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embolism, and myocarditis could be obtained with dedicated protocol acquisition in a new concept of “quadruple rule out.” However, the potential renal injury related to the higher amount of contrast agent required should be carefully evaluated on a case-by-case basis. To the best of our knowledge, this is the first report showing the use of CCT as part of a comprehensive cardiopulmonary assessment during the COVID-19 pandemic, thus, potentially minimizing exposure to healthcare providers and personal protective equipment utilization.

Gianluca Pontone, MD, PhD\*  
 Andrea Baggiano, MD  
 Edoardo Conte, MD  
 Giovanni Teruzzi, MD  
 Nicola Cosentino, MD  
 Jeness Campodonico, MD  
 Mark G. Rabbat, MD  
 Emilio Assanelli, MD  
 Anna Palmisano, MD  
 Antonio Esposito, MD  
 Daniela Trabattoni, MD

\*Centro Cardiologico Monzino  
 IRCCS, Via C. Parea 4  
 20138 Milan  
 Italy

E-mail: [gianluca.pontone@ccfm.it](mailto:gianluca.pontone@ccfm.it)

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* author instructions page.

## REFERENCES

1. Grasselli G, Pesenti A, Cecconi M. Critical care utilization for the COVID-19 outbreak in Lombardy, Italy: early experience and forecast during an emergency response. *JAMA* 2020 Mar 13 [E-pub ahead of print].
2. Esposito A, Palmisano A, Antunes S, et al. Cardiac CT with delayed enhancement in the characterization of ventricular tachycardia structural substrate: relationship between CT-segmented scar and electro-anatomic mapping. *J Am Coll Cardiol Img* 2016;9:822-32.
3. Esposito A, Palmisano A, Barbera M, et al. Cardiac computed tomography in troponin-positive chest pain: sometimes the answer lies in the late iodine enhancement or extracellular volume fraction map. *J Am Coll Cardiol Img* 2019;12:745-8.

## Heart<sup>COVID-19</sup> Lung Interactions

More Common Cause for Right Ventricular Dysfunction Than We Thought



We read with great interest the paper by Argulian et al. (1), describing the prevalence of right ventricular (RV) dilation among 105 patients with coronavirus

disease-2019 (COVID-19). Thirty-one patients were mechanically ventilated, and 32 patients (31%) presented with RV enlargement. The authors (1), however, do not refer on the proportion of mechanically ventilated patients with RV dilation. Mechanical ventilation, vasoactive medication use, and RV enlargement were significantly associated with increased mortality; however, all 3 are interrelated.

Heart-lung interactions may be pronounced in COVID-19 pathophysiology under mechanical ventilation. When lung compliance is relatively normal, as has been reported in COVID-19 lung (2), a substantial amount of the alveolar pressure is transmitted to the pleural pressure. Increased positive end-expiratory pressure (PEEP) levels may induce dead space ventilation and compression of the pulmonary vasculature, leading to increased pulmonary vascular resistance (PVR) and right heart dilation and dysfunction (3). Increased PVRs may be exacerbated in the setting of nonmassive pulmonary embolism (COVID-19 has been related to lung thrombotic events). This could be the case in the 5 patients with confirmed pulmonary embolism (computed tomography angiography) in the study by Argulian et al. (1). Moreover, relatively high PEEP, according to current guidelines (4), in a non-recruitable lung with almost normal compliance (as in COVID-19) may significantly increase pleural pressure and have a detrimental impact on hemodynamics by deteriorating venous return (5). The effects are exaggerated when the patients are relatively hypovolemic in the initial phase after intubation (fever in the preceding days); restricted fluid resuscitation is also indicated in acute respiratory distress syndrome to keep the lung “dry” in an effort to improve oxygenation and avoid intubation (4).

Increased PVRs and a reduction in venous return decrease the cardiac output. The first reaction to correct hypotension, in an intensive care unit setting, is vasopressor initiation. At the tissue level, hypovolemia and vasoconstriction induce hypoperfusion and end-organ damage, which can lead to multiorgan dysfunction syndrome and an unfavorable outcome. Our rationale is that RV dilatation and dysfunction, vasopressors, and mechanical ventilation are interchangeably related in patients with COVID-19. Argulian et al. (1) nicely report that all 3 factors are associated with increased mortality. It would be informative if the authors could provide data on: 1) RV dimensions before and after initiation of mechanical ventilation; 2) PEEP levels in patients with and without RV enlargement; and 3) possible associations between RV dimensions and PEEP levels. Of note, in Figure 1, both patients are mechanically ventilated.

Mechanical ventilation may be a major factor explaining ventricular dilation. Echocardiography can significantly influence decision-making in “adjusting the ventilator settings” in COVID-19.

Vasiliki Tsolaki, MD, PhD\*  
George E. Zakyntinos, MD

\*Intensive Care Unit  
University Hospital of Larissa  
University of Thessaly  
Faculty of Medicine  
Mezourlo, Larissa 41110  
Greece

E-mail: [vasotsolaki@yahoo.com](mailto:vasotsolaki@yahoo.com)

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#### REFERENCES

1. Argulian E, Sud K, Vogel B, et al. Right ventricular dilation in hospitalized patients with COVID-19 infection. *J Am Coll Cardiol Img* 2020 May 15 [E-pub ahead of print].
2. Marini JJ, Gattinoni L. Management of COVID-19 respiratory distress. *JAMA* 2020 Apr 24 [E-pub ahead of print].
3. Repesse X, Charron C, Vieillard-Baron A. Acute respiratory distress syndrome: the heart side of the moon. *Curr Opin Crit Care* 2016;22:38-44.
4. Alhazzani W, Møller M, Arabi Y, et al. Surviving Sepsis Campaign: guidelines on the management of critically ill adults with coronavirus disease 2019 (COVID-19). *Intensive Care Med* 2020;46:854-87.
5. Michard F. Changes in arterial pressure during mechanical ventilation. *Anesthesiology* 2005;103:419-28.

#### THE AUTHORS REPLY:



We thank Drs. Tsolaki and Zakyntinos for their interest in our study (1) and their insights into possible underlying mechanisms of right ventricular (RV) dilation in patients with coronavirus disease-2019 (COVID-19) infection. The authors summarize the underlying pathophysiology of the interplay of mechanical ventilation, pulmonary vascular resistance, and consecutive changes in hemodynamics as one of the many possible mechanisms of RV dilation in these patients. They also postulate that mechanical ventilation by itself, especially with high levels of positive end-expiratory pressure (PEEP), may be a major contributor to the observed findings. In our study, 31 patients (30% of the study population) were mechanically ventilated at the time of echocardiography. Among these patients, 14 patients had RV dilation (44% of all patients with RV dilation).

Interestingly, the median PEEP was 10 cm H<sub>2</sub>O (interquartile range: 8 to 14 cm H<sub>2</sub>O) among patients with RV dilation compared with 12 cm H<sub>2</sub>O (interquartile range: 6 to 14 cm H<sub>2</sub>O) among patients without RV dilation, with no significant difference ( $p = 0.98$ ) and no meaningful correlation between PEEP and RV dimensions. Of note, 24% of the remaining nonventilated patients had RV dilation. We acknowledge that these findings are derived from a small sample size and are observational in nature with inherent limitations.

Although mechanical ventilation and ventilation settings are possibly contributory, RV dilation is likely a multifactorial phenomenon. Other echocardiographic studies reported high rates of RV dilation and dysfunction in patients with COVID-19 infection. One study reported RV dilation in 41% of 74 patients, of whom 78% were mechanically ventilated at the time of echocardiography (2). Another study described RV dilation and dysfunction in 39% of 100 patients, of whom only 10% were mechanically ventilated at the time of echocardiography (3). Importantly, on follow-up echocardiogram, RV enlargement and dysfunction were common in deteriorating patients in that study.

The underlying mechanisms of RV dilation in patients with COVID-19 infection extend beyond mechanical ventilation and possibly include pulmonary thromboembolism, microthrombi in pulmonary vasculature, persistent hypoxic pulmonary vasoconstriction, and direct viral damage. Dedicated studies to understand these underlying mechanisms will provide more insights into the complex pathophysiology of this disease.

Karan Sud, MD  
Jagat Narula, MD, PhD  
Edgar Argulian, MD, MPH\*

\*Mount Sinai Morningside  
Icahn School of Medicine at Mount Sinai  
1111 Amsterdam Avenue  
New York, New York 10025

E-mail: [edgar.argulian@mountsinai.org](mailto:edgar.argulian@mountsinai.org)

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1. Argulian E, Sud K, Vogel B, et al. Right ventricular dilation in hospitalized patients with COVID-19 infection. *J Am Coll Cardiol Img* 2020 May 15 [E-pub ahead of print].