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# The liaison between respiratory failure and high blood pressure: evidence from COVID-19 patients

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**In COVID-19 patients respiratory failure is associated with increased systemic blood pressure, conceivably due to the modulation of the renin-angiotensin-aldosterone system by SARS-CoV-2 infection** https://bit.ly/3cINsHB

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*To the Editor*:

Expanding from China around the world, coronavirus 2019 (COVID-19) is the disease caused by severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2). COVID-19 primarily manifests by hypoxic normo-hypocapnia with preserved lung compliance [1]. In the absence of targeted treatment, sub-intensive clinicians support patients with noninvasive ventilation and anti-inflammatory/anti-viral agents waiting for status improvement. Angiotensin-converting enzyme (ACE)2, highly expressed on the external membrane of lungs, heart, kidney and gastrointestinal tract cells, displays the binding site for the spike protein of SARS-CoV-2 [2]. ACE2, identified as a counterpart of the Renin-Angiotensin-Aldosterone System (RAAS), converts angiotensin (Ang) II to Ang-(1-7) and Ang I to Ang-(1-9). ACE2 activity induces vasodilatation and reduces cell growth and inflammatory response. In experimental models that mimic viral acute respiratory distress syndrome, the absence of *Ace2* led to inflammation, vascular permeability and lung injury *via* activation of the Ang II pathway [3, 4]. The decrease in ACE2 activity by SARS-CoV-2 can unleash a cascade of injurious effects through a heightened imbalance in the actions of the products of ACE *versus* ACE2. Moving to a clinical setting, the ACE2 downregulation may be one of the pathways sustaining arterial hypertension [5] and pulmonary arterial hypertension [6]. Therefore, it is conceivable that in COVID-19 a cleavage of membrane ACE2 along with its circulatory levels could impact on the disease progression and clinical worsening [7]. Thus, to support a pathophysiological role of ACE2, the

present report shares clinical data from an observational study conducted on 40 patients with a diagnosis of COVID-19, hospitalised in the Cardiorespiratory Sub-Intensive COVID-19 Unit at the Fondazione IRCCS Ca' Granda Policlinico Hospital (Milan, Italy).