

## *Acute and long term cardiovascular effects of basilic vein transposition in chronic renal failure patients*

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### **ABSTRACT**

#### **Acute and long term cardiovascular effects of basilic vein transposition in chronic renal failure patients**

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Factors affecting cardiac function in dialysis patients include arterial blood pressure, anemia, intravascular volume and the arteriovenous fistula (AVF). We investigated the acute and chronic effects of basilic vein transposition (mean upper arm brachial artery-basilic vein anastomosis) on both the cardiovascular system and the oxygen status.

Sixteen patients with end stage renal failure were enrolled in this study. Patients with heart failure, pericardial effusion or valvular heart disease were not included in the study. Echocardiography (preoperatively and six months after, stages 1 and 2 respectively) and a Swan-Ganz catheter (preoperatively) were used to assess the hemodynamic status during the phases of AVF construction. Flow measurements were made in the parts of the AVF system before, during and after the construction of the AVF. Moreover, at the same time phases blood sampling from the arterial line and the pulmonary artery catheter was performed, in order to assess oxygen and acid-base status.

Cardiac output, cardiac index, stroke volume, stroke volume index, left ventricular stroke work increased statistically significant 20 and 30min after the AVF construction and they returned to baseline at 40min after the procedure. On the contrary, systemic vascular resistance decreased statistically significant 20 and 30min after the operation and they remained decreased for the whole study

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period. No other differences were recorded regarding the hemodynamic, oxygen and acid-base status. As far as the echocardiographical findings are con-

cerned, ejection fraction showed a tendency to increase but not significantly in contrast to the interventricular septum which increased statistically significant in both systole and diastole ( $p < 0.01$ ). Posterior wall thickness decreased minimally in both systole and diastole. Regarding the AVF system diameters, there were no changes recorded during the study.

Basilic vein transposition in selected patients provides a well functioning AV fistula and is not an appreciable cause of circulatory or pulmonary congestion for at least a period of six months.

## INTRODUCTION

Brescia and colleagues were the first surgeons to describe radiocephalic fistula in the nondominant arm for hemodialysis<sup>1</sup>. This still remains the first choice procedure for vascular access in patients who require long term hemodialysis. Lack of suitable cephalic veins, led the surgeons to investigate alternative procedures, such as basilic vein transposition and prosthetic vascular access grafts (polytetrafluoroethylene [PTFEE])<sup>2</sup>.

Use of a brachio basilic arteriovenous (AVF) fistula with transposed basilic vein was first described in 1976 by Dagher<sup>3</sup>. Due to the subfascial localization of the vein, protecting it from venipuncture, basilic vein is usually available in the upper arm region. The primary patency of this fistula is about 60% to 70% and the long-term patency is similar to that of a graft<sup>4,5</sup>.

This special type of AVF is used, when other AVFs have failed or when the patient's vessels are not suitable for the construction of a distal AVF<sup>6,7,8</sup>. It is well known that many patients undergoing haemodialysis, usually show an in-

crease in cardiac output, which may be due either to the presence of AVF or to anaemia<sup>9-15</sup>.

Cardiac output and other hemodynamic parameters can be studied by both non-invasive techniques, i.e. echocardiography, as well as by invasive techniques e.g. by using a Swan-Ganz catheter.

Most literature studies have investigated the hemodynamic parameters during surgical occlusion or manual compression of AV fistula. Furthermore, there are not many reports on the acute and late effects after basilic vein transposition.

The purpose of this study was to evaluate immediate and long term (after six months) cardiac effects of basilic vein transposition, by using echocardiography (preoperatively and six months after the creation of AVF) and a Swan Ganz catheter perioperatively.

## MATERIAL AND METHODS

This study was carried out in 16 patients with end-stage renal disease (ESRD) in the Nephro-

logy Department (Hemodialysis Unit) of AHE-PA University Hospital, Thessaloniki, Greece. Informed written consent was taken from each patient. The institutional ethical committee on human research and the University thesis approval committee approved the study.

Patients were already on a hemodialysis program using a hemodialysis catheter. A two lumen (12Fr. X 6''-16cm) hemodialysis catheter (Arrow International) was already inserted in the jugular or subclavian vein, as their AVFs for hemodialysis were not functioning properly. The previous AVFs had been constructed in various sites in the upper limb e.g. between the radial artery and cephalic vein or brachial artery to cubital vein, but basilic vein transposition has never been performed in these patients.

All such patients were scheduled for an AVF construction using the basilic vein (basilic vein transposition); 6-12hrs preoperatively they underwent hemodialysis.

Patients with heart failure, pericardial effusion or valvular heart disease were not included in the study.

Pre-operative duplex ultrasound investigation (SSD-2000, Aloka Co. Ltd, Tokyo, Japan) was performed according to a standard protocol by experienced vascular technicians, measuring the diameters of brachial arteries and basilic veins in the upper arm.

An echocardiographic evaluation of cardiac function was performed within 6hrs after hemodialysis in order to achieve a water balance close to the "dry" weight. Shortly thereafter patients were transferred to the operating theater for the scheduled procedure (stage 1). Left ventricular ejection fraction (EF%), posterior wall of left ventricular during diastole (PLV-Wd), posterior wall of left ventricular during systole (PLVWs), left ventricular dimension during systole (LVDs), left ventricular dimension during diastole (LVDd), interventricular septum during systole (IVSs) and interventricular septum during diastole (IVSs) were measured and recorded.

On the day of the study, standard monitoring included electrocardiography (ECG), pulse oxymetry (SpO<sub>2</sub>) and invasive measurement of arterial pressure (IABP). In addition, a special thermodilution Swan-Ganz catheter (OptiQ SvO<sub>2</sub>/CCO Abbott Laboratories North Chicago, IL, USA) was inserted for monitoring of central filling pressures, continuous monitoring of cardiac output and SVO<sub>2</sub>. The catheter was inserted by using the pre-existing vascular access for hemodialysis through an introducer sheath (Seldinger method). Measurements were made at the individual phases of AVF construction (0-5): before (phase=0), during (phase=1) and 10 min (phase=2), 20 min (phase=3), 30 min (phases=4), 40 min (phase=5) min after construction of the AVF.

Flow measurements in the parts of the AVF system (1–2cm from anastomoses), were made by using a portable Doppler unit (ES-1000 SPM Smartdop, Koven Technology, probe P8 M05S8A, 8Hz) and a caliper meter, at phases 0, 1 and 2.

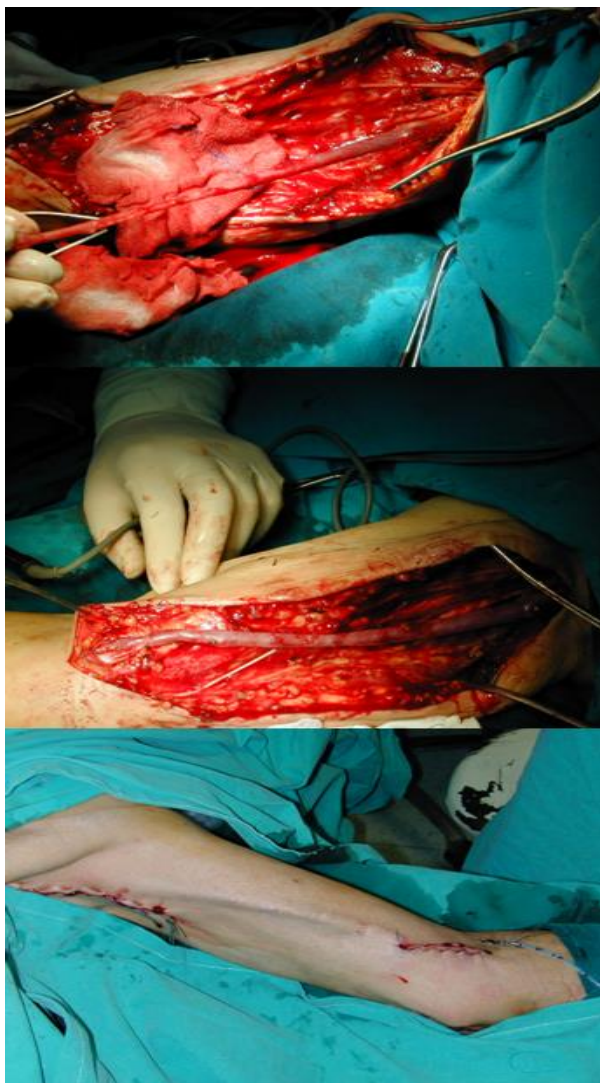
Results were calculated by using the following equation: Blood flow =  $\pi \cdot V \cdot d^2$  (Abbreviations:  $\pi = 3,14$ ,  $V =$  Velocity in cm/min,  $d =$  vessel's diameter in cm). Detailed measurements were made in the basilic vein and brachial artery before and after the AVF construction and in the transposed basilic vein with the feeding artery closed (in this case the flow is called retrograde).

Blood samples were drawn at phases 0, 1 and 2 from the peripheral arterial line and from the pulmonary artery in order to assess the oxygen and acid-base status during AVF construction. All the procedures for the AVF formation were performed under local anesthesia and sedation (small doses of propofol) by using the “one stage procedure technique. The incision was started at the antecubital crease in a vertical fashion just medial to the brachial artery pulse. The incision was extended through the subcutaneous tissue; the fascia was incised and the brachial artery exposed. The medial nerve was always identified and preserved. Proceeding slightly more medially and still deep to the fascia, the basilic vein was visualized and traced proximally, all the way to its junction with the

axillary vein. Occasionally, usually in the proximal third of the arm, the medial cutaneous nerve was encountered and preserved. Once the basilic vein was mobilized and all its branches were ligated, it was divided as far distal to the arm as possible and brought superficial to the medial cutaneous nerve. Then, the vein was tunneled in a more lateral, subcutaneous location. Proximal and distal control of the brachial artery was obtained and the patient was given heparin. A 6- to 7-mm arteriotomy was then made and an end-to-side anastomosis between the basilic vein and the brachial artery was constructed using fine, nonabsorbable monofilament suture. Clamps were then released and flow established in the arterialized basilic vein. The deep fascia was then closed with interrupted, absorbable sutures, keeping the basilic vein superficial to the fascia. The basilic vein was positioned in the subcutaneous pocket, thereby relocating it in a more lateral and more superficial position (Figure 1). There was also a continuous supply of oxygen via a face mask (6-8l/min).

The echocardiographic evaluation of cardiac function was performed six months later by the same operator (precisely) within 6hrs after the hemodialysis session (stage 2). The exact timing of the second echocardiography evaluation was important in order to preserve a water balance close to the “dry” weight, as in the first study (stage 1).

**Figure 1.** Different stages of basilic vein transposition and AVF construction



*AVF: arteriovenous fistula*

Equations were used to calculate the following cardiovascular parameters: cardiac index (CI), stroke volume index (SVI), systemic vascular resistance (SVR), systemic vascular resistance index (SVRI) pulmonary vascular resistance (PVR), pulmonary vascular resistance index (PVRI), left ventricular cardiac work (LVS<sub>W</sub>), left ventricular cardiac work index (LVS<sub>WI</sub>), right ventricular cardiac work (RVS<sub>W</sub>) and rig-

ht ventricular cardiac work index (RVS<sub>WI</sub>). Equations were also used to calculate the following oxygen transport indicators: arterial oxygen content (CaO<sub>2</sub>), Mixed Venous Blood Content (CvO<sub>2</sub>), Pulmonary Capillary O<sub>2</sub> Content (Cc'O<sub>2</sub>), Alveolar Oxygen Tension (PA-O<sub>2</sub>), Arterial-Venous Content Difference (C(a-v) O<sub>2</sub>), Venous Admixture (Q<sub>s</sub>/Q<sub>t</sub>, Shunt %), Oxygen Delivery (DO<sub>2</sub>) and Oxygen Consumption (VO<sub>2</sub>).

The t-test, paired t-test and repeated measures ANOVA were used for statistical analysis. Their nonparametric analogues; the Wilcoxon-Mann-Whitney, Wilcoxon signed rank, and Friedman tests were used for the validation of the results due to the small sample used in the study. Statistical analysis was performed with SPSS 14.0".

## RESULTS

A total of 16 patients with ESRD (8 males and 8 females; mean age 48 years with range of 25 – 75 years) were studied.

Demographic data are shown in Table 1.

Vessel's diameters in the AVF system are depicted in Table 2 and flow measurements in the parts of the AVF system in Table 3.

As expected, blood flow in the basilic vein increased statistically significant after the AVF construction. Likewise, blood flow in the pro-

ximal artery increased but not in a statistically significant manner.

As far as the hemodynamic parameters are concerned, CO, CI, SV and SVI increased in a statistically significant manner after the AVF construction, namely at phases 3 and 4, and they returned back to their preoperative values at phase 5. On the contrary, SVR decreased statistically significant at phases 3 and 4 and they remained decreased throughout the study period. LVSW showed similar alterations to CO and increased at phases 3 and 4 and returned back to baseline at phase 5.

**Table 1.** Patient's demographic data

	Mean ± SD	Median
<b>Ages (yrs)</b>	59,47±12,43	62
<b>Weight(Kg)</b>	69,3±11,16	71
<b>Height(m)</b>	1,66±0,06	1,64
<b>BMI</b>	25,3 ± 4,79	26,39
<b>BSA (m<sup>2</sup>)</b>	1,74±0,11	1,74
<b>Patients on hemodialysis (yrs)</b>	3,84±4,51	1,5
<b>K<sup>+</sup> (mEq/dL)</b>	5,1±0,61	5,3
<b>Na<sup>+</sup>(mEq/dL)</b>	143,4±4,73	144
<b>BUN (mg/dL)</b>	146,55±45,66	123
<b>Cr (mg/dL)</b>	6,64±2,31	6,3
<b>Blood glucose (mg/dL)</b>	124,73±24,9	119
<b>Ht (%)</b>	32,6±2,99	32,6

*BMI: body mass index, BSA: body surface area, K<sup>+</sup>: potassium, Na<sup>+</sup>: sodium, BUN:Blood urea nitrogen, Cr: creatinine, Ht: haematocrit*

There were no changes recorded regarding the rest of the hemodynamic parameters of the study protocol and the oxygen status indicators. (Table 4, Figures 2, 3).

**Table 2.** Vessels diameters in AVF system

	Diameter (cm)	
	Mean ± SD	Median
<b>Basilic Vein</b>	0,36±0,07	0,36±0,07
<b>Brachial artery</b>	0,4±0,06	0,4±0,06
<b>Transposed Basilic Vein</b>	0,61±0,18	0,61±0,18
<b>Feeding Artery after AVF Construction</b>	0,4±0,04	0,4±0,04

*AVF:arteriovenous fistula*

**Table 3.** Blood flow in the AVF system

	Blood flow (ml/ min)	
	Mean ± SD	Median
Basilic vein blood flow before AVF <b>phase 0</b>	317,8± 89,1	324
Arterial blood flow before AVF <b>phase 0</b>	313,2±107,1	321
Blood flow in the transposed basilic vein with the feeding artery "open" <b>phase 2</b>	888,83± 240	790
Blood flow in the transposed basilic vein with the feeding artery "closed" <b>phase 2</b>	274,34± 168	234
Blood flow in the feeding artery <b>phase 2</b>	457,67± 291	389

*AVF:arteriovenous fistula*

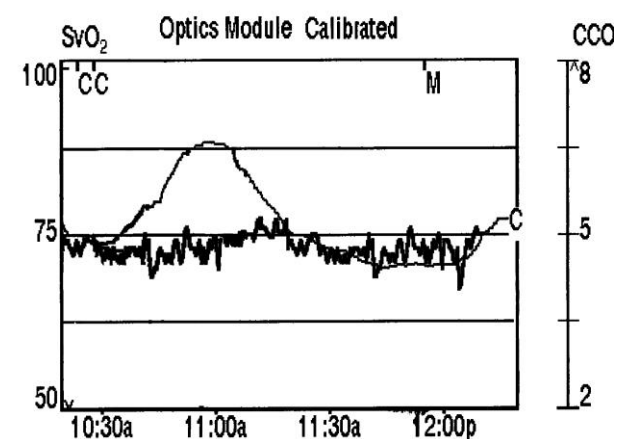
With regard to the echocardiographic findings, EF% showed a tendency to increase but without any significance, whereas IVSs increased statistically significant in both systole and diastole (p<0,01).

**Table 4.** CO, CI, SV, SVI, SVR and LVSW alterations during the study

	CO (l/min)	CI (l/min/m <sup>2</sup> )	SV (ml)	SVI (ml/m <sup>2</sup> )	SVR (dyn.sec/cm <sup>5</sup> )	LVSW (g-m/b)	
PHASES	0	5,49 ±1,24	3,22 ±0,84	74,29 ±26,63	43,56 ±16,39	2101,6 ±683,3	89,40 ±43,29
	1	5,32 ±1,32	3,12 ±0,87	71,97 ±27,07	42,17 ±16,48	2044,2 ±796,4	81,74 ±40,90
	2	5,56 ±1,26	3,26 ±0,83	77,87 ±28,95	45,66 ±17,61	1879,83 ±636,6	87,08 ±38,83
	3	7,7** ±1,66	4,54** ±1,13	106,5** ±38,46	62,7** ±24,16	1333,7** ±422	115,9** ±50,96
	4	7,9** ±1,32	4,67** ±0,83	108,4** ±31,87	63,5** ±19,46	1273,1** ±279,1	120,1** ±41,48
	5	5,77 ±1,11	3,37 ±0,70	78,12 ±22,75	45,78 ±14,17	1729,2** ±337,8	90,08 ±33,87

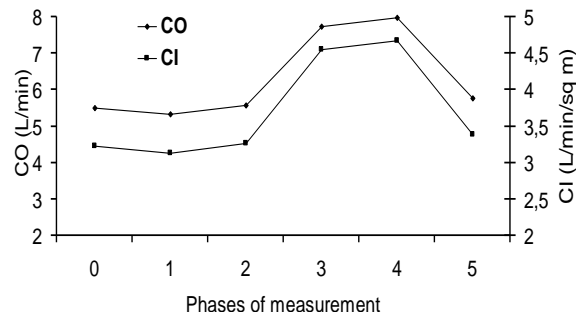
\*\**p*<0,01: comparison with baseline, CO: cardiac output, CI:cardiac index, SV:stroke volume, SVI: stroke volume index, SVR: systemic vascular resistance, LVSW:left ventricular cardiac work. Values are mean±SD.

**Figure 2.** CCO and SvO<sub>2</sub> trend in one patient of the study.



CCO:continuous cardiac output, SvO<sub>2</sub>: Mixed venous oxygen saturation.

**Figure 3.** CO and CI alterations during the study phases



CO:cardiac output, CI:cardiac index

The PLVW decreased minimally in both systole and diastole. The individual elements of the echocardiographic evaluation at stages 1 and 2 are summarized in Table 5.

**Table 5.** Echocardiographic Parameters

	EF (%)	PLV Ws	PLVWd (cm)	LVDs (cm)	LVDd (cm)	IVSs (cm)	IVSd (cm)
Stage 1	66,6 ±6,9	1,66 ±0,3	1,16 ±0,15	3,08 ±0,5	5 ±0,7	1,58 ±0,3	1,05 ±0,1
Stage 2	69,8 ±6,8	1,52 ±0,3	1,21 ±0,11	2,71 ±0,7	4,82 ±0,6	1,8 ±0,3	1,37 ±0,2
Stage 1 Vs Stage 2	NS	NS	NS	NS	NS	*	**

\* *p*<0,01, \*\* *p*<0,001, EF: ejection fraction, PLVWs: posterior wall of left ventricular dimension during systole, PLVWd: posterior wall of left ventricular dimension during diastole, LVDs: left ventricular dimension during systole, LVDd: left ventricular dimension during diastole, IVSs: interventricular septum during systole, IVSd: interventricular septum during diastole. Values are mean±SD.

All of the AVFs were functioning adequately at the time of re-evaluation. There were no infections nor thrombosis or other complications.

## DISCUSSION

AVFs are the preferred vascular access in dialysis patients due to their higher blood flow rates, superior patency and low incidence of infections compared with either arteriovenous grafts or catheters<sup>16,17,18</sup>.

Basilic vein transposition, a kind of AVF above the elbow, allows the patient to have a native fistula, while preserving deep brachial venous systems for future placement of forearm or upper arm PTFE arteriovenous grafts. It is also well documented, that transposition of a basilic vein and creation of brachio-basilic arteriovenous fistula, result in a satisfactory patency rate and provide good blood flows<sup>19,20,21</sup>. This type of AVF is usually considered as the last option for autologous vascular access for hemodialysis (HD) on the upper extremity.

Depending on the diameter of the AV communication and the size of the artery feeding it, an AVF increases venous return to the heart and at the same time it decreases peripheral vascular resistance<sup>22,23</sup>. This, in accordance with Starling's law, increases CO and work of the heart. The impact on CO correlates tightly with the blood flow through the AVF.

In patients with compromised cardiac function, the aforementioned hemodynamic changes could result in cardiomegaly and congestive heart failure<sup>24,25,26</sup>. However, elevated CO alone does not cause symptoms of congestive heart failure or high output cardiac failure.

Blood volume increase results eventually to a gradual increase of the right atrial pressure, pulmonary artery pressure and LV end-diastolic pressure until myocardium decompensates, LV dilates, EF% declines and the patient shows symptoms of heart failure.

Both LV dilation as well as LV hypertrophy have been reported to occur with high-output cardiac failure. Currently there are no guidelines as to what extent AVF flows constitute a potential cardiac risk for patients, and besides that there are no guidelines to define high-access flows<sup>22</sup>.

AVF effects had been studied before in the literature by occlusion studies in patients with a well-established AVF<sup>9</sup>.

However, there are no studies in the literature with data obtained from direct measurements (with a Swan Ganz catheter) of the individual hemodynamic parameters during AVF construction.

Moreover, in our study, we focused on basilic vein transposition and brachial artery-basilic vein anastomosis and we studied its acute and chronic hemodynamic effects in patients with ESRD without any underlying cardiac disease.



In the present study, there were no differences between preoperative and postoperative diameters in the AVF system. AVF blood flow was high enough for haemodialysis (mean flow = 888,83 ml/min). In addition to this, it was found to be higher compared to the flow in the proximal artery, which can be easily explained by Poiseuille's law;  $\Delta\Pi = P_1 - P_2 = Q \cdot 8 \cdot \mu \cdot l / \pi \cdot \alpha^4$  (where Q=blood flow,  $\Delta\Pi$ =pressure gradient between two points,  $\pi$ =constant 3.14,  $\alpha$ =radius of the vessel,  $l$ =distance between the two points,  $\mu$ =viscosity). The retrograde flow (retrograde flow=274,34 ml/min) was low and there were no symptoms of vascular insufficiency at all. There was no difference in the heart rate during the AVF construction (phases 0-5). This is in agreement with other studies<sup>20</sup>. Arterial blood pressure showed a tendency to decrease (phases 2-5) but not in a statistically significant manner. This is explained by the SVR decrease as a consequence of the AVF<sup>28</sup>.

Central venous pressure showed no change during our study and this is also in accordance with the literature<sup>29</sup>.

CO increased significantly during phases 3 and 4 and returned back to preoperative levels at phase 5. This is explained by SVR decline; the bigger the SVR decline (and therefore the higher the AVF flow) the more intense the CO increase<sup>27,28-30</sup>. SV, SVI and CI showed similar alterations to those of CO. SVR decreased

during phases 4 and 5 as a result of the AVF formation. The same results were recorded by Ori et al<sup>20</sup>. Unlike the SVR, PVR showed no change, which is in accordance with other studies<sup>21</sup>.

LVS<sub>W</sub> increased significantly during phases 3 and 4, while RVS<sub>W</sub> showed a tendency to increase during the same phases (but not significantly). These alterations have been previously recorded by Crowe et al in experimental studies<sup>30</sup>.

There were no differences neither in SpO<sub>2</sub> nor in SvO<sub>2</sub> at any phase of the study. This can be attributed to the continuous supply of oxygen during the operation via face mask in relation to the sigmoid curve of haemoglobin oxygen saturation.

Six months after basilic vein transposition, all the fistulas were functioning well, there were no clinical signs of pulmonary oedema or other cardiac disease (e.g. heart failure) and echocardiographic evaluation revealed:

- The cardiac EF showed a tendency to increase, which has been recorded by other authors<sup>31,32</sup>.
- IVSs and IVSd increased statistically significant.
- PLVWs, PLVWd, LVDs and LVDd did not show any significant change, which is in agreement with other literature studies<sup>33,34</sup>.

The recorded increase of the thickness of the

IVS does not constitute a specific feature of LV hypertrophy, since both the ventricular wall thickness and the left ventricular dimensions did not change. Therefore, establishing an AVF by transposition of the basilica vein, does not hamper the cardiac function in terms of cardiomegaly or congestive heart failure.

## CONCLUSION

In conclusion, our study suggests that basilic vein transposition in this selected group of hemodialysis patients provides an excellent patency rate, very good blood flows and does not cause any significant changes regarding any cardiac parameters for at least a period of six months. Moreover, according to our results, this specific type of AVF cannot be considered as a cause of LV hypertrophy, circulatory congestion, pulmonary oedema or cardiac failure. Our future goal is to re-evaluate all these patients by echocardiography to identify any long term cardiac effects of the AVF.

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