The right ventricle after cardiac surgery: Hypotheses, evidence, and the role of advanced echocardiography modalities

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Related article
by Wejner-Mik et al.

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For decades, the right ventricle (RV) was considered not essential for cardiac function and therefore it was virtually ignored [1]. The RV, contrarily to the left ventricle, works under low-pressure high-volume physiology, with a wide degree of adaptation to altered preload. The RV comprises three compartments, the inlet, the trabeculated apex, and the outlet allowing contraction to occur with a peristaltic-like motion from the inflow to the outflow chamber [2].

The relation of the RV function to symptom occurrence and prognosis in a wide variety of cardiac conditions emphasizes the usefulness of comprehensive RV assessment. Notably, isolated RV dysfunction is associated with a significantly poorer prognosis as compared to left ventricular dysfunction [3, 4]. The evaluation of the RV is largely carried out by conventional 2D echocardiography in daily clinical practice; however, RV assessment might be difficult owing to its complex morphology, structure, and function. Recent modalities in echocardiography such as myocardial deformation, three-dimensional imaging, or exercise echocardiography are needed to overcome the potential limitations inherent in two-dimensional imaging [5]. In addition, cardiac magnetic resonance imaging provides a valuable opportunity to image the RV without the limitation of echogenicity.

In the present issue of the *Kardiologia Polska* (*Kardiol Pol*, *Polish Heart Journal*), Paulina Wejner-Mik et al. [6] describe interesting echocardiographic observations regarding alterations of RV morphology and function after cardiac surgery, with emphasis on speckle tracking and 3D echocardiography benefit in this context. The authors observed reversible changes in the geometry of the RV, reduced longitudinal right ventricular function after uncomplicated cardiac surgery, and a simultaneous compensatory increase in other components of RV function, namely the RV shortening fraction.

When RV dysfunction occurs during the postoperative phase of cardiac surgery, it represents a significant clinical challenge because of the high prevalence of morbidity and mortality [7]. Cardiac imaging by echocardiography during the postoperative phase is often restricted owing to mediastinal air, drains, dressings, patient noncompliance with supine position along with possible artificial ventilation. In addition, echocardiography examinations in the postoperative period are sometimes performed as point-of-care ultrasound (POCUS), sometimes via a portable or handheld ultrasound machine. Accordingly, RV dysfunction in the postoperative phase after cardiac surgery might well be underdiagnosed [7].

Shortening fraction is an echo parameter classically applicable for assessment of left ventricular function. It was used by Paulina Wejner-Mik et al. [6] to document a compensatory increase in the non-longitudinal component of the RV function. RV shortening fraction in the paper was calculated as the percentage shortening of the mid-cavity linear dimension of the RV in the 4-chamber apical view. Reduction in RV longitudinal function post-cardiac surgery was already reported, and a compensatory increase in transverse

strain also was documented in this context [8, 9]. The authors showed that global RV function was not affected, using 3D RV ejection fraction, RV indexed stroke volume, and fractional area change.

The micro-anatomy of the RV shows that myocytes are predominantly oriented in the longitudinal direction in the subendocardial layer, whereas circumferentially oriented myocytes are found in the thinner subepicardium; accordingly, the RV contraction pattern is predominantly longitudinal [10]. As a consequence, the RV output is mostly engendered by longitudinal contraction in physiological conditions.

Wejner-Mik et al. [6] used the RV shortening fraction as a simple surrogate of transverse strain to assess the non-longitudinal component of RV function, and this is a noteworthy approach when transverse and radial strain imaging is not pertinent (the authors mentioned in the Limitation section that transverse strain was not performed).

Mechanisms of RV dysfunction after cardiac surgery are multifactorial, ranging from per-operative myocardial depression to factors affecting afterload or preload, along with potential effects of inflammatory cytokines on endothelial function, etc. [10]. Nevertheless, in uncomplicated cardiac surgery, like in the studied population presented by Wejner-Mik et al. [6], we estimate that the impact of these factors is absent or minimal. Possible hypotheses that explain the reduction in RV longitudinal performance include the postoperative geometrical changes of the RV chamber related to the interventricular septal paradoxical motion and the drop in ventricular interdependence [11].

The so-called ventricular interdependence is explained by the basic concept that the size, morphology, and function of one ventricle affect the other and vice versa, both in systole and diastole. Notably, diastolic ventricular interdependence is mainly due to the pericardium, and systolic interdependence is mainly mediated by the interventricular septum [12]. For example, left ventricular stroke volume ultimately matches with RV preload; in this regard, it is estimated that 20%–40% of RV stroke volume is dependent on left ventricular contraction [13]. This interdependence is related to the pericardium that embraces both ventricles, the continuity between RV and LV myocardial fibers and muscular layers, the pulmonary and systemic circulation, the shared blood supply, and the interventricular septum [12].

In the article authored by Paulina Wejner-Mik et al. [6], the causes of change in the RV architecture and morphology were not quite elucidated. The 3D echocardiography did not show any change in RV volume in the postoperative phase, therefore, the change in RV geometry was not related to any change in preload a priori. Because of that, we hypothesize that changes in RV geometry are correlated with the drop in ventricular interdependence, namely the architectural changes consequent on the paradoxical septal motion and the reduced pericardial constraint after cardiac surgery [12].

The message reinforced by Paulina Wejner-Mik et al. [6] is that the assessment of RV function post-cardiac surgery should not rely only on parameters for longitudinal assessment (e.g., tricuspid annular longitudinal excursion, the systolic velocity of the tricuspid annulus, free wall longitudinal strain). Relying exclusively on longitudinal contraction after cardiac surgery might lead to overdiagnosis of global RV dysfunction. Moreover, after cardiac surgery, the RV should be regarded differently, consequent on the drop in ventricular interdependence, and therefore should be assessed differently.

Article information

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REFERENCES

1. Jurcut R, Giusca S, La Gerche A, et al. The echocardiographic assessment of the right ventricle: what to do in 2010? Eur J Echocardiogr. 2010; 11(2): 81–96, doi: 10.1093/ejechocard/jep234, indexed in Pubmed: 20124362.
2. Via G, Tavazzi G, Bendjelid K. The right ventricle after cardiopulmonary bypass: new insights on its adaptive physiology. J Clin Monit Comput. 2020; 34(6): 1133–1134, doi: 10.1007/s10877-020-00497-9, indexed in Pubmed: 32157492.
3. Bootsma IT, Scheeren TWL, de Lange F, et al. Impaired right ventricular ejection fraction after cardiac surgery is associated with a complicated ICU stay. J Intensive Care. 2018; 6: 85, doi: 10.1186/s40560-018-0351-3, indexed in Pubmed: 30607248.
4. Lella LK, Sales VL, Goldsmith Y, et al. Reduced right ventricular function predicts long-term cardiac re-hospitalization after cardiac surgery. PLoS One. 2015; 10(7): e0132808, doi: 10.1371/journal.pone.0132808, indexed in Pubmed: 26197273.
5. Surkova E, Peluso D, Kasprzak JD, et al. Use of novel echocardiographic techniques to assess right ventricular geometry and function. Kardiol Pol. 2016; 74(6): 507–522, doi: 10.5603/KP.a2016.0041, indexed in Pubmed: 27040010.
6. Wejner-Mik P, Kasprzak JD, Szymczyk E, et al. Complexity of changes in right ventricular morphology and function in patients undergoing cardiac surgery - 3D echocardiographic study. Kardiol Pol. 2021; 80(2): 156-162, doi: 10.33963/KP.a2021.0195, indexed in Pubmed: 34970985.
7. Estrada VH, Franco DL, Moreno AA, et al. Postoperative right ventricular failure in cardiac surgery. Cardiol Res. 2016; 7(6): 185–195, doi: 10.14740/cr500e, indexed in Pubmed: 28197291.
8. Korshin A, Grønlykke L, Holmgaard F, et al. Right ventricular transverse displacement increases following cardiac surgery: possibly compensating loss in tricuspid annular plane systolic excursion (TAPSE). J Clin Monit Comput. 2020; 34(6): 1139–1148, doi: 10.1007/s10877-020-00466-2, indexed in Pubmed: 31983013.
9. Grønlykke L, Korshin A, Holmgaard F, et al. Severe loss of right ventricular longitudinal contraction occurs after cardiopulmonary bypass in patients with preserved right ventricular output. Int J Cardiovasc Imaging. 2019; 35(9): 1661–1670, doi: 10.1007/s10554-019-01616-7, indexed in Pubmed: 31049752.
10. Levy D, Laghlam D, Estagnasie P, et al. Post-operative right ventricular failure after cardiac surgery: a cohort study. Front Cardiovasc Med. 2021; 8: 667328, doi: 10.3389/fcvm.2021.667328, indexed in Pubmed: 34195233.
11. Tamborini G, Muratori M, Brusoni D, et al. Is right ventricular systolic function reduced after cardiac surgery? A two- and three-dimensional echocardiographic study. Eur J Echocardiogr. 2009; 10(5): 630–634, doi: 10.1093/ejechocard/jep015, indexed in Pubmed: 19252190.
12. Mandoli GE, Cameli M, Novo G, et al. Right ventricular function after cardiac surgery: the diagnostic and prognostic role of echocardiography. Heart Fail Rev. 2019; 24(5): 625–635, doi: 10.1007/s10741-019-09785-2, indexed in Pubmed: 30982175.
13. Santamore WP, Dell'Italia LJ, Heerdt PM, et al. Left and right ventricular systolic function and exercise capacity with coronary artery disease. Am J Cardiol. 1991; 67(13): 1079–1083, doi: 10.1016/0002-9149(91)90869-m, indexed in Pubmed: 2024597.