

CORRESPONDENCE

Chronic lung disease and the perception of breathlessness: clinical perspective

To the Editor:

I was very impressed by the timely review entitled "Chronic lung disease and the perception of breathlessness: clinical perspective" [1].

However, I do not agree with some aspects of the conclusions. The authors concluded that "there is no convincing evidence that any of the drugs used in this endeavour have any specific effect in modifying the perception of breathlessness". We have recently reported that an inhaled anticholinergic drug, oxitropium bromide, reduces the sensation of dyspnoea during exercise in patients with chronic obstructive pulmonary disease (COPD) [2]. Similar effects of the drug on breathlessness have also been investigated by others [2, 3]. NOSEDA *et al.* [4] have reported that dyspnoea on exercise in COPD patients is closely related to inspiratory flow, suggesting that increased inspiratory flow by anticholinergic inhalation may lessen the sensation of dyspnoea with exercise.

In addition, a combination of decreased functional residual capacity with increased expiratory flow, following inhalation of the drug, may increase the diaphragm fibre length, allowing the diaphragm to generate more pressure for a given neural output, and subsequently contributing to reduction of dyspnoea in COPD.

Although the respiratory work during exercise following the drug administration may be decreased due to improvements of both expiratory flow and diaphragmatic motion, the anticholinergic drug may have other effects on the sensation of dyspnoea relevant to parasympathetic nervous function. The involvement of

parasympathetic nervous function and vagal reflexes in the sensation of dyspnoea in COPD patients has not been clearly assessed. However, there is a possibility that blocking the parasympathetic activity may, at least in part, contribute to relief of breathlessness. Therefore, the inhaled anticholinergic drug may be a candidate drug for modifying the perception of dyspnoea in COPD patients.

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References

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REPLY

From the authors:

We thank Dr. Teramoto for his kind remarks regarding our paper "Chronic lung disease and the perception of breathlessness: a clinical perspective" published recently in the Journal (*Eur Respir J* 1994; 7: 1342-1349).

The literature on the use of drugs to reduce breathlessness is peppered with many reports demonstrating a reduction in the sensation of breathlessness following the use of various pharmacological agents. The use of oxitropium has received attention in the last year or two as Dr Teramoto comments. We stated "there is no convincing evidence that any of the drugs used in this endeavour have any specific effect modifying the perception of breathlessness", because we wished to make the point that we believe that no study to date has demonstrated a reduction in breathlessness, without changing the mechanics or control of breathing in some way.

In the studies quoted by Dr Teramoto, the use of oxitropium has increased the forced expiratory volume

in one second (FEV₁) as one would expect. It is difficult, therefore, to be absolutely sure that oxitropium, or any other drug for that matter, has been responsible for changing the sensation of breathlessness directly, either at the peripheral or central processing level. In other words, the way in which it is perceived independent of changes in the mechanics and control of breathing. It seems more likely that, in these cases, the sensation of breathlessness has been reduced secondary to changes in airway calibre, ventilatory drive or work. Certainly, we would agree that many pharmacological agents, including oxitropium, anxiolytics and opiates, will reduce the sensation of breathlessness, and may be useful in selected cases in clinical practice for this reason. However, the reduction in breathlessness occurs only at the expense of changes in mechanics and control of ventilation.

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