Pulmonary Vasoconstriction Due to Nonocclusive Distention of Large Pulmonary Arteries in the Dog

By Albert L. Hyman, M.D.

ABSTRACT

The pulmonary vascular changes caused by distention of the pulmonary artery with the lumen fixed were compared to those produced by occlusive arterial distention. In 13 methane-anesthetized dogs, nonocclusive distention was produced by distention of a corrugated rubber cuff around a hollow metal cylinder affixed to the end of a cardiac catheter. In 10 other dogs, occlusive distention was produced by inflation of a standard balloon catheter. Pressures were measured in large and small pulmonary veins and in the left and right atria with transseptal techniques, and in the pulmonary and femoral arteries. Simultaneously, pulmonary blood volume and flow were measured with dye-dilution techniques. Pulmonary arteriovenous shunting was studied with the hydrogen-electrode technique. Nonocclusive distention of a pulmonary artery greatly increased pulmonary arterial and venous pressure and decreased pulmonary blood volume without changing left atrial pressure or cardiac output. Pulmonary vascular resistances increased, and pulmonary arteriovenous shunting was detected. Occlusive distention of one pulmonary artery caused smaller increases in pressure in the pulmonary artery and vein of the unobstructed lung. Vascular resistances in the unobstructed lung decreased, while blood volume and flow in that lung doubled. Pulmonary arterial distention leads to active constriction of the pulmonary arteries and veins, but these responses may be obscured by the passive responses to increasing blood flow and volume.

ADDITIONAL KEY WORDS
pulmonary baroreceptors
pulmonary veins
nonocclusive cuffed catheter
arteriovenous shunting
pulmonary blood volume
pulmonary phlebography

The distribution of stretch receptors in the pulmonary arteries and their afferent nerve fibers have been extensively studied (1-3). Although the systemic vascular response to stimulation of these receptors has been the subject of several reports (4-8), relatively little attention has been devoted to the pulmonary vascular response. Distention of one pulmonary artery by a balloon that also completely occludes its lumen causes a 30 to 33% increase in pulmonary arterial pressure proximal to the site of occlusion in open-chest dogs (9). The cause of this pulmonary hypertension is not well understood (10). A pulmopulmonary reflex has been suggested as a cause, since this pressor response can be blocked by infiltration of the pulmonary arterial wall with Xylocaine but cannot be blocked with ganglionic blocking agents, atropine, reserpine, or bilateral cervical vagotomy (10). Distention of one pulmonary artery by a balloon without occlusion of the arterial lumen has also been shown to cause pulmonary arterial hypertension (7) in open-chest dogs. However, no measurements of other intrathoracic vascular pressures, pulmonary blood flow, or volume were obtained in that study. The pulmonary hemodynamic response to stretching the pulmonary arterial wall while keeping the lumen fixed in the intact dog has not been reported.
With recent improvements in transseptal catheterization, pressures in both atria, the small and large pulmonary veins, and the pulmonary artery can be measured, and the pulmonary blood volume and flow can be estimated simultaneously in the intact dog, thereby avoiding the circulatory and respiratory changes that accompany thoracotomy (11, 12). This report describes the changes in pulmonary hemodynamics, studied by transseptal catheterization, after acute distention of a pulmonary artery with a nonocclusive cuffed cardiac catheter and compares these changes to those produced in a second group of dogs by both distention and occlusion of a pulmonary artery.

Methods

Thirty-five mongrel dogs (17 to 24 kg) were lightly anesthetized with urethane, 1.0 g/kg, and were strapped in the supine position to a fluoroscopic table. They spontaneously breathed either room air, 30% oxygen, or 50% oxygen through an endotracheal tube. A Kifa catheter, 2.85 mm o.d., was passed from a jugular vein, through the atrial septum, and into a large pulmonary vein from the left lower lobe. A semi-rigid polyvinyl catheter, 0.9 mm o.d., was passed through the Kifa catheter and wedged into a small pulmonary vein in the left lower lobe of the lung. The polyvinyl catheter was then withdrawn 2 to 3 cm from the wedged position until an abrupt fall in pressure was observed, and fixed in position with an occlusive adapter. Special precautions were taken to ensure that measurements with the polyvinyl catheter were made in veins 1.5 to 2.5 mm in diameter without wedging. Descriptions of this technique have been previously reported (11, 13). A second Kifa catheter, with a rounded distal end and with side openings near the distal end was passed through the Kifa catheter and wedged into the pulmonary artery just beyond the pulmonary valve. Teflon catheters, 1.0 mm o.d., with side openings and closed ends, were then introduced into the pulmonary artery just beyond the pulmonary valve, into the right atrium, and into the one femoral artery. An additional Teflon catheter was introduced into the pleural space using an occlusive adapter and a blunted needle to avoid pneumothorax. Pressures were recorded by Statham P23D pressure transducers connected to an oscillographic recorder. Cardiac output was measured by the dye-dilution method: known quantities of indocyanine green were injected into the pulmonary artery, and blood was drawn through the left atrial catheter and a cuvette densitometer at 38.2 ml/min. The dye curves were inscribed on a linear recorder, and blood was immediately reinjected into the dog after each measurement. Pulmonary blood volume, which included the small quantity in the left atrium, was calculated by the method used in this laboratory (14). Standard formulas were used for calculating vascular resistances (units = mm Hg/[ml/min/kg body weight]) and average distending pressure (one half of the sum of mean pressure in the pulmonary artery and in the left atrium) (14, 15).

FIGURE 1
Side view of the tip of a catheter (A) with a hollow metal cylinder attached to the side opening. The outer surface of the cylinder is covered by the rubber cuff. B and C show the rubber cuff in the collapsed and distended states, respectively.
In 13 dogs, the pulmonary vascular response to distention of the pulmonary arterial wall at a constant lumen was studied, using a nonocclusive double lumen 8.5 F cardiac catheter with a hol-

FIGURE 2
Distended nonocclusive cuff catheter (A), and distended occlusive balloon catheter (B) in the left pulmonary artery. Left anterior oblique position.

FIGURE 3
Bronchogram of left lung illustrating the absence of airway obstruction during nonocclusive cuff distention of the left pulmonary artery (A) and after collapse of the cuff (B) (left anterior oblique position). Similar bronchographic studies, obtained with main and right pulmonary arterial distention, showed no airway obstruction.
In both groups, after the control vascular and pleural pressure, pulmonary blood flow and volume were measured, the pulmonary artery was acutely distended during fluoroscopic observation either by distention of the nonocclusive rubber cuff of the cylinder with 3 ml, or by injecting 5 ml of sodium diatrizoate into the balloon. Care was exercised at fluoroscopy to ensure that the cuff did not distend eccentrically, which could have caused obstruction to flow. Measurements were made at intervals of 1, 5, and 10 minutes after distention. The balloon and rubber cuff were then acutely collapsed, and further observations were made 1 and 10 minutes later. Oxygen saturation, pH, Pco₂, and Pco₃ of blood samples from the left atrium were measured before, during, and after distention of the cuff and the balloon. Since mechanical obstruction of neighboring airways would affect the gaseous content of the blood in pulmonary veins in that region, blood samples from the left pulmonary vein were similarly examined in nonocclusive distentions of the right and main pulmonary artery, and in four of the six distentions of the left pulmonary artery.

After completion of each experiment with nonocclusive arterial distention, the dogs were given specific pharmacologic blocking agents, and the distention was repeated. Each dog served as its own control. Two dogs were given methylergolide, 50 mg/kg, 1 hour before distention; this dose prevented the systemic arterial pressure response of serotonin, 4.0 μg/kg iv, when tested immediately before distention. Two were given dibenzyline, 2.0 mg/kg, 2 hours before distention, and when tested immediately before distention, norepinephrine, 2 μg/kg iv, caused an increase in systemic arterial pressure of only 3 to 4 mm Hg, i.e., 94% blockade. Two, which were given hexamethonium, 25 g iv, 20 minutes before distention, and when tested immediately before distention, bilateral occlusion of the carotid arteries failed to produce a systemic hypertensive response. Two, which were given hexamethonium, 250 mg iv, developed a sustained hypotension of 65 mm Hg and had a 90% suppression of the hypertensive response to bilateral occlusion of the carotid arteries, when tested immediately before cuff distention. Two were given sodium salicylate, 25 g iv, 20 minutes before distention, and when tested immediately before distention, the systemic arterial depressor effect of 1 μg/kg of bradykinin was only 3 mm Hg, i.e., 96% blockade. Two were given diphenhydramine hydrochloride, 5 mg/kg iv, 20 minutes before distention, and when tested immediately before distention, the systemic arterial depressor effect of histamine, 3.5 μg/kg.

In a second group of 10 dogs, the effects of distention of the pulmonary arterial wall with complete occlusion of its lumen were studied with a standard 8 F balloon catheter, which had been passed from the femoral vein into the first part of the right pulmonary artery. Bronchographic studies in intact dogs were made at intervals of 1, 5, and 10 minutes after distention. The balloon and rubber cuff could not be passed beyond the upper pulmonary artery, just beyond the bifurcation of the main pulmonary artery, and in the other 6, into the first part of the left pulmonary artery, just beyond the bifurcation (Fig. 2). That this catheter produced only slight, if any, mechanical impedance to flow after distention of the cuff was indicated by the identity of systolic pressures in the right ventricle and pulmonary artery beyond the cylinder and by inspection of the catheter in place after each experiment. Although the rubber cuff was near the pulmonary veins from the left upper lobe during distention of the left pulmonary artery, distention or obstruction of those vessels was not apparent at thoracotomy. In addition, obstruction to blood vessels in the vicinity of the distended nonocclusive rubber cuff was not seen during the arterial and venous phases of pulmonary cineangiography. Bronchographic studies in intact dogs during distention and after collapse of the cuff (Fig. 3) did not reveal obstruction of neighboring bronchi. Inspection of the mediastinum of dogs during thoracotomy also indicated that these distended rubber cuffs did not compress or obstruct the neighboring airways.

In a second group of 10 dogs, the effects of distention of the pulmonary arterial wall with complete occlusion of its lumen were studied with a standard 8 F balloon catheter, which had been passed from the femoral vein into the first part of the left pulmonary artery, just beyond the bifurcation (Fig. 2). (When introduced from this site, this catheter always entered the left pulmonary artery and could not be passed into the right pulmonary artery.) The external diameter of the distended balloon in situ was 22 to 23 mm, and the length was 19 to 21 mm. In these dogs, the same transseptal technique was used to measure pulmonary vascular flows, pressures, and blood volumes, except that the pulmonary venous pressure was always measured in the right lung.

**References**

1. United States Catheter & Instrument Co., Glens Falls, N. Y.

2. Hypaque, Winthrop Laboratories, New York, N. Y.
was only 4 mm Hg, i.e. 95% blockade. Two were given atropine, 2.0 mg/kg iv, after bilateral cervical vagotomy, and when tested immediately before cuff distention, no systemic arterial response to 20 µg/kg of acetylcholine was observed.

In three other dogs, pulmonary arteriovenous shunting was investigated by the hydrogen-electrode technique (16, 17) during nonocclusive arterial distention.

In five other dogs, the response of the pulmonary veins to nonocclusive distention of the pulmonary artery was also studied by selective cineangiography using a technique similar to that described by Parker and associates (18). A Kifa catheter without side holes was passed transseptally into the vein from the left lower lobe, 2 to 3 cm dorsal to the left atrial border. Control angiograms (35-mm film at 48 frames/sec) were obtained in the left anterior oblique position by injecting 8.0 ml of 75% sodium diatrizoate at an injector pressure of 30 lb/inch\(^2\) with an Amplatz injector. \(^7\) Injections were always begun at the end of expiration. After satisfactory control angiograms were obtained, the nonocclusive rubber cuff was distended, and venous angiography was repeated using exactly the same technique and without changing the position of the Kifa catheter. In one dog, the main pulmonary artery was distended; in one, the right pulmonary artery just beyond the bifurcation was distended; and in three, the left pulmonary artery just beyond the bifurcation was distended. Additional venous angiograms were obtained 10 minutes after the cuff had been deflated. From each study, the angiograms obtained during the control period, during period of distention, and after collapse of the cuff were studied by projection. Frames which had been taken 0.5, 0.85, and 1.2 seconds after the beginning of the injection in each of the periods were printed and compared.

Additional studies were done in two other dogs to determine the effect of acute nonocclusive distention of the main pulmonary artery or the right or left pulmonary artery just beyond the bifurcation in 13 dogs are shown in Figure 4 and Table 1. Mean pressures in the pulmonary artery and in the small pulmonary vein increased within 2 to 3 seconds of cuff distention and fell within 3 to 4 seconds of collapse of the cuff (Fig. 5). Since there was no difference in the magnitude of rise in these pressures whether the main pulmonary artery or the right or left pulmonary artery just beyond the bifurcation was distended, the data have been pooled.

Pressure changes in the left atrium, in the large pulmonary vein adjacent to the left atrium, and in the pleural space were small and inconsistent, and the changes in their means were not significant \((P > 0.5)\). Within 1 minute of distention, the \(P_O_2\) of left atrial blood decreased (Fig. 6) and returned toward control values only after collapse of the cuff. Similar decreases in \(P_O_2\) of pulmonary venous blood regularly occurred. Respiratory rate, minute respiratory volume, blood pH, and \(P_CO_2\) did not change significantly \((P > 0.5)\).

![Figure 4](http://circres.ahajournals.org/)

**Figure 4**

Average changes in mean pressures in 13 intact dogs with nonocclusive distention of either the main pulmonary artery, or the right or left branch just beyond the bifurcation.
TABLE 1
Changes in Pulmonary Hemodynamic Data after Nonocclusive Distention of Pulmonary Artery in Thirteen Dogs

<table>
<thead>
<tr>
<th></th>
<th>Measured parameters (mm Hg)</th>
<th>Vascular resistance (mm Hg [ml/min/kg])</th>
<th>Average distending pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pulm. crtery</td>
<td>Left atrium</td>
<td>Small pulm. vein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>19.7</td>
<td>2.1</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td>1.14</td>
<td>0.33</td>
<td>0.32</td>
</tr>
<tr>
<td>Distention</td>
<td>32.4*</td>
<td>1.7</td>
<td>14.5*</td>
</tr>
<tr>
<td></td>
<td>1.69</td>
<td>0.32</td>
<td>0.60</td>
</tr>
<tr>
<td></td>
<td>31.1*</td>
<td>2.9</td>
<td>13.8*</td>
</tr>
<tr>
<td></td>
<td>1.73</td>
<td>0.47</td>
<td>0.65</td>
</tr>
<tr>
<td></td>
<td>30.3*</td>
<td>2.0</td>
<td>13.9*</td>
</tr>
<tr>
<td></td>
<td>1.9</td>
<td>0.45</td>
<td>0.72</td>
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<tr>
<td>Distention</td>
<td>19.8</td>
<td>2.1</td>
<td>8.3</td>
</tr>
<tr>
<td></td>
<td>0.07</td>
<td>0.46</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>18.5</td>
<td>1.7</td>
<td>7.0</td>
</tr>
<tr>
<td></td>
<td>0.79</td>
<td>0.40</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Values given are means ± se. *P < 0.01.

*P < 0.01.
Similar phenomena were observed after nonocclusive distention of the pulmonary artery in dogs that had received the blocking agents.

In the two other dogs in which smaller pulmonary arteries were distended, the pulmonary arterial pressure rose only 1 to 3 mm Hg. Pulmonary venous pressure and left atrial blood Po2 were unchanged. Data obtained in those two dogs were not included in the tables and figures.

Changes in mean pressure after distention of the occlusive balloon are shown in Figure 7 and Table 2. Although the mean pulmonary arterial pressure increased abruptly with distention of the balloon and fell abruptly with its collapse, the increase was significantly less (P<0.01) than the increase caused by nonocclusive distention. The pressure in the small pulmonary vein increased slightly as the balloon was distended, but returned to control values within 5 minutes. Complete, or almost complete, occlusion of the left pulmonary artery was suggested by the fall in arterial pressure beyond the site of distention to the level of left atrial pressure and by the 34% increase in mean arterial pressure proximal to the obstruction (9). The Po2 of left atrial blood was not significantly (P>0.5) changed by occlusive distention.

Cardiac output was not significantly (P>0.5) changed by either type of distention (Tables 1 and 2). The circulating pulmonary blood volume significantly (P<0.01) decreased with nonocclusive distention, but was unchanged with occlusive balloon distention (Tables 1 and 2).

The total pulmonary vascular resistance and the resistance to blood flow offered by the segment of pulmonary vein in which it was measured rose significantly (P<0.01) with nonocclusive distention (Table 1). Calculations of resistances to blood flow in the right lung in the experiments with occlusive distention of the left pulmonary artery were based on the assumption that during the control period the blood flow and resistances to blood flow in one lung were about equal to those of the other lung. Calculations based on this assumption indicate that the total pulmonary vascular resistance of the right lung and the resistance offered by the segment of pulmonary vein studied in that lung significantly (P<0.01) decreased when the left pulmonary artery was distended and occluded (Table 2). The average pulmonary vascular distending pressure significantly (P<0.05) increased after both nonocclusive and
TABLE 2

Changes in Pulmonary Hemodynamic Data after Occlusive Distention of Left Pulmonary Artery in Ten Dogs

<table>
<thead>
<tr>
<th></th>
<th>Mean pressure (mm Hg)</th>
<th>Vascular resistance (mm Hg/ml/min/kg)</th>
<th>Average distending pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pulm. artery</td>
<td>Left atrium</td>
<td>Small pulm. vein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>20.9</td>
<td>2.9</td>
<td>7.2</td>
</tr>
<tr>
<td>Control</td>
<td>1.14</td>
<td>0.60</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distention</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 minute</td>
<td>27.7*</td>
<td>2.7</td>
<td>11.4*</td>
</tr>
<tr>
<td></td>
<td>1.17</td>
<td>0.59</td>
<td>0.59</td>
</tr>
<tr>
<td>5 minutes</td>
<td>29.8*</td>
<td>2.7</td>
<td>10.3</td>
</tr>
<tr>
<td></td>
<td>1.17</td>
<td>0.52</td>
<td>0.52</td>
</tr>
<tr>
<td>10 minutes</td>
<td>25.8*</td>
<td>2.8</td>
<td>10.5</td>
</tr>
<tr>
<td></td>
<td>0.89</td>
<td>0.50</td>
<td>0.52</td>
</tr>
<tr>
<td>Deflation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 minute</td>
<td>21.0</td>
<td>2.9</td>
<td>10.5</td>
</tr>
<tr>
<td></td>
<td>0.77</td>
<td>0.61</td>
<td>0.61</td>
</tr>
<tr>
<td>5 minutes</td>
<td>20.6</td>
<td>2.4</td>
<td>9.2</td>
</tr>
<tr>
<td></td>
<td>0.99</td>
<td>0.51</td>
<td>0.71</td>
</tr>
</tbody>
</table>

*P < 0.01, †P < 0.05.

Values given are means ± se.
occlusive distention of the pulmonary arterial wall (Tables 1 and 2).

During the control period, no hydrogen signal was detected in the blood of the left pulmonary vein or the left atrium after injection of hydrogen-saturated saline into the right ventricle. After nonocclusive distention of the pulmonary arterial wall, a small, but definite, signal could repeatedly be detected in the blood at these sites. No hydrogen signal was detected during occlusive distention.

Selective pulmonary venous angiograms obtained during nonocclusive distention of the pulmonary artery revealed abrupt tapering and narrowing of medium-size veins, and a marked decrease in retrograde filling of the peripheral veins and capillaries (Fig. 8). In addition, the large pulmonary veins were moderately reduced in size. Angiograms taken after constriction of the cuff were similar to the control angiograms.

Discussion

In the intact urethane-anesthetized dog, segmental distention of the pulmonary arterial wall while keeping the lumen fixed caused a large increase in pulmonary arterial pressures. These data confirm previous observations in dogs with thoracotomy (7), but also demonstrate a smaller, but highly significant, increase in pressure in the small pulmonary veins. These data additionally indicate that since cardiac output and left atrial pressure were not significantly affected, and since average
Pulmonary vascular distending pressure increased while pulmonary blood volume decreased, this pressor response to distention of the arterial wall may be attributed to pulmonary vasoconstriction. The site of this vasoconstriction within the pulmonary vessels is not evident from these data. The increase in pressure difference between the small pulmonary vein and the left atrium, however, suggests constriction of that segment of pulmonary vein. The increase in pressure difference between the pulmonary artery and the small pulmonary vein suggests constriction of vessels upstream to the small pulmonary veins as well, presumably in the small pulmonary arteries. Pulmonary venular constriction is unlikely since these vessels contain no recognizable smooth muscle.

The mechanism producing the pressor response to nonocclusive distention of the pulmonary artery is not evident from these data. Obstruction of pulmonary veins in the vicinity of the distended cuff is not likely to have caused this response since compression of these vessels during distention was not seen at thoracotomy, and since distention of the right pulmonary artery caused the same pressor response in the small pulmonary veins of the left lower lobe as distention of the left pulmonary artery. In addition, pulmonary venous angiograms revealed a decrease in size of these vessels in the left lower lobe during distention of either the right or left pulmonary artery. Mechanical obstruction of these veins by the distended cuff was not seen on these angiograms. The possibility that bronchial obstruction in the vicinity of the distended cuff caused the pressor response is also
unlikely, since no obstruction was found on bronchograms during distention, or after collapse of the cuff, or at inspection of the mediastinum during thoracotomy. Obstructive occlusion of the arterial lumen by eccentric distention of the cuff is improbable since the systolic pressures proximal and distal to the distending site were identical, and angiographic studies of the pulmonary arterial vessels revealed no obstruction. In addition, this pressor response in the pulmonary artery distal to the distended cuff and in the small veins of the left lower lobe occurred during distentions of the right and the left pulmonary artery. Furthermore, the pressor response in the vessels of the unobstructed lung to occlusive distention of the left pulmonary artery were unlike those which occurred during nonocclusive distention. During occlusive distention, the pressor response in the small pulmonary vein of the unobstructed lung was small and transient, and the pulmonary arterial pressor response was less than that during nonocclusive distention.

In similar experiments in dogs with thoracotomy, previous investigators have shown that this pressor response is blocked by infiltration of Xylocaine into the wall of the pulmonary artery (10) and by removal of the adventitia of the artery at the site of distention (7). In those experiments (10), the response was not blocked by hexamethonium, reserpine, bilateral cervical vagotomy, or by denudation of the hilum opposite to the site of distention (7). These investigators have suggested that the pressor response is mediated by pulmopulmonary axon reflexes originating in the pulmonary arteries, and that the afferent limb is contained in the adventitia of the artery. The present experiments in intact dogs confirm the previous observation that this pressor response is not blocked by hexamethonium, reserpine, or bilateral cervical vagotomy, and additionally suggest that serotonin, bradykinin, and histamine are not primarily involved. However, further study of the mechanism producing this pressor response is needed, since blocking agents are more effective against chemical agents given intravenously than against those released locally.

Other studies in dogs with thoracotomy have shown systemic pressor or depressor responses to deformation of baroreceptors in the bifurcation of the pulmonary artery and adjacent areas of the right and left pulmonary arteries (1, 4-7). Few baroreceptors have been found in the main pulmonary artery of the dog, and none have been found in peripheral pulmonary arteries (1); a significant number, however, exist in the main pulmonary artery of the cat (10). Those systemic responses were blocked by bilateral cervical vagotomy. In these experiments on intact dogs, no significant changes in systemic arterial pressure was found with nonocclusive distention of either the main pulmonary artery or its major branches. However, nonocclusive distention in these three sites caused the same pulmonary pressor response, and this response was not blocked by bilateral cervical vagotomy. In addition, a smaller but consistent pulmonary pressor response was observed with nonocclusive distention of the peripheral pulmonary arteries. The reasons for this difference are not clear. However, these experiments, in addition to previous studies (10), suggest that mediation of the systemic and pulmonary vascular responses is different.

The response of the vessels in the right lung to balloon distention of the left pulmonary artery with occlusion of its lumen was unlike the response to nonocclusive distention of the left pulmonary artery. Occlusive distention caused a smaller increase in both pulmonary arterial and pulmonary venous pressures; also, the increase in pressure in the small pulmonary veins was transient. This arterial pressor response in the dogs with occlusive distention is largely passive, as a result of the great increase in blood flow and blood volume in the right lung (9, 20). The significant decrease in pulmonary vascular resistance and the modest increase in average distending pressure after a doubling of blood volume indicate that these vessels had become dilated. These data suggest that, with
occlusive distention of the left pulmonary arterial wall, the increased blood flow and volume in the vessels of the right lung largely overcame the tendency toward vasoconstriction and the observed response was vasodilation. The response of the pulmonary vessels to occlusive distention resembles that of pulmonary vessels to rapid blood transfusion more than it does the response to nonocclusive arterial distention (14).

Since changes in resistance to flow reflect only the changes in caliber of resistance vessels, and since pressure-volume determinations could not be obtained after blocking this pressor response, these data do not completely exclude some increase in vascular tone in the unobstructed lung during occlusive distention. Additionally, the assessment of pulmonary vascular tone in the intact dog is complicated by the inability of available techniques to furnish data regarding the segmental distribution of this increased volume in intact dogs. However, the abolition of this pressor response by infiltration of Xylocaine into the arterial wall of dogs with thoracotomy (9) suggests that vascular tone might have increased during occlusive distention in spite of the increased blood flow and volume.

Although the cause of the fall in $P_{O_2}$ of left atrial blood within 1 minute of nonocclusive distention of the pulmonary artery is not established, the data suggest that this response is due to abrupt opening of pulmonary arteriovenous communications. The possibility that this response was caused by venous admixture in small atelectatic areas in the vicinity of the distended cuff is less likely. Atelectasis in the vicinity of the distended cuff has not caused the abrupt decrease in $P_{O_2}$ of blood in the pulmonary vein from the left lower lobe, or the appearance of the hydrogen signal in the blood of that vessel after right ventricular injection of hydrogen-saturated saline. Similarly, airway obstruction in the vicinity of the cuff-distended left pulmonary artery should have consistently caused a greater fall in $P_{O_2}$ of blood in the left pulmonary vein than that of blood in the left atrium. In addition, inspection of the mediastinum at thoracotomy in dogs with positive-pressure ventilation indicated that the distended cuff in the left pulmonary artery was not in the vicinity of the left lower lobe. Moreover, similar occlusive distentions of the left pulmonary artery in these experiments, and in previous studies (21), did not cause a fall in $P_{O_2}$ of left atrial blood.

Conclusions
In the intact dog, distention of a segment of the pulmonary arterial wall, without changing the diameter of its functioning internal lumen leads to an increase in the pressure in the pulmonary arteries and veins. The data indicate that both arteries and veins constrict. Opening of preterminal arteriovenous communications during distention is suggested by detection of shunted hydrogen and fall in $P_{O_2}$ of left atrial blood.

Distention and occlusion of the pulmonary artery to one lung causes a significantly smaller increase in pulmonary arterial pressure, with a small transient rise in pulmonary venous pressure in the other lung. The increase in flow and blood volume and the fall in resistance to flow offered by vessels in the other lung indicate that these vessels have been passively dilated.

References
ARTERIAL STRETCH RECEPTORS


Circulation Research, Vol. XXIII, September 1968
Pulmonary Vasoconstriction Due to Nonocclusive Distention of Large Pulmonary Arteries in the Dog

ALBERT L. HYMAN

Circ Res. 1968;23:401-413
doi: 10.1161/01.RES.23.3.401

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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