Haemodynamic Changes in Severe Preeclampsia. A Comparative Study Using Thoracic Electrical Bioimpedance

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Abstract

Objective: to describe and compare hemodynamic changes in normal pregnancy and in women with severe preeclampsia using thoracic electrical bioimpedance.

Methods: A prospective and cross-sectional study in 60 pregnant patients between 28-40 weeks of gestation. We selected 35 healthy pregnant women (group A) and 25 patients with severe preeclampsia (group B) controlled at the Mexico’s General Hospital. In order to measure hemodynamic parameters we used thoracic electrical bioimpedance. We described the variables through the median and range. The statistical significance of the variables between the healthy pregnant and the severe preeclampsia group was assessed with T student test.

Results: In the preeclampsia group we noticed significantly higher difference in the systolic blood pressure (125.8 ± 12.5 vs. 117.5 ± 10.6), diastolic blood pressure (80.1 ± 12.1 vs. 72 ± 9.6), mean arterial pressure (113.4 ± 11.7 vs. 87.9 ± 10.1) and systemic vascular resistance index in preeclamptic patients was also significantly higher than in healthy pregnant women (4883 ± 4571 vs. 2894 ± 1509). There were not any significant differences of cardiac output, heart rate, thoracic fluid content, and left cardiac work between healthy pregnant women and preeclamptic patients.

Conclusions: We obtained interesting data about the haemodynamic changes in severe preeclampsia. The major changes in the maternal circulation of patients with severe preeclampsia were systolic blood pressure, diastolic blood pressure, mean arterial pressure and systemic vascular resistance index.

INTRODUCTION

Bioimpedance Overview

Bioimpedance refers to the electrical properties of a biological tissue, measured when current flows through it. This impedance varies with frequency and different tissue types [1]. The thoracic electrical bioimpedance (TEB) is based on old principles of physics established in Russia in the 1940s and applied to the measurement of cardiac output. Revived first by NASA in the 1960s to measure the cardiac output of astronauts in the space program [2]. The basis for its use was later pioneered by Lababidi in 1970 with significant software refinements and technical improvements over the following decades based on animal and human research. In the 1980s, Sramek et al.
developed a less cumbersome impedance cardiography device with a new SV equation that substituted the cylindrical model of the chest used by Kubicek et al. with that of a truncated cone. In 1986, Bernstein modified the equation of Sramek et al. y introducing into the formulae the actual in addition to ideal weight, thus accounting for deviations from ideal body weight. The purpose was to determine more accurately the volume of the thorax [3-6]. The technique finally became popularized in the 1990s when its use in clinical settings was eval- uated by several multicentre studies reporting improvement in determination of left ventricular ejection time, change in impedance with systole and other markers of systole and diastole providing greater accuracy of noninvasive haemodynamic data [7,8].

The underlying theory behind the bioimpedance cardiography is that thorax is considered as a cylinder perfused with fluid (blood) which has a specific resistivity. The technique is based on the measurements of impedance (or resistance) to transmission of a small electrical current throughout the body (whole-body bioimpedance) or chest area (thoracic bioimpedance). Bioimpedance is therefore the electrical resistance to a high-frequency low-amplitude current (e.g., 1.4e1.8 mA at 30e75 kHz) transmitted from electrodes placed on the upper and lower thorax Conduits of low impedance (lowest resistance, equals high conductance) are blood and plasma (150 and 63 ohm/cm).

Resistance of electrical current is higher (lower conductance) for cardiac muscle, lungs (reflecting air) and fat (750, 1275, and 2500 ohm/cm). When alternating low-level electrical current is applied to the whole body or thoracic area, the primary distribution is to the blood and extracellular fluid. Changes in the body’s resistance to electrical current flow over time (in milliseconds) are associated with dynamic changes in the blood and plasma. As the aortic valve opens and blood is ejected rapidly into the aorta and the arterial branches, impedance to electrical current flow is decreased. During diastole, impedance to electrical flow returns to baseline. Therefore, the changes in impedance that are noted by a thoracic bioimpedance cardiography device reflect an increase in aortic pressure during systole, whereas changes in whole-body impedance reflect a proportional increase in the measurable conductance of the whole body during systole [9,10].

The measurement principle is to apply an alternating current of low intensity (painless for the patient) and high frequency at the base of the thorax from another set of skin electrodes. The electric current flowing preferentially through the liquid media (large intrathoracic blood vessels and heart chambers), continuous recording of the electrical impedance (ie, the resistances applied to an alternating current) and heart chambers), continuous recording of the electrical impedance (ie, the resistances applied to an alternating current) and heart rate. Advances in hardware and software, including digital signal processing and new algorithms, have improved the quality of the results obtained. The accuracy and repeatability of the results have been confirmed in comparative studies with results obtained through invasive methods and echocardiography [10,11].

**Thoracic Electrical Bioimpedance In Pregnancy**

The TEB in obstetrics investigation makes a great contribution to the basic information available about the pregnancy circulatory system, which eventually is helpful in the evaluation of patients with preeclampsia. Pregnancy causes major changes in maternal cardiovascular system, which requires an appropriate adaptation for the normal course of pregnancy. In the absence of these physiological hemodynamic changes, some complications may appear, such as preeclampsia [12].

The evaluation of the haemodynamic state of the preeclamptic patient has always been subject of interest to clinicians, but because of the invasive techniques it has been difficult to obtain haemodynamic data. Thermodilution is considered the gold standard technique to measure the cardiac output. Since an invasive technique, it may be dangerous for both fetus and mother and is not applicable in healthy pregnant women [12]. TEB is a simple technique, which does not require a highly specialized operator to perform and makes continuous registration possible. This technique has shown a very good correlation with invasive techniques (r=0.91, p<0.001) such as thermodilution, which is considered the gold standard for the measurement of cardiac output and systolic volume [11,12].

The aim of this study was to describe and compare hemodynamic changes in normal pregnancy and in woman with preeclampsia using TEB.

**MATERIAL AND METHODS**

We realized a prospective and cross-sectional study in 60 pregnant patients between 28-40 weeks of gestation. We selected 35 healthy pregnant women (group A) and 25 patients with severe preeclampsia (group B) controlled at our Obstetrics Unit and in the Obstetric Intensive Care Unit. The preeclampsia group was established in the clinic phase, corresponding to the last period of the disease.

Severe preeclampsia was diagnosed by the presence of one or more of the following: [13]

- Hypertension: systolic blood pressure > 160 or diastolic blood pressure >110 on two occasions at least 4 hours apart while the patient is on bed rest (unless antihypertensive therapy is initiated before this time).
- Thrombocytopenia (platelet count <100,000).
- Impaired liver function (elevated blood levels of liver transaminases to twice the normal concentration), severe persistent epigastric pain unresponsive to medication and not accounted for by alternative diagnoses, or both.
- New development of renal insufficiency (elevated serum creatinine greater than 1.1 mg/dl, or doubling of serum creatinine in the absence of other renal disease).
- Pulmonary edema.
The Cardio Dynamics Bioz TEB monitor calculates cardiac output, cardiac Index, stroke index, stroke volume, systemic vascular resistance, systemic vascular resistance index, heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and thoracic fluid content, velocity index, acceleration index, left cardiac work, left cardiac work index, systolic time ratio, pre ejection period and left ventricular ejection time.

Statistical analysis

Statistical analysis was performed with SPSS packages (version 18.0 for Windows; SPSS, Chicago, IL, USA). We described the variables through the median and standard deviation. The statistical significance of the variables between the healthy pregnant and the severe preeclampsia group was assessed with Student’s t test. Significance was considered to be achieved when \( p < 0.05 \).

RESULTS

We enrolled 60 patients in the study; divided in two groups, group A with 35 healthy pregnant women and group B with 25 preeclamptic patients. The general characteristics of the studied groups are described in (Table 1). Haemodynamic characteristics of the patients of both groups are summarized in (Table 2).

We noticed a significant difference in the systolic blood pressure, diastolic blood pressure, mean arterial pressure and in the systemic vascular resistance index between both groups. We found no differences in cardiac output, cardiac Index, stroke index, stroke volume, systemic vascular resistance, thoracic fluid content, velocity index, acceleration index, left cardiac work, left cardiac work index, systolic time ratio, pre ejection period and left ventricular ejection time.

DISCUSSION

In this study we elucidate that the main haemodynamic differences between healthy and preeclamptic patients are

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**Table 1: General characteristics.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A (n=34)</th>
<th>Group B (n=25)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>26.1 ± 6.4</td>
<td>27.1 ± 7.27</td>
<td>NS</td>
</tr>
<tr>
<td>Body mass index</td>
<td>28.5 ± 2.9</td>
<td>27.6 ± 3.9</td>
<td>NS</td>
</tr>
<tr>
<td>Weeks at birth</td>
<td>38.2 ± 4.25</td>
<td>34.3 ± 7.4</td>
<td>0.05</td>
</tr>
<tr>
<td>Fetal birth weight</td>
<td>2868.43 ± 501.7</td>
<td>1812 ± 974.9</td>
<td>0.039</td>
</tr>
<tr>
<td>Apgar at 1 minute</td>
<td>7.92 ± 0.43</td>
<td>6.02 ± 1.42</td>
<td>0.04</td>
</tr>
<tr>
<td>Apgar at 5 minutes</td>
<td>8.91 ± 0.25</td>
<td>0755 ± 1.68</td>
<td>0.05</td>
</tr>
</tbody>
</table>

* Mann Whitney U

**Table 2: Haemodynamic characteristics of both groups of study.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A (n=34)</th>
<th>Group B (n=25)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>80.3 ± 15.1</td>
<td>77.1 ± 12.6</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>117.5 ± 10.6</td>
<td>125.8 ± 12.5</td>
<td>0.02</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>72 ± 9.6</td>
<td>80.1 ± 12.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>87.9 ± 10.1</td>
<td>113.4 ± 11.7</td>
<td>0.001</td>
</tr>
<tr>
<td>Cardiac Index (min/m²)</td>
<td>5.5 ± 3.1</td>
<td>2 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>3.8 ± 0.9</td>
<td>3.4 ± 1.4</td>
<td>NS</td>
</tr>
<tr>
<td>Stroke index (ml/m²)</td>
<td>32.1 ± 9.7</td>
<td>26.8 ± 11.4</td>
<td>NS</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>119.1 ± 40.8</td>
<td>45.3 ± 19.5</td>
<td>NS</td>
</tr>
<tr>
<td>Systemic vascular resistance (dyn sec cm⁻²)</td>
<td>1865.5 ± 903.4</td>
<td>2911.3 ± 2690.3</td>
<td>NS</td>
</tr>
<tr>
<td>Systemic vascular resistance index (dyn sec/m²)</td>
<td>2893.7 ± 1509</td>
<td>4883.4 ± 4570.5</td>
<td>0.05</td>
</tr>
<tr>
<td>Acceleration index (sec)</td>
<td>96.1 ± 35.1</td>
<td>67.5 ± 36.9</td>
<td>NS</td>
</tr>
<tr>
<td>Thoracic fluid content (kohm)</td>
<td>42.7 ± 13.5</td>
<td>50.1 ± 13.1</td>
<td>NS</td>
</tr>
<tr>
<td>Left cardiac work (kg/m²)</td>
<td>4.3 ± 1.2</td>
<td>3.8 ± 1.6</td>
<td>NS</td>
</tr>
<tr>
<td>Left cardiac work index (kg/m²)</td>
<td>2.7 ± 0.7</td>
<td>2.1 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic time ratio</td>
<td>0.4 ± 0.09</td>
<td>0.3 ± 0.09</td>
<td>NS</td>
</tr>
<tr>
<td>Pre ejection period (msec)</td>
<td>106.6 ± 15.6</td>
<td>103.1 ± 22.2</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular ejection time (msec)</td>
<td>271.7 ± 30.6</td>
<td>1.8 ± 27.1</td>
<td>NS</td>
</tr>
</tbody>
</table>

* Student’s t test.
related with the blood pressure (systolic, diastolic and mean), we also notice a significant increase in the vascular resistance index in the preeclampsia group, which is the resistance imposed by vasculature on each beat of the heart, and according to our results we identified that severe preeclampsia appears to be a more intensely vasoconstricted state than normal pregnancy.

A relevant point is that we did not notice significantly changes in the cardiac output, but previous studies demonstrated that in gestations complicated by preeclampsia at the time of its diagnosis (third trimester), the cardiac output was significantly lower than that found in uncomplicated gestations in the third trimester [14]. In the literature exist different results, Easterling and Benedetti [15] proposed a hyperdynamic model of preeclampsia. In their study of 179 pregnant women who were monitored during pregnancy, 9 developed preeclampsia and these had a higher cardiac output with a compensatory vasodilation, prior to the onset of clinical phase. Benedetti et al. in 1980 and Groenendijk et al. in 1984 describe a high cardiac output in their results. However, this occurred after the plasma volume was increased with colloids and after a vasodilator such as hydralazine was administered to reduce the peripheral resistances, which were high prior to the treatment [16,17].

The main study limitation is that subject numbers may be too small to gain the significant differences for cardiac output between normal pregnant women and preeclamptic women.

CONCLUSIONS

This study aimed to show the main hemodynamic changes in preeclamptic patients in comparison with healthy pregnancy with a non-invasive technique. Although we collected a reduced number of patients in the study (60), we obtained interesting data about the haemodynamic changes in severe preeclampsia. We noticed that the major changes in the maternal circulation of patients with severe preeclampsia were systolic blood pressure, diastolic blood pressure, mean arterial pressure and systemic vascular resistance index. We did not notice any change in the cardiac output, heart rate, thoracic fluid content or left cardiac work.

Larger studies should be performed to obtain more accurate values and to evaluate the role of TEB in the pregnancy complicated with severe preeclampsia, eclampsia or HELLP syndrome.

An interesting point is that despite abundant literature on TEB for almost 60 years, it remains rarely used in the evaluation of the haemodynamic state of the preeclamptic patient in the ICU.

REFERENCES


Cite this article