Supplementary Online Content

Marini JJ, Gattinoni L. Management of COVID-19 respiratory distress. *JAMA*. doi:10.1001/jama.2020.6825

This supplementary material has been provided by the authors to give readers additional information about their work.

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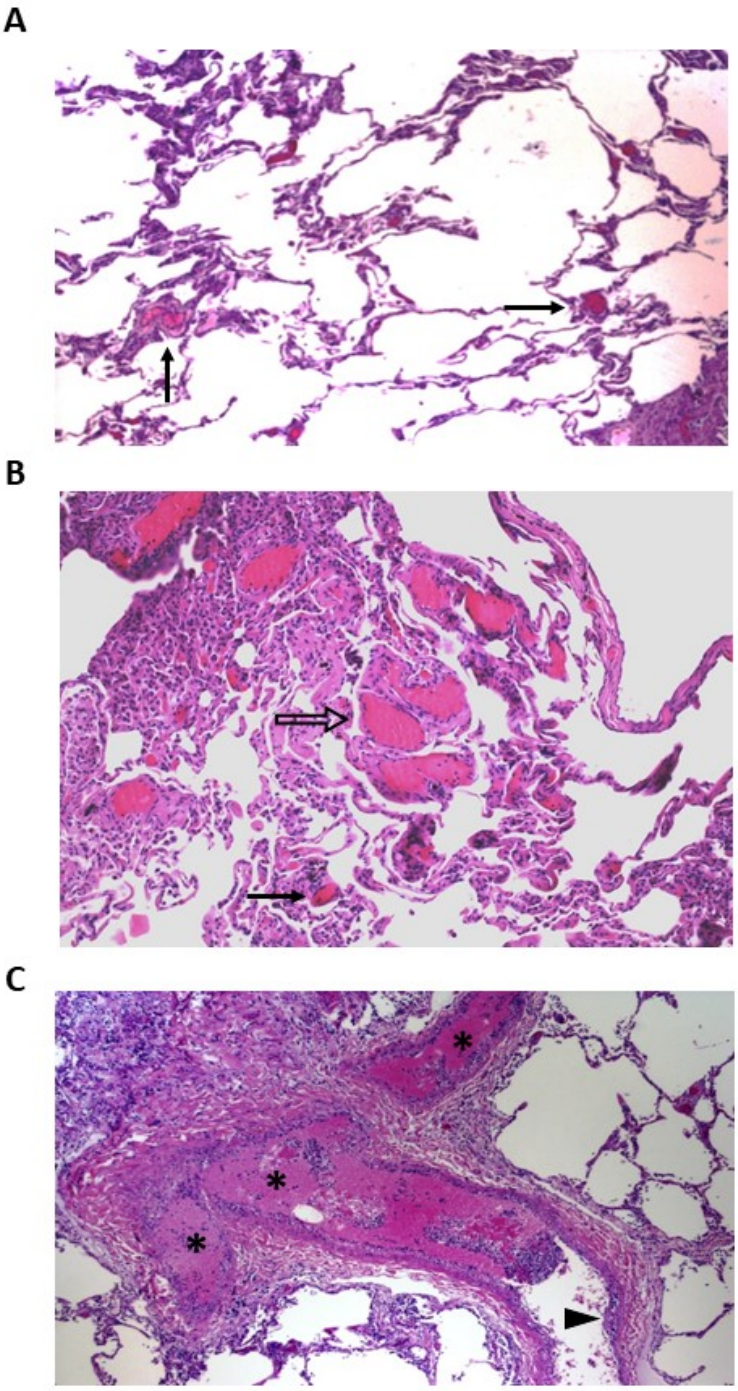
eFigure 1. Postmortem Lung Histological Findings in COVID-19 Patients



A, Emphysematous changes and diffuse thrombosis of intra-septal microvessels (arrows). B, Small sized arterioles (open arrow) with complete luminal thrombosis and occlusive thromboses of intraseptal capillaries (closed arrow). C, medium size arteries (asterisks) with complete luminal thrombosis, one of them showing stratification of inflammatory cells on a destroyed endothelial layer (triangle). Courtesy of A. Sonzogni and A. Gianatti Ospedale Papa Giovanni XXIII (Bergamo, Italy); L. Carsana and M. Nebuloni of Ospedale Luigi Sacco (Milan, Italy).

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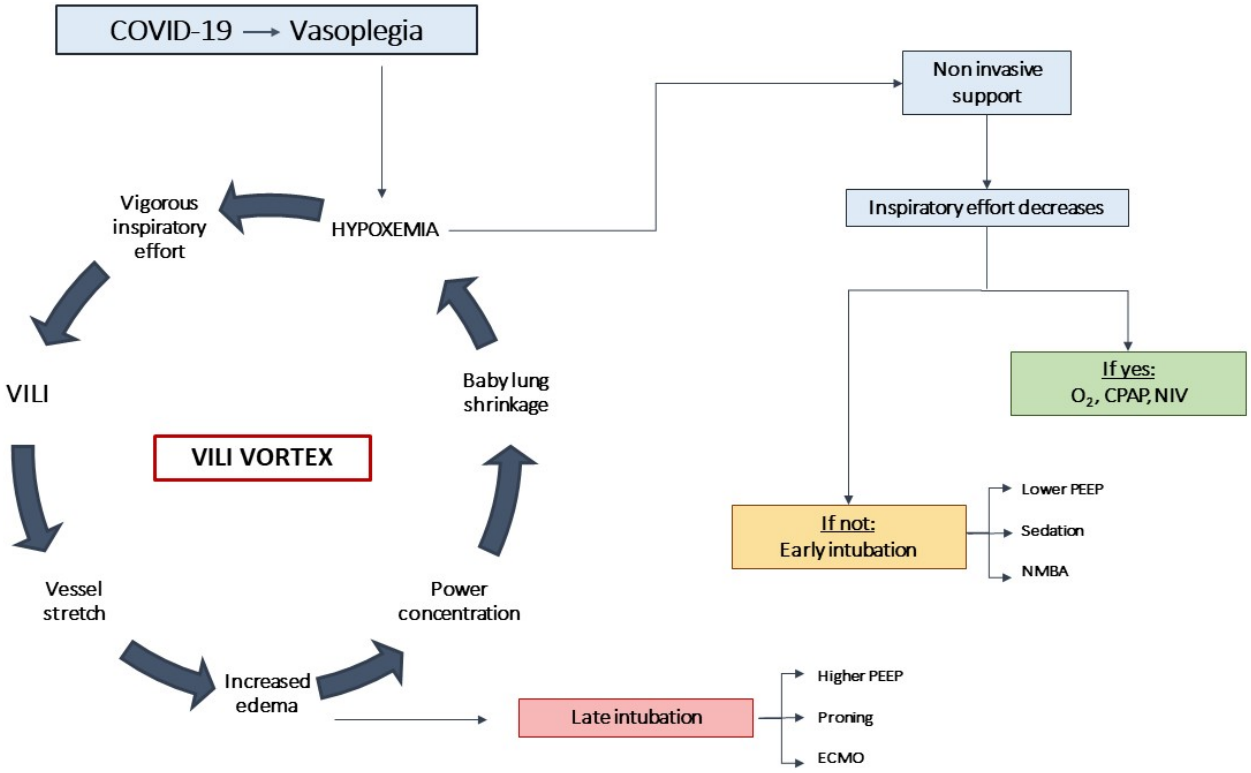
eFigure 2. Drivers and Interrupters of Progressive Lung Injury in COVID-19 Infection



The COVID-19 infection induces vasoplegia, VA/Q mismatch, and hypoxemia. This, in turn (together with viral disease–related stimuli), increases respiratory drive and strong breathing efforts. To avoid initiating a downward spiral of progressive lung shrinkage and injury (the 'VILI vortex'), noninvasive support may be applied, such as low or high flow nasal O_2, continuous positive airway pressure (CPAP), or noninvasive ventilation (NIV). If transpulmonary stresses are relieved, the patient may be adequately treated in this way. However, if strong inspiratory efforts persist, early intubation, sedation, and/or paralysis is indicated. If intubation is significantly delayed, the VILI vortex progresses toward full-blown ARDS. Of note, damage secondary to COVID-19 itself may progress, regardless of respiratory treatments.

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