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Accepted 1 December 2009

Published Online First 23 September 2010

Thorax 2011;**66**:261–262.

doi:10.1136/thx.2009.128751

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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 PE³ 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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Accepted 24 August 2010

Published Online First 23 September 2010

Thorax 2011;**66**:262. doi:10.1136/thx.2010.149682**REFERENCES**

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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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Accepted 28 January 2010

Published Online First 14 October 2010

Thorax 2011;**66**:262. doi:10.1136/thx.2009.133884**REFERENCE**

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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

but must be openly discussed, communicated and documented. A predicted moderate to high risk of death from community-acquired pneumonia is a highly relevant piece of information required to mount an ethically valid treatment recommendation and decision, particularly in those patients with pneumonia regarded to be a terminal event. Nevertheless, we recalculated the predictions of the CRB-65 score excluding all those who died without having received any ventilator support during hospitalisation. The results are: overall death rate 8618, 2.5%, CRB-65 risk class 1: 0.5%, risk class 2: 1.7% and risk class 3: 12.2%. These numbers support the following conclusions: (1) the CRB-65 score remains useful in predicting deaths in a three class pattern; (2) obviously, virtually no previous study on community-acquired pneumonia truly excluded all patients with treatment limitations.

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Competing interests None.

Provenance and peer review Not commissioned; not externally peer reviewed.

Accepted 24 August 2010
Published Online First 23 November 2010

Thorax 2011;**66**:262–263.
doi:10.1136/thx.2010.149716

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Pulmonary rehabilitation in patients with MRC Dyspnoea Scale 2

The recent INTERCOM study emphasises the point that community-based rehabilitation is effective, even in patients with chronic obstructive pulmonary disease (COPD) with less advanced airflow obstruction.¹ However, COPD and pulmonary rehabilitation guidelines recommend offering pulmonary rehabilitation (PR) to patients who consider themselves functionally disabled (usually defined as MRC Dyspnoea Scale grades 3 or above).^{2–3} We wished to test whether less breathless patients with COPD (ie, MRC Dyspnoea Scale grade 2) also benefit from PR.

METHODS

All patients with MRC grade 2 dyspnoea referred to the Lambeth & Southwark Community Pulmonary Rehabilitation Team

Table 1 Effects of pulmonary rehabilitation in patients with MRC 2 and MRC 3/4 dyspnoea

| Outcomes | Change following PR | | p Value |
|---|---------------------|-------------|---------|
| | MRC 2 | MRC 3/4 | |
| Mean (SD) ISW (m) | 83 (7) | 68 (5) | 0.08 |
| Median (25 th , 75 th centile) ISW% change | 27 (12, 45) | 33 (9, 68) | 0.07 |
| Mean (SD) CRQ-D | 0.75 (0.11) | 0.75 (0.07) | 0.96 |
| Median (25 th , 75 th centile) HAD-anxiety | −1 (−3, 1) | −1 (−3, 0) | 0.74 |
| Median (25 th , 75 th centile) HAD-depression | 0 (−2.5, 1) | −1 (−3, 0) | 0.46 |

HAD, Hospital Anxiety and Depression Scale; ISW, incremental shuttle walk; PR, pulmonary rehabilitation.

between the years 2004–7 were included in the study. Patients were offered PR at one of two hospital or five community sites. Each programme consisted of two supervised sessions per week for 8 weeks (with one unsupervised home session) delivered by the same team. Outcome measures were the incremental shuttle walk (ISW), the Chronic Respiratory Disease Questionnaire Dyspnoea score (CRQ-D) and the Hospital Anxiety and Depression Scale (HAD-Anxiety and HAD-Depression). Patients with COPD with MRC dyspnoea grades 3 or 4 undertaking PR over the same time period acted as controls. Changes in outcomes between patients with MRC grade 2 and those with MRC grades 3 or 4 dyspnoea before and after PR were compared using t tests or Mann-Whitney tests.

RESULTS

The results were analysed for 126 patients with MRC grade 2 dyspnoea and 316 with MRC grades 3/4 dyspnoea who completed PR (attended >8 supervised sessions). The groups were well matched for age (mean 69 vs 68 years), gender (50% vs 43% male) and mean forced expiratory volume in 1 s (58% vs 54% predicted), although the MRC grade 2 group had increased ISW (304 vs 201 m; $p < 0.001$), less dyspnoea (median CRQ-D 3.2 vs 2.6) and reduced anxiety and depression scores (median HAD-Anxiety 6.0 vs 9.0; median HAD-Depression 5.0 vs 8.0). Following PR, the MRC grade 2 dyspnoea group showed similar improvements in ISW, CRQ-D, HAD-Anxiety and HAD-Depression to the MRC grades 3/4 dyspnoea group (table 1).

DISCUSSION

Although patients with MRC dyspnoea grade 2 referred for PR have better exercise capacity and fewer symptoms of dyspnoea, anxiety or depression than patients with MRC dyspnoea grades 3/4, they show similar improvements with PR. Exercise-based interventions for COPD should not ignore less severe patients (either in terms of lung function or subjective dyspnoea).

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Funding WD-CM is supported by a National Institute for Health Research Clinician Scientist award. The views expressed in this publication are those of the authors and not necessarily those of the NHS, The National Institute for Health Research or the Department of Health.

Competing interests None.

Provenance and peer review Not commissioned; not externally peer reviewed.

Accepted 8 February 2010
Published Online First 29 September 2010

Thorax 2011;**66**:263. doi:10.1136/thx.2010.136085

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The potential danger of a solely interferon- γ release assay-based approach to testing for latent *Mycobacterium tuberculosis* infection in children

The study reported by Lucas *et al*¹ is a valuable addition to recent publications that have compared the performance of commercial interferon- γ release assays (IGRAs) with that of the tuberculin skin test (TST) for the diagnosis of latent tuberculosis infection (LTBI) in high-risk children.^{2–3} However, we believe that the principal conclusions are not supported by the data provided and that a more guarded interpretation is warranted.

In agreement with previous studies in children,^{3–5} Lucas *et al* found significant discordance between the results of IGRAs and TST. Specifically, of 420 T-SPOT.TB and 460