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STATEMENT OF INTEREST

None declared.

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Excessive daytime sleepiness in patients with sleep-disordered breathing

To the Editors:

I read with interest the article by MEDIANO *et al.* [1] about excessive daytime sleepiness (EDS) in patients with obstructive sleep apnoea (OSA). In order to investigate the determinants of EDS in OSA patients, the authors compared the demographics and polysomnographic variables of two groups of OSA patients who had a similar apnoea/hypopnoea index (AHI), age and body mass index (BMI). Nocturnal hypoxaemia was reported to be a major determinant of EDS in patients with OSA.

I would like to point out that the possible coexistence of obesity hypoventilation syndrome (OHS), which is a known cause of EDS, was not ruled out in the studied group [2]. HIDA *et al.* [3] have shown that OHS patients have a significantly higher Epworth Sleepiness Scale score compared with pure OSA patients matched for age, BMI and AHI. It is well documented that 70–90% of patients with OHS have predominantly obstructive events during sleep [4]. Therefore, to rule out coexisting OHS, measurement of daytime blood gases and nocturnal monitoring of carbon dioxide arterial tension, as well as the observation for oxygen desaturation during sleep that is not explained by apnoeic or hypopnoeic events, become imperative.

MEDIANO *et al.* [1] reported that patients with EDS had lower nocturnal oxygen saturation and mean arterial saturation. However, the desaturation index was not reported. As patients with hypoventilation may have frequent episodes of desaturation without obstructive apnoeic or hypopnoeic events, it

becomes important to compare the desaturation index in both groups. The group with EDS was found to have a longer apnoea duration, which the authors attributed to intrinsic differences in arousal thresholds or increased sleep pressure [1].

It is known that patients with OHS exhibit periods of sustained sleep hypoxaemia [5]. Sustained hypoxaemia has been suggested as a cause of delayed arousal from sleep [6], which may explain the longer apnoea duration in the EDS group. The fact that patients with and without EDS had a similar age, BMI and AHI does not imply that they have the same underlying sleep-disordered breathing [1]. A recent large study [7] reported no difference in AHI between OHS patients and pure OSA patients matched for BMI. In another recently published study [8], patients with OHS and hypercapnic respiratory failure were compared with patients with pure OSA matched to each subject using BMI, age and AHI measured at the time of diagnosis. Compared with pure OSA patients, OHS patients spent more time with arterial oxygen tension <90%, had lower mean saturation, a higher desaturation index and their lowest recorded oxygen saturation was significantly lower [8]. We agree with MEDIANO *et al.* [1] that nocturnal hypoxaemia may play a role in the pathogenesis of EDS; however, we feel that OHS was not ruled out as a cause of EDS in the studied group.

Data suggest that obesity hypoventilation syndrome is both under recognised and underdiagnosed [2]. To overcome these problems, the serum bicarbonate level should be measured in patients with sleep-disordered breathing, excessive daytime

sleepiness and significant desaturation. An elevated serum bicarbonate level suggests the presence of obstructive sleep apnoea and should warrant measurement of daytime blood gases [9].

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STATEMENT OF INTEREST
None declared.

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Pulmonary embolism at home

To the Editors:

We wish to express serious concern about an article in a recent issue of the *European Respiratory Journal*, in which DAVIES *et al.* [1] reported on an outpatient treatment study in patients with acute pulmonary embolism (PE). In this study [1], the authors identified criteria for the safe discharge of PE patients, and these criteria were validated in a series of 157 patients with confirmed PE. At the end of acute low-molecular-weight heparin treatment, there were no in-hospital adverse events (thromboembolic recurrences, bleeding complications or deaths).

First, some variables that have been associated with prognosis in patients with PE were not considered by the clinicians responsible for patient care. Data from large observational studies of PE have confirmed systolic arterial hypotension as the most significant prognostic indicator of outcome in PE patients [2, 3], but this was not an explicit exclusion criterion in the study of DAVIES *et al.* [1]. Conversely, deep venous

thrombosis has been associated with prognosis in one study [4], but not in another [5].

Secondly, the number of adverse events in both phases of the study was extremely low as compared with any treatment trial [6] or PE registry [3]. For example, the 30-day mortality rate of the PE severity index low risk strata has ranged from 1.4 [7] to 3.1% [8].

The present authors validated this score in a prospective series of 805 consecutive patients with an objective diagnosis of acute symptomatic PE. Of these, 350 patients were excluded due to the presence of proximal deep venous thrombosis. Additional patients were excluded due to active bleeding (n=19), previous PE (n=44), hypotension (n=17), hypoxaemia (n=162), chronic obstructive pulmonary disease (n=9), congestive heart failure (n=8) and haemodynamic instability (n=6). The prevalence of adverse events during the first 10 days of treatment in the remaining 190 (24%) patients is shown in table 1.

We share the idea that a highly selected group of pulmonary embolism patients could be treated at home. However, data from the literature cause us to exercise caution when interpreting the results of this study [1]. We are concerned that an excessively optimistic message diverges from the complex reality of pulmonary embolism patients.

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TABLE 1 Prevalence of adverse events during the first 10 days after diagnosis	
	Prevalence (95% CI)
Death	4.7 (1.1–8.2)
Venous thromboembolism recurrence	1.0 (0.7–2.7)
Major bleeding	2.1 (0.3–4.5)
Data are presented as %. CI: confidence interval.	