1.13.8 Arrhythmias

The importance of magnesium in ventricular, supraventricular and digitalis-related arrhythmias has been the matter of extensiveattention. Magnesium deficiency which is regularly accompanied by hypokalemia produces prolongation of QT interval,-ST-segment depression, and low-amplitude T waves (Brugada, 2000).

Increased magnesium levels lead to bradycardia, increased conduction time, and diminished automatism. Magnesium possibly effects transport of potassium,sodium,and calcium across the cell.

Magnesium may eradicate ventricular arrhythmia during acute myocardial ischemia due to prevention of conduction slowing by an anti-ischemic action (Fazekas *et al.*, 1993; Ho, 2008). While hypomagnesemia is related with hyponatremia, hypocalcemia, and hypophosphatemia, the relationship with hypokalemia is the best known link.

Both potassium and magnesium tend to be depleted with thiazide diureticsand spared with potassium-sparing diuretics.

This is particularly true for the elderly and for patients on high doses of diuretics, and may be true for others already at risk for electrolyte abnormalities, including alcoholics, diabetics, those with congestive heart failure, or those with a recent myocardial infarction. It has been shown that although serum potassium rises with replacement therapy, the level of potassium in muscle will not increase unless magnesium is replaced as well. The benefit of replacing potassium and magnesium in patients with deficits in both seems to be larger than for either alone, particularly in patients treated with non-potassium-sparing diuretics. Patients on non-potassium-