Effect of Neck Compression on Sodium Excretion in Subjects with Congestive Heart Failure

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Previous studies on normal subjects have shown that neck compression causes increased urinary excretion of sodium in the sitting position, but has little or no effect in the recumbent position. In the present study, patients with congestive heart failure have exhibited no increase in urinary sodium excretion after neck compression.

Previous studies 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. Neck compression of the same subjects in the horizontal and head-down (Trendelenburg) positions had little or no effect. 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 which regulates the volume of extracellular fluid by altering sodium excretion, and is apparently activated by changes in the volume of body fluids rather than by changes in cardiac output. The present study was performed to learn whether such a mechanism is active in patients with congestive heart failure.

Methods

Four patients in congestive heart failure were studied. None had recently received mercurial diuretics. Each subject came to the laboratory in the fasting state, and ingested 200 ml. of 0.14 percent sodium chloride every 30 minutes. Urine was collected at hourly intervals for a period of nine hours, and one small chocolate bar was consumed every two hours. Serum sodium analyses were done at the beginning and end of the studies. These observations will be reported later.

Experiments dealing with compression of the neck were conducted in the same manner but were performed a few days later. A blood pressure cuff was wrapped around the subject's neck and sustained at a pressure of 20 mm. Hg.

Sodium analyses were done according to the method described by Mosher and co-workers using the flame photometer.

Results

Since the results were essentially similar for all four subjects, they are presented as averages in figure 1. For the purpose of comparison, similar observations made on normal subjects are also presented.

Urine Volume. This function increased shortly after the subjects began to drink the dilute sodium chloride solution. Diuresis attained a peak at the end of the second hour in the normal subjects, but the congestive heart failure patients did not show maximal diuresis until the sixth hour. The total diuresis was much smaller in the patients exhibiting heart failure. After maximal diuresis was reached, the normals continued to excrete water in excess of intake, whereas the congestive heart failure subjects failed to do so.

Neck compression was not associated with significant alterations in urine volume of either group. The diuresis noted in the normal subjects is compatible with the studies of Verney, who demonstrated inhibition of the posterior pituitary consequent to the ingestion of hypotonic solution. A lack of diuresis in the patients with heart failure was perhaps due to the presence of excessive amounts of an antidiuretic substance.

Sodium Excretion. During the control studies,
the normal subjects displayed a progressive rise in sodium output which soon exceeded the intake (fig. 1). On the contrary, the patients with congestive heart failure were unable to excrete the sodium, and at no time did the output equal or exceed the intake. As the observations continued, a decline in sodium excretion was noted.

Compression of the neck in normal subjects produced a significant increment in sodium excretion which was maximal at the end of the second hour. This procedure failed to increase sodium excretion in subjects with heart failure.

The patients with congestive heart failure failed to exhibit increased sodium output when the neck was compressed in the sitting position. Normal subjects displayed this response in the sitting position but not in the horizontal position. The reason for these variations is not clear at the present time. In any case the observations suggest either (1) that the postulated volume regulatory mechanism is inactive in patients with congestive failure, or (2) that if this mechanism is active, its effects are overshadowed by some more potent mechanism, tending to cause sodium retention.

**SUMMARY**

1. Neck compression, under conditions which cause significant increments in sodium excretion of normal subjects, failed to increase sodium excretion in four patients with congestive heart failure.

2. In patients with congestive failure, the previously postulated intracranial “volume center” concerned with the regulation of the volume of extracellular fluid appears to be either inactive or overshadowed by some more powerful mechanism favoring sodium retention.

3. Patients with congestive failure exhibited impairment of excretion, not only of sodium but also of water.

**Sumario Español**

Previos estudios en sujetos normales han mostrado que la compresión al cuello causa aumento en excreción de sodio en la posición sentada, pero tiene poco o ningún efecto en la posición recostada. En el presente estudio, pacientes en decompenación cardíaca no mostraron aumento en excreción de sodio después de compresión al cuello.

**REFERENCES**


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