Letters to the Editor

Squatting: The Hemodynamic Change Is Induced By Enhanced Aortic Wave Reflection

To the Editor:
Murakami 1 has shown convincingly that wave reflection is increased by squatting, and is responsible for increase in aortic systolic pressure. The author argues persuasively that this mechanism in patients with tetralogy of Fallot could explain reduced right-to-left shunting and relief from cyanotic episodes. It is surprising that this mechanism has not been suggested in the past, but investigators could have been misled by amplification of the pressure waveform, which, with squatting, could have caused a lesser increase in brachial than in aortic systolic pressure. The authors measured both brachial and aortic systolic pressure before and after squatting, but only reported changes in aortic systolic pressure. What was the change in brachial systolic pressure with squatting? I expect that such change was considerably less than the change in aortic systolic pressure.

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Reference


In Reply:
I thank Dr. O’Rourke for his comments on my study. 1 I reanalyzed my data about the changes of blood pressure (BP) in brachial artery and central aorta by squatting (Table 1). As expected by Dr. O’Rourke, the increments of systolic BP and pulse pressure in brachial artery were significantly less than those in the central aorta. Therefore, the reason why the increased wave reflection by squatting has not been reported might be the difficulty of central pressure waveform analysis. The development of sphygmoangiographic methods should have made the analysis easy.

Another reason why this mechanism has not been reported may be that few pediatric cardiologists had been interested in the pulsatile property of the arterial pressure wave.

Table 1. The increments of systolic blood pressure and pulse pressure by squatting (n = 12)

<table>
<thead>
<tr>
<th></th>
<th>Brachial Artery</th>
<th>Central Aorta</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>4.0 ± 3.5</td>
<td>8.2 ± 2.9</td>
<td>.0100</td>
</tr>
<tr>
<td>PP</td>
<td>1.1 ± 3.7</td>
<td>5.2 ± 2.6</td>
<td>.0129</td>
</tr>
</tbody>
</table>

SBP = systolic blood pressure; PP = pulse pressure.

Data are given as mean ± SEM.

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Reference


Cardiac Angiotensin II: An Intracrine Hormone?

I write to comment on the recent comprehensive review of cardiac angiotensin by Schuijt and Danser.1

First, the authors state “...the lack of intracellular generation of Ang II does not mean that Ang II has no intracellular effects. It merely means that Ang II is not an intracrine hormone, that is, a hormone that is synthesized and acts intracellularly.” We coined the term intracrine in 1984 to describe the action of a peptide hormone in its cell of synthesis but soon thereafter explicitly expanded the definition to include any intracellular action of a peptide hormone—whether that hormone is internalized or retained in its cell of synthesis.2,3 This definition has the advantage of permitting the term to be used in the case of a peptide hormone that is synthesized and secreted by a cell and then taken up by that same cell to act in the intracellular space. Also, it is remarkable that many intracrine peptide hormones and growth factors operate in both intracrine modes and this dual functionality is likely an important feature of their biology.4 Therefore, until and unless a consensus to the contrary is
reached, we would again argue that the term be used to describe both forms of intracellular peptide action.

Second, the authors appear to have overinterpreted the results of our recent study showing that expression of a construct encoding a nonsecreted angiotensinogen stimulates the growth of hepatoma cells. The fact that the introduction of an angiotensinogen construct was used to demonstrate this effect in these cells does not mean that complete endogenous angiotensin-generating systems do not exist in other cells. Our study was not designed to answer that question. It is known that angiotensin is found in association with physiologically relevant intracellular sites such as euchromatin. This intracrine angiotensin is either internalized or synthesized intracellularly. Neither mechanism has been universally excluded or confirmed in all cells, and there are data to support each in specific cases. In the case of the heart, angiotensin internalization and nuclear binding by cardiac myocytes was demonstrated decades ago, but the totality of available data also suggests cardiac intracellular angiotensin production. This remains an open question.

Finally, it is refreshing that, as this review demonstrates, both the existence of local renin-angiotensin systems and the notion of intracellular angiotensin action, are gaining currency within the research community.

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