INFLUENCE OF PULMONARY VASCULAR PRESSURE ON BRONCHIAL COLLAPSIBILITY OF EXCISED DOG LUNGS

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Abstract  The main bronchi of excised dog lobes were obstructed with beads, 5 to 6 cm from their origin so that they did not communicate with the peripheral air spaces. Both pulmonary artery and vein were cannulated and both pulmonary vascular pressures were controlled. With the lobe held at constant transpulmonary pressure, bronchial pressure-volume curves were studied during acute pulmonary vascular engorgement. The bronchial compliance was reduced at higher vascular pressure and the effect of vascular engorgement on the bronchial collapsibility was larger at higher transpulmonary pressure: bronchial compliance at vascular pressure 10 cm H_2O were 70, 84, 98, and 100% of the bronchial compliance at vascular pressure —40 cm H_2O at transpulmonary pressure 20, 10, 5, and 0 cm H_2O, respectively. We concluded that vascular engorgement increased parenchymal radial traction to the bronchi when the bronchi reduced its diameter, although it appeared that vascular engorgement resulted in little change in the lung elastic recoil pressure.

We recently studied (TAKISHIMA et al., 1975) that bronchi were much less collapsible in situ than when dissected free because of radial traction of the peribronchial tissues. In our previous experiment, the pulmonary vascular system was not taken into consideration and the pulmonary vessels were kept open to pleural pressure. However, the parenchymal radial stress on collapsing bronchi would be the summed effect of alveolar tissues and pulmonary vessels.

Since the experiment of VON BOSCH (1889), it has been accepted that vascular engorgement results in a very limited change in recoiling force of the lung (FRANK, 1959) and that the anatomical arrangements of airway, alveolar tissues and blood vessels appear to exert minimal stress upon each other. However, mechanical interaction (the interdependence) among airway, alveolar tissues and pulmonary vascular vessels, has not been studied.
In the present study, we studied the effect of the pulmonary vascular engorgement on bronchial collapsibility using the same technique as the previous experiment. This time both pulmonary artery and vein were cannulated and the bronchial pressure-volume relationships were recorded at different vascular pressures.

METHODS

The experiments were carried out on excised right or left lower lobes of five mongrel dogs, 12–19 kg, sacrificed by exsanguination. The dog's own heparinized (500 units/kg) arterial blood was used for perfusion of the isolated lobe. The lobar extrapulmonary bronchus was tied to a cannula with an outside diameter of 14 mm, and the lobe was hung in a lucite box. At zero transpulmonary pressure the pulmonary artery and vein of the lobe were cannulated with the tips of the cannulae lying just beneath the pleural surface so that there would be no traction or distortion of the pulmonary vessels. Any air that had entered into the artery and vein of the lobe was replaced with blood via small polyethylene catheters inserted in the peripheral vessels. Both artery and vein were connected to blood reservoirs outside the lucite box (Fig. 1). The zero level of hydrostatic pressure of the vascular system was set at the mid-point of the height of the bronchi studied, about 3 cm below the hilum. At transpulmonary pressure 5 cm H₂O, blood flow 100 ml/min was maintained for one minute at arterial pressure +5 cm H₂O and venous pressure -30 cm H₂O. Then, both arterial and venous pressures

![Block diagram of apparatus](image-url)
were lowered to $-40$ cm H$_2$O.

The orifices of the first 10 to 15 branches of the main bronchus were closed and made airtight by beads coated with tissue glue while the lobe was inflated to transpulmonary pressure 20 cm H$_2$O by negative box pressure. The length of the obstructed bronchus was 5 to 6 cm of which 0.2 to 0.4 cm were extrapulmonary. The uppermost bronchial branch was cannulated with a polyethylene catheter (PE 240) to provide a collateral route of ventilation to the alveoli and to keep the alveolar pressure at atmospheric pressure. Thus, we could separate the main bronchi from peripheral bronchi and alveoli. It took about 30 min to obstruct the main lobar bronchi. After obstructing the bronchi with beads, pulmonary perfusion was again resumed for 5 min at transpulmonary pressure 5 cm H$_2$O with arterial pressure 5 cm H$_2$O and venous pressure $-30$ cm H$_2$O.

The obstructed main bronchus was filled with physiological saline and connected both to a pressure transducer (Electro-manometer, LPU—0.1, Nihon Kohden) for measurement of intrabronchial pressure and to a linear displacement transformer (535 DL 1000 Bm Sanborn) coupled with a 30 ml syringe for measurement of volume change of the bronchial segment. The bronchial pressure-volume curves were plotted on an X-Y recorder (F-32, Rikendensi). Bronchial volume was measured with an accuracy of 0.02 ml with electrical magnification of the linear transducer signal. The zero reference of the bronchi was set at the mid-point of height of the bronchi examined, and therefore was the same level as the vascular system.

Recordings of bronchial pressure-volume curves were made at transpulmonary pressure 20, 10, 5, and 0 cm H$_2$O which were maintained by negative box pressure and zero alveolar pressure; at each transpulmonary pressure, bronchial pressure-volume curves were obtained at vascular pressures 10, 0 and $-40$ cm H$_2$O. At transpulmonary pressure 20 cm H$_2$O both arterial and venous vascular pressures were changed simultaneously by maintaining both arterial and venous reservoirs to a given level, and after waiting for one minute, bronchial volume was deflated slowly and continuously for 30 sec until the bronchial pressure became as low as $-85\pm 5$ cm H$_2$O. After waiting for 30 sec at the end of the deflation, bronchial pressure-volume curves during inflation were recorded slowly and continuously for about 3 min up to the original zero bronchial pressure. This method permitted us to minimize a stress adaptation of the curve compared to deflation curves. In dogs 1 and 2, the bronchial pressure-volume curves were obtained first at lower vascular pressure and then at higher vascular pressure ($-40, 0, 10$ cm H$_2$O). In dogs 3 to 5, they were obtained first at higher vascular pressure and then at lower vascular pressure ($10, 0, -40$ cm H$_2$O). As stress adaptation is reduced with repeated maneuvers (FuKAYA et al., 1968), the effect of repetition in dogs 3 to 5 would cause an overestimate of the observed differences among curves at different vascular pressures. While, in dogs 1 and 2, the effect of repetition would cause an underestimate of the differences. However, between each curve, the lung was
inflated to transpulmonary pressure 20 cm H\textsubscript{2}O, so that the effect should not be large. At transpulmonary pressures 10, 5, and 0 cm H\textsubscript{2}O, the bronchial pressure-volume curves were also measured in the same way. At zero transpulmonary pressure bronchial pressure-volume curves reached a plateau at bronchial pressure $-30$ cm H\textsubscript{2}O and we assumed this plateau to be zero bronchial volume. The experiments were carried out at room temperature 20°C and continued for about 3 hr.

At the end of the experiment, to examine the existence of lung edema, the parenchyma dissected from the bottom of the lobe was fixed with formalin. After fixation, random blocks of the tissue were taken from the slices and embedded in paraffin. Sections 4 μ thick were cut from the paraffin blocks, mounted on glass slides, stained with hematoxylin and eosin, and examined by light microscopy.

**RESULTS**

Typical example of bronchial pressure-volume curves are shown in Fig. 2. At transpulmonary pressure 20 cm H\textsubscript{2}O during waiting for 30 sec at the end of deflation, the initial bronchial pressure $-85\pm5$ cm H\textsubscript{2}O decreased to a less negative bronchial pressure, accordingly, the bronchial pressure-volume curves during inflation were eventually studied from bronchial pressure $-65\pm5$ cm H\textsubscript{2}O. Such stress adaptation of bronchial pressure tended to be great at higher transpulmonary pressure. Bronchial pressure-volume curves obtained from five dogs are shown in Fig. 3. Bronchial volume at transpulmonary pressure 20 cm H\textsubscript{2}O, bronchial pressure 0 cm H\textsubscript{2}O, and vascular pressure 10 cm H\textsubscript{2}O was taken as 100% bronchial volume. The bronchi were less collapsible at higher transpulmonary pressure than at lower transpulmonary pressure and this characteristic tended to be much

![Fig. 2. Bronchial pressure ($p_{br}$)-volume ($v_{br}$) curves were measured at transpulmonary pressure ($p_L$) 20, 15, 5, and 0 cm H\textsubscript{2}O in dog 4. In dog 4 at the end of the experiment, we partially cut away parenchyma and peripheral airways and recorded a partially dissected bronchial pressure-volume curve. Note that at the same bronchial volume ($v_{br}$), the curves at higher vascular pressure ($p_{vas}$) showed more negative bronchial pressure ($p_{br}$) than the curves at lower vascular pressure ($p_{vas}$).](image-url)
VASCULAR PRESSURE AND AIRWAY COMPLIANCE

Fig. 3. Bronchial pressure-volume curves at vascular pressure 10 cm H2O (closed circles with continuous lines), 0 cm H2O (open circles with dashed lines), and -40 cm H2O (cross marks with dotted lines).

more intense when vascular pressure was increased. At higher transpulmonary pressure there were large and consistent differences between the curves at vascular pressures 10 and -40 cm H2O, and at lower transpulmonary pressure there was little difference of slopes at different vascular pressures. At transpulmonary pressure 20 cm H2O, vascular congestion slightly decreased or did not change the bronchial volume at zero bronchial pressure.

Since there is no simple way to describe whole S-shaped and complex bronchial pressure-volume curves, we plotted log bronchial volume versus bronchial pressure (Fig. 4). Log bronchial volume was linearly related to bronchial pressure except at high bronchial volume as previously shown (Takishima et al., 1975). Regression lines for log bronchial volume on bronchial pressure over the range of 80 to 30% of initial bronchial volume were calculated using the least squares method. The slopes of bronchial pressure versus log bronchial volume curve at vascular pressure 10 and 0 cm H2O were expressed as percentages of the slope at vascular pressure -40 cm H2O at each transpulmonary pressure (Fig. 5). Figure 5 showed that the bronchial compliance was reduced at higher vascular pressure and the effect of vascular engorgement was more prominent at higher transpulmonary pressure. Average values for vascular pressure 10 cm H2O were 70, 84, 98 and 100% at transpulmonary pressure 20, 10, 5 and 0 cm H2O, respectively. Average values for vascular pressure 0 cm H2O were 87, 96, 100, and 100% at transpulmonary pressure of 20, 10, 5, and 0 cm H2O, respectively.
Fig. 4. Bronchial pressure versus exponential bronchial volume curves are linearized except for their segments near zero bronchial pressure. Marks are the same as Fig. 3.

Fig. 5. At each transpulmonary pressure bronchial compliances at vascular pressure 10 and 0 cm H₂O were divided by bronchial compliance at vascular pressure −40 cm H₂O and percentaged. Marks are the same as Fig. 3. Horizontal bars are mean values.

Histological studies at the end of the experiments did not reveal any fluid in alveoli or even around the peripheral airways, arteries or veins, as in the pulmonary edema reported by Staub et al. (1967).
DISCUSSION

In the present study, we have demonstrated that vascular engorgement made a systematic increase of radial traction to the collapsing bronchi.

Pulmonary edema

A possible explanation of the present result was a fluid accumulation in the lung which might affect the radial traction by a change of parenchymal visco-elastic properties. Although it was confirmed by the histological examination that there was no fluid in the alveoli, peripheral bronchi and vessels, it was suggested that early pulmonary edema was patchy and localized (Staub et al., 1967). Some fluid might have accumulated in the proximal broncho-vascular sheath of the bronchi studied which we did not prepare for histological studies. Hogg et al. (1972) did not observe fluid completely surrounding the airways even when foam was frothing from the trachea. We examined sections from the dependent portions of the lobe. Therefore, if some fluid was accumulated in the proximal broncho-vascular sheath, sections from dependent portions would offer some evidence. Since it seemed unlikely that fluid accumulation due to vascular congestion would be rapidly reversible, such fluid, if present in the broncho-vascular sheath could not explain the present consistent results in dogs 3 to 5, in which bronchial pressure-volume curves at lower vascular pressure was measured after the curves at higher vascular pressure. We did not increase vascular pressure more than 10 cm H2O in order to avoid pulmonary edema, nevertheless the bronchial compliance was systematically reduced.

Mechanism

Lung volume. Since the experiments of von Bosch (1889), it has been reported that pulmonary vascular congestion influences the mechanical behavior of the lungs, but to a very limited extent. Frank (1959) confirmed von Bosch's effect, demonstrating that at larger lung volumes the recoiling force increased as if the congested blood vessels had the effect of constricting lung parenchyma. In the present study, the lung volume might be changed by different vascular pressures and such a change of lung volume might reduce the bronchial compliance. Since those former reports were mostly carried out at positive alveolar pressures, and the applied vascular pressures and the procedures were not strictly comparable to our present experimental procedures, we re-examined the influence of changing vascular pressure on lung volumes at transpulmonary pressures of 20, 10, 5, and 0 cm H2O. One excised lower lobe was set up just as in the present experiment except that the bronchi were not obstructed. We observed no detectable change of lung volume for the same magnitude of change of vascular pressure that we had studied. Thus we supposed that present results could not be explained by the change of the lung volume.

Longitudinal traction. Since we observed bronchial collapsibility by using
bronchial volume, the effect of vascular engorgement might be summed results not only of bronchial radial traction but also of bronchial longitudinal traction. Frank (1959) observed in cat's lung that the vascular volume increased about 10 ml or more with vascular engorgement from the collapsed condition of the vessels. Therefore, if the vascular engorgement caused the gravitational effect, longitudinal traction resulting from an increase of blood volume in pulmonary vessels should be taken into account in our hanging lobe preparation. To exclude a weight increment due to vascular blood at high vascular pressure, we distended the vessels with air in one lobe, and we could observe almost the same results as for the preparation perfused by blood. This observation denies the bronchial longitudinal traction with the vascular blood increment as a factor responsible for our present results.

Radial traction. Then how can vascular engorgement produce greater radial traction to the bronchial collapse? In the present method, we could separate the three different tension systems in the lung; the parenchymal, vascular and bronchial systems. Using the condom and the spring model, Mead et al. (1970) presented a theoretical analysis of interdependence between the bronchi and the parenchyma. Takishima and Mead (1972) proved their theory experimentally. The model of Mead et al. (1970) might explain the present results, suggesting that the radial traction of the peribronchial parenchyma is a summed effect of condom or spring-like radial stress of both vascular and alveolar tissues. Anatomically the proximal vascular structures appear to run together with the bronchi in the broncho-vascular sheath (Staub, 1963). Macklin (1946) and Howell et al. (1961) separated pulmonary vasculature into alveolar and extra-alveolar portions. If we accept the condom or spring models of the extra alveolar vessels, proposed by Mead et al. (1970), fully expanded vessels with high vascular pressure could increase the radial traction of surrounding tissues on the bronchi and the extra-alveolar vessels would exert increased radial traction at higher transpulmonary pressure rather than at lower transpulmonary pressure. Another possible explanation of the effect of vascular pressure at different transpulmonary pressure could be the bronchial deformation. Using stereoscopic measurements, Hughes et al. (1974) observed the collapsible bronchial area during dynamic expiratory flow maneuvers and reported that the bronchi during expiratory flow markedly distorted their shape in slightly expanded lungs while a slight change took place in a well expanded lung. In a preceding paper, we observed that the bronchi could not keep their circular shape when completely collapsed, even when the lung was fully expanded. At lower transpulmonary pressure, the vessels within the broncho-vascular sheath could become engorged at high vascular pressure and thus prevent bronchial collapse, but the alveolar tissues could be easily deformed around the collapsed bronchi, so that the bronchi might easily change their shape resulting in the same bronchial collapsibility at different vascular pressure.
Airway resistance

At zero bronchial pressure, vascular engorgement tended to decrease bronchial volume at transpulmonary pressure 20 cm H₂O. Hogg et al. (1972) reported that raising left atrial pressure affected the distribution of airway resistance with the major effect on the peripheral airways. This increase was most likely due to competition for space between arteries and small airways in the broncho-vascular sheath. Our experiments were examined upon the limited section of the larger bronchi, so our results could not be compared with the results of Hogg et al. (1972) who have studied the smaller bronchi. We did not observe systematic narrowing of our bronchi with vascular engorgement as reported by Hogg et al. (1972) in the small bronchi.

Implications to expiratory flow limitation

Wilhelmsen and Varnaukaskas (1967) have reported that pulmonary vascular infusion of up to a liter of Rheomacrodex into normal subjects causes a decrease in maximal expiratory flow rate without clinical pulmonary edema. Using a two-dimensional flow model, Takishima and Sasaki (1972) suggested that maximal expiratory flow was inversely proportional to the bronchial compliance and bronchial resistance. The present study suggests that pulmonary vascular engorgement increases peripheral bronchial resistance (Hogg et al., 1972) but decreases bronchial compliance. Accordingly, vascular engorgement might possibly affect the maximal expiratory flow but in either direction. Since the effect of vascular engorgement on the bronchial collapsibility is larger at higher transpulmonary pressure, and smaller at lower transpulmonary pressure, and an increase of peripheral airway resistance is greater at lower transpulmonary pressure (Hogg et al., 1972), then the maximum expiratory flow rate, especially at middle and low lung volumes, might be expected to decrease; however, at larger lung volume, expiratory flow rate would be expected to increase.

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REFERENCES


