STUDIES IN EDEMA. II. THE EFFECT OF CONGESTIVE HEART FAILURE ON SALIVA ELECTROLYTE CONCENTRATIONS

BY ABRAHAM G. WHITE,¹ HAROLD GORDON,² AND LOUIS LEITER

(From the Medical Division, Montefiore Hospital, New York City)

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In congestive heart failure the urinary excretion of sodium and chloride following the administration of sodium chloride is decreased as compared with the normal (1–4). Similarly, the sweat concentration of sodium chloride has been shown to be lower than normal in cases of congestive heart failure (5). To explore the frequency of these disturbances of electrolyte excretion it seemed of interest to study the concentrations of electrolytes in the saliva of patients with congestive heart failure on a low-salt regimen and on a regular salt intake. Similar control studies were performed on non-cardiac subjects.

EXPERIMENTAL

Twenty-nine observations were made on 27 congestive heart failure patients receiving one gram of sodium chloride per day in their diet. There were 13 studies on 11 cardiac failure patients on a regular diet containing 10 grams of salt per day. Of these 11 cardiac patients on the regular diet four received supplemental sodium chloride (4 grams daily for periods up to 16 days). Twelve control observations on ten non-cardiac subjects receiving the salt-poor regimen were obtained, while 16 non-cardiac subjects on the regular diet were studied.

METHODS

Saliva was collected in the morning from the fasting patient who chewed paraffin for 15 to 20 minutes, yielding about 30 cc. of saliva. Whole, mixed saliva was analyzed. Sodium and potassium concentrations were obtained with an internal standard Perkin 52A flame photometer. Chloride concentrations were measured by the Van Slyke-Hiller iodometric method (6).

Standard methods of statistical analysis were employed (7).

RESULTS

Results are presented in Table I.

When data from studies on the low-salt regimen and on the regular diet are combined, it is seen that congestive heart failure is associated with lowered sodium, lowered chloride, and higher potassium concentrations (mean concentrations) in saliva than is the case for the controls. There is no significant difference in electrolyte concentrations between congestive heart failure patients on a low-salt regimen and those on a regular diet. In contrast, non-cardiac, control subjects on a low salt regimen show a significantly lower average concentration of salivary sodium and chloride than those controls receiving a regular diet.

Thus, the salivas of subjects on salt-poor diets do not show significant difference in mean electrolyte concentrations between congestive heart failure patients and non-cardiac controls. On the other hand, studies of subjects on regular diets emphasize the lowered saliva sodium and chloride concentrations in congestive heart failure noted above in reporting the combined data.

In this study, no correlation was found between the electrolyte concentrations of saliva and blood.

DISCUSSION

The saliva concentrations of sodium, chloride and potassium in the controls on the regular diet agree well with those reported previously (8–10). McCance (11) observed that salt deficiency (produced during a period of seven to ten days by restriction of dietary salt intake and sweating in a radiant-heat bath for two hours every day) in five normal subjects produced a consistent fall in the concentration of salivary sodium and rise in the concentration of potassium, while the changes in chloride concentration were inconsistent. In each of our seven control subjects who were changed directly from a normal diet to a salt-poor regimen, the sodium and chloride concentrations fell while that of the potassium rose.

Do the lower mean concentrations of sodium and chloride in saliva in chronic congestive heart failure represent an increased adrenal cortical activity, as has been postulated for sweat (5, 12)? This might explain why increasing the daily salt
intake at least tenfold in cardiac failure patients for periods up to 16 days did not affect the saliva electrolyte concentrations. Increased adrenal cortical activity might promote retention of sodium and chloride by the salivary glands just as it does by the kidneys and sweat glands, despite an increased intake of salt.

Desoxycorticosterone acetate (10 mgm. in oil, daily for three days) was administered to three cardiac failure patients on a low-salt regimen without any effect upon saliva electrolyte concentrations. This, too, is to be expected if we postulate an already enhanced adrenal cortical activity in these patients.

To investigate this question of enhanced adrenal cortical activity affecting salivary electrolyte concentrations, studies are in progress on the administration of desoxycorticosterone acetate for longer periods to normal subjects on a regular diet, and on patients receiving cortisone and ACTH. The salivary electrolyte concentrations in disorders of the adrenal cortex such as Addison’s disease and Cushing's syndrome will be of interest in this connection.

Since mercurial diuretics increase the excretion of sodium and chloride in urine, their effect on saliva electrolyte concentrations was studied. In two cardiac failure patients mercurial diuresis was without effect on the saliva electrolytes, determined 24 hours after the intramuscular injection of the diuretic. However, the saliva electrolytes were determined after the peak of mercurial diuresis. This study will be repeated with saliva specimens collected at earlier intervals following the injection of the diuretic.

### CONCLUSIONS

1. Congestive heart failure is associated with lowered sodium, lowered chloride, and higher potassium average concentrations in saliva.

2. Studies of subjects on regular diets emphasize the lowered saliva sodium and chloride concentrations in congestive heart failure, but tend to minimize the higher potassium concentrations.

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### TABLE I

The effect of congestive heart failure and diet on saliva electrolyte concentrations

<table>
<thead>
<tr>
<th>Patients</th>
<th>Diet</th>
<th>Number observations</th>
<th>Na mEq/L</th>
<th>Cl mEq/L</th>
<th>K mEq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>$\bar{x}$</td>
<td>$S_x$</td>
<td>$S_K$</td>
</tr>
<tr>
<td>Cong. ht. fail. (27)</td>
<td>Salt-poor</td>
<td>29</td>
<td>18.6</td>
<td>9.3</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>Regular</td>
<td>13</td>
<td>16.8</td>
<td>7.6</td>
<td>2.1</td>
</tr>
<tr>
<td></td>
<td>t</td>
<td>0.64</td>
<td>0.62</td>
<td>0.77</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controls (10)</td>
<td>Salt-poor</td>
<td>12</td>
<td>17.6</td>
<td>10.2</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>Regular</td>
<td>16</td>
<td>33.1</td>
<td>13.4</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>t</td>
<td>2.95</td>
<td>0.006</td>
<td>3.91</td>
<td>0.0005</td>
</tr>
<tr>
<td></td>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cong. ht. fail. (28)</td>
<td>Combined</td>
<td>42</td>
<td>18.0</td>
<td>8.7</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td>Combined</td>
<td>28</td>
<td>26.5</td>
<td>14.3</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>t</td>
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<td>0.006</td>
<td>3.02</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>p</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$\bar{x}$ = mean.  
$S_x$ = Standard deviation.  
$S_K$ = Standard error of the mean.  
$t = \frac{\bar{d}}{S_d}$, where $\bar{d}$ = difference between the means  
$(S_d)^2 = (S_x)^2 + (S_K)^2$.  
$p$ = probability if $p = 1.0$, then the observed difference is entirely a chance occurrence.  
$p$ = probability if $p = 0.0$, then there is no element of chance.
3. Salivas of subjects on salt-poor diets do not show significant difference in electrolyte concentrations between congestive heart failure subjects and controls.

4. There is no relationship between serum electrolyte concentrations and those of saliva.

**BIBLIOGRAPHY**


