Being the major route of excretion , kidney plays a major role in magnesium homeostasis. Normally, when 80% of total plasma magnesium is ultrafiltrable, 84 mmol of magnesium is filtered daily, of this amount 95% is reabsorbed, of them 15-20% is reabsorbed in the proximal tubules, 65-70% in thick ascending loop of Henle and the rest in distal tubules (Swaminathan, 2003; Pasternak *et al.*, 2010; Jahnen-Dechent & Ketteler, 2012). In proximal tubules , magnesium is reabsorbed passively, this process mainly depends on sodium/water reabsorbtion and on luminal magnesium concentration .In thick ascending loop of Henle , magnesium reabsorbtion depends on sodium/chloride reabsorbtion and the positive luminal charge created by active cellular pumps and passive paracellular diffusion . On the other side, magnesium reabsorbtion is active and transcellular in the distal tubules . Magnesium reabsorbtion is inversely related to the rate of fluid flow in the tubules (Wester, 1987; Rude, 1998; Pasternak *et al*., 2010; Jahnen-Dechent & Ketteler, 2012).

1.6 Hypomagnesaemia

Magnesium deficiency , also known as hypomagnesemia is an electrolyte disorder that is frequently undetected owing to the fact that hypomagnesemia is often asymptomatic or a secondary disorder . Therefore, recent studies shows that hypomagnesemia is present more than previously thought (Swaminathan, 2003; Shah *et al*., 2014).In general, clinical manifestations of magnesium deficiency are usually not seen until serum magnesium concentration decreases to 0.5 mmol/L or lower, Table(5) shows a list of magnesium deficiency clinical manifistations (Rude, 1998; Jahnen-Dechent & Ketteler, 2012; Zittermann, 2013).