To the Editor: Xie et al. analyzed warfarin doses in pulmonary embolism (PE) and its adverse effects in patients with obstructive sleep apnea hypopnea syndrome (OSAHS). The key findings were that OSAHS patients required a significantly higher dose of warfarin than their non-OSAHS counterparts (4.73 mg vs. 3.61 mg, \( P < 0.001 \)) and that PE recurrence was higher in OSAHS than in non-OSAHS groups after withdrawal of warfarin (21.43% vs. 6.78%, \( P = 0.047 \)). They conclude that OSAHS patients may present with hypercoagulation and relatively high risk of recurrence of PE after cessation of 6-month warfarin treatment. We have read this with great interest.

However, we consider that there are some controversial aspects that can influence the results and that must be taken into account: Firstly, there is scarce information such as: Inability to control patient’s food intake (leading to the intake of certain foods that can influence warfarin levels), poor compliance, concurrent medications that could decrease the absorption or increase the clearance of warfarin, and consumption of diet rich in Vitamin K. These are the major reasons for warfarin resistance that can influence the results described by authors. Additionally, there is no information if patients received any education program to increase warfarin dosage.

Secondly, gender difference is an important aspect. There is a higher percentage of men in the OSAHS group, leading to higher body mass index, which could bias the final results. The OSAHS group also has more patients with hypertension, which can increase the risk of PE. In this study, OSAHS patients have a high prevalence of comorbidities associated with PE development. Hence, it would be interesting to evaluate whether OSAHS remain an independent risk factor for PE after covariates adjustment.

Thirdly, there is a prevalent number of nonadherence of OSAHS individuals to home mechanical ventilation with positive airway pressure (PAP) that could influence the stability of OSAHS and differences among the two groups. Several studies have shown that continuous PAP (CPAP) therapy seems to be related to reduction in mortality risk and cardiovascular events, appearing to be protective in older and male severe obstructive sleep apnoea (OSA) patients.\(^{[3,4]}\) It would be essential to ensure the adhesion of individuals to PAP, to assess if this procedure alone is enough to decrease the hypercoagulable state of patients and thereby lower the risk of PE recurrence.

Fourthly, some known risk factors for PE have not been investigated, such as the presence of hemostatic changes and/or inherited thrombophilia, and smoking habits.

Therefore, whether this relationship is casual or a consequence of common risk factors to OSAHS and PE is not totally clear. Part of the answer for the association may lie in common pathways that promote both OSAHS and PE.\(^{[5]}\)

We consider that it would be interesting to conduct similar studies with more characterized groups and better OSAHS control, to assess the maintenance of hypercoagulation state, higher warfarin doses need, and higher risk of PE recurrence in these patients.

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Conflicts of interest
There are no conflicts of interest.
CPAP reduces hypercoagulability, as assessed by thromboelastography, in severe obstructive sleep apnoea. The association between obstructive sleep apnea hypopnea syndrome (OSAHS) and pulmonary embolism (PE) has already been revealed, although the pathophysiological mechanism is far from being clear. The major concern is that whether OSAHS is an independent risk factor of PE, or just a companion of other diseases which may lead to thrombotic lesions, such as diabetes, hypertension, and heart disease. Whether CPAP, the golden strategy to treat OSA, will contribute to the prophylaxis of thrombotic issues is a super interesting topic, which is currently under investigation. To the best of our knowledge, CPAP has been proved to possess the potential to reduce the risk of thrombotic issues, but the effect is not yet clear. Therefore, more thrombosis-related research is needed. CPAP reduces hypercoagulant state in patients with OSAHS.

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The international normalized ratio and warfarin dose is influenced by many factors, such as medication, which is a challenge for both researchers and clinicians. Exclusion of all potential influential factors is not logistically feasible during the follow-up, although the pathophysiological mechanism is far from being clear. The major concern is that whether OSAHS is an independent risk factor of PE, or just a companion of other diseases which may lead to thrombotic lesions, such as diabetes, hypertension, and heart disease. Whether CPAP, the golden strategy to treat OSA, will contribute to the prophylaxis of thrombotic issues is a super interesting topic, which is currently under investigation. To the best of our knowledge, CPAP has been proved to possess the potential to reduce the risk of thrombotic issues, but the effect is not yet clear. Therefore, more thrombosis-related research is needed.

In this study, even though we did not find a significant increase in the risk of PE in CPAP users, the trend indicates a negative impact of CPAP on PE. Whether this trend is just a false-positive result or a true effect needs further studies. The major concern is that whether OSAHS is an independent risk factor of PE, or just a companion of other diseases which may lead to thrombotic lesions, such as diabetes, hypertension, and heart disease. Whether CPAP, the golden strategy to treat OSA, will contribute to the prophylaxis of thrombotic issues is a super interesting topic, which is currently under investigation. To the best of our knowledge, CPAP has been proved to possess the potential to reduce the risk of thrombotic issues, but the effect is not yet clear. Therefore, more thrombosis-related research is needed.

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