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Conclusion: This study provides the first direct evidence that aortic stiffness not arterial remodeling is an independent predictor of fatal and non-fatal cardiovascular events in patients with CKD stage 2-5.

1.6

ARTERIAL STIFFNESS IS AN INDEPENDENT DETERMINANT OF COMPENSATORY HYPERFILTRATION AFTER KIDNEY DONATION

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After kidney donation, the remaining kidney tends to hyperfiltrate thus limiting the initial loss of renal function. However, the potential determinants of this compensative hyperfiltration (CHF) and the possible influence of arterial function are not known.

In 26 normotensive healthy kidney donors (51 +/- 9 yrs [mean +/- SD], 22 females), glomerular filtration rate (GFR) was measured by the clearance of continuously infused Tc99m-DTPA and timed urine collections at baseline -i.e. before donation- and 1 year after donation. CHF was computed as post-donation GFR minus half of baseline GFR. Arterial function was assessed at baseline through carotido-femoral pulse wave velocity (PWV) and carotid augmentation index (Alx).

After kidney donation, there were no significant changes in blood pressure (BP), but 2 subjects became hypertensive. GFR decreased from 104 +/- 17 to 71 +/- 11 mL/min/1.73m² and mean CHF was 19 +/- 9 mL/min/1.73m². In univariate analysis, CHF was inversely correlated to baseline age ($r^2=0.15$, $p=0.049$) and PWV ($r^2=0.23$, $p=0.012$), but not mean BP or Alx. In multivariate analysis, CHF remained inversely correlated to PWV ($p=0.020$), independently of baseline age, mean BP and GFR (model $r^2=0.45$, $p=0.002$).

In conclusion, in healthy subjects, increased arterial stiffness seems to be associated with a limited magnitude of post-donation hyperfiltration. This could reflect an influence of arterial function on renal reserve, providing further insights into the relationship between macrocirculation and renal microcirculation.

Oral Session 3

Young Investigators' Presentations

3.1

PRE-PREGNANCY CARDIOVASCULAR RISK IN WOMEN WITH PREVIOUS PREECLAMPSIA (PET)/INTRAUTERINE GROWTH RESTRICTION (IUGR)

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Women with history of preeclampsia (PET) and delivery of intrauterine growth restriction (IUGR) babies are at increased risk of cardiovascular events later in life. This could be related to endothelial dysfunction, altered arterial stiffness or metabolic abnormalities resulting from pregnancy complications. Alternatively, pre-existing abnormalities in the cardiovascular system may predispose them to both pregnancy related complications and cardiovascular disease later in life. We compared pre-pregnancy arterial stiffness and central haemodynamics between women with previous PET/IUGR and those with previous uncomplicated pregnancies.

Method: 46 women planning to conceive were prospectively recruited in two groups: (1) 34 parous women with previous normal pregnancies (controls) (2) 12 women with previous PET (ISSHP guideline) requiring delivery at <34 weeks or requiring treatment for severe preeclampsia and/or who delivered an IUGR baby. Brachial and central blood pressures, pulse wave analysis (Alx), carotid-femoral pulse wave velocity (aPWV) and cardiac output were assessed along with routine biochemistry.

Results: Women with previous PET/IUGR had higher brachial diastolic blood pressure ($P=0.03$), central systolic blood pressure ($P=0.02$), MAP ($P=0.01$), and PVR ($P=0.03$) compared to controls but had lower PP amplification ($P=0.04$). There was no difference in the adjusted Alx, aPWV or in the cholesterol/lipid profile between the groups.

Conclusion: Vascular dysfunction as reflected by higher central blood pressure and peripheral resistance exists pre-pregnancy in women with previous PET/IUGR compared to normal healthy women. Further prospective pre-pregnancy studies prior to development of PET/IUGR are needed to assess the mechanism of the vascular dysfunction that persists following these pregnancy complications.

Table 1 Pre-pregnancy haemodynamics in two groups:

Cardiovascular parameter	Previous normal pregnancies (n=34)	Previous PET/IUGR (n=12)	P*
Age (yrs)	37 ± 4	34 ± 5	0.07
Brachial SBP (mm Hg)	108 ± 8	114 ± 9	0.07
Brachial DBP (mm Hg)	71 ± 7	77 ± 8	0.03*
Brachial PP (mm Hg)	37 ± 6	37 ± 4	0.7
Central SBP (mm Hg)	100 ± 9	107 ± 9	0.02*
PP (mm Hg)	28 ± 5	29 ± 5	0.4
PP amplification	1.4 ± 0.1	1.3 ± 0.1	0.04*
MAP (mm Hg)	84 ± 9	92 ± 9	0.01*
Heart rate (beats/min)	67 ± 9	67 ± 13	0.9
CO (L/min)	5.5 ± 1.0	5.1 ± 1.1	0.3
SV (ml)	77 ± 13	64 ± 25	0.1
PVR (dynes.s ⁻¹ .cm ⁻⁵)	1258 ± 239	1508 ± 334	0.03*
Alx _a (%)	23 ± 6	27 ± 7	0.06
aPWV _b (m/sec)	5.4 ± 0.1	5.5 ± 0.2	0.8

Data are means ± S.D. a=adjusted for age, height and heart rate. b=adjusted for MAP. * $P < 0.05$ is statistically significant. (SBP= Systolic blood pressure, DBP= Diastolic blood pressure, PP= pulse pressure, MAP= Mean arterial pressure, CO=cardiac output, SV= stroke volume, PVR= peripheral vascular resistance)

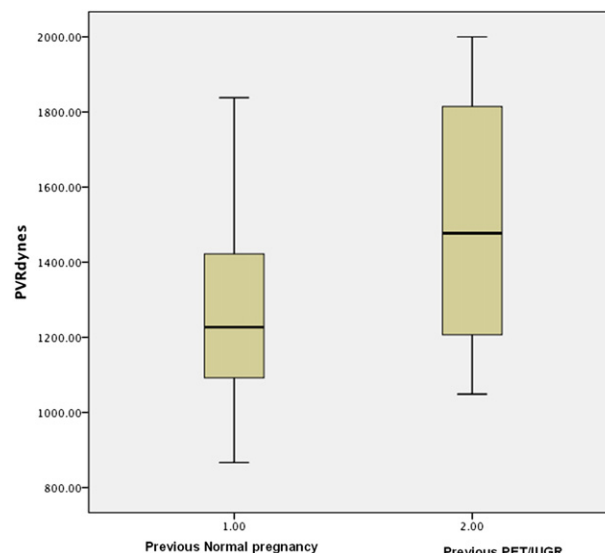


Figure 1 Differences of mean PVR in the two groups ($P=0.03$)

3.2

SPECTRAL ANALYSIS OF CAROTID DISTENSION RATE AND R-R INTERVAL (SPONTANEOUS BAROREFLEX ACTIVITY) PREDICTS CORONARY HEART DISEASE RISK IN PATIENTS WITH MODERATE CHRONIC KIDNEY DISEASE AND IN THOSE WITH NORMAL RENAL FUNCTION: THE EPP3 STUDY

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The classic technique to evaluate the spontaneous baroreflex is the spectral analysis of finger blood pressure and heart rate. However, baroreceptors respond to deformation and not to pressure per se and the results obtained with this technique can be influenced by the vascular component of the baroreflex. Recently, the spectral analysis of carotid distension rate has been used to study the neural baroreflex. The aim of this study is to test this new technique to predict the coronary heart disease (CHD) risk in patients with moderate chronic kidney disease (CKD) and in those with normal renal function.