did not discuss ‘salt’ or ‘volume’ at all. He implies we were ignorant of these factors, and of the prolonged survival of haemodialysis-treated patients in a non-standard way (long, slow dialysis with dietary salt restriction—La recette de Tassin). This is not the case, as we ourselves have reported the survival of a similarly treated cohort of patients [2]. The reason for the omission of these factors is a simple one. There are no randomized controlled trials (RCTs) of these interventions in patients on dialysis with appropriate hard end-points, and it was RCTs of this nature that our article focussed on (something made clear in the article and its companion). Dr Stanley Shaldon should not conflate single-centre retrospective reports with generalizable interventions tested by RCTs. Underpinning the rationale for the NIH trial of quotidian dialysis is the realization that one explanation for the remarkable survival in some dialysis centres offering long, slow dialysis is patient selection bias. Assertion from eminences grises that such therapies are ‘better’ cannot replace the need for high-quality evidence from clinical trials, at least not in 2008.

While the data from Cook et al. [3] are very interesting, we doubt their relevance to patients on dialysis programmes. There are many very good reasons to theorize that better control of salt, water, volume, etc. will be of survival benefit to dialysis patients, but no one, not even someone with as long and distinguished a pedigree in this arena as Dr Stanley Shaldon, has yet got around to proving this.

As for his last comment—well we would defy anyone (else) reading our article, which basically asserts that newer dialysis techniques and expensive drugs and interventions offer no survival advantage, to infer that we had a bias of high-quality evidence from clinical trials, at least not in 2008.

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A wild zebra chase

Sir,

Dr Woywodt et al. [1] should be commended for this interesting and well-written report. We have the following comments. In the teaching point, it was implied that amiodarone produces renal phospholipidosis similar to that in Fabry disease. Although amiodarone is well known to produce cornea verticillata and lamellated phospholipid inclusions in multiple organs such as lung, liver and eyes, significant nephrotoxicity and in particular myeloid bodies in human kidneys had not yet been described in the literature. In another statement about the paucity of the manifestation of Fabry disease, the negative serum α-galactidase A level should have precluded ‘unnecessary and useless’ tests. It is worth mentioning that the diagnosis of Fabry disease can be difficult even in a homozygous male. Some patients, renal variants, may lack classic manifestation of Fabry disease such as acroparesthesia, angiokeratoma and corneal lesion [2]. This difficulty is compounded in a heterozygous female who may be variably affected from lyonization. It should also be recognized that serum α-galactosidase A is frequently normal in females. Dr Woywodt et al. are correct in their emphasis on searching for a more common disease first, rather than for obscure and rare entities such as Fabry disease. However, the true incidence of iatrogenic renal phospholipidosis is not known, with only four case reports so far [1,3–5], and this might well be a rare side effect of chloroquine and hydroxychloroquine. As demonstrated in the paper by morphology, one cannot distinguish between genetic and iatrogenic causes of renal phospholipidosis. Therefore, the diagnosis requires measurement of enzymatic activity and/or mutational analysis of the α-galactosidase gene. These studies are necessary and useful in obtaining the correct diagnosis.

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