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Letters to the Editor

Intra-aortic balloon pump as a vent in VA-ECMO; lower risk, but beware

To the Editor,

I read with great interest the recent expert review in which the authors discussed left ventricular (LV) decompression strategies in patients supported by venoarterial extracorporeal membrane oxygenation (ECMO).[1] Left ventricular decompression is essential for the prevention of increased LV pressure with ensuing stasis, left atrial distention, pulmonary edema, pulmonary hemorrhage, ventricular arrhythmias, and LV and aortic root clot formation.[2] The LV venting strategies vary by institution, but usually begin with modification of ECMO flows, pharmacologic interventions on preload, contractility and afterload, and changes in positive end-expiratory pressure.[3] If these methods are unsuccessful, other interventions may be necessary to avoid the complications of LV distention.[3] Of particular interest and controversy as a venting strategy is the intra-aortic balloon pump (IABP). As stated by the authors, the IABP is placed through a small arteriotomy, is rapidly deployable, and has the added benefit of not being intraventricular.[4] The IABP is a potentially ideal method of mechanical LV decompression in patients who have mechanical aortic valves or LV thrombus burden, situations in which Impella use is contraindicated.[5,6]

It is important to remember that some intrinsic LV function must be present for the IABP to provide support. This contrasts to transvalvular LV assist devices (ie, Impella), which do not require intrinsic myocardial function to decompress the LV. This is especially important in a patient supported by venoarterial-ECMO, because an IABP will give the appearance of pulsatility on an arterial pressure waveform regardless of whether the aortic valve is opening.[7] Thus, it is important to be vigilant for increases in pulmonary capillary wedge pressure, pulmonary edema, and ventricular arrhythmias as clues for insufficient venting. One method of quickly deciding if the aortic valve is opening is very briefly setting the IABP to standby and examining the arterial pressure waveform for pulsatility.[7] If the arterial pressure waveform is flat with the IABP on standby, and the patient is otherwise optimized, suspicion should be high for inadequate venting of the LV (or impending worsening LV failure), and echocardiographic assessment should be considered with the IABP active to assess for aortic valve opening, LV stasis, and aortic root thrombus. Importantly, if the aortic valve is only opening with IABP use, it may be wise to upgrade support to avoid further myocardial decompensation and subsequent LV stasis.

Declaration of Competing Interest

None.

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Correlations of Before and After Event Echocardiographic Parameters with Troponin and BNP in Hospitalized COVID-19 Patients With Cardiovascular Events

To the Editor:

Right and left ventricular (RV and LV, respectively) dysfunction was found to be significantly related to adverse outcomes in patients with COVID-19.[1-7] There is no study to specifically evaluate echocardiography in the cohort of patients who suffered cardiovascular events. We examined the echocardiographic parameters before and after cardiovascular events to predict myocardial injuries so that clinicians could be better prepared to manage pending cardiovascular events or their sequalae.

Forty-nine patients from a multicenter, retrospective database of 700 adult patients hospitalized with a diagnosis of COVID-19 from March 7, 2020, to July 1, 2020 who suffered ≥1 cardiovascular event and underwent echocardiography were selected.[8,9] Cardiovascular events included heart failure, cardiogenic shock, acute myocardial infarction, cardiomyopathy, myocarditis, cardiac arrhythmias, cerebrovascular events, pulmonary embolism, pulmonary edema, deep vein thrombosis, and cardiac arrest. Clinical diagnoses of cardiovascular events were made by individual physicians at each site. In this cohort, 30 patients (61%) had echocardiography performed before their cardiac event, and 19 (39%) had echocardiography performed after their cardiac event. Our endpoints of analysis were admission troponin, peak troponin, and brain natriuretic peptide (BNP) concentrations. The echocardiographic parameters and laboratory values were stratified by the timing of the echocardiography. Correlations among the before-event echocardiographic parameters and admission troponin are detailed in Table 1. Significant correlations were found among LV ejection fraction, LV volume, mitral valve E/E', and admission troponin. Correlations among the before-event echocardiographic parameters and BNP also are shown in Table 1. No before-event echocardiographic parameters were found to have significant correlations with BNP. Correlations of the after-event echocardiographic parameters with peak troponin and BNP are detailed in Table 2. A significant correlation was found between the RV diameter and peak troponin (r = 0.98; p = 0.019); however, no after-event echocardiographic parameters were found to be correlated significantly with BNP. Thus, LV systolic and diastolic dysfunction (indicated by an increased mitral valve E/E' ratio) before a cardiovascular event were correlated with initial myocardial injury levels as indicated by admission troponin. An increase in RV size on after-event echocardiography was correlated with maximal myocardial injury, as indicated by peak troponin in COVID-19 patients.

Table 1
Pearson Correlations Between Before-event Echocardiographic Parameters and Log Admission Troponin and Log BNP

	Log Troponin		Log BNP	
Variable	r*	p Value	r*	p Value
LV ejection fraction, %	−0.68	< 0.001	−0.14	0.51
LV volume, mL	0.45	0.039	0.04	0.87
LA volume, mL	−0.34	0.14	−0.04	0.89
RV diameter, cm	0.25	0.28	0.42	0.08
RA volume, mL	0.24	0.27	0.18	0.45
Ascending aorta size, cm	0.04	0.91	−0.46	0.35
Peak E-wave, m/s	−0.07	0.74	−0.35	0.09
TV TAPSE	0.07	0.75	−0.05	0.84
Estimated RVSP, mmHg	−0.15	0.59	0.16	0.54
Estimated RAP, mmHg	0.14	0.60	0.32	0.21
MV E' Lateral Velocity, m/s	−0.11	0.60	−0.08	0.71
MV E/E' Lateral	0.43	0.038	0.08	0.72

Abbreviations: BNP, brain natriuretic peptide; LA, left atrium; LV, left ventricle; MV, mitral valve; RA, right atrium; RAP, right atrial pressure; RV, right ventricle; RVSP, right ventricle systolic pressure; TAPSE, tricuspid annular plane systolic excursion; TV, tricuspid valve.

* Pearson correlation coefficient.

Table 2
Pearson Correlations Between After-event Echocardiographic Parameters and Log Peak Troponin and Log BNP

	Log Troponin		Log BNP	
Variable	r*	p Value	r*	p Value
LV ejection fraction, %	−0.20	0.61	−0.41	0.11
LV volume, mL	−0.65	0.11	0.29	0.36
LA volume, mL	−0.62	0.38	−0.08	0.86
RV diameter, cm	0.98	0.019	0.55	0.10
RA volume, mL	0.32	0.54	0.22	0.51
Ascending aorta size, cm	−0.42	0.31	−0.39	0.13
Peak E-wave, m/s	−0.79	0.42	0.10	0.81
TV TAPSE	0.03	0.96	−0.31	0.39
Estimated RVSP, mmHg	−0.06	0.89	−0.35	0.24
Estimated RAP, mmHg	−0.29	0.63	−0.31	0.33
MV E' Lateral Velocity, m/s	0.80	0.10	0.51	0.13
MV E/E' Lateral	−0.20	0.61	−0.41	0.11

Abbreviations: BNP, brain natriuretic peptide; LA, left atrium; LV, left ventricle; MV, mitral valve; RA, right atrium; RAP, right atrial pressure; RV, right ventricle; RVSP, right ventricle systolic pressure; TAPSE, tricuspid annular plane systolic excursion; TV, tricuspid valve.

* Pearson correlation coefficient.

The novelties of this current study included the identification of before-event echocardiographic parameters, which correlated with initial myocardial injury levels, and the discovery of after-event echocardiographic parameters, which correlated with maximal myocardial injury levels. Changes in these echocardiographic parameters should alert physicians to imminent hemodynamic instability and mortality.

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Conflict of Interest

None.

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Comparison of Residual Tricuspid Regurgitation Severity Assessed by Intraprocedural and Postprocedural Echocardiography in Patients Undergoing Transcatheter Tricuspid Valve Repair

To the Editor:

TRANSCATHETER TRICUSPID valve repair (TTVr) using edge-to-edge clipping devices has emerged recently to treat high-risk symptomatic patients with severe tricuspid regurgitation (TR).[1] Inaccurate grading of TR severity during the procedure may result in suboptimal results, leading to significant residual TR. Thus, an accurate estimation of intraprocedural TR is essential. Because systemic venous return and cardiac output are frequently decreased during anesthesia,[2] we hypothesized that residual TR during the procedure would be underestimated. This study compared the severity grades and color jet area (CJA) of residual TR, as assessed by the intraprocedural transesophageal echocardiography (TEE) and post-procedural transthoracic echocardiography (TTE), in patients undergoing TTVr. Patients who underwent isolated TTVr using the MitraClip system (Abbott Vascular) in our institution between January 2017 and November 2021 were included. Although baseline and postprocedural TTE were performed under conscious conditions, intraprocedural TEE was conducted under general anesthesia during mechanical ventilation. The baseline, intraprocedural, and postprocedural TR grades were collected from the echocardiography reports. The TR grades were classified by experienced cardiologists based on a multiparametric integrative approach in accordance with current guidelines,[3-5] as none/trace, mild, mild-to-moderate, moderate, moderate-to-severe, severe, and massive/torrential. The CJA of TR was measured by tracing the largest jet area in mid-systole on color Doppler images, with an aliasing velocity of 50-to- 60 cm/s from the apical 4-chamber or right ventricle (RV) inflow view on TTE and the midesophageal four-chamber or RV inflow-outflow view on TEE.

Ninety-two patients were reviewed retrospectively. The etiology of TR was functional (n = 70), pacemaker lead-related (n = 16), and degenerative (n = 6). The number of implanted clips was 2.2 ± 0.8. The median time interval between

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