

**Figure 1** Pulmonary embolism (PE) protocol. COPD, chronic obstructive pulmonary disease; HR, heart rate; PESI, pulmonary embolism severity index; RR, respiratory rate; SaO<sub>2</sub>, saturation of oxygen; SBP, systolic blood pressure; T, temperature.

In an attempt to identify patients who can be appropriately managed in a semioutpatient (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)<sup>5</sup> 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. Haemodynamic instability has significant prognostic impli-

cations for patients diagnosed as having acute pulmonary embolism (PE), and guidelines generally recommend consideration of treatment with thrombolytic agents.<sup>2</sup> 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, future studies should address the usefulness of hsTnT and risk stratification models, alone or in combination, for identifying lowrisk 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

## David Jiménez, on behalf of all coauthors

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# Authors' response

I appreciate the interest of Dr Ahmad *et al*<sup>1</sup> in the article by Jiménez *et al*<sup>2</sup> and the accompanying editorial<sup>3</sup> in which it has been pointed out that the use of echocardiography, laboratory findings and venous ultrasonography should be encouraged in patients with suspected high-risk pulmonary embolism (PE), and management decisions should be taken on all collected data on a case-bycase basis. Due to the high hospital mortality in patients with PE, it is important to select those at the highest risk, who cannot be treated in an outpatient setting and require close monitoring or even more aggressive therapy.<sup>4 5</sup>

Dr Ahmad *et al* suggest using their protocol, where individual components are based on published evidence, in order to attempt to identify patients who can be appropriately managed in a semi-outpatient ambulatory manner and, at the other extreme, patients for active thrombolysis. I agree that the use of initial troponin as a sensitive but not specific triage tool addressing right heart strain would add to the value of prognostic assessment of

PE. I also agree that a further validated use of highly sensitive cardiac troponin (hsTnT) would be desirable in the future. Of course a right ventricle (RV)-sensitive troponin would be preferable.

However, I would like to clarify the term 'overuse' of echo. 'Reduce the overuse' does not mean 'no use' but a 'better use' of echocardiography. Although the assessment of RV function can be challenging even with good acoustic echo windows as well as other alternative techniques such as CT scan, RV dysfunction and dilatation have been reported as robust prognostic factors in acute PE with normal or abnormal troponins. The particular approach (echo or CT) may depend on the available hospital resources. Moreover, while CT provides information on RV dilatation only, echocardiography gives some information on contractility also. To date, we don't have a uniformly accepted definition of the criteria for echocardiographically detected RV dysfunction to give a conclusive answer on the prognostic significance of decreased RV performance in haemodynamically stable patients with PE. Nonetheless, available echocardiographic parameters of RV dysfunction can be carefully assessed and interpreted to judge a possible RV involvement. Furthermore, with recent advances in Doppler and tissue Doppler echocardiography, new methods for measuring regional and global RV function or contractility have been suggested<sup>6</sup> and may enter the clinical routine in the future.

We realise that a combination of imaging modalities with cardiac biomarkers may optimise risk stratification by a two-test or three-test approach. More sophisticated biochemical assays of troponin hopefully will come, but in pulmonary heart disease we certainly cannot neglect a detailed and reliable morphofunctional RV assessment.

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