The Effect of Posture and of Compression of the Neck on Excretion of Electrolytes and Glomerular Filtration: Further Studies

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Compression of the neck of the sitting subject caused increase in urinary sodium output without change in glomerular filtration (creatinine clearance) or apparent cardiac output (electrokymograph). The possible existence of an intracranial mechanism sensitive to alterations in volume of extracellular fluid and regulating the volume of extracellular fluid is suggested.

Previous studies from this laboratory,\(^1\)\(^2\) have indicated: (a) that under comparable conditions the urinary excretion of sodium, like that of chloride\(^3\) and of water,\(^4\) is less in the sitting than in the recumbent position, and (b) that compression of the neck of the sitting subject may cause increase in sodium excretion. Since renal clearances were not measured, the previous work provided no evidence concerning the relative importance of alterations in filtration and in reabsorption.

The purposes of the present communication are to extend the previous observations, particularly in relation to: (a) investigations of glomerular filtration and cardiac output, (b) studies of the excretion of chloride and of potassium, as well as of sodium and water, and (c) measurements during congestion of the head under different circumstances (induced by tilting the body or by compression of the neck of the recumbent subject). It is hoped that such observations may furnish information concerning some of the mechanisms regulating sodium excretion under physiologic conditions, in order that these mechanisms may later be investigated in patients with circulatory disorders.

Methods

The subjects were normal males, aged 22 to 30. Various attempts were made to maintain constancy of sodium excretion by previous regulation of either the intake of "added" sodium only, or by utilizing a diet of known sodium content and also measuring the "added" sodium. None of the various plans employed resulted in a high degree of constancy of diurnal and nocturnal sodium output. The least unsatisfactory procedure involved the utilization of a diet containing 1 Gm. of sodium, plus the addition of 7.0 Gm. of sodium chloride to the food at the regular meals (2 Gm. at breakfast and 2.5 Gm. at the other two meals). Even with this procedure, considerable variations of sodium output occurred. Such variations are probably to be ascribed to incidental alterations in activity, posture, endocrine balance, environmental temperature, and other factors.

On the date of the experiments the subject came to the laboratory without breakfast, and ate no food other than one-half of a chocolate bar every hour or one egg every two hours until the termination of the experimental procedure which lasted 6 to 12 hours (usually 10). In an attempt to attain reasonable constancy of urine volume and of sodium excretion, 200 ml. of a loading solution of hypotonic (0.14 per cent) sodium chloride were ingested at hourly or half hourly intervals. However, these functions varied widely in the different subjects, and they displayed unexplained hourly variation in the same subject. Of the several different variations employed in the loading procedures, the least unsatisfactory appeared to be the ingestion of the solution at hourly intervals in the recumbent position and at
half hourly intervals in the sitting posture. It was also found that, under the conditions of the experiments, even approximate constancy of excretion of sodium and water was not minimum. However, it was found that this technic was highly unsatisfactory because the sitting subjects often displayed an initially positive sodium balance during the procedure.

Attained until after two or more hours of loading had passed (fig. 1).

In the earlier experiments a given individual was studied on successive days in the hope that incidental variations in sodium excretion (due to alterations in activity, environmental temperature, etc.) might thereby be kept at a

![Graph of renal excretion of sodium and water by recumbent subjects]

**Fig. 1.** The upper graphs indicate the inconstancy of the output of sodium and water during the initial hours and the lesser degree of variability after the third hour under the conditions of the experiment (ingesting 200 ml. of 0.14 per cent sodium chloride solution every hour in the recumbent posture).

The lower graphs display similar findings for the sitting posture when twice as much of the same loading solution was ingested. When the same amount was ingested as in the recumbent position (the experiment indicated by an encircled E) the excretion of both sodium and water continued to increase during the entire six hour period.

Despite the lesser intake of sodium in the recumbent posture the usual negative sodium balance and the usual increase in excretion (as compared with that occurring in the same subjects when sitting) was encountered in this position.

and the recumbent subjects exhibited a markedly negative sodium balance (fig. 2), which altered the results of the subsequent days. Therefore, the procedure of allowing two or more days to elapse between successive experiments on the same subject was adopted for the later experiments.
(a) **Sodium** was measured by the method described by Hoffman and Osgood, with the slight modifications mentioned in the previous study.  
(b) **Potassium** determinations were done by the chloroplatinic acid titrimetric method. The method of Consolazio and Talbot was utilized as described, with the exception of the use of 1 and 2 ml urine samples rather than 0.2 ml samples. By this alteration in technic, a sample containing 0.0008 mEq. or more was assured, thus obviating the 10 to 20 per cent recovery loss produced by the use of samples containing less potassium.  
(c) **Chloride** was determined by a modification of the Volhard-Harvey open Carius titrimetric method.  
(d) **Creatinine**. Endogenous creatinine clearance was determined by the Bonsnes and Taussky modification of the Jaffe reaction, utilizing the modified Folin-Wu tungstic acid method of protein precipitation advocated by Brod and Sirota. It should be pointed out that a correction in technic was effected early in the course of these experiments which led to somewhat higher clearance values. The order of magnitude of the change was quite small, and did not affect the values on a given day.  
(e) **Cardiac output**. Directional changes in cardiac output were measured by the electrokymographic technic as described by Ring and co-workers, with certain modifications which will be discussed in detail elsewhere. It should be pointed out that regardless of the absolute accuracy of this procedure, it apparently furnishes a reliable index of directional changes.

**Results**

In the various figures and tables the findings in those experiments which yielded positive results are presented in some detail, while the negative results are presented in summarized form.

1. Electrolyte Excretion, Glomerular Filtration and Cardiac Output in the Recumbent and Sitting Postures. The data on glomerular filtration and electrolyte excretion are presented in table 1. The urine volume was relatively greater (in comparison to the water intake) in the recumbent posture. Although the sodium intake was twice as great in the sitting as in the recumbent posture, the output of sodium was greater in the latter position (table 1, figs. 1 and 2). It is evident that the excretion of chloride tended to parallel that of sodium which, as previously reported, tends to be markedly less in the sitting than in the recumbent position.

Potassium excretion displayed no consistent variations with posture (table 1, fig. 2). In both positions the urinary potassium exhibited a decline after the first four or five hours of

![Fig. 2. The subjects ingested the loading solution (0.14 per cent sodium chloride) in amounts of 400 (sitting) or 200 (recumbent) ml. per hour for a 10 hour period. In the upper graph the urinary output of sodium (columns) and potassium (bars) during this period is depicted for the first and last of three successive days of loading. The values for the second day, which were intermediate in all instances, are not shown. In both subjects the sodium balance was positive in the sitting position during the first day's experiment but negative on the third day, indicating that the previously retained sodium was now being excreted. When, after a 10 day interval, the procedure was repeated with the subjects in the recumbent position, the initial day's sodium balance was markedly negative and this excessive excretion of sodium was still present, but in much lesser degree, on the third day. In contrast to sodium the excretion of potassium did not display consistent alterations either as the result of changes in posture or of continuing the procedure for three successive days. In the lower graph are shown data on the hourly excretion of potassium in the two postures. The initial rise in potassium excretion was inconstant but the later decline was a consistent finding in most of the experiments.](http://circ.ahajournals.org/)

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TABLE 1.—Electrolyte Excretion and Creatinine Clearance in the Sitting and Recumbent Postures.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Position</th>
<th>Date</th>
<th>Hour of Ingestion</th>
<th>Creatinine Clearance</th>
<th>Urine Volume per hr.</th>
<th>Electrolyte Excretion</th>
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<td>Na mEq.</td>
<td>Cl mEq.</td>
<td>K mEq.</td>
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<td>B. O.</td>
<td>Recumbent</td>
<td>9/26</td>
<td>3</td>
<td>109</td>
<td>164</td>
<td>14.6</td>
</tr>
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<td>4</td>
<td>104</td>
<td>291</td>
<td>16.0</td>
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<td></td>
<td>10/4</td>
<td>3</td>
<td>139</td>
<td>209</td>
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<td>455</td>
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<td>545</td>
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<td>574</td>
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<td>109</td>
<td>490</td>
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<td>400</td>
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<td>113</td>
<td>192</td>
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<td>4</td>
<td>103</td>
<td>286</td>
<td>7.0</td>
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<td>W. V.</td>
<td>Recumbent</td>
<td>9/29</td>
<td>3</td>
<td>131</td>
<td>261</td>
<td>19.9</td>
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<td>319</td>
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<td>143</td>
<td>506</td>
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<tr>
<td>S. E.</td>
<td>Recumbent</td>
<td>9/29</td>
<td>3</td>
<td>107</td>
<td>236</td>
<td>7.6</td>
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<td>192</td>
<td>9.4</td>
</tr>
<tr>
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<td>10/5</td>
<td>3</td>
<td>114</td>
<td>246</td>
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<td>114</td>
<td>302</td>
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<tr>
<td></td>
<td>Sitting</td>
<td>11/16</td>
<td>3</td>
<td>144</td>
<td>252</td>
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<td>4</td>
<td>111</td>
<td>316</td>
<td>8.8</td>
</tr>
</tbody>
</table>

The subjects drank 200 ml. of 0.14 per cent sodium chloride solution hourly when recumbent and half hourly when sitting. The data shown are for the third and fourth hours after starting to ingest this loading solution.

TABLE 2.—Effect of Compression of the Neck of Sitting Subjects on Creatinine Clearance and on Renal Excretion of Sodium.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Conditions</th>
<th>Creatinine Clearance (ml. per min.)</th>
<th>Sodium Excretion (mg. per hr.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before* During (hrs.)</td>
<td>After (hrs.)</td>
</tr>
<tr>
<td>B. O.</td>
<td>Control</td>
<td>117 103 111 112 111 108 107</td>
<td>198 165 159 157 166 213 168</td>
</tr>
<tr>
<td></td>
<td>Neck compressed</td>
<td>132 131 129 123 119 118 125</td>
<td>189 154 150 138 123 174 135</td>
</tr>
<tr>
<td>T. L.</td>
<td>Control</td>
<td>119 143 115 108 109 143 111</td>
<td>93 154 150 138 123 174 135</td>
</tr>
<tr>
<td></td>
<td>Neck compressed</td>
<td>122 122 125 110 124 107 95</td>
<td>179 219 229 264 224 167 140</td>
</tr>
<tr>
<td>S. E.</td>
<td>Control</td>
<td>145 139 145 144 147 145 147</td>
<td>161 191 243 237 261 259 411</td>
</tr>
<tr>
<td></td>
<td>Neck compressed</td>
<td>111 112 112 111 106 114 112</td>
<td>225 250 280 270 252 200 207</td>
</tr>
<tr>
<td>W. V.</td>
<td>Control</td>
<td>141 143 133 138 141 138 137</td>
<td>206 216 226 214 270 237 255</td>
</tr>
<tr>
<td></td>
<td>Neck compressed</td>
<td>124 133 131 132 127 128 125</td>
<td>170 220 283 268 271 135 130</td>
</tr>
</tbody>
</table>

* The third hour of ingesting the loading solution and the hour immediately preceding compression of the neck.
gesting the potassium-free loading solution (fig. 2).

As judged by creatinine clearance, glomerular filtration exhibited rather wide and unexplained variations in the same subject on different others, who have found minimal or no difference between the two positions, that the marked variations in sodium excretion must be ascribed to alterations in tubular activity and not to changes in filtration.

![Diagram](http://circ.ahajournals.org/)

**Fig. 3.** The sitting subjects ingested the usual loading solution for a period of 10 hours during the last eight of which hourly urine specimens were obtained.

During the control studies (white columns) sodium excretion tended to rise and potassium excretion to fall. When the experiments were repeated and the neck was compressed for four hours (shaded columns) during the middle of the eight hour period, sodium excretion increased in each instance over that immediately before and after and also over that which occurred during the corresponding period in the control experiments. Chloride excretion behaved similarly. Potassium excretion seemed to be slightly increased during compression of the neck in 2 of the subjects but not in the other 2.

The effects of posture on directional changes in cardiac output were studied also. These observations will be published in detail elsewhere. Cardiac output exhibited the usual decline in the sitting posture, as previously found by others using different methods. The average of two or three cardiac output determinations was obtained in the recumbent
and in the sitting position, for each of the 4 subjects. The average of these four cardiac output values was 6.3 liters per minute in the recumbent position, and 5.8 liters per minute in the sitting position. Thus an 8 per cent decrease in cardiac output occurred in changing from the recumbent to the sitting posture.

2. The Effect of Compression of the Neck of Sitting Subjects on Electrolyte Excretion and on Glomerular Filtration. The data concerning sodium (table 2, fig. 3) are in accord with those previously reported, an increase being found upon the application of a pressure of 20 mm. Hg to the necks of the several subjects. Similar increments in the excretion of chloride were observed when the neck was compressed (fig. 3). On the other hand, the excretion of water was not affected significantly. Inflation of the cervical cuff produced no significant immediate changes in pulse rate or blood pressure. Hence it is unlikely that alterations in carotid sinus

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**Fig. 4.** Effect of compression of the neck on cardiac output. The subjects assumed the sitting position and drank 200 ml. of 0.14 per cent sodium chloride solution every 30 minutes for 10 hours. Cardiac output was measured at the third and fourth hours. The cuff was applied about the neck and inflated to 20 mm. Hg and cardiac output measured hourly for the subsequent 4 hours. The cuff was then released and cardiac output measured for 2 additional hours.

The average of the first two cardiac output values was used as the control value. The cardiac output values subsequently obtained were plotted as percentage change from the control.

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**Fig. 5.** The recumbent subjects ingested the standard loading solution for a period of 10 hours during the last eight of which urinary sodium was measured.

Elevation of the foot of the bed to an angle of 20 degrees for a period of four hours had no consistent effect on sodium excretion as compared with that in the preceding and succeeding two hour periods. Compression of the neck of recumbent subjects was attended by a slight rise in sodium ex-
activity were concerned in the effects on electrolyte excretion.

Consistent results as regards potassium were not obtained by compression of the neck. In 2 of the subjects urinary potassium rose, while in the other 2 the usual decrease occurred as in the control observations when the neck was not compressed. In view of the failure of cervical compression to cause a decline in potassium excretion, it seems unlikely that the increased excretion of sodium was mediated via the adrenal cortex.

Glomerular filtration (table 2) remained unaffected by compression of the neck, as did cardiac output (fig. 4). It is, therefore, clear that the observed alterations in electrolyte excretion are to be ascribed to variations in renal tubular activity, and that such variations are not to be ascribed to changes in cardiac output.

3. The Effect of the Head-Down (Trendelenburg) Position on Sodium Excretion. The results obtained when the foot of the bed was elevated at an angle of approximately 20 degrees are presented in figure 5. Consistent alterations in sodium excretion did not occur in the 4 subjects. Filtration rate did not change significantly in the one experiment in which it was measured. In view of the negative results, the excretions of chloride and of potassium were not measured.

4. The Effects of Compressing the Neck of Recumbent Subjects on Sodium Excretion. The failure of sodium excretion to increase in the head-down as compared with the horizontal position was surprising, in view of the previous observations of the effects of posture and of compressing the necks of sitting subjects. Consequently, additional experiments were performed, the neck being compressed by a pressure of 20 mm. Hg while the subject was in the recumbent posture. The results (fig. 5) of these experiments were in sharp contrast to those obtained in the sitting position (fig. 3). The rise in sodium excretion occurring upon compression of the neck in the sitting experiments was not observed in the same subjects when recumbent.

Discussion

Excretion of electrolytes is conditioned by numerous variables. To control all of them would require accurate standardization not only of dietary and of fluid intake, but also of environmental temperature, physical and psychic activity, emotional state, endocrine balance, and probably of various other as yet unknown factors which influence the activity of the homeostatic mechanisms of the body. Since such elaborate standardization has been impractical in our work, it has been necessary to determine by trial and error the experimental conditions which can be expected to yield reasonably uniform results.

The data in figures 1 and 2 indicate that a satisfactory degree of constancy of excretion of sodium and of water can be maintained for several hours, if the following conditions are fulfilled: (1) The intake of sodium in the diet should be controlled within a fairly narrow range, and a known amount of sodium chloride should be added to the food for several days prior to the actual experiments. (2) Observations should not be made on the same subject on successive days, as the sodium balance of the initial day (markedly negative when recumbent and usually positive when sitting) may have a striking influence on excretion of sodium on the subsequent days (fig. 2). (3) The experiments should preferably be started in the basal state, and only minimal amounts of food (of constant sodium content) should be ingested during the experiments. (4) Data are not available with various loading solutions but when the solution employed in these experiments (0.14 per cent sodium chloride solution) is ingested in the amounts indicated (200 ml. hourly and half hourly, respectively, for recumbent and sitting postures), the output of water and of sodium rises rapidly for a period of two to three hours, and then maintains reasonable constancy or changes slowly for several additional hours (fig. 1). Hence if one wishes to have a preliminary control period before an experimental procedure, it is necessary that the third or, preferably, the fourth hour after the onset of loading be considered...
as the control period. Even when these several precautions are observed, striking constancy of results cannot be expected. Thus, under the conditions described, the subject often tends to have, after the first few hours of loading, an initially positive sodium balance when sitting (figs. 1 and 3) and an initially negative balance when recumbent (figs. 1 and 5), these alterations often being followed by gradual increase and decrease, respectively, in the sodium output during the later hours of prolonged experiments. Likewise, the hourly sodium and chloride outputs vary from day to day, and the potassium excretion may undergo an initial rise (during the early hours of loading), usually exhibiting a steady decline during subsequent hours (fig. 2).

The data may clarify somewhat the relative quantitative importance of some of the several factors determining sodium output. Over a period of 8 to 12 hours, the posture of the subject was a more important factor than was the current intake of sodium. Thus the recumbent subjects who were ingesting only one half as much sodium excreted much more than did the sitting subjects (figs. 1 and 2, table 1). On the other hand, the degree of saturation of the sodium stores of the body, i.e., the sodium balance during the several preceding days, was more important than posture. Thus, when the procedure was repeated daily in the same posture, the striking difference between sodium excretion during the initial day spent in two positions had disappeared by the third day, as the result of respective accumulation and depletion in the sitting and recumbent postures (fig. 2).

Mechanism of Postural Effect on Urine Volume. Brunn and his co-workers have presented convincing evidence that the decline in urine volume in the standing position is related to increased activity of the antidiuretic hormone of the posterior pituitary. When allowance is made for the time lag in the action of this hormone, it would appear that it is released as the result of a rapidly acting mechanism—probably situated in the hypothalamus. It appears likely that the hypothalamic-posterior pituitary mechanism is concerned also in producing the relative oliguria of the sitting position.

The data in figure 1 indicate that in both postures the urine volume tended to increase during the first several hours of the experiments, and became maximal three to four hours after the subjects began to ingest the hypotonic loading solution. It is also evident, from figure 1, that during this period the water load (i.e., the amount of retained water) was increasing more rapidly than was the sodium load (amount of retained sodium), and hence that there was a tendency toward decline in osmotic pressure of extracellular fluid. Since the hypothalamic-posterior pituitary mechanism is very sensitive to slight changes in osmotic pressure, this disproportion would tend to reduce the activity of the mechanism responsible for water retention.

Compression of the neck had no consistent effect on urine volume.

Mechanism of Postural Changes in Sodium Excretion. During the past several years considerable difference of opinion has existed concerning the relative importance of filtration and of reabsorption in relation to alterations of sodium excretion. The observations of the present study indicate that the changes of sodium excretion induced in healthy subjects by change in posture and by compression of the neck are independent of filtration, and hence must be attributed to alterations in tubular activity.

The suggestion that changes in cardiac output constitute a major controlling factor in determining sodium excretion is not supported by these studies. The sodium output of the sitting subject could be increased either by lying down, which caused an increase in cardiac output, or by compression of the neck, which caused no increase (fig. 4).

Compression of the neck by the cuff was not attended by significant immediate changes in blood pressure or pulse rate. It therefore seems unlikely that alterations in the activity of the carotid sinus mechanism were responsible for the effects of cervical compression on sodium excretion.

It is of interest to note that the decline in sodium excretion in the sitting as compared
with the recumbent posture (table 1) was greater than the increase produced by compression of the neck of sitting subjects (table 2). In other words, the latter procedure was only partially effective in overcoming the decline produced by the sitting posture. Since it has been shown that venous congestion of the legs causes decline in sodium excretion, and since the sitting position tends to produce such congestion, this is one possible factor. Another is the increase in renal venous pressure, which has been shown to decrease sodium excretion, and which would be expected to be present in the sitting as compared to the recumbent posture. However, compression of the neck which caused increased excretion of sodium, could have no direct effect on the degree of congestion of the legs or on the level of renal venous pressure. Hence it would appear that some effect on the intracranial contents is probably also concerned in the postural changes in sodium excretion.

If the tentative conclusion is drawn that the increase in sodium excretion produced by compression of the neck of sitting subjects is the result of some alterations within the cranial cavity, and that the same mechanism constitutes one factor in the decline in sodium excretion produced by the sitting position, the question arises as to the precise nature of the mechanism involved. On first thought, it might appear that sodium excretion tends to parallel intracranial venous pressure, which is diminished by the sitting posture and increased by compression of the neck. The decline in sodium excretion, in states of peripheral circulatory failure which are usually accompanied by decline in venous pressure, is in keeping with such an assumption. On the other hand, heart failure is accompanied by elevation of venous pressure and by decline in sodium excretion. Likewise, intravenous administration of hypertonic albumin solution causes rise in venous pressure but decline in sodium excretion. It seems unlikely, therefore, that alterations in intracranial venous pressure are directly concerned in the regulation of sodium excretion.

Cerebral blood flow was not measured in these experiments, and the possibility that alterations in this function may have been concerned in the effects on sodium excretion cannot be entirely excluded. This possibility involves, however, the unlikely assumption that compression of the neck and shift to the horizontal posture cause the same directional changes in cerebral blood flow.

The observations on the effects of posture and of compression of the neck are compatible with the concept of an intracranial "volume center," activated by decline in the volume of fluid (blood or extravascular) in the cranial cavity, and functioning in such a way as to tend to maintain constancy of the volume of body fluids. Such a concept is in accord with the retention of sodium in states of peripheral circulatory failure. The retention of sodium in patients with heart failure is not contrary to this concept, because heart failure is initially associated with redistribution of blood to the central portions of the vascular bed, i.e., to the heart and lungs. Such redistribution will necessarily be attended, in the initial stages, by deficit of blood and extravascular fluid in the periphery (and, presumably, in the cranial cavity). Unfortunately, there are no methods of measuring fluid volumes in the cranial cavity, and in the absence of such measurements this concept can be supported only by indirect evidence.

If an intracranial mechanism regulating volume of body fluids actually exists, the data reported in this paper, and the studies of others, offer certain indications concerning its function.

(1) The reason for the discrepancy between the effects of compression of the neck in recumbent subjects, and in the same subjects when sitting, is not clear and requires further investigation.

(2) In determining the level of sodium output other factors, such as the previous intake of sodium, are of greater quantitative importance than posture and compression of the neck, which appear to act through the assumed central mechanism. The latter would, therefore, appear to be analogous to the fine adjustment of a microscope, the coarse adjustment cor-
responding to such factors as the sodium stores of the body and the endocrine balance.

(3) If it is assumed that the changes in sodium excretion were conditioned, in some manner, by changes in intracranial fluid volume, the question arises as to whether alteration in the amount of blood or of extravascular fluid was responsible. In order to distinguish between these possibilities it would be necessary to establish conditions in which the intravascular and extravascular fluid volumes varied in opposite directions. Such was not done in our experiments, but has been done by others\(^1\) who found that the injection of hypertonic albumin solution caused decline in the excretion of sodium but not of water. Since, following the administration of hypertonic albumin solution, the intravascular volume would be expected to increase at the expense of the extravascular volume, this observation, when considered in relation to our findings, suggests that decline in extravascular fluid volume (or some closely related factor) within the cranial cavity causes reduction in sodium excretion. The fact that potassium, a predominantly intracellular ion, did not exhibit the consistent changes observed with sodium, might be interpreted as indicating that the postulated volume regulating the mechanism is related to changes in the volume of extracellular rather than of intracellular fluid in the cranial cavity.

The observations throw no light on the intermediate mechanisms whereby the postulated central mechanism might affect the reabsorption of sodium by the renal tubules. The data in the previous\(^2\) and the present studies (table 2) indicate that the alterations in sodium output produced by changes in posture and by cranial compression develop relatively slowly, being greater in the second and third hours than in the first. On the other hand, the reduction in sodium excretion following removal of pressure from the neck appeared rapidly, and was pronounced during the first hour (table 2). These effects are perhaps suggestive of a chemical or neurochemical mechanism. They appear to be more rapid than might be anticipated if the adrenal cortex were concerned. The failure to demonstrate an inverse relationship between the excretion of potassium and of sodium is also against the assumption that the changes in sodium excretion were mediated through the adrenal gland. Observations on patients with Addison’s disease and with Simmonds’ disease may be of value in elucidating the intermediate mechanisms. Such studies are in progress.

The starting point of this and the preceding report\(^2\) was the desire to investigate the concept of a homeostatic mechanism concerned with sodium retention as a means of protecting the body against various types of circulatory failure.\(^25-24\) Our studies thus far have been limited to normal subjects and are not, therefore, directly applicable to patients with circulatory failure, or to such problems as the importance of the orthopneic position in relation to edema formation. In so far as can be judged by the data on normal subjects, it would appear that a central homeostatic mechanism concerned with sodium conservation does exist, and that this mechanism is brought into play not by decline in cardiac output but rather by alterations in the distribution and volume of body fluids.

**Summary**

Various procedures were studied in an endeavor to secure constancy of hourly urinary excretion of sodium, chloride, and potassium. A high degree of constancy was not obtained but certain conditions were found to reduce the extent of variability. These are described.

The change from the sitting to the recumbent posture produced well marked increment of sodium and chloride excretion, but the change from recumbency to the head-down (Trendelenburg) position caused no significant increase. Compression of the neck of the sitting subjects caused increased output of sodium and chloride, but the same procedure had little or no effect in recumbent subjects. The conditions which regularly altered the excretion of sodium and of chloride did not have significant effects on the output of potassium.

Consistent alterations in creatinine clearance were not induced by procedures which caused well-marked change in the urinary excretion of sodium and of chloride.

Cardiac output, as estimated by the electrokymograph, changed in the same direction as
sodium excretion when posture was altered. The rise in sodium excretion produced by compression of the neck of sitting subjects was not associated with measurable change in cardiac output.

The suggestion is offered that there is a central mechanism which functions as a "volume center," and which tends to maintain homeostasis by increased tubular reabsorption of sodium when conditions occur which reduce the volume of intracranial extracellular fluid.

The experiments offer no support to the concept that decline in cardiac output is of importance in relation to sodium retention. In so far as can be judged from observations on healthy subjects, the data suggest that homeostatic retention of sodium should not be ascribed to alterations in blood flow unless alterations in the distribution of body fluids can be eliminated as causative mechanisms.

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