analyses of several large clinical trials, with cardiovascular disease (CVD) end-points, in the general population [2].

In the above-mentioned post hoc analyses, apart from the increased risk for type 1 errors [2], there are several limitations. Renal failure patients were excluded [1] and results regarding CKD progression are often based on fewer patients than initially included in the study [3]; glomerular filtration rate is indirectly estimated; adjustment for main factors influencing CKD progression is often incomplete and, when it is, changes the initial results [4], while data for albuminuria or microalbuminuria are frequently absent [5]. If the negative results of the ASCOT-LLA [6], VA-HIT [7] and ALERT [8] studies are added to these limitations, a motivated, based on post hoc analyses, reconsideration of the CPG seems, up to now, less urgent.

On the other hand, considering the impressive results achieved by statin treatment in studies with CVD end-points in the general population and taking into account the dyslipidaemia patterns in CKD patients, a few points of interest in this particular population are worth highlighting.

The main target, regarding lipids, in the majority of the studies in the general population, was elevated low-density lipoproteins (LDL) and total cholesterol (tChol). At initial stages of CKD, a similar dyslipidaemia pattern—due to nephrotic syndrome or to common causes of CKD such as diabetes, atherosclerosis, etc.—is frequently observed. Results based on studies in the general population can potentially be extrapolated (regarding hypolipidaemic treatment for CVD prevention) in CKD patients with a similar dyslipidaemic profile. The improvement of cardiovascular status—at least the haemodynamic benefit—should also be important for the stabilization or retardation of CKD progression. Furthermore, the pleiotropic effects of these drugs and specifically their actions on endothelial function, oxidative stress, inflammation, etc. might also be beneficial in slowing progression of CKD in this subgroup of patients.

In contrast, in patients with severe CKD in whom the dyslipidaemic pattern approaches that of patients with end-stage renal failure (ESRF) in renal replacement treatment (who usually have elevated triglycerides, low high-density lipoproteins and normal or low tChol and LDL), extrapolation of the general population study results might no longer be appropriate (mainly because this dyslipidaemia profile does not exist in the populations included in these studies). Furthermore, hypertriglyceridaemia is better treated with fibrates or hypolipidaemic drugs other than statins, which do not seem to have any beneficial effects on CKD progression [7]. Moreover, cholesterol lowering in this subgroup of CKD patients might even be inappropriate, especially if the ‘reverse epidemiology’, regarding the detrimental role of low tChol in ESRF patients’ morbidity and mortality, is valid and also extended to the pre-dialysis population. Finally, malnutrition and aggravation of micro-inflammation, frequently observed in these late CKD stages, might be inter-related with dyslipidaemia and also have to be taken in consideration.

In any case, only studies in CKD populations (such as SHARP and PREVEND-IT [1]), with end-points related to renal function in time, can eventually give a definite answer to the question of whether lipid-lowering treatment is beneficial for the progression of CKD.

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Bedside testing of CAPD fluid for bilirubin to aid diagnosis of visceral perforation

Sir,

Peritonitis is a major complication of continuous ambulatory peritoneal dialysis (CAPD). Visceral perforation accounts for 1–10% of such complications and can be a difficult diagnosis to make [1,2]. The delay in diagnosis has a significant impact on mortality and morbidity [1]. We would like to highlight a case of CAPD peritonitis that was associated with visceral perforation.

A 47-year-old patient presented to a district general hospital with abdominal pain and was originally treated for CAPD peritonitis. He was admitted because of a previously undiagnosed end-stage renal failure due to immunoglobulin-A nephropathy. There was no history of peptic ulcer disease nor was there steroid, proton pump inhibitor or H2 antagonist use. Initial clinical examination revealed diffuse tenderness across the abdomen without rebound or guarding and he was started on antibiotics for CAPD peritonitis.

He was transferred to the regional renal unit and his clinical
condition had deteriorated. On examination at this stage his abdomen was tender with guarding. His CAPD fluid was tested using 'dipstix' testing and was found to be positive for bilirubin. The patient also had a chest X-ray, which showed air under the diaphragm. This can be normal in the CAPD population, but the chest X-ray was repeated after a temporary dialysis line was inserted and the air was no longer present. Due to the clinical picture and patient's condition he was taken to theatre where a perforated duodenal ulcer was found. He had a protracted post-operative course with an ITU stay and multiple intra-abdominal abscesses, but has now recovered.

Normal methods for diagnosis including erect chest X-rays have been shown to be of limited use in these patients and are not a reliable indicator of visceral perforation [3]. One of the more common indicators of visceral perforation is multiple enteric organisms on culture of CAPD fluid. This in itself can take a few days, which is also delaying the patient's treatment. We feel that the bedside testing of CAPD fluid for bilirubin may help with the diagnosis of visceral perforation. If we can shorten the time to surgery for these patients we may be able to make an impact on the mortality and morbidity.

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Recurrent hydronephrosis causing acute uraemia in a renal transplant donor without the presence of stones or stricture

Sir,

When the kidney donor has been thoroughly examined according to the usual standards [1], complications in the short and the long term are seldom seen. This, together with the favourable outcome for the recipient, justifies living donor transplantation.

However, a presumed complication could have been fatal to a 63-year-old woman who donated her right kidney to her brother in January 2003. In the following May and June, she had severe pain in the left flank and, when vomiting, general oedema and ultimately anuria occurred, she was admitted to the urological department, where the serum level of creatinine was found to be 841 μmol/l. Ultrasound showed hydronephrosis and, after nephrostomy, the serum creatinine dropped to 150 μmol/l. Antegrade pyelography revealed no stones or stricture and the catheter was removed. Unfortunately, the patient was readmitted with similar symptoms some weeks later. Nephrostomy was again performed and no abnormalities in the urinary tract were seen on the pyelogram. The woman had had no problems whatsoever from the urinary tract before donation, and hydronephrosis of the kind caused by a valvular effect over an aberrant artery passing the ureteropelvine junction was suspected. After a period with a JJ stent, she underwent a pyeloplasty operation in August and, 1 year after donation was well with a serum creatinine of 123 μmol/l.

Before donation, the patient reduced her weight from 102 to 79 kg to achieve a body mass index (BMI) of 31. Intravenous pyelography showed a small extrarenal pelvis on the left side, which expert radiologists judged to be completely normal, as was the renogram. Arteriography showed two arteries on the left side.

The 65-year-old recipient had no urological complications, and 1 year after transplantation his serum creatinine was 142 μmol/l.

This kidney donor, who developed acute uraemia 5 months after donation, was at first believed to have passed a stone unknowingly. After a second similar episode with no stone or abnormalities in the urinary tract, another less obvious explanation had to be sought. She was used to drinking about four litres a day; all urine had to pass through one kidney, and it was suspected that a slight hydronephrosis was kinked over the aberrant artery, permitting complete obstruction now that surplus water could not be eliminated by a second kidney [2]. The weight loss with a decrease in the surrounding fatty capsule may have resulted in less physical support for the kidney, thus adding to the problem.

In any case, although kidney donors have a life expectancy longer than that of the general population and should not, in our opinion, have limitations set on their lives [3], health personnel, including physicians in primary care, must be very thorough in their diagnostic work-ups, when a person with a single kidney presents with symptoms from that region. Obstruction might have caused serious damage to the remaining kidney within a relatively short time.

We do not think that this potential complication has a frequency that should lead to limitation of kidney donation. However, when the remaining kidney has multiple vessels and an extrarenal pelvis, the risk of ureteropelvic junction obstruction may be slightly increased [4,5].

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