Pathophysiological Changes in the Lungs During Extracorporeal Circulation

By Robert S. Cartwright, M.D., Thomas P. K. Lim, M.D., Ph.D., Ulrich C. Luft, M.D., and William E. Palich, D.V.M.

Management of the lungs during heart-lung bypass for cardiac surgery should be directed toward minimizing possible adverse effects resulting from gross alterations of perfusion and ventilation of the organ, thus permitting normal gas exchange to be resumed immediately after termination of bypass. So far the approach to this end has been mainly empirical, and various forms of static inflation, intermittent ventilation, or passive deflation have been employed without knowledge of the merits of each. Kottmeier and associates reported an increased incidence of peribronchial hemorrhage and patchy atelectasis when pulmonary ventilation was omitted during animal heart-lung bypass procedures. The detrimental effects of an elevated pulmonary venous pressure on the lungs during and following bypass have been frequently demonstrated. Schramel et al. found pulmonary diffusion capacity to be decreased following thoracic operations and believed this deficiency was greater when heart-lung bypass was utilized. Recent data suggested a significant reduction in pulmonary compliance following open-heart surgery due to disturbed mechanics of breathing.

The present study was undertaken (a) to ascertain the physiological implications of bypass to pulmonary function with regard to the quantity and quality of bronchial collateral blood flow, metabolic requirements of the lung tissue, and alterations of pulmonary compliance, and (b) to establish the most suitable management for maintaining an adequate condition in the lungs and the systemic circulation. Some of the observations gained in the experimental series were extended to several clinical cases.

Methods

A total of 29 experimental and 68 donor dogs was used. The experimental animals were anesthetized with pentobarbital sodium (30 mg./Kg., I.V.) and tracheotomized. Following heparinization (2 mg./Kg.), the femoral vessels were exposed bilaterally to monitor arterial and venous pressures and also to return the oxygenated blood from the pump. Room air (nonhumidified) was used in artificial respiration. In all animals, the chest was opened by a vertical incision splitting the sternum and entering both pleural spaces.

A modified Kay-Cross rotating disk oxygenator primed with blood from the donor dogs was utilized. A gas mixture having 2.0 ± 0.2 percent CO2 in O2 was delivered to the oxygenator at a rate of 3 to 4 L./min. Total body perfusion was conducted for 60 to 80 minutes at flow rates of 50 to 100 ml./Kg./min. maintaining a mean arterial pressure of 80 to 110 mm. Hg and an average esophageal temperature of 37 C.

Two animal preparations were used. In the first preparation, systemic, arterial and caval blood flows and pressures were continuously monitored, while the coronary circulation was excluded from the perfusion by a clamp across the aorta and pulmonary artery at the level of the semilunar valves with resulting hypoxic arrest of the heart. In addition, the left atrial blood was allowed to drain into a container (preparation A) (fig. 1). The second preparation differed from the first in that coronary flow was left intact. Caval blood flow including azygos venous return was directed to the oxygenator. Thus, the heart maintained its sinus rhythm throughout bypass (preparation B). In both preparations, care was taken to maintain left atrial pressure 1 to 4 em. H2O greater than right atrial pressure by manually adjusting the height of the veins receiving reservoir located between the oxygenator and the venae cavae. This was done to avoid a possible reversal of blood flow from the superior vena cava into the pulmonary veins by way of azygos-branchial venous communication when the caval pressure exceeds the left atrial pressure.

During bypass, the lungs were either (1) statically inflated with the surface of the lungs reaching the upper margin of the chest walls (7 to 15 cm. H2O pressure), (2) ventilated at 1 to 6 L./min., or (3) allowed to remain deflated, the sequence being varied at random. In each instance, after a steady state of 20 minutes, blood
samples were taken anaerobically from the systemic artery, vena cava, and pulmonary vein and were analyzed for 
\( O_2 \) saturation, \( pCO_2 \), and pH. The expired air during artificial respiration was collected in a Douglas bag, and the respiratory and blood gases were analyzed according to methods described previously.\(^7\) In those animals having intact coronary flow, the right atrial blood was similarly analyzed. The total peripheral resistance was calculated by Frank’s formula in absolute units.\(^8\) In addition, pulmonary compliance was measured by stepwise increases of lung volume from the passively deflated condition up to 800 ml and by concomitant measurement of static transpulmonary pressure. The latter was obtained by a water manometer. Statistical methods used in this work follow Snedecor.\(^9\)

**Results**

**Effects of varied insufflation of bypassed lungs on blood gases**

Three types of artificial lung insufflation, namely passive deflation, static inflation, and artificial respiration, were administered to 10 animals of preparation A. Since the systemic and the coronary venous returns were prevented from entering the pulmonary vascular beds in this preparation (see fig. 1), the bronchial collateral flow constituted the sole perfusate of the lungs. The data on the blood gases during these maneuvers are summarized in table 1. When the pulmonary venous blood was compared with the systemic arterial blood during passive deflation, the former showed consistently lower \( O_2 \) saturation and higher \( CO_2 \) tension and hydrogen ion concentration than the latter. These differences amounted to 28 per cent of \( O_2 \) saturation, 7 mm. Hg of \( pCO_2 \), and 0.02 units of pH and were suggestive of appreciable exchanges of \( O_2 \) and \( CO_2 \) between the lung tissues and the pulmonary perfusate.

With static inflation of the bypassed lungs with room air, the \( O_2 \) saturation, \( pCO_2 \), and pH of the pulmonary venous blood approached those noted in the systemic arterial blood, as shown in table 1. Except for \( O_2 \) saturation which was still 89 per cent, static inflation restored the chemical values (\( CO_2 \) content, \( pCO_2 \), and pH) of the pulmonary venous blood to practically the same levels as the systemic arterial blood.

With artificial respiration, it was possible to bring the \( O_2 \) saturation of the pulmonary venous blood to the level of the systemic arterial blood. Nevertheless, this was achieved only after considerable depletion of the \( CO_2 \) reservoir as reflected in lowered \( CO_2 \) and high pH.

In preparation B, in which the lung perfusate consisted of both coronary venous and bronchial collateral flow, \( O_2 \) saturation of pulmonary venous blood was markedly reduced to 42.6 per cent during passive deflation of the lungs (table 1). This level was much lower than that of the mixed venous blood (approximately 20 per cent below) due
Effect of Various Artificial Respiration on Blood Gases During Heart-Lung Bypass*†

<table>
<thead>
<tr>
<th>Sample</th>
<th>O₂ content (vol. %)</th>
<th>O₂ capacity (vol. %)</th>
<th>O₂ saturation (%)</th>
<th>CO₂ content (plasma, mM/L.)</th>
<th>pH</th>
<th>pCO₂ (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparation A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.V.</td>
<td>11.5 ± 1.8</td>
<td>19.88 ± 2.0</td>
<td>50.4 ± 6.8</td>
<td>21.6 ± 2.2</td>
<td>7.24 ± 0.06</td>
<td>48.0 ± 1.9</td>
</tr>
<tr>
<td>P.V.</td>
<td>14.0 ± 2.5</td>
<td>19.88 ± 2.0</td>
<td>68.5 ± 11.1</td>
<td>18.9 ± 2.4</td>
<td>7.26 ± 0.08</td>
<td>41.1 ± 4.1</td>
</tr>
<tr>
<td>S.A.</td>
<td>19.7 ± 1.1</td>
<td>19.88 ± 2.0</td>
<td>96.7 ± 4.7</td>
<td>17.5 ± 2.2</td>
<td>7.38 ± 0.06</td>
<td>35.7 ± 0.7</td>
</tr>
<tr>
<td>Preparation B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.V.</td>
<td>12.2 ± 3.5</td>
<td>19.88 ± 2.0</td>
<td>62.2 ± 11.0</td>
<td>21.4 ± 2.2</td>
<td>7.29 ± 0.06</td>
<td>48.0 ± 1.8</td>
</tr>
<tr>
<td>P.V.</td>
<td>17.3 ± 1.9</td>
<td>19.88 ± 2.0</td>
<td>80.3 ± 5.8</td>
<td>18.4 ± 3.0</td>
<td>7.36 ± 0.11</td>
<td>32.2 ± 6.4</td>
</tr>
<tr>
<td>S.A.</td>
<td>19.3 ± 2.2</td>
<td>19.88 ± 2.0</td>
<td>96.7 ± 3.6</td>
<td>18.4 ± 2.8</td>
<td>7.35 ± 0.07</td>
<td>35.3 ± 3.3</td>
</tr>
</tbody>
</table>

I. Passive deflation  
II. Static inflation (7 to 15 cm. H₂O)—room air  
III. Artificial respiration (1.3—4.4 L./min.)—room air  

*Figures are arithmetic mean ± standard deviation.

**Abbreviations:** M.V. = mixed venous samples; P.V. = pulmonary venous samples; and S.A. = systemic arterial samples.

Primarily to the low O₂ saturation of the coronary venous blood.

Interestingly enough, in contrast to preparation A, static inflation did not markedly improve the aeration of the pulmonary venous blood. The O₂ saturation remained at 51.6 per cent which was still below that of the mixed venous blood, while the CO₂ content, pH, and pCO₂ were almost the same as those of the mixed venous blood. Thus, it was apparent that during heart-lung bypass with coronary blood flow circulating through the pulmonary vasculature, static inflation did not provide a normal physiological environment to the bypassed lungs.

It was only with the aid of artificial respiration that both the O₂ saturation and the acid-base parameters of pulmonary venous blood could be maintained close to those of the systemic arterial blood. Employing varying amounts of artificial respiration which ranged from 0.7 to 6.5 L./min., it was possible to bring the O₂ saturation up to 90.1 per cent with somewhat lower CO₂ content and pCO₂ and a higher pH than those of the arterial blood (table 1).

BRONCHIAL COLLATERAL FLOW

Normally the pulmonary perfusate consists of three components: vena caval flow, coronary venous return, and bronchial collateral flow. By placing a clamp at the level of the aortic valve during bypass, as in preparation A, with a precautionary measure of maintaining left atrial pressure 1 to 4 cm. H₂O higher...
### Hemodynamic Changes During Bypass†

<table>
<thead>
<tr>
<th>Preparation A (body wt. = 14.3 ± 2.0 Kg., n = 10)</th>
<th>Preparation B (body wt. = 12.3 ± 1.8 Kg., n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I. Passive deflation</strong></td>
<td><strong>I. Passive deflation</strong></td>
</tr>
<tr>
<td>Perfusion rate (L/min.)</td>
<td>Perfusion rate (L/min.)</td>
</tr>
<tr>
<td>1.2 ± 0.4</td>
<td>1.2 ± 0.2</td>
</tr>
<tr>
<td>Esoph. temp. (°C)</td>
<td>Esoph. temp. (°C)</td>
</tr>
<tr>
<td>37.0 ± 0.7</td>
<td>37.0 ± 0.7</td>
</tr>
<tr>
<td>T1</td>
<td>T1</td>
</tr>
<tr>
<td>100 ± 13</td>
<td>112 ± 15</td>
</tr>
<tr>
<td>T2</td>
<td>T2</td>
</tr>
<tr>
<td>4.2 ± 4.5</td>
<td>−3.3 ± 4.3</td>
</tr>
<tr>
<td>T3</td>
<td>T3</td>
</tr>
<tr>
<td>7.8 ± 5.5</td>
<td>4.8 ± 1.2</td>
</tr>
<tr>
<td>T4</td>
<td>T4</td>
</tr>
<tr>
<td>7.5 ± 3.4</td>
<td>52 ± 13</td>
</tr>
</tbody>
</table>

| **II. Static inflation**                         | **II. Static inflation**                         |
| Perfusion rate (L/min.)                          | Perfusion rate (L/min.)                          |
| 1.1 ± 0.1                                        | 1.2 ± 0.2                                        |
| Esoph. temp. (°C)                                | Esoph. temp. (°C)                                |
| 37.0 ± 0.7                                       | 36.9 ± 0.7                                       |
| T1                                               | T1                                               |
| 111 ± 11                                        | 103 ± 18                                         |
| T2                                               | T2                                               |
| 1.4 ± 2.3                                        | −3.0 ± 2.1                                       |
| T3                                               | T3                                               |
| 2.6 ± 1.8                                        | 4.6 ± 3.7                                        |
| T4                                               | T4                                               |
| 6.3 ± 4.2                                        | 47 ± 17                                          |

| **III. Artificial respiration**                  | **III. Artificial respiration**                  |
| Perfusion rate (L/min.)                          | Perfusion rate (L/min.)                          |
| 1.2 ± 0.3                                        | 1.2 ± 0.2                                        |
| Esoph. temp. (°C)                                | Esoph. temp. (°C)                                |
| 37.2 ± 0.7                                       | 36.8 ± 0.9                                       |
| T1                                               | T1                                               |
| 97 ± 30                                          | 94 ± 19                                          |
| T2                                               | T2                                               |
| 3.7 ± 4.2                                        | −1.0 ± 4.7                                       |
| T3                                               | T3                                               |
| 7.3 ± 5.3                                        | 3.6 ± 3.2                                        |
| T4                                               | T4                                               |
| 10.7 ± 5.5                                       | 32 ± 19                                          |

**Abbreviations:**  
- $\bar{P}_a$: mean systemic arterial pressure;  
- $\bar{P}_v$: mean caval pressure;  
- $\bar{P}_l$: left atrial pressure;  
- $\dot{Q}_l$: blood flow through left atrium.

† Figures are arithmetic mean ± standard deviation.

‡ Significant difference.
than right atrial pressure (to prevent back-flow through azygos-bronchial venous connections), it was possible to quantify the bronchial collateral flow by continuously draining the blood from the left atrium by gravity. The data from 10 animals of preparation A are shown in the last column of table 2. The average body weight of the animals was 14.3 ± 2.0 Kg. (range: 11.4 to 17.3 Kg.) with corresponding mean bronchial flow rates (QJB) of 7.5, 6.3, and 10.7 ml./min. during passive deflation, static inflation, and artificial respiration, respectively. As indicated by the paired comparison in table 2, the differences between these values were not statistically significant. Therefore, the data were pooled together yielding an average bronchial flow of 8.8 ± 4.9 ml./min. (range: 2.4 to 19.0 ml./min.), which was equivalent to 0.7 per cent of the total systemic flow. An attempt to reveal correlation between the gross body weight and the bronchial flow in these animals was unsuccessful, perhaps due to the narrow range of body weight studied and the accompanying variations during the surgery. Furthermore, in subsequent studies in five other dogs, varying the superior vena caval pressure from —10 to +40 cm. H2O relative to the left atrial pressure did not significantly affect the bronchial flow as shown in figure 2.

**METABOLIC RATE OF THE LUNG TISSUES**

The experimental conditions of preparation A were particularly suited for the metabolic study of the lungs. Since the lungs were solely perfused by fully oxygenated blood (i.e., bronchial arterial blood) in this preparation, it was possible to determine the O2 consumption of this organ by collecting and analyzing the expired air during artificial ventilation. The O2 consumption of the lungs measured in this fashion revealed an average value of 1.6 ± 0.8 ml./min. (range: 0.4 to 3.3 ml./min.), with the mean CO2 output of 4.3 ml./min. (range: 2.7 to 6.0 ml./min.). Considering an average body weight of 14.3 Kg. for the animals and a mean respiratory gas exchange prior to bypass of 99 ml./min. of O2 and 81 ml./min. of CO2, it is concluded that the metabolic requirement of the lungs is quite small, amounting to only 1 to 2 per cent of the total metabolic rate.

**HEMODYNAMIC CHANGES DURING BYPASS**

The blood collected from the left atrium in preparation B averaged 52, 47, and 32 ml./min. during passive deflation, static inflation, and artificial respiration, respectively, as shown in table 2. Although the greater amount of drainage seen in this preparation as compared to that in preparation A was undoubtedly due to the large coronary venous return in the former, these volume figures in preparation B did not warrant accurate quantitation of the coronary venous return due to probable escape of blood through the left ventricle into the aorta.

The mean values of systemic arterial (Psa), vena caval (Pcv), and left atrial (Pva) pressures during bypass are also shown in table 2. In preparation A, these pressures were maintained at practically the same levels, whereas the systemic arterial pressure was somewhat lower during artificial respiration...
in preparation B. Since the sequence of lung manipulation was purposefully altered at random, the latter phenomenon could not be attributed to the time factor. The estimation of total peripheral resistance from the pressure gradient and flow rate in 16 animals revealed mean values of 7,540; 7,860; 6,980; and 6,610 dynes/sec./cm.² after 5, 20, 40, and 60 minutes of bypass, respectively, as shown in figure 3. The statistical comparison of these values at 20 and 40 minutes of bypass with that at 5 minutes of bypass indicated no significant difference. However, the value at 60 minutes of bypass was significantly different at the 5 per cent level. Thus, it may be inferred that in our preparation, the total peripheral resistance remained fairly constant up to 40 minutes of bypass and after that showed a tendency to decrease.

**CLINICAL OBSERVATION**

The experimental study described above clearly demonstrates that passive deflation of the bypassed lungs is unphysiological and possibly detrimental to the welfare of the patient. Under this guiding principle, our observations were extended to five patients undergoing open-heart surgery (table 4). In all cases, except J. L., blood samples were drawn while the coronary return flow was perfusing the lungs, the condition being, therefore, comparable to those of preparation B. In J. L., the lungs were maintained statically inflated while the rest of the patients were ventilated at the rates of 2.0 to 5.0 L./min. during bypass. The systemic arterial, mixed venous, and left atrial samples were analyzed, and in two cases (D. G. and L. G.) the right atrial sample was also taken.

In the case of J. L., where coronary venous blood was diverted to the oxygenator by suction, static inflation of the lungs with a gas mixture of 50 per cent helium in O₂ gave satisfactory O₂ saturation but did not completely correct acid-base disturbance of the left atrial blood. On the other hand, in the remaining patients, the samples taken while the coronary sinus blood circulated through the lungs showed the rate of artificial respiration employed to be excessive, as manifested by the low pCO₂ and high pH values. The results suggest that, when using a high concentration of O₂, the optimal rate of assisted ventilation during bypass with intact coronary flow is in the neighborhood of 2.0 L./min.

**Discussion**

Pulmonary complications have been reported as a major cause of mortality and morbidity following cardiopulmonary bypass. Although some of these features may be explained on the basis of maladjustment of the pulmonary vasculature following corrective surgery, there is enough evidence to suggest that a source of such disturbance may reside in the lack of factual knowledge concerning
proper management of the lungs during bypass. Dodrill listed various theories of pulmonary complications encountered following extracorporeal circulation and reported pathological findings of "collapsed lungs" with altered elastic fibers. This type of pulmonary collapse was quite different from ordinary postoperative atelectasis because the former had no segmental distribution and was characterized histologically by areas of collapse surrounded by aerated alveoli. Norlander et al. observed not only pulmonary edema caused by the overloading of the lungs during bypass, but also a type of postoperative lung complication which had patchy areas of increased density on the chest roentgenogram with decreased O₂ saturation of peripheral arterial blood. The concurrent frothy secretions from the lungs of these patients led the authors to believe that there was damage to the alveolar membranes.

As shown in our study, the composition of pulmonary venous blood could deviate markedly from the physiological range in both preparations when adequate support of artificial respiration was not provided. The vasoconstrictor effect of hypoxia on pulmonary capillary beds has been reported. In experimental studies in which systemic and lesser circulations were controlled independently, a reduction of temperature to a subnormal level or an increase in acidity (by means of CO₂ inhalation) of lung perfusate has been found to increase pulmonary vascular resistance. Thus, hypoxia, hypercapnia, and hypothermia all exert influences on the pulmonary vasculature. The avoidance of these adverse features during heart-lung bypass may be achieved either by static inflation of the lungs when the heart is arrested or by artificial respiration at a rate of approximately 2 L/min when the heart is not arrested. It is common practice to divert the coronary sinus blood to the oxygenator by suction from the right atrium. In such a case, since the lungs are solely perfused by the bronchial collateral flow, static inflation of the lungs should be sufficient to maintain normal physiological conditions.
Several attempts have been made in the past to measure the bronchial blood flow in experimental animals and in man, utilizing either direct or indirect methods. The indirect method is based on the simultaneous measurements of the right and left ventricular outputs, and the direct measurement is necessarily marked by the excessive dissection and ligation of the blood vessels. The employment of extracorporeal circulation seems to offer one of the best physiological conditions in which the bronchial flow can be quantitated directly. The elimination of caval and coronary venous returns leaves only the bronchial flow (and negligibly minute thebesian return) to empty into the left heart, and the average value of 8.8 ml./min. under normal arterial pressure and body temperature compares well with the values reported by others. Although the anatomical complexity of the bronchial circulation is apparent, and the possible anastomosis between mediastinal vessels and small bronchial veins is cited by Hayek, the quantity of bronchial blood flow lost to the right heart through this route is assumed to be negligibly small.

Our findings of constant bronchial flow during three different types of lung manipulation with or without cardiac arrest tends to negate the concept expressed by Muller and his associates, who believed that the manual movement of the lungs in open-chest preparation increased the bronchial flow. In agreement with our observations, Horisberger and Rodbard reported that the bronchial blood flow did not change until the lung insufflation pressure reached an excessive level of 20 to 30 cm. H₂O in dogs.

The constancy of bronchial flow despite increased systemic venous pressure, as shown in figure 2, contrasts with the observation made by State and his co-workers, who noted a positive relationship between systemic venous pressure and bronchial collateral flow. The reason for this difference is unknown except that the pulmonary circulation was artificially maintained by a pump in the latter, while it was devoid of main flow in our preparation.

In the past, oxygen consumption of the lung has been estimated using either slices of lung tissue by Warburg’s technique or isolated lung preparations. Differing from these customary methods, our technique is unique in that (1) it allows estimation of oxygen consumption of whole lungs, (2) it is done in situ, and (3) the lungs are perfused with oxygenated blood (bronchial arterial blood). Some technical aspects in our method which deserve further attention are (a) that the difference in oxygen content of systemic arterial and pulmonary venous blood is practically nil during artificial respiration of the bypassed lung (i.e., 19.4 versus 19.2 volume per cent of O₂ with no statistical difference)—this fact suggests that the oxygen consumed by the lung tissue is rapidly supplemented from the air by the diffusion process; (b) that the O₂ content of expired air in the Douglas bag averaged 20.82 per cent (range: 20.63–20.88 per cent) which indicates that although the amount of oxygen consumption is indeed small, its magnitude is sufficiently large to be detected by the standard method of gas analysis (Scholander technique with ± 0.03 per cent of accuracy); and (c) in contrast to the average O₂ consumption of 1.6 ml./min., the corresponding CO₂ output was 4.3 ml./min., which gave a respiratory quotient of 2.7. This was due to the "Auspummpung" of CO₂ during the artificial respiration which ranged from 1 to 4 L./min. and was also perhaps due to higher CO₂ storage capacity of the lungs relative to oxygen.

The mean value for oxygen consumption of canine lung in this study (i.e., 1.6 ± 0.8 ml./min. in 10 dogs having an average body weight of 14.3 Kg.) compares well with that reported by Bostroem and Lochner. During perfusion of the pulmonary artery of an isolated lung with arterial blood of the donor’s dog, the latter found 1.4 ± 0.5 ml./min. of O₂ consumption of whole lungs in nine dogs having an average body weight of 11.4 Kg. Recent data by Aviado gives an average value of 0.64 ml./min./100 Gm. of wet lung. Assuming the wet weight of the lungs in our...
EXTRACORPOREAL CIRCULATION

preparation is 150 Gm., this provides approximately 1.0 ml./min. of O2 uptake of whole canine lung.

Among many factors which influence vasomotor tone, arterial and venous pressures, perfusion rate, and circulating blood volume are of prime importance. In addition, the temperature, acidity, viscosity of perfusing blood, and the depth of anesthesia contribute significantly to the vasomotor regulation. As a preliminary approach to the problem, the total peripheral resistance was calculated over 60 minutes of bypass in our preparation. The result indicated a fairly constant resistance up to 40 minutes of bypass and a slight reduction at the end of one hour of bypass under conditions of normothermia and normotension (fig. 3, table 2).

Contrary to our findings, Moffitt et al.4 reported a gradual increase in peripheral resistance during bypass in patients. Their rate of perfusion, however, was rather low, the mean perfusion rate being 1.7 L./min./M.2 in contrast to the preoperative cardiac index of 4.2 L./min./M.2. Furthermore, the mean arterial pressure during bypass ranged from 27 to 50 mm. Hg, with an average reduction of the patient’s rectal temperature of 1.7 C. Cordell and his associates5 found a gradual increase in peripheral resistance in seven dogs during 30 minutes of bypass under normothermia and normotension. They also perfused the animal at a low rate of approximately 800 ml./min. In an extensive study on vasomotor regulation during bypass in man, Sanger et al.6 observed a somewhat phasic alteration in peripheral resistance: namely, an initial reduction during 5 minutes of bypass, an increase during 10 to 40 minutes, and a gradual decrease during 40 to 80 minutes of bypass. Their rate of perfusion was 50 ml./Kg./min. in adults and 70 to 100 ml./Kg./min. in children. Thus, it appears that, other factors being kept constant, the peripheral resistance tends to increase when the low flow rate of perfusion is used, whereas the trend is reversed with the employment of high flow rate. This conclusion is in agreement with that of Ankney and his associates7 who showed that peripheral resistance progressively declines as the pumping rate is increased. Cloves28 suggests that this may be due to the effect of “buffer” nerves.

In a previous investigation,7 pulmonary compliance of anesthetized intact (closed-chest) dogs was found to be 34.1 ± 2.8 ml./cm. H2O in 10 animals (mean body weight, 15 Kg.). In the present study, the average figure in anesthetized open-chest dogs gave a mean value of 36.0 ± 7.0 ml./cm. H2O in 13 animals (average body weight, 13.4 Kg.) (table 3), revealing close agreement of values for pulmonary compliance in the two groups.

Contrary to our findings, a much higher lung compliance was reported by Guastavino and his associates4 who found a value of 134 ml./cm. H2O (range: 60 to 240 ml./cm. H2O) in 10 anesthetized open-chest dogs having an average body weight of 15.8 Kg. This figure is almost three times as great as our own, approaching values recorded in human beings. The reason for this discrepancy is not known, the only apparent difference in the two studies being the use of air for lung insufflation in our work, while oxygen was used in the other study. It must also be mentioned that leakage of gas from the lungs due to surgical trauma will reduce the static transpulmonary pressure, giving an erroneously high “pulmonary compliance.”

Summary

An attempt has been made to determine the optimal method of managing the lungs during cardiopulmonary bypass. The chemical behavior (oxygen saturation, acid-base balance) of the pulmonary venous blood indicates (a) that static inflation of the lungs with either a gas mixture of high oxygen content or ambient air is sufficient to maintain a proper internal milieu for the pulmonary vasculature when the bronchial collateral flow is the sole pulmonary perfusate, and (b) that in adult human beings artificial respiration up to 2 L./min. is necessary when the pulmonary perfusate consists of the coronary venous blood in addition to the bronchial collateral flow.

The effective application of the heart-lung
bypass procedure in physiological investigation is depicted by the studies on metabolic rate of the lung tissues and the measurement of bronchial flow. It is estimated that the bronchial collateral flow amounts to 8.8 ml./min. or 0.7 per cent of the total systemic flow, and that the oxygen consumption of the lungs averages 1.6 ml./min. or 1 to 2 per cent of the total metabolic rate in the open-chest dogs. The pulmonary compliance did not change following either passive deflation or static inflation, but is transiently reduced after artificial respiration.

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