Short-term Physiologic Consequences of Regional Pulmonary Vascular Occlusion in Pigs

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ABSTRACT

Background: Acute unilateral pulmonary arterial occlusion causes ventilation-perfusion mismatch of the affected lung area. A diversion of ventilation from nonperfused to perfused lung areas, limiting the increase in dead space, has been described. The hypothesis was that the occlusion of a distal branch of the pulmonary artery would cause local redistribution of ventilation and changes in regional lung densitometry as assessed with quantitative computed tomography.

Methods: In eight healthy, anesthetized pigs (18.5 ± 3.8 kg) ventilated with constant ventilatory settings, respiratory mechanics, arterial blood gases, and quantitative computed tomography scans were recorded at baseline and 30 min after the inflation of the balloon of a pulmonary artery catheter.

Results: The balloon always occluded a branch of the left pulmonary artery perfusing approximately 30% of lung tissue. Physiologic dead space increased (0.37 ± 0.17 vs. 0.43 ± 0.17, P = 0.005), causing an increase in Paco₂ (39.8 [35.2 to 43.0] vs. 41.8 [37.5 to 47.1] mmHg, P = 0.008) and reduction in pH (7.46 [7.42 to 7.50] vs. 7.42 [7.38 to 7.47], P = 0.008). Respiratory system compliance was reduced (24.4 ± 4.2 vs. 22.8 ± 4.8 ml · cm H₂O⁻¹, P = 0.028), and the reduction was more pronounced in the left hemithorax. Quantitative analysis of the nonperfused lung area revealed a significant reduction in lung density (−436 [−490 to −401] vs. −478 [−543 to −474] Hounsfield units, P = 0.016), due to a reduction in lung tissue (90 ± 23 vs. 81 ± 22, P < 0.001) and an increase in air volume (70 ± 22 vs. 82 ± 26 ml, P = 0.022).

Conclusions: Regional pulmonary vascular occlusion is associated with a diversion of ventilation from nonperfused to perfused lung areas. This compensatory mechanism effectively limits ventilation perfusion mismatch. Quantitative computed tomography documented acute changes in lung densitometry after pulmonary vascular occlusion. In particular, the nonperfused lung area showed an increase in air volume and reduction in tissue mass, resulting in a decreased lung density.

This article is accompanied by an editorial on p. 226. Supplemental Digital Content is available for this article. Direct URL citations appear in the printed text and are available in both the HTML and PDF versions of this article. Links to the digital files are provided in the HTML text of this article on the Journal’s Web site (www.anesthesiology.org).

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studies performed in the context of preoperative assessment of thoracic surgery patients have described a shift of pulmonary ventilation from the nonperfused to the perfused lung. This compensatory mechanism partially limits the ventilation–perfusion mismatch after pulmonary embolism and is due both to a regional bronchoconstriction and to a reduction in lung compliance (pneumconstriction) triggered by local hypocapnia.

Studies investigating the respiratory modifications secondary to pulmonary embolism were mainly performed through complete unilateral occlusion of the pulmonary artery, a condition that is rarely encountered in the clinical setting. Furthermore, although lung imaging was performed in a similar experimental context through the use of radioactive krypton gas, no study so far employed chest computed tomography imaging and its quantitative analysis to measure changes in lung density and aeration occurring at a regional level.

The aim of the present experimental study was to describe the short-term respiratory and cardiovascular consequences of regional pulmonary vascular occlusion performed through the balloon of a pulmonary artery catheter in a pig model with controlled mechanical ventilation. We hypothesized that also with an occlusion of a distal pulmonary artery branch, regional redistribution of ventilation would occur, limiting the increase in dead-space fraction. To test our hypothesis, we performed advanced physiologic monitoring and chest computed tomography to quantitatively describe the characteristics of pulmonary parenchyma and assess regional respiratory mechanics before and after regional pulmonary vascular occlusion.

Materials and Methods

The study was conducted at the animal research facility at the Fondazione IRCCS Ca’ Granda Ospedale Maggiore Policlinico of Milan. The study protocol was approved by the Institutional Animal Care Committee, and the experiments were conducted according to Italian national regulations. The pigs were studied, because this research animal has been previously used successfully in similar experimental settings. The pigs were housed under standard environmental conditions (air-conditioned room at 20°C with 50% relative humidity). The experiments were performed during the morning, after 12 h of fasting with free access to water. At the end of the experiments, pigs were euthanized through an intravenous administration of 40 milliequivalents of potassium chloride. A study flow chart is reported in Supplemental Digital Content 1 (http://links.lww.com/ALN/B927).

Animal Preparation

Eight healthy female pigs (18.5 ± 3.8 kg, 2 to 3 months of age) were anesthetized via intramuscular injection of medetomidine (0.025 mg · kg⁻¹) and tiletamine/zolazepam (5 mg · kg⁻¹). Thereafter, an auricular vein was cannulated and propofol injected (a bolus of 2 mg · kg⁻¹ followed by a continuous infusion of 3 to 6 mg · kg⁻¹ · h⁻¹). A surgical tracheostomy was performed after additional local infiltration with 1% lidocaine, and animals were mechanically ventilated in volume-controlled mode (Engstrom Carestation, GE Healthcare, USA) using a fractional inspired oxygen tension (Fio₂) of 0.5 at 0 cm H₂O of positive end-expiratory pressure, tidal volume (Vₜ) of 12 ml · kg⁻¹, inspiratory to expiratory ratio of 1:2, and respiratory rate of 20 breaths · min⁻¹. Medetomidine (2.5 to 10.0 μg · kg⁻¹ · h⁻¹) and pancuronium (0.3 to 0.5 mg · kg⁻¹ · h⁻¹) were continuously infused for analgesia and paralysis, respectively. All animals received an intravenous infusion of lactated Ringer’s (100 ml · h⁻¹ during surgery and thereafter 50 ml · h⁻¹).

A 5-F arterial catheter (Arrow International, USA) was surgically inserted in the right carotid artery. Two 5-F pulmonary artery catheters (Edwards Lifesciences, USA) were surgically placed through the right internal jugular vein in the pulmonary artery under pressure guidance: one served for hemodynamic monitoring, whereas the other was subsequently used to occlude a branch of the pulmonary artery. Correct positioning of endovascular catheters was verified on lung computed tomography scan images (see section on Baseline Measurements and Calculation of Physiologic Variables). Body temperature was monitored and kept constant between 37.5 and 38.5°C using an external warming pad.

Experimental Protocol

After surgical preparation, the animals were turned to the prone position, and an adequate stabilization time (approximately 1 h) was allowed. Thereafter, a recruitment maneuver was performed to eliminate any atelectasis, as previously described. The procedure was made as follows: 1 min of ventilation in pressure-controlled mode with inspiratory pressure of 40 cm H₂O, respiratory rate of 10 breaths · min⁻¹, positive end-expiratory pressure of 5 cm H₂O, Fio₂ 0.5, and inspiratory to expiratory ratio of 1:1. Thereafter, previous ventilatory settings were resumed, and ventilation was kept constant throughout the experiment. Baseline measurements were collected 15 min after the recruitment maneuver.

Baseline Measurement and Calculation of Physiologic Variables

Recorded hemodynamic variables included heart rate, systemic and pulmonary arterial pressure, central venous pressure, and cardiac output via thermodilution technique (Vigilance, Baxter Edwards Critical Care, Edwards E6 Lifesciences, USA). Recorded respiratory variables included static respiratory system compliance, airway resistance, and carbon dioxide output (VCO₂) through volumetric capnography (Respirionics NM5 Monitor, Philips, The Netherlands). An arterial blood sample was collected anaerobically for blood-gas analysis (ABL800 Flex, Radiometer...
Chest computed tomography scans (Siemens Somatom Definition 64 slices, Syngo CT2008G as acquisition software) were acquired at baseline, during a respiratory hold performed at end inspiration and end expiration. The acquired images were processed off-line for quantitative analysis. Briefly, left and right lung boundaries were manually drawn on each slice and analyzed using a dedicated software program (Maluna 3.17, Germany). After processing each slice of a series, left and right lung volume, tissue mass, and frequency distribution of lung computed tomography numbers expressed in Hounsfield units were computed.\(^{17-19}\)

Furthermore, regional (left lung and right lung) \(V_T^{\text{reg}}\) was calculated as follows in Equation 1:

\[
\text{Regional } V_T^{\text{reg}} = (\text{Regional end-insp } V_{\text{air}}) - (\text{Regional end-exp } V_{\text{air}})
\]

where “Regional end-insp \(V_{\text{air}}\)” indicates the volume of air of the specific lung region during an end-inspiratory hold, measured with quantitative computed tomography, and “Regional end-exp \(V_{\text{air}}\)” indicates the volume of air of the specific lung region during an end-expiratory hold, measured with quantitative computed tomography.

Regional respiratory system compliance was calculated as follows in Equation 2:

\[
\text{Regional respiratory system compliance } = \frac{\text{regional } V_T}{\Delta P}
\]

where \(\text{regional } V_T\) indicates the regional tidal volume as measured in Equation 1, and \(\Delta P\) indicates the difference between the plateau pressure and the end-expiratory pressure as measured during an expiratory hold.

**Filling of the Balloon of One Pulmonary Artery Catheter: Controlled Regional Pulmonary Vascular Occlusion**

After the acquisition of baseline parameters, including chest computed tomography scans, the balloon of one pulmonary artery catheter was filled with 0.5 ml of 0.9% NaCl and kept filled. All measurements performed at baseline were repeated 30 min after having inflated the balloon of one pulmonary artery catheter. This time point was labeled “wedge time.”

**Subregional Quantitative Computed Tomography Analysis**

To quantify the lung portion where blood flow was interrupted and to study the local effects of a regional occlusion of a pulmonary artery branch, we performed a subregional quantitative computed tomography analysis. The fluid-filled balloon of the pulmonary artery catheter was identified in transverse chest computed tomography scans performed at wedge time and was used as an anatomical landmark to divide the lung in which the balloon was wedged in two distinct portions:

1. **Nonperfused lung portion:** the portion of the lung distal to this anatomical landmark, therefore including the territory of the occluded branch of the pulmonary artery.
2. **Perfused lung portion:** the portion of the same lung, proximal to the fluid filled balloon.

The same anatomical regions (“nonperfused” and “perfused”) were identified also on baseline computed tomography scans. Given the lack of anatomical landmarks in baseline computed tomography scans, the balloon was not fluid-filled, the nonperfused and perfused lung regions were identified based on a computed tomography slice count. In other words, it was assumed that the number of slices constituting the abovementioned lung regions at wedge time would be equal to the number of slices constituting the same regions at baseline. The size of the nonperfused lung region was expressed as a percentage of total lung weight.

**Statistical Analysis**

The normality of data distribution was tested using the Shapiro–Wilk test. Normally distributed data are expressed as means \(\pm SD\), whereas nonnormally distributed data are expressed as median and interquartile range. The presence of outliers was assessed during evaluation of distribution of data; however, no action was foreseen. No blinding methods were used in the study. Variables recorded before (baseline) and after vascular occlusion (wedge time) were compared via paired \(t\) test or signed rank sum test, as appropriate. Mean difference and its 95% CI were calculated for normally distributed data. For nonnormally distributed variables, median difference and its 95% CI were estimated by Hodges–Lehmann’s median analysis.\(^{20}\) Frequency distribution of Hounsfield units before and after vascular occlusion were compared by two-way repeated-measure ANOVA on ranks. Because using ranks in this approach may result in inferences with increased type-I error, if a statistically significant Time \(\times\) HU interaction was observed, a Tukey’s test was used to confirm differential effects in pairwise adjusted multiple comparisons. No statistical power calculation was conducted before the study. The sample size was based on previous experience with this model. All tests were two-tailed, and statistical significance was defined as \(P < 0.050\). Analysis was performed with SigmaPlot v.12.0 (Systat Software Inc., USA) and SAS 9.2 (SAS Institute Inc., USA).

**Results**

In all cases, the inflated balloon occluded a branch of the left inferior pulmonary artery. The region distal to the filled balloon (nonperfused lung portion) accounted for 29 ± 14% of total lung weight.

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are summarized in Table 1. At wedge time, a significant increase in physiologic dead-space fraction was observed (0.37 ± 0.17 vs. 0.43 ± 0.17, \( P = 0.005 \)). Arterial \( \text{PCO}_2 \) increased (39.8 [35.2–43.0] vs. 41.8 [37.5–47.1] mmHg, \( P = 0.008 \)), and arterial pH decreased (7.46 [7.42–7.50] vs. 7.42 [7.38–7.47], \( P = 0.008 \)). No variations in \( \dot{V}_{\text{CO}_2} \) were observed (123 ± 29 ml vs. 122 ± 25 ml, \( P = 0.879 \)).

Mean pulmonary arterial pressure and pulmonary vascular resistance did not change significantly. We observed a significant reduction in mean arterial pressure and systemic vascular resistance, whereas there were no differences in heart rate and cardiac output between groups.

Computed tomographic images were successfully acquired in seven animals, whereas the data of one animal could not be acquired correctly due to a technical problem. On quantitative computed tomography analysis, a small but statistically significant reduction of the weight of the left lung, i.e., the lung in which a branch of the pulmonary artery was occluded, was recorded (Table 2). In contrast, the weight of the right lung did not change. The filling of the balloon of one pulmonary artery catheter resulted in a significant reduction in static respiratory system compliance. Regional quantitative computed tomography–based analysis showed a more pronounced compliance reduction of the left side of the respiratory system (Table 2).

A marked regional change in lung densitometry was observed after the vascular occlusion. Figure 1 shows a representative computed tomography scan with color-mapped qualitative analysis. The subregional quantitative analysis showed, at wedge time, a significant shift of the frequency distribution of computed tomography numbers of the perfused area toward higher values, indicating an increase in lung density (Fig. 2A). On the contrary, the nonperfused

### Table 1. Respiratory and Hemodynamic Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>Wedge Time</th>
<th>( P ) Value</th>
<th>Difference</th>
<th>Lower</th>
<th>Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}_{\text{CO}_2}, \text{ml} \cdot \text{min}^{-1} )</td>
<td>123 ± 29</td>
<td>122 ± 25</td>
<td>0.879</td>
<td>−1</td>
<td>−10</td>
<td>9</td>
</tr>
<tr>
<td>Dead-space fraction</td>
<td>0.37 ± 0.17</td>
<td>0.43 ± 0.17</td>
<td>0.005</td>
<td>0.03</td>
<td>0.07</td>
<td>0.11</td>
</tr>
<tr>
<td>Arterial pH</td>
<td>7.46 [7.42–7.50]</td>
<td>7.42 [7.38–7.47]</td>
<td>0.008</td>
<td>−0.03</td>
<td>−0.09</td>
<td>−0.02</td>
</tr>
<tr>
<td>( \text{PaCO}_2, \text{mmHg} )</td>
<td>39.8 [35.2–43.0]</td>
<td>41.8 [37.5–47.1]</td>
<td>0.008</td>
<td>3.6</td>
<td>1.9</td>
<td>11.9</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>14 ± 3</td>
<td>16 ± 4</td>
<td>0.351</td>
<td>1</td>
<td>−2</td>
<td>4</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>114 [109–123]</td>
<td>107 [102–121]</td>
<td>0.016</td>
<td>−5</td>
<td>−12</td>
<td>−2</td>
</tr>
<tr>
<td>CVP, mmHg</td>
<td>1 ± 2</td>
<td>1 ± 2</td>
<td>0.857</td>
<td>0</td>
<td>−2</td>
<td>2</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>125 ± 34</td>
<td>130 ± 26</td>
<td>0.348</td>
<td>6</td>
<td>−7</td>
<td>18</td>
</tr>
<tr>
<td>Cardiac output, l \cdot \text{min}^{-1}</td>
<td>3.4 ± 0.6</td>
<td>3.9 ± 0.4</td>
<td>0.101</td>
<td>0.3</td>
<td>−0.1</td>
<td>0.8</td>
</tr>
<tr>
<td>PVR, dyn \cdot s \cdot \text{cm}^{-5}</td>
<td>261 ± 54</td>
<td>240 ± 81</td>
<td>0.618</td>
<td>−12</td>
<td>−85</td>
<td>62</td>
</tr>
<tr>
<td>SVR, dyn \cdot s \cdot \text{cm}^{-5}</td>
<td>2.652 ± 483</td>
<td>2.159 ± 467</td>
<td>0.035</td>
<td>−374</td>
<td>−711</td>
<td>−38</td>
</tr>
</tbody>
</table>

\( \dot{V}_{\text{CO}_2} \), total carbon dioxide production as measured with volumetric capnometry.

### Table 2. Lung Weights and Respiratory System Mechanics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>Wedge time</th>
<th>( P ) Value</th>
<th>Difference</th>
<th>Lower</th>
<th>Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total lung weight, g</td>
<td>331 ± 65</td>
<td>324 ± 75</td>
<td>0.375</td>
<td>−7</td>
<td>−27</td>
<td>12</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
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<tr>
<td>Lung weight left, g</td>
<td>141 ± 29</td>
<td>129 ± 30</td>
<td>0.032</td>
<td>−12</td>
<td>−23</td>
<td>−2</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
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<tr>
<td>Lung weight right, g</td>
<td>190 ± 37</td>
<td>195 ± 46</td>
<td>0.289</td>
<td>5</td>
<td>−6</td>
<td>16</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
<td></td>
<td></td>
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<tr>
<td>( R_{aw} ), cm \text{H}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}^{-1} )</td>
<td>27 ± 8</td>
<td>29 ± 6</td>
<td>0.250</td>
<td>2</td>
<td>−2</td>
<td>6</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
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<tr>
<td>( C_v ), total, cm \text{H}_2\text{O} \cdot \text{l}^{-1} )</td>
<td>24.4 ± 4.2</td>
<td>22.8 ± 4.8</td>
<td>0.028</td>
<td>−1.6</td>
<td>−2.9</td>
<td>−0.2</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
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<tr>
<td>( C_v ), left, cm \text{H}_2\text{O} \cdot \text{l}^{-1} )</td>
<td>9.1 ± 1.6</td>
<td>7.9 ± 2.1</td>
<td>0.060</td>
<td>−1.3</td>
<td>−2.6</td>
<td>0.1</td>
</tr>
<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
<td></td>
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</tr>
<tr>
<td>( C_v ), right, cm \text{H}_2\text{O} \cdot \text{l}^{-1} )</td>
<td>11.8 ± 1.9</td>
<td>10.6 ± 2.8</td>
<td>0.290</td>
<td>−1.0</td>
<td>−3.2</td>
<td>1.2</td>
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<tr>
<td>(n = 7)</td>
<td>(n = 7)</td>
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</table>

The lung weight indicates volume of tissue mass as measured at quantitative computed tomography, assuming a specific lung weight of 1 g · ml⁻¹.

\( C_v \), left, respiratory system compliance of the left respiratory system calculated as described in Equation 2; \( C_v \), right, respiratory system compliance of the right respiratory system calculated as described in Equation 2; \( C_v \), total, respiratory system compliance of the whole respiratory system measured as set tidal volume/driving pressure; \( R_{aw} \), inspiratory airway resistance.
area of the left lung was shifted toward lower computed tomography numbers, indicating a reduction in lung density (fig. 2B). As a result, the average computed tomography number of the nonperfused area was significantly reduced (−436 [−490 to −401] vs. −478 [−543 to −474] HU, \( P = 0.016 \)) at Wedge time as compared to baseline (fig. 3A). The reduction in lung density of the nonperfused area observed at wedge time was associated with both a reduction in lung tissue mass (90 ± 23 g vs. 81 ± 22 g, \( P < 0.001 \); fig. 3B) and an increase in lung air volume (70 ± 22 ml vs. 82 ± 26 ml, \( P = 0.022 \); fig. 3C).

**Discussion**

We have described, through selected physiologic measurements and the use of quantitative computed tomography scan...
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analysis, the short-term physiologic effects of regional pulmonary vascular occlusion in a large, healthy, animal model with controlled mechanical ventilation. The experimental vascular occlusion of about 30% of total lung parenchyma resulted in an actually measured increase in dead-space fraction of only 5%. This marked discrepancy between the size of the anatomical region of nonperfused lung and the observed increase in physiologic dead space was likely due to a quick diversion of ventilation from nonperfused to perfused lung areas. Indeed, when a pulmonary vessel is occluded, the alveolar supply of carbon dioxide through its bloodstream is instantly zeroed. Although some carbon dioxide might still be delivered to the nonperfused lung regions through rein- spiration of dead-space gas and through an intact bronchial circulation, the result of pulmonary vascular occlusion is alveolar and bronchial hypocapnia, leading to regional bronchoconstriction and pneumoconstriction. These local hypocapnia-induced modifications reduce the ventilation of the nonperfused lung portion, effectively limiting the ventilation–perfusion mismatch and thus the increase in functional dead-space fraction.

Total minute ventilation was constant before and after vascular occlusion in our experimental setting. Therefore, the increase in dead space was mirrored by a decrease in alveolar ventilation, causing the observed increase in Paco₂ and decrease in arterial pH (table 1). Of note, Vco₂ did not change, underlining the fact that a new steady state was reached.

In all animals, two pulmonary artery catheters were positioned. This allowed us to perform the occlusion and to measure its effects on pulmonary arterial pressure, cardiac output, and pulmonary vascular resistance. Not unexpectedly, pulmonary arterial pressure and pulmonary vascular resistance did not increase after the inflation of the pulmonary catheter, underlining, in our large animal model, the high reserve capacity of pulmonary arterial vessels. Nevertheless, other researchers in a similar setting have found a significant increase in pulmonary arterial pressure, likely due to a greater region of vascular occlusion. Of note, in pathologic conditions the filling of the balloon of the pulmonary artery catheter might, in rare cases, increase pulmonary vascular resistance to a level leading to right heart failure and hemodynamic collapse.

We observed a significant reduction in systemic blood pressure and systemic vascular resistance (table 1). These acute hemodynamic alterations are likely due to the increase in Paco₂ with consequent decrease in arterial pH. Qualitative color-mapped computed tomography images changed markedly after vascular occlusion, mainly due to a decrease in the nonperfused portion (fig. 1). These qualitative results were confirmed by quantitative computed tomography analysis, which revealed a marked variation in subregional lung densitometry (fig. 2). Indeed, a shift toward an increased density was observed in the perfused portion (fig. 2A), possibly explained by an increased vascular filling/perfusion. On the contrary, a shift toward more negative computed tomography numbers, lower density, was observed in the nonperfused portion (fig. 2B). As a result, the measured mean density of the nonperfused portion was significantly reduced (fig. 3A). Because a reduction in density could be due both to a reduction in mass and an increase in air volume, we analyzed these two factors separately: both a decrease in lung tissue mass (fig. 3B) and an increase in air volume (fig. 3C) were observed. On the one hand, the decrease in lung tissue mass was likely caused by a decrease in regional perfusion. On the other, it is conceivable that the hypocapnia-induced increase in expiratory resistance led to some air trapping (dynamic hyperinflation), increasing the absolute air content of the nonperfused lung area. A typical feature of pigs, i.e., the lack of collateral ventilation, might have contributed to the observed air trapping.
We observed a slight but significant reduction in total respiratory system compliance. The regional analysis performed through quantitative computed tomography data on left and right lungs showed a more pronounced decrease in compliance in the left hemithorax, i.e., on the side of the vascular occlusion (table 2). During acute respiratory failure, a reduction in lung compliance is associated with an increase in lung density at quantitative computed tomography due to edema accumulation.32 However, because the overall weight of the lungs did not change with partial vascular occlusion (table 2), we can exclude edema accumulation as a mechanism contributing to the observed reduction in lung compliance.

In our experiments, instead, the reduction in lung compliance was associated with a decrease in lung density. The physiologic mechanisms possibly involved in the observed finding are therefore two: a local reduction in lung compliance (pneumoconstriction)33 and regional overdistension due to air trapping.

Limitations

Our study has some limitations that need to be acknowledged. First, the 30-min pulmonary vascular occlusion, obtained through the filling of a balloon-tipped catheter, was experimental and was not intended to mimic a clinical situation. Second, we had some variability regarding the size of the nonperfused lung parenchyma (29 ± 14%) and therefore in the weight and volume of air in the regions of study (fig. 3). This variability can be explained by some difference in the size of the experimental animals and by the fact that we did not control the site of vascular occlusion. Third, the translatability of our findings to the clinical scenario is limited both by the sample size and by the lack of an inflammatory response typical of clinical embolism. Indeed, a clinical setting that resembles more closely our experiments is the measurement of the wedge pressure through the use of a pulmonary artery catheter, which, however, requires significantly less time. The acquisition of all physiologic and imaging variables, however, did not allow to perform an earlier time point, and it was our aim to acquire variables during a steady-state condition. To visualize the immediate ventilatory changes after vascular occlusion an imaging technique allowing for continuous breath-by-breath analysis, such as chest electrical impedance tomography, would be required.34 Furthermore, we limited our study to 30 min, without exploring later time points, because we were interested in the acute physiologic changes. Finally, we did not study the reversibility of these changes upon balloon deflation.

Conclusions

In conclusion, the experimental vascular occlusion caused (1) an increase in dead-space fraction significantly lower than the estimated size of nonperfused lung and (2) acute changes in lung densitometry. In particular, the area of vascular occlusion showed a decrease in lung density, due both to an absolute reduction in lung tissue and to an increase in air volume. These acute densitometric changes were likely caused by regional hypocapnia-induced broncho- and pneumoconstriction, acting to minimize ventilation perfusion mismatch.

Acknowledgments

The authors are indebted to Lawrence R. Goodman, M.D., F.A.C.R., Cardiothoracic Imaging Section, Department of Radiology and Department of Pulmonary Medicine and Critical Care, Medical College of Wisconsin, Milwaukee, Wisconsin, for his useful suggestions; to Eleonora Carlesso, M.Sc., Department of Pathophysiology and Transplantation of the University of Milan, Milan, Italy, for her valuable statistical support and support with imaging processing; and to Stefano Gatti, M.D., and Daniele Dondossola, M.D., from the Center for Preclinical Research, Fondazione IRCCS Ca’ Granda—Ospedale Maggiore Policlinico of Milan, for their valuable support regarding surgical preparation.

Research Support

Supported by institutional funds of the Fondazione IRCCS Ca’ Granda Ospedale Maggiore Policlinico, Milan, Italy.

Competing Interests

The authors declare no competing interests.

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References


