ARTERIAL STIFFNESS : AN INDEPENDENT DETERMINANT OF
ADAPTIVE GLOMERULAR HYPERFILTRATION AFTER KIDNEY
DONATION

Pierre Fesler 1,3; Georges Mourad 2,3; Guilhem du Cailar 1; Jean Ribstein 1,3; Albert Mimran1,3.

1 Department of Internal Medicine, Hopital Lapeyronie, Montpellier, France.
2 Department of Nephrology, Hopital Lapeyronie, Montpellier, France.
3 Université Montpellier 1, France.

Running Head: Arterial stiffness and adaptive glomerular hyperfiltration

Authors contribution:


Correspondence: Pierre Fesler, MD
Department of Internal Medicine, Hôpital Lapeyronie
34295 Montpellier Cedex 5, France
Phone +33 (0)4 67 33 84 43 - Fax +33 (0)4 67 33 84 53
E-mail: p-fesler@chu-montpellier.fr


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ABSTRACT

After kidney donation, the remaining kidney tends to hyperfiltrate thus limiting the initial loss of renal function. The potential determinants of this adaptive glomerular hyperfiltration (GHF) and specifically the influence of arterial function are poorly known.

In 45 normotensive healthy kidney donors (51 +/- 10 yrs [mean+/-SD], 39 females), glomerular filtration rate (GFR) was measured as the clearance of continuously infused $^{99m}$Tc-DTPA and timed urine collections at baseline -i.e. before donation- and 1 year after donation. GHF was computed as post-donation GFR minus half of baseline GFR. Arterial function was assessed as baseline carotido-femoral pulse wave velocity (PWV) and carotid augmentation index (AIX).

After kidney donation, no significant change in blood pressure (BP) was observed, but 2 subjects developed hypertension. GFR decreased from 107 +/- 19 to 73 +/- 15 mL/min/1.73m² and mean GHF was 20 +/- 10 mL/min/1.73m². In univariate analysis, GHF was inversely correlated to age ($r^2$=0.24, p=0.01), baseline PWV ($r^2$=0.23, p=0.001) and AIX ($r^2$=0.11, p=0.031). Nevertheless, GHF was not correlated to baseline peripheral or central BP. In multivariate analysis, baseline PWV, but not AIX, remained inversely correlated to GHF, independently of age, baseline mean BP and GFR (model $r^2$=0.34, p<0.001).

In healthy subjects selected for renal donation, increased arterial stiffness is associated with decreased post donation compensatory hyperfiltration.

Key words: Renal transplantation, Kidney donation, arterial stiffness, adaptive glomerular hyperfiltration
INTRODUCTION

Due to the shortage of cadaveric organs, living donors kidney transplantation is gaining increasing interest. Long-term follow-up studies have shown that kidney donors have a similar life expectancy and no increased risk of developing end-stage renal disease (ESRD) when compared to matched nondonors (16, 24). However, despite a careful workup performed before donation, a rapid decline in renal function that could lead to ESRD may occur in some kidney donors (7), with a higher frequency in African Americans (14). In the Minnesota study conducted in 3698 kidney donors followed during a mean period of 12 yrs after donation, a decline in glomerular filtration rate (GFR) to less than 60mL/min/1,73m² was associated with older age and higher body mass index (BMI) (16).

In recent years, several cross-sectional studies have suggested a relationship between arterial stiffness assessed by pulse pressure (35) or carotid-to-femoral pulse wave velocity (PWV) (17, 18, 31, 33) and mild deterioration of renal function. In a longitudinal study conducted in 132 never-treated patients with essential hypertension, we have observed that pretreatment pulse pressure, a marker of arterial stiffness and wave reflection (27), emerged as an important predictor of the yearly decline in measured GFR within a 6-year period of treatment associated with satisfactory control of hypertension (11).

Following uninephrectomy, the function of the remaining kidney tends to be higher than 50% of the baseline GFR (21) due to an adaptive mechanism of hyperfiltration. In the present study, we investigated the influence of arterial stiffness and wave reflection on the magnitude of adaptative glomerular hyperfiltration (GHF) after uninephrectomy in healthy subjects selected for kidney donation.
METHODS

Study population

The study population consisted in 45 healthy subjects (39 women and 6 men, aged 24 to 70 yrs) who were considered for kidney donation. None of them had ever taken antihypertensive drugs. However, 8 subjects were receiving other medications such as statins (n=1), hormonal contraception (n=5) or hormonal replacement therapy (n=2). Patients with clinical evidence of atherosclerosis (stroke, coronary and peripheral artery disease), heart failure, renal failure (serum creatinine >120 μmol/L), diabetes mellitus (fasting blood glucose ≥ 7 mmol/L), marked obesity (body mass index [BMI] ≥ 35 kg/m²), or a history of alcohol abuse (≥ 5 drinks/day) were excluded. Electrocardiogram and echocardiography were obtained in all subjects in order to control for the absence of valvular lesions as well as cardiac functional and morphologic abnormalities. All procedures were performed in accordance with the Declaration of Helsinki and the Declaration of Istanbul; and informed consent was obtained prior to studies.

Determination of renal function

Renal function was measured at baseline and one year after kidney donation. On the day of investigation, patients came to the ward with a 24-h urinary collection for the determination of creatinine, electrolytes and albumin (nephelometry). As previously described (20), GFR was estimated by urinary clearances of technetium-labelled diethylene triaminopentaacetic acid ($^{99}$mTc-DTPA) using the constant infusion technique and 3 to 4 timed urine collections obtained every 30 min by spontaneous voiding. Blood samples were obtained prior to clearance determinations for the measurement of serum creatinine (enzymatic method based on the conversion of creatinine to ammonia and N-methylhydantoine by creatinine deiminase), glucose, uric acid and lipid levels. In a previous study conducted in 20 subjects, the reproducibility of day-to-day measurements of GFR, expressed as coefficient of variation, was 6.4 % (9).
Adaptive glomerular hyperfiltration (GHF) was calculated as 1-yr post-donation GFR minus 50% of whole baseline GFR, as proposed by Tan et al (34).

**Determination of blood pressure**

Hemodynamic measurements were performed at baseline by the same observer (PF) in a quiet room maintained at constant temperature (21° Celsius). BP was measured every 3 min with an automatic device (Model Dinamap V100, GE Healthcare, Little Chalfont, UK) (28). Reported values are the average of at least 10 measurements following a 10-min period of rest in the supine position. Normotension was defined as systolic blood pressure (BP) < 140 mmHg, and diastolic BP < 90 mmHg (1).

**Pulse wave velocity**

Femoral, carotid and radial pressure waveforms were recorded by applanation tonometry using the SphygmoCor system (AtCor Medical, Sidney, Australia). From carotid and femoral waveforms, wave transit time was calculated using the R wave of a simultaneously recorded electrocardiogram as a reference frame and the foot of the waves determined with intersecting tangent algorithm. Distances from the suprasternal notch to carotid and femoral sites were measured and transit distance was calculated by subtraction of the two distances; thus allowing correction for parallel transmission. Carotid-to-femoral PWV was then calculated as transit distances divided by transit time (25).

**Pulse wave analysis**

Carotid pressure waveform was used as an estimate of central aortic waveform without the use of transfer function. Carotid pressure waveform was calibrated using mean and diastolic pressure values of the radial pressure waveform; which was itself calibrated with cuff-measured systolic and diastolic brachial BP. Augmented pressure (AP) was determined as the pressure difference between first and second peaks of carotid waveform. Augmentation index (AIx) was
calculated as AP / central pulse pressure (26).

As evaluated by Wilkinson et al (39) and using Bland-Altman plots, within-observer variability is 0.07±1.17 m/sec for carotid-to-femoral PWV and 0.49±5.37 % for AIx.

**Statistical analysis**

SPSS for Windows version 11.0 software (SPSS Inc., Chicago, IL) was used for statistical analysis. Due to skewed distribution, urinary albumin excretion values were log-transformed prior to statistical analysis. Within subjects comparisons before and after kidney donation were performed using paired t-test.

The relationship between GHF and baseline parameters was first tested by linear univariate regression analysis. Multivariate linear regression was used to detect independent relationship between GHF and arterial parameters after adjustment for age and mean arterial BP.

The population was then categorized into tertiles of large artery function parameters. GHF was used as the dependent variable in analysis of covariance (ANCOVA), with age, baseline mean BP, fasting blood glucose, cholesterol, triglycerides but also pre-donation GFR as additional possible explanatory variables. The presence of a linear trend across tertiles of large artery function parameters was assessed by contrast analysis.

Two-tailed p<0.05 was considered statistically significant. Unless otherwise stated, results are expressed as mean ± standard deviation or median (interquartile range) for descriptive statistics and mean (95% confidence interval) for results of multivariate analysis.

**RESULTS**

**Characteristics at baseline and after kidney donation**

As summarized in Table 1, peripheral BP, BMI, lipid profile, fasting blood glucose and natriuresis were unchanged after uninephrectomy, except for triglycerides that were slightly but
significantly increased post-donation. Two subjects progressed to hypertension and were adequately controlled by drug treatment. Urinary excretion of albumin was unchanged after uninephrectomy and none of the subjects developed microalbuminuria (≥ 30 mg/24-hr) at 1-yr.

Of note, kidney donation had no influence on large artery function parameters, namely carotid-to-femoral PWV, central BP and AIx. More specifically, post-donation PWV was significantly correlated with the pre-donation value ($r^2=0.20$, $p=0.015$). There was no relationship between baseline PWV and the change in BP associated with kidney donation.

Serum creatinine increased from 65±10 to 93±18 µmol/L ($p<0.001$) and measured GFR decreased by 32%, from 107±19 at baseline to 73±15 mL/min/1.73m² after nephrectomy ($p<0.001$).

Baseline one-kidney GFR (half of whole GFR) was 53±10 mL/min/1.73m², thus resulting in a mean value of adaptive glomerular hyperfiltration (GHF) of 20±10 mL/min/1.73m² (i.e. 38% of the pre-donation one-kidney GFR) (Figure 1). Of interest, serum uric acid increased from 263±68 to 300±70 µmol/L after uninephrectomy.

**Determinants of adaptive glomerular hyperfiltration.**

As shown in Table 2, in univariate analysis, age was significantly and inversely correlated to GHF ($r^2=0.24$, $p=0.001$). Gender as well as baseline BMI, fasting blood glucose, serum uric acid, lipid parameters, urinary urea or sodium were not related to GHF. Interestingly, neither pre-donation serum creatinine nor albuminuria was associated with GHF.

No significant influence of any component of peripheral or central blood pressures was detected. Importantly, an inverse relationship between the magnitude of GHF and baseline carotid-to-femoral PWV ($r^2=0.23$, $p=0.001$) or AIx ($r^2=0.11$, $p=0.031$) was found. The higher baseline PWV or AIx the lower the post nephrectomy GHF.

**Multivariate analysis**

As shown in Table 3, the association between baseline PWV and GHF remained significant
after adjustment to age and baseline mean BP, two well-known covariates of arterial stiffness (model 1). This association remained unchanged after replacement of baseline mean BP by baseline peripheral (model 2) or central pulse pressure. In a model including mean BP, peripheral PP, heart rate and age together with PWV, only age and PWV remained significantly associated with GHF. Concerning AIx, the association with GHF was no longer significant after adjustment to age and baseline BP.

As illustrated in Figure 2, when the population was divided into tertiles of baseline carotid-to-femoral PWV, the association with post-donation GHF was significant (p linear trend = 0.017) after adjustment to age and baseline values of body mass index, mean BP, fasting blood glucose, total cholesterol, triglycerides and baseline whole GFR. In addition, the adjusted magnitude of GHF (mean±SEM) decreased from 25±2 (tertile 1) to 17±2 (tertile 3) mL/min/1.73m² (p linear trend = 0.032), corresponding to a GHF of 47 and 32 % of the pre-donation one-kidney GFR in subjects with the lowest and highest baseline PWV, respectively.

**DISCUSSION**

In the present longitudinal study conducted in healthy kidney donors, an inverse relationship was observed between the level of compensatory glomerular hyperfiltration (GHF) of the remaining kidney and the most accessible marker of aortic stiffness, i.e. carotid-to-femoral PWV (25).

As shown in meta-analysis studies, development of hypertension (6) and proteinuria (13) may be facilitated by unilateral nephrectomy; however, no modification of survival was observed (16). No increase in the risk in ESRD, when compared to the general population, was reported (16). In the present study, none of the patients had a 1-yr GFR lower than half of the pre-donation GFR, thus suggesting the absence of deleterious functional effect of nephrectomy on the remnant kidney within this short interval.

Following uninephrectomy, hyperfiltration of the remnant kidney develops within days. In 57
healthy kidney donors, and within 6 months after uninephrectomy, a 40 to 45% rise in GFR (clearance of iothalamate) was observed; and no difference between subjects aged <= 45 years and subjects aged >=55 years was detected (34). The present observation of a measured GHF of 20 mL/min/1.73m² (i.e. 38% of one-kidney baseline GFR) is in line with previous studies (29, 34, 37, 38).

The occurrence of GHF might reflect pre-donation renal functional reserve which is commonly defined as the ability of the kidney to increase GFR in response to low-dose dopamine infusion (36) or amino acid load (5). Renal reserve is known to be impaired in essential hypertension (40), in the presence of chronic kidney disease (3), and in elderly subjects (8, 12). In 125 kidney donors, renal reserve capacity was shown to contribute to the prediction of post-donation GFR (30). Of interest, the renal reserve in response to dopamine was blunted after nephrectomy (21), thus suggesting that part of the GHF might be the consequence of intra-renal vasodilation and increased renal plasma flow.

Previous studies have shown that large artery function is related with renal function. In 305 never-treated hypertensive subjects with serum creatinine < 120 µmol/L, creatinine-based estimated GFR was negatively correlated with carotid-to-femoral pulse wave velocity (PWV) after adjustment for age and mean BP (31). In addition, carotid-to-femoral PWV was positively correlated to the level of albuminuria in 70 never-treated hypertensive subjects (22). Our observation of a lack of change of arterial function parameters in response to uninephrectomy suggests that an isolated moderate decline in GFR has limited impact on large arteries. In a population of 264 subjects with chronic kidney disease stages 3-5, the contribution of estimated GFR to explain variability of carotid-to-femoral PWV was less than 1% in models adjusting for age, SBP, diabetes, heart rate and BMI (32).

Peripheral pulse pressure, considered as a rough indicator of arterial stiffness in subjects over 40 years, has emerged as the most influencing component of BP on the rate of decline of measured
GFR in hypertensive subjects (11, 35). The present observation of a relationship between pre-donation PWV and the decrease in the magnitude of GHF, independent of age, is another argument for a deleterious effect of large artery dysfunction on the kidney.

Arterial stiffness could influence post-donation GHF partly through a decrease in the baseline renal reserve. Limited data are available of the potential impact of large artery function on renal hemodynamics. In a cross-sectional study conducted in 49 healthy nomotensive subjects, we observed that albuminuria and filtration fraction were positively correlated with central augmentation index and reflexion magnitude (10). It was hypothesized that an increase in wave reflection could promote intra-glomerular hypertension as suggested by its relation to filtration fraction (23) and albuminuria (4). Unfortunately, in the present study, renal plasma flow was not measured.

Alternatively, it is possible that the change in GFR following uninephrectomy could be related to the number of nephrons. It has been shown that low-birth weight may be related to an increase in arterial stiffness (19) as well as a decrease in nephrons number (15). Such a common determinant could explain the previously observed association between kidney donors aortic PWV and renal outcome of related graft recipients (2).

In the present study, the hypothesis that high arterial stiffness in the kidney donor could predict poor adaptive GHF in response to uninephrectomy was successfully tested. Large vessel function determination prior to kidney donation could be proposed as a tool to assess vascular health beyond blood pressure and GFR. Measurement of PWV may help identify potential kidneys donors with high cardiorenal risk.

In conclusion, arterial stiffness may be an important contributor of the renal hemodynamic adaptation to a reduction in nephron number in healthy subjects without diabetes or any other cause of renal disease. Further longitudinal studies assessing the long-term response of GFR to uninephrectomy according to the level of PWV may be of crucial interest.
DISCLOSURES

No author has any potential conflict of interest to disclose in relation to this manuscript.

REFERENCES


Table 1: Population characteristics at baseline and one year after kidney donation.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>1-yr Post UNx</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female / Male (n)</td>
<td>39/6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>51 ± 10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>24.2 ± 3.4</td>
<td>24.4 ± 3.9</td>
<td>0.20</td>
</tr>
<tr>
<td>Serum creatinine (µmol/L)</td>
<td>65 ± 10</td>
<td>93 ± 18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum uric acid (µmol/L)</td>
<td>263 ± 68</td>
<td>300 ± 70</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting blood glucose (mmol/L)</td>
<td>4.9 ± 0.6</td>
<td>4.8 ± 0.5</td>
<td>0.31</td>
</tr>
<tr>
<td>Total cholesterol (g/L)</td>
<td>2.22 ± 0.43</td>
<td>2.23 ± 0.40</td>
<td>0.78</td>
</tr>
<tr>
<td>HDL-cholesterol (g/L)</td>
<td>0.64 ± 0.18</td>
<td>0.65 ± 0.22</td>
<td>0.62</td>
</tr>
<tr>
<td>Triglycerides (g/L)</td>
<td>0.91 ± 0.35</td>
<td>1.02 ± 0.45</td>
<td>0.037</td>
</tr>
<tr>
<td>Urinary sodium (mmol/24-h)</td>
<td>130 ± 50</td>
<td>132 ± 68</td>
<td>0.84</td>
</tr>
<tr>
<td>Urinary albumin excretion (µg/min)</td>
<td>4 (3-6)</td>
<td>3 (3-6)</td>
<td>0.41</td>
</tr>
<tr>
<td>Peripheral systolic blood pressure (mmHg)</td>
<td>122 ± 12</td>
<td>122 ± 13</td>
<td>0.97</td>
</tr>
<tr>
<td>Peripheral diastolic blood pressure (mmHg)</td>
<td>70 ± 9</td>
<td>70 ± 8</td>
<td>0.65</td>
</tr>
<tr>
<td>Peripheral pulse pressure (mmHg)</td>
<td>52 ± 7</td>
<td>52 ± 10</td>
<td>0.74</td>
</tr>
<tr>
<td>Peripheral mean blood pressure (mmHg)</td>
<td>88 ± 10</td>
<td>87 ± 9</td>
<td>0.78</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>66 ± 8</td>
<td>66 ± 8</td>
<td>0.91</td>
</tr>
<tr>
<td>Carotid-to-femoral pulse wave velocity (m/s)</td>
<td>7.2 ± 1.3</td>
<td>6.8 ± 1.1</td>
<td>0.74</td>
</tr>
<tr>
<td>Central Augmentation Index (%)</td>
<td>24 ± 14</td>
<td>26 ± 14</td>
<td>0.68</td>
</tr>
<tr>
<td>Central systolic blood pressure (mmHg)</td>
<td>115 ± 15</td>
<td>115 ± 17</td>
<td>0.57</td>
</tr>
<tr>
<td>Central pulse pressure (mmHg)</td>
<td>46 ± 11</td>
<td>46 ± 13</td>
<td>0.39</td>
</tr>
</tbody>
</table>

Values are expressed as Mean ± Standard Deviation or Median (Interquartile Range). Unx: uninephrectomy.
Table 2: Univariate analysis between the magnitude of adaptive glomerular hyperfiltration and pre-donation parameters

<table>
<thead>
<tr>
<th>Dependent variable: Adaptive Glomerular Hyperfiltration (mL/min/1.73m²)</th>
<th>β-coefficient</th>
<th>r²</th>
<th>T</th>
<th>p</th>
</tr>
</thead>
</table>

**Independent variables:**

<table>
<thead>
<tr>
<th>Variable</th>
<th>β-coefficient</th>
<th>r²</th>
<th>T</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (female = 0, male = 1)</td>
<td>4.18</td>
<td>0.02</td>
<td>0.96</td>
<td>0.34</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td><strong>-0.48</strong></td>
<td><strong>0.24</strong></td>
<td><strong>-3.72</strong></td>
<td><strong>0.001</strong></td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>-0.29</td>
<td>0.01</td>
<td>-0.67</td>
<td>0.51</td>
</tr>
<tr>
<td>Serum creatinine (µmol/L)</td>
<td>-0.20</td>
<td>0.04</td>
<td>-1.37</td>
<td>0.18</td>
</tr>
<tr>
<td>Serum uric acid (µmol/L)</td>
<td>-0.037</td>
<td>0.07</td>
<td>-1.74</td>
<td>0.089</td>
</tr>
<tr>
<td>Fasting blood Glucose (mmol/L)</td>
<td>-4.70</td>
<td>0.08</td>
<td>-1.88</td>
<td>0.067</td>
</tr>
<tr>
<td>Total cholesterol (g/L)</td>
<td>-2.71</td>
<td>0.01</td>
<td>-0.78</td>
<td>0.44</td>
</tr>
<tr>
<td>HDL-cholesterol (g/L)</td>
<td>-13.8</td>
<td>0.06</td>
<td>-1.69</td>
<td>0.099</td>
</tr>
<tr>
<td>Triglycerides (g/L)</td>
<td>-3.61</td>
<td>0.02</td>
<td>-0.85</td>
<td>0.40</td>
</tr>
<tr>
<td>Urinary sodium (mmol/24-h)</td>
<td>0.009</td>
<td>0.002</td>
<td>0.28</td>
<td>0.78</td>
</tr>
<tr>
<td>Urinary albumin excretion (µg/min, log-transformed)</td>
<td>4.05</td>
<td>0.007</td>
<td>0.51</td>
<td>0.61</td>
</tr>
<tr>
<td>Peripheral systolic blood pressure (mmHg)</td>
<td>-0.11</td>
<td>0.02</td>
<td>-0.88</td>
<td>0.39</td>
</tr>
<tr>
<td>Peripheral diastolic blood pressure (mmHg)</td>
<td>-0.079</td>
<td>0.005</td>
<td>-0.48</td>
<td>0.64</td>
</tr>
<tr>
<td>Peripheral pulse pressure (mmHg)</td>
<td>-0.19</td>
<td>0.02</td>
<td>-0.92</td>
<td>0.37</td>
</tr>
<tr>
<td>Peripheral mean blood pressure (mmHg)</td>
<td>-0.10</td>
<td>0.01</td>
<td>-0.67</td>
<td>0.51</td>
</tr>
<tr>
<td>Heart Rate (bpm)</td>
<td>0.11</td>
<td>0.007</td>
<td>0.56</td>
<td>0.58</td>
</tr>
<tr>
<td>Carotid-to-femoral pulse wave velocity (m/s)</td>
<td><strong>-3.70</strong></td>
<td><strong>0.23</strong></td>
<td><strong>-3.59</strong></td>
<td><strong>0.001</strong></td>
</tr>
<tr>
<td>Central augmentation index (%)</td>
<td><strong>-0.24</strong></td>
<td><strong>0.11</strong></td>
<td><strong>-2.23</strong></td>
<td><strong>0.031</strong></td>
</tr>
<tr>
<td>Central systolic blood pressure (mmHg)</td>
<td>-0.070</td>
<td>0.01</td>
<td>-0.69</td>
<td>0.49</td>
</tr>
<tr>
<td>Central pulse pressure (mmHg)</td>
<td>-0.17</td>
<td>0.03</td>
<td>-1.15</td>
<td>0.26</td>
</tr>
</tbody>
</table>
Table 3: Multivariate analysis of the relationship between adaptive glomerular hyperfiltration and its potential determinants.

<table>
<thead>
<tr>
<th>Dependent variable: Adaptive Glomerular Hyperfiltration (mL/min/1.73m²)</th>
<th>β-coefficient (95% CI)</th>
<th>p</th>
</tr>
</thead>
</table>

Independent variables:

Model 1: \( r^2 = 0.34 \), \( p < 0.001 \)
- Age (yrs) \(-0.358 (-0.634 – -0.082) 0.012\)
- Mean Blood Pressure (mmHg) \(-0.108 (-0.372 – 0.157) 0.42\)
- Carotid-to-Femoral Pulse Wave Velocity (m/s) \(-2.41 (-4.61 – -0.20) 0.033\)

Model 2: \( r^2 = 0.34 \), \( p = <0.001 \)
- Age (yrs) \(-0.326 (-0.607 – -0.045) 0.024\)
- Peripheral Pulse Pressure (mmHg) \(0.093 (-0.461 – 0.275) 0.61\)
- Carotid-to-Femoral Pulse Wave Velocity (m/s) \(-2.59 (-4.79 – -0.39) 0.022\)
FIGURES LEGEND

Figure 1: One-kidney glomerular filtration rate (GFR) before (half of whole GFR) and 1-yr after uninephrectomy (UNx). Results are expressed as mean ± standard deviation.

Figure 2: Adaptative glomerular hyperfiltration (GHF) according to tertiles of baseline carotid-to-femoral pulse wave velocity (PWV). Mean values are adjusted to age and baseline values of body mass index, mean blood pressure, fasting blood glucose, total cholesterol, triglycerides and glomerular filtration rate. Results are expressed as mean and 95% confidence interval.
Adaptive GHF (ml/min/1.73m²)

Tertiles of baseline PWV (m/sec)

I: [4.6-6.7] 25
II: [6.8-7.7] 18
III: [7.8-11.6] 17

p linear trend = 0.032