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Commentary: Invasive, but indispensable: Hemodynamic assessment to comprehend interventricular interactions

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It was almost a century ago, in 1929, that Dr Werner Forssmann, intrigued by the recording of intracardiac pressures in horses reported 70 years previously, would pass a ureteral catheter through his own antecubital vein and obtain an X-ray showing it in his right atrium.¹ A decade or so later, stimulated by Forssmann's groundbreaking work, pulmonologists Drs André Cournand and Dickinson Richards formed a famous collaboration to quickly establish invasive hemodynamic assessment of their patients as the basis of modern cardiology.² The 3 physicians would later share the Nobel Prize in Medicine in 1956.

There followed an explosion of basic and clinical research establishing invasive hemodynamic and functional assessment as the gold standard to describe right and left ventricular function individually, as well as forming the basis for our understanding of the complex interplay between the right and left ventricles as a functional unit. This is true both in the normal heart and those with various abnormal pressure and volume loads.^{3,4}

In 1956, Dr Abraham Rudolph would become the first congenital cardiologist to perform a cardiac catheterization in a young infant, subsequently helping to establish it, then, as the primary tool for diagnosis and, more recently, treatment of congenital heart disease.⁵ Straddling the era of invasive hemodynamic research and the understandable concentration on noninvasive diagnosis

CENTRAL MESSAGE

This article demonstrates the occurrence of adverse interventricular interactions in both the volume- and pressure-loaded right ventricle.

and functional assessment, the use of invasive assessment of complex hemodynamics as a foundational gold standard in congenital diseases was, perhaps, abandoned too soon. We ourselves have jumped on the bandwagon in exploring and promoting noninvasive means toward understanding hemodynamics and functional parameters, which, until recently, could only accurately be assessed invasively.^{6,7}

We commend Jani and colleagues⁸ for bringing us back to our foundational roots in this excellent, albeit small, cohort animal study on the effects of pressure and volume loading of the right ventricle on interventricular interactions. Many of the adverse findings reported in this study are consistent with noninvasive investigations in patients with repaired tetralogy of Fallot, both those with pressure- or volume-loaded right ventricles. For example, it has been demonstrated in this population with a volume-loaded right ventricle that diastolic interventricular septal flattening correlates with increased pulmonary regurgitation, right ventricular volumes, and QRS duration, as well as decreased right and left ventricular ejection fractions.⁹ Diastolic interventricular septal flattening in combination with the common occurrence of right ventricular restrictive physiology in this population has also been correlated to impaired left ventricular filling as assessed by echocardiography.¹⁰ The current study demonstrated prolonged left ventricular filling time due to elevated left ventricular end-diastolic pressure and an increase in isovolumic relaxation time in animals with a volume-loaded right ventricle.⁸ It is only by the additional insight provided in

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these types of invasive studies that will help to complement and fill in the gaps in knowledge regarding this complex ventricular–ventricular interplay, which involves a combination of mechanical, electromechanical, and neurohormonal coupling.⁴

Have we missed the opportunity to perform such studies in our patients? We think not. A thorough understanding of the consequences of our actions after the surgical and nonsurgical treatment of congenital heart diseases will continue to unfold over decades. We clearly are still learning about those consequences, primarily using noninvasive assessments, and largely in the absence of foundational invasive observations. We would argue that intermittent cardiac catheterization studies, particularly using the sophisticated pressure–volume catheters described by Jani and colleagues⁸ and by us in patients previously,¹¹ cannot only be justified on a risk–benefit basis, but that we owe it to our patients that they form a component of the fundamental assessment of the unique hemodynamic consequences of their congenital heart diseases and its treatment.

In summary, while we would not be so bold as Dr Forssmann, reportedly walking himself up one flight of stairs to the X-ray department to document his self-catheterization, we would, however, be bold enough to risk accusations of being “dinosaurs” in appealing to our field to selectively restore invasive hemodynamics as a gold-standard research and clinical

tool to complement and better understand our noninvasive assessments.

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