Peripheral and Myocardial Microcirculation

To The Editor:

We do not agree with Bøttcher and colleagues \(^1\) when they conclude that the results of their study indicate “...different mechanisms of microvascular activation or regulation...” in coronary and brachial circulation.

The assessment of endothelium-dependent and endothelium-independent vasomotor responses in systemic and coronary arteries has confirmed the association between vascular risk factors and endothelial dysfunction.\(^2,3\)

In their study, Bøttcher and colleagues \(^1\) used two completely different stimuli to induce hyperemia in coronary and brachial circulation. Coronary microvascular dysfunction was measured by the response to the vasoactive substance dipyridamole using positron emission tomography, and brachial artery vasoreactivity was assessed ultrasonographically by postocclusion hyperemia. The effects of these two stimuli on microvascular function are completely different.

In the myocardium, the hyperemic response is mediated by the accumulation of adenosine and the stimulation of adenosine A\(_2\) receptors.\(^4\) Ischemia-induced accumulation of vasodilating metabolites is assumed to be responsible for the postocclusion hyperemia of the brachial artery.\(^5\)

No evidence is provided to conclude that the results of the study indicate different mechanisms controlling microvascular activation or regulation in peripheral and coronary circulation. In contrast, it would be appropriate to state that there is no correlation of vasomotor responses in the two vascular beds if assessed by these two methods.

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Response

We thank Drs Auer, Robert, and Eber for their interest in our article. They raise important issues regarding the interpretation of our findings and particularly regarding our statement about different mechanisms controlling vasodilatation in the peripheral vessels and in the coronary circulation.

We fully agree that our conclusions regarding the lack of correlation between the regulation of the coronary and peripheral circulation confine themselves to the two methods used. This was specifically the purpose of the study and is stated several times throughout the article. It is also stressed in the article that different mechanisms are in play during the two different stimulations, and the mechanisms are as correctly reiterated in the letter. We also agree with their comments regarding endothelial function, which seems to be related in the brachial and coronary circulation. This, however, was not the objective of our study.

We certainly found that both the ultrasound-based evaluation of brachial artery flow and positron-emission tomography-based perfusion measurements in the myocardium are valuable tools in the investigation of cardiovascular disease. However, the ability to extrapolate between the two techniques needs to be kept in mind.

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