in severe ARDS patients who are unable to protect their airways' from aspiration. Finally, the swift time period is still controversial.

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Cut-off point for switching from non--invasive ventilation to intubation in severe ARDS. Fifty shades of grey?

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Key words: acute respiratory distress syndrome, severe ARDS, non-invasive ventilation

Sir, I would like to thank Drs Skoczyński and Esquinas for their comments. Firstly, let us turn to their secondary points:

1. The initial intention was to pre-oxygenate a severely hypoxic patient before tracheal intubation [1] with a Respironics ventilator in the emergency department (ED), not to manage the whole case under non-invasive ventilation (NIV) with an Evita 4 XL ventilator in the Critical Care Unit (CCU). Nevertheless, the ventilatory discoordination disappeared almost immediately following the initiation of NIV, calling for an *iterative* re-assessment of preconceived strategy.
2. This case was *not* acute respiratory distress syndrome (ARDS), but acute hypoxemic non-hypercapnic respiratory failure: the opacities required by the Berlin definition could not be seen on the chest x-ray taken minutes after admission to the ED.
3. Although the patient was conscious, cooperative and drowsy (Glasgow 14), he was fully able to answer questions, and denied repeatedly having inhaled heroin. Esquinas [2] reported intubation with Glasgow ≤ 11. Thus, unconsciousness is irrelevant.
4. The arrhythmia was not sinus tachycardia, but supraventricular arrhythmia: no P waves were observed on the oscilloscope using a *high*-speed display. Nevertheless, arrhythmia was, presumably, a consequence of hypoxia, a trivial issue not further discussed in the report [3]. Magnesium followed by amiodarone was aimed at isolating, as early as possible upon presentation, a «pure» ventilatory distress *vs*. a combined ventilatory and

circulatory distress. Lung toxicity of a single dose of 450 mg of amiodarone awaits documentation.

5. The interface was a standard oro-nasal mask.
6. High PEEP (up to 20 cm H_2O) generated neither leak nor clinical gastric overdistension, in *this* patient. I recently handled acute hypoxia (SaO_2 = 39%) due to postoperative atelectasis, with PEEP increased over 2 h from 5 to 24 cm H_2O (Drager Evita 4XL, low pressure support: PS to Pplat < 30 cm H_2O, FiO_2 = 1), allowing the pneumologist to perform a bronchoscopy under spontaneous ventilation (SaO_2 = 100% when beginning bronchoscopy), without leaks or gastric distension. The reader will decide whether this is again deliberate malpractice or careful, minute by minute, observation.
7. A high tidal volume (Vt) under PS is no trivial issue [4]. At variance with high PS in the setting of chronic obstructive pulmonary disease (COPD) [5], minimal PS (≤ 8 cm) to compensate for the valves and tubing [6] will generate a low Vt: following the setting up of a high PEEP the lung operates on the highest slope of the pressure-volume curve [7]. The observed Vt was 250–500 mL (not 800–1200 mL as stated by Skoczynski), compatible with permissive hypercapnia (46–69 mm Hg) in a quiet patient with respiratory drive depressed by heroin. This technique was delineated earlier [8]. Guldner proposed similar analysis in animals [9]: see note added in proof [3].
8. Skoczynski and Esquinas question the use of excessively high FiO_2 (FiO_2 = 1). However, the definition of excessive use of O_2 is an FiO_2 > 0.5 when SaO_2 is > 92%, for up to 12–30 h, and excluding the "first 6 h of shock" [10]. Given a P/F≈57, in the ED, the patient received FiO_2 = 1, *en route* toward intubation and controlled mechanical ventilation. As SaO_2 remained < 90% for at least ≈5 h, this does not fit with excessively high FiO_2. Subsequently, FiO_2 was reduced to 0.4 within ≈10 h. As severe hypoxia (PaO_2 = 19–36 mm Hg) is compatible with life in elite climbers [11], the question may be posed whether benign neglect should be extended to an unstable patient presenting with acute cardio-ventilatory distress (P/F ≈ 57 on zero PEEP, 30 L min^{-1} on high O_2 concentration mask; P/F = 75 on PEEP = 15 after 2 h on NIV). Moreover, should SaO_2 = 88–92% be aimed at in the present patient, as proposed in a fully *stabilized* patient [12]?

The modified NIH table [13] (tab. 1) uses high PEEP-low FiO_2 in stabilized *intubated mechanically ventilated patients* (SaO_2≈88–95%), at variance with the questionable combination of high FiO_2-low PEEP [10, 12]:

Accordingly, in a non-intubated unstabilized patient, PEEP was increased up to 20 cm H_2O over 4 h, while FiO_2 was lowered to 0.4 over 8 h, *after* stabilization : "*the practice of using higher FiO_2 cannot be considered unreasonable under these settings*" [10].

The effect of O_2 on the *respiratory rate (RR) as a function of PaO_2* under spontaneous ventilation-PS [14] in the *setting of ARDS,* is to be taken into account to lower the work of breathing, at variance with COPD. Therefore, setting a 88–92% goal in the setting of invasive controlled mechanical ventilation in ARDS in stabilized intubated patients [12] does not apply to the early use of high PEEP-spontaneous ventilation in an unstabilized patient under NIV.

As to the question whether high FiO_2 acts synergistically with other insults to worsen alveolar damage, a "*safe level and duration of O_2 exposure has not been established even in normal humans*" [12]. Accordingly, a cut-off point of $FiO_2 \leq 0.6$ for 8 h 45 could not be retrieved from the reference [12] provided by Skoczynski and Esquinas. Avoiding the closing-opening of alveoli (atelectrauma) with high PEEP presumably avoided inflammation and terminated swiftly the disease. Any synergistic effect of high FiO_2 and inflammation appears irrelevant, given the *short* time course of the disease.

Can 9 to 10 h be considered a swift recovery? To my surprise, the intensivist in charge on day 2 terminated the NIV at 08 h 30 am. In the setting of ARDS, P/F increases over 72 h or more [15, 16]. Thus, the reader may decide whether a recovery time over 10 h is swift or not (day 1, 10 pm : P/F≈57 on zero-PEEP, high O_2 concentration mask; day 2, 08 45 am: P/F = 240, PEEP = 15, FiO_2 = 0.4).

Secondly, *how far should NIV go without being detrimental?* Let's consider Esquinas' data: a) «*in the NIV group, P/F and RR became significantly higher and lower 3–4 hours after randomization*» (Fig. 3 in [2]). b) the avoidance of intubation is reported in 54% of the patients with a P/F = 116 ± 38 [17]: given the standard deviation, some of his patients had a low P/F ≈40–60, as in our report [3]. Indeed, Pichot [3] observed the phenomenon described by Esquinas [2, 17]. Nevertheless, the use of NIV in acute respiratory failure demands caution [18]. Firstly, in the setting of severe ARDS (P/F = 126), 84% of the patients needed intubation [19]. Does this imply that the remaining 16% should be intubated upfront or should they simply observed even more closely to proceed to intubation if appropriate? Secondly, following extubation after respiratory failure, NIV is associated with a 10 h delay re: re-intubation and a higher mortality (NIV: 38%; standard treatment + reintubation: 22%) [20]. Thus, NIV should not be used (except perhaps in COPD or immuno-compromised patients, or as a bridge to intubation). A sober interpretation only implies that patients presenting a second exacerbation of acute respiratory failure after extubation should be very closely re-assessed, e.g. at least hourly, and their trachea intubated early, as needed, should NIV fail. *Individualized minute-by-minute observation in one considered patient (3) does not necessarily agree with epidemiologic findings* [20].

Altogether, NIV is detrimental when extended too far. Indeed, one referee complimented our non-invasive management: "*avoid tracheal tubes, minimize sedation, prevent ventilator-induced lung injury and nosocomial infections*" [21]. Conversely, another referee considered this [3] management as malpractice (*P* 140, l 7). Again, the reader will decide whether our concluding insistence on minute by minute re-assessment in a highly restricted subset [3] was conservative enough.

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The role of Argentine Federation of Associations of Anaesthesia, Analgesia and Reanimation

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Szpital Kielecki św. Aleksandra w Kielcach

Due to previous friendly relationships with Argentinean anesthesiologists, especially with Pedro Klinger, MD, PhD, with whom I worked in the past in Ibiza, Spain, as well as an invitation to participate in the 15th *World Congress of Anaesthesiologists* (WCA), I had an opportunity to familiarize myself with the organization of anaesthesiology care in Argentina. Moreover, this year I was pleased to visit this wonderful country and be hosted by my Argentinean friends, including Marisa Bard, MD, a specialist in anaesthesiology with Polish roots. Argentina is acountry of emigrants and has accepted in past a lot of Polish people, several of whose descendants have become outstanding figures in the medical world.