heavy atherosclerotic burden in these patients [2]. This may be secondary to risk factors commonly observed in patients with chronic kidney disease (calcium-phosphate abnormalities, inflammation and traditional cardiovascular risk factors), especially in the setting of a lengthy dialysis vintage.

Understandably, one may be hesitant to start such a dyslipidemic patient on peritoneal dialysis; however, the aim of our manuscript was to simply describe our experience in a patient with a unique metabolic abnormality. Given the rarity of this disease and conflicting reports to date, it would be prudent to develop a worldwide database of patients with LCAT deficiency on renal replacement therapy, to further delineate this fascinating disease.

Conflict of interest statement. None declared.

Sir,

Helmut Schiffl’s study, published in this journal [1], provides further confirmation of the difficulties of prescribing and delivering a urea-based dose of intermittent haemodialysis (IHD) in the critically ill patient. Using an anthropometric (Watson) estimate of body water [2] to help define the prescription, and the simplified Daugirdas II formula [3] to define dose delivery, significant prescription–delivery shortfalls were apparent in this population of critically ill maintenance HD patients.

The findings of this study are of interest but the approach and conclusions invite some comment.

Firstly, the distinction between ESRD and Acute Kidney Injury (AKI) is probably artificial—critical illness, rather than the cause of dialysis dependence, is likely to be the relevant factor. A prescription–delivery shortfall should thus not be unexpected, as shown by others [4] as well as by us [5]. The use of the Watson estimate of body water (developed from a non-uraemic population) may compound under-prescription of HD—and thus overestimate the prescription–delivery shortfall—in these patients who are often significantly fluid-loaded.

Secondly, the suggestion that critical illness violates the fundamental assumptions of urea kinetic modelling (UKM) warrants examination. Steady-state assumptions, with regard to urea generation and urea distribution volume, allow delivered dose to be assessed at relatively infrequent intervals in stable, chronic HD patients. In the only published evaluation of formal UKM in the critically ill AKI patient undergoing IHD [5], we have demonstrated wide inter- and intra-individual variability in these key factors. Despite this, we were able to derive an equilibrated Kt/V using formal, iterative double-pool UKM (rather than shortcut formulae) and were able to demonstrate that a target eKt/V could be prescribed to within a median absolute error of <0.14 Kt/V using practical prescription algorithms. Rather than violating urea kinetic assumptions, the presence of critical illness actually required the application of such dosing techniques on a frequent (i.e. sessional) basis, to account for the non-steady state.

Although low-molecular weight toxin clearance (using urea as a surrogate) is only one aspect of ‘dose’ in renal replacement therapy for the critically ill [6], a urea kinetic approach is entirely possible, if applied with the appropriate frequency and technique.

Conflict of interest statement. None declared.

Department of Renal Medicine, N.S.
Newcastle-upon-Tyne Hospitals Kanagasundaram Trust, UK

E-mail: suren.kanagasundaram@nuth.nhs.uk
doi: 10.1093/ndt/gfm746

Advance Access publication 6 November 2007

Reply

Sir,
The correct prescription of dialysis dose for critically ill end-stage renal disease (ESRD) patients is still unsolved.