Swift recovery of severe hypoxemic pneumonia upon morbid obesity

C. Galland (*), F. X. Ferrand (*), A. Cividjian (**), B. Sergent (*), C. Pichot (**), M. Ghignone (***), and L. Quintin (**)

Abstract: A morbidly obese (body mass index = 55.5) female patient presented with severe hypoxemic community acquired pneumonia [PaO2/FiO2 (P/F) = 57] with primarily right basal atelectasis, but without bilateral opacities in the upper lobes on the chest X-ray. Major O2 desaturations led the nurses to object to moving the patient to the prone position: muscle relaxation combined to prone position was impossible. Therefore, stringent 60° reverse Trendelenburg legs down position was constantly maintained during mechanical ventilation through the endotracheal tube, using low pressure support (pressure support = 5-10 cmH2O) and high positive end-expiratory pressure (PEEP). PEEP was progressively increased to 20 cmH2O, and little or no sedation was used. A P/F improvement from 57 to 200 over three days allowed removing the tracheal tube. The patient was discharged 13 days after admission. In this paper, the use of high PEEP in the context of morbid obesity, and low pressure support are discussed.

Key words: Community acquired pneumonia; hypoxemic pneumonia; severe hypoxic distress; P/F < 100; acute respiratory distress syndrome; morbid obesity; positive end-expiratory pressure; high PEEP; spontaneous ventilation; pressure support; weaning; “Smart Care”; alpha-2 agonist; clonidine.

Acute respiratory distress syndrome (ARDS) in morbidly obese patients is challenging (1). At rest, the increased weight of the chest wall and the high intra-abdominal pressure reduce the distending pressure of the lung (namely, the “transpulmonary pressure”), which becomes less positive. This generates atelectasis, impaired ventilation/perfusion ratio, increased breathing work, and lowered respiratory reserve (1). Therefore, in the setting of ARDS, high PEEP is necessary to prevent de-recruitment (1). When monitoring esophageal pressure (2) in order to separate chest wall and lung elastance, plateau pressures (Pplat) higher than 30 cmH2O can be applied without any overdistension (3). Indeed, an end-inspiratory transpulmonary pressure approaching 27 cmH2O, at partial inspiration, is acceptable in healthy volunteers (4).

Severe [PaO2/FiO2 (P/F) < 100] acute respiratory distress syndrome (ARDS) is increasingly dealt with muscle relaxation for 48 h (5) and prone position (6), leading to a remarkable reduction in 90 days mortality (~22% and -43%, respectively). The authors emphasized early weaning (7), e.g. after 3 days (5, 8) if appropriate. They also emphasized that teams should be fully accustomed with prone positioning (6).

We present an severe acute hypoxic respiratory failure. Given morbid obesity combined to threatening O2 desaturations, the nurses of our community hospital objected to moving the patient to the prone position. Her pathology was presumably linked to community-acquired pneumonia. Therefore, heterodox management, without muscle relaxation and without prone position, was proposed, and followed by a swift recovery.

Case report

A 50 years old female patient experienced dyspnea at home. Upon arrival of the medical team, she had an O2 saturation (SaO2) of 70% in room air, increasing to 89% when receiving O2 at a rate of 9 L.min⁻¹ through a high O2 concentration mask. Her respiratory rate was 30 cycles.min⁻¹ (c.min⁻¹), heart rate was 120 b.min⁻¹, and arterial blood pressure 186/111 mmHg. A history of bronchitis, two weeks prior to admission, with possible treatment with antibiotics was the only anamnestic.


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finding, without any other detail. In the emergency
department of the community hospital where she
was admitted, respiratory rate was 25-30 c.min⁻¹,
arterial blood pressure was 190/120 mmHg, and
heart rate 110 b.min⁻¹. Past medical history included
morbid obesity (body weight: 130 Kg, height: 153 cm, body mass index: 55, with a 30 kg increase
over the past 3 years), chronic arterial high blood
pressure, type 2 diabetes, smoking (15 pack-year),
and alcohol use (2 oz per day). Usual medications
daily dose) were gliclazide 60 mg, sitagliptine
50 mg, metformine 1000 mg, irbesartan 150 mg,
and hydrochlorothiazide 12.5 mg. She was diag-
nosed by the emergency physician as having
pulmonary edema. Consequently, furosemide
80 mg, and a 2 mg nitroglycerin bolus followed by
a continuous infusion at a rate of 1 mg.h⁻¹, were
prescribed by the emergency department physician.
Echocardiography by an experienced cardiologist
did not show any increase in left ventricle filling
pressure, evidenced normal left ventricle systolic
and diastolic function (E/E’ < 8), as well as normal
or high normal estimated systolic pulmonary
pressure (circa 20 mmHg added to right filling
pressure). The inferior vena cava was not visualized.
She lost consciousness at the time of the instauration
of continuous positive airway pressure ventilation.
This motivated the insertion of an orotracheal tube
into the trachea, as well as sedation using a
midazolam-sufentanyl continuous infusion. Upon
examination by the intensivist, major cyanosis was
observed, with a SaO₂ lower than 70% despite a
100% FiO₂. At that time, the peak inspiratory
pressure (Ppeak) was 65–70 cmH₂O in the supine
position and using a tidal volume (Vt) of 350 ml and
a PEEP of 5 cmH₂O. Patient examination revealed
severe bilateral wheezing, as well as major bilateral
dermatitis with erythema that was more marked on
the right leg and lower abdomen. The patient had
several episodes of SaO₂ below 70% occurred. In
Trendelenburg legs down position. At that time,
Trendelenburg
bolus of cis–atracurium,
and stringent 60° reverse
position and using a tidal volume (Vt) of 350
ml and
pressure (Ppeak) was 65–70
position and using a tidal volume (Vt) of 350
ml and
a PEEP of 5 cmH₂O. Patient examination revealed
severe bilateral wheezing, as well as major bilateral
dermatitis with erythema that was more marked on
the right leg and lower abdomen. The patient had
also severe lower extremities and lumbar edema
(“anasarque”).

In the critical care unit (CCU), given the
precarise state, mechanical ventilation still
required 100% FiO₂, 60–70 cmH₂O Ppeak, and 50–
60 cmH₂O Pplat despite the 60° reverse
Trendelenburg legs down position. At that time,
several episodes of SaO₂ below 70% occurred. In
front of this scene, the nurses argued that prone
positioning would lead to cardiac arrest. Therefore,
an heterodox treatment (Table 1) was planned,
consisting in the administration of a single 20 mg
bolus of cis–atracurium, and stringent 60° reverse
Trendelenburg (9) during the entire intubation
period. A Drager Evita 4/ XL respirator served to
perform controlled mechanical ventilation. The
mode of ventilation was a volume-controlled mode,
using a Vt of 5 ml.kg⁻¹. The patient received
repeated subcutaneous administration of 150 μg of
clonidine to lower arterial blood pressure and heart
rate. This allowed discontinuing nitroglycerin
administration, and lowering midazolam-sufentanyl
requirements (10). Additional furosemide (80 mg
followed by 250 mg) was given, as well as 125 mg
of methylprednisolone, 2 g of amoxicillin 2 g
3 times in a day, 200 mg of clavulanic acid 3 time in
da day, 5 mg of terbutaline 8 times in a day, 0.5 mg
of ipratropium 8 times in a day, and 1 mg of
budesonide 8 times in a day. Given the precarious
state and severe mycosis on the groin, no attempt
was made to perform a CT scan or insert a central
venous line to assess O₂ saturation in the superior
vena cava. After 24 h, following improvement of
hypoxia and repeated doses of clonidine, arterial
blood pressure and heart rate normalized to 120–
130/70–80 mmHg, and 70–80 b.min⁻¹, respectively.
Daily echocardiography repeatedly confirmed the
observations made in the emergency department,
confirming the absence of right ventricle dilation
that would have been favored by the high PEEP
(20 cmH₂O ; see below). Given the absence of right
ventricle dilation, elevated left ventricle filling
pressure, and systolic and diastolic dysfunction, and
based on a 2.4 μg.ml⁻¹ D dimer (normal < 0.5), a
400 pg.ml⁻¹ B-natriuretic peptide, a 0.27 ng.ml⁻¹
procalcitonin, and 30 mg.L⁻¹ C reactive protein
plasma level, as well as a 9580 mm⁻³ white cell
count (WCC), a 37.4°C temperature, an intact
kidney and liver function, and a 35 simplified acute
physiology score II (SAPS II), the diagnosis was
reset to community-acquired pneumonia and
validated by the cardiologist and chest physician in
charge of the critical care unit. On day 3, endotracheal
cultures revealed a pneumococcus and a staphylo-
coccus that were adequately treated with the
association of amoxicillin and clavulanate.

Over a few hours following admission to the
CCU, discontinuation of sufentanyl and lowering of
midazolam infusion to 1 mg.h⁻¹ allowed switching
from controlled mechanical ventilation to pressure
support, using a 100% automatic tube compensation,
lowest trigger, 8-10 cmH₂O of pressure support, and
first a 5 followed by a 10 cmH₂O PEEP. After 24 h,
under pressure support and stringent positioning,
Pplat decreased from 50-60 to 30-35 cmH₂O. As a
consequence, PEEP was increased to 20 cm H₂O.
Given the inability to measure Pplat when the
pressure support mode of the Drager ventilator is
used, Ppeak was equated to Pplat. The PaCO₂

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always remained below 60-65 mmHg. Low respiratory rate below 20-25 cpm was achieved, following progressive oxygenation and lowered O₂ consumption, for more than 48 h. External cooling to 35-37°C was achieved. P/F increased from 57 upon arrival in the critical care unit and under the above-described ventilation parameters [PEEP of 10 cmH₂O, Vt of 5-6 mL.kg⁻¹, 100% FiO₂, in line with (11)], to 76 at day 3 (PEEP of 15), 106 at day 2 (PEEP of 20), 155 and 180 at day 3 (PEEP of 15), and 200 at day 4 (PEEP of 15). FiO₂ changed from 100% to 40% within 3 days. Starting at day 3, PEEP was progressively changed from 20 to 10 cmH₂O at the time of endotracheal tube removal on day 4. Given the improvement in clinical status, P/F, and chest X-ray (see right part of Fig.), the midazolam was stopped on the morning of day 2, while clonidine administration was maintained only upon requirements day 3 to day 4. Methylprednisolone, terbutaline, ipratropium, and budesonide were progressively tapered over 3 days. Furosemide was maintained until discharge to ward. Pressure support was lowered from 8-10 cmH₂O on day 1, to 5-8 cmH₂O on day 2. In addition, to avoid high Vt under pressure support mode (12, 13), the “Smart Care” function (14) of the Evita Respirator was used during day 2, 3, and 4. This ventilation mode generated low pressure support (3-6 cmH₂O), low Vt (300-350 mL), no sternal notch retraction, no thoraco-abdominal dys-coordination, or no increased respiratory rate (appendix). The trachea was extubated on day 4, that is after 74 h. Non-invasive ventilation (NIV) was then instituted, using a PEEP of 10, the same as the one used immediately before extubation. The patient was discharged to pneumology on day 7. At that time, she was receiving 6 L.min⁻¹ of O₂, and she had lost 10 Kg. She still had major peripheral edema. On day 13, she was discharged from hospital. Within two months, she lost more than 25 Kg, quit tobacco, and normalized oxygenation. Indeed, at that time, she had a PCO₂ of 31 mmHg, a PO₂ of 89 mmHg in

**Table 1**

Clinical course over time - mains events related to ventilation and sedation with approximate time

<table>
<thead>
<tr>
<th>day</th>
<th>approximate time</th>
<th>event</th>
<th>Mode of Ventilation</th>
<th>FiO₂</th>
<th>Vt (ml)</th>
<th>PS (cm H₂O)</th>
<th>PEEP (cm H₂O)</th>
<th>P/F</th>
<th>sedation</th>
<th>Ramsay</th>
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<tbody>
<tr>
<td>1</td>
<td>10 AM</td>
<td>arrival to ED</td>
<td>SV</td>
<td>02 = 9 L.min⁻¹</td>
<td>500 then 350</td>
<td>5</td>
<td>etomidate-succinylcholine then midazolam 5 mg.h⁻¹ sufentanyl 10 mcg.h⁻¹</td>
<td>no sedation</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>1 PM</td>
<td>intubation ED</td>
<td>CMV</td>
<td>1</td>
<td>350</td>
<td>10</td>
<td>57</td>
<td>d/c sufentanyl, midazolam 2 mg.h⁻¹ clonidine 150 mcg s.c as required, cis-atracurium 20 mg</td>
<td>6</td>
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</tr>
<tr>
<td></td>
<td>3 PM</td>
<td>arrival CCU</td>
<td>CMV</td>
<td>1</td>
<td>350</td>
<td>10</td>
<td>57</td>
<td>d/c sufentanyl, midazolam 2 mg.h⁻¹ clonidine 150 mcg s.c as required, cis-atracurium 20 mg</td>
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<td>7 PM</td>
<td>PS</td>
<td>1</td>
<td>8</td>
<td>15</td>
<td>76</td>
<td>midazolam 1 mg.h⁻¹, clonidine as required</td>
<td>6</td>
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<td>2</td>
<td>8 AM</td>
<td>PS</td>
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<td>10 AM</td>
<td>PS</td>
<td>1</td>
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<td>20</td>
<td>5-6</td>
<td>8 then as per smart care</td>
<td>4-5</td>
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<td>8 then as per smart care</td>
<td>20</td>
<td>4-5</td>
<td>3-4</td>
<td>clonidine s/c as required</td>
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<td>6 PM</td>
<td>PS</td>
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<td>clonidine s/c as required</td>
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<td>4</td>
<td>8 AM</td>
<td>PS</td>
<td>0.4</td>
<td>300-350</td>
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<td>200</td>
<td>d/c clonidine</td>
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<tr>
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<td>extubation</td>
<td>0.4</td>
<td>5 then “Smart Care”</td>
<td>10</td>
<td>2</td>
<td>2</td>
<td>no sedation</td>
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<td>no sedation</td>
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room air during late afternoon. She had also minimal nocturnal desaturation episodes.

**Discussion**

A swift recovery of severe hypoxemic pneumonia was observed in a patient with multiple co-morbidities, using a treatment that differs from the conventional practice of muscle relaxation and prone positioning. In the following discussion, our heterodox management using high PEEP, spontaneous ventilation with pressure support, a steep reverse Trendelenburg position, and light sedation will be examined, one element after the other.

*Differential diagnosis between fluid overload, pulmonary embolism, ARDS, and severe hypoxemic pneumonia*

Fluid overload was suspected, based on the extensive peripheral edema. However, experienced echocardiographists ruled out, early on and repeatedly, left ventricle systolic dysfunction, elevated left ventricle filling pressure, or heart failure with preserved ejection fraction. Therefore, increased pulmonary veno-capillary pressure was not present. In this hypertensive and obese patient, the repeatedly demonstrated absence of diastolic dysfunction was striking. Even though, and before echocardiography, furosemide was given in the emergency department. It was also administered again in the CCU, despite the absence of elevated left ventricle filling pressure. The reasons were the presence of threatening desaturation episodes, and major peripheral edema. Hypovolemia increases West zones 1 & 2, and alveolar dead space. This effect could have worsened desaturation. However, this was not the case, presumably because of the beneficial effect of positioning, spontaneous ventilation with pressure support, and increasing PEEP.

Absence of right ventricle dilatation excluded major pulmonary embolism. Doppler examination of the right leg ruled out peripheral venous thrombosis. By exclusion, a patient is qualified "as having ARDS, as long he/she has respiratory failure that is not fully explained by cardiac failure or fluid overload" (15).

The low P/F was established according to published criteria (11). However, the Berlin definition of ARDS (15) requires the presence of bilateral opacities consistent with pulmonary edema on the chest radiograph. No, or minimal, bilateral opacities were observed in the upper lobes (see left part of Fig. 1). Therefore, rather than true severe ARDS, this patient presumably presented a severe hypoxemic community-acquired pneumonia, or "lobar" (16) ARDS limited to the lower lobes. The recovery within 3 days of mechanical ventilation and antibiotic therapy fits with this diagnosis. By contrast, severe ARDS generally necessitates 9 days (range : 5-17) of mechanical ventilation (15).

The discrepancy between this mild pulmonary infection and such a severe hypoxia is probably related to a shunt effect. The relationship between occlusion of the bronchial airway and magnitude of the shunt is not linear, and a 55% occlusion leads to

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![Fig. 1. — Chest X-rays. Left : 1 h before loss of consciousness and intubation. Right : a few hours before extubation after 3 days of low level pressure support with high PEEP, and strict reverse 60° Trendelenburg position. The initial pulmonary diffuse opacities were primarily localized at both lower lobes (community-acquired pneumonia or “lobar” (16) ARDS). Subsequent clearing occurred within 3 days, and is compatible with recruitment of atelectasis by high PEEP in this morbidly obese patient.](image-url)
an 80% shunt (17). This infection-generated shunt can be worsened by a reduced transpulmonary pressure and end-expiratory collapse (i.e. atelectasis). As mentioned above, reduced transpulmonary pressure is worsened, in the morbidly obese patient, due to elevated intra-abdominal pressure (1). The lowered reserve under resting conditions (1) is strained upon infection.

Positive end-expiratory pressure management

The chest X ray shows a lobar community acquired pneumonia, which was limited to the basis (see left part of Fig. 1). The PEEP, in that case, should be in the range of 5 ± 2 cmH₂O (16). Such a PEEP was used in our patient during her stay in the emergency department, but did not worked. Morbid obesity generates high intra-abdominal pressure, which is transmitted through the diaphragm by a factor 0.5 (range: 0.25-0.8) (18) in the supine position. Morbid obesity increases intra-abdominal pressure to 18 cm H₂O on average (18). Therefore, only a much higher PEEP (up to 20 cmH₂O for 24 h on day 2) allowed recruiting some of the atelectasis observed at the basis of the lungs. Such a high PEEP counteracted the elevated intra-abdominal pressure (18).

When setting a PEEP level, caution must be paid to avoiding right ventricle dilatation, and this was the case in our patient. To ventilate the lungs themselves adequately, and partition between lungs and chest wall mechanics (2), an esophageal balloon should be used (2). It allows measuring end-inspiratory transpulmonary pressure, and guiding PEEP adjustment. Using that technique, PEEP can be sometimes raised to high levels. As an example, it has been set to 22 cmH₂O in patients (3 obese out of 7 subjects) that were candidate to receiving extracorporeal membrane oxygenation (ECMO) (3). This maneuver increased P/F from 67 to 180 over 30 min, and allowed avoiding extracorporeal membrane oxygenation in those patients (3). Similarly, increased end-expiratory transpulmonary pressure avoids cyclic collapse, and is able to ameliorate P/F over 72 h (19).

PEEP level can also be set without measuring transpulmonary pressure. A very high PEEP can be empirically used to increase P/F over a few hours (20). When not able to identify the inflexion point on the P-V curve, or before performing a CT scan, experienced investigators set PEEP at 15-16 cmH₂O (21, 22). Therefore, in a community hospital, our much less sophisticated approach (trial PEEP) reproduced these (21, 22) observations. Here, high PEEP improved P/F over 3 days, as reported elsewhere (3, 19, 20, 23). This swift recovery, in one patient, contrasts with slower recoveries, in large trials, where low PEEP (control group) (21) or muscle relaxation (5) associated to prone position were used [time to successful extubation was 17 ± 16 days or 19 ± 21 days (6)].

The use of spontaneous ventilation

When considering the Pplat of 50 cmH₂O on day 1, predicted mortality rate is 65-70% (24). This risk may even be further increased by morbid obesity. The combination of high Pplat, threatening desaturations, young age, morbid obesity, and P/F of 57 should normally have led to combine prone position (6), muscle relaxation (5), NO/almitrine, or even ECMO (25). As muscle relaxation (5) and prone position (6) reduce mortality, one may question whether departing from this therapeutic option is ethically acceptable. This must be nuanced by the fact that prone positioning requires knowledge during severe ATRDS (6). In our case, the nurses, de facto, objected to prone the patient. The intensivist was left with possible cardiac arrest upon prone positioning or heeding to a 65-70% risk of death (24), majored by morbid obesity. The choice was dictated by necessity.

Spontaneous ventilation cured hypoxia within 3 days. Insofar as it preserved a diaphragm function, it helped to unfold the alveoli of the lower lobes. Spontaneous ventilation should be seen in a continuum (26) with continuous mandatory ventilation associated with muscle relaxation, the latter being briefly used when acute cardiorespiratory distress or elevated respiratory rate are preeminent (26). Indeed, upon arrival to the CCU, major desaturations led to a single bolus of muscle relaxants, allowing early transition to spontaneous ventilation. This observation contrasts with usual practice (5, 6).

Leading groups advocated early transitioning from controlled mechanical ventilation to spontaneous ventilation (7). Indeed, proponents of muscle relaxation recommend stopping sedation as soon as possible after the initial 48 h and switching ventilation mode to pressure support (8). Accordingly, in the context of ARDS, spontaneous ventilation has been proposed (27-34). Indeed, an early use of intermittent mandatory ventilation is associated with a 16% mortality rate (35). Patients presenting with a P/F lower than 300 can be managed using pressure support, when respiratory rate does not reach too high values (36). Therefore,
spontaneous ventilation combined with pressure support (23, 37), as early as possible, is heterodox, but not heretic. In contrast, a minimization of transpulmonary pressure and the ventilation on the deflation limb of the P-V curve (38, 39), used here (see Fig. 2), are orthodox.

**Position of the patient**

A stringent 60° reverse Trendelenburg legs down position was maintained to improve respiratory mechanics (9). As gravity directs flow, a “zone 3” may have led to better VA/Q ratio upon quasi-upright position. The inference is that standing lowers intrathoracic pressure by 5-10 cm H$_2$O (41). The inference is also that transitioning from controlled to spontaneous ventilation lowers the intrathoracic pressure by 5-10 cm H$_2$O (18). These simple tricks may have reduced Pplat, allowing high PEEP.

**Management limitations**

The peak temperature of 37.9 °C in our patient was compatible with mild pulmonary infection. Given her minimally increased O$_2$ consumption, external cooling (42, 43) further lowered respiratory rate. Indeed, during mild ARDS, weaning from controlled mechanical ventilation using pressure support is only possible when respiratory rate is low (36). With the same idea in mind, our heterodox strategy cannot not be used when severe metabolic acidosis is present, because acidosis generates a high respiratory rate (26).

In contrast to a previous report (23), sedation with alpha-2 agonist was minimized, in line with a policy of no/little sedation (44, 45). Indeed, s.c. clonidine was used during the first 48 h to lower arterial blood pressure and heart rate, taper nitroglycerin, lower O$_2$ (46) and sedative (10) requirements, without impairing ventilation (47). This allowed switching to spontaneous ventilation, lowering Pplat from 50-60 to less than 30-35 cmH$_2$O, and increasing PEEP from 5 to 20 cmH$_2$O. Our patient tolerated the endotracheal tube without combativeness. Hence, the present observation goes one step beyond a first report in the setting of early severe ARDS (23): the mode of ventilation (pressure support-high PEEP) appears more important than the sedative regime in this non-combative patient.

To conclude, muscle relaxation-prone position (5, 6) is the current and accepted practice. Our highly limited observation has no power enough to convey any clinical message. It only goes beyond previous group report (23) and hypotheses (37, 26). Little or no sedation regimes (44, 45) may be considered in non-combative patients. Low VO$_2$ is a key factor for the success of pressure support-high PEEP combinations. Evidence-based documentation is awaited.

**References**


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Appendix

“Smart Care” during severe hypoxic pneumonia: additional comments on Figure 2

On d2, despite a high PEEP of 20 cmH2O, the “Smart Care” mode with pressure support (PS) (1) was set to avoid high tidal volumes (VT) (2, 3). To our surprise, “Smart Care”, over a few hours, lowered PS level to a much lower level than previously set by the physician (from 5-8 to 3-4 cmH2O, 100% automatic tube compensation, trigger to the lowest value), with no/minimal respiratory effort (no sternal notch retraction, no use of accessory muscles, no thoraco-abdominal desynchronization). The patient was perfectly comfortable, and alert with a Ramsay score between 2 and 3. On the pressure-time diagram, the PS level was barely discernible above the PEEP level. Given the high PEEP, one could have thought that the “Smart Care” mode was not functioning appropriately. The session was initiated again, several times, on day 2 and 3, using a PEEP of 20, followed by a PEEP of 15 cmH2O. Again, ventilation was characterized by a very low PS level, and no respiratory fatigue. Blood gases and SaO were in an acceptable range. “Smart Care” was turned off during the night on day 2 and 3.

Low PS level in the setting of high PEEP and ARDS was explained (4, 5) as follows: above critical closing pressure [loop B, figure 5 in (6)], the alveoli remain open upon end-expiration. As the slope of the pressure-volume curve is steep, witnessing a good compliance, above critical closing pressure (7), a small pressure gradient generates a large volume, and therefore large VT (8). However, PS may lead to high transmural pressure (2) and high VT (3) if the subject is not monitored closely. Therefore, the “Smart Care” mode (1) was used to avoid high VT under PS and high PEEP. To our surprise, very low level of PS was needed once the alveoli were opened by the high PEEP (15-20 cmH2O). This complements previous observations (5) and fits with a low resistive work (9) needed when the lung is “in a position of optimal function” (10). Two remarks are necessary: the 100% automatic tube compensation (11) presumably overcomes the resistive work generated by the small diameter of the tracheal tube (12), and the “Smart Care” presumably mimics the low PS level set to counteract the resistive work added by valves and circuits (3-5 cmH2O), when physiological experiments are performed under non-invasive ventilation (13). Whether, upon severe hypoxic pneumonia or ARDS under high PEEP-low PS, “Smart Care” functions well, requires extensive validation.

References appendix

10. Kirby R. R., Continuous positive airway pressure: to breathe or not to breathe, Anesthesiology, 63, 578-80, 1985.