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Electrolyte Balance of the Blood in Ménière's Disease.

JOHN H. TALBOTT, MADELAINE R. BROWN, FREDERICK S. COOMBS AND WILLIAM V. CONSOLAZIO. (Introduced by H. Jackson, Jr.)

From the Medical Clinic, Massachusetts General Hospital, and the Fatigue Laboratory, Harvard University.

Mygind and Dederding[1] associated the pathogenesis of Ménière's disease with a disturbance of water and salt equilibria. From their investigations they concluded that a dehydrating and an acid-forming diet was indicated in the treatment of this condition. Two years later Furstenberg, Lashmet and Lathrop[2] extended these observations, showed evidence which indicated that sodium was the offending ion and recommended a regime with a low sodium diet and intermittent periods of ammonium chloride ingestion. This regime, where employed, has been successful,[3, 4] but we believe from our data that a lowering of the sodium content of the body fluids is not its mode of action.

In this communication blood studies from 14 patients with Ménière's disease are presented. The diagnosis was confirmed by one or more clinicians and satisfied most of the criteria discussed by Crowe.[5] In all of the patients the concentration of serum total base, sodium and potassium was determined and in 4 the concentration of serum protein and hydrogen ion also. Eight patients were seen during periods of acute symptoms and bloods were taken at such times. In 6 patients, bloods were taken after the institution of a low sodium regime or during periods of freedom from symptoms after admission to the hospital. The results are given in Table I. It is observed that the concentrations of serum sodium and total fixed base are within the range for normals. The average concentration of serum sodium is slightly less in the patients with acute symptoms than in those without acute symptoms. The concentration of potassium is somewhat higher in the patients with acute symptoms.

1 Mygind, S. H., and Dederding, D., *Acta Otolaryngologia*, 1932, **17**, 424.

2 Furstenberg, A. C., Lashmet, F. H., and Lathrop, F., *Ann. Oto. Rhin. and Laryng.*, 1934, **43**, 1035.

3 Brown, M. R., *J. A. M. A.*, 1937, **108**, 1158.

4 Furstenberg, A. C., *Transactions of the Twenty-fourth Annual Meeting of the Pacific Coast Ophthalmological Society*, 1936, **21**, 150.

5 Crowe, S. J., *Medicine*, 1938, **17**, 1.

TABLE I.

	Total fixed base	Sodium	Potassium
Concentrations are expressed in m.eq. per liter.			
Electrolytes of serum taken from 8 patients during acute symptoms.			
Max.	155.8	141.5	15.8
Min.	151.0	131.5	3.5
Aver.	153.6	138.3	6.7
Electrolytes of serum taken from 6 patients after institution of low sodium regime.			
Max.	155.8	144.8	4.7
Min.	148.0	135.0	4.4
Aver.	153.2	140.0	4.5
Range for normals as determined in this laboratory			
Max.	155	142	6
Min.	150	138	4

Four patients during symptom-free periods were given sodium salts in an attempt to precipitate vertigo, severe tinnitus or nausea. Large doses of soda bicarbonate by mouth or sodium racemic lactate by vein were used. In these patients the average concentration of serum sodium was increased from 140.8 to 143.7 m.eq. per liter, pHs from 7.40 to 7.43, and the serum water from 943 to 947 gm. per liter. None of the patients had an exacerbation of symptoms following these experiments.

From these data is is difficult to implicate the sodium ion as the offender in the precipitation of acute Ménière's disease. Since the clinical benefit of the so-called low sodium regime has been verified, a more satisfactory explanation was sought. In devising a palatable diet with a low sodium content some increase in the amount of potassium is unavoidable. It seemed reasonable, therefore, to try a high potassium intake and note the effect. For this purpose 5 patients have been placed on an average diet without restriction of sodium content but their potassium intake has been increased by the ingestion of 6 gm. of potassium chloride each day. In all of this group the clinical improvement has been comparable to that obtained with the Furstenberg regime.

In conclusion, it is believed that neither alkalosis, hydration nor a high sodium intake is associated with the pathogenesis of Ménière's disease.