Cardiovascular Research turns the spotlight onto the right ventricle

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This month’s issue of Cardiovascular Research is dedicated to the right ventricle. As introduced by the guest editors, this has long been considered the ‘lesser’ ventricle. A PubMed search for ‘left ventricle’ (LV) shows 25,217 publications since 2010, whereas a search for ‘right ventricle’ (RV) results in 21,684 publications in the same time period. However, most documents include both terms, so if we compare the publications that exclusively cover the ‘left ventricle’ we get 6,720 documents, and in contrast there are only 2,737 publications specifically on the ‘right ventricle’.

Even if this is a crude approximation, it aligns with a message that is present in several of the review articles of this Spotlight Issue, namely that our knowledge of the RV is less than that of the LV. At the same time, available knowledge clearly indicates the particularities of the RV and underscores that we cannot simply ‘copy-paste’ insights from the LV to the RV.

In the past years, we also noticed that the RV was less often the topic of submitted manuscripts but seemed to gain attention. We have browsed the recent issues of Cardiovascular Research, bringing together relevant publications in this virtual issue including selected articles from Europace and European Heart Journal, to complement the series of reviews.

Filling of the RV and diastolic functions were studied by Pérez Del Villar et al., focusing on the restoring forces and the role of suction.1,2 The interaction between LV and RV function, focusing on diastolic dysfunction, was recently underscored in a study on HFpEF patients demonstrating impaired RV functional reserve.3

The causes and mechanisms underlying arrhythmogenic RV disease are still being unravelled,4 and diagnostic testing presents challenges.5,6 The power of advanced and next generation sequencing can support differentiating impaired RV functional reserve.7

Mechanisms of remodeling and targets for treatment are mostly studied in models of RV hypertrophy and failure consequent on pulmonary hypertension (PHT).9 Within the RV, apoptosis and loss of myocytes may temporarily be compensated by the remaining myocytes.10 Targeting pathways that reduce PHT and vascular remodelling are associated with improved RV remodelling. While this could be due to the reduced haemodynamic load, some of these approaches may involve some direct effects on the RV, as e.g. the neuregulin pathway.11 Other mechanistic studies emphasize the tight link between the vascular remodelling and RV remodelling, as for miR-223-IGF-IR.12,13 Thrombospondin-1 may be important in signalling within the lungs and pulmonary vascular smooth muscle cells in response to hyoxia,14,15 but interrupting this pathway benefits cardiac function as well.16,17

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References


