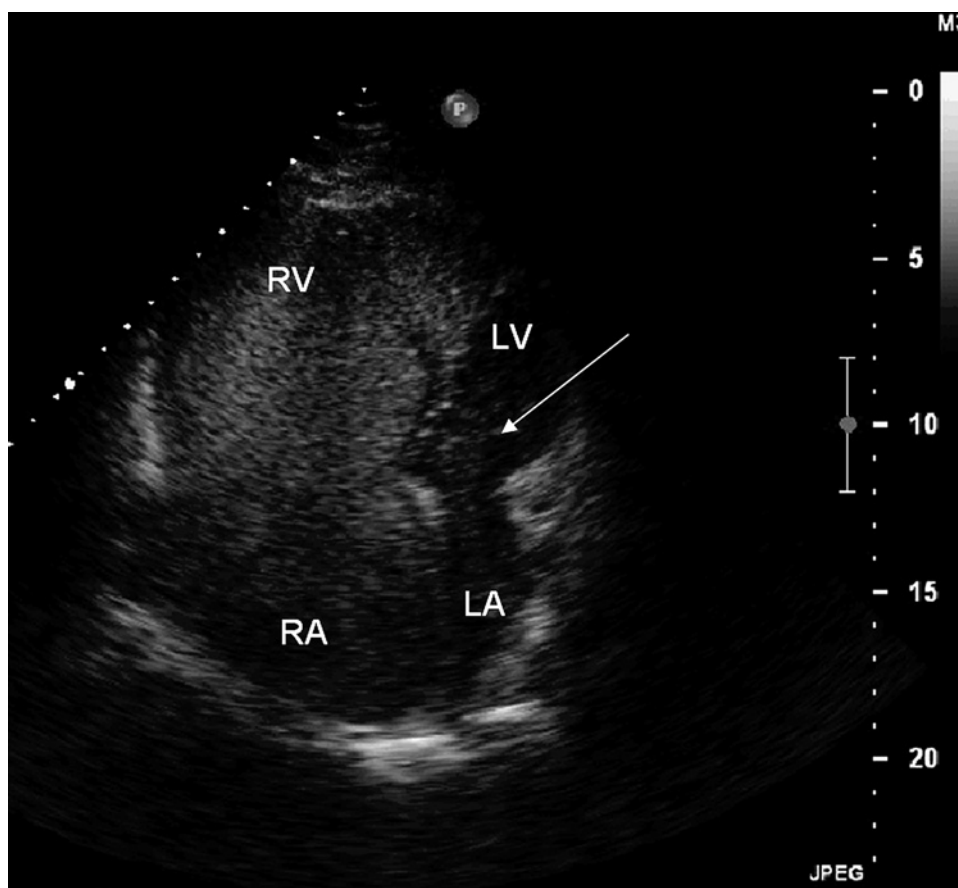**Figure 1** Saline-contrast transthoracic echocardiogram showing right-to-left shunt. LA, left auricle; LV, left ventricle; RA, right auricle; RV, right ventricle.

in ventilation—perfusion heterogeneity, as seen in patients treated with a high dose of intravenous epoprostenol in whom IPS and severe hypoxaemia occurred.[5] In these cases, an accurate diagnosis and drug down-titration or discontinuation allowed a rapid recovery of the symptoms. In conclusion, sildenafil may be associated with development of IPS and hypoxaemia in PAH patients. In these cases, an SC-TTE should be performed in order to disclose previously undiagnosed IPS.

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Risk stratification in pulmonary embolism: an algorithmic tool approach

It is with much interest we read the article by Jiménez *et al*[1] and the accompanying editorial[2] Focusing investigation on patients with symptomatic pulmonary thromboembolism (PTE) but who are normotensive at presentation, it reminds us that work still needs to be undertaken for the 95% of patients (including the 15% with submassive disease) who remain haemodynamically stable and excluded from thrombolysis, if current guidelines are followed.[3] Anecdotally, with the increased use of CT pulmonary angiography, clinicians more readily visualise thrombus burden and, despite the lack of scientific evidence, consider thrombolytic therapy ahead of heparin even with submassive PTE. Although the mortality benefits from thrombolysis in this group are debatable, it does help improve the right ventricular function more rapidly than anti-coagulation alone, reducing complications of chronic thromboembolic pulmonary hypertension[4] The paper by Jiménez[1] recognises and evaluates the prognostic tools currently being used in risk analysis, reminding us that the use of a two-test strategy has a higher specificity and positive predictive value of pulmonary embolism-related death than any single test itself, whether using cardiac biomarkers such as troponin (cTnI/T), cardiac ECHO or lower extremity complete compression ultrasound. Importantly, it also clarifies that although there is a trend to better evaluation with all three tests used together, the difference comparing a two-test approach with a three-test approach is not statistically significant.
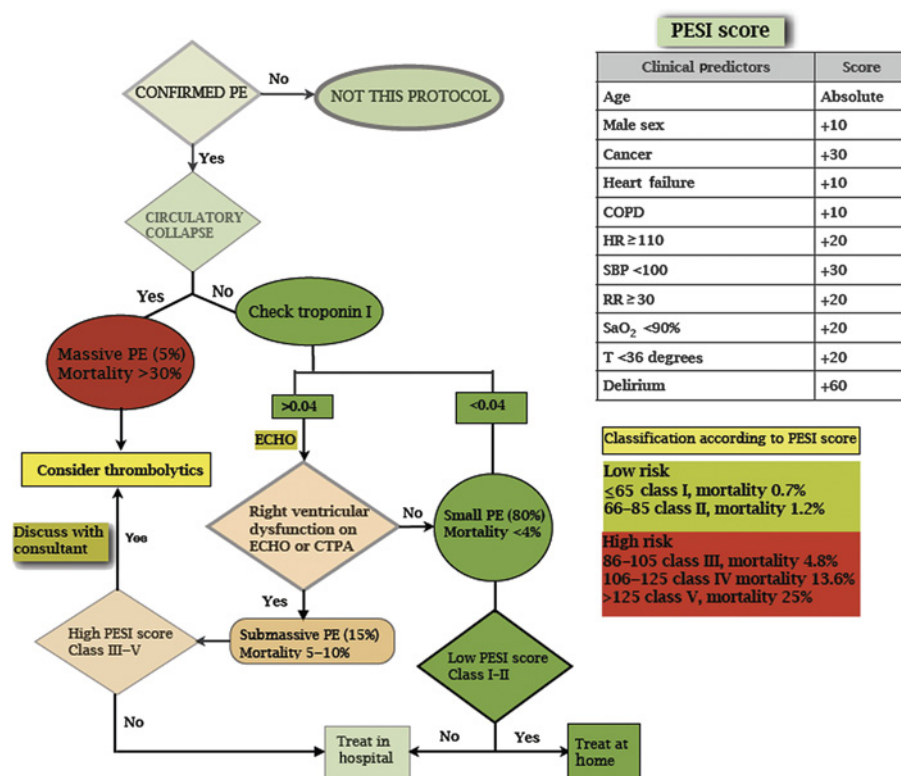
PESI score

Clinical predictors	Score
Age	Absolute
Male sex	+10
Cancer	+30
Heart failure	+10
COPD	+10
HR ≥110	+20
SBP <100	+30
RR ≥30	+20
SaO$_2$ <90%	+20
T <36 degrees	+20
Delirium	+60

Classification according to PESI score

Low risk
≤65 class I, mortality 0.7%
66–85 class II, mortality 1.2%

High risk
86–105 class III, mortality 4.8%
106–125 class IV mortality 13.6%
>125 class V, mortality 25%

Figure 1 Pulmonary embolism (PE) protocol. COPD, chronic obstructive pulmonary disease; HR, heart rate; PESI, pulmonary embolism severity index; RR, respiratory rate; SaO$_2$, saturation of oxygen; SBP, systolic blood pressure; T, temperature.

In an attempt to identify patients who can be appropriately managed in a semi-outpatient (after day 2) ambulatory manner and, at the other extreme, patients for active thrombolysis, we have used the enclosed protocol (figure 1) where individual components are based on published evidence but not necessarily guidelines. More specifically it incorporates the pulmonary embolism severity index (PESI)[5] in the two-test approach and gives more confidence, particularly when thrombolysis becomes an option in those with high severity (class IV and V) scores. Using the initial troponin, as a sensitive but not specific triage tool addressing right heart strain, reduces the overuse of ECHO and adds to the value of the pathway as there will still be patients who can be discharged diagnosed with a small PTE and low PESI score (class I and II) and therefore low risk of mortality. The future may see further validated use of highly sensitive cardiac troponin (hsTnT) and CT assessment of the right heart, but it is likely that a two-test approach will be maintained in risk stratification.

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Authors' response

We thank Dr Ahmad and colleagues for their thoughtful comments.[1] Haemodynamic instability has significant prognostic implications for patients diagnosed as having acute pulmonary embolism (PE), and guidelines generally recommend consideration of treatment with thrombolytic agents.[2] [3] At the other end of the spectrum, different studies suggest that risk stratification models (particularly the Pulmonary Embolism Severity Index (PESI) and the simplified PESI) may accurately identify patients at low risk of death within the first 3 months after the diagnosis of PE.[4] [5] One study found that the addition of troponin testing to the PESI did not increase the prognostic value of the PESI for the identification of low-risk patients who might benefit from a shortened hospital stay or outpatient therapy.[6] Although recent data suggest that the use of a highly sensitive troponin T (hsTnT) assay may improve the risk stratification of PE,[7] future studies should address the usefulness of hsTnT and risk stratification models, alone or in combination, for identifying low-risk patients who can be discharged early from the hospital and treated as outpatients. Our recent study adds to the body of evidence that a combination of cardiac biomarkers, echocardiographic findings and lower limb ultrasound testing are useful for fine-tuning risk stratification in the subgroup of intermediate-risk patients with acute symptomatic PE.[8]

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