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The sick right ventricle in endurance athletes: What is the contribution of the pulmonary vascular bed?

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For a long time, the right ventricle was referred to as the "*forgotten ventricle*". In recent years, this has changed significantly. A growing number of studies have focussed on the investigation of the right ventricle in many diseases such as pulmonary hypertension or heart failure, and the pathophysiology of the right ventricle has gained a particularly high degree of attention in exercise medicine and science.[1–7] Right ventricular properties have been shown consistently to play a central role in training-dependent adaptation processes in response to altered loading conditions, and represent an important determinant of individual exercise performance. Intense endurance exercise has been demonstrated to trigger acute dysfunction of the right ventricle, usually followed by a complete short-term recovery. In contrast, the left ventricle remains unaffected by such an acute form of physical stress.[4,7,8] The differences regarding the capacities of the ventricles may be attributable to a high workload and wall stress of the right ventricle compared to the left ventricle.[5] In some athletes, chronic changes of the right ventricular morphology (e.g. right ventricular enlargement) and the right ventricular myocardial structure (e.g. increased myocardial fibrosis) as well as a persistently reduced function and an increased frequency of arrhythmias have been demonstrated.[4,7,8] In addition, significant interindividual and sex differences have been reported with regard to these right ventricular adaptations to endurance exercise. Thus, athletes performing similar amounts of exercise may demonstrate extremely different structural and functional right ventricular changes, varying from no to severe dysfunction.[7,8] Interestingly, both an enhanced right ventricular contractile reserve as well as a less impaired right ventricular function in response to intense endurance exercise have been shown in female athletes as compared to male athletes.[7]

The mechanisms underlying these interindividual differences are still not well understood. One suggested explanation is pulmonary vascular adaptation processes. In particular, the capability of recruiting anatomical arterial-venous shunt vessels and the extent of the dilatation of the pulmonary capillaries during exercise have been discussed in this respect. These changes would reflect a larger pulmonary vascular reserve and, consequently, a lower exercise-related increase in pulmonary artery systolic pressure during exercise.[6,8,9]

This hypothesis has now been further investigated by Sanz-de la Garza et al. in an exploratory study, recently published in the *European Journal for Preventive Cardiology*.[10] The authors evaluated the transpulmonary transit of agitated saline (PTAS) as a parameter of vascular reserve by echocardiography immediately after a symptom-limited incremental cardiopulmonary exercise test on a cycle ergometer in 100 highly trained female and male endurance athletes aged 20–45 years. For the final analyses, the data of 88 participants were available (11 athletes were excluded due to an intracardiac shunt at rest and one due to a chronic obstructive pulmonary disease). The athletes were stratified into two groups according to a high or low PTAS. The major findings of the study were that a high PTAS capability was associated with a significantly larger right ventricular contractile reserve, a larger pulmonary vascular reserve and an enhanced maximal exercise capacity. Athletes with a high PTAS capability also showed a more pronounced biventricular remodelling than those with a low PTAS. This was seen in both female and male athletes. Interestingly, female sex was the only significant independent determinant of a high PTAS capability in the multivariate analysis, while age and history of intense dynamic exercise training during childhood were additional significant correlates of a high PTAS only in the univariate analyses.

With their results, Sanz-de la Garza et al.[10] make an important contribution to clarifying the question of why there are such large interindividual differences in

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the adaptation processes of the right ventricle to high-dose endurance training. This very well planned and carried out study provides further evidence for the hypothesis that the capability of the pulmonary vascular bed to adapt during endurance exercise, indeed, plays a crucial role in determining whether and to what extent an impairment of the right ventricular performance develops in this situation.

However, while some open questions have not yet been answered sufficiently by this study either, these limitations illustrate very well the need for future research in this field. Firstly, some methodological issues have to be discussed. Thus, pulmonary artery pressure was estimated only indirectly but could not be assessed accurately in this study. The explanation of the authors for this limitation was that most of the participants showed minimal tricuspid regurgitation with inadequate Doppler signals for using the regurgitant tricuspid flow method. Therefore, this study gives only a hint towards a decreased pulmonary arterial pressure during exercise in those with a larger pulmonary vascular reserve as an explanation for an improved right ventricular adaptation and a higher exercise capacity. Ideally, this issue should be investigated by invasive measurements of the pulmonary pressure during exercise as the gold standard in future studies. In addition, it would also be desirable to assess PTAS immediately *during* peak exercise; although, this is an enormous practical and methodological challenge. In the present study, this was done after termination of the exercise test (measurements within the first two minutes during the recovery phase were used).

The second, still open, question is the interplay between the right and left ventricle, which could not be investigated in sufficient detail in this study. *We definitively should not forget the left ventricle, now!* Future investigations should include an accurate assessment of left ventricular stiffness to evaluate the impact of left ventricular compliance on the enhanced right ventricular contractile reserve and the potentially lower increase in pulmonary arterial pressure in athletes with a high PTAS capability.

An additional important task is to learn more about the determinants of PTAS; specifically, how it can be improved and to which extend acute and lifetime training volume as well as type of exercise training may be influencing factors. For example, it was a bit surprising that training load was not a significant determinant (at least, in the multivariable analysis of a high PTAS), although it is well known to have a large impact on ventricle adaptations in athletes.[2,3]

Finally, as also concluded by the authors themselves,[10] the long-term implications of the absence, presence and extent of pulmonary shunting, and the subsequent effects on right ventricular afterload increase during (endurance) exercise, remain to be determined by subsequent investigations.

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