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RESEARCH ARTICLE

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Assessing the optimal MAP target in pre-capillary PH patients with RV failure: A retrospective analysis

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Abstract

Right ventricular failure (RVF) in pre-capillary pulmonary hypertension (PH) is associated with high morbidity and mortality. While mean arterial pressure (MAP) goals have been well established in critical care literature, the optimal MAP target for patients with RVF secondary to pre-capillary PH remains unknown. The objective of this study was to evaluate the difference in outcomes between patients who were managed with different MAP targets. We retrospectively analyzed records of 60 patients who were admitted to the intensive care unit for decompensated RVF secondary to pre-capillary PH. The records were stratified into two groups: 30 patients who were treated with a static MAP goal of either 65 or 70 mmHg ($MAP_{65/70}$) and 30 patients who received a dynamic MAP goal (MAP_{CVP}) determined by invasively obtained central venous pressure or right atrial pressure. The dynamic MAP group had a statistically significant decrease in in-hospital mortality and incidence of acute kidney injury compared to the static MAP cohort.

KEYWORDS
acute kidney injury, cardiogenic shock, central venous perfusion, pulmonary arterial hypertension, right ventricular failure

BACKGROUND

Pre-capillary pulmonary hypertension (PH) is a progressive disease with high morbidity and mortality. The right ventricle (RV) is constantly adapting to the increased afterload that defines PH, and RV function is a major determinant of prognosis and functional capacity.[1] Patients with PH often require hospitalization during the course of their disease as the RV ultimately decompensates, becoming dilated, hypokinetic and fibrotic.[1] RV failure (RVF) requiring admission to an intensive care unit (ICU) has an inpatient mortality rate

Abbreviations: CI, cardiac index; CO, cardiac output; CVP, central venous pressure; eRVSP, estimated right ventricular systolic pressure; ICU, intensive care unit; IQR, interquartile range; KDIGO, Kidney Disease Improving Global Outcomes; LV, left ventricle; MAP, mean arterial pressure; PAC, pulmonary artery catheter; PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; RAP, right atrial pressure; RHC, right heart catheterization; RRT, renal replacement therapy; RV, right ventricle; RVF, right ventricle failure; $ScvO_2$, central venous oxygen saturation; SD, standard deviation; SPP, systemic perfusion pressure; SvO_2, venous oxygen saturation; TAPSE, tricuspid annular plane systolic excursion; TLC, triple lumen catheter; TR, tricuspid regurgitation; TTE, transthoracic echocardiogram.

of over 40%, making it the leading cause of death in PH patients. Management requires optimization of RV preload, afterload, and contractility.[2] Invasive hemodynamic monitoring via a pulmonary artery catheter (PAC) and/or clinical and laboratory findings, such as creatinine and lactate, paired with jugular venous pressure on physical exam, can guide treatment. The management of RVF and subsequent shock is complicated, and there is considerable overlap in management strategies in patients with left ventricular (LV) failure as well.[2] There are currently no guidelines for what the optimal perfusion target is in patients with a failing RV. Establishing an adequate mean arterial pressure (MAP) in patients with decompensated RVF becomes essential as it promotes perfusion to the RV myocardium, perfuses the kidneys by maintaining systemic perfusion pressure (SPP), increases LV afterload and reduces the amount of right-to-left shunting in patients with a patent foramen ovale.[3–6] However, the optimal MAP target has not been well established, and there is no substantial evidence to suggest an exact goal. While a MAP target of 65 mmHg is universally used for all forms of shock, the literature behind this target predominantly comes from studies on septic shock.[4,5] Some suggest incorporating the central venous pressure (CVP) into a MAP target, whereas others will recommend a higher MAP goal, similar to how clinicians approach the management of hepatorenal syndrome.[2,6] Given the sparsity of evidence to support specific MAP targets in pre-capillary PH patients with RVF, we sought to describe the relationship between MAP and morbidity and mortality in this critically ill patient population. The aim of this study was to assess outcomes in pre-capillary PH patients admitted with RVF who were treated with a static MAP target ($MAP_{65/70}$) versus those managed with a dynamic MAP goal (MAP_{CVP}).

METHODS

Study design

This was a retrospective study approved by the Hartford HealthCare Institutional Review Board. Records of patients were identified by the International Classification of Diseases 10th Revision (ICD-10) using the diagnosis of RVF (I50.810) secondary to pre-capillary PH (I27.0, I27.20) at a single tertiary academic center from October 2020 to March 2023. Patients with pre-capillary PH, defined by a mean pulmonary artery pressure (mPAP) ≥ 20 mmHg, pulmonary capillary wedge pressure (PCWP) ≤ 15 mmHg, and pulmonary vascular resistance (PVR) > 3 Wood units, were included in the study, and those with a component of post-capillary PH were excluded.[7] Primary outcomes included in-hospital mortality and development of acute kidney injury (AKI). Secondary outcomes were hospital and ICU length of stay (LOS).

Establishing a diagnosis of RVF

The ICD-10 diagnosis of RVF was further confirmed by echocardiographic and invasive hemodynamic data. Criteria used to confirm evidence of RVF included RV dysfunction identified on echocardiogram along with elevated right atrial pressure (RAP) or CVP > 15 mmHg and reduced Fick cardiac index (CI) < 2.0.[8–13] In patients where PAC was not placed, surrogate data was obtained from a triple lumen catheter (TLC), where CVP was used interchangeably with RAP (14/60, 23.3%) and central venous oxygen saturation ($ScvO_2$) served as a surrogate (14/60, 23.3%) for venous oxygen saturation (SvO_2).

Treatment of RVF

Management of RVF was guided by a specific protocol. Patients were treated based on clinician judgment in a multidisciplinary approach, including PH consult service, nephrology, and/or advanced heart failure. Specific vasopressor or inotrope use was not specified except for avoidance of phenylephrine, as it causes vasoconstriction of the pulmonary vascular bed, further increasing PVR and worsening RV performance.[14–16] With respect to the MAP_{CVP} group, the primary focus was to optimize SPP based on the formula: SPP = MAP − CVP. Lastly, preload was optimized with diuresis for a target RAP or CVP goal of 8–12.[17]

Statistical analysis

The outcomes analyzed for this study included: mortality, end-organ damage (assessed by lactic acid), AKI defined by Kidney Disease Improving Global Outcomes (KDIGO) classification (a minimal increase in creatinine by ≥0.3 mg/dL or 50% from baseline),[18] and ICU LOS. Descriptive statistics were generated for each episode or admission and aggregated within each of the two MAP groups. They comprised means and standard deviations (SD) for normally distributed continuous variables, median and interquartile ranges (IQR) for non-normally distributed continuous variables, and frequencies for categorical/dichotomous variables. Inferential statistics were used to evaluate differences in the outcomes listed above between the two MAP groups.

Categorical variables were compared with a Fisher's exact test. ICU LOS was evaluated with a Mann–Whitney *U* test. All analyses were conducted with SPSS v. 29 (IBM). Results yielding $p < 0.05$ were deemed statistically significant.

RESULTS

Demographic and baseline clinical characteristics

A total of 60 consecutive pre-capillary PH patients with RVF requiring admission to the ICU were identified. The initial thirty patients identified who were treated in a conventional manner targeting a static MAP goal of either 65 or 70 mmHg ($MAP_{65/70}$) and the initial 30 patients who were managed with a dynamic MAP goal based on invasively measured RAP or CVP (MAP_{CVP}) were retrospectively evaluated. Of the 30 patients in the $MAP_{65/70}$ group, 20 (66.7%) had a MAP target of 65 mmHg, and 10 (33.3%) had a MAP target of 70 mmHg. The MAP_{CVP} group had a changing MAP goal that was adjusted every 12 h based on the patient's transduced CVP or RAP, and it was calculated based on the equation of MAP = 60 + CVP (or RAP).

Baseline characteristics demonstrated a male predominance (56.7%) and a mean age of 64.8 years (Table 1). The predominant etiology of pre-capillary PH was World Health Organization (WHO) Group 1 (57%), followed by WHO Group 3 (15%), combined WHO Group 1 and 3 (13%), WHO Group 4 (12%), and WHO Group 5 (3%). Thirty-eight patients (63%) were not on PH-specific therapy at the time of admission. Baseline echocardiographic data, including estimated right ventricular systolic pressure (eRVSP), estimated RAP, and tricuspid annular plane systolic excursion (TAPSE), were similar between the two cohorts. Likewise, baseline invasive hemodynamic data were similar between the two groups.

Primary and secondary outcomes between $MAP_{65/70}$ and MAP_{CVP}

There was a statistically significant difference in the primary outcome of in-hospital mortality between the $MAP_{65/70}$ and MAP_{CVP} groups, with decreased mortality in the dynamic MAP cohort ($MAP_{65/70}$: 43.3%, MAP_{CVP}: 16.7%; $p = 0.047$; Table 2). Similarly, 73.3% of $MAP_{65/70}$ patients developed AKI compared with 3.3% in the MAP_{CVP} group ($p < 0.001$). As for secondary outcomes, the $MAP_{65/70}$ patients had a non-significantly shorter median LOS for index hospitalization ($MAP_{65/70}$: 14 days,

TABLE 1 Baseline characteristics.

	$MAP_{65/70}$	MAP_{CVP}	*p*-Value
Sample (*n*, %)	30 (50%)	30 (50%)	---
Age, years (mean ± SD)	65.1 ± 10.2	64.4 ± 13.8	0.807[A]
Gender (*n*, %)			
Male	18 (60%)	16 (53.3%)	
Female	12 (40%)	14 (46.7%)	0.795[B]
Race/ethnicity (*n*, %)			
W	22 (73.3%)	21 (70%)	
B/AA	5 (16.7%)	6 (20%)	0.945[C]
H	3 (10%)	3 (10%)	
WHO Group			**0.025**[C]
1	22 (73.3%)	12 (40.0%)	
3	5 (16.7%)	4 (13.3%)	
4	1 (3.3%)	6 (20.0%)	
5	1 (3.3%)	1 (3.3%)	
1 & 3	1 (3.3%)	7 (23.3%)	
On PH therapy	13 (43.3%)	9 (30%)	0.422[B]
Baseline TTE measurements			
eRVSP	80.7 ± 21.2	87.8 ± 21.3	0.197[A]
TAPSE	1.3 ± 0.3	1.3 ± 0.2	0.963[A]
RAP*			1.000[B]
8 (moderately dilated)	9 (30%)	9 (30%)	
15 (severely dilated)	21 (70%)	21 (70%)	
Baseline RHC measurements			
RAP	18.9 ± 3.8	18.4 ± 3.3	0.615[A]
RVSP	76.9 ± 19.0	80.2 ± 15.2	0.465[A]
Mean PA	44.8 ± 9.8	44.9 ± 10.1	0.979[A]
PAOP	9.7 ± 3.8	9.3 ± 3.0	0.708[A]
CI (F)	1.9 ± 0.3	1.8 ± 0.3	0.459[A]
PVR (F)	10.6 ± 3.5	10.8 ± 2.8	0.855[A]
PA Sat	53.0 ± 6.4	52.9 ± 6.4	0.958[A]

Note: Values in bold are statistically significant at $p < 0.05$. A = Student's *t*-test; B = Fisher's exact test; C = chi square.

Abbreviations: CI, cardiac index; eRVSP, estimated right ventricular systolic pressure; MAP, mean arterial pressure; PA, pulmonary artery; PAOP, pulmonary artery occlusion pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RHC, right heart catheterization; SD, standard deviation; TAPSE, tricuspid annular plane systolic excursion; TTE, transthoracic echocardiogram; WHO, World Health Organization.

Values in bold are statistically significant at $p < 0.05$.

*These were estimated, and could only be 8 or 15.

TABLE 2 RVF admission outcomes.

	$MAP_{65/70}$	MAP_{CVP}	*p*-Value
Peak ProBNP [median (IQR)]	5915.0 (1690.8–11,480.8)	6081.0 (3145.2–17,001.0)	0.375[B]
Peak lactate [median (IQR)]	2.6 (2.0–4.2)	2.4 (1.6–2.8)	0.189[B]
Admission Cr (mean ± SD)	1.1 ± 0.4	1.3 ± 0.7	0.130[A]
RAP	18.9 ± 3.8	18.4 ± 3.3	0.615[A]
CVP*	13.9 ± 5.2	12.3 ± 4.0	0.639[A]
CI (F) (mean ± SD)	1.9 ± 0.3	1.8 ± 0.3	0.459[A]
SvO_2	53.0 ± 6.4	52.9 ± 6.4	0.958[A]
$ScvO_2$**	57.4 ± 6.8	53.7 ± 9.1	0.465[A]
LOS overall	14.0 (7.0–20.2)	15.0 (9.8–22.0)	0.310[B]
LOS ICU	6.0 (4.0–10.0)	6.5 (4.8–9.2)	0.783[B]
Need for ventilator	5 (16.7%)	4 (13.3%)	1.000[C]
Development of AKI	22 (73.3%)	1 (3.3%)	**<0.001**[C]
Need for RRT	0 (0%)	1 (3.3%)	1.000[C]
In-hospital mortality	13 (43.3%)	5 (16.7%)	**0.047**[C]

Note: Values in **bold** are statistically significant at $p < 0.05$. A = Student's *t*-test; B = Mann–Whitney *U* test; C = Fisher's exact test.

Abbreviations: AKI, acute kidney injury; CI (F), Fick cardiac index; CVP, central venous pressure; ICU, intensive care unit; IQR, interquartile range; LOS, length of stay; MAP, mean arterial pressure; RAP, right atrial pressure; RRT, renal replacement therapy; SD, standard deviation; SvO_2, venous oxygen saturation.

*CVP was used in 14 cases ($MAP_{65/70}$ $n = 11$; MAP_{CVP} $n = 3$) where this value was measured.

**$ScvO_2$ was used in 13 cases ($MAP_{65/70}$ $n = 10$; MAP_{CVP} $n = 3$) where this value was measured.

MAP_{CVP}: 15 days; $p = 0.310$) and shorter median ICU LOS ($MAP_{65/70}$: 6 days, MAP_{CVP}: 6.5 days; $p = 0.783$).

Vasopressor and inotrope management of RVF

The most frequently utilized initial vasopressor in the entire cohort was norepinephrine (40/60, 66.7%), followed by vasopressin (10/60; 16.7%), and a total of 22 patients required more than 1 vasopressor to achieve the designated MAP goal (Table 3). 36 patients also required support with an inotrope, where dobutamine was more frequently administered compared to milrinone (21 [35%] versus 15 [25%], respectively; $p = 0.319$]. A total of 16 patients were initiated on advanced prostacyclin therapy (intravenous treprostinil or epoprostenol) during the index hospitalization as well.

DISCUSSION

Clinical implications

The universally accepted MAP goal of 65 mmHg for critically ill patients originates primarily from the literature surrounding sepsis and subsequent shock.[4,5] However in specific clinical conditions, the MAP goal may need to be adjusted to optimize perfusion pressure, such as in hepatorenal syndrome.[3,7] When it comes to RVF as a result of decompensated pre-capillary PH, an optimal MAP target has not been well studied. Patients with RVF exhibit systemic congestion with an elevated CVP or RAP. This condition is frequently overlooked as patients do not typically display obvious hypotension, and symptoms only become pronounced when it reaches a severe stage.[19–21] Reduced SPP results in inadequate organ perfusion. Therefore, investigation of a dynamic MAP goal by taking CVP or RAP into consideration is paramount for promoting perfusion to the RV myocardium, maintaining SPP primarily to the kidneys, and increasing LV afterload, which in turn counteracts interventricular septal flattening and encourages normal cardiac geometry.[3] In this retrospective study, our data suggests the importance of having a dynamic MAP target instead of a fixed goal when it comes to RVF where optimization of RV preload, afterload, and contractility is key.

Campo et al.[2] previously showed that the presence of renal dysfunction is a significant indicator of poor outcomes in RVF patients. Patients with PH are at risk of developing AKI due to multiple factors, including low cardiac output, venous congestion, activation of the renin–angiotensin–aldosterone system, and hypoxia.[2] An

TABLE 3 Vasopressor, inotrope, and prostacyclin management of RVF admission.

	$MAP_{65/70}$	MAP_{CVP}	*p*-Value
Initial vasopressor administered	21 (70%)	30 (100%)	**0.002**[A]
Specific vasopressor administered			0.303[B]
Norepinephrine	15 (71.4%)	25 (83.3%)	
Vasopressin	6 (28.6%)	4 (13.3%)	
Epinephrine	0 (0%)	1 (3.3%)	
Phenylephrine	0 (0%)	0 (0%)	
>1 vasopressors administered	10 (33.3%)	12 (40%)	0.789[A]
>2 vasopressors administered	4 (13.3%)	6 (20%)	0.731[A]
Initial inotrope administered	19 (63.3%)	17 (56.7%)	0.792[A]
Specific inotrope administered			**0.008**[A]
Dobutamine	7 (36.8%)	14 (82.4%)	
Milrinone	12 (63.2%)	3 (17.6%)	
Both	0 (0%)	0 (0%)	
"Rescue" prostacyclin administered	6 (20%)	10 (33.3%)	0.382[A]

Note: Values in bold are statistically significant at $p < 0.05$. A, Fisher's exact test; B, chi square.

Abbreviations: CVP, central venous pressure; MAP, mean arterial pressure; RVF, right ventricle failure.

additional cause of worsening renal function in these cases may be related to diuresis, even though it is a mainstay of volume optimization therapy. Pre-capillary PH patients with an elevated CVP or RAP often require high-dose diuretics though clinicians may be inclined to abandon such therapy if there is a worsening trend in kidney function. However, the developing renal dysfunction may not be a result of over-diuresis but, instead, poor SPP in the setting of RVF. The findings from our study support this theory by showing that the dynamic MAP cohort, which focused on optimizing SPP based on the patient's changing CVP or RAP, had significantly less development of AKI.

Strengths and limitations

The present study has several limitations. The data are retrospectively collected from a single center with a relatively small sample size, which may introduce bias and limit the generalizability of results. MAP was measured both invasively and noninvasively, potentially affecting the precision of these measurements. Most importantly, the dynamic MAP group had a higher use of invasive hemodynamic measurements via PAC compared to static MAP group (MAP_{CVP} $n = 27$; $MAP_{65/70}$ $n = 19$) potentially leading to better utilization of diuretics and vasoactive drugs, including inotropes and intravenous prostacyclin agents. As such, there was a statistically significant difference between the type of inotrope used between the dynamic and static MAP groups without a significant difference in the overall number of inotropic support between groups. While Dobutamine and Milrinone have well-established and similar outcomes in cardiogenic shock, they have not been studied head-to-head in pre-capillary PH.[16,22,23] Similarly, there was a trend toward higher usage of intravenous prostacyclin in the dynamic MAP group compared to static MAP as well.

Despite these limitations, this study includes patients with a diverse demographic background with multiple etiologies of pre-capillary PH that were investigated over a multi-year period. Moreover, the findings from the study serve to introduce the novel concept of adjusting the MAP target in RVF patients based on dynamic changes in invasive hemodynamics, which is quite plausible from a physiologic perspective.

CONCLUSION

There is a high prevalence of RVF in pre-capillary PH, resulting in prolonged admissions to the ICU that are accompanied by significant morbidity and mortality. We found that targeting a dynamic MAP goal, instead of the traditional MAP target of 65 mmHg, resulted in a significantly lower incidence of AKI and significantly lower in-hospital mortality compared to a static MAP target in pre-capillary PH patients admitted with RVF. Refinement of this clinical approach and subsequent investigation through a multi-center study and perhaps a randomized controlled trial is needed to further evaluate these findings.

AUTHOR CONTRIBUTIONS

Niala Moallem: Conceptualization; data curation; investigation; methodology; validation; writing—original draft; writing—review & editing. **Garrett Fiscus**: Conceptualization; data curation; investigation; methodology; validation; writing—original draft, review, editing. **David M. O'Sullivan**: Conceptualization; data curation; formal analysis; methodology; writing—original draft, review, editing. **Michael Perkins**:

Conceptualization; data curation; investigation; methodology; validation; writing—original draft, review, editing. **Andrew Scatola**: Conceptualization; data curation; investigation; methodology; validation; writing—original draft, review, editing. **Raj Parikh**: Conceptualization; investigation; methodology; validation; writing—original draft, review, editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

ETHICS STATEMENT

This material is the authors' own original work, which has not been previously published elsewhere or currently being considered for publication elsewhere. The article reflects the authors' own research and analysis in a truthful and complete manner. The authors give consent for publication.

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REFERENCES

1. Ryan JJ, Huston J, Kutty S, Hatton ND, Bowman L, Tian L, Herr JE, Johri AM, Archer SL. Right ventricular adaptation and failure in pulmonary arterial hypertension. Can J Cardiol. 2015;31(4):391–406. https://doi.org/10.1016/j.cjca.2015.01.023
2. Campo A, Mathai SC, Le Pavec J, Zaiman AL, Hummers LK, Boyce D, Housten T, Lechtzin N, Chami H, Girgis RE, Hassoun PM. Outcomes of hospitalisation for right heart failure in pulmonary arterial hypertension. Eur Respir J. 2011;38(2):359–67. https://doi.org/10.1183/09031936.00148310
3. Farkas J. Right ventricular failure due to pulmonary hypertension [Internet]. EMCrit Project. 2023 [cited 2023 April 22]. https://emcrit.org/ibcc/rv/
4. Burstein B, Tabi M, Barsness GW, Bell MR, Kashani K, Jentzer JC. Association between mean arterial pressure during the first 24 hours and hospital mortality in patients with cardiogenic shock. Crit Care. 2020;24(1):513. https://doi.org/10.1186/s13054-020-03217-6
5. Ameloot K, Jakkula P, Hästbacka J, Reinikainen M, Pettilä V, Loisa P, Tiainen M, Bendel S, Birkelund T, Belmans A, Palmers PJ, Bogaerts E, Lemmens R, De Deyne C, Ferdinande B, Dupont M, Janssens S, Dens J, Skrifvars MB. Optimum blood pressure in patients with shock after acute myocardial infarction and cardiac arrest. JACC. 2020;76(7):812–24. https://doi.org/10.1016/j.jacc.2020.06.043
6. Tonelli AR, Plana JC, Heresi GA, Dweik RA. Prevalence and prognostic value of left ventricular diastolic dysfunction in idiopathic and heritable pulmonary arterial hypertension. Chest. 2012;141(6):1457–65. https://doi.org/10.1378/chest.11-1903
7. Condon DF, Nickel NP, Anderson R. The 6th World Symposium on Pulmonary Hypertension: what's old is new. F1000Res. 2019;19(8):888. https://doi.org/10.12688/f1000research.18811.1
8. Ghio S, Pazzano AS, Klersy C, Scelsi L, Raineri C, Camporotondo R, D'Armini A, Visconti LO. [Internet] Clinical and prognostic relevance of echocardiographic evaluation of right ventricular geometry in patients with idiopathic pulmonary arterial hypertension. Am J Cardiol. 2011;107:628–32. [cited 2023 May 1]. Available from: https://doi.org/10.1016/j.amjcard.2010.10.027
9. Forfia PR, Fisher MR, Mathai SC, Housten-Harris T, Hemnes AR, Borlaug BA, Chamera E, Corretti MC, Champion HC, Abraham TP, Girgis RE, Hassoun PM. Tricuspid annular displacement predicts survival in pulmonary hypertension. Am J Respir Crit Care Med. 2006;174(9):1034–41. https://doi.org/10.1164/rccm.200604-547OC
10. Lee CY, Chang SM, Hsiao SH, Tseng JC, Lin SK, Liu CP. Right heart function and scleroderma: insights from tricuspid annular plane systolic excursion. Echocardiography. 2007;24(2):118–25. https://doi.org/10.1111/j.1540-8175.2007.00365.x
11. D'Alonzo GE, Barst RJ, Ayres SM, Bergofsky EH, Brundage BH, Detre KM, Fishman AP, Goldring RM, Groves BM, Kernis JT, Levy PS, Pietra GG, Reid LM, Reeves JT, Rich S, Vreim CE, Williams GW, Wu M. Survival in patients with primary pulmonary hypertension. results from a national prospective registry. Ann Intern Med. 1991;115(5):343–9. https://doi.org/10.7326/0003-4819-115-5-343
12. Anderson MB, Goldstein J, Milano C, Morris LD, Kormos RL, Bhama J, Kapur NK, Bansal A, Garcia J, Baker JN, Silvestry S, Holman WL, Douglas PS, O'Neill W. Benefits of a novel percutaneous ventricular assist device for right heart failure: the prospective RECOVER RIGHT study of the Impella RP device. J Heart Lung Transplant. 2015;34(12):1549–60. https://doi.org/10.1016/j.healun.2015.08.018
13. Von Siebenthal C, Aubert J-D, Mitsakis P, Yerly P, Prior JO, Nicod LP. Pulmonary hypertension and indicators of right ventricular function. Front Med. 2016;3:23. https://doi.org/10.3389/fmed.2016.00023
14. Rich S, Gubin S, Hart K. The effects of phenylephrine on right ventricular performance in patients with pulmonary hypertension. Chest. 1990;98(5):1102–6. https://doi.org/10.1378/chest.98.5.1102
15. Jentzer JC, Coons JC, Link CB, Schmidhofer M. Pharmacotherapy update on the use of vasopressors and inotropes in the intensive care unit. J Cardiovasc Pharmacol Ther. 2015;20(3):249–60. https://doi.org/10.1177/1074248414559838
16. Mathew R, Di Santo P, Jung RG, Marbach JA, Hutson J, Simard T, Ramirez FD, Harnett DT, Merdad A, Almufleh A, Weng W, Abdel-Razek O, Fernando SM, Kyeremanteng K, Bernick J, Wells GA, Chan V, Froeschl M, Labinaz M, Le May MR, Russo JJ, Hibbert B. Milrinone as compared with dobutamine in the treatment of cardiogenic shock. N Engl J Med. 2021;385(6):516–25. https://doi.org/10.1056/NEJMoa2026845
17. Su L, Pan P, Li D, Zhang Q, Zhou X, Long Y, Wang X, Liu D. Central venous pressure (CVP) reduction associated with

higher cardiac output (CO) favors good prognosis of circulatory shock: a single-center, retrospective cohort study. Front Med. 2019;6:216. https://doi.org/10.3389/fmed.2019.00216
18. KDIGO clinical practice guideline for Acute Kidney Injury. [cited 2023 Apr 22]. Available from: https://kdigo.org/wp-content/uploads/2016/10/KDIGO-2012-AKI-Guideline-English.pdf
19. Mar PL, Nwazue V, Black BK, Biaggioni I, Diedrich A, Paranjape SY, Loyd JE, Hemnes AR, Robbins IM, Robertson D, Raj SR, Austin ED. Valsalva maneuver in pulmonary arterial hypertension. Chest. 2016;149(5):1252–60. https://doi.org/10.1016/j.chest.2015.11.015
20. Chebib N, Mornex JF, Traclet J, Philit F, Khouatra C, Zeghmar S, Turquier S, Cottin V. Pulmonary hypertension in chronic lung diseases: comparison to other pulmonary hypertension groups. Pulm Circ. 2018;8(2):1–10.
21. Song JW, Song JK, Kim DS. Echocardiography and brain natriuretic peptide as prognostic indicators in idiopathic pulmonary fibrosis. Respir Med. 2009;103(2):180–6.
22. Tavares-Silva M, Alaa M, Leite S, Oliveira-Pinto J, Lopes L, Leite-Moreira AF, Lourenço AP. Dose-response head-to-head comparison of inodilators dobutamine, milrinone, and levosimendan in chronic experimental pulmonary hypertension. J Cardiovasc Pharmacol Ther. 2017;22(5):485–95. https://doi.org/10.1177/1074248417696818
23. Rajagopal S, Ruetzler K, Ghadimi K, Horn EM, Kelava M, Kudelko KT, Moreno-Duarte I, Preston I, Rose Bovino LL, Smilowitz NR, Vaidya A, American Heart Association Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, and the Council on Cardiovascular and Stroke N. Evaluation and management of pulmonary hypertension in noncardiac surgery: a scientific statement from the American Heart Association. Circulation. 2023;147(17):1317–43. https://doi.org/10.1161/CIR.0000000000001136

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