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Ventilation of COVID-19 patients in intensive care units

About 5–15% of patients with COVID (corona virus disease)-19 infection require intensive care surveillance and ventilatory support. Current recommendations suggest early intubation of COVID-19 patients mainly for two reasons: (1) severe hypoxemia with PaO_2/FiO_2 often <200 mm Hg, fulfilling Berlin criteria of moderate-to-severe acute respiratory distress syndrome (ARDS); and (2) to protect staff from viral transmission [1, 2]. Mortality during mechanical ventilation appears to be high, however, and lung-protective ventilation is mandatory. Gattinoni et al. reported that many patients with COVID-19 pneumonia are initially characterized by a relatively well-preserved lung compliance despite severe hypoxemia, which is generally not observed in typical ARDS [3, 4]. Their concept of "L-type" and "H-type" pneumonia was presented at an ESICM webinar on April 2, 2020 [5]. These contributions and current recommendations form the basis of this brief review.

The proposed COVID-19 pathophysiology is given in more detail elsewhere [4]. In brief, viral infection may lead to subpleural inflammation, an increase in vascular permeability, and interstitial oedema. Impaired (regional) vasoreactivity with vasoplegia may counteract hypoxic vasoconstriction and further increase shunt fraction, which can be easily estimated (**Fig. 1**, ①). The physiologic response to hypoxemia is increased ventilation with increased tidal volume and an increase in respiratory rate. An increased metabolic drive from inflammation, high fever, and a rise in oxygen consumption and demand further increases respiratory drive and breathing work. Patients need careful surveillance for early detection of deterioration (**Fig. 1**,②). Biomarkers are helpful in assessing the clinical trend. Current suggestions reserve the use of high-flow nasal oxygen (HFNO) or noninvasive ventilation (NIV) for mild hypoxemia, with airborne precautions and a low threshold for intubation to avoid viral nosocomial transmission to staff [1]. In addition, similar to ventilator-induced lung damage (VILI), stress and strain on the lung associated with noninvasively generated high tidal volumes may also cause patient self-induced lung injury (P-SILI). Unlike ARDS, the majority of COVID-19 lungs are not small and stiff ("baby lung"), but have a near-normal compliance and will likely not benefit from high positive end-expiratory pressure (PEEP). For COVID-19 patients, the precise role of early HFNO or NIV therapy in moderate to severe hypoxemia should be clarified in a clinical trial setting with a special focus on viral transmission to healthcare workers.

In worsening hypoxemia, additional end-organ failure, or in the presence of contraindications such as delirium, invasive mechanical ventilation is required. Timely intubation should be carried out by the most skilled operator in a small drilled team, preferably avoiding bag-mask ventilation, using rapid sequence induction, video-laryngoscopy, personal protective equipment, and end-tidal capnometry. Ventilator settings initially with lower PEEP and higher tidal volume than in typical severe ARDS can be adapted with targets as indicated (**Fig. 1**, ③④), with a PEEP of 8 cm H_2O, driving pressure <15 cm H_2O, and a plateau pressure to achieve tidal volumes of about 8 ml/kg predicted body weight especially in hypercapnic patients to begin with. The PEEP can then be gradually up-titrated to about 15 cm H_2O as needed (**Fig. 1**,⑤), keeping driving pressure (ΔP) low. Importantly, ΔP is not simply the difference between PEEP and plateau pressure, but the resulting tidal volume (VT) should be normalized to the respiratory system compliance (CRS), i.e., ΔP = VT/CRS, and hence be adjusted to the size of the aerated lung. Tidal volume too low for the size of the aerated lung may lead to hypoventilation and atelectasis. A low compliance indicates a functional baby lung and usually responds to higher PEEP (**Fig. 1**,⑥). By contrast, COVID-19 patients often do not have baby lungs. Instead, they usually benefit from early prone positioning for 16 h or longer depending on the effect, in order to improve ventilation–perfusion (V/Q) mismatch by redistributing pulmonary perfusion (**Fig. 1**,⑦). Indeed, the concept of *L-type* and *H-type* COVID-19 pneumonia was proposed because many patients initially have *low* elastance (= high compliance), a *limited* PEEP response and *low* recruitability, and a *low* V/Q matching (L-type).

Some patients may develop "hyperinflammation" and aggravation of pulmonary oedema, partly due to viral infection, bacterial superinfection, VILI or P-SILI, heart failure, fluid overload, multi-organ dysfunction, or a combination of all of these factors. Worsening inflammation may indicate transition toward the H-type, characterized by *high* elastance (= low compliance), a *higher* recruitability and PEEP response, and a *high* right-to-left shunt [4, 5], i.e. typical ARDS. These patients may benefit from a further stepwise increase in

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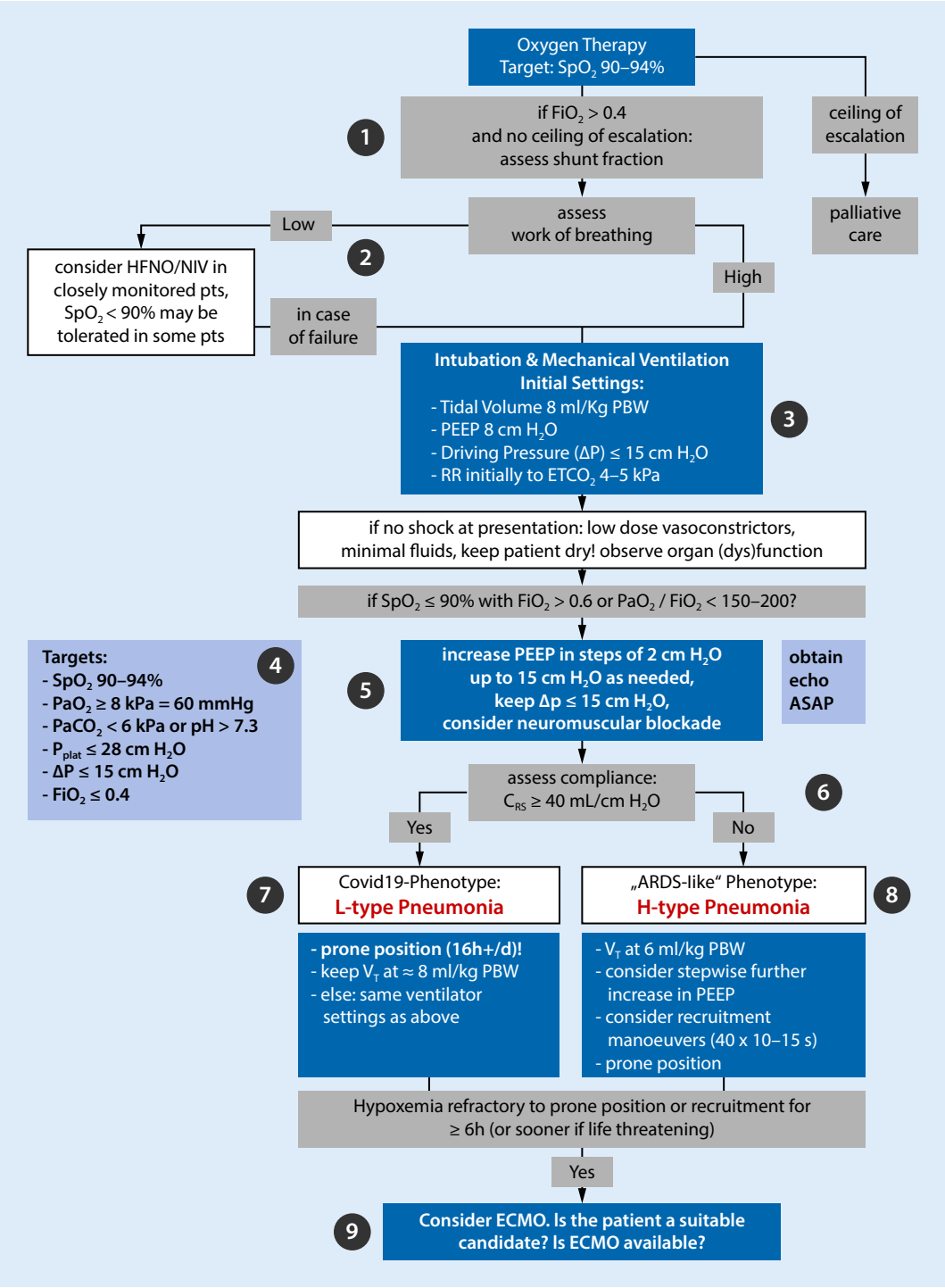

Fig. 1 ◀ Illustration of a ventilation strategy for COVID-19 patients. *SpO2* O_2 saturation, *NIV* noninvasive ventilation, *MV* mechanical ventilation, *PBW* predicted body weight, *pts* patients, *HFNO* high-flow nasal oxygen, *ΔP* driving pressure, *RR* respiratory rate, *ETCO2* end-tidal CO_2, *VT* tidal volume, *PEEP* positive end-expiratory pressure, *CRS* compliance of the respiratory system, *kPa* kilopascal (to convert to mm Hg, use factor 7.5), *(vv-)ECMO* veno-venous extracorporeal membrane oxygenation. (Modified from [5])

PEEP even beyond 15 cm H_2O every 15–30 min, or possibly from recruitment maneuvers (◘ **Fig. 1**,⑧). Neuromuscular blockade may improve transpulmonary pressure and has an anti-inflammatory effect. Fluids should be restricted depending on cardiac pre-load, with a negative daily balance to reduce pulmonary oedema. Heparin should be given to prevent pulmonary embolism and micro-thrombosis in this presumably pro-thrombotic disease. Other causes of respiratory failure should be repeatedly assessed. Clinical evidence of neurotropic effects of the virus should not be missed. If oxygenation is still inadequate and the patient is deteriorating, vv-ECMO (veno-venous extracorporeal membrane oxygenation) must be considered (◘ **Fig. 1**,⑨).

In summary, ventilation of COVID-19 ICU patients is challenging because of the heterogeneous lung pathology that requires an individualized lung-protective ventilation strategy to improve outcome.

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Conflict of interest. S. Möhlenkamp and H. Thiele declare that they have no competing interests.

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