Chronic Alkalosis with Damage to the Central Nervous System

Samuel Natelson

Recently an anomalous type of sodium retention in a case of encephalitis in an adult has been reported (1). This case resembles closely the type of chronic alkalosis observed in certain patients with injury to the central nervous system, previously reported by the author (2).

It is the purpose of this paper to illustrate this syndrome with a few typical examples, which came to autopsy, and to suggest a possible mechanism for the observed phenomena associated with this syndrome. For purposes of identification this syndrome will be referred to as the "alkalosis syndrome."

The most obvious characteristics of this syndrome are: 1. Damage to a particular portion of the central nervous system—apparently the hypothalamus. 2. A chronic alkaline blood pH, aggravated by administration of saline solutions, and only temporarily relieved with ammonium chloride or potassium chloride in large amounts. 3. A disproportion in the sodium to chloride ratio in the urine, in that much larger amounts of chloride are excreted than sodium. 4. When in alkalosis, large volumes of urine and other drainage fluids are obtained.

Table 1 illustrates this disproportion in sodium and chloride excretion in the urine in several selected cases. One case, where gastric contents were assayed, is also listed.

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From The Department of Biochemistry, Rockford Memorial Hospital, Rockford, Ill.
Present address: St. Vincent's Hospital, Department of Biochemistry, New York, N. Y.
The author wishes to acknowledge the invaluable aid rendered by M. O. Alexander, M.D., pathologist at Rockford Memorial Hospital, in performing the autopsies on the cases of this study.
Received for publication Aug. 10, 1957.
Table 1. Composition of Fluids Excreted by Certain Patients in Chronic Alkalosis Associated with Brain Injury Illustrating Disproportion in Sodium and Chloride Excretion

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Fluid</th>
<th>Na (mEq/L)</th>
<th>Cl (mEq/L)</th>
<th>K (mEq/L)</th>
<th>Fluid pH</th>
<th>Blood pH</th>
<th>Volume (ml/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Urine</td>
<td>6.5</td>
<td>96</td>
<td>15.2</td>
<td>5.1</td>
<td>7.70</td>
<td>2050</td>
</tr>
<tr>
<td>2</td>
<td>Urine</td>
<td>13.5</td>
<td>100</td>
<td>34.1</td>
<td>5.5</td>
<td>7.52</td>
<td>1850</td>
</tr>
<tr>
<td>3</td>
<td>Urine</td>
<td>15.0</td>
<td>140</td>
<td>28.2</td>
<td>5.3</td>
<td>7.68</td>
<td>2800</td>
</tr>
<tr>
<td>4</td>
<td>Urine</td>
<td>12.3</td>
<td>96</td>
<td>30.0</td>
<td>4.7</td>
<td>7.60</td>
<td>1750</td>
</tr>
<tr>
<td>5</td>
<td>Stomach drainage</td>
<td>18.0</td>
<td>140</td>
<td>32.0</td>
<td>1.5</td>
<td>7.70</td>
<td>125</td>
</tr>
</tbody>
</table>

In the case of Rowntree, cited above (1), no blood pH measurements, nor blood chlorides, are listed, but loss of sodium and chloride, in mEq./24 hr., in the urine, when not under treatment, were reported as 11.7 and 78.7; 2.9 and 50.4; 30.5 and 61.0; respectively. It is probable that this patient must also have had an alkaline blood pH. In the normal adult the excretion of sodium and chloride in 24 hours is approximately 1:1 when expressed in mEq./24 hr., as reported (2), and determined by the author on several hundreds of cases. The urine pH in these patients ranged from 4.7–5.5. Rowntree reports the pH of the urine to be 5.0 and 5.5 in his case.

While more than 30 such cases have been noted in the last 7 years, a few have been selected to illustrate a particular point. A typical case of brain damage resulting from cerebral thrombosis is illustrated in Table 2. The table shows the effects of administration of pitressin, which lowers urinary output and total salt output (3). The urinary Na/Cl ratio improved but did not return to normal.
Table 2. Effect of Pitressin on Blood and Plasma Levels of Pertinent Constituents in Case of Cerebrovascular Accident

Autopsy showed infarction of right thalamic and internal capsule area, and extension subcortically into occipital lobe.

<table>
<thead>
<tr>
<th>Hospital day</th>
<th>Na (mEq/L)</th>
<th>K (mEq/L)</th>
<th>Cl (mEq/L)</th>
<th>Protein (%)</th>
<th>Blood pH</th>
<th>O2 (mM/L)</th>
<th>Hematocrit (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>133</td>
<td>3.9</td>
<td>83</td>
<td>5.0</td>
<td>7.60</td>
<td>28</td>
<td>50</td>
</tr>
<tr>
<td>6</td>
<td>134</td>
<td>4.9</td>
<td>88</td>
<td>4.8</td>
<td>7.59</td>
<td></td>
<td>42</td>
</tr>
<tr>
<td>7</td>
<td>136</td>
<td>3.9</td>
<td>93</td>
<td>4.9</td>
<td>7.52</td>
<td></td>
<td>43</td>
</tr>
<tr>
<td>8*</td>
<td>140</td>
<td>3.6</td>
<td>80</td>
<td>5.7</td>
<td>7.70</td>
<td>22</td>
<td>47</td>
</tr>
<tr>
<td>10*</td>
<td>139</td>
<td>4.2</td>
<td>105</td>
<td>5.1</td>
<td>7.36</td>
<td>22</td>
<td>42</td>
</tr>
</tbody>
</table>

*Before Pitressin.
*After Pitressin.

Clinical abstract (Table 2): This 67-year-old female was admitted in coma with a flaccid left hemiplegia. Since kidney function was normal (e.g., urea, N 14 mg. per 100 ml.; urine, neg.; sp. gr., 1.025) and the patient was not diabetic, she was chosen for study, when it was found that urinary output exceeded total fluid intake with a chronic alkaline blood pH. An example of the urine composition is shown in Table 1 for the seventh day. On the eighth and ninth days, 1 ml. Pitressin tannate (Parke-Davis Company, 5 units/ml.) was administered intramuscularly. After Pitressin, urinary electrolytes were Na 30.6 mEq./L., K 19.3 mEq./L., Cl 65 mEq./L. Urine volume dropped to 1200 ml. on a 2000-ml. intake containing Ca gluconate, KCl and 10 Gm. NaCl. Plasma chloride rose and pH became normal. Death occurred on the eleventh day. Autopsy showed a thrombosis of the lenticulostriate branch of the right middle cerebral artery with a fairly recent infarct of the right thalamic and internal capsule area and extension subcortically into the occipital lobe. (Case of John O. Heald, M.D.)

Table 3 illustrates the blood levels of pertinent chemical constituents of the plasma of an infant admitted with left hemiparesis. This case was of interest, since the levels were normal on admission. The infant later developed the high salt syndrome (4), followed by a period of chronic alkalosis.

The case of Table 4 was unique because of the apparent paradox of uremia without acidosis. Diagnosis was lower nephron nephrosis following surgical shock. Autopsy confirmed the diagnosis, but also revealed multiple acute abscesses in the brain, including the hypothalamic area.

In Table 5 the effect of Diamox (acetazolamide) is demonstrated in a case of cerebrovascular accident. The effect of Diamox is to inhibit carbonic anhydrase. Thus, a greater concentration of bicarbonate ion is available for increased excretion of sodium (5). This is illustrated in the table. In the case of Rowntree (1), the same phenomenon was observed. Note that the sodium retention is not always obvious in the Na/C1 ratio in the serum. This may indicate movement of Na into tissue cells and bones (6).
Table 3. Blood and Plasma Levels in Infant with Hemiparesis, Chronic Alkalosis, and Congenital Heart Disease. Autopsy showed Cerebral Infarct

<table>
<thead>
<tr>
<th>Blood &amp; Plasma Levels</th>
<th>Hemiparesis with Alkalosis</th>
<th>Congenital Heart Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pH</td>
<td>7.17</td>
<td>7.50</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>16.4</td>
<td>16.0</td>
</tr>
<tr>
<td>Urea N</td>
<td>6.3</td>
<td>4.8</td>
</tr>
<tr>
<td>Protein</td>
<td>189</td>
<td>128</td>
</tr>
<tr>
<td>Na (mEq./L)</td>
<td>5.3</td>
<td>5.1</td>
</tr>
<tr>
<td>K (mEq./L)</td>
<td>100</td>
<td>80</td>
</tr>
<tr>
<td>Cl (mEq./L)</td>
<td>11</td>
<td>20</td>
</tr>
<tr>
<td>CO2 (mM/L)</td>
<td>7.72</td>
<td>7.56</td>
</tr>
<tr>
<td>Hct.</td>
<td>51</td>
<td>41</td>
</tr>
</tbody>
</table>

*After NH4Cl (0.5 Gm.), and 450 ml. 0.85% NaCl.

Clinical abstract (Table 3): This 9-month-old, 16-lb. infant, known to have a cyanotic type of congenital heart disease, was admitted because of left hemiparesis. On oral feedings the infant's condition remained the same, until the thirteenth day when it was dehydrated clinically and in acidosis as evidenced by the pH and CO2 levels, but not with imbalance in sodium and chloride levels. With fluids administered intravenously, and some blood, the infant became well hydrated by the following day, but a sharp rise in serum Na and Cl levels (checked twice that day) was noted. The clinical condition of the infant was much improved, however. Fluid administration for the next 24 hours without NaCl resulted in alkalosis. This could be corrected only temporarily with NH4Cl and NaCl. On subsequent days, in addition to the fluids containing Ca gluconate, KCl, plasma, protein hydrolysate, vitamins, invert sugar, and NaCl, 0.2-1.5 Gm. of NH4Cl were added daily. Alkalosis persisted until the twenty-fourth day when just prior to death a respiratory acidosis was observed. Autopsy showed the heart anomaly to consist of a patent foramen ovale and complete atresia of the tricuspid opening. High in the interventricular septal wall there was an oval defect 7 × 5 mm. The right cerebral hemisphere, from the basal nuclei out, was soft and necrotic in appearance. There was no line of demarcation between the white and gray matter. Near the longitudinal fissure there was fresh subarachnoid hemorrhage. Beneath the area of subarachnoid hemorrhage in the superior occipital parietal region there was also brain hemorrhage extending 2 cm. into the substance of the brain. (Case of Homer F. Weir, M.D.)

When in alkalosis, these patients show a tendency to increased secretion and excretion of fluids. This is illustrated in Fig. 1, where an attempt was made to meet the rising output, both in total volume and electrolytes, with a corresponding increase in intake. Only when the pH was adjusted to normal with ammonium chloride was balance achieved.

Referring to Fig. 1 for the seventeenth day, before NH4Cl administration, more chloride (533 mEq.) was excreted than sodium (514 mEq.) in all fluids, at a time when the blood pH was 7.65, chloride was 65 mEq./L and sodium level was essentially normal. The only form in which sodium and chloride was administered, in this case, was in the form of saline.
Table 4. Blood and Plasma Levels in Case of Lower-Nephron Nephrosis Syndrome Following Surgical Shock

Multiple Acute Abscesses in Brain Including Basal Nuclear Area Were Demonstrated at Autopsy

<table>
<thead>
<tr>
<th>Post-operative day</th>
<th>Urea N (mg/100 ml.)</th>
<th>Na (mEq/L.)</th>
<th>K (mEq/L.)</th>
<th>Cl (mEq/L.)</th>
<th>Protein (Gm./100 ml.)</th>
<th>O2 (mM/L.)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>...</td>
<td>122</td>
<td>3.6</td>
<td>84</td>
<td>6.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>43</td>
<td>133</td>
<td>4.5</td>
<td>90</td>
<td>5.6</td>
<td>20</td>
<td>7.45</td>
</tr>
<tr>
<td>8</td>
<td>80</td>
<td>142</td>
<td>4.5</td>
<td>84</td>
<td>5.8</td>
<td>27.1</td>
<td>7.52</td>
</tr>
<tr>
<td>9</td>
<td>80</td>
<td>142</td>
<td>4.5</td>
<td>84</td>
<td>5.5</td>
<td>24.5</td>
<td>7.38</td>
</tr>
<tr>
<td>10</td>
<td>70</td>
<td>128</td>
<td>4.4</td>
<td>83</td>
<td>5.8</td>
<td>19.1</td>
<td>7.38</td>
</tr>
<tr>
<td>11</td>
<td>86</td>
<td>141</td>
<td>5.5</td>
<td>81</td>
<td>5.3</td>
<td>21.6</td>
<td>7.48</td>
</tr>
<tr>
<td>12</td>
<td>140</td>
<td>139</td>
<td>6.2</td>
<td>81</td>
<td>5.5</td>
<td>20.4</td>
<td>7.48</td>
</tr>
<tr>
<td>14</td>
<td>137</td>
<td>130</td>
<td>6.7</td>
<td>79</td>
<td>4.9</td>
<td>18.0</td>
<td>7.38</td>
</tr>
</tbody>
</table>

Clinical abstract (Table 4) (see case 1, Table 1): Cholecystectomy was performed on this white male, age 60. Preoperatively, no evidence of kidney dysfunction was evident. Urine analysis was negative and serum urea N was 12 mg./100 ml. On the sixth postoperative day a sudden drop in urine output was noted. Urine output was 300 ml. on the sixth day, 200 ml. on the seventh, and none on the eighth. Combined output from Wangensteen drainage and a T tube connected for draining the common duct was 4770 ml. on the seventh postoperative day, with a total intravenous intake of 3450 ml. (200 mEq. NaCl, 60 mEq. KCl). Because of the alkaline blood pH (7.62) on the eighth day, NH₄Cl (120 mEq.) was administered with the fluids. Increasing amounts of urine and correspondingly decreasing amounts of drainage fluids were obtained so that by the eleventh postoperative day, urinary output increased to 2100 ml., and drainage fluid was nil. In spite of the fact that urine volume was large, 2700 on the fourteenth day, 2900 on the fifteenth and 2500 on the sixteenth, urea levels remained high. Fluids administered, did not contain sodium lactate or bicarbonate, but blood pH remained normal or slightly alkaline. The patient went into acidosis on the seventeenth day and died. Autopsy diagnoses included septicaemia, with multiple acute abscesses in the brain and kidneys, and microscopic mycotic pulmonary lesions (aspergillus). The brain abscesses were of random distribution in cortical, subcortical, and basal nuclear areas, including the thalamus. (Case of M. P. Rogers, M.D.)

Table 5. Effect of Carbonic Anhydrase Inhibitor on Blood pH and Sodium to Chloride Ratio in Urine in Patient with Brain Damage

<table>
<thead>
<tr>
<th>Urine</th>
<th>Serum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na (mEq/L.)</td>
<td>K (mEq/L.)</td>
</tr>
<tr>
<td>-------</td>
<td>-------</td>
</tr>
<tr>
<td>Before Diamox</td>
<td>12</td>
</tr>
<tr>
<td>After Diamox</td>
<td>64</td>
</tr>
</tbody>
</table>

Clinical abstract (Table 5): This 68-year-old male was admitted with left hemiplegia. Consultants' diagnosis was cerebral thrombosis with left hemiplegia. The patient was comatose during the period of the experiment, 6 days after admission. 2500 ml. of fluids containing 6 Gm. of NaCl were given by stomach tube. At the end of 24 hours, urine (1600 ml.) and blood were analyzed. 250 mg. of Diamox were given by stomach tube and the same fluids were administered. The urine (1400 ml.) and the blood were again analyzed. Results are shown in Table 5. It is of interest to note that for the next few days the blood pH climbed slowly and reached 7.57 at 84 hours. Urine Na/Cl ratio dropped steadily and was 11/38 at 84 hours. (Case of E. J. McKinney, M.D.)
As another example, a 22-year-old male, weighing 170 pounds, was being treated, in another hospital, for severe burns suffered in an explosion. On the forty-eighth day of treatment, the patient was in alkalosis (pH 7.68), with tetany, although the serum calcium level was at 10 mg./100 ml. Oral and intravenous fluids at the time had been raised to 8.7 liters, in an attempt to meet a rising fluid output. The patient received no alkaline fluids, yet showed a chronic alkalosis. Death was attributed to severe pneumonia and generalized septicemia. Autopsy findings included edema of the brain with generalized hyperemia, and diffuse hyperemia of the hypothalamus and the pituitary. The adrenals showed considerable degree of hyperplasia of all zones of the cortex with focal areas of hemorrhage.

In this case and that of Fig. 1, a vicious cycle was set up with saline administration. The more alkaline the patient’s blood became, the

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**Fig. 1.** Effect of blood pH adjustment on fluid output in a child weighing 12 kg. Block diagram shows composition of fluid output for the seventeenth day before NH₄Cl administration. Lower chart shows changing blood electrolyte levels. Clinical abstract: Following surgery for an inflamed, perforated appendix this 4-year-old female became distended and was unable to retain oral feedings. Wangensteen suction did not reduce the distention and an ileostomy was performed on the eleventh postoperative day. Feeding by intravenous route was regulated in accordance with the analysis of the total output as to total volume, and electrolyte (Na, K, Cl) and nitrogen content. Blood was administered from time to time to maintain the erythrocyte count. Although the child was disoriented from the eleventh to the nineteenth day, no obvious clinical signs could be elicited to indicate brain damage to a particular area. In spite of increased fluid intake, a rising voluminous output could not be matched. On the seventeenth day, when the patient's condition was grave, Ca gluconate and 10 Gm. of NH₄Cl were added to the fluids, and 2 Gm. of NH₄Cl given on the eighteenth day. A prompt decrease in fluid output was observed. Ileostomy output was 250 ml. on the eighteenth day, less than 25 ml. on the nineteenth day, and by the twenty-first day the ileostomy had sealed itself. The patient's condition was sufficiently improved so that she could be returned to surgery where adhesions, which had caused the ileum to be kinked in several places, could be divided. Recovery was uneventful. (Case of T. F. Krause, M.D.)
greater the fluid output. The greater the output of fluids, the more salt was administered. However, chloride loss with respect to sodium was always greater. Thus, continued saline administration aggravated the alkalosis.

Figure 2 illustrates the practical value of the knowledge that fluid output may be controlled in this type of patient by adjusting the pH of the blood. Change of total fluid output could be repeatedly demonstrated in this patient, with change in pH.

An interesting case was that of an infant, born with Rh incompatibility with the mother (the patient of W. L. Crawford). Exchange transfusion was delayed for 48 hours, at which time the bilirubin level rose to 53 mg./100 ml. Twenty-four hours after exchange transfusion, alkalosis (pH 7.65; Na 129; Cl 82 mEq./l.) was at first attributed to the sodium citrate administered in the blood. Saline and calcium gluconate brought temporary relief from tetany and lowered the pH to 7.52 but 24 hours later when alkalosis was again severe, ammonium chloride was added to the fluids for 3 consecutive days. Each morning the infant was again in alkalosis. On the fourth day the pH remained at 7.38, and henceforth could be maintained with hypotonic saline solutions. For 30 days it was suspected, from neurologic examination, that the infant had suffered permanent brain damage. At 1 year of age the infant was normal. Because of the common find-

![Fig. 2. Illustration of fluid output control by pH adjustment with NH₄Cl. Clinical abstract: Cerebrovascular accident occurred in this 88-year-old female weighing 49 kg., while in a standing position. In falling she experienced a fracture of the left inferior ramus of the pubis. On admission, she had lost her swallowing reflex and was tube fed for 36 days and then on intravenous feedings because she had become distended unable to tolerate tube feedings. From the thirty-sixth to sixty-third day after admission she was given 8 Gm. of NaCl daily and KCl, in accordance with her potassium loss. Serum K levels ranged from 4.5-5 mEq./L. 50 Gm. of protein hydrolysate and 100 Gm. of glucose were given daily. pH was controlled by repeated administration of NH₄Cl as indicated. Patient was comatose from the thirty-sixth to sixtieth day. At 60 days the patient was alert and could take oral feedings. Discharge, in a wheel chair, occurred on the seventy-sixth day. Two years later the patient was apparently still well, ambulatory, and active. (Case of John O. Heald, M.D.)](image-url)
ing of kernicterus in such infants at autopsy (7), it is suggested that the chronic alkalosis was a result of insult to the brain.

Rowntree reports edema with the sodium retention (1), and says, "It is of interest to note that, when the patient tolerated the headache and puffiness, that were symptoms of salt and water retention, and did not resort to the use of a diuretic, spontaneous diuresis occurred." In the light of our observations this might be interpreted that when the patient had gone into a moderately severe alkalosis, the diuresis occurred.

Cognizant of the fact that low serum potassium levels and tissue cell depletion of potassium have been often observed associated with alkalosis (8), apparently adequate amounts of potassium chloride were used in the fluids administered as indicated by serum potassium levels (see Tables 2-5). The volume of fluids and the amount of K, Na, and Cl to be administered were determined from the measured losses. Additional amounts of these ions were given if serum levels were low. These were calculated assuming the extracellular fluid volume to be 20 per cent of body weight. The possibility, however, that intracellular K deficiency persisted is not eliminated.

If the alkalosis were treated with large amounts of KCl (80–200 mEq./24 hours), then the alkalosis and the hypokalemia were both corrected and the fluid output decreased (9). This was mainly due, as determined by analysis, to the loss of potassium into the intestinal drainage fluids and the urine, and retention of the chloride. The same effect could be obtained with smaller amounts of KCl (30–60 mEq./24 hours) and larger amounts of NH₄Cl (80–200 mEq./24 hours). In other words, both KCl and NH₄Cl solutions are acidifying solutions. Potassium is readily excreted in the urine, leaving the chloride behind, and the ammonia is converted to urea to produce the same effect. Caution must be exercised in the use of both substances in that potassium intoxication may result if oliguria should develop and ammonium ion should not be allowed to exceed its toxic level, as occurs in certain liver conditions.

In these studies, pH determination was done with the glass electrode, on serum from clotted blood, as previously described (10). Sodium and potassium determination were done with the flame photometer (11). Chloride was analyzed by mercurimetric titration (12). Protein was estimated by the biuret method (13). Carbon dioxide content was done with the microgasometer (14). The diacetyl method was used for urea estimation (15).
DISCUSSION

In the "alkalosis syndrome" (Tables 1–5) the problem appears to be an inability to excrete sodium ions, particularly in the urine. The condition is different from primary hyperaldosteronism (16), in that adrenal tumors were not observed at autopsy. Stimulation or inhibition of adrenal secretions, however, must play some part in this condition. Chronic severe alkalosis is not uniformly observed in Addison's disease, nor when more than the minimal requirement of aldosterone was administered to patients with Addison's disease (17). Conflicting observations on the electrolyte effects observed in different cases of hyperaldosteronism may possibly be the result of central nervous system changes in some cases, and not in others. The antidiuretic hormone will also not correct the disparity between sodium and chloride excretion in the urine (Table 2).

Alkalosis, resistant to treatment with NaCl, has been observed in certain patients with Cushing's disease (18, 19, 20). Willson (19) points out that in Cushing's disease, "Demonstrable abnormalities of electrolytes occur only exceptionally." (Three of 30 cases showed chronic alkalosis.) It is fair to inquire whether these patients, also, suffered impairment of some particular portion of the brain, as in the cases cited in this paper.

Diamox, a carbonic anhydrase inhibitor, is very effective in the treatment of the condition described, in that it: (1) Corrects the imbalance in urine sodium to chloride excretion, (2) lowers the total CO₂ level, (3) adjusts the blood pH level, and (4) lowers the total volume of output. However, when Diamox is used, care must be taken not to deplete the patient of potassium.

Point 4 is of interest because it is known that Diamox, in large quantities, will tend to cause a diuresis in certain patients with edema (21). In our cases, urine volume did not always increase, and in many cases decreased on small doses of Diamox. The patients we were dealing with were, in general, somewhat dehydrated from excessive loss of fluids, and had elevated total CO₂ levels associated with the alkalosis. It is of interest to note that 250 mg. of Diamox given orally will continue to show its effect in tending to maintain normal blood pH levels in these patients for 2–3 days. A 25–50 mg. dose daily is adequate to maintain the adult patient in this respect.

In any scheme designed to explain acid base and electrolyte balance, in the human, the brain must be included. Brain damage may result in changes of sodium and chloride levels, as in the case of the
high salt syndrome (4), and as pointed out here, may result in alkalo-
sis. It is more than likely that the brain is the center of a servo-
mechanism designed through its action on the lungs, kidneys, and
cell metabolism through the target glands, to maintain the human at
constant pH and constant salt levels.

Regardless of the true nature of the mechanism causing the phe-
nomena discussed above, it must be stressed that a chronic alkalo-
sis, with abnormal sodium to chloride ratios in the urine, should alert the
physician to the possibility of brain damage. Conversely, where brain
damage is apparent, the physician should be alert to the possibility
of electrolyte and pH dyscrasias, and be prepared to correct them.

**SUMMARY**

A condition of chronic alkalo-sis has been described with central
nervous system damage. This syndrome is characterized by alkaline
blood pH, and elevated total CO2 levels, associated with central ner-
vous system damage. It is apparently caused by sodium retention as
indicated from the sodium to chloride ratios in the urine excreted by
these patients. In alkalo-sis, the output of fluid is often greater than
the input. Sodium chloride solutions intravenously tend to aggra-
vate the alkalo-sis. Potassium chloride or ammonium chloride will re-
lieve the alkalo-sis only temporarily. Pitressin will not correct the ab-
normal sodium to chloride ratios in the urine. A carbonic anhydrase
inhibitor (Diamox) will lower the CO2 levels, and correct the blood
pH. Its action seems to be due to its ability to correct the sodium
to chloride ratios in the urine excreted. The effect of Diamox will
persist for several days after its administration.

*Rockford Memorial Hospital*
*2400 No. Rockton Ave.*
*Rockford, Ill.*

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