Magnesium and Arterial Stiffness

To The Editor:

We read with interest the paper by S. Laurent et al1 dealing with structural and genetic bases of arterial stiffness. The authors reviewed data concerning the heritability of arterial stiffness and propose an integrated view of the structural and genetic determinants of arterial stiffness based on a candidate gene approach and recent studies on gene expression profile.1

It is well documented that large-artery stiffness is the main determinant of pulse pressure.2 In addition, aortic stiffness has independent predictive value for total and cardiovascular mortality, coronary morbidity and mortality,3 and fatal stroke4 in patients with essential hypertension.5 In this context, we could show that a magnesium deficiency is of pathogenetic importance when essential hypertensive patients developed high pulse pressure values (P<0.01).5 In addition, we demonstrated that a magnesium deficiency is involved in the pathogenesis of primary hypertension2,4 in combination with altered structural determinants.8 In a recent paper by our group we also described a loss of elastic material in smooth muscle cells of magnesium-deficient spontaneously hypertensive rats of the Münster strain.8

In conclusion, the review of Laurent et al is excellent; however, the role of a magnesium deficiency resulting in structural changes of vascular vessels has to be taken in account, too. In addition, we agree that signaling pathways and transport mechanisms (eg, TRPM6 and TRPM7) are of special interest for the development of primary hypertension, pulse pressure values, and arterial stiffness.

Klaus Kisters
Medical Clinic I
St Anna Hospital
Herne, Germany

Bernhard Gremmler
Cardiology Department
Marienhospital
Bottrop, Germany

Martin Hausberg
Med. Univ. Poliklinik D
Münster, Germany


Response

We read with interest the letter by Kisters et al1 concerning the potential involvement of magnesium in arterial stiffness. The authors have gathered indirect evidence that magnesium deficiency could increase arterial stiffness. Indeed, they observed low plasma magnesium values in patients with high brachial pulse pressure (PP)2 and abnormal elastic material in the aortic wall of magnesium-deficient spontaneously hypertensive rats of the Münster strain.3 The increase in PP may be due either to a higher arterial stiffness or to earlier wave reflections caused by changes in vasomotor tone of resistive arteries, modifying the reflectance point. Indeed, Adrian et al4 showed an acute vasodilatation of resistive mesenteric arteries in rats in response to an increase in magnesium concentrations together with a reduction in the stiffness of wall material. However, mesenteric arteries are resistive arteries and play only a minor role in systemic stiffness, in contrast to conductance elastic arteries, like the carotid arteries. The same group5 showed that magnesium deficiency did not modify the arterial distensibility of the rat carotid artery, despite increased intima-media thickness. Thus, a direct response to the issue of whether magnesium deficiency is associated with an increased arterial stiffness would be afforded by studying the elastic properties of the carotid artery in the magnesium-deficient spontaneously hypertensive rats of the Munster strain and the demonstration of asignificant correlation between aortic pulse wave velocity and plasma magnesium values in humans, independently of confounding factors.

Stéphane Laurent
Department of Pharmacology and Inserm
Hôpital Européen Georges Pompidou
Paris, France

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Klaus Kisters, Bernhard Gremmler and Martin Hausberg

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