Effects of Lung Inflation on Blood Flow During Cardiopulmonary Resuscitation in the Canine Isolated Heart-Lung Preparation

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Using an isolated, fibrillated canine heart-lung preparation, we studied the effects of simultaneous lung inflation and chest compression on blood flow in a model of cardiopulmonary resuscitation. The heart and lungs were placed in an artificial thorax with the great vessels and trachea exteriorized and attached to an artificial perfusion circuit and respirator, respectively. The blood volume of the system was adjusted to obtain various levels of static equilibrium pressure. Blood flow was obtained by cyclically raising and lowering the pressure in the artificial thorax, simulating the changes in pleural pressure that occur during cardiopulmonary resuscitation. Lung inflation during the compression phase caused an increase in cardiopulmonary resuscitation blood flow when the change in pleural pressure was small and when static equilibrium pressure was high. In contrast, lung inflation caused a decrease in blood flow when changes in pleural pressure were high and when blood volume was low. These results suggest that the driving pressure for blood flow during chest compression may be increased by lung inflation when the pulmonary blood vessels are filled with blood. However, blood may become trapped in the right heart and unavailable for transfer to the periphery during chest compression if lung inflation causes the alveolar blood vessels to collapse. (Circulation Research 1986;59:676–683)

The traditionally accepted mechanism for cardiopulmonary resuscitation blood flow is direct compression of the heart. Certain evidence suggests that under most but not all conditions cardiopulmonary resuscitation (CPR) blood flow is produced by the thoracic pump mechanism. External chest compression causes an increase in the pressure in the compliant vascular structures in the thorax, creating a pressure gradient for the flow of blood to the extrathoracic vasculature. Venous valves and collapse of the great veins prevent retrograde blood flow to the extrathoracic veins. When chest compression is released, blood returns to the thorax by passive discharge of the compliant portions of the peripheral vasculature.

Simultaneous application of high airway pressure and chest compression has been shown to generate greater blood flow than conventional CPR. This has been attributed to the generation of higher pleural pressures during the duty cycle, which in turn causes higher pressures in the thoracic vasculature. However, it is possible that changes in airway pressure may also affect CPR blood flow independent of its effects on pleural pressure. With conventional CPR, lung gas volume decreases during chest compression since pleural pressure, which surrounds the lungs, increases relative to alveolar pressure. With the application of high airway pressure with chest compression, lung gas volume may increase, decrease, or remain unchanged, depending on whether alveolar pressure rises, falls, or remains unchanged relative to pleural pressure.

Lung inflation may cause blood to be expelled from pulmonary vessels in the alveolar compartment, since the surrounding pressure of these vessels increases relative to pleural pressure as the lung inflates. Therefore, we hypothesized that for the same change in pleural pressure, chest compression with simultaneous lung inflation would generate greater blood flow than conventional CPR. Using an isolated heart-lung preparation, we have studied the effects of lung inflation on CPR blood flow independent of its effects on pleural pressure.

Materials and Methods

Surgical Preparation

Twenty mongrel dogs of either sex weighing 20–25 kg were anesthetized intravenously with 30 mg/kg pentobarbital, with supplemental doses given as required to maintain adequate anesthesia. The animals were intubated and mechanically ventilated with supplemental oxygen by a volume respirator (Harvard Apparatus, Braintree, Mass.). The chest was opened by midsternal thoracotomy. The azygous vein and distal esophagus were ligated and divided, and the remaining vessels entering or leaving the thorax were isolated. Beef heparin (10,000 U) was administered, and the
superior vena cava and brachiocephalic artery were cannulated. The cannulae were connected to a mechanical analogue of the peripheral vascular system (Figure 1). The dog was exsanguinated into the perfusion circuit, which was primed with 6% dextran-saline solution. The subclavian artery, descending aorta, and inferior vena cava were ligated and divided. Thereafter, the heart was removed from the thorax, and the pericardium was excised. A fluid-filled polyethylene catheter was placed in the left ventricle through a stab wound and was secured with a purse-string suture.

**ARTIFICIAL THORAX.** The isolated heart-lung preparation was placed in a 23 x 23 x 28 cm plexiglass chamber that served as the artificial thorax. The arterial and venous cannulae were briefly clamped and disconnected from their connecting conduits, exteriorized, and reconnected to the peripheral perfusion circuit. The tracheal cannula was also exteriorized and connected to an artificial pressure source by which tracheal pressure could be cyclically raised and lowered. The chamber was connected to a second pressure source by which chamber pressure could be cyclically raised and lowered.

**PERFUSION CIRCUIT (FIGURE 1).** After fibrillation of the heart with a 60 Hz AC current, CPR was simulated by cyclically raising and lowering the chamber pressure. When chamber pressure was raised, blood flowed from the heart and lungs through the brachiocephalic cannula and the connecting silastic tubing to an electromagnetic flow probe (EM600, Carolina Medical, King, N.C.). Blood then passed through a Starling resistor with a surrounding pressure of 30 mm Hg, represented the venous valves, which prevent retrograde flow in the intact animal. 

**DEPENDENT VARIABLES.** The static equilibrium pressure of the system (Ps) is analogous to mean systemic pressure in the intact animal, and was measured at the base of the reservoir with chamber and tracheal pressures constant at 0 mm Hg. The level of Ps was adjusted by adding or removing blood from the reservoir. The levels to which the chamber (PPl) and tracheal (PT) pressures were raised during the duty cycle were independently adjusted according to the protocols that follow.

**INDEPENDENT VARIABLES.** Pressures were measured in the left ventricle (Plv), the extrathoracic arterial conduit (Pao), the base of the reservoir (Pres), and the extrathoracic venous conduit (Pra) between the one-way valve and the chamber. All pressures were measured with Statham P23dB pressure transducers and were referenced to the base of the reservoir. Blood flow was measured with a square wave electromagnetic flow meter (Carolina Medical Electronics, King, N.C.). Stroke volume was determined by electronic integration of the flow signal. Physiologic variables were continuously recorded on a Gould multichannel recorder.

**Protocol**

**SERIES 1** (INTERACTION BETWEEN CHANGES IN PPL AND PT). Eight preparations were studied at a frequency of compression of 30 cycles/min with a duty cycle (compression time/total cycle time) of 0.5. Blood volume in the system was adjusted so that Ps equalled one of the following: 7.5, 15.0, or 22.5 mm Hg. At each level of Ps CPR blood flow was obtained by cyclically raising and lowering Ppl with each of the following swings in pressure (ΔPpl): 0 to 10, 0 to 20, 0 to 30, 0 to 40, 0 to 50, and 0 to 60 mm Hg. The effects of four different
degrees of lung inflation during chamber compression were compared at each level of Ppl by adjusting the airway pressure source to provide each of the following swings in Pj (ΔPT): 0, 0 to 10, 0 to 20, and 0 to 30 mm Hg. The difference between ΔPj and ΔPpl (ΔPT − ΔPpl) indicates the level of lung inflation during the duty cycle, LI. Thus, for each of the three levels of Ps, there were 24 combinations of ΔPpl and LI. Under each of these 72 conditions, the cycles were continued until CPR blood flow attained a steady level (approximately 10–12 cycles), and then recordings were made.

**SERIES 2 (EFFECTS OF SINGLE STEP CHANGES OF Ppl AND Pj).** These experiments (12 animals) were designed to separate the effects of lung inflation on blood flow during the compression phases of the CPR cycle from the effects of lung inflation on venous return during the relaxation phases. The static equilibrium pressure was adjusted to 15 mm Hg and clamps were placed on the arterial and venous conduits. Pleural pressure was increased to either 15 or 60 mm Hg, and LI was adjusted to one of the following levels: 0, 10, 20, or 30 mm Hg. With Ppl and LI fixed at the respective levels, the arterial clamp was removed and blood was allowed to move from the chamber to the peripheral reservoir. The volume of blood leaving the chamber was calculated from the product of reservoir compliance and the change in Pres. When the transfer of blood from chamber to reservoir was complete (after approximately five minutes), Ppl and Pj were lowered to 0 mm Hg, and the venous clamp was removed allowing the system to return to the initial condition. Another combination of step changes in Ppl and Pj was then applied. This was repeated for the eight combinations of Ppl and Pj.

**Statistics**
In Series 1, the independent and interactive effects of lung inflation and changes in Ppl on CPR blood flow were compared by two-way analysis of variance for repeated measures. In Series 2, the effects of lung inflation were analyzed by one-way analysis of variance. The data are presented as mean values. Statistical significance was inferred when p < 0.05.

**Results**
**SERIES 1.** Recordings from a single experiment in Series 1 are shown in Figure 2, in which Pj is raised and lowered by the same amount as Ppl. Therefore, LI equals 0 mm Hg, indicating that lung gas volume does not increase during the duty cycles; this is so regardless of the levels to which Ppl and Pj rise.

In Figure 3, Pj is raised during the duty cycle to levels that exceed Ppl by 20 mm Hg. Therefore, in contrast to Figure 2, lung gas volume increases during the compression phases shown in Figure 3 (LI = 20 mm Hg). Regardless of the levels to which Ppl and Pj rise, the levels to which the lungs inflate are the same, determined by the 20 mm Hg increment in Pj relative to Ppl. In both Figures 2 and 3, the stroke volumes increased, as the pleural pressure swings were increased from 10 to 30 or 40 mm Hg. However, when the pleural pressure swings were increased to higher levels, there were no further increases in stroke volume, and in some cases a fall in stroke volume was observed.

For each combination of ΔPpl and LI, mean CPR blood flow (Q) was determined from the product of the stroke volume and frequency (30 cpm for Series 1).

**Figure 2.** Tracings from a single experiment in Series 1. The static equilibrium pressure (Ps) was 22.5 mm Hg. Chamber (pleural) pressure is cycled at 30 cpm with duty cycle of 0.5. Q = flow and SV = stroke volume. Other symbols defined in Figure 1. Swings in pleural pressure (ΔPpl) of 10–60 mm Hg; LI (ΔPj–ΔPpl) = 0 mm Hg, indicating no change in lung gas volume during the duty cycle.
The relation between $Q$ and $\Delta P_{pl}$ when $L_I$ was 0 mm Hg is shown in Figure 4. For each of the three levels of $P_s$, the $Q-\Delta P_{pl}$ relation was biphasic. There was an initial rising phase in which $Q$ increased with $\Delta P_{pl}$. This was followed by a plateau phase in which further increases in $\Delta P_{pl}$ caused either no further increases in $Q$ or in some cases a slight decrease in $Q$. Increasing levels of $P_s$ were associated with higher levels of $Q$ at every level of $\Delta P_{pl}$. In addition, when $P_s$ was elevated, the level of $\Delta P_{pl}$ at which $Q$ attained its maximum level also increased.

The relation between $Q$ and $\Delta P_{pl}$ for the four levels of $L_I$ is shown in Figure 5, in which the $P_s$ was 15 mm Hg. Biphasic relations between $Q$ and $\Delta P_{pl}$ were observed when the lungs were inflated, as well as when $L_I$ was 0. The effects of lung inflation on $Q$ were variable and depended on the magnitude of $\Delta P_{pl}$. When $\Delta P_{pl}$ was low (10 mm Hg), lung inflation resulted in higher $Q$. However, at the higher levels of $\Delta P_{pl}$ lung inflation caused $Q$ to decrease. There was a significant interaction between $\Delta P_{pl}$ and $L_I$ ($p < 0.05$), as well as significant independent effects on $Q$ of both $\Delta P_{pl}$ ($p < 0.01$) and $L_I$ ($p < 0.01$).

The relation between $Q$ and $\Delta P_{pl}$ at the high and low levels of $P_s$ (22.5 and 7.5 mm Hg, respectively) is shown in Figure 6. When $P_s$ was high, the rising phases predominated. Increasing $\Delta P_{pl}$ caused greater $Q$ until $\Delta P_{pl}$ was 40 or 50 mm Hg, after which $Q$ remained constant or decreased. When $P_s$ was low, the plateau phases predominated. In this case, $Q$ rose only until $\Delta P_{pl}$ was 20 or 30 mm Hg. The effects of lung inflation on $Q$ were qualitatively similar regardless of the level of $P_s$. When $\Delta P_{pl}$ was low, lung inflation resulted in higher levels of $Q$; when $\Delta P_{pl}$ was high, lung inflation caused $Q$ to decrease. When $P_s$ was high, there was a significant interaction between $\Delta P_{pl}$ and $L_I$ ($p < 0.005$) and also a significant independent effect of $\Delta P_{pl}$ on $Q$ ($p < 0.005$). However, there was no significant independent effect of $L_I$ on $Q$. When $P_s$ was low, there was no significant interaction between $\Delta P_{pl}$ and $L_I$. However, there were significant independent effects of both $\Delta P_{pl}$ ($p < 0.05$) and $L_I$ ($p < 0.005$) on $Q$.

During the first several cycles of each run of CPR, $P_{es}$ was observed to rise from the level of $P_s$ and attain a new mean level that was maintained during the remainder of the run, indicating that blood had redistributed from the central compartment (heart, lungs, and thoracic vena cava and aorta) to the periphery. For each level of $P_s$, the levels of $P_{es}$ that were maintained after the first several cycles were dependent on the magnitude of $\Delta P_{pl}$ and also of $L_I$. The levels obtained when $P_s$ was 7.5 mm Hg are shown in Table 1.
Qualitatively similar relations occurred when $P_s$ was 15 and 22.5 mm Hg. Higher levels of $\Delta P_p$ caused greater shifts in blood volume from the central to the peripheral compartments. The effects of lung inflation on $P_{es}$, however, were variable and were similar to the effects on $Q$ (Figures 5 and 6). At the lower levels of $\Delta P_p$, lung inflation caused a greater rise in $P_{es}$, indicating a larger shift in blood volume from the central to the peripheral compartment. At the higher levels of $\Delta P_p$, the effects of lung inflation were reversed.

Series 2. These experiments were designed to explore further the mechanism by which lung inflation affects blood flow during the compression phase of the CPR cycle, apart from the possible effects of lung inflation on venous return during the relaxation phase. The volumes of blood that were transferred after the step changes in $\Delta P_p$ and $L_I$ are shown in Table 2. The effects of lung inflation were qualitatively similar to those that occurred during the CPR cycles (Figures 5 and 6). When $\Delta P_p$ was low, lung inflation caused a greater transfer of blood to the periphery. When $\Delta P_p$ was high, however, the opposite occurred. This latter effect suggested that under these conditions lung inflation caused blood to be trapped in the central compartment.

Table 1. Mean Peripheral Reservoir Pressures During Cardiopulmonary Resuscitation

<table>
<thead>
<tr>
<th>$L_I$</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta P_p$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$P_s = 7.5$ mm Hg</td>
<td>8.69±0.39</td>
<td>9.13±0.34</td>
<td>10.5±0.57</td>
<td>11.0±0.29</td>
<td>11.43±0.54</td>
<td>11.28±0.54</td>
</tr>
<tr>
<td>0</td>
<td>8.56±0.24</td>
<td>9.25±0.25</td>
<td>10.5±0.62</td>
<td>10.5±0.60</td>
<td>11.0±0.51</td>
<td>11.06±0.51</td>
</tr>
<tr>
<td>10</td>
<td>8.50±0.31</td>
<td>9.44±0.43</td>
<td>10.0±0.48</td>
<td>10.0±0.56</td>
<td>10.13±0.65</td>
<td>10.36±0.52</td>
</tr>
<tr>
<td>20</td>
<td>8.86±0.35</td>
<td>9.57±0.38</td>
<td>9.79±0.42</td>
<td>9.79±0.52</td>
<td>9.64±0.40</td>
<td>9.79±0.39</td>
</tr>
<tr>
<td>30</td>
<td>14.44±0.22</td>
<td>15.44±0.37</td>
<td>16.75±0.62</td>
<td>18.06±0.78</td>
<td>18.5±0.80</td>
<td>18.69±0.78</td>
</tr>
<tr>
<td>$P_s = 15$ mm Hg</td>
<td>14.56±0.31</td>
<td>15.69±0.44</td>
<td>17.25±0.76</td>
<td>18.31±0.94</td>
<td>18.6±0.88</td>
<td>18.69±0.71</td>
</tr>
<tr>
<td>10</td>
<td>14.63±0.54</td>
<td>15.69±0.81</td>
<td>16.94±1.02</td>
<td>18.13±1.01</td>
<td>18.3±0.90</td>
<td>18.19±0.94</td>
</tr>
<tr>
<td>20</td>
<td>15.29±0.48</td>
<td>16.29±0.67</td>
<td>17.07±0.68</td>
<td>17.64±0.62</td>
<td>17.8±0.57</td>
<td>17.86±0.55</td>
</tr>
<tr>
<td>30</td>
<td>27.0±0.46</td>
<td>22.63±0.57</td>
<td>23.81±0.70</td>
<td>25.4±0.60</td>
<td>26.3±0.73</td>
<td>27.2±0.98</td>
</tr>
<tr>
<td>$P_s = 22.5$ mm Hg</td>
<td>21.4±0.76</td>
<td>22.0±0.98</td>
<td>28.3±1.15</td>
<td>24.9±1.23</td>
<td>25.9±1.43</td>
<td>26.4±1.57</td>
</tr>
<tr>
<td>10</td>
<td>21.4±0.76</td>
<td>22.31±1.96</td>
<td>23.6±1.21</td>
<td>24.3±1.16</td>
<td>25.6±1.27</td>
<td>25.4±1.56</td>
</tr>
<tr>
<td>20</td>
<td>22.3±0.58</td>
<td>22.79±1.03</td>
<td>23.9±1.16</td>
<td>24.9±1.18</td>
<td>25.3±1.22</td>
<td>25.4±1.21</td>
</tr>
</tbody>
</table>

Values of $P_{es}$ during CPR given as mean ± SEM in mm Hg.

Discussion

In these experiments, as well as in previous studies, $Q$ has been shown to vary with the magnitude of the swings in $P_p$$^1$–$^3$ and also with the level of $P_s$. We have also shown that lung inflation may cause either an increase or a decrease in $Q$, depending on the levels of both $P_s$ and $\Delta P_p$.

When $P_p$ rises relative to atmospheric pressure, vascular pressures in the thoracic structures rise relative to systemic vascular pressure, causing blood to...
Table 2. Volume of Blood Transferred After Step Changes in Ppl and LI

<table>
<thead>
<tr>
<th>LI (mm Hg)</th>
<th>ΔPpl</th>
<th>Final reservoir (ml)</th>
<th>Ppl (mm Hg)</th>
<th>PRATM</th>
<th>PLVTM</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>7.75 ±0.43 (12)</td>
<td>16.57 ±1.25 (7)</td>
<td>1.27 ±0.70 (11)</td>
<td>PplVTM final</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>8.08 ±0.45 (12)</td>
<td>17.0 ±1.40 (7)</td>
<td>0.36 ±1.32 (11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>9.00 ±0.70 (11)</td>
<td>17.5 ±1.79 (10)</td>
<td>0.70 ±1.79 (10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>9.00 ±1.00 (7)</td>
<td>18.0 ±1.90 (7)</td>
<td>1.86 ±1.90 (7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p &gt; 0.05</td>
<td>p &gt; 0.05</td>
<td>3.09 ±0.56 (11)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>7.58 ±0.31 (12)</td>
<td>16.29 ±0.89 (7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>8.00 ±0.33 (12)</td>
<td>16.8 ±0.89 (11)</td>
<td>5.45 ±0.89 (11)</td>
<td></td>
<td></td>
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<tr>
<td>20</td>
<td>10.82 ±0.54 (11)</td>
<td>17.0 ±1.38 (10)</td>
<td>10.10 ±1.38 (10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>16.57 ±1.25 (7)</td>
<td>17.5 ±1.79 (10)</td>
<td>14.43 ±2.07 (7)</td>
<td></td>
<td></td>
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<tr>
<td>p &lt; 0.01</td>
<td>p &lt; 0.01</td>
<td>3.09 ±0.56 (11)</td>
<td></td>
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<td></td>
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<tr>
<td>0</td>
<td>116.4±6.13 (12)</td>
<td>174.7 ±15.66 (7)</td>
<td>336.5 ±25.2 (11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>128.8±5.28 (12)</td>
<td>174.7 ±15.66 (7)</td>
<td>284.7 ±26.80 (11)</td>
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<td>20</td>
<td>144.5±6.90 (11)</td>
<td>200.2 ±15.55 (10)</td>
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<tr>
<td>30</td>
<td>145.7±6.96 (7)</td>
<td>174.7 ±15.66 (7)</td>
<td>144.5 ±6.90 (11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p &lt; 0.05</td>
<td>p &lt; 0.01</td>
<td>3.09 ±0.56 (11)</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

All numbers mean ± SEM (n). p values given for effect of lung inflation by one-way analysis of variance. PplVTM = left ventricular transmural pressure. PRATM = right arterial transmural pressure.

flow from the central to the peripheral compartments. It was not surprising, therefore, that increasing levels of ΔPpl resulted in greater levels of Q. However, Q reached a maximum value when a critical level of ΔPpl was attained. Further increases in ΔPpl caused no further increases in flow, and in some cases stroke volume and Q decreased (Figures 5 and 6). This phenomenon of CPR flow limitation has been attributed in the intact dog to collapse of the arterial vessels near the thoracic outlet. In many respects, flow limitation during CPR is similar to the limitation of gas flow which occurs with forced expiration from the lungs. The three Q–ΔPpl relations obtained at different levels of Ps shown in Figure 4 may be considered analogous to a family of isovolume pressure–flow relations of the lung constructed at varying levels of elastic recoil.

Lung inflation during the CPR duty cycle caused Q to increase when the swings in Ppl were in the low to moderate range (Figures 5 and 6). This may have occurred if lung inflation caused a decrease in the resistance to emptying of the compliant vascular structures in the thoracic compartment. This seems unlikely, however, for the following reasons. First, in the Series 2 experiments, there was an increase in transferred volume with lung inflation when ΔPpl was 15 mm Hg. If the sole effect of lung inflation was a decrease in the resistance to emptying, then blood would have transferred more quickly but by the same amount as when LI was 0 mm Hg. Second, the slopes of the Q–ΔPpl relations (Figures 5 and 6) are inversely related to the resistance to emptying of the thoracic structures. These relations show that this resistance actually increases as a result of lung inflation.

A more likely explanation for the increases in Q that occurred with lung inflation is that lung inflation causes an increase in the driving pressure for blood flow during the duty cycle. If lung inflation occurs when the alveolar vessels are filled, there is an increase in alveolar pressure, which surrounds the alveolar blood vessels, relative to the pressure in the alveolar vessels. Under these conditions, the alveolar vascular compartment would be compressed, and this would contribute to the increase in pressure in the thoracic vasculature relative to the pressure in the peripheral vessels. The alveolar vessels would be filled to a greater extent when the Ps is high. It is not surprising, therefore, that the greatest increases in Q that occurred with lung inflation were observed when Ps was 22.5. At each level of Ps, central volume translocates to the peripheral compartments as the swings in Ppl rise. This may be responsible, in part, for the largest increases in Q with lung inflation occurring at the lowest levels of ΔPpl.

When the swings in Ppl were high, lung inflation caused Q to decrease. This effect was pronounced and occurred over the widest range of ΔPpl when Ps was low. In contrast, lung inflation caused Q to decrease by only modest amounts when Ps was high, and only when ΔPpl was 50 and 60 mm Hg. We observed that the lungs did not deflate completely during the relaxation phase of the CPR cycle and that higher levels of LI were associated with higher levels of functional residual capacity. It seemed possible, therefore, that higher levels of pulmonary vascular resistance, which are associated with increases in functional residual capacity, might have impeded the flow of blood from the periphery to the central compartment during the relaxation phase. However, inhibition of venous return in this manner would be expected to cause blood to accumulate in the peripheral circulation. The relations between Pres and ΔPpl show that the opposite occurred.

The slopes of the Q–ΔPpl relations (Figures 5 and 6) suggest that the resistance to flow from the central compartment may increase with lung inflation. While this mechanism might help to explain the fall in Q under the cyclic conditions of Series 1, it cannot explain the decreases in transferred volume observed with lung inflation in Series 2. In this group, an increase in the resistance to emptying could explain a decrease in the rate of flow but could not account for the decrease in total volume that was transferred from the central to the peripheral compartment.

The results of the Series 2 experiments suggest that lung inflation causes a decrease in Q in the central compartment during the duty cycle, preventing its transfer to the periphery. Blood is not trapped in the left heart, since the final transmural pressure of the left ventricle was not affected by lung
inflation. However, the final right atrial transmural pressure was higher when the lungs were inflated, indicating that blood was retained in the right heart. This was especially pronounced when the swings in Ppl were 60 mm Hg, but was apparent also at the lower level of APpl when LI was 30 mm Hg.

Blood could have become trapped in the right ventricle and atrium, if lung inflation caused an increase in the back-pressure to flow from the right heart. When LI was 0 mm Hg, the alveolar vessels were not compressed by inflation of the lungs and were likely, therefore, to have remained patent throughout, or for the majority, of the duty cycle. Therefore, the back-pressure to flow from the right heart would have been the pressure in the Starling resistor of the peripheral circuit under the nonflow-limited conditions, or the critical "collapse" pressure of the aorta or brachiocephalic artery after flow limitation occurred. However, when the lungs were inflated, the alveolar vessels may have become completely emptied and collapsed before the end of each duty cycle. If this occurred, the back-pressure to flow from the right heart would have become alveolar pressure, and this would prevent further flow from the right heart. Higher levels of lung inflation during the duty cycle would, therefore, cause higher back-pressures to flow from the right heart. This, in turn, would cause greater amounts of blood to be trapped in the right heart, and, therefore, smaller amounts of blood to be available for transfer to the periphery.

Whether lung inflation during the duty cycle causes a net increase or decrease in Q depends on the magnitudes of its two opposing effects. Increases in Q with lung inflation would be favored by conditions in which the alveolar vessels are filled, such as when Ps is high or when the swings in Ppl are low. In contrast, decreases in Q with lung inflation would be favored by conditions in which the alveolar vessels are relatively empty, such as when Ps is low or when ΔPpl is high. These opposing effects of lung inflation during the duty cycle are depicted in the model shown in Figure 7. If there is a small increase in Ppl without lung inflation, the alveolar vessels contain an appreciable volume of blood at the end of the duty cycle (A to B). If the lungs inflate during the duty cycle under these conditions, there is an increase in the pressure surrounding the alveolar blood vessels and, therefore, a greater discharge of blood from this region (A to C). If there is a larger increase in Ppl without lung inflation, the alveolar vessels contain less blood at the end of the duty cycle (A to D). The potential for expelling more blood by inflating the lungs under these conditions is less than when the swings in Ppl are small. The combination of high ΔPpl and lung inflation combine to discharge completely the alveolar compartment before the completion of the duty cycle. The collapsed alveolar vessels then prevent further discharge of blood from the right heart, and net decreases in stroke volume and Q occur.

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