The Effect of Posture on the Excretion of Water and Sodium by Patients with Congestive Heart Failure

By THOMAS A. LOMBARDO, M.D.

Four patients with congestive heart failure were studied in the sitting and the recumbent positions. After hypotonic loading maximal diuresis set in at six hours, as compared with three hours in the normal subjects. Heart failure patients never excreted more than 50 per cent of water intake in either position. Likewise, these patients never equalled sodium intake with urinary excretion of sodium. Sodium excretion in the two positions was approximately the same. Increase in venous pressure, lengthening of circulation time, decline of vital capacity, and decline of serum sodium concentration were noted after each investigation period. These results are interpreted as indicating that the previously postulated intracranial volume regulating center is either inactive or overpowered by a more powerful mechanism in the patient with congestive heart failure.

A NUMBER of recent investigations have been concerned with the possible relationship between alterations in fluid volume and sodium excretion. Thus studies on normal subjects have shown that the decline in sodium excretion which occurs in the sitting position, as compared with recumbency, can be partially but not completely prevented by compression of the neck. Also, removal of small amounts of blood from sitting subjects caused reduction of sodium excretion which could be prevented by compression of the neck. These observations were interpreted as indicating the existence of a central homeostatic mechanism concerned in regulating the volume of body fluids. The finding of hypernatremia and hyperchloremia with little or no salt excretion in the urine of patients with brain damage indicates that a center in the brain may exist, which responds to tonicity changes. Furthermore, Levy and Gassman have produced hyperchloremia and hyperchloruria, but not polyuria, in cats by inducing lesions in the parapontic nuclei.

Welt and Orloff have shown that increases in plasma volume as much as 51 per cent, using salt-poor albumin, are not associated with increase in the renal excretion of sodium. Recently, Strauss and co-workers have shown that hypotonic expansion of the extracellular fluid in normal recumbent subjects is uniformly effective in augmenting the renal excretion of sodium, without changes in creatinine clearance. However, no effect was observed in the sitting position. These data were interpreted as indicating that an increase in extracellular fluid volume in the cephalic portion of the body produces an increase in sodium excretion. Conversely, a contraction of extracellular volume in the cephalad portion of the body may be a stimulus for sodium retention. On the other hand the data indicate that changes in total plasma volume and total extracellular volume are without effect unless associated with corresponding local changes in the cephalic portion of the body.

Since neck compression fails to increase sodium excretion in sitting patients with congestive heart failure, it is possible that the postulated volume regulatory mechanism is inactive in such patients, or that if the mechanism is active, its effects are overshadowed by some more potent mechanism tending to cause sodium retention. In order to test the hypothesis, it was decided to study the effect of posture on sodium excretion in patients with congestive heart failure.

METHODS

Four patients in congestive heart failure were studied on separate days, in the sitting and recum-
bent positions. Dietary control was not attempted, and none of the patients had recently received mercurial diuretics. Each subject came to the laboratory in the fasting state and ingested 200 ml. of 0.14 per cent sodium chloride solution every 30 minutes. Similar observations on normal subjects ingesting 200 ml. of 0.14 per cent sodium chloride solution every hour in the recumbent position, and 400 ml. of the same solution in the sitting position have been reported.\(^2\)

Urine was voided and collected every hour for a nine-hour period, and one small chocolate bar was consumed every two hours. Ten milliliters of blood were drawn at the beginning and the end of each study. Also, venous pressure (saline manometers at right heart level), vital capacity, and circulation time (arm-to-tongue-Decholin) were measured at the beginning and the end of the observations. Serum analysis for sodium was done according to the method described by Mosher and associates,\(^3\) using the flame photometer.

**Results**

Since all four subjects showed the same directional changes in both the sitting and the recumbent positions, the results are presented as averages in figure 1. For the purpose of comparison, similar observations made on normal subjects\(^4\) are also presented.

**Urine Volume in the Sitting and Recumbent Positions.** In both the sitting and the recumbent positions, urinary output increased shortly after the subjects began to ingest the dilute sodium chloride solution. Observations in the sitting position revealed that the normal subjects attained maximal diuresis at the end of the third hour, whereas, the patients with congestive heart failure did not show maximal diuresis until the sixth hour (fig. 1). After the peak of diuresis was reached, the normal subjects continued to excrete water in excess of intake, but the congestive failure subjects failed to do so. Even though the intake was the same for both groups in the sitting position, the patients with congestive failure retained an average of approximately 50 per cent of the water ingested.

Although the water intake of the recumbent patients with congestive failure was twice as great as the normal subjects in the same position, urine volume was considerably less. The normal recumbent subjects excreted a volume greater than their intake, in contrast to the subjects with congestive heart failure who excreted less than 50 per cent of their intake.

No significant differences in total volume output were noted in the subjects with heart failure in the recumbent as compared with the sitting position. However, maximal diuresis was reached at the end of the fourth hour in the recumbent position, and at the end of the sixth hour in the sitting position.

![Fig. 1. Mean alterations in the renal excretion of water and sodium in the sitting and recumbent positions in four normal subjects and four subjects with congestive heart failure.](http://circ.ahajournals.org/content/5/6/92/F1.large.jpg)
Sodium Excretion in the Sitting and the Recumbent Positions. Studies in the sitting position revealed that normal subjects displayed a progressive rise in sodium output which almost equaled their intake. On the contrary, the patients with congestive heart failure were unable to excrete significant amounts of sodium, and at no time did the output equal or exceed the intake (fig. 1).

In the recumbent position, the subjects with heart failure ingested twice as much sodium as the normals. In spite of this, sodium excretion was much greater in the normal subjects. The normal subjects exhibited a greater output of sodium in the recumbent as compared with the sitting position, but the congestive heart failure subjects failed to show this response to a significant degree (fig. 1).

Serum Sodium Content. On both days of the observations, the serum sodium content averaged 139.0 mEq. per liter in the four subjects before ingesting the dilute sodium chloride solution. An average decline in serum sodium of 6.6 mEq. per liter was noted at the end of the sitting studies. This represents a 5 per cent decline in serum sodium concentration (table 1).

In the recumbent position, an average decline in serum sodium was 5.5 mEq. per liter after ingesting the hypotonic solution or a 4 per cent decline (table 1). Therefore, the fall in serum sodium concentration occurring with the ingestion of hypotonic saline did not differ significantly in the sitting and recumbent positions.

Venous Pressure, Circulation Time, and Vital Capacity. The average venous pressure observed before beginning the sitting and recumbent studies was 233 and 224 mm. H₂O, respectively. After ingesting the dilute saline solution in the sitting position, the average rise in venous pressure was 61 mm. H₂O, or a 27 per cent increase. An average rise of 33 mm. H₂O was noted after ingesting the dilute solution while in the recumbent position. This rise represents 19 per cent increase above the level before commencing the study. All subjects demonstrated a rise in venous pressure at the end of the sitting and recumbent observations (table 1).

After ingesting the dilute saline solution, the arm-to-tongue (Decholin) circulation time increased by an average of five seconds in the sitting position and by 28 seconds in the recumbent position. At the same time the vital capacity declined under both conditions studied. The average decline in the vital capacity was 300 and 400 ml. in the sitting and recumbent positions, respectively (table 1).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Serum Sodium mEq./L.</th>
<th>Venous Pressure mm. H₂O</th>
<th>Circulation Time arm-to-tongue (Decholin)</th>
<th>Vital Capacity liters</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sitting Position</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J.A. Before</td>
<td>135.5</td>
<td>190</td>
<td>35</td>
<td>2.5</td>
</tr>
<tr>
<td>After</td>
<td>135.0</td>
<td>265</td>
<td>35</td>
<td>2.1</td>
</tr>
<tr>
<td>B.M. Before</td>
<td>135.0</td>
<td>260</td>
<td>70</td>
<td>3.1</td>
</tr>
<tr>
<td>After</td>
<td>128.0</td>
<td>410</td>
<td>75</td>
<td>3.0</td>
</tr>
<tr>
<td>C.H. Before</td>
<td>139.0</td>
<td>240</td>
<td>25</td>
<td>3.0</td>
</tr>
<tr>
<td>After</td>
<td>132.0</td>
<td>260</td>
<td>30</td>
<td>2.7</td>
</tr>
<tr>
<td>C.W. Before</td>
<td>135.0</td>
<td>190</td>
<td>20</td>
<td>2.5</td>
</tr>
<tr>
<td>After</td>
<td>134.0</td>
<td>240</td>
<td>30</td>
<td>2.2</td>
</tr>
<tr>
<td><strong>Recumbent Position</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J.A. Before</td>
<td>133.0</td>
<td>250</td>
<td>28</td>
<td>2.8</td>
</tr>
<tr>
<td>After</td>
<td>129.0</td>
<td>250</td>
<td>44</td>
<td>2.0</td>
</tr>
<tr>
<td>B.M. Before</td>
<td>135.0</td>
<td>410</td>
<td>43</td>
<td>2.8</td>
</tr>
<tr>
<td>After</td>
<td>129.0</td>
<td>425</td>
<td>125</td>
<td>2.6</td>
</tr>
<tr>
<td>C.H. Before</td>
<td>141.0</td>
<td>160</td>
<td>25</td>
<td>3.0</td>
</tr>
<tr>
<td>After</td>
<td>138.0</td>
<td>170</td>
<td>30</td>
<td>2.8</td>
</tr>
<tr>
<td>C.W. Before</td>
<td>145.5</td>
<td>160</td>
<td>25</td>
<td>2.5</td>
</tr>
<tr>
<td>After</td>
<td>135.5</td>
<td>180</td>
<td>35</td>
<td>2.2</td>
</tr>
</tbody>
</table>

Discussion

The patients differed from normal persons subjected to the same procedures in several significant respects: (1) They exhibited a strikingly positive balance both for sodium and water, in both the recumbent and sitting positions. (2) They developed a well-marked decline in the sodium concentration of extracellular fluid. (3) There was relatively little difference between the two positions as regards sodium output; normal subjects excrete a significantly greater amount of sodium when recumbent. (4) Within the course of several hours significant prolongation of circulation.
time, elevation of venous pressure, and reduct-
on of vital capacity, occurred as a conse-
quency of retention of fluid during the pro-
cedure.

These observations furnish additional evi-
dence for the concept that retention of water
by patients with congestive failure is not neces-
arily secondary to sodium retention, but
tends to occur independently. This confirms
the observations of Fremont-Smith and of
Miller. The observations indicate that the
normal delicate homeostatic mechanisms regu-
lating sodium and water excretion are greatly
disturbed in patients with congestive failure.
It has been previously shown that such pa-
tients do not display the usual increase in
sodium excretion produced by compression of
the neck. The present study indicates an absent
or impaired effect of posture, and also an in-
ability of these patients to prevent significant
osmolar dilution when hypotonic saline is in-
gested. The observations can perhaps be ex-
plained by the assumption that there is some
powerful mechanism, active in subjects with
heart failure, but absent (or inactive) in nor-
mal subjects, and tending to overcome the
normal delicate adjustments to changes in
posture, to alterations in intracranial fluid
volume, and to slight changes in osmolar
concentration. The nature of such a mechanism,
if it actually exists, is obscure at the present
time and can only be elucidated by further
investigations.

SUMMARY

1. Patients with congestive heart failure
ingesting hypotonic solution of sodium chlor-ide displayed relatively greater retention of
water than of sodium and developed significant
decline in serum sodium. It is believed that
such subjects have primary as well as second-
ary (to sodium) water retention.

2. The effect of posture on the excretion of
sodium and of water is absent or markedly
diminished in patients with congestive heart
failure as compared with normal subjects. The
previously postulated intracranial volume regu-
lating mechanism appears to be inactive or
overshadowed in such subjects.

SUMARIO ESPAÑOL

Cuatro pacientes con decompensación cardia-
ca fueron estudiados en posición sentada y
reclinada. Después de haber sido cargados hipo-
tonicamente la diuresis comenzó a las seis horas,
comparado con tres horas en sujetos normales.
Pacientes con decompensación cardiaca nunca
eliminaron más de 50% del agua ingerida en
ninguna posición. De igual manera, estos paci-
etnes nunca igualaron la cantidad de sodio
consumida a la cantidad eliminada en la orina.
Excreción de sodio en las dos posiciones fue
aproximadamente igual. Aumento en la presión
venosa, prolongación del tiempo de circulación,
diminución de la capacidad vital y diminución
en la concentración del sodio en el suero fueron
observados luego de cada período de investi-
gación. Estos resultados han sido interpretados
como indicativos de que el postulado centro
intracranial de volumen o es inactivo o sub-
yugado a un mecanismo mas poderoso en el
paciente con decompensación cardiaca.

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The Effect of Posture on the Excretion of Water and Sodium by Patients with Congestive Heart Failure

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Circulation. 1953;7:91-95
doi: 10.1161/01.CIR.7.1.91

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/7/1/91

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