Alterations in Exchangeable Sodium Content, "Sodium$^{24}$ Space" and Body Weight During The Treatment of Congestive Failure

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Serial determinations of the exchangeable sodium content and sodium$^{24}$ (Na$^{24}$) space were made by the radioisotope dilution method in patients with congestive failure during the course of intensive therapy. In most instances, the decrease in body weight was too great to be accounted for solely on the basis of the change in sodium$^{24}$ space or exchangeable sodium content. This excess weight loss is attributed to loss of intracellular fluid.

INCREASE in body weight, which occurs during congestive heart failure, was generally assumed to be due to the retention of sodium and water in the extracellular fluid compartment. However, some recent studies have suggested that changes also occur in the intracellular phase.$^{1,2}$ The external balance method, which has been used in most previous studies of this problem does not permit the direct measurement in vivo of the sodium content of the body. This difficulty can be overcome by the use of the isotope dilution technic, and one group of investigators$^3$ has previously reported such an application of this technic.

The purpose of the present study was to determine, by the use of the in vivo isotope dilution technic, whether or not the weight loss which occurs during diuresis can be accounted for solely on the basis of a decrease in the extracellular content of sodium and water. Serial determinations of the exchangeable sodium content were made and the changes in this value were correlated with the alterations in body weight. Observations were also made on the effects of intensive therapy with mercurial diuretics on sodium and potassium metabolism.

Material and Methods

Subjects. Thirteen patients, 9 men and 4 women, with advanced congestive failure were studied. Their ages ranged from 53 to 77 years. In five subjects, the cause of the congestion was pulmonary fibrosis and emphysema with cor pulmonale; in six subjects, coronary arteriosclerotic heart disease; and in two, hypertensive cardiovascular disease. Most of these individuals had previously been followed in the Outpatient Clinic and had received maintenance doses of digitalis, ammonium chloride and aminophylline, and periodic injections of a mercurial diuretic. Only one patient (case 2) had not previously been treated with digitalis or a mercurial diuretic. When admitted to the hospital, all were edematous.

The general plan was to make serial determinations of the exchangeable sodium content, the radiosodium space, the serum concentrations of sodium and potassium, and the body weights during hospitalization.

All patients received a salt-free or low-salt (200 to 400 mg.) diet; water was given without restriction. Except in one case, mercaptomerin sodium (Thiomerin) was administered subcutaneously or intramuscularly whenever mercurial diuretics were indicated. A summary of the clinical data and the type of therapy employed is given in table I.

Isotopes. Isotopic sodium (Na$^{24}$)* was prepared for injection in the manner previously described.$^4$

Measurement of Radioactivity. The activity of the urine and serum specimens was determined with a well-type scintillation counter and a scaling circuit.

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Dr. Aikawa is an Established Investigator of the American Heart Association.
A total of 10,000 counts were made on each sample. All determinations were corrected for decay.

**Determination of Serum Sodium and Potassium.** The total sodium and potassium concentrations in the serum were determined with a Baird flame photometer, using the lithium internal standard method.

**Procedure**

**Determination of Exchangeable Sodium Content (Na-e).** Each subject received from a calibrated syringe 1.5 μ of Na24 per Kg. of body weight, contained in sterile 0.9 per cent sodium chloride solution. All urine voided for the next 24 hours was collected, and the Na24 content of the pooled specimen was determined. A blood specimen was obtained 24 hours after the injection of Na24 and the specific activity of the sodium in the serum was determined.

**Calculations.** The following formula was used to calculate the value for the exchangeable sodium content of the body:

\[
Na-e = \frac{Na-i^{24} - Na-u^{24}}{Na-s^{24}/Na-s^{23}}
\]

- \(Na-e\) = quantity of exchangeable sodium in milliequivalents (mEq.).
- \(Na-i^{24}\) = quantity of radiosodium administered.
- \(Na-u^{24}\) = quantity of radiosodium excreted in the pooled specimen of urine.
- \(Na-s^{24}\) = concentration of radiosodium in the serum at 24 hours.
- \(Na-s^{23}\) = concentration of nonradioactive sodium in the serum at 24 hours.
- \(Na-s^{23}/Na-s^{24}\) = specific activity of the serum at 24 hours.

Preliminary studies in this laboratory revealed that the Na-e measurement was reproducible within five per cent in edema-free, hospitalized subjects with various chronic diseases whose condition was stabilized. This finding agrees with those previously reported by Miller and Wilson.1

**Sodium24 Space.** The volume of dilution of the injected Na24 was calculated as follows:

\[
Na^{24} \text{ space in liters} = \frac{\text{total sodium}^{24} \text{ activity injected}}{\text{serum Na}^{24} \text{ concentration per liter at 24 hours}}
\]

A total of 35 determinations of exchangeable sodium content and radiosodium space were made on 13 subjects, usually at weekly, but occasionally at biweekly intervals.

**Results (table 1 and 2)**

**Weight Changes.** All 13 subjects lost weight during the period of observation. The maximum decrease in body weight (25 per cent of the original value) occurred in the subject (case 2) with the shortest duration of acute congestive heart failure (one week). This subject, on intensive mercurial therapy, rapidly lost 14.1 Kg., in spite of which the serum sodium concentration remained normal and unchanged. Transient small increases in weight (1 and 3 per cent) occurred in two subjects (cases 3 and 4), and were followed by subsequent decreases in weight. One of these subjects (case 4) died in congestive heart failure four days after completion of the third determination of exchangeable sodium content (Na-e).

**Exchangeable Sodium Content.** The mean exchangeable sodium content in older adult male subjects with congestive heart failure has been previously reported to be 56.7 ± 1.9 mEq. per kilogram.3 In the present series, the mean of the initial values was 65.3 ± 3.2 mEq. per kilogram. In only one instance (case 13) was the original value for Na-e/Kg. within the normal range (40.6 ± 1.5 mEq. per kilogram).4 This subject was a markedly obese woman who had clinical evidences of congestive heart failure and in whom the Na-e value subsequently decreased during therapy.

In 11 of the 13 patients, the final values for exchangeable sodium content per kilogram were above the range previously reported (3) in individuals who had cardiovascular disease without congestive heart failure (42.8 ± 2.4 mEq. per kilogram.)

In all instances except one (case 4), the absolute values for Na-e decreased during the period of observation. This decrease was greater than 30 per cent of the original value in five subjects (cases 2, 3, 8, 9 and 11); in absolute values the decrease ranged from 1310 to 2132 mEq. The patient who failed to show any decrease was a man who did not respond to intensive treatment and who died four days after the last determination (case 4).

In figure 1 the change in weight has been plotted against the change in exchangeable sodium content and serum sodium concentration. If 1 Kg. of weight loss is assumed to represent 1 L. of extracellular fluid water, then each kilogram of weight loss should be accompanied by a loss of sodium equivalent to its concentration in the serum at that instant. This nor-
<table>
<thead>
<tr>
<th>Case</th>
<th>Age (Yrs)</th>
<th>Sex</th>
<th>Cause of Congestion</th>
<th>Pertinent Clinical Data</th>
<th>Therapy*</th>
<th>Days of Hospitalization</th>
<th>Weight (kg)</th>
<th>Exchangeable Sodium Content</th>
<th>Radiosodium Space</th>
<th>Serum Concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1, 4, 7 (2 ml. q day x 7)</td>
<td>5 12</td>
<td>53.6 46.8</td>
<td>4282 3478</td>
<td>80.5 74.3</td>
<td>31.3 24.3</td>
</tr>
<tr>
<td>1</td>
<td>53</td>
<td>M</td>
<td>ASHD</td>
<td>Chr. congest.; readmitted 2 wks. after previous discharge. Disoriented between 5th and 12th hospital days.</td>
<td>1, 3, 4, 7 (2 ml. q o.d. x 19)</td>
<td>13 34 45</td>
<td>68.2 63.6 59.5</td>
<td>5236 5462 2376</td>
<td>76.9 85.9 55.0</td>
<td>38.7 40.9 24.7</td>
</tr>
<tr>
<td>2</td>
<td>72</td>
<td>M</td>
<td>ASHD</td>
<td>One prev. episode 2 yrs. ago. Duration present bout, 1 wk.</td>
<td>1, 6, 3, 7 (2 ml. x 2)</td>
<td>2 9 15 16 20</td>
<td>67.8 45.0 70.9 60.5</td>
<td>4897 430 7614</td>
<td>72.2 68.3 35.1 50.4</td>
<td>52.9 50.4 54.7 50.4</td>
</tr>
<tr>
<td>3</td>
<td>77</td>
<td>M</td>
<td>ASHD</td>
<td>Chr. congest., initially refractory to therapy.</td>
<td>1, 3, 4, 7 (2 ml. q o.d. x 3 in 4 days)</td>
<td>8</td>
<td>73.5</td>
<td>5923</td>
<td>80.6</td>
<td>45.6</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>M</td>
<td>Cor P.</td>
<td>Chr. edema; died on 19th hospital day.</td>
<td>1, 3, 4, 7 (2 ml. q o.d. x 1)</td>
<td>13</td>
<td>68.2</td>
<td>5246</td>
<td>76.9</td>
<td>38.7</td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>F</td>
<td>Cor P.</td>
<td>Congest. for 4 yrs. Severe dyspnea 2 wks. Obese</td>
<td>1, 6, 4, 7 (2 ml. x 2)</td>
<td>2</td>
<td>67.8</td>
<td>4897</td>
<td>72.2</td>
<td>52.9</td>
</tr>
<tr>
<td>6</td>
<td>62</td>
<td>M</td>
<td>Cor P.</td>
<td>Congest. for 3 mos.</td>
<td>1, 6, 3, 7 (2 ml. q o.d. x 9)</td>
<td>8 16</td>
<td>56.4 51.4</td>
<td>3591 3220</td>
<td>63.7 62.7</td>
<td>27.0 23.0</td>
</tr>
<tr>
<td>7</td>
<td>54</td>
<td>M</td>
<td>Cor P.</td>
<td>Congest. for 10 yrs. Parox. dyspnea for 10 yrs.</td>
<td>1, 3, 4, 7 (2 ml. x 1)</td>
<td>1 8</td>
<td>45.5 42.3</td>
<td>2887 2230</td>
<td>63.5 52.7</td>
<td>21.2 18.1</td>
</tr>
<tr>
<td>8</td>
<td>68</td>
<td>M</td>
<td>ASHD</td>
<td>Periph. edema, 1 yr. Dyspnea, 2 wks.</td>
<td>1, 3, 4, 8, 7 (2 ml. x 1)</td>
<td>2 9</td>
<td>77.3 68.6</td>
<td>4893 3344</td>
<td>63.3 48.7</td>
<td>37.0 22.5</td>
</tr>
<tr>
<td>9</td>
<td>60</td>
<td>M</td>
<td>HCVD</td>
<td>Congest. 4 yrs. Massive periph. edema, 6 wks.</td>
<td>1, 3, 4, 7 (1 ml. q o.d. x 11)</td>
<td>7 14 21</td>
<td>68.2 60.5 55.0</td>
<td>4314 3497 2680</td>
<td>63.3 57.8 48.7</td>
<td>30.2 25.8</td>
</tr>
<tr>
<td>10</td>
<td>75</td>
<td>F</td>
<td>ASHD</td>
<td>Increasing dyspnea and leg edema for 2 mos.</td>
<td>1, 4, 6, 7 (2 ml. x 3)</td>
<td>16 24 28</td>
<td>65.2 60.3 52.7</td>
<td>4117 3713 2387</td>
<td>63.1 61.6 45.2</td>
<td>31.1 27.1</td>
</tr>
<tr>
<td>11</td>
<td>63</td>
<td>F</td>
<td>HCVD</td>
<td>Massive edema for 3 wks.</td>
<td>1, 4, 7 (2 ml. x 3)</td>
<td>4 12</td>
<td>73.5 61.8</td>
<td>4245 2835</td>
<td>57.8 47.5</td>
<td>32.4 23.1</td>
</tr>
<tr>
<td>12</td>
<td>74</td>
<td>M</td>
<td>Cor P.</td>
<td>Congest. continually for 1 yr.</td>
<td>1, 7 (1 ml. q o.d. x 7)</td>
<td>7 22</td>
<td>60.9 57.3</td>
<td>3100 2946</td>
<td>50.9 51.4</td>
<td>23.3 20.8</td>
</tr>
<tr>
<td>13</td>
<td>77</td>
<td>F</td>
<td>ASHD</td>
<td>Congest. 4 mos. Marked obesity.</td>
<td>1, 7 (1 ml.)</td>
<td>3 11</td>
<td>83.6 77.7</td>
<td>3328 2651</td>
<td>39.8 34.1</td>
<td>25.2 19.4</td>
</tr>
</tbody>
</table>

*1 = Digitalis in maintenance doses.
2 = Rapid digitalization while hospitalized.
3 = Aminophylline suppositories.
4 = Ammonium chloride.
5 = Potassium chloride.
6 = Diamox.
7 = Dosage of mercurial diuretic (Thiomerin).
Exchangeable sodium content, "sodium space" and body weight

TABLE 2—Changes in Exchangeable Sodium Content, Radiosodium Space and Body Weight During Intensive Therapy of Congestive Heart Failure

<table>
<thead>
<tr>
<th>Case</th>
<th>Day of Hospitalization</th>
<th>Change in Weight* (kg.)</th>
<th>Change in Exchangeable Na* (mEq.)</th>
<th>Change in Radiosodium Space* (L.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>-13</td>
<td>-6.8</td>
<td>-19</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>+3</td>
<td>+2.0</td>
<td>-2</td>
</tr>
<tr>
<td>4</td>
<td>34</td>
<td>-7</td>
<td>-4.6</td>
<td>+4</td>
</tr>
<tr>
<td>5</td>
<td>45</td>
<td>-13</td>
<td>-8.7</td>
<td>-38</td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>+1</td>
<td>+0.5</td>
<td>+7</td>
</tr>
<tr>
<td>7</td>
<td>15</td>
<td>-3</td>
<td>-2.1</td>
<td>+1</td>
</tr>
<tr>
<td>8</td>
<td>9</td>
<td>-11</td>
<td>-7.3</td>
<td>-16</td>
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<td>-5.0</td>
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<tr>
<td>10</td>
<td>16</td>
<td>-7</td>
<td>-3.2</td>
<td>-23</td>
</tr>
<tr>
<td>12</td>
<td>22</td>
<td>-22</td>
<td>-17.3</td>
<td>-25</td>
</tr>
<tr>
<td>13</td>
<td>11</td>
<td>-11</td>
<td>-7.7</td>
<td>-19</td>
</tr>
</tbody>
</table>

* Compared with initial determination.

The normal relationship is indicated by the diagonal line. It is apparent that in 17 of 22 instances the weight changes were greater than could be accounted for solely on the basis of the sodium changes, whereas, in five instances, the decrease in body weight was less than could be accounted for solely on the basis of a change in exchangeable sodium.

Na24 space. Warner and his co-workers have previously reported that the mean sodium24 space in elderly adults with decompensated cardiovascular disease was 41.7 per cent of the body weight. The mean of the initial values obtained in the present series (48.6 ± 2.4 per cent) was slightly higher than that in Warner's series. In all 13 subjects the sodium24 space decreased by at least 10 per cent of the original value. The maximum decrease (49 per cent) was observed in case 2.

In most instances the value for Na24 space per kilogram tended to decrease during therapy of congestive failure. In 9 of the 13 subjects, the changes in weight were too great to be accounted for solely on the basis of a change in Na24 space. This relationship parallels that observed with the exchangeable sodium content.

Serum Sodium Concentration. The initial serum sodium levels were below 135 mEq. per liter in seven subjects. In all of these individuals, subsequent values obtained during therapy of the congestive heart failure were higher. In case 11, the initial value of 131.2 mEq. per liter dropped to 127 mEq. per liter and then rose to 139 mEq. per liter; satisfactory diuresis was obtained at the time the serum sodium concentration was lowest.

Initially normal values decreased to 129.1 mEq. per liter and 123.0 mEq. per liter, respectively, in two subjects (cases 7 and 9) during diuresis; no abnormal clinical symptoms or signs appeared at this time. There was no correlation between the presence of a serum sodium concentration below 135 mEq. per liter and the development of the symptoms and signs of the "low salt syndrome," such as lethargy, weakness, and somnolence.

The changes in serum sodium concentration...
were unpredictable and could not be correlated with the amount of mercurial diuretic which was given.

**Serum Potassium Concentration.** Serum potassium concentrations as low as 3.3 and 2.7 mEq. per liter were observed on only two occasions (cases 1 and 3). The value of 3.3 mEq. per liter was observed in case 3 at a time when the patient was responding poorly to treatment and was lethargic and somnolent. A dramatic clinical improvement occurred before the second determination, when the serum potassium concentration had risen to 5.1 mEq. per liter. Diuresis, however, did not occur for another week. The value of 2.7 mEq. per liter was obtained in case 1 during intensive mercurial therapy. This patient became disoriented and somnolent; potassium given orally in small amounts produced dramatic improvement, and he again became alert.

**Comment**

The exchangeable sodium content is a measure of the sodium in the body which is available for exchange with the radioisotopic atom and reflects a functional component of the body rather than an anatomic unit. Although there is some question as to whether this determination measures the total body content of sodium and whether the injected radioactive isotope of sodium ever reaches complete equilibrium with all the native sodium in the body, the values obtained are reproducible and the method is practical for serially following changes in sodium metabolism in the living subject.

The initial values for exchangeable sodium content per kilogram in the present series were higher than those previously reported,

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The initial values for exchangeable sodium content per kilogram in the present series were higher than those previously reported, this suggests that the subjects in the present study were retaining more sodium. The results indicate that patients with exchangeable sodium contents twice as high as the usual value found in chronically ill, edema-free patients may have normal serum sodium concentrations. Indeed, in several instances, the serum sodium concentration was abnormally low at a time when the total body content of sodium was markedly increased. Clinically, most of the subjects did not attain an edema-free state during the period of observation, and this fact is reflected by the final values for exchangeable sodium content per kilogram, which were higher than those reported in edema-free subjects.

A decrease in body weight which exceeds the relative decrease in exchangeable sodium content and the radiosodium space can be most logically explained on the basis of some alteration within the intracellular compartment or in the non-aqueous tissues of the body, such as body fat. The interpretation that such a decrease in body weight is due to a loss of intracellular fluid is consonant with the conclusion reached by other investigators, using the external balance method.

In five instances the change in exchangeable sodium was greater than the change in total body weight due to loss of extracellular fluid. The most likely explanation of the discrepancy in this direction would be that, at the time of the first measurement, the patients had an abnormally high intracellular sodium content, possibly associated with potassium depletion; with diuresis and the associated improvement in the congestive state, some of this intracellular sodium was replaced with potassium.

In spite of intensive therapy with mercurial

\[ \text{Serum Potassium Concentration.} \]

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In spite of intensive therapy with mercurial
diuretics, no untoward effects such as somno-
ience, weakness and disorientation, which have
been previously associated with the "low salt
syndrome,"8 were observed concomitantly with a
decrease in serum sodium concentration.
Such symptoms and signs, in the present study,
were noted in two subjects at a time when the
serum potassium concentration was decreased;
in one of these patients a dramatic improve-
ment occurred when oral supplements of potas-
sium were administered. This observation
suggests that some of the adverse side effects
of intensive mercurial therapy may be more
closely related to a depletion of the body store
of potassium than to a depletion of sodium.

SUMMARY

The radioisotope dilution method was used
to make serial determinations of the exchange-
able sodium content of the body in 13 sub-
jects with congestive failure. The mean value
(65.3 ± 3.2 mEq. per kilogram) was consider-
ably higher than in elderly non-edematous
subjects (40.6 ± 1.5 mEq. per kilogram). Body
weight and exchangeable sodium content de-
creased with intensive therapy. In most in-
stances the decrease in body weight was too
great to be accounted for solely on the basis of
the change in radiosodium space or exchange-
able sodium content. This weight loss, in excess
of the amount which can be explained by the
decrease in sodium values, is attributed to loss
of intracellular fluid.

SUMMARIO IN INTERLINGUA

Le metodo del dilution de radioisotopos
esseva usate in determinaciones serial del
contento de natrium excambiabile in le corpore
de 13 individuos con dysfunctionamento con-
gestive. Le valor median (65,3 ± 3,2 milli-
-equivalentes per kilogramma) esseva con-
siderablemente plus alte que in non-edematose
individuos de etate avantiate (40,6 ± 1,5 milliequivalentes per kilogramma). Le pesos
corporee e le contentos de natrium excambiabile
se abassava in le mesura que le therapia esseva
intensificate. In le majoritate del casos le
reduction del peso corporee esseva plus grande
que lo que haberea essite explicable integre-
mente super le base del alteration del spatio
of radionatrium o del contento de natrium ex-
cambiabile. Le excesso del perdita de peso—i.e.
le perdita de peso non explicabile per le reduc-
del valores de natrium—es attribuite per
nos al perdita de fluido intracellulare.

REFERENCES

1 Miller, G. E.: Water and electrolyte metabolism
in congestive heart failure. Circulation 4: 270,
1951.
2 Squires, R. D., Crosley, A. P., and Elkinton,
J. R.: The distribution of body fluids in con-
gestive heart failure. III. Exchanges in patients
3 Warner, G. F., Dobson, E. L., Rodger, C. E.,
Johnston, M. E., and Pace, N.: The measure-
ment of total "sodium space" and total body
sodium in normal individuals and in patients
4 Akawa, J. K.: Comparison of the thiocyanate
and radiosodium spaces in disease states. Am.
5 Miller, H., and Wilson, G. M.: The measure-
ment of exchangeable sodium in man using the
theoretic considerations of electrolyte
space measured by the tracer method in intact
7 Iseri, L. T., Boyle, A. J., and Myers, G. B.: Water
electrolyte balance during recovery
from severe congestive failure on a 50 milli-
8 Schroeder, H. A.: Renal failure associated with
low extracellular sodium chloride; Low salt
Alterations in Exchangeable Sodium Content, "Sodium\(^{24}\) Space" and Body Weight During The Treatment of Congestive Failure
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