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LETTERS TO THE EDITOR

Hypertension and the Prothrombotic State

We read with interest the report by Pini et al. (1) that stated patients with isolated systolic hypertension had a higher prevalence of cardiac hypertrophy and carotid atherosclerosis than did those with diastolic hypertension. Indeed, hypertensive left ventricular hypertrophy is the most evident manifestation of hypertensive target organ damage, and such patients are at particularly high risk for strokes and heart attacks. We would like to propose an additional interpretation of their important observations.

Despite the vessels being exposed to high pressures, the main complications of hypertension (strokes, myocardial infarction) are, paradoxically, thrombotic rather than hemorrhagic — the so-called thrombotic paradox of hypertension or the Birmingham paradox (2). The findings by Pini et al. (1) would actually strengthen our view that hypertension confers a prothrombotic or hypercoagulable state by fulfilling the three different components of Virchow's triad for thrombogenesis. With regard to the latter, there ought to be changes in the blood flow, changes in the vessel wall, and changes in the blood constituents, for increased thrombogenesis. "Abnormal flow" is evident in hypertension, with blood vessels exposed to blood flow under high pressures, as well as abnormal coronary flow reserve and microcirculatory changes (3). We had previously reported abnormalities in prothrombotic factors, endothelial function, and platelet activation in patients with isolated systolic hypertension, comparable to that observed with systolic–diastolic hypertension (3–5).

The study by Pini et al. (1) certainly confirms the presence of "vessel wall abnormalities" with the high prevalence of cardiac hypertrophy and carotid atherosclerosis. Furthermore, hypertensive patients with target organ damage (5) show evidence suggestive of an even greater prothrombotic state, which would contribute to the high risk of vascular complications in such patients.

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REPLY

I have read the interesting letter by Profs. Nadar and Lip. Their speculation on the results of our study adds an additional interpretation of our data. However, our study focused on cardiac and vascular remodeling rather than on prothrombotic factors; thus, because we did not analyze whether isolated systolic hypertension was associated with direct evidence of a prothrombotic state we cannot provide substantive comments on the interesting speculations of Profs. Nadar and Lip.

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Questions Remain Regarding Patients With Aortic Stenosis and Severe Pulmonary Hypertension

Malouf et al. (1) have presented data in an uncommon but clinically important subgroup of patients with severe aortic stenosis and severe pulmonary hypertension. In their study, one subgroup of patients had aortic valve replacement and another was treated medically. The data are very interesting and important.

However, to understand fully the study groups and their outcomes, the investigators need to present additional information about the two subgroups:

1. How was left ventricular ejection fraction measured?
2. What was the actual calculated pulmonary artery systolic pressure (PASP) (mean ± SD, and range)?
3. What were the number and percentage of patients who underwent selective coronary arteriography? Of those who underwent coronary arteriography, what were the number and percentage who had significantly obstructive coronary artery disease? Also, did all patients with significantly obstructive coronary artery disease have coronary bypass graft surgery? It should be noted that the mean patient age was 78 ± 8 years.
4. What was the five-year survival, including operative mortality (mean ± SE) and the p value for the difference in survival?

These investigators are to be congratulated on this valuable study in a group of patients in whom additional data were needed.

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