Cardiopulmonary effects of positive end-expiratory pressure during one-lung ventilation in anesthetized dogs with a closed thoracic cavity

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Objective—To evaluate the effects on oxygen delivery (Do_2) of 2.5 and 5 cm H_2O of positive end-expiratory pressure (PEEP) applied to the dependent lung during one-lung ventilation (OLV) in anesthetized dogs with a closed thoracic cavity.

Animals—7 clinically normal adult Walker Hound dogs.

Procedure—Dogs were anesthetized, and catheters were inserted in a dorsal pedal artery and the pulmonary artery. Dogs were positioned in right lateral recumbency, and data were collected during OLV (baseline), after application of 2.5 cm H_2O of PEEP for 15 minutes during OLV, and after application of 5 cm H_2O of PEEP for 15 minutes during OLV. Hemodynamic and respiratory variables were analyzed and calculations performed to obtain Do_2, and values were compared among the various time points by use of an ANOVA for repeated measures.

Results—PEEP induced a significant decrease in shunt fraction that resulted in a significant increase in arterial oxygen saturation. However, it failed to significantly affect arterial oxygen content (Cao_2) or cardiac output. Thus, Do_2 was not affected in healthy normoxemic dogs as a net result of the application of PEEP.

Conclusions and Clinical Relevance—The use of PEEP during OLV in anesthetized dogs with a closed thoracic cavity did not affect Do_2. Use of PEEP during OLV in dogs with a closed thoracic cavity is recommended because it does not affect cardiac output and any gain in Cao_2 will be beneficial for Do_2 in critically ill patients. (*Am J Vet Res* 2005;66:978–983)

One-lung ventilation (OLV) is the isolation and selective ventilation of 1 lung.[1] One-lung ventilation has been primarily used to facilitate thoracic surgery and minimally invasive thoracic surgery.[2] One-lung ventilation is typically initiated at the request of a surgeon once the thoracic cavity of the patient has been opened. Indications for starting OLV in patients with a closed thoracic cavity have been reported.[2,3] Absolute indications for the use of OLV include an infected lung, a bronchopleural or bronchocutaneous fistula, an opened conducting airway, bullae or cysts, any tracheobronchial disruption that may cause pneumomediastinum, and certain cases of pneumothorax and hemothorax after chest trauma.[1,2,4] In those situations, OLV is established in patients with a closed thoracic cavity as a maneuver to isolate injured areas of the lungs and to stabilize the patient before surgery is performed.

One-lung ventilation induces cardiopulmonary changes that result in a substantial augmentation of **shunt fraction (Q_s/Q_T)**, which in turn causes a substantial reduction in Pao_2 and alters **arterial oxygen saturation (Sao_2)**.[5-7] Use of a high concentration of oxygen,[8,9] application of continuous positive airway pressure to the nonventilated lung,[10] an increase in tidal volume,[11] and application of **positive end-expiratory pressure (PEEP)** to the dependent or nondependent lung[11-13] have been used to prevent hypoxemia during OLV. Positive end-expiratory pressure has been used to rapidly increase **arterial oxygen content (Cao_2)** by increasing the resting lung volume at the end of expiration. It increases **functional residual capacity (FRC)**, which contributes to recruitment of alveoli and thus prevents airway closure and improves gas exchange.[14-16]

The amount of oxygen available for tissues is determined by the amount of oxygen delivered. **Oxygen delivery (Do_2)** is dependent on **cardiac output (CO)** and Cao_2.[17,18] Use of high values of PEEP in patients with a closed thoracic cavity has been related to a reduction in CO, which may decrease Do_2.[1,19,20]

The use of PEEP in the dependent lung during OLV has the risk of causing volume-induced compression of intra-alveolar vessels, which will increase **pulmonary vascular resistance (PVR)** that diverts blood away from the ventilated lung. It will increase shunting and decrease oxygenation.[1] Therefore, the net effect of PEEP on Do_2 may be compromised when a reduction in Do_2 is greater than the reduction in CO, compared with the degree of improvement in Cao_2.

The objective of the study reported here was to determine changes in hemodynamic and respiratory variables when PEEP is applied to the dependent lung during OLV in anesthetized dogs with a closed thoracic cavity. We hypothesized that application of PEEP during OLV would affect Do_2 in clinically normal dogs.

Materials and Methods

Animals—Seven adult Walker Hound dogs were included in the study. Dogs were healthy as determined on

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the basis of results of a physical examination, CBC count, and serum biochemical analysis. Dogs were sexually intact, of both sexes, and weighed between 25.6 and 29.2 kg. Dogs were between 2 and 5 years of age and were used to evaluate changes induced by OLV with a closed chest immediately before inclusion in the study reported here. Food was withheld from each dog beginning 12 hours before the onset of the study. The study was approved by the Colorado State University Animal Care and Use Committee.

Procedure—Each dog served as its own control animal. Each dog was used to determine the changes induced when **2.5 cm H_2O of PEEP ($PEEP_{2.5}$)** and **5.0 cm H_2O of PEEP ($PEEP_5$)** were applied to the dependent lung during OLV.

The dogs received no medications prior to induction of anesthesia. A catheter was inserted in a cephalic vein, and induction of anesthesia was accomplished by IV administration of propofol[a] (3 to 4 mg/kg) and diazepam[b] (0.3 mg/kg). Each dog was intubated with a standard endotracheal tube, and an appropriate plane of anesthesia was maintained with an end-tidal concentration of isoflurane[c] of 1.85% to 1.95% (approx 1.5 times the minimum alveolar concentration) in oxygen, which was measured by use of an agent analyzer.[d] To facilitate intermittent positive-pressure ventilation in these dogs, atracurium[e] was administered (0.2 mg/kg, IV as a bolus, followed by 0.1 mg/kg, IV, repeated as needed to maintain muscle paralysis). An esophageal temperature probe[f] was advanced to the region of the heart base to measure core body temperature. Maintenance fluids consisted of IV administration of lactated Ringer's solution[g] (5 mL/kg/h) and a solution of dextrans[h] (5 mL/kg/h).

Hemodynamic and cardiorespiratory variables—The dogs were positioned in right lateral recumbency. A volume-limited ventilator[i] was adjusted to provide a baseline $PaCO_2$ of 35 to 45 mm Hg. **End-tidal partial pressure of carbon dioxide ($PETCO_2$)** was monitored by use of a side-stream capnograph[j] connected to the endotracheal tube. A $PaCO_2$ of 35 to 45 mm Hg was maintained, and respiratory rate and tidal volume did not change during the study.

A catheter was inserted in the dorsal pedal artery. Another catheter[k] was inserted in the pulmonary artery through an introducer[l] that had been inserted in a jugular vein; characteristic waveforms were used to guide proper placement of the catheter in the pulmonary artery. Each catheter was connected to a fluid-filled pressure transducer[m] and calibrated (ie, 0) at the level of the right atrium. Systolic pulmonary artery pressure, diastolic pulmonary artery pressure, **mean pulmonary artery pressure (MPAP)**, **pulmonary artery wedge pressure (PAWP)**, systolic arterial pressure, diastolic arterial pressure, **mean arterial pressure (MAP)**, and **right atrial pressure (RAP)** were recorded. Heart rate was recorded. Cardiac output was measured by use of a thermodilution technique.[n] Ten milliliters of ice-cold saline (0.9% NaCl) solution was injected, and the mean of 3 measurements was determined.

Blood gas analysis[o] was performed on heparinized arterial and mixed-venous blood samples. Arterial blood samples were collected via catheters inserted in the pedal and pulmonary arteries, respectively, and samples were analyzed immediately after collection to determine PaO_2, pH, bicarbonate concentration, acid-base excess, and SaO_2.

Values were calculated for several variables. The value for **pulmonary end-capillary oxygen content ($Cc'O_2$)** was calculated by use of the following equation:

$$Cc'O_2 = ([1.36 \times Hb \times 100]/100) + (0.003 \times PAO_2),$$

where Hb is the hemoglobin concentration and PAO_2 is the partial pressure of oxygen in the alveoli. The value for PAO_2 was calculated by use of the following equation:

$$PAO_2 = (FIO_2 \times [PB - PH_2O]) - (1.2 \times PaCO_2),$$

where FIO_2 is the inspired fraction of oxygen, PB is the barometric pressure, and PH_2O is the partial pressure of water vapor.

The Q_S/Q_T was calculated by use of the following equation:

$$Q_S/Q_T = (Cc'O_2 - CaO_2)/Cc'O_2 - C\bar{v}O_2),$$

where $C\bar{v}O_2$ is the mixed-venous oxygen content. The value for CaO_2 was calculated as $CaO_2 = (1.36 \times Hb \times SaO_2) + (0.003 \times PaO_2)$, whereas the value for $C\bar{v}O_2$ was calculated by use of the following equation:

$$C\bar{v}O_2 = (1.36 \times Hb \times S\bar{v}O_2) + (0.003 \times P\bar{v}O_2),$$

where $S\bar{v}O_2$ is the mixed-venous oxygen saturation and $P\bar{v}O_2$ is the mixed-venous partial pressure of oxygen.

The value for **alveolar-arterial oxygen pressure gradient ($PA\text{-}aO_2$)** was calculated as $PAO_2 - PaO_2$. The value for DO_2 was calculated as $CO \times CaO_2 \times 10$. The value for **cardiac index (CI)** was calculated by use of the following equation:

$$CI = CO/BSA,$$

where BSA is body surface area.

The value for **oxygen extraction ratio (O_{2ER})** was calculated as $(SaO_2 - S\bar{v}O_2)/SaO_2$. **Pulmonary vascular resistance index (PVRI)** was calculated as (MPAP – PAWP)/CI. **Systemic vascular resistance index (SVRI)** was calculated as (MAP – RAP)/CI. **Dead space (V_D/V_T)** was calculated by use of the following equation:

$$V_D/V_T = ([PaCO_2 - PETCO_2]/PaCO_2) \times 100.$$

Collection of data—The left bronchus of each dog was obstructed by use of a bronchial blocker.[p] Bronchoscopy[q] was used to ensure appropriate placement of the blocker and adequate obstruction of the bronchus. After the left bronchus was blocked, 15 minutes was allowed for equilibration and data were then collected for the OLV period (baseline). Following these measurements, $PEEP_{2.5}$ was applied, a similar equilibration period was provided, and measurements were again obtained. Finally, $PEEP_5$ was applied, a 15-minute equilibration period was provided, and measurements were recorded.

Statistical analysis—An ANOVA for repeated measurements[21] was used to evaluate the effects of $PEEP_{2.5}$ and $PEEP_5$ on hemodynamic and respiratory variables during OLV. Data were reported as mean ± SE. Values of $P < 0.05$ were considered significant. When the P value was significant, comparisons were made among treatment groups by use of the Fisher least significant difference test.

Results

The use of OLV-$PEEP_5$ caused a significant ($P = 0.022$) augmentation of SaO_2, compared with SaO_2 during baseline (**Table 1**). When compared with baseline values, there was a significant augmentation during OLV-$PEEP_{2.5}$ and OLV-$PEEP_5$ for RAP ($P = 0.004$ and $P = 0.009$, respectively), PAWP ($P = 0.007$ and $P < 0.001$, respectively), and MPAP ($P = 0.01$ and $P = 0.004$, respectively; **Table 2**).

Cardiac index was not significantly ($P = 0.416$) affected by the application of PEEP (**Table 3**). This

Table 1—Mean ± SE values for respiratory variables of 7 clinically normal anesthetized dogs with a closed thoracic cavity during one-lung ventilation (OLV), OLV with 2.5 cm H_2O of positive end-expiratory pressure (OLV-$PEEP_{2.5}$), and OLV with 5 cm H_2O of positive end-expiratory pressure (OLV-$PEEP_5$).

Variable	OLV	OLV-$PEEP_{2.5}$	OLV-$PEEP_5$	*P* value*	Power
Pa_{O_2} (mm Hg)	170 ± 25	207 ± 26	245 ± 37	0.096	0.110
Pa_{CO_2} (mm Hg)	44 ± 2	42 ± 2	43 ± 3	0.402	0.427
pHa	7.30 ± 0.02	7.30 ± 0.02	7.30 ± 0.03	0.869	0.878
HCO_3^-a (mEq/L)	22.0 ± 0.4	21.0 ± 0.4	22.0 ± 0.5	0.069	0.080
ABEa (mEq/L)	–4.1 ± 0.6	–4.9 ± 0.5	–4.7 ± 0.6	0.110	0.125
Sa_{O_2} (%)	98.6 ± 0.2[a]	99.0 ± 0.2	99.2 ± 0.2[b]	0.022	NA
P_{ETCO_2} (mm Hg)	35 ± 2	35 ± 2	36 ± 2	0.656	0.676

*Represents a *P* value of general effect.
pHa = Arterial pH. HCO_3^-a = Arterial bicarbonate concentration. ABEa = Arterial acid-base excess. Sa_{O_2} = Arterial oxygen saturation. P_{ETCO_2} = End-tidal partial pressure of carbon dioxide. NA = Not applicable.
[a,b]Within a row, values with different superscript letters differ significantly ($P < 0.05$).

Table 2—Mean ± SE values for hemodynamic variables of 7 clinically normal anesthetized dogs with a closed thoracic cavity during OLV, OLV-$PEEP_{2.5}$, and OLV-$PEEP_5$.

Variable	OLV	OLV-$PEEP_{2.5}$	OLV-$PEEP_5$	*P* value*	Power
SAP (mm Hg)	108 ± 6	109 ± 6	111 ± 6	0.826	0.838
DAP (mm Hg)	57 ± 3	60 ± 3	61 ± 2	0.452	0.477
MAP (mm Hg)	74 ± 3	76 ± 4	77 ± 3	0.516	0.540
RAP (mm Hg)	5.0 ± 0.3[a]	7.0 ± 0.7[b]	7.0 ± 0.3[b]	0.007	NA
PAWP (mm Hg)	6 ± 1[a]	8 ± 1[b]	9 ± 1[b]	< 0.001	NA
SPAP (mm Hg)	21 ± 1[a]	24 ± 1[b]	23 ± 1[b]	0.033	NA
DPAP (mm Hg)	11 ± 1[a]	12 ± 1	13 ± 1[b]	0.049	NA
MPAP (mm Hg)	16 ± 1[a]	17 ± 1[b]	18 ± 1[b]	0.011	NA
HR (beats/min)	117 ± 4	117 ± 5	115 ± 6	0.327	0.3515
Core body temperature (°C)	36.5 ± 0.25	36.5 ± 0.28	36.7 ± 0.26	0.280	0.470

SAP = Systolic arterial pressure. DAP = Diastolic arterial pressure. MAP = Mean arterial pressure. RAP = Right atrial pressure. PAWP = Pulmonary arterial wedge pressure. SPAP = Systolic pulmonary arterial pressure. DPAP = Diastolic pulmonary arterial pressure. MPAP = Mean pulmonary arterial pressure. HR = Heart rate.
See Table 1 for remainder of key.

Table 3—Mean ± SE values for respiratory and hemodynamic calculated variables of 7 clinically normal anesthetized dogs with a closed thoracic cavity during OLV, OLV-$PEEP_{2.5}$, and OLV-$PEEP_5$.

Variable	OLV	OLV-$PEEP_{2.5}$	OLV-$PEEP_5$	*P* value*	Power
Ca_{O_2} (mL/dL)	21.6 ± 0.8	21.8 ± 0.9	22.0 ± 0.9	0.058	0.067
$Q_S/\dot{Q}_T$ (%)	30 ± 2[a]	28 ± 1[b]	25 ± 1[b]	0.015	NA
Do_2 (mL/kg/min)	1,028 ± 136	1,105 ± 111	996 ± 144	0.472	0.496
PVRI (mm Hg/L/min/m^2)	2.0 ± 0.2	2.0 ± 0.2	2.0 ± 0.3	0.141	0.158
SVRI (mm Hg/L/min/m^2)	15.0 ± 1.7	13.0 ± 0.7	15.0 ± 1.0	0.157	0.176
O_{2ER} (%)	12.40 ± 0.01	12.30 ± 0.01	12.50 ± 0.06	0.966	0.968
CI (L/min/m^2)	5.1 ± 0.6	5.4 ± 0.5	4.8 ± 0.6	0.416	0.441
V_D/V_T (%)	21.00 ± 0.02	18.40 ± 0.02	18.00 ± 0.01	0.227	0.249
P_{A}-a_{O_2} (mm Hg)	375 ± 27	340 ± 27	301 ± 37	0.089	0.102
P_{AO_2} (mm Hg)	545 ± 6	547 ± 6	546 ± 7	0.402	0.427

Ca_{O_2} = Arterial oxygen content. Q_S/Q_T = Shunt fraction. Do_2 = Oxygen delivery. PVRI = Pulmonary vascular resistance index. SVRI = Systemic vascular resistance index. O_{2ER} = Oxygen extraction ratio. CI = Cardiac index. V_D/V_T = Physiologic dead space. P_{A}-a_{O_2} = Alveolar-arterial oxygen difference. P_{AO_2} = Alveolar oxygen tension.
See Table 1 for remainder of key.

resulted in nonsignificant ($P = 0.141$) changes in PVRI. Furthermore, the significant increase of Sa_{O_2} with the application of $PEEP_5$ did not result in a significant ($P = 0.058$) augmentation of Ca_{O_2}. Application of $PEEP_{2.5}$ and $PEEP_5$ resulted in a significant ($P = 0.045$ and $P = 0.005$, respectively) reduction of Q_S/Q_T, compared with the baseline value. As a net result of PEEP on hemodynamic and respiratory variables, Do_2 was not significantly ($P = 0.47$) altered.

Discussion

Application of PEEP did not reduce CO during OLV in dogs with a closed thoracic cavity. Positive end-expiratory pressure reduced the amount of pulmonary shunting; however, Ca_{O_2} did not improve in dogs of the study reported here. Therefore, Do_2 was not improved because Ca_{O_2} was not increased by a clinically important amount. Oxygen content would most likely be improved in patients with pulmonary disease

with reduced Sa_{O_2}. Therefore, we recommend the use of PEEP during OLV in clinically affected dogs with a closed thoracic cavity because it was not detrimental to cardiac function and D_{O_2} during OLV in clinically normal dogs with a closed thoracic cavity.

It has been established that PEEP can cause reductions in CO. Possible mechanisms include a reduction in venous return because of the increase in intrathoracic pressure,[1] an increase in right ventricular afterload,[22,23] leftward displacement of the interventricular septum,[24] depressed lung stretch reflex,[25,26] decreased left and right ventricular preload,[27] and altered ventricular function.[28,29] The exact mechanism for this effect is controversial, and major adverse hemodynamic effects generally are evident.[19,25,27,29-33] The negative effect of PEEP on hemodynamic variables has been documented in dogs with a closed thoracic cavity.[24,28,34] In animals with an intact rib cage, PEEP increases intrathoracic pressure and has a negative effect on cardiac function; however, the situation during OLV is not the same as that during two-lung vetilation. During OLV, PEEP will be applied only to the ventilated lung, whereas the nonventilated lung will become atelectatic. Absorption atelectasis of the nonventilated lung should allow expansion of the ventilated lung and limit the augmentation of intrathoracic pressure. Therefore, PEEP during OLV in a dog with a closed thoracic cavity probably provided sufficient space for the ventilated lung to expand, which prevented most of the negative effects of PEEP on hemodynamic variables.

Application of PEEP did not affect CO in the study because it did not have an effect on PVR. Pulmonary vascular resistance is a function of CO, MPAP, and PAWP.[22] In the study reported here, MPAP and PAWP were significantly increased but CO was unchanged, leading to a net effect of no change in the calculated PVR (Ohm's law). Volume of the lung and alveoli influences PVR because it alters transmural pressure at the level of the capillaries.[35] Pulmonary vascular resistance is increased when lung volume is increased or decreased from FRC.[23,34-36] Positive end-expiratory pressure can affect PVR because of modification of FRC.[12,37] Thus, when alveoli collapse, PVR increases as a result of collapse of the alveolar capillaries. When alveoli are subsequently reexpanded by use of PEEP, the expanded alveoli exert traction on the capillary wall to open the capillaries, thereby decreasing PVR. However, when the alveoli are overexpanded by use of excessive amounts of PEEP, transmural pressure increases and collapses the capillaries, which results in an increase in pulmonary arterial pressure. In this study, PVR was not affected by the amount of PEEP used, probably because we reexpanded collapsed alveoli without inducing overdistension. Therefore, $PEEP_5$ did not affect PVR, and we believe that $PEEP_5$ can be safely used during OLV with a closed thoracic cavity.

Pulmonary vascular resistance is also affected by transmural pressure,[35] recruitment of capillaries with increases in CO,[38,39] and hypoxic vasoconstriction.[40,41] It was beyond the scope of our study to evaluate the effect of PEEP on PVR. However, it has been reported[42] that application of PEEP at < 5 cm H_2O is unlikely to increase PVR.

An increase in PVR in the ventilated lung as a result of PEEP applied during OLV can increase the amount of pulmonary shunting by redistributing blood flow from the ventilated lung to the nonventilated lung.[1] Values of PEEP in excess of 5.0 cm H_2O can increase PVR in the ventilated lung.[42] During OLV, the nonventilated lung represents a great potential for redistribution of blood flow after application of PEEP. However, during OLV, the nonventilated lung is subjected to **hypoxemic pulmonary vasoconstriction (HPV)** that reduces the amount of shunting by 30% to 40%.[1] Dogs have intense HPV[43] that should protect them against redistribution of blood flow during PEEP and OLV. However, anesthesia achieved by use of isoflurane or sevoflurane abolishes this phenomenon in a dose-dependent manner.[19,44] Because the dogs in our study were anesthetized with isoflurane, there could have been redistribution of blood flow to the nonventilated lung. We can assume that HPV did not change during the study because anesthesia was maintained at a constant depth. Because Q_S/Q_T and $P_{A\text{-}a}O_2$ improved and PVR did not change in this study, we can assume that the pulmonary blood flow was not redistributed to the nonventilated lung and that PEEP did not overstretch the recruited alveoli.

Shunt fraction improved after application of $PEEP_5$, indicating improvement in ventilation-perfusion matching and a reduction of pulmonary shunting. However, this effect was not sufficient to induce a significant reduction of $P_{A\text{-}a}O_2$ and a significant augmentation of Pa_{O_2}. Application of PEEP increased the distention of poorly ventilated alveoli and recruited collapsed alveoli. Reduction of shunting increased Pa_{O_2} with $PEEP_5$; however, it did not induce a significant increase in Sa_{O_2} because of the sigmoid shape of the oxygen-dissociation curve. The values of Pa_{O_2} observed in this study corresponded to the upper plateau of the oxygen-dissociation curve such that the increase in Pa_{O_2} did not significantly increase Sa_{O_2}. Consequently, augmentation of the calculated Ca_{O_2} was extremely limited because Pa_{O_2} is only a negligible component in the equation to calculate Ca_{O_2}. In a study[37] in humans, the application of PEEP improved Pa_{O_2} only in patients with a low Pa_{O_2} (< 80 mm Hg) but not in patients with an initial Pa_{O_2} > 80 mm Hg.[37] The proposed reason for this effect is that hypoxemic patients are more likely to have lung volumes below the FRC with increased PVR. With the use of PEEP, lung volume approaches FRC, which results in a decrease of PVR and an increase of blood flow to the dependent lung.[11,37] The effect of PEEP would be more important in patients with pulmonary disease and low Sa_{O_2}. The negligible effect of PEEP on Sa_{O_2} in our study limited any potential benefits of PEEP on D_{O_2}.

The study reported here had several limitations. The small number of dogs entered in the study gave a limited power to our statistical analysis for the effect of PEEP on CO and D_{O_2}. However, O_{2ER}, which is another variable used to evaluate D_{O_2}, had excellent power. Therefore, the fact that O_{2ER} was not significantly affected in this study would confirm that D_{O_2} was not significantly affected by the application of PEEP during OLV. A second limitation is the fact that we evaluated healthy

dogs that did not have cardiopulmonary disease. Consequently, the detrimental effects of OLV on Sa_{O_2} were minimal, which limited the opportunity for PEEP to exert a beneficial effect on D_{O_2}.

A third limitation of the study was that the treatment groups were not randomized. Each dog served as its own control animal. As recommended in another report,[45] dogs were maintained on OLV for 15 minutes before recording data and then applying $PEEP_{2.5}$. The sequential application of $PEEP_{2.5}$ followed by $PEEP_5$ was used to mimic the clinical situation in which stepwise increases in PEEP are typically applied. It is possible that the beneficial effects of $PEEP_5$ were in part attributable to the preceding application of $PEEP_{2.5}$. Nevertheless, investigators in other studies[6,24,46,47] of cardiopulmonary effects of PEEP have generally recorded data after incremental increases in PEEP. It has also been recommended that PEEP been applied in an incremental fashion while concurrently measuring D_{O_2} to enable clinicians and researchers to optimize the effects of PEEP.[48]

a. PropoFlo, Abbott Laboratories, North Chicago, Ill.
b. Diazepam, Elkins-Sinn Inc, Cherry Hill, NJ.
c. IsoFlo, Abbott Laboratories, North Chicago, Ill.
d. Ohmeda 5330 agent monitor, Datex-Ohmeda, Louisville, Colo.
e. Atracurium besylate injection, Faulding Pharmaceutical Co, Elizabeth, NJ.
f. Reusable temperature probe, Yellow Springs Instrument Co, Yellow Springs, Ohio.
g. Lactated Ringer's injection, Abbott Laboratories, North Chicago, Ill.
h. 6% Gentran 70 and 0.9% sodium chloride injection, Baxter Healthcare Corp, Deerfield, Ill.
i. Narkomed DA, 2-L volume limited ventilator, North American Drager, Telford, Pa.
j. Side-stream end-tidal CO_2 sensor, Model 20021, Medical Data Electronics, Arleta, Calif.
k. Opticath catheter, Abbott Critical Care Systems, Abbott Laboratories, North Chicago, Ill.
l. Arrow percutaneous sheath introducer system, Arrow International Inc, Reading, Pa.
m. CDXpress, Argon, Maxxim Medical, Athens, Tex.
n. Explorer oximetry computer, Baxter Healthcare Corp, Edwards Critical-Care Division, Santa Ana, Calif.
o. IRMA blood analysis system, series 2000, Diametrics Medical Inc, Saint Paul, Minn.
p. Arndt endobronchial blocker, Cook Critical Care, Bloomington, Ind.
q. Evis bronchovideoscope, Olympus BF type 240 series, Olympus America Inc, Melville, NY.

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