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COMMENTARY

Aortic constriction and the relevance of physiologic research

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A casual reader might be tempted to skip over the article by Apitz and colleagues in this issue, dismissing it as irrelevant to clinical practice. The title is also somewhat off-putting because “aortic constriction” evokes only bad images in the mind of any cardiac surgeon. In fact, however, this elegant yet traditional cardiac physiology experiment shows that chronic aortic constriction not only improves right ventricular (RV) function in the setting of chronic RV pressure overload but also leads to less maladaptive RV remodeling. This article does inform on highly relevant themes, such as ventricle-ventricle interaction, timing of RV remodeling, and the molecular signature of

RV profibrotic reactions. It may also open the door to a very untraditional way of managing the remodeled RV. The problem of the hypertrophied yet not decompensated RV is typically solved by an early repair of the anatomy, such as early repair of tetralogy of Fallot for example. No one would consider “banding” the aorta to allow for reverse RV remodeling. What, however, about the thick, fibrotic RV in an older patient with unrepaired pulmonary stenosis? Should we consider, akin to pulmonary artery banding and left ventricular training in an older patient with transposition of the great arteries, a period of aortic afterload increase to reposition the septum and maybe reverse some of the myocardial profibrotic signaling? What about managing the failing postoperative RV? In the setting of good left ventricular function, and in the absence of any current drugs that can directly increase RV contractility, systemic afterload increase by pharmacologic means may in fact have a role to play.

At a time when therapeutic answers are being sought mostly in genetics and tissue engineering, this study reminds us that physiologic research remains an essential and vital tool.

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