The Polyuria of Paroxysmal Atrial Tachycardia

By Michael J. Kinney, M.D., Richard M. Stein, M.D., and Vincent A. Discala, M.D.

SUMMARY

Two patients with paroxysmal atrial fibrillation and an associated polyuria were studied to delineate the mechanism of the increase in urine flow. A striking saluresis was noted in both patients. The increased sodium excretion was probably due to decreased sodium reabsorption, perhaps at proximal tubular nephron sites. This inhibition of sodium reabsorption could explain both the saluresis and some part or all of the polyuria. Re-evaluation of earlier case reports reveals patterns of concomitant salt and water excretion consistent with this mechanism. The saluresis cannot be explained by the previously favored hypothesis of antidiuretic hormone inhibition.

Additional Indexing Words:

Diuresis  Solute diuresis  Atrial pressure  Atrial distension

**A STRIKING POLYURIA** is often associated with the paroxysmal atrial tachycardias (including atrial flutter and fibrillation). The profuse urine production associated with any of these states of atrial hyperactivity has been noted since at least 1927 and the known clinical and hemodynamic features of this association have been reviewed previously. Dr. Wood suggested that the polyuria represents a water diuresis secondary to antidiuretic hormone (ADH) inhibition. However, a significant saluresis was found in the two cases he reported and is found in every reported case for which urinary data exists. Since ADH inhibition alone would not explain the saluresis, the renal mechanism producing this polyuria has remained unclear. Analysis of our two cases suggests that the polyuria can best be explained by a decrease in renal sodium and water reabsorption.

**Methods**

All subjects freely granted consent to the renal studies. Full disclosure was made of the nontherapeutic nature of the studies and the potential complications. As soon as possible after the start of a spontaneous episode of tachycardia-polyuria, urine collections were begun and priming and sustaining infusions of concentrated inulin and parahypophosphite (PAH) were administered by vein using a Bowman type constant infusion pump at the low fixed rate of 0.65 or 1.1 cc/min throughout the study. Periodic blood specimens were drawn for clearance measures through a separate heparin-locked venous catheter. After these initial venous catheterizations the patients were not disturbed and were comfortably recumbent throughout the study.

Urine and plasma specimens were analyzed for osmolality, sodium, potassium, urea, inulin and PAH concentrations. Osmolality was measured by the method of freezing point depression using a Fiske G62 osmometer. Sodium and potassium concentrations were determined with a model 143 internal lithium standard flame photometer, manufactured by Instrumentation Laboratories. Inulin concentration was measured by the resorcinol method with alkali treatment, PAH and creatinine by standard methods.

Creatinine and inulin clearance (UV/P) is an index of glomerular filtration rate (GFR). Osmolar clearance (Cosm) less urinary flow rate (V) is water freed of solute and reabsorbed (T'H2O). V-Cosm = CH2O or water freed of solute and excreted. CNa+ is the clearance of sodium. Dividing either V or CNa+ by 100 ml of GFR yields the fraction of the filtered water or sodium, respectively, excreted. Such factoring will not of course eliminate changes in renal function evoked by changes in glomerular filtration rate; it will only adjust for variations in supply to the nephrons.

**Case 1 Report**

This 64-year-old white married male American seaman (S. B.) had a two-year history of paroxysms of polyuria with accompanying paroxysmal atrial fibrillation. The frequent passage of large volumes of urine might last 3 to 6 hours and might be accompanied by a few pounds of weight loss but not by thirst. He never noted his arrhythmia but was aware of the polyuria. The paroxysm would occur as often as twice a week or might only bother him twice in a quarter year. He prevented his polyuria with reasonable success by a combination of digitalis and dilantin for his concomitant paroxysmal atrial fibrillation. Acute paroxysms of polyuria were treated successfully with quinidine, until a few weeks prior
to his admission. At that time his medications began to fail and his paroxysms became more frequent and then stopped, as he was left with a persistent atrial fibrillation. The patient never noted any cardiorespiratory or other urinary symptoms and knew of no renal disease. He had noted no symptoms suggestive of hyperthyroidism.

In the past this patient had occasionally been treated for mild labile hypertension of unknown etiology and had been treated for late latent lues.

Physical examination revealed a strong, well nourished, elderly male of 184 lbs weight, able in appearance. Brachial arterial blood pressure was 150/86 and the pulse was initially irregularly irregular at 80 beats per minute. No thrymegaly or sign of hyperthyroidism was found. He had a faint (grade ½) apical systolic heart murmur, otherwise the cardiorespiratory examination was normal; there was no sign of heart failure. The urine contained a few calcium oxalate crystals and a rare WBC but showed no growth on culture for bacteria. The blood-urea-nitrogen (BUN) was 18 mg%. Serum electrolytes and chest X-ray were interpreted as essentially normal. The electrocardiogram initially revealed simple atrial fibrillation with intermittent left bundle branch block.

Hematocrit was 41%. The serological test for syphils (VDRL) was nonreactive. Fasting blood sugar (FBS) equaled 94 mg%. Serum cholesterol was 240 mg%. A liver profile, sedimentation rate, and WBC count were all normal. While in the hospital on a standard daily diet of 110-175 mEq of Na+ per day, the patient had 24 hour total urine excretions of sodium of 110, 144, and 164 mEq.

The Paroxysm

During hospitalization patient S. B.'s atrial fibrillation was corrected to regular sinus rhythm with digoxin alone. After conversion to a regular rhythm, and while on digoxin 0.125 mg twice daily and quinidine 300 mg at six hour intervals, he awoke one morning at 12:30 A.M. and noted polyuria. By 1:30 he had urinated several times an estimated total volume of a little more than 300 cc (approximately 5 cc/min). His urinary bladder was catheterized shortly after 2 A.M. and timed measured urine collections initiated. Each urine specimen collected was by washing out the bladder three times with 30 to 50 cc of air. It had now been at least seven hours since the patient had eaten and some five hours since he last took fluids. He remained without food or fluids by mouth during this studied paroxysm and slept intermittently. The patient's apical heart rate was an irregular 110 beats/min and the brachial rate approximately 84 beats/min. An electrocardiogram revealed atrial fibrillation with a ventricular rate varying from 80 to 135 beats/min. The particular ventricular rate appeared totally unrelated to the concomitant urinary output and the arrhythmia outlasted the polyuria. A few hours prior to this paroxysm of fibrillation and polyuria the regular sinus rate had been 72 beats/min.

Several weeks later, after return to sinus rhythm, this patient underwent bladder catheterization and a constant infusion of insulin to measure baseline renal function. For these latter studies he was food and fluid deprived overnight and studied in the early morning.

Results of Renal Studies

Table 1 summarizes the renal findings during the paroxysm and the subsequent control studies. During the paroxysm there was a marked increase in urine flow (V) and

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solute clearance (Cosm). The latter was due almost entirely to an increased sodium clearance (CNa+). The CNa+ rose to 6.9 cc/min and from the 2xUNa+/Uosm ratio we can estimate that more than 70%, and as much as 89%, of the urinary solute was sodium and its accompanying anions. In subsequent studies when the subject was in regular sinus rhythm, his urine output and pattern of sodium and water excretion were normal (table 1). Serum electrolytes and osmolality are not included in table 1 as they never changed significantly from normal values.

Figure 1 depicts the urinary excretion pattern during the paroxysm. Note the similar behavior of the urinary flow rate (V), the sodium clearance (CNa+), and the rate of water reabsorption (CH2O or TTH2O).

To correct the nephron rates of water and sodium reabsorption for variability in supply to the nephron, figure 2 depicts our measures of water (V or urine flow) and sodium clearance (CNa+) after they have been related to a standard 100 ml of glomerular filtrate, by dividing them by the creatinine clearance. Note that the diuresis-saluresis persists and that at its peak more than 4.9% of the filtered sodium was excreted (CNa+/100 ml GFR).

The total amount of sodium excreted during the paroxysm was approximately 263 mEq in 1,933 cc of urine.

**Case 2 Report**

This 72-year-old white widowed female had been having paroxysmal atrial fibrillation for six years. Its onset was often noted by the patient as a sense of “pounding” and “congestion” in the chest, frequently with a nonproductive cough. These episodes were often followed within 15 minutes by voluminous urination that typically lasted 1-2 hours. The poluryra never occurred without the cardiac hyperactivity and never preceded it. But, as with our first patient, the cardiac hyperactivity usually outlasted the polyuria by any time from four hours to two days. These paroxysms occurred irregularly with symptom-free intervals ranging from one month to only one day. The paroxysms of fibrillation were less frequent when the patient took quinidine, 200 mgm t.i.d., and quinidine gluconate, 350 mg at bedtime. Paroxysms of both cardiac symptoms and urination were often terminated by taking 200 mg of quinidine at four hour intervals during the attacks.

There was no history of angina-like chest pain, orthopnea, paroxysmal nocturnal dyspnea, or ankle edema. The patient did admit to one flight exertional dyspnea. There were no symptoms of hyperthyroidism. No history of respiratory diseases, diabetes mellitus, myocardial infarctions, rheumatic fever or joint diseases could be elicited. The past history revealed two episodes of cystitis and one of thrombophlebitis as well as the onset of a mild bilateral hearing defect late in life.

Physical examination on admission to the hospital disclosed a well developed elderly, slightly obese, white female. She was in no distress and had a brachial arterial blood pressure of 110/70 with a regular pulse rate of 70 beats/min. The heart’s apical impulse was not displaced and the heart was not enlarged to percussion. There were no heart murmurs. No abdominal organopathy was palpated. No peripheral venous distention or edema was found. Pulses at the carotid, femoral, brachial and pretilibial sites were adequate and symmetrical. Neurological examination, including the optic fundi, was within normal limits. No sign of hyperthyroidism existed.

Urinalysis showed a few WBCs but no significant growth on culture; BUN = 13 mg%; protein bound iodine = 6.2 gamma %; FBS = 97 mg%; cholesterol = 231 mg%; total proteins were 6.0 g% with an albumin of 4.1 g%; Hct = 39%. A chest X-ray revealed atherosclerotic tortuosity of the aorta and old stable healed parenchymal fibrotic changes in the left inferior base. There was neither cardiomegaly nor cardiac calcifications. The initial electrocardiograms revealed a regular rhythm with minimal ST and T wave changes ascribable to quinidine.

The Paroxysm

Patient E. K.‘s paroxysm of polyuria occurred after her

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

The pattern of urinary salt and water excretion during the polyuria associated with paroxysmal atrial fibrillation in the first case. Over the time course (abscissa) the clearance of sodium (CNa+), urinary flow (V), and water reabsorption (TTH2O) are plotted. Urinary collection times are indicated by the dots.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

As in figure 1, the clearance of sodium and the urinary flow are depicted over the time course of the polyuria associated with the paroxysm of atrial fibrillation in case #1. Patient S. B. These values are factored here by 100 ml of creatinine clearance to adjust for differences in renal supply. The excretion fraction of the filtered sodium (CNa+/Ccr) and water (V/Ccr) behave similarly.
8:00 A.M. breakfast, so that she had ingested a modest amount of salt and water some 2½ hours prior to the paroxysm. However this hospital diet was limited to 1 g total Na+ content per day, hence her early morning Na+ intake prior to the paroxysm was not more than 14 mEq at the most. She had also received a 300 mg quinidine tablet at 6 A.M. and received another 400 mg early in our study of the paroxysm. Nothing else was taken by mouth during the study.

At about 9:45 the ward staff noted that an irregular pulse had replaced her early morning regular pulse. Shortly after 10:30 the patient noted the first passage of a large volume of pale urine. This was saved and studied. Subsequent specimens were obtained by freely voiding and their time of passage and volume noted.

During the studied paroxysm of fibrillation no change in her physical state was noted other than the presence of an irregularly irregular heart rate which ranged from 70 beats/min to 130 beats/min. As with the first patient the polyuria bore little relationship to the concomitant ventricular rate. The continuous ECG monitor revealed persistent atrial fibrillation that oscillated the polyuria. The B.P. was stable at 110/70. There were no signs or symptoms during the paroxysm to suggest heart failure, angina, or any other difficulty.

Results of Renal Studies

Table 2 reveals a relatively short-lived polyuric (V) episode, once again composed of both water and sodium. In addition to the increase in urine flow rate (V) and clearance of solute free water (CH4O) there was a solute diuresis, (Cosm), which could be explained entirely by the increased sodium clearance (CNa+). At the peak of the diuresis the fraction of filtered sodium excreted (CNa+/100 ml GFR) rose to 5.7% and sodium, with its attendant anion, accounted for more than 70% of the solute excreted (2xU+Na+/Uosm). As in the first patient the behavior of both urine flow and saluresis in this patient were similar during the paroxysm of polyuria. Figure 3 depicts the behavior of water excretion (V) and sodium clearance (CNa+) during the paroxysm; both have been factored by the creatinine clearance, an index of the filtration rate. The changes in CNa+/100 ml GFR, or fraction of the filtered sodium excreted parallel those noted in V/100 ml GFR, or the fraction of the filtered water excreted. A total Na+ excretion of 42 mEq and water excretion of 1,357 cc was found during this three hour paroxysm of polyuria in this salt restricted patient—more than three times the maximal intake of sodium during the preceding 12 hours.

Discussion

Both of our patients produced a marked saluresis in addition to their water excretion. Reviewing all the other patient studies‡ of the associated polyuria and atrial tachycardia for which there are sufficient urinary data, we found always both a saluresis and diuresis and both closely associated. Nowhere does a diuresis occur from a paroxysmal atrial tachycardia without a saluresis. This saluresis persists after factoring the sodium clearance by an index of the glomerular filtration rate so it is not due to a simple increase in sodium
supply to the renal nephrons. The saluresis is not secondary to a solute diuresis from some substance other than sodium, for the 2xUNa+/Uosm ratios in both cases exceeded 70%.

It appears that the saluresis results from a decrease in renal tubular sodium reabsorption. The fraction of the filtered sodium load (C
\textsubscript{Na}/100 ml GFR) appearing in the urine in these studies rose to levels as high as 4.9 and 5.7%. In both instances this represents a marked decrease in the fraction of the filtered sodium load reabsorbed in the tubules. Although it is hazardous to analyze nephron sites of altered tubular sodium reabsorption with clearance methods under conditions in which antidiuretic hormone activity is neither maximal nor minimal, we have plotted in figure 4 urine flow rate (V) versus solute clearance (Cosm) in our two cases (dashed lines). Also depicted in this figure as solid lines are the mean curves obtained in normal subjects in our laboratory and elsewhere\textsuperscript{12} during mannitol loading under hydropenic conditions (maximal ADH activity) and under hydrated conditions (suppression of ADH activity). Data from our two patients with polyuria fall on either side of the isosmotic line (dotted) and tend to parallel the corresponding normal mean curve on that side of the isosmotic line. In patient #1 T\textsuperscript{3}H\textsubscript{2}O is increased throughout most of the increase in Cosm whereas in patient #2, CH\textsubscript{2}O is increased with Cosm. A rise in T\textsuperscript{3}H\textsubscript{2}O or CH\textsubscript{2}O accompanying an increase in Cosm during a solute diuresis under hydropenic or hydrated conditions, respectively, is generally thought to reflect an inhibition of proximal tubular sodium and water reabsorption, with a consequent increase in ascending limb sodium supply and transport (T\textsuperscript{3}H\textsubscript{2}O or CH\textsubscript{2}O). Since the directional changes noted in our patients seem to parallel the hydropenic and hydrated mean normal curves, it may be proposed that the increase in solute clearance in these patients represents an inhibition of proximal tubular sodium and water reabsorption with a subsequent increase in ascending limb and distal tubular nephron sodium supply and transport. This would be most plausible if one were to assume that in each patient the increase in solute (sodium) clearance was superimposed on a relatively stable but submaximal level of ADH. In fact, when our data are compared to findings previously reported in the dog,\textsuperscript{13} the curves of our patients are indeed very similar to those noted in dogs who received submaximal doses of antidiuretic hormone during a solute diuresis. Thus, with respect to the renal mechanism of the polyuria associated with paroxysmal tachycardia, there is a significant increase in solute excretion due almost exclusively to an increase in sodium excretion, occurring in a setting of what appears to be a variable suppression.

![Figure 3](image)

**Figure 3**

The clearance of sodium (C\textsubscript{Na}/C\textsubscript{cr}) and the urinary flow (V/C\textsubscript{cr}) are depicted over the time course of the polyuria associated with the paroxysm of atrial fibrillation in case #2, patient E. K. Values are factored by 100 ml of creatinine clearance to adjust for differences in renal supply.

![Figure 4](image)

**Figure 4**

The relationship of urinary flow (V) to solute clearance (Cosm) in the human. The solid line curves for maximal dehydration and hydration are mean curves from many other studies. The case studies reported here are depicted as dashed lines. The dotted line represents isosmotic urine excretion. The difference between isotonicity and any one curve of Cosm vs V represents either free water excreted (CH\textsubscript{2}O) or reabsorbed (T\textsuperscript{3}H\textsubscript{2}O).
of antidiuretic hormone activity. The increase in sodium excretion is consequent to a decrease in the fraction of the filtered sodium load reabsorbed. Inferences from clearance data suggest that this represents, at least in part, an inhibition of sodium reabsorption within the proximal tubule. In addition, the pattern of the increase in both sodium and potassium excretion is also consistent with an inhibition of proximal tubular sodium reabsorption.

Although we cannot exclude an additional decrease in sodium reabsorption at the ascending limb of the nephron, it is relatively unlikely since the second patient, a 72-year-old female, was able to lower her urine osmolality to 92 mosm/kg and increase her CH₂O to 6.35 cc/min (table 2). Since the water clearing sites of the nephron responsible for lowering the urine osmolality and increasing CH₂O are the ascending limb and early distal tubule, the findings in this patient suggest no major defect at these sites.

It is instructive to consider the possible atrial mechanisms leading to the decreased renal sodium reabsorption. There are no known striking central hemodynamic changes in paroxysmal atrial fibrillation, flutter, or tachycardia that alone might produce such a saluresis. In addition, in the arrhythmias of other than atrial origin, there are no striking renal excretory changes. When hemodynamic measurements have been obtained during specific paroxysms of atrial tachycardia-polyuria, they have not been consistently aberrant. Cardiac output has increased, or remained the same. We found peripheral blood pressure unchanged during the paroxysm as did Luria. Wood reported a fall in blood pressure. Glomerular filtration rate is usually well maintained during the paroxysm. Right atrial pressure can rise or decrease. Apical and peripheral heart rate can vary, as we have seen in our cases, from 80 to more than 170 beats/min. Luria lowered the pulse rate with propranolol during a paroxysm and noted no influence on the polyuria. There appears to be no consistent relationship between atrial or ventricular rate and the polyuria. In the one case (2) in which it was measured, left atrial pressure rose, suggesting that atrial distension may occur. Thus it appears that the only certain relationship of the renal polyuria of the paroxysmal atrial tachycardias is to the atrium itself, to atrial stimulation/distension.

That the atria represent ideally located sites for volume receptors and contain neurally mediated stretch receptors has been previously noted. That the proximal tubule has already been pinpointed as the site of nephron function most amenable to changes in extracellular and intravascular blood volume is well known. That a similar saline excretion pattern results from the infusion of saline — commonly referred to as "volume expansion" — is also generally accepted. It is intriguing to speculate that atrial stimulation in this syndrome may be signaling an excess of volume to the kidney.

In agreement with this speculation is the general rule that the polyuria occurs after the onset of the atrial paroxysm and is always of the same or a shorter duration than the atrial tachycardia. But the diuresis-saluresis ceases while the atrial activity persists. We suspect that this is because the salt and water losses become significant enough to involve other mechanisms for the renal control of volume or sodium excretion. Neither of our patients were significantly disturbed by their rather large losses of sodium and water but both were inactive and recumbent. Symptoms of weakness, malaise, and syncope previously reported with this syndrome may be related either to the atrial tachycardia or to the salt and water losses.

There is a group of other human experimental conditions which should be just mentioned as possible analogues to our cases of paroxysmal atrial hyperactivity with polyuria. They have in common the ability to alter the pressure across the walls of the atria and major pulmonary veins or stimulate-distend the atria and concomitantly increase renal water and salt excretion. Intermittent negative pressure breathing, immersion in thermo-indifferent water, and states of weightlessness, as in space, are examples.

These renal effects of atrial distension or stimulation in the human are in striking contrast to dog studies of left atrial distension. For in the dog there is a water diuresis without a saluresis and the mediating renal mechanism for the polyuria is probably antidiuretic hormone (ADH) inhibition. Although ADH activity has not been measured in the human cases of paroxysmal atrial tachycardia-polyuria, Luria infused hypertonic solutions and exogenous vasopressin during the paroxysm of atrial hyperactivity and polyuria and noted similar normal renal responses to both. So that in these human studies the normal osmoreceptor-ADH-collecting duct permeability system seems at least intact. But we believe that there is little reason to implicate changes in antidiuretic hormone as being of major importance in the clinical cases of polyuria due to atrial hyperactivity. For not only is the saluresis unexplained by changes in ADH but also the water diuresis (e.g. case #2) may be seen as secondary to the saluresis.

We speculate that human atrial hyperactivity from an atrial arrhythmia represents, in the clinical cases studied and reviewed here, a natural experiment in man simulating volume expansion, and signaling the kidney, through entirely unknown means, to decrease renal tubular sodium and water reabsorption, probably at proximal nephron sites.

Circulation, Volume 50, September 1974
POLYURIA OF PAT

Addendum

Recently two separate groups have studied the polyuria of tachycardia by artificially pacing atrial heart rates in dogs. (Abstracts 265 A-4 and 572 A-3 in Clinical Research 22; April 1974).

A diuresis was produced in these studies only when left atrial pressure rose with the pacing tachycardia. The diuresis was predominately, if not entirely, a water diuresis, most likely due to the inhibition of Antidiuretic Hormone. These studies confirm our prior report that a pure water diuresis is produced by direct distension of the dog's left atrium with a balloon. As noted above in the text these dog studies stand in sharp contrast to human studies, where it appears that a sodium-solute diuresis occurs.

Acknowledgment

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References
