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REVIEW

Right ventricular failure in the ICU : a practical approach

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Abstract
Right ventricular (RV) failure is an often undertreated entity due to its multifactorial and complex pathophysiology. In contrast to the muscular high-pressure generating left ventricle, the thinwalled crescend-shaped RV optimizes venous return and enables continuous ejection of blood into a low-resistance, highly compliant pulmonary vascular system. RV dysfunction leads to impaired RV filling with increased right atrial pressures and venous congestion. Additionally, progressive RV overload will cause leftward shifting of the intraventricular septum with reduced LV filling, low cardiac output and multi organ failure. Besides treatment of major potentially reversible precipitants, supportive treatment remains the cornerstone in the management of RV failure and comprises 1. optimizing RV preload and cardiac output and 2) reducing RV afterload. This review presents a comprehensive approach of patients with RV failure in the ICU.

Introduction
Right ventricular (RV) failure is a highly underestimated problem among critically ill patients. Intensivists are not sufficiently aware of this clinical entity and experience difficulties in using adequate diagnostic tools. These aspects contribute importantly to undertreatment of RV failure, resulting in significant in-hospital mortality of up to 17%.[1] Moreover, medical treatment itself can be extremely challenging due to its multifactorial aetiology. Important cornerstones in the management of RV failure are 1) the improvement of RV cardiac output and 2) the reduction of RV afterload.
In this overview, we discuss the pathophysiology and correct diagnosis of acute RV failure. We focus on management options of RV failure in the intensive care unit within the context of a great diversity of potential triggers, reviewing current guidelines and existing evidence.

Physiology of the right ventricle
In the past, the right ventricle was considered a relatively non-essential passive conduit between the systemic and pulmonary circulation. However, in the 1970s, Cohen described deleterious effects of RV failure following myocardial infarction of the right ventricle, which shed a new light on the clinical importance of RV failure.[2] The primary function of the right ventricle consists of optimising venous return by maintaining a constant low right atrial pressure (RAP), as well as enabling a continuous ejection of blood into a low-resistance, highly compliant pulmonary vascular system. In contrast to the left ventricle, which generates high pressure pulsatile flow, the right ventricle is 'designed' for a low pressure system. It is a thin-walled, crescent-shaped structure, with a tight interaction to the left ventricle. Due to a shared interventricular septum and surrounding pericardium, RV ejection is augmented up to 40% by left ventricular (LV) ejection. RV coronary perfusion is mainly supplied by the right coronary artery (RCA) and occurs during both systolic and diastolic phases. RV contraction contains sequential inward movement of the free wall, followed by contraction of longitudinal fibres with apical movement of the tricuspid annulus as a result of LV contraction.[3] Due to all these structural differences, the right ventricle has a different capacity to adapt to sudden changes in preload and afterload when compared with the left ventricle. The muscular left ventricle tolerates abrupt increases in afterload quite well, in contrast to sudden increases in preload. The more compliant right ventricle, however, can compensate for vigorous increases in venous return but poorly tolerates sudden increases in pulmonary vascular resistance (PVR).[4,5]

The most common causes of RV failure in the ICU include decompensation of pre-existing congestive heart failure or pulmonary (vascular) disease, as well as massive pulmonary embolism, sepsis, cardiopulmonary bypass surgery and ARDS.[6-9] In addition, RV failure can significantly contribute to difficulties to wean from mechanical ventilation.[10]

In general, the pathophysiology of RV failure can be divided into three main categories: 1) overwhelming preload, 2) insufficient myocardial contractility and 3) excessive afterload. In most cases, RV failure results from a combination of chronic disease together with an acute derangement in one or more of these categories.

Pathophysiology of RV failure

Increase in preload

RV preload and diastolic filling pressures affect contractility and consequently RV cardiac output via the Frank-Starling mechanism. A close interaction of the interventricular septum and ventricular contraction causes 'ventricular interdependence', which means that loading conditions in one ventricle depend on passive filling of the contralateral ventricle. Unlimited or rapid increase in preload may overwhelm RV compensatory mechanisms leading to RV dilation, impaired contractility and increased right-sided filling pressure. This will result in leftward shifting and flattening of the intraventricular septum ('D-shape') with impaired LV filling and decreased cardiac output. As a consequence, systemic hypotension will lead to both organ failure and coronary hypoperfusion, further reducing cardiac performance. In addition, equalisation of RAP and mean systemic filling pressure opposes venous return, causing progressive venous congestion with multi-organ failure and poor prognosis.[11-13]

Reduced RV contractility

Besides an increased preload, intrinsically reduced RV contractility plays an important role. RV contractile forces are generally reduced by two important mechanisms: mechanical overstretching of RV free wall myocytes and myocardial ischaemia, such as in acute RCA occlusion. Right ventricular infarction is associated with an in-hospital mortality of up to 53%.[14] In chronic heart disease and/or pulmonary hypertension, however, contractility of the slowly dilating right ventricle will decline over time with a more gradual onset of symptoms.

Excessive afterload

An increase in RV afterload mainly results from functional and structural alterations of the pulmonary circulation, often associated with worsening of a pre-existing cardiovascular or pulmonary (vascular) disease.

In healthy individuals, PVR is generally less than 90% of the systemic vascular resistance (SVR). When PVR rises gradually, myocardial hypertrophy will ensue and maintain an adequate stroke volume. However, in the case of a sudden increase in RV afterload, there is no time for compensatory RV remodelling to occur resulting in pulmonary hypertension, and haemodynamic collapse. In addition, circulating vasoactive substances, such as interleukin-6, endothelin and serotonin, play a prominent role in the pathogenesis of arterial pulmonary hypertension. During sepsis and ARDS, deranged activity of prostacyclin, nitric oxide (NO) and phosphodiesterase type 5 constitute important targets for specific medical therapy.[17,18] Finally, pathophysiological factors associated with vasoconstrictive properties, such as hypoxaemia, hypercapnia, increased alveolar dead space and reduced functional residual capacity, were all found to significantly increase PVR.[19-23]

Diagnosis and monitoring of RV failure in the ICU

Clinical aspects of right-sided heart failure

Although less reliable in the ICU patient, a thorough physical examination and consideration of medical history are important elements in diagnosing potential RV dysfunction. Jugular venous distention, ascites, hepatomegaly and peripheral oedema might easily be recognised. Cardiac auscultation can reveal splitting of the second heart sound and a systolic murmur indicating tricuspid regurgitation, which is best heard over the right sternal border.

Echocardiography

Echocardiography is an easily accessible bedside tool and is therefore a hallmark in diagnosing acute RV failure in the ICU.[24,25] Primarily, overall cardiac dimensions and function should be assessed, as well as the detection of any pericardial effusion. RV dimensions should be assessed from all available views, including parasternal, apical four-chamber and subcostal views. The presence of RV hypertrophy (wall thickness >5 mm) indicates longer existing pressure overload, whereas RV enlargement (RV end-diastolic diameter (EDD)/ LV EDD > 1.0 in apical four-chamber view) indicates RV volume overload, especially in combination with septal D-shaping of the left ventricle in the parasternal short-axis view. Quantitative assessment of RV systolic function is recommended in the guidelines by the following parameters: tricuspid annular plane systolic excursion (TAPSE; <17 mm indicates RV systolic dysfunction), tissue Doppler-derived tricuspid lateral annular systolic velocity (s'; velocity <9.5 cm/s indicates RV systolic dysfunction) or RV index of myocardial performance (RIMP; > 0.54 indicates RV systolic dysfunction).[26] Second, RV filling pressures should be estimated. The right atrial pressure (RAP) is estimated by assessing the diameter and the respiratory collapse of the inferior vena cava and is best measured 15–20 mm caudal to the hepatic vein junction *(table 1)*.[27] In addition, fluid responsiveness in both ventilated and spontaneously breathing patients, can be accurately predicted by a respiratory variation in inferior caval vein diameter of > 48%.[28,29] The systolic pulmonary arterial pressure (PAP) can be reliably quantified by adding together the trans-tricuspid pressure gradient and RAP.

Table 1. Estimation of RA pressure by assessing diameter and respiratory collapse of the inferior vena cava

RA pressure (mmHg)	IVC diameter (mm)	Respiratory collapse
0-5	< 15	Collapse > 50%
5-10	15-25	Collapse > 50%
10-15	15-25	Collapse < 50%
15-20	> 25	Collapse < 50%
> 20	> 25	No collapse

Pulmonary artery catheter (PAC)

Although the PAC does not positively affect outcome, it is still very useful in specific cases, e.g. after complex cardiac surgery or in patients with circulatory failure of combined aetiology.[30] Although alternative less invasive monitoring devices have been introduced in the past decades, PAC facilitates continuous bedside monitoring of therapeutic effects on cardiac output and right-sided filling pressures and is therefore recommended in the guidelines.[25,31] In contrast to echocardiography, PAC monitoring enables a more comprehensive haemodynamic assessment of RV failure which may manifest by a central venous pressure (CVP) exceeding pulmonary capillary wedge pressure (PCWP) or an increasing CVP and PVR with decreasing cardiac output.

Regarding the complexity of RV failure, treatment should be based on a well-structured approach, which will be discussed in the following section.

Management of RV failure in the ICU

Treatment of underlying disease

Management of RV failure starts with identification of major potentially reversible precipitants that should be managed urgently to avoid further deterioration.

Supportive treatment

Fluid management and preload optimisation

Initially, patients with acute RV failure may be preload responsive and fluid administration can improve cardiac output by increasing RV end-diastolic volume without measurable changes in transmural pressures. However, too much preload may overwhelm RV compensatory mechanisms leading to quick elevation of RAP and CVP. Therefore, the trend in CVP is a useful parameter for bedside monitoring during volume loading in such patients. In contrast, in cases of clear volume overload and venous congestion, diuretics are recommended provided the mean arterial pressure is adequate. In addition to an adequate preload, an appropriate heart rate and rhythm are also important prerequisites for optimal RV filling. As right atrial contraction contributes up to 40% to RV filling, maintenance or restoration of sinus rhythm is essential.

Vasopressors and inotropic support

Aside from optimisation of RV preload, it is of utmost importance to maintain an adequate aortic root pressure to allow sufficient right coronary arterial perfusion. This is of particular importance when the PVR approaches or exceeds SVR which in turn jeopardises coronary perfusion in a potentially vicious circle. This goal is achieved with vasopressor support while aiming for a mean arterial pressure >65 mmHg; in patients with chronic pulmonary hypertension, however, treatment should focus on increasing systemic arterial pressure above the RV systolic pressure.[32] Noradrenaline is a potent vasoconstrictor stimulating both alpha-$_1$ and beta-$_2$ adrenergic receptors. Activation of the alpha-$_1$ receptors, however, also causes pulmonary vasoconstriction, especially at doses exceeding 0.5 µg/kg/min.[33] In addition, positive inotropic effects through beta$_{-1}$ receptor agonism were found to improve cardiac output and RV performance in patients with pulmonary hypertension and sepsis.[34] Arginine vasopressin (AVP) is a selective vasoconstrictor that mainly targets the vasopressinergic (V1) receptor. AVP is an attractive alternative vasoconstrictor in the treatment of RV failure as it also causes release of nitric oxide with selective pulmonary vasodilation thereby reducing RV afterload.[35,36] In addition, AVP causes less tachyarrhythmia compared with noradrenaline and should be used in difficult 'noradrenaline-resistant' cases. However, high-dose vasopressin causes coronary vasoconstriction-induced ischaemia.[37]

In addition to optimising coronary perfusion, RV myocardial function can be further improved using inotropes and/or inodilators. Sympathicomimetic inotropes (e.g. adrenaline, dopamine, dobutamine and isoprenaline) increase myocardial contractility through beta-$_1$ stimulation at lower doses at a cost of unfavourable alpha-agonism causing pulmonary vasoconstriction at higher doses.

Dopamine has dose-dependent pharmacological effects and although it was found to increase cardiac output in patients with pulmonary hypertension, it is not recommended in the current guidelines as it was also found to be associated with increased arrhythmic events and mortality in patients with cardiogenic shock.[38-40] Dobutamine is a beta-$_1$ agonist that, in doses up to 5 µg/kg/min, augments RV contractility and reduces afterload due to concomitant beta-$_2$ stimulation induced vasodilation. It improves RV performance in patients with RV infarction and in pulmonary hypertension-related RV failure.[41] However, it causes deleterious side effects including increased myocardial oxygen consumption, susceptibility for arrhythmias, reduced diastolic filling times and a trend towards increased mortality.[42] Inodilators including phosphodiesterase (PDE) III inhibitors

and levosimendan increase myocardial contractility due to an increase in intracellular cyclic adenosine monophosphate (cAMP), while simultaneously causing dilation of systemic and pulmonary vascular smooth muscles. PDE III inhibitors such as enoximone and milrinone are good alternatives in the treatment of both patients with decompensated heart failure and following cardiac surgery as they have proven to be equally effective compared with dobutamine.[43,44] Because of their combined effect on cardiac contractility and PVR, they are the preferred drugs in patients with RV failure secondary to increased pulmonary afterload.[45] Levosimendan increases cardiac contractility by sensitising cardiac troponin C to the effects of intracellular calcium without increasing oxygen consumption. In patients with biventricular failure or following acute myocardial infarction, levosimendan has been shown to improve RV contractility and diastolic function with reduction in afterload.[46] Morelli et al. observed a reduction in PVR and mean PAP with improvements in cardiac index and RV ejection fraction in 35 ARDS patients treated with levosimendan.[47] In general, systemic vasodilation and hypotension caused by all above-mentioned inotropes and inodilators necessitate combined treatment with vasopressors in order to avoid systemic hypotension.

Afterload reduction

Measures in minimising RV afterload include reduction of PVR either by non-pharmacological or pharmacological methods.

Table 2. Vasopressors, inotropics, and agents used to reduce RV afterload

Drug	Dosage	Drug characteristics	Duration of action ($t_{1/2}$)	Potential side effects
Vasopressors				
Noradrenaline	0.2–1.0 µg/kg/min	systemic vasoconstriction through stimulation alpha-$_1$ and beta-$_1$ receptors	1-2 min	excessive vasoconstriction may worsen tissue perfusion, pulmonary vasoconstriction at dose > 0.5 µg/kg/min
Arginine vasopressin	20 units/ml dose 1-4 units/hour	* systemic vasoconstriction through stimulation V1 receptor * Selective pulmonary vasodilation due to release NO	4- 20 min	excessive vasoconstriction may worsen tissue perfusion and cause coronary ischemia [50,51]
Sympathicomimetic inotropics				
Dopamine	2-20 µg/kg/min	Dose dependent stimulation: * 2-5 µg/kg.min: dopaminergic receptor * 5-10 µg/kg.min:beta-$_1$ receptor * 10-20 µg/kg.min: alpha-$_1$ receptor	2 min	arrhythmic events and increased mortality in patients with cardiogenic shock [53]
Dobutamine	2–20 µg/kg/min	beta-1 and beta-2 stimulation	2-3 min	tachyarrhythmia, increased myocardial oxygen consumption, systemic vasodilation
Inodilators				
Enoximone	5-20 µg/kg/min	Increase intracellular cAMP	6-8 hours	pulmonary and systemic dilation of smooth muscles with hypotension
Milrinone	0.375-0.75 µg/kg/min	Increase intracellular cAMP	1-2 hours	pulmonary and systemic dilation of smooth muscles with hypotension, tachyarrhythmia
Levosimendan	0.1–0.2 µg/kg/min (optional bolus of 6–12 µg/kg bolus in 10 min; not recommended if SBP<90 mmHg)	Sensitizing cardiac troponin C to effects of intracellular calcium	1 hour	strong systemic dilation with severe hypotension
Reduction of afterload				
Inhaled				
NO	1-40 ppm	Reduction of pulmonary vascular motor tone bij increasing production of cGMP	15-30 sec	Methemoglobinemia and rebound pulmonary hypertension after abrupt withdrawal
Epoprostenol	5-20 µg/kg/min	Reduction of pulmonary vascular motor tone bij activation of cGMP	2-3 min	
Iloprost	2.5-5 µg 6-9 times/day		30 min	
Intravenous				
Epoprostenol	titrate upward in 2 ng/kg/min increments according to effect	Reduction of pulmonary vascular motor tone bij activation of cGMP	2-3 min	Systemic hypotension, increased V/Q mismatch, increased bleeding due to antiplatelet effect, flushing
Iloprost	1-5 ng/kg/min		30 min	Systemic hypotension, increased V/Q mismatch, increased bleeding due to antiplatelet effect, flushing
Oral				
Sildenafil	10-20 mg 3 times/day	Reduction of pulmonary vascular motor tone bij blocking degradation of cGMP	3-4 hours	As above, with less side affects when compared to intravous administration

Definition of abbreviations: $t_{1/2}$= elimination half time; NO=nitric oxide; cAMP= cyclic adenosine monophoshate; cGMP= cyclic guanosine monophosphate; V/Q= ventilation/perfusion

Non-pharmacological treatments should focus on appropriate ventilation strategies preventing hypoxia and hypercapnia.
Pharmacological treatment includes pulmonary vasodilators that can be classified according to their mechanism of action. Aside from pulmonary vasodilation, systemic administration of these agents also causes severe reduction in systemic blood pressure, which means they should only be used after optimisation of cardiac output. As an alternative, inhaled vasodilators should be considered as they hardly affect systemic pressure. As these agents only act in selectively ventilated lung units, they reduce intrapulmonary shunting.[48]

Pulmonary vasodilator therapy

Inhaled nitric oxide (NO) is very potent in selectively reducing pulmonary vasomotor tone by increasing the production of cGMP. Unlike systemic pulmonary vasodilators, NO does not increase intrapulmonary shunting as effects are limited to ventilated areas only.[49,50] In combination with systemic inodilators, NO may act synergistically, while discontinuation of NO should be done with caution to avoid rebound pulmonary hypertension. Prolonged administration is limited by methaemoglobinaemia but is rarely observed in daily practice.[51] Although NO has been shown to reduce PVR with transient improvement of oxygenation in patients with ARDS, there are no survival benefits.[52]

Prostacyclin derivatives include prostaglandin-I2 (prostacyclin, PGI2) with its analogues (iloprost) and prostaglandin E1 (PGE1, alprostadil). Prostacyclins reduce pulmonary vasomotor tone through activation of cGMP and administration by inhalation reduces systemic vasodilator effects and intrapulmonary shunting.[53] Published experience in the ICU is limited and epoprostenol remains the only therapy that has been shown to improve survival in patients with pulmonary hypertension.[54] Inhaled iloprost was also found to be beneficial in RV failure following cardiac surgery.[55]
Oral phosphodiesterase-5 inhibitors (sildenafil) block the degradation of cGMP, resulting in a rapid reduction in PVR.[56] Moreover, oral sildenafil has been found to facilitate weaning of inhaled and parental pulmonary vasodilators by limiting rebound pulmonary hypertension in patients following implantation of a left ventricular assist device (LVAD)[57] and it reduces PVR in patients with pulmonary hypertension after cardiac surgery.[58,59] Although PDE5 expression was found to be absent in the normal heart, PDE5 was found to be significantly upregulated in patients with RV hypertrophy, suggesting a focus for positive inotropic effects of phosphodiesterase-5 inhibitors, requiring further clinical evaluation.[60] In addition, in a small cohort of ARDS patients, oral sildenafil improved RV haemodynamics but worsened oxygenation.[61]
Table 2 shows an overview of all mentioned agents and their characteristics.

Mechanical ventilation

As stated earlier, important non-pharmacological goals to reduce PVR are the prevention of hypoxia, hypercapnia and compression of pulmonary vasculature at extremes of lung volumes. Both atelectasis and high volume ventilation should therefore be avoided. In general, positive pressure ventilation (PPV) decreases LV preload by limiting venous return, while high tidal volume (Vt) ventilation with high positive end-expiratory pressure (PEEP) may increase PAP and RAP, worsen tricuspid regurgitation and increase RV afterload.[23] Should PPV be necessary, then a 'right ventricle protective ventilation strategy' is appropriate, including: 1) Aiming for the lowest possible Vt and plateau pressure (<27 cmH2O) with driving pressure (plateau pressure – total PEEP) <18 cmH2O; 2) Limiting pulmonary vasoconstriction by strict O2 targets (oxygen saturation > 90%) and CO2 control (aim at paCO2 < 48 mmHg) and 3) Prone positioning to unload the right ventricle through improved alveolar ventilation and oxygenation with decreasing airways pressures.[62,63]
In addition, the optimal values of haemoglobin and haematocrit in patients with RV failure remain a matter of debate. Given the likelihood that anaemia, especially in the presence of hypoxia, may worsen RV failure, it is the authors' perception that it is reasonable to aim for haemoglobin levels >6 mmol/l.

Mechanical support

In cases of severe or refractory RV failure, despite all above-mentioned strategies, mechanical support can be considered in individual cases. The decision to perform mechanical support should be taken by a multidisciplinary team and performed in patients with potentially reversible causes of RV failure before initiation of irreversible end-organ failure.[64] The Survival After Veno-arterial ECMO (SAVE) score can help to predict survival for patients receiving extracorporeal membrane oxygenation (ECMO) for refractory cardiogenic shock (online calculator at http://www.save-score.com).[65] Early transfer of the patient to an appropriate centre is essential and device selection depends on the anticipated duration of mechanical support. In selective cases, mechanical circulatory support can be used as a bridge to recovery or as a bridge to heart or lung transplant.

ECMO and intra-aortic balloon pump (IABP)

Venovenous (VV) ECMO may be used in patients with isolated severe refractory respiratory failure.[66] In ARDS patients, VV ECMO has shown to lower RV afterload by the ability to reduce ventilator settings.[67] In cases of concomitant severe RV cardiac dysfunction, a combined treatment of VV ECMO and IABP could be beneficial.[68] However, short-term RV mechanical support can also be supplied by veno-arterial (VA) ECMO, which simultaneously unloads the right ventricle and supports pulmonary function.[69] In selective cases, ECMO can be used either as a bridge to lung transplant or as extended postoperative application in lung transplant recipients to control reperfusion injury.[70,71]

Right ventricular assist device (RVAD)

An RVAD (inserted either surgically or percutaneously) drains blood from the caval vein or right atrium and returns it into the pulmonary artery, which results in supporting the failing right ventricle. These devices are appropriate for longer-term use (e.g. several weeks) and can be combined with an oxygenator in case of severe pulmonary dysfunction. They have been successfully used in patients with RV failure after cardiopulmonary bypass or after RV infarction.[72] In addition, following LVAD implantation, RV failure can occur due to changes in the geometrical shape of the right ventricle and increase in preload after unloading the left ventricle. Severe cases can be bridged with a temporary RVAD, although one-year survival is still worse compared with patients not requiring an RVAD.[73] Most patients, however, will either die from complications such as bleeding, thromboembolic events, infection, or multi-organ failure. More recently, the use of commercially available devices for permanent RV support has been reported to be clinically feasible but long-term results are lacking.[74,75]

The systematic approach of acute RV failure in the ICU and appropriate treatment are summarised in *figure 1.*

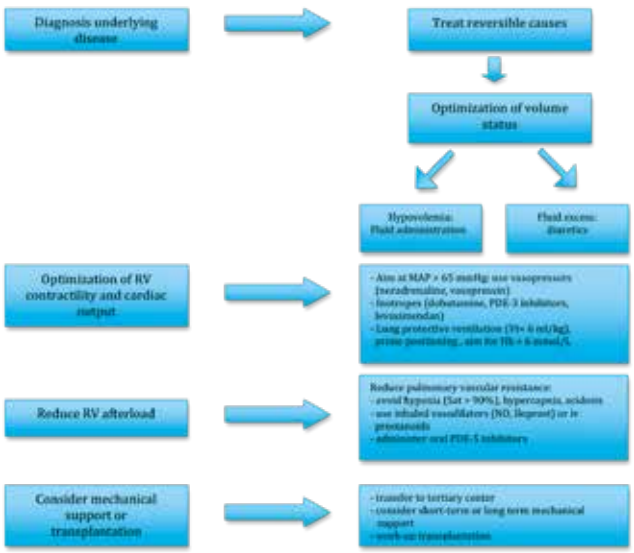

Figure 1: Systematic approach of acute right ventricular failure in the ICU Definition of abbreviations: MAP= mean arterial pressure; PDE = phosphodiesterase; Vt= tidal volume, Hb= hemoglobine; sat= oxygen saturation; NO= nitric oxide; iv= intravenous.

Conclusion

RV failure in the ICU is often caused by different aetiologies and scientific evidence of pharmacological treatment in isolated RV failure is limited. In addition to treatment of the reversible causes of acute RV failure, management mainly consists of supportive treatment. At present, despite advances in treatment, RV failure refractory to supportive therapy remains a lethal condition without a cardiac transplant.

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List of abbreviations

ARDS	Acute Respiratory Distress Syndrome
AVP	Arginine Vasopressin
cAMP	cyclic Adenosine Monophosphate
cGMP	cyclic Guanosine Monophosphate
CVP	Central Venous Pressure
ECMO	Extracorporeal Membrane Oxygenation
EDD	End Diastolic Diameter
IABP	Intra-Aortic Balloon Pump
ICU	Intensive Care Unit
LV	Left Ventricle
LVAD	Left Ventricular Assist device
NO	Nitric Oxide
PAP	Pulmonary Arterial Pressure
PAC	Pulmonary Artery Catheter
PCWP	Pulmonary Capillary Wedge Pressure
PDE	Phosphodiesterase
PEEP	Positive End-Expiratory Pressure
PGE1	Prostaglandin E1
PGI2	Prostaglandin-I2
PH	Pulmonary Hypertension
PPV	Positive Pressure Ventilation
PVR	Pulmonary Vascular Resistance
RA	Right Atrial
RAP	Right atrial pressure
RCA	Right Coronary Artery
RIMP	Rv Index of Myocardial Performance
RV	Right Ventricle
RVAD	Right ventricular assist device
RVH	Right Ventricular Hypertrophy
RVSP	Right Ventricular Systolic Pressure
SVR	Systemic Vascular Resistance
TAPSE	Tricuspid Annular Plane Systolic Excursion
Vt	Tidal Volume
VA	Veno-Arterial
VV	Veno Venous