Effects of inertial load and countermeasures on the distribution of pulmonary blood flow

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Chornuk, Myron A., Susan L. Bernard, John W. Burns, Robb W. Glenny, Don D. Sheriff, Scott E. Sinclair, Nayak L. Polissar, and Michael P. Hlastala. Effects of inertial load and countermeasures on the distribution of pulmonary blood flow. J Appl Physiol 89: 445–457, 2000.—We assessed the influence of cranial-to-caudal inertial force (+Gz) and the countermeasures of anti-G suit and positive pressure breathing during G (PBG) specifically during +Gz, on regional pulmonary blood flow distribution. Unanesthetized swine were exposed randomly to 0 G (resting), +3 Gz, +6 Gz, and +9 Gz, with and without anti-G suit and PBG with the use of the Air Force Research Laboratory centrifuge at Brooks Air Force Base (the gravitational force of the Earth, that is, the dorsal-to-ventral inertial force, was present for all runs). Fluorescent microspheres were injected into the pulmonary vasculature as a marker of regional pulmonary blood flow. Lungs were excised, dried, and diced into ~2-cm3 pieces, and the fluorescence of each piece was measured. As +Gz was increased from 0 to +3 Gz, blood flow shifted from cranial and hilar regions toward caudal and peripheral regions of the lung. This redistribution shifted back toward cranial and hilar regions as anti-G suit inflation pressure increased at +6 and +9 Gz. Perfusion heterogeneity increased with +Gz stress and decreased at the higher anti-Gz suit pressures. The distribution of pulmonary blood flow was not affected by PBG. ANOVA indicated anatomic structure as the major determinant of pulmonary blood flow.

pulmonary perfusion; spatial heterogeneity; extended coverage anti-G suit; acceleration

PULMONARY GAS EXCHANGE and ultimately tissue oxygenation are largely affected by the distribution of blood flow in the lung. Since the early 1960s, it has been thought that a vertical hydrostatic gradient due to gravity is the primary determinant of pulmonary blood flow. This was predicted by Permutt et al. (35) and observed experimentally by West et al. (42). The gravitational model divides blood flow into three zones according to the interaction of arterial, alveolar, and venous pressures. As a result, flow should increase from nondependent to dependent lung regions. Evidence from low spatial resolution studies supports the zone model (2, 6, 16, 31, 32). However, other studies have concluded that nongravitational factors may play the major role in determining pulmonary blood flow (24–26, 29, 36). Recently, Glenny et al. (20) used a high spatial resolution technique to show considerable heterogeneity within isogravitational planes. Several additional studies suggest that gravity may play only a small role in determining pulmonary perfusion (3, 4, 27, 34, 41).

A method of testing the influence of gravity on the distribution of pulmonary blood flow, and thus the zone model, is to increase the hydrostatic pressure gradient in the lung by increasing the magnitude of gravity using a centrifuge. Several low spatial resolution studies have examined regional pulmonary blood flow during increased levels of acceleration.1 A reduction of blood flow to nondependent regions of the lung during cranial-to-caudal inertial force (+Gz) stress was observed in human subjects by Bryan et al. (7), Glaister (15), and von Nieding et al. (40) and in miniature swine by Whinnery et al. (43, 44). These studies measured perfusion to relatively large regions of the lung by averaging flow within isoinertial planes. More recently, Hlastala et al. (28) exposed unanesthetized miniature swine to increasing levels of dorsal-to-ventral direction force (−Gz). Using high spatial resolution methods, Hlastala showed that, as −Gz stress was increased, perfusion heterogeneity increased and blood flow decreased in both ventral and dorsal regions of the lung and increased in hilar regions. A high-resolution study on the role of increased inertial force in the +Gz

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1Blood and tissue displacements are due to the inertial force, which is equal in magnitude but opposite in direction to acceleration. Standard notation of inertial force vectors used in acceleration research is as follows: +Gz (ventral-to-dorsal), −Gz (dorsal-to-ventral), +Gx (right-to-left), −Gx (left-to-right), +Gy (cranial-to-caudal), and −Gy (caudal-to-cranial), where G denotes Earth’s normal gravitational acceleration of 9.8 m/s2.
vector on pulmonary blood flow distribution has not been performed.

High-performance aircraft pilots are routinely subjected to high inertial stress during aerial combat maneuvering. In this environment, pulmonary ventilation-perfusion mismatching and decreased oxygen saturation may be accentuated by the increased G stress; therefore, a more complete understanding of the effects of increased $+G_x$ on the redistribution of blood flow in the lung is needed. To maintain brain perfusion during $+G_z$, several countermeasures have been developed, including anti-G suits, positive pressure breathing during G (PBG), specifically during $+G_z$, and anti-G straining maneuvers (AGSM; formerly known as M-1 and L-1). There is little information on how these countermeasures influence regional blood distribution in the lung during increased $+G_z$ stress.

The purpose of this study, therefore, was to evaluate how pulmonary blood flow redistributes during increased inertial loads in $+G_z$ in unanesthetized miniature swine using the high-resolution fluorescent microsphere method. In addition, the effects of countermeasures (anti-G suit and PBG) on pulmonary blood flow redistribution were assessed.

METHODS

Animal Preparation

Six female miniature swine (43.3 ± 6.8 kg) were chemically restrained with ketamine, intubated, and placed on positive-pressure ventilation (tidal volume of 10–15 ml/kg, frequency adjusted to maintain arterial carbon dioxide tension ($P_{aCO_2}$) of 35–40 Torr) with ~1% isofluorane (to effect). A surgical plane of anesthesia was maintained at all times during catheter placement. A Swan-Ganz catheter was introduced into each external jugular vein and advanced into the pulmonary artery. The pulmonary arterial catheters were used to determine cardiac output (thermal dilution) and core body temperature, withdraw reference pulmonary arterial blood samples, and measure pulmonary arterial pressure and central venous pressure. The proximal port of one of the Swan-Ganz catheters was used for microsphere injection. A catheter was placed into the right carotid artery to measure arterial blood pressure and obtain arterial blood gases. An esophageal balloon was introduced to monitor respiration and intrathoracic pressure. Surgical sites were closed and infiltrated with 2% bupivacaine. The animals were given 5,000 IU of heparin intravenously, the endotracheal tube was removed, and electrocardiogram leads were attached. The animals were fitted with a custom-made, extended-coverage anti-G suit (ECGS) and pressure breathing mask, and placed in a prone position on a form-fitted fiberglass couch. Lateral thoracic X-rays were obtained to determine lung orientation relative to the couch. The animal and couch were transported to the centrifuge facility, and the couch was attached to the animal arm of the Air Force Research Laboratory centrifuge. During centrifugation, the animal couch was maintained in a horizontal position (relative to the floor) at a constant $-1 G_z$ (Fig. 1), which contrasts with the human gondola that rotates to keep the $+G_z$ force in line with the cranial-to-caudal axis.

Pulmonary Blood Flow Distribution

Fluorescent 15-μm-diameter microspheres (FluoSpheres, Molecular Probes, Eugene, OR) of eight different colors (blue, blue-green, green, yellow-green, orange, red, crimson, and scarlet) were used to measure regional pulmonary blood flow. The remote control infusion system used for injecting the microspheres consisted of an infusion pump (model 600-000, Harvard Apparatus) and a catheter (injection link) containing a suspension of $2 \times 10^6$ microspheres in saline, dextran, and Tween 20 (0.2% solids in 2 ml). The solution containing microspheres was modified to have a specific gravity equal to the microspheres (1.04) to prevent settling during centrifugation. To conserve this specific gravity, two small air bubbles isolated the solution of microspheres from 1 ml of saline.

![Fig. 1](http://jap.physiology.org/)

Fig. 1. Schematic diagram of G forces on miniature swine on centrifuge. When the centrifuge is at rest, there is no inertial force in the cranial-to-caudal direction ($-G_x$); however, normal Earth gravitational acceleration in the dorsal-to-ventral direction ($+G_z$) is always present.
dead space. The microspheres were injected through the proximal port of the Swan-Ganz catheter by remote control.

**Experimental Protocol**

Once fully awake, the animals were exposed to 0 Gz (resting), +3 Gz, +6 Gz, and +9 Gz, with and without anti-G suit and PBG as shown in Table 1. Earth’s normal gravity (−1 Gz) is always present, so inertial loads in the cranial-to-caudal direction are not pure +Gz exposures (Fig. 1). However, this is the standard convention used for quadrupeds in acceleration research. Because of the presence of the Earth’s gravity vector (−1 Gz) in a direction perpendicular to the experimentally altered +Gz vector, a resultant inertial vector will be the vector sum of the two. Although this resultant vector will change in magnitude and angle with respect to the cranial-to-caudal direction, it can be dealt with using its vector components. Changes in pulmonary blood flow are analyzed along the z (cranial-to-caudal)-axis as well as the x (ventral-to-dorsal)-axis. The major analysis of this study deals with the z component of the inertial vector. During all +Gz exposures (at 3, 6, and 9), the anti-G suit was inflated to 1.5, 6.0, and 10.5 psi, respectively, as is standard in the United States Air Force (1.5 psi/G beginning at +2 Gz). Pressures used for PBG (when applied) were 60 Torr at 0 Gz, 24 Torr at +6 Gz, and 60 Torr at +9 Gz (12 Torr/G beginning at +4 Gz). PBG is not operationally used at loads under +4 Gz, but we included it at 0 Gz to isolate the influence of PBG on blood flow distribution. In addition, the anti-G suit was inflated to 1.5 psi during 0 Gz PBG exposures to minimize blood pooling in dependent regions. All runs were randomized, except those at +9 Gz, which were always performed at the end of the experiment. Some runs were repeated within an animal. For each +Gz exposure, once the centrifuge reached a stable Gz exposure sequence, the Next color of microspheres was measured with a blood-gas analyzer (Nova Biomedical ABC inertial force). PBG, positive pressure breathing during +Gz exposures, respectively (+Gz is cranial-to-caudal inertial force). PBG, positive pressure breathing during +Gz exposures at 60, 24, and 60 Torr at 0 Gz, +6 Gz, and +9 Gz exposures, respectively.

**Lung Tissue Processing**

After the last G maneuver, the animals, in less than 30 min, were deeply anesthetized, intubated, ventilated, and given 10,000 IU of heparin and 350 mg of papaverine intravenously. The animals were exsanguinated via arterial catheters, and intravascular volume was replaced with normal saline containing heparin. Once sufficiently hemodiluted, the animals were killed with pentobarbital sodium. A thoracotomy was performed, large-bore catheters were placed into the left atrium and pulmonary artery, the thoracic aorta was occluded, and the lungs were perfused with 2% dextran in saline. This procedure cleared the lungs of blood but did not dislodge the microspheres. The lungs were removed from the chest, reinflated, and allowed to dry at total lung capacity on warm, continuous positive airway pressures of 25–30 cmH2O for 5 days. Samples of heart and kidney were removed to determine whether shunting of the microspheres through the lungs occurred.

When dry, the lungs were coated with Kwik Foam (DAP, Dayton, OH), suspended vertically in a plastic-lined square box, and embedded in rapidly setting urethane foam (International Sales, Seattle, WA) to provide a rigid, orthogonal reference system. The lateral chest X-ray was used to align the lungs in the box so that slicing produced transverse planes. Because of the presence of Earth’s normal gravity, transverse planes are not true isoinertial planes. True isoinertial planes would be perpendicular to the resultant of the inertial vector and Earth’s gravitational vector. The use of a miter box, the foam block was cut into cubes (n = 1932 ± 222, mean ± SD), −2 cm³ in volume. Fifteen percent of all lung pieces were discarded (pieces with airways occupying >25% of the volume, as determined by visual inspection). Weight, spatial coordinates, and lobe designation for each piece were recorded. Lung pieces were soaked in 1.5 ml of 2-ethoxyethyl acetate to extract the fluorescent dyes from the microspheres, and the fluorescent dye concentrations were measured with a luminescence spectrophotometer (model LS-50B, Perkin-Elmer, Norwalk, CT). See Ref. 18 for additional information concerning this technique. A matrix-inversion method was used to correct for fluorescent spillover from adjacent colors (37).

**Data Processing**

Blood flow was corrected for weight by dividing the flow to each lung piece by the weight of that piece. Relative blood flow was then determined by dividing the weight-normalized

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**Table 1. +Gz exposure sequence**

<table>
<thead>
<tr>
<th>Run</th>
<th>Pig 1</th>
<th>Pig 2</th>
<th>Pig 3</th>
<th>Pig 4</th>
<th>Pig 5</th>
<th>Pig 6</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>0 Gz</td>
<td>+3 Gz</td>
<td>+3 Gz</td>
<td>0 Gz</td>
<td>+6 Gz</td>
<td>0 Gz</td>
</tr>
<tr>
<td>2</td>
<td>+6 Gz PBG</td>
<td>0 Gz</td>
<td>+6 Gz</td>
<td>+3 Gz</td>
<td>+6 Gz PBG</td>
<td>0 Gz</td>
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<tr>
<td>3</td>
<td>+3 Gz</td>
<td>0 Gz</td>
<td>0 Gz</td>
<td>+6 Gz PBG</td>
<td>0 Gz</td>
<td>+6 Gz</td>
</tr>
<tr>
<td>4</td>
<td>+6 Gz</td>
<td>+6 Gz PBG</td>
<td>0 Gz PBG</td>
<td>0 Gz PBG</td>
<td>0 Gz</td>
<td>+3 Gz</td>
</tr>
<tr>
<td>5</td>
<td>0 Gz PBG</td>
<td>+6 Gz</td>
<td>+6 Gz PBG</td>
<td>+6 Gz</td>
<td>+6 Gz PBG</td>
<td>+3 Gz</td>
</tr>
<tr>
<td>6</td>
<td>0 Gz</td>
<td>+3 Gz</td>
<td>+3 Gz</td>
<td>0 Gz</td>
<td>+6 Gz</td>
<td>+6 Gz</td>
</tr>
<tr>
<td>7</td>
<td>+9 Gz</td>
<td>+9 Gz PBG</td>
<td>+9 Gz PBG</td>
<td>+9 Gz</td>
<td>+9 Gz PBG</td>
<td>+9 Gz</td>
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<tr>
<td>8</td>
<td>+9 Gz PBG</td>
<td>+9 Gz</td>
<td>+9 Gz PBG</td>
<td>+9 Gz</td>
<td>+9 Gz PBG</td>
<td>+9 Gz</td>
</tr>
</tbody>
</table>

Anti-G suit was inflated to 1.5, 6.0, and 10.5 psi during all +3 Gz, +6 Gz, and +9 Gz exposures, respectively (+Gz is cranial-to-caudal inertial force). PBG, positive pressure breathing during +Gz, used pressures of 60, 24, and 60 Torr at 0 Gz, +6 Gz, and +9 Gz exposures, respectively.
blood flow of each piece by the mean weight-normalized flow of all pieces. Weight-normalized relative flows are used for all analyses and are hereafter referred to as blood flow or perfusion. The radial distance from the ipsilateral hilum to each piece \((D_p)\) was calculated as the Euclidean distance (17).

**Statistical Analysis**

Flow heterogeneity. Pulmonary blood flow heterogeneity was characterized by the coefficient of variation (CV = 100%·SD·mean \(^{-1}\)). The relationship of heterogeneity to \(G\) was analyzed by plotting \(+G\) as the independent variable and CV as the dependent variable.

Linear trends of pulmonary blood flow. A simple linear regression model of flow vs. each spatial distance (ventral-to-dorsal direction \((x)\), right-to-left direction \((y)\), cranial-to-caudal direction \((z)\), and \(D_p\)) was performed to identify linear gradients (expressed as regression slopes). The relationship of these slopes to \(+G\) was analyzed by plotting \(+G\) as the independent variable and slope as the dependent variable.

Center of flow. The center of flow was determined as the weighted mean of \(x\), \(y\), and \(z\) (separately), in which the weights were the blood flow for each piece.

Sources of flow variation. The variation in blood flow across \(+G\) levels and countermeasures was classified into four parts: 1) a component due to position of the piece within the lung (anatomic structure), 2) a component due to the combined effect of \(+G\) and the anti-G suit, 3) a component due to PBG, and 4) a residual variation component. The component of variation due to position can be thought of as variation arising from a biological pattern that endured across the \(+G\) levels and countermeasures being considered. The second and third components of variation represent the changeable portion of flow across changes in \(+G\) levels and anti-G suit and in PBG, respectively. The residual variation is due to time, methodological noise, and the three-way interaction of piece position, \(+G\) and anti-G suit, and PBG.

The components of flow variation were estimated from the following model using four conditions (+6 G with anti-G suit at 6.0 psi, +9 G with anti-G suit at 10.5 psi, both with and without PBG).

\[
\text{Flow}_{ijk} = \alpha_i + \beta_j + \gamma_k + (\alpha\beta)_{lj} + (\alpha\gamma)_{ik} + (\beta\gamma)_{jk} + \epsilon_{ijk} \quad (1)
\]

Flow \(_{ijk}\) is the perfusion for piece \(i\) under inertial load \(j\) and PBG condition \(k\); \(\alpha\), \(\beta\), and \(\gamma\) are the main effects of inertial force and anti-G suit, PBG, and piece (structure), respectively. In addition, the terms \((\alpha\beta)_{lj}\), \((\beta\gamma)_{jk}\), and \((\alpha\gamma)_{ik}\) represent the various two-way interaction effects of inertial force and anti-G suit, PBG, and piece. The term \((\alpha\beta\gamma)_{ijk}\) is the three-way interaction effect, and \(\epsilon_{ijk}\) represents the random variation due to time and measurement error (5).

Because we normalized blood flow to a mean of 1.0 for each condition, \(\alpha_i = \beta_j = (\alpha\beta)_{lj} = 0\). Not all exposures were repeated for a given animal (Table 1); thus the variance of \(\epsilon_{ijk}\) could not be estimated separately from the variance of \((\alpha\beta\gamma)_{ijk}\). We used \((\alpha\beta\gamma)_{ijk}\) to represent the sum of these two terms so \(\epsilon_{ijk}\) was dropped. Equation 1 is thus reduced to

\[
\text{Flow}_{ijk} = \gamma_k + (\alpha\beta)_{lj} + (\alpha\gamma)_{ik} + (\alpha\beta\gamma)_{ijk} \quad (2)
\]

where \(\gamma\) represents the piece effect, \((\alpha\beta)_{lj}\) represents the effect of PBG in modifying the distribution of blood flow across pieces, \((\alpha\gamma)_{jk}\) represents the effect of inertial force and anti-G suit in modifying the distribution of blood flow across pieces, and \((\alpha\beta\gamma)_{ijk}\) represents the residual error.

The variance estimates were based on standard analysis of variance (ANOVA) methods for mixed models with \(+G\) and PBG as fixed effects and piece and its interactions as random effects. Use of an anti-G suit and PBG did not change the variance estimates or the ANOVA assumptions were met here because of statistical dependence of flow among pieces and main effects constrained to be zero. Nevertheless, the estimates of the variance components are valid, descriptive measures for components of total flow variation. The estimation of each variance component was derived from equations involving sums of squares and mean squares. Setting each mean square equal to its expected value yields a simple set of simultaneous equations in the variance components of interest. Each variance component can be estimated from the solution of these equations. The relative contribution of each component to the total flow variation in the experiment was calculated as a percentage.

**RESULTS**

**Physiological Data**

Basic physiological data for the animals are shown in Table 2. Gas exchange deteriorated with increasing inertial load, as indicated by a decrease in \(P_aO_2\). The increase in \(P_aCO_2\) with inertial stress indicates hyperventilation. Use of an anti-G suit and PBG did not improve blood gases. Alveolar-artrial \(O_2\) differences are shown in Table 2. Physiological parameters

<table>
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<tbody>
<tr>
<td>Temp, °C</td>
<td>37.8±0.7</td>
<td>38.5±0.3</td>
<td>38.1±0.4</td>
<td>38.6±0.5</td>
<td>39.0±0.5</td>
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<tr>
<td>(P_aO_2), Torr</td>
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<td>70.1±2.4</td>
<td>48.4±2.4</td>
<td>39.1±6.3</td>
<td>31.5±2.3</td>
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<tr>
<td>(P_aCO_2), Torr</td>
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<td>241.1±4.7</td>
<td>50.0±4.3</td>
<td>57.9±4.7</td>
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<tr>
<td>pH</td>
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<td>7.33±0.03</td>
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<td>7.24±0.05</td>
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<tr>
<td>A-aPO2, Torr</td>
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<td>42.7±4.7</td>
<td>42.8±5.1</td>
<td>44.3±7.1</td>
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<tr>
<td>CO, l/min</td>
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<td>4.5±0.9</td>
<td>7.2±1.5</td>
<td>4.6±0.4</td>
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<td>5.3±1.9</td>
<td>6.7±1.5</td>
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<tr>
<td>HR, beats/min</td>
<td>116.8±12.8</td>
<td>131.8±19.9</td>
<td>170.2±18.7</td>
<td>165.8±21.9</td>
<td>182.5±14.9</td>
<td>186.0±6.3</td>
<td>167.5±29.9</td>
<td></td>
</tr>
<tr>
<td>SAP, mmHg</td>
<td>127.4±5.2</td>
<td>159.1±8.5</td>
<td>180.9±7.3</td>
<td>261.7±9.2</td>
<td>243.0±19</td>
<td>303.8±10.9</td>
<td>301.5±12.3</td>
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<td>Pap, mmHg</td>
<td>25.3±1.1</td>
<td>77.5±1.1</td>
<td>29.0±5.7</td>
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<td>160.6±4.6</td>
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<td>CVP, mmHg</td>
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<td>19.0±5.2</td>
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<td>137.2±9.1</td>
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<tr>
<td>Pes, mmHg</td>
<td>2.6±1.34</td>
<td>51.7±4.6</td>
<td>22.2±3.0</td>
<td>95.6±13.9</td>
<td>115.6±6.1</td>
<td>159.3±23.6</td>
<td>185.9±10.3</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE. Temp, body temperature; \(P_aO_2\), arterial \(O_2\) tension; \(P_aCO_2\), arterial \(CO_2\) tension; pH, arterial pH; A-aPO2, alveolar-arterial \(O_2\) gradient; CO, cardiac output (determined from reference blood not thermal dilution); HR, heart rate; SAP, mean systemic arterial pressure; Pap, mean pulmonary arterial pressure; CVP, mean central venous pressure; Pes, mean esophageal pressure. Statistical significance between conditions (0 G and +3 G, +3 G and +6 G, +6 G and +9 G) determined by paired t-test: \(1^P = 0.02, 2^P = 0.004, 3^P = 0.03, 4^P = 0.04, 5^P = 0.005, 6^P = 0.03, 7^P = 0.0007, 8^P = 0.0006, 9^P = 0.0001, 10^P = 0.001, 11^P = 0.0005, 12^P = 0.002, 13^P = 0.008, 14^P = 0.001, 15^P = 0.007, 16^P = 0.02.\)
were calculated by using respiratory quotients of 0.85. The thermal dilution did not work well, so cardiac output was determined from fluorescent microspheres with the use of a reference withdrawal pump (21). Heart rate, mean systemic arterial pressure, and mean pulmonary arterial pressure increased with inertial force.

Heterogeneity

The blood flow of each lung piece for a representative animal is plotted as frequency histograms in Fig. 2. At 0 Gz, the distribution of pulmonary blood flow is centered around the mean relative flow of 1.0. As the inertial load increased to +3 Gz with an anti-G suit pressure of 1.5 psi, blood flow heterogeneity substantially increased. This is represented by a broader histogram (Fig. 2B) and a CV of 62%. However, at +6 and +9 Gz, as anti-G suit pressure increased, heterogeneity substantially decreased and the histograms became more narrow (Fig. 2, C and D). Heterogeneity of pulmonary blood flow did not change with positive pressure breathing (Fig. 3 and Table 3; histograms not shown).

These trends in perfusion heterogeneity were observed in all animals (Fig. 3 and Table 3). On average, perfusion heterogeneity increased from 45.3% at 0 Gz to 59.5% at +3 Gz (Fig. 3A). With the increased anti-G suit pressure at +6 and +9 Gz, heterogeneity decreased to 36.6 and 45.0%, respectively, despite the increase in +Gz stress. Positive pressure breathing did not have a significant effect on perfusion heterogeneity, as shown in Fig. 3B.

Pulmonary Blood Flow

Linear gradients of pulmonary blood flow distribution were identified using least-squares linear regression of blood flow vs. each spatial direction.

Cranial-to-caudal gradients (z coordinate; +Gz). Blood flow vs. z-coordinate plots for one animal are shown in Fig. 4. At 0 Gz (Fig. 4A), the slope of the linear regression was negative, indicating greater blood flow in cranial lung regions compared with caudal regions. Note the high degree of heterogeneity within each perpendicular slice along the cranial-caudal axis (transverse planes). At +3 Gz (Fig. 4B), the slope became positive, demonstrating a blood flow shift toward caudal lung regions, in the direction of the inertial force. Heterogeneity decreased greatly within cranial-caudal planes in the most cranial part of the lung. As +Gz was increased further to +6 and +9 Gz and the anti-G suit pressure increased, blood flow redistribution was minimized, as shown in Fig. 4, C and D. Although regression slopes at 0 and +6 Gz are similar, the differences in heterogeneity within transverse planes are great. Blood-flow redistribution did not change with positive pressure breathing (Table 3; plots not shown).

These trends in perfusion redistribution in the cranial-to-caudal direction were observed in all animals (means shown in Table 3) and are made more clear when the regression slopes are plotted as a function of +Gz level. As shown in Fig. 5A, pulmonary blood flow shifted from cranial-to-caudal lung regions when inertial load was increased from 0 to +3 Gz. With increased
DISTRIBUTION OF PULMONARY BLOOD FLOW DURING +Gx STRESS

Plots. The mean regression slope of all animals at 0 Gx was slightly positive, indicating blood flow in the dorsal regions of the lung greater than that in the ventral regions. With an increase in inertial stress to +3 Gx, the slope became more positive, demonstrating increased blood flow to dorsal regions of the lung. As +Gx was increased further to +6 and +9 Gx and anti-G suit pressure increased, blood flow shifted minimally. Blood-flow redistribution did not change with positive pressure breathing. All vertical shifts in blood flow mentioned above were small and statistically nonsignificant (Table 3).

Right-to-left gradients (y coordinate). A small positive slope in the right-to-left direction was seen across all animals at 0 Gx. The flow was slightly higher in the left lung than in the right lung. The effects of increased +Gx, anti-G suit, and positive pressure breathing on pulmonary blood flow in the right-to-left direction were nonsignificant (Table 3).

Radial gradients (Dx). Pulmonary blood flow shifted from central (hilair) to peripheral regions of the lung when inertial stress was increased from 0 to +3 Gx (Fig. 6 and Table 3). Anti-G suit pressure at +6 Gx (6 psi) caused blood flow to shift back toward hilar regions. Blood flow again shifted to peripheral regions of the lung at +9 Gx and anti-G suit pressure of 10.5 psi. Positive pressure breathing did not have a significant effect on blood flow redistribution (Table 3).

Center of Flow

The center of flow was calculated as a spatial average of blood flow for the x, y, and z coordinates. Changes in the center of flow are shown in Table 4. With increased inertial force, large shifts in the center of flow occurred in the direction of the inertial force. High anti-G suit inflation pressure reversed this shift. Significant changes in the center of flow were not observed in the x and y coordinates. The overall shift in center of flow was large for inertial load and anti-G suit and small for PBG.

Blood Flow Variation

Variation in pulmonary blood flow was separated into that due to anatomic structure, +Gx, and anti-G suit, and PBG. As shown in Table 5, the strongest

Table 3. CV and spatial dimension gradients by G level and countermeasure

<table>
<thead>
<tr>
<th>Condition</th>
<th>n</th>
<th>CV, %</th>
<th>x Gradient</th>
<th>y Gradient</th>
<th>z Gradient</th>
<th>Dx Gradient</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 Gx</td>
<td>6</td>
<td>45.3 ± 3.9(^4)</td>
<td>0.006 ± 0.006</td>
<td>0.002 ± 0.005</td>
<td>-0.011 ± 0.006(^3,4)</td>
<td>-0.023 ± 0.007(^5)</td>
</tr>
<tr>
<td>0 Gx, PBG</td>
<td>5</td>
<td>46.1 ± 2.3</td>
<td>-0.024 ± 0.011</td>
<td>0.002 ± 0.006</td>
<td>-0.023 ± 0.006</td>
<td>-0.036 ± 0.008</td>
</tr>
<tr>
<td>+3 Gx</td>
<td>6</td>
<td>59.5 ± 2.7(^\text{a,b})</td>
<td>0.030 ± 0.008</td>
<td>0.006 ± 0.008</td>
<td>0.042 ± 0.006(^3,5,6)</td>
<td>0.036 ± 0.011(^7,8)</td>
</tr>
<tr>
<td>+6 Gx</td>
<td>6</td>
<td>36.6 ± 1.2(^2)</td>
<td>0.021 ± 0.004</td>
<td>0.001 ± 0.004</td>
<td>-0.006 ± 0.006(^5,6)</td>
<td>0.031 ± 0.010(^9,10)</td>
</tr>
<tr>
<td>+6 Gx, PBG</td>
<td>5</td>
<td>38.8 ± 1.7</td>
<td>0.029 ± 0.002</td>
<td>-0.002 ± 0.004</td>
<td>0.000 ± 0.006</td>
<td>-0.025 ± 0.010</td>
</tr>
<tr>
<td>+9 Gx</td>
<td>5</td>
<td>45.0 ± 5.7</td>
<td>0.031 ± 0.010</td>
<td>0.002 ± 0.004</td>
<td>0.019 ± 0.011(^3,4,6,7)</td>
<td>0.006 ± 0.019(^8)</td>
</tr>
<tr>
<td>+9 Gx, PBG</td>
<td>4</td>
<td>47.1 ± 5.1</td>
<td>0.033 ± 0.011</td>
<td>0.005 ± 0.003</td>
<td>0.019 ± 0.019</td>
<td>0.009 ± 0.031</td>
</tr>
</tbody>
</table>

Values are means ± SE. Gradient values are regression slopes of blood flow vs. x, y, z, and Dx dimensions expressed as weight-normalized relative flow units per centimeter. CV, coefficient of variation; x, ventral-to-dorsal direction; y, right-to-left direction; z, cranial-to-caudal direction; Dx, distance from hilum. Statistical significance between conditions determined by paired t-test: \(^1P = 0.02, ^2P = 0.004, ^3P = 0.0004, ^4P = 0.03, ^5P = 0.0004, ^6P = 0.04, ^7P = 0.002, ^8P = 0.002, ^9P = 0.04.\)
The piece effect explains a mean of 61% of the total flow variance. The combined effects of $+G_z$ and anti-G suit explain a considerably smaller 20% of the total variance. PBG accounts for only 6% of variation in flow. The residual variation is 14%. The ordering of these effects is consistent across the four animals, with fairly similar magnitudes, despite a twofold difference in total variance (0.16–0.32).

**DISCUSSION**

This is the first study to use a high-resolution technique to describe the redistribution of pulmonary blood flow with $+G_z$ stress, anti-G suit, and PBG. The primary findings are as follows: 1) heterogeneity of pulmonary blood flow in unanesthetized miniature swine increases as $+G_z$ increases; 2) pulmonary blood flow shifts from cranial and central regions of the lung toward caudal and peripheral regions as $+G_z$ increases; 3) inflation of anti-G suit at high $+G_z$ minimizes blood flow redistribution along the cranial-to-caudal axis seen at high $+G_z$ without the anti-G suit (0 and $+3 G_z$); 4) positive pressure breathing during $+G_z$ has no measurable effect on blood flow heterogeneity and redistribution; and 5) anatomic structure is the major determinant of pulmonary blood flow across all considered $+G_z$ levels and countermeasures.

**Methodological Issues**

We used 15-μm-diameter fluorescent-labeled microspheres as markers of pulmonary blood flow. Use of fluorescent microspheres was recently validated for measurement of regional pulmonary blood flow in dogs (18). The resulting excitation and emission spectra are easily separated, providing a method for measuring perfusion at multiple physiological conditions. In addition, techniques for injection of microspheres into conscious animals undergoing centrifugation have been previously developed and successfully employed (33, 43, 44). In the present study, microspheres were completely entrapped by the pulmonary circulation, as confirmed by the absence of fluorescence in the kidney and heart samples. To limit the effect of methodological error, we injected $2 \times 10^6$ microspheres, providing $>400$ microspheres per lung piece when blood flow was $\geq 25\%$ of average flow (8).
To examine flow gradients and relative flow distributions, we normalized each lung piece to the mean flow of all pieces per animal. Flow to each piece was also normalized by the weight of the piece to correct for variations in piece size. Lung pieces that contained more than 25% airways were excluded from the analysis because airway cartilage has a higher density than lung parenchyma. Inclusion of these pieces would result in large errors in weight normalization.

We dried the lungs at total lung capacity with a pressure of 25–30 cmH₂O; therefore, all alveoli were nearly uniform in size. The process of weight normalization of each lung piece thus provided blood flow per alveolar volume. The configuration of the lungs after drying may be different from that of an in vivo lung. The volume of the dried lung was slightly greater than in vivo, which increased the linear dimensions slightly. The geometry of the lung after it is dried will be slightly different from that in the intact lung because of the lack of the physical constraints of the chest wall and diaphragm. Drying the lungs at total lung capacity (vs. functional residual capacity) will have a negligible influence on the interpretation of overall heterogeneity.

The geometry of the lung at the time of microsphere injection may be different from that at the time of lung drying. Lung parenchyma is an elastic tissue with a density of 0.2–0.3 g/cm³ and will distort under increased inertial loads. It is likely that cranial regions of the lung were stretched during +Gz stress, whereas caudal regions were compressed. Inflation of the anti-G suit can greatly elevate the diaphragm; therefore, caudal lung regions may be further compressed.

The miniature swine has been developed as an excellent human analog for acceleration research (12). The miniature swine have anatomic structures similar to humans, including heart-to-brain distance, cardiovascular system, and lung size. In addition, miniature swine instinctively perform straining maneuvers that resemble the AGSM performed by high-performance aircraft pilots during high-G stress. Straining increases systemic arterial blood pressure through increases in intrathoracic pressure, as does PBG (9). Intrathoracic pressure generated from anti-G suit is an active Valsalva-type maneuver that uses the intercostal and the abdominal muscles, an outside-to-inside pressure on the lungs. However, pressure breathing is a passive inside-to-outside lung pressure that results from tracheal pressurization. When supported with the anti-G suit, the effect of straining on the distribution of pulmonary blood flow is similar to PBG and not significantly different.

**Pulmonary Blood Flow and Increased +GZ**

The overall heterogeneity of perfusion for all animals at 0 Gz (baseline) was 45%. Previous studies have observed perfusion heterogeneity of 41–47% in anesthetized dogs (21, 34), 31% in unanesthetized horses (4, 27), 30% in unanesthetized sheep (41), 57% in anesthetized pigs (1), and 65% in anesthetized upright baboons (19). Lung pieces were of similar size (1.0–1.9 cm³) in these studies because heterogeneity increases with decreasing piece size (22).

In the present study, increases in the +Gz level caused significant increases in blood flow heterogeneity.

![Fig. 5. Cranial-to-caudal flow gradient vs. +Gz level for all animals (A) and vs. with or without positive pressure breathing (B).](http://jap.physiology.org/)

![Fig. 6. Radial flow gradient (distance from hilum, Dh) vs. +Gz level for all animals.](http://jap.physiology.org/)
ity. As seen in Fig. 3A, heterogeneity increased as +Gx stress was increased from 0 to +3 Gx. The CV increased by an average of 4.7% per unit increase in +Gx. In a recent study, Hlastala et al. (28) exposed miniature swine to −Gx. With the use of the same high-resolution techniques as the present study, a similar trend in heterogeneity was seen. Hlastala et al. (28) found a baseline CV of 38% in miniature swine and an increase in CV of 16.7% per unit increase in −Gx. The difference in the rate of CV increase between the two studies may be due to differences in lung compression. An anti-G suit was inflated in the present (+Gx) study, which contributed to a minimizing of rib and abdominal expansion in the caudal lung regions. As a result, patterns of vascular resistance and thus perfusion were different. The direction of acceleration during +Gx acceleration is coincident with cranial-caudal axis of trachea and large airway. In the −Gx acceleration, the direction is perpendicular to the major airway axis, perhaps causing a greater parenchymal displacement. Lung structure (vascular branching) with respect to the direction of the inertial force may play a role.

The directional reference frame used by acceleration physiologists is one that is fixed to the anatomic orientation of the subject. It is defined with the x-axis oriented in the cranial-caudal direction or perpendicular to a coronal plane, the y-axis oriented in the right-to-left direction or perpendicular to a sagittal plane, and the z-axis oriented in the cranial-caudal direction or perpendicular to a transverse plane. The orientation of the inertial vector with respect to lung geometry must be considered when comparing acceleration studies from animals to humans. We have made comparisons in the following way: 1) miniature swine at +Gx (Fig. 7A) to human at +Gx (Fig. 7C) so that the inertial vector is parallel to long axis of both lungs (cranial-caudal axis) and 2) miniature swine at +Gx (Fig. 7A) to human at ventral-dorsal inertial flow (+Gx) (Fig. 7D) so that the inertial vector is horizontal with respect to normal posture for both subjects.

Results from human exposures to +Gx stress are conflicting. Hoppin et al. (30) found that +4 and +8 Gx stress had no effect on the distribution of blood flow in three human subjects. However, Glaister (14) and von Nieding et al. (40) showed that pulmonary blood flow shifted in an inertial-dependent manner. Exposure to +Gx decreased blood flow to ventral lung regions and increased flow to dorsal regions.

Three earlier studies that exposed human subjects to +Gx observed a shift in pulmonary blood flow in the direction of the inertial vector. Bryan et al. (7) investigated the effect of inertial stress up to +4 Gx in separate human subjects. Blood flow decreased in the upper (cranial) regions of the lung in agreement with zone model predictions. Flow at the base of the lung increased but became fixed at levels higher than +2 Gx. Glaister (15) also found that blood flow was depressed in cranial lung regions of human subjects but to an extent that was larger than that shown by Bryan et al. (7). Glaister observed that the upper half of the lung was not perfused at +3 Gx. Results obtained by von Nieding et al. (40) found similar shifts in blood flow.

In the present study, a redistribution of pulmonary blood flow in the direction of the inertial vector was observed. As seen in Fig. 4, A and B, pulmonary blood flow shifted from cranial regions of the lung toward caudal regions as inertial load was increased from 0 to +3 Gx. The cranial-caudal gradient increased by an average of 0.018 relative flow units per centimeter per unit increase in +Gx (Fig. 5A). With increases in −Gx
stress, Hlastala et al. (28) observed a shift in blood flow from dorsal regions toward ventral regions of the lung. This dorsal-to-ventral gradient increased by 0.035 relative flow units per centimeter per unit increase in $-G_x$. Differences in vasculature branching with respect to the direction of the inertial force is most likely the reason for the rate differences between the two studies.

We also found a redistribution of blood flow from central lung regions to peripheral regions as $+G_z$ stress was increased. Because the spherical radial distance is expressed as $D_h$, changes in pulmonary blood flow to the dorsal-caudal regions of the lung have a large influence on the central-to-peripheral flow gradient. Because the inertial force was in the cranial-to-caudal direction, the central-to-peripheral shift in blood flow is most likely due to an increase in flow in the dorsal-caudal pieces (most distant from the hilum), which is expected with $+G_z$ acceleration. With increases in $-G_x$, Hlastala et al. (28) found a peripheral-to-central shift in blood flow. This gradient is opposite to the findings of the present study and may be due to a decrease of pulmonary blood flow to the dorsal-caudal regions, which is expected with $-G_x$. Alternatively, lung structure (vascular branching) with respect to the direction of the inertial force may play a role.

ANOVA reveals that anatomic structure is the major determinant of pulmonary blood flow, accounting for a mean of 61% of the flow variation across the considered $+G_z$ levels and countermeasures. This is illustrated for a representative animal in Fig. 8, in which blood flow at $+3 G_z$ is plotted against blood flow at $0 G_z$. Here, lung pieces with high blood flow tend to maintain high flow and pieces with low flow maintain low flow ($r = 0.8$). Inertial force plays a secondary role (20% of total variance), and the effect of PBG is minor (6%).

Heterogeneity of pulmonary blood flow within coronal planes was recently found in miniature swine exposed to $-G_x$ stress (28) but was not detected in earlier $-G_x$ studies of pulmonary blood flow. The difference in results between earlier studies and those more recent can be explained by the level of spatial resolution available. Earlier studies used radiolabeled gas (14, 15), macroaggregated albumin (7, 30), and microspheres (13, 23, 40, 43, 44) to determine the distribution of pulmonary blood flow. External scintillation counters with poor spatial resolution measured radioactivity across the chest; thus heterogeneity within planes perpendicular to the inertial vector could not be detected.

The results from a low-spatial resolution study by Glaister (14) are shown in Fig. 9A. Here, the average distribution of relative flow per alveolus for three human subjects at $+1$ and $+5 G_z$ is plotted against position along the ventral-dorsal axis. As $+G_z$ increased, blood flow decreased in ventral regions and increased in dorsal regions of the lung and hence shifted in the direction of the $+G_z$ vector. Data were obtained from external lung scans and represent mean flow in coronal planes (perpendicular to the inertial vector). Information on heterogeneity within coronal planes could not be collected in Glaister’s experimental setup.

For comparison, we reduced the spatial resolution of the present study by averaging observations within transverse planes. Figure 9B shows $0$ and $+3 G_z$ data from one animal at a low-spatial resolution, similar to that of Glaister. Here, blood flow shifts in the direction of the $+G_z$ vector along the cranial-caudal axis. Heterogeneity within transverse planes is not apparent. However, when the flow distribution of individual pieces is displayed, heterogeneity within each transverse plane becomes apparent (Fig. 4).

**Anti-G Suit**

The primary hemodynamic effect of $+G_z$ stress to aircrew is a decrease in arterial blood pressure and...
flow to the brain. To offset this effect, anti-G suits have been developed as a countermeasure. Anti-G suits improve $+G_z$ tolerance by increasing systemic arterial blood pressure and minimizing blood pooling in dependent regions, thus assisting venous return to the chest. Similar to humans, miniature swine do not tolerate high $+G_z$ stress without protection. In the present study, the miniature swine wore a custom-made ECGS, which was designed from the US Air Force human ECGS, called the advanced technology anti-G suit (ATAGS). The anti-G suit was always inflated during $+3$, $+6$, and $+9$ $G_z$ exposures. As inertial load was increased from $+3$ to $+6$ $G_z$, perfusion heterogeneity decreased despite the increase in $G$ level. This suggests that the increase in anti-G suit pressure from 1.5 psi at $+3$ $G_z$ to 6.0 psi at $+6$ $G_z$ countered the effects of increased $+G_z$ on perfusion heterogeneity. The unique contribution of the anti-G suit cannot be specified numerically. However, on the basis of the reversal of the increase in heterogeneity and the change in the cranial-to-caudal gradient, the effect of the anti-G suit is quite large. It is probable that, in the absence of the anti-G suit at $+6$ and $+9$ $G_z$, perfusion heterogeneity and the cranial-caudal gradient would continue to increase.

An explanation for the anti-G suit effect is that high inflation pressures compress the abdominal contents and thus elevate the diaphragm. This in turn compresses caudal lung regions, resulting in increased vascular resistance and decreased perfusion. Blood flow cannot shift; therefore, the distribution resembles that at $0$ $G_z$.

In earlier studies, Whinnery et al. exposed anesthetized miniature swine (with anti-G suit protection) to $0$ and $+7$ $G_z$ (44) and loads ranging from $-4$ to $+8$ $G_z$ (43). As $+G_z$ increased, pulmonary blood flow decreased not only in the most cranial lung regions but also in the most caudal regions. Increases in pulmonary perfusion were observed in the midlung. The anti-G suit used in these studies was a simple abdominal bladder suit and not an ECGS. Our observations regarding central-peripheral shifts are consistent with Whinnery’s findings.
Positive-Pressure Breathing

Positive pressure breathing during +GZ is a countermeasure designed to aid in +GZ tolerance by increasing intrathoracic pressure, which leads to increased arterial blood pressure. Thus less effort is required when performing AGSMs. The result is less workload and fatigue, thereby improving the pilot’s ability to sustain prolonged or recurrent exposures to high +Gz (9, 11, 38, 39). The increased intrathoracic pressure caused by PBG restricts venous return so blood pools in the lower extremities. Therefore, PBG is used in conjunction with an inflated anti-G suit in the high-performance aviation environment. A recent study showed that ATAGS in combination with PBG protects human subjects to +9 Gz, and no straining maneuvers were necessary (10). PBG does not alter regional pulmonary blood flow patterns from those seen without PBG when venous return and peripheral resistance are supported by anti-G suit inflation. PBG does not improve gas exchange efficiency as seen in alveolar-arterial O2 differences. However, it has been shown that PBG reduced the rate of arterial desaturation in humans (11).

Role of Countermeasures

Overall, our data demonstrate that vascular structure is the dominant factor in determining the distribution of blood flow in the lung during acceleration stress. Increasing cranial-to-caudal (z-axis) G stress (0 to +3 Gz) results in an increased CV (45.3 ± 3.9 to 59.5 ± 2.7; Table 3), an increased z gradient (−0.011 ± 0.006 to 0.042 ± 0.006; Table 3), and a caudal shift in center of flow (2.04 ± 0.34 cm; Table 4). These changes are due to the increased cranial-to-caudal inertial load that affects both the hydrostatic column within the pulmonary vasculature and the effect on expansion of caudal rib cage dimension and due to the caudal shift in diaphragm position and abdominal contents.

Further increasing G stress from +3 to +6 Gz causes the surprising effect of shifting blood flow distribution in the cranial direction: decreased CV (59.5 ± 2.7 to 36.6 ± 1.2), decreased z gradient (0.042 ± 0.006 to −0.006 ± 0.006), and a cranial shift in center of flow (−1.83 ± 0.28 cm). The expected caudal shift in blood flow did not occur with increased G stress from +3 to +6 Gz. It appears that inflation of the anti-G suit and subsequent return of the basal rib cage and abdominal and diaphragm toward their control position offset the caudal shift in pulmonary blood flow. No significant differences in either CV or z gradient were shown when +6 Gz (with ECGS) was compared with 0 Gz. The center of flow shifts in the cranial direction by only 0.21 ± 0.13, which, for a +6 Gz difference, is only 10% of the caudal shift of that between 0 and +3 Gz with minimal anti-G suit pressure. Increasing from +6 Gz to +9 Gz results in no significant change in CV (36.6 ± 1.2 to 45.0 ± 5.7; Table 3) and an increase in z slope (−0.006 ± 0.006 to 0.019 ± 0.011; Table 3). The center of flow shifted 0.99 cm in the caudal direction.

Further support for the finding that gravity has only a minimal direct effect on pulmonary blood flow distribution is found in the components of flow variation listed in Table 5. Analysis of the variability of blood flow within individual regions shows that more than 60% of flow variation is due to pulmonary vascular structure. Inertial force and anti-G suit account for less than 20% of the flow variation.

One key difference between the present study and earlier studies is the use of a more advanced anti-G suit, which was designed to minimize the downward movement of the diaphragm and outward movement of the chest wall. This may account for the qualitative differences between our findings of a minimal effect of gravity and those of Glaister (15) and Bryan et al. (7); both studies found a greater influence of gravity on pulmonary blood flow distribution.

The data indicate that there is only a minimal effect of gravity on pulmonary blood flow distribution when the chest wall and abdomen configuration are held constant with anti-G suit. The apparent shift of blood flow with minimal anti-G suit inflation is due to the effect of gravity on configuration of the chest wall, diaphragm, and abdominal contents.

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The animals involved in this study were procured, maintained, and used in accordance with the Animal Welfare Act and the Guide for the Care and Use of Laboratory Animals prepared by the Institute of Laboratory Animal Resources, National Research Council. The Air Force Research Laboratory is accredited by the American Association for Accreditation of Laboratory Animal Care.

Opinions, interpretations, conclusions, and recommendations are those of the authors and are not necessarily endorsed by the United States Air Force.

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