Mechanism of lobar alveolar pressure decline during forced deflation in canine regional emphysema

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Mink, S. N. Mechanism of lobar alveolar pressure decline during forced deflation in canine regional emphysema. J. Appl. Physiol. 82(2): 632–643, 1997.—A canine model of unilobar papain-induced emphysema was used to examine the extent to which differences in alveolar pressures (PA) would develop between an emphysematous right lower lobe (RLL) and normal left lower lobe (LLL) during forced vital capacity (FVC) deflation. RLL and LLL PA (PARLL and PALLL, respectively) were measured by the alveolar capsule technique. During forced deflation, PA and lobar flows were determined between 95 and 20% FVC. A choke point common to both lower lobes was observed at >40% FVC. The results showed that deflation compliance (C) was altered for the RLL such that <90% lobar vital capacity, PARL > PALL, whereas >90% lobar vital capacity, PARL < PALL. At 95 and 90% FVC, the initial RLL PA decline was greater than that for the LLL (P < 0.05). However, large differences in PA were prevented because of the effect of interdependence of regional expiratory flow (IREF). IREF caused a relative decrease in RLL flows and increase in LLL flows that limited PA differences. Between 80 and 50% FVC, as PARL became greater than PALL, and because of the initial effect of IREF, PARLL was ~PALLL. At ≤40% FVC, without IREF, lobar differences in PA widened. These findings indicate that IREF may affect the dynamics of flow limitation in regional lung disease.

maximum expiratory flow; nonuniform emptying; flow limitation

IN PULMONARY EMPHYSEMA, it is often observed that alveolar destruction occurs nonhomogeneously throughout the lung and that some lung regions are severely diseased, whereas other regions are relatively spared (1, 18). This nonuniform process results in a lung in which mechanical properties are variable between regions. Lung mechanical properties, such as recoil pressure (Pel), frictional resistance, and bronchial pressure-airway behavior are the primary determinants of maximal expiratory flow (Vmax) (8). Whereas alveolar destruction in emphysema characteristically results in a decrease in Pel and an increase in compliance over most of the vital capacity (VC) (1, 18), it was previously demonstrated in a papain model of emphysema that at very high lung volumes [VL; i.e., >90% total lung capacity (TLC)], compliance was actually lower than that found in healthy lungs (10, 11, 14). At these very high VL, it appears that nonelastic collagen is the predominant factor limiting further alveolar distension in this model, and therefore a relative lower compliance is found. Accordingly, the shape of the static pressure-volume curve is altered in papain-induced emphysema so that at very high VL compliance is lower, whereas over the remaining volumes, compliance is greater compared with values obtained from healthy lungs.

Such changes in compliance in regional emphysema would be expected to produce markedly nonuniform alveolar pressures (PA) during forced deflation. During the initiation of expiration, because compliance at very high VL is lower in emphysematous lung, the fall in PA (Pel + pleural pressure) of emphysematous regions may occur to a greater extent than that of normal lung units. On the other hand, with continuing deflation, compliance would relatively increase in emphysematous units so that, at low VL, PA would eventually rise to exceed that of normal lung units.

Whereas alterations in lung compliance in regional emphysema would provide the mechanism by which differences in PA would develop between regions, because fractinal resistance does not appreciably increase in this model (10), it has been proposed that opposing this development of nonhomogeneous deflation would be the effect of regional interdependence of expiratory flow (IREF). Wilson et al. (20) proposed that if regions shared a common site of flow limitation (i.e., choke point), then there would be interdependence of flow between these regions. If differences in PA developed between regions, then the units with the higher PA would relatively increase their contribution to total flow to compensate for units with the lower recoil. Because flow would relatively increase from the region with the higher recoil, the fall in PA in this region would occur to a greater extent than without this compensation, and lung deflation would, to some degree, remain homogeneous. However, the extent to which IREF may be important in nonuniform lung disease to preserve homogeneity of PA has not been experimentally shown.

In the present study, lobar deflation was examined in a regional model of papain-induced emphysema (11). The alveolar capsule technique of Fredberg et al. (3) was used to monitor PA of the right lower lobe (PARLL) and left lower lobe (PALLL) during a forced VC maneuver (4, 5, 7, 13, 19). Emptying of the normal and emphysema lobes was compared at different fractions of the VC. The objectives were to determine how regional emphysema altered Vmax and parameters of flow limitation and to assess the extent to which IREF contributed to these findings.

METHODS

Unilobar emphysema model. The details of this canine lobar emphysema model have previously been described and will only be briefly delineated here (10, 11, 14). Unilobar emphysema was produced in 12 mongrel dogs (20–30 kg) by the instillation of the enzyme papain into the RLL on four occasions ~2 wk apart. On the basis of measurements obtained in previous studies in which this model was used, although Pel is altered in this model, lobar frictional resis-
Fluorescence is not appreciably increased. Moreover, a central choke point can be identified at the trachea at the high $V_l$ (>40% VC) in most dogs, whereas at lower $V_l$, choke points are identified at lobar bronchi; these locations approximate those found in normal dogs. Because flow limitation occurs at a common airway (i.e., the trachea) over a large part of the VC in this model, it was therefore possible to assess the extent to which interdependence of flow between the emphysematous RLL and normal LLL may contribute to the findings during forced deflation.

During the papain instillation, the animals were anesthetized with pentobarbital sodium (30 mg/kg) and were placed in the supine position. A flexible bronchoscope was passed into the trachea and advanced down the right lung until the bronchopulmonary segments of the RLL were visualized. A solution that contained ~2.5 ml of the enzyme papain (type IV, Sigma Chemical, St. Louis, MO) mixed in ~25 ml of normal saline was placed into a localized area of the RLL such that, after the fourth instillation, the entire lobe would be injured.

After each of the four instillations, the animal was ventilated for 6–7 h with its right side maintained in the dependent position and with its head slightly elevated to prevent the papain mixture from spilling to the other lung lobes. When the papain is given in this manner, it has been observed that a rather diffuse unilobar emphysematous lesion is produced (14). The animals were returned to their cages when stable.

In six other control dogs, 25 ml of normal saline solution rather than the papain mixture were administered into the RLL at similar time intervals. The dogs were randomly allocated to either of the two groups.

Animal preparation. The basic methods and preparation were similar to those previously described (4–6, 9, 10, 15). On the day of the study, the animal was anesthetized with pentobarbital sodium (30 mg/kg) and was placed in the supine position. The chest was widely opened, and the trachea was cannulated with a large-bore steel tube that just entered the thoracic cavity. The animal was heparinized and phlebotomized, after which the heart was carefully removed. It has previously been shown that lung mechanics in this protocol are stable.

Measurements were obtained with the animal placed into a pressure-corrected volume-displacement plethysmograph (4–6, 9, 10, 15). $V_l$ were measured by a Krogh spirometer, and flow was measured by a pneumotachygraph (Fleisch no. 4) mounted between the plethysmograph and spirometer. Pressure at the airway opening ($P_{ao}$) was referenced relative to plethysmographic box pressure to obtain transpulmonary pressure ($P_{tp}$), which was measured, in turn, with a differential pressure transducer (MP-45, Validyne, Northridge, CA). The lungs could be inflated from a positive-pressure source with air or forcibly deflated (~100 to ~200 mmHg) by a negative-pressure reservoir attached to the airway opening. The frequency response of this system has been found to be adequate in phase and amplitude and has previously been described (9, 15).

The technique of Fredberg et al. (3) was used to measure $P_{ao}$ (7, 19). A pressure capsule (13-mm surface diameter) with a 5-mm hole, continuous with a 5-mm threaded sleeve, was glued to the parenchymal surface of each of the lower lobes. The lung parenchyma visible through the hole in the capsule was punctured with a small needle. A miniature differential pressure transducer (8510B; Endevco, San Juan Capistrano, CA) was screwed into the threaded sleeve of the capsule. $P_{ao}$ was recorded on an oscillograph and displayed on a storage oscilloscope (Tektronix, Beaverton, OR).

A Pitot static tube (1.5-mm diameter and 2.5-cm length) was used to locate airway sites of flow limitation (choke point) over the VC deflation (4, 6, 9, 10, 13, 16). The objective of the Pitot static measurement was to determine whether choke points were identified in an airway that was common to the RLL and LLL over most of the VC or, alternatively, whether individual lobar choke points would be found. Two polyethylene tubes (Intramedic PE-205, 1.6-mm inner diameter, 65-cm length, Parsippany, NJ), with numbered markings to identify airway locations of interest, were attached to the respective lateral and end-on ports of the Pitot static tube, whereas the other ends were connected to individual pressure transducers (Validyne MP-45).

The Pitot static tube was advanced down the airway by a thread attached to the front end of this device. The other end of the thread was pulled out the pleural surface of the RLL as previously described (4–6, 9, 10, 13). After a given $V_l$, choke point location was identified by the following criteria (6, 9, 15). The lateral port of the Pitot static tube was positioned at an airway site where lateral pressure ($P_{lat}$) did not vary with negative $P_{ao}$, but slightly downstream $P_{lat}$ decreased abruptly and varied with negative $P_{ao}$. $P_{ao}$ measured at the choke point was termed $P^*$; total or end-on pressure at choke point was termed $P_{end}^*$.

Airway pressure losses due to convective acceleration ($P_{ca}$) were calculated as the difference between $P_{end}^*$ and $P_{lat}^*$ (4–6, 9, 10, 14, 16). The Bernoulli equation [$P_{ca} = 1/2 \rho V^2$], where $\rho$ is gas density (1.12 x 10$^{-3}$ gm/cm$^3$), and $V$ is the flow subtended by the Pitot-static tube was used to determine cross section at choke point (termed $A^*$). At $V_l$ at which a central choke point was found in the trachea (see results), the flow subtended by the Pitot static tube was total flow. When lobar choke points were identified, the flow subtended was from the lower lobe (see calculation below). At a given $V_l$, frictional resistance to the RLL lobar bronchus was calculated from ($P_{ao} - P_{end}^*)/V$, where $V$ is the subtended flow and $P_{ao}$ is lobar end-on pressure.

The protocol was as follows. The lungs were twice inflated to TLC ($P_{tp} \sim 30$ cmH$_2$O) to standardize for volume history. After the third inflation, the airway was opened to the negative pressure reservoir, which forcibly deflated the lungs. Volume-time, and $P_{ao}$ were recorded at 200 mm/s on the oscillograph, whereas flow and $P_{ao}$ could be plotted as a function of volume on the oscilloscope. The Pitot static tube was pulled down the airway to identify choke point locations over the course of the VC deflation.

After whole lung Vmax-volume curves were performed, quasi-static capsular pressure-volume curves were determined from the RLL and LLL, during which airways to all lobes except the lobe of interest were transiently occluded with cotton tape (4–6, 13). Moreover, over a range of lobar volumes (30, 50, and 75% lobar VC), it was determined that capsular pressures measured from the respective lobes were the same during static and dynamic deflations and that static pressures measured by capsular pressure and $P_{ao}$ were also the same. This allowed one to relate capsular pressure to alveolar volume during static and dynamic measurements (4, 6, 13, 19).

$P_{ao}$ obtained during the deflations were differentiated with respect to time ($dP_{ao}/dt$) at the specific alveolar volumes analyzed in the individual experiments (4–6, 7, 13). Multiplying $dP_{ao}/dt$ by the slope of the static volume-pressure curve ($dV/dP_{ao}$) measured at the same absolute volume or $P_{ao}$ for each lobe allowed computation of lobar Vmax (Vmax); $[(dP_{ao}/dt) \times (dV/dP_{ao})] = dV/dt$. When this method has been used in previous studies, the agreement between measured and calculated values has been reasonably good (4, 13).
In the respective emphysema and control groups (see results), a two-way analysis of variance (ANOVA) for two repeated measures (within-within ANOVA) was used to assess differences between RLL and LLL parameters. The interaction between factor A (i.e., lobes) and factor B (i.e., either time from deflation or percent whole lung VC) was also determined. If a significant interaction was present, then the specific results were analyzed by paired \( t \)-test in which a Bonferroni correction was used for multiple comparisons. When parameters between groups were compared, a two-way split-plot ANOVA (between-within) or unpaired \( t \)-test (corrected for multiple comparisons) was used. Results are reported as means \( \pm \) SE.

RESULTS

A central choke point was found in 9 of the 12 emphysema experiments. In these nine experiments, a choke point was identified at the trachea at \( V_l > 40\% \) whole lung VC and at lobar bronchi at the lower \( V_l \). These nine dogs were analyzed together and constituted the emphysema group. In the other three dogs, no central choke point was found, and these three dogs were analyzed as additional emphysema experiments. The six dogs in which normal saline was instilled constituted the control group. In the control group, choke points at the respective \( V_l \) were identified at locations similar to those described in the emphysema group. Whole lung VC measured 2.2 \( \pm \) 0.23 liters in the emphysema group vs. 2.1 \( \pm \) 0.17 liters in the control group.

In all groups, there was no difference in lobar pressure-volume behavior when curves were obtained statically and dynamically. The dynamic lobar pressure-volume curves are shown in Fig. 1 during which all the other lobes were tied off. In the emphysema group (A), RLL volume was greater than LLL volume at all levels of \( P_A \). On the other hand, in the control group (B), pressure-volume curves were not different between the lower lobes. Although the dogs were randomized according to body weight, lobar volumes in the control group probably started out slightly larger than those in the emphysema group. This accounts for the slightly higher mean LLL volume in the control group, which was not significantly different between groups.

In the control and emphysema groups, lobar compliances were determined over multiple intervals of the pressure-volume curve. These results are shown in Table 1. In the emphysema group, for the emphysematous RLL, between 0 and 5 cmH\(_2\)O, lobar compliance was significantly higher than that found for the LLL, whereas it was significantly lower, between 10 and 20 cmH\(_2\)O and between 20 and 30 cmH\(_2\)O, respectively. In the control group, there were no differences in compliances between RLL and LLL in any intervals of pressure-volume curve.

Figure 2 shows a dynamic \( P_A \) vs. time curve obtained for a control (B; dog 4) and an emphysema dog (A; dog 6).

The mean values are shown in Fig. 3. At TLC, mean \((\pm \text{SE})\) \( P_{\text{RLL}} \) and \( P_{\text{ALL}} \) in the emphysema group averaged 30.4 \( \pm \) 0.4 and 30 \( \pm \) 1.2 cmH\(_2\)O, respectively, whereas corresponding values in the control group were 30.4 \( \pm \) 1.4 vs. 30 \( \pm \) 1.6 cmH\(_2\)O. In the emphysema group, over the course of deflation, there was significant interaction \((P < 0.01)\) between \( P_A \) and time. At 25 ms postdeflation, mean \( P_{\text{RLL}} \) were lower than \( P_{\text{ALL}} \). Over the remaining course of the deflation, \( P_{\text{RLL}} \) rose relative to \( P_{\text{ALL}} \). The LLL had emptied in all experiments by ~350 ms, whereas the RLL continued to empty in most experiments. In the control group, there was no difference in volume between lobes at a given pressure. *Significantly different RLL vs. LLL, \( P < 0.05 \) [2-way within-group analysis of variance (ANOVA)]. Between emphysema and control groups at all pressures, respective differences in RLL and LLL volumes were statistically significant \((P < 0.05)\) by between-groups ANOVA.

![Graph showing dynamic \( P_A \) vs. time curve](image)

**Fig. 1.** Lobar alveolar pressure \((P_A)\)-volume curves for right (RLL; solid line) and left lower lobes (LLL; dotted line) in control (B; \( n = 6 \) dogs) and emphysema groups (A; \( n = 9 \) dogs). Values are means \( \pm \) SE.

**Fig. 4A.** Dynamic \( P_{\text{RLL}} \) and \( P_{\text{ALL}} \) measured in the emphysema group are plotted as a function of the whole
lung VC. By two-way ANOVA, there was a significant interaction ($P < 0.0001$) between lobar PA (i.e., factor A) and VL (i.e., factor B). Interaction was examined to determine whether the relationship of PA to whole lung VC changed during the course of deflation. Interaction should be distinguished between the decreases in PA that occurred as VL decreased during deflation, which is given by factor B and which was statistically significant in both groups.

In the emphysema group (see Fig. 4A), at very high VL (i.e., 95 and 90% VC), $P_{\text{RLL}}$ were lower than $P_{\text{LLL}}$; that occurred as VL decreased during deflation, which is given by factor B and which was statistically significant in both groups.

### Table 1. Lobar compliances in emphysema and control groups

<table>
<thead>
<tr>
<th>Transpulmonary Pressure Range</th>
<th>Emphysema group (n = 9)</th>
<th>Control group (n = 6)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>RLL, l/cmH$_2$O</td>
<td>LLL, l/cmH$_2$O</td>
</tr>
<tr>
<td>0–5 cmH$_2$O</td>
<td>0.120 ± 0.02*†</td>
<td>0.026 ± 0.005</td>
</tr>
<tr>
<td>5–10 cmH$_2$O</td>
<td>0.262 ± 0.005</td>
<td>0.041 ± 0.008</td>
</tr>
<tr>
<td>10–20 cmH$_2$O</td>
<td>0.01 ± 0.002*†</td>
<td>0.022 ± 0.005</td>
</tr>
<tr>
<td>20–30 cmH$_2$O</td>
<td>0.0034 ± 0.0002*</td>
<td>0.007 ± 0.002</td>
</tr>
</tbody>
</table>

Values are means ± SE. RLL and LLL, right and left lower lobes, respectively. *Significantly different RLL vs. LLL within a group, $P < 0.05$ (paired t-test corrected for multiple comparisons). †Significantly different between RLL vs. LLL groups, $P < 0.05$ (unpaired t-test corrected for multiple comparisons in which respective differences in RLL and LLL compliances were compared).
at VL =< 40, whole lung VC and Vmax obtained in the emphysema group were significantly lower than control group values.

Figure 7 shows the lobar flows calculated in the emphysema and control groups over the intervals of the whole lung VC. In the emphysema group, at 95 and 90% whole lung VC, RLL flows were less than LLL flows, whereas combined RLL and LLL flows were not different between groups. On the other hand, at VL =< 40% whole lung VC, RLL flows were greater than LLL values. In the control group, there were no differences in flows between lobes measured over the whole VC range.

At 95 and 90% whole lung VC, because in the emphysema group the respective P_{RLL} were lower than P_{LLL}, it was determined whether the reductions in RLL flows observed at these VL were appropriate for the corresponding lower PA. In this analysis, lobar over the middle range of VL (i.e., 80 to 50% VC), P_{RLL} were similar to P_{LLL}; and at the lower VL, P_{RLL} were higher than P_{LLL}. In the control group (see Fig. 4B), there was no interaction between PA and VL, and P_{RLL} were similar to P_{LLL} at all VL examined.

In Fig. 5A, dPA/dt obtained in the emphysema group are plotted as a function of whole lung VC, and there was a significant interaction between dPA/dt and VL. In the emphysema group, at VL =< 40, whole lung VC and dPA/dt for RLL were significantly higher than dPA/dt for LLL. These findings were significantly different from those observed in the control group, in which there was no interaction between dPA/dt and VL.

In Fig. 6, whole lung maximum expiratory flow (Vmax) are plotted for the emphysema and control groups over intervals of the VC. At VL =< 40%, whole lung VC and Vmax were similar between groups, while

**Fig. 4.** PA plotted against % whole lung vital capacity for control (B; n = 6 dogs) and emphysema (A; n = 9 dogs) groups. Values are means ± SE. For emphysema group, there was a significant interaction (P < 0.001) between PA obtained for RLL and LLL and lung volume. This interaction was not found for control group. Significantly different RLL vs. LLL: *P < 0.05 within a group (Bonferroni corrected paired t-test); †P < 0.05 between groups (Bonferroni corrected unpaired t-test in which differences in PA between lobes were compared in 2 groups).

**Fig. 5.** Rates of PA change with respect to time (dPA/dt) plotted against % whole lung vital capacity for control (B; n = 6 dogs) and emphysema (A; n = 9 dogs) groups. Values are means ± SE. For emphysema group, there was a significant interaction (P < 0.01 by ANOVA) between dPA/dt obtained for RLL and LLL and lung volume that was not found in control group. Significantly different RLL vs. LLL: *P < 0.05 (Bonferroni corrected paired t-test); †P < 0.05 (Bonferroni corrected unpaired t-test, in which differences between RLL and LLL dPA/dt in 2 groups were compared).
flows calculated at 95 and 90% whole VC in the emphysema group were compared with respective flows in the control group, in which the $P_A$ of the emphysema group were slightly greater or equal to those in the control group. If, for comparable PARLL in the emphysema and control groups, RLL flows in the emphysema group were still lower than control group values, then other factors (such as interdependence of regional expiratory flow; see DISCUSSION) would need to be considered to explain the lower RLL flows found in the emphysema group. In the emphysema group, (see Fig. 4) the mean $P_{RLL}$ measured at 95% was $\sim 13.5$ cmH$_2$O, which was comparable to the $P_A$ of 13 cmH$_2$O found at 90% whole lung VC in the control group. At 90% whole lung VC, $P_{RLL}$ in the emphysema group was $\sim 10$ cmH$_2$O, which was slightly higher than the 8 cmH$_2$O $P_{RLL}$ found at 80% whole lung VC in the control group. The corresponding RLL flows measured at 95 and 90% whole lung VC in the emphysema group were significantly reduced in emphysema group, $* P < 0.05$ (2-way ANOVA between groups). *Significantly different between groups, $P < 0.05$ (Bonferroni corrected unpaired t-test).

In both the control and emphysema groups, choke points were identified at the trachea for $V_L > 40$% whole lung VC, whereas for $V_L \leq 40$%, choke points were identified at approximately lobar bronchi. Table 2 shows the values of $A^*$, $P^*$, and $P_{end}^*$ obtained at the multiple $V_L$ in the emphysema and control groups. There were no differences in these parameters between the two groups. In addition, RLL frictional resistances (see Table 2) were also not different in the two groups, although in the control group, frictional resistances measured at $V_L \leq 40$% whole lung VC tended to be greater than those in the emphysema group (see DISCUSSION).

In three dogs with unilobar emphysema, a tracheal choke point was not identified (additional emphysema experiments) so that there was no interdependence of flow between lobes. There was no apparent difference in the degree of emphysema produced in these dogs compared with that found in the emphysema group, as determined by RLL vs. LLL volume and compliance changes.

Figure 8 shows the $P_{RLL}$ and $P_{A_{LLL}}$ vs. whole lung VC curves obtained in these three experiments. Unlike in the emphysema group, these relationships were inconsistent between experiments. In dogs 2 and 3,
choke points were identified at lobar bronchi between 95 and 20% whole lung VC. In contrast to the emphysema group in which, during the initiation of deflation, \( P_{ARLL} \) fell to a greater extent than \( P_{ALLL} \), in dogs 2 and 3 \( P_{ARLL} \) were greater than \( P_{ALLL} \) by \(-2 \) cmH\(_2\)O during the entire deflation period (see DISCUSSION). In dog 1, choke points were identified in mainstem bronchi at \( V_L >40\% \) whole lung VC. In a manner similar to that found in the emphysema group, in dog 1 during initial deflation \( P_{ARLL} \) fell more rapidly than \( P_{ALLL} \). However, unlike in the emphysema group, \( P_{ARLL} \) remained much lower than \( P_{ALLL} \) by \(-2 \) to \(-3 \) cmH\(_2\)O until very late in deflation (30\% whole lung VC), after which \( P_{ARLL} \) became \( >P_{ALLL} \).

The \( V_L \) at which \( P_{ARLL} \) became equal to \( P_{ALLL} \) was compared in the emphysema group and additional emphysema experiments. In the emphysema group, this \( V_L \) averaged 69 \(+4\% \) whole lung VC, whereas in the additional emphysema experiments it occurred at the end of the VC maneuver and averaged 10 \(+10\% \) whole lung VC (\( P < 0.001 \) between groups). Moreover, in the additional emphysema experiments, \( V_{max} \) at all \( V_L \) appeared lower than those measured in the control group and were significantly reduced at 95 and 90\% whole lung VC (see Table 3).

### DISCUSSION

In the emphysema group, the results showed that only at the extremes of the whole lung VC maneuver were \( P_A \) different between the emphysematous and normal lower lobes. During early expiration, because compliance of the emphysematous lobe was lower at high \( P_{tp} \), a gradient in \( P_A \) was quickly established between the lower lobes, and, at 95 and 90\% whole lung VC, \( P_{ARLL} \) were lower than \( P_{ALLL} \) (see Fig. 4). As deflation continued, \( P_{ARLL} \) rose relative to \( P_{ALLL} \), and between 80 and 50\% whole lung VC, \( P_{ARLL} \) approximated \( P_{ALLL} \). Eventually, however, \( P_{ARLL} \) became higher than \( P_{ALLL} \) so that, at \( \geq40\% \) whole lung VC, the expire came predominantly from the emphysematous RLL for the remainder of the deflation. Moreover, in the emphysema group, despite the changes in lobar \( P_A \) that occurred between lobes, \( V_{max,\text{tot}} \) were not different between the emphysema and control groups until the lower \( V_L \) were reached. The results obtained in the emphysema group are examined below in terms of possible mechanisms that would regulate emptying of the normal and emphysematous regions over the course of the VC maneuver in this model.

It could be argued that in the emphysema group, the changes in \( P_{tp} \) and flows that were observed between lobes during deflation reflected only the effect of the RLL compliance changes and that there was no evidence that flow interaction between lobes had contributed to these findings. In that case, compared with the LLL, because the compliance of the emphysematous RLL lobe was smaller at high \( P_{tp} \), \( P_A \) of the emphysematous lobe decreased faster during early deflation. However, because the compliance of the emphysematous lobe was relatively greater at low \( P_{tp} \), RLL emptying continued whereas that of the LLL had already ceased. There would be no need to invoke other mechanisms such as flow interdependence between regions to explain any of the findings observed in the emphysema group.

However, the results shown in Fig. 7, in which lobar flows are plotted as a function of \( % \) whole lung VC, contradict this view. In the emphysema group, compared with the values obtained in control group, there were marked changes in RLL and LLL flows at the high lung regions that would support a role for a flow interdependent mechanism between regions. The rationale for this conclusion is as follows.

As previously discussed, \( P_{ARLL} \) were less than \( P_{ALLL} \) during early expiration because of the lower compliance of the emphysematous lobe (see Fig. 4). Wilson et al. (20) indicated that when regions share a common downstream airway, flow from each region depends on the driving pressure for other regions, and flow from the region with the higher driving pressure is favored. For a mean \( P_A \) (averaged among the different regions), flows from the region with the higher \( P_A \) would increase, whereas flow from the other would decrease compared with results obtained when \( P_A \) were similar between regions. Solway et al. (17) showed similar results in a transistor model of nonhomogeneous airflow obstruction. When regions shared a common choke point, if flows from one region were reduced by an obstruction, then the other region would increase its flow rate. In my laboratory, a similar conclusion was

### Table 2. Choke-point variables in emphysema and control groups

<table>
<thead>
<tr>
<th>% Whole Lung Vital Capacity</th>
<th>95</th>
<th>90</th>
<th>80</th>
<th>70</th>
<th>60</th>
<th>50</th>
<th>40</th>
<th>30</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emphysema group</strong></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>( P^* ), cmH(_2)O</td>
<td>10.8±0.8</td>
<td>6.4±1.4</td>
<td>-1.0±1.4</td>
<td>-3.5±1.1</td>
<td>-5.0±1.1</td>
<td>-7.7±1.6</td>
<td>-3.7±1.4</td>
<td>-3.0±1.8</td>
<td>-2.5±1.1</td>
</tr>
<tr>
<td>( P_{END}^* ), cmH(_2)O</td>
<td>13.9±2.4</td>
<td>9.7±0.6</td>
<td>2.4±1.1</td>
<td>0.7±0.7</td>
<td>0.5±0.9</td>
<td>-0.9±0.9</td>
<td>0.4±1.0</td>
<td>-0.9±1.2</td>
<td>-1.0±1.0</td>
</tr>
<tr>
<td>( A^* ), cm(^2)</td>
<td>3.6±1.4</td>
<td>4.1±0.9</td>
<td>4.2±1.1</td>
<td>3.3±0.9</td>
<td>2.2±0.45</td>
<td>1.7±0.3</td>
<td>0.5±0.25</td>
<td>0.30±0.11</td>
<td>0.2±0.11</td>
</tr>
<tr>
<td>( R_{fr} ), cmH(_2)O·l·s(^{-1})</td>
<td>0.33±0.22</td>
<td>0.525±0.500</td>
<td>0.55±0.49</td>
<td>0.659±0.162</td>
<td>0.80±0.400</td>
<td>0.74±0.018</td>
<td>0.76±0.126</td>
<td>0.32±0.161</td>
<td>0.701±0.360</td>
</tr>
<tr>
<td><strong>Control group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( P^* ), cmH(_2)O</td>
<td>12.0±4.0</td>
<td>4.0±1.4</td>
<td>-2.3±3.0</td>
<td>-6.3±2.9</td>
<td>-7.6±2.7</td>
<td>-9.0±0.44</td>
<td>-6.9±2.3</td>
<td>-5.3±0.3</td>
<td>0.33±2</td>
</tr>
<tr>
<td>( P_{END}^* ), cmH(_2)O</td>
<td>15.8±2.7</td>
<td>7.8±13</td>
<td>0.8±2.5</td>
<td>-0.7±1.9</td>
<td>-2.1±2.3</td>
<td>-3.3±1.7</td>
<td>-3.2±2.0</td>
<td>-0.6±1.1</td>
<td>2.0±1.5</td>
</tr>
<tr>
<td>( A^* ), cm(^2)</td>
<td>3.9±0.9</td>
<td>5.6±1.4</td>
<td>3.0±0.6</td>
<td>2.9±0.6</td>
<td>2.7±0.35</td>
<td>2.4±0.35</td>
<td>0.4±0.37</td>
<td>0.30±0.5</td>
<td>0.5±0.75</td>
</tr>
<tr>
<td>( R_{fr} ), cmH(_2)O·l·s(^{-1})</td>
<td>0.35±0.37</td>
<td>0.59±0.392</td>
<td>0.55±0.120</td>
<td>0.60±0.119</td>
<td>0.70±0.01</td>
<td>0.731±0.86</td>
<td>0.176±0.867</td>
<td>0.32±0.85</td>
<td>1.78±1.10</td>
</tr>
</tbody>
</table>

Values are means ± SE; \( n = 5 \), 3, and 3 dogs in emphysema group at 40, 30, and 20\% whole lung vital capacity, respectively, and 5, 2, and 2 dogs in control group at 40, 30, and 20\% whole lung vital capacity, respectively. \( P^* \) and \( P_{END}^* \), lateral and end-on pressures at choke point, respectively; \( A^* \), cross section at choke point; \( R_{fr} \), frictional resistance to right lobar bronchus.
reached in a canine model of nonhomogeneous airflow obstruction (12).

In the emphysema group, at 95 and 90% whole lung VC, because a choke point was common to both lower lobes and because the downstream pressure (i.e., \( P^* \)) was the same for both lobes, in terms of the results of Wilson et al. (20) the LLL would be favored for flow because \( P_{\text{LLL}} > P_{\text{RLL}} \). Therefore, in the emphysema group, LLL flows were significantly greater than RLL flows at 95 and 90% whole lung VC (see Fig. 7).

Yet, in the emphysema group, how does one know whether this reduction in RLL flows would have occurred without invoking a mechanism such as IREF? That is, if flow from both lobes were not interdependent, and if \( P_{\text{RLL}} < P_{\text{LLL}} \) because of a lower RLL compliance during early deflation, then what would have been the resulting RLL and LLL flows in the emphysema group?

First of all, it can be observed in Fig. 7 that LLL flows obtained in the emphysema group at 95 and 90% whole lung VC were greater than corresponding LLL flows in the control group. In the canine lung, flows are fairly constant from TLC to ~50% VC (see Fig. 6) so that there would be few changes in LLL flows expected in the emphysema group for the small differences in \( P_{\text{LLL}} \) found between groups at these VL. Accordingly, why should LLL flows increase in the emphysema group if not due to a mechanism such as IREF?

Second, at 95% whole lung VC, \( P_{\text{RLL}} \) in the emphysema group averaged ~13 cmH\(_2\)O and RLL flows averaged ~2.5 l/s. On the other hand, in the control group, at 90% VC, \( P_{\text{RLL}} \) measured ~11 cmH\(_2\)O, and RLL flows measured ~5 l/s. Thus, for similar \( P_{\text{RLL}} \) measured in the two groups, RLL flows were much lower that those found in the control group. It appears that in the emphysema group, LLL flows increased by ~2 l/s (~30%), whereas RLL flows decreased by ~2 l/s, and these reciprocal changes are exactly what was predicted by Wilson et al. (20).

Moreover, Wilson et al. (20) showed that when resistance was unchanged between regions (see Table 2 at high VL), the relative flows between these regions \( (V_{\text{RLL}} - V_{\text{LLL}})/V_{\text{avg}} \) would be determined by their corresponding differences in PA \( ((P_{\text{RLL}} - P_{\text{LLL}}))/(P_{\text{avg}} - P^*)) \). In the emphysema group, \( \Delta P \) between lobes at 95% whole lung VC was nearly equal to ~3 cmH\(_2\)O (see Fig. 4), whereas \( P_{\text{avg}} - P^* \) was 15 – 10.8 = 4.2 cmH\(_2\)O (see Table 2). This would give a pressure ratio of ~0.71, which was slightly less than the measured flow ratio of ~0.88 calculated from the lobar flows in Fig. 7 [i.e., (2.5 – 6.5)/4.5]. Accordingly, the reciprocal changes in LLL and RLL flows observed in the emphysema group are in agreement with the predictions of Wilson et al. (20) and support the conclusion that there was interaction of expiratory flows between lobes that preserved uniform PA decline in the emphysema group.

Furthermore, the results are also in agreement with those of Topulos et al. (19), who showed that PA differences between lobes developed very early during the course of deflation. In the condition in which a common choke point is present, near-maximal interlobar pressure differences coincided with peak flow. Thus the maximum effect of IREF was observed at high VL in the emphysema group.

Accordingly, without a flow interdependent mechanism, RLL flow measured at 95 and 90% VC in the
The high mid-VC range (i.e., 80 to 50% whole lung VC) in the emphysema group.

In the emphysema group, Fig. 6 shows that at >40% whole lung VC, the respective \( V_{\text{max,\tot}} \) values were not different from those found in the control group. Between 80 and 50% whole lung VC, because \( P_A \) were not different between the emphysema and control groups and because RLL resistances were also not different between groups, the pressure-head measured at the choke point (\( P_{\text{end,*}} \)) was unchanged in the emphysema group (see Table 2). Because wave speed was reached at the same airway site with a similar \( P_{\text{end,*}} \) in the two groups, wave-speed variables and hence \( V_{\text{max}} \) were unchanged at these \( V_L \) in the emphysema group (2).

In the emphysema group, although IREF maintained a relatively uniform \( P_A \) decline between lobes over the mid-whole lung VC range, what is the evidence that IREF contributed to the unchanged \( V_{\text{max,\tot}} \)? In the IREF-absent condition, it was previously shown (see above) that at \( V_L >40\% \) whole lung VC, differences in \( P_A \) would further widen compared with values found in the emphysema group. Then, if IREF were not present, what would be the effect of this widening on \( V_{\text{max,\tot}} \) and lobar flows compared with values obtained during homogeneous deflation? At 90, 80, and 70% whole lung VC, it seems unlikely that there would be much of a difference. Because flows for both lobes are relatively constant (i.e., 5 l/s) between \( P_A \) of 3 and 20 cmH2O (see Fig. 7B), substitution of the \( P_A \) calculated to occur in IREF-absent condition for those measured during homogenous deflation would not alter the sum of lobar \( V_{\text{max}} \) very much. In either case, the sum of RLL and LLL flows would average \( \sim 10 \) l/s.

However, at 60 and 50% whole lung VC, there is evidence that IREF could play a role in the maintenance of \( V_{\text{max,\tot}} \). During the IREF-absent condition (see Appendix), it was predicted that at 60% whole lung VC, \( P_{\text{A,\RLL}} \) would be 2.5 cmH2O and \( P_{\text{A,\LLL}} \) would be 4 cmH2O, respectively. At a \( P_A \) of 2.5 cmH2O (i.e., which corresponds to a \( V_L \) between 40 and 30% whole lung VC), RLL flows would be \( \sim 2 \) l/s (see data in Fig. 7 at 30% whole lung VC). At a \( P_{\text{A,\LLL}} \) of 4 cmH2O (i.e., which corresponds to 60% VC in Fig. 4), LLL flows would be \( \sim 3 \) l/s (see data at 40% VC in Fig. 7). One can observe that the combined lobar flows of 5 l/s would be \( <8 \) l/s measured at 60% whole lung VC during homogeneous deflation, in which RLL and LLL flows were 5 and 3.5 l/s, respectively (see Fig. 7B).

At 50% whole lung VC, in the IREF-absent condition (see Appendix), \( P_{\text{A,\RLL}} \) was predicted to be 2 cmH2O (which corresponds to 30% VC in Fig. 4), and \( P_{\text{A,\LLL}} \) was

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**Table 3. Whole lung maximum expiratory flow in additional emphysema experiments**

<table>
<thead>
<tr>
<th>% Whole Lung Vital Capacity</th>
<th>( 95 )</th>
<th>( 90 )</th>
<th>( 80 )</th>
<th>( 70 )</th>
<th>( 60 )</th>
<th>( 50 )</th>
<th>( 40 )</th>
<th>( 30 )</th>
<th>( 20 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_{\text{max},\tot} )</td>
<td>7.2 ± 1.0*</td>
<td>8.1 ± 0.7*</td>
<td>7.5 ± 1.4</td>
<td>5.4 ± 1.2</td>
<td>4.6 ± 1.2</td>
<td>3.5 ± 1.2</td>
<td>2.9 ± 0.9</td>
<td>2.5 ± 1.0</td>
<td>1.5 ± 0.6</td>
</tr>
</tbody>
</table>

*Values are means ± SE in l/s; \( n = 3 \) dogs. *Significantly different vs. control group, \( P < 0.05 \) (unpaired t-test corrected for multiple comparisons).
predicted to be 3.0 cmH\textsubscript{2}\text{O} (which corresponds to between 50 and 40\% VC in Fig. 4). The respective flows obtained from Fig. 7B would be 1.5 and 3 l/s. The combined predicted lobar flows of 4.5 l/s are <8 l/s that were measured at 50\% whole lung VC when deflation was homogeneous in Fig. 7.

Thus this analysis shows that there would be little change in \( \text{Vmax}_{\text{tot}} \) at high \( \text{Vl} \) (i.e., >60\% whole lung VC), if IREF were not present. Because the canine flow-volume curve is relatively constant over a large range of \( \text{Pa} \), widening of the \( \text{Pa} \) difference that would occur in the IREF-absent condition would not substantially change the sum of lobar flows compared with those found during homogeneous deflation. However, as the lung deflates, choke points start to jump into lobar bronchi, and then small differences in \( \text{Pa} \) may cause large differences in lobar flows. In Fig. 7B, particularly for the RLL, flows decreased from 5 to 2 l/s when \( \text{Pa} \) reached a critical value of <3 cmH\textsubscript{2}\text{O}. Then, IREF would prevent \( \text{P}_{\text{A}_{\text{RLL}}} \) from reaching this critical value at too high a \( \text{Vl} \), and in turn, this effect would preserve RLL flows at 60 and 50\% whole lung VC.

In the emphysema group, the sum of RLL and LLL flows measured at 60\% whole lung VC in Fig. 7 were slightly greater than what would be predicted during the IREF-absent condition (6 vs. 5 l/s). At 50\% whole lung VC, the sum of lobar flows in the emphysema group (4.5 l/s) were similar to those calculated for the IREF-absent condition. In terms of this analysis, preservation of \( \text{Vmax}_{\text{tot}} \) was observed mainly at 60\% whole lung VC. Thus the effect of IREF on \( \text{Vmax} \) in this emphysema model was relatively modest.

The canine lung might be relatively insensitive to IREF because over the upper one-half of the VC maneuver, flows are relatively constant, despite large decreases in lung recoil. The extent to which IREF will preserve regional flows depends on the precise nature of the pressure-flow relationships that are found in the different regions, which will, in turn, determine the reciprocal changes in regional flow that will occur. For instance, in the human lung, flow is more sensitive to changes in \( \text{Vl} \). In that case, without IREF, an increase in \( \text{Pa} \) in the normal lobe might not offset the decrease in \( \text{Pa} \) in the emphysema lobe so that a decrease in \( \text{Vmax}_{\text{tot}} \) would be observed over a greater proportion of the VC maneuver in nonhomogeneous disease. Moreover, in some experiments in the present study (see additional emphysema experiments), a central choke point did not occur despite the fact that a lesion similar to one found in the emphysema group was produced. Thus, under different conditions, the effect of IREF on \( \text{Vmax}_{\text{tot}} \) may vary in importance. It is difficult to make general inferences about the effect of IREF on \( \text{Vmax}_{\text{tot}} \) in regional lung disease without making the measurements.

At \( \text{Vl} \leq 40\% \) whole lung VC, a central choke point was not found in the emphysema group, and \( \text{Vmax}_{\text{tot}} \) measured were less than control group values (see Fig. 6). At these \( \text{Vl} \), choke points were identified at lobar bronchi and there was no interdependence of flow between regions. In the emphysema group, because at the high \( \text{Vl} \) most of the early deflation had come from the LLL and because RLL compliance was increased relative to LLL compliance, \( \text{P}_{\text{A}_{\text{LLL}}} < \text{P}_{\text{A}_{\text{RLL}}} \) at the low \( \text{Vl} \) (see Fig. 4). Because frictional resistance is not increased in this model, the pressure head at the RLL choke point [(\( \text{Pa} \) – frictional pressure (Pfr))] would be greater than that for the LLL. Hence, RLL flow was less than LLL flow. In the emphysema group, combined flows from the RLL and LLL were less than those found in the control group so that \( \text{Vmax}_{\text{tot}} \) were smaller at the low \( \text{Vl} \) in the emphysema group.

Furthermore, at \( \text{Vl} \leq 40\% \) whole lung VC, frictional resistances measured in the control group appeared to be slightly greater than those in the emphysema group, although the numbers were small and there were wide SE in the two groups. At these \( \text{Vl} \), because \( \text{P}_{\text{A}_{\text{RLL}}} \) values obtained in the emphysema group were slightly higher than corresponding values found in the control group, it is likely that airway diameter was large and therefore frictional resistances were lower in the emphysema group. Finally, it is important to note that when choke points move into lobar bronchi, flow from the subtended region would be governed by local choke point pressure-area behavior. This behavior is difficult to predict and may vary between regions. This behavior may be abnormal in emphysema because of destruction of tissue attachments to bronchi (10) (see below).

In the additional emphysema experiments, regional interdependence of flow was not a factor in the regulation of RLL and LLL flow. In dog 1, choke points were identified at mainstem bronchi. In this dog, as in the emphysema group, \( \text{P}_{\text{A}_{\text{RLL}}} \) fell faster than \( \text{P}_{\text{A}_{\text{LLL}}} \) at the beginning of expiration. The difference between \( \text{P}_{\text{A}_{\text{RLL}}} \) and \( \text{P}_{\text{A}_{\text{LLL}}} \) averaged 2–3 cmH\textsubscript{2}\text{O} over most of the VC. This difference in \( \text{Pa} \) appeared bigger than values found in the emphysema group, and moreover, \( \text{P}_{\text{A}_{\text{RLL}}} \) did not equal \( \text{P}_{\text{A}_{\text{LLL}}} \) until 30\% whole lung VC was reached. This greater nonuniformity of deflation is consistent with what was predicted in the previous analysis when regional interdependence of flow was not a factor in controlling lobar emptying in the emphysema group (see above).

In dogs 2 and 3, lobar choke points were observed throughout the whole VC range. In these dogs, in contrast to what was observed in the emphysema group, \( \text{P}_{\text{A}_{\text{RLL}}} \) did not decrease faster than \( \text{P}_{\text{A}_{\text{LLL}}} \) at the beginning of expiration. In dogs 2 and 3, the changes in RLL mechanical properties were similar to those produced in the emphysema group. The reason for the slower \( \text{P}_{\text{A}_{\text{RLL}}} \) decline in dogs 2 and 3 is that, when lobar choke points are found in emphysematous dogs, choke point pressure-area behavior may be altered. For a given pressure head, choke point area and hence \( \text{Vmax} \) are less than what would be predicted from a normal airway. In a previous study (13), this finding was ascribed to loss of bronchial tissue attachments due to destruction in this papain model. Thus in dogs 2 and 3 during early deflation, \( \text{P}_{\text{A}_{\text{RLL}}} \) decline was less than that found for the LLL, despite reduced compliance at high \( \text{P}_{\text{tp}} \) in the emphysema lobe.

The present study shows that despite the changes in compliance produced in the emphysematous lobe, \( \text{Pa} \)
decline over the high mid-vital range was relatively uniform in the emphysema group. This occurred because of a flow interdependence mechanism that helped to maintain Pa pressures between regions quite homogeneous. In the emphysema lobe, at high VL, compliance was lower than normal values, whereas at low VL compliance was higher than normal values. On the other hand, there were no changes in frictional resistances between lobes. The papain model resembles panacinar emphysema rather than centrilobular emphysema (10, 18). Centrilobular emphysema is associated with cigarette smoking and often is accompanied by an increase in frictional resistance (10, 18). Because frictional resistance may cause upstream movement of choke points (9, 16, 17), a common choke point among lobes may not occur in human disease, and the present results must be applied cautiously to humans.

The present study shows that when a common choke point was present, a flow interdependence mechanism limited the extent to which differences in Pa developed between regions in a canine model of regional emphysema. IREF appeared to maintain choke points centrally over a greater proportion of the whole lung VC sema. IREF appeared to maintain choke points centrally over a greater proportion of the whole lung VC.

Restimation of PARLL and PALLL in IREF-Absent Condition

The analysis performed in DISCUSSION indicates that without IREF, PARLL and PALLL measured at 90% whole lung VC in the emphysema group would have been 7.5 and 17.5 cmH2O, respectively. In the analysis below, a similar approach was used to estimate what the resulting lobar Pa would be when IREF was absent between 80 and 50% whole lung VC in the emphysema group.

If, at 90% whole lung VC, the predicted PARLL and PALLL in the emphysema group were 7.5 and 17.5 cmH2O, respectively, then flow from the RLL at a Pa of 7.5 cmH2O would be 5 l/s, and flow from the LLL at a Pa of 17.5 cmH2O would be also be 5 l/s. This is because lobar flows predicted to occur without IREF would be those found in the control group. Then, at a PARLL of 7.5 cmH2O, whole lung VC and hence lobar VC taken from Fig. 4B would be 70% because deflation is homogeneous in the control group. In turn, lobar flow measured at 70% whole lung VC from Fig. 7B would be ~5 l/s. Similarly, at a PALLL of 17.5 cmH2O (which corresponds to a whole lung and lobar VC >95% see Fig. 4), LLL flow would also be ~5 l/s (from Fig. 7B).

Furthermore, Vmaxtot is the sum of RLL flow, LLL flow, and the combined flows from the remaining lobes. The combined flows from the remaining lobes between 90 and 80% whole lung VC would be [60% × LLL flow (5 l/s)] = 3 l/s. The sum of RLL flow, LLL flow, and flow from the remaining lobes would yield a predicted Vmaxtot of ~13 l/s. In that case, the time for deflation between 90 and 80% whole lung VC would be 0.015 s (i.e., 0.2 liter / 13 l/s). Of the 0.2 liter expired between 90 and 80% whole lung VC, 75 ml (i.e., 5 l/s × 0.015 s) would come from the RLL, and 75 ml (i.e., 5 l/s × 0.15 s) would come from the LLL, whereas the remainder would occur from the other lung lobes.

From the RLL and LLL pressure volumes curves obtained in the emphysema group (see Fig. 1A), expiration of an additional 75 ml from the respective lobes would yield PARLL of ~5 cmH2O and PALLL of ~8 cmH2O.

Similarly, between 80 and 70% whole lung VC, the RLL at a Pa of 5 cmH2O would be deflating at ~5 l/s (Figs. 4B, 7B), whereas LLL flow at a Pa of 8 cmH2O would also be deflating at ~5 l/s. Then, predicted Vmaxtot would again be 13 l/s. Of the 0.2 liter expired, 75 ml would come from the RLL (5 l/s × 0.015 s) and 75 ml (5 l/s × 0.15 s) would come from the LLL. The resulting PARLL would be ~4 cmH2O, and the PALLL would be ~6 cmH2O.

Between 70 and 60% whole lung VC, PARLL of 4 cmH2O would again be deflating at ~5 l/s and PALLL of 6 cmH2O would also be deflating at ~5 l/s. The predicted Vmaxtot would again be ~13 l/s. Seventy-five milliliters would be expired from each lobe, and PARLL and PALLL would fall to 2.5 and 4 cmH2O, respectively. Finally, between 60 and 50% whole lung VC, RLL flow measured at Pa of 2.5 cmH2O would fall to 1.5 l/s, whereas LLL flow at Pa of 4 cmH2O would be 3.0 l/s. The predicted Vmaxtot (6.8 l/s) would be the sum of the RLL flow (2.0 l/s), LLL flow (3.0 l/s) and flow from the remaining lobes (6.0 × 3.0 l/s). The time to deflate the next 0.2 liter would be 0.025 s. The RLL would expire 63 ml and the LLL 88 ml. PARLL would fall to 2 cmH2O, and PALLL would fall to 3 cmH2O.

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