Several different experimental animal models have shown that magnesium deficiency is cardiotoxic, resulting in gross pathologic changes and histologic changes from injury. Postmortem evaluation has shown that with a variety of insults, magnesium and potassium leave cardiac tissue and calcium and sodium accumulate in cardiac tissue. Any of these changes could cause necrosis (Purvis & Movahed, 1992; Kupetsky-Rincon & Uitto, 2012; An *et al*., 2014).

1.13.7 Congestive Heart Failure

Patients with congestive heart failure (CHF) regularly have magnesium due to increased urinary excretion. Magnesium deficiency worsens hyperaldosteronism, which may lead to fluid retention. Magnesium loss also compounds hypokalemia, which could theoretically produce ventricular arrhythmias and hemodynamic deterioration in CHF. Magnesium depletion may worsen cardiac function by weakening contractility, increasing vasoconstriction,or by depleting energy stores (Douban *et al*., 1996; Qu *et al*., 2013).

Not all evidence supports the importance of magnesium in CHF. Studies have shown that in ambulatory patients with dilated cardiomyopathy and CHF, the incidence of hypomagnesemia is fairly low and serum, circulating mononuclear cell, skeletal muscle, and myocardial magnesium concentrations relate poorly with each other (Douban *et al.*, 1996; An *et al.*, 2014).