

period. Magnesium infusion in patients with suspected myocardial infarction could reduce myocardial oxygen demand and limit the infarct size. The preinfarction magnesium status is unclear, because serum and intracellular magnesium levels have been found to be decreased, no different from, or increased, compared to patients without myocardial function (Fazekas *et al.*, 1993; Altura *et al.*, 2013; An *et al.*, 2014).

An association between hypomagnesemia and postinfarction ventricular arrhythmias has been revealed in most, but not all, studies. Supraventricular tachycardia and atrial fibrillation have been found more frequently in hypomagnesemic patients, and atrioventricular blocks and supraventricular bradycardias have been found more frequently in hypermagnesemic patients (Purvis & Movahed, 1992; Rude, 1998; Kupetsky-Rincon & Uitto, 2012).

### **1.13.6 Cardiomyopathy**

Magnesium deficiency has been suggested as the cause of cardiomyopathy. Pathologic relations as well as epidemiologic, histological, and animal studies, have implicated magnesium deficiency in a variety of cardiomyopathies (Purvis & Movahed, 1992; Rude, 1998; Kupetsky-Rincon & Uitto, 2012).

For instance, patients with hypoparathyroidism can manifest a cardiomyopathy which responds to calcium and magnesium replacement. Alcoholic patients are known to have both cardiomyopathy and magnesium deficiency (Purvis & Movahed, 1992; Kupetsky-Rincon & Uitto, 2012; An *et al.*, 2014).