What is the most useful and cost-effective strategy to screen for left ventricular systolic dysfunction in clinical practice?: reply

We agree with many of the comments made by Yamada et al.,1 namely that the ECG and NTproBNP are fundamental tests used in the diagnosis of heart failure and that as tests become cheaper they become more cost-effective. We further agree that our data show that hand-held echocardiography gives the greatest sensitivity, ECGs give intermediate sensitivity, and NTproBNP normal range cuts off the worst sensitivity in detecting left ventricular systolic dysfunction (LVSD).2 We also agree that as screening sensitivity falls, some subjects with the screened-for condition will fail to have it diagnosed. Although we clearly stated that the screening programmes described would miss cases of LVSD, we did not make it clear that this was a function of screening sensitivity, an important point that we are pleased Yamada et al. have emphasized. Thus, NTproBNP-driven screening would leave more subjects with LVSD undiagnosed than ECG- or hand-held echocardiography-driven screening. However, as ECG-driven screening has much lower specificity (thus higher false positive rates) and hand-held echocardiography costs more than NTproBNP per test, our conclusion that NTproBNP or ECG screening prior to hand-held echocardiography prior to traditional echocardiography is the most cost-effective strategy, at both current test costs and future likely test costs, remains correct. Furthermore, despite its lower sensitivity, NTproBNP-driven screening might be justifiable in practice, as subjects with normal NTproBNP levels have extremely low cardiac morbidity and mortality whatever their left ventricular function,3,4 although long-term follow-up of our subjects would be required to confirm this. Finally, we agree that if the price of hand-held echocardiography approached that of the ECG, hand-held echocardiography would clearly become the screening method of choice. However, whether this would ever happen is unclear, and furthermore were this to happen then whether enough fully trained sonographers would be available to accurately screen for LVSD using this technology is also unclear, a major potential future issue.

References

1. Yamada A, Yamada Y, Okada K. What is the most cost-effective strategy to screen for left ventricular systolic dysfunction: natriuretic peptides, the electrocardiogram, hand-held echocardiography, traditional echocardiography, or their combination? [Letter]. Eur Heart J 2006, in press.


Gavin Galasko
Department of Cardiovascular Medicine
Northwick Park Hospital
Level 9V
Watford Road
Harrow
Middlesex HA1 3UJ
UK

Roxy Senior
Department of Cardiovascular Medicine
Northwick Park Hospital
Level 9V
St Marks
Watford Road
Harrow
Middlesex HA1 3UJ
UK

Tel: +44 208 869 2547
Fax: +44 208 864 0075
E-mail address: roxy.senior@virgin.net

Incidence of recognized and unrecognized myocardial infarction in men and women aged 55 and older: the Rotterdam Study

The Rotterdam Study5 might well be a ‘wake-up call’ for those authors of management guidelines5 for myocardial infarction (MI), who do not sufficiently stress the potential for saving lives if the non-chest pain presentations of MI are promptly recognized and evaluated for eligibility for thrombolysis and other therapeutic strategies. Our National Service Framework (NSF) for coronary heart disease recognizes that many patients initially thought to have MI may have other causes for their chest pain. Nevertheless, it does not stress that, as a corollary, MI might have a pain-free presentation characterized, instead, by sudden onset of unexplained dyspnoea and by unexplained collapse.2 These alternative clinical stigmata of MI are well described in the elderly,3 and might well have been a feature of many of the patients in the Rotterdam Study. The other major shortcoming of our NSF emerges in the outline of models of care to be used in hospital-wide protocols, and here the advice on the assessment of eligibility for thrombolysis deals with management of those MI patients who present with chest pain without referring to the potential benefits of thrombolysis in those who do not have chest pain.4 Nowhere is there a recognition that a pain-free MI patient who presents to the hospital promptly with sudden onset dyspnoea or collapse might well be within the therapeutic time window for thrombolysis if the electrocardiographic criteria for such treatment are met.4

References


Risk assessment in acute pulmonary embolism

We read with great interest the paper by Aujesky et al., validating the prognostic model comprising 11 routinely available clinical parameters in patients with pulmonary embolism (PE). However, the presented model is of low cost but also complex. Moreover, some parameters like presence of cancer, altered mental status, severity of heart failure, and chronic lung disease, can be difficult to assess and are observer-dependent. It is also remarkable that the presented model does not include increased creatinine level reflecting impaired renal function, which was reported to be an important prognostic factor in acute PE patients. According to the authors, this important prognostic factor in acute PE depends on the haemodynamic compromise mostly determined by the level of right ventricle overload. Natriuretic peptides and troponin are well-established markers of cardiovascular mortality. It is generally accepted that they reflect the severity of acute heart dysfunction in PE. In 2005, there were two papers that proposed inclusion of laboratory parameters. Our model is most useful in identifying patients with clinically massive embolism and reached 33%. Importantly, both biomarkers helped to stratify 40-day prognosis in acute PE for both low and high-risk groups. Therefore, on the basis of the biomarkers model, stratifying the risk in PE seems to be the option, which provides objective and accurate prognosis assessment.

Maciej Kostrubiec
Department of Internal Medicine, Hypertension, and Angiology
The Medical University of Warsaw
Banacha 1a
02-097 Warsaw
Poland

Anna Kaczyńska
Department of Internal Medicine, Hypertension, and Angiology
The Medical University of Warsaw
Banacha 1a
02-097 Warsaw
Poland

Piotr Pruszczyk
Department of Internal Medicine, Hypertension, and Angiology
The Medical University of Warsaw
Banacha 1a
02-097 Warsaw
Poland

References