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ORIGINAL ARTICLE

Ventilation Practices in Subarachnoid Hemorrhage: A Cohort Study Exploring the Use of Lung Protective Ventilation

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Abstract
Background Acute respiratory distress syndrome (ARDS) is common following aneurysmal subarachnoid hemorrhage (SAH), but the influence of mechanical ventilator settings on its development is unclear. We sought to determine adherence to lung protective thresholds in ventilated patients with SAH and describe the association between ventilator settings and subsequent development of ARDS.
Methods We conducted a retrospective cohort study of consecutive patients receiving mechanical ventilation within 72 h of SAH at a single academic center. Ventilator settings and blood gas data were collected twice daily for the first 7 days of ventilation along with ICU and hospital outcomes. Lung protective ventilation was defined as follows: tidal volume $\leq$8 mL/kg of predicted body weight, positive end-expiratory pressure (PEEP) $\geq$5 cm H_2O, and peak or plateau pressure $\leq$30 cm H_2O. The development of ARDS was ascertained retrospectively by PaO_2/FiO_2 $\leq$300 with new bilateral lung opacities on chest X-ray within one day of hypoxemia.
Results We identified 62 patients who underwent early mechanical ventilation following SAH. PS and Continuous Positive Airway Pressure were common ventilator modes with a median tidal volume of 7.8 mL/kg [interquartile range 6.8–8.8], median peak pressure of 14 cm H_2O [IQR 12–17], and median PEEP of 5 cm H_2O [IQR 5–6]. Adherence to tidal volumes $\leq$8 mL/kg was seen in 64 % of all observations and peak pressures $<$30 cm H_2O were 94 % of all observations. All three lung protective criteria were seen in 58 % of all observations. Thirty-one patients (50 %) were determined to have ARDS. ARDS patients were more frequently ventilated with a peak pressure $>$30 cm H_2O (11.3 % of ARDS ventilation days vs. 0 % of non-ARDS ventilation days; $p < 0.01$). Initial tidal volume was not associated with subsequent development of ARDS in univariate ($p = 0.6$) or multivariate analysis ($p = 0.49$). Only the number of ARDS risk factors was independently associated with the development of ARDS (Adjusted Odds Ratio 2.8 per additional risk factor [95 % CI 1.2–6.5]).
Conclusion Patients with SAH requiring mechanical ventilation frequently breathe spontaneously, generating tidal volumes above usual protective thresholds regardless of meeting ARDS criteria. In patients with SAH, the presence of an additional ARDS risk factor should prompt close screening for the development of ARDS and consideration of adjustment of ventilator settings to meet lung protective thresholds.

Keywords Mechanical ventilation · Subarachnoid hemorrhage · Acute respiratory distress syndrome

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Abbreviations

APACHE II	Acute physiology and chronic health evaluation II
ARDS	Acute respiratory distress syndrome
CPAP	Continuous positive airway pressure
ICP	Intracranial pressure
ICU	Intensive care unit
IQR	Interquartile range
$PaCO_2$	Arterial partial pressure of carbon dioxide
PS	Pressure support ventilation
PEEP	Positive end-expiratory pressure
SAH	Subarachnoid hemorrhage
WFNS	World federation of neurological surgeons

Introduction

Acute respiratory distress syndrome (ARDS) is a common complication of aneurysmal subarachnoid hemorrhage (SAH) in patients admitted to the intensive care unit (ICU) [1], and has been associated with poor clinical outcomes [1, 2].

Lung protective ventilation (targeting tidal volumes of 6 mL/kg of predicted body weight (PBW), positive end-expiratory pressure (PEEP) $\geq$5 cm H_2O, and peak or plateau Pressure $\leq$30 cm H_2O) has improved outcomes among patients with established ARDS [3] and may help to prevent ARDS in patients at risk [4, 5]. Injurious ventilator settings have been associated with the development of ARDS in patients with normal pulmonary function [6].

Neurologic factors and initial ventilator settings are thought to increase the risk of ARDS in patients with SAH [7]. Studies examining ventilation strategies in neurologic patients have demonstrated the application of lung protective ventilation to be inconsistent [7, 8]. Accordingly, data describing the influence of ventilator settings on the development of ARDS in patients with SAH are lacking.

In this study, we sought to determine adherence to lung protective thresholds in ventilated patients with SAH and describe the association between ventilator settings and subsequent development of ARDS.

Materials and Methods

We studied patients admitted to the Toronto Western Hospital ICU, which is a 24 bed mixed medical surgical and neurosurgical intensive care unit in an academic neurovascular centre. The Research Ethics Board at the University Health Network approved the study protocol.

Cohort Assembly

We conducted a retrospective observational study of early mechanical ventilation (defined as the first seven days of ventilation) in consecutive patients with aneurysmal SAH. Patients were identified by cross-linking hospital administrative databases and the ICU clinical database using an admitting diagnosis of SAH (ICD-10 code I60). A study period of two fiscal years was selected to facilitate access to administrative data.

We included patients with 18 years of age or older with documented SAH on digital subtraction or computerized tomographic angiography on admission to hospital. Patients were excluded if admission to the ICU occurred greater than 72 h after hospital admission. The patient charts and the electronic patient record were reviewed for demographic and clinical data, which included Acute Physiology and Chronic Health Evaluation II (APACHE II) score [9], ICU length of stay, duration of mechanical ventilation, and hospital mortality. Height and gender were extracted to determine PBW. Other ARDS risk factors such as sepsis, shock, inhalational injury, trauma, fat emboli, drug overdose, pneumonia, near drowning, lung contusion, massive transfusion (defined as $>$15 units/24 h), cardiopulmonary bypass, reperfusion pulmonary edema, aspiration, and pancreatitis were collected for each patient on each study day [10]. SAH severity was classified using the World Federation of Neurological Surgeons (WFNS) Grade, [11] and the risk of delayed ischemia was recorded using the modified Fisher Grade [12].

Ventilator settings and arterial blood gas data were extracted twice daily (at 10:00 h and 22:00 h) for the first seven days of mechanical ventilation. We accepted data within 1 h of these time points if no value was recorded at these times. In the absence of recorded peak pressures, the sum of PEEP and driving pressure was used as a conservative estimate. Lung protective ventilation was defined as follows: tidal volume $\leq$8 mL/kg of PBW, PEEP $\geq$5 cm H_2O, and peak or plateau pressure $\leq$30 cm H_2O, according to the most liberal criteria from the ARDS Network's published protocol [3]. We defined injurious tidal volumes to be $\geq$12 mL/kg.

The development of ARDS was ascertained retrospectively by the presence of PaO_2/FiO_2 $\leq$300 and new bilateral lung opacities on chest X-ray [13] according to the Berlin Criteria. If no chest radiograph was available for the day on which the patient met hypoxemia criteria, we accepted new bilateral lung opacities occurring within 1 day of this event. Chest X-rays for all patient days meeting hypoxemia criteria were reviewed independently by two physicians, with a third physician adjudicator in the event of disagreement. The presence of cardiogenic

pulmonary edema or congestive heart failure was ascertained from the clinical notes as per the impressions of the treating physicians using clinical, hemodynamic, and echocardiographic data as available.

The primary outcome of the study was the proportion of ventilation days in which lung protective thresholds were met. Secondary outcomes included the development of ARDS as a dichotomous endpoint, time to development of ARDS, ICU length of stay, duration of mechanical ventilation, ICU mortality, and hospital mortality.

Statistical Analysis

Summary statistics were reported as means with standard deviations or medians with interquartile ranges for continuous data, and proportions for categorical data. Differences between groups were tested using Wilcoxon, Kruskal–Wallis, and χ^2 tests as appropriate. The association between tidal volumes and subsequent development of ARDS after the first day of mechanical ventilation was explored using multivariate logistic regression, adjusting for covariates. The development of ARDS was the binary dependent variable with Age, Sex, APACHE II, WFNS Grade, mean tidal volume over all ventilation days prior to ARDS onset, and the number of ARDS risk factors as independent covariates. Model fit was assessed using the Hosmer–Lemenshow Goodness of Fit Test. All statistical analyses were carried out using the SAS 9.2 software package (SAS Institute, Cary, NC, USA).

Results

We identified 94 patients with admission diagnoses of SAH admitted to the ICU between April 2010 and March 2012. We excluded 32 patients (10 patients did not have identifiable aneurysms, 5 patients were excluded because ICU admission occurred >72 h after hospital admission, and 17 patients did not receive mechanical ventilation), resulting in a final study cohort of 62 patients. Characteristics of the study group are shown in Table 1. The median age of study participants was 59 [IQR 51–67]. Females comprised 60 % of the cohort. The median length of stay in the ICU was 6 days [IQR 3–16], and the median duration of mechanical ventilation was 3 days [IQR 1–11]. Overall, the ICU mortality rate was 27 % and hospital mortality rate was 35 %.

Adherence to Lung Protective Ventilation

We collected 471 observations of ventilator parameters corresponding to 272 ventilated patient days. Ventilation modes where patients were breathing spontaneously (PS and CPAP) accounted for the majority (78 %) of all ventilator observations: CPAP comprised 231 (49 %), pressure support 138 (29 %), pressure control 73 (16 %), and volume control 29 (6 %). Across all observations, the median tidal volume was 7.8 mL/kg [IQR 6.8–8.8] with a median PEEP of 5 cm H_2O [IQR 5–6] and a median peak pressure of 14 cm H_2O [IQR 12–17].

All three lung protective criteria were observed in 58 % of ventilated days (see Table 2). Figure 1 displays the proportion of patients each day whose average tidal volume met lung protective thresholds: daily mean tidal volumes $\leq$8 mL/kg PBW were seen in 64 % of all ventilated days, while daily mean tidal volumes $\leq$6 mL/kg PBW were seen in 18 % of ventilated days. Patients were more likely to have mean tidal volumes >8 mL/kg on PS and CPAP

Table 1 Baseline characteristics of study cohort

Characteristic	$N = 62$
Age (years), median [IQR]	59 [51–67]
Female sex, *N* (%)	37 (59.68)
Height (cm), median [IQR]	165 [158–173]
Predicted body weight (kg), median [IQR]	59.7 [50.6–70.3]
APACHE II score, median [IQR]	12 [8–18]
WFNS grade, *N* (%)	
I	11 (18)
II	13 (21)
III	1 (2)
IV	12 (19)
V	25 (40)
Modified Fisher grade, *N* (%)	
1	20 (32)
2	8 (13)
3	17 (27)
4	17 (27)
Risk factors for ARDS, *N* (%)	
Sepsis	5 (8)
Shock	6 (10)
Pneumonia	16 (26)
Gastric aspiration	2 (3)
Transfusion	1 (2)
Arterial blood gas, median [IQR]	
Day 1 pH	7.41 [7.38–7.45]
Day 1 $PaCO_2$—mmHg	30 [29–35]
Day 1 bicarbonate	20 [18–22]
Duration of ICU stay (Days), median [IQR]	6 [3–16]
Duration of mechanical ventilation (Days), median [IQR]	3 [1–11]
ICU mortality rate, *N* (%)	17 (27)
Hospital mortality, *N* (%)	22 (35)

Table 2 Description of ventilation practices

	$N = 471$ observations
PEEP (cm H_2O), median [IQR]	5 [5–6]
Tidal volume (mL/kg), median [IQR]	7.8 [6.7–8.8]
Peak pressure (cm H_2O), median [IQR]	14 [12–17]
Lung protective ventilation	$N = 272$ ventilated days
Tidal volume ≤6 mL/kg, *N* (%)	50 (18)
Tidal volume ≤8 mL/kg, *N* (%)	174 (64)
Peak pressure <30 cm H_2O, *N* (%)	255 (94)
PEEP ≥5 cm H_2O, *N* (%)	272 (100)
Lung protective ventilation, *N* (%)	159 (58)
V_T >12 mL/kg, *N* (%)	37 (14)

modes (34.2 % of all observations on spontaneous modes vs. 20.6 % of all observations on assist-control modes; $p = 0.029$). A mean peak pressure below 30 cm H_2O was observed in 255 (94 %) of patient days, and mean PEEP ≥5 cm H_2O was identified in 272 (100 %) of ventilation days. Five patients (6 %) received tidal volumes greater than 12 mL/kg at least once during the observation period. Of these patients, 2 were exposed for 1 day, 1 was exposed for 2 days, and 2 were exposed for 4 days.

Development of ARDS

We identified 155 patient days for 45 unique patients where the PaO_2/FiO_2 was ≤300 mmHg. Initial adjudication of chest radiographs for bilateral opacities saw disagreement on 22 X-rays (kappa = 0.58), requiring adjudication by a third physician. From this, new bilateral lung opacities were determined to be present in 90 chest X-rays for 31 unique patients, resulting in 31 cases of ARDS. Of the 31 patients who developed ARDS, 45 % met criteria on the first day of mechanical ventilation. The median time to onset in the 31 patients who developed ARDS was 2 days [IQR 1–3].

Comparison of baseline characteristics, outcome measures, and ventilation practices in the ARDS and non-ARDS groups appears in Table 3. Twenty-one patients (24 %) in the overall study cohort had at least one other ARDS risk factor in addition to SAH. Fifteen of the patients with ARDS (48 %) had at least one additional ARDS risk factor compared to 5 out of the 31 patients (16 %) in the non-ARDS group ($p = 0.02$). No significant difference was found between study groups with regards to WFNS ($p = 0.58$). There were more patients with higher modified Fisher scores in the ARDS group: 22 (71 %) of the ARDS group was modified Fisher 3 or 4, compared to 12 (39 %) of the non-ARDS group, although this did not meet statistical significance ($p = 0.08$).

Patients who developed ARDS had a longer duration of mechanical ventilation (median duration 9 vs. 2 days; $p = 0.0003$), and ICU length of stay (median length of stay 13 vs. 4 days; $p = 0.0001$). The two groups did not differ in regards to ICU mortality (35 % in ARDS group vs. 19 % in non-ARDS group; $p = 0.15$) and hospital mortality (42 % in ARDS group vs. 29 % in non-ARDS group; $p = 0.29$).

ARDS and Associated Ventilation Practices

Overall ventilation settings did not differ significantly between ventilation days meeting ARDS criteria and those that did not. For patients who developed ARDS, there was no difference in ventilation settings in the 39 ventilated days before and the 131 ventilated days after ARDS onset (see Table 4). There was no difference in the proportion of

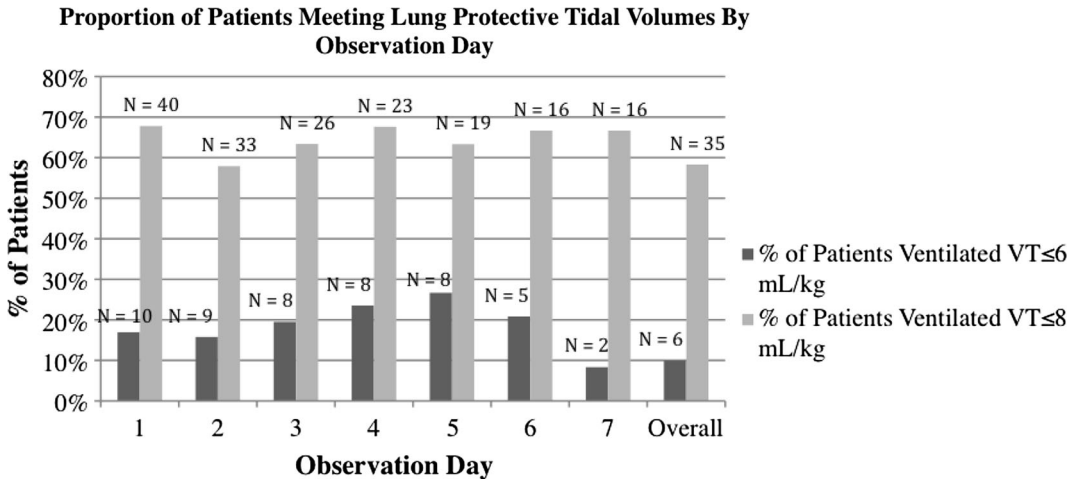

Fig. 1 Proportion of patients meeting lung protective tidal volumes by observation day

Table 3 Characteristics of study cohort stratified by development of ARDS

Characteristic	ARDS group ($N = 31$)	Non-ARDS group ($N = 31$)	
Age (years), median [IQR]	59 [52–68]	59 [50–65]	$p = 0.94$
Female sex, *N* (%)	18 (58)	19 (61)	$p = 0.8$
Height (cm), median [IQR]	166 [160–175]	165 [157–173]	$p = 0.32$
Predicted body weight (kg), median [IQR]	61.5 [52.4–73.3]	58.7 [49.7–68.8]	$p = 0.14$
APACHE II score, median [IQR]	13 [11–21]	9 [6–17]	$p = 0.03$
Duration of ICU stay (Days), median [IQR]	13 [6–22]	4 [2–6]	$p < 0.001$
Duration of mechanical ventilation (Days), median [IQR]	9 [2–20]	2 [1–4]	$p < 0.001$
ICU mortality rate, *N* (%)	11 (35)	6 (19)	$p = 0.15$
Hospital mortality, *N* (%)	13 (42)	9 (29)	$p = 0.29$
WFNS grade, *N* (%)			$p = 0.58$
I	4 (13)	7 (23)	
II	8 (26)	5 (16)	
III	0 (0)	1 (3)	
IV	7 (23)	5 (16)	
V	12 (39)	13 (42)	
Modified Fisher grade, *N* (%)			$p = 0.09$
1	7 (23)	13 (42)	
2	2 (6)	6 (19)	
3	11(35)	6 (19)	
4	11 (35)	6 (19)	
Risk factors for ARDS, *N* (%)			
Sepsis	3 (10)	2 (6)	$p < 0.001$
Shock	5 (16)	1 (3)	$p < 0.001$
Pneumonia	11 (35)	5 (16)	$p < 0.001$
Gastric aspiration	2 (6)	0 (0)	$p < 0.001$
Transfusion	1 (3)	0 (0)	$p < 0.001$
At least 1 risk factor	15 (48)	6 (16)	$p < 0.02$

ventilated days with tidal volumes $\leq$8 mL/kg in patients before and after ARDS onset (64.1 % before ARDS vs. 66.4 % after ARDS; $p = 0.79$). No significant difference was observed in the number of ventilation days with PEEP $\geq$5 cm H_2O (100 % for both) or peak pressure $<$30 cm H_2O (100 % before ARDS vs. 88.7 % after ARDS; $p = 0.10$).

Injurious tidal volumes were administered at least once during 4 (2.9 %) non-ARDS ventilated days versus 8 (6.1 %) ARDS ventilated days ($p = 0.20$). Peak pressures $>$30 cm H_2O were not observed in any non-ARDS ventilated days, but observed in eight (11.3 %) ARDS ventilated days ($p < 0.01$). The proportion of patients meeting any single or combined lung protective ventilation threshold did not vary significantly when stratified by gender, WFNS grade, or modified Fisher grade (data not shown).

Ventilator Settings and Subsequent Development of ARDS

Ventilation settings did not predict the subsequent development of ARDS. In the patients who did not have ARDS on the first day of mechanical ventilation, mean tidal volume did not differ between patients who went on to develop ARDS compared to those who did not (mean tidal volume prior to development of ARDS 8.1 mL/kg [95 % CI 7.3–8.9] and 7.8 mL/kg [95 % CI 7.4–8.1], respectively; $p = 0.49$). Tidal volume was not associated with the subsequent development of ARDS in univariate analysis ($p = 0.6$) or in multivariate analysis ($p = 0.94$) after adjusting for age, sex APACHE II score, SAH grade, modified fisher grade, and the number of ARDS risk factors. Only the number of ARDS risk factors was independently associated with the subsequent development of ARDS (Adjusted Odds Ratio 2.8 per additional risk factor [95 % CI 1.2–6.5]).

Discussion

In this study of 62 patients receiving early mechanical ventilation following subarachnoid hemorrhage, injurious tidal volumes were rare and lung protective tidal volumes were delivered in approximately two-thirds of patients regardless of whether they met criteria for ARDS. ARDS was common and was not predicted by ventilator parameters. Spontaneous ventilation modes (PS and CPAP) were common in our cohort.

A majority of patients in our cohort were ventilated using spontaneous modes. This contrasts with data from a large cohort in which volume control was the most common mode of mechanical ventilation in brain-injured patients, including those with hemorrhagic stroke [8]. Some of this difference may relate to institutional protocols which favor spontaneous respiration in patients in whom intracranial pressure (ICP) is not elevated and tight CO_2 control is not indicated. Although such an approach allowed a mild respiratory alkalosis (pCO_2 30 mmHg) on Day 1 in our cohort, there are few data to suggest tight CO_2 control, and requisite sedation is of benefit in this patient population outside of emergent ICP control [14].

Table 4 Description of ventilation practices in patients stratified by ARDS status

	Never developed ARDS ($N = 102$ ventilated days)	ARDS patients prior to ARDS ($N = 39$ ventilated days)	ARDS patients with ARDS ($N = 131$ ventilated days)	
Average daily pH, median [IQR]	7.44 [7.41–7.46]	7.43 [7.38–7.47]	7.44 [7.40–7.48]	$p = 0.42$
Average daily CO_2, mmHg median [IQR]	31 [28–34]	31 [27–37]	32 [29–37]	$p = 0.27$
Average daily bicarbonate, mmol/L median [IQR]	21 [19–23]	20 [18–24]	22 [19–25]	$p = 0.13$
Average daily tidal volume $\leq$6 mL/kg, *N* (%)	17 (17)	6 (15)	27 (21)	$p = 0.69$
Average daily tidal volume $\leq$8 mL/kg, *N* (%)	62 (61)	25 (64)	87 (66)	$p = 0.75$
Average daily peak pressure $<$30 cm H_2O, *N* (%)	100(98)	39 (100)	116 (89)	$p = 0.01$
Average daily PEEP $>$5 cm H_2O, *N* (%)	99 (100)	39 (100)	131 (100)	N/A
All lung protective Ventilation criteria, *N* (%)	61 (60)	25 (64)	73 (56)	$p = 0.60$
Injurious ventilation (V_T $>$12 mL/kg), *N* (%)	1 (1)	3 (8)	8 (6)	$p = 0.07$

The rate of ARDS in our cohort is higher than previously documented in patients with SAH [1]. Several reasons may account for this: first, our cohort had a high proportion (59 %) of patients in WFNS grades IV and V, and ARDS is associated with higher WFNS grades [1, 15]. A large proportion of ARDS patients in this study met criteria for ARDS on day 1 of mechanical ventilation, suggesting that these patients required mechanical ventilation due to hypoxia in addition to airway protection on account of depressed neurological function. Although a majority of SAH patients are transferred to the ICU already intubated (without record for its indication), it is conceivable that low GCS and hypoxia were the top indications among this study cohort. Secondly, the retrospective study design did not allow for stringent identification of volume overload or other causes of pulmonary edema. Therefore, patients with neurogenic pulmonary edema or congestive heart failure may have been misclassified as having ARDS. It is reassuring, however, that although ARDS occurred early in our study, this was similar to a previous investigation of ARDS in SAH [1]. Despite the possible influence of cardiogenic and neurogenic pulmonary edema, patients in our cohort had similar ARDS outcomes to other studies with regards to ICU length of stay, duration of mechanical ventilation, and mortality [16]. Interestingly, previous investigations have demonstrated that an elevated arterio-alveolar gradient is associated with at least 30 % mortality in patients with SAH. This is consistent with the 35 % mortality observed among ARDS patients in this study cohort [17].

Clinical evidence supports the use of lung protective ventilation to improve survival in patients with established ARDS [3]. The benefit of lung protective ventilation in patients without ARDS remains less clear. There is strong physiological rationale for restricting tidal volumes in order to avoid high pressures and prevent cyclical collapse with PEEP [18].

Ventilator settings are associated with subsequent development of ARDS in a broad range of patients without ARDS [6], and lung protective ventilation has been associated with improved lung function and organ procurement in organ donors [19]. Lung protective ventilation may require increased use of sedatives in patients on spontaneous breathing modes, however, and it is not clear if the benefit of lung protective ventilation outweighs the risk of increased sedation in patients at lower risk. A recent meta-analysis found improved outcomes with the use of low tidal volume ventilation in patients without ARDS [5]. Our observed median tidal volume of 7.8 mL/kg is consistent with findings from large observational cohort studies [20–22] and suggested practice in recent editorial [23], but still remains higher than those recently reported in a study of ventilation in patients with ARDS [24].

Two-thirds of ventilated patient days that met our ARDS case definition received tidal volumes in the lung protective range. This is consistent with a recent cohort study showing that only 41 % of all eligible ventilator settings in patients with ARDS were adherent to lung protective ventilation [24]. It is interesting to note that another study observed that lung protective ventilation was much less common on the first day of ventilation in patient with hemorrhagic stroke compared to patients with other brain injuries [8]. The obstacles to application of lung protective ventilation are not clear but may include a lack of consistent screening for ARDS or ascribing hypoxemia to other causes (cardiogenic or neurogenic pulmonary edema). It is also possible that clinicians were wary of permissive hypercapnia in the face possible raised intracranial pressure. We did not collect data on ICP, although utilization of lung protective ventilation did not vary across severity of brain

injury as documented by WFNS grade or modified Fisher grade.

Although adherence to peak pressures less than 30 cm H_2O was observed in 94 % of our observations, this metric must be interpreted with caution because a majority of patients were breathing spontaneously. Spontaneous respiratory efforts on positive pressure ventilation add to the transpulmonary pressure (the difference of the alveolar and intrapleural pressure). Because we were unable to measure the patient's own respiratory efforts, the peak and plateau pressures as measured at the ventilator likely underestimate the true transpulmonary pressure, or distending pressure. Consequently, safe peak or plateau pressures do not preclude potentially injurious pressures being applied to the lungs by the combination of the ventilator and the patient's own efforts. It is unclear, however, if higher tidal volumes or transpulmonary pressures generated spontaneously by a patient's efforts are as injurious as those applied by positive pressure ventilation.

We did not find a relationship between initial ventilator parameters and the subsequent development of ARDS, contrary to several other studies [4, 25]. It is possible our sample size was too small to allow detection of an independent association between ventilator parameters and development of ARDS and this statistical power may have been reduced by misclassification of ARDS. Assuming the most conservative scenario with an alpha level of 0.05, our sample size would allow us 80 % power to detect a difference of 24, or 60 % power to detect a difference of 20 % in the incidence of ARDS between the two groups. While our data do not suggest a large association between initial ventilator settings and the subsequent development of ARDS, our sample size precludes detection of a small effect size. Our study does suggest, however, that SAH patients with an additional ARDS risk factor are at high risk of developing ARDS and require close screening for this complication, along with careful consideration of ventilation practices targeting lung protective thresholds. This may prevent the onset of ARDS and may improve clinical outcomes in patients without ARDS [5].

There are several limitations to our study. We were not able to rigorously collect data on left ventricular filling pressures or clinical signs of heart failure due to the retrospective design. Our institution does not routinely insert pulmonary artery catheters, and although echocardiograms are routinely performed in patients with subarachnoid hemorrhage, although these data do not reliably inform clinicians about the presence of elevated pulmonary capillary wedge pressures, especially in patients with preserved ventricular function [26]. We excluded patients admitted to ICU greater than 72 h after hospital admission because we wished to explore ventilation strategies early in SAH and thus we may have excluded patients with better WFNS grades but subsequently required mechanical ventilation due to neurological or respiratory deterioration. It is possible that performance in applying lung protective ventilation to patients transferred into the ICU with respiratory failure may have been better as physicians would be more vigilant in looking for ARDS. However, the use of local ventilation protocols, consistent staffing, and ventilator management would make it unlikely that the ventilator management in these patients was significantly different from those patients admitted to the ICU with a primary diagnosis of SAH. Finally, our study was a single centre study, which may limit our ability to generalize our findings concerning the impact of lung protective ventilation.

Conclusions

In conclusion, SAH patients requiring mechanical ventilation frequently breathe spontaneously, often generating large tidal volumes. Currently accepted thresholds for lung protective ventilation were not met in approximately one-thirds of patient days with ARDS suggesting that this is a target for quality improvement. Although we could not identify an independent association between ventilator settings and subsequent development of ARDS, the presence of an additional ARDS risk factor in patients with SAH should prompt screening for the onset of ARDS and consideration for mechanical ventilation targeting lung protective thresholds. The association between high tidal volumes in spontaneous ventilation modes and clinical outcomes including the subsequent development of ARDS requires further study.

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Conflict of interest The authors declare that they have no competing interests.

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