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Editorial

# Echocardiography in myocardial perfusion and mechanics analysis after acute myocardial infarction – Old dog, new tricks

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Infarct-related dysfunction of myocardial tissue is a result of two main mechanisms: irreversible damage is associated with a reversible stunned myocardium. Myocardial stunning is defined as a temporary dysfunction of the myocardium persisting after ischemia-reperfusion with the absence of irreversible histological damage [1]. The diagnosis of stunning is established based on the impairment of contractile function which is reversible and the return of normal coronary blood flow in the dysfunctional territory.

Echocardiography can qualitatively and quantitatively (wall motion score index - WMSI) analyze myocardial contractility and deformation. However, myocardial perfusion assessment requires a special echocardiographic contrast to be used. The idea of using contrast agents in cardiac ultrasound was born in the late 1960s [2] and for the basis of myocardial perfusion analysis were given in works of Keller et al. [3] Modern myocardial contrast echocardiography (MCE) agents are microbubbles composed of high-molecular-weight gas encapsulated in an outer shell. The diameter of these springy microbubbles vary - the majority are less than 6 μm with a mean diameter of around 2–3 μm. The size of the contrast particles allows them to flow easily through microcapillaries, which have a diameter greater than 5 μm. From the echocardiographic technical side, the best detection of the contrast is ensured by using a low- mechanical index harmonic mode. If the myocardial microvessels are damaged, there is no contrast flow and a lack of echocardiographic signal enhancement is observed in this region.

MCE is rather safe, and the side effects are rare [4]. A headache, flushing or back pain is usually relieved on cessation of contrast agent administration. In very rare cases (1:10,000 risk) of a non-IgE related pseudoanaphylaxis and hypersensitivity can be found in literature [5,6].

The technique has some important practical advantages over others (cardiac magnetic resonance, positron emission computed tomography, single photon emission computed tomography etc.), being rapid, less expensive and avoiding the use of ionizing radiation. The sensitivity and specificity are comparable to radiological techniques [2], but none of them can be used at the patient's bedside.

In this issue, Qiu et al. [7] using the integrated imaging approach (MCE, deformation analysis and coronary angiography) show that in some patients with acute coronary syndrome, the region of mechanic abnormalities after ischemia exceed the region of microvascular damage and perfusion-contractility mismatch occurs. In most patients with perfusion-contractility mismatch, an apical ballooning ventricular contractile pattern occurred. Their abnormal wall motion score increased from basal to apical ventricles, and absolute values of myocardial strains descended from basal to apical ventricles, both of which were comparable to the findings in patients with takotsubo syndrome (TTS). TTS is a transient left ventricular dysfunction most often preceded by mental or physical stress [8]. The most reliable mechanism of TTS is catecholamine-mediated myocardial stunning. Physiologically, epinephrine released from the adrenal gland and norepinephrine released from its nerve endings, exert a positive inotropic effect through the G proteins family. With an excess of catecholamines β2 adrenergic receptors, which are located mainly in the apex of the left ventricle, activate cAMP G protein. As a result, myocardial contractility is reduced, which is termed catecholamine toxicity [9]. This mechanism and microcirculatory dysfunction [10] in the ischemic regions may be the cause of the observed apical ballooning effect.

However, contrary to patients without mismatch, due to nonstructural but functional rather than transient failure, the scale of the worsening of systolic function is not on par with a diastolic one and finally with hemodynamic deterioration. For this reason, myocardial contrast echocardiography appears to be a useful tool in establishing an appropriate therapeutical approach to patients.

The observations made by the authors may enrich our understanding of myocardial dysfunction in ACS-TTS *continuum*.

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## References

[1] A.I. Guaricci, G. Bulzis, G. Pontone, et al., Current interpretation of myocardial stunning, Trends Cardiovasc. Med. 28 (4) (2018) 263–271, https://doi.org/10.1016/j.tcm.2017.11.005.
[2] R. Gramiak, P.M. Shah, D.H. Kramer, Ultrasound cardiography: contrast studies in anatomy and function, Radiology 92 (5) (1969) 939–948, https://doi.org/10.1148/92.5.939.
[3] M.W. Keller, S.S. Segal, S. Kaul, B. Duling, The behavior of sonicated albumin microbubbles within the microcirculation: a basis for their use during myocardial

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https://doi.org/10.1016/j.ijcard.2018.11.016

contrast echocardiography, Circ. Res. 65 (2) (1989) 458–467, https://doi.org/10.1161/01.RES.65.2.458.
[4] P. Scisło, J. Kochanowski, Ł. Kołtowski, G. Opolski, Utility and safety of three-dimensional contrast low-dose dobutamine echocardiography in the evaluation of myocardial viability early after an acute myocardial infarction, Arch. Med. Sci. 14 (3) (2018) https://doi.org/10.5114/aoms.2016.58650.
[5] L. Rotaru, T. Nanea, Assessment of Myocardial Perfusion Using Contrast Echocardiography – Case Report, 8(4), 2015 471–475.
[6] S. Orde, A. Mclean, Bedside myocardial perfusion assessment with contrast echocardiography, Crit. Care (2016) https://doi.org/10.1186/s13054-016-1215-7.
[7] Q. Qiu, M. Abdelghany, R. Subedi, et al., Discrepant myocardial microvascular perfusion and mechanics after acute myocardial infarction: characterization of the "Tako-tsubo effect" with real-time myocardial perfusion contrast echocardiograph, Int. J. Cardiol. (November 2018) https://doi.org/10.1016/j.ijcard.2018.09.114.
[8] J. Ghadri, I.S. Wittstein, A. Prasad, et al., International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology, 2018 2032–2046, https://doi.org/10.1093/eurheartj/ehy076.
[9] P.T. Wright, M.H. Tranter, A.C. Morley-smith, A.R. Lyon, Focus Issue on Takotsubo Cardiomyopathy Pathophysiology of Takotsubo Syndrome – Temporal Phases of Cardiovascular Responses to Extreme Stress, 78, July 2014 1550–1558, https://doi.org/10.1253/circj.CJ-14-0623.
[10] E. Verna, S. Provasoli, S. Ghiringhelli, F. Morandi, J. Salerno-Uriarte, Abnormal coronary vasoreactivity in transient left ventricular apical ballooning (tako-tsubo) syndrome, Int. J. Cardiol. 250 (2018) 4–10, https://doi.org/10.1016/j.ijcard.2017.07.032.