

Original Article

## The relationship between the flow of arteriovenous fistula and cardiac output in haemodialysis patients

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### Abstract

**Background.** Satisfactory haemodialysis (HD) vascular access flow (Qa) is necessary for dialysis adequacy. High Qa is postulated to increase cardiac output (CO) and cause high-output cardiac failure. Aim of the present prospective study was to evaluate the relationship between Qa of arteriovenous fistulas (AVFs) and CO in order to have a closer insight into this scarcely explored aspect of HD pathophysiology.

**Methods.** Ninety-six patients bearing an AVF entered the study. All were evaluated a priori for the existence of cardiac failure according to the functional classification of the American College of Cardiology/American Heart Association task force. Qa and CO were measured by means of the ultrasound dilution Transonic Hemodialysis Monitor HD02.

**Results.** The mean Qa of the 65 lower arm AVFs was  $0.948 \pm 0.428$  SD l/min, whereas that of the 31 upper arm AVFs was  $1.58 \pm 0.553$  l/min. The difference was statistically significant ( $P < 0.001$ ). Ten patients were classified as having high-output cardiac failure; seven of them bore an upper arm AVF. Thus, upper arm AVFs were associated with an increased risk of high-output cardiac failure ( $P < 0.04$ ,  $\chi^2$  test). A third-order polynomial regression model best fitted the relationship between Qa and CO. The analysis of the regression equation identified 0.95 and 2.21/min as Qa cut-off points. The receiver operating characteristic curve analysis showed that Qa values  $\geq 2.01$ /min predicted the occurrence of high-output cardiac failure more accurately than two other Qa values (sensitivity 89%, specificity 100%, curve area 0.99) and three Qa/CO ratio values (cardio-pulmonary recirculation—CPR). The better performance among the latter was that of CPR values  $\geq 20\%$  (sensitivity 100%, specificity 74.7%, curve area 0.92).

**Conclusions.** Our prospective study shows that the relationship between Qa of AVFs and CO is complex

and a third-order polynomial regression model best fits this relationship. Furthermore, it is the first study to clearly show the high predictive power for high-output cardiac failure occurrence of Qa cut-off values  $\geq 2.01$ /min.

**Keywords:** arteriovenous fistula—cardiac failure—cardiac output—haemodialysis—ultrasound dilution

### Introduction

Satisfactory haemodialysis (HD) vascular access flow (Qa) is necessary for dialysis adequacy. On the one hand, low Qa are indicative of access dysfunction; on the other hand, high Qa are postulated to increase cardiac output (CO) and cause high-output cardiac failure. The latter is defined as symptoms of cardiac failure (dyspnoea either at rest or with varying degrees of exertion, orthopnoea, paroxysmal dyspnoea and oedema, either pulmonary and/or peripheral) in the presence of an above-normal cardiac index (CI) ( $>3.01$ /min/m<sup>2</sup>) [1]. There is a paucity of literature regarding high-output cardiac failure in HD patients other than a few case reports [2–4].

Krivitski [5] has developed an ultrasound dilution technique to measure Qa (Transonic Hemodialysis Monitor HD02, Transonic Systems Incorporated, Ithaca, NY, USA). There are published data supporting efficacy of Transonic for the regular monitoring of Qa of vascular accesses for HD [5,6]. The same ultrasound dilution technique can also be used to measure CO [7]. The relative dependency of Qa and CO on each other is unknown. At one end of the spectrum, one may hypothesize that Qa drives CO. On the other hand, CO must impact on Qa. For example, if CO were zero, so too would be Qa [8].

Currently, there is no definition of when a Qa is too high either in National Kidney Foundation–Kidney Disease Outcomes Quality Initiative guidelines or in the literature. The concept of using a ratio of Qa to CO

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(cardio-pulmonary recirculation—CPR) has been put forth by Pandeya and Lindsay [8] in their study of stable long-term HD patients. They found that the average Qa was 1.61/min and the average CO was 7.21/min, thus describing an average CPR of 22% [8]. The Vascular Access Society guidelines define an arteriovenous Fistula (AVF) with a high Qa as that having a Qa of 1.0–1.51/min and a CPR >20% [9].

Aim of the present prospective study was to evaluate the relationship between Qa of AVFs and CO in order to have a closer insight into this scarcely explored aspect of HD pathophysiology.

## Subjects and methods

### Study protocol

After written informed consent, all HD patients treated in our Dialysis Unit and bearing an AVF in whom the tests described subsequently could be technically feasible, were enrolled into the present study. Ninety-six patients entered the study. All patients were evaluated a priori for the existence of cardiac failure according to the functional classification of the American College of Cardiology/American Heart Association task force on practice guidelines [10]. Heart failure was classified into four stages (A, B, C and D). Briefly, stage A is that of patients at high risk for heart failure but without structural heart disease or symptoms of heart failure. Such patients have no identified structural or functional abnormalities of the pericardium, myocardium or cardiac valves and have never shown signs or symptoms of heart failure; stage B is that of patients who have developed structural heart disease that is strongly associated with the development of heart failure (e.g. previous myocardial infarction, left ventricular remodelling including left ventricular hypertrophy—LVH—or asymptomatic valvular disease) but without signs or symptoms of heart failure; stage C is that of patients who have current or prior symptoms of heart failure (e.g. shortness of breath, fatigue and reduced exercise tolerance) with structural heart disease, such as those described earlier; stage D is that of patients with refractory heart failure requiring specialized interventions [10]. The evaluation and classification into the four stages of heart failure of patients enrolled into the study was blind and a priori with respect to the tests which they underwent in the following weeks. Stage C classification does not immediately imply high-output cardiac failure. The latter requires the presence of an above-normal cardiac index (CI) (>3.01/min/m<sup>2</sup>) [1].

Measurements were performed on a mid-week dialysis. Qa was evaluated by means of the ultrasound dilution Transonic Hemodialysis Monitor HD02 as previously described [5]. For the measurement of Qa the blood lines were reversed in a sterile manner and a temporary recirculation was created. Qa was determined as the average of three separate measurements taken approximately 5–10 min apart during the first 30 min of dialysis session. In short, a heated (37°C) bolus of 10 ml NaCl 0.9% (indicator) is injected into the venous line with the blood pump rate set at 200 ml/min, and the change in velocity of ultrasound waves produced by the returning dilution curve (S) is detected by a probe attached to the arterial line. By comparing the dilution curve S with a

calibration curve (Scal) produced by injecting 30 ml of isotonic saline in the venous bubble trap, CO is calculated by the formula:  $3 \times \text{blood flow} \times (S/\text{Scal})$  [7]. CPR is Qa/CO ratio. All tests throughout the entire study were performed by the same operator. Furthermore, 24 patients underwent a second Qa and CO measurement 1 week apart with the same modalities described earlier.

### Statistics

The distribution of the data was studied by means of the Kolmogorov–Smirnov test. The relationship between Qa and CO was studied by means of the bivariate analysis of the best-fit model. In case of non-linear best-fit model the analysis of the regression equation should be made in order to calculate the Qa values (cut-off points) where the CO trend significantly changed its slope (points of maximum, minimum or flex of the function). Then, the one-way ANOVA followed by the Tukey's post-hoc test was performed in order to compare the mean CO and CPR values in each Qa category identified by the cut-off points previously calculated. The comparison of the continuous variables between groups was made by means of the Student's *t*-test for unpaired data, while the  $\chi^2$  test was utilized for the distributions between groups of the categorical variables. Finally, the sensitivity, specificity and accuracy of Qa and CPR values in identifying the patients with high-output cardiac failure were assessed by means of the receiver operating characteristic (ROC) curve analysis. All statistical inferences were performed with the use of the SPSS software package, version 10 (SPSS Inc., Chicago, IL, USA). Data were expressed as means  $\pm$  SD, median and ranges and values of  $P < 0.05$  were assumed as statistically significant.

## Results

Demographic, clinical and haemodynamic characteristics of the 96 patients enrolled in the study are reported in Tables 1 and 2. Sixty-five patients had a lower arm AVF (44 distal radio-cephalic and 21 middle-arm) and 31 patients had an upper arm AVF (18 brachio-basilic, 8 brachio-cephalic and 5 Gracz). The percentage of diabetics, the gender distribution, the age and the dialysis vintage were not different when comparing the group with lower arm AVFs with the group with upper arm AVFs (Table 1). The mean Qa of the 65 lower arm AVFs was  $0.948 \pm 0.428$  SD l/min, whereas that of the 31 upper arm AVFs was  $1.58 \pm 0.553$  l/min. The difference was statistically significant ( $P < 0.001$ ) (Table 1). Similarly, CO and CPR were significantly higher in the group with upper arm AVFs than in the group with lower arm AVFs (for both,  $P < 0.001$ ) (Table 1). The blind and a priori evaluation of the 96 patients enrolled in the study led to their stratification into the four stages of heart failure (Table 2). Ten patients were classified as having stage C cardiac failure (Table 2 and Figure 1) [10]. Furthermore, the latter could also be classified as high-output cardiac failure [1] because CI was  $4.73 \pm 0.481$  l/min/m<sup>2</sup> (range 3.8–5.5) [Qa was  $2.31 \pm 0.321$  l/min (range 2.0–2.8) and CO was

**Table 1.** Demographic, clinical and haemodynamic characteristics of the 96 patients enrolled in the study subdivided by location of their AVFs

	Lower arm AVFs (no. 65)	Upper arm AVFs (no. 31)
Age (years)	59.6(9.4)/61 (37–72)	66.4 (11.0)/70 (44–82)
Gender (males)	53.1	55.3
Diabetes mellitus	13.5	16.1
Dialysis duration (months)	56.4 (40.0)/42 (6–154)	60.0 (43.2)/50 (12–196)**
Qa of AVFs (l/min)	0.948 (0.428)/0.890 (0.280–2.0)	1.58 (0.553)/1.34 (0.760–2.8)*
Cardiopulmonary output (CO, l/min)	5.6 (0.8)/5.8 (3.6–8.3)	6.9 (1.1)/6.8 (4–10)*
Cardiopulmonary recirculation (Qa/CO,%)	17 (5)/16 (4–27)	23(5)/23 (11–35)*

Continuous variables are expressed as mean (SD)/median (range) while categorical data as percentages.

\* $P < 0.001$ ; \*\* $P =$  not significant (Student's t-test for unpaired data).

**Table 2.** Demographic, clinical and haemodynamic characteristics of the 96 patients enrolled in the study according to the classification of the American College of Cardiology/American Heart Association task force [10]

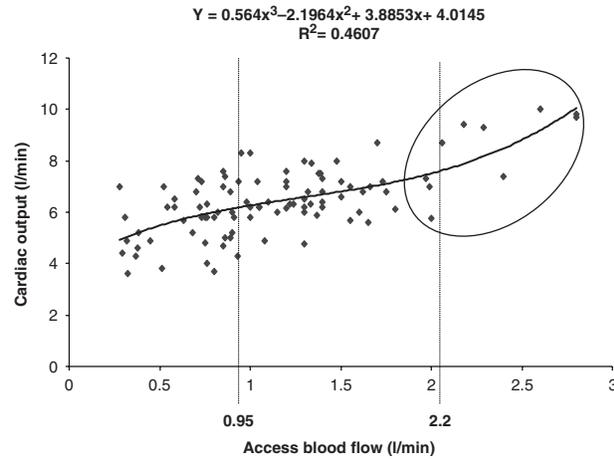
Patients with stage A heart failure [(no.(lower arm/upper arm)]	30 (28/2)		
Patients with stage B heart failure [(no.(lower arm/upper arm)]	56 (34/22)		
Patients with stage C heart failure [(no.(lower arm/upper arm)]	10 (3/7)***		
Patients with stage D heart failure [(no.(lower arm/upper arm)]	0		
		Stage C of heart failure (no. 10)	Stages A and B of heart failure (no. 86)
Mean age (years)	70.8 ± 8.3		62.0 ± 10.0*
Mean HD duration (months)	73.0 ± 43.0		56.5 ± 41.4**
Mean cardiac output (l/min)	8.4 ± 1.5		6.2 ± 1.1#
Mean Qa of AVFs (ml/min)	2.3 ± 0.3		1.0 ± 0.4#
Mean AVF duration (months)	39.0 ± 13.7		31.5 ± 21.4**

$P < 0.01$ ; \*\* $P =$  not significant; # $P < 0.0001$ . Student's t-test for unpaired data was used.

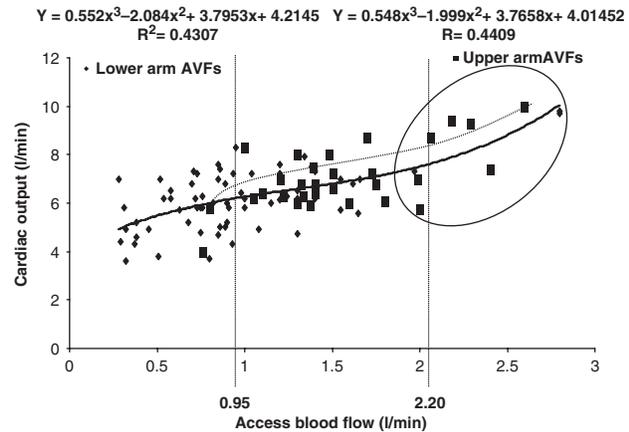
$P < 0.04$ .  $\chi^2$  test was used.

8.43 ± 1.46 l/min (range 5.7–10.0)] (Table 2 and Figure 1). Seven of the 10 patients bore an upper arm AVF. Thus, upper arm AVFs were associated with an increased risk of high-output cardiac failure ( $P < 0.04$ ,  $\chi^2$  test). The ten patients with stage C (and high-output) cardiac failure were significantly older than the other patients ( $P < 0.01$ ); in contrast, their dialysis vintage and their AVF duration were non-significantly different from that of patients without cardiac failure (Table 2).

Coefficient of variation of Qa and CO between subsequent measurements in 24 patients was 4.6 and 8.9%, respectively. A third-order polynomial

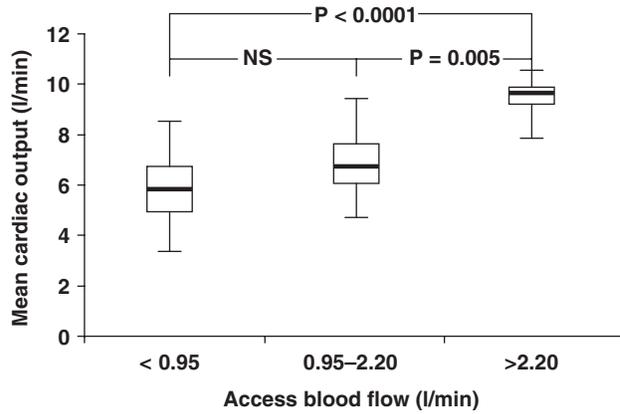


**Fig. 1.** A third-order polynomial regression model best fitted the relationships between vascular access flow and cardiac output in the 96 patients. The ellipse encompasses 10 patients who were classified as affected by stage C cardiac failure (according to reference 10) and by high-output cardiac failure (according to reference 1).

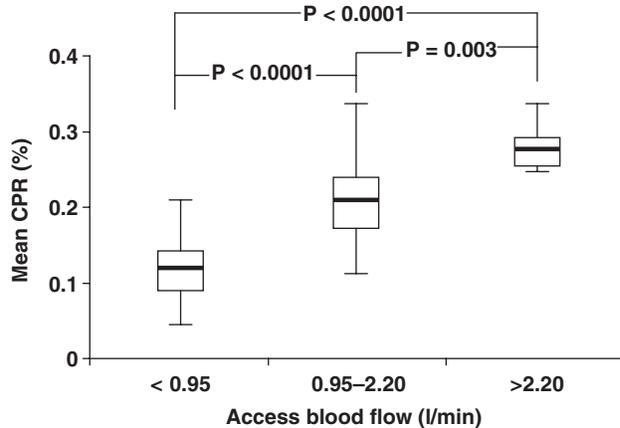


**Fig. 2.** A third-order polynomial regression model best fitted the relationships between vascular access flow and cardiac output, respectively in lower arm AVFs (filled diamond, continuous line) and upper arm AVFs (filled square, dotted line). The ellipse encompasses 10 patients who were classified as affected by stage C cardiac failure (according to reference 10) and by high-output cardiac failure (according to reference 1).

regression model best fitted the relationship between Qa and CO (Figure 1). Also when splitting out the 96 patients between those with a lower arm AVF and those with an upper arm AVF, a third-order polynomial regression model best fitted the relationships between Qa and CO (Figure 2). The ellipses in Figures 1 and 2 encompass the 10 patients who were classified as affected by stage C cardiac failure (according to [10]) and by high-output cardiac failure (according to [1]). The analysis of the regression equation of Figure 1 identified 0.95 and 2.21/min as Qa cut-off points. Actually, the regression curve was graphically characterized by three slopes, the latter being steeper than the first and the second, which presented a plateau (Figure 1). The one-way

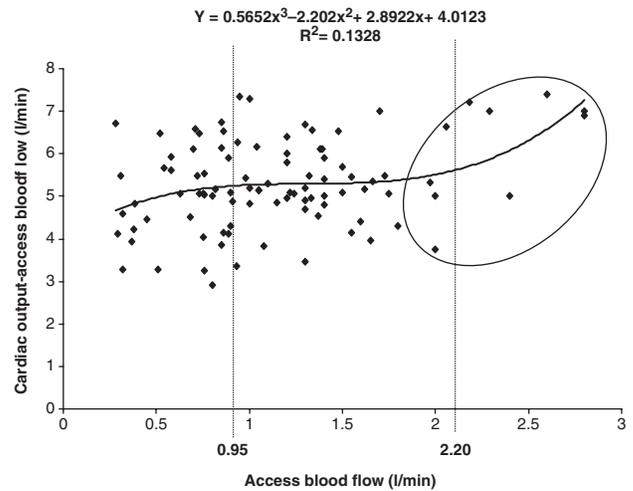


**Fig. 3.** Comparison of the cardiac output values among the patients subdivided according to the vascular access flow cut-off values. The horizontal line represents the median, upper and lower limits of the box including the first and third quartiles, and capped bars indicate minimum and maximum value. The one-way ANOVA followed by the Tukey's post-hoc test was performed in order to compare the mean cardiac output values in each access blood flow category identified by the cut-off points previously calculated.



**Fig. 4.** Comparison of the cardio-pulmonary recirculation (CPR) values among the patients subdivided according to the vascular access flow (Qa) cut-off values. The horizontal line represents the median, upper and lower limits of the box including the first and third quartiles, and capped bars indicate minimum and maximum value. The one-way ANOVA followed by the Tukey's post-hoc test was performed in order to compare the mean CPR values in each Qa category identified by the cut-off points previously calculated.

ANOVA followed by the Tukey's post-hoc test demonstrated that patients with Qa values  $>2.21$ /min had a significantly higher mean CO ( $9.24 \pm 0.71$ /min) than patients with Qa values both  $<0.95$  ( $5.74 \pm 0.71$ /min;  $P < 0.0001$ ) and Qa values ranging from 0.95 to 2.21/min ( $6.76 \pm 0.71$ /min;  $P = 0.005$ ) (Figure 3). A non-statistically significant difference in mean CO was shown by patients having Qa values  $<0.95$  when compared with that of patients with Qa values ranging from 0.95 to 2.21/min (Figure 3). On the contrary, CPR values significantly increased across the Qa categories, the highest being for values  $>2.21$ /min ( $<0.95$ :  $12 \pm 4\%$ ,  $P < 0.0001$ ;  $0.95-2.2$ :  $21 \pm 5\%$ ,  $P = 0.003$ ;  $>2.2$ :  $28 \pm 3\%$ ) (Figure 4).



**Fig. 5.** Best-fit regression model of the relationship between vascular access flow and the systemic flow (cardiac output - access blood flow). The ellipse encompasses 10 patients who were classified as affected by stage C cardiac failure (according to reference 10) and by high-output cardiac failure (according to reference 1).

**Table 3.** Accuracy, sensitivity, specificity and curve area in identifying patients with high-output cardiac failure among three cut-off values of vascular access flow (Qa) and three cut-off values of cardio-pulmonary recirculation (CPR) (ROC curve analysis)

	Qa (l/min)			CPR (%)		
	1.94	2.0	2.2	20	27	30
Accuracy (%)	99	99	95.8	77.9	79.6	91.7
Sensitivity (%)	97	89	55.6	100	55.6	22.2
Specificity (%)	98.9	100	100	74.7	93.1	98.9
Curve area	0.98	0.99	0.833	0.92	0.85	0.76

Figure 5 shows the best-fit regression model of the relationship between Qa and the systemic flow (CO-Qa): there is such a wide dispersion of systemic flow data for a given Qa that a very low value of  $R^2$  (0.13) results, showing very clearly that there is no relationship between the systemic flow and Qa.

The predictive power for high-output cardiac failure occurrence of three different values of Qa and CPR was explored. They were derived from either our data or literature: Qa values chosen were 1.94 (the lowest Qa value among the Qa values of the 10 patients classified as stage C of heart failure), 2.0 [11] and 2.21/min (the second Qa cut-off point); CPR values chosen were 20% [9], 27% (it is the CPR of the AVF with Qa of 1.941/min) and 30% [8]. The ROC curve analysis showed that Qa values  $\geq 2.01$ /min predicted the occurrence of high-output cardiac failure more accurately than the two other Qa values (sensitivity 89%, specificity 100%, curve area 0.99) and the three CPR values (Table 3). The better performance among the latter was that of CPR values  $\geq 20\%$  (sensitivity 100%, specificity 74.7%, curve area 0.92) (Table 3).

## Discussion

CO increases greatly and immediately on opening an AVF in experimental models [12]. This increase in CO is achieved by means of a reduction in peripheral resistance, an increase in sympathetic nervous system activity (increasing contractility) and an increase in stroke volume and heart rate [12]. Therefore, the presence of an AVF lowers systemic vascular resistance, resulting in an increase in stroke volume and CO in order to maintain blood pressure [13]. Circumferential wall stress, calculated with the same value of mean blood pressure ( $96 \pm 14$  mmHg) on the radial artery feeding the AVF and on the contralateral radial artery taken as a control, is significantly increased on the AVF side [14]. A 6-fold increase in mean blood flow is observed on the AVF side compared with the contralateral side [14]. The flow of brachial artery as measured by Duplex Doppler ultrasonography is a reliable expression of Qa of a distal AVF [15]. The mean brachial artery flow increases from  $56.2 \pm 20.0$  ml/min before the creation of an AVF to  $365.0 \pm 129.3$  ml/min 1 day after and to  $720.4 \pm 132.8$  ml/min 28 days after the creation of an AVF [15]. A prospective short-term echocardiographic study was performed in order to assess the influence that the creation of an AVF exerted on cardiac function [14]: a significant elevation in left ventricular end-diastolic diameter (+4%), fractional shortening (+8%) and CO (+15%) occurred when comparing data obtained immediately before and 14 days after the creation of an AVF [16].

In the long run, the AVF of a HD patient can be considered very similar to a 'physiological fistula' created in endurance trained older men [17]: cardiac adaptations are characterized by volume-overload LVH in the latter [17]. Unfortunately, the only way to determine if AVF creation produces LVH in HD patients is with a prospective randomized study comparing a central venous catheter with an AVF, and examining the serial changes in left ventricular dimensions and thickness. Due to the increased morbidity and mortality associated with catheters, this is unlikely to occur [18]. However, several studies showed regression of LVH following AVF closure in renal transplant patients [19–21]. In transplant patients followed for a longer period of time after AVF closure (up to 21 months in one study) [19] there is a regression of eccentric LVH, which implies a reduction in volume overload. What cause(s) is able to transform a volume-overload LVH in cardiac failure is not known. Specific characteristics of either the patients or the AVFs, or both, may pre-dispose to the development of heart failure [22]. Some suggest that cardiac decompensation due to AVFs is likely to only occur in patients with underlying cardiac disease [23].

The risk factors for the development of high Qa AVFs are male gender, upper arm AVFs and previous access surgery [24]. Patients with high-flow AVFs most likely have a greater risk of developing high-output

cardiac failure and are also likely to have greater increases in left ventricular end-diastolic volume (LVEDV) [22]. Preliminary data suggest a trend to increased LVEDV in patients with Qa of AVFs  $>2.01$ /min compared with those with lower Qa ( $<1.01$ /min) [11]. Currently, the evidence linking AVFs to the development of heart failure is indirect, but consistent with what is known about high-output cardiac failure in other kinds of conditions, such as traumatic AVFs. What happens to renal plasma flow in high-output heart failure? We have very few experimental data: a rat model of high-output heart failure due to the creation of an AVF between the aorta and inferior vena cava below the renal arteries led to a reduced effective renal plasma flow [25].

Our data show that the relationship between Qa of AVFs and CO is quite complex: a plateau is present in between two steeper slopes (Figure 1); it is of such a relevance that the mean CO in the Qa range  $0.95$ – $2.21$ /min was not statistically significantly different from the mean CO in the Qa range  $<0.95$ /min (Figure 3). Furthermore, also the relationship between Qa and the systemic flow (CO–Qa) is not linear, as one would expect if the hypothesis is that Qa drives CO. The mechanisms underlying this phenomenon, i.e. why the increase in Qa is not accompanied by a parallel increase in CO and in systemic flow of proportional dimensions, are far from being clear. We can only hypothesize a sort of myocardial functional reserve and, then, of a myocardial adaptation which is able to make the prevalence of high-output cardiac failure in our HD patients (10.4%) much more rare compared with what one may reasonably imagine, when taking also into account the high prevalence of upper arm AVFs (32.3%).

The novel finding of our prospective study is the empirical demonstration of the high predictive power for high-output cardiac failure occurrence of Qa cut-off values  $\geq 2.01$ /min. Even though highly statistically significant, our analysis was based on only 10 patients with stage C heart failure. Thus, more extensive data are needed before accepting these results in the clinical practice.

The other relevant finding of our study, which is, however, not a novelty [26], is that upper arm AVFs are associated with an increased risk of high-output cardiac failure. Even though it must be acknowledged that lower arm AVFs are usually positioned in a type of patient with a different phenotype from those who get an upper arm AVF (among them, usually there are less diabetics, younger people with fewer vascular diseases and cardiac dysfunctions), the fact remains that such an association seems to favour the hypothesis of a causative role of the upper arm AVFs in the pathogenesis of high-output cardiac failure. Even though it is likely that only a small percentage of patients have overt stage D heart failure (one study pointed out that only 2.6% of patients with upper arm AVFs underwent banding or ligation owing to steal or high-output syndromes) [27], the message deriving from our study is clear: the upper extremity AVFs

should be placed as distal as possible, as also underlined by the very recent EBPG guidelines [28].

In conclusion, our prospective study shows that the relationship between Qa of AVFs and CO is complex and a third-order polynomial regression model best fits this relationship. Furthermore, it shows that upper arm AVFs are associated with an increased risk of high-output cardiac failure. Currently, there are no guidelines at what Qa or CPR to intervene to prevent high-output cardiac failure linked to high flow AVFs. Finally, it is the first study to clearly show the high predictive power for high-output cardiac failure occurrence of Qa cut-off values  $\geq 2.0$  l/min. Further prospective studies are required to confirm the biologically plausible concept that high-access flows (Qa  $\geq 2.0$  l/min) with elevated CPR ( $\geq 20\%$ ) may represent cardiac risk.

*Conflict of interest statement.* None declared.

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*Received for publication: 30.4.07*

*Accepted in revised form: 18.7.07*