Electrolyte Disturbance in Acute Alcoholism

With Particular Reference to Magnesium

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A STUDY of serum electrolyte levels in the alcoholic was undertaken in view of the reports from Flink and his associates[1,2] of the frequent occurrence of low serum magnesium levels in the chronic alcoholic with delirium tremens, with the improvement of the tremor, handwriting, and hallucinations in these patients occurring with magnesium therapy. As other electrolyte changes were not documented except for mention of occasional hypopotassemia and hypophosphatemia, and absence of hypocalcemia, it appeared important to study the serum electrolyte pattern.

MATERIAL AND METHODS

Forty-four patients who entered the hospital with acute alcoholism, the majority with hallucinations and severe tremor, were the subjects. On entry 17 patients had hallucinations, 39 had tremor, 2 had convulsions, and 5 were violent, requiring full leather restraints. While an adequate history was not obtained on all patients due to the mental confusion it was ascertained that 21 of the patients were chronic alcoholics, 2 were periodic drinkers, and 5 were acute alcoholics. Only one patient had ascites. The duration of alcoholism was not determined in 16 of the patients.

On entry to the hospital or soon thereafter, blood was drawn for determination of serum magnesium, sodium, potassium, calcium, chloride, phosphate, and serum water levels. Samples were obtained in 30 of the patients prior to any therapy. One or more determinations of serum electrolytes were made in 28 of the patients.

The serum magnesium level was determined by the method of Simonsen.[3§] The other serum electrolytes were determined by methods previously reported from this laboratory.[4] Serum water was determined by the gravimetric method, after drying to constant weight.

Handwriting samples were obtained on entry in all patients who were not violent and who were not restrained (26 patients). Several samples of handwriting were taken during hospitalization in a small group of patients. A clinical estimate of mental state and degree of tremor was also made.

Five of the patients received magnesium therapy. Only serum magnesium levels obtained before magnesium therapy are included in this paper, as the amounts and routes of magnesium therapy were too variable for comment on effect.

The majority of the patients received intensive therapy consisting of hydration, intravenous glucose, vitamins, and Thorazine and were discharged within 24 hours. A few patients required several days of hospitalization.

RESULTS

Table I summarizes the entry serum electrolyte levels in 30 patients, expressed as percentage of the determinations which were low, normal, or elevated. Thirty-six and six-tenths per cent of the serum sodium and 27 per cent of the serum chloride levels were elevated on entry, in contrast to infrequent elevation of the other serum electrolyte levels. Sixty

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§ Flink used both the method of Simonsen and the Titan Yellow method, so his results can be readily compared to ours.

TABLE I

Serum Electrolyte Levels Before Therapy (30 Patients)

	Low	Normal	High
Sodium	6.7	56.6	36.6
Potassium	30	70	—
Magnesium	60	30	10
Calcium	20	80	—
Chlorine	6.7	6.7	26.7
Phosphate[a]	13.6	72.7	13.6

Figures are per cent of total number of determinations.

[a] Twenty-two patients.

per cent of the serum magnesium determinations, 30 per cent of the serum potassium, 20 per cent of the serum calcium, and 13.6 per cent of the serum phosphate levels were below the normal range, while only 6.7 per cent of the serum sodium and chloride levels were below normal.*

All the entry serum water levels were essentially normal except for 4 patients who had a slight decrease in level (88.8–89.5). Severe dehydration was noted clinically in only 4 patients. All of these patients had either normal or elevated serum magnesium levels.

The electrolyte values on days 1–4 in the patients followed serially are summarized in Table II. Following hydration and sedation during the first 24 hours, 90 per cent of the serum chloride levels, 80 per cent of the serum calcium, and 70 per cent of the sodium levels were in the normal range, compared to only 20 per cent of the serum magnesium and 50 per cent of the serum potassium levels. By day 3, in the 8 patients studied, three-fourths or more of the sodium, potassium, calcium, chloride, and phosphate levels were normal, compared to only 50 per cent of the serum magnesium levels.

Handwriting samples were taken on entry in 26 patients, in 11 patients during the first 24 hours in the hospital, in 6 patients on the second hospital day, and in 6 patients after the second day. The handwriting samples were graded into 4 categories (normal, moderate tremor, marked tremor, complete illegibility) by one of the authors who did not know the serum magnesium levels. The classification of the handwriting was then correlated with the serum magnesium level (Table III and Table IV).

On entry, except for the patients whose handwriting was completely illegible and who all had low serum magnesium levels, there appeared to be little general correlation between serum magnesium levels and handwriting. Approximately 50 per cent of the serum magnesium levels were low or normal with either normal or tremulous handwriting. When all the handwriting samples from entry to hospital day 5 were correlated with the serum magnesium levels (see Table IV) the same general lack of correlation was also found, except for those with illegible handwriting.

Illustrations of handwriting correlated with the serum magnesium level are shown in Figures 1 and 2. Figure 1 shows marked improvement in handwriting in 48 hours, with no improvement in the serum magnesium level

* Values reported previously in abstract form[5] gave slightly different percentages, as a few patients were included in the entry group who had received a small amount of intravenous fluid before blood was drawn for the serum electrolyte determinations.

TABLE II

Serum Electrolytes After Entry

	Day 1 (10 patients)			Day 2 (7 patients)			Day 3 (8 patients)			Day 4 (3 patients)		
	Low	Normal	High	Low	Normal	High	Low	Normal	High	Low	Normal	High
Sodium	20	70	10	14	57	29	13	74	13	—	100	—
Potassium	50	50	—	29	71	—	25	75	—	33	67	—
Magnesium	70	20	10	43	57	—	50	50	—	33	67	—
Calcium	20	80	—	43	57	—	13	87	—	33	67	—
Chloride	—	90[a]	10	—	100[c]	—	13	74	13	—	67	33
Phosphate	—	88[b]	12	—	100	—	17[d]	83	—	—	100	—

Figures are per cent of total number of determinations.

[a] 9 patients; [b] 8 patients; [c] 6 patients; [d] 6 patients.

TABLE III

Entry Handwriting and Serum Magnesium Levels (26 Patients)

Character of handwriting	No. patients	% Normal or slightly elevated (1.53–1.84+ meq/l)	% Slightly low (1.26–1.52 meq/l)	% Low (1–1.25 meq/l)	% Very low (under 1 meq/l)
Normal	6	50	17	33	—
Moderate tremor	10	50	20	20	10
Marked tremor	7	43	28.5	28.5	—
Illegible	3	—	—	67	33

(1.01 meq/l on entry and 0.75 meq/l at 48 hours). Figure 2 shows a fairly legible but tremulous handwriting on entry in another patient. At this time the serum magnesium

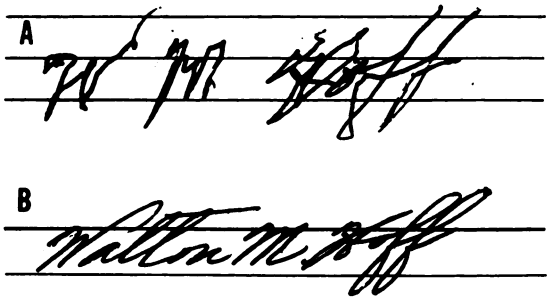

Fig. 1. Improvement in handwriting with no improvement in serum magnesium level. *A*, handwriting at entry; serum magnesium 1.01 meq/l. *B*, handwriting on day 3; serum magnesium 0.75 meq/l. Patient: Walton M. Hoff.

level was 1.42 meq/l. Ten hours later, following hydration, the serum magnesium level was 1.24 meq/l, and the handwriting at this time had become completely illegible.

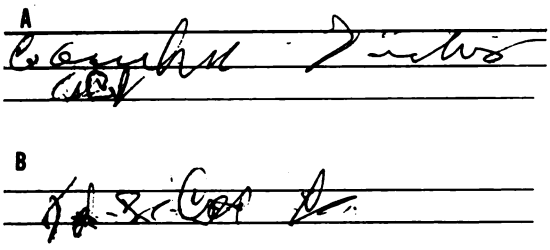

Fig. 2. Change in handwriting (decrease in legibility) with only slight change in serum magnesium level. *A*, handwriting at entry; serum magnesium 1.42 meq/l. *B*, handwriting 10 hours later; serum magnesium 1.24 meq/l. Patient: Concha Fisher.

TABLE IV

Correlation of All Handwriting Samples Obtained and Serum Magnesium Levels (49 Patients[a])

Character of handwriting	No. patients	% Normal or slightly elevated (1.53–1.84+ meq/l)	% Slightly low (1.26–1.52 meq/l)	% Low (1–1.25 meq/l)	% Very low (under 1 meq/l)
Normal	14	43	29	14	14
Moderate tremor	23	43	26	22	9
Marked tremor	8	50	25	25	-
Illegible	4	25	—	50	25

[a] Same patient may be listed more than once if serial magnesium levels were determined.

TABLE V

Correlation of Hallucinations and Tremor with Serum Magnesium Level[a]

Clinical state	No. patients	% Normal or slightly elevated (1.53–1.84+ meq/l)	% Low (1.0–1.52 meq/l)	% Very low (under 1 meq/l)
Hallucinations	16	63	31	6
No hallucinations	48	48	44	8
Tremor	57	46	47	7
No tremor	12	67	25	8

[a] Same patient may be listed more than once if serial magnesium levels were determined.

Little correlation is seen between the serum magnesium level and the presence or absence of hallucinations (Table V). A few more patients with tremor, however, had low serum magnesium levels.

DISCUSSION

Our results confirm the important finding of Flink[1,2] of the occurrence of low serum magnesium levels in the chronic alcoholic with delirium tremens. We were unable to correlate clinical symptomatology with the serum magnesium level in most instances. This was true of some patients in Flink's series also. The improvement in handwriting and tremor which he noted after magnesium therapy occurred in many of our patients who received only

hydration, intravenous glucose, vitamins, and sedation, and no magnesium. The situation may be analogous to that occurring with potassium, where serum levels do not always reflect cellular levels and where the symptoms and signs of potassium deficiency cannot always be correlated with the serum level.

The relationship of serum magnesium changes to other electrolyte changes in the alcoholic, a problem which had not previously been adequately studied, has been determined. Our studies demonstrate the important finding that low serum magnesium levels occur more frequently and last longer than other electrolyte changes.

The occurrence of changes in serum electrolyte levels other than magnesium in the acute alcoholic, however, we feel is significant. Previous studies[6,7] have documented the occurrence of potassium deficiency in the chronic alcoholic with cirrhosis. This change is confirmed by our studies. The occurrence of hypernatremia and hyperchloremia in the acute alcoholic has not to our knowledge been mentioned previously. Further, hypocalcemia as a relatively common facet of the electrolyte problem in the acute alcoholic has not been stressed.

The mechanisms responsible for the changes in the serum electrolytes found in the alcoholic require further study. Several suggestions for these changes might be made, however. The drop in the level of the primarily intracellular electrolytes (potassium, magnesium, phosphate) might occur due to poor food intake, plus poor gastrointestinal absorption, and in some instances loss by vomiting and diarrhea. The effect of starvation on serum electrolyte levels is complicated, and no absolute pattern has been found.[8,9] An important factor might be accelerated urinary loss of electrolytes. While a completely normal man may conserve magnesium rigorously on a virtually magnesium-free intake,[10,11] it takes several days for renal conservation to become fully operative. The same situation occurs with potassium, where renal conservation in the normal man may also become adequate after several days.[12] It is well known, however, that starvation and dehydration lead to accelerated loss of potassium over nitrogen in the urine.[13] The same situation may be true as far as magnesium is concerned. The alcoholic usually has serious nutritional problems.

Poor gastrointestinal absorption secondary to vitamin deficiency and such pathologic changes as alcoholic gastritis might well explain alterations of serum calcium and phosphate. Semistarvation also has been shown to cause a fall in serum calcium levels.[9] Alterations of pancreatic function secondary to pancreatitis might also be a factor but was not present in our series, except in one patient who had acute pancreatitis.

The occurrence of hypernatremia and hyperchloremia in about one-third of the patients is of great interest, and suggests disruption of the normal mechanisms which guard the osmolarity of the body fluids. It has been shown that water diuresis, with loss of more water than electrolyte, with decrease in urine osmolarity, occurs in experimentally induced mild acute alcoholism via a probable effect on antidiuretic hormone.[14] This factor could explain the occurrence of the hypernatremia and hyperchloremia.

As magnesium is vitally concerned with enzyme systems in the liver and skeletal and heart muscle which are necessary for carbohydrate and protein metabolism,[15] the depletion of body magnesium which is probably present when the serum levels are low acquires great importance. Further, magnesium deficiency, particularly in the young animal of many species, has been shown to cause necrosis and hemorrhage in the brain and heart, alterations in liver and kidney (nephrocalcinosis), and poor growth.[16–23]

Potassium deficits also are associated with histologic changes (necrosis) in skeletal and cardiac muscle.[24] Chronic severe potassium deficiency may also lead to distal tubular epithelial change, with resultant polyuria and urinary potassium loss.[25]

These facts suggest the importance of correction of the electrolyte alterations which occur in the alcoholic, by adequate magnesium, calcium, and potassium therapy, and hydration for states of hypernatremia and hyperchloremia.

SUMMARY

Serum electrolyte determinations were made in 30 patients with acute, severe alcoholism before any therapy was given. Sixty per cent of the serum magnesium levels, 30 per cent of the serum potassium levels, and 20 per cent of the serum calcium levels were below the normal range. Approximately one-third of the patients showed hypernatremia and hyperchloremia.

There was little correlation in most patients between serum magnesium levels and such clinical findings as hallucinations, tremor, and tremulous handwriting. All but one of the patients with completely illegible handwriting had low serum magnesium levels.

Possible mechanisms suggested for the alterations in the serum electrolytes included poor food and electrolyte intake, poor gastrointestinal absorption, and accelerated urinary loss of electrolytes due to effects of starvation and dehydration. Water diuresis, a demonstrated effect of alcohol, might explain the occurrence of hypernatremia and hyperchloremia in some patients.

The need for adequate fluid and electrolyte therapy in the treatment of the acute alcoholic was stressed in view of the deleterious effects of altered body electrolytes.

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Water, taken in moderation, cannot hurt anybody.—MARK TWAIN