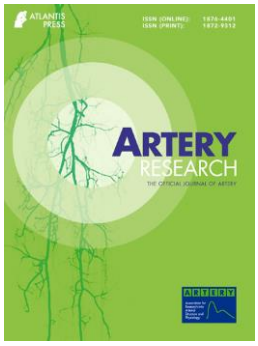

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P5.03: THE CENTRAL HEMODYNAMIC RESPONSE TO POSTURAL STRESS IS UNFAVOURABLY ALTERED IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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measurements as 'baseline' against which changes in normal and complicated pregnancies have been compared. Moreover, the extent of changes in maternal arterial stiffness and central haemodynamics very early in pregnancy is not known.
Our aim was to assess pre-pregnancy to early pregnancy maternal cardiovascular adaptation including arterial stiffness.
Method: 44 women planning to conceive were prospectively recruited (22-40yrs). Cardiovascular assessments were performed pre-pregnancy and repeated very early in pregnancy (2-3 weeks after the first positive pregnancy test) including brachial and central blood pressures, augmentation index (AIx), aortic pulse wave velocity (aPWV) and cardiac output (CO).
Results: While heart rate ($P=0.001$) and cardiac output ($P=0.005$) increased significantly, both brachial and central blood pressure were significantly reduced ($P<0.001$) within 2-3 weeks of conception. AIx was also significantly reduced, even after adjustment for the change in HR ($P=0.005$) along with a reduction in peripheral vascular resistance ($P=0.008$). aPWV was unaltered, even after adjustment for MAP ($P=0.6$).
Conclusion: This is the first study to investigate changes in AIx and PWV very early in pregnancy. We have demonstrated profound changes in brachial and central blood pressures and AIx within 2-3 weeks of conception, with no change in aPWV. Furthermore, late first trimester data cannot be used as 'baseline' data in order to identify the extent of cardiovascular adaptation in pregnancy as has been hitherto assumed.

Table 1 Pre-pregnancy to early pregnancy (2-3 weeks after positive pregnancy test) cardiovascular changes

	Pre-pregnancy	Early pregnancy	$P*$
HR (beats/min)	69 ± 11	73 ± 10	0.001
Brachial SBP (mm Hg)	108 ± 9	103 ± 7	<0.001
Brachial DBP (mm Hg)	70 ± 7	64 ± 5	<0.001
MAP (mm Hg)	84 ± 8	77 ± 5	<0.001
CSBP (mm Hg)	98 ± 9	91 ± 6	<0.001
CO (L/min)	5.5 ± 1.0	5.9 ± 1.1	0.005
SV (ml)	76 ± 10	77 ± 14	0.6
CI (L/min/m^2)	3.2 ± 0.6	3.5 ± 0.6	0.003
PVR (dynes.s^{-1}.cm^{-5})	1253 ± 253	1074 ± 223	0.008
Supine AIx_a (%)	20 ± 9	13 ± 9	<0.001
Supine AIx_b (%)	19 ± 10	13 ± 9	0.005
$aPWV_a$ (m/sec)	5.2 ± 0.6	5.0 ± 0.5	0.1
$aPWV_c$ (m/sec)	5.1 ± 0.5	5.2 ± 0.5	0.6

Data are means ± SD. a= unadjusted. b= heart rate adjusted. c=adjusted for Mean arterial pressure.
*$P<0.05$ is statistically significant
(Heart rate= HR, SBP= Systolic blood pressure, DBP= Diastolic blood pressure, MAP=Mean arterial pressure, CSBP= Central systolic blood pressure, CO=cardiac output, SV= stroke volume, CI= Cardiac Index, PVR= peripheral vascular resistance)

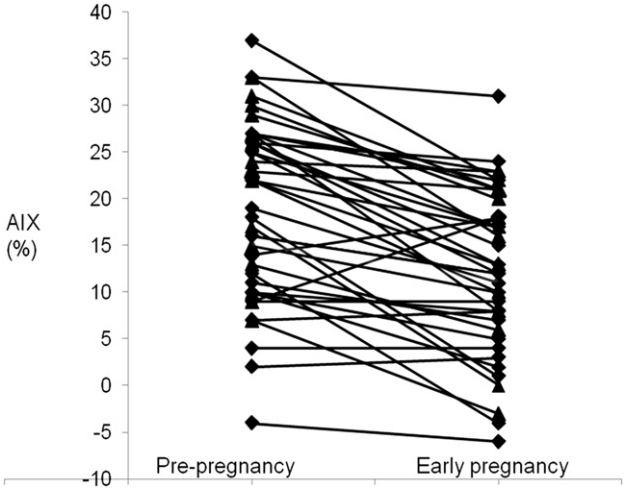

Figure 1 Pre-pregnancy to early pregnancy changes in AIx (%)

P5.02
FREQUENT AND WIDESPREAD VASCULAR ABNORMALITIES IN HUMAN STAT3 DEFICIENCY

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Background: STAT3 deficiency is responsible for autosomal dominant hyper-IgE syndrome characterized by recurrent bacterial and fungal infections, connective tissue abnormalities, hyper-IgE and Th17 lymphopenia. Although vascular abnormalities have been reported in some patients, the prevalence, characteristics and etiology of these features have yet to be described.
Methods We prospectively screened 21 adult STAT3-deficient patients (median age: 26; range 17 - 44) for vascular abnormalities. They were explored with whole-body magnetic resonance imaging angiography, coronary multislice computed tomography and echotracking-based imaging of the carotid arteries. We also assayed for serum biomarkers of inflammation and endothelial dysfunction. Finally, murine models of aortic aneurysm were studied in the presence and absence of inhibitors of STAT3-dependent signaling.
Results: Brain abnormalities (white matter hyperintensities, lacunar lesions suggestive of ischemic infarcts, atrophy) were found in 95% of patients. Peripheral and brain artery abnormalities were reported in 84% of patients, whereas coronary artery abnormalities were detected in 50%. The most frequent vascular abnormalities were ectasia and aneurysm. The carotid intima-media thickness was markedly decreased, with a substantial increase in circumferential wall stress indicating the occurrence of hypotrophic arterial remodeling in this STAT3-deficient population. Systemic inflammatory biomarker levels correlated poorly with the vascular phenotype. In vivo inhibition of STAT3 signaling or blockade of IL-17A resulted in a marked increase in aneurysm severity and fatal rupture in mouse models.
Conclusions Vascular abnormalities are highly prevalent in STAT3-deficient patients. This feature is consistent with the greater susceptibility to vascular aneurysm observed after inhibition of STAT3-dependent signaling in mouse models.

P5.03
THE CENTRAL HEMODYNAMIC RESPONSE TO POSTURAL STRESS IS UNFAVOURABLY ALTERED IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Background. An abnormal brachial BP response to postural stress is associated with increased risk of developing hypertension and stroke. However, the hemodynamic responses contributing to changes in central BP with postural stress are not well characterised, and we aimed to determine these in healthy controls (HC) and patients with type 2 diabetes (T2DM); in whom we hypothesised would have an abnormal postural response..
Methods. 20 HC (53±8yrs,45% male) and 21 T2DM (61±9yrs,48% male) patients underwent measurement of central BP (by radial tonometry), with simultaneous bioimpedance cardiography to determine stroke volume (SV) and cardiac output (CO), in seated and standing postures. Systemic vascular resistance (SVR; mean arterial pressure/CO), and arterial elastance (E_A; end systolic pressure/SV) were calculated. Postural changes were defined as seated minus standing values.
Results. Central pulse pressure (PP) was higher in patients with T2DM and did not significantly change from the seated-to-standing positions ($P=0.461$), whereas there was a significant decrease upon standing in HC ($P=0.022$). The change in central SBP correlated with the change in SVR and E_A in HC ($r=0.67$, $P=0.002$ and 0.68, $P=0.001$ respectively), but not in patients with T2DM ($r=-0.05$, $P=0.790$ and $r=0.03$, $P=0.910$ respectively). SV was the only significant correlate of change in central systolic BP in patients with T2DM ($r=0.62$, $P=0.004$), and this was not observed in HC ($r=-0.08$ $P=0.760$).
Conclusions. Central hemodynamic responses to postural stress are altered in patients with T2DM and result in persistent elevation of central PP while standing. This may contribute to increased cardiovascular risk associated with T2DM.