Venous and arterial behavior during normal pregnancy

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1Department of Anesthesiology, A. Bédère Hospital, 92140 Clamart; and 2Department of Internal Medicine (Medicine 1) and Institut National de la Santé et de la Recherche Médicale, U 337, Broussais Hospital, 75674 Paris Cedex 14, France

Edouard, D. A., B. M. Pannier, G. M. London, J. L. Cuche, and M. E. Safar. Venous and arterial behavior during normal pregnancy. Am. J. Physiol. 274 (Heart Circ. Physiol. 43): H1605–H1612, 1998.—To assess the contribution of the arterial and venous systems in the hemodynamic changes of normal pregnancy, we studied blood flow, vascular resistance, venous tone, and the viscoelastic properties (“creep”) of the upper and lower limbs (using plethysmography), aortic distensibility (using pulse wave velocity measurements), and cardiac dimensions (using echocardiography) in nine healthy women. Studies were longitudinally performed at the first (10–13 wk) and third (33–38 wk) trimesters of pregnancy in comparison with the period between the third and sixth month after delivery. From the first trimester, heart rate significantly increased while systemic blood pressure and limb vascular resistances did not change significantly and aortic distensibility increased (P < 0.05). Lower limb viscoelastic properties decreased at the third trimester (P < 0.05) and venous tone increased from the first trimester (P < 0.01), whereas little changes were observed at the site of upper limbs. The decrease in calf venous tone was significantly correlated with the increase in left ventricular diastolic diameter at the first (P < 0.001) and the third trimester (P < 0.05). The study provides evidence that during normal pregnancy, changes in the arterial and venous sides of the circulation occur independently of pressure alterations. The increase in venous tone, contributing to preload augmentation, and the decrease in aortic stiffness, reducing afterload, both optimize cardiac function until delivery.

venous tone; aortic compliance; arterial distensibility; cardiac function

IT IS WELL ACCEPTED that a normal pregnancy is characterized by a large increase in total blood volume and in cardiac output (31, 32, 48). Blood volume expansion appears early during the pregnancy and rises up to 50%. Cardiac output rises up to 40–50% above nonpregnant values, and the highest increase is reached halfway through gestation. Cardiac output is the product of heart rate and stroke volume. Heart rate is known to reach a 10–27% increase between weeks 4 and 36 of pregnancy (10). However, an increase in stroke volume might also play an important role at the early phase. Stroke volume appears elevated at least until weeks 28–32 (31). The mechanism(s) causing the increase in stroke volume are not yet well established. In pregnant rats and women, an increased cardiac contractility has been suggested (13, 36, 44), but the differentiation between the contractile properties of cardiac muscle itself and the contractile changes that result from altered preload and afterload is difficult to establish. The possibility should be considered that an increase in venous return also plays a major role. In the literature, numerous data indicate a significant increase in venous capacitance (11, 17, 18, 28, 35, 47). However, significant discrepancies have also been reported, depending mainly on the site of measurement of venous variables (40, 46). Furthermore, the early venous changes could not be adequately detected because pregnant women were not studied longitudinally and because there was a large measurement error associated with the method used. Finally, the venous system was often investigated alone in pregnant women. No simultaneous evaluation of vascular resistance and of arterial distensibility was performed, thus allowing a concomitant evaluation of the consequences of the arterial and venous changes on the cardiac structure and function during pregnancy. Our working hypothesis is that alterations of both the arterial and venous sides of the circulation contribute to optimize cardiac structure and function in pregnant women.

The aim of this work was 1) to investigate the upper and lower limb behavior throughout a normal pregnancy in terms of venous tone and viscoelastic properties of the venous wall (“creep,” as derived from the hysteresis of the pressure-volume curve), 2) to analyze in parallel the aortic distensibility, and, finally, 3) to evaluate the cardiac function and dimensions using echocardiography. Because of the well-established role of neurohumoral factors during pregnancy (4, 9, 16, 19, 27, 30, 37, 42), plasma levels of estrogens, progesterone, and several hormones related to the autonomic and renin-angiotensin systems were also repeatedly measured.

METHODS
Subjects. Our subjects were drawn from a specialized obstetrical outpatient visit to A. Bédère Hospital. From 1989 to 1991, 70 women having a confirmed pregnancy of <14 wk were selected. Of the 70 women, 15 were normotensive, accepted the protocol, and were enrolled in the study after providing written consent and reviewing a complete description of the procedure, according to the ethical committee of the hospital. In these 15 women, blood pressure measured before pregnancy constantly indicated values equal to or lower than 140/90 mmHg. No patient suffered or had suffered from varicose veins or lower limb venous disease, as confirmed by clinical history and examination and Doppler investigation. Three of these 15 patients became hypertensive during pregnancy and therefore were finally excluded, and three withdrew before the end of the study for personal reasons. Consequently, nine women were investigated during the entire protocol, and for them all measurements were completed. All newborns were healthy and ranged in normal body weight (between 3,020 and 3,600 g). Five of these women were nulliparous. Mean age and height were, respectively, 28 ± 4 yr (± 1 SD of the mean) and 159 ± 5 cm. At each hemodynamic period and throughout the clinical follow-up of the pregnancy, the supine or sitting blood pressures recorded by mercury sphygmomanometer were below or equal to 0.01, whereas little changes were observed at the site of upper limbs. The decrease in calf venous tone was significantly correlated with the increase in left ventricular diastolic diameter at the first (P < 0.001) and the third trimester (P < 0.05). The study provides evidence that during normal pregnancy, changes in the arterial and venous sides of the circulation occur independently of pressure alterations. The increase in venous tone, contributing to preload augmentation, and the decrease in aortic stiffness, reducing afterload, both optimize cardiac function until delivery.

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METHODS

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130/85 mmHg. The repeated determinations of proteinuria were constantly negative. The hemodynamic study was performed during three outpatient visits in the center, in the same temperature-controlled room (22 ± 1°C), at the first (10–13 wk) and third trimesters (33–38 wk) of their pregnancy and after the third month following their delivery, which was considered the control period. During this control period, no woman was breast-feeding.

Study design. The investigations began at 8:30 AM after the women ate a standard breakfast without tea or coffee. The women were placed in a 30° left lateral recumbent position to avoid any compression of the vena cava during the investigation periods. An indwelling intravenous catheter was immediately placed in a left forearm vein to permit blood sampling. A cuff connected to a semiautomatic oscillometric blood pressure recorder (Dinamap, Critikon, Chatenay Malabry, France) was placed on the left arm to measure blood pressure every 3 min throughout the entire procedure, except during the 15 min before blood sampling. Mean blood pressure (MBP) was calculated from systolic (SBP) and diastolic blood pressure (DBP) as MBP = DBP + (SBP – DBP)/3. Measurements began after a 30-min supine resting period and involved successively the measurement of aortic distensibility, then upper and lower limb plethysmography, and finally echocardiography.

Aortic distensibility determination. The characteristic impedance of an arterial segment is directly related to regional pulse wave velocity (PWV) according to the widely accepted Moens-Korteweg equation: \( PWV = \sqrt{\frac{Eh}{2\mu}} \), where \( E \) is Young's modulus of the arterial wall, \( h \) is wall thickness, \( R \) is lumen arterial radius, and \( \mu \) is blood density (29). Aortic PWV was determined as the foot-to-foot wave velocity measured from the carotid and femoral arteries. Two pulse transducer heads (Electronics for Medicine) were fixed to the skin over the most prominent part of the right common carotid artery and the right common femoral artery in the groin. The heads (Electronics for Medicine) were fixed to the skin over the most prominent part of the right common carotid artery and the right common femoral artery in the groin. The distance between the two sites (D) was measured on the skin. The time delay (or transit time (TT)) was measured on paper between the foot of the two simultaneously recorded pulse waves (at a speed of 150 mm/s). The foot, which contains the high-frequency information, was defined as the point obtained by extrapolating the wavefront downward and measured from the intersection of this line with the straight line extrapolation of the last part of the diastolic curve. PWV was calculated as \( D/TT \). This was averaged over at least one respiratory cycle, that is to say, about 10 beats.

The variability coefficient (SD/mean) and the repeatability coefficient (RC) were calculated according to the British Standards Institution (7) and the Bland and Altman recommendations (6). RC was calculated as \( \sqrt{D/N} \), where \( D \) is the difference within each pair of measurements and \( N \) is sample size. The intraobserver variability coefficient of this method was previously evaluated as 6.2% (3), and the short-term repeatability coefficient was 0.94 m/s (2). We measured the carotid-to-femoral pulse wave velocity over 2 mo in eight normotensive patients (not included in this study). For a mean value of 8.08 ± 0.80 m/s, the repeatability coefficient was 0.80 m/s.

Hemodynamic investigations of upper and lower limbs. Upper and lower limb blood flow, vascular resistance, and venous variables were determined using strain-gauge occlusion plethysmography with gauges placed around the right forearm and calf and pressure controlled with a mercury sphygmomanometer (Perievin, Société Européenne des Techniques Avancées, Noisy-le-Grand, France) (49). The right forearm was placed in 90° abduction, 5–10 cm above heart level (taking into consideration the left lateral recumbent position). An adjustable arm rest ensured that the forearm remained in the same position relative to the heart. The mercury-in-Silastic strain gauge was applied on the right forearm 6 cm distal to the lateral epicondyle of the humerus and then calibrated. The right leg was placed in an adjustable leg rest, ensuring that the calf was maintained 5–10 cm above heart level. The gauge was placed on the prominent part of the calf and then calibrated. Pneumatic cuffs were placed around the right arm and thigh for venous occlusion. Both cuffs were connected to the same air compressor and mercury manometer for measurement of cuff pressure during inflation and deflation. Venous distensibility was first measured simultaneously on the upper and lower limbs, and thereafter blood flow (BF) simultaneously on both limbs. Local resistances (\( R_L \)) were calculated as \( R_L = (MBP/BF)/60 \) (mmHg·ml⁻¹·s⁻¹).

The venous distensibility was determined by a method adapted from Wood and Eckstein (50), as already published (21, 25, 34). Volume changes measured by the gauge are well correlated with radionuclide plethysmography and highly reproducible (26). The cuff pressures were simultaneously increased by steps of 2–3 mmHg until the volume of the limbs started to increase. The cuff pressure just below that value was considered the zero level of effective venous pressure (minimal occluding pressure). The cuff pressure was increased to 5 mmHg, then to 7.5 mmHg, and then by steps of 5 mmHg to 10, 15, 20, 25, and 30 mmHg of effective venous pressure. At each step the pressure was kept constant for a delay corresponding, for each subject, to 10 heartbeats. The plateau corresponding to the last five heartbeats was then recorded on paper. The same technique was used in the deflation phase, during which cuff pressures were decreased by identical steps from 30 to 0 mmHg. The pressure-volume relationship showed a typical hysteresis and then was analyzed to define simple quantitative parameters of venous viscoelastic properties. The first hemodynamic variable was the venous tone, expressed as the slope of the linear part of the volume-pressure relationship (from 5 to 30 mmHg of cuff pressure) during the inflation phase. The slope of this correlation was expressed in millimeters of mercury per milliliter times 100 ml and represented an index of the elasticity of the venous system. The second variable was the extent of isotonic relaxation (creep) determined as the difference in limb volume between inflation and deflation phases at 20 mmHg. The creep [volume increment at a pressure of 20 mmHg (\( \Delta V_{20} \))] was expressed in milliliters per 100 ml and quantified the hysteresis of the venous pressure-volume curve. The third parameter was the relative change of limb volume observed at the second 30-mmHg step of pressure (the 1st step of the deflation) and corresponded to a global index: the venous capacitance [volume variation at a pressure of 30 mmHg (\( VV_{30} \))]. This variable depends on both the venous tone and the width of the curve hysteresis. For each patient three recordings were performed at 5-min intervals, permitting a return to baseline conditions. The mean value of these three consecutive measurements was taken as characteristic for each subject and for statistical evaluation. In a previous study (38) we showed that forearm venous tone determined according to this noninvasive method was strongly correlated in humans with total effective compliance of the vascular bed evaluated from rapid volume expansion.

Forearm and calf blood flows were measured just after the determination of venous distensibility with the same mercury-in-Silastic strain gauges. Venous occlusion was achieved by cuff inflation to 50 mmHg at a constant rate of 5 mmHg/s.
RESULTS

Table 1 shows the values of body weight, blood pressure, heart rate, and pulse wave velocity. Body weight was increased significantly at the third trimester of pregnancy, but postpartum weight remained slightly but significantly higher than the first trimester body weight. Blood pressures did not change throughout the study, whereas heart rate increased as early as the first trimester (P < 0.01). PWV significantly decreased throughout the pregnancy. This was due to a significant increase in the pulse wave TT (control period: 7.1 ± 0.6 10⁻² s; 1st trimester: 7.8 ± 0.8 10⁻² s; 3rd trimester: 8.4 ± 0.8 10⁻² s; P < 0.01) without any significant change in distance between each recording site (data not shown). Thus there was a significant increase in aortic distensibility of up to 10% at the third trimester compared with the postdelivery value.

Figure 1, A and B, shows the venous pressure-volume recorded curves at the site of the lower and the upper limbs. The values of the venous parameters are indicated in Table 2 for both the upper and lower limbs. For the lower limb venous changes (Fig. 1B), there was a significant (P < 0.001) decrease in curve width (ΔVP₂₀) on the third trimester corresponding to decrease in the viscoelasticity, and an early increase in the venous tone (P < 0.01) from the first trimester. These results indicate a global reduced venous distensibility of the lower limb, as shown by the significant decrease (P < 0.01) in VV₃₀. For the forearm (Fig. 1A), only the width of the curve hysteresis (ΔVP₂₀) significantly increased at the third trimester. Note that whereas at the delivery control period, venous tone and ΔVP₂₀ differed significantly (P < 0.01) between the upper and the lower limbs, no difference was observed at the third trimester (Table 2).

During the pregnancy, blood flows and local resistances did not change in both the upper and lower limbs (Table 2).

Table 3 shows the echocardiographic changes. The aorta annulus diameter was increased at the third trimester (P < 0.05). The LVDD also slightly increased at the third trimester (P < 0.05), whereas no significant change in cardiac thickness was observed.

Table 4 indicates the hormone results. Plasma catecholamine and ANF levels did not change, whereas the sex hormone levels (estradiol and progesterone),
plasma renin activity, and aldosterone levels increased markedly during the pregnancy, particularly at the third trimester. Plasma proteins decreased during pregnancy. Thus, when the hormonal levels were analyzed relative to the plasma protein level (data not shown), only plasma estradiol, progesterone, renin activity, and aldosterone were shown to be significantly elevated along the pregnancy. There was no significant correlation relating hormonal plasma levels to the measured hemodynamic variables. In the overall population, we observed a significant positive relationship between the changes in the lower limb venous tone and the change in LVDD between the control period and the first trimester \( (r = 0.91, P < 0.001) \) (Fig. 2). For the two variables, a similar relationship was observed between the control period and the third trimester \( (r = 0.68, P < 0.05) \) (Fig. 3). LVDD did not correlate with any other variable related to the viscoelastic properties of the veins either for the upper or the lower limbs.

Results in Tables 1–4 did not differ when the patients were divided into two subgroups: multiparity \( (n = 4) \) and nulliparity \( (n = 5) \).

DISCUSSION

The principal findings of this longitudinal study of normal pregnancy were that
1) all the distensibility and viscoelastic components of the lower limb veins significantly decreased, whereas no comparable finding was observed in the upper limb, 2) the changes in lower limb venous tone were strongly correlated with the changes in left ventricular diastolic diameter, and 3) aortic distensibility and diameter significantly increased, whereas at the same period of observation, there was no significant change in the concomitant measurements of systemic blood pressure.

Considerations of methods. Important discrepancies have been previously reported for the study of the peripheral venous system during normal pregnancy. Most of the investigations did not analyze the upper and lower limbs in parallel, did not involve longitudinal measurements, or did not include a control period. Furthermore, in most investigations, the venous system of the upper or the lower limbs was analyzed in terms of global indexes of venous capacitance. With this procedure, the cuff was usually inflated at one level of pressure between 30 and 60 mmHg. Subsequently, venous compliance has been shown to be significantly

Table 2. Limb hemodynamic parameters

<table>
<thead>
<tr>
<th></th>
<th>Control Period (A)</th>
<th>First Trimester (B)</th>
<th>Third Trimester (C)</th>
<th>Fisher’s Test P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Upper limb</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BF, ml·s(^{-1})·100 ml(^{-1})</td>
<td>3.85 ± 1.94</td>
<td>3.22 ± 0.98</td>
<td>5.29 ± 2.89</td>
<td></td>
</tr>
<tr>
<td>RL, mmHg·ml(^{-1})·s·100 ml</td>
<td>1,547 ± 617</td>
<td>1,613 ± 531</td>
<td>1,180 ± 482</td>
<td></td>
</tr>
<tr>
<td>VT, mmHg·ml(^{-1})·100 ml</td>
<td>27.8 ± 6.9</td>
<td>28.9 ± 8.1</td>
<td>22.6 ± 6.4</td>
<td></td>
</tr>
<tr>
<td>VV(_{30}), ml/100 ml</td>
<td>1.04 ± 0.2</td>
<td>1.06 ± 0.23</td>
<td>1.21 ± 0.26</td>
<td></td>
</tr>
<tr>
<td>∆VP(_{20}), ml/100 ml</td>
<td>0.25 ± 0.14</td>
<td>0.34 ± 0.08</td>
<td>0.44 ± 0.22</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Lower limb</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BF, ml·s(^{-1})·100 ml(^{-1})</td>
<td>2.47 ± 0.66</td>
<td>2.34 ± 0.82</td>
<td>2.31 ± 0.90</td>
<td></td>
</tr>
<tr>
<td>RL, mmHg·ml(^{-1})·s·100 ml</td>
<td>2,200 ± 766</td>
<td>2,265 ± 789</td>
<td>2,503 ± 882</td>
<td></td>
</tr>
<tr>
<td>VT, mmHg·ml(^{-1})·100 ml</td>
<td>17.4 ± 4.3</td>
<td>24.2 ± 6.5</td>
<td>26.2 ± 5.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>VV(_{30}), ml/100 ml</td>
<td>1.58 ± 0.35</td>
<td>1.26 ± 0.37</td>
<td>1.08 ± 0.27</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>∆VP(_{20}), ml/100 ml</td>
<td>0.65 ± 0.14</td>
<td>0.65 ± 0.19</td>
<td>0.46 ± 0.22</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Values are means ± SD. One-way repeated-measures ANOVA was performed; if significant, Fisher’s LSD post hoc test among periods A, B, and C was performed. BF, blood flow; RL, local vascular resistance; VT, venous tone; VV\(_{30}\), capacitance; ∆VP\(_{20}\), isotonic volume variation at 20 mmHg (creep).
Hormonal plasma levels

Table 4.

<table>
<thead>
<tr>
<th></th>
<th>Control Period (A)</th>
<th>First Trimester (B)</th>
<th>Third Trimester (C)</th>
<th>Fisher's Test P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma protein, g/l</td>
<td>80 ± 4</td>
<td>74 ± 5</td>
<td>62 ± 2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Epi, pg/ml</td>
<td>25.0 ± 21.1</td>
<td>29.3 ± 27.9</td>
<td>24.6 ± 15.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>NE, pg/ml</td>
<td>259 ± 152</td>
<td>360 ± 230</td>
<td>306 ± 174</td>
<td>0.01</td>
</tr>
<tr>
<td>D, pg/ml</td>
<td>33.9 ± 33.6</td>
<td>29.3 ± 27.9</td>
<td>35.7 ± 22.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Estradiol, pg/ml</td>
<td>82 ± 68</td>
<td>2,703 ± 2,398</td>
<td>19,066 ± 6,263</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Progesterone, pg/ml</td>
<td>1.35 ± 3.6</td>
<td>32.6 ± 13.4</td>
<td>128.0 ± 32.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ANF, pg/ml</td>
<td>38.67 ± 12.18</td>
<td>48.67 ± 21.60</td>
<td>29.0 ± 8.07</td>
<td>0.01</td>
</tr>
<tr>
<td>PRA, ng·ml⁻¹·h⁻¹</td>
<td>39 ± 16</td>
<td>214 ± 110</td>
<td>461 ± 303</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Aldosterone, pg/100 ml</td>
<td>20.1 ± 12.0</td>
<td>30.7 ± 13.3</td>
<td>64.5 ± 13.6</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Values are means ± SE. One-way repeated-measures ANOVA was performed; if significant, Fisher’s LSD post hoc test among periods A, B, and C was performed. Epi, epinephrine; NE, norepinephrine; D, dopamine; ANF, atrial natriuretic factor; PRA, plasma renin activity.
because a rise in cardiac output has been noted in the last or midtrimester and might play a role in the regional observed changes. This point could account for the observed lack of change in MBP. Finally, the relatively small number of subjects participating in the overall investigation might have contributed to reduce the statistical power, particularly for cardiac mass determination (33). However, such a difficulty was partly challenged by the use of longitudinal measurements, a point that is detailed below.

Considerations of findings. The principal finding in this study was that all the measured indexes of venous distensibility and viscoelastic properties were decreased in the lower limbs but not in the upper limbs. At the site of the lower limb, the width of the pressure-volume loop, expressed as the creep (or $DVP_{20}$), was significantly decreased at the third trimester. The tonic venous tone increased from the end of the first trimester. The mechanics of such venous changes are difficult to elucidate. The venous alterations appear as a consequence of two different factors: 1) the distending force applied on the vessel wall (which is here largely dependent on the inward arteriolar flow), and 2) the local muscle tone. In the present study, we did not identify a change in limb blood flow or in systemic blood pressure during the pregnancy. Thus the venous changes could not be considered a passive consequence of arteriolar vasodilation. On the other hand, venous changes differed markedly at the site of lower and upper limbs, suggesting that changes in venous pressure could not explain per se the substantial differences in behavior of the two limbs. Thus the contribution of intrinsic structural and/or functional alterations of the venous wall should be considered (43). It is important to mention that the density of smooth muscle cell (SMC) in the vein wall differs greatly from upper limb to lower limb; it is higher in lower limb and higher in the distal part of the limb. Thus it is clear that, with a larger amount of SMC, the same constrictor stimulus will produce a higher degree of constriction of the vein wall. Furthermore, in the venous system, the degree of innervation is considerably less than in the resistance arteries. Moreover, the amount of innervation differs from saphenous veins to iliac veins and is higher in the former. Thus a smaller amount of reactive muscle in the forearm will produce smaller variations in venous viscoelasticity and/or distensibility during pregnancy.

One of the major results of this study was the significant increase in aortic distensibility observed during normal pregnancy, as evidenced from the average 10% decrease in PWV that occurred at the third trimester. Similar findings have been reported in rats (45). The PWV change could not be related to changes in systemic blood pressure, which was unmodified during the pregnancy. Because aortic distensibility is the ratio between aortic compliance and volume, and because an increase in aortic diameter and volume (Table 3) was also observed during the pregnancy, the increase in distensibility indicates intrinsic change in the aortic wall. As for veins, structural and/or functional factors may be implicated in the observed aortic changes. Because estrogens were increased during the pregnancy and because estrogen receptors are present on SMC in arterial wall (5, 22), it might be suggested that this hormone was responsible for the distensibility changes. In humans, arterial vasodilation may be obtained from acute administration of estrogen (15). In postmenopausal women, the loss of estrogens is associated with increased rigidity of the arterial wall (23). In the present study, we did not observe any correlation between estrogens and hemodynamic variables. We found a positive correlation between the increase in the venous tone of the lower limbs and the changes in the LVDD studied both at the end of the first and the third trimesters. Because we found a decrease in lower limb tonic and viscoelastic distensibilities, it is clear that venous return was markedly augmented during pregnancy, a mechanism involving an increase in the filling of the heart and contributing to the increase or maintenance of cardiac output. On the other hand, because a decrease in aortic PWV and an increase in aortic diameter were concomitantly observed, aortic compliance was substantially decreased. Thus both arterial
and venous adjustments contributed to optimize cardiac function during normal pregnancy.

In conclusion, throughout normal pregnancy, the lower limb venous distensibility and viscoelastic properties were decreased and induced and/or maintained the increase in cardiac filling and cardiac output. In contrast, the upper limb venous system showed a late and small increase in wall time-dependent viscoelasticity. This hemodynamic pattern was observed without any change in limb local vascular resistances or change in mean blood pressure. In addition, although aortic distensibility is often dependent on blood pressure level, we observed an increase in aortic distensibility unrelated to mechanical stress and probably due to intrinsic modifications of the arterial wall. This change contributed to preserve heart vessel adjustments during normal pregnancy. The mechanisms by which both the reduced venous distensibility and the increased aortic distensibility may be observed simultaneously in normal pregnancy remain unexplained and require further investigations.

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