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MAGNESIUM METABOLISM

The past decade has brought into prominence the part played by electrolytes in health and disease. So far the main emphasis has been on sodium, chloride, and potassium, and advances in knowledge have been such that clinicians have been able to detect the clinical counterparts of disturbed electrolyte metabolism, readily confirm them biochemically, and institute rational therapy. It is clear that in this field the next step forward will be the elucidation of the part played by magnesium.

Many methods have been developed to estimate the presence of this ion, and most of these are tedious, inadequate and unsuitable for routine use. An instrument is now available which is accurate, relatively simple to use, and able to determine the magnesium content of serum, urine and tissues. The more widespread use of this instrument will greatly increase our knowledge of the role of magnesium in man.

A great deal is already known: The total amount of magnesium in the adult human is about 25 g. One half of this is stored in bone and high concentrations are also found in the liver and striated muscle, the ion being found mainly within the cells. The average daily intake of magnesium in the diet is 250-300 mg. and some of this is unabsorbed and excreted in the faeces, while 60-120 mg. are excreted daily in the urine. There is no known endocrine control of this excretion, but there is evidence that there may be tubular secretion of magnesium as well as filtration by the glomeruli and reabsorption by the tubules.

The normal level of the serum magnesium will vary with the method of estimation used, and each laboratory must establish its own normal levels. These usually lie between 1·5 and 2·0 m.Eq. per litre. It is not known why the level in the cerebrospinal fluid is higher than that in the serum. Part of the serum magnesium is bound to protein (probably about thirty-five per cent). Hypermagnesaemia occurs in renal disease. This may result from the administration of magnesium sulphate to patients with severe chronic renal failure. Hypermagnesaemia may also occur in acute oliguric renal failure, and it is possible that some of the manifestations of depression of the central nervous system in uraemia may be due to an elevated serum magnesium. Magnesium salts should not be given to patients with severe renal disease. However, a low serum magnesium may also occur in renal disease. Hypomagnesaemia has been observed in the recovery phase of acute oliguric renal failure (acute tubular necrosis) and occurs occasionally in pyelonephritis.

Fifty per cent of patients with primary hyperaldosteronism have a low serum magnesium. This state of affairs may also be found post-operatively in patients who receive prolonged parenteral feeding and it tends to occur much more readily in children than in adults. Prolonged diarrhoea in a child may deplete the body stores of magnesium. A percentage of patients after parathyroidectomy are found to be in negative magnesium balance, possibly because magnesium accompanies calcium when it is rapidly deposited in bone. However, some patients are in negative balance even before the parathyroids are removed. A lowered serum magnesium may be associated with tetany, neuromuscular irritability, and confusion, and this may be corrected by the administration of magnesium. Most workers have failed to confirm that magnesium deficiency plays any part in epilepsy or alcoholism, and the low serum magnesium sometimes found in acute pancreatitis or diabetic acidosis has no known important accompanying symptoms or signs. There are no known electrocardiographic changes due to magnesium deficiency in man.

The next few years should yield the answers to many of the unknown facts about magnesium metabolism.

DIE HIPERVENTILASIE-SINDROOM

Die hiperventilasie-sindroom, of respiratoriese alkalose, is 'n algemene toestand. Die insidensie van die toestand in algemene hospitale en in die praktyk van interniste word beraam op 5-10 persent.[1,2] Nagenoeg 75 persent van die pasiënte is volwassenes van oor die dertig jaar, en vrouens is meer daaraan onderhewig as mans in die verhouding van 3 : 2.[1]

Die simptomatologie van die toestand berus op die feit dat hiperpnee 'n oormaat kooldioksied deur die longe laat verlore gaan vanweë oormatige alveolêre ventilasie. Dit lei tot 'n daling in die kooldioksiedtensie (pCO_2) van die plasma en gevolglik 'n verandering in die elektroliet- en pH-waardes met 'n versteuring in die verskillende metaboliese funksies van die organisme.

Die simptome wissel soms baie, maar afgesien van 'n opvallende hiperventilasie, kla die pasiënte aanvanklik van duiseligheid, mislikheid, en floute. Daar is 'n dofheid en domheid van die vingerpunte met 'n styfheid en prikkelende gevoel om die lippe. Hierop volg 'n gevoel van beklemming in die borskas en sametrekkings van die spiere, hartkloppings, gesuis, en dofheid van gesig. Die vel mag koud en klam wees en sianose mag sigbaar word. Omdat die daling in die pCO_2 van die bloed 'n belangrike prikkel van die asemhalingsentrum verwyder, volg hipokapnie spoedig.

Die beeld mag baie lyk op dié van 'n skoktoestand, veral die stygende polsspoed, dalende bloeddruk en die koue, klam sianotiese vel. Vir die klinikus word die beeld verder gekompliseer deurdat die elektrokardiogram omkering van T-golwe of afdrukking van ST-segmente met afplatting van die T-golf mag toon. Die Q-T-tyd mag verleng wees. Al hierdie veranderings is omkeerbaar deur inaseming van 'n 5 persent CO_2-mengsel.[1]

Pasiënte wat aan hierdie toestand ly is dikwels gespanne mense en hulle het soms 'n verborge vrees vir siekte of die dood. Deur aan hulle te toon dat die simptome na willekeur te voorskyn geroep kan word deur doelbewus te hiperventileer, kan hulle dikwels, indien nie genees nie, baie beter word.[3]

Dit is egter belangrik om te onthou dat hiperventilasie nie *noodwendig* op hierdie basis verklaar moet word nie. Aronson[4] byvoorbeeld, beskryf agt gevalle waar die hiperventilasie-sindroom te voorskyn geroep is deur organiese letsels soos miokardiale infarksie, hiatusbreuk, spontane pneumotoraks, en akute cholesistitis. Waar die sindroom dus in die meeste gevalle 'n psigogene patogenese het, moet 'n mens tog versigtig wees om nie die paar gevalle waar ernstige organiese letsels die verantwoordelike faktore is, sonder meer as psigogene hiperventilasie te bestempel nie.

As daar 'n organiese letsel teenwoordig is, sal dit heelwaarskynlik intratorakaal geleë wees, *of* nèt onder die diafragma, en 'n röntgenologiese en elektrokardiografiese ondersoek mag dus van groot waarde wees.

1. Forteza, M. E. (1957): Medicina (B. Aires), **17**, 132.
2. Rice, R. L. (1950): Amer. J. Med., **8**, 691.
3. Harrison, T. R. *et al.* (1958): *Principles of Internal Medicine*, 3de uitgawe, p. 304. Londen: McGraw-Hill.
4. Aronson, P. R. (1959): Ann. Intern. Med., **50**, 554.