CLINICAL STUDIES OF MAGNESIUM DEFICIENCY IN EPILEPSY

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INTEREST in the physiology and chemistry of the mineral magnesium has gradually increased over the past ten years. As early as 1944, a correlation between magnesium, tetany, and petit mal seizures were reported. Numerous studies in the deficiency of magnesium have been made since that time by many investigators.

My original work was directed toward the role of magnesium in bone apatite, and, while investigating that field, it became obvious that magnesium might play a vital role in the physiology of the central nervous system.

Beck observed in 1952 that a deficiency of magnesium might well produce effects similar to hypophysectomy. Martin, Meke and Wertman did a very comprehensive study on the clinical influence of magnesium metabolism, and again noted that in the state of epilepsy, there was a deficiency of serum magnesium. Numerous investigators have noted a form of tetany and occasional petit mal in this syndrome where a low magnesium intake was noted.

After reviewing most of the literature, not very extensive according to references, it became very obvious to me that very little work had been done in this most important, and extremely active mineral.

Until the work of Martin, Meke, and Wertman normal serum levels had not been accurately determined. In approximately 500 cases, our normal for this area was 1.60 mEq. All of these determinations were done by means of the flame spectrophotometer. We, too, have observed deficiency signs in such other diseases as renal pathology, diabetic states, congestive heart failure, postoperative patients, intestinal obstruction, toxemia of pregnancy, asthma, stravation, and depressed metabolic states.

In order to arrive at some basis for this study, we recalled all of the previously diagnosed children, both petit mal and grand mal type of seizures for review. In presenting these facts, I would like to point out that I feel additional studies are indicated to follow the magnesium levels, as well as the clinical response of these patients. Also, to determine the renal threshold of the mineral magnesium. In future studies, it is my plan to do both urinary and fecal excretions in order to determine the threshold of each individual case.

The basic studies included PBI, serum magnesium, serum calcium, serum sodium, potassium, and, in most cases, the total 17 Ketosteroids excretion and bone age.

Of thirty children studied, all of them showed a serum magnesium level of 1.5 mEq and below. After clinical studies on other types of disturbance, we have arrived at 1.5 mEq as being the lower limit of normal for people residing in this area. The chemistry of the mineral magnesium is to find an intracellular element classed as an anesthetic. It was my intention in these cases to uncover a deficiency in magnesium, if such existed, and to correct that deficiency by means of an oral preparation. It was hoped the magnesium supplement would control their petit mal and grand mal seizures. The results obtained in this series have been encouraging. For your consideration, I am listing cases of previously diagnosed epilepsy in children:

CASE NO. 1: Four year old boy, with history of petit mal seizures, particularly during stress periods, with onset at the age of eighteen months. Laboratory findings were as follows: PBI 2.8; Serum Magnesium 1.1 mEq, serum calcium 4.5 mEq, serum sodium 138 mEq, serum potassium 4.2 mEq, total 17 Ketoseroid 2.5; bone age retarded. Therapy consisted of 450 mg. of magnesium by mouth, in addition to his diet, together with thyroid extract. Within two weeks, the serum magnesium level had risen to 1.6 mEq, and his clinical signs were greatly improved. This child has been maintained on this regime for a period of three years without remission.

CASE NO. 2: S. M., age thirteen, history of grand mal seizures since the age of three. These seizures were under fair control with anti-convulsant drugs. However, the individual had marked depression and signs of mental retardation. Laboratory findings, initially, were as follows:

PBI 1.2, serum magnesium 1 mEq, serum calcium 4.6 mEq, serum sodium 142 mEq. serum potassium 4.8 mEq. 17 Ketosteroid 4.5.

Again high oral intake of magnesium was instituted along with correction of the metabolic state. Within a period of three months, all anti-convulsant drugs had been withdrawn, and the patient was maintained at a level of 1.6 mEq. normal serum magnesium. Clinically, he was much improved mentally and neurologically.

Twenty-eight other cases, with approximately the same studies and treatment, have been observed. In all cases with the exception of one, clinical improvement has been obtained by cor-

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recting the magnesium deficiency. Serum calcium levels were within normal limits in all cases. Approximately half of these children had a low PBI and a low steroid level. Since most of the children had not reached puberty, the steroid level was not given much consideration. All anti-coagulant drugs were removed over a period of time.

In presenting these findings, I would like to point out the following::

1. The possible role of magnesium therapy in the control of petit and grand mal epileptic seizures in children.

2. The need for further studies to determine the effect of the mineral magnesium upon the vasomotor center.

3. The desirability of studies to determine the serum magnesium levels in the hyper-irritable child that does not present clinical findings of petit or grand mal seizures.

4. The need for clinical studies to determine secondary conditions such as metabolic derangement, where a deficiency of serum magnesium might be present.

5. The presence of some evidence that damage to the hypothalamus and adjacent areas may well result in a clinical deficiency of magnesium, resulting in a hyper-irritability of the central nervous system, which in turn might manifest itself either as petit or grand mal seizures.

SUMMARY

This is a presentation of laboratory and case findings in epileptic children presenting deficient magnesium levels. These studies have been done to ascertain, if possible the role of serum magnesium on the central nervous system, and especially the hypothalamus. There is clinical evidence to support the possibility that it has a very vital role. There is also clinical evidence to suppose that deficient serum magnesium levels may be raised by the adequate intake of some suitable magnesium compound.

In all cases, serum calcium levels were within normal range, by using this approach to epilepsy, in addition to the complete neurological examination, which should always be done, it may be possible to control many cases without using depressant drugs. By balancing their physiological state, with special emphasis on intracellular chemistry, an easier and finer control of these unfortunate individuals may be possible.

Additional correlative studies are now in progress, and it is hoped that within the near future, they will provide further clinical and laboratory data of use to the clinician.

NOTE: mEq is the concentration in terms of reactive particles per liter and is a term used where injectibles are employed;

mg is the concentration in terms of weight per volume.

To convert from mEq to mg: Divide the mEq by the following conversion factors:

Sodium	.435
Potassium	.256
Calcium	.500 (both total & ionizable)
Magnesium	.833
Phosphorus	.580
Chloride	.282
NaCl	.171

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