Anesthesiology
55:376–380, 1981

Unilateral Lung Lavage: Blood Flow Manipulation by Ipsilateral Pulmonary Artery Balloon Inflation in Dogs

David D. Alfery, M.D., Barry G. Zamost, M.D.,* Jonathan L. Benumof, M.D.†*

The authors attempted to favorably manipulate the distribution of pulmonary blood flow during unilateral atelectasis and during unilateral lung lavage by nonocclusive inflation of an ipsilateral pulmonary artery catheter balloon (PAB). Six mongrel dogs were anesthetized, intubated with a double-lumen endotracheal tube, and following a thoracotomy, pulmonary artery and left lung blood flows ($\dot{Q}_t$ and $\dot{Q}_{LL}/\dot{Q}_t$, respectively) were measured electromagnetically; right lung blood flows ($\dot{Q}_{RL}/\dot{Q}_t$) were derived by difference. A PAB was positioned in the right main pulmonary artery. The experimental sequence consisted of seven steps: 1) both lungs ventilated; 2) ventilation of the left lung (LL vent), right lung (RL) atelectatic, PAB deflated; 3) LL vent, RL atelectatic, PAB inflated; 4) LL vent, RL lavaged, PAB deflated; 5) LL vent, RL lavaged, PAB inflated; 6) LL vent, RL drained, PAB deflated; and 7) LL vent, RL drained, PAB inflated. At each step the shunt fraction ($\dot{Q}_s/\dot{Q}_t$) was determined. Inflation of the PAB during LL vent and RL atelectatic (step 3) caused $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$ to decrease and $\dot{Q}_{LL}/\dot{Q}_t$ and Pa_{O_2} to increase significantly (compared to step 2). There were no significant differences in Q_{RL}/Q_t, $\dot{Q}_{LL}/\dot{Q}_t$, $\dot{Q}_s/\dot{Q}_t$, and Pa_{O_2} during RL lavage with the PAB deflated (step 4) compared to RL lavage with the PAB inflated (step 5). Inflation of the PAB during RL drainage (step 7) caused $\dot{Q}_{RL}/\dot{Q}_t$ to decrease and $\dot{Q}_{LL}/\dot{Q}_t$ to increase significantly compared to their values during periods of RL drainage with the PAB deflated (step 6). This resulted in a significant increase in Pa_{O_2} and decrease in $\dot{Q}_s/\dot{Q}_t$. These results demonstrate that the distribution of pulmonary blood flow in dogs can be favorably manipulated by nonocclusive ipsilateral PAB inflation and support a trial of use in selected patients during one-lung anesthesia and ventilation. (Key words: Equipment: catheters, pulmonary artery. Hypoxia: hypoxic pulmonary vasoconstriction. Lung: atelectasis; blood flow; lavage; pulmonary artery; shunting. Oxygen: blood levels.)

UNILATERAL LUNG LAVAGE has been employed with good success for the removal of lipoproteinaceous material from severely infiltrated alveoli in patients with alveolar proteinosis.[1-9] Infrequently, lung lavage may be performed on patients with asthma[4,7,8] and cystic fibrosis.[1,7,10] Unilateral lung lavage is most often performed under general anesthesia, and in every case a double-lumen endotracheal tube and one-lung ventilation are utilized.

During periods of lung lavage, lavage fluid infusion pressure (the alveolar pressure of the lavaged lung [P_A]) usually equals or exceeds the pulmonary artery pressure (PAP), and nonventilated (lavaged) lung blood flow is diverted, in part, to the ventilated lung.[8,9] Conversely, during periods of lung drainage, PAP exceeds P_A, and nonventilated lung blood flow, which is shunt flow, is reestablished. Thus, the degree of hypoxemia is greatest during the period of lung drainage.[1,2,7-9] We attempted to favorably manipulate the distribution of pulmonary blood flow in dogs during unilateral lung lavage (during periods of infusion and drainage) by nonocclusive inflation of an ipsilateral pulmonary artery catheter balloon (PAB). In addition, we examined the effects of ipsilateral PAB inflation on nonventilated (atelectatic) lung blood flow during one-lung ventilation.

Methods

Six mongrel dogs weighing 18–23 kg were anesthetized with 25 mg/kg pentobarbital, iv, intubated with a standard endotracheal tube, paralyzed with 1.0 mg/kg pancuronium, iv, and mechanically ventilated with 100 per cent O_2 by a dual-piston Harvard® respirator. The respiratory rate was adjusted to achieve a Pa_{CO_2} of 40 ± 3 torr (mean ± SE). A thoracotomy was performed through the left fifth intercostal space and the full length of the sternum. Electromagnetic flow probes (Statham® SP7515) were placed around the main (sizes 12 and 14 mm) and left main (size 8 mm) pulmonary arteries. All flow probes had been calibrated *in vitro* with known blood flows through excised vessels and the main pulmonary artery flow probe calibrations were confirmed *in vivo* with simultaneous thermodilution cardiac outputs. Over the range of 1000–3000 ml/min the main pulmonary artery flow probe reading was always ≤ ± 6 per cent of the simultaneously determined thermodilution cardiac output. Right main pulmonary artery blood flow was calculated as the difference between the directly measured main and left main pulmonary artery blood flows. Through a conventional tracheostomy a left sided double-lumen endotracheal tube was passed (Raush® #382050) and directly seated and tied into the left mainstem bronchus by hand from within the chest.

A balloon-tipped pulmonary artery catheter (PAB) was positioned in the right main pulmonary artery (fig. 1) and the location of the PAB confirmed by palpation. Mean pulmonary artery pressure (PAP) was always measured from the distal port of this catheter. Left atrial pressure (LAP) was measured by a catheter inserted into the left atrium via the left atrial appendage and the correct location of this catheter was confirmed by palpation.

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* Fellow in Anesthesia.

† Associate Professor of Anesthesia.

Received from the Department of Anesthesia, the University of California, San Diego, La Jolla, California 92093. Accepted for publication March 18, 1981. Supported by NIHGMS #24674A.

Address reprint requests to Dr. Jonathan L. Benumof: Anesthesia Research Laboratory T-001, University of California, San Diego, La Jolla, California 92093.

Airway pressure (P_A) was measured at the carina by a catheter within the tracheal tube. The PAP, LAP and P_A catheters were zeroed at the vertical level of the left atrium. Temperature and systemic arterial pressure were also continuously measured and all pressures and blood flows were continuously recorded (Hewlett Packard® eight-channel recorder). During the experimental sequence steps in which the PAB was inflated (see below), 1.0 ml of air was always used for inflation and the PAP trace always retained a phasic, but diminished in amplitude (decreased pulse pressure) character.

The experimental sequence was performed in the supine position and consisted of seven steps: 1) both lungs ventilated; 2) ventilation of the left lung (LL vent), right lung (RL) atelectatic (induced by O_2 absorption following clamping of right lung airway), PAB deflated; 3) LL vent, RL atelectatic, PAB inflated; 4) LL vent, RL lavaged, PAB deflated; 5) LL vent, RL lavaged, PAB inflated; 6) LL vent, RL drained, PAB deflated; and 7) LL vent, RL drained, PAB inflated. Steps 4-5 (RL lavaged) *vs.* 6-7 (RL drained) were performed in random order. During periods of lavage, fluid infusion pressure was constant at 23 torr (fig. 1). Arterial and mixed venous blood samples were taken 10 min after each step change. Blood O_2 contents were calculated from the measured oxygen partial pressures (Corning® 165 1/2) and hemoglobin concentrations (Coleman,® junior II) according to the formula:

$$O_2 \text{ content} = 1.39\ ([Hb])\ (\text{Per cent Sat}) + 0.0031(P_{O_2}).$$

Per cent saturation was determined from the oxygen-hemoglobin dissociation and was corrected for *p*H, P_{CO_2}, and temperature deviations from 7.4, 40 torr, and 37°C, respectively. Right-to-left shunt ($\dot{Q}_s/\dot{Q}_t$) was calculated by the Berggren formulation:

$$\dot{Q}_s/\dot{Q}_t = C\dot{c}_{O_2} - Ca_{O_2}/C\dot{c}_{O_2} - C\bar{v}_{O_2}.^{11}$$

End pulmonary capillary blood O_2 partial pressure ($P\dot{c}_{O_2}$) was calculated from:

$$P\dot{c}_{O_2} = P_{A_{O_2}} = (P_{atm} - 47)(1.0) - Pa_{CO_2}/R.$$

P_{atm} was measured daily (Sargent-Welch). Results are expressed as means ± SE and were analyzed by the F test, Student's paired *t* test, and with bivariable linear regression.

Results

The pulmonary blood flow distribution results are shown in figure 2. Prior to lung lavage, changing from two-lung ventilation (step 1) to only left lung ventilation (step 2) caused right lung blood flow ($\dot{Q}_{RL}/\dot{Q}_t$) and arterial oxygenation (Pa_{O_2}) to decrease ($P < 0.05$ and $P < 0.025$, respectively) and left lung blood flow ($\dot{Q}_{LL}/\dot{Q}_t$) and $\dot{Q}_s/\dot{Q}_t$ to increase ($P < 0.05$ and $P < 0.01$, respectively) significantly. Inflation of the PAB during LL vent and RL atelectatic (step 3) caused $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$ to decrease and $\dot{Q}_{LL}/\dot{Q}_t$ and Pa_{O_2} to increase significantly ($P < 0.01$) (compared to step 2).

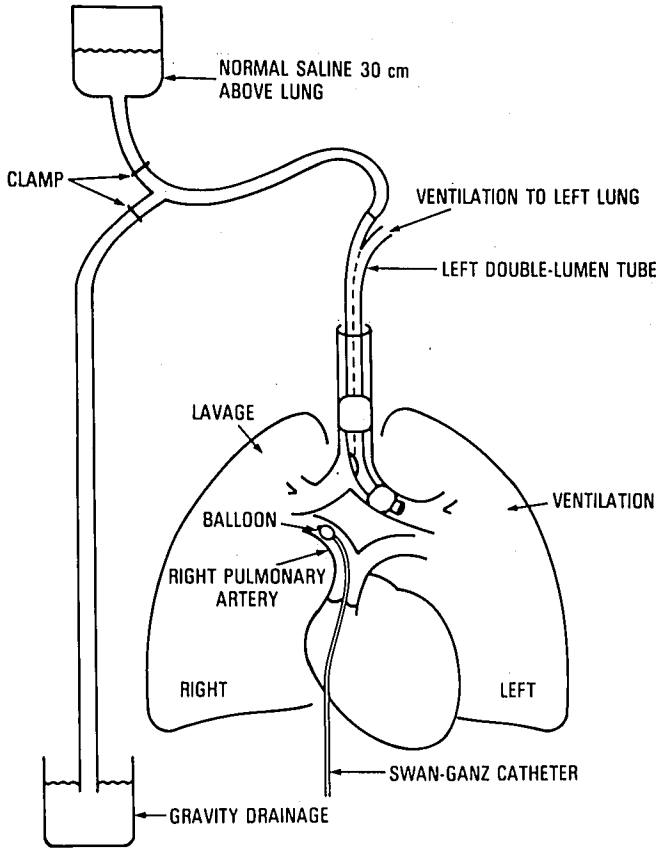

FIG. 1. Schematic of the experimental preparation. A canine double-lumen endotracheal tube allows ventilation of the left lung while the right lung is lavaged. Saline is infused at a constant pressure of 23 torr (30 cm H_2O) and then drained by gravity pressure. A balloon-tipped catheter is located in the right main pulmonary artery.

Right lung lavage caused a significant decrease in $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$ and a significant increase in $\dot{Q}_{LL}/\dot{Q}_t$ and Pa_{O_2} ($P < 0.001$; step 4 compared to step 3). There were no significant differences in $\dot{Q}_{RL}/\dot{Q}_t$, $\dot{Q}_{LL}/\dot{Q}_t$, $\dot{Q}_s/\dot{Q}_t$, and Pa_{O_2} during RL lavage with the PAB deflated (step 4) compared to RL lavage with the PAB inflated (step 5). With the PAB deflated, RL drainage (step 6) compared to RL lavage (step 4) caused $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$ to increase and $\dot{Q}_{LL}/\dot{Q}_t$ and Pa_{O_2} to decrease significantly ($P < 0.001$). Finally, inflation of the PAB during RL drainage (step 7) caused $\dot{Q}_{RL}/\dot{Q}_t$ to decrease and $\dot{Q}_{LL}/\dot{Q}_t$ to increase significantly ($P < 0.001$) compared to their values during periods of RL drainage with the PAB deflated (step 6). This resulted in a significant increase in Pa_{O_2} and decrease in $\dot{Q}_s/\dot{Q}_t$ ($P < 0.001$). All

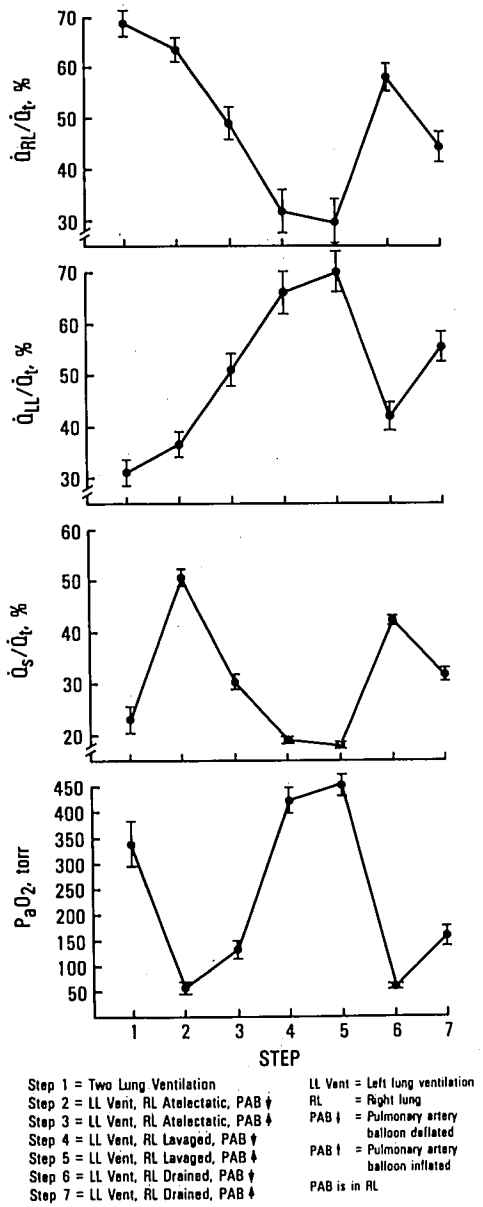

FIG. 2. Results of ipsilateral PAB inflation and deflation on right and left lung blood flows, transpulmonary shunt, and arterial oxygenation. See text for details.

single-step changes in $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$, (range of single-step change = 1–28 per cent) caused by atelectasis, PAB inflation and lung lavage and drainage correlated extremely well; r = 0.97 and $P < 0.001$.

Right lung lavage (step 4) caused a significant increase in PAP and decrease in Q_t ($P < 0.05$) compared to step 1 (table 1). During step 4 $\dot{Q}_{RL}/\dot{Q}_t$ was a linear function of PAP; $\dot{Q}_{RL}/\dot{Q}_t = 1.2$ (PAP) + 0.9, with r = 0.70 and $P < 0.025$. Inflation of the PAB (steps 3, 5, and 7) caused increases in PAP and decreases in cardiac output which were not significantly different from the immediately preceding steps. There were no significant changes in LAP during the seven method steps.

Discussion

Our experimental simulation of unilateral lung lavage used electromagnetic flow probes and $\dot{Q}_s/\dot{Q}_t$ determinations as the primary methods of evaluation of the effects of nonocclusive PAB inflation in the nonventilated lung. Specifically, we wished to correlate the changes in the distribution of pulmonary blood flow with changes in right-to-left shunt. In steps 2 through 7 the magnitude of our flow probe measurement changes agreed closely with our shunt measurement changes over a very wide range. Thus, we are confident that our flow probes recorded both the magnitude and direction of blood flow changes accurately.

In our experimental model, during steps 2–7 $\dot{Q}_{RL}/\dot{Q}_t$ should have been equal to or less than $\dot{Q}_s/\dot{Q}_t$, since it was possible for some $\dot{Q}_s/\dot{Q}_t$ to originate from the ventilated left lung as well as from Thebesian and bronchial veins. We found, however, that $\dot{Q}_{RL}/\dot{Q}_t$ was consistently greater (12 per cent to 18 per cent) than $\dot{Q}_s/\dot{Q}_t$; thus, the absolute magnitude of our $\dot{Q}_{RL}/\dot{Q}_t$ measurement was probably in error. Since the measurements necessary for the $\dot{Q}_s/\dot{Q}_t$ calculation are relatively straightforward and our $\dot{Q}_s/\dot{Q}_t$ values agreed well with our Pa_{O_2} values,[12] and because our main pulmonary artery flow probe agreed well with simultaneous *in vivo* thermodilution cardiac outputs, it is probable that our left main pulmonary artery flow probe caused the modest constant $\dot{Q}_{RL}/\dot{Q}_t$ error. The larger than normal $\dot{Q}_{RL}/\dot{Q}_t$ (69 per cent) that was found during control two-lung ventilation (step 1) indicates that the 8-mm left pulmonary artery flow probe was excessively constricting. This contention is supported by the fact that no discrepancies between $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$ occurred in an identical preparation in which a 10-mm left lung flow probe was used.[13] Thus, the *in vitro* calibrations for the 8-mm left main pulmonary artery flow probe were probably inaccurate *in vivo*, causing a constant $\dot{Q}_{RL}/\dot{Q}_t$ error. Since our conclusions do not depend on the exact partitioning of blood flow between each lung, but do depend on blood flow distribution *changes*, and both the magnitude and direction of the $\dot{Q}_{RL}/\dot{Q}_t$ changes provide strong confirmatory evidence for the mechanism of $\dot{Q}_s/\dot{Q}_t$ changes, we view the modest absolute magnitude error in left lung flow probe function as a relatively minor experimental design defect.

During periods of lavage, P_A clearly approximates the lavage fluid infusion pressure (23 torr).[8,9] If the lavage fluid pressure equals or exceeds PAP it should cause compression of the small intra-alveolar vessels in the lavaged lung and thereby divert blood flow to the ventilated lung.[14] However, during step 4 (LL vent, RL lavaged),

TABLE 1. Hemodynamic Changes during One-lung Ventilation, Lavage and Pulmonary Artery Balloon Inflation*

Method Step	PAP (torr)	LAP (torr)	$\dot{Q}_t$ (ml/min)
Step 1: two-lung ventilation	15.8 ± 2.7	6.2 ± 1.1	2010 ± 275
Step 2: LL ventilation, RL atelectasis, PAB ↓	15.8 ± 2.0	6.1 ± 0.9	2010 ± 399
Step 3: LL ventilation, RL atelectasis, PAB ↑	16.4 ± 2.5	6.1 ± 1.0	1941 ± 404
Step 4: LL ventilation, RL lavage, PAB ↓	25.3† ± 2.8	5.8 ± 1.1	1591† ± 224
Step 5: LL ventilation, RL lavage, PAB ↑	27.2† ± 3.1	5.7 ± 1.2	1387† ± 249
Step 6: LL ventilation, RL drainage, PAB ↓	17.3 ± 1.7	6.2 ± 1.4	1954 ± 281
Step 7: LL ventilation, RL drainage, PAB ↑	18.5 ± 2.3	6.2 ± 1.3	1885 ± 273

Values are means ± SE.
* PAP = pulmonary artery pressure; $\dot{Q}_t$ = cardiac output; PAB = pulmonary artery balloon; LL = left lung; RL = right lung.
† $P < 0.05$ compared to step 1.

PAP (25 torr) slightly exceeded right lung P_A (23 torr) and we therefore found a small, although greatly reduced, $\dot{Q}_{RL}/\dot{Q}_t$. The linear regression equation relating $\dot{Q}_{RL}/\dot{Q}_t$ to PAP during step 4 clearly shows that as PAP increased (relative to a constant right lung P_A), $\dot{Q}_{RL}/\dot{Q}_t$ increased significantly. Inflation of the PAB during periods of lung lavage (step 5) failed to decrease the minimal persisting step 4 $\dot{Q}_{RL}/\dot{Q}_t$. We speculate that PAP and $\dot{Q}_{RL}/\dot{Q}_t$ would be even greater during periods of PAB inflation and lung lavage if the ventilated left lung were diseased.

Conversely, during periods of lung drainage, PAP greatly exceeds P_A, and nonventilated lung blood flow, which is shunt flow, is reestablished. We found during step 6 (LL vent, RL drained) that PAP (17 torr) greatly exceeded right lung P_A (atmospheric) and caused a relatively normal $\dot{Q}_{RL}/\dot{Q}_t$. Thus, the period of highest $\dot{Q}_s/\dot{Q}_t$ and lowest Pa_{O_2} occurred during periods of lung drainage. Inflation of the PAB during periods of lung drainage (step 7), which presumably resulted in an incomplete mechanical vascular obstruction, caused a dramatic, significant reduction in $\dot{Q}_{RL}/\dot{Q}_t$ and $\dot{Q}_s/\dot{Q}_t$ and a greater than twofold increase in Pa_{O_2}.

Prior to performing lung lavage, steps 1, 2, and 3 were performed to evaluate the effect of an ipsilateral PAB inflation on nonventilated (atelectatic) lung blood flow. The change from two-lung ventilation (step 1) to left lung ventilation alone (step 2) resulted in an expected decrease in $\dot{Q}_{RL}/\dot{Q}_t$, presumably by the mechanism of hypoxic pulmonary vasoconstriction (HPV).[15] The decrease in $\dot{Q}_{RL}/\dot{Q}_t$ was only modest and was consistent with previous single-lung HPV experience.[15] Additionally, the small single-lung HPV response may have been due to the fact that both lungs had been manipulated and the right lung was made atelectatic only one time; the magnitude of HPV is known to increase following repeated hypoxic challenges.[16,17] Indeed, $\dot{Q}_{RL}/\dot{Q}_t$ was less during step 6 compared to step 2. One-lung ventilation was accompanied by an expected increase in $\dot{Q}_s/\dot{Q}_t$ and decrease in Pa_{O_2}. Inflation of the PAB in the nonventilated lung (step 3) caused Pa_{O_2} to markedly improve and $\dot{Q}_s/\dot{Q}_t$ to decrease as a result of a further reduction in $\dot{Q}_{RL}/\dot{Q}_t$.

The risks and benefits of PAB inflation in a lavaged or atelectatic lung in humans must be considered. The risk of pulmonary artery catheter insertion for primarily cardiac function measurements are low and well-documented;[18] the risk of pulmonary artery catheter insertion for the purpose of pulmonary blood flow manipulation should be no different. The risk of short periods of PAB inflation in terms of pulmonary vascular rupture, pericatheter clotting, and possible pulmonary embolization should be no different than, and perhaps less than those associated with pulmonary artery occlusion pressure measurements, since we always retained a slight phasic character on the PAP trace.

Under the conditions of our experiment, the benefits of PAB inflation are a decreased $\dot{Q}_s/\dot{Q}_t$ and an increased Pa_{O_2}. These changes probably occur quite rapidly since the pulmonary blood flow distribution change is almost immediate. However, we speculate that the increase in Pa_{O_2} caused by PAB inflation in humans would be less than we found in our animals since the degree of nonventilated lung vascular obstruction would be less in the larger human vessels. In some patients undergoing one-lung ventilation the ventilated lung may be abnormal.

In these patients, even when ventilation to the remaining lung and cardiac output are optimal, hypoxemia may be severe. Under these circumstances, ipsilateral PAB inflation may be the only available means of increasing Pa_{O_2}.

The authors acknowledge the technical assistance of Frank Trousdale.

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