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50 Years Ago in *THE JOURNAL OF PEDIATRICS*

Magnesium and Birth Asphyxia

Engel RR, Elin RJ. Hypermagnesemia from birth asphyxia. J Pediatr 1970;77:631-7

Engel and Elin reported that there is a correlation between oxygen deprivation, hypermagnesemia, and high potassium levels in both neonates with perinatal asphyxia and anoxic dogs. In view of their finding of elevated magnesium and potassium levels from placental blood after hypoxic deliveries and from dog pups subjected to anoxia, the authors speculated whether magnesium could be used as a marker of perinatal asphyxia from fetal scalp blood.

An increase in potassium post-asphyxia can be explained by the movement of intracellular potassium into the extracellular fluid in the presence of acidosis. This is frequently seen in asphyxiated newborns, and the levels spontaneously decrease as the acidosis improves, unless there is secondary renal failure. Regarding the magnesium levels, more recent publications report both hyper- and hypomagnesemia—but mainly hypomagnesemia, and mainly in samples taken from the neonate and not from the umbilical cord or placenta. That could explain the findings in the study of Engel and Elin. In current practice, when addressing hypomagnesemia or normal levels of magnesium following birth asphyxia, it is standard of care to give magnesium to achieve levels just over normal. The goal is to stabilize the motor membrane and the sodium–potassium–ATPase enzyme system, thus having a favorable effect on seizures and arrhythmia. The findings of Engel and Elin highlight the fact that 50 years later we still do not have all the answers regarding magnesium regulation in asphyxia. It also does highlight how perception and treatment changes, as we now know that magnesium has potentially stabilizing effects, and slightly greater levels than normal might be desirable. Magnesium sulfate ($MgSO_4$) given antenatally is today recommended for perinatal neuroprotection during threatened preterm labor. $MgSO_4$ also has been given postnatally to prevent post-hypoxic brain injury by blocking glutamate receptors within the calcium ion channel without long-term effects in clinical trials. High doses of $MgSO_4$ may trigger hypotension.[1]

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