Pulmonary Blood Volume in Experimental Unilateral Pulmonary-Artery Occlusion

By Pedro Aramendia, M.D., Jose D. Fermoso, M.D., and Alberto C. Taquini, M.D.

The behavior of the vascular bed appears to be contradictory. A number of papers indicate that the pulmonary circulation in man shows slight variations in arterial pressure during muscular exercise,¹⁻³ after pulmonary resections⁴⁻⁶ or following the occlusion of a main branch of the pulmonary artery.⁷⁻¹¹ Conversely, other experimental and clinical studies appear to demonstrate that the pulmonary vascular tree is a practically rigid system.¹²⁻¹⁵ This report pertains to the effects of the ligation of a main branch of the pulmonary artery, on blood flow, blood volume and vascular pressures for the pulmonary circulation of anesthetized dogs.

Methods

Dogs received a premedication of morphine (0.1 mg./Kg.) and atropine (0.1 mg./Kg.). One hour later sodium thiopental (25 mg./Kg.) was injected intravenously. A left lateral thoracotomy was performed and a loose ligature was inserted around the left pulmonary artery. Catheters were then passed into the pulmonary artery and the ascending aorta. Constant pulmonary ventilation with room air was maintained with a mechanical respirator during the whole experiment. The intravascular pressures were recorded in a Sanborn Twin-Viso using Statham strain-gage P 23 AA transducers.

A 0.5 per cent solution of T 1824 was injected in the pulmonary artery in less than one second through a Cournand catheter. Immediately afterwards, serial samples, one every second, were collected from a polyethylene catheter lying on the aorta into heparinized test tubes. Time intervals were measured with a metronome and the tubes were manually operated. The amount of dye introduced was determined by weighing the syringe before and after injection and the amount remaining in the pulmonary artery catheter estimated by weighing of the same catheter.

Collection of expired air in a Douglas bag connected to the respiration pump was started just before the dye injection and completed in 2 minutes; gas samples were analyzed for oxygen and carbon dioxide with a Scholander microanalyzer. Ten minutes later, the left pulmonary artery was ligated and once the pressures were stabilized (usually within 1 or 2 minutes) all the measurements were repeated. Blood samples were then centrifuged, the plasma was separated and its optical density was measured in a DU Beckman spectrophotometer at a lengthwave of 625 A. The dye concentrations derived were plotted in semilogarithmic paper with extrapolation of the straight descending limb prior to recirculation. Cardiac output was estimated by the formula:

\[ Q = \frac{I \times 60}{c \times t} / (100 - H_t) \]

where \( I \) = amount of dye injected, \( c \) = mean concentration of dye, \( t \) = transit time of the curve, and \( H_t \) = hematocrit. Mean circulation time was estimated by Hamilton's equation: \( MCT = \frac{\Sigma c \times t}{\Sigma c} \), where \( c \) = dye concentration every second and \( t \) = corresponding time. The pulmonary blood volume was calculated by the formula:

\[ Q_o = MCT \times CO/60 \]

where \( CO \) = cardiac output in 1 minute. All these procedures were described in a previous paper.¹⁶

Stroke volume was estimated by dividing cardiac output by cardiac frequency; the latter was taken from the pressure records obtained during the blood flow determinations. The estimation of the blood gases was carried out with the van Slyke technic for the arterial oxygen content and saturation. At the end of the experiments the animals were sacrificed to verify position of catheters.

Results

Table 1 summarizes the results, before and after ligation of the left pulmonary artery. The cardiac output showed a large variation relative to the body weight of the animals, with a mean value of 83.4 ml./min./Kg. ± 8.62 (S.B.). After ligation the mean was 84.6 ml./min./Kg. ± 9.71. The mean circulation time prior to ligation averaged 9.93 ± 0.86 seconds; after ligation, 8.82 ± 0.66 seconds.
Table 1

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<th>Dog no.</th>
<th>Control</th>
<th>Weight (Kg.)</th>
<th>Cardiac output (L/min.)</th>
<th>Cardiac output (ml/min./Kg.)</th>
<th>Central blood volume (ml./Kg.)</th>
<th>Mean circulation time (sec.)</th>
<th>Oxygen saturation (%)</th>
<th>Pulmonary arterial pressure (mm. Hg)</th>
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The mean circulation time was reduced in 8 cases and was increased in 3.

The intrapulmonary blood volume showed a mean of 12.17 ml./Kg. ± 0.86 before, and reached 11.35 ml./Kg. ± 0.97 after the occlusion of one pulmonary artery (fig. 1). There was a slight tendency towards reduction of the central blood volume in each experiment. The systolic pressure in the pulmonary artery did not alter in 6 cases, and increased by 4 to 6 mm. Hg in the 5 remaining cases. The arterial oxygen saturation was reduced in all cases. The oxygen consumption beforehand was 7.58 ml./min./Kg. ± 0.32, and afterwards 7.67 ml./min./Kg. ± 0.49. The individual variation was not statistically significant.

Discussion

The estimations of the volume of intrapulmonary blood in our experiments agree with those obtained by direct methods,17-20 being about 12 ml./Kg. body weight.

Bearing in mind that there was little change in the blood volume after ligation of the left branch of the pulmonary artery, a real increase in the blood content of the opposite lung must be admitted. The inclusion of the volume of the left heart does not alter appreciably the estimation of the pulmonary blood volume, considering that it is relatively small in size, and that stroke volume is not altered by occlusion of the left pulmonary artery.

This behavior has been described previously by Salonikides in pneumonectomized patients, in whom he observed no alteration in the amount of central blood compared with preoperative estimations.6

The pressure of the pulmonary artery did not increase overtly after ligation, which agrees with observations by numerous authors,7-11 but disagrees with the findings of others.21, 22 The changes in pressure observed above were not related to the alterations of
cardiac output. It must be noted, however, that following arterial ligation there was an immediate increase in pressure, followed by a progressive fall to previous levels, or stabilization at a slightly higher level with the exception of dogs 2, 3 and 9 which showed no variation throughout the whole procedure.

The adaptation of the pulmonary arterial pressure to vascular occlusion can be explained by the following factors:

1. The extravascular pressure produced by the respirator on the vascular bed of a congested lung will act in an important manner, and may perhaps be responsible for the increase in pressure maintained, in some cases, after the initial rise.

2. The existence of pulmonary vascular pressoreceptors, producing bradycardia and vasodilatation with increased flow, would seem to be proven and they appear to be localized in the pulmonary artery trunk. In our experiments, this area does not increase its flow, which is entirely diverted to the patent branch. No receptors have been identified in the principal branches of the pulmonary artery or in pulmonary veins. Nevertheless, it seems difficult to rule out that a reflex mechanism of this sort plays any part in the stabilization of pressures.

3. It is possible that the diversion of blood flow caused by the elimination of the circulation of one lung may, at first, utilize some of its kinetic energy in the dilatation of the vascular bed, overcoming the critical opening pressure of some vessels which are nonfunctional with the lung at rest. This interpretation seems to agree with experiments on isolated lungs and the adaptation of pulmonary vascular pressures during exercise in human beings.

We think that the deficient arterial oxygen saturation found after pulmonary ligation with an open thorax is due to the maintenance of a constant ventilation (by means of the artificial respirator), in contrast with the increased flow of the lung with circulation intact. This situation creates at the same time an alteration in the ventilation/perfusion relationship, which may be responsible for the disturbance in the arterial saturation. On the other hand, in pulmonary ligation with a closed thorax and spontaneous respiration there occurs hyperventilation which maintains normal alveolar ventilation.

**Summary**

In a series of 11 dogs in which the left pulmonary artery was ligated, the blood from the occluded lung was absorbed by the remainder of the vascular bed and, consequently, there was no significant alteration in the pulmonary blood volume. This adaptation took place, apparently, with only slight modifications in the circulation time and the pressure of the system. Examination of the variables investigated suggests, to a certain extent, that the increased flow brings about the opening-up of new vascular channels.

**References**

2. HICKAM, J. B., AND CABGILL, W. H.: Effect of exercise on cardiac output and pulmonary arterial pressure in normal persons and in patients with cardiovascular disease and pul-

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**Figure 1**

Central blood volume before and after occlusion of one main branch of the pulmonary artery. The rectangles represent standard deviation. Mean figures for both control and occlusion periods and its standard errors are shown at the bottom.
UNILATERAL PULMONARY ARTERY OCCLUSION


15. SANZETTA, S. M., AND RAKITA, L.: Response of pulmonary artery pressure and total pul-


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