Effects of ADH on single-nephron glomerular filtration rates in the avian kidney

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Effects of ADH (arginine vasotocin) on the total-kidney glomerular filtration rate (GFR) and on single-nephron glomerular filtration rates (SNGFR) of reptilian type (RT) and mammalian type (MT) nephrons were studied in desert quail, Lophortyx gambelii. Low, probably physiological, doses (10 and 50 μg/kg) of arginine vasotocin (AVT) had no effect on mean arterial blood pressure, but caused reduction in total-kidney GFR primarily as a result of the decrease in the number of filtering RT nephrons. High, probably pharmacological, doses (200 μg/kg) of AVT produced increases, decreases, or biphasic changes in mean arterial blood pressure. With decreases or biphasic changes in mean arterial blood pressure, total-kidney GFR, SNGFR of MT nephrons, and the number of filtering RT nephrons decreased. With increases in mean arterial pressure, total-kidney GFR decreased, SNGFR of MT nephrons increased, and RT nephrons ceased filtering. The possible changes in intrarenal blood flow responsible for these effects on SNGFR, the possible relationship of effects of AVT and salt load on SNGFR, and the possible relationship of changes in the number of filtering RT nephrons to concentrating ability are discussed.

The avian kidney contains nephrons resembling those seen in mammalian kidneys and nephrons resembling those seen in reptilian kidneys. The reptilian-type (RT) nephrons, which are located superficially in the kidney, empty at right angles into collecting ducts and do not function together to contribute directly to the concentrating mechanism of the kidney. The mammalian-type (MT) nephrons are situated deeply in relation to the reptilian-type nephrons. They have long loops of Henle which are bound together with vasa recta and collecting ducts in structures termed medullary cones. This arrangement, as in mammalian kidneys, appears to permit the avian kidney to produce a concentrated urine (8, 18). The details of these anatomical relationships have been described previously (2). Only a small fraction of the nephrons are of the mammalian type (about 10% in the desert quail) (2), and the concentrating ability of the avian kidney is quite limited.

In previous work with the desert quail, Lophortyx gambelii (2), we measured the single-nephron glomerular filtration rates (SNGFR) of both the MT and RT nephrons using de Rouffignac, Dics, and Bonvalet’s (7) modification of Hanssen’s (9) sodium ferrocyanide technique. The filtration rates of individual MT nephrons were more than twice those of individual RT nephrons during a control diuresis. In the same study, the intravenous infusion of a hyperosmotic sodium chloride solution produced a fall in total-kidney GFR, resulting almost entirely from a decrease in the number of filtering RT nephrons. The factors responsible for regulating this reduction in the number of filtering RT nephrons are unknown. However, arginine vasotocin (AVT), the naturally occurring antidiuretic principle in all birds, reptiles, and amphibians, appears to regulate the number of filtering nephrons in reptiles (4) and amphibians (10, 20). Earlier studies (3, 18) showed no effect of even massive doses of AVT on avian total-kidney GFR. However, more recent studies (1) have suggested that AVT may reduce avian GFR. Moreover, recent studies have suggested that, in mammals, antidiuretic hormone may enhance the filtration rate of juxtamedullary nephrons (6). Since the hyperosmotic sodium chloride infusion increased the plasma osmolality as would dehydration, it seemed possible that its glomerular effects might be mediated by the release of AVT.

To examine this hypothesis, we administered AVT to desert quail during the same control diuresis as that used previously and measured the total-kidney GFR and the SNGFRs of the MT and RT nephrons. The results indicate that: 1) even small, apparently physiological, doses of AVT can produce a reduction in total kidney GFR which results primarily from a reduction in the number of filtering RT nephrons; and 2) high, probably pharmacological, doses of AVT produce alterations in mean arterial blood pressure, a reduction in total-kidney GFR, a reduction in the number of filtering RT nephrons, and also significant changes in the SNGFR of MT nephrons.

Methods

Animals and operative procedures. Desert quail, Lophortyx gambelii, were used as experimental animals. The quail weighed from 131 to 167 g (mean weight: 151 g). They were trapped in their native habitat near Tucson, Ariz. (Trapping Permit no. 49 from Arizona Game and Fish Department) and were maintained in an outside aviary exposed to natural environmental conditions. Twelve birds were used in the present study. In captivity, the birds had free access to a cracked-grain mixture and green fodder and were allowed water ad libitum. Operative procedures were essentially the same as those described previously (2).
Briefly, the animals were anesthetized with Equithesin (Jensen-Salsbery, Kansas City, Mo.) (3 ml/kg) prior to the operative procedures. A 9% lidocaine solution was used as a local anesthetic during the operations. During the operative procedures and experiments, the birds were restrained by taping their legs and wings to a specially designed bird board. The left brachial vein and artery were cannulated for intravenous infusions and collections of arterial blood samples, respectively. Ureteral urine was collected by exteriorizing the ureters and cannulating them with PE-50 polyethylene cannulas as previously described (2). Dead space in each ureteral cannula was 18–20 μl.

Renal function studies. A control diuresis (mean value 0.182 ± 0.0077 ml/kg per min; Table 1) was produced, as in the previous study (2), by a constant intravenous infusion of 2.5% mannitol at 0.4 ml/kg per min. Both total-kidney glomerular filtration rates (GFR) and single-nephron glomerular filtration rates (SNGFR) were estimated with sodium ferrocyanide- 14C. The validity of this substance as a marker for estimating glomerular filtration rates in these animals was established previously (2). The single-nephron glomerular filtration rates were determined with the modification of Hansen’s (9) technique developed by De Rouffignac, Diess, and Bonvalet, (7). A complete description of the technique as modified in our laboratory for use in measuring SNGFRs in avian kidneys has been published before (2).

In the experiments described here, a priming dose of 2 μCi sodium ferrocyanide-14C (Schwarz/Mann, Orangeburg, N.Y.; sp act: 11–32 mCi/mmol depending on the lot used) was given followed by a sustained infusion of 20 μCi/ml in the intravenous mannitol infusion. At least 40 min were allowed for equilibration of the isotope before the control clearance periods were started. Eight 5-min control clearance periods were taken before the appropriate dose of arginine vasotocin was administered. The hormone was given through the venous cannula and washed in with the 2.5% mannitol infusion. Two 5-min clearance periods were taken after the hormone was given. A final arterial blood sample was taken 9 min after the AVT was administered.

TABLE 1. Renal function during control periods

<table>
<thead>
<tr>
<th>Arginine Vasotocin Dose, μg/kg</th>
<th>10</th>
<th>50</th>
<th>200</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine flow*</td>
<td>0.209</td>
<td>0.153</td>
<td>0.169</td>
<td>0.182</td>
</tr>
<tr>
<td>GFR*</td>
<td>1.108</td>
<td>1.230</td>
<td>0.890</td>
<td>1.025</td>
</tr>
<tr>
<td>Osm (U/P)r.M</td>
<td>0.025</td>
<td>0.0397</td>
<td>0.0309</td>
<td>0.0247</td>
</tr>
<tr>
<td></td>
<td>± 0.25</td>
<td>± 0.397</td>
<td>± 0.309</td>
<td>± 0.247</td>
</tr>
<tr>
<td></td>
<td>0.0402</td>
<td>0.0857</td>
<td>0.0403</td>
<td>0.0300</td>
</tr>
<tr>
<td></td>
<td>± 0.0402</td>
<td>± 0.0857</td>
<td>± 0.0403</td>
<td>± 0.0300</td>
</tr>
<tr>
<td></td>
<td>1.090</td>
<td>0.890</td>
<td>0.917</td>
<td>0.850</td>
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<td>± 0.0402</td>
<td>± 0.0857</td>
<td>± 0.0403</td>
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<td>± 0.0402</td>
<td>± 0.0857</td>
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All values are means ± 1SEM with the number of clearance periods in parentheses. * ml/kg per min.

RESULTS

Effect of arginine vasotocin on mean arterial blood pressure. The mean arterial blood pressure before the administration of AVT averaged 112 mmHg for all the animals studied (range: 100–130 mmHg). No change in mean arterial pressure occurred following the administration of 50 ng or less of AVT per kilogram of body weight (see Fig. 1 for representative recording of effect of this dose of AVT). Mean arterial blood pressure did change following the administration of 200 ng AVT/kg, but the response varied in different animals. In some animals there was an immediate increase of about 20 mm Hg in the mean arterial pressure followed by a slow return to the control levels (see Figs. 1 and 2 for representative recordings of this response). In other animals, the mean arterial pressure decreased by as much as 30 mmHg and then returned to control levels in
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FIG. 1. Changes in blood pressure, GFR, urine flow, and $T_{\text{H}_{2}\text{O}}/\text{GFR}$ following administration of 2 doses of AVT in a single animal. Note that scales on ordinate (except that for blood pressure) are logarithmic.

FIG. 2. Representative recordings showing 3 systemic blood pressure responses to intravenous injections of 200 ng AVT/kg. A depressor response is shown at A, a biphasic response at B, and a depressor at C.

less than 10 min (see Fig. 2 for a representative recording of this response). In still other animals, the blood pressure response following this dose of AVT was biphasic. The initial response in these animals was a decrease in mean arterial pressure to about 10 mmHg below the control level. Secondarily, the pressure increased to about 10 mmHg above the control level. The pressure then returned to control levels following this overshoot (see Fig. 2 for a representative recording of this response). These varied responses of the mean arterial blood pressure to the administration of 200 ng AVT/kg were not related to the sex of the birds.

Effect of arginine vasotocin on urine flow, GFR, and relative free-water clearance. The renal responses to AVT were evaluated at each of the following dose levels: 1) 10 ng/kg body wt or about 2.25 vasopressor mU/kg; 2) 50 ng/kg or about 11.25 vasopressor mU/kg; 3) 200 ng/kg or about 45 vasopressor mU/kg. The values for urine flow and total-kidney GFR during control periods prior to the administration of each of these doses of AVT are shown in Table 1. The mean values for urine flow and total-kidney GFR for all control values are also shown in Table 1. These control values are nearly identical with those obtained previously (2) under the same conditions in desert quail (previous control urine flow: $0.186 \pm 0.008$ ml/kg per min for 101 clearance periods; previous total-kidney GFR: $0.882 \pm 0.036$ ml/kg per min for 97 clearance periods).

Urine flow and total-kidney GFR decreased following the administration of each dose of AVT (Fig. 3). Ten minutes after the injection of AVT, at the time when the single-nephron glomerular filtration rates were measured, the urine flow had decreased to about 30–60% of the control level in each case, although the decrease tended to be greatest at the highest dose level (Fig. 3). At this time, the total-kidney GFR had also decreased to about 60–80% of the control level, the greatest decrease occurring after the administration of 200 ng AVT/kg (Fig. 3). This pattern of decreases in urine flow and total-kidney GFR following the administration of 50 ng AVT/kg and 200 ng AVT/kg is shown for a single animal in Fig. 1.

Two of the experimental groups of animals, those receiving 10 ng AVT/kg and those receiving 200 ng AVT/kg, were producing a urine hyposmotic to the plasma during the control clearance periods before the administration of AVT. This is shown by the osmolar urine-to-plasma (U/P) ratios in Table 1. The free-water clearance ($C_{\text{H}_{2}\text{O}}$) in both these groups of animals was slightly positive and averaged about 3–5% of the filtration rate during the control periods (Fig. 4). This relative free-water clearance ($C_{\text{H}_{2}\text{O}}/\text{GFR}$) decreased markedly 10 min following the administration of 200 ng AVT/kg (Fig. 4), as the urine became essentially isosmotic with the plasma. Following the administration of 10 ng AVT/kg, $C_{\text{H}_{2}\text{O}}/\text{GFR}$ actually increased slightly (Fig. 4), but the change was so small that it is probably not meaningful. In the group of animals receiving 50 ng AVT/kg, the urine was essentially isosmotic with the plasma during the control periods (Table 1). Following the adminis-
Effect of arginine vasotocin on SNGFR. Following the administration of AVT, changes occurred in the filtration rates of individual mammalian-type and reptilian-type nephrons and in the total number of filtering RT nephrons. After the injection of 10 ng AVT/kg body wt, the SNGFRs of both the MT and RT nephrons were reduced compared to the control values determined previously (Fig. 5) (2). The mean SNGFR for the MT nephrons was 11.3 nl/min compared to a mean control value of 14.6 nl/min. The difference between these mean SNGFRs is significant at the 0.01 level of probability. The SNGFR for the RT nephrons also was reduced from a mean control value of 6.4 nl/min to a mean value of 4.7 nl/min (Fig. 5), but the difference between these mean values is not statistically significant (0.1 < P < 0.2). There was, however, a reduction in the fraction of filtering RT nephrons from 71% during the control periods to 52% following the administration of 10 ng AVT/kg (Fig. 5). The fraction of RT nephrons filtering was determined, as in the previous study (2), from the number of dissected RT nephrons in which the blue ferrocyanide precipitate was found. This was also confirmed in the previous study (2) by histological examination of the number of open and closed proximal tubule lumina.

No statistically significant change in the mean SNGFR of MT or RT nephrons from the control level occurred following the administration of 50 ng AVT/kg (Fig. 5). Moreover, the mean SNGFR for the MT nephrons (16.5 nl/min) was significantly higher (P < 0.001) than that following the administration of 10 ng AVT/kg (Fig. 5). However, the fraction of filtering RT nephrons fell to 26% following the administration of 50 ng AVT/kg compared to 71% during control periods and 52% following the administration of 10 ng AVT/kg (Fig. 5).

There appeared to be two major patterns of changes in SNGFRs following the administration of 200 ng AVT/kg. One pattern occurred in two animals in which a decrease or biphasic response in mean arterial blood pressure was observed. In one pattern, the mean SNGFR for the MT nephrons (7.3 nl/min) was significantly lower (P < 0.001) than the mean control value (14.6 nl/min) (Fig. 5). At the same time, the mean SNGFR for the RT nephrons was unchanged from the control level (Fig. 5). However, the fraction of filtering RT nephrons was reduced to 43% compared to 71% during the control periods (Fig. 5).

The second pattern was observed in two animals in which an increase in mean arterial blood pressure appeared to predominate. In this pattern, no RT nephrons appeared to be filtering (Fig. 5). A similar pattern for the RT nephrons was observed previously when the quail were given an intravenous salt load (Fig. 5) (2). However, the SNGFRs for the MT nephrons actually increased, compared to the control values, in these animals following the administration of 200 ng AVT/kg (Fig. 5). These SNGFRs for the MT nephrons were divided into two groups for the purposes of presentation in Fig. 5. This could be done because most of the SNGFRs were tightly grouped around a mean value of 24.7
ney GFR, the SNGFR, and the number of filtering reptilian-type nephrons. Although the sodium ferrocyanide technique makes possible the direct evaluation of the glomerular filtration rates of individual mammalian-type and reptilian-type nephrons throughout the avian kidney (2), it does not permit evaluation of SNGFRs in the same kidney both before and after an experimental manipulation. Therefore, the SNGFRs determined following the administration of AVT had to be compared with control values obtained in other animals. The control SNGFRs used for comparison in the present study were those obtained during an identical control infusion with animals showing this pattern of response and also higher than the measured clearance from the same kidneys. As noted above (see RESULTS), the total-kidney GFR and the urine flow during the control infusion in the present study were almost exactly the same as those observed in the previous study (2).

The total-kidney GFR following the administration of 10 ng AVT/kg was calculated from the SNGFRs was about 25% higher than the measured clearance from the same kidneys. The total-kidney GFR following the administration of 50 ng AVT/kg calculated from the SNGFRs was almost identical with the measured clearance for the same kidneys. As noted previously (2), the glomerular counts are difficult to perform accurately, and it is even more difficult to determine the proportions of MT and RT nephrons exactly. Considering the possible variables in these measurements, the total-kidney GFRs calculated from the SNGFRs in the present study are in quite good agreement with the measured GFRs. Moreover, this agreement between calculated and measured GFRs is as good as or better than that found for the control and salt-loaded animals in the previous study (2). As noted above (see RESULTS), the total-kidney GFR and the urine flow during the control infusion in the present study were almost exactly the same as those observed in the previous study (2).

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crease could be explained by dead space errors. In addition, the very close agreement between the measured total-kidney GFR and that calculated from the SNGFRs strongly supports the idea that the decrease in GFR is a real one even at low dose levels. This contrasts with the findings of Ames et al. (1) in domestic chickens, in which decreases in GFR following low doses of AVT (5-30 ng/kg) appeared to result largely from dead-space errors. Although some of this difference might have been the result of a difference between species in sensitivity to AVT, the quail in the present study were also in a less-marked water diuresis than the chickens in the study by Ames et al. (1). Under these circumstances, a small dose of AVT might have been more likely to produce a decrease in total-kidney GFR. However, as noted above, the control diuresis in the present study was established to match that in the previous study (2), in which an infusion of sodium chloride produced a depression of GFR. With higher doses of AVT (above 40 ng/kg), a real decrease in total-kidney GFR occurred in domestic chickens (1) as it did in the quail in the present study.

The chicken neurohypophysis contains about 400 vaso-pressor milliunits of AVT (14). If the quail neurohypophysis contains a comparable amount of AVT, then all doses used in the present study could have been contained in the neurohypophysis. However, it seems most likely that only the lower doses (10 ng or about 2.25 mU/kg and 50 ng or about 11.25 mU/kg) could have been released under physiological circumstances. The lowest dose level (10 ng or 2.25 mU/kg) especially would seem to be well within the range of physiological release. It would seem most unlikely, however, that an amount as large as 200 ng (about 45 mU/kg), enough to cause major changes in the mean arterial pressure, would be released except under very unusual circumstances. Thus, although it is conceivable that such an amount could be released from the neurohypophysis, it would probably have to be considered a pharmacological dose.

Following the administration of 10 ng AVT/kg, there was a slight decrease in the SNGFR for MT nephrons, an even smaller decrease in the SNGFR for RT nephrons, and a marked decrease in the number of filtering RT nephrons. Previous work (2) indicated that when RT nephrons ceased filtering, blood could bypass them into the peritubular capillary network. Possible glomerular bypass vessels near the glomeruli in domestic fowl have been suggested by the anatomical studies of Siller and Hindle (17). With the administration of 10 ng AVT/kg there could have been some slight constriction of the afferent arterioles of those glomeruli still filtering. Complete constriction of afferent arterioles of other RT nephrons could have shunted blood through small bypass vessels near the glomeruli into the peritubular capillaries.

The mean SNGFRs of the MT and RT nephrons did not change significantly from the control levels following the administration of 50 ng AVT/kg, although that of the MT nephrons tended to increase slightly. However, there was a much more marked decrease in the number of filtering RT nephrons than occurred following 10 ng AVT/kg. It appears possible that constriction of larger arterial branches could have occurred with this dose of AVT, cutting off larger areas of RT nephrons. This could have led to shunting of greater blood flow to the MT nephrons and, perhaps, have accounted for some slight increase in the mean SNGFR of MT nephrons. That this pattern may have occurred is suggested by the photograph of a portion of a kidney from a quail following the administration of 50 ng AVT/kg (Fig. 6). This photograph shows the branching of a larger arterial vessel proximal to the origin of the glomeruli. Ferrocyanide precipitate is apparent in one portion of the vessel but has not entered the side branch, suggesting that blood flow may have been cut off in that branch. This same pattern is supported by the photograph shown in Fig. 7. This shows a portion of the surface of a kidney following the administration of this same dose of AVT. Some areas of the surface are dark from the presence of ferrocyanide precipitate while other sections

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**Fig. 6.** Photomicrograph showing evidence of shunting of arterial blood away from RT nephrons following administration of 50 ng AVT/kg. Artery at A contains Prussian blue precipitate, but after bifurcation at C only artery at B contains precipitate. Artery at D which leads to an area of RT nephrons contains no precipitate. (Original magnification ×10.)
show no evidence of any ferrocyanide precipitate. This suggests that whole portions of the surface areas of the kidney where the RT nephrons are located were not being supplied with arterial blood. Control kidneys show a uniform pattern of dark precipitate on the surface ((2) and unpublished observations). Moreover, previous work with salt-loaded animals indicated that when RT nephrons ceased filtering (apparently as a result of constriction of afferent arterioles) and blood was shunted into the peritubular capillaries, the surface of the kidney still showed a uniform pattern of dark blue precipitate ((2) and unpublished observations). Thus, it seems most likely that with these higher dose levels, blood was shunted away from some superficial areas of the kidneys. However, the exact vascular sites of action of AVT are unknown.

In those animals in which the mean arterial blood pressure fell following the administration of 200 ng AVT/kg, the mean SNGFR for the MT nephrons fell almost to the level of that for the RT nephrons. The percentage of filtering RT nephrons was also reduced from the control level. It appears possible that constriction of larger arterial branches again shunted blood away from areas of RT nephrons and that the reduced arterial pressure was insufficient to maintain filtration at the normal level in the MT nephrons. The same pattern of distribution of precipitate on the kidney surface shown for the 50 ng/kg dose (Fig. 7) was observed in these kidneys.

In those animals in which a vasopressor response predominated following the administration of 200 ng AVT/kg, there were no filtering RT nephrons and the SNGFR of the MT nephrons was greatly increased. It appears likely that most of the blood flowing through the kidneys was shunted away from the superficial RT nephrons to the MT nephrons where greater flow with increased hydrostatic pressure produced an increase in SNGFR. Again, distribution of precipitate on the kidney surface was very spotty. However, as noted above, nothing is yet known about the exact sites of action of AVT within the avian kidney. Moreover, although variable blood pressure responses to high doses of vasopressin and other neurohypophysial peptides have been observed in birds as well as other nonmammalian vertebrates (21), the mechanism underlying these responses is not understood.

In the previous study (2), the continuous intravenous infusion of a hyperosmotic sodium chloride solution produced a fall in total-kidney GFR, which appeared to result primarily from a decrease in the number of filtering RT nephrons. In fact, at the point during the infusion at which SNGFRs were determined (following administration of 40 meq/kg), all RT nephrons appeared to have ceased filtering (Fig. 5). In the present study, this appeared to occur only with the highest doses of AVT, at which point there was also a marked increase in the SNGFR for the MT nephrons. In the case of the salt load, the SNGFR for the MT nephrons actually decreased somewhat (2) (Fig. 5). This pattern of MT nephron function is closer to that observed in the present study with low doses of AVT (10 ng/kg). Also, as noted above, with high doses of AVT ferrocyanide precipitate was not uniformly distributed on the surface of the kidney. However, when RT nephrons ceased filtering following a salt load and the precipitate was distributed in the peritubular capillaries, the surface of the kidney showed a uniform pattern of precipitate ((2) and unpublished observations). This is again a pattern more similar to that observed following low doses of AVT (10 ng/kg) than to those observed following high doses of AVT (50 ng/kg or more). Presumably, both with a salt load and with the administration of low doses of AVT, constriction of the afferent arterioles leads to cessation of filtration in RT nephrons and shunting of blood through bypass vessels into the peritubular capillaries. It is conceivable that some intermediate dose of AVT would mimic the effects of a salt load exactly, with cessation of filtration in all RT nephrons without shunting of blood away from whole masses of kidney tissue. However, it is also possible that the effects of a sodium chloride load on the number

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**FIG. 7.** Photomicrograph of kidney surface showing irregular distribution of Prussian blue precipitate after administration of 50 ng AVT/kg suggesting internal shunting of blood. Areas of heavy blue precipitate are outlined for identification on black and white photograph. Area at A is broken surface of kidney. (Original magnification X 7.)
of filtering RT nephrons may not be mediated through the action of AVT.

What importance might these observed effects of AVT have for water conservation by avian kidney? Arginine vasotocin is considered to be a true antidiuretic hormone in birds (15). Its antidiuretic effect has been considered to result primarily from an increase in the permeability of the distal tubule and collecting duct to water (1, 15, 18). However, this effect on tubular permeability to water has never been demonstrated directly for avian nephrons. Moreover, the distal nephron of many reptiles shows little change in permeability to water with dehydration or administration of AVT (5). Thus, it at least seems possible that the permeability of the distal tubule of the RT nephrons in the avian kidney might not be altered in the presence of AVT. In the present study, reductions in the number of filtering RT nephrons occurred even with small, probably physiological, doses of AVT. Since these nephrons do not function together to contribute directly to the concentrating ability, it appears reasonable that they might cease filtering with the need to conserve water. However, all RT nephrons contribute to the fluid flowing through the collecting ducts in the medullary cones. Using the mean values for SNGFRs and percents of filtering RT nephrons shown in Fig. 5, and assuming that the fraction of filtrate reabsorbed before the collecting ducts are reached is unchanged following the administration of AVT, we calculate that the volume flow through the collecting ducts is reduced by about 40% from the control level following the administration of 10 or 50 ng AVT/kg. These changes, which result primarily from changes in the number of filtering RT nephrons, may be more important than changes in tubular permeability to water in enhancing the concentrating ability of the avian kidney.

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