

Improving Oxygenation during One-lung Ventilation in Dogs: The Effects of Positive End-Expiratory Pressure and Blood Flow Restriction to the Nonventilated Lung

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During one-lung ventilation in dogs, the authors studied the individual and combined effects of 5, 10, and 15 cm H₂O of O₂ PEEP to the nonventilated lung and nonocclusive inflation of a pulmonary artery catheter balloon (PAB) in the main pulmonary artery of the nonventilated lung on PaO₂, \dot{Q}_v/\dot{Q}_t , and blood flow to the nonventilated lung (\dot{Q}_{vent}/\dot{Q}_t). Periods of one-lung ventilation alone resulted in PaO₂ of 61 ± 5 torr (mean ± SE), \dot{Q}_v/\dot{Q}_t of 50 ± 3 per cent, and \dot{Q}_{vent}/\dot{Q}_t of 41 ± 6 per cent. They found that 5 and 10 cm H₂O of O₂ PEEP caused a greatly decreased \dot{Q}_v/\dot{Q}_t (22 ± 4 and 21 ± 4 per cent, respectively) and increased PaO₂ (327 ± 43 and 330 ± 40 torr, respectively) while \dot{Q}_{vent}/\dot{Q}_t remained unchanged, whereas 15 cm H₂O of O₂ PEEP caused similar PaO₂ and \dot{Q}_v/\dot{Q}_t changes while \dot{Q}_{vent}/\dot{Q}_t significantly decreased (to 19 ± 6). Nonocclusive inflation of the PAB alone caused moderate favorable changes in PaO₂ and \dot{Q}_v/\dot{Q}_t (but less than those caused by any level of O₂ PEEP) which were due to a decreased \dot{Q}_{vent}/\dot{Q}_t (to 22 ± 5 percent). Inflation of the PAB during any level of O₂ PEEP did not change PaO₂ or \dot{Q}_v/\dot{Q}_t significantly, but still decreased \dot{Q}_{vent}/\dot{Q}_t significantly. Probable mechanisms of efficacy of nonventilated lung manipulations are as follows: low levels of O₂ PEEP maintain the patency of airways, allowing nonventilated lung alveoli to participate in gas exchange; high levels of O₂ PEEP act by this mechanism as well as by causing blood flow diversion to the ventilated lung; PAB inflation acts by blood flow diversion to the ventilated lung alone. The authors conclude that low levels of O₂ PEEP to the nonventilated lung should be used when possible if hypoxemia occurs during one-lung ventilation. Flow diversion caused by PAB inflation may be uniquely useful in situations in which O₂ PEEP to the nonventilated lung is impractical (e.g., surgical interference, pulmonary hemorrhage) or impossible (e.g., pulmonary lavage). (Key words: Equipment: catheters, pulmonary artery. Hypoxia. Lung: atelectasis, blood flow, pulmonary artery; shunting. Oxygen: blood levels. Ventilation: apnea, positive end-expiratory pressure.)

ONE-LUNG VENTILATION is often used as a method for isolating a diseased lung from a normal lung and improving operating conditions. There is, however, a very large and variable alveolar-to-arterial oxygen tension difference [P(A-a)_{O₂}] which develops as a necessary consequence of this intervention.¹⁻⁷ Use of a high inspired oxygen concentration has been tried as a means of minimizing hypoxemia during one-lung ventilation.^{1,3-5,7,8} In addition, intermittent two-lung ventilation may be re-

quired for short periods of time if severe hypoxemia persists.⁸

Very recently, two new promising approaches to improving the matching of ventilation and perfusion during one-lung anesthesia have been reported. First, selective application of 10 cm H₂O positive end-expiratory pressure with 100 per cent oxygen (O₂ PEEP) to the nonventilated lung (apneic oxygenation) has significantly increased arterial oxygenation (PaO₂).⁹ Second, we have recently shown that nonocclusive inflation of a balloon at the tip of a 7-Fr. pulmonary artery catheter positioned in the main pulmonary artery of the nonventilated lung significantly diverts blood flow from the nonventilated lung to the ventilated lung and thereby improves PaO₂ during one-lung ventilation.¹⁰

The purpose of this study was to critically examine these two new techniques in a canine one-lung ventilation model and to answer five specific, but related, questions: 1) What is the mechanism whereby O₂ PEEP to the nonventilated lung improves PaO₂? 2) Is there an optimal level of PEEP that should be used? 3) Does insufflation of oxygen at zero end-expiratory pressure (ZEEP) improve PaO₂? 4) Is one of these techniques, namely, pulmonary artery balloon inflation or O₂ PEEP to the nonventilated lung, superior to the other? 5) Is the combination of these two techniques superior to either method when used alone?

Methods

Our experimental preparation has been previously described¹⁰ and will only be summarized here. Six mongrel dogs (19-26 kg) were anesthetized with pentobarbital, intubated, and mechanically ventilated with 100 per cent O₂ by a dual-piston Harvard® respirator. Following a wide thoracotomy, electromagnetic flow probes (Statham® SP7515, see previous calibration procedures¹⁰) were placed around the main (sizes 12 and 14 mm) and left main (size 10 mm) pulmonary arteries. Right main pulmonary artery blood flow was calculated as the difference between the directly measured main and left main pulmonary artery blood flows. Placement of a double-lumen endotracheal tube and confirmation of proper location and function was achieved as before.¹⁰

A pulmonary artery balloon (PAB) on the tip of a 7 Fr. pulmonary artery catheter was positioned in the right

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TABLE 1. Experimental Sequence

Step 1. Two-lung ventilation.	
Segment A	Step 2. Nondependent lung atelectasis and PAB down.*
	Step 3. O ₂ insufflation at ZEEP and PAB down.
	Step 4. Nondependent lung atelectasis and PAB up.†
Segment B	Step 5. 5 cm H ₂ O O ₂ PEEP and PAB down.
	Step 6. 5 cm H ₂ O O ₂ PEEP and PAB up.
	Step 7. Nondependent lung atelectasis and PAB down.
Segment C	Step 8. 10 cm H ₂ O O ₂ PEEP and PAB down.
	Step 9. 10 cm H ₂ O O ₂ PEEP and PAB up.
	Step 10. 10 cm H ₂ O N ₂ PEEP and PAB down.
	Step 11. Nondependent lung atelectasis and PAB down.
Segment D	Step 12. 15 cm H ₂ O O ₂ PEEP and PAB down.
	Step 13. 15 cm H ₂ O O ₂ PEEP and PAB up.
	Step 14. Nondependent lung atelectasis and PAB down.

* PAB down = pulmonary artery balloon deflated.

† PAB up = pulmonary artery balloon inflated.

(four animals) or left (two animals) main pulmonary artery and the location of the PAB confirmed by palpation. Pressures in the pulmonary artery (PAP), left atrium (LAP), airway (P_A), and systemic artery were measured and recorded (along with the two pulmonary blood flows) as described previously.¹⁰ The PAB was inflated as described previously¹⁰ during several of the experimental steps (see below). Each experimental step was 10 min in duration; thus, the entire experimental test sequence took approximately 2.5–3 hours to complete.

The entire experimental sequence (table 1) was carried out in the lateral decubitus position and with the PAB positioned in the main pulmonary artery of the nonventilated nondependent lung. All steps subsequent to the control situation of two-lung ventilation (step 1) were carried out with only the dependent lung being ventilated (tidal volume = 12 ml/kg), and all experimental manipulations were carried out on the nondependent lung. The application of PEEP or ZEEP to the nonventilated nondependent lung was achieved by terminating the deflation phase of a single 20 ml/kg breath (sigh) at the desired end-expiratory pressure (ZEEP or PEEP) and maintained (without tidal ventilation) by insufflation of O₂.

Section A of the experimental sequence was always performed first (table 1), but steps 2, 3, and 4 within this section were performed in random order: step 2, nondependent lung atelectasis and PAB deflated (PAB down); step 3, five l/min O₂ insufflation at ZEEP and PAB down; and step 4, nondependent lung atelectasis and PAB inflated (PAB up). Following section A, sections B, C, and D were performed in random order.

Sections B, C, and D each contained a period of nondependent lung atelectasis and PAB down (steps 7, 11, and 14, respectively). Each of these three periods with nondependent lung atelectasis and PAB down were preceded by administration of a different level of O₂ PEEP to the nondependent lung. During each different level of O₂ PEEP the PAB was either deflated or inflated (random order) as follows: in section B, with 5 cm H₂O O₂ PEEP (steps 5 and 6, respectively); in section C, with 10 cm H₂O O₂ PEEP (steps 8 and 9, respectively); and in section D with 15 cm H₂O O₂ PEEP (steps 12 and 13, respectively). In addition, the effects of administering 10 cm H₂O PEEP with 100 per cent N₂ (N₂ PEEP) and PAB down (step 10, section C) were studied.

Right to left shunt (\dot{Q}_s/\dot{Q}_t) was calculated by the Berggren formulation as described previously¹⁰ and pulmonary blood flows and pressures were read only at the end of each step change. Results are expressed as means \pm SE and were analyzed by the F test and Student's paired *t* test, with *P* < 0.01 considered statistically significant.

Results

Changing from two-lung (step 1) to one-lung ventilation and PAB down (step 2) caused a significant decrease in PaO₂ (fig. 1). Insufflation of O₂ at ZEEP with the PAB down (step 3) did not cause the PaO₂ to change significantly from step 2. However, inflation of the PAB

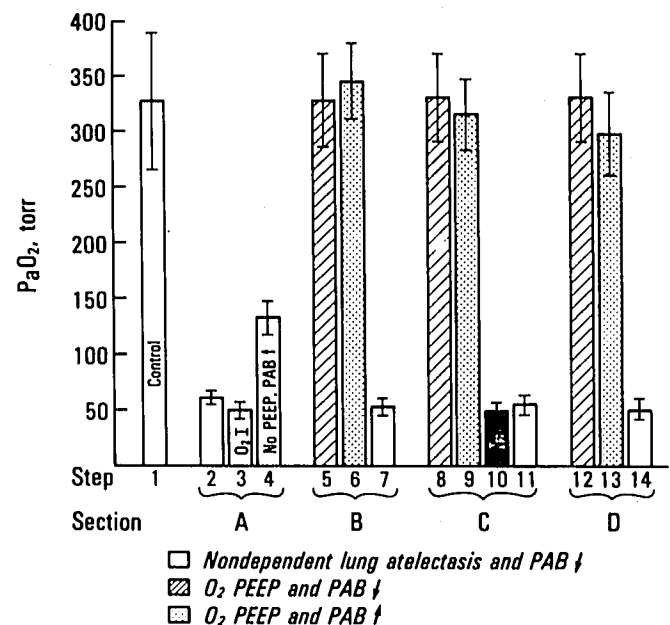


FIG. 1. The PaO₂ results during each experimental sequence step. Control = two-lung ventilation; O₂ I = O₂ insufflation at ZEEP; No PEEP, PAB ↑ = nonventilated lung atelectasis and pulmonary artery balloon (PAB) inflated; N₂ = 10 cm H₂O N₂ PEEP and PAB deflated. See table 1 and text for full explanation of steps and significant results.

(step 4) caused a significant increase in Pa_{O_2} as compared to steps 2 and 3. The application of 5, 10, and 15 cm H_2O O_2 PEEP with the PAB down (steps 5, 8, and 12, respectively) resulted in significant increases in Pa_{O_2} as compared to periods of nondependent lung atelectasis and PAB down (steps 7, 11, and 14, respectively). In addition, and most importantly, for every O_2 PEEP level with the PAB down (steps 5, 8, and 12) there was a significantly greater Pa_{O_2} compared to the period of nondependent lung atelectasis and PAB up (step 4). The Pa_{O_2} obtained during any given O_2 PEEP level was not statistically different from the Pa_{O_2} obtained during any other O_2 PEEP level. Inflation of the PAB during 5, 10, and 15 cm H_2O O_2 PEEP (steps 6, 9, and 13, respectively) did not cause a significant change in Pa_{O_2} compared to these O_2 PEEP levels with the PAB down (steps 5, 8, and 12, respectively). Finally, application of 10 cm H_2O N_2 PEEP (step 10) did not cause Pa_{O_2} to change significantly from the value obtained during the corresponding period of nondependent atelectasis and PAB down (step 11). All the O_2 and the single N_2 PEEP levels to the nonventilated lung did not cause any significant change in Pa_{CO_2} .

Changing from two-lung ventilation (step 1) to one-lung ventilation and PAB down (step 2) caused \dot{Q}_s/\dot{Q}_t to significantly increase (fig. 2). Insufflation of O_2 at ZEEP with the PAB down (step 3) did not cause the \dot{Q}_s/\dot{Q}_t to change significantly from step 2. However, inflation of the PAB (step 4) caused a significant decrease in \dot{Q}_s/\dot{Q}_t as compared to either steps 2 and 3. The application of 5, 10, and 15 cm H_2O O_2 PEEP with the PAB down (steps 5, 8, and 12, respectively) resulted in

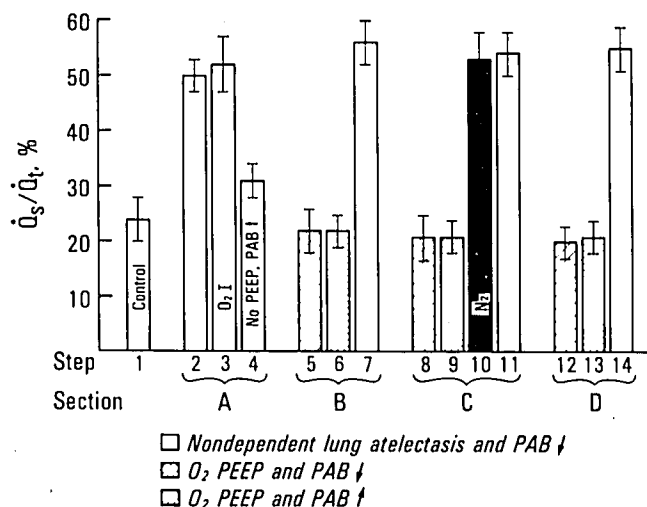


FIG. 2. The \dot{Q}_s/\dot{Q}_t results during each experimental sequence step. Control = two-lung ventilation; O_2 I = O_2 insufflation at ZEEP; No PEEP, PAB ↑ = nonventilated lung atelectasis and pulmonary artery balloon (PAB) inflated; N_2 = 10 cm H_2O N_2 PEEP and PAB deflated. See table 1 and text for full explanation of steps and significant results.

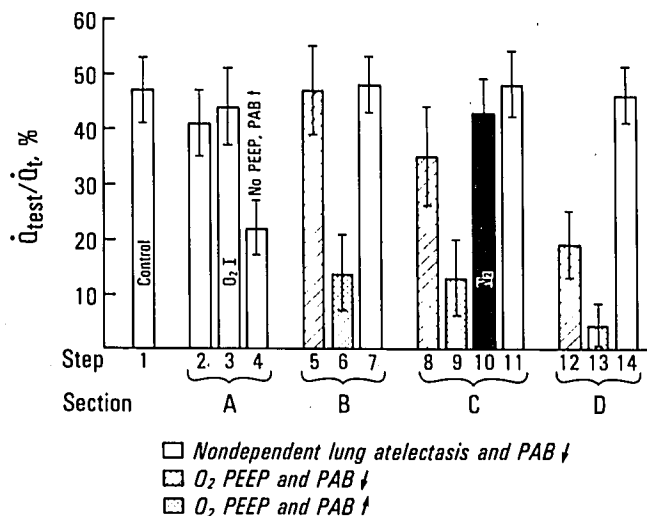


FIG. 3. Blood flow to the test (nonventilated, nondependent) lung as a per cent of cardiac output (\dot{Q}_{test}/\dot{Q}_t) during each experimental sequence step. Control = two-lung ventilation; O_2 I = O_2 insufflation at ZEEP; No PEEP, PAB ↑ = nonventilated lung atelectasis and pulmonary artery balloon (PAB) inflated; N_2 = 10 cm H_2O N_2 PEEP and PAB deflated. See table 1 and text for full explanation of steps and significant results.

significant decreases in \dot{Q}_s/\dot{Q}_t as compared to periods of nondependent lung atelectasis and PAB down (steps 7, 11, and 14, respectively). In addition, and most importantly, for every O_2 PEEP level with the PAB down (steps 5, 8, and 12) there was a significant reduced \dot{Q}_s/\dot{Q}_t compared to the period of nondependent lung atelectasis and PAB up (step 4). As was found for Pa_{O_2} , the \dot{Q}_s/\dot{Q}_t obtained during any given O_2 PEEP level was not statistically different from the \dot{Q}_s/\dot{Q}_t during any other O_2 PEEP level. Inflation of the PAB during 5, 10, and 15 cm O_2 PEEP (steps 6, 9, and 13, respectively) did not cause a significant change in \dot{Q}_s/\dot{Q}_t compared to these O_2 PEEP levels with the PAB down (steps 5, 8, and 12, respectively). Finally, application of 10 cm H_2O N_2 PEEP (step 10) did not cause \dot{Q}_s/\dot{Q}_t to change significantly from the value obtained during the corresponding period of nondependent lung atelectasis and PAB down (step 11).

Figure 3 shows the blood flow to the test (nonventilated, nondependent) lung as a fraction of cardiac output (\dot{Q}_{test}/\dot{Q}_t) for each experimental sequence step. Changing from two-lung ventilation (step 1) to one-lung ventilation and PAB down (step 2) or O_2 insufflation at ZEEP with the PAB down (step 3) did not cause \dot{Q}_{test}/\dot{Q}_t to change significantly. However, inflation of the PAB (step 4) caused a significant reduction in \dot{Q}_{test}/\dot{Q}_t as compared to steps 1-3. During steps 2 and 3 \dot{Q}_{test}/\dot{Q}_t was slightly less than \dot{Q}_s/\dot{Q}_t and step 4 caused identical decreases in \dot{Q}_{test}/\dot{Q}_t and \dot{Q}_s/\dot{Q}_t .

The administration of 5 and 10 cm H₂O O₂ PEEP and 10 cm H₂O N₂ PEEP with the PAB down (steps 5, 8, and 10, respectively) did not cause any significant change in $\dot{Q}_{\text{test}}/\dot{Q}_t$ compared to periods of nondependent lung atelectasis and PAB down (steps 7 and 11, respectively). Inflation of the PAB during 5 and 10 cm H₂O O₂ PEEP (steps 6 and 9, respectively) caused a significant decrease in $\dot{Q}_{\text{test}}/\dot{Q}_t$ compared to periods of nondependent lung atelectasis and PAB down (steps 7 and 11, respectively). The application of 15 cm H₂O O₂ PEEP with the PAB down (step 12) did cause a significant decrease in $\dot{Q}_{\text{test}}/\dot{Q}_t$ compared to the corresponding period of nondependent lung atelectasis and PAB down (step 14) and 5 and 10 cm H₂O O₂ PEEP with PAB down (steps 5 and 8, respectively). Finally, inflation of the PAB during 15 cm H₂O O₂ PEEP (step 13) caused a further, significant decrease in $\dot{Q}_{\text{test}}/\dot{Q}_t$ compared to 15 cm H₂O O₂ PEEP with the PAB down (step 12).

Mean left atrial pressure and cardiac output did not change significantly from the control value during two-lung ventilation (step 1) of 8.8 ± 1.2 torr and $2,045 \pm 384$ ml/min, respectively, with any of the subsequent 13 experimental steps. However, mean pulmonary artery pressure was increased significantly from 19.8 ± 1.1 to 23.8 ± 1.4 torr when comparing nondependent lung atelectasis and PAB down (step 2) to PAB up (step 4). Similar but slightly smaller increases in mean pulmonary artery pressure occurred when the corresponding periods of nondependent lung atelectasis and PAB down (steps 7 and 11, respectively) were compared to either 5 or 10 cm H₂O O₂ PEEP and PAB up (steps 6 and 9, respectively).

Discussion

This study had two methodologic/results features that deserve comment. First, of the four nondependent lung atelectasis and PAB down periods (steps 2, 7, 11, 14), only step 2 demonstrated a small decrease in $\dot{Q}_{\text{test}}/\dot{Q}_t$ (approximately 12 per cent decrease compared to step 1) and therefore evidence of hypoxic pulmonary vasoconstriction (HPV). Since step 2 was the first exposure of the test lung to hypoxic conditions and the first hypoxic exposure elicits the least HPV^{11,12}, and since single lung HPV does not cause a large flow redistribution,¹³ the small step 2 result is not surprising. Since our experimental sequence did not include a return to control two-lung ventilation there are no normoxic (and non-PEEP) $\dot{Q}_{\text{test}}/\dot{Q}_t$ values available with which to directly compare steps 7, 11, and 14. It is possible that during the course of the experiment the dependent lung became diseased, and the normoxic $\dot{Q}_{\text{test}}/\dot{Q}_t$ increased (compared to the value in step 1). Thus, it is possible that our steps 7, 11,

and 14 hypoxic $\dot{Q}_{\text{test}}/\dot{Q}_t$ values could actually be decreased compared to an unmeasured but increased, normoxic $\dot{Q}_{\text{test}}/\dot{Q}_t$. Second, we found no discrepancy between $\dot{Q}_{\text{test}}/\dot{Q}_t$ and \dot{Q}_s/\dot{Q}_t as we had previously.¹⁰ In this experiment we used a less constricting 10-mm left lung flow probe compared to our previous use of an 8-mm flow probe.¹⁰

The purpose of this study was to critically examine two new techniques which increase PaO₂ and decrease \dot{Q}_s/\dot{Q}_t during one-lung ventilation. The first method involves the selective application of O₂ PEEP to the nonventilated, nondependent lung.⁹ In the present study we performed dose (level of O₂ PEEP)-response (oxygenation effect) curves and sought to determine the mechanism of the therapeutic effect of nonventilated lung O₂ PEEP. The second method for improving PaO₂ and decreasing \dot{Q}_s/\dot{Q}_t involves nonocclusive inflation of a PAB in the nonventilated lung.¹⁰ In this study we have reconfirmed the efficacy of nonventilated lung PAB inflation, compared the benefits of nonventilated lung PAB inflation with nonventilated lung O₂ PEEP, and tested the effects of combining both therapeutic maneuvers.

First, what is the likely mechanism whereby O₂ PEEP to the nonventilated lung increases PaO₂? Since there were no significant $\dot{Q}_{\text{test}}/\dot{Q}_t$ changes during 5 and 10 cm H₂O O₂ PEEP, the mechanism for \dot{Q}_s/\dot{Q}_t and PaO₂ improvement at these O₂ PEEP levels must have been due to oxygenation of the blood perfusing the nonventilated lung (apneic oxygenation). Although we did not measure differential lung volumes or lung O₂ uptake, we attribute the easily achieved transfer of oxygen at 5 and 10 cm H₂O O₂ PEEP to the fact that PEEP was initiated during the deflation phase of a large tidal ventilation (20 ml/kg) and thereby was able to maintain the preexisting patency of airways. Since the PEEP was maintained by an inflow of fresh O₂, the alveoli were able to participate in continuous O₂ exchange. Ten cm H₂O N₂ PEEP may have maintained the patency of airways but could not, of course, contribute to oxygen transfer. We speculate that low levels of O₂ PEEP would not be as efficacious if applied following a period of respiratory quiescence, for under these circumstances previously closed airways might not open and allow transfer of oxygen.

Since there were significant $\dot{Q}_{\text{test}}/\dot{Q}_t$ changes with 15 cm H₂O O₂ PEEP, at least two mechanisms for \dot{Q}_s/\dot{Q}_t and PaO₂ improvement were likely operative at this PEEP level. First, it is likely that 15 cm H₂O O₂ PEEP allowed O₂ exchange in the nonventilated lung by holding open airways and alveoli, as with 5 and 10 cm H₂O O₂ PEEP. Second, the blood flow which was diverted from the nonventilated to the ventilated lung must have participated in gas exchange in the ventilated lung. Since there were no differences in \dot{Q}_s/\dot{Q}_t and PaO₂ during 5

and 10 cm H₂O O₂ PEEP and 15 cm H₂O O₂ PEEP there must not have been any significant difference in the efficiency or amount of oxygen transferred to the blood perfusing the PEEP distended nonventilated lung compared to the blood perfusing the normally ventilated lung. The unchanged Pa_{CO₂} during steps 2-14 underscores the expected efficiency of the single ventilated lung to remove CO₂.¹⁴

Is there an optimal level of PEEP that should be used? There was no difference in the efficacy (decreased \dot{Q}_s/\dot{Q}_1 , increased Pa_{O₂}) between the various levels of O₂ PEEP tested. A high level of O₂ PEEP, however, might be associated with surgically bothersome lung distension. Thus, the use of surgical packs and retractors and high levels of PEEP are probably incompatible maneuvers. In addition, although cardiac output was not significantly changed in our study, a high level of PEEP might cause cardiovascular depression in some patients. A low level of PEEP would, therefore, appear to be preferable to a high level of PEEP in most instances. Since all levels of O₂ PEEP alone improved Pa_{O₂} and \dot{Q}_s/\dot{Q}_1 more than PAB inflation alone, and PAB inflation necessitates potential hazards associated with pulmonary artery catheterization and manipulation, low levels of O₂ PEEP appears much preferable to PAB inflation in the vast majority of clinical situations. PAB flow diversion alone may be uniquely useful in situations in which O₂ PEEP to the nonventilated lung interferes with surgical resection, or is impractical, as in massive pulmonary hemorrhage, or is impossible, as in pulmonary lavage.

Finally, what is the effect of combining nonventilated lung PAB inflation and O₂ PEEP together? We found that inflation of the PAB in the nonventilated lung during each level of O₂ PEEP tested did not cause any significant change in Pa_{O₂} and \dot{Q}_s/\dot{Q}_1 even though $\dot{Q}_{\text{test}}/\dot{Q}_1$ was decreased significantly. Our findings demonstrate that the precise partitioning of pulmonary blood flow between the nonventilated and ventilated lung does not matter as long as the blood flow to each lung can participate in the uptake of alveolar oxygen. In this sense, neither PAB inflation nor 15 cm H₂O O₂ PEEP are beneficial additions to 5 or 10 cm H₂O O₂ PEEP with PAB down. Additionally, insufflation of oxygen to the atelectatic nondependent lung at ZEEP failed to increase Pa_{O₂} and decrease \dot{Q}_s/\dot{Q}_1 probably because many nonventilated lung airways were collapsed and the insufflated oxygen failed to reach the alveolar gas exchanging regions.

In summary, we found that the insufflation of O₂ at ZEEP was not beneficial during one-lung ventilation. In contrast, both nonventilated lung O₂ PEEP and PAB inflation were efficacious during one-lung ventilation; however, the former maneuver improved Pa_{O₂} and \dot{Q}_s/\dot{Q}_1 more so than the latter. The probable mechanisms of efficacy of these nonventilated lung manipulations are: low levels of O₂ PEEP maintain the patency of nonventilated lung airways and thus permit gas exchange; high levels of O₂ PEEP act by this mechanism as well as by causing blood flow diversion to the ventilated lung; PAB inflation causes blood flow diversion to the ventilated lung.

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