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Ventilate or not?…Is this really the question?… Or do not ventilate at all?

COVID-19 virus enters alveolar epithelial cell type II via angiotensin-converting-enzyme type two (ACE2) receptors, which is pretty abundant in the lung. The destruction of the surfactant follows but without much of the fibrosis[1]. However, the toxicity of the viral particles seems to be more dependent on non-receptor binding elements of virion by driving unfavorable immune response[1]. Interestingly, the immune response resembles monocyte activation syndrome (MAC) followed by leukopenia in patients, especially in cases of dim prognosis[2,3]. Also, vasculitis and intravascular coagulopathy of pulmonary endothelium seem to be one of the primary drivers[3,4].
The natural history of pulmonary symptoms of COVID-19 is highly heterogenous. No treatment can modify the outcomes. Chest X-ray is often negative initially, yet computer tomography can detect a ground-glass opacity so characteristic that some suggest using them for a diagnostic test, especially in sick patients early [3,5,6].
The initial symptom of the pulmonary demise of COVID-19 is hypoxemia[5]. The patient lung compliance is relatively satisfactory. It is a surprise to the author of this article then to see the prevalent perception of COVID-19 as a classical ARDS-like illness. The patients' symptoms are far from those observed in a classical "stiff" lung. They resemble more refractory hypoxia secondary to a diffusion block. COVID-19 may be a completely different disease, then we believed. We did not appreciate that COVID-19 is an exquisite and unique disease, as demonstrated by different pathogenesis and "atypical" clinical radiographical evidence. Instead, we carbon copied the clinical decision process from seemingly similar illnesses. In the case of COVID-19, the clinical decision mimics those seen in classical ARDS management, including early initiation of mechanical ventilation, muscule relaxation, heavy sedation, and proning. However, these may not be the only choices, and the COVID-19 pandemic spurred an incredible amount of innovation. The shortage of ventilators forced creative thinking.
First of all, we started to appreciate the benefit of nasal cannulae. Also, non-invasive ventilation is much more appreciated. High O_2 content can be delivered in a variety of ways, including high-flow nasal canulae, BiPAP, or a high oxygen tent. Due to the calling COVID-19, an ARDS like disease a ventilator is a must. However, a ventilator serves TWO purposes. Delivery of high inspired oxygen and/or improvement in the work of breathing. If compliance is high, the latter

function of the ventilator is not pivotal to improve respiratory compromise. In those cases, the ventilator may be hurting the patient by impairing the evacuation of the fluid from the bronchial tree by suppressing the natural processes of coughing and mucus evacuation. The high mortality among the patients placed on a ventilator (up to 86% according to the Chinese data) may demonstrate abnormal pathology and mismatched tool to improve outcomes. This puzzling finding, coupled with a potential lack of ventilators, forced re-examination of using non-ventilators strategies. Potentially, this may be an excuse to use more ECMO except that this strategy is extremely resource-intensive.

Let us re-examine the ramification of the statement that COVID-19 is not "classical ARDS". For once the management of the respiratory system has to change. Second, we might have enough ventilators to provide to them who genuinely will benefit.

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