High-sensitivity troponin is—per definition—highly sensitive to detect all sorts of myocardial injury. This does not necessarily mean that permanent damage has been done to the myocyte. It is known that the troponin level may well be elevated after exceptional physical exercise, like in marathon runners. Of course, long-term prognosis will not be compromised in these athletes, but also in non-coronary conditions like aortic valve or mitral valve disease, elevated troponin values were detected, with different implications on prognosis (table 1).

A variety of studies addressed the frequent finding of elevated biomarker values following coronary angiography and percutaneous interventions with or without stent deployment in patients with stable coronary artery disease. Potential mechanism of periprocedural infarcts are (1) side branch occlusion, (2) distal embolisation, (3) prolonged or multiple balloon inflation, (4) coronary dissection with slow flow or (5) microthrombi and no reflow.

However, the definition of periprocedural myocardial injury varies among different authors and the interpretation of these data may prove difficult. In particular, as an isolated troponin elevation might have less prognostic impact if compared with true myocardial necrosis with a creatine kinase MB (CK-MB) rise. Tricoci and colleagues compared the prognostic impact of Creatine-kinase-MB (CK-MB) and troponin rise. Interestingly enough, the mortality risk of a CK-MB rise >3× upper limit of normal (ULN) was comparable to a cTroponin rise >60× ULN.

In the interventional community, it is widely accepted that an isolated minor troponin rise following percutaneous coronary procedures will not affect prognosis. Therefore, no guidelines recommend routine evaluation of biomarkers in patients with an uneventful postinterventional course. However, the European Society of Cardiology defined the percutaneous coronary intervention (PCI)-associated myocardial ischaemia as a Type 4a infarct. The Type 4a infarct is characterised by an elevation of troponin values >5×99th percentile ULN in patients with normal baseline values and (1) symptoms suggestive of myocardial ischaemia, (2) new ischaemic ECG changes or new left bundle branch block, (3) angiographic loss of patency of a major coronary artery or a side branch or persistent flow or no flow or embolisation or (4) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality.

In the present issue of ‘Open Heart’, Hamaya and colleagues investigate the impact of high-sensitivity I troponin elevation. Their study included 538 stable patients who underwent a diagnostic coronary angiogram. The authors identified patients with minor procedure-related myocardial necrosis and those with major procedure-related myocardial necrosis with troponin elevation >3–5x ULN. The troponin was measured just before the angiogram and 18–24 hours postprocedure. Importantly, in patients with significant coronary artery disease, any revascularisation procedure was rescheduled for a second session.

The main findings of this study were that patients with troponin elevation were older, female, had previous coronary interventions and a longer procedural time. Patients with major elevations of troponin had higher levels of N-terminal -Brain Natriuertic Protein (NT-proBNP) and a higher left ventricular enddiastolic pressure. Moreover, aortic stenosis or pressure wire measurements were associated with a troponin rise. In addition, the authors conclude that a major troponin rise was associated with a worse long-term outcome.

Indeed, it is not surprising that older and sicker patients will experience a more pronounced troponin rise. If this troponin rise does translate into a worse outcome remains somehow speculative. Unfortunately, the patient number in the present study is too small to elucidate this research question.

In general, the interpretation of the presented data is impeded by several potential unmeasured confounders. In particular, the outcome of the revascularisation procedure
It is hard to believe that a troponin rise following a diagnostic procedure should impact on survival, while a minor isolated troponin elevation after percutaneous intervention is considered to be negligible?

In conclusion, it is unlikely that this study will change current clinical practice.

Contributors GMF: Idea, draft of the manuscript. DML: Table and proof reading.

Provenance and peer review Commissioned; internally peer reviewed.

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REFERENCES
1. Shave R, Baggish A, George K, et al. Exercise-Induced cardiac troponin elevation. J Am Coll Cardiol 2010;56:169–76.
2. Tricoci P, Leonardi S, White J, et al. Cardiac troponin after percutaneous coronary intervention and 1-year mortality in non-ST-segment elevation acute coronary syndrome using systematic evaluation of biomarker trends. J Am Coll Cardiol 2013;62:242–51.
3. Ndrepepa G, Colleran R, Braun S, et al. High-Sensitivity troponin T and mortality after elective percutaneous coronary intervention. J Am Coll Cardiol 2016;68:2259–68.
4. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Eur Heart J 2012;33:2551–67.
5. American College of Cardiology. Determining myocardial infarction after PCI: ck-mb, troponin, both or neither? http://www.acc.org/latest-in-cardiology/articles/2014/07/18/14/53/determining-myocardial-infarction-after-pci-ck-mb-troponin-both-or-neither
6. Saito T, Hojo Y, Hirose M, et al. High-sensitivity troponin T is a prognostic marker for patients with aortic Stenosis after valve replacement surgery. J Cardiol 2013;61:342–7.
7. Rosja H, Andreassen J, Edvardsen T, et al. Prognostic usefulness of circulating high-sensitivity troponin T in aortic Stenosis and relation to echocardiographic indexes of cardiac function and anatomy. Am J Cardiol 2011;108:88–91.
8. Chin CW, Shah AS, McAllister DA, et al. High-sensitivity troponin I concentrations are a marker of an advanced hypertrophic response and adverse outcome in patients undergoing percutaneous repair of mitral valve regurgitation. PLoS One 2015;10:e0137464.
9. Thygesen K, Mir J, Katus H, et al. Recommendations for the use of cardiac troponin measurement in acute cardiac care. Eur Heart J 2010;31:2197–204.
10. Bocchiampi N, Audibert G, Rangeld O, et al. Serum troponin ic values in organ donors are related to donor myocardial dysfunction but not to graft dysfunction or rejection in the recipients. Int J Cardiol 2009;133:80–6.
11. Marasco SF, Kras A, Schulberg E, et al. Impact of warm ischemia time on survival after heart transplantation. Transplant Proc 2012;44:1385–9.
17. De Santo LS, Torella M, Romano G, et al. Perioperative myocardial injury after adult heart transplant: determinants and prognostic value. *PLoS One* 2015;10:e0120813.