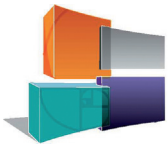
Anaesth Crit Care Pain Med 38 (2019) 265–276



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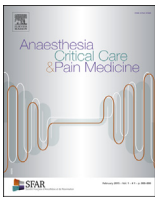

Review article

# Acute respiratory distress syndrome after chest trauma: Epidemiology, specific physiopathology and ventilation strategies

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**Contents**



## 1. Background

Traumatic chest injuries are responsible for significant morbidity and the cause of trauma-related death in 20%–25% of cases [1]. Traffic accidents and falls are thus the most causes of these injuries [1]. Thoracic trauma can include multiple injuries, mainly osseous (ribs, sternal fractures, flail chest), pulmonary contusions or lacerations, pneumothoraxes and pleural effusions, and sometimes involve wounds to the heart and vessels (aortic dissection, cardiac contusion) or diaphragm [2]. Chest trauma is also often associated with severe injuries to other regions, particularly to the head, abdomen or extremities [3]. Following trauma, patients with thoracic injuries are at risk of developing acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). This worsening of respiratory function can lead to requirement for mechanical ventilation [4]. ARDS may thus occur because of direct injury to the lung or due to secondary mechanisms induced by the trauma setting. In addition, changes to gas exchange may also be generated or aggravated by mechanical ventilation as a result of volotrauma barotrauma, biotrauma, or ventilation-associated pneumonia [5]. Many mechanical ventilation strategies have been tried in trauma patients in the last 30 years to determine the optimal method of maximizing gas exchange with minimal lung damage [6]. The aim of the present work has been to establish the current state of ARDS in relation to chest trauma and to review the different mechanical ventilation strategies used in this setting. Non-invasive ventilatory support and ventilatory weaning are two complex study fields with an available literature very limited. These topics could be the subject of another systematic review and will not discussed in the present work.

## 2. Study design and literature search

By selecting several major key topics, our aim was to investigate the indications for several alternative management of conventional mechanical ventilation in severe or multiple traumas, as well as their foremost effects.

We only included data from studies that enrolled adults (aged > 18 years) affected by ARDS induced by blunt or

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https://doi.org/10.1016/j.accpm.2018.09.009

*Abbreviations*

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|---|---|
| APRV | airway pressure release ventilation |
| ARDS | acute respiratory distress syndrome |
| ECMO | extracorporeal membrane oxygenation |
| HFO | high-frequency oscillation ventilation |
| HFJV | high-frequency jet ventilation |
| HFPV | high-frequency percussive ventilation |
| ILV | independent lung ventilation |
| MOF | multi-organ failure |
| PEEP | positive end-expiratory pressure |
| TRALI | transfusion-related acute lung injury |

penetrating trauma, into an emergency department, a trauma service or an intensive care unit (ICU). Randomised and non-randomised controlled trials, as well as observational studies including cohort, case-control and case series, were analysed. The list of studies was updated by a number of clinical databases, including PUBMED, MEDLINE and EMBASE, from January 1990 until today. The selected keywords were mechanical ventilation and trauma, which were cross-referenced with flail chest, pulmonary contusion, chest injury, blunt chest trauma, acute lung injury (ALI), acute respiratory distress syndrome (ARDS), respiratory failure, airway pressure ventilation (APRV), high frequency oscillation (HFO), high frequency percussive ventilation (HFPV), independent lung ventilation (ILV), high frequency jet ventilation (HFJV), extracorporeal membrane oxygenation (ECMO), extracorporeal life support (ECLS) and multi organ failure (MOF). Because this was a retrospective review, ethical approval was deemed not necessary for data collection.

## 3. Epidemiology of ARDS in chest trauma

Blunt chest trauma is involved in nearly one-third of acute trauma admissions to the hospital. Traditionally, respiratory failure and ARDS are frequent following chest trauma, but are poorly documented in the literature. Chest trauma is often caused by high-velocity blunt trauma, which explains the adverse consequences. Patients with thoracic injuries are at high risk for ARDS [7], occurring in 10%–25% of cases depending from trauma severity [8,9]. ARDS in trauma patients account for about 5% of all ARDS [10]. The delay in the onset of ARDS is variable according to its cause; ARDS may indeed occur several days after a trauma. Mortality attributable to trauma-related ARDS varies from 20% to 80% with the severity of injuries [11]. Moreover, ARDS in trauma patients has been associated with longer hospital stay, increased costs, and worse long-term health-related quality of life [12]. In cases of chest trauma, initiation of mechanical ventilation during the initial management is also very variable (ranging from 23% to 75%) because it depends on the severity of the trauma, the degree of underlying lung disease, associated injuries, and early surgical requirements [13]. Similar to the medical setting, mechanical ventilation in cases of trauma is well known to be associated with a high risk of nosocomial pneumonia (between 30% and 50%), leading also to respiratory failure and ARDS [14].

Many risk factors for early and delayed respiratory failure have been described for chest trauma, including thoracic injuries, but also several extra-thoracic injuries. Deformation of the thoracic osseous cage caused by rib and sternum fractures or flail chest has been strongly associated with ARDS, as well as the presence and the amount of pulmonary contusions, pneumothoraces and hemothoraces [15]. A pulmonary contusion surface of 20% of the overall pulmonary parenchyma was documented as a robust predictive threshold for ARDS (positive predictive value 80%) [16]. Lung ultrasonography may help diagnose pulmonary contusions as soon as admission and follow their evolution, in order to identify trauma patients at risk of developing ARDS [17]. In addition, many extra-thoracic injuries (compartment abdominal syndrome, major long bone fractures and unstable pelvic fractures, and head trauma) were also identified as independent risk factors of ARDS [18]. Among other risk factors of ARDS, age older than 55 years, male gender, active smoker, important comorbidities such as diabetes have been clearly determined, as well as initial massive fluid therapy, volume and type of blood products administered, number of surgical procedures [19].

## 4. Physiopathology of ARDS in chest trauma

Multiple causes have been associated with the development of ARDS in cases of chest trauma. Hypoxemic thoracic injuries lead to local and systemic aggravating mechanisms that induce ARDS in the hours after trauma, with an interval of several hours or days. Fig. 1 summarises the different phenomena that lead to ARDS.

### 4.1. Primitive thoracic damage

Many thoracic injuries can lead to ALI and alteration of gas exchange. All these injuries can induce a decrease in functional residual capacity and alveolar recruitment, leading to ventilation-perfusion mismatch and intrapulmonary shunt [20].

Pulmonary contusion is a nosologic entity often found in patients with chest trauma (70%–80%). Alveolar derecruitment may result from direct alveolo-capillary membrane changes. In addition with alveolar blood filling, an increasing alveolo-capillary membrane permeability indeed occurs, leading to alveolar oedema, surfactant destruction and hypoxemia [21].

Alveolar collapse may also be a consequence of impairment in thoracic expansion and change in the mechanics of spontaneous breathing. Shallow tidal breathing and a lack of deep inspirations promote atelectasis, V/Q mismatch, and hypoxemia, leading to pneumonia and respiratory failure. Sternal and ribs fractures, flail chest and thoracic spine fractures are the main injuries involved in this pathogenic phenomenon. Impairment of ventilatory mechanics is thus strongly affected by pain [22]. Rib fractures are reputed to be very painful and to limit deep breathing. Optimal analgesia in these patients was found to be associated with reduced need for mechanical ventilation and lower incidence of ARDS [22]. In this setting, regional anaesthesia, such as thoracic epidural analgesia, paravertebral nerve block may clearly improve survival of trauma patients [23]. In addition, osseous injuries, diaphragmatic wounds or ruptures may also lead to distortion of diaphragmatic movement and ventilatory problems [24]. Reduction of shallow tidal breathing and inspiratory amplitude therefore promotes atelectasis, ventilation-perfusion ratio mismatch and ARDS.

Alveolar derecruitment may be generated by extrinsic lung compression; pleural effusions (pneumothoraces and hemothoraces) and intraperitoneal organs in cases of diaphragmatic hernia are the main causes [25]. Furthermore, tracheal or bronchial injuries may also increase the compressive mechanisms in cases of massive air leaks [1].

### 4.2. Biomarkers of pathogenesis

ARDS is a progressive inflammatory disease of the lungs. The acute phase is characterised by an important and uncontrolled

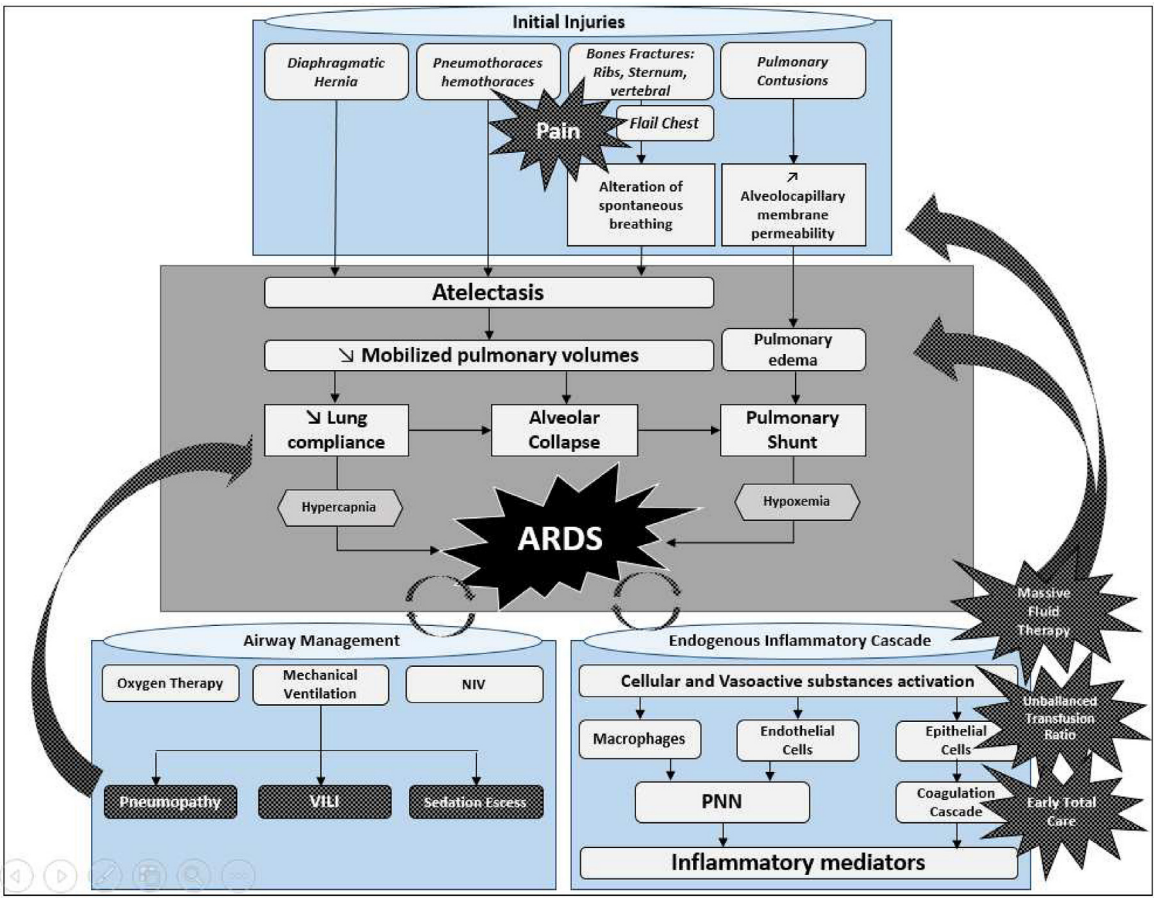


**Fig. 1.** Physiopathogenesis of acute respiratory distress syndrome (ARDS) related to trauma. This figure explains the different physiopathological mechanisms leading to ARDS in cases of chest trauma, including initial and delayed ARDS. Secondary aggression mechanisms are also represented.

inflammatory response in the lung tissues. It results a significant accumulation of neutrophils and macrophages [26]. The intense and prolonged activation of the immune system in response to initial injury leads to immunological reaction. The importance of this inflammatory response depends obviously on the degree of the trauma, but also on the genetic profile of patients. A previous work by Tompkins et al. [27] showed in severe trauma population that the early leukocyte genomic response was associated with simultaneously increased expression of genes involved in the systemic inflammatory, innate immune, and compensatory anti-inflammatory responses, as well as in the suppression of genes involved in adaptive immunity. All these modifications induce a massive release of damage-associated molecular pattern molecules from injured tissues. These specific biomarkers are different according to the kind of damaged cells [28]. Endothelial injuries generate a blood accumulate in the pulmonary alveoli associated with a raise of vascular permeability and inflammatory cells. Among the main endothelial biomarkers, Ang-2, ICAM-1, selectins and VEGF can be cited [29]. Epithelial injuries involve biomarkers leading to dysfunction of type I, type II alveolar cells and clara cells. Massive release of epithelial biomarkers such as sRAGE are responsible for dysfunction of capillary-alveolar barrier [30]. Biomarkers such as surfactant protein B also play critical roles in ARDS developing by the reducing of alveoli surface tension and repair phenomena of injured lung tissues [31]. Finally, the increasing of several clara cells, such as clara cell protein 16, decreases protection of lung tissues against inflammation, oxidative stress and fibrosis in ARDS [32].

### 4.3. Delayed phenomena during trauma management

Several harmful factors can subsequently lead to the occurrence or aggravation of ARDS in the days after the initial chest trauma. Pulmonary oedema results from extravascular diffusion because of increased capillary permeability, and decrease in oncotic pressure [33]. Limitation of capillary pressure by fluid restriction has been shown in favour of a decreasing of lung oedema [34]. Similarly, the volume and the type of blood products administrated (i.e. fresh frozen plasma) have long been blamed for lung damage, so-called transfusion-related acute lung injury (TRALI). However, a recent prospective trial has proved that aggressive administration of fresh frozen plasma and platelets in patients with massive bleeding patients was not associated with a higher rate of respiratory failure [35]. Large volumes of fluid therapy administrated concomitantly with massive transfusion are probably the origin in previous reports of ARDS development. Restrictive fluid strategy applied in coagulopathic patients was indeed associated recently with a lower incidence of TRALI. Another significant risk factor of ARDS is the early total care strategy that may potentiate the systemic inflammation cascade and its adverse consequences [36]. Intramedullary nailing may thus cause an increase in intramedullary pressure leading to release of bone marrow and fat into the venous blood system, which dramatically increased the incidence of ARDS and MOF [37]. Early intramedullary femoral nailing increases for example the incidence of ARDS more than four times in cases of severe chest trauma (33% versus 8%) [38]. Finally, Ventilator-associated pneumonia is a frequent complication may lead to ARDS. This may increase the risk of multiple organ failure and death [14].

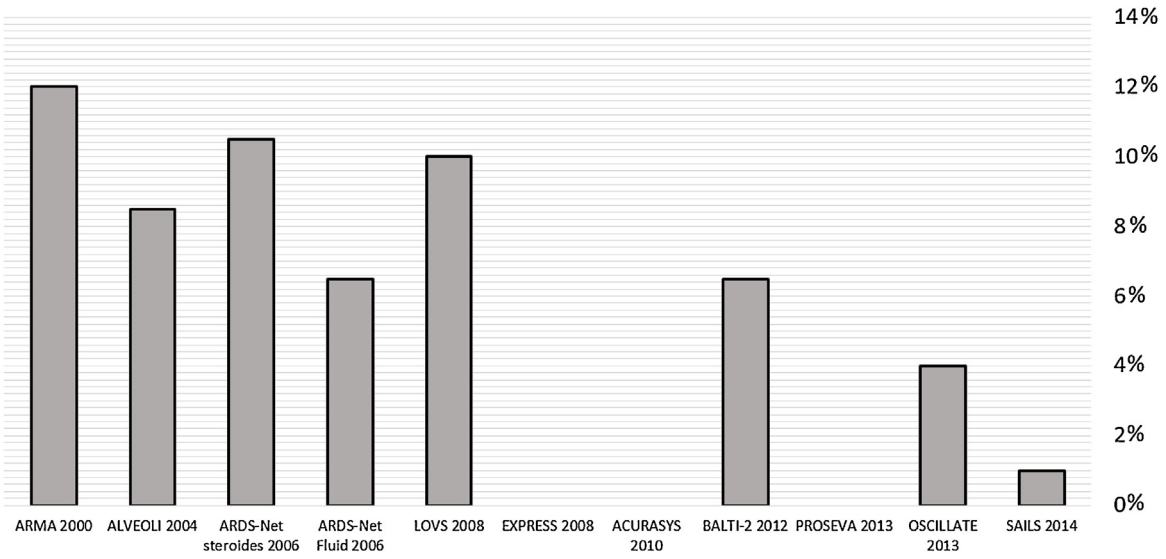


**Fig. 2.** Multicenter double-blind trials of acute respiratory distress syndrome (ARDS): percentage of trauma patients included.

## 5. Mechanical ventilation and chest trauma

Chest trauma is associated with the need for mechanical ventilatory support, especially in the presence of pulmonary contusions, multiple rib fractures or flail chest [39]. However, mechanical ventilation may cause itself or potentiate ventilator-induced lung injury [40]. Indeed, in patients with ARDS, excessive pressure or volume applied to the lung may cause regional parenchymal over distention, also called barotrauma and volotrauma. Cyclic opening and closing may also create shear stresses at the interface between open and closed lung regions (atelectrauma) [40]. Finally, local activation of the inflammatory cascade leads to destruction of the extracellular matrix and biotrauma. Consequently, ventilator strategies aimed at reducing parenchymal damage and the inflammatory response. Recent studies have shown significant benefits of lung-protective ventilation strategies [41]. As a consequence, most experts strongly recommend the use of a physiological low tidal volume of 6–8 mL/kg (predicted body weight) and limiting the inspiratory plateau pressure under 30 $cmH_2O$ in trauma patients [42]. Positive end-expiratory pressure (PEEP) seems to be moreover indispensable to optimize oxygenation. The level of PEEP must thus be titrated in order to define the optimal PEEP [43]. The use of rescue therapies such as neuromuscular blockade improves outcomes in ARDS patients, limitation of work of breathing, reduction of resistance from the chest wall and abdomen, and improvement in ventilator synchrony [44]. In patients with severe ARDS, early application of prolonged prone-positioning sessions significantly decreased mortality and improve oxygenation in various degrees of lung injury [45,46]. However, these strategies have never been specifically studied in case of chest trauma with associated ARDS, especially in case of multiple ribs fractures or flail chest [47]. Indeed, no study is available on the target trauma population concerning benefit of protective ventilation, the use of neuromuscular blockade, the goal of sedation, or the use of prone position. Therefore, management in trauma patients is based on an extrapolation of the medical setting since they were established on patients with ARDS from multiple origins, post-traumatic ARDS representing indeed only 8% to 13% of cohorts (Fig. 2).

Conversely, the open lung concept has been studied more with regard to trauma. The aim of open lung is to maintain open alveoli during inspiration and expiration using of significant PEEP, and to recruit atelectasis alveoli by increasing inspiratory pressure for a short period. High-frequency inverse-ratio ventilation was described in chest trauma as an alternative to external PEEP, which allows to generate alveolar and intrinsic PEEP trapping [48]. Recruitment manoeuvres reported in several studies used predefined opening pressures (i.e. 50, 65, 80 $cmH_2O$) with a targeted goal of arterial pressure oxygenation, considered as a robust indicator of optimal alveolar recruitment [48]. Many results indicate that application of the open lung concept is a reasonable strategy for ventilation in cases of severe chest trauma [49]. However, the use of high PEEP levels may be associated with important stress and strain in lung parenchyma, severe hypotension and a substantial reduction of cardiac output [50]. Furthermore, injured lung parenchyma is well-known to be heterogeneous in the trauma setting, leading to overdistention of healthy alveoli in case of too high PEEP. High PEEPs and recruitment manoeuvres may generate a massive air leak in cases of tracheobronchial rupture or bronchopleural fistula, strengthening the necessity to determine the optimal ventilation according to lesional status.

## 6. Alternatives to conventional ventilation

Conventional mechanical ventilation may be not efficient in some cases of ARDS related to chest trauma. Alternative methods of ventilation can then be used. These ventilatory strategies were thus studied in this specific setting. The results of several trials are summarized in Table 1.

### 6.1. Airway pressure release ventilation (APRV)

APRV is a ventilator strategy that may be used in critically ill patients affected by ARDS [51,52]. APRV involves bi-level positive airway pressure with a double-valve flow system; it is a pressure-limited, time-cycled method of ventilation that permits spontaneous breathing at any time throughout the respiratory cycle. This method allows alveolar recruitment beyond the inspiration phase [53]. The main difference from the traditional pressure mode is that the lower pressure level is short (e.g. 1–3 s), whereas the upper pressure level is particularly prolonged (e.g. 7–12 s). The tidal volume does not therefore depend only on differences between the two pressure levels, but is mainly generated by spontaneous breathing activity.

A prospective randomised trial comparing APRV and ARDS network protocols in trauma patients proved that APRV was safe

and provided the same quality of oxygenation ($PaO_2/FiO_2$ ratio) as the conventional method [54]. On the other hand, Maung et al. [55] found that duration of ventilation was increased with APRV compared the conventional methods in trauma patients with respiratory failure. Similarly, a systematic review comparing early APRV initiation versus a conventional ventilator strategy in trauma patients at high risk of ARDS showed a reduction of mortality using early APRV [56]. Despite increasing evidences on improved oxygenation and haemodynamics, patient comfort, and safety, many issues regarding this unconventional method remain unanswered. Reduction in mortality with APRV in cases of ARDS is indeed still under debate, which requires cautious for a large use of this mode in chest trauma.

### 6.2. High-frequency oscillatory ventilation (HFO)

HFO is a rescue method for patients with severe respiratory distress, which uses the concept of gas exchange diffusion and

**Table 1**
Important characteristics of major clinical studies of alternative ventilatory strategies in ARDS related to trauma (2001–2015).

| Study | Year | Design | No. in cohort | Initial $PaO_2/FiO_2$ | Overall survival (%) | Study protocol | Comment |
|---|---|---|---|---|---|---|---|
| Airway pressure release ventilation (APRV) | | | | | | | |
| Maxwell et al. [45] | 2010 | Prospective controlled randomized | 63 | 350 ± 150 | 93 | APRV ($n$ = 31) vs conventional mechanical ventilation (CMV) ($n$ = 32)<br>Ventilatory setting:<br>CMV group:<br>Tidal volume before:<br>6.4 ± 1.2 mL/kg<br>PEEP: 10 $cmH_2O$<br>Plateau pressure: < 30 $cmH_2O$<br>APRV group:<br>Inspiratory time: 4–5 s<br>Expiratory time: 0.4–1 s<br>Respiratory rate: 10–30/min<br>Time to APRV: immediately after initiation of mechanical ventilatory support<br>Duration of APRV: during control-assisted ventilatory support | Few patients presented ARDS in each group (35%–45%)<br>No significant difference was found between the 2 groups |
| Maung et al. [46] | 2012 | Retrospective observational | 309 | 186 ± 106 | 100 | APRV ($n$ = 75) vs. conventional mechanical ventilation ($n$ = 234)<br>Time to APRV: 24 h<br>Duration of APRV: not communicated | Patients presented minor or moderate ARDS in the APRV group<br>Many patients in the conventional mechanical group did not present ARDS (mean $PaO_2/FiO_2$: 243 ± 120)<br>Ventilator days were greater in the APRV group (19 vs. 10 days) |
| High-frequency oscillation (HFO) | | | | | | | |
| Funk et al. [51] | 2008 | Retrospective observational | 70 | 96 ± 11 | 87 | Ventilatory setting: not communicated<br>Time to HFO: not communicated<br>Duration of HFOV: 5 ± 1.5 days | Most patients had pulmonary contusions<br>A significant improvement in oxygenation index was found after HFO initiation<br>A significant improvement in $PaO_2/FiO_2$ was found after HFO initiation |
| Vrettou et al. [52] | 2013 | Prospective observational | 13 | 86 ± 12 | 53 | Conventional ventilatory setting:<br>Tidal volume before HFO initiation: 8.3 ± 1.3 mL/kg<br>Minute ventilation before HFO initiation: 15.0 ± 2.9 L/min<br>PEEP before HFO initiation:<br>14.6 ± 2.6 $cmH_2O$<br>Plateau pressure:<br>30.4 ± 4.5 $cmH_2O$<br>Time to HFO: 35 h<br>Duration of HFO: not communicated | Most patients presented ARDS and a traumatic brain injury<br>A significant improvement in $PaO_2/FiO_2$ was found after initiation of HFO |
| High-frequency percussive ventilation (HFPV) | | | | | | | |
| Salim et al. [55] | 2004 | Retrospective observational | 10 | 92 ± 13 | 90 | Conventional ventilatory setting was not communicated<br>Time to HFPV: immediately in cases of refractory hypoxaemia under conventional mechanical ventilation<br>Duration of ILV: until $PaO_2$ recovery according to study physicians | Most patients presented ARDS and a traumatic brain injury<br>A significant improvement in $PaO_2/FiO_2$ was found |

**Table 1** (*Continued*)

| Study | Year | Design | No. in cohort | Initial $PaO_2/FiO_2$ | Overall survival (%) | Study protocol | Comment |
|---|---|---|---|---|---|---|---|
| Eastman et al. [54] | 2006 | Retrospective observational | 12 | 90 ± 50 | 58 | Initial management by conventional ventilatory setting:<br>Tidal volume before HFPV initiation was not communicated<br>PEEP before HFPV initiation: 10–20 $cmH_2O$<br>Plateau pressure before HFPV initiation: 30 ± 8 $cmH_2O$<br>Time to HFPV: immediately in cases of refractory hypoxaemia under conventional mechanical ventilation<br>Duration of HFPV: until $PaO_2$ recovery according to study physicians | A significant improvement in the oxygenation index was found after initiation of HFPV<br>A significant improvement in $PaO_2/FiO_2$ was found after initiation of HFPV |
| Independent lung ventilation (ILV) | | | | | | | |
| Katsaragakis et al. [58] | 2005 | Case report | 2 | 48 | 87 | Conventional ventilatory setting was not communicated<br>Synchronous and asynchronous ventilation were used<br>Time to ILV: 25 h<br>Duration of ILV: 5 days | Patients presented unilateral or asymmetrical lung trauma<br>A significant improvement in $PaO_2/FiO_2$ was found after initiation of ILV |
| Sawulski et al. [59] | 2012 | Case report | 1 | 123 | 100 | Synchronous then asynchronous ventilation settings were used<br>Time to ILV: 48 h<br>Duration of ILV: 2 days | Patient presented a unilateral chest lesion<br>Improvement in ventilatory settings was found after initiation of ILV |
| High-frequency jet ventilation (HFJV) | | | | | | | |
| Riou et al. [62] | 2001 | Retrospective observational | 9 | 73 ± 24 | 56 | Conventional ventilatory setting:<br>Tidal volume: 659 ± 151 mL<br>PEEP: 10 ± 3 $cmH_2O$<br>Time to HFJV: 7 ± 6 h<br>Duration of HFJV: ± 7 days | Patients with severe pulmonary contusions<br>A significant improvement in $PaO_2/FiO_2$ was found after initiation of HFPV<br>A significant improvement in acid base correction was found after initiation of HFPV |
| Extracorporeal membrane oxygenation (ECMO) | | | | | | | |
| Cordell-Smith et al [73]. | 2006 | Retrospective observational | 28 | 62 | 71 | Heparinization: systemic heparin was adapted to activated clotting time<br>Time to extracorporeal life support (ECLS): 69 h<br>Duration of ECLS: 141 h | Mean time to ECMO was 61 h for survivors versus 87 h for non-survivors |
| Arlt et al. [75] | 2010 | Prospective observational | 10 | 47 | 60 | VV-ECMO: 7 patients<br>VA-ECMO: 3 patients<br>Heparinization: no heparin was used after initiation of ECMO<br>Time to ECLS was not communicated<br>Duration of ECLS: 5 days | Patients presented active bleeding<br>A significant improvement in $PaO_2/FiO$ was found after initiation of ECMO |
| Bonacchi et al. [67] | 2013 | Prospective observational | 14 | 62 | 71 | VV-ECMO: 4 patients<br>VA-ECMO: 10 patients<br>Heparinization free time on ECLS: 20.7 ± 19.8 h<br>Systemic heparin was adapted to activated clotting time<br>Time to ECLS: 351.8 ± 242 min<br>Duration of ECLS 128.7 ± 113 h | A significant improvement in cardiac index, mean arterial pressure, blood lactate, $PaO_2$, $PaCO_2$, and acid base correction within 3.5 ± 1.5 h of initiation of ECMO was found |
| Ried et al. [64] | 2013 | Retrospective observational | 52 | 63 | 71 | VV-ECMO: 26 patients<br>pECLA: 26 patients<br>Heparinization: systemic heparin was adapted to reach a partial thromboplastin time of 40–50 s<br>Time to ECLS: 5 ± 8 h<br>Duration of ECLS: 7 ± 4 days | pECLA is an extracorporeal carbon dioxide removal system<br>A significant improvement in cardiac index, mean arterial pressure, blood lactate, $PaO_2$, $PaCO_2$, and acid base correction after initiation of ECMO was found |

**Table 1** (*Continued*)

| Study | Year | Design | No. in cohort | Initial $PaO_2/FiO_2$ | Overall survival (%) | Study protocol | Comment |
|---|---|---|---|---|---|---|---|
| Biderman et al. [68] | 2013 | Retrospective observational | 10 | 62 | 70 | ILA circuit: 5 patients<br>ECMO: 5 patients<br>Heparinization: 3 (30%) patients received systemic heparin in the first 48 h<br>Time to ECLS: 3 (1–7) days<br>Duration of ECLS: 10 ± 5 days | ILA is an extracorporeal carbon dioxide removal system<br>Patients presented traumatic brain injury<br>A significant improvement in $PaO_2/FiO_2$ was found after initiation of ECMO<br>A significant improvement in acid base correction was found after initiation of ECMO |
| Wu et al. [72] | 2014 | Prospective observational | 20 | 56 | 70 | VV-ECMO: 20 patients<br>Heparinization: systemic heparin was adapted to activated clotting time<br>Systemic heparin strategy was utilized in 55% of patients<br>Time to ECLS: 69 h<br>Duration of ECLS: 10 ± 5 days | 7 cases of massive haemorrhage during ECMO, 3 were lethal<br>A significant improvement in $PaO_2/FiO_2$ was found after initiation of ECMO |
| Guirand et al. [69] | 2014 | Retrospective controlled | 102 | 52 | 65 | Two groups were compared: ECMO group ($n$ = 26) and conventional mechanical ventilation ($n$ = 26) ARDSNet protocol goals were used in conventional group<br>No heparinization was used in cases of emergent surgery or traumatic brain injury<br>Time to ECLS: 5 days<br>Duration of ECLS: 224 h | A significant improvement in survival was found in the ECMO group: 64.7% vs 23.5%<br>A significant improvement in survival was also found in the ECMO group after matching for age and Injury Severity Score |
| Wu et al. [71] | 2015 | Retrospective observational | 19 | 60 | 69 | VV-ECMO: 9 patients<br>VA-ECMO: 10 patients<br>Heparinization: 16 (84%) patients received systemic heparin<br>Time to ECLS: 69 h<br>Duration of ECLS: 141 h | Five patients had traumatic brain haemorrhage (3/5 survived)<br>Five patients with systemic heparinization died (31%) |
| Jacobs et al. [74] | 2015 | Retrospective observational | 85 | 60 ± 3 | 74 | VV-ECMO: 63 patients<br>VA-ECMO: 22 patients<br>Heparinization: systemic heparin from 4 mg/kg to 1.5 mg/kg was used<br>Time to ECLS: 95 ± 13 h<br>Duration of ECLS: 207 ± 24 h | Haemorrhagic complications occurred in 25 cases (29.4%)<br>A shorter duration of ECMO and the use of venovenous ECMO were predictive criteria for survival |
| Bosarge et al. [70] | 2016 | Prospective controlled | 29 | 63 | 66 | Two groups were compared : ECMO group ($n$ = 15) and conventional mechanical ventilation ($n$ = 14)<br>ARDSNet protocol goals were used in conventional group<br>Heparinization: systemic heparin was adapted to thromboelastogram<br>Time to ECLS: 48 h<br>Duration of ECLS: 8 days | Haemorrhagic complications occurred in 5 cases (33.3%)<br>A significant improvement in survival was also found in the ECMO group |

Brownian motion. A constant mean airway pressure is maintained throughout the respiratory cycle, which is reputed to keep the alveoli open. A pressure differential around the mean pressure is generated that induces a very low tidal volume with a very high frequency [57]. HFO offers two potential advantages over conventional mechanical ventilation for patients with ARDS. First, the removal of gas convection and tidal volumes allow reduction of lung overdistention causing dynamic stress. Second, decarboxylation and oxygenation are decoupled in HFO. Decarboxylation mainly depends on the pressure differential around the mean pressure and oscillation frequencies. Oxygenation depends on the mean pressure level and $FiO_2$. Specific adverse consequences using HFO have however to be considered: more numerous atelectasis caused by airway debris and secretions, and higher haemodynamic instability caused by the absence of alternate pressures phases. If HFO is nowadays the standard of care for refractory hypoxemia in neonatology care, its use in adults with moderate to severe ARDS was associated with a higher mortality compared to conventional ventilation even if in another multicentre study, the use of HFO had no significant effect on 30-day mortality in patients undergoing mechanical ventilation for ARDS [58]. In trauma patients, HFO ventilation was proposed as a rescue strategy when conventional mechanical ventilation did not provide sufficient oxygenation [59,60]. Nevertheless, some medical trials highlighted a higher mortality rate associated with the use of HFO in cases of ARDS not related to trauma [58]. Consequently, despite several physiological benefits in cases of chest trauma, the use of HFO is not recommended in clinical practice.

### 6.3. High-frequency percussive ventilation (HFPV)

HFPV can be considered as a hybrid technique between conventional pressure control ventilation and HFO [61]. A special valve called a phasitron is used to generate diffusive gas exchange due to a high frequency (200−900/min). HFPV mainly differs than HFO by the presence of two different steady pressure levels. The frequency of alternating between two pressure levels is usually fixed at 10−15/min, similar to conventional methods [62]. This cyclic variation of the airway pressure level limits the traditional adverse consequences induced by HFO. Other benefits of HFPV include the generation of intra-bronchial vibrations, airway turbulence and higher airflow. All these flow characteristics may enhance mobilisation and clearance of airway debris and secretions, allowing an improvement in alveolar recruitment without increasing dynamic stress. Recently, Godet et al. [63] have confirmed these benefits in CT scan study in patients with early non-focal ARDS. HFPV mode has adequate humidification of administrated gases, which allows safe prolonged. The imprecision about the parameter settings and the risk of drifting are the main reasons of the low use of this method.

In the setting of chest trauma, HFPV showed on small samples a trend of improved oxygenation in patients with ARDS, without concomitant increases in respiratory pressures [64]. These results seem contradictory with ARDS-related pneumonia, which was associated with lower oxygenation ability and lower survival [65]. Although further trials are necessary to support greater use in trauma setting, HFPV seems to be an acceptable temporary ventilation strategy in several specific critical cases, such as refractory hypoxemic pulmonary contusions or broncho-pleural disruption.

### 6.4. Independent lung ventilation (ILV)

The distribution of inspired air is dictated by the rule of minimal workload. It is mainly dependent on the properties of different lung areas. Lung compliance in cases of chest trauma is frequently altered heterogeneously or asymmetrically. In such cases, mechanical ventilation will drive a large proportion of the tidal volume towards the less affected areas, which may induce both serious overdistension of healthy parenchyma and an important ventilatory shunt in injured areas. The likelihood of volotrauma is then increased in the normal parenchyma; moreover recruitment manoeuvres are inefficient and harmful [66]. ILV is provided either by double-lumen endotracheal tube or additional endobronchial blocker, and may be synchronous and asynchronous.

Only a small number of case reports have been published on ILV in cases of ARDS after trauma [67]. In these reports, $FiO_2$ requirements and airway pressures were reduced, and $PaO_2/FiO_2$ and ventilation/perfusion ratio improved. Preferential use of ILV in trauma seems to be in order to establish different ventilation regimes for each lung according to their mechanical properties. This attractive concept has however been moderated by constraints of implementation. ILV is indeed challenging to achieve at the bedside; it requires accurate adjustment and cautious and specific surveillance. Furthermore, re-intubation is necessary in most cases that may be a real risk-taking in a hypoxemic patient. Tube position has to be regularly verified. Finally, although the indications for IVL are poorly defined in the literature for chest trauma, it seems reasonable to reserve this complex strategy for rare refractory patients with a unilateral severe air leak or unilateral hypoxemic massive contusions [68,69]. In these specific cases, the combination of conventional ventilation with other methods such as HFPV, or with extracorporeal support has also been described.

### 6.5. High-frequency jet ventilation (HFJV)

HFJV aims to deliver high-frequency gas pulses at high velocity and high pressure with an injector either directly through the trachea or an endotracheal tube. This is an air-leak ventilation that maintains sufficient passive gas expiration. Basal settings are the working pressure (1–4 bar), injection frequency (1–5 Hz) and inspiration/expiration ratio. HFJV has been described in the critical care and surgical settings as a rescue ventilatory strategy in cases of massive air leak generating respiratory failure [70]. Broncho-pleural or pleuropulmonary fistulae were the main causes. The major danger with HFJV is the volotraumatic risk. Most devices do not have a pressure limitation detector, which may lead to rapid and threatening pulmonary hyperinflation if injected gases cannot be expired. A second important disadvantage with HFJV is the total absence of parameter monitoring. Finally, the main limitation of HFJV is the absence of rewarming and humidification system for injected gases for that forbids a prolonged use.

Only a few cases of HFJV use were reported in cases of chest trauma [71]. HFJV was thus proposed with success in the presence of traumatic tracheobronchial rupture, during the initial management while waiting for emergent surgical repair [72]. HFJV was not studied in cases of ARDS related to trauma. HFJV has therefore a limited place in trauma, mainly as a temporary and rescue ventilation strategy. The risks of volotrauma and mechanic tracheitis are indeed too important. Technical advances are required to warrant safe prolonged use of HFJV in critically ill patients.

### 6.6. Extracorporeal assistance

Despite small tidal volumes strategy, harmful plateau pressures may be unavoidable, refractory hypoxaemia may occur, and carbon dioxide removal may be insufficient. Extracorporeal assistance may in this case break the vicious cycle and allow a lung-protective strategy to be maintained. Extracorporeal assistance is used as a bridge to provide sufficient ventilation, oxygenation, and $CO_2$ removal while limiting lung damage and favouring recovery of respiratory function [73]. Two extracorporeal systems are available to date: extracorporeal carbon dioxide removal, called $ECCO_2R$ or extracorporeal membrane oxygenation (ECMO). $ECCO_2R$ is a technique of partial respiratory support that achieves removal of $CO_2$ from the blood (extracorporeal output 0.4−2 L/min) with poor influence on oxygenation. ECMO offers in contrast a total respiratory support due to a significant extracorporeal blood flow (3−7 L/min) [74]. ECMO requires therefore the placement of a large cannula via surgical dissection and frequently requires significant heparinisation to avoid filter clotting. ECMO can be deployed in a veno-arterial or veno-venous configuration. In medical cases of ARDS, veno-venous ECMO is mainly used in the presence of refractory massive hypoxemia under conventional mechanical ventilation and/or uncontrolled hypercapnia or acidosis [75].

In the context of trauma, ECMO was essentially studied as a rescue strategy with interesting results (Table 1). Ried et al. [73] described 52 patients requiring ECMO because of a refractory hypoxemia under conventional mechanical ventilation. The median $PaO_2/FiO_2$ ratio was immediately and systematically increased after initiation of ECMO, however the mortality rate was particularly low in this series (15%). These findings corroborate results from other trials in cases of trauma [76]. Guirand et al. [77] have also shown, using a propensity score design, that veno-venous ECMO was independently associated with higher survival. This increased survival was reported in two other studies [73]. Nevertheless, ECMO support remains controversial in cases

of trauma, especially due to fear morbid haemorrhaging in patients affected by coagulopathy or injuries with a bleeding risk. Wu et al. [78] showed that haemorrhagic complications induced during ECMO were non-negligible. Several major or lethal cases of bleeding were observed in the presence of an activated partial thromboplastin time ratio between 1.5 and 2 [79]. Several major or lethal cases of bleeding were observed under ECMO support using a reasonable anticoagulation (aTTPr between 1.5 and 2) [79-81]. Anticoagulation has been however proved to be not indispensable in the first days following initiation of extracorporeal assistance. Arlt et al. [82] have thus demonstrated that heparin-free ECMO improves outcome in severe trauma patients with an acceptable survival duration (mean, 5 days; range, 0.5 – 11 days). Robba et al. [83] also showed that the heparin-free veno-venous ECMO was a safe and valid option in patients with a high-risk of bleeding. The choice of cannulation sites may however lead to specific technical issues in trauma. The two main configurations are femoro-jugular and femoro-femoral cannulation. Several injuries may therefore limit or forbid the insertion of draining cannula via the femoral site; for example limb, pelvic, vascular or caval injuries. In this case, extracorporeal assistance can nonetheless be performed either by the placement of a dual-lumen cannula (one draining cannula and one infusion cannula) in superior vena cava, or by the use of two single-lumen cannulas in superior caval area. To summarise, the evidences supports to date that veno-venous ECMO has to be considered in chest trauma as the referent rescue strategy when hypoxaemia is refractory under maximal mechanical ventilation and/or hypercapnia is uncontrolled [84,85]. These considerations were reinforced by results of EOLIA study, which found a benefice among patients with very severe ARDS even if mortality was not significantly lower with earlier ECMO than with a strategy of conventional mechanical ventilation that included ECMO as rescue therapy [86]. Major bleeding risk will then guide the level of systemic heparinisation. In addition, with this robust indication, prophylactic use of ECMO is proposed by some experts in order to maintain ultra-protective ventilation and avoid the noxious effects of aggressive ventilation in cases of ARDS. Prospective studies are however necessary in cases of traumatic ARDS to confirm the potential benefits of this strategy.

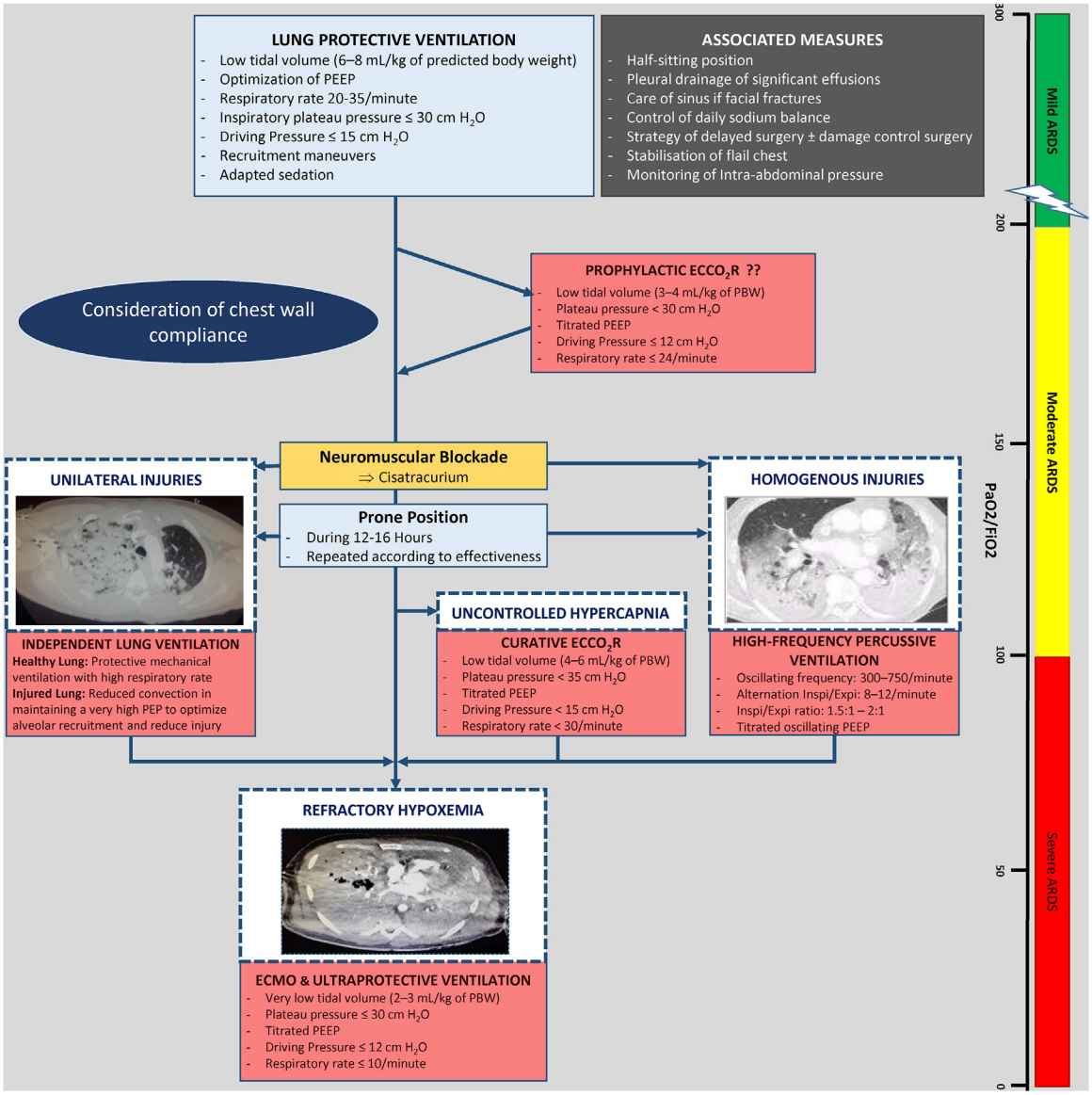


**Fig. 3.** Proposal of management algorithm for acute respiratory distress syndrome after chest trauma. PEEP: positive end-expiratory pressure; PBW: predicted body weight; alternation Inspi/Expi: alternation between inspiratory and expiratory phases; Inspi/Expi ratio: ratio between inspiratory time and expiratory time; ECMO: extracorporeal membrane oxygenation.

## 7. Conclusions

Although it has its own specificities, post-traumatic ARDS has not received as much investigations as ARDS in the medical setting in recent years. The development of ARDS in cases of chest trauma has multiple causes. Its occurrence is favoured by the inflammatory cascade induced by trauma and strengthened by secondary aggression. The management of ARDS related to trauma needs to be treated on an individual basis, depending on the location and type of lung or chest injury (Fig. 3). Mechanical ventilation of chest trauma may be a real challenge because of the difficulties in achieving adequate gas exchange and lung protection. Alternative strategies to standard mechanical ventilation have a modest place in clinical practice, mainly temporally and in rescue context. ECMO should be considered in trauma patients as the main alternative when mechanical ventilation failed and survival is compromised. However, many studies are necessary to consolidate current state of knowledge.

## Consent for publication

Not applicable.

## Availability of data and material

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

**Funding**

The authors declare that they have no sources of funding.

## Authors' contributions

J.C. performed the draft of the study, S.R. and J.C. wrote the article, which S.J and X.C. read and approved the final manuscript.

**Disclosure of interest**

The authors declare that they have no competing interest.

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