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Endocrinological derangement in COPD

To the Editors:

With the greatest interest I have read the comprehensive review “endocrinological derangements” in chronic obstructive pulmonary disease (COPD) by LAGHI *et al.* [1]. Arriving at the last page, where the authors discussed the renin–angiotensin–aldosterone system, I have read: “the presence of sympathetic neuropathy...” and “...the possibility that autonomic neuropathy is caused by intraneural hypoxaemia”.

In COPD and other diseases such as heart failure and obstructive sleep apnoea the concept of autonomic nervous system dysbalance with increased sympathetic activation and reduced parasympathetic activity has been recognised [2–6]. Recent work published in this journal on normoxic COPD patients demonstrated that slow breathing reduces sympathoexcitation and improves baroreflex sensitivity, raising possible therapeutic implications concerning the autonomic nervous system [7]. There is a strong mutual interaction between angiotensin and the sympathetic nervous system [8]. This interaction may partly account for the activation of the renin–angiotensin–aldosterone system LAGHI *et al.* [1] competently described in COPD patients.

Interestingly, work published in the same volume as the review on endocrinological derangements in COPD demonstrated marked alterations in cardiac autonomic control in patients with pulmonary artery hypertension [4]. The authors and an adjoining editorial discussed various causes of the observed findings, such as sympathetic overactivity related to an overactive chemoreflex and baroreflex, increased filling pressure and blood gas alterations [4, 6].

Thus, there is growing evidence demonstrating a dysfunction of the autonomic nervous system in COPD patients [2]. This evidence stems from evaluating the baroreflex sensitivity, heart rate variability and muscle sympathetic nerve activity, and is corroborated by research on other diseases such as heart failure, sleep apnoea and pulmonary hypertension [3, 6]. This evidence can not be readily explained by intraneuronal hypoxaemia. However, in very severe COPD patients and

long-standing severe hypoxaemia without supplemental oxygen alterations of the peripheral nervous system may ensue.

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