Case report: paradoxical ventricular septal motion in the setting of primary right ventricular myocardial failure

Présentation de cas: mouvement paradoxal du septum interventriculaire dans le contexte d'une défaillance myocardique primaire du ventricule droit

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Abstract

Purpose *In this report, a case of right ventricular (RV) failure, hemodynamic instability, and systemic organ failure is described to highlight how paradoxical ventricular systolic septal motion (PVSM), or a rightward systolic displacement of the interventricular septum, may contribute to RV ejection.*

Clinical features *Multiple inotropic medications and vasopressors were administered to treat right heart failure and systemic hypotension in a patient following combined aortic and mitral valve replacement. In the early postoperative period, echocardiographic evaluation revealed adequate left ventricular systolic function, akinesis of the*

RV myocardial tissues, and PVSM. In the presence of PVSM, RV fractional area of contraction was $\geq 35\%$ despite akinesis of the primary RV myocardial walls. The PVSM appeared to contribute toward RV ejection. As a result, the need for multiple inotropes was re-evaluated, in considering that end-organ dysfunction was the result of systemic hypotension and prolonged vasopressor administration. After discontinuation of phosphodiesterase inhibitors, native vascular tone returned and the need for vasopressors declined. This was followed by recovery of systemic organ function. Echocardiographic re-evaluation two years later, revealed persistent akinesis of the RV myocardial tissues and PVSM, the latter appearing to contribute toward RV ejection.

Conclusions *This case highlights the importance of left to RV interactions, and how PVSM may mediate these hemodynamic interactions.*

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Résumé

Objectif *Dans ce compte-rendu, un cas de défaillance ventriculaire droite (VD), d'instabilité hémodynamique et de défaillance organique systémique est décrit afin de souligner la manière dont le mouvement systolique paradoxal du septum interventriculaire (PVSM), ou un déplacement systolique de la cloison interventriculaire vers la droite, peut jouer un rôle dans l'éjection du VD.*

Éléments cliniques *De nombreux médicaments inotropes et vasopresseurs ont été administrés pour traiter une défaillance cardiaque droite ainsi qu'une hypotension systémique chez un patient à la suite d'une chirurgie valvulaire aortique et mitrale combinée. Au début de la période postopératoire, l'évaluation échocardiographique a révélé une fonction systolique adéquate du ventricule*

gauche, une akinésie des tissus myocardiques du VD, et un PVSM. En présence du PVSM, la fraction de contraction du VD était de $\geq 35\%$ malgré l'akinésie des parois myocardiques primaires du VD. Par conséquent, les prestataires de soins ont réévalué le besoin d'agents inotropes multiples et émis l'hypothèse que la dysfonction des organes résultait de l'hypotension systémique et de l'administration prolongée de médicaments vasopresseurs. Après avoir interrompu l'administration d'inhibiteurs de la phosphodiesterase, le tonus vasculaire est redevenu normal et les besoins en médicaments vasopresseurs ont diminué. Une récupération de la fonction des différents organes s'est ensuivie. Une réévaluation échocardiographique deux ans plus tard a révélé une akinésie persistante des tissus myocardiques du VD et un PVSM, lequel semblait contribuer à l'éjection VD.

Conclusion *Ce cas souligne l'important des interactions entre les ventricules gauche et droit et la façon dont le PVSM pourrait les médier.*

Since the early 1940s, it has been known that impairment or complete abolition of RV free wall contraction produces only modest effects on systemic hemodynamics when right ventricular (RV) afterload is low.^{1,2} Over the last 60 years, both experimental data and clinical experience show that a significant portion of the RV systolic pressure is a result of the left ventricle (LV) generating pressure that is transmitted through the interventricular septum (IVS).^{3–6} This left to right “ventricular assistance” is influenced by RV volume and pressure and the transeptal pressure gradient.^{7,8} The clinical impact of this interaction increases with increasing RV dysfunction.^{4–6,8–11} A number of investigations describe this interventricular interaction in terms of the IVS position, highlighting its impact on both LV and RV functions.^{4–6,8–11}

With the patient's informed consent, we present a case of severe primary RV failure that was successfully resuscitated with no significant improvement in the systolic function of the primary RV myocardial tissues. Of note, was the occurrence of paradoxical ventricular systolic septal motion (PVSM), during which the IVS moved toward the RV free wall during systole and appeared to contribute substantially to RV ejection. From this point forward, PVSM will refer to rightward movement of the IVS during ventricular systole.

Case report

A 54-year-old male was scheduled for a repeat sternotomy for replacement of both the aortic valve (AVR; AV stenosis) and the mitral valve (MVR; mitral regurgitation), as well as for tricuspid valve repair (TVR; tricuspid valve

regurgitation). Six years earlier, the patient underwent a two-vessel coronary artery bypass grafting (CABG). During the procedure, the left internal mammary artery was used to bypass a diseased left anterior descending artery (LAD), and a segment of saphenous vein (SVG) was used to bypass a lesion in the second obtuse marginal artery (OM2). The right coronary artery was totally occluded proximally and was filled distally by collateral flow from the LAD. Prior to the first cardiac surgical procedure, the patient had an inferior wall myocardial infarction, which was complicated by RV dysfunction and a presumed infarction. Prior to the current hospitalization, preoperative coronary angiography showed that both bypasses were patent, and there was good collateral flow from the LAD to the right coronary artery. No new coronary lesions were found, nor were any bypass grafts planned.

The preoperative echocardiogram reported an LV ejection fraction (LVEF) of 50% with inferior and septal wall hypokinesis and moderate global RV hypokinesis. The estimated pulmonary artery peak systolic pressure was 35 mmHg.

The baseline electrocardiogram (ECG) revealed a sinus rhythm with a left bundle branch block (LBBB) at a rate of 80 beats/min. There was no significant baseline pulmonary dysfunction. The preoperative blood urea nitrogen (BUN) and serum creatinine were 15.4 mmol/L and 140.8 μ mol/L, respectively.

Following an uneventful induction of general anesthesia and endotracheal intubation, a pulmonary artery catheter (PAC) and transesophageal echocardiography (TEE) probe were placed. Hemodynamic data are presented in Table 1. Pre-cardiopulmonary bypass (CPB) TEE confirmed preoperative echocardiographic findings. In addition, the tricuspid annular plane systolic excursion (TAPSE) was measured to 1.4 cm (normal >1.8–2.0 cm), which is consistent with reduced RV systolic function. Otherwise, the position, shape, and movement of the IVS were considered normal (with the exception of the hypokinetic inferior septal wall). The RV appeared to be mildly enlarged, based on measurement of the RV short-axis diameter relative to the LV short-axis diameter (>0.5); however, RV volumes were not quantitatively assessed.

Prior to median sternotomy, the patient was placed on CPB via femoral artery and venous cannulae. Placing patients on CPB prior to median sternotomy has become standard practice at our institution. The sternotomy was complicated by lacerations of the SVG and the RV free wall. The surgical procedure included placement of a 21 mm St Jude mechanical AVR and a 29 mm St Jude mechanical MVR, as well as DeVega repair of the tricuspid valve, CABG $\times 1$ to the OM2, and repair of the RV free wall tear. Total CPB time was 362 min, and time for the aortic cross-clamp was 203 min.

Table 1 Hemodynamic and laboratory data for the case presented during the perioperative and postoperative period along with are the vasoactive medications administered to maintain hemodynamic data

	Pre CPB	Post CPB	POD 1	POD 3	POD 4	POD 6	POD 9	POD 21	Two years
HR/pacing mode	70SR	90DOO	100DOO	100DDD	100DDD	100DDD	85DDD	80DDD	80DDD
BP, mmHg	100/60	95/60	100/60	95/50	100/50	124/52	145/58	110/55	
CVP, mmHg	8	19	18	18	18	15	13		
PAP, mmHg	24/15	30/18	30/19	33/22	32/23	30/16	30/15		
PCWP, mmHg		16	15	16	16	14	14		
CO/CI, L/min; L/min · m ²	3.4/1.7	2.9/1.5	2.5/1.3	5.0/2.6	5.1/2.6	5.1/2.6	4.5/2.3		
MvO ₂ %		63	60	62	64	65	65		
SVR dynes/s · cm ⁻⁵		1517	1760	780	741	957	1546		
PVR dynes/s · cm ⁻⁵		166	256	148	157	110	124		
Vasoactive		Vasopress 0.1 units/min	Vasopress 0.1 units/min	Vasopress 0.1 units/min	Dopa 5 µg/kg/min	Dopa 5 µg/kg/min	Dopa 5 µg/kg/min		
Medications		Dopa 5 µg/kg/min	Dopa 5 µg/kg/min	Dopa 5 µg/kg/min	Epi 3 µg/min	Epi 3 µg/min	Epi 3 µg/min		
		Epi 3 µg/min	Epi 3 µg/min	Epi 3 µg/min	NTG 15 µg/min	NTG 20 µg/min	Lasix 2 mg/h		
		Norepi 4 µg/min	Norepi 8 µg/min	Norepi 6 µg/min					
		NTG 15 µg/min	NTG 15 µg/min	NTG 15 µg/min					
		Amrin 5 µg/kg/min	Amrin 5 µg/kg/min	Milrin 0.5 µg/kg/min					
		NTG 20 µg/min	Flolan® 55 ng/kg/min	Flolan® 55 ng/kg/min					
Platelets xK/mL	205,000	92,000	61,000	44,000	26,000	103,000	133,000	442,000	
BUN/Creat (mg/dL)	43/1.6	24/1.7	38/3.5	79/6.9	94/7.7	57/3.4	42/2.5	50/2.5	45/2.0

Amrin amrinone, *BP* systemic blood pressure, *BUN* blood urea nitrogen, *Creat* creatinine, *CVP* central venous pressure, *CO/CI* cardiac output/cardiac index, *DDD* pacemaker demand mode, *DOO* pacemaker asynchronous mode, *Dopa* dopamine, *Epi* epinephrine, *Milrin* milrinone, *MvO₂* mixed venous oxygen saturation, *Norepi* norepinephrine, *NTG* nitroglycerin, *PAP* pulmonary artery pressure, *PCWP* pulmonary capillary wedge pressure, *POD* postoperative day, *PVR* pulmonary vascular resistance, *SVR* systemic vascular resistance, *Vasopress* vasopressin

In anticipation of significant cardiac dysfunction, intravenous infusions of dopamine (5 µg/kg/min), norepinephrine (0.06 to 0.12 µg/kg/min), and amrinone (5 µg/kg/min; after a 100 mg loading dose) were started prior to weaning from CPB to provide inotropic stimulation and maintenance of systemic blood pressure. Intravenous nitroglycerin was also administered with the hope of reducing RV pressures and wall tension. After the patient was weaned from CPB and the norepinephrine infusion was increased to 0.12 µg/kg/min, vasopressin (0.1 units/min; 0.0014 units/kg/min) and epinephrine infusions (0.05 µg/kg/min) were added to increase systemic blood pressure and myocardial contractility. Hemodynamic goals included a systolic blood pressure of ≥ 90 mmHg (or mean blood pressure ≥ 55 mmHg) and a cardiac index ≥ 2.5 L/min · m². The atrium and ventricle were asynchronously paced at a rate of 90 beats/min. Ventricular pacing wires were placed toward the apical portion of the RV beyond the repaired laceration. Post CPB TEE demonstrated normal functioning prosthetic AV and MV. There was mild-to-moderate tricuspid regurgitation. The LVEF was 45%. The IVS appeared to be akinetic. There was severe RV systolic dysfunction with akinesis of the RV base, free wall, and outflow tissues. Tricuspid annular plane systolic excursion was severely depressed (i.e., <0.5 cm). The RV fractional area of contraction was $<20\%$. Although the RV volume was not measured, the ratio of short-axis diameters of the RV to the LV in the mid-esophageal four-chamber view was greater than 1.0, which is consistent with RV enlargement. The IVS appeared to be flat toward the LV during both systole and diastole, consistent with both pressure and volume overload of the RV. There was no echocardiographic evidence of RV or LV outflow tract obstruction. Hemodynamic data are shown in Table 1. Although an intra-aortic balloon pump was considered, the surgeon was not able to access the femoral arterial system and was not partial to having a balloon pump placed in the pulmonary artery.

On arrival to the intensive care unit, continuous inhaled PGI₂ and an intravenous nesiritide infusion were administered empirically to reduce and/or to attenuate any increases in pulmonary vascular resistance (PVR). The QRS complex on the postoperative electrocardiogram appeared to be similar to that seen prior to surgery. Given the lack of response to intravenous fluid (IVF) administration (decrease or no change in systemic blood pressure and cardiac index and a rise in the central venous pressure, CVP) over the first 10 h, fluid administration was limited with a goal to keep the CVP < 20 mmHg. Over the next 2 days, the patient's kidney and liver function deteriorated, and the serum platelet count declined (Table 1). Due to persistent need for higher dose vasoconstrictors, the nesiritide was discontinued on

postoperative day (POD) one. In the face of thrombocytopenia, the amrinone infusion was replaced with a milrinone infusion. With the exception of the PAC, all heparin was removed. Subsequent assays for the diagnosis of heparin-induced thrombocytopenia were negative. Epinephrine, dopamine, norepinephrine, and vasopressin infusions were continued. Atrio-ventricular sequential pacing continued at a rate of 100 beats/min. On POD three, systemic organ failure worsened, and the patient required hemodialysis (Table 1).

A transthoracic echocardiogram (TTE) was performed on POD three (Fig. 1; Supplementary material—Videos 1 and 2). Examination showed normal prosthetic valve function, mild TR, an LVEF of 45%, and persistent RV wall akinesis (Fig. 1; Supplementary material—Videos 1 and 2). Significant PVSM was noted. Compared to the intraoperative exam, the RV chamber size appeared to be smaller relative to the LV, i.e., the ratio of RV to LV short-axis diameters was <1.0 . The IVS during diastole appeared to be convex toward the LV. Aside from inferior wall hypokinesis, other LV myocardial segments were normal or hyperdynamic. The QRS pattern on the ECG was unchanged from the operating room.

After reviewing the echocardiogram, it was felt that the PVSM contributed toward RV ejection. Although the primary RV myocardial walls were akinetic, from a four-chamber view, the RV fractional area change (RV FAC) was calculated to be 40%.

In light of these data, caregivers questioned the need for multiple inotropic medications and considered that systemic end-organ dysfunction could be the result of prolonged vasopressor therapy, reduced systemic blood pressure, and systemic organ perfusion pressure. The infusion of milrinone was discontinued. Within 16 h, the patient's systemic blood pressure increased, and the vasopressors were weaned to off by POD four. Given the significant improvement in hemodynamic function, inhaled Flolan[®] was discontinued at the end of POD four. Infusions of epinephrine and dopamine were continued. A repeat TTE was not significantly different from the one performed on POD three.

As systemic blood pressure increased and vasopressor therapy declined, the urine output improved. In conjunction with this, the BUN and serum creatinine decreased, the platelet count increased, and liver function tests returned toward normal. Hemodialysis was no longer necessary. The patient was weaned from mechanical ventilation and his trachea extubated on POD six. By POD 11 and 13, epinephrine and dopamine were discontinued. Since the underlying rhythm showed persistent third-degree heart block, a permanent dual-chamber synchronous pacemaker was placed on POD 21. The patient was discharged fully intact on POD 29.

Fig. 1 Images from the postoperative transthoracic echocardiographic exam obtained on postoperative day five. These parasternal long (top three panels—A, B, C) and short (bottom panels—D, E, F) axis views of the left (LV) and right (RV) ventricle demonstrate the abnormal leftward diastolic shift (arrows) of the interventricular septum (IVS), which is convex toward the LV (Panels—A and D).

On two-year follow-up, the patient was in stable condition. The serum creatinine was 176.0 $\mu\text{mol/L}$. The ECG showed a paced rhythm with LBBB. Repeat echocardiogram demonstrated akinesis of the RV myocardium, an LVEF of 45% with significant PVSM, and inferior wall hypokinesis. The overall RV ejection fraction was estimated to be approximately 35–40%.

Discussion

A case of acute almost chronic RV failure is presented in order to describe PVSM as a mechanism by which the left heart contributes toward RV systolic function. The immediate post CPB period was characterized by elevated CVP, relatively low pulmonary artery pressure (PAP), and systemic hypotension requiring multiple vasoactive medications. At this time, PVSM was not considered significant. However, during echocardiographic evaluation 3 days later, significant PVSM was noted and appeared to contribute toward RV ejection. In light of this observation, caregivers reconsidered the regimen of vasoactive medications and speculated that systemic organ dysfunction

This is followed by paradoxical motion of the IVS starting in early systole, with the IVS moving toward the RV and a reduction in RV area (Panels—B and E). This is followed by contractility of the left ventricle, a reduction in the LV area, and a further reduction in the RV (Panels—C and F)

may have been related to low perfusion pressure and prolonged use of vasopressors. Ultimately, the echocardiographic appearance of PVSM and its role in LV–RV interaction altered medical management. The novelty of this case lies in the echocardiographic demonstration of the benefit of PVSM on RV ejection.

Assessing RV function

Echocardiographic evaluation provides information regarding RV chamber size, pressures, and function. Two-dimensional imaging may not provide accurate volume assessment due to the relatively complex three-dimensional structure of the RV; however, the ratio of short-axis dimensions (RV: LV) assessed from four-chamber views is normally <0.5 . Although exact chamber volumes and, therefore, accurate ejection fractions are difficult, it is possible to calculate the fractional area of contraction by tracing the endocardial area and to perform wall motion analyses similar to that of the LV.¹² A TAPSE >1.9 cm is associated with normal RV function, while severe RV failure is noted in patients with a TAPSE <1.4 cm.¹³ In this study, RV function was assessment-based on chamber

diameter, wall motion analysis, calculation of the FAC, and by TAPSE. These data were consistent with severe RV dysfunction. While right heart pressures and diastolic function can be estimated using Doppler echocardiography, these data are not available for the case presented. Hemodynamic data included a relatively high CVP in comparison with the pulmonary artery diastolic pressure, and PCWP. A ratio of the CVP/PCWP > 1 is also consistent with severe right heart dysfunction and hemodynamic instability.¹⁴ In the face of severe RV dysfunction, the generation of pulmonary artery pressures is reduced, perhaps explaining the relatively low PAP in the postoperative period despite extensive left heart surgery.

Managing RV failure

In 1943, Starr *et al.* suggested that the RV can function as a passive conduit to transport blood from the venous system to the low resistance pulmonary vasculature and, subsequently, to the LV.¹ This was supported by the success of procedures designed to bypass the RV, such as the Fontan operation.^{15,16} Based on these reports, treatment of primary right heart failure consisted of volume loading and maintenance of low PVR. However, this may not be effective for all patients with acute RV failure, and excessive IVF may result in further deterioration of heart function.^{4,17} For these patients, RV ejection can be maintained as a result of left-to-right systolic ventricular interactions.^{4–6,18} Experimental data show that these interactions are impaired with increasing RV preload and afterload,⁴ which is consistent with clinical experience.¹⁷ Based on these data, management of RV failure should consider early administration of vasoactive therapy with less aggressive fluid therapy.^{4,9,17,19–21} However, the selection of vasoactive medications needs to be tailored toward each specific situation.

The administration of a phosphodiesterase inhibitor to improve cardiac function and systemic hemodynamics in the face of RV pressure and volume overload has been described.^{22,23} Reductions in systemic blood pressure are known and may require concurrent administration of vasopressors.²³ However, the importance of maintaining systemic blood pressure and, subsequently, right coronary pressure and flow in the face of RV dysfunction may not be as well appreciated.⁹ This has been demonstrated in experimental models of RV failure, where an increase in right coronary flow resulted in improved RV function.^{9,19,20} This benefit was independent of LV cardiac output.⁹ Although the use of vasopressors can be beneficial, its prolonged administration can also contribute to systemic organ failure.²² Specifically, prolonged administration (more than 48 h) of vasopressin has been linked to hepatic failure, renal failure, and thrombocytopenia.²² For the case

presented, systemic organ dysfunction seemed to be related, at least in part, to prolonged administration of vasopressin and not failure of the RV to eject. After considering the benefits of PVSM on right heart ejection, the milrinone infusion was discontinued to allow return of native vasomotor tone.

Paradoxical ventricular septal motion

Paradoxical ventricular systolic septal motion immediately after heart surgery is not uncommon. In the great majority of cases, it is related to either an alteration in the ventricular conduction pattern and/or the release of pericardial restraints after pericardiectomy.^{24–26} In general, PVSM is typically transient, not due to septal ischemia or infarct, and resolves within the first year as pericardial adhesions form or as conduction abnormalities resolve. For most cases, PVSM offers no significant benefit in the patient with otherwise normal heart function. Instead, it is likely to be associated with reduced LV performance.^{28,29} For the case presented, PVSM could have been related to release of pericardial constraints and/or abnormal inter-ventricular conduction patterns. However, based on intraoperative TEE, significant PVSM did not occur immediately but was noted 72 h after surgery in the setting of persistent RV myocardial akinesis, suggesting that the changes in the transeptal systolic pressure gradient contributed significantly toward its development. Furthermore, follow-up examination demonstrated persistent PVSM and severe RV myocardial wall motion abnormalities 2 years later. During this time period, there were no gross changes in the QRS configuration seen on ECG; however, no formal electrophysiologic study was performed to delineate specific conduction patterns.

LV–RV interaction and the IVS

A number of experimental studies have demonstrated the importance of the IVS in mediating LV–RV interaction.^{4,6,8–11} In an acute RV free wall infarct model, elevated RV pressure and volume throughout the cardiac cycle caused a leftward shift of the IVS, resulting in both LV diastolic and systolic dysfunction.⁸ Right ventricular preload reduction using a Glenn Shunt reduced RV volume and pressure, normalized the IVS position during diastole, and increased LV filling and ejection and systemic blood pressure.⁸

In another RV infarct model using different loading conditions, the IVS was noted to paradoxically ‘bulge’ toward the RV free wall at the onset of ventricular systole (PVSM) causing an increase in RV dP/dt, a reduction in RV volume, and forward ejection of blood from the RV.^{10,11,30} These authors speculated that the systolic

pressure increase in the normally functioning LV was unopposed by the reduced pressure generation in the depressed RV, thereby increasing the left-to-right pressure gradient across the IVS. This resulted in a rightward systolic shift of the contracting IVS (PVSM) toward the RV free wall.^{10,11} The reduction in the distance between the IVS and the RV free wall increases RV dP/dt, RV ejection, and pulmonary blood flow, despite RV free wall akinesis.^{6,10,11,18}

The motion of the IVS can also be affected by abnormal or altered inter-ventricular conduction patterns, as seen in the first case. While inter-ventricular conduction delays and PVSM are not beneficial for patients with reduced LVEF, they may have a positive effect for patients with primarily RV failure. In several animal models of RV volume^{31,32} or pressure³³ overload, pacing-induced inter-ventricular conduction delays improved right heart function and systemic hemodynamics.^{31–33} Although it is not definitively clear whether it is better to pace the RV or the LV first, it is evident that nonsimultaneous pacing improves cardiac hemodynamics in the face of RV dysfunction.^{31–33} Clinical benefits were demonstrated for patients with right heart dysfunction^{34–36} with or without a pre-existing right bundle branch block.^{34,35} For those with a right bundle branch block, pacing the RV first narrowed the QRS and improved RV dP/dt and systemic hemodynamics.^{34,35}

Importantly, the benefit of the IVS is due not only to movement but also to contractility and the overall systolic performance of the LV, which, when increased, can produce a more vigorous movement of the IVS toward the right. Inactivation or akinesis of the IVS in models of RV dysfunction results in a 30–40% reduction in RV performance (RV dP/dt).^{9,10} In this experiment, a dopamine infusion increased LV contractility and generated LV systolic pressure. As a result, there was increased rightward systolic motion of the akinetic IVS with significantly greater RV dP/dt and stroke work.¹⁰

Taken together, both experimental and clinical data show the effects of cardiac loading conditions on trans-septal pressure gradients and subsequent shifts in IVS position. In the face of acute right heart failure, a reduction in RV preload and pressure not only improves left heart function³ but may promote PVSM^{10,11} and improve RV ejection.^{6,10,11,18,30} Although past opinion suggested that aggressive fluid therapy was a most important component of therapy for RV failure, more recent data support a multimodal approach directed at controlling or even reducing RV volume and pressure and reducing PVR. Attempts to increase RV contractility are potentially beneficial, as long as systemic blood pressure and right coronary perfusion are not compromised.^{9,19,20,23,30} As seen in the present case, the primary RV tissues may not

have significant reserve function for their contractility to improve. Therefore, contribution of the LV toward RV function becomes more significant. Reducing the loading conditions on the RV, while increasing the contractility and pressure generated by the LV, improves left-to-right assistance and should be considered when managing severe RV failure.^{6,7,10,11,30}

In conclusion, this case highlights the difficulties in managing patients with severe RV dysfunction. Evaluation with echocardiography and an understanding of the physiologic principles of right and left heart interaction can help guide management for such patients. As described experimentally in previous case reports and in the case presented, PVSM and left heart function significantly contribute toward RV ejection, especially in the presence of primary RV failure. For the patient with severe RV failure, manipulations of right heart loading conditions, maintenance of LV systolic function, and PVSM have therapeutic benefits.

Conflicts of interest None declared.

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