Unrecognized Hyponatremia and Hypochloremia

By George C. Griffith, M.D. and Oscar Magidson, M.D.

With comments by: Eugene A. Stead, Jr., M.D. and Louis Leiter, M.D.

DR. GRIFFITH: “To reduce edema, to decrease, insofar as possible, the sodium content of the body”: From the time the role of sodium in the retention of fluid was first established, this has been a cardinal prescription in the management of congestive heart failure and other conditions in which there is an extracellular accumulation of fluid. This precept is rooted in the belief that fluid in the peripheral tissues superimposes an added burden upon an already handicapped heart and should, therefore, be removed as speedily as possible, by whatever means or combination of means are at hand.

The mistaken belief that congestive failure can exist only in the presence of excessive sodium retention, coupled with the conviction that salt restriction is not in itself harmful, has led to abuses of a regimen that, judiciously employed, is invaluable in the treatment of heart failure. Years ago, before we learned to remove salt from the diet efficiently, before the time of powerful mercurial diuretics, and before the development of ion-exchange resins, hyponatremia and hypochloremia were observed on but few occasions. The methods then in use were too crude to reduce levels of sodium and chlorides in the body to a dangerous level. But today our tools are more precise, and constant safeguards are necessary.

In each of the cases to be presented, the patient was from the first under the care of experienced and able physicians; yet, in each instance, symptoms of electrolyte depletion were overlooked until such time as the salt depletion syndrome had become irreversible and death was inevitable. Since Dr. Magidson is well acquainted with the cases to be discussed, I shall ask him to present the salient facts in each case and aid in the discussion. Dr. Magidson, will you please present the first case.

DR. MAGIDSON: The patient, a 51-year-old female, first was seen in November, 1952, at which time she complained of orthopnea, shortness of breath and palpitation on slightest exertion, easy fatigability, complete nervous exhaustion, and stiffness of the hands and feet. Swelling of the abdomen, legs and back had been present for about a week. The purpose of the consultation was to determine whether heart surgery would be beneficial.

History: Although cyanosis in early infancy suggested the presence of a congenital heart lesion, the patient was apparently in good health until her thirty seventh year, when she suffered a severe episode of pneumonia. Digitalis was prescribed at that time, and had been continued to the date of the examination. The patient’s gall bladder was removed in her forty fourth year. At 48, she underwent thyroidectomy, which resulted in an episode of profound shock. Although the thyroidectomy apparently had been of decided benefit, episodes of heart failure requiring recurrent hospitalization dated from that operation and necessitating discontinuance of her employment as a typist-clerk. At the time of her examination, she had just returned from the hospital.

In addition to digitoxin, the patient was taking mercaptomerin every third day. She reportedly drank but one cup of coffee a day, plus a little ginger ale and 7-Up; she had been drinking distilled water for a week. She used no tobacco or alcohol.
Physical examination: The patient, who spoke slowly as if under a sedative, was pale and icteric, with dry skin and dry hair. Veins in her neck were distended, pulsating freely, even when the patient was sitting upright. Pulse rate was rapid and the rhythm totally irregular. Blood pressure was 120/80. On percussion, the lungs showed dullness below the angle of the scapula. Many fine, moist rales were audible throughout both lung fields.

The heart was enormously enlarged, the point of maximum impulse occurring in the third and fourth left intercostal spaces, 3 cm. to the left of the mid-line. The apex beat was located in the sixth intercostal space at the midaxillary line. Rhythm was totally irregular (auricular fibrillation). The aortic first and second sounds could be heard clearly, and the pulmonic second sound was accentuated many times over the aortic. A grade II systolic murmur was audible in the tricuspid area, and a moderately soft grade II apical systolic murmur could be heard in the left midaxillary line. Venous pressure was markedly increased (estimated as 160 mm. water, minimum). The liver could be palpated 10 cm. below the costal margin. The abdomen contained free fluid. Pitting edema was present over the sacrum, and thighs and legs showed marked swelling.

Fluoroscope: The lung fields were very cloudy. The heart was greatly enlarged, with the enlargement most marked in the region of the right ventricle. The pulmonary arteries and veins were markedly prominent. No enlargement of the left atrium was observed.

Electrocardiogram: Right ventricular hypertrophy pattern and uncontrolled auricular fibrillation were reported.


Dr. Griffith suggested that the patient might be a candidate for closure of the septal defect after she had been thoroughly “dried out” and compensated, provided the defect would be confirmed by cardiac catheterization. He recommended that the patient be placed on a low sodium diet, and that digitalis be increased to a point where the ventricular rate would remain in the seventies.

The patient was admitted to the Los Angeles County Hospital on November 11, 1952, for a stay of nine days. During this time she was maintained on a low sodium diet (approximately 300 mg.) but received no mercurials. Her condition improved considerably under this regimen, and she was discharged from the hospital with the proviso that she remain on the low sodium diet and reenter the hospital at a later date for cardiac catheterization.

On November 30, 1952, the patient reentered the hospital and was again found to be in increasing congestive failure, in spite of the low sodium diet she had been following at home. The low sodium diet was continued during the 14 days of this hospitalization and, on four occasions, the patient was given intramuscular injections of 2 cc. of mercurallure sodium solution (Meruhydrin sodium solution) with good diuretic response. The patient was discharged on Dec. 12, 1952, after cardiac catheterization, with instructions to continue the low sodium diet.

The patient’s condition was diagnosed as rheumatic heart disease in the cardiac conference held on December 22. The low sodium diet was continued in an effort to clear the congestive failure. By Jan. 12, 1953, gross failure was no longer apparent, but the patient was instructed to continue the low sodium diet at home.

On Jan. 19, 1953, the patient was rehospitalized because of increasing dyspnea and orthopnea, which had been noted for three days. Signs of severe cardiac failure were present. Again, the patient was maintained on a low sodium diet, and 2 cc. Meruhydrin was administered without response although water intake was adequate. On January 23, the patient became confused and lethargic. Lethargy continued and deepened until, on January 26; the patient responded only to painful stimuli.

Blood chemistry was reported for the first time on January 26. Non-protein nitrogen was 27 mg. per 100 cc.; serum carbon dioxide, 33 mEq. per liter; serum chlorides, 50 mEq. per liter; serum sodium, 109 mEq. per liter; and serum potassium, 2.6 mEq. per liter. Urine output was only 550 ml. on this day.

Presence of the salt depletion syndrome was first suspected at this time, and 1000 cc. of 3 per cent solution of sodium chloride with 1 Gm. of potassium chloride added was administered intravenously. Later the same day, 500 cc. of a 5 per cent glucose solution containing 0.5 Gm. potassium chloride was administered. The following day, an additional 1000 cc. of the sodium chloride solution with 2 Gm. of potassium chloride was administered. The patient expired on Jan. 28, 1953.

This case encompasses most of the features usually present in instances where the salt depletion syndrome has escaped detection: 1. The patient suffered from edema of cardiac origin but was apparently free from renal pathology and was, therefore, an appropriate candidate for dehydration therapy. 2. Dehydration therapy had worked for a time for this patient, but had broken down. 3. Breakdown was ascribed not to the salt-depletion syn-
drome, but to the need for more vigorous methods for promotion of diuresis. 4. Mercu-
rial solutions were used to increase diuresis, forcing increased salt leakage.

By the time electrolyte depletion is suspected, the condition has often become irre-
versible and terminates in death. In view of the very low level recorded, potassium deple-
tion may have been a factor in this patient’s death.

Dr. Griffith: The maintenance of this patient on a low sodium diet for over three
months without the safeguard of frequent blood chemistry studies and other laboratory
determinations is insupportable. Yet, because clinicians are often unaware of the importance
of maintaining serum sodium and chlorides at levels sufficient to permit relatively unimpaired
renal function, they often neglect to request laboratory determinations, or purposely omit
these in order to spare the patients “needless expense.”

I should like to call your attention to the period of increasing dyspnea and orthopnea in this
patient, which followed a period of satisfactory diuresis, during which interval the patient was free from gross signs of cardiac
failure. Reappearance of symptoms of cardiac failure erroneously was interpreted as a sign
that more vigorous diuretic measures were needed, and the low sodium diet was supple-
mented by administration of mercurials. Recurrence of edema in the patient should have put
the physician on notice that serum sodium and chloride levels had dropped to dangerously
low levels. Sudden increase in body weight by water retention after a period of successful
diuresis should always be suspect, particularly if accompanied by an increase in serum non-
protein nitrogen or a rise in blood urea nitrogen, indication either that urinary volume is not
sufficient to carry away the nonprotein nitrogen or that renal disease is present. In either case,
dehydration therapy should have been terminated at once.

Clinical signs and symptoms should have suggested the probability of salt depletion. In spite of the excess fluid in their peripheral
 tissues, patients suffering from salt depletion complain of thirst, but they experience dryness
of the throat which makes swallowing difficult. They show signs of lassitude, apathy, weakness
and anorexia. They vomit, or complain of muscular cramps. In cases of extreme salt deple-
tion, patients often become disoriented and even psychotic.

As persons on low sodium diets are often receiving digitalis, the salt depletion syndrome
is occasionally mistaken for a toxic reaction to the digitalis. Measurement of plasma sodium
and chloride levels sometimes will distinguish between the two conditions; however, the two
conditions may coexist. If potassium depletion also is present, response to digitalis may be
intensified.

Once the condition has been recognized, correction is best accomplished by oral ad-
ministration of salt or, if vomiting precludes oral salt administration, through intravenous
salt administration. Since infusion of an isotonic solution would not raise sodium and chloride
levels of the body fluids sufficiently unless very large amounts were administered—a hazardous
procedure in the patient with congestive failure—a hypertonic solution is used. A 3 or 5 per
cent concentration of sodium chloride, administered slowly, is usually satisfactory; its
administration causes no significant or constant elevation of venous pressure. Unless the
condition has persisted for such a time that it has become irreversible and death is imminent,
as in the cases here presented, clinical improvement parallels closely the restoration of
electrolyte balance.

Potassium is administered to these patients for two purposes: (1) to restore potassium
levels; and (2) because alkalosis is refractory to sodium chloride therapy if potassium defficiency
continues. Infusions of saline or glucose without the addition of potassium would increase
the potassium loss, as the kidneys preferentially excrete potassium rather than sodium. Dr.
Magidson, will you please present the next case.

Dr. Magidson: This patient, a 39-year-old male formerly employed as a carpenter, was
transferred to the Los Angeles County Hospital on May 9, 1950, from a nearby private
hospital, where he had been under treatment for heart disease and subacute bacterial endo-
carditis for a period of seven weeks. The state
of the electrolytes and the treatment are shown graphically in figure 1.

**History:** The patient suffered penile trauma at the age of three, and symptoms of penile obstruction were noted in his early teens. Otherwise, he is alleged to have been in good health until April, 1949, when he was hospitalized for a week because of the development of jaundice, ascites and edema of the ankles. Three months later, the patient consulted a urologist because of pyuria observed since discharge from the hospital. Retrograde studies disclosed a urethral stricture, which subsequently was dilated satisfactorily. In October, 1949, the patient was hospitalized because of a urinary tract infection for a 30 day period. On admission, his temperature registered 104 F. During a short hospital stay in January, 1950, the patient’s condition was diagnosed as heart disease with ascites and edema of the ankles, accompanied by subacute bacterial endocarditis. A recurrence of subacute bacterial endocarditis in April, 1950, resulted in the seven-week period of hospitalization which was referred to at the opening of this case history, after which he was transferred to the Los Angeles County Hospital.

**Physical examination:** Upon admission, the patient was noted to be dyspneic and ascitic, with marked edema of the legs. Blood pressure was 145/40 mm. Hg. Pulse rate was 80; respiratory rate, 20. Crepitant rales were heard over both lung bases. The heart was moderately enlarged, with sinus rhythm. Evidence of free aortic incompetence was noted. The abdomen was moderately distended from ascites. The liver was not felt to be enlarged. Four plus pitting edema of the legs and abdominal wall was present. Venous pressure was estimated to be 300 mm. of water. Using Decholin, the arm to tongue circulation time was found to be 30 seconds.

**Laboratory:** Hemoglobin was 17.9 Gm. per 100 cc.; white blood cells, 14,800, of which 86 per cent were polymorphonuclear leukocytes. A trace of albumin was present in the urine, together with numerous pus cells and red blood cells.

Two days following his admission to the Los Angeles County Hospital, the patient was reported to be “paranoid and psychotic.” He was put on a low sodium diet, and aminophylline and 2 cc. of Mercuhydrin were administered intravenously. The following day, 3500 ml. of sanguineous fluid was removed from the abdomen by paracentesis, and another 8 cc. aminophylline and 2 cc. Mercuhydrin were administered. On May 13, the patient was started on a rice diet, on which he was maintained for the next 41 days. During this period, he drank 1 to 1.5 liters of distilled water daily. The diuretic effect of the rice diet was reinforced on the fifteenth, seventeenth, nineteenth and twenty fourth day of May by intravenous injections of aminophylline, 10 cc., and Mercuhydrin, 2 cc. On May 30, 600 ml. of sanguineous fluid was removed by paracentesis from the left side of the chest.

The day following hospitalization at the Los Angeles County Hospital, blood chemistry was as follows: serum chlorides (as sodium chloride), 89.6 mEq. per liter; serum potassium, 4.1 mEq. per liter; and nonprotein nitrogen, 48 mg. per 100 cc. Carbon dioxide combining power was 21.8 mEq. per liter. Icterus index was 26 units.

Serum sodium was not measured separately until June 13, at which time it was 112 mEq. per liter. By this date, nonprotein nitrogen had climbed to 91 mg. per 100 cc., and serum chloride values had slipped to 77.2 mEq. per liter. Potassium concentration and carbon dioxide combining power remained unchanged. The patient refused food. Cerebral depression was marked, and hospital attendants commented on the patient’s lethargy. A 3 per cent solution of sodium chloride (500 ml.) was administered intravenously, raising sodium and chloride values slightly but resulting in no clinical improvement.

Corticotropin (ACTH), 15 mg., was administered on each of four successive days, starting with June 20. Although the patient seemed slightly more alert on the first day of this medication, his condition deteriorated rapidly, and he became icteric (icteric index had climbed to 58 units on June 22). On June 23, the rice diet was discontinued, and he was returned to a low sodium diet. Death occurred on June 30, 1950.

**Dr. Griffith:** In the case just presented, dangerously low levels of serum sodium and chlorides were ignored, and drastic measures were employed in an attempt to rid the patient of excess extracellular fluid: low sodium diet, the even more severe rice diet for a period of 41 days, and the administration of aminophylline.
and mercurial diuretics on six occasions. Sodium and chloride levels were depleted further by abdominal paracentesis on May 12 and thoracic paracentesis on May 30. This case serves to re-emphasize a point I have always made to my students: edema in the peripheral tissues is not a danger to the patient which calls for immediate, heroic measures. The "drying out" process should occupy an extended period, rather than hazard the safety of the patient. If vigorous measures are employed, a close watch should be kept for clinical signs and symptoms of distress, and for laboratory values which indicate that sodium and chloride levels have reached perilously low levels.

**DR. MAGIDSON:** I should like to raise a question or two about the case just presented. Do we keep a close watch for **perilously low levels of sodium and chloride**, or can we adopt a policy which forestalls this finding? Obviously, the object of vigilance in these cases is to prevent occurrence of these levels. When the patient is on a low sodium intake and has had mercurials, if he fails to respond adequately to mercurials and urinary output falls below the fluid intake, and if symptoms begin to occur, we must immediately suspect salt depletion and measure the electrolytes and blood urea nitrogen. I am beginning to feel that failure to take note of a falling urine output in the face of deterioration leads to most trouble.

It is generally acknowledged that low sodium and chloride levels result ultimately in impaired renal function and the events we are discussing. Questions I should like to ask are these: Can electrolyte levels be used to **predict** the events in any one case? How can we assess the flexibility of the kidneys from case to case? In two patients with similarly low electrolytes and similar treatment, one keeps a good urine volume and does well and the other goes into the low salt syndrome. Do you not agree that this may be the case? Another question: Is it the individual with some renal damage who gets into trouble more readily? In the occasional case, may he not manifest a "better" electrolyte pattern than the patient with relatively uninjured kidneys?

**DR. GRIFFITH:** These are certainly points to be kept in mind and I agree as to their significance. May we have the third case, please, Dr. Magidson.

**DR. MAGIDSON:** As a third instance of the unsuspected presence of the low salt syndrome, we present the case of a 64-year-old male, in a state of cardiac decompensation from prolonged hypertension, who was already in an irreversible condition of electrolyte depletion on April 19, 1953, when he was admitted to Good Samaritan Hospital with the complaint of shortness of breath, cough, weakness, and swelling of the legs.

**History:** The patient was apparently well until December of 1951, when he sought medical advice because of ringing in the ears and occasional frontal headaches. Examination at that time revealed an enlarged heart and elevated blood pressure. The physician in attendance advised weight loss and prescribed a diet low in cholesterol.

In August, 1952, shortness of breath again became evident, progressing to orthopnea and accompanied by episodes of acute pulmonary edema. The patient complained of pain in the epigastrium and his legs became swollen. In the next few months, the patient's condition remained stationary; blood pressure averaged 190/130, save for a temporary fall to 120/80 following acute illness. The pulse remained elevated, ranging between 100 and 125, except for a brief period after an acute illness. The heart was observed to be enlarged, with gallop rhythm. A grade II apical systolic murmur was present, and many rales could be heard at the bases of both lungs. A pleural effusion on the right side was drained. Edema of the legs was 2 plus. Treatment included the administration of oxygen and ouabain and rest in bed. The patient was maintained on a low sodium diet and given mercurial diuretics in an effort to clear the failure. Oral medication with hexamethonium drugs in an attempt to maintain blood pressure at a lower level was unsuccessful.

Studies done at an institution in Mexico City revealed elevated nonprotein nitrogen, and blood urea nitrogen of 53 to 63 mg. per 100 cc. One to 2 mg. of blood creatinine was present per 100 cc. A blood Wasserman test was negative. A 24-hour sample of the patient's urine contained 2 Gm. of albumin and many hyalin casts. An electrocardiographic tracing done at this time disclosed an incomplete left bundle branch block, with evidence of left ventricular hypertrophy and suggestion of an old anteroseptal myocardial infarction. Hexamethonium drugs were tried again without success. The patient was considered a poor candidate for sympathectomy.

The patient was brought from Mexico to Los Angeles in the hope that he might be benefited by the lower altitude. The findings, treatment and course follow.
Physical examination: The patient was extremely alert. His color was good. His eyes showed grade II retinopathy. His nose and throat were healthy. Respiratory rate was 32; pulse rate, 110. Blood pressure was 150/120. Many rales were present at the bases of both lungs. His heart showed grade III enlargement with a definite gallop rhythm. A grade II to grade III apical systolic murmur was present. The liver was slightly tender, and could be palpated 8 cm. below the costal margin. The spleen was just palpable. Two plus pitting edema was present. The bladder was empty.

X-ray examination: Marked enlargement of the left ventricle could be observed. A small amount of fluid was present at the bases of both lungs.

Electrocardiogram: Tracing showed a complete left bundle branch block, with probability of an old anteroseptal myocardial infarction.

Laboratory: Blood urea nitrogen was 21 mg. per 100 cc.; blood creatinine, 1.5 mg. per 100 cc.; serum sodium, 119 mEq. per liter; and serum potassium, 5.1 mEq. per liter. Serum chloride determination was not requested.

The day following his admission to the Good Samaritan Hospital, the patient was noted to be cyanotic, and a uremic odor was present on his breath. Weight was 170 pounds, and blood pressure was 160/120 in both arms. Pleural effusion was present on the right side, and rales were audible over the left lung base.

A solution of 5 per cent sodium chloride (500 ml.) was administered intravenously and then a solution containing 5 per cent glucose (500 ml.). Injection of a mercurial diuretic followed. Only a 2 per cent weight loss resulted, therefore administration of additional saline solution preceded further attempts to promote diuresis. Serum sodium concentration was 122 mEq. per liter at this time, and nonprotein nitrogen measured 79 mg. per 100 cc. The following day, the patient's heart tones were good and his lungs were dry. Blood pressure was 140/80 at this time. Additional saline was administered cautiously, but the patient's condition worsened, edema increased and, on May 30, 1953 convulsive twitching was noted. The patient died on the following day.

Dr. Griffith: Long-time maintenance on a salt-poor diet in an attempt to reduce hypertension and to clear congestive failure secondary to the hypertension had disturbed the already precarious electrolyte balance in this patient, and he had passed from the therapeutic to the toxic stage of salt-restriction therapy some time before. At the time he was brought to the Good Samaritan Hospital for treatment, the condition was no longer remediable.

Comments

Dr. Magidson: The so-called low salt syndrome should be suspected whenever patients who have been maintained on a low sodium diet and/or have responded to mercurial diuretics demonstrate a marked depletion of urinary volume over a period of several days, together with a rapid, progressive gain in weight. Clinical symptoms include thirst which cannot be slaked by water, lethargy, apathy, weakness, drowsiness or restlessness, nausea and mental confusion. The Schales and Schales test for sodium chloride in the urine usually will show little or no precipitate with silver nitrate unless there has been pre-existing renal tubular damage. Anorexia may operate to limit food intake, and vomiting to further depress electrolyte concentration. Decline in plasma chloride values will usually precede fall in plasma sodium concentrations. Elevation of blood urea nitrogen and serum nonprotein nitrogen occurs under these conditions because urinary volume is not adequate to carry away the nonprotein nitrogen.

Dr. Griffith: Symptoms of salt depletion result from either sodium depletion, or depletion of serum chlorides; imbalance between the two ions may contribute to acidosis or alkalosis. Under certain conditions, the salt depletion syndrome may occur even if dietary intake of salt is not restricted, i.e., if profuse vomiting occurs, in the presence of excessive diarrhea, if an intestinal fistula is present, or if continuous suction drainage has been established. Any of these conditions results in disproportionate loss of sodium and chloride ions, which may disturb electrolyte balance and initiate the low salt syndrome. Continued loss of gastric juice, for example, will result in hypochloremic alkalosis, which may be accompanied by a rise in the plasma bicarbonate level as a result of the combination of the freed sodium with carbonic acid. On the other hand, continued loss of fluid from the gut distal to the pylorus results in a proportionately greater loss of sodium ions, with resulting acidosis and decrease of plasma bicarbonate. Vomiting or diarrhea during salt restriction therapy will, of course, intensify electrolyte loss. Administration of large amounts of salt free liquid, either orally or
parenterally, also lowers electrolyte concentrations in the body fluids.

Whenever dietary salt is restricted as a therapeutic measure, and whenever conditions exist which operate to derange electrolyte balance within the body, a close check must be kept on electrolyte levels at all times. In addition, levels of nonprotein nitrogen or blood urea nitrogen should be checked for indications of severe dehydration. In hyponatremia blood urea nitrogen may reach 60 to 90 mg. per 100 cc., or even higher.

If salt replacement is indicated, it is important that sodium chloride be given cautiously, in divided doses; in a few cases, the administration of one-third to one-half of the dosage calculated to restore sodium and chloride levels to normal has proved sufficient. The amount of sodium needed is calculated by the following formula:

Patient's weight in kilograms \( \times \frac{100}{106} \times (\text{normal mEq} - \text{patient's mEq}) \), in which body water is considered to be 60 per cent of body weight.

Dr. Magidson: I should like to have this conference on hyponatremia and hypochloremia come to a close without some mention of the abuses of dietary restriction of sodium chloride that occur all too frequently. It is important to remember that dietary restriction of sodium is not justified in every form of heart disease. If the heart disease in the patient is not associated with sufficient failure to produce renal retention of salt and water, restriction of the amount of salt in the diet not only is of no value, but may be detrimental to the patient from both psychologic and nutritive standpoints. We have all seen patients who suffer from valvular heart disease uncomplicated by congestive heart failure—rheumatic heart disease or subacute bacterial endocarditis, for example—who have been placed on low sodium diets that are valueless from a physiologic point of view and serve only to focus the patient's attention on his cardiac disability and thus increase his tension and anxiety.

Dr. Griffith: Edema is not invariably due to congestive failure. When edema is present, it is important to determine whether the accumulation of fluid is in fact due to cardiac decompensation or whether it is the result of peripheral venous or lymphatic disease unaccompanied by cardiac decompensation.

Dr. Magidson: It has been our experience that even in the presence of congestive heart failure, diets prescribed are often too low in salt content. Although diets providing 0.5 Gm. of sodium per day or less are justified in the initial treatment of moderately severe to severe congestive failure, the correction of aggravating factors such as hyperthyroidism, inflammatory disease processes or obesity will usually permit some relaxation of dietary salt restriction.

Dr. Griffith: We urge frequent assessment of cardiac status to determine whether heart action has improved to a point where less rigid salt restriction may be observed. Although persons with organic disease who have suffered congestive failure with edema rarely can return to an entirely unrestricted diet, cardiac function may be restored sufficiently that only mild dietary restriction is necessary.

I think we have covered most of the points we wish to make here today. I should like to leave you with a statement I made earlier in this discussion: Edema in the peripheral tissues is not a danger to the patient which calls for immediate removal by drastic, even heroic measures. The "drying out" process should occupy an extended period of time, rather than hazard the safety of the patient and only after adequate rest and digitalization have been evaluated.

Discussion by Eugene A. Stead, Jr., M.D.

Salt restriction never causes harm when congestive heart failure is the central problem, if it is not combined with mercurial diuretics. If one has primarily renal failure, with heart failure being more or less incidental, salt restriction may lead to difficulty.

In many chronic illnesses, low sodium states develop without salt restriction. If the chronic illness is one like congestive failure, or cirrhosis, edema will be present. In brain tumors or malignant hypertension, edema will be absent. These disturbances in electrolyte concentration are in some way related to the severity of illness,
and they will develop in most patients with heart failure if they die slowly. Adding salt to the diet results in an expansion of extracellular fluid volumes with no change in electrolyte concentration but with progressive edema. These conditions are not comparable to salt-losing nephritics as they will not excrete excess amounts of salt on a low salt diet.

Although I agree that the patients reported on were treated without proper laboratory controls, the fact that none of them responded to the administration of salt weakens the argument that they died from salt depletion. As two of the patients were jaundiced, my guess would be that pulmonary infarctions were also important.

I do not agree with the concept that patients who recover from congestive failure are necessarily best treated by liberalizing the salt intake. In many hypertensive patients, keeping the sodium intake below 200 mg. will cause a considerable reduction in blood pressure and a great decrease in heart size. Appreciable reduction in heart size may also occur in the absence of hypertension. A decrease in heart size is of great advantage to the heart mechanically. Adding salt to the diet of a patient with a large heart is undesirable from the cardiovascular side: it is always a compromise to gain other things.

Discussion by Louis Letter, M.D.

The three cases described illustrate the necessity of differentiating the hyponatremic state due to primary sodium loss or depletion, from the dilution type of hyponatremia which follows excessive retention of water in severe heart failure. The depletion type of hyponatremia should be treated with concentrated salt solution for obvious reasons. The dilution type of hyponatremia in cardiac patients seems to represent excessive antidiuretic hormone activity and abnormal behavior of volume regulating centers in response to increased congestive failure. In this situation the urgent need is not hypertonic saline but improved myocardial function, to promote the excretion of the excess water. This happens without loss of sodium so that the serum level rises by concentration of the extracellular fluid. The daily injection of long-acting Pitressin in patients in controlled heart failure can, by inducing primary water retention and aggravating the congestive state, closely mimic the natural form of dilution hyponatremia. Here, too, addition of sodium chloride is of no therapeutic value. Withholding Pitressin leads to prompt water diuresis and rise in serum sodium level to the control values.

Since the dilution type of hyponatremia is a manifestation of severe heart failure and not of sodium deficiency, its treatment must be directed toward increasing myocardial efficiency, whether by adequate digitalization, restoration of tissue potassium content, control of intercurrent infection, relief of anoxia or other measures. The administration of hypertonic saline solution can be very harmful.

In a given case of hyponatremia, even with extensive laboratory data available, it may be problematic whether the patient is suffering from sodium depletion or sodium dilution, or both. Close clinical observation of the sequence of events may help in this differentiation. Thus, a low serum sodium level in a cardiac patient who has lost weight and mobilized edema rapidly is probably due to depletion. When to this a change in psyche is added and signs of peripheral collapse and gastrointestinal symptoms, the diagnosis becomes quite certain. On the other hand, a low serum sodium in an edematous cardiac who is developing signs of increasing congestive failure and gaining weight, while responding poorly to mercurials, is strongly suggestive of dilution hyponatremia. Careful check of the fluid balance and search for known factors in the aggravation of heart failure will help in the diagnosis. In case of doubt or where the patient presents a mixture of the two syndromes, slow intravenous administration of 5 per cent saline solution in divided doses to elevate the extracellular sodium level by only 5 to 10 mEq. per liter, may significantly improve the condition of a depleted patient and not seriously harm the patient with dilution hyponatremia. Fluid intake must be sharply restricted on the day of the infusion in order to obtain the maximum increase in serum sodium concentration. A
mercurial diuretic should not be administered at this time unless there is pulmonary edema.

Confusion in diagnosis and treatment of a hyponatremic state is compounded by simultaneous hypokalemia and hypochloremic alkalosis, as was likely in case 1. This patient was given a large amount of sodium chloride and very little potassium. In severe heart failure effective digitalization may be impossible without appropriate restoration of tissue potassium depleted by poor intake, mercurial or other diuresis, and emaciation. Attention to potassium deficits may be more important therapeutically than attempts to elevate the serum sodium level. One must consider the physiological balance between these two cations.

Because of the unusual severity of sodium restriction on the Kempner rice diet, no patient should be placed on this regimen without a preliminary determination of the serum electrolyte pattern. In case 2, one may estimate from the sum of serum bicarbonate and chloride concentrations, 111 mEq. per liter, that the serum sodium level was probably as low as 125 mEq. per liter even before paracentesis, the use of the rice diet and mercurials. Under these circumstances, the subsequent downhill course is not surprising. The 250 mEq. of sodium given when the serum sodium level was 112 mEq. per liter would be inadequate in an individual of average weight.

The administration of hypertonic saline in case 3 in an attempt to potentiate mercurial diuresis involves the mistaken notion that a successful diuresis will result in a net negative sodium balance for the period. This is not the case because the increased urinary excretion of sodium never exceeds the amount infused and usually falls far short of it. Often, the patient ends up with more edema than before the infusion because of a limited diuretic response to the mercurial and failure to restrict fluid intake after the administration of hypertonic saline. Although the data available for case 3 are incomplete, it would seem that the patient was retaining water and diluting his extracellular sodium. It is also likely that he was given an excessive amount of hypertonic salt solution for the wrong reasons.
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