Anestezjologia Intensywna Terapia
2016, tom 48, numer 1, 66–68
ISSN 0209–1712
www.ait.viamedica.pl

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

Luc Quintin

*Department of Physiology, University of Lyon, Lyon, France*

**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

Należy cytować wersję: *Quintin L:* Cut-off point for switching from non-invasive ventilation to intubation in severe ARDS. Fifty shades of grey? Anaesthesiol Intensive Ther 2016; 48: 62–64. doi: 10.5603/AIT.2016.0011.

**Table 1.** High PEEP group (after protocol change to use high PEEP; reproduced from [13])

| $FiO_2$ | 0.3 | 0.3 | 0.4 | 0.4 | 0.5 | 0.5 | 0.5–0.8 | 0.8 | 0.9 | 1.0 |
|---|---|---|---|---|---|---|---|---|---|---|
| PEEP | 12 | 14 | 14 | 16 | 16 | 18 | 20 | 22 | 22 | 22–24 |

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.

## ACKNOWLEDGEMENTS

Conflict of interest: Luc Quintin holds a US patent 8 703 697, April 22 2014: Method for treating early severe diffuse acute respiratory distress syndrome.

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**Adres do korespondencji:**
*Luc Quintin MD, PhD*
*Physiologie*
*Campus de la Doua*
*8 Rue R Dubois*
*69 622 Villeurbanne, France*
*e-mail: lucquintin@yahoo.com*

Anestezjologia Intensywna Terapia
2016, tom 48, numer 1, 68–70
ISSN 0209–1712
www.ait.viamedica.pl

# Rola Argentyńskiej Federacji Stowarzyszeń Anestezji, Analgezji i Reanimacji w ochronie statusu ekonomicznego jej członków

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

Wojciech Stanisław Pietrzyk

*Szpital Kielecki św. Aleksandra w Kielcach*

Dzięki wcześniejszym przyjaźniom z argentyńskimi anestezjologami — a w szczególności znajomości z dr. Pedro Klingerem, z którym pracowałem wcześniej na Ibizie w Hiszpanii — i ich zaproszeniu do uczestnictwa w 15. Światowym Kongresie Anestezjologów (WCA), mogłem zapoznać się z organizacją opieki anestezjologicznej Argentynie. Także i w tym roku miałem przyjemność odwiedzić ten wspaniały kraj i gościć w domach moich argentyńskich przyjaciół, w tym mającej polskie korzenie specjalistki anestezjologii dr Marisy Bard. Argentyna jako kraj imigrantów, przyjął wielu Polaków, których potomkowie osiągnęli niekiedy znaczącą pozycję w świecie medycznym.

Choć powierzchnia Argentyny jest wielokrotnie większa niż Polski, kraje te łączy wiele podobieństw, jak na przykład wielkość populacji. Wielu argentyńskich anestezjologów, tak jak wielu Polaków wyspecjalizowanych w tej dziedzinie, pracuje stale lub czasowo za granicą.

W samej Argentynie, według danych ustnych z roku 2012, praktykuje około 3800 specjalistów anestezjologii, z czego około 3750 należy do Argentyńskiej Federacji Stowarzyszeń Anestezji, Analgezji i Reanimacji (FAAAAR, *Federación Argentina de Asociaciones de Anestesia, Analgesia y Reanimación*). Z kolei według Rocznika Statystycznego 2012 liczba specjalistów anestezjologii i intensywnej terapii w Polsce wynosi 3414 i plasuje nasz kraj na 5. miejscu wśród innych specjalności. Biorąc jednak pod uwagę istotne nakłady finansowe Ministerstwa Zdrowia na rezydentury z anestezjologii, można przyjąć, że liczba anestezjologów w kraju wkrótce znacząco się zwiększy.

W Argentynie, gdzie szkoleniem specjalizacyjnym anestezjologów zajmuje się FAAAAR, liczba specjalistów jest dostosowywana do potrzeb rynku usług ochrony zdrowia. Było to zresztą przyczyną małego skandalu w trakcie otwarcia 15. WCA w Buenos Aires. Trudna do zdefiniowania politycznie grupa demonstrantów protestowała przed wejściem do centrum kongresowego przeciwko „monopolowi" FAAAAR w kształtowaniu „mafijnej" pozycji anestezjologii w strukturze argentyńskiej ochrony zdrowia. Towarzyszyło temu dosłowne „zasypanie" uczestników zdążających na uroczyste otwarcie przez maszyny rozpylające tysiącami ulotek informujących o olbrzymich dochodach anestezjologów i ich Stowarzyszenia. Do tego z zainstalowanych wielkich głośników słychać było ścieżkę dźwiękową z filmu „Ojciec chrzestny" Coppoli. Bez wątpienia profesjonalnie zorganizowana grupa demonstrantów osiągnęła swój cel — zaintrygowała wielu zagranicznych anestezjologów, w tym i mnie, do pogłębienia tematu.

Należy cytować anglojęzyczną wersję: *Pietrzyk WS:* The role of Argentine Federation of Associations of Anaesthesia, Analgesia and Reanimation. Anaesthesiol Intensive Ther 2016; 48: 64–66. doi: 10.5603/AIT.2016.0012.