Postoperative right ventricular dysfunction—Integrating right heart profiles beyond long-axis function

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Consequences (A-D) and mechanism (E-H) of right ventricular failure.

Central Message

Precision perioperative RV and pulmonary artery pressure monitoring integrates 3D volumetric assessment, RV strain, splanchnic and renal venous congestion repercussions, and air embolic burden.

See Article page 1430 in the April 2020 issue.

Right ventricular (RV) failure has been long recognized by cardiac surgeons as a major cause of morbidity and mortality.¹⁻⁷ Through the years, studies have focused on identifying more sensitive markers of RV dysfunction. The study of Singh and colleagues⁸ in this issue of the *Journal* brings novel insights into perioperative changes in RV function, focusing on annular, longitudinal shortening (RV myocardial strain) as well as area changes. Its strength lies in describing the dynamic of RV changes at key perioperative moments and in variable types of surgery. Almost 30 years ago, Popp and associates⁹ observed impairment in tricuspid annular excursion following cardiac surgery. One important question raised by this observation was whether longitudinal RV function reflects global systolic function. In fact, one of the challenges in assessing RV function resides in its complex geometry. By means of 3-dimensional echocardiography, Tamborini and coworkers¹⁰ showed that despite the postoperative reduction of tricuspid annular excursion, 3-dimensional RV ejection fraction remained preserved at 3 postoperative months. The study of Singh and colleagues⁸ also demonstrates that RV fractional area change is less impaired than annular or strain indices, at least in patients undergoing coronary revascularization.

Systolic parameters are, however, only one component of right heart performance.¹¹ Other components can be divided as consequences or mechanism of RV failure (Figure 1). Hemodynamic elements, such as central venous pressure and the mean pulmonary arterial pressure to systemic arterial pressure ratio (yielding insights into both the pulmonary and systemic circulations¹³⁻¹⁷) are useful to monitor; however, combined pulmonary artery and RV pressure waveform (Figure 1, A)¹⁸ appears to be a promising method for detecting abnormal RV function.¹⁹ The current understanding of RV failure combines the

ventricular and the arterial elastance as an index of RV-arterial coupling.²⁰ When this RV-arterial coupling is less than 1, then the RV will enlarge to maintain cardiac output, but at the expense of venous congestion.

This concept in recent years has led to an emphasis beyond systolic and diastolic RV parameters toward both forward (low cardiac output) and backward (venous congestion) consequences of RV dysfunction. This can lead to a vicious cycle of decreased overall tissue perfusion and congestion to the brain, liver, spleen, gut, and kidney.^{21,22} (Figure 1, B through D) In 2 recent prospective studies in which portal venous flow Doppler was interrogated before, during, and after cardiac surgery,^{23,24} the presence of portal pulsatility greater than 50% (used as a surrogate for splanchnic congestion) was associated with all the commonly used hemodynamic and echocardiographic variables related to RV dysfunction. In addition, both abnormal portal and renal venous pulsilities were associated with prolonged duration of stay in the intensive care unit, excessive bleeding, reoperation, acute renal failure, and increased overall complication rate.^{23,24}

Finally, air embolization (Figure 1, E through H) should always be considered as contributing mechanism of RV

Consequences of RV failure

Mechanism of RV failure

FIGURE 1. Consequences (A-D) and mechanisms (E-H) of right ventricular (RV) failure. **A**, Simultaneous pulmonary artery pressure (PAP) and right ventricular pressure (RVP) waveforms. Numbered features: 1, Normal right ventricular pressure (Prv) waveform with rapid systolic pressure changes; 2, normal horizontal Prv diastolic slope; 3, abnormal right ventricular pressure Prv diastolic slope greater than 4 mm Hg; 4, square root sign with RV pulsus tardus; 5, diastolic equalization between the Prv and the diastolic pulmonary artery pressure (Ppa). **BIS**, Basal inferior section; **MIS**, midinferior section; **APS**, apical section; **AVC**, aortic valve closure; **SV**, splenic vein; **PWD**, pulsed-wave Doppler; **MCA**, middle cerebral artery; **TCD**, transcranial Doppler; **PSV**, peak systolic velocity; **MV**, mean velocity; **EDV**, end-diastolic velocity; **PI**, pulsatility index; **HITS**, high-intensity transient signals; **RCA**, right coronary embolism; **HA**, hepatic artery; **CWD**, continuous-wave Doppler. Adapted by permission of Taylor and Francis Group, LLC, a division of Informa plc, from: Denault A, Vegas A, Lamarche Y, Tardif J, Couture P. Basic Transesophageal and Critical Care Ultrasound. Taylor and Francis, CRC Press, Boca Raton, FL. 2018.¹²

dysfunction after cardiac surgery. Beyond direct visualization of air emboli in the coronary artery, ST elevation in the inferior lead on weaning from cardiopulmonary bypass with simultaneous hyperintensity transient signals in the middle cerebral artery on transcranial Doppler^{25,26} and in the hepatic artery may also be suggestive of such a mechanism.

In conclusion, we are currently entering an era of precision right heart monitoring. This monitoring integrates RV and pulmonary artery pressure monitoring, 3-dimensional volumetric and strain assessments, assessment of congestive end-organ repercussion, as well as perioperative monitoring for air embolic burden.

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