3. O'Donaghue M, Kenney P, Oestreicher E, et al. Usefulness of aminoterminal pro-brain natriuretic peptide testing for the diagnostic and prognostic evaluation of dyspneic patients with diabetes mellitus seen in the emergency department (from the PRIDE study). Am J Cardiol 2007;100:1336–40.
4. Azevedo A, Bettencourt P, Pimenta J, et al. Clinical syndrome suggestive of heart failure is frequently attributable to non-cardiac disorders—population-based study. Eur J Heart Fail 2007;9:391–6.
5. Androne AS, Hryniewicz K, Hudaihed A, et al. Relation of unrecognised hypervolaemia in chronic heart failure to clinical status, haemodynamics, and patient outcomes. Am J Cardiol 2004;93:1254–9.
6. Chakko S, Woska D, Martinez H, et al. Clinical, radiographic, and haemodynamic correlations in chronic congestive heart failure: conflicting results may lead to inappropriate care. Am J Med 1991;90:353–9.
7. Black ER, Panzer RJ, Mayewski RJ, Griner PF. Characteristics of diagnostic tests and principles for their use in quantitative decision making. In: Panzer RJ, Black ER, Griner PF, editors. Diagnostic Strategies for Common Medical Problems. Philadelphia, PA: American College of Physicians 1991:1–16.
8. Wu AH, Smith A, Wieczorek S, et al. Biological variation for N-terminal pro- and B-type natriuretic peptides and implications for therapeutic monitoring of patients with congestive heart failure. Am J Cardiol 2003;92:628–31.
9. Tang WHW, Girod JP, Lee MJ, et al. Plasma B-type peptide levels in ambulatory patients with established chronic symptomatic systolic heart failure. Circulation 2003;108:2964–6.
10. Pruszczyk P. N-terminal pro-brain natriuretic peptide as an indicator of right ventricular dysfunction. J Card Fail 2005;11 Suppl:S65–9.
11. Leuchte HH, Baumgartner RA, Nounou ME, et al. Brain natriuretic peptide is a prognostic parameter in chronic lung disease. Am J Respir Crit Care Med 2006;173:744–50.

Reply

We would like to thank Drs. MacFadyen and Chuen for their interest in our study (1). They seem to conclude that our population-based report advocates screening for all persons with dyspnea. Unfortunately, the majority of the issues they raise are based on this false assumption. We believe that it is necessary to await the results from studies evaluating the cost-effectiveness before introducing pro–B-type natriuretic peptide (proBNP) as a screening tool of all breathless persons in the community. We agree that if one sought to examine the cost-effectiveness of the natriuretic peptides in primary care, they should include the additional information that is obtainable for the general practitioner.

The aim of our study was to examine whether or not plasma proBNP could be used in discriminating between cardiac and pulmonary dysfunction in the general population with dyspnea. Our findings that plasma proBNP is increased in left ventricular dilation, hypertrophy, systolic and diastolic dysfunction, but unaffected by pulmonary dysfunction, should be viewed in this perspective. Drs. MacFadyen and Chuen also point out that 48% of the population in our study reported dyspnea without having an abnormal conventional echocardiogram or pulmonary function test. It is important to emphasize that a normal conventional echocardiogram does not rule out cardiac dysfunction. Most of these people (83%) were only complaining of mild dyspnea. Compared with their nondyspneic counterparts, they had a higher frequency of ischemic heart disease and risk factors for ischemic heart disease. Conventional echocardiography does not provide much information about longitudinal myocardial contractility, which is impaired initially in ischemic heart disease. In fact, we suspect that breathless persons with a high level of plasma proBNP have a high risk of cardiac dysfunction, although they might have normal conventional echocardiograms. Further research with more advanced modalities (e.g., tissue Doppler imaging) within such groups is needed.

Intraindividual and interindividual variability of plasma proBNP should not be extrapolated from studies of BNP or N-terminal proBNP. The interindividual variability is one of the major reasons for the assay's gray zone reported in our study, whereas the intraindividual variability of the assay is small (2). Regarding the ability to rule out parenchymal lung disease by spirometry, pulse oxymetry, and clinical history, we agree that additional information from computed tomography scans and lung biopsy would have been interesting but obviously not possible in this population study. Concerning the last issue of pulmonary hypertension in parenchymal lung disease, we have previously shown that plasma proBNP only increases at elevated mean pulmonary artery pressures >50 mm Hg in patients with terminal parenchymal lung disease (3).

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REFERENCES

1. Mogelvang R, Goetze JP, Schnohr P, et al. Discriminating between cardiac and pulmonary dysfunction in the general population with dyspnea by plasma pro-B-type natriuretic peptide. J Am Coll Cardiol 2007;50:1694–701.
2. Damgaard M, Goetze JP, Norsk P, Gadsboll N. Altered sodium intake affects plasma concentrations of BNP but not proBNP in healthy individuals and patients with compensated heart failure. Eur Heart J 2007;28:2726–31.
3. Goetze JP, Videbaek R, Boesgaard S, Aldershvile J, Rehfeld JF, Carlsen J. Pro-brain natriuretic peptide as marker of cardiovascular or pulmonary causes of dyspnea in patients with terminal parenchymal lung disease. J Heart Lung Transplant 2004;23:80–7.

Plaque Burden, Intravascular Ultrasound, and Distal Embolization Phenomenon

Although embolization of atherothrombotic material during stent deployment is probably ubiquitous, its causes and consequences remain poorly characterized. In a recent issue of the *Journal*, both Kawaguchi et al. (1) and Kawamoto et al. (2) suggested that vessels most likely to sustain distal embolization could be identified using virtual histology (VH) intravascular ultrasound (IVUS) to quantify