Assessment of maternal vascular stiffness indices in three trimesters of normal pregnancy

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Abstract
Normal pregnancy is associated with intense alterations in the maternal cardiovascular system. The aim of the present study was, to assess the influence of normal pregnancy on maternal central aortic pressures, arterial stiffness, and arterial wave reflection using non-invasive PC based cardiovascular risk analysis system (Periscope™). The current study was conducted on 137 women with, normotensive, healthy, singleton pregnancies at first trimester (n=42), second trimester (n=48), third trimester (n=47) of pregnancy and 35 age matched non-pregnant controls. There was no significant correlation between the estimated means for age and systolic and diastolic blood pressure. There was progressive and significant increase in BMI as pregnancy progresses (p=0.0001). Heart rate rose significantly from Pre-pregnant to second and second to third trimesters (P<0.003). There were no significant changes observed in central aortic diastolic pressure (AoDiaBP) as pregnancy progressed (p=0.235) however Post Hoc comparisons showed a significant increase in central aortic systolic blood pressure (AoSysBP) and central aortic pulse pressure (AoPP) during first trimester when compared with non pregnant control group (p=0.039 and 0.048 respectively). There was significant increase in central aortic augmentation pressure (AoAugP) in first trimester compared to non pregnant control group (p=0.024). All the parameters of central aortic pressures were increased in the first trimester but decreased in the second trimester and again increased in the third trimester of pregnancy. There was a significant drop in Brachial-Ankle Pulse wave Velocity (baPWV) during first trimester of pregnancy compared to non-pregnant control group (p=0.0001) after that there is a progressive increase in baPWV in second and third trimester of pregnancy. In the third trimester baPWV is increased to more than non-pregnant control group but it was non significant (p=0.562) however it was significantly higher than first trimester (p=0.0001). Carotid-femoral Pulse wave velocity (cfPWV) also followed the same sequence as BaPWV but the drop in cfPWV during first trimester was not significant (P=0.135). All of the variables of hemodynamic and arterial compliance differed between participants with various trimester of pregnancy and non-pregnant control group. A significant up and down changes in Augmentation index (Aix) was observed from control to first, second and third trimester of pregnancy (3.14 to 6.74 to 2.63 to 10.51 respectively, P<0.0001). To summarize our report show that normal pregnancy is associated with a significant cardiovascular adaptation indicated by alteration in central aortic blood pressure, augmentation index and pulse wave velocity.

Introduction
Normal pregnancy is linked with increased blood volume, heart rate, cardiac output and marked
decrease in peripheral vascular resistance and a decreased mean arterial blood pressure (1-2). The decrease in peripheral vascular resistance and generalized vasodilatation is linked with increased aortic compliance (3-4). Aortic compliance can be quantified by assessing arterial stiffness which in turn be evaluated by measuring Augmentation index (Alx), Pulse wave velocity and central aortic pressure profiles. Arterial stiffness is one of the core determinants of central aortic pressure and is a self-sufficient predictor of adverse cardiovascular outcome (10). Several studies in non-pregnant participants have shown that arterial stiffness is increased in patients with risk factors for cardiovascular disease such as hypertension, hypercholesterolemia, and diabetes mellitus (6-7). Hypertensive disorders in pregnancy are associated with systemic endothelial dysfunction leading to impaired physiological vasodilatation. Recent evidence has shown central aortic pressures obtained through pulse wave analysis, at less than 14 weeks of gestation, to be predictive of pre-eclampsia (16). In addition, some other studies in pregnancy suggested that preeclampsia is characterized by increased arterial stiffness during pregnancy (8-9). Moreover, higher arterial stiffness is associated with preeclampsia compared with normal pregnancies (18). Preeclampsia is a common cardiovascular problem during pregnancy that leads to significant maternal and fetal mortality and morbidity. Within healthy, normotensive pregnancy, pulse wave velocity is more directly associated with birth weight than mean arterial pressure, signifying that arterial stiffness may represent maternal adjustment to pregnancy better than blood pressure (15).

The physiological basis of altered hemodynamic of the heart and systemic arteries in pre-eclamptic patients has not been well described (17). Insufficient cardiovascular adjustment in early pregnancy usually predicts its clinical presentation and it is associated with an increased long-term risk of maternal cardiovascular disease (19). Arterial stiffness amongst females is affected by the influence of both estrogen and progesterone on structural and functional components of the arterial system (11). There is inadequate information available regarding maternal central hemodynamic, central aortic pressure and arterial stiffness in pregnancy and sequence of maternal adaptation in various trimesters of gestation. Augmentation index and pulse wave velocity (PWV) are the key measures of central arterial pressure and arterial stiffness. Evaluation of augmentation index (Alx) and arterial stiffness is possible noninvasively by the simple, cost effective, PC based technique of cardiovascular risk assessment (5). With periscopic arterial waveform, it is likely to assess central aortic pressures and augmentation index (Alx), a measure of arterial wave reflection while with PWV it is possible to assess the stiffness carotid-femoral pulse wave velocity (cfPWV) in the carotid to femoral part of the arterial system and brachial ankle pulse wave velocity (baPWV) in the Brachial to ankle part of arterial system. What are the physiological basis of such alterations during various trimester of gestations are not well known and also less information available. However some investigators reported that in the pre-pubertal and post-menopausal years, the female sex steroids is less therefore arterial stiffness is more amongst women than age-matched men (12). During the reproductive years, female sex steroid hormones fluctuate periodically during the menstrual cycle and increase considerably in pregnancy. Some other human studies show the early effect of pregnancy reducing arterial stiffness is well recognized (13). On the other hand, data are inconsistent with reference to the effect of sex steroids on arterial stiffness for the duration of later gestation (14). Understanding the relationship between various trimester of pregnancy and arterial stiffness may, therefore, not only inform understanding of its physiology but may also increase our understanding of the association between preeclampsia and subsequent cardiovascular disease. The aim of the present study was, to assess the influence of normal pregnancy on maternal central aortic pressures, arterial stiffness, and arterial wave reflection using non-invasive PC based cardiovascular risk analysis system (Periscope™).

Materials and Methods

Study design

The study was conducted on women with viable,
singleton pregnancies and without any major or minor medical or surgical illnesses. The study participants were recruited from Anti-natal clinic of Obstetrics & Gynecology department of BPS Govt. Medical College for women, Khanpur Kalan, Sonepat and followed up till delivery and puerperium. The study was approved by the Institutional Ethics Committee of BPS Govt. Medical College for women, Khanpur Kalan, Sonepat. A prior written consent was obtained from all the study participants. The study population was comprised mostly of rural and semi urban population of Sonepat district. Targeted sample sizes of 200 eligible pregnant women were enrolled into the study from antenatal clinics. A suitable sampling of study participants looking for antenatal care at the outpatient clinics were screened for study recruitment. The final sample size of the study was 137 having accounted for around 30% dropout rate and 35 age matched non-pregnant controls. The sub groups of the study participants includes first trimester (n=42), second trimester (n=48), third trimester (n=47) of pregnancy and 35 age matched non-pregnant controls. Exclusion criteria included multiple pregnancy, fetal anomalies, and a history of essential hypertension, previous pre-eclampsia, renal disease, autoimmune disorders, diabetes or women with medication that could affect blood pressure. Data entry was done by two independent investigator of the research project in 2 separate access databases. Data analysis was carried out using SPSS version 14.0. Analysis included distribution of sample characteristics in relation to various trimesters of pregnancy and non pregnant control in proportions and central tendencies; and their crude associations using mixed models for correlated data. Data analysis was done by One-way ANOVA and Tukey Krammer post-hoc test is applied for multiple comparisons between various groups of pregnancy with non pregnant control group. 95% Confidence Interval was taken for all the variables. The mean difference was significant at the 0.05 level.

**Recruitment criteria**

At selection of the study group, basic demographic profiles such as marital status, education, and employment was collected. Women who were unsure about study participation were contacted within the week and after 2 failed attempts of telephonic contact within 2 weeks; they were deemed ‘not contactable’ and therefore considered ineligible for inclusion. Women who had given written consent for the study and later miscarried or decided to deliver in a different place were considered as ‘ineligible’.

**Study Procedures**

After written consent, the study participants were followed-up till delivery. All cardiovascular assessments were performed in the same room at room temperature. Participants were restricted from tea, coffee or any kind of liquid intake that leads to cardiovascular impact on the day of the study and rested for at least 10 minutes prior to the measurements. During periscopy, the women did not move or speak. Central aortic blood pressures, arterial stiffness parameters and augmentation index being the exposure of interest were measured over 3 study visits at 12-14, 20-24 & 32-36 weeks of gestation. Study follow-up for outcome ascertainment were continued till hospital discharge after delivery.

Main exposure under study were central aortic pressure, Arterial Stiffness Index (ASI), brachial-ankle Pulse-Wave velocity (baPWV), Carotid Femoral Pulse-Wave velocity (cfPWV), Ankle Brachial Index (ABI), and augmentation index (Alx). These parameters were assessed by a non-invasive PC based Arterial Health Assessment Analysis system (PeriScope™ RMS Chandigarh). The outcomes under follow-up were gestational hypertension and pre-eclampsia. Periscopic evaluations were repeated at 12-14, 20-24 & 32-36 weeks of gestation in all cases and 6 weeks post-partum in pre-eclamptic patients only. Other measures include lifestyle factors such as age of the patient, smoking, physical exercise, height and weight measurements. Liver Function Test, Renal Function Test, urinary protein and complete blood count were performed in all the study population.
**Flowchart for Study Procedures**

Recruitment & Consent taking  
Study procedure at each visit

**Physical Measurements**: Height, Weight and Blood Pressure

**Questionnaires**: Lifestyle, sleep and past medical history

**Clinical Examination**: Medical, Surgical, obstetrical, Gynecological

**Complete blood profile**: RBC count, WBC Count, Hb%, ABO, Rh, Blood sugar

**Urine**: Routine and protein

**U/S Doppler Periscopy**: Cardiovascular Risk analysis (PWVs, ASI, ABI, Alx, CA Stifness etc)

Repeat Procedure at 12-14, 20-24 & 32-36 weeks gestation

Questionnaires used to gather information pertaining to socio-economic, lifestyle, sleep and maternal coping during pregnancy. Interviewer administered structured questionnaires were used to collect demographic data on variables such as maternal and partner’s date of birth, race, education, occupation, type of housing, household size and income; lifestyle variables such as alcohol and coffee intake, physical exercise and daily activities and smoking habits; and lastly personal or family history of gestational diabetes, hypertension or pre-eclampsia. At study enrolment, baseline information on socio-economic, personal, medical and obstetric history was obtained through an interviewer administered questionnaire.

For exclusion of gestational hypertension and pre-eclampsia, the criteria specified by working group on high blood pressure were applied (20).

**Results**

The baseline characteristics of the recruited study participants are shown in Table I. There were no significant differences in baseline characteristics among these women recruited in the three trimesters. There was no significant correlation between the estimated means for age and systolic and diastolic blood pressure. There was progressive and significant increase in BMI as pregnancy progressed (p=0.0001; Table I). Heart rate rose significantly from Pre-pregnant to second and second to third trimesters (P<0.003; Table I). We compared the central aortic pressure parameters in all the three trimesters of pregnancy with non pregnant control group. There were no significant changes observed in central aortic diastolic pressure (AoDiaBP) as pregnancy progressed (p=0.235, Table II) however Post Hoc comparisons showed a significant increase in central aortic systolic blood pressure (AoSysBP) and central aortic pulse pressure (AoPP) during first trimester when compared with non pregnant control group (p

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (n=35)</th>
<th>First trimester (n=42)</th>
<th>Second trimester (n=48)</th>
<th>Third trimester (n=47)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25.57±3.97</td>
<td>24.79±2.95</td>
<td>25.39±3.97</td>
<td>24.72±3.11</td>
<td>0.608</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.98±2.74</td>
<td>24.17±3.69</td>
<td>25.67±3.41</td>
<td>27.46±4.05***</td>
<td>0.0001</td>
</tr>
<tr>
<td>Heart Rate (beat/min)</td>
<td>68.71±13.93</td>
<td>71.19±16.96</td>
<td>77.67±20.06º</td>
<td>80.89±13.92***</td>
<td>0.003</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>115.20±7.70</td>
<td>120.26±21.20</td>
<td>116.88±20.33</td>
<td>115.77±17.85</td>
<td>0.585</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>79.37±6.85</td>
<td>81.86±11.57</td>
<td>79.83±10.56</td>
<td>82.66±10.39</td>
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</tr>
<tr>
<td>PP (mmHg)</td>
<td>35.83±6.46</td>
<td>38.40±12.78</td>
<td>37.04±11.89</td>
<td>33.11±10.31</td>
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Data analysis was done by One-way ANOVA and Tukey Krammer post-hoc test is applied for multiple comparisons between various groups of pregnancy with non pregnant control group. *P<0.05, **P<0.005, ***P<0.0005; 'P<0.05, 'P<0.005, 'P<0.0005, <=0.05 between control and second.* represents comparison between Control and Third trimester, º represents comparison between first trimester and Third trimester. BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, PP: Pulse pressure.
There was a significant decrease in Brachial-Ankle Pulse wave Velocity (baPWV) during first trimester of pregnancy compared to non-pregnant control group (p=0.0001; Table III) after that a progressive increase in baPWV in second and third trimester of pregnancy was observed. In the third trimester baPWV is increased to more than non-pregnant control group but it was not significant (p=0.562) however it was significantly higher than first trimester (p=0.0001; Fig. I & Table IV, V). Carotid-femoral Pulse wave velocity (cfPWV) also followed the same sequence as BaPWV but the drop in cfPWV during first trimester was not significant (p=0.135; Table III). There was a progressive increase in cfPWV in second and third trimester of pregnancy however it was not significant (p=0.166, 0.562 respectively; Table III). All of the variables of hemodynamic and arterial compliance differed between the non-pregnant control and women with various trimester of pregnancy (Table III). A significant increase and decrease in Augmentation index (Aix) was observed from control to first, second and third trimester of pregnancy (3.14 to 6.74 to 2.63 to 10.51 respectively, p<0.0001; Table III). There were no significant differences observed in Ankle brachial index from control to any gestations (Table III).

### Discussion

The study has demonstrated that normal pregnancy is associated with a significant increase in central systolic blood pressure, pulse pressure and central aortic augmentation pressure during first trimester. After first trimester there was decrease in the central aortic pressures, reached their nadir at mid-pregnancy, and rose thereafter to approximately either non-pregnant levels or more than non-pregnant level by the third trimester of pregnancy. Healthy

### Table II: Central aortic pressure in the different study group participants.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (n=35)</th>
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<th>Second trimester (n=48)</th>
<th>Third trimester (n=47)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AoSysBP (mmHg)</td>
<td>107.23±8.27</td>
<td>117.83±2.99**</td>
<td>108.02±20.47</td>
<td>111.17±18.61</td>
<td>0.039</td>
</tr>
<tr>
<td>AoDiaBP (mmHg)</td>
<td>73.74±7.93</td>
<td>77.38±11.74</td>
<td>73.83±10.56</td>
<td>76.74±10.47</td>
<td>0.235</td>
</tr>
<tr>
<td>AoPP (mmHg)</td>
<td>33.49±6.45</td>
<td>40.31±14.53*</td>
<td>31.92±15.05</td>
<td>34.23±11.14</td>
<td>0.013</td>
</tr>
<tr>
<td>AoAugP</td>
<td>4.51±2.73</td>
<td>9.05±10.02*</td>
<td>5.15±8.37</td>
<td>5.45±6.22</td>
<td>0.030</td>
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Data analysis was done by One-way ANOVA and Tukey Kramer post-hoc test is applied for multiple comparisons between various groups of pregnancy with non pregnant control group. *<P<0.05, **<P<0.005, ***<P<0.0005; *<P<0.05, **<P<0.005, ***<P<0.0005, * represents comparison between Control and first trimester, # represents comparison between first trimester and second trimester. AoSys BP: Central Aortic Systolic Blood Pressure, AoDia BP: Central Aortic Diastolic Blood Pressure, AoPP: Central Aortic Pulse Pressure, AoAugP: Central Aortic Augmentation pressure.

### Table III: Pulse wave velocity, ankle brachial index and augmentation index in the study participants.

<table>
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<th>Parameters</th>
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<th>Second trimester (n=48)</th>
<th>Third trimester (n=47)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>cfPWV (cm/sec)</td>
<td>890.70±140.27</td>
<td>777.89±164.83</td>
<td>856.19±315.03</td>
<td>892.11±317.09</td>
<td>0.146</td>
</tr>
<tr>
<td>baPWV (cm/sec)</td>
<td>1398.64±159.88</td>
<td>1145.36±152.34***</td>
<td>1302.35±259.67***</td>
<td>1455.30±314.31</td>
<td>0.0001</td>
</tr>
<tr>
<td>ABI</td>
<td>1.11±0.050</td>
<td>1.10±0.082</td>
<td>1.11±0.102</td>
<td>1.10±0.099</td>
<td>0.9403</td>
</tr>
<tr>
<td>Aix</td>
<td>3.14±4.16</td>
<td>6.74±4.26***</td>
<td>2.63±8.01</td>
<td>10.51±7.72***</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Data analysis was done by One-way ANOVA and Tukey Kramer post-hoc test is applied for multiple comparisons between various groups of pregnancy with non pregnant control group. *<P<0.05, **<P<0.005, ***<P<0.0005; *<P<0.05, **<P<0.005, ***<P<0.0005, * represents comparison between Control and first trimester, # represents comparison between first trimester and second trimester. * represents comparison between Control and first trimester, # represents comparison between First trimester and Second trimester. cfPWV: Carotid Femoral Pulse wave velocity, baPWV: Brachial Ankle Pulse wave velocity, ABI: Ankle Brachial Index, Aix: Augmentation Index.
pregnancy is associated with minimal changes in peripheral systolic and diastolic blood pressure, which reaches its lowest levels at second trimester as confirmed in our study (21). Hence, it seems that alterations in aortic central blood pressure in pregnancy are more pronounced than peripheral BP. This phenomenon is likely to be attributable to a reduction in the impact of wave reflection on the central pressure waveform (22). The results of the Maria L et al (2008) also demonstrated that normal pregnancy is associated with alterations in central BP, despite relatively little change in the peripheral BP, and a delay in wave reflection within the arterial tree (23). Another study by Wykrętowicz M et al (2011) reported that healthy pregnancy is associated with increased pulse pressure amplification as well as diminished wave reflection, which results in lower central augmentation index and augmentation pressure. They also observed that women in the third trimester of pregnancy have slightly higher arterial stiffness in comparison with healthy non-pregnant, age- and height-matched controls. They explained that the increased value of measures of arterial stiffness might be secondary to a known physiological increase of cardiac output and the amount of circulating blood (24).

Another study has demonstrated that noninvasively-determined central pulse pressure is more strongly related to vascular hypertrophy, atherosclerosis, and cardiovascular events than is peripheral blood pressure (25). Therefore, assessing changes in central aortic blood pressure may be more beneficial in the early detection of pregnancies complicated by hypertensive disorders (26). This, however, needs further investigation. During normal pregnancy, arterial stiffness increases from the mid trimester to term or third trimester of pregnancy (27). We have also shown that normal pregnancy is associated with a significant decrease in the carotid-femoral and brachial-ankle pulse wave velocity (indicator of maternal arterial properties). Mersich B et al (2005) in his study observed that normal pregnancy is associated with decreased maternal carotid femoral pulse wave velocity (cfPWV) compared to non-pregnant controls (28). This was very much similar to our current observation however Mersich B et al did not investigated the role of brachial-ankle pulse wave velocity that is a very strong indicator of arterial compliance along with carotid femoral pulse wave velocity. Maternal pulse wave velocity has also been assessed by Elvan-Taçspinar A et al in 2004 (29) in pregnancies complicated by preeclampsia and they found that there is increase in PWV, but this increase could have been a mere result of the elevated mean arterial pressure associated with preeclampsia. In our current study, we observed that there was a significant drop in Brachial-ankle Pulse wave Velocity (baPWV) during first trimester of pregnancy compared to non-pregnant control group after that there is a progressive increase in baPWV in second and third trimester of pregnancy. In the third trimester baPWV is increased to more than non-pregnant control group but it was non significant however it was significantly higher than first trimester (p=0.0001; Table III). Carotid-femoral Pulse wave velocity (cfPWV) also followed the same sequence as baPWV but the drop in cfPWV during first trimester was not significant (P=0.146; Table III). When we compared Elvan-Taçspinar A et al observation with our results, we found a difference in arterial properties indicated by cfPWV and baPWV these differences can be explained by the fact that our study is in normal healthy and normotensive women and the study by Elvan-Taçspinar A et al was amongst pregnancies complicated by preeclampsia. These comparative study findings may offer a new insight in the maternal adjustment to pregnancy and may prove to be useful in the early detection of pregnancies complicated by hypertensive disorders. This may imply that the hemodynamic changes in pregnancy with increased aortic diameter, at the level of the left outflow tract (21), and increased circulating blood volume, attributable to volume expansion (30), may mask the alterations and consequently the usefulness of these parameters in the assessment of arterial stiffness in normal pregnancy. A possible explanation of maternal arterial adaptation during pregnancy is dramatic change in arterial compliance during pregnancy. Arterial compliance increases during the first trimester and remains elevated throughout the remainder of pregnancy. Thus, both steady and pulsatile afterload decrease occur during normal pregnancy (31).

Our results of a rise in arterial stiffness from the
second trimester to term are supportive of previous studies using brachial-ankle Pulse wave velocity (baPWV) as a composite measure of systemic and central stiffness and augmentation index (32, 33). Other studies report no change in Pulse wave velocity with gestation or a general decrease in pulse wave velocity and augmentation index during pregnancy (34, 35). However these studies were conducted earlier during third third-trimester than in our study.

A significant increase in the first trimester and then decrease in the second trimester and again further significant increase in the third trimester of pregnancy in augmentation index (Alx) was observed in our study. A previous cross-sectional study by Roman MJ et al (2007) on 53 pregnant women, from 17 to 36 weeks of gestation, reported that Alx was lower throughout these gestations compared to nonpregnant controls (25). However, the investigators did not investigated augmentation index in first trimester and also did not examine the relationship of Alx in various trimester of pregnancy and also did not adjust Alx for maternal age, heart rate, and blood pressure, all of which are known determinants of Alx. A very much similar report was observed by Macedo ML et al (2008), they confirms that in normal pregnancy, augmentation index (Alx) varies throughout pregnancy, reaching its nadir in the second trimester and rising again in the third (36,37). Individual women followed longitudinally throughout pregnancy showed a significant fall in augmentation index (Alx) in the second trimester, with a significant rise again in the third. These changes were not linked to variations in blood pressure, which in our study group did not change significantly through pregnancy. These observations advocate that changes in arterial stiffness may result from changes in the levels of vaso-active substances such as progesterone and relaxin, and a large expansion of fluid volume during pregnancy.

The decrease Alx in second trimester of pregnancy in our study may be a effect of the enhanced nitric oxide (NO) production induced by estrogen may increase reflection site distance within the vasculature and reduce wave reflection amplitude associated with normal pregnancy (38, 39). However a significant increase of Alx in third trimester of pregnancy may be explained by inhibition of nitric oxide (NO) theory as investigated by Wilkinson IB et al (40), Weber T et al (41), McEniery CM et al (42) and Holden DP et al (43).

The decrease in arterial stiffness during first trimester of pregnancy compared with non pregnant control is likely to arise from several factors. Estrogen has encouraging effects on the endothelium and vascular smooth muscle cells, with many of the hemodynamic changes observed in normal pregnancy being mimicked in non-pregnant animals chronically exposed to estrogen (44). Both the endothelium and vascular smooth muscle cells express receptors for estrogen and progesterone through which they can regulate vascular tone (45). Therefore, they are likely to influence arterial stiffness through effects on mean arterial pressure, as well as structural changes to elastin, collagen, and smooth muscle in the arterial wall (46). Progesterone has often been thought to have opposing vascular effects to estradiol, although it has favorable vascular effects in vitro and in vivo (45). However, despite the increased serum estradiol and progesterone concentrations with advanced gestation, we report an increase in arterial stiffness in the third trimester of pregnancy that we postulate that it is attributed to factors other than sex steroids.

Conclusion

The aim of the present study was to investigate the maternal cardiovascular adaptation during various trimesters of pregnancy by assessing arterial stiffness, central aortic pressure and augmentation index in normotensive pregnant women. The results of the present study demonstrate that normal pregnancy is associated with profound alterations in central blood pressure, despite relatively little change in the peripheral blood pressure, and a delay in wave reflection within the arterial tree. Furthermore, in the present study we cannot comment on the longitudinal changes of maternal arterial stiffness during pregnancy complicated by pre-eclampsia. However, our results should encourage further research, involving larger number of women, to establish the physiology of maternal adaptation to pregnancy. Our findings offer a further insight in the pathophysiology of maternal cardiovascular adaptation to pregnancy.
The degree to which this information will be useful in the detection of pathological pregnancies particularly pregnancy induced hypertension remains to be determined.

**Future perspectives**

Using the noninvasive PC based cardiovascular risk assessment technique (Periscope™); we have shown that normal pregnancy is associated with profound alterations in central blood pressure, despite relatively little change in peripheral blood pressure, and a delay in wave reflection within the arterial tree. This is a significant new step over the already established knowledge in this area, which is so far based on the assessment of peripheral blood pressure in pregnancy. It is also of particular interest because previous research has demonstrated that central and peripheral blood pressure are not the same and several factors can exert some degree of different effects on them (19). These findings offer a new insight in the maternal adaptation to pregnancy and may prove to be useful in the early detection of pregnancies complicated by hypertensive disorders.

**References**


