- 4 Janda S, Park K, Fitzgerald JM, *et al.* Statins in COPD: a systemic review. *Chest* 2009; 136: 734–743.
- 5 Anthonisen NR, Skeans MA, Wise RA, *et al.* The effects of a smoking cessation intervention on 14.5– year mortality: a randomised clinical trial. *Ann Intern Med* 2005; 142: 233–239.
- 6 Young RP, Hopkins RJ, Christmas T, *et al.* COPD prevalence is increased in lung cancer independent of age, sex, and smoking history. *Eur Respir J* 2009; 34: 380–386.
- 7 Dasari V, Gallup M, Lemjabbar H, *et al.* Epithelial-mesenchymal transition in lung cancer: is tobacco the “smoking gun”? *Am J Respir Cell Mol Biol* 2006; 35: 3–9.
- 8 Shintani Y, Maeda M, Chaika N, *et al.* Collagen 1 promotes epithelial-to-mesenchymal transition in lung cancer cells *via* transforming growth factor- β signalling. *Am J Respir Cell Mol Biol* 2008; 38: 95–104.
- 9 Yende S, Waterer GW, Tolley EA, *et al.* Inflammatory markers are associated with ventilatory limitation and muscle dysfunction in obstructive lung disease in well functioning elderly subjects. *Thorax* 2006; 61: 10–16.
- 10 Lee TM, Lin MS, Chang NC. Usefulness of C-reactive protein and interleukin-6 as predictors of outcomes in chronic obstructive pulmonary disease receiving pravastatin. *Am J Cardiol* 2008; 101: 530–535.

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From the authors:

We thank R. Young and co-workers for their constructive comments on our review [1] and we fully agree with them on the potential benefits of statins in chronic obstructive pulmonary disease (COPD) patients. The new information they provide about the potential for statins in reducing lung cancer is particularly important, as this is one of the most common causes of death amongst COPD patients, particularly in those with severe disease. The potential anti-inflammatory effects of statins highlighted in our review might account for the reduction in cancer, although it is possible that statins have additional effects on the signalling pathways which lead to epithelial cancers [1]. For example, lovastatin inhibits the growth of lung cancer cell lines *in vitro*, in part, by increasing activity of the tumour suppressor protein p53 and the cell-cycle check-point inhibitors p21^{WAF} and p27^{KIP} [2]. Although the pleiotropic effects of statins provide a convincing scientific

rationale for their potential benefit in COPD patients, a note of caution should be applied as the evidence for the benefits of statins is derived solely from observational studies that may have a selection bias. For example, patients with a better prognosis or who receive more intense medical attention may be more likely to be selected for statin therapy. What is needed now is a large, long-term, randomised placebo-controlled study investigating the addition of statins to usual therapy across a range of COPD disease severity; measuring disease progression, exacerbations, comorbidities and all-cause mortality. However, the high costs of such a study and the availability of generic statins mean that such a trial may be difficult to fund. In the meantime, further research investigating the molecular mechanisms of action of statins may be useful in identifying novel targets for the development of new treatments for COPD and its comorbidities [3]. It should also be remembered that there are differences between statins in the range of pleiotropic effects, thus it may not be appropriate to analyse all statins together.

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REFERENCES

- 1 Barnes PJ, Celli BR. Systemic manifestations and comorbidities of COPD. *Eur Respir J* 2009; 33: 1165–1185.
- 2 Maksimova E, Yie TA, Rom WN. *In vitro* mechanisms of lovastatin on lung cancer cell lines as a potential chemopreventive agent. *Lung* 2008; 186: 45–54.
- 3 Barnes PJ.: Future treatments for COPD and its comorbidities.: *Proc Am Thorac Soc* 2008; 857–864.

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Penetrating missile pulmonary embolisation

To the Editors:

We read with interest the review by JORENS *et al.* [1] highlighting the causes, diagnosis and treatment of nonthrombotic pulmonary embolism. Certainly, particulate material embolisation, especially from an iatrogenic source, is becoming more common. In fact, another well recognised source of embolisation of cyanoacrylate glue and lipiodol is following endoscopic injection of gastric or oesophageal varices [2, 3].

Embolisation of foreign material related to military trauma or weapons ammunition has become more topical with the presence of worldwide military conflicts in recent years [4]. The management of these patients can be very challenging given the presence of multiple injuries and contaminated wounds. Following adequate resuscitation, total body computed tomography (CT) imaging is often advocated to guide and prioritise treatment for the most life-threatening condition. The CT images in figure 1 show a 36-yr-old male injured by a