

LESS IS MORE IN INTENSIVE CARE



“Less is More” in mechanical ventilation

Luciano Gattinoni^{1*} , Michael Quintel¹ and John J. Marini²

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Though formed in the late 1960s, the field of intensive care medicine solidified its place in the 1970s. Critical care practitioners were typically young and enthusiastic, but lacked the benefit of guidance from an established scientific literature or the experience of “older mentors,” as simply they did not exist. The rational basis of this new specialty was systems physiology and short-term observation, on which most of its monitoring and interventions are based. Interestingly, not only were the “intensivists” young, but so were the patients, compared to now. Very importantly, we naively assumed uniformity of diseases and disease mechanisms and translated our familiar deep knowledge of normal physiology to the pathologic state.

Energy, enthusiasm and the pioneering attitudes of the young intensivists were associated with a widespread tendency to “exceed” the confines of prior experience. Adverse consequences gradually became evident. This exuberance characterized many elements of practice: in septic patients, if milligrams of corticosteroids are good, grams might be better [1]. In nutrition, if 2000 kcal/day is good, 5000 must be better [2]. The same applies to sedations and fluids administration. In hemodynamics, supra-normal values of oxygen transport must be better [3], etc. Essentially, the intensivists of that era were doing the same things as we are now, but with far greater dosage, extent and intensity. ARDS is one of the best examples of our evolution from “more-to-less,” nurtured by the difficult lessons of our experience (Fig. 1).

Inspired Oxygen Fraction (FiO₂)

In the 1970s, when ARDS became the signature challenge of intensive care, the assumed goal was improved O₂

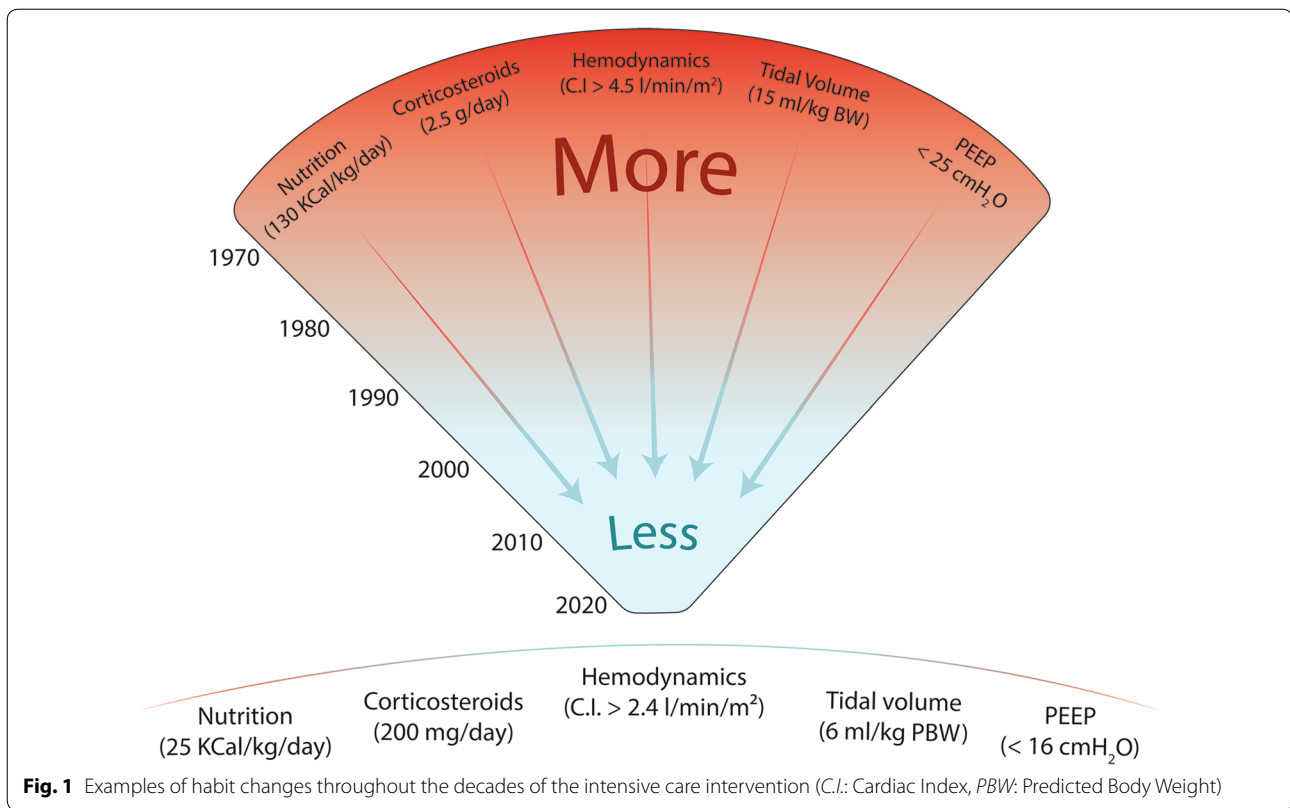
delivery and a key risk was considered pulmonary O₂ toxicity. Sixty percent FiO₂ demarcated the threshold to the danger zone. Accordingly, the first ECMO trial [4] was designed to decrease the risk of high FiO₂ while maintaining O₂ delivery, regardless of tidal volume and pressures. In subsequent years, when the dominating roles of high tidal volumes and pressures in causing lung damage were recognized, FiO₂ receded to the background, largely because of diverted interest. Only three decades later were the potential risks of sustaining high FiO₂ re-evaluated [5].

Tidal Volume (TV)

In the 1970s, tidal volumes of 12–15 ml/kg of observed body weight were recommended by the most prestigious groups treating ARDS [6]. This approach stemmed primarily from the assumed need to maintain normal PaCO₂ and the observation that higher tidal volumes often produced less atelectasis and better arterial oxygenation in ARDS [7], as already known in the anesthesia practice at that time. It was rather quickly realized, however, that higher pressures and volumes were accompanied by a worrisome incidence of barotrauma [8]. In the late 1970s, we proposed to provide lung rest in severe ARDS by extracorporeal removal of CO₂ [9]. Gentler lung ventilation for ARDS was advocated for the clinical setting by Hickling [10], who, following the experience of Perret with severe asthmatics [11], proposed reducing the intensity of conventional mechanical ventilation by allowing PaCO₂ to rise. The superiority of gentler lung ventilation in observational and laboratory studies was confirmed a decade later by results from the ARMA trial of the ARDS network (6 vs 12 ml/kg of PBW). This unmitigated success, however, followed several inconclusive randomized studies with less rigorous separation of cohorts and narrower differences between them in the strength of the tested VT variable [12]. This sequence exemplifies the oft-repeated pattern of theory, anatomic knowledge, experiment, and experience leading the way

*Correspondence: gattinoniluciano@gmail.com

¹ Department of Anaesthesiology, Emergency and Intensive Care Medicine, University of Göttingen, Göttingen, Germany
Full author information is available at the end of the article



toward down-regulated therapeutic dosing—not dichotomous RCTs conducted in a broadly defined population sample.

Though now widely accepted by the intensive care community, even 6 ml/kg has been reported to be potentially dangerous in a subgroup of severe ARDS patients [13], leading to the current concept of “hyper-protective” ventilation [14]. In some settings, exuberant embracing of ECMO is a reflection of the impetus to “protect the lungs”—“less ventilation is more.” In so doing we now risk yielding to the same simplistic logic of the early 1970s, just with the opposite sign: if less is good, lesser must be better. Unfortunately, extremely low ventilation may be associated with several undesired effects [15].

Positive End Expiratory Pressure (PEEP)

Given the positive effect of PEEP on oxygenation, using high levels of PEEP (even “super PEEP” up to 25 cm H₂O) was proposed early on [16]. The importance of the chest wall properties and body position was seldom considered at that time. The price paid to apply high level of PEEP to maintain “acceptable” arterial oxygenation and O₂ delivery was not immediately appreciated (and still is not by some clinicians). Indeed, PEEP ranging from 6 cm H₂O (prevention of volutrauma) to 15 cm H₂O (prevention of “atelectrauma”) led to similar results in three

large randomized trials, suggesting that the competing risks of volutrauma and atelectrauma are offsetting across this range of PEEP in an unselected ARDS population [17]. However, at the higher levels of PEEP set after a recruitment maneuver, the risks of volutrauma and hemodynamic compromise appear to exceed the risk of atelectrauma. Indeed, the higher PEEP treatment group experienced significantly higher mortality than did the control [18].

Respiratory rate

Of itself, using higher respiratory frequency has not traditionally been considered a problem, and it is set to maintain the PaCO₂ within certain limits. However, caution is advised, especially at high levels of strain and power; in the 1970s, after some preliminary experimental reports, great enthusiasm was generated concerning high-frequency jet ventilation [19] soon abandoned, due to lack of improvement. Two decades later, however, HFOV at mean airway pressures sufficient to provide a lung volume close to total lung capacity was suggested as an ideal form of “open lung” protection (tidal volume of few ml). Results from clinical trials, however, proved discouraging [20]. We believe that the role of respiratory rate in generating VILI merits careful reevaluation,

as it is an essential determinant of the mechanical power delivered to the lung [21].

Conclusion

“Less is More” is a theme that characterizes the evolution and painful lessons of intensive care practice. Yet, aggressive interventions are often well justified in the stabilization phase, and it is extremely unlikely that “Lesser is invariably more than Less.”

Author details

¹ Department of Anaesthesiology, Emergency and Intensive Care Medicine, University of Göttingen, Göttingen, Germany. ² University of Minnesota and Regions Hospital, Minneapolis/St. Paul, MN, USA.

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Compliance with ethical standards

Conflicts of interest

The authors declare that they have no competing interests.

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