The Distribution of Body Fluids In Congestive Heart Failure

II. Abnormalities in Serum Electrolyte Concentration and in Acid-Base Equilibrium

By R. D. Squires, M.D., R. B. Singer, M.D., G. R. Moffitt, Jr., M.D., and J. R. Elkinton, M.D.

The distribution of values of serum concentrations of sodium, chloride, potassium, and total carbon dioxide were studied in a large group of patients with congestive heart failure, and compared with the values in a smaller group of normal subjects used as controls. The depression of sodium and chloride concentration, and the elevation of total carbon dioxide content were evaluated in terms of renal function and mercurial therapy. In 11 of the cases with high serum contents of total carbon dioxide the acid-base equilibrium was precisely defined by measurement of pH in cutaneous whole blood and calculation of the pressure of carbon dioxide and of the concentration of buffer base.

The plasma or serum of blood is the most readily sampled portion of the body fluids. It would therefore seem logical to begin an investigation of disturbances in distribution of water and electrolytes in any clinical condition by observing the concentration of some of the principal electrolytes in plasma or serum. Such a survey in patients with congestive heart failure is the subject of this report, and is the first step in a study of some of the problems outlined in the preceding paper.

**Experimental Material**

Fifty-four samples of serum from 44 patients with congestive heart failure were analyzed for sodium, chloride, carbon dioxide, and potassium. Multiple samples in the same patient were obtained, with two exceptions, on different admissions to the hospital; in the two exceptions, the second sample was obtained after a therapeutic regime of many days not involving the use of sodium solutions. The patients were selected only insofar as they were patients with clinical heart disease associated with predominately peripheral edema*; patients with pulmonary edema only were not included. Likewise, patients with right heart failure and edema which was secondary to primary pulmonary disease (cor pulmonale) were excluded from the series. This was done to obviate the effects of primary pulmonary dysfunction on carbon dioxide elimination and hence on the acid-base balance.

The etiologies of the heart disease in the group, in order of decreasing frequency, were as follows: chronic rheumatic heart disease, 16; hypertensive heart disease, 15; arteriosclerotic heart disease, seven; syphilitic heart disease, two; constrictive pericarditis, two; active rheumatic myocarditis, one; and congenital heart disease, one. No attempt is made to present

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* The cardiac patients studied in this and in the following papers had clinical evidence of congestive failure; that is, increased venous pressure as shown by jugular distention or by direct measurement, as well as by peripheral edema.
the serum concentrations of electrolytes according to organic etiology of the heart disease since no particular correlations were apparent on preliminary analysis.

FIG. 1. The distribution of serum concentrations of sodium in normal subjects and in patients with "cardiac edema," that is, congestive heart failure.

In the presentation of the data as curves of distribution of each electrolyte concentration considered (figs. 1 to 4), the observations are labeled according to certain pathologic variables or conditions that seemed most likely to affect the phenomena being analyzed. The symbols for these variables are designated in the legend of each figure and were assigned according to the following criteria. Patients who had received mercurial diuretics are scored according to whether the last dose had been given within two days or two weeks of the time the serum analysis was made; no indication is given of the duration or frequency of mercurial therapy. All of the patients had been, and were, on low sodium diets. Patients with renal insufficiency were arbitrarily separated from the others by the presence of azotemia as in-

FIG. 2. The distribution of serum concentrations of chloride in normal subjects and in patients with "cardiac edema."

FIG. 3. The distribution of serum contents of total carbon dioxide in normal subjects and in patients with "cardiac edema."

FIG. 4. The distribution of serum concentrations of potassium in normal subjects and in patients with "cardiac edema."
dicated by a blood urea nitrogen content of more than 20 mg. per 100 cc. These patients were then subdivided into those who did or did not have intrinsic renal disease, according to the history, physical examination for evidence of extensive vascular damage and hypertension, and laboratory findings. One patient who had lost gastrointestinal fluids by vomiting or diarrhea within one week of the time of study was so designated.

Eleven of the patients were selected for detailed blood studies of their acid-base balance. These patients were mainly from those who had exhibited an elevation in the total carbon dioxide content of venous serum.

In 19 of the patients extensive studies were made of the internal and external transfers of water and electrolytes, by the balance technic. These studies are presented in the subsequent communications.

Serum concentrations of electrolytes were determined in 21 control subjects for purposes of comparison with the patients with congestive heart failure. These control subjects were healthy, active young adult male and female technicians, nurses, and physicians.

**CHEMICAL METHODS**

Sodium and potassium in serum were determined by use of a Barclay internal standard flame photometer. In our hands the standard deviation (σ) about the mean of duplicates was for sodium, ±0.75 per cent, and for potassium, ±1.36 per cent. Significant differences, therefore, are those that are greater than ±2σ, or approximately ±2.1 mEq. per liter of sodium and ±0.11 mEq. per liter of potassium.

Chloride was measured by the method of Eisenman and the total carbon dioxide content of serum by the method of Van Slyke and Stadie. The acid-base equilibrium of cutaneous whole blood was determined by the microtechnic of Shock and Hastings. From the pH, total carbon dioxide content, and hematocrit values were calculated the pressure of carbon dioxide (pCO₂) and the concentration of buffer base (BBB) according to the nomogram of Singer and Hastings.

**RESULTS**

The results of the study are presented in figures 1 to 5 and in table 1. In the distribution curves in figures 1 to 4, the patients with "cardiac edema," that is, with congestive heart failure, who had no evidence of renal insufficiency or vomiting, are compiled in group B; all the cardiac patients are compiled in group C. The mean values and their standard deviations are listed beside each group. The range of two standard deviations from the mean of the control group A is indicated over the curves of groups B and C by the dotted lines; any individual value in these groups that lies outside this range is taken to have less than a 5 per cent probability of being in the control group (p = 0.05).

**Sodium.** The concentration of sodium in serum was lower than normal in many of the cardiac patients; in only two instances was the level elevated (fig. 1). The mean value in all

![Fig. 5. The acid-base data on 12 edematous cardia

The cardiac cases (group C) was 132.8 ± 8.1 mEq. per liter. This is a highly significant difference between the means of these two groups, the p value being < 0.001. The mean value for the cardiac patients without azotemia or vomiting (group B) was 134.7 ± 7.2 mEq. per liter and also differed significantly from the mean of the control group (p < 0.01). Within group B only two of the seven cases who had different mercurials lay outside the normal range. On the other hand, the cases who had received mercurials were distributed over both normal and depressed values.

**Chloride.** The concentration of chloride in serum was, with one exception, normal or low (fig. 2). The mean value for all the cardiac
cases (group C) was 91.5 ± 11.2 mEq. per liter, compared to the mean for the controls (group A) of 100.9 ± 1.6 mEq. per liter. The mean value for the selected cardiae (group B) was 92.5 ± 7.2 mEq. per liter. Both of these mean values differed significantly from that of the controls (p < 0.01 and < 0.001 respectively). In the cardiae selected for absence of vomiting and azotemia (group B) six of the seven cases who had not received mercurials were essentially within the normal range, whereas all but one of those who had received mercurials were below the normal range.

**Total Carbon Dioxide.** The total content of carbon dioxide in venous serum spread over a wide range of values in all the cardiae, but tended to be elevated in the selected cardiac patients (group B) (fig. 3). This latter distribution is a reflection of the fact that the concentration of chloride was depressed to an extent greater than that of sodium, and indicates that

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**Table 1. Analysis of Acid-Base Equilibrium in Blood of Patients with Heart Disease and Congestive Failure, Who Had No Antecedent Vomiting or Alkali Ingestion, and no Recognized Pulmonary Emphysema or Fibrosis**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Cardiac diagnosis</th>
<th>Venous serum</th>
<th>Cutaneous whole blood</th>
<th>Acid-base diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>CO₂</td>
<td>Cl</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>mM. per liter</td>
<td>mM. per liter</td>
<td>cell volume</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>mM. per liter</td>
<td>mM. per liter</td>
<td>CO₂</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>pH</td>
<td>pCO₂</td>
<td>buffer base</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>mm. Hg</td>
<td>mEq. per liter</td>
<td></td>
</tr>
<tr>
<td>Normal adult</td>
<td></td>
<td></td>
<td>22 ± 2</td>
<td>7.40 ± 0.06</td>
<td></td>
</tr>
<tr>
<td>A. W.</td>
<td>M</td>
<td>Constrict.</td>
<td>26.5</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>E. G.*</td>
<td>M</td>
<td>Hypertensive</td>
<td>28.4</td>
<td>42.0</td>
<td></td>
</tr>
<tr>
<td>G. B.</td>
<td>F</td>
<td>Chr. rheumatic</td>
<td>32.0</td>
<td>19.5</td>
<td></td>
</tr>
<tr>
<td>S. M.</td>
<td>M</td>
<td>Chr. rheumatic</td>
<td>39.5</td>
<td>22.9</td>
<td></td>
</tr>
<tr>
<td>J. C.</td>
<td>M</td>
<td>Arteriosclerotic</td>
<td>33.4</td>
<td>44.0</td>
<td></td>
</tr>
<tr>
<td>F. P.†</td>
<td>M</td>
<td>Chr. rheumatic</td>
<td>35.1</td>
<td>20.1</td>
<td></td>
</tr>
<tr>
<td>E. S.</td>
<td>F</td>
<td>Hypertensive</td>
<td>33.4</td>
<td>44.5</td>
<td></td>
</tr>
<tr>
<td>H. H.‡</td>
<td>F</td>
<td>Arteriosclerotic</td>
<td>—</td>
<td>29.6</td>
<td></td>
</tr>
<tr>
<td>J. B.</td>
<td>M</td>
<td>Chr. rheumatic</td>
<td>35.1</td>
<td>22.0</td>
<td></td>
</tr>
<tr>
<td>G. C., I§</td>
<td>F</td>
<td>Chr. rheumatic</td>
<td>40.5</td>
<td>7.55</td>
<td>37 57</td>
</tr>
<tr>
<td>G. C., II§</td>
<td>F</td>
<td>Chr. rheumatic</td>
<td>37.8</td>
<td>55.5</td>
<td>49 62</td>
</tr>
<tr>
<td>L. D.</td>
<td>F</td>
<td>Hypertensive</td>
<td>33.8</td>
<td>54.5</td>
<td>52 57</td>
</tr>
</tbody>
</table>

* Lobar pneumonia of left lower lobe and right pyothorax present.
† Pulmonary infarcts found at autopsy.
‡ Bilateral bronchopneumonia present.
§ Pleural effusion.
|| In these patients, studies of pulmonary function by Comroe did not show the increased residual air capacity and lack of uniformity of alveolar gas mixing which are characteristic of emphysema. In all except patient J. B., the arterial O₂ saturation while breathing room air was low.

In general the concentration of other undetermined anions was not increased. The mean value for all the cardiac cases (group C) was 29.45 ± 7.45 mM. per liter, compared with a mean for the controls (group A) of 28.71 ± 1.47 mM. per liter. These two values are not significantly different. The mean value for the cardiae without azotemia or vomiting
(group B), however, was 32.17 ± 4.87 mEq. per liter, and differs significantly from the control value (p < 0.01). Patients who had not received mercurials, as well as those who had, were distributed within and without the range of control values.

Undetermined Anion Concentration. The difference between the serum concentration of sodium and carbon dioxide plus chloride, used as a measure of undetermined anion concentration, was 9.9 ± 4.7 mEq. per liter for group B compared to 9.5 ± 2.4 for the controls, group A; both of these values are within normal limits of 0 to 11 mEq. per liter.\(^9\) This value for all the cardiax, group C, was 11.7 ± 6.4, and is slightly higher than the control group because of the presence of renal insufficiency. These data are not presented graphically.

Potassium. The concentration of potassium in serum was, with few exceptions, normal or elevated (fig. 4). The mean value for all the cardiax (group C) was 4.62 ± 1.04 mEq. per liter compared with a control mean (group A) of 4.00 ± 0.33 mEq. per liter. The mean for the selected cardiax (group B) was 4.17 ± 0.47 mEq. per liter, and does not differ significantly from the control group. In group B very few of those cases who had, or who had not, received mercurials lay outside the range of control values.

Acid-Base Equilibrium. The data on 11 selected patients are given in table 1, and are presented graphically in figure 5. Most of these patients had elevated concentrations of total carbon dioxide in venous serum. In nine of the 12 studies the pH of cutaneous blood was elevated (above 7.46). The values for the partial pressure of carbon dioxide and buffer base were calculated to evaluate the respiratory and metabolic factors involved in the acid-base disturbance. If the normal range of carbon dioxide pressure in cutaneous blood is taken to be 35 to 47 mm., six of the 12 samples show an abnormally low carbon dioxide pressure, or a carbon dioxide deficit resulting from hyperventilation, and four show an abnormally high carbon dioxide pressure or carbon dioxide retention. The values of whole blood buffer base concentration are in the normal range of 46 to 52 mEq. per liter (normal for a red cell volume of 45 per cent) in only two cases; in the remaining ten there is an excess of buffer base, or metabolic alkalosis. The co-existence of carbon dioxide deficit and buffer base excess in four of the patients indicates that both the respiratory and metabolic factors have undergone a primary disturbance, which can be called a “mixed alkalosis.” In the three samples characterized by carbon dioxide retention and buffer base excess it is impossible to state with certainty whether both disturbances are primary or whether one is secondary to the other (compensatory elevation of the buffer base in response to primary carbon dioxide retention, or vice versa).

**Interpretation of Results**

The interpretation of the results of these serum and blood studies depends to a large extent on the clinical evaluation of the patients and on the balance data obtained during treatment of some of them. Although the latter are to be presented subsequently, certain statements appear justified from the results just detailed.

Serum sodium concentration was definitely lowered in many of these cardiax and for the group as a whole, even when those with vomiting or renal insufficiency were excluded. Thus these two latter conditions, while augmenting the hyponatremia, do not account for it. On the other hand, six of the seven patients who had received no mercurial diuretics had serum sodium levels within the normal range. The inference is clear that the mercurial therapy was directly related to the hyponatremia. Unfortunately, the paucity of cardiax who had not received mercurial diuretics prevents subjecting this inference to a statistical analysis. But it would appear that, at least in the edematous cardiac who has received mercury, hyponatremia is a common finding. No index of frequency, as opposed to recency, of mercurial injection is available in this study. Yet this may be the more significant factor of the two since neither patient F. J. in the succeeding study\(^2\) nor the subjects observed by Blumgart and his co-workers\(^8\) developed hyponatremia during massive diuresis resulting from a single dose of mercurial drug. Whatever the effective factors may be in respect to mercurial
administration, such hyponatremia must be due to deficit of sodium (and total electrolyte in the body fluid) relative to water even though the total quantities of both of these substances in the body are in excess of normal. Whether this hypotonicity of the body fluids is due to loss of salt relative to water, or to retention of water relative to salt, cannot be stated from the data presented above. The known effect of mercury on the renal excretion of salt suggests the former explanation; the observation in patients reported in the succeeding paper of falling serum sodium levels when the sodium balance was in equilibrium or positive, suggests the latter explanation, that is, that water retention at least in part is responsible for the hyponatremia.

The serum chloride concentration was depressed to a greater extent than that of sodium, and the carbon dioxide content was elevated. This fact can hardly be explained by a primary effect of mercury on sodium excretion or by a retention of water. In the absence of renal failure and vomiting, the following possible explanations may be considered. (1) In mercurial diuresis in edematous patients chloride and sodium are excreted in approximately equivalent amounts, and therefore more chloride than sodium is lost relative to the usual proportions of sodium to chloride of 1.3 to 1.0 in extracellular fluid. The net effect of such losses of water, chloride, and sodium would be a fall in the concentration of chloride relative to sodium. This explanation of the hypochloremic alkalosis seems valid in view of the data presented in the next paper. (2) Chloride is excreted and the bicarbonate concentration of extracellular fluid thereby elevated, as a secondary response to a primary respiratory retention of carbon dioxide. The presence of a low carbon dioxide pressure in blood in a significant number of cases renders this explanation invalid for those cases and unlikely for the group as a whole. (3) Chloride is displaced by other undetermined anions. This is not a usual physiologic adjustment since bicarbonate ion (HCO3-) is usually displaced under these circumstances; the absence of a significant increase in group B over group A in the difference between the serum concentrations of sodium and carbon dioxide plus chloride makes this explanation untenable. (4) A metabolic alkalosis with hypochloremia was present as a result of intracellular potassium deficiency (according to the theory of Darrow). This possibility cannot be ruled out by the data above, and, as will be seen in the subsequent paper, many cardias retain potassium when administered and, therefore, are probably deficient in the ion. However, the absence of hypokalemia in these cases and the failure to maintain the normal concentration of chloride and carbon dioxide following potassium therapy, do not support this hypothesis.

The respiratory alkalosis indicated by the low carbon dioxide pressure calculated in some of the patients is the result of some stimulus or stimuli to the respiratory center other than a metabolic acidosis, since the latter was not present in the group without renal insufficiency. These stimuli cannot be identified completely from these data; presumably they are reflexes from the congested lung and/or anoxemia such as was found to be present in two of these cases (table 1). Respiratory retention of carbon dioxide (respiratory acidosis) was present in some of the cases. This may have been due primarily to poor ventilation of the alveoli although no clinical or functional evidences of permanent lung changes, notably emphysema and fibrosis, could be adduced in the particular cases in which the carbon dioxide pressure was elevated (table 1). In patient G. C. (1), who was anoxemic, the presence of pleural effusion and possibly pulmonary congestion may have interfered with the excretion of carbon dioxide. Patient J. B. was not anoxemic and had no functional evidence of emphysema (table 1). It is possible that in this patient and in patient L. D. the respiratory retention of carbon dioxide was a secondary and compensatory response to the metabolic alkalosis which was so frequently present, although such a response seldom occurs to any great magnitude.

**Discussion**

These findings were not entirely unexpected. Although Blumgart and his associates found the serum concentration of sodium and chloride to be essentially unchanged after mercurial diuresis, others have found the concentration of these ions to be lowered. Similar observa-
tions have led to the use of the term "low-salt syndrome" to describe the edematous patient who has become refractory to mercurial diuretics and who usually has a lowered concentration of sodium and of chloride in serum.\textsuperscript{17, 18} The physiologic mechanism by which this state is induced and its therapeutic implications are discussed in a subsequent paper.\textsuperscript{5} It appears to the authors that hyponatremia in cardiacs who have been receiving mercurials is more frequent than the low salt syndrome. This is true insofar as that syndrome is identified by a response to hypertonic sodium solutions in terms of improvement of the circulation, improvement of renal function, and diuresis of the edema. It is possible, therefore, that in some of the patients the hyponatremia represents the setting of a new level of total electrolyte concentration in body fluids, possibly by changes in cellular osmolarity, rather than by a systemic depletion of sodium.\textsuperscript{1, 5} But whatever may be the mechanism involved, hyponatremia and hypochloremia appear to be frequent sequelae of mercurial therapy.

Hypernatremia, on the other hand, was conspicuously absent in this group of patients. Peters\textsuperscript{19} has suggested that the renal abnormality in respect to sodium excretion in the cardiac is a mistaken "dehydration reaction," that is, an increased tubular reabsorption of sodium and chloride under conditions of water deprivation and rising concentration of sodium and chloride in serum.\textsuperscript{20} Presumably such a response would tend to result in a slightly elevated concentration of sodium in serum. Unfortunately these data cast no light on this problem because of the insignificant number of determinations of serum sodium concentration in patients with congestive heart failure who had received no mercurial diuretics during the preceding two weeks. Sodium chloride may have been retained in excess of or to an equivalent degree to water in the initial stages of the disease in these patients. But at the time these observations were made, almost always following mercurial therapy, the hyponatremia in the presence of edema can only mean that the excess of water exceeded that of salt.

The elevation of the concentration of serum bicarbonate in association with hypochloremia has been reported in cardiacs in the past.\textsuperscript{10, 21, 22} Peters, Bulger, and Eisenman\textsuperscript{22} interpreted this finding as indicating a secondary response to a primary pulmonary retention of carbon dioxide similar to that found in patients with emphysema and pulmonary fibrosis. Many of their patients had extensive pulmonary disease, but unfortunately no measurements of pH were recorded; it is not possible, therefore, to ascertain whether or not the carbon dioxide pressure was sufficiently elevated to confirm this interpretation. Winkler and Crankshaw\textsuperscript{22} interpreted in a similar manner the data which they obtained on a patient with congestive heart failure. Such a secondary response to a primary carbon dioxide retention in patients with heart failure usually has been thought to be restricted to those patients who also had extensive structural changes in the lungs, namely, emphysema and fibrosis. Our data indicate that an elevated serum bicarbonate concentration and hypochloremia frequently are not secondary responses to carbon dioxide retention occurring in those cardiacs who have extensive pulmonary disease. In only three of the 12 patients in whom a detailed study of the acid-base equilibrium was made, was there evidence of a pulmonary lesion other than passive congestion. And in at least one-half of the 12 patients the conjunction of a lowered carbon dioxide pressure with elevated buffer base rules out the latter as being a compensatory response to respiratory retention of carbon dioxide. A carbon dioxide deficit (or respiratory alkalosis due to a diminished carbon dioxide pressure in the alveoli) has been described\textsuperscript{24-26} and may be explained as the result of an anoxemia or abnormal pulmonary reflex stimulus to hyperventilation, but the compensatory renal response should be a retention of chloride, with a reduction of the buffer base, or a secondary metabolic acidosis. The metabolic alkalosis in these cases and in those in which the carbon dioxide pressure was normal, cannot be explained as a secondary or compensatory response to a primary retention of carbon dioxide by the lungs.

It is not unreasonable to inquire whether the metabolic alkalosis found in some of these patients may not be related to disturbances in intracellular electrolyte content, since evidence is presented in the following papers that disturbances of intracellular fluid do occur in pa-
tients with edema and heart failure. Darrow and co-workers have shown that in the rat under conditions of the steady state of uninterrupted renal function, the experimental production of intracellular potassium deficiency and sodium excess results in a metabolic alkalosis. In patients showing this phenomenon the renal threshold for chloride is low. Although many of these cardiacs have been shown in the balance studies to retain potassium when administered, the deficiency of the ion, if it existed, was not sufficiently great to result in a lowering of the potassium concentration in the serum and extracellular fluid, nor was the uptake of potassium always associated with the restoration to normal of the bicarbonate level. Thus, a relationship between intracellular electrolyte disturbances and the metabolic alkalosis seems unlikely, but is not entirely ruled out, since it is known that the extracellular concentration of potassium does not always mirror the state of cellular stores of the ion, and since the extracellular alkalosis may be more directly related to changes in intracellular sodium than potassium.

In view of these facts and of the frequent finding of normal or low values for carbon dioxide pressure (pCO₂) in many of these patients, the most likely explanation of the metabolic alkalosis as defined by the elevated buffer base values, is that under the influence of mercurial therapy and diuresis, chloride is excreted in greater amounts than sodium relative to their proportional contents in extracellular fluid. This phenomenon has been recorded by other workers and was observed in the patients reported in the next paper. The acid-base disturbance in any given cardiac patient would appear to be compounded of a variety of factors: respiratory stimuli (central and peripheral), pathologic changes in pulmonary structure, prior treatment with salt and diuretics, and possibly intracellular electrolyte abnormalities.

SUMMARY AND CONCLUSIONS

The serum concentrations of certain electrolytes were studied in 51 samples from 44 patients with peripheral edema due to congestive heart failure. The patients without renal insufficiency are presented in group B and all of the patients are grouped together in group C. Most of the patients had had mercurial diuretics within two weeks of the time of sampling. The results are compared to values obtained from a control series of normal subjects, group A.

The mean concentration of serum sodium was significantly lower in groups B and C. The mean concentration of serum chloride was depressed to a greater extent than was that of sodium, in both groups B and C. The mean content of carbon dioxide was elevated in group B only. The mean concentration of serum potassium was not altered significantly in either group.

In 12 studies of acid-base equilibrium in 11 patients, nine cases were found to have an elevated buffer base concentration. Six of these were cases of primary metabolic alkalosis; and in four of these six, the carbon dioxide pressure was low, indicating the coexistence of a primary respiratory alkalosis. In three of the cases with an elevated buffer base concentration this change might have been secondary to the high carbon dioxide pressure (primary carbon dioxide retention or respiratory acidosis). The remaining two cases had a normal buffer base concentration but had a low carbon dioxide pressure (primary carbon dioxide deficit or respiratory alkalosis).

It is concluded: (1) that hyponatremia and hypochloremia are common findings in edematous cardiacs who have received mercurial diuretics; (2) that the relatively greater degrees of hypochloremia and the elevated bicarbonate concentration (the metabolic alkalosis) frequently found, (a) usually are not explicable as a secondary response to a primary carbon dioxide retention, but (b) are probably due to excretion of chloride in relatively greater amounts than of sodium during mercurial therapy and diuresis.

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