However, a potential important confounding factor may explain a part of their results: undiagnosed pulmonary embolism (PE), mimicking (or induced by) COPD exacerbation. Troponin and BNP are factors associated with poor prognosis in PE.[2] COPD is associated with an increased risk of deep venous thrombosis and PE (particularly during exacerbation) and with an increased risk of fatal PE.[3] In particular, COPD is associated with increased risk of death from undiagnosed PE.[4]

The real incidence of PE during exacerbation of COPD is not clearly known, ranging from 1.5% to 24.7%[5] corresponding to the incidence of elevated troponin and BNP, as noted by Chang *et al* in their cohort. Therefore, it would be of great interest if Chang *et al* could provide us some precise answers:

- In how many of the 250 patients a PE has been evoked and/or eliminated?
- How many patients were under efficient anticoagulant drugs at inclusion?
- How many patients received thromboprophylaxis, as a significant number of patients included presented other PE risk factors such as malignancy or cerebrovascular diseases?

Because of reserved prognosis of COPD patients with PE, and of the availability of preventive and curative specific drugs, COPD patients admitted with exacerbation and with abnormal cardiac biomarkers may require a PE screening and effective thromboprophylaxis if PE has been ruled out.

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Competing interests None.

Contributors LB, PM and HD analysed the data and drafted the manuscript.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 26 May 2011
Published Online First 18 June 2011

Thorax 2012;**67**:177–178.
doi:10.1136/thoraxjnl-2011-200416

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Authors' response

We thank Bertoletti and colleagues for raising the important issue of pulmonary embolism (PE) in the exacerbation of chronic obstructive pulmonary disease (COPD).[1] Although we did not routinely investigate for PE in our cohort, we excluded any patients with suspected or confirmed PE from the study.[2] Unfortunately, it is difficult to detect thromboembolic events in this population and it is possible that we included some patients with subclinical pulmonary emboli. It is also plausible that this contributed to the association between elevated cardiac biomarkers and mortality. However, we think that this is unlikely to be the only mechanism.

Thromboprophylaxis was administered to some patients during their admission depending on their immobility and other risk factors, but this would not have influenced the NT-proBNP or troponin T results obtained on presentation. We did not collect information on pre-existing anticoagulation therapy on admission to the study.

Further research into the mechanism linking elevated cardiac biomarkers and mortality in COPD exacerbation is needed. We agree with Bertoletti and colleagues that investigating the contribution of concurrent PE is important, as this is something that can be treated.

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Competing interests None.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 25 May 2011
Published Online First 18 June 2011

Thorax 2012;**67**:178.
doi:10.1136/thoraxjnl-2011-200512

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CORRECTIONS

doi:10.1136/thoraxjnl-2011-201054c.151corr1

Thorax 2011;**66**:A128–A129 doi:10.1136/thoraxjnl-2011-201054c.151. P151 Cost of pulmonary rehabilitation is offset by reduction in healthcare utilisation. The author list and author affiliations for this poster should read: [1] S Kibe, [1] D Ford, [2] S Hart. 1 Scarborough General Hospital, Scarborough, UK; 2 Castle Hill Hospital, Hull, UK.

doi:10.1136/thoraxjnl-2011-201054c.163corr1

Thorax 2011;**66**:A133–A134 doi:10.1136/thoraxjnl-2011-201054c.163. P163 Factors influencing histological confirmation of diagnosis in lung cancer patients. The author list for this poster should read: S Chandramouli, M Cheema, J Corless. Wirral Lung Unit, Arrowe Park Hospital, Wirral CH49 5PE, UK.

doi:10.1136/thoraxjnl-2011-201054c.233corr1

Thorax 2011;**66**:A162–A163 doi:10.1136/thoraxjnl-2011-201054c.233. P233 Judicious use of oximetry can help deliver cost effective sleep service. The author list and affiliation for this poster should read: C L Collins, B Balakrishnan, J Madieros, M Sovani. Queen's Medical Centre, Nottingham University Hospitals, Nottingham, UK.

doi:10.1136/thoraxjnl-2011-201054c.179corr1

Thorax 2011;**66**:A140 doi:10.1136/thoraxjnl-2011-201054c.179. P179 The changing numbers and indications of mediastinoscopy procedures performed following the introduction of endobronchical ultrasound at a UK tertiary centre. The author list and affiliations for this poster should read: [1]M Bakir, [2]R Breen, [2]A Quinn, [2]J King, [1]G Santis. 1 Kings College London, London, UK; 2 Guy's and St Thomas' NHS Foundation Trust, London, UK.