**Correspondence**

Letters to the Editor must not exceed 400 words in length and must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Authors of letters selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication. Please submit three typewritten, double-spaced copies of the letter to Herbert L. Fred, MD, % the Circulation Editorial Office. Letters will not be returned.

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**Serum Myoglobin in Pulmonary Embolism**

To The Editor:

N. Kucher and S.Z. Goldhaber discuss the potential role of cardiac troponins and brain natriuretic peptides in the risk stratification of patients with acute pulmonary embolism (APE). Increased levels of cardiac troponins indicate myocardial damage in an overloaded right ventricular myocardium. Interestingly, right ventricular infarctions were reported in patients who died from massive APE and in survivors with angiographically normal coronary arteries. Myoglobin, a highly sensitive marker of myocardial injury, can be elevated after myocardial damage, even before any detectable rise of cardiac troponin levels occurs. However, to our knowledge, measurement of serum myoglobin levels has not been evaluated in APE. Therefore, we checked the prevalence and prognostic significance of elevated serum myoglobin levels in 46 patients with major APE. Our study showed that on admission, myoglobin serum concentrations are elevated in 45% of APE patients. All 7 in-hospital deaths occurred in the group with elevated serum myoglobin, and in one fatal case, cardiac troponin T measured simultaneously was negative. Moreover, elevated serum myoglobin was a significant predictor of fatal outcome (odds ratio 25, 95% confidence interval 1.3 to 474.2). We think that both cardiac troponins and myoglobin, a biochemical marker of myocardial injury, are powerful predictors of increased risk of fatal outcome in major pulmonary embolism.

Anna Bochowicz, MD
Maciej Kostrubiec, MD
Piotr Pruszczyk, MD
Department of Internal Medicine and Hypertension
The Medical University of Warsaw
Banacha 1a
02–096 Warsaw, Poland
piotr.pruszczyk@amuw.edu.pl

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Anna Bochowicz, Maciej Kostrubiec and Piotr Pruszczyk

Circulation. 2004;109:e194
doi: 10.1161/01.CIR.0000127106.85030.4A
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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