Letters to the Editor

Heparin-Releasable Platelet Factor 4 in Patients with Coronary Artery Disease

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To the Editor:

We have read with interest the article of Sadayasu and co-workers[1] on heparin-releasable platelet factor 4 (HR-PF4) in patients with coronary artery disease. However, we believe the following comments are in order:

1. We were surprised to see such a mismatch between the control group and patients in the ratio of males to females. The female patients were 17%, while in the control group they were 40%. It has been shown by O'Brien[2] and by us[3] that there is a statistically significant difference in HR-PF4 between males and females.

2. There is no mention of the platelet count distribution in controls and patients. Platelets are the major determinant of HR-PF4.[4, 5]

3. In spite of the statistically significant difference in HR-PF4 mean levels between controls and patients, it would be interesting to see how many patients had HR-PF-4 above the normal range and whether there were causes in addition to coronary artery disease (CAD) that could be involved in platelet activation. We found that the majority of patients with well documented severe CAD had a normal HR-PF4. Only a few patients with a combination of clinical disorders had an increased HR-PF4.

4. It seems very unlikely that aspirin can influence *in vivo* HR-PF4. It has been shown by Ludlam[6] and Minar[7] that platelet specific proteins are not affected *in vivo* by antiplatelet agents, and by us[4] that these drugs do not affect HR-PF4. Although a significant reduction in HR-PF4 mean levels before and after aspirin treatment was seen by Sadayasu *et al.*, three of their patients had an increased level of HR-PF4 after aspirin, while in the others the decrease seems to be small. A possible explanation could be that different platelet counts were present at the time of the study and that less *in vitro* release is taking place during samples collection in patients taking aspirin.

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Authors' reply:

We are pleased with Drs. Cella, Luzzatto, and Strauss's attention to our article. Though there is some truth in their comments, our results were different than theirs. In our study there was no statistical difference in platelet count between controls and patients (opposed to comment #2). Other comments seem to originate from the essential inconsistency of *in vivo* study and its statistical analysis. We would like to proceed with our study on the basis of their comments.

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