research. Since the work of AXFORD *et al.* [5], there has been very limited published work along this topic, despite the fact that the technical understanding of lung radiotherapy has advanced considerably. A model can be conceived in which dose and fractionation are designed to limit the syndrome of pneumonitis, and resulting in regional fibrotic shrinkage. We agree with the proposed mechanism of the authors, believe that this is an area which is ripe for clinical research and call for a broad discussion on this topic.

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From the authors:

We thank J.A. Murray and J. Lyons for their insightful comments on our recent case report dealing with dyspnoea relief in a patient with chronic obstructive pulmonary disease (COPD) and presumed lung cancer following radiation treatment [1]. Despite the impressive improvements in symptoms, exercise tolerance and pulmonary function in our patient, we cannot generalise our findings to the COPD population as a whole. Clinical experience has taught us that radiation-induced toxicity, when superimposed on the baseline respiratory impairment of COPD, may lead to further clinical deterioration in some patients. Thus, it is well established that radiation injury to the lungs initiates a cascade of events that create a self-sustaining cycle of chronic oxidative stress and inflammation [2]. Inflammation of the airways, pulmonary vasculature and lung parenchyma ultimately results in negative effects on pulmonary gas exchange (something we also observed in our patient). Nevertheless, some patients may, paradoxically, derive symptomatic improvement due to radiation-induced lung deflation [1, 3], but this response is likely to be variable given the vast pathophysiological heterogeneity of COPD.

There is a paucity of data on the effects of modern radiotherapy techniques on detailed respiratory physiology, exercise performance and respiratory symptoms in patients with combined COPD and lung cancer. Before we can even consider radiotherapy as a noninvasive alternative to lung volume reduction surgery, we need to refine selection criteria to help predict who is most likely to derive sustained symptomatic benefit. In order to do this, we need to be able to adequately characterise COPD phenotypes by comprehensive clinical evaluation, which includes detailed radiological and physiological assessment. This should be followed by a careful evaluation of both the short- and long-term effects of radiotherapy on respiratory symptoms, perceived health status, lung structure and function. We are therefore in agreement with J.A. Murray and J. Lyons that further study is required to determine whether radiotherapy-induced lung volume reduction can be considered in the management of selected patients with COPD and lung hyperinflation who report incapacitating dyspnoea.

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