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The impact of ventilation – perfusion inequality in COVID-19: a computational model

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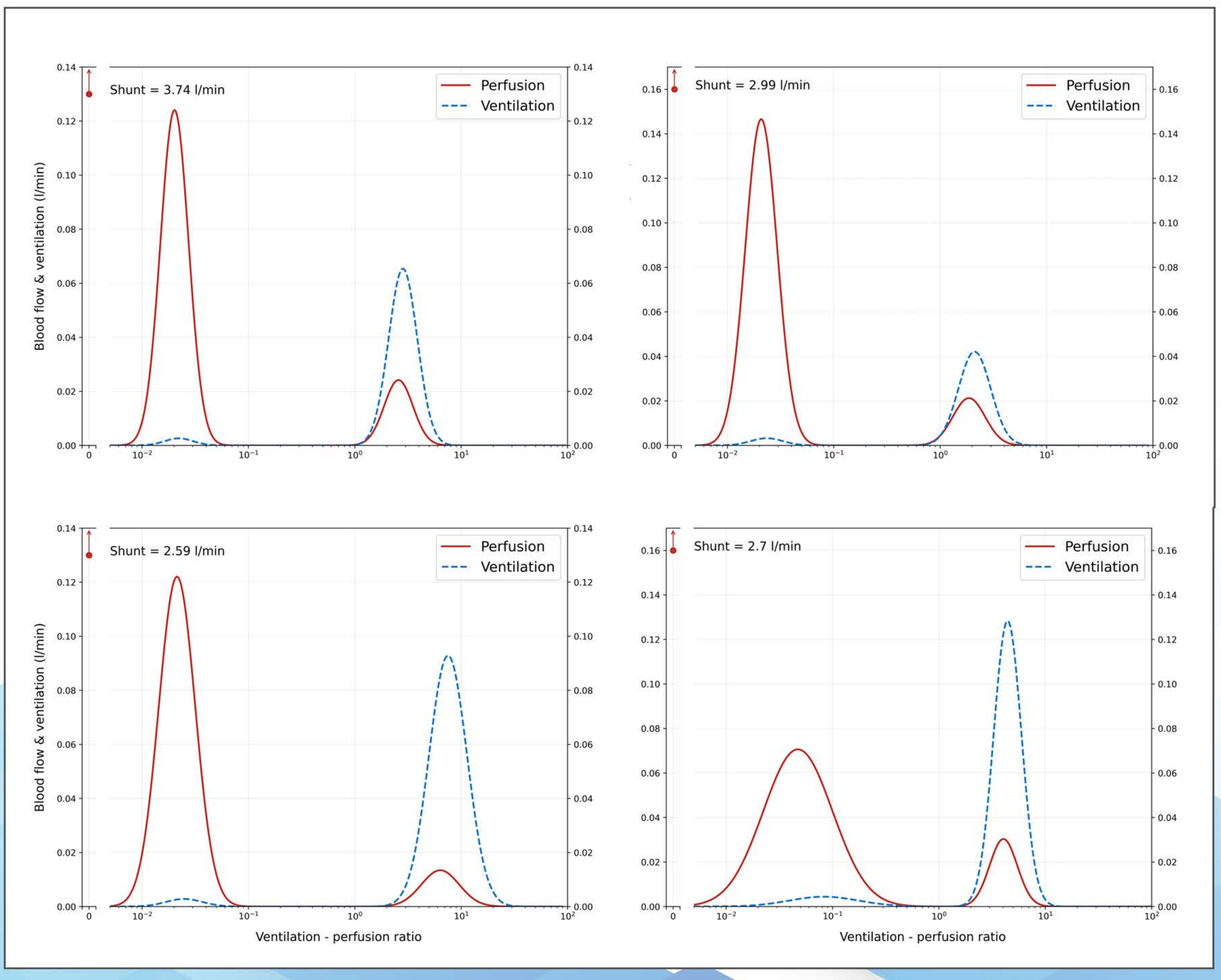
Introduction

COVID-19 infection may lead to an Acute Respiratory Distress Syndrome where severe gas exchange derangements may be associated, at least in the early stages, only with minor pulmonary infiltrates. This implies that mechanisms other than right-to-left shunt play a role in CARDS pathophysiology.

Methods

We designed an algorithm ($Vent_{ri}Q_{lar}$), based on the same conceptual grounds described by J.B West in 1969. We set 499 ventilation-perfusion (V_A/Q) compartments and, after calculating their blood composition, we randomly chose 10^6 combinations of parameters controlling a bimodal distribution of associated blood flow. The solutions were accepted if the predicted PaO_2 and $PaCO_2$ were within 10% of the patient's values. We assumed that shunt fraction equaled the fraction of non-aerated lung tissue at the CT quantitative analysis.



Results

Five critically-ill patients were studied. The PaO_2/FiO_2 was 91.1±18.6 mmHg and $PaCO_2$ 69.0±16.1 mmHg. Cardiac output was 9.58±0.99 l/min. The fraction of non-aerated tissue was 0.32±0.07 vs a calculated venous admixture of 0.43±0.08. The model showed that a large fraction of the blood flow was likely distributed in regions with very low V_A/Q (Q_{mean} = 0.06±0.02) and a smaller fraction in regions with moderately high V_A/Q. LogSD, Q was 1.74±0.14, suggestive of high V_A/Q inequality. Shunt alone cannot completely account for the observed hypoxemia and a significant V_A/Q inequality must be present in COVID-19. The high cardiac output and the extensive microthrombosis found at the autopsy further support the hypothesis of a pathological perfusion of non/poorly aerated lung tissue.