Letters to the Editor

Brain Natriuretic Peptide Increases Acutely and Much More Prominently than Atrial Natriuretic Peptide during Coronary Angioplasty

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A. ANTONIADIS, N. I. NIKOLAOU, D. T. KREMASTINOS:
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To the Editor:

Kyriakides *et al.* report an increase of the natriuretic peptides ANP and BNP in the systemic circulation during PTCA. While this is an interesting observation, its mechanism is not clear. In addition to the authors' speculations, an acute volume load due to radiocontrast administration should be included/discussed as a plausible explanation for the observed increase in the secretion of natriuretic peptides. We have shown a natriuresis during coronary angiography with and without PTCA that was associated with an increase in plasma ANP and particularly with the urinary excretion of the renal natriuretic peptide urodilatin.¹

Because of the hypertonicity of most radiocontrast agents, the physiologically relevant intravascular volume load exceeds the volume of the administered compound. Therefore it would be pertinent for the interpretation of the reported findings to know (a) the volume and (b) the osmolality of the radiocontrast agents employed by Kyriakides *et al.*

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Reference

1. Haller C, Meyer M, Scheele T, Koch A, Forssmann WG, Kübler W: Radiocontrast-induced natriuresis associated with increased urinary urodilatin excretion. *J Internal Med* 1998;243:155–162

Authors' reply:

We appreciate the comments made by Drs. Haller and Meyer regarding our paper about cardiac natriuretic peptides during coronary angioplasty. Their main concern is in regard to the mechanisms underlying the increased secretion of cardiac natriuretic peptides during coronary angioplasty. In our study we used 100–300 ml of a contrast agent containing sodium ioxaglate 19.65 g/100 ml and meglumine ioxaglate 39.3 g/100 ml with osmolality 600 mosmol/kg (Hexabrix, Guerbet, France).

Haller *et al.*¹ used contrast agents with higher osmolality in a population with high incidence of left ventricular dysfunction. However, hypertonicity of contrast material may indeed impose an acute volume load, and therefore cannot be excluded as a factor contributing to enhanced peptide secretion.

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References

1. Haller C, Meyer M, Scheele T, Koch A, Forssmann WG, Kübler W: Radiocontrast-induced natriuresis associated with increased urinary urodilatin excretion. *J Internal Med* 1998;243:155–162

True-Positive Exercise Electrocardiogram/ False-Negative Thallium-201 Scintigram: A Proposal of a Mechanism for the Paradox

J. E. MADIAS, P. KNEZ, M. T. WIN: *Clin Cardiol* 2000;23:625–629

To the Editor:

I would like to compliment the authors, Dr. Madias and colleagues, on their excellent article. The illustrations, especially Figure 6, are excellent and clearly demonstrate important elements of the pathophysiology of exercise-induced ischemia.

The statement that ECG positive/thallium negative scintigram tests on patients with ischemia are rare has not been experience, however. In a small unpublished study of about 200 patients comparing coronary angiograms with thallium scintigraphy, we found the false negative rate of thallium to be approximately 40%. In our facility never a month goes by that one of our cardiologists doesn't present a case in which negative thallium occurred in a patient with severe coronary artery disease, often 1- or 2-vessel disease as well as 3-vessel disease. Many of our patients are referred for nuclear testing after having had an abnormal exercise test in a private office; thus a work-up bias may explain some, but not all, of these patients. I believe there must be other explanations for this finding, besides balanced hypoperfusion, that have not yet been elucidated.

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(continued on page 716)