From the authors:

We thank L. Mascitelli and colleagues for their interesting comments about the role of the renin–angiotensin system in severe chronic obstructive pulmonary disease. Our review [1] was intended to provide guidance on current established therapies rather than consider potential future treatment options and hence we did not include a comment relevant to this interesting but, as yet, unproven treatment approach. The only data we are aware of that directly addresses this were published recently in the *European Respiratory Journal* and did not find improvements in pre-specified outcomes after blockade of the renin–angiotensin system [2]. This does not preclude a role for this system in some settings in chronic obstructive pulmonary disease; however, it did make us cautious about commenting specifically on the role of these drugs in a review of current disease management.

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STATEMENT OF INTEREST

None declared.

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Massive haemoptysis: the definition should be revised

To the Editors:

Massive haemoptysis represents one of the most challenging conditions in clinical practice. The condition is potentially lethal and, therefore, warrants clear understanding and precise definition. The definition of massive haemoptysis has not been completely agreed upon and varies widely in the literature. It is unfortunate that almost all previous definitions of massive haemoptysis relied only on the volume of expectorated blood. The use of expectorated blood volume alone to define massive haemoptysis is often misleading and confusing for three main reasons. First, no cut-off volume has been agreed upon in the literature. While AMIRANA *et al.* [1] proposed an amount of 100 mL of expectorated blood in 24 h to define massive haemoptysis, COREY and HLA [2] defined massive haemoptysis as expectoration of $\geqslant$1,000 mL of blood over 24 h. In the middle of the spectrum we find other studies that use 200 mL [3], 240 mL [4], 500 mL [5] or 600 mL [6] as a cut-off volume for the definition of massive haemoptysis. Secondly, in real practice, the quantification of haemoptysis is often difficult and, from a clinical point of view, such criteria are not useful [7]. In many instances the amount of expectorated blood may be exaggerated by patients. Furthermore, in a majority of patients, quantification of expectorated blood volume may underestimate the overall amount of blood loss because the volume of blood engulfing the involved lobes or lungs is not quantified and may be significant [8]. Thirdly, morbidity and mortality in patients with haemoptysis depend on not only the volume of expectorated blood but also the rate of bleeding, the ability of the patient to clear blood from the airways and the extent and severity of any underlying lung disease [9]. The confusion created by the arbitrary use of the volume of expectorated blood to define massive haemoptysis has led other authors to consider the magnitude of effects (namely airway obstruction and hypotension) as the defining factors [10, 11].

I feel that the confusion will persist if we continue to use the word ''massive''. The term ''massive haemoptysis'' is a general term that was originally selected to describe the magnitude of life-threatening bleeding. Over time, this term became a loosely applied descriptor for the condition as the word ''massive'' necessitates the identification of a specific volume of blood. Therefore, in order to precisely define this serious condition, we should move away from using the word massive. The term ''life-threatening haemoptysis'' may provide a fascinating and rich understanding of the condition. Thus, life-threatening haemoptysis may be defined as any haemoptysis that: 1) is >100 mL in 24 h; 2) causes abnormal gas exchange/airway obstruction; or 3) causes haemodynamic instability. The cut-off volume of 100 mL per 24 h has been selected because it is the smallest amount of haemoptysis that is reported in literature to threaten the life of the patient.

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STATEMENT OF INTEREST

None declared.

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Mortality predictive capacity of the 6-min walk distance

To the Editors:

In a recent issue of the *European Respiratory Journal* we read with interest the article by COTE *et al.* [1] on the capacity of the 6-min walk distance to predict mortality in chronic obstructive pulmonary disease patients. We noted a striking difference between the cut-off value reported by COTE *et al.* [1], corresponding to 350 m, and a cut-off value previously reported as the mortality predictor in patients with idiopathic pulmonary fibrosis, which was 207 m [2].

Even considering the obvious diversities between the two diseases, such a difference is quite surprising. The two populations cannot be compared by forced expiratory volume in one second values because such data are lacking in the article by LEDERER *et al.* [2], but in both studies there are the data concerning forced vital capacity (FVC). Thus, we are permitted to compare the lung function by this parameter, where a remarkable difference can also be found: COTE *et al.* [1] reported a mean FVC value of 72.7±21% predicted, whereas LEDERER *et al.* [2] reported a mean value of 51±17% pred. The mortality rates were approximately two-fold in the study by COTE *et al.* [1] and four-fold in the study by LEDERER *et al.* [2]. In particular, the mortality rates recorded by LEDERER *et al.* [2] were significantly lower in the fourth quintile, patients walking 314–395 m, which included the allocated 350 m cut-off reported by COTE *et al.* [1].

As physicians currently working in a pulmonary rehabilitation setting we would like to learn more about the prognostic significance of the distance walked during the 6-min walk distance test in different respiratory diseases, which was not discussed in the study by COTE *et al.* [1].

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STATEMENT OF INTEREST

None declared.

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From the authors:

We have read with interest the observations by G.G. Riario Sforza and C. Incorvaia regarding the differences in the threshold values used to predict mortality that were provided by LEDERER *et al.* [1] for patients with idiopathic pulmonary fibrosis (IPF) and those that were reported for patients with chronic obstructive pulmonary disease (COPD) [2].

First, G.G. Riario Sforza and C. Incorvaia correctly point out that LEDERER *et al.* [1] quote a threshold of 207 m as being more specific for waiting-list mortality at 6 months but, in the discussion, LEDERER *et al.* [1] also state that the 350-m cut-off remained more sensitive for waiting-list mortality, a value that is very close to the one we reported [2].

Secondly, G.G. Riario Sforza and C. Incorvaia show surprise about the difference in mortality between the patients with IPF and those with COPD in our study. This has several likely explanations. To begin with, the patients reported by LEDERER *et al.* [1] were all on the waiting list for transplantation due to IPF, whereas ours were patients with different severity of COPD attending regular clinics, that is to say, healthier [2]. Furthermore, LEDERER *et al.* [1] do not mention the use of corticosteroids or immunosuppressants in their patients but it is very likely that patients with such severity of IPF may have been on agents capable of inducing muscle dysfunction. Finally, patients with COPD, even on transplant lists, have a better prognosis than patients with IPF or other underlying disease, thereby making direct comparisons difficult.