PATHOGENESIS OF THE CLINICAL SYMPTOMS OF HYPOMAGNESAEIA IN RUMINANTS

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ABSTRACT


Recent investigations of the pathogenesis of hypomagnesaemic tetany have shown that the clinical symptoms are of central origin and particularly related to a reduction in Mg content of the CSF (and probably also the intercellular fluid of the brain). Reduction in the level of plasma Mg is an important feature of these changes, but is not the cause of acute symptoms. The reduced level of plasma Ca - often associated with hypomagnesaemia - may modify the clinical picture, but it is not a major feature in the induction of the acute stages. The influence of so-called trigger substances for the onset of clinical symptoms (ammonia, phosphate, citric acid) could not be confirmed in acute experiments.

INTRODUCTION

As Sjollema & Seehles (1930) have already shown, a fall in blood magnesium is an essential precondition for the development of clinical symptoms in hypomagnesaemia. At the present time, the development of hypomagnesaemia can be explained in terms of an imbalance between Mg intake (defined by the Mg concentration in the feed, the quantity of feed eaten and the percentage absorption) and Mg loss (see: Meyer, 1963; Simesen, 1970; Seidel and Wirtler, 1974); but the pathogenesis of the condition and the nature of the disturbances at cellular level remain poorly understood. As numerous
studies (e.g. Hemingway and Ritchie, 1963) have shown, deficiency symptoms in magnesium shortage do not show a simple correlation with blood magnesium levels. Some animals will show clinical effects with magnesium levels as high as 1 mg %, while others remain clinically unaffected even with chronic blood levels below 0.5 mg %.

PRESENT STATE OF KNOWLEDGE

It has not so far been possible to explain this inconsistent relationship between blood magnesium levels and clinical effects, principally because the necessary pathophysiological conditions for the appearance of deficiency symptoms were not known. No convincing proofs have yet been provided for the hypothesis that there is a disturbance of impulse transmission at the neuromuscular junction in magnesium deficiency. A fall in blood magnesium

Fig. 1. Relationship between blood Ca and CSF Mg levels and the development of clinical symptoms (Scholz and Meyer, 1972).