which will recover spontaneously over the following days and weeks; however, there seems to be a fluid transition between repetitive stunning and classic myocardial hibernation, and the ideal timing of the test is unclear. Similar to the detection of ischaemia, the most crucial issue is local availability and expertise, again outweighing the differences in diagnostic accuracy of the individual test.

Supplementary material
Supplementary material is available at European Heart Journal online.

References
The list of references is available in the online version of this paper.

Fatal fulminant myocarditis associated with novel influenza A (H1N1) infection
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A 51-year-old male presented to a local hospital with deteriorating general fatigue. The patient had reportedly been well until 2 days earlier when he began to have general fatigue. Because the blood pressure was as low as 70/40 mmHg, he was transferred to our hospital. Laboratory data showed WBC of 25 500/μL with 93% of neutrophil, creatine phosphokinase of 434 U/L, and Troponin I of 0.16 ng/mL. An electrocardiogram revealed sinus rhythm, poor R progression, and low voltage. Transthoracic echocardiography (TTE) revealed left ventricular dysfunction with an ejection fraction of 30% and marked thickening of the left ventricular wall (Panel A). A TTE performed 4 months prior to the admission had shown normal thickness of the wall and an ejection fraction of 62% (Panel B). He was treated with aggressive pharmacological support and mechanical circulatory support. However, he underwent cardiac arrest 8 h and 30 min after arrival in our hospital. At autopsy, the left ventricle showed concentric hypertrophy and diffuse patchy haemorrhage (Panel C). There was no significant stenosis in epicardial coronary arteries. A microscopic examination of the myocardium revealed infiltration of the inflammatory cell consisting of CD3-positive lymphocytes, macrophages, and occasional neutrophils (Panel D). Reverse transcriptase–polymerase chain reaction using autopsy specimen showed that influenza A (H1N1) M2 gene unique to novel influenza was positive in the left ventricle, the right ventricle, and the left lung (Panel E). In conclusion, we report a case with fluminant myocarditis associated with novel influenza A (H1N1) who had no response to intensive pharmacological and mechanical supports.