understanding of cholestasis, many problems remain. Anatomical lesions may in fact represent the secondary results of the disordered secretion of bile. For example, there are other forms of cholestasis, associated with haemolytic syndromes, severe sepsis, and major operations, which could be due to changes in the composition of the bile itself.

Magnesium Deficiency

The metabolic role of magnesium has been studied for many years, but our knowledge remains remarkably incomplete. The fourth most abundant cation, it is an important intracellular component and has a concentration of about 15 mEq/l. The plasma concentration is 1.5 to 2.0 mEq/l. The main reservoirs of the 2,000 mEq of the metal in the body are bone and muscle. The principal sites of the ion are the mitochondria and cell nuclei, and magnesium is known to be essential to the activity of many enzymes,[1] particularly those concerned with phosphorylation and energy transfer. In addition, magnesium has a curare-like action at the neuromuscular junction.[2]

Dietary deficiency is rare, since magnesium is present in most foods. Of the daily intake of 17–34 mEq 35–40% is absorbed, a similar amount is excreted in the urine, and the remainder is excreted in the faeces. Renal conservation of the ion is extremely efficient.[3] However, renal excretion appears to be linked with that of calcium, and possibly the two elements share a common reabsorptive path in the renal tubule and gut.[4,5] Parathyroid hormone also influences magnesium excretion.[6] Therefore a combination of factors is required for the development of magnesium deficiency in man—deficient intake, failure of absorption, and excessive losses from the renal or alimentary tract—which in turn may be affected by hormonal disturbances, especially of parathormone and aldosterone.

Thus magnesium deficiency has been recorded in malnutrition with gastroenteritis in children, idiopathic steatorrhoea, ulcerative colitis, Crohn's disease, intestinal resection, ileostomy dysfunction, fistulae, ureteric transplantation, renal tubular acidosis, diabetic acidosis, prolonged diuresis, chronic alcoholism, acute pancreatitis, hyperparathyroidism, and primary aldosteronism.[7–16] In many cases the deficiency developed as a relatively late postoperative phenomenon, following prolonged infusions of magnesium-free fluids.

The symptoms of magnesium deficiency are not specific, and the clinical picture is usually complicated by symptoms of concurrent imbalance of other electrolytes. Perhaps most important in diagnosis is an awareness of those conditions in which the deficiency is likely to arise. Secondly, a readily available, reliable method for estimating magnesium is required. Hitherto lack of such a method has been a stumbling-block, and indeed accounts in part for our ignorance of magnesium metabolism. Atomic absorption spectrophotometry[17] should help remedy this situation. Obviously, hypomagnesaemia is suggestive of deficiency, but intracellular deficiency may exist without hypomagnesaemia. Finding a low urinary excretion[3] or abnormally high retention after administration of magnesium may assist diagnosis.[18] The clinical features are mental disturbances such as depression, confusion, agitation, and hallucinations ; weakness ; and neuromuscular irritability, as shown by tremor, athetoid movements, and convulsive seizures, which may prove fatal.[7,10,12,19–21] Tetany has been described in normocalcaemic hypomagnesaemia,[22] but I. MacIntyre[4] claims that the painful muscular cramps typical of hypocalcaemic tetany do not occur, Trousseau's sign being negative, though a positive Chvostek's sign may be elicited. Hyperactive changes in the E.E.G.[12] and low voltages in the E.C.G.[10] have also been noted.

Treatment[9,23] depends on the severity of the case. Magnesium sulphate or chloride 20–80 mEq in saline daily may be given intravenously, and in the less acute stages oral supplements such as magnesium hydroxide suspension, 20 ml. daily in divided doses, are suitable. Large doses are cathartic. A close watch on plasma magnesium levels during therapy is vital in patients with renal impairment.

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Amputation and Substitutes for Limbs

Amputation should no longer be regarded as an ablative and mutilating procedure but recognized as a means of getting a patient to resume his customary activities as completely and as soon as possible. When time allows, a patient who is going to need an amputation should be prepared mentally and physically for the life he will lead after he has lost his limb. In cases of injury the process of mental adjustment may be much more difficult because of the suddenness of events and because of the sometimes dominant influence of culpability. If an artificial limb is to be accepted as a natural appendage it should be as unobtrusive as possible and match as closely as possible the natural movements of the part it replaces. Engineers may in some cases be able to outdo the mechanical efficiency of nature, but at the cost of producing a limb of unacceptable appearance, especially for a woman. A recent symposium at the Royal College of Surgeons of England[1] showed clearly and in detail how the interests and responsibilities of operating surgeons, limb-makers, limb-