Gastrointestinal Absorption of Aluminium

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Sir,

Aluminium (Al) is now recognised as an important toxin causing considerable morbidity and mortality, particularly in patients with chronic renal failure. Though Al toxicity tends to occur when the gastrointestinal barrier is circumvented, measurable amounts of Al are absorbed from the gut in healthy subjects [1] and intoxication has developed in patients with uraemia treated with Al-containing phosphate binding gels [2].

The mechanisms that affect gastrointestinal uptake of Al are poorly understood. Intestinal permeability is increased in the neonatal period in humans [3] and this may account for the increased susceptibility of infants to Al intoxication. In man and animals various factors have been shown to promote Al absorption including parathyroid hormone [4], dihydroxyvitamin D3 [5], zinc deficiency [6] and citrate ingestion [7]. As Al-containing phosphate binders, used in chronic renal failure, have ampho-teric properties gastric acid secretion may affect absorption. In vitro studies have shown that pH affects the ability of these substances to bind phosphate [8] and the gastric acid secretory status of the stomach may affect phosphate binding by these substances in vivo [9].

Ten stable and compliant patients with chronic renal failure, with no significant residual renal function, established on continuous ambulatory peritoneal dialysis for greater than 6 months underwent a standard pentagastrin stimulation test where basal and maximal stimulated acid secretion was measured. No patients were taking H2 blockers. Fasting blood was drawn for serum Al estimation by atomic absorption spectrophotometry and the patients were then prescribed 20 ml of aluminium hydroxide (Aludrox) to be taken 3 times daily with meals for 2 weeks; the blood samples were then repeated.

Eight patients had normal gastric acid secretory profiles, 1 had high basal and maximal acid secretion and 1 p = < 0.01

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Fig. 1. Serum aluminium levels before and after oral ingestion of aluminium hydroxide.

had basal achlorhydria and low stimulated acid secretion. There was a significant rise in serum
Al over 2 weeks in the group as a whole (fig. 1). In the 3 patients in whom serum Al rose to
above 2 µmol/l (54 µg/l) gastric acid secretion was low, normal and high, respectively.

Serum Al can only be used as an indirect measure of Al absorption due to deposition of the
element in tissues. Bearing this caveat in mind our findings suggest that gastric acid secretion
may not play a significant clinical role in modulating Al absorption in patients with chronic renal
failure.

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This is an interesting book for the nephrologist. It deals with all possible uses of shock wave
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second generation lithotripsy results and (6) a final group of research papers.

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