres (Kowallik et al., Circ. 83: 974, 1991). However, we needed to verify whether we could quantitatively recover the spheres from tissues as required for their enumeration (by dye extraction and spectrophotometry). We accomplished this in vitro, by injecting known volumes of spheres into a variety of tissues and digesting and extracting the spheres in a single container (as described by Cheung and Young (Workshop of the 1991 FASEB meeting)). The variables that proved important

in obtaining an accurate recovery were a) using a sufficient volume (at least 100 µl) of spheres so that sampling error was minimized, b) adequate tissue digestion (24 h at 40-45°C), and c) small enough tissue samples (<0.5 g) so that the filters did not clog. Unfortunately, additional difficulties arose when applying this approach in vivo in 50 g birds, including difficulty in advancing a cannula to the heart, cannulas puncturing internal blood vessels, and settling of the spheres in the cannulas (with the possibility of uneven infusion or clogging the small cannula tips). We were thus unable to accumulate satisfactory data on regional blood flow using this technique, though we are now convinced of its utility, perhaps in anesthetized and certainly in larger animals.

We did measure kidney function, blood pressure, and heart rate of animals infused with either 125 or 550 mM NaCl. Despite the reduction in GFR (described above), and presumably the concomitant increase in renal vascular resistance, mean arterial pressure did not differ between the two infusions: 96 $\pm$ 13 mm Hg on 125 mM, 91 $\pm$ 13 mm Hg on 550 mM (N = 19 and 11, respectively; all values mean $\pm$ s.d.). Neither did heart rate differ: 457 $\pm$ 60 (N = 18) and 456 $\pm$ 51 (N = 10) beat/min on 125 and 550 mM NaCl, respectively. This suggests that the reduction of renal blood flow is compensated either by a reduced stroke volume (to lower cardiac output) or by a reduced resistance (increased flow) in some other vascular beds. One of these beds is surely the blood supply to the salt glands. However, even with the high blood flow these organs receive (up to 20 ml/g•min), their small size (0.3 g/gland in the birds we measured) limits the contribution they can make to blood pressure regulation. It is possible that in petrels, as in ducks (which do not reduce GFR during saline infusion; Gerstberger et al., Am. J. Physiol. 248: F663, 1985), salt infusion induces a quite widespread increase in blood flow to the visceral organs (Kaul et al., J. Comp. Physiol. 149: 457, 1983).

To examine the extent to which AVT could stimulate the responses elicited by infusion of 550 mM saline, we added varying doses of this hormone to the 125 mM NaCl infusion. Analysis of the plasma concentrations of AVT produced by these infusions (or by the 125 and 550 mM NaCl infusions) is not yet complete. Nevertheless, the rates of infusion we used (ranging from 0.15 to 150 ng/kg•min) should have produced physiological to supraphysiological concentrations of AVT in the plasma (based on previous studies of normal AVT concentrations and of the relation between AVT infusion rates and plasma AVT concentrations in other species). We were unable to duplicate the effects of 550 mM NaCl by infusing AVT. Regressions of physiological variables against AVT infusion rate revealed that all variables changed in predicted directions: GFR ($r = -0.2$, $P < 0.5$), urine flow rate ($r = -0.1$, $P < 0.8$), and plasma osmolarity ($r = -0.5$, $P < 0.05$) dropped, whereas urinary osmolarity ($r = 0.4$, $P < 0.1$), urinary sodium concentration ($r = 0.7$, $P < 0.001$), and blood pressure ($r = 0.2$, $P < 0.4$) rose (N = 13 - 18 for all regressions). However, note that most of these relationships were weak and only two (urinary sodium and plasma osmolarity) were statistically significant. These latter two effects could have occurred by a renal tubular (rather than vascular) mechanism of AVT action. The reason for lack of a more pronounced effect of AVT could involve the concomitant expansion and dilution of the extracellular fluid by the 125 mM saline. Birds in general vigorously defend extracellular fluid volume, and this signal may have overridden the AVT effect.

In conclusion, the present studies confirm earlier findings that renal filtration is markedly reduced in Leach's storm petrel during saline loading. This is accomplished without any effect on systemic blood pressure or heart rate, and probably involves increased blood flow through other vascular beds. Colored microspheres could be effectively used to measure such changes, though doing so is difficult in unanesthetized small animals. AVT may be involved in the renal response to salt loading. However, if this is so then mechanisms exist to counter the hormone's effects on the renal vasculature during times of extracellular volume expansion.

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