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Authors' reply

We welcome the letter from Dr Iles and the opportunity to make additional comments on the subject of our recent editorial.[1] It is correct that the mortality rate in normotensive patients with acute pulmonary embolus (PE) reported in the recent study by Boca *et al*[2] refers to all-cause mortality at 3 months and not inpatient mortality, and we are pleased to have the opportunity to clarify this. A wide range of mortality rates in acute PE have been reported in published studies, depending on whether hypotensive and normotensive patients are included together or reported separately and whether inpatient, 30-day or 3-month mortality is quoted as an end point. Furthermore, identifying the exact cause of death in studies of PE is very difficult and few if any have been able to provide accurate data on this, therefore most report all-cause mortality. In the recent European Society of Cardiology guidelines on acute PE[3] it states that the risk of early mortality in normotensive patients with acute PE is dependent on the presence of right ventricular dysfunction (RVD) on transthoracic echocardiography, with studies reporting rates of 3–15% in those who are normotensive with RVD and <3% in those without RVD. Indeed, in the ICOPER study,[4] 50% of normotensive patients with acute PE had RVD and the mortality in that group was 10%, much higher than in those who were normotensive but without evidence of RVD. These observations imply that, even in normotensive patients, clot burden as implied by the presence of RVD contributes to the risk of early death. This suggests that death after PE in those normotensive at presentation is not simply down to other diagnoses such as cancer but that cardiorespiratory comorbidities are likely to contribute to the risk in an additive way. In a recent study by Ibrahim and colleagues which included >15 000 patients with acute PE, the 30-day mortality rate in normotensive patients not receiving thrombolysis was 7.7% and the in-hospital mortality rate for normotensive patients who did not receive thrombolysis was 7.2 per 1000 person days.[5] We therefore believe that it would be wrong to underestimate the early acute PE-associated mortality risk, even in normotensive patients. We believe this is significant in those with objective evidence on echocardiography or on cardiac biomarkers of RVD. It is this association which is eloquently described in the original article on which our editorial was based, stressing its importance to the literature.[6]

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Predicting CAP-related mortality with CRB-65

Ewig *et al* are to be commended for their very large study of 388 406 patients admitted with community-acquired pneumonia (CAP) in German hospitals from 2005 to 2006.[1] Using the CRB-65 tool (confusion, respiratory rate ≥30 min, low blood pressure (either systolic <90 mm Hg or diastolic ≤60 mm Hg) and age ≥65 years), the authors found 30-day mortality rates of 2.4, 13.4 and 34.4% in those with 0 points, 1–2 points and 3–4 points, respectively. As a result, the authors promote this tool as being accurate for predicting CAP-related deaths.

However, while this appears impressive, it is notable that of the >54 700 deaths, only 29.0% were classed as high risk, whereas 68.1% were only intermediate risk and 2.8% were low risk. In addition, many of those patients who died had treatment limitations applied and only 15.7% of the patients who died received ventilatory support. These two points raise the question of how clinically useful this tool really is. If over two-thirds of deaths were classed as having clinically 'moderate' CAP, then the tool cannot really be described as being accurate for this purpose. Furthermore, if the vast majority of people who died did so after active treatment was withdrawn, then the identification of such patients does not appear to serve much purpose. It would be more relevant to assess such a tool for its ability to identify those patients in whom every effort is made to save their lives—that is, those admitted to the intensive care unit.

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Authors' response

We thank Dr Charles for his important comments.[1]

He raises the important question of whether CRB-65 is a useful tool to advise treatment limitations. If only 29% of those who finally died were at high risk of death at initial presentation (CRB-65 risk class 3), such a tool may be of limited value in this regard. In fact, we agree that the CRB-65 score (like any other such as the PSI) is not helpful for the decision to apply treatment restrictions. Such restrictions up to fully palliative treatment cannot be based primarily on considerations about the current risk of death but should be the result of a careful evaluation of the clinical state and overall prognosis of the patient, both initially and during follow-up, and such decisions should be decided with the patient or his legal social worker.

In this context, the CRB-65 severity score remains important as part of the initial clinical evaluation of all patients. Treatment restrictions must not follow a hidden agenda