USE OF MAGNESIUM SULPHATE IN CRITICAL CARE

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Magnesium sulphate has long been used in the treatment of eclampsia to control the fits. It has been shown to reduce the central nervous system irritability and to enhance the threshold of the central neurones to fire spontaneously. This observation led to many investigators to search any possible role for magnesium sulphate in the prevention of eclamptic fits by using it in pre-eclamptic patients. However the results were conflicting. The tocolytic effects of this drug in this category of patients led to many unwanted consequences, including prepartum / post-partum haemorrhage and uterine atony. This effect was presumed to be due to its effect on uterine smooth muscle as well as blood vessels. No effect was found on the coagulation profile itself.

An interesting co-finding of use of the drug in eclamptic patients was that the drug had a protective role against cerebral palsy in very low birth weight infants. Nelson and Greathner confirmed this finding in their retrospective study. They found that overall survival was improved in infants, weighing <1500g, born to mothers, who received Mg SO4 due to preeclampsia or as a tocolytic agent. This association was also confirmed by Blair and co-workers.

Magnesium sulphate has a role in the treatment of premature infants, admitted to neonatal critical units with persistent pulmonary hypertension. It has been used with satisfaction for this purpose. The drug causes significant decrease in AaDo2 at 36 hours, but the decrease of oxygenation index does not reach significance even after 72 hours.

Over the past few years the role of magnesium sulphate has been widely investigated in the treatment of acute asthma. Beta-adrenergic agonists are useful for the emergency treatment of asthma, but these have unwanted side-effects, e.g. increased systolic blood pressure, corrected QT interval (QTc), serum glucose and insulin and decreased RR interval, diastolic blood pressure, serum potassium, phosphate, and calcium. Concurrent use of magnesium sulphate with terbutaline has been shown to enhance these effects, modestly. But in patients with stable cardiac and metabolic function, no serious adverse actions have been reported. It has been shown that when given with standard protocol drugs, magnesium sulphate improves FEV1 at 120 to 240 minutes, and reduces hospital admission rate. This effect is more pronounced in acute severe asthmatics, whereas moderate asthma and chronic stable asthma did not respond to any significant levels. The drug may safely be used as adjunctive treatment for acute exacerbation of chronic obstructive pulmonary disease. A dose of 1.2g over 20 minutes after beta-agonist administration, is safe and modestly efficacious in the treatment of acute exacerbations of chronic obstructive pulmonary disease, and its bronchodilator effect is greater than that of a beta-agonist given alone and lasts beyond the period of magnesium sulfate administration.

The use of magnesium sulphate in acute stroke has been demonstrated recently. Magnesium ions act at endogenous vasodilators of the cerebral circulation and act pharmacologically as noncompetitive antagonists of the N-methyl-D-aspartate receptor by virtue of their role as endogenous voltage-sensitive blockers of the ion channel. The preclinical efficacy of magnesium has been demonstrated in standard models of stroke. It has been shown that the drug has neuroprotective action in focal brain ischaemia and reduces early mortality and has no deleterious haemodynamic effects. Further trials are required to confirm efficacy. It has been suggested that to gain optimum benefits out of this regimen, a loading dose of 16mmol, followed by 65mmol over 24 hours gives best results.

The role of magnesium in treating acute myocardial infarction (AMI) has been controversial. Several small clinical trials indicate that magnesium may have a role in treating AMI early, whereas the other results suggest...
that magnesium is of questionable benefit. Chirstenson
and co-workers have suggested that magnesium in-
fusion during a coronary occlusion has a significant ben-
et in reducing the infarct size (IS). Magnesium may
have a beneficial clinical role in AMI, especially if ad-
ministered before reperfusion as bolus followed by a
constant infusion. 

Fourth International study of Infarct Survival (ISIS-4) compared efficacy of early oral captopril,
oral mononitrate, and intravenous magnesium sulphate
in 58,050 patients with suspected acute myocardial in-
farction, and concluded that magnesium sulphate was
equal if not superior to captopril, as far as the mortality
rate was concerned. This is achieved probably by di-
rect vasodilatory effect, thereby reducing the infarct size,
and also by an antithrombotic effect. 

Although MgSO4 has been claimed to be effective in
different cardiac arrhythmias, especially in patients
suspected of having myocardial ischaemia, only pro-
longation of RR interval at AV nodal level in atrial tachy-
cardia could be proven to be prolonged. It had no effect
on ventricular fibrillation, ventricular tachycardia, atrial
fibrillation or heart block of any degree. The reduction in
mortality that has been shown with this form of treat-
ment is not attributable to suppression of life threaten-
ing rhythm disturbances. 

Magnesium sulphate, once regarded only as a laxa-
tive agent, has thus found many uses, and has emerged
as a useful tool in the hands of critical care physician.

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