

Reply

Sir,

We thank Dr Shaldon for his comments, but find it slightly disappointing that he focuses on minutiae and semantics that are not related to the main focus of our study. We agree that some of the issues raised in the introductory paragraph regarding management of anaemia in chronic kidney disease can be debated. These are issues from the published literature and certainly not the 'claim' of the authors of this article. The focus of the publication was 'antibody-mediated pure red cell aplasia' and was certainly not a discussion on the much wider topic of management of anaemia in dialysis patients [1].

Dr Shaldon's letter refers to publications from the 1960s relating to management of CKD with dialysis and intravenous iron. As evident from the published literature, quality of life (QoL) then was less of an issue in clinical practice, and the tools to measure QoL were even less standardized than they are today. 'Maintaining patients' Hb levels between 9–10 g/100 ml on IV iron alone' has to be assessed against this background. There has always been a view that long dialysis reduces the need for transfusion, and while this is an admirable achievement, this is not something that is appropriate for, or acceptable to, all patients.

Randomized controlled multicentre trials since the advent of EPO have confirmed the improvement in QoL with this agent in dialysis patients, and have also indicated that this is not maximized at a Hb of 9–10 g/100 ml [2–4]. It is widely accepted that administration of intravenous iron leads to improvements in haematocrit levels in haemodialysis patients and enhances Hb responsiveness when used in conjunction with epoetin [5]. Our use of EPO is consistent with recognized standards and practice elsewhere.

Although there are several survival studies suggesting no benefit in normalizing Hb in chronic kidney disease patients, no study has ever been conducted in a large-enough sample size to investigate whether EPO therapy increases survival compared with standard practice in the pre-EPO era using regular blood transfusions and/or IV iron, but it would take a very brave nephrologist to suggest that the minimization of blood transfusions and avoidance of iron overload by EPO has not impacted on survival.

The declared conflict of interest relates only to one of the authors, whose involvement was only in the investigation of pure red cell aplasia and not in the direct clinical care of the patient. Even this author has espoused the benefit of IV iron with EPO, a practice which has been shown to decrease the use of EPO [5]. Volume discounting is widely used in all 'markets', including health care, and made use of by both the buyers and the sellers.

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Statins and renal function. Is the compound and dose making a difference?

Sir,

Atthobari *et al.* [1] conclude that statin (pravastatin) treatment was not associated with a significant improvement in glomerular filtration rate (GFR) in subjects with modestly impaired GFR. This conclusion deserves some debate.

The Heart Protection Study showed that allocation to simvastatin significantly attenuated the fall in estimated GFR (e-GFR) in diabetic and non-diabetic subjects compared with placebo [2]. A pooled analysis of the Cholesterol And Recurrent Events, Long-term Intervention with Pravastatin in Ischemic Disease and West Of Scotland Coronary Prevention Study trials comparing pravastatin vs placebo also showed a decreased deterioration of renal function [3]. In the GREEK atorvastatin and coronary heart disease evaluation (GREACE) study, atorvastatin treatment significantly increased e-GFR and reduced serum uric acid levels, whereas renal function deteriorated in untreated patients [4,5,6]. In GREACE [4,5,6], the effect on renal function was rapid, more evident in patients with serum creatinine levels at the upper end of the reference range, more pronounced at higher doses of atorvastatin used (10–80 mg/day) and contributed to the reduction in vascular events (multivariate analysis). The results of a *post hoc* analysis of the Treating to New Targets trial showed that instead of the expected decline of about 5 ml/min over the 5 year study period, there was a significant increase in e-GFR with both the 10 mg/day (by 5.6%) and the 80 mg/day dose of atorvastatin (by 8.4%) [7].

Thus, the effect of statins on renal function may depend on the statin used, the extent of hypolipidaemic effect and the patient category. These factors need to be resolved to ensure the provision of best treatment. Given that renal and coronary disease may progress in parallel, [4,5,6] appropriate statin treatment at appropriate dosing may be beneficial to both the heart and kidneys.