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EDITORIAL

Leave me alone: the right ventricle in anterior myocardial infarction

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This editorial refers to 'Right ventricular involvement in anterior myocardial infarction: a translational approach', by V. Bodi *et al.*, pp. 601–608, this issue.

Even though rates have been declining with the turn of the millennium, about 150 000 patients (500 per million) in the USA and 475 000 patients (800 per million) in Europe suffer an ST segment elevation myocardial infarction (STEMI) each year.[1,2] The distribution of these STEMIs is fairly evenly split between anterior, left anterior descending (LAD)-related events (40–50%) and inferior, right coronary artery (RCA)-related events (approximately 40%).[3,4] In general, the prognosis is thought to be better with inferior than with anterior MIs.[5] This, however, is not the case with concomitant involvement of the right ventricle (RV), which indicates a two to three times higher risk of arrhythmia, cardiogenic shock, and mortality in inferior MI.[6] Moreover, predominantly RV-related cardiogenic shock is associated with a prognosis that is as detrimental as left ventricle (LV)-related cardiogenic shock (>50% in-hospital mortality).[7] Reperfusion therapy improves these outcome statistics to those patients without RV involvement (2% in-hospital mortality).[8] Despite these implications, the RV has, for the most part, remained the 'forgotten chamber'—in need of 'wake-up calls' to pay greater attention to its involvement in MI.[9,10]

Bodi *et al.*[11] respond to this call in a translational manner. In both the experimental and the clinical approach, they found that approximately one-third of the RV (i.e. the anterior wall) was at risk with LAD occlusion. With reperfusion, however, 94% of the area at risk was salvaged and the resulting infarct size was only 2% of the RV. With inferior STEMIs, the RV area at risk was greater (50% of the RV), the amount of salvaged RV myocardium was smaller (85%), and the infarct size was greater (6%). Intriguingly, the incidence of RV infarction by magnetic resonance imaging (MRI) was quite the same with anterior and inferior STEMI (40 and 50%, respectively). Anterior STEMI patients with and without RV infarction did not differ with regards to clinical, ECG, and angiographic characteristics. However, all arrhythmic events were noted exclusively in patients with RV infarction.

The authors are to be commended for their efforts and their findings of how frequent RV involvement occurs with anterior MI—as frequent as with inferior MI. As depicted in *Figure 1*, these observations are explained by the fact that the LAD is linked to the perfusion of the anterior RV as much the posterior descending artery is linked to the posterior and inferior wall and the marginal branches to the lateral wall.[12] In agreement with the current study, Jensen *et al.*[13] showed that RV infarction is common in anterior STEMI (65 vs. 47% in inferior STEMI) and commonly not extensive (< 4 of 12 segments in 77%). Especially smaller degrees of RV infarction were not recognized by ECG and echocardiography, and hence there was only moderate agreement between these modalities and MRI. As it appears, Bodi *et al.* did not assess these parameters, and neither study utilized nuclear imaging or right heart catheterizations. In either way, though, MRI allows a much more sophisticated analysis of RV involvement including global and regional contractile dysfunction, tissue oedema, microvascular obstruction, and infarction. Hence, MRI seems to outperform the traditional diagnostic techniques and may be considered the new gold standard for RV assessment.[14] When, how much, and at which cost MRI will impact clinical outcome remains to be defined.

The current study indicates that clinical parameters such as vital signs, Killip class, and cardiac troponin levels do not differ between those with and without MRI evidence of RV infarction. These data suggest that MRI increases the sensitivity to detect RV involvement even when it is not clinically expected or apparent. This prompts the question of how extensive RV infarction would have to be to translate into clinical presentation. One may argue that even if the LAD provides for one-third of RV perfusion, it still provides for half of the LV perfusion and hence the impact and signs of LV involvement may outweigh those of any RV involvement. Furthermore, LV dysfunction may impact RV function by increasing pulmonary pressures, making it difficult to distinguish primary from secondary impairment. On the contrary, the RCA supplies two-thirds of the RV (in a right dominant coronary artery system) and one quarter to a third of the LV. For this reason, RV infarction may become relatively more noticeable with inferior MIs, and generations of physicians have been taught to recognize it in this setting. These considerations apply in particular to the acute and/or non-revascularized stage. With revascularization, the permanent damage is to be very small, and it is difficult to conceive that this would impact prognosis. Even factoring in the prognostic implications of microvascular obstruction, the area still remains

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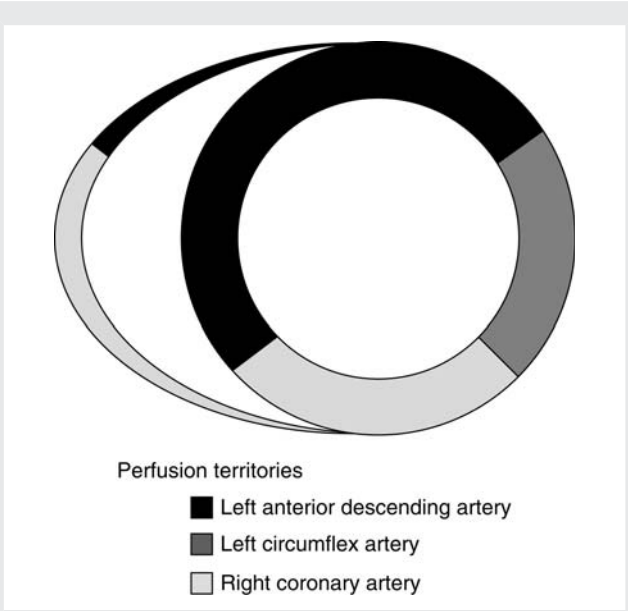

Figure 1 Illustration of coronary artery perfusion territories.

relatively small. Most of the acute injury, as shown in the current study, is tissue oedema and should therefore be reversible.

Indeed, the recovery potential of the RV in the setting of acute MI has been deemed to be very good to begin with due to its low nutrient demand, its oxygen supply by diffusion, and an elaborate collateral circulation. One could argue that especially if all goes well from a reperfusion standpoint, no one has to worry about the RV and may, in fact, simply 'forget' about it. Is there, then, any clinical benefit in obtaining an MRI some days after complete revascularization of an acute MI? This question was not addressed in the current study but the work by Jensen *et al.*[13] points out that MRI evidence of RV infarction after primary PCI indicates a 16-fold higher risk of major adverse cardiac events and even more so in anterior MIs. As most of these future events were revascularization procedures, RV infarction might simply indicate more proximal LAD and more extensive coronary artery disease and may not by itself be a prognostic indicator. However, Larose *et al.*[15] found that a reduction in RV ejection fraction to less than 40% beyond the first 30 days after MI predicts a three times higher long-term mortality risk independent of other variables. This persistent reduction in the RV function was observed in 14% of all MIs with equal contribution of anterior and inferior MIs. There was no correlation with LV infarct extension, and RV infarction was noted in only 16% of these patients. In keeping with these and the findings of Bodi *et al.*, the final RV infarct size in acute MI seems to be small and not the main factor for the prognostically relevant, persistent RV function impairment. As the next step, it will be important to study those factors that impair the recovery potential of the RV and lead to its sustained dysfunction. The current study provides an important impulse to not forget about the RV even in acute anterior MI. Future studies will have to take it further to the bench and the bedside.

Conflict of interest: none declared.

References

1. Movahed MR, John J, Hashemzadeh M, Jamal MM, Hashemzadeh M. Trends in the age adjusted mortality from acute ST segment elevation myocardial infarction in the United States (1988–2004) based on race, gender, infarct location and comorbidities. *Am J Cardiol* 2009;**104**:1030–1034.
2. Widimsky P, Wijns W, Fajadet J, de Belder M, Knot J, Aaberge L *et al.* Reperfusion therapy for ST elevation acute myocardial infarction in Europe: description of the current situation in 30 countries. *Eur Heart J* 2010;**31**:943–957.
3. Laarman GJ, Suttorp MJ, Dirksen MT, van Heerebeek L, Kiemeneij F, Slagboom T *et al.* Paclitaxel-eluting versus uncoated stents in primary percutaneous coronary intervention. *N Engl J Med* 2006;**355**:1105–1113.
4. Svilaas T, Vlaar PJ, van der Horst IC, Diercks GF, de Smet BJ, van den Heuvel AF *et al.* Thrombus aspiration during primary percutaneous coronary intervention. *N Engl J Med* 2008;**358**:557–567.
5. Mehta SR, Eikelboom JW, Natarajan MK, Diaz R, Yi C, Gibbons RJ *et al.* Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am Coll Cardiol* 2001;**37**:37–43.
6. Hamon M, Agostini D, Le Page O, Riddell JW, Hamon M. Prognostic impact of right ventricular involvement in patients with acute myocardial infarction: meta-analysis. *Crit Care Med* 2008;**36**:2023–2033.
7. Jacobs AK, Leopold JA, Bates E, Mendes LA, Sleeper LA, White H *et al.* Cardiogenic shock caused by right ventricular infarction: a report from the SHOCK registry. *J Am Coll Cardiol* 2003;**41**:1273–1279.
8. Bowers TR, O'Neill WW, Grines C, Pica MC, Safian RD, Goldstein JA. Effect of reperfusion on biventricular function and survival after right ventricular infarction. *N Engl J Med* 1998;**338**:933–940.
9. Rigolin VH, Robiolio PA, Wilson JS, Harrison JK, Bashore TM. The forgotten chamber: the importance of the right ventricle. *Cathet Cardiovasc Diagn* 1995;**35**:18–28.
10. Pfisterer M. Right ventricular involvement in myocardial infarction and cardiogenic shock. *Lancet* 2003;**362**:392–394.
11. Bodi V, Sanchis J, Mainar L, Chorro FJ, Nunez J, Monmeneu JV *et al.* Right ventricular involvement in anterior myocardial infarction. A translational approach. *Cardiovasc Res* 2010;**87**:601–608.
12. Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right ventricular function in cardiovascular disease, part I: anatomy, physiology, aging, and functional assessment of the right ventricle. *Circulation* 2008;**117**:1436–1448.
13. Jensen CJ, Jochims M, Hunold P, Sabin GV, Schlosser T, Bruder O. Right ventricular involvement in acute left ventricular myocardial infarction: prognostic implications of MRI findings. *AJR* 2010;**194**:592–598.
14. Burgess MI, Bright-Thomas RJ, Ray SG. Echocardiographic evaluation of right ventricular function. *Eur J Echocardiogr* 2002;**3**:252–262.
15. Larose E, Ganz P, Reynolds HG, Dorbala S, Di Carli MF, Brown KA *et al.* Right ventricular dysfunction assessed by cardiovascular magnetic resonance imaging predicts poor prognosis late after myocardial infarction. *J Am Coll Cardiol* 2007;**49**:855–862.