benefit. We totally agree about the importance of circulatory compensatory mechanisms and that a steady state of relative blood volume (RBV) may not have been attained during the brief period of exercise we employed. What we in fact suggested was that our findings may not extrapolate to older patients subjected to more prolonged or intense exercise at high ultrafiltration (UF) rates. We still think this is a reasonable caveat. We quoted evidence that exercise in the later stages of haemodialysis sessions employing UF may not be quite so well tolerated [1]. Indeed, Dasselaar et al. in their concluding remarks tacitly recognize this possibility stating that ‘patients who experience cardiovascular instability during intra-dialytic exercise may benefit from exercise programmes in the inter-dialytic interval’.

Their second major point was that exercise-induced changes in the F-cell ratio provide a possible explanation for the discrepancy between the changes in haemoglobin and total protein concentrations we reported. We agree. We alluded to this possibility both in the paper’s introduction and in the discussion but concluded that ‘while it is not possible to exclude …an effect on RBV of haematocrit redistribution, we have not found any direct evidence. . .’. We still do not feel that the small differences we found justify a more definite conclusion.

We wholeheartedly concur with Dasselaar et al.’s remarks that ‘exercise during haemodialysis should be promoted due to it positive effects. . .’. Our paper described haemodynamic responses during exercise on haemodialysis comparable with those seen during exercise in normal individuals. In our view, this enhances the case for intra-dialytic exercise.

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Trends in coronary artery calcification in peritoneal dialysis and transplant patients

Sir,

In a recent issue of Nephrology Dialysis and Transplantation, an interesting paper by Moe and co-workers was published that has shown a substantial difference in trends of coronary artery calcification (CAC) progression between patients remaining on dialysis and those who underwent renal transplantation (RTx) [1]. We would like to share our experience on this topic.

We performed CAC assessment using multi-slice spiral computed tomography (MSCT; Somatom Plus 4 Volume Zoom, Siemens AG, Erlangen, Germany) in 61 patients (28 female, 33 male, mean age 50.4 ± 13.6 years, on dialysis for a period of 17.5 ± 19.8 months) treated exclusively with peritoneal dialysis (PD). After 12 months, CAC evaluation was repeated in 60 patients. In addition, the broad-spectrum risk profile of biochemical markers that may impact on development and progression of atherosclerosis and calcification was repeatedly analysed. During the mentioned period, one patient died, 10 underwent renal transplantation (PD/RTx), 47 remained on PD and three were transferred to haemodialysis (HD). The comparison was performed between patients continuing PD and those who were transplanted. Unlike in the study of Moe et al., the baseline CAC assessment was performed in PD/RTx patients not at the time of RTx, but preceding it by 5.6 ± 2.3 months; the second MSCT followed RTx after 6.4 ± 3.5 months.

The coronary artery calcification score (CaSc) for the whole group (PD and PD/RTx patients) equalled 298.9 ± 805.2 Agatston units (median 11.5, range 0–550.2) at baseline and increased to 361.9 ± 813 units (median 25.8, range 0–5001.3) after 1 year (P < 0.05), with a ΔCaSc of 62.9 ± 281.4 units (median 0, range −501 to +1368). Among patients who remained on PD, CaSc increased from 355.9 ± 877.2 (median 22.6, range 0–550.2) to 435.4 ± 879.2 units (median 84.0, range 0–5001.3) (P < 0.05), with a ΔCaSc of 79.5 ± 307 units (median 0, range −501 to +1368). In contrast, among PD/RTx patients, CaSc decreased from 31.2 ± 66.9 (median 0, range 0–197.4) to 16.3 ± 34.6 units (median 0, range 0–90.3) (P = NS), with a ΔCaSc of −14.3 ± 33.9 units (median 0, range −107.1 to 0). The difference in CAC between subjects that were transplanted and those who remained on PD was already significant at baseline (P = 0.04), and became more pronounced on follow-up (P = 0.004). When comparing the risk profile between the two groups at study initiation, we found significantly lower values of C-reactive protein (CRP; P < 0.001), interleukin-6 (IL-6; P < 0.0001) and tumour necrosis factor-α (TNF-α; P < 0.05) in patients who were further transplanted. Patients who underwent transplantation were significantly younger (39.4 ± 11.2 vs 52.7 ± 12.8 years; P < 0.005) and displayed fewer co-morbid conditions. After 12 months, the differences became even more pronounced in terms of CRP, IL-6 and TNF-α and, in addition, became significant for leptin, fibrinogen, phosphates and Ca × P product.

The results obtained are largely consistent with those of Moe et al. As in their study, our patients on dialysis substantially progressed in CAC, whereas no change was noted in PD/RTx subjects (although they benefited from RTx for <6.5 months on average and were additionally exposed to dialysis between two CAC assessments). Similarly, all patients without calcification at baseline remained calcification-free on follow-up, regardless of the group (14 among PD patients and seven among those transplanted later).

As can be concluded from the previous publication of Moe et al., patients who were transplanted and those who remained on dialysis until repeated CAC assessment differed significantly in baseline characteristics. RTx patients were younger, had spent less time on dialysis and were more frequently treated with PD (P < 0.005); there were also fewer smokers and Blacks in this group. Some significant differences were also present in terms of biochemical and inflammatory parameters between the two groups [2].

Moe and co-workers do not attempt to separate the impact of transplantation itself from the influence of baseline differences observed between the groups and provide no speculation on this issue. Although the authors conclude that CAC stabilizes in subjects receiving RTx, they do not discuss whether RTx itself is responsible for this (influence of a functioning kidney), or whether stabilization results from the mentioned differences in risk profile. The difference in CAC between the originally included groups was insignificant, although CAC tended to be higher in the dialysis group. Similarly, more CAC-free patients were among the RTx group (37 vs 21%), although this difference was also insignificant [2]. In the present study the Δ value for the difference in CAC at baseline and follow-up between groups is not provided. Presuming it was significant or borderline sig-
nificant, and in the context of a strong association between baseline CAC and ΔCaSc in the dialysis group, it seems likely that the baseline profile of the RTx group (including baseline CAC) is at least equally important for stabilizing CAC progression.

Data from the literature suggest that the impact of RTx on the vascular system should be interpreted with caution. De Lima et al. demonstrated substantial improvement in echocardiographic parameters in end-stage renal disease (ESRD) patients undergoing RTx, whereas they failed to document a similar amelioration within the vascular system when using common carotid artery ultrasound [3]. Unfortunately, no other results can be discussed regarding CAC after RTx, as the study of Moe et al. and possibly this letter seem to remain the only available data.

In conclusion, we can cite and uphold Moe et al.’s finding that ‘coronary artery calcification stabilizes in subjects who had received a functioning renal allograft’, although whether the observed amelioration is the true effect of RTx, or is biased by a substantial difference in baseline risk profile remains to be clarified.

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Reply

Sir,

We thank Dr Stompor and colleagues for their comments regarding our publication. We agree with them that our findings of a lack of progression of vascular calcification in the transplant recipients could be due to more ‘favourable’ patient characteristics (younger, less time on dialysis) rather than a true effect of transplant. It was for that very reason that we purposely did not attempt to directly compare patients who remained on dialysis with those who underwent renal transplantation.

The similarity in the observation of no progression in transplant recipients cited in their letter, despite the follow-up representing a mixed dialysis and transplant treatment period, is encouraging. We agree, larger studies are needed to confirm these observations.

In addition we would like to note two errors in the published manuscript: The author, Dr Martina Reslerova’s name was mis-spelled, and the p-value in the legend of Table 1 is 0.085 not 0.85. The p value in the text is correct.

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