The Effect of Posture and of Congestion of the Head on Sodium Excretion in Normal Subjects

By J. M. Lewis, Jr., R. M. Buie, M.D., S. M. Sevier, M.D., and T. R. Harrison, M.D.

In healthy young subjects the sodium excretion is decidedly less in the sitting than in the recumbent posture. The difference can be partially overcome by compression of the neck in the sitting position. Some possible implications of these observations will be considered in the subsequent communications.

The present study is the first of several dealing with some of the factors controlling sodium excretion in normal subjects and in patients with congestive heart failure. Only the more pertinent and recent literature will be reviewed.

One of the significant advances of recent years in regard to the nature of congestive heart failure has been the development of a concept that normal homeostatic mechanisms play an important role in the production of some of the outstanding manifestations of this disorder. Thus it has been suggested that inadequacy of the cardiac output in relation to the needs of the body leads to retention of sodium, excess of extracellular fluid, hypervolemia, edema, and aggravation of congestive phenomena. Some have ascribed such sodium retention to decline in glomerular filtration. Others have considered increased tubular reabsorption the predominant factor.

This general concept is supported by the demonstration of decline in cardiac output (either absolute or relative to metabolic needs) in the vast majority of patients with heart failure. The demonstration that retention of sodium occurs not only during heart failure but also during peripheral circulatory failure, which is accompanied by well-marked decline in cardiac output, lends further support to this hypothesis. On the other hand, there are certain objections to this concept. Among them are:

1. Absolute reduction in cardiac output is absent in many instances of heart failure. Reduction of cardiac output relative to metabolic needs leads to decline in tissue oxygen tension. If such a decline is responsible for sodium retention as has been suggested, one would expect edema to occur during states of severe arterial anoxia. Such is not the case, as most individuals with congenital heart disease or advanced pulmonary fibrosis display no edema despite extreme reduction of arterial saturation. (Calculations of venous oxygen tension in such cases, even allowing for high levels of cardiac output and displacement of the oxygen dissociation curve, indicate values at least as low as those calculated for patients with heart failure.) Furthermore, recent investigations indicate that anoxic anoxia increases sodium excretion.

2. Under certain conditions heart failure may occur despite high levels of cardiac output, both absolute and relative to metabolic needs.

3. Marked reduction in glomerular filtration due to intrinsic renal disease is often unassociated with edema. (This objection appears to be valid in regard to the concept of reduced glomerular filtration as the cause of sodium retention, but is not applicable to the alternate concept of increased tubular reabsorption brought about by unknown mechanisms consequent to decline in cardiac output.)

4. The administration of digitalis to normal persons causes a decline in cardiac output as

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great as that existing in many patients with congestive failure but does not produce edema.

(5) Clinical experience seems to indicate that mechanisms for maintaining blood volume (which necessarily involve retention of sodium) exist independently of alterations in cardiac output. Thus, in menstruating women and in patients with bleeding hemorrhoids the blood volume may remain at normal levels despite frequent loss of blood in amounts much less than the amount required to produce a measurable decline in cardiac output. If a homeostatic mechanism exists which maintains normal levels of blood volume under such conditions, one might suspect that the same mechanism might be concerned in the alterations of blood volume occurring in patients with heart failure.

These considerations appear to cast doubt on the concept that inadequacy of cardiac output institutes homeostatic mechanisms which lead to increase in extracellular fluid volume. They do not, however, constitute evidence against the presence of a homeostatic mechanism brought into play by some stimulus other than inadequacy of cardiac output. If such a mechanism exists, one might expect it to involve the central nervous system, which is so intimately concerned with homeostasis, and one might expect it to be initiated by some functional alteration which is common to peripheral circulatory failure and heart failure. The question, therefore, arises as to what possible alteration in the nervous system might exist in both of these conditions.

Peripheral circulatory failure probably tends to be associated with loss of blood (and extravascular fluid) from the head consequent either to an absolute decline in blood volume, or to a distributional shift of blood to a dilated peripheral vascular bed. Heart failure is attended by an initial accumulation of blood in the central portions of the vascular bed (i.e., in the heart, lungs, and great veins). Such a distributional shift in blood will necessarily tend to lead to deficit of blood in the other portions of the vascular bed. If the patient remains in the sitting position because of dyspnea, the tendency toward loss of blood (and extravascular fluid) from the head will presumably be enhanced. It, therefore, seems probable (although not proved) that in their earlier stages both peripheral failure and heart failure are accompanied by a tendency toward deficit of blood within the cranial cavity. If other factors remain constant, loss of blood from the vessels in the cranial cavity will lower the average capillary pressure and will favor reabsorption of extravascular fluid from the tissues in this region.

Thus the possibility is suggested that intracranial deficit of blood and/or extravascular fluid may exist during the initial phases of heart failure and of peripheral failure, and the question arises as to whether such a deficit could be a factor in causing retention of sodium and water by the kidney. Unfortunately, there are no methods of measuring intracranial fluid volumes. It seems likely, although unproved, that the upright position will tend to reduce the volume of blood and/or extravascular fluid in the cranial cavity, and that compression of the neck will tend to have the opposite effect. The experiments to be reported were, therefore, devised in order to study the effects of these procedures on normal subjects.

Methods

The sodium excretion of healthy young male medical students was compared in the sitting and recumbent postures. Each subject drank 200 ml. of a 0.14 per cent sodium chloride solution every 30 minutes, and urine was collected hourly for three to nine hours. (This concentration was selected because the experiments of Wolf proved it seems likely that more constant results would be secured with it than with other solutions.) Each experiment was preceded by an hour of loading, during which time the subjects ingested 400 ml. of the loading solution, the urine excreted during this preliminary hour being discarded.

A given subject was studied in one position (sitting or recumbent) on a given day, and a few days later was studied in the other position.

Experiments dealing with compression of the neck were conducted in the same manner. A blood pressure cuff was wrapped around the sitting subject's neck and inflated to pressures of 15 to 35 mm. Hg in different experiments.

No attempt at preliminary dietary control was made. The subjects did not usually eat on the day of the six-hour experiments, but one small chocolate bar was consumed midway through the nine-hour experiments. The three-hour experiments on com-
pression of the neck were conducted in the early evening, the last meal having been at noon, some five hours previously.

Blood pressures and pulse rates were recorded every 30 minutes.

The temperature of the room in which the experiments were conducted never varied more than 3 C.

Sodium was measured by a slight modification of the technic described by Hoffman and Osgood.\textsuperscript{18}

The modifications were as follows:
1. Protein was removed by the addition of 1 ml. of a 20 per cent trichloracetic acid solution to 5 ml. of urine and filtering through a Whatman No. 42 ashless filter.
2. Either 0.5 ml. or 1.0 ml. of the above filtrate was then added to 10 ml. of recently filtered uranyl zinc acetate solution and the mixture stirred vigorously for two minutes.
3. Only one ether washing was performed.
4. To the completely washed precipitate 11 ml. of triple distilled water were added and the tubes inverted until solution had occurred. The solution was recentrifuged at 1200 revolutions per minute for 10 minutes.
5. The solution was carefully decanted into colorimeter tubes, which were placed in a water bath held at a temperature of 34 C. for 20 minutes. The tubes were then placed in the Evelyn photoelectric colorimeter and read, using a 520 filter.
6. Instead of reading the solutions against a blank of distilled water, a blank of triple distilled water was carried through each procedure and the readings made against it.

RESULTS

1. The Effect of Variations in Posture on the Excretion of Water and of Sodium. The data for normal subjects are summarized in table 1.

In both the sitting and the recumbent positions the urine volume increased shortly after the subject began to drink the dilute solution of sodium chloride. Diuresis began within the first hour but did not attain a peak until several hours had passed. These findings, when considered in relation to those of Verney,\textsuperscript{29} are compatible with the assumption that the increase in urine volume was mediated by inhibition of the antidiuretic hormone of the posterior pituitary, presumably consequent to the ingestion of hypotonic solutions.

The findings of others\textsuperscript{17, 19} were confirmed, for the urine volume usually was greater in the recumbent than in the sitting position.

Sodium excretion was greater in the recumbent position in seven of eight comparisons, a minimal change in the reverse direction being encountered once.* The degree of change in sodium excretion with posture tended to be somewhat greater than that in urine volume in most instances, but this finding was inconsistent.

The time factor appeared to be of importance. Thus, although the expected individual variation was encountered, the average values indicated a decreased excretion of sodium in the sitting position of about 30 per cent during the first hour, and of about 50 per cent during subsequent hours (table 1). The delay in onset of the maximal effect suggests the possibility that a chemical mechanism of some type may be set off by changes in posture.

2. The Effect of Compression of the Neck on the Excretion of Water and of Sodium in the Sitting Position. The data are summarized in table 2.

The changes in urine volume were variable and no general trend was observed. In several instances the urine volume decreased sharply during a period of headache induced by the application of a pressure of 35 mm. Hg. Headache occurring spontaneously in a migrainous subject was also found to decrease urine volume. Since it has been shown\textsuperscript{20} that emotions cause release of antidiuretic hormone from the posterior pituitary, this observation is not surprising.

When the pressure in the cuff was 35 mm. Hg, the sodium excretion increased in seven of eight comparisons. Since this pressure produced considerable discomfort, investigations were made at lower levels of compression.\textsuperscript{†} Fifteen mm. of pressure produced no discomfort, but led to little or no increase in sodium excretion during a period of three hours. On the other

* The apparent discrepancy between these results and those of Kattus and associates\textsuperscript{7} is readily explained. Those investigators found no alteration in sodium excretion on changing from the recumbent to the sitting position. Our observations suggest that the lag in the effect is greater than the duration of the period (20 minutes) during which their subjects were studied in the sitting posture.

† Because of the thickness of the intervening tissues, it is uncertain what fraction of the pressure in the cuff was transmitted to the internal jugular vein.
TABLE 1.—Urinary Excretion of Sodium and of Water of Normal Subjects in Recumbent and Sitting Postures

<table>
<thead>
<tr>
<th>Subject</th>
<th>Duration of experiment in hours</th>
<th>Fluid Ingested</th>
<th>Recumbent</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Compsition % NaCl</td>
<td>Volume</td>
<td>1st hour</td>
<td>2nd hour</td>
</tr>
<tr>
<td>TT</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>Urine ml.</td>
</tr>
<tr>
<td>DS</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
<tr>
<td>RP</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
<tr>
<td>F6</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
<tr>
<td>EB</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
<tr>
<td>BC</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
<tr>
<td>JL</td>
<td>6</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
<tr>
<td>SMS</td>
<td>6</td>
<td>0.14</td>
<td>400</td>
<td>Sodium mg.</td>
</tr>
</tbody>
</table>

TABLE 2.—Effect of Compression of the Neck of Sitting Subjects on the Urinary Excretion of Sodium and of Water

<table>
<thead>
<tr>
<th>Subject</th>
<th>Length of Experiment, hours</th>
<th>Cuff Pressure, mm. Hg</th>
<th>Fluid Ingested</th>
<th>Sitting Position with Cuff</th>
<th>Sitting Position without Cuff</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Conc. NaCl</td>
<td>Volume, ml/hr.</td>
<td>Total Urine Volume, ml.</td>
<td>Total Sodium, mg.</td>
<td>Total Urine Volume, ml.</td>
</tr>
<tr>
<td>RP</td>
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<td>0.14</td>
<td>400</td>
<td>1123</td>
<td>809</td>
</tr>
<tr>
<td>TT</td>
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<td>0.14</td>
<td>400</td>
<td>635</td>
<td>556</td>
</tr>
<tr>
<td>DS</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1509</td>
<td>1542</td>
</tr>
<tr>
<td>FG</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1232</td>
<td>450</td>
</tr>
<tr>
<td>EB</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1300</td>
<td>524</td>
</tr>
<tr>
<td>BC</td>
<td>3</td>
<td>0.14</td>
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<td>623</td>
<td>620</td>
</tr>
<tr>
<td>SMS 4</td>
<td>6</td>
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<td>400</td>
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<td>1698</td>
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<tr>
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<td>0.14</td>
<td>400</td>
<td>2180</td>
<td>1515</td>
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<td>9</td>
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<td>2468</td>
</tr>
<tr>
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<td>400</td>
<td>1299</td>
<td>780</td>
</tr>
<tr>
<td>DS</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1323</td>
<td>882</td>
</tr>
<tr>
<td>RP</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>828</td>
<td>668</td>
</tr>
<tr>
<td>ZS</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1219</td>
<td>484</td>
</tr>
<tr>
<td>BJ</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1411</td>
<td>1273</td>
</tr>
<tr>
<td>TG</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>114</td>
<td>318</td>
</tr>
<tr>
<td>VK</td>
<td>3</td>
<td>0.14</td>
<td>400</td>
<td>1132</td>
<td>601</td>
</tr>
</tbody>
</table>

hand, pressures of 20 mm. were tolerated readily, and produced well marked increase in sodium excretion during nine-hour experiments. Measurements of pulse rate and of blood pressure yielded no significant changes when the neck was compressed. It is, therefore, unlikely that the carotid sinus mechanism was concerned in the changes in sodium excretion.
Discussion

The starting point of these studies was the idea that certain alterations in the distribution of body fluids might be reflected in changes in sodium excretion. The data which have been presented are compatible with this concept. As compared to recumbency, the sitting position caused decline in sodium excretion. This decline could be partially prevented by compression of the neck of sitting subjects. These findings could be interpreted as suggesting the existence within the cranial cavity of a volume regulating mechanism. However, the data are deficient in certain respects: sodium intake was not standardized prior to the experimental periods, and it would be desirable to know whether elimination of the variable would result in more uniform results. The effects of compression of the neck were studied only in the sitting position, and it is uncertain whether this procedure would influence the sodium output of recumbent subjects.

Likewise, it is not clear from the data whether the degree of cervical compression of the neck required to induce changes in sodium excretion is within or without the range of venous congestion occurring in health and in disease. For these reasons, and others which will be discussed in subsequent communications, it would seem desirable to defer interpretation of the results until additional studies have been completed. Such studies are in progress and will be reported in a subsequent communication.

SUMMARY

1. Healthy young men ingesting hypotonic sodium chloride solution displayed a well marked decline in the excretion of water and of sodium in the sitting as compared to the recumbent position.

2. This effect of the sitting posture on sodium excretion could be partially but not entirely overcome by compression of the neck.

3. Further investigations are needed in order to determine whether the observations are pertinent to the mechanisms controlling sodium excretion in health and in disease.

REFERENCES


15. HAMILTON, W. F., MOORE, J. W., KINSMAN, M. M., AND SPURLING, R. G.: Studies on the circu-


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