The Effect of an Acute Increase in Renal Perfusion Pressure on Sodium Transport in the Rat Kidney

ROBERT T. KUNAU, JR., M.D., AND NORBERT H. LAMEIRE, M.D., PH.D.

SUMMARY We used micropuncture techniques to examine the intrarenal response to an acute elevation of the renal perfusion pressure. In one series of studies (epinephrine, group I) the renal perfusion pressure was acutely increased by intravenous epinephrine infusion; in another series, by bilateral carotid occlusion and vagotomy. The results indicate that the increase in sodium transport pressure cannot be attributed to enhanced sodium delivery from superficial cortical late distal tubule occurred. These data suggest that the natriuresis which follows an acute elevation of the renal perfusion pressure cannot be attributed to enhanced sodium delivery from superficial cortical late distal tubule. Furthermore, we found that epinephrine infusion at a constant renal perfusion pressure (epinephrine, group II) did not affect fractional sodium excretion, although a small, but significant, decrease in the CFR and sodium delivery from the superficial cortical late distal tubule occurred. In neither study, however, did we find that the increase in renal perfusion pressure changed the glomerular filtration rate (GFR) (both kidneys) or fractional sodium delivery from the superficial cortical late distal tubule. Furthermore, we found that epinephrine infusion at a constant renal perfusion pressure (epinephrine, group I) did not affect fractional sodium excretion, although a small, but significant, decrease in the GFR and sodium delivery from the superficial late distal tubule occurred. These data suggest that the natriuresis which follows an acute elevation of the renal perfusion pressure cannot be attributed to enhanced sodium delivery from superficial cortical nephrons but must result from (1) inhibition of sodium reabsorption in inner cortical nephrons or (2) an effect on sodium transport in the collecting system.

1.0% ($P < 0.001$) following carotid occlusion and vagotomy. In neither study, however, did we find that the increase in renal perfusion pressure changed the glomerular filtration rate (GFR) (both kidneys) or fractional sodium delivery from the superficial cortical late distal tubule. Furthermore, we found that epinephrine infusion at a constant renal perfusion pressure (epinephrine, group I1) did not affect fractional sodium excretion, although a small, but significant, decrease in the GFR and sodium delivery from the superficial late distal tubule occurred. These data suggest that the natriuresis which follows an acute elevation of the renal perfusion pressure cannot be attributed to enhanced sodium delivery from superficial cortical nephrons but must result from (1) inhibition of sodium reabsorption in inner cortical nephrons or (2) an effect on sodium transport in the collecting system.

ACUTE ELEVATIONS in the renal perfusion pressure have been shown to result in parallel increases in the urinary excretion of sodium in several conditions under a variety of circumstances. As this effect of perfusion pressure occurs in the absence of a detectable change in the filtered sodium load, it has been suggested that renal perfusion pressure can directly influence tubular sodium transport. Although micropuncture studies of the proximal convoluted tubule in the dog and rat have shown that an acute elevation in renal perfusion pressure can diminish fractional sodium reabsorption at this site, there is also strong, albeit indirect, evidence to indicate that sodium reabsorption in some segment(s) of the distal nephron also may be affected.

Our present studies were performed to further characterize changes in sodium transport that occur in response to an acute elevation in renal perfusion pressure. In these micropuncture studies, sodium transport in the distal convoluted tubule of the rat nephron was examined prior to and after the renal perfusion pressure was increased acutely by epinephrine infusion and by bilateral carotid occlusion and vagotomy. The results indicate that the increase in sodium excretion observed in these models cannot be attributed to an increase in sodium delivery from the superficial distal convoluted tubule but must reflect an effect of perfusion pressure on sodium transport in deeper cortical nephrons or in the collecting system.

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Methods

Male Sprague-Dawley rats weighing between 300 and 358 g were used. The rats were deprived of food for 12–16 hours prior to study but were permitted free access to water. Following intraperitoneal anesthesia with sodium ethyl(1-methylpropyl)malonilythiourea (Inactin, Promonta, Hamburg) (100 mg/kg of body weight), a tracheostomy was performed and catheters were inserted into the left femoral and right carotid arteries, the left femoral and left external jugular veins, and into the dome of the urinary bladder. These catheters permitted the administration of infusion solutions, the recording of the arterial blood pressure, and the collection of arterial blood and urine samples for analysis. The arterial blood pressure was determined with Statham P23Db pressure transducers, a Hewlett-Packard model 301 amplifier recorder, and a Stentor model BPM-1 recorder. The rat then was transferred to a thermostatically heated micropuncture table where the left kidney was exposed by a lateral abdominal incision. A length of suture was placed around the aorta cephalad to the renal arteries and drawn through polyethylene (PE-90) tubing. This “aortic snare” provided a means by which renal perfusion pressure could be controlled by exerting tension on the protruding ends of the suture and compressing the tubing against the aorta. The left kidney then was placed in a Lucite cup in which it was bathed in mineral oil heated to 38°C. Following the surgical preparation, a solution consisting of 25 mM NaHCO₃, 120 mM NaCl, and 4 mM KCl was infused at a rate of 200 μl/min throughout the experiment. The same solution, with 10% inulin added, was administered at 20 μl/min through another syringe. D-Aldosterone and aqueous vasopressin were added to this latter solution in an amount sufficient to increase the arterial pressure 35–45 mm Hg above the mean baseline femoral arterial pressure observed during period A. The amount of epinephrine required to achieve these pressure levels ranged from approximately 0.5 to 1.7 μg/min. Fifteen minutes later tubular fluid samples again were collected from the previously studied distal convoluted tubules and arterial blood and urine samples also were obtained (period B).

EPINEPHRINE, GROUP I

This group was studied in a manner identical to that for group I through period A. After period A, the epinephrine was infused as in group I, but the perfusion pressure to the kidney, as measured by the femoral artery pressure, was maintained constant by adjustment of the tension on the aortic snare. The carotid artery pressure, however, was increased to an extent comparable to the increment observed for group I.

BILATERAL CAROTID OCCLUSION AND VAGOTOMY

To examine the effect of an acute increase in renal perfusion pressure induced by an intervention other than epinephrine infusion, in a separate group of experiments the perfusion pressure was increased by bilateral carotid occlusion and vagotomy. This group was studied as were epinephrine groups I and II through period A. Thereafter, the left carotid artery was ligated and a bilateral vagotomy was performed. Although the arterial pressure following carotid occlusion and vagotomy tended to be less stable than after epinephrine, we used data from only those experiments in which the mean arterial pressure during period B was 30 mm Hg above the mean of period A. In this group, tubular fluid samples were obtained only from the superficial late distal tubule.

In all experiments period A was between 20 and 25 minutes in duration. To obtain samples over as stable an interval as possible following the elevation of the arterial pressure, period B was limited to 15–17 minutes. The earliest and latest distal convolutions of the same distal convoluted tubule were localized as previously described. In previous studies we were able to obtain tubular fluid samples from both the earliest and latest convolutions of the same distal convoluted tubule, the oil droplet from the late convolution readily progressing downstream in all tubules used so that the sample obtained from the early convolution was collected at a time when flow downstream was unobstructed. In contrast, in the present studies, after collection of the tubular fluid sample from the late distal convolution during elevated arterial pressure (period B), the oil droplet frequently failed to move downstream. For this reason, early and late distal tubular fluid samples were obtained from separate distal convoluted tubules. To determine the single nephron glomerular filtration rate (SNGFR), the tubular fluid samples obtained from the early distal sites were collected over an accurately timed interval of 1–2 minutes. The volume of this collection was determined in a calibrated quartz microcapillary with a constant internal diameter. Tubular fluid samples were analyzed for sodium with a helium glow photometer and for inulin by the microfluorescent method of Vurek and Pegram. The sodium
concentration in arterial plasma and urine was determined with an Instrumentation Laboratories model 143 flame photometer. Inulin in plasma and urine was determined by the anthrone method of Fillér et al. The inulin concentration in plasma was corrected for plasma water; the plasma solids concentration in arterial plasma and urine was determined with an Instrumentation Laboratories model 143 flame photometer. A comparison of the results was performed with Student's t test, using paired and unpaired tests as indicated. A P value of less than 0.05 was considered to be significant.

Results

EPINEPHRINE STUDIES, GROUPS I AND II

The clearance and micropuncture results from these two groups are presented in Tables 1 and 2, respectively.

During period A the renal perfusion pressure was comparable in both groups, being 119 ± 1.0 (SEM) mm Hg in group I and 117 ± 2.7 mm Hg in group II. Following epinephrine infusion in group I, renal perfusion pressure rose to 166 ± 1.9 mm Hg. In group II, in period B, epinephrine infusion increased the carotid arterial blood pressure to 162 ± 1.1 mm Hg, whereas renal perfusion pressure was maintained constant at 118 ± 2.9 mm Hg by adjustment of the suture placed about the aorta.

As shown in Table 1, the acute elevation in renal perfusion pressure in group I resulted in a marked increase in both fractional and absolute sodium excretion but did not affect the total kidney glomerular filtration rate (GFR) (both kidneys). In contrast, the administration of epinephrine while the renal perfusion pressure was maintained constant (group II, period B) resulted in a fall in both urinary sodium excretion and GFR (Table 1).

Figure 1 demonstrates the change in SNGFR which occurred between periods A and B in groups I and II. The individual values for group I are randomly distributed about the line of identity indicating, as was true for total GFR, that epinephrine infusion and the resultant increase in renal perfusion pressure did not affect the SNGFR as measured at the early distal convoluted tubule. In contrast, epinephrine infusion during constant renal perfusion pressure (group II, period B) resulted in a modest but significant decrease in SNGFR from 73.9 ± 2.7 to 64.1 ± 2.6 nl/min, P < 0.01.

The early distal tubular fluid to plasma (TF/P) ratios for Na⁺ were essentially identical in both periods for the two groups (Table 2). In group I, the early distal TF/P ratio for inulin in period A was similar to that in period B. In contrast, the early distal TF/P ratio for inulin increased significantly following epinephrine infusion in group II. Fractional sodium delivery to the early distal tubule was unaffected by the epinephrine infusion and resultant increase in renal perfusion pressure which occurred in group I (Fig. 2). However, primarily as a result of the increase in the TF/P ratios for inulin fractional sodium delivery to the early distal tubule decreased following epinephrine infusion in group II, from 8.31 ± 0.68% to 6.58 ± 0.54% (P < 0.005) (Fig. 2).

The mean TF/P ratios for both Na⁺ and inulin in the

### Table 1 Epinephrine Studies: Clearance Data

<table>
<thead>
<tr>
<th></th>
<th>GFR (ml/min)</th>
<th>FE (%)</th>
<th>Uᵢₑᵥₑ (μEq/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Period A</td>
<td>Period B</td>
<td>Period A</td>
</tr>
<tr>
<td>Group I (9 rats)</td>
<td></td>
<td></td>
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<tr>
<td>SEM</td>
<td>3.62*</td>
<td>3.65</td>
<td>2.31</td>
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<tr>
<td>P</td>
<td>&gt;0.8</td>
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<td>Group II (7 rats)</td>
<td>3.77</td>
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<td>2.10</td>
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<td>SEM</td>
<td>0.22</td>
<td>0.17</td>
<td>0.35</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.001</td>
<td></td>
<td>&lt;0.1 &gt; 0.05</td>
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</table>

GFR = glomerular filtration rate; FE = fractional excretion of sodium; Uᵢₑᵥₑ = absolute sodium excretion.

* GFR represents values from both kidneys.

### Table 2 Epinephrine Studies: Micropuncture Data

<table>
<thead>
<tr>
<th>SNGFR (ml/min)</th>
<th>Na Early distal TF/P</th>
<th>In Early distal TF/P</th>
<th>Na⁺/In × 100</th>
<th>Na⁺/In × 100</th>
<th>Late distal TF/P</th>
<th>Na⁺/In × 100</th>
<th>Na⁺/In × 100</th>
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</thead>
<tbody>
<tr>
<td>A</td>
<td>B</td>
<td>A</td>
<td>B</td>
<td>A</td>
<td>B</td>
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<td>Group I</td>
<td>72.0</td>
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<td>0.35</td>
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<tr>
<td>SEM</td>
<td>1.9</td>
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<td>0.01</td>
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<td>0.09</td>
<td>0.21</td>
<td>0.47</td>
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<tr>
<td>P</td>
<td>&gt;0.1</td>
<td>&gt;0.6</td>
<td>&gt;0.5</td>
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<td>&gt;0.4</td>
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<tr>
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<td>14/8</td>
<td>17/9</td>
<td>14/8</td>
<td>17/8</td>
<td>20/9</td>
<td>17/8</td>
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<tr>
<td>Group II</td>
<td>73.9</td>
<td>64.1</td>
<td>0.30</td>
<td>0.28</td>
<td>0.34</td>
<td>0.32</td>
<td>0.68</td>
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<tr>
<td>SEM</td>
<td>2.7</td>
<td>2.6</td>
<td>0.01</td>
<td>0.01</td>
<td>0.14</td>
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<tr>
<td>P</td>
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<td>14/7</td>
<td>11/6</td>
</tr>
</tbody>
</table>

A and B = experimental periods A and B; SNGFR = single nephron glomerular filtration rate measured in the early distal tubule; TF/P = tubular fluid to plasma ratio; In = inulin; Na⁺/In × 100% = fractional delivery of Na⁺ as percent of filtered load; n = number of tubules/number of rats.
superficial late distal tubule are not significantly different when one compares period A with period B in groups I and II (Table 2). However, when the ratios are considered together (Table 2 and Fig. 3) fractional sodium delivery to the late distal tubule was observed to decrease significantly in group II, from $3.71 \pm 0.46\%$ to $2.58 \pm 0.53\%$ ($P < 0.05$). In contrast, the increased renal perfusion pressure which was induced by the epinephrine infusion in group I did not alter fractional sodium delivery to the late distal tubule (Fig. 3, Table 2).

**BILATERAL CAROTID OCCLUSION AND VAGOTOMY**

The results for group I of the epinephrine series indicated that sodium excretion may increase following an acute elevation in the renal perfusion pressure without an associated change in superficial late distal sodium delivery. To examine the effect of an acute increase in renal perfusion pressure, induced by a means other than epinephrine, on the relationship between superficial late distal sodium delivery and fractional sodium excretion, in six experiments the pressure was elevated by carotid occlusion and vagotomy. The clearance and micropuncture results from these six studies are shown in Table 3.

Following ligation of the left carotid artery and bilateral vagotomy, the renal perfusion pressure increased from $122 \pm 5.9$ mm Hg (period A) to $169 \pm 3.1$ mm Hg (period B), values comparable to those after epinephrine (group 1). Although the GFR did not increase significantly, both fractional and absolute sodium excretion rose dramatically and significantly after the acute elevation in the renal perfusion pressure. Again, as in the epinephrine, group I, series, fractional sodium delivery to the superficial late distal tubule was similar prior to and following bilateral carotid occlusion and vagotomy ($3.55 \pm 0.41\%$ in period A and $4.24 \pm 0.42\%$ in period B; $P > 0.1$) ($n = 13$ tubules).

In Figure 4 a comparison between fractional sodium

| Table 3 Bilateral Carotid Occlusion and Vagotomy Studies |
|----------------|----------------|
|                | Period A       | Period B       |
| GFR (ml/min)   | $3.69 \pm 0.22$| $4.32 \pm 0.19$|
| FE (%)         | $1.80 \pm 0.71$| $6.40 \pm 1.0$|
| $U_{me} \cdot V$ (µEq/min) | $9.3 \pm 3.4$| $38.2 \pm 4.6$|
| Late distal TF/P$_{au}$, $\times 100\%$ | $3.55 \pm 0.41$| $4.24 \pm 0.42$|

Abbreviations used have the same meaning as in Tables 1 and 2. These data, expressed as mean ± SEM, were obtained from six rats; 13 superficial late distal tubules were studied.
delivery to the end of the superficial late distal tubule and fractional sodium excretion can be made for each period in each group of studies. In this figure the late distal values are not the mean of the individual observations, but are derived by using the mean value for each experiment. In period A for all three series of studies, fractional sodium delivery to the superficial late distal tubule was significantly greater than the simultaneously measured fractional sodium excretion (P < 0.02, P < 0.05, and P < 0.001 for the groups presented in Figure 4). In period B the fractional sodium excretion of 5.11 ± 0.65% was significantly greater than fractional sodium delivery to the superficial late distal tubule, 3.90 ± 0.55% in group I of the epinephrine studies (P < 0.01). In all six of the studies in which bilateral carotid occlusion and vagotomy were used to increase the renal perfusion pressure, although the magnitude of the increase in arterial pressure was less between periods A and B. In group I of the epinephrine studies and following bilateral carotid occlusion and vagotomy, the increment in fractional sodium excretion from period A to B was significantly greater (P < 0.001) for both, than the modest increases in fractional sodium delivery to the superficial late distal tubule. This clearly indicates that the observed increase in sodium excretion which resulted from the acute elevation in the renal perfusion pressure was significantly greater than could be accounted for by the change in sodium delivery to the superficial late distal tubule. In group II of the epinephrine studies, the change in late distal sodium delivery was not different from the observed fall in fractional sodium excretion (P > 0.5).

Discussion

In rats undergoing a modest saline diuresis, an acute elevation in the renal perfusion pressure resulted in a marked increase in both fractional and absolute sodium excretion. Furthermore, this increased natriuresis occurred without a change in either the total GFR or superficial SNGFR, and, undoubtedly, reflected an effect of perfusion pressure on tubular sodium transport. Of particular note was that the greater natriuresis observed after the elevation in the renal perfusion pressure was not the result of an increase in sodium delivery from the superficial late distal tubule, but the fractional sodium delivery to this site was unaltered by the increased renal perfusion pressure. The enhanced sodium excretion seen in this experimental setting, therefore, must be related either to an inhibition of sodium reabsorption in inner cortical nephrons or to an effect on sodium transport in the collecting system. In contrast, elevation of the systemic arterial blood pressure, while the renal perfusion pressure was kept constant, did not significantly affect fractional sodium excretion.

In the first two series of studies, intravenous epinephrine was selected as the means to increase the renal perfusion pressure because by this it was possible to carefully control the increase in arterial blood pressure to the desired level and keep it constant (within 5 mm Hg) without effecting a change in GFR. This method of increasing the renal perfusion pressure when performed in the presence of a modest saline diuresis permitted changes in sodium transport of a magnitude which could be readily detected with micropuncture techniques.

As there is a qualitative similarity in the response of urinary sodium excretion to an acute enhancement of the renal perfusion pressure induced by a number of different experimental maneuvers, it can be suggested that it is the change in renal perfusion pressure and not the means by which it is accomplished that is primarily responsible for the effect of perfusion pressure on sodium excretion. In the present studies it is assumed that it was the ability of epinephrine to increase the renal perfusion pressure, and not an intrarenal effect of the drug, which was responsible for the increased natriuresis observed after its administration. The results of the group II studies provide evidence in favor of this contention. Further support is obtained from the studies in which bilateral carotid occlusion and vagotomy were used to increase the renal perfusion pressure. Although the magnitude of the increase in arterial pressure was less
predictable and the pressure less stable following bilateral carotid occlusion and vagotomy than after epinephrine, the results (Fig. 4) are remarkably similar and permit similar conclusions to be drawn.

The fact that epinephrine is, of itself, not natriuretic is further supported by the observations of others. Nickel et al.\textsuperscript{13} reported that the intravenous administration of nonpressor doses of epinephrine to man resulted in a decrease in renal plasma flow and the urinary excretion of sodium without a detectable change in the GFR. In addition, when studied in either the isolated frog skin preparation\textsuperscript{14} or the isolated perfused dog kidney,\textsuperscript{15} no consistent effect of epinephrine on sodium transport has been observed. Finally, it should be noted that the natriuretic effect of pure $\beta$-adrenergic stimulation is minimal at best and much less than the natriuretic effect observed in group I.\textsuperscript{16} Also, in view of the observation that the GFR fell in group II after epinephrine infusion it would appear that the $\alpha$-adrenergic effect of epinephrine likely predominated, an effect which is felt to be antinatriuretic.\textsuperscript{17}

The data provided by this and by previous studies do not permit a choice to be made between the two possible means by which an increase in renal perfusion pressure increases sodium excretion. Sodium transport in the mammalian collecting system, as in many other transporting epithelia, would appear to be characterized by bidirectional sodium movement\textsuperscript{18} with sodium efflux from the lumen usually greatly exceeding influx. However, it appears, at least in the medullary collecting duct, that under certain conditions the rate of sodium influx into the lumen may exceed the rate of efflux and result in net addition of solute as well as of water to the collecting duct urine.\textsuperscript{19} The present findings could be explained if an acute increase in renal perfusion pressure were to enhance the rate of sodium influx relative to sodium efflux. The demonstration of "net addition" of sodium between the end of the superficial distal convoluted tubule and the final urine would then obviously depend upon the rate of sodium influx relative to sodium efflux.

Alternatively, the increased natriuresis observed in period B of group I and following carotid occlusion and vagotomy may be accounted for if sodium reabsorption in some portion of the inner cortical nephrons were responsive to and decreased by the acute increase in renal perfusion pressure. If this is the case, an increase in sodium delivery to the collecting system distal to the entrance of the superficial distal tubule would occur.

The present study should not be considered to contradict the previously reported results of micropuncture studies which demonstrate a decrease in fractional sodium reabsorption in the proximal tubule of superficial nephrons after acute elevation of the renal perfusion pressure.\textsuperscript{20} If one assumes that an increase in sodium delivery from the proximal convolution occurred in the present experiments, as in the other micropuncture studies, this increment must have been reabsorbed in the superficial loop of Henle because sodium delivery to the early distal tubule was not enhanced by the elevated renal perfusion pressure.

Finally, it should be noted that the results of the present experiments differ in several aspects from the observations made on the rat kidney subjected to a chronic increase in blood pressure. In the unclamped kidney of a rat with one-kidney Goldblatt hypertension, delivery of sodium to the early distal tubule was increased when compared to either the contralateral kidney subject to the arterial clamp or an appropriate control kidney.\textsuperscript{21} These results, which are not in agreement with the conclusions of the present study, were taken to indicate that sodium reabsorption was decreased in the superficial loop of Henle by the chronic hypertensive state. In addition, the GFR in these unclamped kidneys was enhanced as a result of an increase in the filtration rate of juxtamedullary nephrons,\textsuperscript{22} an increase felt to be related to the increased blood pressure. As shown above, the GFR in the present studies was not altered by the acute increase in renal perfusion pressure.

In summary, our results confirm the observations of previous studies which indicate that sodium excretion can increase dramatically following an acute increase in renal perfusion pressure. This increase in sodium excretion is not the result of an enhancement of sodium delivery from the superficial distal tubule but represents an effect of perfusion pressure on sodium transport in inner cortical nephrons or the collecting system.

References

The Myocardial Energetic Active State

I. Oxygen Consumption during Tetanus of Cat Papillary Muscle

GEORGE COOPER, IV, M.D.

SUMMARY The potential role of active state maintenance as a determinant of myocardial oxygen consumption (MVO₂) has not been defined. Right ventricular papillary muscles from 15 cats were studied in a polarographic myograph at 23°C in a Krebs-Ringer solution containing 7.5 mM Ca²⁺ and 10 mM caffeine. MVO₂ was determined for isometric tetani of 1-5 seconds' duration. Increases in tetanus duration related linearly to increments in both active tension time (active tension) and MVO₂. In order to examine the oxygen cost of active state maintenance not attributable to associated tension generation, both the same isometric and 2.5- to 10.0-second lightly preloaded isotonic tetani were produced in nine muscles. For each tetanus duration the contribution throughout the contraction of developed force (preload) to MVO₂ could be subtracted from overall isotonic MVO₂. In the absence of the MVO₂ associated with force development, the active state duration was related linearly to MVO₂, with a mean active state MVO₂ of 2.42 ± 0.29 nL O₂/mg dry muscle/sec of isotonic tetanus; this MVO₂ is 68% of the value of 3.58 ± 0.42 nL O₂/mg dry muscle/sec that was obtained for isometric tetanus at Lmax. This study identifies active state maintenance as the major determinant of MVO₂ during myocardial tetanus and, furthermore, suggests the possibility that alterations in active state intensity and duration may be the biochemical mechanism by which other determinants of MVO₂ act in a more physiological setting.

THE HEART is almost exclusively an aerobic organ; therefore, steady state oxygen consumption provides a reasonably precise estimate of overall myocardial metabolism. In a recent review¹ tension development, contractile state, and heart rate were stated to be the major determinants of myocardial oxygen consumption, with current information suggesting that other factors are less important. However, tetanus of cardiac muscle, with variation of active state duration independent of changes in other determinants of myocardial oxygen consumption, has not previously been possible. Therefore, the potential contribution of active state maintenance to myocardial oxygen consumption has not been determined. The purpose of this investigation was to combine a recently described method for myocardial tetani² with the polarographic papillary muscle bath³,⁴ to define what role active state maintenance may have in determining myocardial oxygen consumption.

Methods

MEASUREMENT OF MECHANICAL ACTIVITY AND OXYGEN CONSUMPTION OF CAT PAPILLARY MUSCLES

The procedures and myograph used were essentially those described by Coleman,⁵ with any modifications described below.

Rapid cardiectomy was performed on adult cats (weighing 1.36-2.72 kg) after anesthesia was induced with sodium pentobarbital (30 mg/kg, ip). The infundibulum of the right ventricle then was opened and flushed with Krebs-Ringer solution saturated at 25°C with 95% O₂-5% CO₂. A papillary muscle was excised from the right ventricle and mounted in the polarographic myograph as previously described.⁶ Mean papillary muscle dimensions at Lmax (that length at which maximum isometric tension is generated) were: length, 6.11 mm (range 4.27-9.56 mm), and cross-sectional area, 0.77 mm² (range 0.46-1.12 mm²). Muscles of these dimensions are not diffusion-limited so that adequate metabolic support is assured. The ratio of resting tension to total tension (active tension + resting tension) at Lmax has been used as an index of the mechanical suitability of any particular cat papillary muscle preparation. This ratio for these 15 muscles, contracting at Lmax at a rate of 0.2 Hz at 23°C in the bathing solution described below, was 15.34 ±

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