

animal studies, magnesium administration prior to reperfusion led to a reduction in infarct size (Touyz, 2004; Geiger & Wanner, 2012; Mhaskar *et al.*, 2013).

In recent years, the use of intravenous (I.V.) magnesium can be considered as a major breakthrough in the treatment of myocardial infarction. It is found that in patients of myocardial infarction, who became critical and who died suddenly, had low serum magnesium levels. Similarly, life-threatening arrhythmias were found to be more frequent in patients with acute myocardial infarction with low serum magnesium levels. It was also shown that the magnesium content of the infarcted/ischemic myocardium was much lower (about 40-50%) as compared to that of normal heart muscle. It has been shown that magnesium depletion modifies coronary blood flow, blood clotting, and atherogenesis. Magnesium lowers systemic vascular resistance, dilate coronary arteries, decrease platelet aggregation, improve myocardial metabolism, protect against catecholamine-induced myocardial necrosis, and stabilize cell membranes. It is also cheap and easy to handle. Thus, it would appear to be an excellent contender for a place in the routine treatment of myocardial infarction, but it has not achieved this status yet. Therefore, the use of magnesium in myocardial infarction is a worthy topic of serious consideration. We, therefore, decided that we would evaluate the effect of I.V. magnesium supplement therapy in patients admitted for acute myocardial infarction and if this would be helpful in reducing the morbidity and mortality in patients.

Magnesium deficiency appears to be extremely important in the peri-infarction period. In addition to previous uncontrolled studies, several more recent controlled studies have evaluated the effect of magnesium infusion in the peri-infarction