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Review Article

Anesthetic Management of Critical COVID-19 Infection: A Narrative Review of Concepts and Evidence-Based Clinical Practices

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Introduction

Anesthesiologists are on the frontline in the war against the global COVID-19 pandemic, providing airway, ventilatory, and hemodynamic support to acute patients suffering from severe and critical COVID-19 infection. This is despite facing enhanced risk for cross-infection from patient respiratory secretions while performing intubations at high volume. The Chinese Society of Anesthesiology reports a 20% cross-infection rate among anesthesiologists who performed intubations on COVID-19 patients during the early phase of the pandemic [1]. Furthermore, 12% of New York anesthesiology residents reported presumed, suspected, or confirmed infection-higher than any other specialty including emergency medicine, ophthalmology, and otolaryngology [2]. In addition to providing life-saving intubation for severe COVID-19 patients, anesthesiologists have comforted critically ill patients in isolation from family due to IPC-mandated visitation restrictions. Many have dedicated their time and expertise in the critical care environment, serving as ancillary ICU staff or on hospital intubation teams. This review details these unique viral features and reviews the evidence base regarding infection prevention and control (IPC) in the anesthesia workspace. It also outlines the conceptual foundations behind airway, ventilatory, and hemodynamic management recommendations and the supplementary protocols for COVID-19 management. When necessary, critical appraisal of the evidence base surrounding these topics is offered, as well as suggested changes to current guidelines and topics for further investigation.

Clinical virology

Coronaviridae (CoV) is a zoonotic family of positive-sense single-stranded RNA viruses that exhibit strong genetic diversity and pandemic potential. This is likely due to the more error-prone RNA-dependent RNA polymerases causing frequent genomic recombination events during viral replication. This genomic diversity enables coronaviruses to evolve and periodically infect the respiratory tract of human hosts.

The precise pathogenesis from pneumonia to COVID-19 ARDS (CARDS) is unclear. Studies have reported significantly elevated levels of inflammatory signaling molecules IL-6, IL-10, G-CSF, and TNF-α among CARDS patients suggesting cytokine release syndrome (CRS), an inflammatory syndrome characterized by fever and multiple organ dysfunction, commonly in the setting of immunosuppressive drug therapy following transplantation [3]. Immune hyperreactivity paradoxically suppresses leukocyte production and attenuates cell-mediated immunity. The precipitous decline from baseline to critical status among patients with CARDS may be linked to underlying CRS causing depressed CD8+ T-Cell mediated viral clearance, prolonged neutrophil-mediated lung destruction, and upregulated macrophage-mediated fibroproliferative damage [4]. Autopsy studies also point to endothelial cell dysfunction, thrombotic microangiopathy, and virally-mediated lung damage in addition to hyperinflammatory injury [5].

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Virulence and transmission dynamics

Virulence has been described as the degree to which a pathogen elicits disease in exposed individuals and is a measure of individual outcomes, such as the case fatality rate (CFR) and infection fatality rate (IFR), rather than population-level mortality. Though less virulent than its predecessors SARS-CoV-1 and MERS-CoV, which exhibited CFRs of 9.60% and 34.4% respectively [6,7]. SARS-CoV-2 exhibits higher overall virulence than influenza viruses [8-10]. Furthermore, COVID-19 demonstrates various degrees of attenuated or magnified virulence that make it clinically challenging and unpredictable. It is been posited that the heterogeneity and presentation among patients with COVID-19 are partially attributable to the differential interplay between individual susceptibility and the array of different viral species [11]. While some patients decompensate within a week, a large subset of patients remain asymptomatic throughout the course of infection (Table 1) [12] Children, in particular, are more likely to have few or no symptoms yet remain infectious carriers [13,14].

Perioperative implications

Surgical risk: Operating and performing anesthesia on COVID-19 positive patients, particularly those with comorbidities, pose significant health risks to both patients and providers. Anesthesia providers are at enhanced risk since endotracheal intubation exposes them to virally dense upper airway secretions. Perioperative assessment based on clinical screening is confounded by a significant portion of COVID-19 patients who portend little or no symptoms. As nations begin the transition towards pre-pandemic surgical volume, the potential for presymptomatic or asymptomatic transmission to healthcare providers may increase, especially if the predicted resurgence in COVID-19 cases coincides with this timeline.

Operating and performing anesthesia on COVID-19 positive patients also may also confer a significant risk to patients. The evidence-base regarding clinical outcomes for COVID-19 positive patients undergoing surgery is limited to one retrospective case series of 34 patients with comorbidities who were not symptomatic at the time of screening and underwent non-emergent vascular and GI surgeries [15]. An alarming proportion went on to develop ARDS (32%), shock or secondary infection (29%), arrhythmia (24%), acute cardiac injury (50%), and acute kidney injury (6%), with 21% ultimately dying [15] These patients endorsed comorbidities predictive of critical COVID-19 infection—specifically hypertension [13,16-19] cardiovascular disease [17,19] diabetes [13,17-20] malignancy [21,22] and age [23-25] > 65. Despite the dearth of other studies on surgical risks to infected patients, these findings are nevertheless concerning since early patient cohorts scheduled for medically necessary surgeries likely have similar chronic conditions and may have a similarly elevated risk for negative health outcomes.

Table 1: Intubating patients with COVID-19: Consistently Reported Recommendations.

1. Intubation should be reserved for the most experienced clinician in airway management, preferably an anesthesiologist [109–117].
2. Avoid manual ventilation through preoxygenation and rapid sequence induction [109–117].
3. High Aa gradient or contraindications to succinylcholine ⟶ anticipate manual ventilation during apnea but use smaller title volumes to avoid aicr leaks along the mask interface [109–117].
4. Avoid laryngeal mask airways which do not provide a sufficient seal to prevent aerosol leak [109–117].
5. Consider routine use of videolaryngoscopy [109–117] for the first attempt at intubation to maximize the distance between the airway operator and the patient.
6. Exercise caution during resuscitative chest compressions to not generate coughing or aerosol. Specifically, position oneself away from the mouth of the patient [109–117].
7. For all aerosolizing procedures in patients with unknown COVID-19 status, use airborne precautions: N95 mask, PAPR (if available), and a negative pressure room (if available) [109–117].

Preoperative screening: Given both the dangers to patients and providers and the unreliability of clinical assessment alone for identifying presymptomatic or symptomatic carrier states, supplemental molecular-based screening is essential. Despite their utility in supplementing standard history and physical exam during preoperative evaluation, the precise utility of current RT-PCR requires accurate population prevalence estimates to help determine the post-test negative predictive value. Moreover, the true prevalence of infectious carriers will remain elusive unless governments institute population-level zero surveillance to determine the proportion of infectious presymptomatic, asymptomatic, mildly symptomatic carriers that go largely unreported [26]. While the current rate of confirmed infectious carriers worldwide is approximately 0.6%, rates of unconfirmed cases have been estimated at anywhere from 20-70% [27–29]. of Assuming an RT-PCR sensitivity of 70% and specificity of 90%, the negative predictive value of the test would drop from 99.27%, or 1 case in 137, to 98.41%, or 1 case in 63, as the true population prevalence increases from 1% to 5% [30] This constitutes a two-fold increase in the risk of performing surgery and administering anesthesia to the naïve COVID-19 positive patient.

Until representative prevalence rates on a regional level are accurately estimated, universal serial testing for those patients with higher pre/post-test probabilities represents the most risk-averse preoperative screening modality—depending on when the test is administered in the disease process, the sensitivity of RT-PCR may be only marginally superior to clinical assessment alone [31-37]. Molecular screening and clinical assessment should be used synergistically to enhance the overall sensitivity of the initial evaluation, and any subtle indication of infection or close contact from history and physical should be met with a high index of suspicion and a low threshold for retesting (PARIS score [38], is a pre-test probability assessment with demonstrated external validity). These recommendations are consistent with Anesthesia Patient Safety Foundation guidelines [39]. Additionally, if an emergent aerosol-generating medical procedure (AGMP) precludes testing or if RT-PCR is unavailable, donning full PPE with N95 should be considered prudent practice [40].

When retesting a negative screen in the background of high clinical suspicion for viral infection (i.e. sick contacts, viral symptoms), the WHO recommends repeat testing after

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2-4 days to account for the latent period when the degree of viral shedding has not yet crossed the critical threshold for an adequate sample [41,42]. According to a robust meta-analysis of 1330 tests from 7 previously published studies, this window is represented by a steady decline in the false-negative rate of 100% on day one to a nadir of 20% on day eight post-exposure. This curve inversely mirrors the timeline of viral shedding described by He and colleagues (Figure 1) [43]. Another important consideration is the considerable variation in nasopharyngeal and oropharyngeal RT-PCR sensitivities depending on the initial location of viral inoculation [44]. Smaller viral aerosols preferentially colonize the LRT, while larger droplets colonize mucous membranes or the URT. The IDSA recommends that patients with negative URT samples but who have high post-test probabilities for infection undergo retesting of the LRT, either through sputum analysis [45].

Infection prevention and control

Preventing droplet transmission: Prevention measures against droplet transmission of SARS-CoV-2 focus on three sequential points of viral dissemination: (1) identifying infected patients (2) preventing patients presumed infectious from dispersing droplets and (3) maintaining adequate distance from droplet trajectory should dispersion occur. These first two measures invariably counter contact transmission as well. A type 1 surgical mask should be placed on the patient, and hospital staff should wear surgical masks at all times. This alone is sufficient to prevent direct inoculation by larger respiratory droplets (Figure 2) [46].

Preventing contact transmission: The two primary goals in IPC of indirect contact-transmission of SARS-CoV-2 are (1) prevent fomites via disposable physical barriers and (2) decontaminating surfaces that are likely to have fomites (such as monitors and anesthesia workstations) with viricidal agents. SARS-CoV-2 is a relatively bulky lipid-enveloped RNA virus that is exquisitely sensitive to low-and intermediate-level disinfectants, despite its generating resilient fomites on environmental surfaces [47-51]. Low-level disinfection results in significant decreases in pathogen density with more frequent and targeted cleaning of high-touch surfaces [52-54]. Furthermore, simulation studies found that double-gloving prevents cross-contamination of the anesthesia work environment after intubation [55,56]. Lastly, compliance with hand-hygiene protocols was greater in groups where disinfectants were readily available, with reported decreases in hourly noncompliance rates [57] from 50% to 20%.

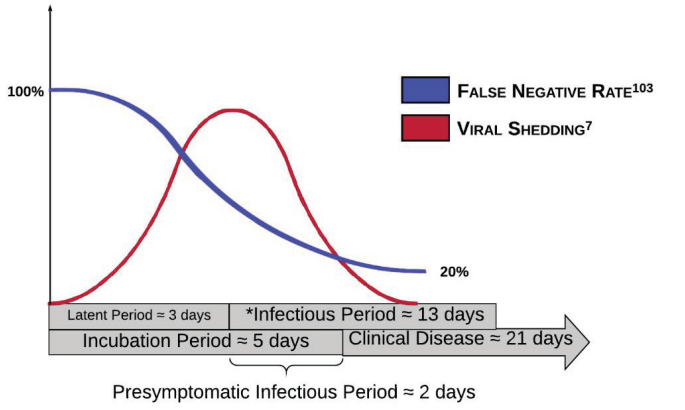

Figure 1: The drop in the false-negative rate according to data from Kucirka et al correlates with the degree of viral shedding according to data from He, et al.

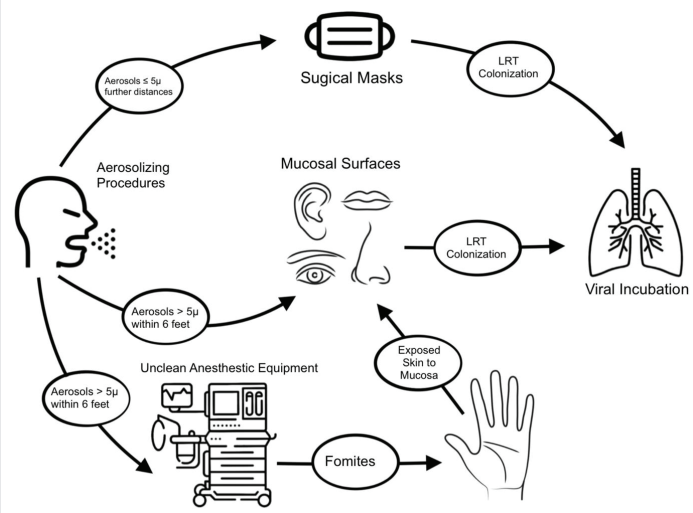

Figure 2: General schematic of the potential modes of droplet, contact, and airborne transmission common in the anesthesia workspace.

Preventing airborne transmission: Noninvasive positive pressure ventilation presents more of a theoretical risk for aerosolization of pathogenic droplets because pressured air delivery can potentially compromise the device interface and leak if not firmly secured to the patient's face. This risk is amplified if NIPPV causes dynamic hyperinflation of the noncompliant lung– high distending pressures are transferred across the patient-mask interface. If initiated, a full-face mask is preferred to minimize particle dispersion [58]. On average, the dispersal distance for NIPPV was reported as 90 cm versus only 10 cm for HFNC, with a greater reported risk of far-reaching aerosol dispersal if there was a compromise in the mask interface [58]. If possible, HFNC should be administered over NIPPV for initial treatment of acute hypoxic respiratory failure. Furthermore, since there is currently a global shortage of PPE, an N95 should be rationed to one per day and they should be kept in a "splash-proof" container for storage [59].

Epidemiology of critical infection

Predictors for critical outcomes: Comparative studies on hospitalized versus ICU cases consistently reported significantly higher rates of hypertension [13,16-19], cardiovascular disease [17,19], diabetes [13,17-20], cerebrovascular disease [17,60,61] chronic lung disease [19,62,63] malignancy [21,22] obesity [64,65] and age [23–25] > 65 among critically ill groups, though some of these studies did not adjust for baseline confounding factors between groups [66]. Additionally, intractable dyspnea and tachypnea were consistently reported in the literature as significantly associated with critical outcomes [13,17,19,67]. but not mortality [13]. A systematic review demonstrated that lymphopenia [18,68-71], on admission was associated with ARDS and death, especially in younger [71] patients. Studies also demonstrate that COVID-19 pneumonia can predispose

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patients to acute venous thromboembolism [5,72-77] and myocardial injury [17,19,67,78,79] even in the absence of risk factors with respectively elevated lab markers [76,80,81,81-83] notably, the SOFA [19,84] qSOFA [19] CURB65 [19], and APACHE II [84] scoring systems were all strong predictors of ICU mortality in patients with pneumonia from COVID-19 (Figure 3).

Critical outcomes: The most common critical outcome among patients admitted to the ICU for COVID-19 is ARDS. Chinese, US, and Italian studies demonstrate robust rates of ARDS progression among ICU admissions [16,63,85,86] (40-70%). An alarmingly high portion of patients who presented with ARDS decompensated to severe ARDS within 3-7 days [12,16,17,63,87]. Rates for shock [17,67,85,87] and cardiomyopathy [17,67,85] were 20-30% among ICU cohorts as well. High rates for these two concomitant pathologies should determine an appropriate induction agent. Mortality rates were consistent between studies, with around 50% of patients admitted to the ICU for COVID-19 infection.

Early interventions

Patient self-inflicted lung injury: Amplified respiratory drive during AHRF and ARDS is superimposed on NIPPV to generate large tidal volumes that, while normally tolerable in AHRF of extrapulmonary etiology, cause stress fractures and regional strains that result in intra-tidal shifts of gas to dependent areas of lung [88] with regional overdistension despite deceivingly modest elevations in global lung volume [89]. As lung aeration and oxygen saturation decline, more vigorous spontaneous efforts correspond with increasingly negative intrathoracic pressures that enhance venous return and augment transmural vascular hydrostatic pressures in the setting of increased capillary permeability from de novo ALI, worsening pulmonary edema [90] Lastly, excessive concentric shortening of the diaphragmatic muscles due to patient effort and insufficient mechanical support can cause muscle inflammation and myofibrillar damage [91].

NIPPV: Early initiation of NIPPV is associated with an approximately 50% reduction in the need for intubation in ARDS but does not affect mortality [92,93]. Studies on NIPPV outcomes in CARDS patients found similar failure rates of 13% [94] and 52% [60]. The inability to improve P/F after 1 hour of therapy is the greatest predictor of the need for endotracheal intubation and invasive mechanical ventilation [95]

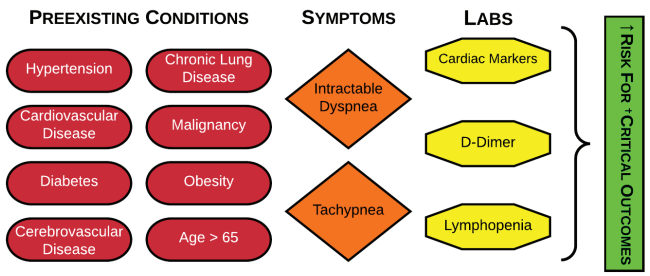

Figure 3: Predictors of poor critical outcomes.

HFNC: Conventional low-flow oxygen devices provide 100% FiO2 at a rate of 15 L/min less than normal physiologic inspiratory flow rates, resulting in dilution of oxygen concentration as room air mixes with pure oxygen to fulfill a respiratory demand [96]. This differential is exaggerated during respiratory distress, resulting in the entrainment of a much larger proportion of room air relative to pure oxygen [97]. HFNO overcomes these flow limitations by delivering up to 60 L/min of humidified gas which typically meets or exceeds the physiologic inspiratory flow of 30L/min to minimize room air entrainment and maximize FiO2 [98] Compared to NIPPV, HFNO is more comfortable for patients and facilitates self-proning [99-102] expectoration, oral suctioning, mucociliary clearance [103] and airflow conductance [104] Two studies reported HFNC failure rates for CARDS patients with a P/F < 200 mm Hg of 63% [105] and 100% [106]; conversely, CARDS patients with a P/F > 200 had a failure rate of 0% [105]. Additionally, a retrospective case-control study comparing HFNC with NIPPV in 73 patients with COVID-19 pneumonia found that failure rates for HFNC and NIPPV were 22% and 100% respectively [84].

Self-proning: A pilot study carried out in a single urban emergency department in New York City found that patients with moderate to severe hypoxemia related to COVID-19 lung injury demonstrated improvement in their SpO2 after being placed in the prone position for 30 to 120 minutes followed by 30 to 120 minutes in the left lateral decubitus position while on HFNC [101]. Other anecdotal studies have reported similar successes with self-proning of COVID-19 patients on HFNC [107,108].

Intubation and ventilatory management

The following guidelines have been founded on multiple literature reviews and expert recommendations regarding the minimization of aerosolization during intubation of patients with COVID-19:

Special considerations for COVID-19 patients: Critically ill patients with respiratory failure have the inadequate functional residual capacity (expiratory reserve volume + residual volume) to provide the necessary oxygen stores to comfortably endure the apneic phase of intubation [118]. Furthermore, as de novo lung injury progresses to ARDS, stress fractures along the alveolar-capillary interface compromise ventilation resulting in shunt physiology [119]. These patients are less amenable to preoxygenation than the typical "medically optimized" surgical patient. These patients can therefore not endure long apneic episodes, which can precipitate anoxic brain injury, cardiovascular collapse, and death [118]. Though manual ventilation is considered an AGMP, wearing appropriate airborne PPE and maintaining a tight mouth-mask seal will more effectively denitrogenate reserve lung volume for a shorter manual ventilation time [120] which, in turn, lowers viral aerosolization and transmission risk [121]. Supplemental HFNC while ventilating has the added benefit of delivering positive pharyngeal pressure (essentially low-grade PEEP ≈ 8 cm H2O) that prevents post-expiratory atelectasis common during denitrogenation of noncompliant lung [122].

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Driving pressure: Ventilator-induced lung injury (VILI) is due to (1) excessive volume mechanically delivered to distal air spaces that produce stress-strain fractures at the capillary alveolar interface (volutrauma); (2) a systemic inflammatory response that increases pulmonary capillary permeability and pulmonary edema (biotrauma); and (3) shear injury from cyclical lung collapse during expiration (atelectrauma) [123-125]. The driving pressure is the difference between the plateau pressures and PEEP. Alternatively, it can be expressed as the ratio of tidal volume to respiratory system compliance, and is proportional to the ratio of the tidal volume to the FRC [126].

$$\Delta P = \left(P_{PLAT} - PEEP\right) = \left(\frac{Vt}{C_{rs}}\right) \propto \left(\frac{Vt}{FRC}\right)$$

A study by the New England Journal of Medicine found that driving pressure was the greatest predictor of ventilator injury among patients with ARDS [127,128] which was later confirmed in two meta-analyses [129,130].

Ventilating the CARDS patient: As discussed in the section on P-SILI, volumetric information provided by global ventilatory readings (Vt) does not accurately reflect the degree of regional volutrauma and localized lung strain common in patients with ARDS. Gattinoni and colleagues speculate that 70% of patients with severe COVID-19 have only slightly decreased pulmonary compliance and consequently do not maintain the high-driving pressures typical of "traditional" ARDS [131-133]. This group of patients is termed the "L-" or "Class I" phenotype of CARDS. Because compliance is only marginally reduced, delivering high PEEP in these patients can theoretically increase lung volume and precipitate VILI. The other 30% of patients are hypothesized to demonstrate the classic ARDS profile which consists of low compliance, high driving pressures, and higher recruit ability. These patients are said to possess the "H-phenotype" of CARDS. Nevertheless, most guidelines recommend using the same lung-protective ventilation strategy for both subtypes, i.e. low tidal volumes to prevent volutrauma and biotrauma and high PEEP to prevent atelectrauma.

Corticosteroids: The WHO offers weak best-practice recommendations [134] against the use of corticosteroids in patients with SARI from COVID-19 pneumonia, except in patients with concomitant shock. These recommendations are extrapolated from other viral studies which showed no survival benefit of corticosteroids among SARS patients [135] no reduction in mortality among influenza patients [136,137] and delayed viral clearance of the LRT in MERS-CoV patients [138]. In regards to the latter study, there is no evidence that delayed viral clearance in MERS-CoV is reflective of delayed clearance among patients with COVID-19 taking corticosteroids. Moreover, a more recent study on CARDS patients by Fang and colleagues found that low-dose corticosteroid therapy did not delay SARS-CoV-2 viral clearance [139]. Studies have shown reduced mortality and shorter length of stay among severe ARDS cases administered high dose methylprednisolone [87,140,141].

Proning: ARDS is associated with unevenly distributed inflammation, edema, and atelectasis that results in a greater transpulmonary pressure gradient that more sharply increases from dependent to nondependent areas of the lung. In the prone position, the pleural pressure is less negative in the nondependent lung and less positive in the dependent lung, reducing the pleural pressure gradient and by extension the transpulmonary pressure gradient. The result is more homogenized alveolar ventilation and less V/Q mismatch, decreasing the intra-pulmonary shunt fraction. Proning should be initiated within 12-24 hours if patients continue to demonstrate P: F ratios < 150 mmHg as reported in the PROSEVA trial (Figure 4) [142].

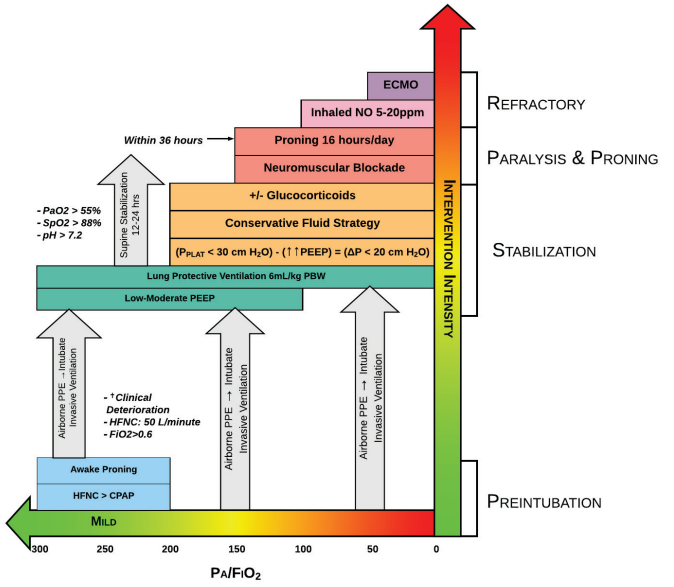

Figure 4: Managing COVID-19 patients in the ICU setting.

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