**Blood pressure variations assessed by continuous 24-hour monitoring in menopausal and climacteric women**


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**Abstract.** – Changes in the metabolic hormonal balance during the climacteric and menopause, especially surgically induced menopause, increase the risk of acute cerebrocardiovascular complications. This major risk may be linked to changes in blood pressure. In this study we performed twenty-four ambulatory blood pressure monitoring in climacteric (C), menopausal (PM), and surgically induced menopausal women (SM) to determine mean diurnal and nocturnal systodiastolic levels and percentage peaks, as variations in the pressure profile may be linked to organ damage. Our results showed that the entire series presented mainly diastolic increments (mDBP: HPM = 104.4 ± 5.1; HSM = 106.3 ± 2.9; HC = 100.2 ± 3.1), and that this rise was greater in surgically induced menopausal women. In addition, these subjects presented the highest diastolic and systolic pressure peaks (HSM 37/42 versus HPM 35/36 and HC 29/31) also during the night (nocturnal peak: HSM 15/19 versus HPM 10/12 and HC 5/15). Non dippers seem more exposed to cerebrocardiovascular disease.

Our results revealed that climacteric patients affected by arterial hypertension (mSBP = 162.2 ± 4.1; mDBP = 100.2 ± 3.1; 24 h systolic peak % = 24, diastolic peak % = 24) during the climacteric presented the same levels as observed in conclaimed menopause (mSBP 165.2 ± 5.5; mDBP = 104.2 ± 5.1; 24 h systolic peak % = 28,diastolic peak % = 29). Therefore, 24 h blood pressure monitoring is able to show that pressure changes in hypertensive climacteric and menopausal women and could detect women who are at a greater risk of organ damage.

**Key Words:**
Hypertension, Climacteric, Menopause, Monitoring blood pressure, Cerebrocardiovascular risk.

**Introduction**

The effect of the menopause leads to cardiac ischemia and stroke becoming the main causes of death in women1-7 and this may be related to the complex variations that act on diverse factors (lipid balance, weight, etc.) and on blood pressure. The results of the Framingham Study8-12 indicated that the physiological menopause played a role in increased cardiovascular risk as a result of a reduction in the metabolic hormonal protective balance13. However, this study did not reveal significant variations in blood pressure related to the menopause and these observations were confirmed by other studies14-21. On the contrary, other investigations showed that elevated percentages of menopausal women were affected by arterial hypertension22-25 and it was reported26 that arterial hypertension developed in about 70% of climacteric women and some years after the onset of menopausal. Clinical data revealed that the menopause, especially early menopause, is accompanied by elevated diastolic pressure27, and that surgically induced menopause (ovariectomy) increased the cerebrocardiovascular risk28-33.

Therefore, we conducted continuous 24 hour pressure monitoring of blood pressure in climacteric and menopausal women in order to detect clinical signs that could represent potential risk factors, since organ damage, including cerebrovascular damage, seems to be closely correlated with variations in blood pressure34-42.
Materials and Methods

Subjects

We enrolled 98 climacteric and menopausal women (age range 39-56 years; mean 47.5 years) in the study series. Twelve of these patients were normotensive and made up the control group. The remaining 86 were hypertensive (blood pressure over 160/90 mmHg according to criteria set by the World Heart Association) and were divided into three groups depending on their menopausal status according to the Consensus of Menopause Research: 43 hypertensives were in physiological menopause (HPM); 22 were climacteric (HC); and 21 were in surgical menopause (HSM) defined. Only women with sure diagnosis (case history or general practitioners' reports) of onset of arterial hypertension during the climacteric or menopause were enrolled in the study. Duration of hypertension was less than a year in menopausal women. None of the women presented case history or signs of cerebrovascular, cardiac or renal complications, organ damage, severe concomitant cardiovascular or non cardiovascular diseases, severe arrhythmia, or body mass < 27/kg/m². All the subjects were non smokers. Some of the hypertensive women had not initiated pharmacological treatment, while the women on antihypertensive or oestroprogestinic treatment suspended it 10 days before the study. Patients were enrolled if they presented blood pressure levels over 160/90 mm Hg in agreement with criteria set by the World Heart Association. The study was in line with the Helsinki declaration and all the study series gave their informed consent.

Measurements and data analysis

Continuous 24 h blood pressure monitoring was performed using the oscillometric method AND TM 2421 devices. Diurnal and nocturnal blood pressure were measured every 15 min and every 30 min, respectively. Mean 24 h systodiastolic pressure, mean diurnal and nocturnal systodiastolic pressure, and the percentages of diurnal and nocturnal systodiastolic peaks were measured in all the hypertensive subjects and in the controls. Assessment and definition of pressure peaks was related to the percentage of blood pressure values over the standard systolic and diastolic levels set by the WHO. This study followed the lines of our previous investigations and adopted well-known blood pressure ranges and heart rate.

Statistical analysis was performed using one way variance method (ANOVA) for unpaired study groups. Student's t test for paired data was used to compare mean pressure peak variability and to calculate the approx p levels of statistical significance (p < 0.005, p < 0.001).

Results

Our results are shown in Table I. The three groups of patients presented systodiastolic hypertension and constantly elevated percentages of systodiastolic pressure peaks over the 24 h period (HPM = 28/29%; HSM = 31/37%; HC = 24/24%). Pressure peaks were mainly diastolic during the day in all three hypertensive groups (HPM = 36%; HSM = 42%; HC = 31%). Overlapping mean systolic (mSBP) and diastolic blood pressure increases (mDBP) were observed in HC (mSBP = 162.2 ± 5.5; mDBP 100.2 ± 3.1) and HPM (mSBP = 165.2 ± 4.1; mDBP 104.2 ± 5.1). The highest mean 24 h systodiastolic pressure was observed in HSM (mSBP = 167.1 ± 4.7; mDBP 106.3 ± 2.9). Analysis of pressure peaks distribution (ANOVA) in all the three hypertensive groups revealed statistically significant percentages compared with the controls, and the highest percentages were seen in HSM women (mean 24 h peaks 31/37%: diurnal peaks 37/42%; nocturnal peaks 15/19%). Nocturnal diastolic peaks, 24 h diastolic peaks and diurnal peaks presented statistically intergroup differences exceeding 5% and were as follows:

HSM versus HC  t = 10.53  t = 8.31  t = 6.36
HSM versus HPM t =  7.28  t = 7.15  t = 6.24
HPM versus HC  t = 11.37  t = 7.46  t = 6.53

These results showed that HSM subjects presented higher t values and therefore greater risk than HPM and HC patients. Nocturnal diastolic peaks were more statistically significant in HC than in HPM women.
Discussion

Controlled trials have indicated that blood pressure rises between the late fertile period, the premenopause and claimed menopause22,23,25 in women and that these variations are induced by changes in the oestrogen and biohumoral protective balance. Even if recent studies revealed interesting correlations between progressive variations of the hormonal balance and some blood pressure regulating factors, such as the neurovegetative system, the RAA system, endothelial function, etc2,3,6,25,27,52, numerous mechanisms can determine the presumed increase in blood pressure during the menopause and it has not been clarified how the menopause influences blood pressure. Nevertheless, it is known that these changes in menopausal women increase the risk of cerebrocardiovascular diseases and that this risk, that seems greater in HSM subjects, may be reduced by hormone replacement therapy22,25,31,35.

Twenty four hour pressure monitoring confirmed that surgical menopausal women had a high risk pressure profile with an elevated percentage of diastolic peaks: they also presented nocturnal peaks. A greater incidence of non dippers was observed in hypertensive menopausal women56. The loss of protection caused by nocturnal pressure drops could determine organ damage and this is complicated by the presence of more silent complications (silent or non diagnosed infarction) in women than in men9. Our data revealed that non dippers were mainly HSM subjects and this may partly explain why they are more at risk of cardiovascular diseases.

Changes in blood pressure are involved in organ damage, and studies35,36 reported that damage was greater in patients presenting greater pressure variations than in patients with the same mean 24 h blood levels. A significant correlation between increased cardiovascular complications related to hypertension and the degree of pressure variations at different times during the day and night has

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**Table I.** Pressure changes in normotensive menopausal women with hypertensive climacteric, menopausal and surgically induced menopausal women.

<table>
<thead>
<tr>
<th></th>
<th>Normotensive menopause (NM) n = 12</th>
<th>Hypertensive physiological menopause (HPM) n = 43</th>
<th>Hypertensive surgical menopause (HSM) n = 21</th>
<th>Hypertensive climacteric (HC) n = 22</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>51.0 ± 4.5</td>
<td>53.9 ± 4.4</td>
<td>52.5 ± 4.1</td>
<td>38.0 ± 9.8</td>
</tr>
<tr>
<td>BMI Kg/m²</td>
<td>24.7 ± 0.6</td>
<td>26.4 ± 0.5</td>
<td>25.2 ± 0.2</td>
<td>23.8 ± 0.6</td>
</tr>
<tr>
<td>mSBP</td>
<td>134.1 ± 5.3</td>
<td>165.2 ± 5.5</td>
<td>167.1 ± 4.7</td>
<td>162.2 ± 4.1</td>
</tr>
<tr>
<td>mDBP</td>
<td>89.2 ± 5.3</td>
<td>104.2 ± 5.1</td>
<td>106.3 ± 2.9</td>
<td>100.2 ± 3.1</td>
</tr>
<tr>
<td>HR</td>
<td>68.1 ± 2.8</td>
<td>72.4 ± 2.2</td>
<td>74.1 ± 2.5</td>
<td>78.6 ± 19</td>
</tr>
<tr>
<td>24 h Peak% syst diast</td>
<td>9</td>
<td>28</td>
<td>31</td>
<td>24</td>
</tr>
<tr>
<td>24 h Peak% diast</td>
<td>8</td>
<td>*29‡</td>
<td>*37†</td>
<td>‡24†</td>
</tr>
<tr>
<td>Diurnal syst PEAK % diast</td>
<td>10</td>
<td>35</td>
<td>37</td>
<td>29</td>
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<tr>
<td>Diurnal syst PEAK % diast</td>
<td>11</td>
<td>**36‡‡</td>
<td>**42††</td>
<td>‡‡31††</td>
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<tr>
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<td>1</td>
<td>10</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Nocturnal syst PEAK % diast</td>
<td>5</td>
<td>§12</td>
<td></td>
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</tr>
</tbody>
</table>

Pressure data are expressed as mean ± SD. Peaks are expressed in %.
BMI = Body mass index; mSBP = Systolic blood pressure mean; mDBP = Diastolic blood pressure mean; HR = Heart rate.
Approx p is < 0.05 for each peak variable investigated.
Student’s t test > 5% (* t = 7.15; † t = 8.31; ‡ t = 7.46; § t = 7.28; || t = 10.53; ||| t = 11.37; ** t = 6.24; †† t = 6.36; ‡‡ t = 6.53;)
been observed. Moreover, hypertension induced damage was closely linked to the frequency of hypertensive peaks occurring in the 24 h period, and longitudinal studies adopted these variations as prognostic markers.

Various trials are investigating the clinical indications of non invasive blood pressure monitoring. This method not only reveals non dippers, but also detects an elevated risk of complications in some groups of patients early on. It confirms that HSM women, who have long been considered at higher risk of cerebrocardiovascular complications, present greater pressure variations and elevated nocturnal peaks.

Our study revealed a high percentage of pressure changes in both HC women and HPM subjects where fewer oscillations were observed. Although there are reports of significant increments in pressure during the transition period between premenopause and advanced menopause, this may be due to hormonal fluctuations that do not occur during claimed menopause. However, the scant data on hypertension in pre and postmenopausal do not allow any conclusions to be drawn and further studies on larger and homogeneous study series are required.

Our data indicate that arterial hypertension is present in the climacteric and menopausal women, especially in surgically induced menopause, and that it is characterised by great blood pressure variations and elevated pressure peaks that could cause organ damage. Future prospective studies will reveal if hypertensives are more exposed to organ damage and cardiovascular disease, as reported in the literature. In conclusion, 24 h pressure monitoring can reveal marked changes in blood pressure and high percentage of pressure peaks in climacteric and menopausal patients. This method is easy to perform, low in cost and could be used as an effective screening method to detect menopausal patients where hormonal changes lead to increased risk of cerebrocardiovascular events.

References

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