Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Letters

Beta-Blockers in COVID-ARDS

Inflammation or Hemodynamic?

We read with interest the study by Clemente-Moragón et al (1) evaluating the use of metoprolol in COVID-19-related acute respiratory distress syndrome (ARDS) (COVID-ARDS). Clemente-Moragón et al (1) conclude that metoprolol is associated with reduced pulmonary inflammation and improved oxygenation, the latter explained by decreased biomarkers of inflammation in bronchoalveolar lavage. Although the use of β-blockers is supposed to have a putative anti-inflammatory effect in critically ill patients (2), we would like to point out that, in this study, clinical improvement may reflect a correction of the ventilation-perfusion ratio rather than a decrease in alveolar inflammation.

Intensivists have known for more than 40 years that a decrease in cardiac output (CO) is a direct mechanism of reduction of intrapulmonary shunt and therefore better oxygenation (3). We recently reported in COVID-ARDS that oxygenation improvement related to CO decrease may not be desirable because it is associated with a paradoxical decrease in oxygen delivery (4). Thus, caution is required when analyzing the effect of a treatment on Pao_2 or the ratio of Pao_2 to the fraction of inspired oxygen, especially when the treatment has well-known hemodynamic effects likely to affect CO. This is obviously the case for cardioselective β-blockers such as metoprolol. CO is not directly reported in the study by Clemente-Moragón et al (1). However, considering left ventricular outflow tract velocity time integral and heart rate reported in their supplemental data, we may assume that metoprolol use is associated with a drastic reduction in CO of 20% on average. This association may explain the observed effect on oxygenation whatever the effects on inflammation may be.

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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 Author Center.

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