Red Blood Cell Sodium Transport and Phosphate Release in Uremia

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Dear Sir,

In a recent article, Walter and Becht [Nephron 34: 35–41, 1983] observed that red blood cell sodium transport and ouabain-sensitive phosphate release was diminished in uremics. I would like to make two comments about this article:

(1) It is not universal to find a decreased red cell sodium transport in uremics. In a recent article [4], we observed that erythrocyte sodium content was diminished in 15 uremic patients and this was associated with a decreased ouabain-sensitive efflux rate of sodium, a normal ouabain-sensitive efflux rate constant of sodium, a normal Na^+ , K^+ -ATPase activity and reduced number of ouabain-binding sites. On closer analysis of the data, it was observed that in a small group of uremic patients with low erythrocyte sodium, the ouabain-sensitive efflux rate constant of sodium was increased and in another group of patients with a more severe degree of uremia the ouabain-sensitive efflux rate constant, efflux rate Na^+ , K^+ -ATPase activity and ouabain-binding capacity were all reduced. We interpreted these findings as suggestive of a series of changes during the development of renal failure. During the progression of chronic renal failure, there is initially an increase in the activity of sodium pumps due to a 'stimulator' or absence of 'inhibitor'. With further deterioration in renal function, activity of sodium pumps as well as membrane permeability decrease. A low erythrocyte sodium content has been observed previously in chronic renal failure [1, 2, 5].

(2) Walter and Becht measured ouabain-sensitive phosphate release and found it to be related to rate constant of ouabain-sensitive sodium efflux. The phosphate release was used as an index of Na^+ , K^+ -ATPase activity in intact red cells. We agree that measurement of Na^+ , K^+ -ATPase activity of hemolysates, ghosts or membrane is not indicative of the activity in intact cells. As suggested by Walter and Becht the phosphate release is not entirely satisfactory as there may be variable reutili-

zation of phosphate released. We have used a different approach to this problem by studying the disappearance of glucose. In a recent study of 25 healthy subjects, it was observed that the rate constant of sodium efflux and ouabain-binding sites were significantly related to ouabain-sensitive glucose consumption ($r = 0.433$ $p < 0.05$, and $r = 0.5575$ $p < 0.01$ respectively [Richardson and Swaminathan, unpubl. results]). The correlation co-efficient, although significant, was not high as between Na^+ , K^+ -ATPase of red cell ghosts and ouabain-binding sites ($r = 0.94$) [3]. Possible reasons are (a) the glucose consumption method is not sensitive enough, and (b) a small number of observations with a narrow range of sodium pump activity (range for rate constant 0.201–0.432 and for ouabain-binding sites 187–404). Further studies with more precise techniques are in progress to verify these possibilities.

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