However, we are fortunate to work in a large teaching hospital with both major centres for viral hepatitis and HIV and a large liver transplant programme and have experienced transplanting patients with hepatitis B, hepatitis C and HIV. Serving a ethnically diverse population, we have numerous patients who regularly dialyse abroad, and suspending all such patients for 3 months, as suggested, would not only increase the transplant assessment workload, but in practice would lead to otherwise healthy patients remaining suspended for >3 months. As such, we would advocate an intensive surveillance programme post-transplantation for patients deemed at risk. Importantly, the diagnostic window period for the detection of hepatitis C can be reduced on patients deemed at risk. Importantly, the diagnostic window period for the detection of hepatitis C can be reduced on return to the unit, to ~20 days, by hepatitis C RNA testing [3], thus allowing more rapid reinstatement on the transplant list for those units who currently suspend their holiday dialysis patients.

Conflict of interest statement. None declared.

Royal Free Hospital and Royal Free University College Medical School

Anna Maria Geretti
Andrew Davenport

E-mail: a.geretti@medsch.ucl.ac.uk

2. Good Practice Guidelines for Renal Dialysis/Transplantation Units. Prevention and Control of Blood-borne Virus Infection: 95 Department of Health (UK): Recommendations of a working group convened by the Public Health Laboratory Service (PHLS) on behalf of the Department of Health, 2002

doi: 10.1093/ndt/gfn113

Advance Access publication 29 February 2008

Decreased access resistance in haemodialysis patients with upper arm arteriovenous fistulae

Sir,

We have read with interest the recent publication of Basile et al. [1], who identified high vascular access flow (Qa) as a risk factor for high output failure in haemodialysis patients. Furthermore, they reported that the cardiopulmonary recirculation (CPR, Qa/CO ratio) is higher in upper arm arteriovenous fistulae (AVFs) than in forearm AVFs. We confirm these findings and made relevant additional observations in our haemodialysis population (1) by validating the transonic CO measurement technique, (2) by including a group of patients with PTFE graft and (3) by calculating the access resistances.

Because quantification of CO by the transonic technique has not yet been validated against another modality, we compared this technique to echocardiography (using standard transthoracic two-dimensional and Doppler echocardiographic recordings). The measurements were performed during the first hour of a dialysis session, in random order and shortly after each other. We compared the techniques in 35 stable haemodialysis patients. The correlation coefficient was high ($r = 0.95, P < 0.001$), and Bland–Altman analysis showed no regression of the differences versus the means ($r = 0.03, Figure 1$), indicating that measurements obtained with both techniques do not differ significantly.

Secondly, we wished to address the idea that the finding that CO is higher in upper arm AVF is indeed explained by the AVF and not by a higher systemic blood pressure or a lower systemic resistance. In the study of Basile et al. [1] no blood pressure values were presented. We measured CO, Qa and mean arterial pressure (MAP, contralateral arm) in 32 patients with upper arm AVFs, 39 with forearm AVFs and 15 with PTFE grafts. Subsequently, we calculated total peripheral vascular resistance (TPVR = MAP/CO), access resistance (AR = MAP/Qa) and systemic vascular resistance (SVR = MAP/(CO – Qa)).

Similar to the findings of Basile et al. [1], the relation between CO and Qa could be described by a third-order polynomial ($y = 3.2 + 2.0x + 0.44x^2 - 0.20x^3; R^2 = 0.45, P < 0.001$). As shown in table 1, Qa, CO, CPR and Qa adjusted for CO (by analysis of covariance) were significantly higher in the AVF upper arm group than in both the AVF forearm and the PTFE groups, whereas AR was significantly lower (one-way ANOVA, studentized Newman–Keuls post hoc test). In contrast, MAP and SVR were almost numerically equal in the three groups, being far from significantly different.

We validated the transonic CO technique, and we confirm and extend the findings presented by Basile et al. [1] by showing that the higher CO in upper arm AVF is a result of the lower TPVR, caused by a lower AR and not by a lower SVR.

Conflict of interest statement. None declared.

Table 1. Shunt flows and resistances of haemodialysis patients with various shunt types

<table>
<thead>
<tr>
<th>AVF upper arm</th>
<th>AVF forearm</th>
<th>PTFE graft</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>32</td>
<td>39</td>
</tr>
<tr>
<td>Qa (L/min)</td>
<td>1.8±0.2</td>
<td>1.0±0.1*</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>6.6±0.3</td>
<td>5.3±0.3†</td>
</tr>
<tr>
<td>CPR (ratio)</td>
<td>0.28±0.02</td>
<td>0.17±0.01*</td>
</tr>
<tr>
<td>Adjusted Qa (L/min)</td>
<td>1.5±0.1</td>
<td>1.0±0.1*</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>95±3</td>
<td>91±3</td>
</tr>
<tr>
<td>TPVR (mmHg L⁻¹)</td>
<td>16±1</td>
<td>19±1</td>
</tr>
<tr>
<td>AR (mmHg L⁻¹)</td>
<td>71±9</td>
<td>142±16*</td>
</tr>
<tr>
<td>SVR (mmHg L⁻¹)</td>
<td>23±2</td>
<td>23±1</td>
</tr>
</tbody>
</table>

Significances versus AVF upper arm group: *P < 0.001; †P < 0.01; P < 0.02.

Qa adjusted by analysis of covariance for variations related to variations in CO.


Conflict of interest statement. None declared.
Fig. 1. Bland–Altman plot of CO measured by transonic and Doppler echo techniques.

Departments of Nephrology and Cardiology, University Medical Center Utrecht, PO Box 85500 3508 GA Utrecht, The Netherlands

Wil A.M.A. van der Mark
Peter Boer
Maarten J.M. Cramer
Peter J. Blankestijn

E-mail: P.J.Blankestijn@umcutrecht.nl

doi: 10.1093/ndt/gfn016

Advance Access publication 20 February 2008

Reply

Sir,

We read with interest the letter by Blankestijn et al. in response to our article [1]. We are very grateful to them for their letter, which confirms our findings as far as at least two points are concerned: (1) cardiopulmonary recirculation is higher in upper arm arteriovenous fistulae (AVFs) than in forearm AVFs; (2) a third-order polynomial regression model fits best the relationship between vascular access flow (Qa) and cardiac output. Furthermore, they found a decreased access resistance (AR) in upper arm AVFs. This finding is actually not unexpected if we take into account that Poiseuille's law states that the blood flow in any vessel, and therefore also Qa, is determined by the following relationship [1]:

\[ Qa = \frac{\pi \Delta P r^4}{8\eta l} \]

where \( \Delta P \) is the pressure difference between the extremities of the vessel, \( r \) the radius of the vessel, \( \eta \) the viscosity of the fluid and \( l \) the length of the vessel. Now, we know that AR is expressed by the ratio

\[ AR = \frac{Mean\ arterial\ pressure\ (MAP)}{Qa}. \]

We can rewrite this relationship in the following way:

\[ AR = MAP \times \frac{8\eta l}{\pi \Delta P r^4}. \]

It is clearly evident that, among all factors involved, \( r \) (at power 4) of a vessel plays the most important role in determining AR. Now, the brachial artery utilized for an upper arm AVF must necessarily have a higher \( r \) than the radial artery utilized for a lower arm AVF. Consequently, the AR of an upper arm AVF must be evidently lower than that of a forearm AVF.

In conclusion, we think that the data presented by van der Mark et al. (with an elegant statistical inference) confirm our previous findings and that the evaluation of the peripheral, systemic and access resistance provides further interesting explanations about the determinism of the haemodynamic processes occurring in an AVF.

Conflict of interest statement. None declared.