Venous Distensibility During Pregnancy
Comparisons Between Normal Pregnancy and Preeclampsia

Kazuhiko Sakai, Tsutomu Imaizumi, Hirotaka Maeda, Hideaki Nagata, Kiyomi Tsukimori, Akira Takeshita, Hitoo Nakano

Abstract Preeclampsia is associated with increased peripheral resistance. This study was performed to determine whether an increase of venous distensibility occurs as well in preeclampsia. We obtained venous distensibility by measuring the venous pressure-volume relation in the forearm with a water-filled plethysmograph. Twenty-one women with normal pregnancy, 12 women with severe preeclampsia, and 8 women with mild preeclampsia were studied during the third trimester and/or 6 weeks after the delivery. Ten nonpregnant normotensive women were also studied. Venous distensibility was greater (P<.01) in normal pregnant women and smaller (P<.01) in women with preeclampsia during pregnancy than postpartum. The magnitude of the decrease of venous distensibility correlated with the severity of preeclampsia. Venous distensibility was similar between normotensive nonpregnant women, women with normal pregnancy during the postpartum period, and women with preeclampsia during the postpartum period. Thus, venous distensibility increased during normal pregnancy. In preeclampsia, the decrease of venous distensibility occurred during pregnancy but was reversed postpartum. These results may suggest that a decrease of venous distensibility occurs during preeclampsia. These venous abnormalities may contribute to impaired control of hemodynamics in preeclampsia during pregnancy. (Hypertension. 1994;24:461-466.)

Key Words • human • forearm • hypertension, pregnancy-induced • plethysmography

Normal pregnancy is characterized by arterial1,2 and venous3,4 vasodilation and by increased intravascular volume.5-8 In contrast, preeclampsia is associated with increased peripheral resistance5,8 and decreased intravascular volume.9,10 Changes in venous distensibility in women with preeclampsia are less well documented.

The venous system may be important in control of intravascular volume and blood pressure. It has been documented that essential11 and borderline12 hypertension are associated with decreased venous distensibility. Decreases in venous distensibility shift the intravascular volume from the peripheral to the central circulation. Thus, it is possible that impaired venous distensibility may contribute to altered hemodynamic status in preeclampsia.

Several previous investigators examined venous distensibility in women with preeclampsia. Weinstein et al13 and Hunyor14 reported no difference between normal pregnancy and preeclampsia. Stainer et al15 reported decreased venous distensibility during preeclampsia. However, this study was cross-sectional, and they did not examine whether it was reversible postpartum. Pickles et al16 reported increased venous distensibility during preeclampsia. Thus, the reported results are variable. The aim of this study was to further evaluate peripheral venous distensibility in preeclampsia during pregnancy and postpartum and in groups with different severities of preeclampsia.

Methods

Subjects

The subjects were Japanese women cared for in the Maternity and Perinatal Care Unit, Kyushu University Hospital, Fukuoka, Japan. Twenty-one normal pregnant women, 12 women with severe preeclampsia, and 8 women with mild preeclampsia participated in the study. The study protocol was fully explained, and informed consent was obtained from each subject. This study was approved by the Human Research Committee of the Research Institute of Angiocardiology and Cardiovascular Clinic.

Preeclampsia was diagnosed according to definitions by the American College of Obstetrics and Gynecology.17 Preeclampsia was defined as proteinuria >0.2 g/d and a rise in blood pressure from <140/90 mm Hg before 20 weeks of gestation to ≥140/90 mm Hg after 20 weeks of gestation. Preeclampsia was arbitrarily divided into two groups: mild preeclampsia with blood pressure <160/110 mm Hg and severe preeclampsia with systolic blood pressure ≥160 mm Hg and/or diastolic blood pressure ≥110 mm Hg. The clinical profiles of women with normal pregnancy and with preeclampsia are summarized in the Table. The blood pressures shown in the Table are those at the time of measurement of venous distensibility. There were no differences in age and the number of para or gravida. All women with severe preeclampsia had severe hypertension, proteinuria, and edema. Women with mild preeclampsia had less severe hypertension, proteinuria, and edema than women with severe preeclampsia. The edema and proteinuria of women with preeclampsia disappeared after delivery. There was no edema or proteinuria in normal pregnant women throughout the pregnancy. There was no evidence of renal or cardiovascular disease in any subject. No drugs had been administered for at least several days before the measurement of venous distensibility during pregnancy or postpartum in any subjects.

Ten nonpregnant women also participated in the study for comparison. The mean age of the normotensive nonpregnant
### Clinical Profiles of Women With Normal Pregnancy and Women With Preeclampsia During Pregnancy and Postpartum

<table>
<thead>
<tr>
<th>Group and Patient</th>
<th>Age, y</th>
<th>G</th>
<th>P</th>
<th>Weeks</th>
<th>SBP, mm Hg</th>
<th>DBP, mm Hg</th>
<th>UP</th>
<th>E</th>
<th>VP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal pregnant women</strong>&lt;br&gt; 28-33 weeks (n=10)&lt;br&gt; Mean±SEM</td>
<td>30.1±2.6</td>
<td>29.0±1.2</td>
<td>116.8±7.1</td>
<td>73.8±6.6</td>
<td>-</td>
<td>-</td>
<td>5.4±1.4</td>
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<tr>
<td>34-39 weeks (n=10)&lt;br&gt; Mean±SEM</td>
<td>30.1±4.3</td>
<td>36.2±1.3</td>
<td>117.4±8.4</td>
<td>70.0±3.6</td>
<td>-</td>
<td>-</td>
<td>5.9±0.6</td>
<td></td>
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</tr>
<tr>
<td>Postpartum (n=10)&lt;br&gt; Mean±SEM</td>
<td>29.6±3.6</td>
<td>5.3±0.6</td>
<td>119.6±7.3</td>
<td>74.4±5.0</td>
<td>-</td>
<td>-</td>
<td>4.5±0.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mild preeclampsia</strong>&lt;br&gt; During pregnancy (n=8)&lt;br&gt; I.O.</td>
<td>27</td>
<td>1</td>
<td>0</td>
<td>37</td>
<td>146</td>
<td>104</td>
<td>+</td>
<td>-</td>
<td>6</td>
</tr>
<tr>
<td>K.M.</td>
<td>28</td>
<td>1</td>
<td>0</td>
<td>33</td>
<td>136</td>
<td>90</td>
<td>+</td>
<td>-</td>
<td>7</td>
</tr>
<tr>
<td>C.N.</td>
<td>32</td>
<td>1</td>
<td>0</td>
<td>34</td>
<td>144</td>
<td>80</td>
<td>+</td>
<td>-</td>
<td>9</td>
</tr>
<tr>
<td>T.A.</td>
<td>26</td>
<td>2</td>
<td>1</td>
<td>37</td>
<td>150</td>
<td>88</td>
<td>-</td>
<td>+</td>
<td>6</td>
</tr>
<tr>
<td>K.K.</td>
<td>26</td>
<td>1</td>
<td>0</td>
<td>39</td>
<td>148</td>
<td>100</td>
<td>+</td>
<td>+</td>
<td>6</td>
</tr>
<tr>
<td>Y.T.</td>
<td>35</td>
<td>2</td>
<td>1</td>
<td>38</td>
<td>140</td>
<td>86</td>
<td>+</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td>T.M.</td>
<td>35</td>
<td>4</td>
<td>0</td>
<td>37</td>
<td>136</td>
<td>94</td>
<td>+</td>
<td>+</td>
<td>5</td>
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<tr>
<td>C.U.</td>
<td>39</td>
<td>2</td>
<td>1</td>
<td>35</td>
<td>150</td>
<td>100</td>
<td>-</td>
<td>-</td>
<td>6</td>
</tr>
<tr>
<td><strong>Mean±SEM</strong></td>
<td>31.1±1.8</td>
<td>36.6±0.7</td>
<td>144.0±1.9</td>
<td>92.7±2.9</td>
<td>6.3±0.5</td>
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<td><strong>Severe preeclampsia</strong>&lt;br&gt; During pregnancy (n=10)&lt;br&gt; Y.K.</td>
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<td>2</td>
<td>0</td>
<td>28</td>
<td>170</td>
<td>106</td>
<td>++</td>
<td>+</td>
<td>7</td>
</tr>
<tr>
<td>S.K.</td>
<td>29</td>
<td>1</td>
<td>0</td>
<td>30</td>
<td>160</td>
<td>98</td>
<td>++</td>
<td>++</td>
<td>9</td>
</tr>
<tr>
<td>A.U.</td>
<td>39</td>
<td>1</td>
<td>0</td>
<td>32</td>
<td>190</td>
<td>100</td>
<td>++</td>
<td>++</td>
<td>12</td>
</tr>
<tr>
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<td>29</td>
<td>2</td>
<td>1</td>
<td>31</td>
<td>170</td>
<td>110</td>
<td>++</td>
<td>++</td>
<td>6</td>
</tr>
<tr>
<td>J.M.</td>
<td>25</td>
<td>2</td>
<td>1</td>
<td>34</td>
<td>170</td>
<td>92</td>
<td>++</td>
<td>++</td>
<td>10</td>
</tr>
<tr>
<td>Y.S.</td>
<td>28</td>
<td>1</td>
<td>0</td>
<td>35</td>
<td>160</td>
<td>90</td>
<td>++</td>
<td>++</td>
<td>10</td>
</tr>
<tr>
<td>K.T.</td>
<td>29</td>
<td>1</td>
<td>0</td>
<td>36</td>
<td>190</td>
<td>106</td>
<td>++</td>
<td>++</td>
<td>11</td>
</tr>
<tr>
<td>M.N.</td>
<td>32</td>
<td>2</td>
<td>1</td>
<td>37</td>
<td>190</td>
<td>94</td>
<td>++</td>
<td>++</td>
<td>8</td>
</tr>
<tr>
<td>Y.S.</td>
<td>29</td>
<td>1</td>
<td>0</td>
<td>35</td>
<td>160</td>
<td>100</td>
<td>+</td>
<td>+</td>
<td>6</td>
</tr>
<tr>
<td>F.M.</td>
<td>38</td>
<td>2</td>
<td>1</td>
<td>37</td>
<td>170</td>
<td>110</td>
<td>++</td>
<td>++</td>
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<tr>
<td><strong>Mean±SEM</strong></td>
<td>31.7±1.6</td>
<td>33.8±1.0</td>
<td>173.0±4.0</td>
<td>100.6±2.3</td>
<td>8.2±0.8</td>
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<td></td>
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<tr>
<td><strong>Postpartum (n=8)</strong>&lt;br&gt; J.M.</td>
<td>25</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>130</td>
<td>80</td>
<td>-</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Y.K.</td>
<td>39</td>
<td>2</td>
<td>1</td>
<td>6</td>
<td>130</td>
<td>80</td>
<td>-</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Y.S.</td>
<td>28</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>130</td>
<td>78</td>
<td>-</td>
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<td>5</td>
</tr>
<tr>
<td>K.T.</td>
<td>29</td>
<td>1</td>
<td>1</td>
<td>7</td>
<td>118</td>
<td>78</td>
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<tr>
<td>S.K.</td>
<td>29</td>
<td>1</td>
<td>1</td>
<td>6</td>
<td>128</td>
<td>74</td>
<td>-</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>A.U.</td>
<td>39</td>
<td>1</td>
<td>1</td>
<td>7</td>
<td>128</td>
<td>80</td>
<td>-</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>N.Y.</td>
<td>20</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>120</td>
<td>60</td>
<td>-</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>N.F.</td>
<td>28</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>114</td>
<td>70</td>
<td>-</td>
<td>-</td>
<td>7</td>
</tr>
<tr>
<td><strong>Mean±SEM</strong></td>
<td>29.6±2.3</td>
<td>5.5±0.5</td>
<td>124.8±2.3</td>
<td>76.3±3.0</td>
<td>4.0±0.6</td>
<td></td>
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</tr>
</tbody>
</table>

G indicates gravida; P, para; Weeks, gestational or postpartum weeks at sampling; SBP, systolic blood pressure; DBP, diastolic blood pressure; UP, proteinuria (by AlbuStix): ++, ≥0.3 g/dL; +, ≥0.03 g/dL; +, 0.01-0.02 g/dL; -, no proteinuria; E, edema: +, generalized edema; +, pretibial edema; -, no edema; and VP, venous pressure.
Measurement of Venous Distensibility

Venous distensibility was determined by obtaining venous pressure-volume curves as previously described. A single-chamber water-filled plethysmograph was used for recording volume changes in a segment of the left forearm. Subjects wore light clothing and felt comfortably warm at a room temperature of 22°C to 24°C.

Subjects rested in the supine position. The left forearm was enclosed in an acrylic plastic plethysmograph. Warm water (temperature, 32°C to 34°C) was added to 26 cm above the upper aspect of the forearm. A rubber membrane separated the forearm from the water. The external water pressure initially collapsed the veins under these conditions, but the arterial inflow caused the venous pressure to reach a level slightly higher than that of the external water pressure. Previously we have shown that an external pressure of 20 mm Hg was sufficient to collapse the veins under resting conditions. The difference between the pressure within the veins and the external water pressure surrounding them is the distending or transmural pressure. Venous pressure was measured through a polyethylene tube inserted into a superficial vein in the segment of the forearm enclosed in the plethysmograph. Transmural venous pressure was measured by placing the reference level of the pressure transducer at the surface of the water in the plethysmograph. Transmural venous pressure in subjects at rest under these conditions is <1.5 mm Hg. The blood volume in the forearm vessels at this low transmural venous pressure is approximately 1.3 milliliters per deciliter of forearm volume. Under resting conditions, transmural venous pressure and volume are constant and reproducible in a given subject and are similar between subjects.

Changes in the forearm blood volume were recorded during stepwise increases in the transmural venous pressure to 30 mm Hg by inflating a cuff on the upper arm proximal to the plethysmograph. Transmural venous pressure was increased slowly to minimize nonuniform filling of the veins and was held constant at each step until changes in forearm blood volume were stabilized (Fig 1). Changes in the venous volume were reflected by the changes in forearm blood volume, which were measured by recording changes in the height of water with a displacement transducer (DT 4812, San-ei). Increases in volume in response to congestion of the forearm occur primarily in vessels in which resting pressure is <10 mm Hg. Venous pressure-volume curves were constructed by plotting changes of forearm blood volume (milliliters per deciliter) against corresponding levels of transmural venous pressure. Venous distensibility was defined as the changes in the forearm volume when transmural venous pressure was increased from 0 to 30 mm Hg. Measurements of venous distensibility were repeated three times by the same examiner (K.S.), and the intraobserver coefficient of variation was 6.3%.

Venous distensibility was determined during 28 to 33 weeks and 34 to 39 weeks of gestation and 6 weeks after delivery, but subjects did not undergo every measurement of venous distensibility at all three stages. A total of 30 measurements were made in 21 normal pregnant women. These 30 measurements consisted of 10 in 10 women during 28 to 33 weeks of gestation, 10 in another 10 women during 34 to 39 weeks of gestation, and 10 in 10 women during the postpartum period. Measurements were made in 9 of 21 normal pregnant women both during the third trimester and postpartum. Eighteen measurements were made in 12 women with severe preeclampsia both during the third trimester and postpartum. Eight measurements were made in 8 women with mild preeclampsia during the third trimester.

The data are expressed as mean±SEM. Statistical analysis for comparisons between pregnant women was performed by one-way or two-way ANOVA. When they were significantly different by ANOVA, t test was used with Bonferroni's correction to determine the location of the difference. Venous distensibility between nonpregnant women and women with normal pregnancy was compared by unpaired t test. A value of $P<.05$, $**P<.01$. $*P<.05$, $**P<.01$.

Results

The values of venous distensibility of normal pregnant women, women with mild preeclampsia, and women with severe preeclampsia during the third trimester are summarized in Fig 2. The decreases in venous distensibility correlated with the severity of preeclampsia. For comparison, the venous distensibility

\[ \begin{align*}
\text{Forearm Venous Volume} & \quad (\text{mL}) \\
\text{Transmural Venous Pressure} & \quad (\text{mm Hg}) \\
\end{align*} \]

![Fig 1. Representative recording of the changes in venous volume in the forearm during a stepwise increase in transmural venous pressure from 0 to 30 mm Hg.](image-url)
of normotensive nonpregnant women is also shown in Fig 2. The venous distensibility of normal pregnant women was significantly greater than that of normotensive nonpregnant women (P<.01).

The chronological changes in venous distensibility of normal pregnant women and women with severe preeclampsia are summarized in Fig 3. Venous distensibility during normal pregnancy was significantly greater than that of normotensive nonpregnant women (P<.01). The venous distensibility during pregnancy was significantly smaller during pregnancy than postpartum (P<.05). Venous distensibility during pregnancy was significantly smaller in women with severe preeclampsia than in normal pregnant women (P<.01) but was not different between the two groups postpartum. We could obtain serial venous distensibility during the third trimester and postpartum in 9 of 21 normal pregnant women and 6 of 12 women with severe preeclampsia, which is shown in Fig 4. The sequential changes in the same subject support the changes found for group data. The venous distensibility of normal pregnant women postpartum was similar to that of normotensive nonpregnant women.

Venous pressure is shown in the Table. The venous pressure of women with severe preeclampsia was slightly but significantly (P<.05) higher than that of normal pregnant women during pregnancy. Venous pressure was similar between normotensive nonpregnant women (4.5±0.9 mm Hg, n=7), normal pregnant women postpartum, and women with severe preeclampsia postpartum.

**Discussion**

We confirmed that venous distensibility increased during normal pregnancy compared with that postpartum. The major findings of this study are as follows: (1) venous distensibility decreased during preeclampsia compared with that postpartum; (2) venous distensibility of women with preeclampsia was much smaller than that of women with normal pregnancy; and (3) the magnitude of decreases in venous distensibility correlated with the severity of preeclampsia. These results may suggest that transient decrease of venous distensibility occurs during preeclampsia.

**Methodological Considerations**

We used a single-chamber plethysmograph and measured volume changes at 30 mm Hg of transmural venous pressure. Volume increase measured by a single-chamber water plethysmograph is greater than that measured by a double-chamber plethysmograph because of an edge effect occurring at the proximal end of the single-chamber plethysmograph at the same transmural pressure. This is the phenomenon of volume increase preceding pressure increase, which causes an artifact in plotting the pressure-volume curve. A double-chamber water plethysmograph alleviates this effect. However, as Wood and Eckstein described, there was no difference of venous volume changes between a single- and double-chamber water plethysmograph when transmural pressure was increased to 30 mm Hg. Thus, the measurement of venous distensibility with a single-chamber plethysmograph is acceptable when transmural venous pressure is increased to 30 mm Hg.

Venous pressure was measured in one of the superficial veins. The venous pressure in this superficial vein may not have been representative. However, it has been demonstrated that uniform pressure changes in superficial veins may be achieved by inflating a cuff very slowly at <0.25 mm Hg/s. In this study we inflated the cuff and increased transmural venous pressure by 5 to 10 mm Hg in approximately 40 seconds (0.125 to 0.25 mm Hg/s). Thus, we assume that we achieved uniform filling of the veins.
When we measured changes in forearm volume, we did not correct them for net water loss (capillary filtration). Capillary filtration may be significant with the slow rate of increase of transmural venous pressure. We examined capillary filtration rate in our previous study, which was 0.05 mL/min per deciliter of forearm volume in young healthy subjects when transmural venous pressure was kept constant at 30 mm Hg. Because in this study we completed one measurement of venous distensibility in 2 to 3 minutes, the expected maximal water loss would have been smaller than 0.15 mL/dL of forearm volume. This value was small compared with the value of venous distensibility. Although it has been reported that capillary filtration is smaller in women with preeclampsia than in women with normal pregnancy, the difference of capillary filtration is small between nonpregnant women, women with normal pregnancy, and women with preeclampsia compared with the value of venous distensibility. Thus, the decreased venous distensibility in women with preeclampsia was not likely due to the difference in capillary filtration.

Although Litter and Wood reported that blood volume at low transmural pressure (unstressed volume) is small and reproducible in a given subject and similar between subjects, this finding may not hold for preeclampsia, in which venous pressure is elevated. However, we have previously shown that unstressed volume is constant when external water pressure is higher than 10 mm Hg. In this study we applied 20 mm Hg of external water pressure, which would have minimized unstressed volume even in the condition of preeclampsia with slightly elevated venous pressure to approximately 8 mm Hg.

Because studies were performed in the supine position, venous distensibility could have been affected by hemodynamic changes caused by compression of the inferior vena cava. However, it is unlikely that compression of the inferior vena cava caused changes in venous distensibility in opposite directions between normal pregnancy and preeclampsia. Thus, the conclusions regarding increases in venous distensibility in normal pregnancy and decreases in venous distensibility in preeclampsia may have been qualitatively correct. We did not perform the study in the left lateral position because the left arm was compressed and venous drainage was impaired in the left lateral position. Therefore, we could not obtain stable and reproducible pressure-volume curves.

**Venous Distensibility During Normal Pregnancy**

We demonstrated that venous distensibility of the forearm in normal pregnancy increased. Our findings are compatible with those of many previous studies in which increased venous distensibility during normal pregnancy was demonstrated. Although we studied venous distensibility only during the third trimester, previous studies have demonstrated that venous distensibility increases as pregnancy progresses and reaches maximal values at the third trimester, when increases in the plasma volume are maximal. The mechanisms by which venous distensibility increases during normal pregnancy are not well understood. Several mechanisms are considered. It is suggested that the increase of venous distensibility in normal pregnancy reflects adaptation to the changes in circulation, such as an increase of plasma volume. Hormonal changes may be responsible because it has been demonstrated that there was an increase of venous distensibility during oral contraceptive therapy and that the venous distensibility curve parallels the steroid curves of pregnancy.

**Venous Distensibility During Preeclampsia**

Venous distensibility during the third trimester was much smaller in women with preeclampsia than in women with normal pregnancy. Our findings agree with those of some investigators who reported smaller venous distensibility in women with preeclampsia than in women with normal pregnancy. However, Weinstein et al and Hunyor reported no difference in venous distensibility between normotensive pregnant and hypertensive pregnant women. Pickles et al reported larger venous distensibility in women with preeclampsia before they developed preeclampsia. Thus, the reported results are variable.

Venous distensibility during the third trimester in women with preeclampsia was not only smaller than in women with normal pregnancy but also smaller than during the postpartum period. Venous distensibility returned to the normal level postpartum. Our findings suggested that a reversible decrease of venous distensibility occurred during pregnancy in women with preeclampsia. Most previous investigators did not examine the change in venous distensibility postpartum in women with preeclampsia. Hunyor was the only investigator who reported the change in venous distensibility postpartum in women with preeclampsia. In his study venous distensibility during pregnancy was similar between women with normal pregnancy and those with preeclampsia. The change in venous distensibility was reversible postpartum in normal pregnant women but not in women with preeclampsia. Thus, venous distensibility remained larger postpartum in women with preeclampsia than in women with normal pregnancy. Hunyor did not provide an explanation for the greater venous distensibility postpartum in women with preeclampsia than in women with normal pregnancy.

We compared venous distensibility not only between normal pregnancy and preeclampsia but also before and after delivery. We clearly demonstrated that venous distensibility was smaller in women with preeclampsia than in those with normal pregnancy and smaller during preeclampsia than postpartum, indicating the reversibility after delivery (Fig 3). In Fig 4, the sequential and individual data points of venous distensibility of normal pregnant women and women with severe preeclampsia both during the third trimester and postpartum are shown, which support the changes for the group shown in Fig 3. We further demonstrated that the magnitude of the decrease in venous distensibility correlated with the severity of preeclampsia, which has not been described previously.

It is not known from our study why venous distensibility decreased in preeclampsia. Several mechanisms may be considered. Structural changes of the vein are not likely because the decreased venous distensibility was reversible postpartum. It is interesting to note that venous pressure was slightly but significantly higher in women with severe preeclampsia than in women during normal pregnancy. Thus, it is possible that increased...
venous pressure may have affected venous distensibility in women with severe preeclampsia. Although we cannot deny this possibility, some other factors must be present because the venous distensibility of women with mild preeclampsia was still smaller than that of women during normal pregnancy despite comparable venous pressure between the two groups.

Because preeclampsia is associated with increased peripheral resistance and increased vascular responsiveness to angiotensin II in vivo and in vitro, some circulating factors may be present in preeclampsia. Abnormal endothelial function may be implicated in the pathogenesis of preeclampsia. It has been documented in preeclampsia that vascular synthesis of prostacyclin of fetal and maternal vessels is decreased and that production of thromboxane A2 is increased. Tsukimori et al suggested that preeclampsia is characterized by the presence of a serum factor cytotoxic to endothelial cells. Furthermore, Pinto et al reported that endothelial release of endothelin-derived relaxing factors was impaired in preeclampsia. Thus, an imbalance between vasorelaxing and vasoconstricting factors may underlie increased peripheral resistance and decreased venous distensibility in preeclampsia.

It is possible that the decreased venous distensibility was due to tissue edema because women with severe preeclampsia had edema of the hand. We cannot deny this possibility. Although women with mild preeclampsia had no apparent edema of the hand, we still cannot deny the possibility that the decreased venous distensibility was due to subclinical edema since Zelis demonstrated that patients with heart failure and no apparent edema still had decreased venous distensibility after maximal venodilator stimuli.

Preeclampsia is associated with decreased plasma volume and cardiac output compared to those of normal pregnancy. Thus, it is possible that the vein constricts in a compensatory manner to maintain cardiac filling pressure and subsequently to maintain cardiac output.

In conclusion, the results of this study suggest that a reversible decrease of venous distensibility occurs in preeclampsia. Although the mechanisms of the decrease in venous distensibility are unclear, our findings may be pertinent to understanding the pathophysiology of preeclampsia.

References

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Hypertension. 1994;24:461-466
doi: 10.1161/01.HYP.24.4.461
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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