Table 2
Left ventricular (LV) structure and function parameters in RA patients and controls.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>RA patients (n = 44)</th>
<th>Controls (n = 35)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal thickness mm</td>
<td>10 ± 2</td>
<td>9.3 ± 1.2</td>
<td>0.02</td>
</tr>
<tr>
<td>Posterior wall thickness mm</td>
<td>8.9 ± 1.2</td>
<td>8.2 ± 1.2</td>
<td>0.01</td>
</tr>
<tr>
<td>LV end-diastolic dimension mm</td>
<td>47 ± 7</td>
<td>47 ± 4</td>
<td>0.93</td>
</tr>
<tr>
<td>LV end-systolic dimension mm</td>
<td>25 ± 3.4</td>
<td>27 ± 7</td>
<td>0.23</td>
</tr>
<tr>
<td>Ejection fraction %</td>
<td>62 ± 7</td>
<td>64 ± 4.4</td>
<td>0.26</td>
</tr>
<tr>
<td>LV mass, g</td>
<td>155 ± 47</td>
<td>140 ± 31</td>
<td>0.012</td>
</tr>
<tr>
<td>LV mass index, g/m²</td>
<td>140 ± 31</td>
<td>92 ± 25</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LV mass index, g/m²</td>
<td>43 ± 12</td>
<td>31 ± 10</td>
<td>0.0043</td>
</tr>
<tr>
<td>DT msec</td>
<td>11 ± 0.7</td>
<td>17 ± 0.6</td>
<td>0.0003</td>
</tr>
<tr>
<td>DT index</td>
<td>3 ± 1.2</td>
<td>5 ± 1.2</td>
<td>0.0005</td>
</tr>
<tr>
<td>Distensibility cm² dyn⁻¹ 10⁻⁶</td>
<td>8.3 ± 4.5</td>
<td>5 ± 2.9</td>
<td>0.0002</td>
</tr>
<tr>
<td>Strain %</td>
<td>8.1 ± 3.1</td>
<td>13 ± 7</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

Antigen carbohydrate 125 in heart failure: A promising clinical tool
Julio Núñez *, Gema Miñáez, Eduardo Núñez, Juan Sanchis
Servicio de Cardiología, Hospital Clínico Universitario, INCLIVA, Universitat de Valencia, Valencia-Spain

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Dear editor,

We want to acknowledge Topatan and Başaran for their contribution toward a better understanding of the pathophysiology of antigen carbohydrate 125 (CA125) in heart failure (HF) [1].

First, we want to clarify to the authors that longitudinal results presented in our letter entitled “Antigen carbohydrate 125 in heart failure: Not just a surrogate for serosal effusions?” were misinterpreted and no methodological issues are present in our calculation [2]. These results (which are also presented in figure 1 – see explanation at the bottom of the figure), were expressed as median (interquartile range), and not as median (min-max range).

Second, our disagreement with the above authors can be summarized into two aspects: 1) the role of CA125 within the very complex and poorly understood pathophysiological cascade that ultimately leads to an elevation of pro-inflammatory markers in acute heart failure syndromes (AHF), and; 2) clinical usefulness of CA125 serum levels in HF.

There is plenty of evidence showing a significant elevation in CA125, proinflammatory markers as well as systemic volume expansion in AHF [3–6]. How these factors are inter-related is still a matter of controversy. Is the mesothelial cells activation by volume expansion/serosal effusions the main mechanism for triggering the production of CA125, or is it the already heightened background inflammation in AHF that triggers the activation of the mesothelial cells, leading ultimately to CA125 elevation? We believe that the amount of evidence available is insufficient to dismiss the exact role of CA125 within the (most likely) multifactorial cascade that ultimately leads to an elevation of pro-inflammatory markers in AHF. Therefore, it seems unfair to conclude that CA125 is no more than a simple surrogate for the presence of serosal effusions and mesothelial stimulation. We cautiously have suggested that CA125 may increase in HF patients, not only as a consequence of serosal effusion but, perhaps...
Effects of vascular nitric oxide pathway on vascular smooth muscle cell proliferation

Lian Chen, Nan Dong, Jinyu Huang, Zhansheng Zhu, Minchen Wang, Kaiyun Wu

Department of Anatomy, Medical College of Soochow University, Suzhou 215123, Jiangsu Province, China
Department of Orthopaedics, the First Affiliated Hospital of Soochow University, Suzhou 215006, China

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Previous studies have demonstrated that sympathetic nerves contain NO neuron, but it is unclear whether the NO pathway of sympathetic nerves exerts effects on vascular smooth muscle cell (VSMC) proliferation. To investigate its effect on VSMC proliferation, five groups were used in this study (1): the 1 day model group in which rats were sacrificed after 1 day (2); the 5 day model group in which rats were sacrificed after 5 days (3); the L-NNA group that received intraperitoneal injection of 15 mg/kg L-NNA (a blocker of NOS, used to confirm the effect of nitroxidergic nerve on VSMC proliferation by blocking NO nerves), twice per day, for a total of 6 successive days; (4) the sympathectomy group in which the right common carotid artery was fully exposed, and the nerves that dominate the common carotid artery were all severed, to confirm the effect of sympathetic nerves on VSMC proliferation; and (5) the sham surgery group in which 1 μL physiological saline was added to 2 mm×1 mm filter paper. One week after the experiment, the rats were perfused with 4% paraformaldehyde, and the common carotid artery was harvested. Three animals from each group were used to evaluate mRNA using reverse transcription-polymerase chain reaction (RT-PCR). VSMC proliferation was

The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [11].

References