ORIGINAL REPORT

Effects of two methods of heat therapy on the acute vascular response and hemodynamics in healthy subjects

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KEYWORDS
Heat;
Ultrasound;
Vascular function;
Hemodynamics

Abstract
Objective: Recently, non-pharmacological resources to relieve pain like hot packs and ultrasound (US) have become common in clinical practice. However, little experimental evidence is available about the possible mechanisms through which these methods bring about pain relief. We aimed to determine the effects of hot packs and US on the acute vascular response and on hemodynamic parameters in healthy subjects.

Materials and methods: We conducted an experimental study in 20 healthy subjects (10 men and 10 women; mean age, 22.54 ± 1.70 years). The two interventions were randomly applied: (a) hot packs (n = 10): 15 min at 60°C and (b) US (n = 10): 15 min at 1 MHz. Before and after each intervention, the following vascular parameters were measured in the brachial artery using Doppler ultrasonography with a 7 MHz probe: arterial compliance, elastic modulus, beta stiffness index, systolic and diastolic arterial diameters, systolic flow velocity and diastolic flow velocity, systolic/diastolic ratio, resistance index, and pulsatility index. The following hemodynamic parameters were monitored: heart rate and blood pressure (systolic, diastolic, and mean).

Results: After the application of hot packs, we observed changes in diastolic flow velocity and in the pulsatility index (p < 05). After the application of US, we observed changes in diastolic flow velocity, systolic/diastolic ratio, resistance index, and arterial compliance (p < 05). No changes in hemodynamic parameters were observed after either intervention.

Conclusions: Applying hot packs or US modifies the physiology of the vascular system but does not affect hemodynamic parameters in healthy subjects.

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Efectos de dos métodos de termoterapia sobre la respuesta vascular aguda y parámetros hemodinámicos en un grupo de sujetos sanos

Resumen
Objetivo: Recientemente determinados recursos no farmacológicos, como el paquete caliente (PC) y los ultrasonidos (US), son utilizados en el alivio del dolor con alto grado de aceptación en la práctica clínica. Sin embargo, la evidencia experimental que apoye los posibles mecanismos por los que se producen estos beneficios es escasa. El objetivo de este estudio fue comprobar los efectos de dos métodos de termoterapia (PC y US) sobre la respuesta vascular aguda y parámetros hemodinámicos en sujetos sanos.

Materiales y métodos: Estudio experimental en 20 sujetos sanos (10 hombres y 10 mujeres; edad media, 22,54 ± 1,70 años). Se aplicaron dos intervenciones de forma aleatoria: a) PC (n = 10): 15 min a 60 °C, y b) US (n = 10): 15 min a 1 Mhz, y se realizaron mediciones vasculares mediante ecografía doppler con transductor de 7 Mhz, antes y después de cada intervención.

Parámetros vasculares: distensibilidad arterial (Da), módulo elástico (Ep), índice beta de rigidez (β), diámetro arterial sistólico y diástólico, velocidad del flujo sistólico (Vfs) y diastólico (Vfd), relación sístole/diástole (RSD), índice de resistencia (IR) e índice de pulsatilidad (IP) en la arteria braquial, y monitorización hemodinámica de la frecuencia cardíaca y de las presiones arteriales sistólica, diastólica y media.

Resultados: Se encontraron diferencias en la Vfd y en el IP (p < 0,05), en la intervención con PC, mientras que la intervención con US mostró cambios en la Vfd, la RSD, el IR y la Da (p < 0,05). No se encontraron cambios en los parámetros hemodinámicos.

Conclusiones: La fisiología del sistema vascular se modifica por la aplicación del PC y los US, sin afectar los parámetros hemodinámicos en sujetos sanos.

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Introduction
Non-pharmacological treatments (physical agents or modalities) have been recently used to relieve pain with a high level of acceptance in clinical practice.1 These resources, based on sophisticated equipment, promote physiological effects such as local and superficial increase in temperature, increase in blood flow, reduction in joint stiffness, and tissue regeneration among others, because in many traumatic and non-traumatic injuries, inflammatory processes appear as the first and immediate response to the injury, allowing extravasation of proteins out of the vessels, causing vascular changes that result in a local increase in blood flow.1-5

Several non-pharmacological agents such as local heat therapy, ultrasound (US) and hot pack [HP; silica gel wrapped in a plastic bag that is warmed up in hot water and is applied locally] have been proposed to accelerate the healing of tissue damage by increasing the supply of blood, oxygen and nutrients into the lesion site. Additionally, this mechanism helps remove from the injured area the products of cell metabolism that increase nociception.6 Because US and HP are inexpensive and easy to apply, they could become an important alternative for providing analgesia, as they apparently show no adverse or side effects and can be controlled and tolerated by the patient.7,8

Although physical modalities, especially HP and US, have been long and widely used for the treatment and management of different conditions, there is a lack of medical literature supporting their application. This could be explained in part by the fact that controlled clinical and experimental trials have focused only on the effects on analgesia, inflammation, skin microcirculation and superficial temperature,6-8 but have not examined in depth the physiological effects that could explain some of the benefits observed, such as the vascular and hemodynamic response of deep tissues induced by these methods.

A novel approach in rehabilitation would be to experimentally investigate the physiological effects induced by this type of therapeutic agents, given their widespread acceptance in clinical practice, even though little data exist on their effectiveness or physiological effects. In this study we hypothesize that “the application of US or HP increases the vascular perfusion of large vessels and this effect is accompanied by hemodynamic changes”. The purpose of this study is therefore to determine the effects of two methods of thermotherapy (HT and US) on the acute vascular response and on hemodynamic parameters in healthy subjects using Doppler ultrasonography.

Patients and methods
Participants
An experimental study was conducted between November and December 2010 in 20 healthy subjects (10 men and 10 women; mean age, 22.54 ± 1.70 years; range, 18–23 years) from a higher education institution in Cali (Colombia). Following a call for participation, purposeful sampling was used to select the participants. Individuals with cardiovascular syndromes, overweight or obesity, or other types of endocrine disorder (type 1 or 2 diabetes mellitus, metabolic syndrome, etc.) that might affect the acute vascular response were excluded. Written informed consent was obtained in all study subjects, and the study was approved by the ethics committee of the academic center.
The study was performed in accordance with the deontological principles laid down in the Declaration of Helsinki and the regulations in force in Colombia regarding human research (Resolution 008430 of 1993 from the Ministry of Health).

**Clinical and anthropometric evaluation**

The following data were obtained from all patients: (a) familial history of cardiovascular risk, (b) past medical history, (c) basic anthropometric evaluation (weight and height) by means of standardized technique, and (d) blood pressure measurements, always obtained by the same examiner using the same device (Dinamap® oscillometric method). The blood pressure was measured in the right arm, with the patient lying down comfortably; the cuff at heart level and adjusted to the arm circumference. Three measurements were obtained from each patient and the average value was calculated. Mean arterial pressure was calculated using the equation: diastolic pressure + [(systolic pressure – diastolic pressure)/3].

**Physiological measurements using Doppler ultrasonography**

A SIEMENS SG-60® ultrasound system with EchoTracking® software was used. Measurements were performed in the brachial artery, 5 cm above the elbow joint, with the patient positioned supine and at a room temperature of 22–25 °C, following the procedure described by Celermajer et al.7 and Correti and Anderson.10 The brachial artery was selected for being a relevant body segment that is close to the glenohumeral joint, and for being a site with a high incidence of lesions.13 At least 5 min of rest in supine were needed before starting the examination (Fig. 1). The 7 MHZ linear transducer was positioned in a way that the artery was imaged in the longitudinal plane until maximization of the echoes from the media-adventitia interface. When this dividing interface was clearly visualized, the two markers of the system were placed at diametrically opposed points in the interface. Once in place, the markers were moved simultaneously with the arterial wall providing a recording of the distance between the two points as a function of time. ECG gating was used during image acquisition so the system could detect the beginning of the systolic and diastolic flow velocity (Sfv and Dfv, respectively). By using this technique the following information was obtained: (a) graphic recording of pulse wave, (b) maximum (systolic) arterial diameter (Sd), which corresponds to the highest point of each pulse, with peak pressure in the artery (systolic blood pressure [SBP]), and (c) minimum (diastolic) arterial diameter (Dd), which corresponds to the lowest point, with lowest pressure in the artery (diastolic blood pressure [DBP]).12 All the measurements were performed by the same reader with more than 3 years of experience in the technique (Fig. 1), 3 min before (baseline measurement) and immediately after each intervention. The software applied a number of equations on the data collected to calculate the three parameters related to arterial elasticity:

- Arterial compliance (Ac) \( (\text{mm}^2/\text{kPa}) = \pi \left( \frac{Sd^2 - Dd^2}{4} \right) \)
- Elastic modulus (Em) \( (\text{kPa}) = \left( \frac{SBP - DBP}{Dd - (Sd - Dd)} \right) \)
- Stiffness index \( \beta = \ln(\frac{SBP - DBP}{(Sd - Dd)/Dd}) \)

Additionally, the indices most commonly used for quantitative analysis of the flow velocity waveforms were estimated.

- Pulsatility index (PI) = \( \frac{(S - D)}{C} \)
- Systolic/diastolic (S/D) ratio = \( \frac{S}{D} \)
- Resistance index (RI) = \( \frac{(S - D)}{S} \)

where S is the maximum velocity peak at end-systole, D is the maximum velocity peak at end-diastole, and C is the average of mean velocity over one cardiac cycle.
Hemodynamic measurements

The electrocardiogram, heart rate and blood pressure were monitored 2 min before the application of the protocol, during and immediately after each intervention.

Interventions

The two interventions were randomly applied to the 20 participants: 10 subjects were applied HP (n = 5 women and n = 5 men), and 10 subjects were applied US (n = 5 women and n = 5 men). The patients were asked to refrain from drinking alcohol and from exercising for 24 h before the study. The patients were then placed in an air-conditioned room at 22 °C with controlled humidity and deprived of visual and auditory stimuli. After acclimatization and a 5 min pre-treatment period, in supine position, HP or US were applied to the arm using the physical parameters described in Table 1.

Baseline measurements were performed during 5 min, each method was applied for 15 min, and post-treatment measurements were performed during 3 min, yielding a total of 23 min.

Statistical analysis

The statistical analysis was performed using SPSS software, version 15. The results were expressed as mean ± standard deviation, and the Kolmogorov–Smirnov test was used to evaluate data distribution. A non-parametric factorial ANOVA was used to evaluate the changes between baseline and post-treatment measurements for each protocol. For all the studies, a p value < 0.05 was considered statistically significant.

Results

No differences were found in the clinical and anthropometric parameters evaluated (Table 2). Regarding the measurements of physiological parameters, statistically significant differences were found in those patients treated with HP (n = 10; 5 women and 5 men) in the diastolic flow velocity (4.01 ± 0.85 vs 3.27 ± 1.05, p = 0.020; decrease of 22.6%) and the pulsatility index (1.88 ± 0.41 vs 2.36 ± 0.83, p = 0.029; increase of 20.3%) between baseline and post-intervention measurements, respectively (Table 3).

On the other hand, significant differences were found in those patients treated with US (n = 10; 5 women and 5 men) in the diastolic flow (8.19 ± 9.67 vs 3.30 ± 1.32, p = 0.031; decrease of 57.8%), systolic/diastolic ratio (7.73 ± 4.41 vs 12.07 ± 4.83, p = 0.005; increase of 36.0%), and resistance index (0.79 ± 0.16 vs 0.90 ± 0.04, p = 0.009; increase of 12.2%) and arterial compliance (0.07 ± 0.06 vs 0.10 ± 0.04, p = 0.034; increase of 42.9%), etc between baseline and post-intervention measurements, respectively (Table 4).

Discussion

The results of the current study indicate that the administration of HP or US modifies the physiology of the vascular system but does not affect hemodynamic parameters. The vasodilation secondary to the use of thermal agents is acute and appears in response to muscle and vascular hyperemia. This and other changes can be measured using high

Table 1 Application parameters of the physical modalities.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Duration</th>
<th>MHz/temperature (°)</th>
<th>Objective</th>
</tr>
</thead>
<tbody>
<tr>
<td>HP</td>
<td>15 min</td>
<td>60 °C</td>
<td>Ability to penetrate superficial and deep tissue layers and cause vascular changes</td>
</tr>
<tr>
<td>US</td>
<td>15 min</td>
<td>1 MHz</td>
<td>Ability to penetrate deep tissue layers and cause vascular changes</td>
</tr>
</tbody>
</table>

Table 2 Clinical and anthropometric data of the participants (n = 20).

<table>
<thead>
<tr>
<th>Variables</th>
<th>US group (mean ± SD) (n = 10)</th>
<th>HP group (mean ± SD) (n = 10)</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex distribution of each group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Men n = 5</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>22.49 ± 1.82</td>
<td>22.88 ± 1.71</td>
<td>0.10</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>61.70 ± 9.08</td>
<td>62.21 ± 10.43</td>
<td>0.89</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.60 ± 9.08</td>
<td>169.40 ± 9.15</td>
<td>0.45</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.22 ± 1.71</td>
<td>20.83 ± 1.86</td>
<td>0.99</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>125.60 ± 15.74</td>
<td>121.55 ± 12.94</td>
<td>0.34</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>71.70 ± 7.10</td>
<td>67.40 ± 9.61</td>
<td>0.39</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>144.21 ± 13.12</td>
<td>138.01 ± 12.84</td>
<td>0.31</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>72.50 ± 10.50</td>
<td>69.30 ± 9.19</td>
<td>0.17</td>
</tr>
</tbody>
</table>

HR, heart rate; BMI, body mass index; DBP, diastolic blood pressure; MAP, mean arterial pressure; SBP, systolic blood pressure; HP, hot packs; US, ultrasound.
resolution plethysmography, a simple technique used to measure vascular function and structure, with the advantage of being minimally invasive, easy to apply and offering reproducible results. Several authors have reported that the changes in the superficial arterial flow correlate with higher values of endothelium-dependent vasodilation in the extremities treated with physical agents such as US.\textsuperscript{13,14} Nonetheless, as far as we know, plethysmography has not been used to assess the effect of HP and US on the vascular physiology and morphology.\textsuperscript{15,16}

After a period of HP or US-induced heat shock, considerable arterial dilation occurs, with the consequent changes in the arterial flow. This phenomenon is known as reactive hyperemia, which is dependent on endothelium-mediated vasodilation and independent of vascular innervation.\textsuperscript{17} Hyperemia is in part due to myogenic relaxation of the vasculature and the release of mediators and metabolites from the treated tissue,\textsuperscript{18} such as prostaglandins\textsuperscript{19} and nitric oxide\textsuperscript{20}, which seem to operate in combination.\textsuperscript{21-24} This study showed statistically significant differences in the physiology of the vascular system between baseline measurements and measurements obtained after the application of HP and US. These changes can be explained in part by the endothelial response to forces related to

### Table 3  Vascular physiology data from the hot pack group (n=10).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>Post intervention</th>
<th>% change</th>
<th>F</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>125.60 ± 15.32</td>
<td>120.80 ± 14.85</td>
<td>4.0</td>
<td>1.011</td>
<td>0.321</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>71.70 ± 6.91</td>
<td>69.60 ± 5.77</td>
<td>3.0</td>
<td>1.087</td>
<td>0.304</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>142.20 ± 18.29</td>
<td>136.10 ± 18.18</td>
<td>4.5</td>
<td>1.119</td>
<td>0.297</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>72.50 ± 9.80</td>
<td>71.60 ± 8.43</td>
<td>1.3</td>
<td>0.997</td>
<td>0.757</td>
</tr>
<tr>
<td>Sd (mm)</td>
<td>3.15 ± 0.48</td>
<td>3.34 ± 0.50</td>
<td>5.7</td>
<td>1.475</td>
<td>0.232</td>
</tr>
<tr>
<td>Dd (mm)</td>
<td>3.04 ± 0.47</td>
<td>3.24 ± 0.50</td>
<td>6.2</td>
<td>1.678</td>
<td>0.203</td>
</tr>
<tr>
<td>SFV (cm/s(^2))</td>
<td>34.67 ± 9.53</td>
<td>32.39 ± 14.81</td>
<td>7.0</td>
<td>0.335</td>
<td>0.566</td>
</tr>
<tr>
<td>DfV (cm/s(^2))</td>
<td>4.01 ± 0.85</td>
<td>3.27 ± 1.05</td>
<td>22.6</td>
<td>5.949</td>
<td>0.020</td>
</tr>
<tr>
<td>S/D</td>
<td>8.62 ± 2.35</td>
<td>10.02 ± 3.60</td>
<td>14.0</td>
<td>2.118</td>
<td>0.154</td>
</tr>
<tr>
<td>RI</td>
<td>0.87 ± 0.03</td>
<td>0.88 ± 0.05</td>
<td>1.1</td>
<td>0.113</td>
<td>0.739</td>
</tr>
<tr>
<td>PI</td>
<td>1.88 ± 0.41</td>
<td>2.36 ± 0.83</td>
<td>20.3</td>
<td>5.152</td>
<td>0.029</td>
</tr>
<tr>
<td>Stiffness index ((\beta))</td>
<td>55.95 ± 14.27</td>
<td>62.09 ± 15.76</td>
<td>9.9</td>
<td>1.663</td>
<td>0.205</td>
</tr>
<tr>
<td>Ac (mm(^2)/kPa)</td>
<td>0.07 ± 0.01</td>
<td>0.07 ± 0.01</td>
<td>0.0</td>
<td>0.052</td>
<td>0.821</td>
</tr>
<tr>
<td>Em (kPa)</td>
<td>206.41 ± 68.08</td>
<td>225.87 ± 80.00</td>
<td>8.6</td>
<td>0.686</td>
<td>0.413</td>
</tr>
</tbody>
</table>

\(\text{Ac}\), Arterial compliance; \(\text{Dd}\), minimum or diastolic arterial diameter; \(\text{Sd}\), maximum or systolic arterial diameter; \(\text{Em}\), elastic modulus; \(\text{F}\), Snedecor’s \(F\); \(\text{HR}\), heart rate; \(\text{PI}\), pulsatility index; \(\text{RI}\), resistance index; \(\text{kPa}\), kilo Pascal; \(\text{bpm}\), beats per minute; \(\text{DBP}\), diastolic blood pressure; \(\text{MAP}\), mean arterial pressure; \(\text{SBP}\), systolic blood pressure; \(\text{S/D}\), systolic/diastolic ratio; \(\text{DfV}\), diastolic flow velocity; \(\text{SFV}\), systolic flow velocity. 

The results are presented as mean±SD.

### Table 4  Vascular physiology data from the ultrasound group (n=10).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>Post intervention</th>
<th>% change</th>
<th>F</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>120.50 ± 12.60</td>
<td>116.80 ± 10.88</td>
<td>3.2</td>
<td>0.987</td>
<td>0.327</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>67.40 ± 9.36</td>
<td>67.30 ± 8.14</td>
<td>0.1</td>
<td>0.001</td>
<td>0.971</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>138.20 ± 14.78</td>
<td>133.29 ± 12.22</td>
<td>3.7</td>
<td>1.306</td>
<td>0.260</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>69.30 ± 8.94</td>
<td>67.80 ± 7.93</td>
<td>2.2</td>
<td>0.315</td>
<td>0.578</td>
</tr>
<tr>
<td>Sd (mm)</td>
<td>3.34 ± 0.39</td>
<td>3.36 ± 0.46</td>
<td>0.6</td>
<td>0.021</td>
<td>0.884</td>
</tr>
<tr>
<td>Dd (mm)</td>
<td>3.20 ± 0.37</td>
<td>3.27 ± 0.43</td>
<td>2.1</td>
<td>0.298</td>
<td>0.588</td>
</tr>
<tr>
<td>SFV (cm/s(^2))</td>
<td>34.59 ± 11.79</td>
<td>34.73 ± 7.61</td>
<td>0.4</td>
<td>0.002</td>
<td>0.965</td>
</tr>
<tr>
<td>DfV (cm/s(^2))</td>
<td>8.19 ± 1.67</td>
<td>3.30 ± 1.32</td>
<td>148.2</td>
<td>5.013</td>
<td>0.031</td>
</tr>
<tr>
<td>S/D</td>
<td>7.73 ± 4.41</td>
<td>12.07 ± 4.83</td>
<td>36.0</td>
<td>8.788</td>
<td>0.005</td>
</tr>
<tr>
<td>RI</td>
<td>0.79 ± 0.16</td>
<td>0.90 ± 0.04</td>
<td>12.2</td>
<td>7.619</td>
<td>0.009</td>
</tr>
<tr>
<td>PI</td>
<td>2.24 ± 1.15</td>
<td>2.50 ± 0.53</td>
<td>6.7</td>
<td>0.319</td>
<td>0.575</td>
</tr>
<tr>
<td>Stiffness index ((\beta))</td>
<td>49.60 ± 16.92</td>
<td>48.65 ± 35.16</td>
<td>2.0</td>
<td>0.012</td>
<td>0.914</td>
</tr>
<tr>
<td>Ac (mm(^2)/kPa)</td>
<td>0.07 ± 0.06</td>
<td>0.10 ± 0.04</td>
<td>42.9</td>
<td>3.144</td>
<td>0.034</td>
</tr>
<tr>
<td>Em (kPa)</td>
<td>181.74 ± 70.12</td>
<td>174.69 ± 121.19</td>
<td>4.0</td>
<td>0.051</td>
<td>0.823</td>
</tr>
</tbody>
</table>

\(\text{Ac}\), Arterial compliance; \(\text{Dd}\), minimum or diastolic arterial diameter; \(\text{Sd}\), maximum or systolic arterial diameter; \(\text{Em}\), elastic modulus; \(\text{F}\), Snedecor’s \(F\); \(\text{HR}\), heart rate; \(\text{PI}\), pulsatility index; \(\text{RI}\), resistance index; \(\text{kPa}\), kilo Pascal; \(\text{bpm}\), beats per minute; \(\text{DBP}\), diastolic blood pressure; \(\text{MAP}\), mean arterial pressure; \(\text{SBP}\), systolic blood pressure; \(\text{S/D}\), systolic/diastolic ratio; \(\text{DfV}\), diastolic flow velocity; \(\text{SFV}\), systolic flow velocity. 

The results are presented as mean±SD.
pulsating blood flow and shear forces, which are physio-
logical stimuli partially responsible for the release of
vasoactive agents like nitric oxide. It is therefore sug-
gested that both interventions generate similar forces in the
vasculature regarding the plethysmographic parameters, but
do not modify hemodynamic parameters. Another inter-
esting finding was the increase in arterial compliance (increase
of 42.9\%) between baseline and post-intervention measure-
ments. Arterial compliance can be defined as the ability
of the vessel wall to adapt to the changes in volume and
pressure transmitted from the left ventricle.25–27 We can
only speculate on the mechanism behind the increase in
arterial compliance caused by US. Compliance is mainly
determined by the elastic and intrinsic properties of the vas-
culature. The wall elements that determine compliance are
elastin and collagen composition (structural determinants)
and the vasoconstrictive tone exerted by its smooth muscle
cells (functional determinant).28 Since biochemical changes
in elastin and collagen composition occur over the years,
itis unlikely that US could modify arterial compliance in
the short term by means of this mechanism. However, the
increase in pulse pressure and mechanical distension occur-
ing during the application of US (collagen stretching) are
likely to modify the intertwining of collagen, therefore mod-
ifying arterial compliance.29–32 In this way, the findings of the
current study indicate that the vascular system is modified
by these thermal agents, and the duration or intensity of the
physiological processes that determine the morphology and
physiology of the vasculature are modified by endothelial
reactivity, tone and peripheral vascular resistance.

Similarly, our results must be interpreted with caution
due to the limitations of the study. First one is the small
sample size and the limited age range. Second, the eating
patterns, which can modulate the vascular and metabolic
response, were not taken into account. In addition, no
evaluation of the inability to control the plethysmographic
parameters during the application of the thermal agents
was carried out and, therefore, any potential changes that
might have taken place during the intervention were not recorded.
Nonetheless, we highlight that since we hypothesize about
the ability of HP and US to generate changes in the mor-
phology and physiology of the vasculature, this limitation does
not seem relevant.

In conclusion, the results of this study show one of the
first experimental evidences that have used plethys-
mographic measurements to assess the local changes in
morphology and physiology of the great vessels. Our observa-
tions may provide a starting point for future clinical studies
in the field of prevention and rehabilitation using HP and
US. In fact, the use of heat therapy has shown some ben-
etits in cases of restricted mobility. Future studies should
explore these and other physiological and metabolic changes
using larger sample sizes in order to validate the observa-
tions described here, including the no significant changes in
morphological and physiological variables, among others.

Authorship

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2. Conception of the study: RAAZ, RRV
3. Design of the study: RRV
4. Acquisition of data: RAAZ, RRV, AGS, CJMB, CAPG, JGO, CALA
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Conflicts of interest

The authors declare not having any conflicts of interest.

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