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From the author:

Teramoto and colleagues have completely misunderstood the purpose of the ERS Task Force on diagnosis and management of chronic cough [1]. The document deals with patients who have had a cough for >8 weeks. It is not about patients who can't cough. To suggest in their opening paragraph that we neglect cough in the elderly is simply disingenuous. We deliberately separated chronic cough in children from that in adults since the aetiology is different. However, in adults the causes and treatment of chronic cough are not age related and the elderly were frequent attendees in the 13 studies quoted in table 1 which presents the accumulated experience of specialist cough clinics [1].

Decreased cough and aspiration are important clinical problems but they were not the subject of our discussions. Clearly neurological illness [2, 3] and anatomical abnormality [4] can increase the likelihood of aspiration but this is neither age specific nor relevant to clinicians dealing with patients who present with isolated chronic cough.

Finally, an important function of documents such as the Task Force report is to provide a balanced overview of the literature. Teramoto and colleagues seem to have concentrated largely on their own work, which perhaps goes some way to explain the current debate.

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The hepatopulmonary syndrome: NO way out?

To the Editors:

The hepatopulmonary syndrome (HPS) is defined by the triad of chronic liver disease, abnormal pulmonary gas exchange (low arterial oxygen tension (P_{a,O_2}) and transfer factor of the lung for carbon monoxide), and intrapulmonary vascular dilatation [1]. The recent editorial on HPS [2] suggests that ''hunting endogenous vasodilators that reduce pulmonary vascular tone logically became a sound strategy for those whose quest was to unravel the missing 'molecular' link between the diseased liver and the affected lung''. But, is this strategy actually so logical? The key feature of the intrapulmonary vascular dilatation in HPS is the intrapulmonary shunt shown physiologically by a low P_{a,O_2} after 100% oxygen breathing, and anatomically by the passage of radiolabelled albumin macroaggregates (20–60 μm in diameter), or echobubbles, through the pulmonary capillary bed [3]. The striking feature pathologically is gross dilatation of capillaries in the alveolar septum, diameters of 100 μm, as compared with the normal 7–15 μm being described [4]. Is it likely that endogenous vasodilators are responsible for ''relaxing'' alveolar capillaries to such an extent? Of course, endogenous vasodilators may play a part in ''remodelling'' these capillaries.

With regard to pulmonary gas exchange, two factors seem to operate in severe hepatopulmonary syndrome: 1) a