reached the level of statistical significance (Wilcoxon’s \( P=0.055 \)). No reciprocal relations between the above HGF values, their per-dialytic change, enoxaparin dose or HD session duration were found.

In conclusion, we saw that salivary HGF concentration normalized during HD sessions. Our working hypothesis turned out to be apparently incorrect. The specific and important quest for the cause behind periodontal disease epidemics in maintenance HD patients has just begun and should be pursued.

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**The long-term effect of serum magnesium on cyclosporin A toxicity**

Sir,

We read with great interest the study by Holzmacher *et al.* [1], suggesting that low serum magnesium (Mg) levels caused a more rapid decline in allograft function and higher rates of allograft loss in patients with chronic cyclosporin A (CsA) toxicity. We would like to underline several points in this study:

(1) We were surprised by the dose of CsA reported in this paper, as it seems quite high at the time of biopsy, the mean 3.9 ± 0.6 years and 4.3 ± 0.7 years after transplantation, the mean dose of CsA 7.5 ± 2.5 and 8.2 ± 3.6 mg/kg/d for low Mg and the normal Mg groups, respectively, leading to high CsA levels for both groups. Such a high dose of CsA can induce chronic allograft nephropathy due to chronic CsA toxicity.

(2) The authors also reported that although the adjusted relative risk (RR) of graft loss was 35% higher in the low Mg in comparison to the normal Mg group, the RR was not to be 0.65. By definition, RR is a measure of how much a particular risk factor (patients with low Mg) influences the risk of a specified outcome (kidney failure/death). In this study by Holzmacher *et al.*, according to an RR of 0.65, the low Mg group should have had lower death/kidney failure in comparison to the normal Mg group, as seen in Figure 2, bottom panel. In addition, the confidence interval for the RR was 0.4 – 1.4. It is clear that the confidence interval contains the value 1.0 and this is evidence that the RR is not statistically significant.

(3) The median level for the normal Mg group was not given and the mean level was given.

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**To haemodialysis and back: saving a kidney graft by treatment of an arteriovenous fistula**

Sir,

With an incidence of around 16%, arteriovenous fistula (AVF) is a frequent complication of percutaneous renal biopsy (PRB) after kidney transplantation [1]. In only 0–5% of cases, however, an AVF causes clinical signs, such as systolic-diastolic murmur in the area of the graft, haematuria or hypertension [1–3]. A more serious consequence of AVF is a decline in renal function caused by shunting of part of the blood flow directly through the AVF into the venous system, bypassing the glomerular vessels. This leads to a shortage in circulation at the glomerular level in the flow area behind the AVF. The perfusion outside the flow area of the AVF will also be reduced since a substantial part of the blood flow in this region will be shunted towards the affected flow area. Due to this diminished blood flow the affected flow area will activate the renin-angiotensin system causing hypertension and sodium-retention [1,4,5].

Recently a 36-year-old male with end-stage renal failure due to type 1 diabetes received a simultaneous pancreas–kidney transplant in our clinic. Due to limited improvement of renal function (Figure 1) in total three PRBs were performed during follow-up, all taken from the upper pole. The first two biopsies showed signs compatible with cyclosporine and tacrolimus toxicity, respectively, but cessation of these drugs did not improve renal function. After the second PRB a systolic-diastolic murmur was heard above the graft and presence of an AVF was suspected. Colour-coded Doppler sonography (CCDS), however, did not support this diagnosis. Forty-five days after transplantation the patient had to return to haemodialysis and a third PRB was performed, showing signs of focal tubular necrosis.
Clinically there was now hypertension and mild urinary sodium retention, both suspected to have a renovascular cause, which strengthened earlier suspicions of the existence of an AVF despite the negative CCDS. Subsequent angiography revealed two large AVFs with rapid venous outflow in the upper pole of the graft (Figure 2). Both AVFs were coiled using superselective transcatheter embolization, which resulted in disappearance of the murmur, improvement of renal function, normalization of blood pressure and urinary sodium-excretion. Currently, more than 1.5 years after transplantation, the patient has an excellent renal function with a creatinine clearance of 54 ml/min. This is to our knowledge the first report of successful treatment of a patient with 'features of end stage renal failure' after PRB-induced AVF. This case clearly shows that sometimes an AVF can cause serious problems and therefore requires treatment. However, due to the low incidence of symptomatic AVFs after PRB there is little attention to this phenomenon in clinical practice. With this case we want to stress the importance of considering AVF as a complication of a PRB in the differential diagnosis of poorly understood renal dysfunction after kidney transplantation.

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