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Authors' response

I appreciate the interest of Dr Ahmad *et al*¹ in the article by Jiménez *et al*² and the accompanying editorial³ 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-by-case 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.^{4 5}

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 suggested⁶ 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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