

A Description of Intraoperative Ventilator Management and Ventilation Strategies in Hypoxic Patients

James M. Blum, MD,* Douglas M. Fetterman, MD,* Pauline K. Park, MD,† Michelle Morris, MS,* and Andrew L. Rosenberg, MD*

BACKGROUND: Hypoxia is a common finding in the anesthetized patient. Although there are a variety of methods to address hypoxia, it is not well documented what strategies are used by anesthesiologists when faced with a hypoxic patient. Studies have identified that lung protective ventilation strategies have beneficial effects in both oxygenation and mortality in acute respiratory distress syndrome. We sought to describe the ventilation strategies in anesthetized patients with varying degrees of hypoxemia as defined by the P_{aO_2} to fraction of inspired oxygen (F_{iO_2}) (P/F) ratio.

METHODS: We conducted a review of all operations performed between January 1, 2005, and July 31, 2009, using a general anesthetic, excluding cardiac and thoracic procedures, to assess the ventilation settings that were used in patients with different P/F ratios. Patients older than 18 years who received a general anesthetic were included. Four cohorts of arterial blood gases (ABGs) were identified with $P/F > 300$, $300 \geq P/F > 200$, $200 \geq P/F > 100$, $100 \geq P/F$. Using the standard predicted body weight (PBW) equation, we calculated the milliliters per kilogram (mL/kg PBW) with which the patient's lungs were being ventilated. Positive end-expiratory pressure (PEEP), peak inspiratory pressures (PIPs), F_{iO_2} , oxygen saturation (S_{aO_2}), and tidal volume in mL/kg PBW were compared.

RESULTS: A total of 28,706 ABGs from 11,445 operative cases met criteria for inclusion. There were 19,679 ABGs from the $P/F > 300$ group, 5364 ABGs from the $300 \geq P/F > 200$ group, 3101 ABGs from the $200 \geq P/F > 100$ group, and 562 ABGs from the $100 \geq P/F$ group identified. A comparison of ventilation strategies found statistical significance but clinically irrelevant differences. Tidal volumes ranged between 8.64 and 9.16 and the average PEEP varied from 2.5 to 5.5 cm H_2O . There were substantial differences in the average F_{iO_2} and PIP among the groups, 59% to 91% and 22 to 29 cm H_2O , respectively.

CONCLUSION: Similar ventilation strategies in mL/kg PBW and PEEP were used among patients regardless of P/F ratio. The results of this study suggest that anesthesiologists, in general, are treating hypoxemia with higher F_{iO_2} and PIP. The average F_{iO_2} and PIP were significantly escalated depending on the P/F ratio. (Anesth Analg 2010;110:1616–22)

Mechanical ventilation during anesthesia provides for necessary gas exchange, airway control, and a conduit for inhaled anesthetic delivery. Nevertheless, mechanical ventilation is not without risks, including barotrauma (lung injury from high pressure), atelectrauma (lung injury from shear forces on compressed alveoli), and volutrauma (lung injury from excessive volumes).¹ These complications can occur even after relatively short durations of use in the operating room (OR), particularly when anesthesiologists are attempting to treat intraoperative hypoxemia and decreasing P_{aO_2} /fraction of inspired

oxygen (F_{iO_2}) (P/F) ratios, which are conditions common to patients receiving general anesthesia.² Because atelectasis is believed to be the most common contributing factor, adjustment of tidal volumes (V_T) to 10 to 12 mL/kg³ and increasing the F_{iO_2} and positive end-expiratory pressure (PEEP)⁴ have been traditionally advocated for treating relative hypoxemia in the operative setting. In contrast, in intensive care unit (ICU) patients, a subset of patients with hypoxia actually have acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) and management of hypoxia is then based on a lung protective ventilation strategy (LPVS), minimizing additional ventilator-associated lung injury using lower V_T .^{5,6} Along with the low V_T LPVS, aggressive PEEP and recruitment maneuvers can be beneficial in improving oxygenation while reducing the perceived potential toxicity of high-concentration oxygen and reducing alveolar shear stress.⁷

We sought to examine the current management of hypoxemic patients in the OR to determine the frequency at which LPVS is implemented in response to a low P/F ratio and to determine whether other methods were preferentially used to address hypoxia. Using a large, prospectively

From the Departments of *Anesthesiology, and †Surgery, University of Michigan, Ann Arbor, Michigan.

Accepted for publication February 2, 2010.

Address correspondence and reprint requests to James M. Blum, MD, Department of Anesthesiology and Critical Care, The University of Michigan Health Systems, 4172 Cardiovascular Center/SPC 5861, 1500 East Medical Center Dr., Ann Arbor, MI 48109-5861. Address e-mail to jmbalum@umich.edu.

Copyright © 2010 International Anesthesia Research Society
DOI: 10.1213/ANE.0b013e3181da82e1

collected, multi-year set of automatically acquired operative data, we examined the ventilatory variables used in the operative setting. This methodology is devoid of the potential Hawthorne effect and provides insight into the actual modalities that are frequently used by anesthesiologists in patients with a wide range of P/F ratios. Specifically, we evaluated the use of PEEP, peak inspiratory pressures (PIPs) and FiO_2 . We hypothesized that, compared with patients with P/F ratios >300 mm Hg, intraoperative ventilator management of patients with lower P/F would demonstrate significantly lower V_T , no changes in PIP, and higher PEEP levels, similar to what might be demonstrated in ICU ventilator management for hypoxemic patients.

METHODS

IRB approval was obtained for this retrospective cohort study at the University of Michigan Medical Center, a large, tertiary care facility. We examined all operations performed between January 1, 2005, and July 31, 2009, during which at least 1 arterial blood gas (ABG) determination was made. We excluded all cases in which intraoperative ventilator management and/or ABGs are routinely affected by protocolized care for single lung ventilation or cardiopulmonary bypass as well as patients in whom ventilator management is potentially different because of extremes of height and weight where height was not between 60 and 80 in. and body mass index was not between 15 and 60 kg/m^2 .

Preoperative data were collected from routine clinical documentation that was entered into the institutional anesthetic information system (Centricity, General Electric Healthcare, Barrington, IL). The record includes a structured preoperative history and physical examination allowing for coded entry and free text where required. Data obtained from the preoperative history included age, gender, ASA physical status, surgical service, and height. Predicted body weight (PBW) was calculated as whole values using the formula $50 + 2.3 (\text{height [in.]} - 60)$ for men and $45.5 + 2.3 (\text{height [in.]} - 60)$ for women.

The unit of measure was an individually obtained ABG. We chose to use the ABG as the unit of measure because each ABG provided information for the anesthesiologist to adjust ventilator settings, which conceivably could change multiple times through a case. Each patient had 1 to 18 ABGs. The blood gas values were manually entered by the anesthetic team into the structured electronic intraoperative record. Intraoperative physiologic and ventilator data were acquired using an automated, validated electronic interface from the anesthesia machine (ASIS, General Electric Healthcare, Waukesha, WI) and physiologic monitors (Solar 9500, General Electric Healthcare). FiO_2 , PIP, V_T , PEEP, SpO_2 , and end-tidal (ET) CO_2 were collected every minute for 15 minutes after a documented ABG. These data were used to calculate median values for each variable to eliminate spurious and isolated data. From the recorded Pao_2 and median FiO_2 , the P/F ratio was calculated for each ABG. The mL/kg PBW was calculated for each ABG from the median V_T and the PBW.

ABG values with $40 \text{ mm Hg} > \text{Pao}_2 > 600 \text{ mm Hg}$, $20 \text{ mm Hg} > \text{Paco}_2 > 110 \text{ mm Hg}$, $6.99 > \text{pH} > 7.99$, $0.21 > \text{FiO}_2 > 1.00$, associated with a median $20 \text{ mm Hg} > \text{ETCO}_2 > 110 \text{ mm Hg}$, P/F ratio >600 , or a combined $\text{Pao}_2 < 55 \text{ mm Hg}$ with a median SpO_2 value $>93\%$ were excluded.

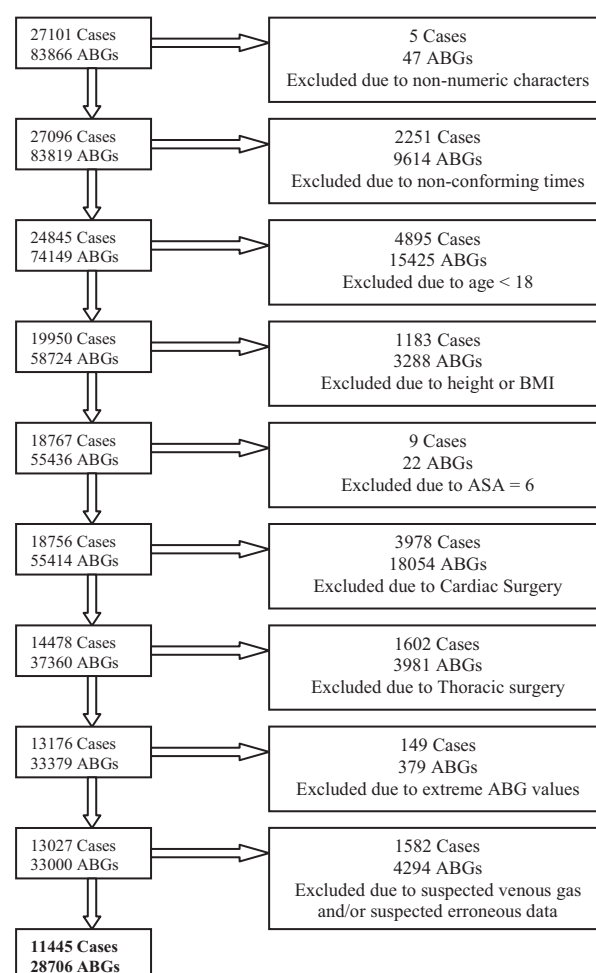


Figure 1. Method of case exclusion: arterial blood gas (ABG) values with $40 \text{ mm Hg} > \text{Pao}_2 > 600 \text{ mm Hg}$, $20 \text{ mm Hg} > \text{Paco}_2 > 110 \text{ mm Hg}$, $6.99 > \text{pH} > 7.99$, $0.21 > \text{fraction of inspired oxygen (FiO}_2) > 1.00$, associated with a median $20 \text{ mm Hg} > \text{end-tidal (ET)CO}_2 > 110 \text{ mm Hg}$, $\text{Pao}_2/\text{FiO}_2$ ratio >600 , or a combined $\text{Pao}_2 < 55 \text{ mm Hg}$ with a median SpO_2 value $>93\%$ were excluded.

Hg with a median SpO_2 value $>93\%$ were excluded because of the high probability of the gas being of venous origin or having erroneous data entry. ABGs associated with $\text{PIP} < 5 \text{ cm H}_2\text{O}$ or $\text{V}_T < 100 \text{ mL}$ were excluded because of potential spontaneous respirations or pressure support ventilation. ABGs from patients with ASA physical status VI were excluded because the ventilation strategy implemented may have been designed to preserve other organs. ABGs with abnormal characters were also removed from the dataset. Cases were validated as having started by using electronically documented start (incision or induction end) times and end (anesthesia end, patient transported from room, or dressing complete) times. ABGs from cases <30 minutes in duration or with nonconforming times (documented outside of the anesthesia start or end times, or from cases with undocumented anesthesia start or end times) were excluded.

Statistical Analysis

Statistical analysis was performed using SPSS version 17 (SPSS, Chicago, IL). Patients were divided into 4 groups by

lowest recorded P/F ratio during the case: ≥ 300 , 200 to 299, 100 to 200, and those ≤ 100 representing normal lung, mild hypoxia, moderate hypoxia, and severe hypoxia, respectively. The mean values and SD for each group including mL/kg PBW, PIP, PEEP, F_{IO_2} , P_{aO_2} , P_{aCO_2} , pH, age, ASA physical status, and PBW were calculated. The groups were compared using either the Student *t* test for normalized distributions or the Mann-Whitney test for nonnormalized distribution of the data. Multiple comparisons between rows were analyzed using analysis of variance and the Bonferroni correction was applied. $P < 0.05$ was considered statistically significant.

Table 1. Demographics of Cohort and Distribution of ABGs by Surgical Service

	Mean	Minimum	Maximum	SD
Age (y)	57.18	18	103	15.62
ASA physical status	2.86	1	5	0.73
Weight (kg)	84.90	35	215	22.39
Height (in.)	67.42	60	80	3.93
PBW	65.26	46	96	10.70
OR time (min)	382.00	30	2191	204.92
Service	Operative cases		ABGs	
Neurosurgery	2260	19.58%	5890	20.52%
General surgery and subspecialties	1849	16.02%	4412	15.37%
Vascular	1497	12.97%	3745	13.05%
Orthopedic	1333	11.55%	2635	9.18%
Otolaryngology/maxillofacial	1321	11.45%	4011	13.97%
Urology	990	8.58%	2246	7.82%
Transplant	799	6.92%	2801	9.76%
Trauma	565	4.89%	1097	3.82%
Other/combined/unknown	428	3.71%	969	3.38%
OB/gyn	294	2.55%	515	1.79%
Plastic	205	1.78%	385	1.34%
Total	11,541	100%	28,706	100%

OR = operating room.

RESULTS

A total of 27,101 operative cases in which at least 1 ABG was acquired during the case were identified. In these cases, 83,866 ABGs were obtained. Operative cases and ABGs that were excluded are shown in Figure 1. Inclusion criteria were met by 28,706 ABG values from 11,445 operative cases. Of the samples taken, 58% were from male patients with an average age of 57 years. Table 1 shows the demographic and surgical services included in the analysis.

The 11,445 operative cases meeting criteria were divided into groups based on the lowest P/F ratio seen during the case: 54% of the total cases had lowest P/F ratios >300 ; 42% of the cases had lowest P/F ratios <300 and >100 ; and 4% had lowest P/F ratios ≤ 100 at some point during the case. Table 2 displays the average and SD of the ventilatory settings and demographics of each group of patients. Bar graphs shown in Figure 2 were generated illustrating the V_T , PIP, PEEP, and F_{IO_2} used in each group for ventilation. The average V_T observed in all cases was 9.07 mL/kg PBW with an average PEEP of 3.13 cm H₂O. Even when faced with P/F ratios <100 , anesthesiologists made minimal changes in ventilation management ($V_T = 8.64$ mL/kg PBW, PEEP = 5.48 cm H₂O).

For patients who had >1 ABG during their operation, we sought to determine whether the presence of hypoxemia was associated with subsequent changes in a given patient's ventilator-delivered V_T . Within each subgroup, we analyzed the changes that were made in ventilatory settings at the beginning and end of their OR course. The average changes in ventilation for each group are shown in Table 3. Four hundred ninety-four cases (14%) with P/F ratios <300 were found to have decreases in their V_T of at least 1 mL/kg PBW during their anesthetic course, and 672 cases (19%) were found to have increases of at least 1 mL/kg PBW. In the lowest P/F group, 25% had an increase in F_{IO_2} of at least 5%, 16% had decreased mL/kg PBW of at

Table 2. Mean and SD Where N= the Number of ABGs

	Normal		Mild hypoxia		Moderate hypoxia		Severe hypoxia	
	P/F >300 (N = 19,679)		300 \geq P/F > 200 (N = 5364)		200 \geq P/F > 100 (N = 3101)		100 \geq P/F (N = 562)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (y)	56.94	16.04	58.64*	14.27	56.70	15.12	54.16*	16.01
ASA physical status	2.80	0.73	2.95*	0.71	2.98*	0.74	3.27*	0.87
Height (in.)	67.28	3.91	67.76*	3.96	67.63*	4.04	68.01*	4.08
Weight (kg)	81.38	20.54	91.75*	23.20	93.60*	25.41	94.56*	27.11
Predicted body weight	64.88	10.61	66.19*	10.74	65.83*	10.98	66.71*	11.06
P/F ratio	427.86	73.44	253.84*	28.48	158.15*	28.21	81.51*	12.63
P_{aO_2} (mm Hg)	249.61	101.13	149.94*	54.03	103.85*	31.38	74.13*	12.21
F_{IO_2} (%)	58.46	21.54	59.21	20.45	66.75*	19.37	91.43*	9.63
PEEP (cm H ₂ O)	2.86	2.30	3.40*	2.56	3.98*	2.95	5.48*	3.63
V_T (mL/kg PBW)	9.05	1.80	9.16*	1.89	9.10	1.97	8.64*	2.17
PIP (cm H ₂ O)	22.18	5.54	24.41*	6.04	26.08*	6.40	28.82*	6.71
P_{aCO_2} (mm Hg)	36.34	4.94	38.21*	5.63	39.93*	6.55	44.79*	9.44
pH	7.43	0.06	7.41*	0.07	7.40*	0.07	7.36*	0.10
Sp_{O_2} (%)	99.29	1.41	98.57*	1.87	97.93*	2.22	96.20*	4.02
Actual V_T (mL)	578.72	112.16	597.49*	117.95	591.46*	129.17	568.17	140.41
ET CO_2 (mm Hg)	32.99	3.58	33.42*	3.89	33.55*	4.51	33.95*	5.91

PEEP = positive end-expiratory pressure; V_T = tidal volume; PIP = peak inspiratory pressures; ABG = arterial blood gas.

* $P \leq 0.001$ when compared with the group where P/F >300 using ANOVA with the Bonferroni correction.

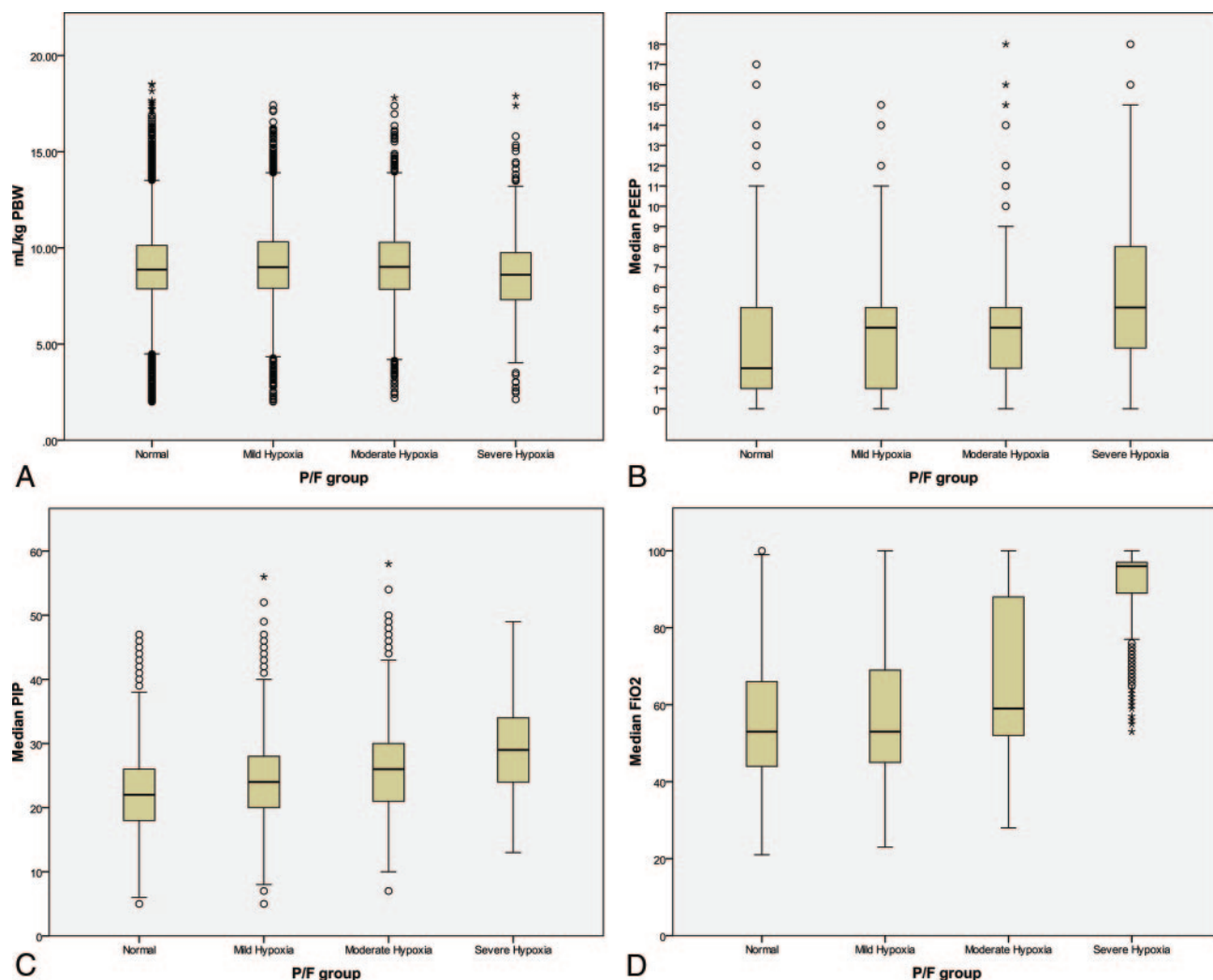


Figure 2. A–D, Distribution of tidal volumes by Pao_2 /fraction of inspired oxygen (F_{iO_2}) (P/F) group. A, Milliliters of ventilation per kilogram predicted body weight (PBW) by hypoxic group. B, Median positive end-expiratory pressure (PEEP) by hypoxic group. C, Median peak inspiratory pressure by hypoxic group. D, Median F_{iO_2} by hypoxic group. Solid lines = values; boxes = interquartile range; T bars = 95% of total sample; ○ = outliers ($1.5-3 \times$ box length). *Extreme outliers ($>3 \times$ box length).

Table 3. Comparison of Ventilator Strategies at the Beginning and End of the Operation

	Normal		Mild hypoxia		Moderate hypoxia		Severe hypoxia	
	P/F > 300 (N = 3659)		300 ≥ P/F > 200 (N = 1858)		200 ≥ P/F > 100 (N = 1308)		100 ≥ P/F (N = 288)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Earliest mL/kg PBW	8.91	1.76	9.07	1.88	9.07	1.88	8.65	1.96
Latest mL/kg PBW	9.01*	1.85	9.21*	1.89	9.14	2.07	8.78	2.16
Earliest PEEP (cm H_2O)	2.48	2.19	2.79	2.37	3.36	2.76	4.64	3.51
Latest PEEP (cm H_2O)	2.81*	2.21	3.28*	2.37	4.14*	2.74	5.92*	3.64
Earliest F_{iO_2} (%)	60.78	21.56	59.47	20.66	64.34	19.86	84.07	17.74
Latest F_{iO_2} (%)	57.42*	21.92	57.74*	21.24	65.23	20.75	83.97	18.51

First and last ABGs for patients with multiple ABGs drawn and corresponding ventilatory parameters.

PBW = predicted body weight; PEEP = positive end-expiratory pressure.

* $P \leq 0.001$ when compared with earliest value using the t test.

least 1 mL/kg PBW, and 32% had an increase in PEEP of at least 2 cm H_2O .

Ventilation strategies used to treat critically ill patients have changed substantially during the past several years.^{8,9} In an effort to determine whether this has had an effect on

the ventilation strategies used in the OR, we examined each year of cases for the fundamental ventilator settings being used on each subset of patients. The results of this analysis are shown in Table 4. Overall, there was a trend toward lower V_T , lower PIP, and higher levels of PEEP over the 5

Table 4. Changes in Ventilation by Calendar Year

Year	Normal		Mild hypoxia		Moderate hypoxia		Severe hypoxia		Total	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Average tidal volumes by hypoxia group in mL/kg pbw										
2005	9.35	1.98	9.51	2.03	9.55	2.20	9.73	2.60	9.41	2.03
2006	9.51*	1.95	9.82*	2.11	9.60	2.04	8.55*	2.20	9.56*	2.00
2007	9.06*	1.78	9.13*	1.82	9.14*	1.84	8.81†	2.16	9.08*	1.80
2008	8.82*	1.60	8.91*	1.71	8.94*	1.85	8.57*	1.81	8.85*	1.66
2009	8.60*	1.62	8.64*	1.67	8.42*	1.76	7.78*	1.89	8.57*	1.66
Total	9.05	1.80	9.16	1.90	9.10	1.97	8.64	2.17	9.07	1.85
Average peak inspiratory pressures by hypoxia group in cm H ₂ O										
2005	25.38	4.99	27.52	5.70	28.91	5.80	31.62	6.40	26.33	5.47
2006	25.61	5.02	27.78	5.49	29.31	5.98	31.69	6.59	26.47	5.44
2007	21.28*	5.32	23.69*	5.91	25.52*	6.65	29.91	6.57	22.34*	5.90
2008	20.11*	4.80	22.35*	5.55	24.00*	5.74	26.13*	6.08	21.10*	5.33
2009	19.99*	4.73	22.50*	5.21	24.27*	5.82	26.63*	5.97	21.06*	5.26
Total	22.18	5.54	24.41	6.04	26.08	6.40	28.81	6.72	23.15	5.97
Average PEEP by hypoxia group in cm H ₂ O										
2005	2.04	1.37	2.43	1.73	2.86	2.07	4.04	2.93	2.25	1.63
2006	2.12	1.34	2.52	1.78	3.06*	2.20	4.40	3.15	2.32*	1.63
2007	2.38*	2.59	2.99*	2.85	3.69*	3.27	5.88*	3.95	2.70*	2.82
2008	3.42*	2.54	4.03*	2.74	4.64*	3.01	6.03*	3.58	3.73*	2.72
2009	4.06*	2.25	4.57*	2.41	5.14*	3.11	6.21*	3.70	4.32*	2.47
Total	2.86	2.30	3.40	2.56	3.98	2.95	5.47	3.63	3.13	2.51

PBW = predicted body weight.

* $P \leq 0.05$, † $P \leq 0.001$ when compared with the 2005 group using ANOVA with the Bonferroni correction.

calendar years of data examined. In the severe hypoxia group, V_T decreased by 2 mL/kg PBW, PIP decreased by 5 cm H₂O, and PEEP increased by >2 cm H₂O.

Finally, because actual body weight (ABW) is often incorrectly substituted for PBW used in LPVS, we calculated the mL/kg of V_T based on ABW. Overall, we found on average 7.15 (SD = 1.66) mL/kg ABW delivered to patients with V_T of 6.34 (SD = 1.88) mL/kg ABW in the severe P/F grouping.

DISCUSSION

In this study of >11,000 anesthetics, there seems to be minimal clinical differences (average V_T 8.6–9.16 mL/kg PBW; PEEP 2.8–5.5 cm H₂O) in how anesthesiologists manage mechanical ventilation in patients with intraoperative hypoxemia compared with those with normal oxygenation. Only in the group of patients with P/F ratios <100 was the V_T delivered significantly lower (9.16 vs 8.6 mL/kg PBW, $P < 0.001$) than those delivered to patients with P/F >300. Our findings suggest that even in severely hypoxic patients, who may have ALI/ARDS, V_T in mL/kg PBW was maintained at a higher level than the 6 mL/kg PBW recommended by an LPVS.^{6,10}

In general, the response by the anesthesiologist to hypoxia was to increase F_{IO_2} , tolerate increased peak airway pressures, and target an $ETCO_2$ of approximately 34 mm Hg. It seems that anesthesiologists limited PIP to <30 cm H₂O, suggesting that attempts to prevent barotrauma may be at the forefront of management with less attention given to volutrauma and atelectrauma. The calendar year analysis shown in Table 4 suggests that anesthesiologists are continuing to decrease V_T (9.4–8.6 mL/kg PBW, $P < 0.001$), decrease PIP (26.3–21.1 cm H₂O, $P < 0.001$), and increase

PEEP (2.25–4.32 cm H₂O, $P < 0.001$), particularly in those patients with severe hypoxia. However, it seems that these changes are not nearly as dramatic in the mild and moderate hypoxia groups for whom a diagnosis of ALI/ARDS could still be considered.

Ventilator management in the OR by anesthesiologists is an integral part of an anesthetic and is receiving increased scrutiny. It is important to consider the literature from the critical care arena supporting the use of LPVS among patients with ALI.^{6,7,11–14} There is a growing body of evidence to suggest that the traditional 10 mL/kg ABW ventilation strategy may be inappropriately high for a large population of patients receiving mechanical ventilation. Several studies in thoracic anesthesia involving low V_T management have shown benefits in outcome,^{15–17} whereas in cardiac surgery patients, the same benefits were not seen.^{18,19} Nevertheless, intraoperative ventilation using high V_T has shown worsened clinical outcome and an increase in biomarkers indicating subclinical injury in previously healthy lungs.^{17,20–22} In 2006, Fernandez-Perez et al.¹⁵ evaluated intraoperative ventilation in pneumonectomy patients. Larger intraoperative V_T values, 8.3 vs 6.7 mL/kg PBW, were correlated with increased respiratory failure and increased hospital mortality after 60 days. Likewise, Michelet et al.¹⁷ showed that a reduction in V_T during one-lung ventilation reduced the duration of postoperative ventilation. Recently, it was also shown that PIP >30 cm H₂O was associated with increased postoperative respiratory failure.²³

We found the use of higher levels of PEEP to be modest in hypoxic patients compared with those used in the ARMA trial in which PEEP values could exceed 20 cm H₂O

by protocol when patients were receiving 100% Fio_2 .⁶ Increasing PEEP increases mean airway pressures, reduces atelectasis, and increases oxygenation while limiting the potentially toxic effects of high concentration oxygen and the effect of denitrogenation, which may actually increase atelectasis-induced shunt.^{24–26} Although there are technical and hemodynamic reasons that high levels of PEEP may be contraindicated during surgery, the average PEEP among patients with the lowest P/F ratios was still <6 cm H_2O . Because average Spo_2 remained $>95\%$, even in cases with lower P/F ratios, the severity of the change in alveolar-arterial gradient may have been underestimated by the practitioners.

One could claim that there was no life-threatening hypoxia in these situations because patients maintained reasonable oxyhemoglobin saturations in most cases. The data also display that, as patients became more hypoxic, they seemed to have less-compliant lungs as the PIP continued to increase in each group, with Vt values that were clinically quite similar, although statistically different. It is also conceivable that the anesthesiologists in many of these cases avoided making ventilatory changes in a patient with unstable hemodynamics or were concerned with hyperinflation.²⁷ It is also possible that, in general, anesthesiologists are not aware of or are not practicing more nuanced ICU ventilator management in the OR. It is a key component in patient management for LPVS to calculate the PBW, and many of the ventilation decisions may have been based on ABW (based on our data) because Vt values were very close to 6 mL/kg ABW in patients with severe hypoxia.

Several risk factors for the development of ALI are encountered in the OR, including transfusion of blood products, large-volume fluid administration, acidemia, history of restrictive lung disease, history of alcohol use, thoracic surgery, sepsis, trauma, peak pressures >30 cm H_2O , and large Vt .^{15,16,23,28} Therefore, we should not ignore the possibility of ALI/ARDS beginning in the setting of anesthesia. Considering the fact that both high Vt and high peak pressures have been associated with ALI developing under general anesthesia, it would seem reasonable to consider that LPVS may benefit patients.

This study has several limitations. First, the data were collected during normal clinical care. The data were from the electronic anesthetic record, and no additional details were available. There were no rigorous processes to validate the entry of data, although the use of automated collection of physiologic data has been accepted in many previous studies. Second, the data are from a single tertiary care center, which may not serve as a representative sample of patients throughout the world. Certain patients undergoing procedures limiting oxygenation, such as orthopedic thoracic spine surgery requiring double-lumen tube placement and single-lung ventilation, would not have been excluded by criteria used in the study. The dataset available to us, although providing substantial investigative power, was limited in recording other key ventilatory variables. For example, the dataset does not provide the choice of ventilator mode (volume control or pressure control), plateau pressures, or the use of inverse ratio ventilation. There

is also no modality to confirm the use of recruitment maneuvers. Finally, those patients who were most hypoxic were potentially ventilated in the OR with an ICU ventilator and would have been excluded because of lack of electronic data collection of ventilator data.

In conclusion, in patients with mild to moderate hypoxia ($100 < \text{P/F} < 300$), we observed the primary mode of ventilation to be unchanged from nonhypoxic patients ($\text{P/F} > 300$). In patients with at least 1 ABG demonstrating severe hypoxia ($\text{P/F} < 100$), the most common ventilator management by the anesthesiologist was to use a higher Fio_2 , tolerate higher peak airway pressures, and target an ETCO_2 of approximately 34 mm Hg. There was little evidence that ALI/ARDS was considered in the differential diagnosis or that LPVS was used. Because intraoperative ventilator settings may affect either the treatment of existing ARDS or the development of ARDS, anesthesiologists should consider both when managing intraoperative hypoxia. ■■

REFERENCES

1. Slinger P. Perioperative lung injury. *Best Pract Res Clin Anaesthesiol* 2008;22:177–91
2. Strachan L, Noble DW. Hypoxia and surgical patients—prevention and treatment of an unnecessary cause of morbidity and mortality. *J R Coll Surg Edinb* 2001;46:297–302
3. Cai H, Gong H, Zhang L, Wang Y, Tian Y. Effect of low tidal volume ventilation on atelectasis in patients during general anesthesia: a computed tomographic scan. *J Clin Anesth* 2007;19:125–9
4. Chapman MJ, Myburgh JA, Kluger MT, Runciman WB. Crisis management during anaesthesia: pulmonary oedema. *Qual Saf Health Care* 2005;14:e8
5. Dos Santos CC. Advances in mechanisms of repair and remodelling in acute lung injury. *Intensive Care Med* 2008;34:619–30
6. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. *N Engl J Med* 2000;342:1301–8
7. Gattinoni L, Vagginelli F, Chiumello D, Taccone P, Carlesso E. Physiologic rationale for ventilator setting in acute lung injury/acute respiratory distress syndrome patients. *Crit Care Med* 2003;31:S300–4
8. Epstein SK. Weaning from ventilatory support. *Curr Opin Crit Care* 2009;15:36–43
9. Tang SS, Redmond K, Griffiths M, Ladas G, Goldstraw P, Dusmet M. The mortality from acute respiratory distress syndrome after pulmonary resection is reducing: a 10-year single institutional experience. *Eur J Cardiothorac Surg* 2008;34:898–902
10. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kairalla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CR. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. *N Engl J Med* 1998;338:347–54
11. Meade MO, Cook DJ, Guyatt GH, Slutsky AS, Arabi YM, Cooper DJ, Davies AR, Hand LE, Zhou Q, Thabane L, Austin P, Lapinsky S, Baxter A, Russell J, Skrobik Y, Ronco JJ, Stewart TE. Ventilation strategy using low tidal volumes, recruitment maneuvers, and high positive end-expiratory pressure for acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. *JAMA* 2008;299:637–45
12. Brochard L, Roudot-Thoraval F, Roupie E, Delclaux C, Chastre J, Fernandez-Mondejar E, Clementi E, Mancebo J, Factor P, Matamis D, Ranieri M, Blanch L, Rodi G, Mentec H, Dreyfuss D, Ferrer M, Brun-Buisson C, Tobin M, Lemaire F. Tidal volume reduction for prevention of ventilator-induced lung injury in acute respiratory distress syndrome. The Multicenter Trial Group on Tidal Volume Reduction in ARDS. *Am J Respir Crit Care Med* 1998;158:1831–8

13. Brower RG, Shanholtz CB, Fessler HE, Shade DM, White P Jr, Wiener CM, Teeter JG, Dodd-o JM, Almog Y, Piantadosi S. Prospective, randomized, controlled clinical trial comparing traditional versus reduced tidal volume ventilation in acute respiratory distress syndrome patients. *Crit Care Med* 1999;27:1492–8
14. Brower RG, Lanken PN, MacIntyre N, Matthay MA, Morris A, Ancukiewicz M, Schoenfeld D, Thompson BT. Higher versus lower positive end-expiratory pressures in patients with the acute respiratory distress syndrome. *N Engl J Med* 2004;351:327–36
15. Fernandez-Perez ER, Keegan MT, Brown DR, Hubmayr RD, Gajic O. Intraoperative tidal volume as a risk factor for respiratory failure after pneumonectomy. *Anesthesiology* 2006;105:14–8
16. Licker M, de Perrot M, Spiliopoulos A, Robert J, Diaper J, Chevalley C, Tschoop JM. Risk factors for acute lung injury after thoracic surgery for lung cancer. *Anesth Analg* 2003;97:1558–65
17. Michelet P, D'Jo XB, Roch A, Doddoli C, Marin V, Papazian L, Decamps I, Bregeon F, Thomas P, Auffray JP. Protective ventilation influences systemic inflammation after esophagectomy: a randomized controlled study. *Anesthesiology* 2006;105:911–9
18. Wrigge H, Uhlig U, Baumgarten G, Menzenbach J, Zinserling J, Ernst M, Dromann D, Welz A, Uhlig S, Putensen C. Mechanical ventilation strategies and inflammatory responses to cardiac surgery: a prospective randomized clinical trial. *Intensive Care Med* 2005;31:1379–87
19. Wrigge H, Uhlig U, Zinserling J, Behrends-Callsen E, Ottersbach G, Fischer M, Uhlig S, Putensen C. The effects of different ventilatory settings on pulmonary and systemic inflammatory responses during major surgery. *Anesth Analg* 2004;98:775–81
20. Choi G, Wolthuis EK, Bresser P, Levi M, van der Poll T, Dzoljic M, Vroom MB, Schultz MJ. Mechanical ventilation with lower tidal volumes and positive end-expiratory pressure prevents alveolar coagulation in patients without lung injury. *Anesthesiology* 2006;105:689–95
21. Gajic O, Dara SI, Mendez JL, Adesanya AO, Festic E, Caples SM, Rana R, St Sauver JL, Lymp JF, Afessa B, Hubmayr RD. Ventilator-associated lung injury in patients without acute lung injury at the onset of mechanical ventilation. *Crit Care Med* 2004;32:1817–24
22. Reis Miranda D, Gommers D, Struijs A, Dekker R, Mekel J, Feelders R, Lachmann B, Bogers AJ. Ventilation according to the open lung concept attenuates pulmonary inflammatory response in cardiac surgery. *Eur J Cardiothorac Surg* 2005;28:889–95
23. Fernandez-Perez ER, Sprung J, Afessa B, Warner DO, Vachon CM, Schroeder DR, Brown DR, Hubmayr RD, Gajic O. Intraoperative ventilator settings and acute lung injury after elective surgery: a nested case control study. *Thorax* 2009;64:121–7
24. Slinger P. Perioperative lung injury. *Best Pract Res Clin Anaesthesiol* 2008;22:177–91
25. Houde J, Clavet M, Jacques A, Pelletier J, Dry R. Alveolar collapse induced by denitrogenation. *Can Anaesth Soc J* 1965;12:531–57
26. Rothen HU, Hedenstierna G. Atelectasis formation during anesthesia: causes and measures to prevent it. *J Clin Monit Comput* 2000;16:329–35
27. Brochard L, Lemaire F, Vasile N, Anglade MC, Mollet JJ, Roupie E, Dambrosio M. Effects of positive end-expiratory pressure and different tidal volumes on alveolar recruitment and hyperinflation. *Anesthesiology* 1997;87:495–503
28. Gajic O, Frutos-Vivar F, Esteban A, Hubmayr RD, Anzueto A. Ventilator settings as a risk factor for acute respiratory distress syndrome in mechanically ventilated patients. *Intensive Care Med* 2005;31:922–6