

Ventilation in Trauma Patients: The First 24 h is Different!

Timothy Craig Hardcastle^{1,2} · David J. J. Muckart^{1,2} · Ronald V. Maier³

Published online: 13 May 2016
© Société Internationale de Chirurgie 2016

Abstract

Introduction Ventilation of major trauma patients is often needed in both the acute (emergency department and early ICU phase) and subsequent phases of trauma care for those who need ICU admission. What is unclear is whether ICU ventilation strategies should be directly extrapolated to the acute phase of treatment.

Methods This paper reviews the ARDS.net study, highlights recent developments in ventilation strategies, and provides practical ventilation guidance to the trauma surgeon for acute phase (in the ED or ICU) and the subsequent phase of ICU care.

Results The acute phase of care in the ED and the ICU is different from the subsequent phases of ICU care as the lung is more recruitable and there are other aspects of resuscitation from metabolic acidosis and traumatic brain injury, which require a different ventilation strategy to the traditional ARDS.net approach.

Discussion and conclusion The acute phase is different from the subsequent phase of care and there appears to be some inappropriate extrapolation of ICU practice to the acute phase. Application of the proposed ventilation strategies should ensure an optimal outcome. It is important to treat patients as individuals during assessment and treatment.

All three authors have presented lectures on this topic at various trauma meetings.

✉ Timothy Craig Hardcastle
Hardcastle@ukzn.ac.za; timothyhar@ialch.co.za

¹ Trauma Service, Inkosi Albert Luthuli Central Hospital, 800 Vusi Mzimela Rd, Mayville, Durban 4091, South Africa

² Department of Surgery, University of KwaZulu-Natal, Durban, South Africa

³ Department of Surgery, Harborview Medical Center, University of Washington, Surgery Clinic, 410 9th Ave, 7th Floor, Seattle, WA 98104, USA

Introduction

Trauma remains a leading cause of non-natural mortality and disability across the world. Major trauma affects between 5 and 15 % [1, 2] of the total trauma burden to as high as 25 % in certain facilities (Seattle, WA). Depending on location, the need for aggressive resuscitation and intensive care admission accounts for a similar percentage of the total injury spectrum. Since pulmonary injury or associated dysfunction is frequently present, it is important that every trauma surgeon and intensive care specialist has a safe and reasoned approach to the management of ventilation of the trauma patient.

Much had been written about the improved concepts of “lung-protective ventilation.” However, some of the concepts are applied incorrectly when these ventilation modes, useful in the post-acute ICU phase, are extrapolated to the

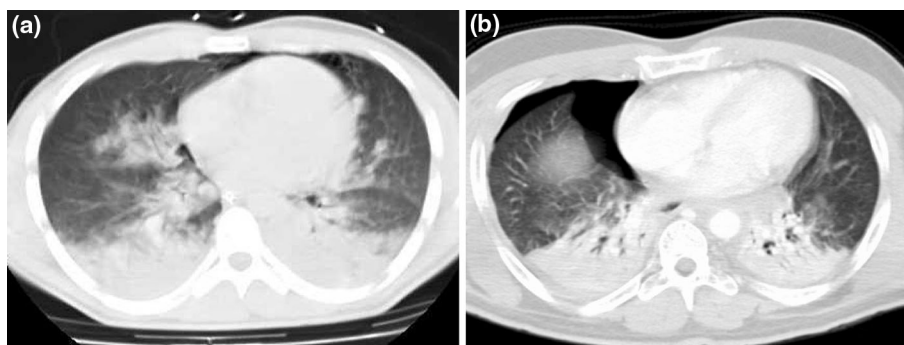


Fig. 1 **a** A patient three weeks after admission for an abdominal stab wound with contamination and repeated episodes of sepsis who now has “ARDS.” **b** A patient just admitted after a motor vehicle collision

as an unrestrained passenger who has a pneumothorax, bilateral lung contusions, a femur fracture, and an associated traumatic brain injury (chest tube not visible on this CT-slice)

prehospital, emergency department, and ICU environment, during the acute resuscitative phase (12–24 h), when the patient needs are far different. The ARDS.net study [3] radically improved the way patients with severe lung injury, also known as Acute Respiratory Distress Syndrome (ARDS), are ventilated, but extrapolation to ventilation prior to the “resuscitated” ICU environment has occurred without rigorous trials to test if these concepts are indeed the best option for the emergency or elective non-ARDS patient requiring ventilation during the acute resuscitation of the unstable patient.

Figure 1a, b demonstrates chest-CT slices of two patients with the clinical need for invasive ventilation, but at two very different stages of care. Both patients are acidotic and hypoxic, and currently mildly hypercarbic. Figure 1a depicts a patient three weeks after admission for an abdominal stab wound with contamination and repeated episodes of sepsis who now has “ARDS,” while Fig. 1b demonstrates a patient just admitted after a motor vehicle collision as an unrestrained passenger who has a pneumothorax, bilateral lung contusions, a femur fracture, and an associated traumatic brain injury (TBI) (the chest tube as treatment for the pneumothorax is not visible on this slice). The question is how best to ventilate these two patients—do the same ARDS.net philosophies apply equally to these two patients or any acute resuscitative patient versus the “stable” resuscitated ICU patient?

Physiologically, the chest examined early after trauma is often more compliant and recruitable, since the patient was previously healthy, which does mean that one must carefully monitor the ventilation settings and titrate the effect. One may err in either direction: too little volume may fail to recruit available, but atelectatic lung tissue, while excessive volume may lead to over-distension and hemodynamic compromise, although this is rare with modern ventilators and good clinical management. Later in the course, the lungs will be stiffer (restrictive phase), making later recruitment more difficult, yet with the changes in

pulmonary pressure having a lesser impact on pressure-induced hemodynamic changes. Additionally, early in the course lung “happiness” may be at the expense of cardiac or brain “happiness,” requiring the clinician to balance more carefully potentially conflicting goals. This paper aims to address these controversies from a practical perspective.

Methods

This paper will review the original ARDS.net trial and the principles that resulted therefrom. It will also review some of the recent literature examining whether the research-gaps left by the ARDS.net trial have been adequately addressed. Furthermore, the authors attempt to place the various aspects of ventilation into perspective relevant to the major trauma patient, with, or without TBI and at various time-points in the management pathway. The paper will attempt to dispel some myths and illogical extrapolation of ventilation concepts to the acute resuscitation phase of care.

Results

ARDS.net: a critical review

The ARDS.net study [3] enrolled patients in the ICU with a clinical diagnosis of ARDS to either 4–6 or 12–15 ml/kg tidal volume ventilation and also compared plateau pressures less than 50 cm H₂O to reduced plateau pressure of less than 30 cm H₂O. The trial was terminated prior to completion of enrollment due to higher mortality and longer length of ventilation in the control arm. Although the study demonstrated lower complications and mortality with the 4–6 ml/kg group with restricted plateau pressures, there was one quartile of static compliance that showed no

difference in outcome, namely the group with near normal compliance. Also, while there was a higher trauma subgroup in the trial cohort (13 vs. 9 %), overall there was a very low trauma-patient group. Randomization in the ICU was on condition that the physician treating the patient agreed and the trauma subgroup excluded patients with a TBI. Any patient with a predicted mortality over 50 % was also excluded. These latter points are critical to the discussion in the context of the severely injured trauma patient. Additionally, a volume-control ventilation strategy was used, currently less favored in modern ICU practice where pressure-control and patient-compliant modes are preferred [4]. Lastly, functional reserve capacity (FRC) recruitment was primarily achieved using PEEP, with a ~1:5 ratio of PEEP:F_iO₂.

Ventilation during resuscitation

During the resuscitation phase of trauma care, the patient is often in hypovolemic shock resulting in anaerobic metabolism, a metabolic acidosis and hyperlactemia. If there is chest trauma, especially blunt chest trauma, aspiration, or secondary inflammatory insults, the resultant loss of alveolar volume may still be recruitable early in the disease process by using tidal volume in addition to PEEP. During the acute phase with required volume resuscitation, there are rapid ongoing changes in the patterns and extent of alveolar loss due to direct injury and ischemia/reperfusion insults. This is very different compared to the ARDS scenario in the ICU, where the degree of pulmonary recruitability is much less and PEEP functions to maintain the limited recruited alveoli that are open.

Most trauma patients that require ventilation are initiated on full mechanical ventilation (MV) with intubation as opposed to non-invasive ventilation (NIV), especially if there is a TBI. Continuous Positive Airway Pressure (CPAP) or non-invasive pressure-support ventilation, by face mask or helmet, should be reserved for patients with mild pathology where support is predicted to be of short duration. If this modality is utilized and despite this the PCO₂ increases or the PaO₂ or saturation decreases, MV should ensue [5]. It is far safer to initiate early intubation and ventilation than to try to recover from a step-wise failure of face mask, to non-rebreather mask, and to non-invasive support leading to potential respiratory arrest. This latter group of patients have much longer ICU stay and more complications.

In this acute phase of care, one is aiming to correct acidosis, maintain normocapnea (at least until TBI is excluded), and recruit available lung volume. To do this, either a pressure-control or volume-control mode may be used, such as synchronized intermittent mandatory ventilation (SIMV), with the focus having moved away from the

peak and plateau pressures, to driving pressure, using the peak pressure minus PEEP as the driving pressure, with a goal of no greater than 25–30 cm H₂O [6] and an ideal of around 18 cm H₂O [7].

In general, the peak or plateau pressure, depending on mode, should not exceed 30 cm H₂O; however, in patients with significant chest wall trauma, with obese abdomens, or secondary to generalized tissue edema from diffuse capillary leak from ischemia/reperfusion secondary to the shock state, this pressure could be exceeded as most of the compliance is determined by factors other than the pulmonary parenchyma. The same applies due to increased abdominal pressures or abdominal compartment syndrome [8]. With extreme acute pulmonary failure, significantly higher peak pressures and PEEP may be required to maintain minimal oxygenation to support organ function and survival.

Pressure support should be added to ensure adequate spontaneous tidal volumes, if possible, as the modern approach to trauma ventilation is to avoid long-term chemical paralytics [9]. The aim is to keep the tidal volumes around 8–10 ml/kg, rather than 6–8 ml/kg, so as to avoid hypercapnea and also recruit acute volume loss. Furthermore, the aim is to correct the acidosis, which enhances coagulation and sensitivity to medications that may be administered. The oxygen requirement is adjusted to achieve at least a saturation greater than 92 % or P_a-O₂ > 60 mmHg/8 kPa.

The endotracheal cuff pressure should be maintained at around 1 cm H₂O above the peak ventilator pressure to prevent an air-leak, and the tube position should be checked at 2–3 cm above the carina with a Chest X-ray [10].

PEEP has been controversial, with some authors advocating against it during the resuscitation phase on the erroneous belief that it reduces cardiac output. PEEP is essential to prevent further atelectasis of the recruited segments of lung and to reduce the risk of atelectrauma. The PEEP may be adjusted upward and an acceptable approach is that based on the ARDS.net philosophy of using the PEEP in a ratio of 1:5 with the F_iO₂ [3]. An example of this would be that for a patient requiring 80 % oxygen, the PEEP would be rapidly advanced to 16 cm H₂O, (known in Seattle as “Maier’s Rule”). Combination of low tidal volume, low or no PEEP and high inspired O₂ concentration can only be expected to worsen atelectasis [11].

This approach is not associated with worsening lung injury in the acute phase and avoids the risk of causing further harm if there is a TBI present. Once the acute acidosis is reversed and the resuscitation is completed, usually within the first 12–24 h, then the ventilator settings can be adjusted to more “lung-protective” values provided there is no TBI to be adversely affected by the concomitant physiologic changes.

Dilemma of neuroprotection versus lung protection

Patients with TBI are a challenging issue, as the risk of worsening the brain perfusion from hypercapnea is significant. Lung-protective ventilation, as espoused in the ARDS.net philosophy, allows for permissive hypercapnea and permissive acidosis [12]. Both acidosis and hypercapnea are detrimental for TBI. The goal is to therefore optimally ventilate and balance the risk–benefit of saving neurological tissue at risk while doing the least harm to the lungs. Recent research suggests that higher tidal volumes (8–10 ml/kg) are at least no more harmful and at most may increase the pneumonia risk only, without affecting mortality, when compared to 4–6 ml/kg, yet with the advantage of avoiding hypercarbia [13, 14]. Hyperventilation of the TBI patient is not advised unless used as a bridge to acute surgical intervention and decompression [15]. Equally controversial has been the use of PEEP. It does appear, however, that when PEEP is set at levels lower than ICP, it does not have a significant detrimental effect on ICP [16].

Complicating all of these factors is the effect recently described where pathology in one compartment (e.g., the head) can affect the pressures and perfusion in the other compartments (e.g., chest or abdomen) and vice versa, a so-called multi-compartment syndrome theory. This must be borne in mind when adjusting ventilation and optimizing end-tidal carbon dioxide control [17].

Recruitment strategies in the early phase of ventilation

Transition to lung-protective ventilation may involve lung recruitment attempts. Traditional recruitment strategies included the 40–40 concept [18], which implied a 40-s breath-hold at 40 cm H₂O using an inspiratory hold on a ventilator or using a bag-valve-mask device. If non-paralyzed, patients find this to be very uncomfortable and therefore alternative methods have been derived. These include slow gentle recruitment with a combination of higher tidal volumes and adequate pressure support, maintaining the recruitment with higher PEEP [19]. Also one can use incremental pressure support/pressure control above PEEP to avoid derecruitment and enable recruitment in the early transition phase of ventilator support [20].

Prevention of lung injury

The concern with large tidal volumes was the induction of various types of lung injury, namely volutrauma (over-expansion), barotrauma (high-pressure alveolar rupture), atelectrauma (inadequate PEEP leading to shear stress from repeated cycles of alveolar collapse), and also bio-trauma (intra-pulmonary inflammation of the non-injured lung

[21]. To prevent this, the ARDS.net study [3] showed that 4–6 ml/kg tidal volumes had a largely protective effect when compared to 12–15 ml/kg. What the ARDS.net study did not examine was whether there was any real difference in the in-between volumes. To date, studies prior to ARDS.net using slightly higher tidal volumes and the subsequent studies have shown mild benefit, or equivalence, for any tidal volume less than 10 ml/kg, while there is increasing harm above 10 ml/kg, including secondary injury to other organ systems [12, 13, 22–24]. Additionally, intra-operative trials of lung-protective ventilation during abdominal surgery have shown mixed results, with limited overall benefit [25, 26].

ARDS in the trauma patient

The Berlin consensus is the most recent document defining the concept of ARDS, listing the causative potentials and the inclusion/exclusion criteria [27]. This new definition sets a timeframe for the development of ARDS (within 7 days of an insult), and suggests imaging criteria (bilateral opacities not explained by effusions, collapse, or nodules) and respiratory failure not explained by cardiac failure or fluid overload. They also removed the Acute Lung Injury (ALI) category of the older classifications, replacing this with a mild/moderate/severe ARDS grouping of <300/<200 and <100 P:F ratios, respectively.

The challenge with the use of the Berlin definition in patients with chest trauma (or trauma in general) appears to be that the trauma would count as the inciting event; however, chest trauma with haemothorax and collapse (lung contusion) would partially fit the exclusion group. Additionally, there are numerous causes of ARDS, with the trauma patient at risk for a number of these including lung contusion, aspiration of gastric content, fat embolism syndrome, massive transfusion, sepsis, and pneumonia. Patients with over 20 % lung contusion have a high incidence of prolonged ventilation and development of ARDS [28]. The incidence is considered to be about 6.5 % in all trauma patients needing in excess of 48-h mechanical ventilation [29]. Patients with ARDS after injury tend to be younger and have fewer comorbid medical conditions [30].

Ventilation for the patient in ICU after trauma

In the patient with TBI, the current best-practice is to offer “neuroprotection” for at least 48 h post trauma, using normocapnea as the goal, along with the other aspects advocated by the Brain Trauma Foundation [15]. For the patient without TBI, once the acute resuscitation phase is complete and the patient has “stabilized,” the application of the ARDS.net lung-protection strategies are completely acceptable. For those who develop the full-blown

syndrome of ARDS, all the additional interventions to recruit and maintain alveolar surface area are important, for example patient head-up 30–45°, prone-positioning if needed, physiotherapy, bronchoscopy, etc. [28]. Once the neurological, chest or other trauma-related indication for ventilation has resolved the patient can be weaned onto a spontaneous mode (such as pressure-support ventilation), prior to extubation. The use of t-piece trials are not advocated as routine practice due to the loss of PEEP during the procedure [31].

Additional optional modes and alternative oxygenation strategies

If standard methods of recruitment, ventilation, and the routine adjuncts in the ICU do not result in adequate oxygenation, there are a number of alternative or newer ventilation or oxygenation strategies available to improve pulmonary function.

Biphasic positive airway pressure (BIPAP) [32] ventilation uses two preset pressure levels with a time-cycled change in the applied pressure level. It is a useful option when weaning as an alternative to pressure-support-type modes, but carries the additional advantage of potentially being combined with other modes, such as airway pressure release ventilation. Bi-level positive airway pressure ventilation (BiPAP) can also be applied in a non-invasive method. Airway pressures are elevated and yet auto-PEEP is less common.

Pressure-regulated volume control (PRVC) uses a decelerating flow pattern with pressure control while ensuring a guaranteed tidal volume. It has shown better outcomes than traditional volume-control ventilation. There is a sensing feedback loop that adjusts the ventilator next breath based on the pressures generated [33].

Airway Pressure Release Ventilation (APRV) [34], is similar to BIPAP, but uses a pressure-control mode with inverse ratio ventilation allowing unrestricted spontaneous breathing. It is mainly used as a rescue-mode for severe ARDS. Some purported advantages include alveolar recruitment (using this so-called open-lung approach), improved oxygenation, and preservation of spontaneous breathing, improved hemodynamics, and potential lung-protective effects. Numerous claimed disadvantages relate to the risks of volutrauma, increased work of breathing, and increased energy expenditure related to spontaneous breathing. Auto-PEEP is very common and sedation is usually required for patient compliance and to avoid ventilator-asynchrony. There remains no proven mortality or ventilation-duration benefit over other modes of treatment.

High-frequency oscillatory ventilation (HFOV) uses miniscule tidal volumes at high frequency to deliver oxygen to the lungs, at a constant relatively high mean airway pressure and is known to be mildly beneficial in neonates

[35]. However, to date trials in adults and trauma patients, in particular, have failed to show a survival advantage or reduced ICU length of stay [36, 37].

Extracorporeal membrane oxygenation (ECMO) is mentioned for completeness as a rescue oxygenation therapy for severe ARDS that has found a small niche in the trauma patient population [38]. ECMO will not be discussed in detail as it is the subject of a separate paper in this IATSIC Symposium [39].

Summary: a practical approach for the surgeon

In summary, the trauma patient should be treated based on the phase of treatment and the underlying injuries. In the early phase of care, recruitment, normocapnoea, and reversal of the associated metabolic acidosis remain the goal. As such, higher tidal volume ventilation with either a synchronized mode that provides adequate support in spontaneous breathing or mandatory volume ventilation in patients with respiratory insufficiency due to any cause along with adequate PEEP is essential. The lung-protective strategies as espoused for ARDS patients in the ICU should not simply be translated to the acute phase of resuscitation. On the other hand, once the patient is out of the acute resuscitation phase, ventilation should be a balance of risk—benefit with lung-protective strategies and rapid weaning to spontaneous modes, provided the TBI patient has received adequate neuroprotective strategies. For the patient who develops ARDS, lung-protection and rescue therapies may offer the ability to achieve adequate tissue oxygenation with minimal iatrogenic pulmonary injury.

Conclusions

A one-size-fits-all approach is not appropriate for the ventilatory management of the trauma patient and the patient's care must be individualized to the phase of treatment.

Compliance with ethical standards

Conflict of interest The authors have no conflict of interest.

References

1. Lutge E, Moodley N, Tefera A et al (2016) A hospital based surveillance system to assess the burden of trauma in Kwa-Zulu Natal Province South Africa. *Injury* 47:135–140
2. Trunkey D (2008) The medical world is flat too. *World J Surg* 32(8):1583–1604
3. The acute respiratory distress syndrome network (2000) Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 342:1301–1308

4. Garneroa AJ, Abbonab H, Gordo-Vidal F (2013) Pressure versus volume controlled modes in invasive mechanical ventilation. *Med Intensiva* 37(4):292–298
5. Papadakos P, Karcz MK (2015) Noninvasive ventilation in trauma. *World J Crit Care Med* 4(1):47–54
6. Harris T, Davenport R, Hurst T et al (2012) Improving outcome in severe trauma: trauma systems and initial management—intubation, ventilation and resuscitation. *Postgrad Med J* 88:588–594
7. Amato MBP, Meade Slutsky AS et al (2015) Driving pressure and survival in the acute respiratory distress syndrome. *N Engl J Med* 372:747–755
8. Gestring M (2015) Abdominal compartment syndrome. <http://www.uptodate.com/contents/abdominal-compartment-syndrome>. Accessed 30 Dec 2015
9. Tripathi SS, Hunter JM (2006) Neuromuscular blocking drugs in the critically ill. *Br J Anaesth CEACCP* 6:119–123
10. Hardcastle TC, Faurie M, Muckart DJJ (2015) Endotracheal tube cuff pressures and tube position in critically injured patients on arrival at a referral centre: avoidable harm? *Afr J Emerg Med*. (IN PRESS)
11. Hedenstierna G, Edmark L (2015) Effects of anesthesia on the respiratory system. *Best Pract Res Clin Anaesth* 29:273–284
12. Laffey JG, O’Croinin D, McLoughlin P et al (2004) Permissive hypercapnia – role in lung protective strategies. *Intensive Care Med* 30:347–356
13. Coppola S, Froio S, Chiumello D (2014) Protective lung ventilation during general anaesthesia: is there any evidence? In: Vincent JL (ed) Annual update in intensive care and emergency medicine, 2014. Springer, Switzerland
14. Sutherasan Y, Vargas M, Pelosi P (2014) Protective mechanical ventilation in the non-injured lung: review and meta-analysis. In: Vincent JL (ed) Annual update in intensive care and emergency medicine, 2014. Springer, Switzerland
15. Brain Trauma Foundation (2007) Guidelines for prehospital management of traumatic brain injury, 2nd edn. <https://www.braintrauma.org/pdf/prehospitalGuideline2ndEdition.pdf>. Accessed December 2012
16. Seder DB, Riker RR, Jagoda A et al (2012) Emergency neurological life support: airway, ventilation, and sedation. *Neurocrit Care* 17:S4–S20
17. Scalea TM, Bochicchio GV, Habashi N et al (2007) Increased intra-abdominal, intrathoracic, and intracranial pressure after severe brain injury: multiple compartment syndrome. *J Trauma* 62(3):647–656
18. Valente Barbas CS (2003) Lung recruitment maneuvers in acute respiratory distress syndrome and facilitating resolution. *Crit Care Med* 31(4 Suppl):S265–S271
19. Borges JB, Okamoto VN, Matos GF et al (2006) Reversibility of lung collapse and hypoxemia in early acute respiratory distress syndrome. *Am J Respir Crit Care Med* 174(3):268–278
20. Póvoa P, Almeida E, Fernandes A et al (2006) Evaluation of a recruitment maneuver with positive inspiratory pressure and high PEEP in patients with severe ARDS. *Acta Anaesthesiol Scand* 48(3):287–293
21. Silva PL, Negrini D, Rocco PRM (2015) Mechanisms of ventilator-induced lung injury in healthy lungs. *Best Pract Res Clin Anaesth* 29:301–313
22. Brochard L, Roudot-Thoraval F, Roupie E et al (1998) Tidal volume reduction for prevention of ventilator-induced lung injury in acute respiratory distress syndrome. *Am J Respir Crit Care Med* 158:1831–1838
23. Stewart TE, Meade MO, Cook DJ et al (1998) Evaluation of a ventilation strategy to prevent barotrauma in patients at high risk for acute respiratory distress syndrome. *N Engl J Med* 338:355–361
24. Brower RG, Shanholtz CB, Fessler HE et al (1999) Prospective, randomized, controlled clinical trial comparing traditional versus reduced tidal volume ventilation in acute respiratory distress syndrome patients. *Crit Care Med* 27:1492–1498
25. Treschan TA, Kaisers W, Schaefer MS et al (2012) Ventilation with low tidal volumes during upper abdominal surgery does not improve postoperative lung function. *Br J Anaesth* 109:263–271
26. Futier E, Constantin JM, Paugam-Burtz C et al (2013) A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. *N Engl J Med* 369:428–437
27. The ARSD Definition Task Force (2012) Acute respiratory distress syndrome: the Berlin definition. *J Am Med Assoc* 307(23):2526–2533
28. Bakowitz M, Bruns B, McCunn M (2012) Acute lung injury and the acute respiratory distress syndrome in the injured patient. *Scand J Trauma Resusc Emerg Med* 20:54
29. Recinos G, DuBose JJ, Teixeira PG et al (2009) ACS trauma centre designation and outcomes of post-traumatic ARDS: NTDB analysis and implications for trauma quality improvement. *Injury* 40:856–859
30. Calfee CS, Eisner MD, Ware LB et al (2007) Acute Respiratory Distress Syndrome Network, National Heart, Lung, and Blood Institute: trauma-associated lung injury differs clinically and biologically from acute lung injury due to other clinical disorders. *Crit Care Med* 35:2243–2250
31. Ladeira MT, Vital FM, Andriolo RB et al (2014) Pressure support versus T-tube for weaning from mechanical ventilation in adults. *Cochrane Database Syst Rev* 5:CD006056
32. Hörmann C, Baum M, Putensen C et al (1994) Biphasic positive airway pressure (BIPAP)—a new mode of ventilatory support. *Eur J Anaesthesiol* 11:37–42
33. Guldager H, Nielsen SN, Peder C et al (1997) A comparison of volume control and pressure-regulated volume control ventilation in acute respiratory failure. *Crit Care* 1:75–77
34. Daoud EG, Farag HL, Chatburn RL (2012) Airway pressure release ventilation: what do we know? *Respir Care* 57:282–292
35. Cools F, Offringa M, Askie LM (2015) Elective high frequency oscillatory ventilation versus conventional ventilation for acute pulmonary dysfunction in preterm infants. *Cochrane Database Syst Rev* 3:CD000104
36. Franco PM, Enders F, Wilson G et al (2015) A comparative effectiveness study of rescue strategies in 1,000 subjects with severe hypoxemic respiratory failure. *Respir Care*. pii: resp-care.04162. [Epub ahead of print]
37. Gu XL, Wu GN, Yao YW et al (2014) Is high-frequency oscillatory ventilation more effective and safer than conventional protective ventilation in adult acute respiratory distress syndrome patients? A meta-analysis of randomized controlled trials. *Crit Care* 18(3):R111
38. Jacobs JV, Hooft NM, Robinson BR et al (2015) The use of extracorporeal membrane oxygenation in blunt thoracic trauma: a study of the Extracorporeal Life Support Organization database. *J Trauma Acute Care Surg* 79:1049–1054
39. Zonies D et al (under review) ECMO in Trauma. *World J Surg*