Supplementary oxygen in acute STEMI: no added benefit rather than potential for harm

Heerajnarain Bulluck[1,2], Derek J Hausenloy[1,2,3,4]

[1]The Hatter Cardiovascular Institute, Institute of Cardiovascular Science, University College London, London, UK
[2]The National Institute of Health Research University College London Hospitals Biomedical Research Centre, London, UK
[3]National Heart Research Institute Singapore, National Heart Centre Singapore, Singapore.
[4]Cardiovascular and Metabolic Disorders Program, Duke-National University of Singapore, Singapore.

Corresponding author:

Dr Heerajnarain Bulluck
The Hatter Cardiovascular Institute, Institute of Cardiovascular Science, NIHR University College London Hospitals Biomedical Research Centre, University College London Hospital & Medical School,
67 Chenies Mews, London,
WC1E 6HX, UK.
Tel: +44 (203) 447 9888
Fax: +44 (203) 447 9505
E-mail: h.bulluck@gmail.com

Word Count: 429

No disclosures.

Key words: ST-segment myocardial infarction, primary percutaneous coronary intervention, oxygen therapy, cardiovascular magnetic resonance, troponin I

The use of high-flow oxygen in the setting of acute myocardial infarction remains controversial. We therefore read with great interest the randomized controlled clinical trial by Stub et al[1], which investigated the effect of supplementary oxygen versus air in normoxic patients presenting with an acute ST-segment elevation myocardial infarction (STEMI). In their study, the authors concluded that supplementary oxygen therapy may increase early myocardial injury and was associated with larger myocardial infarct size assessed by cardiovascular magnetic resonance (CMR) at 6 months in STEMI patients. However, the data provided in the study do not appear to support this conclusion. Firstly, it was based on a difference in peak levels of serum CK between the two groups. However, CK is known to be less specific for myocardial necrosis[2], and the study had not been originally powered to detect this endpoint. The authors had actually powered the study based on peak levels of serum troponin I, a more specific biomarker for myocardial necrosis than CK, and for which there was no significant difference between the 2 groups. Secondly, they reported a difference in myocardial infarct MI size at 6 months in grams measured by CMR, but when this was normalized to left ventricular (LV) mass, there was no significant difference between the two groups. In the absence of T2-weighted CMR imaging to quantify the area-at-risk (AAR), the authors could have used coronary angiographic jeopardy scores to estimate the AAR and calculate myocardial salvage[3, 4], an approach which has previously been used to validate T2-weighted CMR imaging. In summary, the data do not appear to support the conclusion that supplemental oxygen may be harmful in normoxic STEMI patients when compared to air – rather it shows no added benefit with this therapeutic approach.

1. Stub D, Smith K, Bernard S, Nehme Z, Stephenson M, Bray JE, Cameron P, Barger B, Ellims AH, Taylor AJ, Meredith IT, Kaye DM and Investigators* A. Air Versus Oxygen in ST-Segment-Elevation Myocardial Infarction. *Circulation*. 2015;131:2143-50.
2. Tsung SH. Several conditions causing elevation of serum CK-MB and CK-BB. *Am J Clin Pathol* 1981;May 711-5.
3. Fuernau G, Eitel I, Franke V, Hildebrandt L, Meissner J, de Waha S, Lurz P, Gutberlet M, Desch S, Schuler G and Thiele H. Myocardium at risk in ST-segment elevation myocardial infarction comparison of T2-weighted edema imaging with the MR-assessed endocardial surface area and validation against angiographic scoring. *JACC Cardiovascular imaging*. 2011;4:967-76.

4. Wright J, Adriaenssens T, Dymarkowski S, Desmet W and Bogaert J. Quantification of myocardial area at risk with T2-weighted CMR: comparison with contrast-enhanced CMR and coronary angiography. *JACC Cardiovascular imaging*. 2009;2:825-31.