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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 NT-proBNP 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 NT-proBNP 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, NT-proBNP-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 NT-proBNP per test, our conclusion that NT-proBNP 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, NT-proBNP-driven screening might be justifiable in practice, as subjects with normal NT-proBNP 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.

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

The Rotterdam Study1 might well be a ‘wake-up call’ for those authors of management guidelines 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 function, which was reported to be an increased creatinine level reflecting impaired renal function, 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.

Kostrubiec and colleagues criticize that the creatinine level, a known prognostic factor for PE, is not part of our model. To facilitate its applicability, we derived the model using only clinical factors, without the inclusion of laboratory parameters. However, we also assessed several laboratory variables as potential predictors in the logistic regression model. In this more complex model, a blood urea nitrogen value of $\geq 11$ mmol/L (but not a creatinine value of $\geq 133$ µmol/L) was independently associated with mortality. Although the more complex model including laboratory variables achieved a slightly higher discriminatory power for mortality, its overall prognostic performance was not superior to the simpler model.

Our model is most useful in identifying low-risk patients with PE who are potential candidates for outpatient treatment with low-molecular weight heparins. Patients with the highest risk based on our model (risk class V) had a 30-day mortality between 10–25%, resulting in positive predictive values (PPVs) for mortality of 11–14%. While these PPVs are too low to accurately identify high-risk patients with PE, the PPVs of cardiac biomarkers and echocardiography are similarly low (6–44 and 11–25%, respectively). Therefore, the basis of the biomarkers model, stratifying the risk in PE seems to be the option, which provides objective and accurate prognosis assessment.

References


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Risk assessment in acute pulmonary embolism: reply

Kostrubiec and colleagues are concerned that our 11-variable prognostic model for pulmonary embolism (PE) may be too complex to be applied in clinical practice and that some of the variables of the model might be difficult to assess. While we agree that the application of our model probably necessitates the help of a written reminder, the model consists of 11 easily available demographic characteristics and history and physical examination findings, without the need for laboratory or radiographic tests. This should greatly facilitate the practical applicability of the model. While the reproducibility of our model was not yet tested in an interobserver agreement study, all prognostic factors represent explicit, dichotomous variables that can be easily obtained at the patient’s bedside. These factors include the patient’s age, sex, pulse, blood pressure, respiratory rate, temperature, arterial oxygen saturation, an altered mental status (defined as the presence of disorientation, lethargy, stupor, or coma), and a known history of cancer, heart failure, or any chronic lung disease. The severity of heart failure, as implied by Kostrubiec, does not play a role in our model.