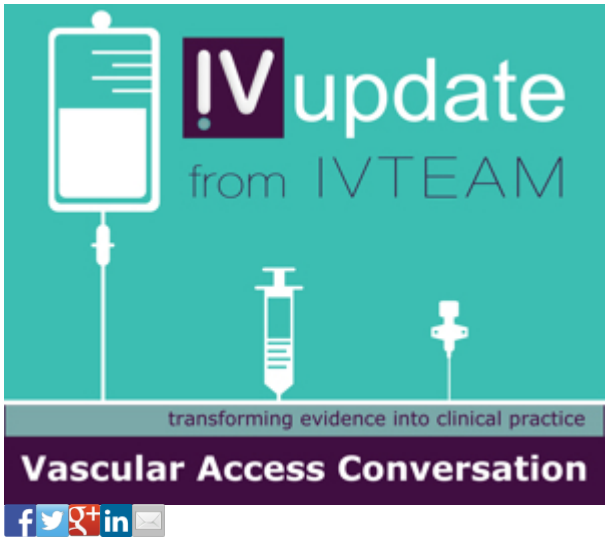

Intravenous literature: Atherton, J.C. (2009) Role of the kidney in acid-base balance. Anaesthesia and intensive care medicine. 10(6), p.276-278.

Abstract:

Correction of disturbances in acid-base balance is achieved by: physicochemical buffering by extracellular and intracellular buffer systems (instantaneous), alveolar ventilation to control pCO2 (rapid), and renal compensation (long term). Buffering and changes in ventilation limit changes in pH but cannot return acidâ€“base status to normal. The kidney has a pivotal role: disturbances can be completely corrected through changes in H+ secretion and HCO3âˆ’ reabsorption and production. HCO3âˆ’ reabsorption is modified by changes in GFR (filtered load), changes in extracellular volume and by hormones which modify Na+ reabsorption via the Na+â€“H+ exchanger in renal tubular cells. Changing the activity of this exchanger influences H+ secretion and, hence, HCO3âˆ’ reabsorption. Chronic (but not acute) changes in pCO2 influence HCO3âˆ’ reabsorption through changes in the filtered load and, in chronic acidosis, by the insertion of more H+ transport proteins in renal tubular cells. Renal HCO3âˆ’ production is linked to H+ excretion: acid buffer salts (phosphate, creatinine), their availability and pK and tubular fluid pH. Formation and excretion of NH4+ buffer salts are important â€“ acidosis stimulates secretion of NH4+ (proximal tubule) and NH3 (collecting duct). There is a reciprocal relationship between extracellular K+ and NH4+ excretion, hence HCO3âˆ’ production.