THE EFFECT OF VARIOUS HORMONES ON THE RENAL EXCRETORY PATTERN OF THE DOMESTIC DUCK (ANAS PLATYRHYNCHOS)

by

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ABSTRACT

The effects of corticosterone, cortisol, aldosterone, oxytocin and vasopressin on the renal excretory pattern of the water-loaded domestic duck (Anas platyrhynchos) were examined. When compared with the results obtained from mammalian studies, the excretory pattern differed little with respect to water and sodium. The domestic duck showed high levels of potassium excretion. It is suggested that the function of the potassium secretory mechanism in the distal tubule may be predominant in this species.

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I am also indebted to the members of my Committee, Dr. C.V. Finnegan, Dr. W.S. Hoar, Dr. R.N. Band and Dr. P.A. Dehnel for their critical reading of this study.

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INTRODUCTION

There is scant information on renal excretory patterns in birds, especially with respect to sodium, potassium and water. The domestic duck is an ideal subject for such investigation since after water loads, the urine is liquid and readily analysed for electrolytes.

The domestic duck is presumably derived from marine ancestors (1), and it demonstrates the capacity to live indefinitely on a diet containing seawater or hypertonic saline as the sole source of drinking water (2, 3). This ability appears to depend on supra-orbital glands which produce a nasal discharge containing highly concentrated electrolytes. Recent investigations, in this laboratory, have shown this extra-renal excretory process to be, at least in part, under the control of adrenocortical, and possibly posterior pituitary hormones (2, 3). The pronounced effects of adrenocortical steroids on the renal tubular excretory process have been demonstrated in those vertebrates so far studied (4). Furthermore, under the influence of adrenocortical hormones, the onset of extra-renal excretion is accompanied by considerable modification of the renal electrolyte excretory pattern (2, 3).

The available data on renal excretion in the domestic duck, obtained in these laboratories and elsewhere (5), had been incidental to studies of the extra-renal mechanism. The present work, therefore, was concerned with an examination of the urinary excretion from water-loaded ducks not subjected to hypertonic diets, in order to establish a normal pattern of renal excretion. The effects of adrenocortical steroids and posterior pituitary hormones on this excretory pattern were investigated.

MATERIALS AND METHODS

Young ducks of the Pekin variety of domestic duck (Anas platyrhynchos) were used in this study. All birds were maintained on "duck grower food", with an unlimited supply of fresh water. Birds were starved for 24 hr. prior to use, but were supplied with drinking water.

The birds were weighed, placed in metabolism cages at zero time, and given 25 ml. of distilled water at 0, 1.5, 3 and 4.5 hr. thereafter. Therefore, each bird received 100 ml. of distilled water, administered in four doses. This was introduced into the glandular stomach (proventriculus) by means of a flexible plastic tube connected to a syringe.

Urine collections were made at 1.5, 3, 4.5 and 6 hr. after zero time and volumes recorded for each bird. The 1.0 I.U. oxytocin treated group (Appendix Table X) consisted of 11 birds. All other groups consisted of 8 birds.

Hormone doses were administered intramuscularly in alternate legs at -1.5, 0, 1.5, 3 and 4.5 hr. Cortisol and corticosterone (Sigma) were prepared as stabilized suspensions in isotonic saline, aldosterone (Ciba) was administered in oil solution and commercial saline preparations of oxytocin and vasopressin (Parke, Davis and Co.) were used.

Aliquots of 3 ml. were taken from the original urine sample and diluted to 50 ml. All diluted samples were then analysed for sodium and potassium with a Zeiss PF5 flame photometer (6). Results were calculated in total milliequivalents of sodium and potassium excreted per sample and concentrations were expressed in m. eq./liter of urine. Standard errors were calculated for all individual values. All 6 hr. values were compared with the corresponding control value using "Student's" t-test (7).

RESULTS

The excretory outputs of water, sodium and potassium in all groups for the individual collecting periods are tabulated in Appendix Tables I-XIII.

Controls

The accumulated excretion of water, sodium and potassium was a linear function of time in all control birds. By plotting these outputs against time and fitting the regressions by the method of least squares the equations were of the form $y = bx + a$, where y = accumulated excretion, x = time in hours, a = the ordinate intercept and b = the slope of the line (Table I). Comparison of the simultaneous rates of sodium and potassium excretion by the analysis of covariance (8) showed no significant difference between the slopes of the sodium and potassium equations. The sodium/potassium ratio for these birds, therefore, was unity and this was similarly indicated by the total output values in Table 2.

Corticosterone (Table 2, Figs. 1, 2 and 3)

The 2.5 mg. dose of corticosterone caused a consistent water diuresis throughout the experimental period and after 6 hr. the output was significantly higher than the controls ($p < 0.001$). This water diuresis was accompanied by a significantly reduced excretion of sodium ($p < 0.001$). The values obtained indicating a simultaneous increase in potassium excretion, were significant only at the 5% level.

Although sodium excretion was again significantly reduced after treatment with a 1.25 mg. dose ($p < 0.001$), the previously observed water diuresis was not apparent. The potassium excretion at the end of 1.5 hr. was 29.4% higher than the corresponding control value and this trend continued until at the

Table 1. The renal excretion of water, sodium and potassium from the water-loaded duck during a 6 hr. observation period. Each bird received 25 ml. distilled water at 0, 1.5, 3.0 and 4.5 hr.

Table 1

Parameter	Regression[1]	Correlation Coefficient "r"	Na & K Covarience		
			"F" value	Degrees of Freedom	"p" value
Volume (ml.)	y = 17.4x - .480	0.984			
Sodium (m.eq.)	y = 0.34x + .021	0.910			
			1.213	1 76	>0.25
Potassium (m.eq.)	y = 0.31x + .028	0.945			

[1] y = bx + a where y = accumulated excretion, x = time (hr.), a = the ordinate intercept and b = the slope of the line.

end of the 6 hr. period the excretion was 43% higher than in the controls. This increase in potassium (2.716 m.eq. vs. 1.897 m.eq. in the controls) was highly significant ($p<0.001$).

Cortisol (Table 2, Figs. 1, 2 and 3)

The administration of 2.5 mg. doses of cortisol brought about the same degree of water diuresis and sodium retention as was observed with similar doses of corticosterone. In contrast, the potassium excretion was unchanged when compared with the controls ($p<0.03$). At the lower dose of cortisol (1.25 mg.) water diuresis was similarly enhanced and sodium retention evident, although at a lower level of significance ($p<0.01$) than had been observed at the higher dose. Potassium excretion, on the other was significantly increased ($p<0.001$) when compared with the controls. This increase was also significant (5% level) when compared with the 2.5 mg. dose level.

This pattern of excretion, after treatment with cortisol, was qualitatively similar to the effects of corticosterone; that is the higher doses enhanced water diuresis and sodium reabsorption but had little effect on potassium excretion, whilst lower doses had the additional effect of enhancing potassium excretion.

Aldosterone (Table 2, Figs. 1, 2 and 3)

At both 100 and 50 µg. dose levels aldosterone had no apparent effect on water diuresis when compared with control birds. The effect on sodium excretion was similar to that of corticosterone and cortisol; that is, a significant retention of sodium at both dose levels. Aldosterone caused a highly significant ($p<0.001$) retention of potassium at both the 50 and 100 µg. levels. Since this hormone influenced the excretion of sodium and potassium

Table 2. The renal excretion of water, sodium and potassium from the domestic duck during a 6 hr. observation period. Each bird received 25 ml. of distilled water at 0, 1.5, 3.0 and 4.5 hr. and the indicated doses of adrenocortical steroids were administered at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Table 2

Treatment	Body wt. kg.	Urine vol. ml.	Total excretion m.eq.		Urine concentration m.eq./liter		Na/K
			Na	K	Na	K	m.eq.
Control	2.14 ±.02	99.9 ±1.9	2.069 ±.185	1.897 ±.119	20.8 ±2.0	19.1 ±1.3	1.107 ±.09
2.5 mg. corticosterone	2.25 ±.04	119.5** ± 4.1	0.855** ±.135	2.466 ±.227	7.1** ±1.0	20.9 ±2.1	0.418** ±.09
1.25 mg. corticosterone	2.15 ±.09	111.3 ± 5.9	0.856** ±.137	2.716* ±.213	7.5* ±0.84	24.4 ±1.4	0.320** ±.04
2.5 mg. cortisol	2.23 ±.03	119.4** ± 3.4	0.604** ±.036	2.057 ±.138	5.1** ±0.41	17.1 ±1.1	0.312** ±.04
1.25 mg. cortisol	2.14 ±.03	124.0** ± 2.6	1.154* ±.210	2.905* ±.338	9.3** ±1.8	23.6 ±2.9	0.428** ±.07
100µg. aldosterone	2.10 ±.08	94.2 ±4.4	0.698** ±.123	0.586** ±.076	7.3** ±0.99	6.1** ±0.65	1.251 ±.22
50µg. aldosterone	2.10 ±.08	99.3 ±3.7	1.068* ±.215	0.706** ±.070	10.3* ±2.1	7.0** ±0.58	1.472 ±.22

** $p < 0.001$
* $p < 0.01$
with respect to the corresponding control values

similarly, the sodium/potassium ratio was not significantly reduced as it was for cortisol and corticosterone.

Oxytocin (Table 3, Figs. 4, 5 and 6)

The two higher doses of oxytocin (5.0 and 1.0 International Units (I.U.)), caused a significant antidiuresis ($p<0.01$ and 0.001 respectively) while the lower dose (0.1 I.U.) significantly enhanced the excretion of water ($p<0.01$). Oxytocin significantly increased and decreased the excretion of sodium and potassium respectively, at all dose levels.

Vasopressin (Table 3, Figs. 4, 5 and 6)

The 5.0 and 1.0 I.U. doses of vasopressin showed marked antidiuretic effects when compared with the controls ($p<0.001$). Indeed, the antidiuretic effect of 1.0 I.U. of vasopressin was significantly greater than the antidiuretic effect of the same dose of oxytocin ($p<0.01$). There was no significant effect on water diuresis with the 0.1 I.U. dose. In contrast to oxytocin, vasopressin had no significant effect at any dose level on the total sodium excretion but, possibly due to water reabsorption, the concentration of sodium in the urine was significantly higher than the controls at the 5.0 and 1.0 I.U. dose levels ($p<0.01$). With respect to potassium reabsorption and/or secretion, the effect at all dose levels of vasopressin was even more pronounced than was the effect of oxytocin. In each case the total excretion of potassium was significantly lower than the controls ($p<0.001$).

A transient nasal secretion was noted after the -1.5 hr. injection in the following groups: corticosterone (both 1.25 mg. and 2.5 mg.), cortisol (1.25 mg.) and oxytocin (1.0 I.U.). The volume was small and the discharge ceased before the birds were placed in their metabolism cages at zero hours.

Table 3. The renal excretion of water, sodium and potassium from the domestic duck during a 6 hr. observation period. Each bird received 25 ml. of distilled water at 0, 1.5, 3.0 and 4.5 hr. and the indicated doses of mammalian posterior pituitary hormones were administered at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Table 3

Treatment	Body wt. kg.	Urine vol. ml.	Total excretion m.eq.		Urine concentration m.eq./liter		Na/K
			Na	K	Na	K	m.eq.
Control	2.14 ±.02	99.9 ±1.9	2.069 ±.185	1.897 ±.119	20.8 ±2.0	19.1 ±1.3	1.107 ±.09
5.0 I.U. Oxytocin	2.29 ±.06	46.5** ±2.0	3.066* ±.457	1.175* ±.195	71.9** ±8.4	25.4* ±1.7	2.904** ±.38
1.0 I.U. Oxytocin	2.24 ±.04	77.7* ±5.7	3.911** ±.307	1.160* ±.158	50.9 ±1.9	14.9 ±1.6	4.195** ±.74
0.1 I.U. Oxytocin	2.14 ±.03	113.4* ± 3.4	3.524* ±.309	1.405* ±.088	30.9* ±2.4	12.5** ±0.8	2.570** ±.28
5.0 I.U. Vasopressin	2.20 ±.08	40.3** ±6.9	1.924 ±.307	0.900** ±.154	56.8 ±9.2	23.7 ±2.3	2.540 ±.52
1.0 I.U. Vasopressin	2.24 ±.06	45.9** ±6.5	1.512 ±.247	1.199** ±.119	33.5* ±3.3	29.8 ±4.4	1.276 ±.17
0.1 I.U. Vasopressin	2.15 ±.06	104.2 ± 2.5	1.813 ±.197	1.074** ±.116	17.4 ±1.9	10.3** ±1.0	1.772 ±.22

** $p < 0.001$
* $p < 0.01$
with respect to the corresponding control values

Figure 1. The accumulated renal outputs of water during a 6 hr. period from the water-loaded domestic duck. Each bird received 25 ml. of distilled water at 0, 1.5, 3.0 and 4.5 hr. and the indicated doses of adrenocortical steroids were administered at -1.5, 0, 1.5, 3.0 and 4.5 hr.

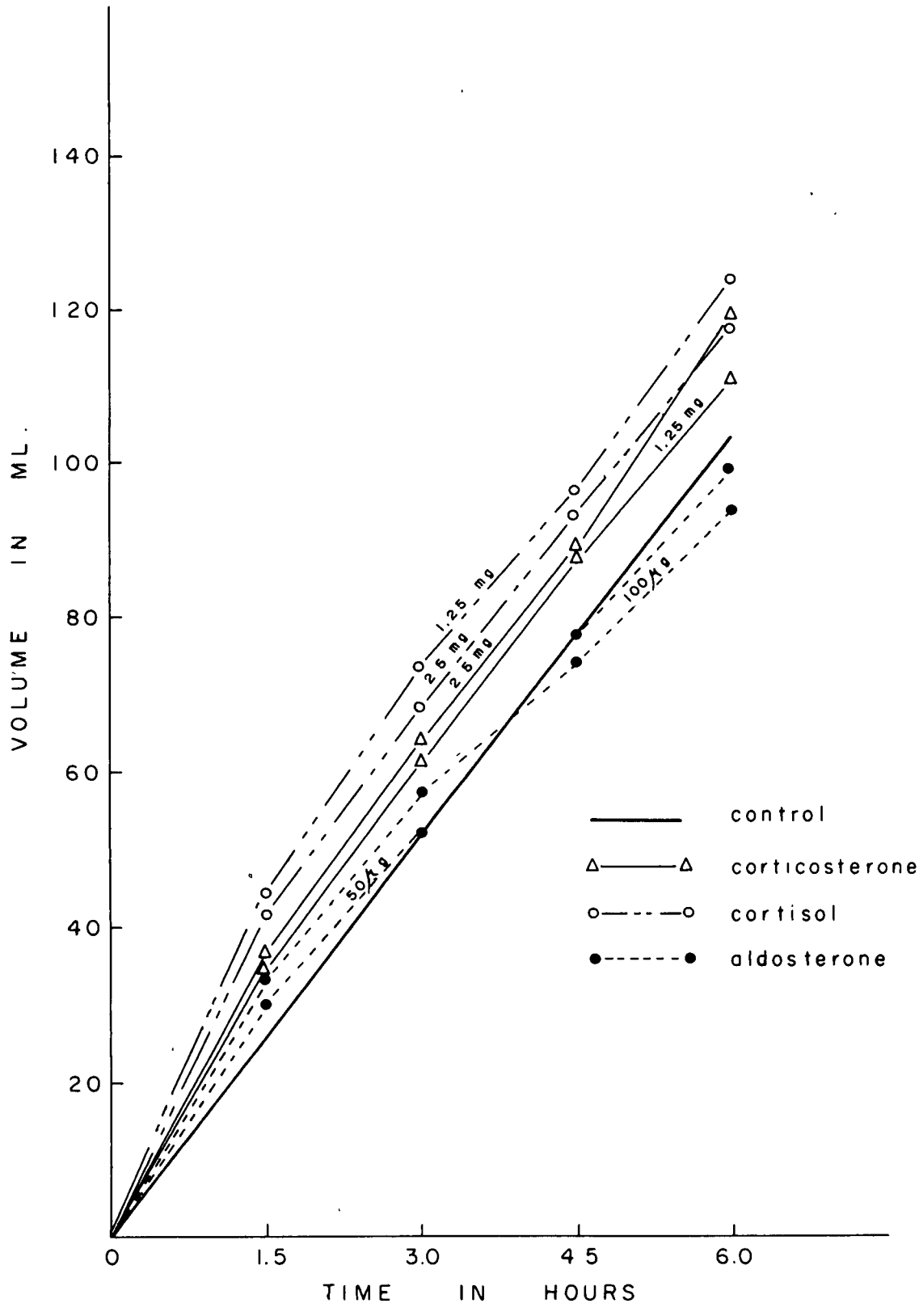

VOLUME IN ML.
140
120
100
80
60
40
20
0
1.5
3.0
4 5
6.0
TIME IN HOURS
1.25 mg
2.5 mg
2.5 mg
1.25 mg
50 µg
100 µg
control
corticosterone
cortisol
aldosterone

Figure 2. The accumulated renal outputs of sodium during a 6 hr. period from the water-loaded domestic duck. Each bird received 25 ml. of distilled water at 0, 1.5, 3.0 and 4.5 hr. and the indicated doses of adrenocortical steroids were administered at -1.5, 0, 1.5, 3.0 and 4.5 hr.

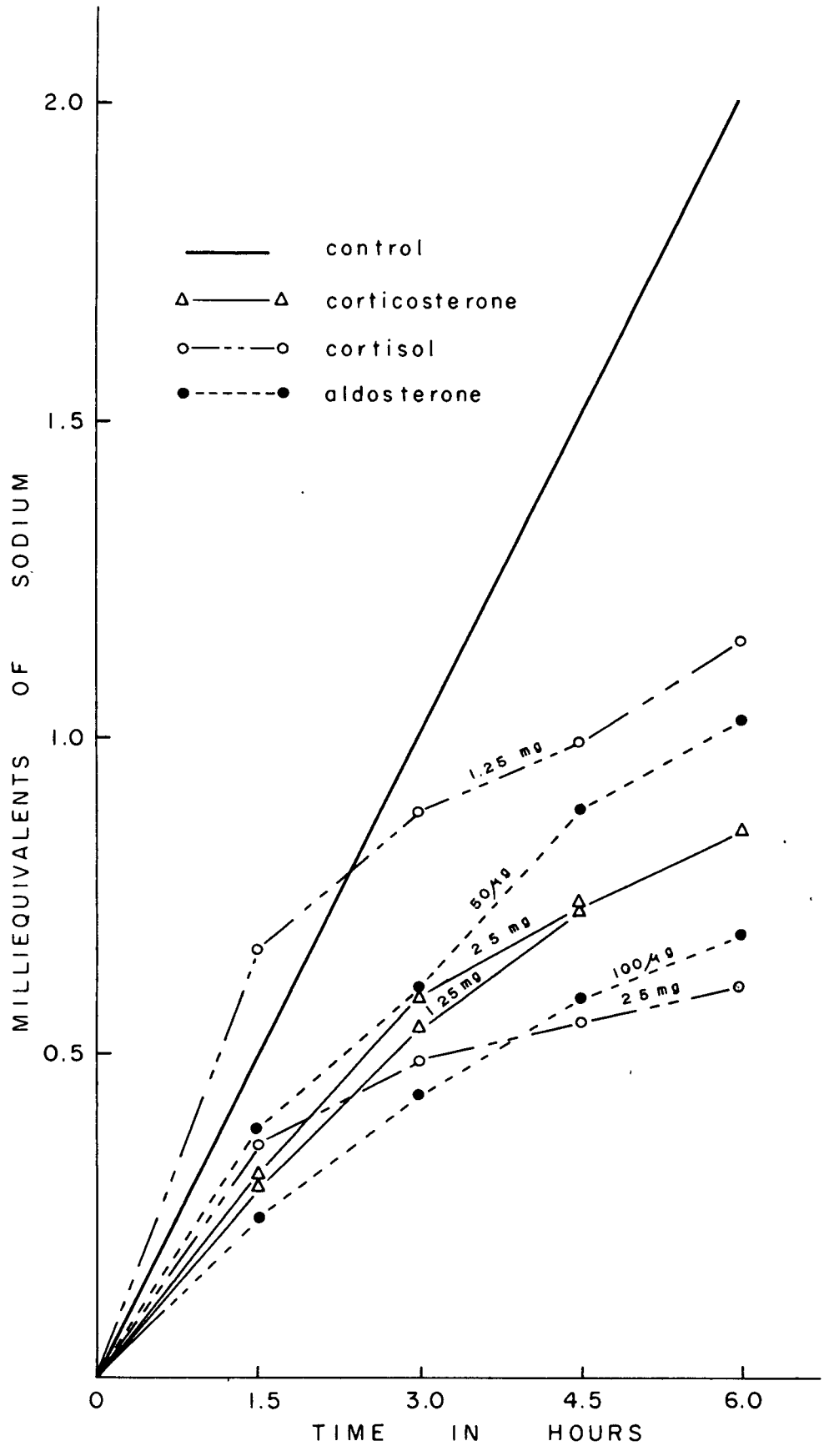
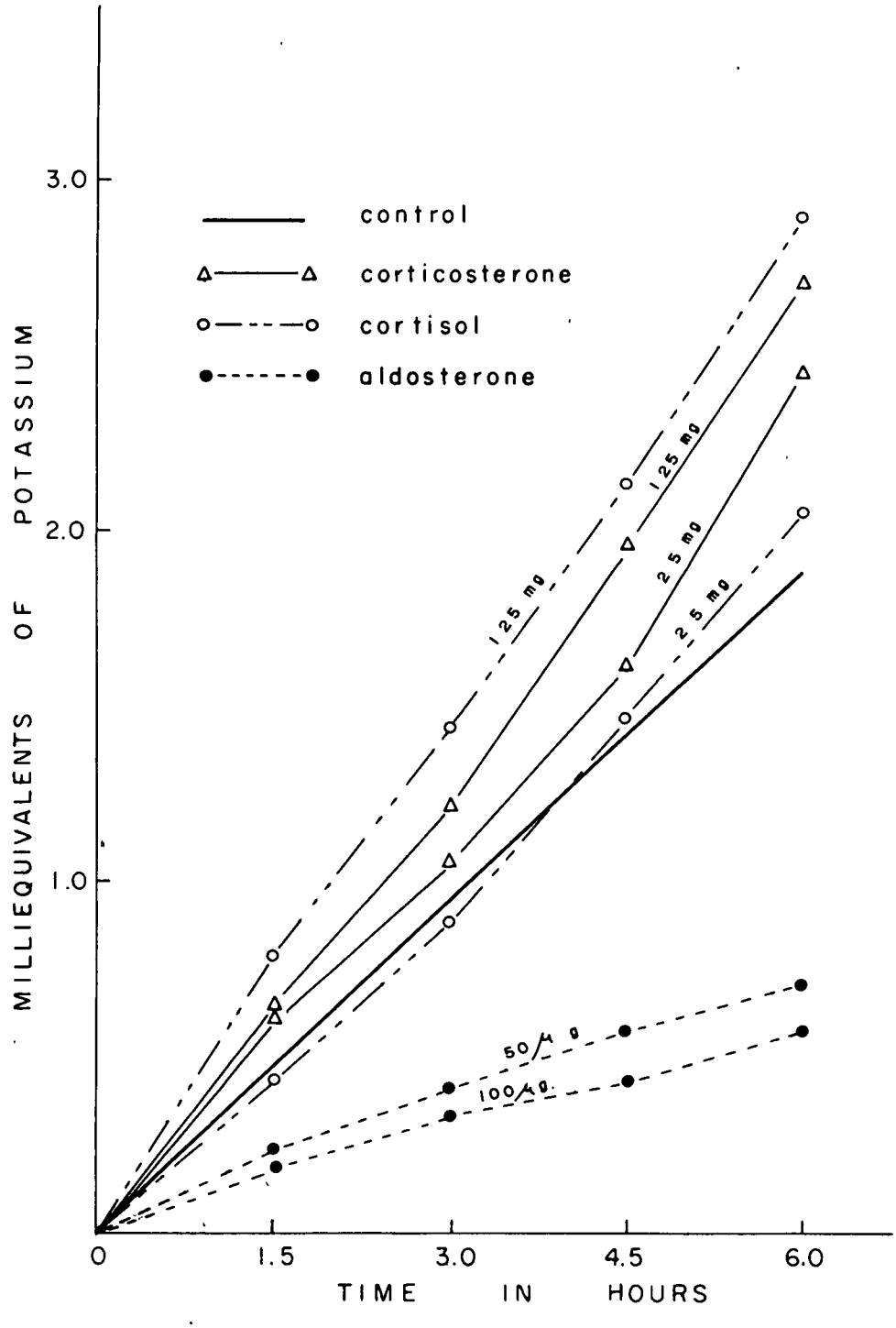
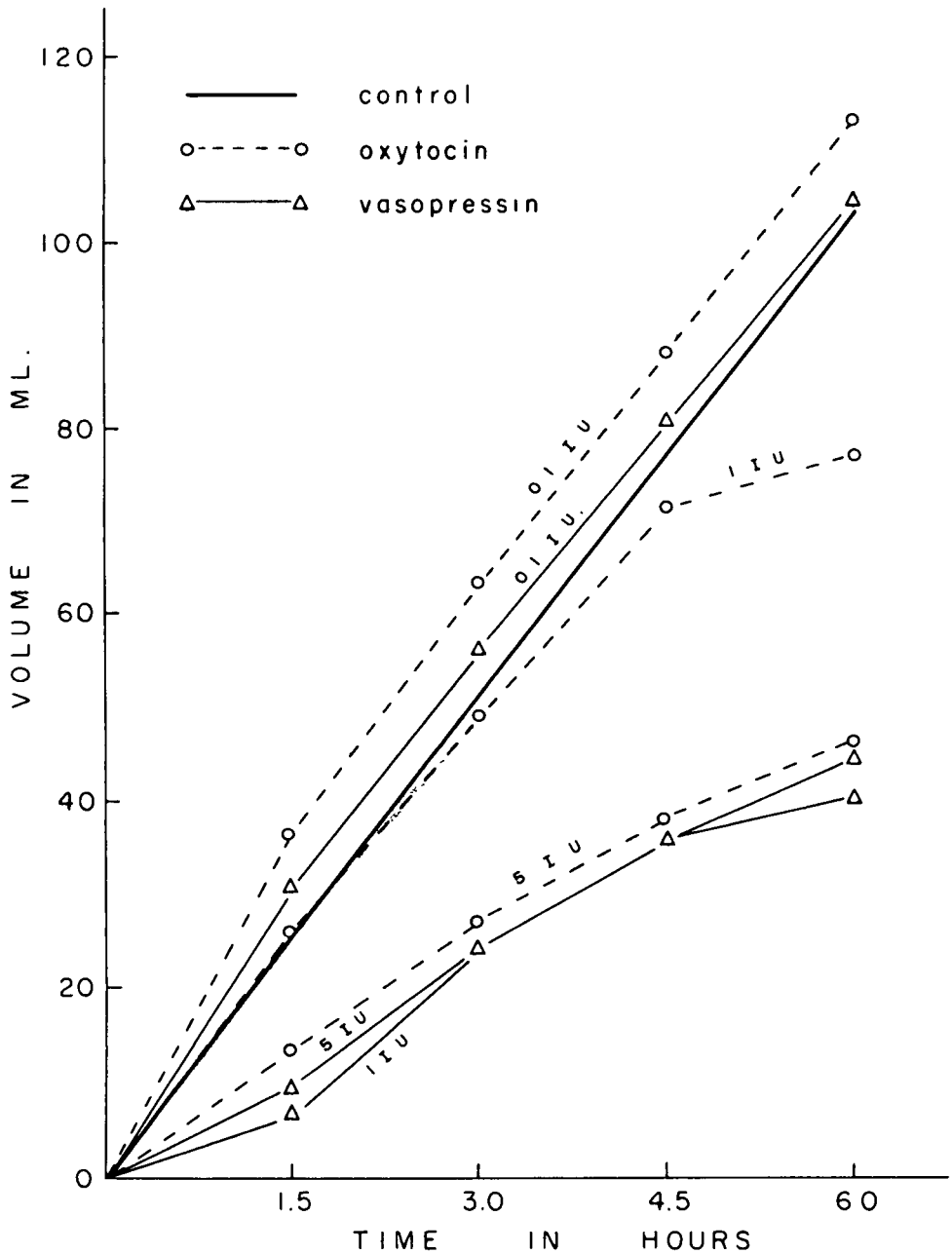

2.0
1.5
1.0
0.5
0
1.5
3.0
4.5
6.0
MILLIEQUIVALENTS OF SODIUM
TIME IN HOURS
control
corticosterone
cortisol
aldosterone
1.25 mg
50 µg
2.5 mg
1.25 mg
100 µg
2.5 mg

Figure 3. The accumulated renal outputs of potassium during a 6 hr. period from the water-loaded domestic duck. Each bird received 25 ml. of the distilled water at 0, 1.5, 3.0 and 4.5 hr. and the indicated doses of adrenocortical steroids were administered at -1.5, 0, 1.5, 3.0 and 4.5 hr.


3.0
2.0
1.0
MILLIEQUIVALENTS OF POTASSIUM
control
corticosterone
cortisol
aldosterone
1.25 mg
1.25 mg
2.5 mg
2.5 mg
50 µg
100 µg
0
1.5
3.0
4.5
6.0
TIME IN HOURS


120
100
80
60
40
20
0
VOLUME IN ML.
control
oxytocin
vasopressin
0.1 I U.
0.1 I U.
1 I U
5 I U
5 I U
1 I U
1.5
3.0
4.5
6.0
TIME IN HOURS

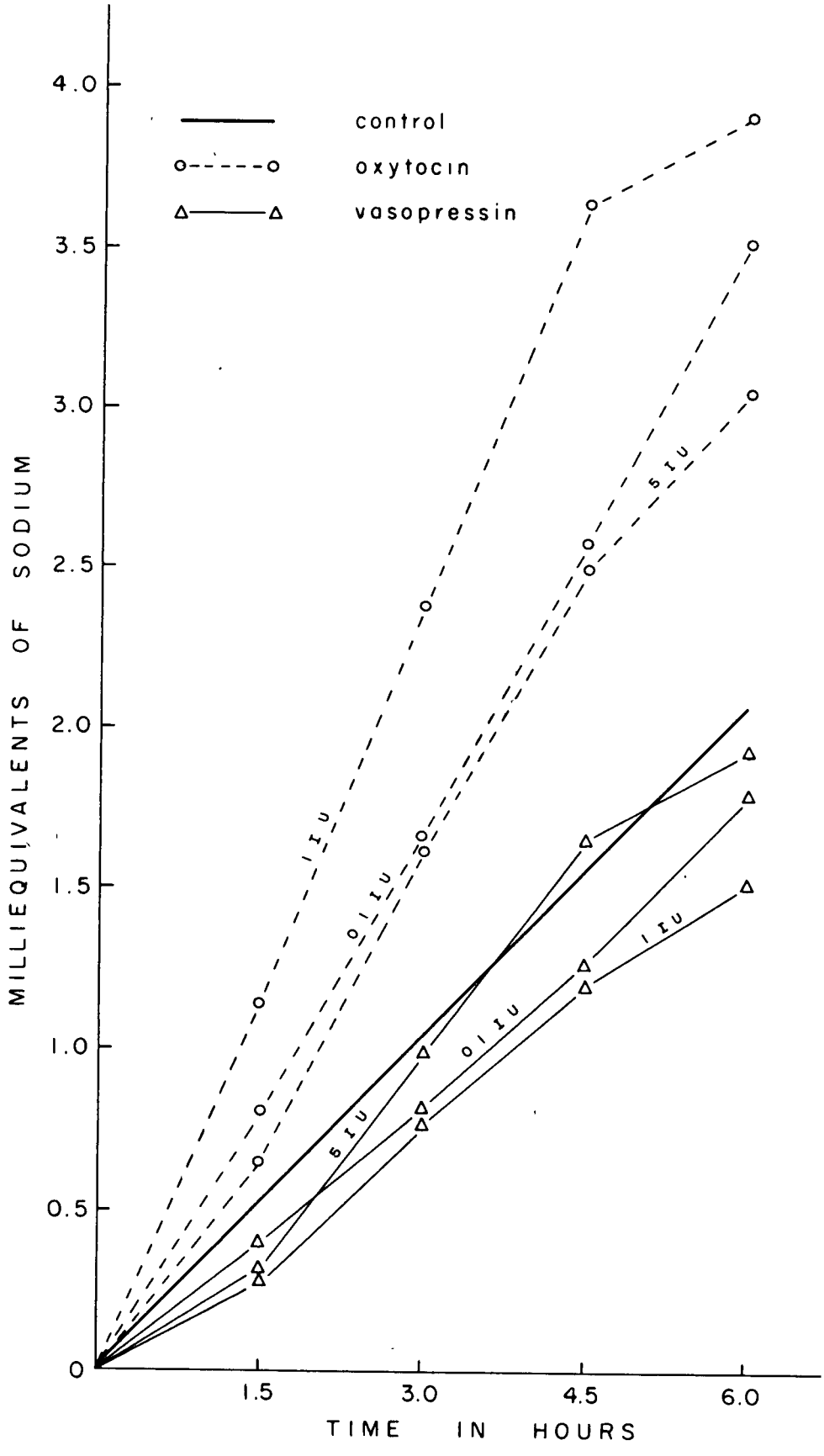
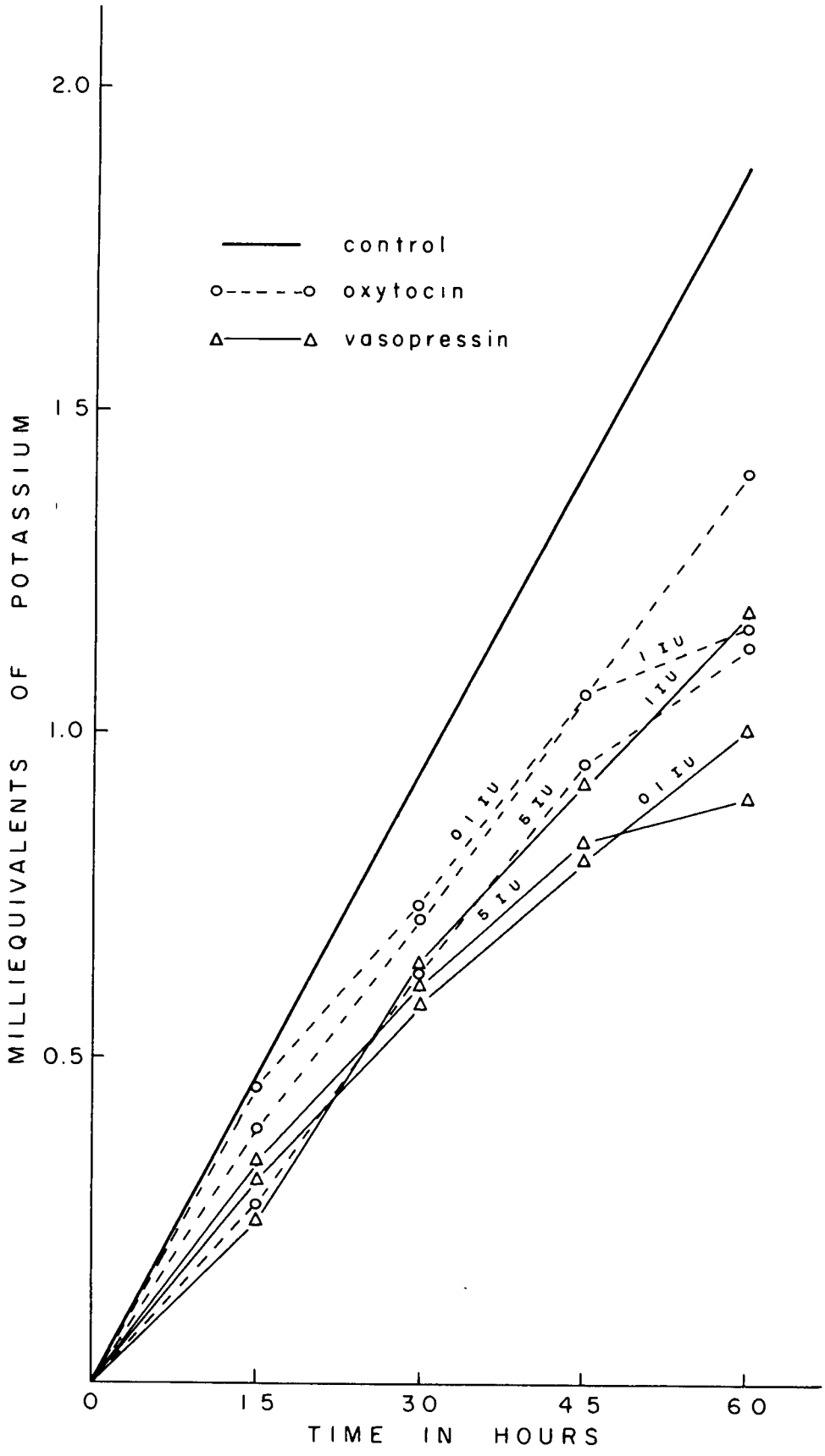

control
oxytocin
vasopressin
MILLIEQUIVALENTS OF SODIUM
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0
1.5
3.0
4.5
6.0
TIME IN HOURS
1 I U
0.1 I U
5 I U
5 I U
0.1 I U
1 I U

Figure 6. The accumulated renal outputs of potassium during a 6 hr. period from the water-loaded domestic duck. Each bird received 25 ml. of distilled water at 0, 1.5, 3.0 and 4.5 hr. and the indicated doses of mammalian posterior pituitary hormones were administered at -1.5, 0, 1.5, 3.0 and 4.5 hr.


2.0
1.5
1.0
0.5
0
15
30
45
60
MILLIEQUIVALENTS OF POTASSIUM
TIME IN HOURS
control
oxytocin
vasopressin
0.1 IU
5 IU
1 IU
0.1 IU
5 IU
1 IU

There was, therefore, no contamination of urine from this source in these birds.

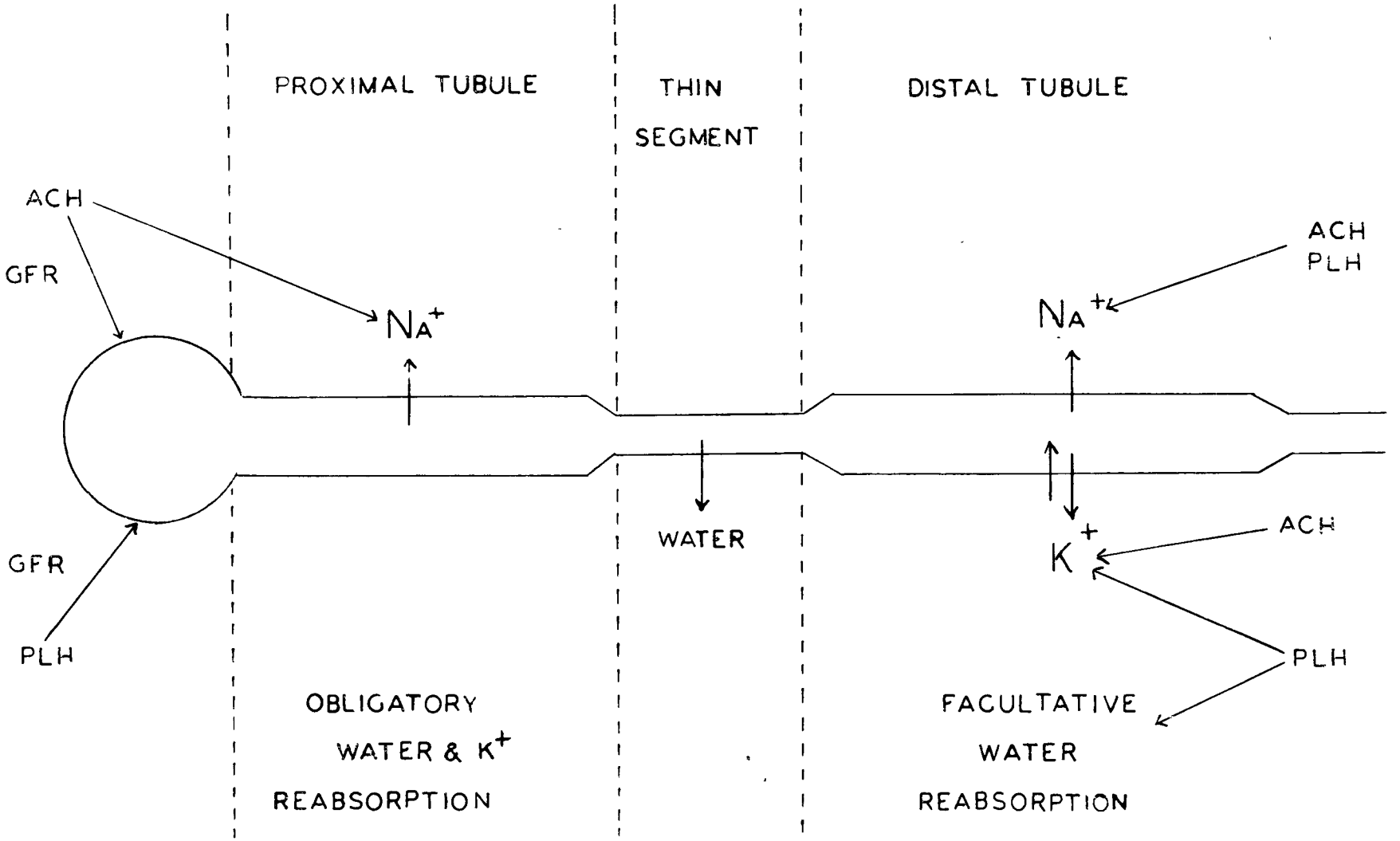
DISCUSSION

The influence of corticosteroids and the hormones of the posterior pituitary on the excretion of water, sodium and potassium in the eutherian mammals has been extensively reviewed (4, 9, 10, 11). Despite this, the explanation of renal variations and the precise delineation of the action of these hormones is by no means complete. In general, water and electrolyte movements between the blood and the lumen of a typical mammalian nephron may be schematically represented as in Figure 7. Possible sites of hormonal action are indicated. In the present investigation the response of the domestic duck to a water load was compared with the well known mammalian pattern. The duck, possibly as representative of the class Aves, shows differences from the expected pattern when this comparison is made. There is, however, a similarity between the two classes in that all the water is readily excreted as urine by the intact bird, over a 6 hr. period.

Cortisol and corticosterone generally increased the excretion of water to result in a negative water balance and we may conclude that these corticosteroids acted by increasing the glomerular filtration rate and/or by having a positive diuretic action. In this latter case, it is suggested that these steroids render the distal tubule less permeable to water, so that optimal water reabsorption does not occur.

Similarities with mammalian reactions can also be found in the results with posterior lobe hormones. In this case, however, the use of mammalian

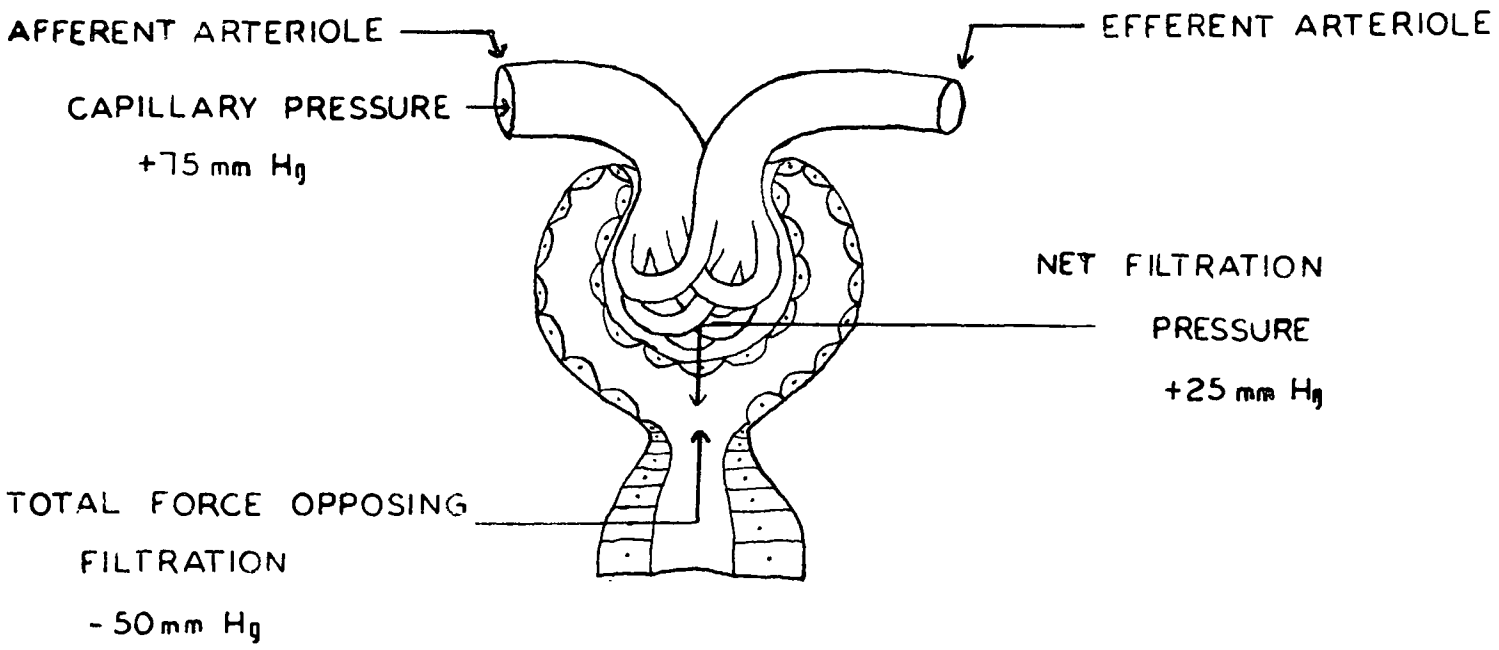
Figure 7. Possible sites of action of hormones on water, sodium and potassium movement between the circulation in the lumen of the nephron. ACH, adrenocortical hormones, PLH, posterior lobe hormones. (Adapted from Chester Jones, 1957).


PROXIMAL TUBULE
THIN SEGMENT
DISTAL TUBULE
ACH
GFR
Na^+
ACH
PLH
Na^+
WATER
GFR
PLH
K^+
ACH
PLH
OBLIGATORY WATER & K^+ REABSORPTION
FACULTATIVE WATER REABSORPTION

and commercial preparations of vasopressin and oxytocin may not completely simulate endogenous hormones of the bird, which we know to be vasotocin and oxytocin (12, 13, 14). In appropriate doses both oxytocin and vasopressin produced an antidiuresis in the duck but it is difficult to say in what measure these were physiological doses, since by mammalian standards they were high. It may be, if they were pharmacological doses in the duck, that the anti-diuresis was occasioned by the action of vasopressin and oxytocin reducing or increasing the diameter of the afferent and/or efferent glomerular arterioles to decrease the renal plasma flow (Fig. 8). Furthermore, oxytocin is known to have a vasodepressor effect in birds (15, 16, 17). Such changes in the systolic blood pressure would cause a general lowering of the effective filtration pressure and consequently reduce the plasma filtration rate across the glomerular membrane. Some support is given to this view by consideration of the lowest doses of both these hormones (0.1 I.U.) after which a slight diuresis was evident, although their effects on electrolyte excretion was still present. The bird is supposed to be capable of producing a hypertonic urine (4), though definitive evidence is lacking. In some birds cloacal urine may be a viscous paste of uric acid crystals (18) and this assumes a mechanism for water reabsorption. Vasotocin would act to aid this process, and, although the site of action is unknown, the distal tubule or cloaca would be possible sites. However, the evidence for the cloaca as a site of water reabsorption has been critisized (19).

The excretion of sodium and potassium depends, both as to rate and amount, on numerous factors. In this investigation we are drawing attention to hormonal influences. The control figures show that sodium and potassium

Figure 8. Factors affecting the net filtration pressure across the glomerular membrane. Reduced systic blood pressure constriction of the afferent arteriole or dilation of the efferent arteriole will result in reduced net filtration pressures. Converse changes in these factors will tend to increase the effective filtration pressure. (Adapted from Harper, Physiological Chemistry, 7th Edition).


AFFERENT ARTERIOLE
EFFERENT ARTERIOLE
CAPILLARY PRESSURE
+75 mm Hg
NET FILTRATION
PRESSURE
+25 mm Hg
TOTAL FORCE OPPOSING
FILTRATION
-50 mm Hg

are excreted, under conditions of water loading, at about the same millimolar concentration. This is unusual among the mammals, and may be among the birds. The normal human daily excretions of sodium and potassium are 135.2 and 73.5 m.eq./day respectively (20). This excretion represents a sodium/potassium ratio of 1.84 i.e. 84% greater than our observed value for the duck. The sodium/potassium ratio of unity in the Pekin duck has been further verified in birds receiving the water load over a 12 hr. period (Holmes and Adams, unpublished (21)). This equimolar excretion means that if we consider a G.F.R. as 2.0 ml./kg./min. (from figures supplied by Dr. A.H. Sykes, Wye College, Kent, England) then over 6 hr. the control ducks could have produced 1.54 liters of urine if there had been no tubular reabsorption. It would appear, therefore, that 94% tubular reabsorption occurred since the control birds produced only 100 ml. of urine. On the usual theory of renal dynamics (4) 80 to 90% of the water (and solutes) would be reabsorbed in the proximal tubule. The remainder of the reabsorption occurs distally. By similar calculations, therefore, if the Pekin duck plasma sodium concentration is 145.6 m.eq./liter (2) then the total glomerular filtrate of 1.54 liters would contain 224 m.eq. sodium. But in 6 hr. we have shown that the urine contained a total of 2.069 m.eq. of sodium. Thus 99% of the filtered sodium was reabsorbed, presumably 80 to 90% in the proximal tubule. These figures compare well with the similar human value of 99.4% (20) and thus it would appear that the handling of sodium by the domestic duck may be similar to the mammalian pattern.

As regards potassium, however, the duck shows an exceptionally large output. If 1.54 liters of glomerular filtrate is formed in 6 hr. it would be expected to contain, at a plasma potassium concentration of 3.79 m.eq./liter (2), 5.84 m.eq. of potassium. The total excretion of potassium in

six hours was, in fact, 1.897 m.eq. This is roughly 30% of the filtered potassium compared with a corresponding value for the human of 12% (20). There are reasons, from mammalian work, to suppose that all filtered potassium is reabsorbed in the proximal tubule (22). This means that potassium occurs in the distal tubular fluid by a process of re-secretion. If this pattern applies to the duck, as well it might, it means that re-secretion was a predominant phenomenon under the conditions of our experiments. In this, then, we may find the clue to the paradoxical effects of the corticosteroids and posterior lobe hormones on potassium excretion. The apparent ability of the duck kidney to excrete higher than normal amounts of potassium suggests a possible function under the condition of high dietary potassium. It has been suggested that the corticosteroids may control the extra-renal excretion of sodium in the marine bird (2, 3). Presumably the hormone under these conditions, would cause a concomitant retention of sodium in the renal tubule. However, this retention would be more than compensated for by the excretion of highly concentrated sodium from the nasal gland. In addition to sodium retention, low doses of cortisol and corticosterone enhance the renal excretion of potassium. Therefore these corticosteroids in low doses may have a dual effect in electrolyte excretion. They may influence the excretion of sodium by an extra-renal mechanism and the excretion of potassium from the kidney. A bird drinking seawater and eating marine animals would not only be presented with the problem of excreting sodium but in addition would have to excrete large amounts of potassium. The corticosteroids, of the type cortisol and corticosterone, in their ability to effect sodium retention and potassium excretion, differ in no way from the results fully documented

for mammals (4). However, with aldosterone, there was the surprising finding that the excretion of both sodium and potassium were diminished. It seems that aldosterone had an effect on the re-secretory mechanisms for potassium, but these mechanisms are unknown. However, Orloff and Burg (23), using the chicken, failed to observe any effects on the renal tubular electrolyte transport mechanism after injection of adrenocortical steroids, including aldosterone.

As far as the posterior lobe hormones are concerned the mammalian literature is not unanimous on their effect on sodium and potassium. The results of Chester Jones (10, 11) indicated that the posterior lobe hormones could enhance sodium excretion and Brooks and Pickford (24) confirmed this for oxytocin, since oxytocin bears a structural relationship to vasotocin, we may then be seeing the same phenomenon in the bird insomuch as the duck showed an increased excretion of sodium in response to 0.1 I.U. oxytocin even in the absence of an antidiuresis. The observed variations in potassium excretion are unexplained but the sensitivity of the potassium secretory mechanism, as postulated above, may well be the prime cause.

Recent observations have suggested that the extra-renal excretory mechanism in the duck is dependant upon a fully functional adrenal cortex (2, 3). The initial transient nasal secretion of the birds treated with cortisol and corticosterone lends support to these findings. The similar response found in ducks treated with 1.0 I.U. oxytocin introduces the interesting possibility that this hormone may be associated with ACTH release in the duck. There is agreement now that oxytocin and vasopressin are neurosecretory products discharged by the hypothalamic axons that terminate in the neural lobe of the hypophysis (25). Since there is anatomical evidence

that some of these axons terminate in the median eminence, it is possible that they may make contact with the hypophysial portal system. The liberated hormones, or some allied substances, might be delivered by the portal vessels to the pars distalis where they function as ACTH release factors (26, 27, 28, 29 and 30). Experiments based on this type of rationale may prove to be a profitable field for further examinations of electrolyte excretion, both renal and extra-renal, in marine birds.

SUMMARY

The effects of posterior lobe and adrenocortical hormones on the renal excretory pattern in the domestic duck were investigated. In the control group all introduced water was excreted during the period of observation. Oxytocin and vasopressin, in the higher doses, caused an antidiuresis but cortisol and corticosterone had a diuretic effect. It may be that these hormones were effecting the G.F.R. and/or the water reabsorption mechanism. Aldosterone had no apparent effect on the excretion of water.

A sodium retention was observed with all hormones except oxytocin, which, despite its antidiuretic effect enhanced sodium excretion.

The urinary output of potassium in the controls was high when compared with mammalian values. Potassium excretion was enhanced by the administration of cortisol and corticosterone. Posterior lobe hormones and aldosterone caused a potassium retention.

The observed effects of cortisol and corticosterone on the renal excretion of potassium and on the extra-renal excretion of sodium suggests a

possible function in marine birds. These steroids may act on the renal distal tubule secretory mechanism for potassium.

An initial transient nasal drip was observed with oxytocin, cortisol and corticosterone. These observations are considered to raise the possibility of oxytocin as an ACTH release factor.

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APPENDICES

Table I. The urinary excretion of water, sodium and potassium from the intact control domestic duck during a six hr. period. Each bird received 4 X 25 ml. doses of distilled water at 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	28.9 ±3.2	18.2 ±2.8	22.2 ±4.1	0.504 ±0.073	0.555 ±0.060	1.008 ±0.169
1.5–3.0	25.4 ±2.4	17.6 ±1.9	15.0 ±1.2	0.452 ±0.052	0.379 ±0.040	1.169 ±0.104
3.0–4.5	21.5 ±1.5	24.5 ±2.0	21.9 ±1.7	0.529 ±0.059	0.456 ±0.032	1.157 ±0.100
4.5–6.0	24.1 ±0.82	24.1 ±2.3	20.8 ±1.4	0.585 ±0.065	0.506 ±0.045	1.167 ±0.085
Total	99.9 ±1.9	20.8 ±2.0	19.1 ±1.3	2.069 ±0.185	1.897 ±0.119	1.107 ±0.089

Table I

(Controls)

Table II. The effect of corticosterone on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. period. Each bird was water loaded similarly to the intact control birds. 2.5 mg. of corticosterone was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0-1.5	36.5 ±3.9	9.1 ±0.8	14.9 ±3.5	0.338 ±0.055	0.561 ±0.162	0.856 ±0.163
1.5-3.0	27.6 ±4.2	8.5 ±1.9	18.5 ±1.8	0.253 ±0.079	0.508 ±0.091	0.533 ±0.169
3.0-4.5	24.8 ±1.6	5.3 ±1.4	22.6 ±2.0	0.133 ±0.039	0.542 ±0.048	0.344 ±0.094
4.5-6.0	30.6 ±2.5	4.3 ±1.1	27.9 ±1.7	0.131 ±0.026	0.855 ±0.091	0.230 ±0.061
Total	119.5 ± 4.1	7.1 ±1.0	20.9 ±2.1	0.855 ±0.135	2.466 ±0.227	0.418 ±0.088

Table II

(2.5 mg. Corticosterone)

Table III. The effect of corticosterone on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water loaded similarly to the intact control birds. 1.25 mg. of corticosterone was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	34.4 ±3.7	9.0 ±1.4	19.3 ±1.7	0.313 ±0.057	0.635 ±0.058	0.529 ±0.105
1.5–3.0	26.8 ±1.7	8.8 ±1.8	21.8 ±2.0	0.237 ±0.064	0.574 ±0.055	0.334 ±0.068
3.0–4.5	27.5 ±1.6	6.7 ±1.1	28.9 ±1.6	0.192 ±0.036	0.754 ±0.080	0.259 ±0.047
4.5–6.0	22.6 ±1.6	4.7 ±0.9	33.1 ±2.3	0.114 ±0.027	0.753 ±0.067	0.144 ±0.029
Total	111.3 ± 6.0	7.5 ±0.8	24.4 ±1.4	0.856 ±0.137	2.716 ±0.213	0.320 ±0.041

Table III

(1.25 mg. Corticosterone)

Table IV. The effect of cortisol on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds and they were injected with 2.5 mg. of cortisol at the same times as the corticosterone treated birds.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	41.7 ±1.9	9.0 ±1.2	11.1 ±1.6	0.367 ±0.049	0.467 ±0.067	0.879 ±0.145
1.5–3.0	26.4 ±0.8	4.8 ±0.7	16.5 ±0.9	0.125 ±0.019	0.433 ±0.016	0.289 ±0.041
3.0–4.5	24.9 ±0.7	2.5 ±0.4	23.1 ±2.1	0.061 ±0.009	0.583 ±0.069	0.160 ±0.057
4.5–6.0	24.9 ±2.6	1.9 ±0.3	21.5 ±1.9	0.050 ±0.010	0.573 ±0.070	0.090 ±0.014
Total	119.4 ± 3.4	5.1 ±0.4	17.1 ±1.1	0.604 ±0.036	2.057 ±0.158	0.312 ±0.035

Table IV

(2.5 mg. Cortisol)

Table V. The effect of cortisol at the 1.25 mg. dose level on the excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded and injected as in the 2.5 mg. series.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0-1.5	44.6 ±2.1	14.3 ±3.1	18.4 ±3.1	0.666 ±0.149	0.799 ±0.126	0.980 ±0.174
1.5-3.0	28.6 ±1.6	7.0 ±1.1	22.5 ±2.4	0.207 ±0.042	0.643 ±0.078	0.339 ±0.069
3.0-4.5	23.5 ±1.1	5.1 ±0.8	30.4 ±3.1	0.121 ±0.021	0.705 ±0.066	0.184 ±0.028
4.5-6.0	27.3 ±2.3	5.8 ±0.9	28.3 ±3.7	0.160 ±0.028	0.758 ±0.121	0.228 ±0.045
Total	124.0 ± 2.6	9.3 ±1.8	23.6 ±3.0	1.154 ±0.210	2.905 ±0.338	0.428 ±0.069

Table V

(1.25 mg. Cortisol)

Table VI. The effect of aldosterone on the excretion of water sodium and potassium from the domestic duck during a six hr. observation period. Each bird was loaded similarly to the intact control birds. 100 µg. of d.l.aldosterone was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	33.5 ±3.6	7.1 ±1.6	5.5 ±0.3	0.252 ±0.082	0.190 ±0.034	1.223 ±0.245
1.5–3.0	23.8 ±1.3	8.8 ±1.2	6.2 ±1.0	0.193 ±0.037	0.143 ±0.022	1.111 ±0.189
3.0–4.5	16.9 ±2.6	9.3 ±2.1	6.1 ±1.1	0.148 ±0.038	0.097 ±0.018	1.913 ±0.629
4.5–6.0	20.0 ±2.7	5.3 ±0.8	7.2 ±1.2	0.105 ±0.019	0.156 ±0.039	0.840 ±0.149
Total	94.2 ±4.4	7.3 ±1.0	6.1 ±0.7	0.698 ±0.123	0.586 ±0.076	1.251 ±0.217

Table VI

(100 μg. d.l. Aldosterone)

Table VII. The effect of aldosterone at the 50 μg. dose level on the excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded and injected as in the 100 μg. series.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0-1.5	30.4 ±3.0	11.2 ±3.1	7.9 ±1.1	0.377 ±0.115	0.247 ±0.047	1.495 ±0.305
1.5-3.0	22.3 ±2.7	12.2 ±3.5	7.9 ±0.7	0.234 ±0.065	0.168 ±0.024	1.411 ±0.288
3.0-4.5	25.3 ±3.3	9.3 ±2.4	6.7 ±1.0	0.274 ±0.058	0.155 ±0.018	1.633 ±0.091
4.5-6.0	21.3 ±0.9	8.4 ±1.5	6.5 ±0.8	0.183 ±0.034	0.136 ±0.015	1.356 ±0.278
Total	99.3 ±.37	10.5 ±2.1	7.0 ±0.6	1.068 ±0.215	0.706 ±0.070	1.472 ±0.224

Table VII

(50 μg. d.l. Aldosterone)

Table VIII. The effect of oxytocin on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds. 5.0 I.U. oxytocin was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0-1.5	13.3 ±3.1	58.4 ±8.4	22.6 ±2.9	0.675 ±0.163	0.273 ±0.064	2.719 ±0.361
1.5-3.0	14.3 ±2.1	69.6 ±8.0	24.0 ±2.9	0.952 ±0.201	0.360 ±0.085	3.267 ±0.620
3.0-4.5	11.0 ±2.0	78.2 ±8.4	28.7 ±2.9	0.876 ±0.183	0.319 ±0.070	3.103 ±0.527
4.5-6.0	7.9 ±1.0	75.8 ±9.3	29.9 ±2.6	0.563 ±0.078	0.223 ±0.024	2.691 ±0.451
Total	46.5 ±2.0	71.9 ±8.4	25.4 ±1.7	3.066 ±0.457	1.175 ±0.195	2.904 ±0.382

Table VIII

(5.0 I.U. Oxytocin)

Table IX. The effect of oxytocin on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds. 1.0 I.U. oxytocin was administered intramuscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	25.3 ±2.2	44.9 ±3.1	15.6 ±2.1	1.104 ±0.111	0.399 ±0.071	3.802 ±0.804
1.5–3.0	24.6 ±2.3	51.7 ±2.4	13.3 ±1.5	1.284 ±0.153	0.321 ±0.050	5.052 ±1.012
3.0–4.5	22.0 ±1.7	55.6 ±2.4	16.3 ±1.9	1.216 ±0.104	0.347 ±0.046	4.325 ±0.793
4.5–6.0	5.8 ±2.0	52.3 ±3.6	16.0 ±2.4	0.307 ±0.010	0.100 ±0.040	3.877 ±0.625
Total	77.7 ±5.7	50.9 ±2.0	14.9 ±1.6	3.911 ±0.307	1.167 ±0.158	4.195 ±0.740

Table IX

(1.0 I.U. Oxytocin)

Table X. The effect of oxytocin on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds. 0.1 I.U. oxytocin was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0-1.5	36.5 ±3.5	22.4 ±3.3	12.5 ±1.6	0.811 ±0.125	0.463 ±0.078	1.904 ±0.281
1.5-3.0	26.8 ±1.8	33.5 ±4.3	10.3 ±0.8	0.854 ±0.083	0.272 ±0.026	3.500 ±0.544
3.0-4.5	25.3 ±1.4	35.4 ±2.5	12.7 ±0.8	0.913 ±0.101	0.318 ±0.023	2.948 ±0.372
4.5-6.0	24.9 ±0.9	37.8 ±2.0	14.2 ±0.4	0.946 ±0.068	0.353 ±0.013	2.691 ±0.189
Total	113.5 ±3.4	30.9 ±2.4	12.5 ±0.8	3.542 ±0.309	1.406 ±0.088	2.579 ±0.282

Table X

(0.1 I.U. Oxytocin)

Table XI. The effect of vasopressin on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds. 5.0 I.U. vasopressin was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0-1.5	9.4 ±2.8	34.8 ±5.2	44.1 ±8.4	0.323 ±0.110	0.340 ±0.085	0.956 ±0.186
1.5-3.0	15.3 ±4.4	49.6 ±8.1	22.8 ±4.7	0.667 ±0.158	0.299 ±0.077	2.454 ±0.417
3.0-4.5	11.9 ±3.4	70.6 ±15.4	20.6 ±5.3	0.660 ±0.171	0.191 ±0.044	3.904 ±0.661
4.5-6.0	3.8 ±1.4	66.2 ±18.6	15.9 ±5.0	0.274 ±0.144	0.069 ±0.037	4.652 ±0.900
Total	40.3 ±6.9	56.8 ±9.2	23.7 ±2.3	1.924 ±0.305	0.900 ±0.153	2.540 ±0.519

Table XI

(5.0 I.U. Vasopressin)

Table XII. The effect of vasopressin on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds. 1.0 I.U. vasopressin was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	7.0 ±2.4	46.6 ±9.5	47.3 ±13.1	0.287 ±0.092	0.257 ±0.077	1.224 ±0.236
1.5–3.0	17.0 ±2.5	32.4 ±7.4	27.9 ±6.1	0.496 ±0.088	0.392 ±0.211	1.509 ±0.320
3.0–4.5	12.4 ±1.5	35.4 ±3.5	24.1 ±3.1	0.422 ±0.052	0.289 ±0.044	1.623 ±0.185
4.5–6.0	9.5 ±1.7	34.3 ±5.4	29.6 ±3.4	0.307 ±0.070	0.258 ±0.044	1.207 ±0.187
Total	45.9 ±6.5	33.5 ±3.3	29.8 ±4.4	1.512 ±0.247	1.199 ±0.119	1.276 ±0.170

Table XII

(1.0 I.U. Vasopressin)

Table XIII. The effect of vasopressin on the urinary excretion of water, sodium and potassium from the domestic duck during a six hr. observation period. Each bird was water-loaded similarly to the intact control birds. 0.1 I.U. vasopressin was administered intra-muscularly at -1.5, 0, 1.5, 3.0 and 4.5 hr.

Time hr.	Volume ml.	Concentration		Total Excretion		
		Na m.eq./l.	K m.eq./l.	Na m.eq.	K m.eq.	Na/K m.eq.
0–1.5	30.8 ±1.8	13.8 ±2.0	11.8 ±2.4	0.405 ±0.049	0.341 ±0.029	1.277 ±0.185
1.5–3.0	25.5 ±1.6	16.4 ±1.9	9.7 ±1.1	0.418 ±0.054	0.244 ±0.032	1.791 ±0.208
3.0–4.5	24.5 ±3.4	19.1 ±2.3	8.0 ±1.6	0.466 ±0.086	0.223 ±0.047	2.222 ±0.297
4.5–6.0	23.4 ±3.1	25.1 ±4.4	13.5 ±3.3	0.524 ±0.091	0.266 ±0.050	2.126 ±0.309
Total	104.2 ±2.5	17.4 ±1.9	10.3 ±1.0	1.813 ±0.197	1.074 ±0.116	1.772 ±0.218

Table XIII

(0.1 I.U. Vasopressin)