Lead Stores in Patients with Renal Insufficiency

B.T.

Emmerson

University of Queensland Department of Medicine, Princess Alexandra Hospital,
Brisbane, Qld, Australia

Prof.B.T. Emmerson, University of Queensland Department of Medicine, Princess Alexandra Hospital, Brisbane, Qld 4102
(Australia)

Dear Sir,

A recent paper by Winterberg et al. [1] entitled 'Lead Overload in Patients with Renal Insufficiency' has considered the question of whether lead overload develops in haemodialysis patients and concluded that the answer is clearly in the affirmative. They presented the bone lead concentrations of control subjects, of patients with chronic renal failure, of haemodialysis patients and of patients after renal transplantation, none of whom were known to have had lead exposure, and concluded that the lead body burden depended on renal function. Our extensive experience in Queensland, Australia, with chronic lead nephropathy following childhood lead poisoning, provides contrary data which we believe is relevant to the current consideration of this question.

Childhood lead poisoning became common in Queensland after 1890 and was followed some years later by the development of chronic renal failure. This varied in severity so that the most severely affected died in their teens and the less severely affected cohorts lived for 40 or 50 years. This childhood lead poisoning resulted in a great increase in the incidence of granular contracted kidneys in Queensland in the first half of this century. After lead paint was outlawed by legislation in the 1920s, this increased incidence of granular contracted kidneys steadily subsided until it was similar to that in the rest of Australia some 30 or 40 years later.

The epidemiological follow-up of subjects with childhood lead poisoning was undertaken by Henderson [2] who also studied the lead content of bone in patients with granular contracted kidneys in Queensland [3]. These studies showed that the bone lead content of persons with chronic renal disease of known aetiology (not due to lead) is the same as for persons who died without chronic renal failure, whereas much higher values were found in those persons with granular contracted kidneys following childhood lead poisoning (chronic lead nephropathy). Later studies of the lead content of bone in subjects without recognized past lead exposure in Queensland and in patients with chronic renal disease showed that the lead content of bone in patients with renal disease not due to lead was within the range of normal subjects, whereas the values of lead in bone for subjects with chronic lead nephropathy were elevated [4]. Additional information was provided by the amount of lead excreted in the urine after a standardized infusion of calcium EDTA. This was measured in normal subjects, in subjects with chronic lead nephropathy and in patients with chronic renal insufficiency attributable to causes other than childhood lead poisoning [5, 6]. This showed that, except possibly in patients with polycystic kidney disease, the lead excreted after EDTA in subjects with chronic renal disease due to causes other than childhood lead poisoning was in the normal range for that population.

When taken together, these studies provide strong evidence that renal function is not a prime determinant of the lead content of bone. Even if renal excretion of lead is one of the factors determining the body lead burden, considerable further evidence is needed before it can be concluded that the 'lead body burden depends on renal function'. However, these studies from Queensland were undertaken before chronic haemodialysis was established, so that the studies of Winterberg et al. [1] refer to the slightly different question of whether lead overload develops in haemodialysis patients. Nonetheless, their data gives the bone lead of their haemodialysis patients as 3.59 ± 2.3 mg/g of wet bone and such a wide standard deviation suggests a wide range of bone lead values for this group. Since environmental and industrial past lead exposure and absorption may vary widely and may not be

234

Emmerson

Emmerson BT, Lecky DS: The lead content of bone in subjects without recognized past lead exposure and in patients with renal disease. *Aust Ann Med* 1963; 12:139–142.

Emmerson BT: Chronic lead nephropathy: The diagnostic use of calcium EDTA and the association with gout. *Aust Ann Med* 1963;12:310–324.

Emmerson BT: Chronic lead nephropathy. *Kidney Int* 1973; 4:1–5.

obvious even on careful questioning, it would be important to exclude these factors and to record the precise distribution of the bone lead data before concluding that any elevated bone lead was due to impairment of the renal excretion of lead.

6

References

Winterberg B, Fischer R, Korte R, Bertram H: Lead overload in patients with renal insufficiency. *Nephron* 1990; 55:96.

Henderson DA: A follow-up of cases of plumbism in children. *Aust Ann Med* 1954;3:219–224.

Henderson DA, Inglis JA: The lead content of bone in chronic Bright's disease. *Aust Ann Med* 1957;6:145–154.