

Is Permissive Hypercarbia Pneumoprotective?

To the Editor:

In their review of how to improve pulmonary care, Futier *et al.*¹ emphasize the pneumoprotective strategies of limiting tidal volume and positive inspiratory pressure and the use of moderate positive end-expiratory pressure and recruitment maneuvers. In theory, and there is some evidence (discussed below), another pneumoprotective strategy that can be used is permissive hypercapnia. Further studies will be needed to verify its effectiveness, but it may be reasonable to aim for, or at least tolerate, hypercarbia, assuming no contraindications (*e.g.*, increased intracranial pressure, right heart failure, hyperkalemia).

Fundamentally, unlike spontaneous ventilation where inspired gases enter alveoli where increased space has been created, positive pressure ventilation forces air into spaces and may cause regional overdistention, resulting in trauma.² Every breath is potentially, and likely, traumatic, and the accumulation of these “micro”-traumatic events can eventually lead to clinically significant “macro”-trauma, that is, pulmonary dysfunction. In theory, reducing the number and/or severity of these micro-traumatic insults may reduce the clinically evident pulmonary dysfunction. To achieve normocarbia, one must achieve sufficient minute ventilation. Minute ventilation is achieved by an adequate tidal volume and respiratory rate. Aiming for a lower minute ventilation allows for a lower tidal volume (as well as peak and mean inspiratory pressures) and/or a lower respiratory rate. Futier *et al.*¹ nicely review how excessive tidal volume and inspiratory pressure are damaging. In humans, less attention has been paid to the role of respiratory rate. In animals, it has been shown that an increased respiratory rate increases pulmonary dysfunction.³ There are at least two potential mechanisms. First, with a higher respiratory rate, there are simply more breaths delivered and hence more cumulative trauma over time. Second, gas flow velocity must be higher to achieve a given tidal volume in a shorter inspiratory time as would typically be the case with a higher respiratory rate. Further studies will be needed to determine whether this excess shear is damaging. A lower respiratory rate may also result in lower peak inspiratory pressures and less auto-positive end-expiratory pressure.

Hypercarbia is common during modern anesthesia practice and seems to be well tolerated. Many patients who breathe spontaneously throughout the course of an anesthetic are hypercarbic, which commonly occurs when a laryngeal mask airway is used or during monitored anesthetic care. Evidence suggests that hypercarbia may be pneumoprotective.^{4,5}

Increasing clinical evidence supports the use of permissive hypercapnia in respiratory disease states. Although there are

reasons to routinely use permissive hypercarbia as a preventive strategy, conclusive outcome studies that demonstrate its benefits and/or hurtfulness are limited. However, there is stronger rationale to more readily tolerate hypercarbia in the more difficult to ventilate patients. These may be the higher risk patients where pneumoprotection may be most important. It may not be worth the pulmonary risk to increase the respiratory rate and/or tidal volume to normalize a mild-to-moderate high $Paco_2$ level that is not causing any significant problems. A more complete discussion of the non-pulmonary benefits and disadvantages of hypercarbia is beyond the scope of this communication. It is worth noting that low volume ventilation was initially found to be of benefit in diseased patients and then found to be of benefit in patients without preexisting pulmonary disease. It is possible that the role of permissive hypercapnia may evolve in the same manner.

I would be interested in the opinion of Futier *et al.*¹ on the current and/or potential role of permissive hypercarbia in a lung protection strategy and whether we should be limiting respiratory rates.

Competing Interests

The author declares no competing interests.

Jonathan V. Roth, M.D., Albert Einstein Medical Center, Philadelphia, Pennsylvania. rothj@einstein.edu

References

1. Futier E, Marret E, Jaber S: Perioperative positive pressure ventilation: An integrated approach to improve pulmonary care. *ANESTHESIOLOGY* 2014; 121:400–8
2. Mead J, Takishima T, Leith D: Stress distribution in lungs: A model of pulmonary elasticity. *J Appl Physiol* 1970; 28:596–608
3. Hotchkiss JR Jr, Blanch L, Murias G, Adams AB, Olson DA, Wangenstein OD, Leo PH, Marini JJ: Effects of decreased respiratory frequency on ventilator-induced lung injury. *Am J Respir Crit Care Med* 2000; 161(2 pt 1):463–8
4. Curley G, Contreras M, Nichol AD, Higgins BD, Laffey JG: Hypercapnia and acidosis in sepsis. 2010; 112:462–72
5. Hedenstierna G: Respiratory physiology, Miller's Anesthesia, 7th edition. Edited by Miller RD. Philadelphia, Churchill Livingstone, 2010, pp 361–91

(Accepted for publication December 29, 2014.)

In Reply:

We sincerely thank Dr. Roth for his interest and positive comments on our recent article¹ on prophylactic perioperative positive pressure ventilation (“P.O.P. ventilation” bundle) to prevent postoperative pulmonary complications, and we are happy to propose the following responses to his letter.

As for patients experiencing acute respiratory distress syndrome (ARDS), evidence is accumulating that lung-protective ventilation, including but not limited to the use of lower tidal