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THE HYponATREMIC SYNDROME

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The hyponatremic syndrome is a phenomenon of recent reported prevalence, perhaps largely due to the facility with which sodium determinations can be performed by the flame photometer in the last decade. The low sodium syndrome or less specifically 'the low salt syndrome' are terms used synonymously. Related to this syndrome are the former descriptions of "hypochloremic azotemia" and also the syndrome of "water intoxication."

The term hyponatremic syndrome implies that there are clinical signs and symptoms which are rather specifically related to a low sodium concentration in the serum and extracellular fluid. In recent years, a low sodium concentration has been found frequently to accompany a wide variety of diseased states. This has led to some confusion about the syndrome, since many of the symptoms and signs may be associated with the primary diseases and not necessarily with the incidental finding of low sodium concentration. Thus the specificity of the clinical symptoms and signs needs careful analysis. If a definition of the syndrome is to be useful, one might hope to develop accurate diagnosis and treatment which would correct specifically the manifestations. Let us approach the problem of definition.

The term low salt syndrome was first popularized by Schroeder in describing some patients with cardiac failure and edema with low serum sodium concentrations. Even though the name of the syndrome seemed to put emphasis on sodium, Schroeder stated plainly that this condition could be brought about by dilution of electrolytes from a high water or low salt intake. Thus he might just as well have called it the 'salt dilution' syndrome. He postulated that this condition followed the use of mercurial diuretics in conjunction with diets low in sodium chloride and that the administration of concentrated sodium chloride was sometimes corrective.

The dictionary says that the word syndrome means 'concurrence,' and when used in medicine it is "a group of signs and symptoms that occur together and characterize a disease." The definition of the hyponatremic syndrome needs clarification in at least four important ways.

**Emphasis on regulation of water as well as sodium concentration.**

If the sodium concentration is abnormally low, the water concentration is abnormally high. Equal emphasis upon water and sodium is important because from the standpoint of etiology it is apparent that the abnormal concentration could be produced either by a loss of sodium or by a gain of water in the extracellular fluid compartment or by both. There is perhaps a tendency today among clinicians to lay the blame on the sodium ion and proceed with therapeutic manipu-

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The problem of internal intercompartmental sodium and water balance regulation.

Secondly, the terms “low” or “high” do not specify in what part of the body sodium and water are increased or decreased. It is conceivable that a low concentration, without alteration in total body content, could be produced by a shift of sodium into cells and/or shift of water out of cells. In addition, the size of the intravascular compartment or the blood volume may be important in the production of some of the circulatory abnormalities of the syndrome. We must examine the mechanisms responsible for the balance of electrolyte and water across the internal boundaries; namely, the capillary and the cell walls. This may be called the problem of internal intercompartmental balance regulation.

Importance of other electrolyte abnormalities.

Third, we have a problem of defining whether certain clinical signs and symptoms are specifically related to alterations in sodium concentration. In many of the diseases accompanied by low sodium concentration there may also be a rearrangement of potassium concentration and frequently a depletion of intracellular potassium. In some cases we might reasonably ask if abnormalities of other electrolytes, for example, of magnesium, may be responsible for some of the symptoms. Furthermore, parts of the clinical picture may be modified by the relative concentrations of chloride to sodium and by the presence of acidosis or alkalosis. All electrolyte abnormalities which accompany the hyponatremia must be examined.

External body sodium and water balance regulation.

Fourth and last, the description of any hyponatremic situation should include knowledge concerning the direction of external total body sodium and water balances. Some patients with hyponatremia can excrete administered sodium and water normally, others cannot. It is not enough to know only how much or how little sodium and water is being excreted by the kidneys or lost by other routes. We must know what proportion of the intake is being lost. We cannot determine by analysis of serum sodium concentration alone how the overall regulation of body sodium or water balance is being conducted. A few determinations of total dietary intakes and total urinary sodium and water outputs are worth more than several serum concentration determinations when managing the patient. Estimation of water balance may be more difficult than of sodium because of variation in insensible water loss; but it is nonetheless important in determining the picture.

In Table I is summarized the points so far considered important when describing any hyponatremic situation.

**TABLE I**

External Total Body Balance Regulation of Sodium and Water

a) Influence of magnitude of intake
b) Renal control of excretion
c) Insensible loss and loss by G.I. tract

TABLE II
Internal Intercompartmental Balance Regulation of Sodium and Water

1. Intravascular _______ Extravascular, Extracellular
   a) Hydrostatic capillary pressure
   b) Tissue pressure and elasticity
   c) Serum protein concentration
   d) Blood loss

2. Extracellular _______ Intracellular
   a) Cell energy and osmotic control
   b) Influence of hormones
   c) Malnutrition
   d) Acid base balance
   e) Anoxia (?)

TABLE III
Associated Electrolyte Derangements
   a) Potassium
   b) Chloride
   c) Magnesium (?)
   d) Metabolic or Respiratory acidosis or alkalosis

The factors involved in the regulation of internal intercompartmental, and of external balance are listed, as well as possible associated electrolyte abnormalities. Of course, it is much easier to list these factors in a table than to determine them in each case.

In the remaining time, I shall attempt to outline the clinical physiological pathology of the hyponatremic edematous cardiac patient, drawing a little from my own experience, and using as much information as I could find from the work of students on this subject. The factors are summarized in Tables A through E.

“HYPONATREMIC SYNDROME”
of the Edematous Cardiac Patient

A. CLINICAL FEATURES:

1. Older patient
2. Severe failure
3. Prolonged failure
4. Poor prognosis
5. Cirrhosis of liver (cardiac)
6. Inelastic tissues
7. Previous low sodium diets
8. Previous mercury diuresis
9. Malnutrition
10. Water given—forced or ad lib
B. PHYSIOLOGICAL FEATURES:

1. Low serum sodium concentration
2. Low serum chloride concentration (Metabolic alkalosis)
3. Azotemia progressive
4. Low serum proteins
5. Water antidiuresis
6. Sodium antidiuresis
7. Cell potassium decreased
8. Total nitrogen deficit
9. Cell sodium increased
10. Cell water increased
11. Abnormal thirst mechanism
12. Low blood pressure (peripheral circulatory insufficiency)

C. SYMPTOMS:

**Non-specific**
1. Weakness, Drowsiness, Lethargy, Apathy
2. Anorexia, Nausea, Occasional vomiting
3. Mental confusion, Psychosis, Coma

**Specific**
1. Thirst
2. Muscular and abdominal cramps
3. Convulsions, Twitchings

D. RESPONSE TO THERAPY:

1. Unresponsive to sodium restriction
2. Unresponsive to more digitalis
3. Aggravated by excess water intake
4. Unresponsive to mercury or
5. Mercury diuresis of potassium with chloride (low potassium toxicity occasionally)
6. Thirst with water restriction
7. Thirst with sodium administration
8. Rise in serum sodium without diuresis on administration of large doses of potassium

E. RESPONSE TO CONC. NaCl ADMINISTRATION:

1. Usually disappointing, no diuresis
2. Demonstrates large sodium space (cellular plus extracellular)
3. Mercury responsiveness (?) Increased diuresis due to excess available chloride
4. More edema (if water not restricted)

There are many other situations besides the edematous cardiac patient in which hyponatremia occurs. Each of these situations must be characterized in terms of the internal and external balance behaviour. The many situations are listed in Table F., and many have been studied extensively with experimental and clinical methods.

F. OTHER HYPONATREMIC SITUATIONS

1. Tuberculosis
2. Malnutrition
3. Hypoadrenalism
4. Post-operative overhydration
5. Hypodermoclysis overhydration
6. Heat cramps—water intoxication
7. Gastrointestinal salt loss
8. Nephrosis
9. Cirrhosis of liver
10. Hypoalbuminemias
11. Pitressin administration
12. Renal disease—salt loss
13. Miscellaneous injuries and illness
14. Experimental dialysis

CONCLUSION:

The hyponatremic syndrome of the edematous cardiac patient is caused primarily by a failure of the mechanisms for maintenance of proper proportions of sodium, chloride, potassium and water in all body fluid compartments. Its onset is accelerated by low sodium diets, mercurial diuretics and high water intake. The mechanisms failing are those that selectively control intake, namely thirst and appetite, those that control selective renal excretions, and those which preserve the internal structure of cells.

No ready solution to the problem and no therapy which is specific can be offered. The best treatment is to avoid it. Many cases are irreversible and represent in the patient with cardiac failure a final dissolution of the mechanisms for regulating body fluid structure.

BIBLIOGRAPHY


