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Steep head-down tilt has persisting effects on the distribution of pulmonary blood flow

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Abstract

Head-down tilt has been shown to increase lung water content in animals and alter the distribution of ventilation in humans; however, its effects on the distribution of pulmonary blood flow in humans are unknown. We hypothesized that head-down tilt would increase the heterogeneity of pulmonary blood flow in humans, an effect analogous to the changes seen in the distribution of ventilation, by increasing capillary hydrostatic pressure and fluid efflux in the lung. To test this, we evaluated changes in the distribution of pulmonary blood flow in seven normal subjects before and after 1 h of 30° head-down tilt using the magnetic resonance imaging technique of arterial spin labeling. Data were acquired in triplicate before tilt and at 10-min intervals for 1 h after tilt. Pulmonary blood flow heterogeneity was quantified by the relative dispersion (standard deviation/mean) of signal intensity for all voxels within the right lung. Relative dispersion was significantly increased by 29% after tilt and remained elevated during the 1 h of measurements after tilt (0.84 ± 0.06 pretilt, 1.09 ± 0.09 calculated for all time points posttilt, $P < 0.05$). We speculate that the mechanism most likely responsible for our findings is that increased pulmonary capillary pressures and fluid efflux in the lung resulting from head-down tilt alters regional blood flow distribution.

Keywords

functional magnetic resonance imaging; arterial spin labeling; relative dispersion; heterogeneity; interstitial pulmonary edema

Regional pulmonary blood flow may be significantly altered by cardiovascular or pulmonary disease. For example, focal lung disease may cause local abnormalities in blood flow, whereas diffuse pulmonary disease may alter blood flow throughout the lung (33). Such alterations can lead to ventilation-perfusion mismatch and inefficient gas exchange (29). Therefore, the ability to quantitatively evaluate such alterations in pulmonary blood flow distribution may provide insight into how pulmonary diseases such as pulmonary edema affect lung function.

Head-down tilt increases pulmonary vascular pressures. In rabbits, 1 h of steep (20°) head-down tilt results in a significant increase in pulmonary interstitial pressure of a magnitude comparable to that seen in the infusion-based models of hydraulic pulmonary interstitial

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edema (20). This observed change in interstitial pressure after head-down tilt suggests fluid accumulation in the interstitial matrix (20, 21). In rats, steep head-down tilt (45°, using tail suspension) results in the development of interstitial pulmonary edema in the alveolar septal areas after 2 h of exposure (24). Moreover, the edema was shown to shift to the areas around pulmonary airways and arteries, which would likely result in alterations in the distribution of ventilation and pulmonary blood flow.

In humans, 1 h of steep, 60° head-down tilt increases ventilatory inhomogeneity (23), and similar changes are found using 1 h of 30° head-down tilt (Olfert IM and Prisk GK, unpublished observations). During 60° head-down tilt, ventilatory inhomogeneity immediately changed but then continued to change over approximately the first 30 min of tilt. This suggests that ventilatory inhomogeneity in the normal human lung during head-down tilt is dependent not only on gravitational reorientation of lung tissue during head-down tilt, but also on some other mechanism with a time course requiring 30 min to reach full effect, possibly because of the increased intrathoracic blood volume and/or the accumulation of extravascular lung water. In keeping with this idea, head-down tilt has been shown to redistribute body fluid from caudal to cephalic regions in humans (11), and an elevated thoracic blood volume would be expected to engorge pulmonary and other major vessels, subsequently altering the geometry of the distal airways.

The effects of head-down tilt on pulmonary blood flow heterogeneity are unknown. We hypothesized that head-down tilt, by increasing capillary hydrostatic pressure and fluid efflux in the lung, would alter the distribution of pulmonary blood flow. To test this hypothesis, we evaluated changes in the heterogeneity of pulmonary blood flow that occurred before and during the hour after 1 h of steep 30° head-down tilt using pulmonary functional magnetic resonance imaging (MRI).

METHODS

Subject selection

This study was approved by the University of California, San Diego Human Research Protection Program and conducted with written, informed consent of all subjects. Seven healthy subjects were included in each of the two experiments comprising this study, with two subjects participating in both experiments (total of 12 subjects). Subjects were included if they had no history of smoking or lung disease, did not have contraindications to MRI, and had normal spirometry.

Protocol

Two experiments were conducted to determine the effects of 1 h of 30° head-down tilt on the heterogeneity of pulmonary blood flow. First, to establish that changes in relative dispersion were a result of head-down tilt and not simply from lying supine, measurements were made on 7 subjects at the beginning and end of lying supine in the scanner for 1 h with no other intervention. Data were acquired in triplicate, and arterial oxygen saturation and heart rate were monitored (3150 MR Monitor; Invivo Research, Orlando, FL). Measurements were made after 10 min of lying supine and then repeated 1 h after the baseline measurement.

During the second experiment, images were acquired at baseline while the subjects were lying supine, after which the subject was placed in a steep (30°) head-down tilt position for 1 h outside the scanner room. At the end of the hour tilt period, the subject was returned to the supine position and transported via gurney to the scanner in a manner such that he or she was always supine (never upright) between the time of tilt and the completion of data collection posttilt. Posttilt images were acquired at ~10, 20, 30, 40, 50, and 60 min after tilt.

Head-down tilt

A commercially available inversion table (Teeter Hang Ups F5000, STL International, Puyallup, WA) was used to place subjects in the head-down tilt position for 1 h. The subjects' body weight was supported by a climbing harness positioned low across the hips, leaving the shoulders, chest, and abdomen able to move freely.

Pulmonary functional MRI

Each subject underwent MRI with arterial spin labeling using a Vision 1.5-T whole-body magnetic resonance scanner (Siemens Medical Systems, Erlangen, Germany). Arterial spin labeling is a technique used for the quantification of organ blood flow. The signal intensity of the image has been shown to be proportional to bulk flow in vitro by use of a tube-flow model (1), and it has been validated in heart with microspheres (26) and skeletal muscle with venous occlusion plethysmography (28) with an excellent linear relationship between arterial spin labeling measurements and the validating technique. Arterial spin labeling inverts the proton magnetization of blood by applying a radiofrequency pulse, allowing these magnetically tagged protons in blood to act as an endogenous tracer for the evaluation of blood flow. During each measurement, two images are acquired (Fig. 1, *A* and *B*) such that in the first image there is intravascular signal and in the second image the intravascular signal has been nulled. The two images are then subtracted. Signal from inverted protons in stationary tissue will be present in both images and thus will cancel out when the two images are subtracted. The signal from each image voxel in the subtracted image is then proportional to the amount of blood that entered the voxel over a specific time period (4, 5).

Pulmonary blood flow was assessed using a 2D arterial spin labeling, flow-sensitive alternating inversion recovery with an extra radiofrequency pulse (ASL-FAIRER) sequence with a half-Fourier acquisition single-shot turbo spin-echo (HASTE) imaging scheme. The ASL-FAIRER sequence was modified from one previously reported (16, 17) to allow for a single subtraction (4) and has been utilized in other studies by our group (4, 12). In essence, two images are acquired. The sequence is cardiac gated such that *1*) both images are acquired during diastole (while flow is minimal) and *2*) one complete systole interval occurs during the time between the two images. In the first image, termed the "selective tag" image, there is intravascular signal. In the second "nonselective tag" image, the intravascular signal has been largely eliminated. This is accomplished as follows: the pulse sequence begins with a preparatory inversion (180°) pulse followed immediately by a selective 90° radiofrequency pulse, centered on the imaging plane. In the selective tag image, the inversion pulse is applied only to the region of the lung that contains the imaging slice. After a delay encompassing one R-R interval, an image is acquired. Because the inversion pulse eliminates signal, moving protons from outside the tagged region flow into the imaging slice and produce signal. The pulse sequence is repeated a second time within the same breath hold, but this time the inversion pulse is applied to the entire torso (nonselective tag), and thus there are no untagged protons that can flow into the image plane to produce signal. Protons from stationary structures within the image plane are inverted in both images. The selective tag and nonselective tag images are then subtracted, producing a map of signal intensity representing protons that have moved into the image slice in the interval between the tag and image acquisition (one R-R interval). Figure 1 shows an image of pulmonary blood flow obtained by this methodology. Note that this technique may contain components of both pulmonary arterial and venous blood flow.

Each subject was placed in a head-first, supine position into the scanner, after a four-element phased-array torso coil was positioned over the thorax. The superior end of the coil was placed directly under the chin to minimize magnetic field drop off in the apical sections of the lung. It is not necessary to determine the inhomogeneity map of the phased-array torso

coil because it is constant within a subject over the experiment. Data were obtained during an 8- to 10-s breath hold at functional residual capacity in a 15-mm-thick image slice in the coronal plane in the posterior one-third of the lung. The posterior edge of the descending aorta was located in the scout image and used as a reference point for selecting the coronal slice to be imaged. The images were acquired during a breath hold to avoid misregistration due to motion artifacts. Each slice was 40 × 40 cm and imaged at a resolution of 256 × 128 pixels; therefore voxels of ~1.5 × 3 × 15 mm (~70 mm^3) were visualized. Three measures were obtained at each time point. All sequence parameters were kept within U.S. Food and Drug Administration guidelines for clinical magnetic resonance examinations.

Data analysis

The signal intensity for each voxel in the images was determined for each image and each time point by using MAT-LAB (The MathWorks, Natick, MA). To minimize motion artifacts from the heart and aorta (see Fig. 1*C*), only data from the right lung were analyzed. Relative dispersion is a global index of the heterogeneity of blood flow and was calculated as the standard deviation divided by the mean signal intensity of all voxels within the right lung. A higher value for relative dispersion indicates a more heterogeneously distributed system (8). The relative dispersion values from the pulmonary blood flow images taken in triplicate were averaged for each time point.

Statistics

Data were analyzed by using an ANOVA (Statview 4.1, SAS Institute, Cary, NC) with one repeated measure (time posttilt, seven levels: $t = 0, 70, 80, 90, 100, 110, 120$) (7). To compare the changes pre- and post-head-down tilt to those observed lying supine in the scanner, a second ANOVA was performed with one repeated measure, pre- and post-intervention, and one randomized group (lying supine or head-down tilt). Where overall significance was determined, Student-Newman-Keuls post hoc ANOVA testing was applied to determine where significant differences occurred. Significance was accepted at $P < 0.05$, two tailed. All data are shown as means ± SE.

RESULTS

Six male subjects and one female subject participated in *experiment 1* (the control study) ($n = 7$). Subject descriptive characteristics for *experiment 1* are shown in Table 1. Relative dispersion values for 7 subjects measured at baseline before and after lying supine in the scanner for 1 h are shown in Fig. 2*A*. After 1 h of lying supine in the scanner, the relative dispersion increased from 0.96 ± 0.08 to 1.07 ± 0.09, a 12% change ($P < 0.05$). The relative dispersion for three of the seven subjects increased only slightly (8% or less), and 1 subject's relative dispersion decreased. Lying supine for 1 h did not have a significant effect on heart rate (64 ± 7 vs. 62 ± 6 beats/min) or oxygen saturation (98 ± 1 vs. 98 ± 1%). The average signal-to-noise ratio for all images for both experiments was 35 ± 3.

Four male subjects and three female subjects participated in *experiment 2* (the head-down tilt intervention). Subject descriptive characteristics for *experiment 2* are shown in Table 2. Pretilt, pulmonary blood flow heterogeneity as measured by the relative dispersion was 0.84 ± 0.06, which is within the range of healthy subjects as previously reported by our group (12) (Fig. 3*A*). At 10 min after 1 h of 30° head-down tilt, the relative dispersion was increased to 1.04 ± 0.07 ($P < 0.05$) and remained elevated compared with pretilt values ($P < 0.05$) for all remaining time points (mean relative dispersion increased by 29% as a result of tilt, or 0.25 relative dispersion units). Relative dispersion increased and subsequently decreased over the hour of posttilt measurements, the time course of which varied from subject to subject. Mean relative dispersion reached a maximum value of 1.13 ± 0.10 at 40

min posttilt (Fig. 3*A*). This change from pretilt was significantly different than the change in mean relative dispersion at the end of the 1 h period of lying supine in the scanner ($P < 0.05$), and all of the subjects showed an increase in relative dispersion at 40 min posttilt (Fig. 2*B*).

One hour of 30° head-down tilt resulted in a 12% decrease in heart rate from 75 ± 5 beats/min pretilt to 66 ± 2 beats/min post tilt ($P < 0.05$); however, there was no further decrease in heart rate over the duration of posttilt measurements (Fig. 3*B*). Arterial oxygen saturation did not change (Fig. 3*C*) over the duration of the experiment.

DISCUSSION

The principal result of this study is that the heterogeneity of pulmonary blood flow was increased by 1 h of 30° head-down tilt to a greater extent than by lying supine for 1 h. Heart rate decreased after 1 h of 30° head-down tilt; however, it did not change as a result of lying supine for 1 h. Oxygen saturation was not affected by either intervention.

Effects of head-down tilt on heterogeneity of pulmonary blood flow

The baseline values of relative dispersion for the healthy subjects in both experiments, ranging from 21 to 48 yr of age, are consistent with data previously obtained in our laboratory (15). Pulmonary blood flow heterogeneity, as measured by relative dispersion, increases linearly with age in healthy, nonsmoking subjects. Specifically, relative dispersion is ~0.85 in healthy, nonsmoking subjects that are 20 yr old, and increases by ~0.07 with each decade of age above 20 yr old (e.g., for a healthy subject 50 yr of age, relative dispersion would be expected to be 0.85 + 0.21, or ~1.05).

The heterogeneity of pulmonary blood flow was increased by 12% as a result of lying supine in the scanner for 1 h, and this increase was more than doubled to a 29% increase after a 1-h period of 30° head-down tilt ($P < 0.05$). Thus 1 h of 30° head-down tilt resulted in a greater heterogeneity of pulmonary blood flow than what would be expected from the postural changes associated with lying supine in the scanner alone. In addition, relative dispersion remained elevated for 1 h after tilt ($P < 0.05$). Although the mechanisms responsible for the elevation in relative dispersion could be quite different, the magnitude of the increase in relative dispersion seen with 1 h of 30° head-down tilt was similar to that observed in individuals who have previously experienced high-altitude pulmonary edema and are exposed to acute hypoxia (12).

One possible explanation for an increase in relative dispersion (i.e., blood flow heterogeneity) could be due to collapsed regions of the lung, or atelectasis, as a result of the change in posture, i.e., head-down tilt. To evaluate this, we estimated lung volumes pretilt and posttilt using the total number of voxels within the right lung on the coronal images. The total number of voxels before and during the hour after head-down tilt remained unchanged (4,257 ± 276 voxels pretilt, 4,313 ± 261 voxels posttilt), suggesting that atelectasis cannot explain the changes in relative dispersion evident as a result of tilt.

Relative dispersion is a robust and reliable index of blood flow heterogeneity (8, 12). Reliability data for relative dispersion of pulmonary blood flow obtained in 45 healthy nonsmoking volunteers by using the same methodology as this study have been presented previously by our group (12). Relative dispersion was determined from a coronal image of blood flow obtained 10 min apart within a single testing session. Results showed that there was a positive, linear correlation between the two measures of relative dispersion for the group of 45 subjects, with a correlation coefficient of 0.96 (12). To date, we have performed

these reliability measurements on a total of 85 healthy nonsmoking subjects and found a correlation coefficient of 0.95.

Effects of head-down tilt on pulmonary physiology

Steep head-down tilt has been shown to increase pulmonary interstitial pressures in rabbits (20, 21) and lung fluid content in rats (24). In humans, steep head-down tilt has been shown to alter ventilatory inhomogeneity, possibly as a result of alterations in acinar geometry due to peribronchial cuffing (23). In this study, 30° head-down tilt resulted in a greater heterogeneity of pulmonary blood flow (relative dispersion) for up to 1 h after tilt. These data collectively would be consistent with the idea that steep head-down tilt increases pulmonary capillary pressures and fluid efflux in the lung, possibly leading to the development of interstitial pulmonary edema and a more heterogeneous distribution of ventilation and perfusion. Because inhomogeneity of ventilation and perfusion is the hallmark of many lung diseases, the imaging techniques used in this study may have important clinical applications. Furthermore, head-down tilt may prove to be a useful model for studying the development of interstitial pulmonary edema and how it transitions to alveolar edema.

Head-down tilt results in a redistribution of body fluids from caudal to cephalic regions of the body, increasing blood volume within the pulmonary vasculature. This increase in blood volume could activate arterial baroreceptors (18, 27) and thus be responsible for the decrease in heart rate that was evident posttilt (Fig. 3*B*). This is also supported by the fact that heart rate did not change as a result of lying supine in the scanner for 1 h but was decreased for all time points posttilt (Fig. 3*B*). To the extent that a lower heart rate may reflect an alteration in cardiac output, this also may affect the regional distribution of pulmonary blood flow.

Fluid redistribution leading to increases in pulmonary blood volume not only can affect heart rate, but also venous return and stroke volume. Unfortunately, in our study we do not know the effect of 1 h of 30° head-down tilt on venous return and stroke volume. However, a study similar to the head-down tilt protocol we used in this study found that 45 min of 30° head-down tilt did not significantly alter stroke volume, heart rate, or cardiac output (34). The intervention, however, resulted in a decreased peak velocity of pulmonary arterial blood flow, perhaps suggesting that pulmonary vascular resistance was elevated as a result of tilt. Heart rate was decreased in our study posttilt; thus cardiac output could be reduced if there was residual activation of arterial baroreceptors. Because in healthy subjects the lungs receive all of the cardiac output, there are other more direct methods that can be used to measure total perfusion, such as soluble gas uptake (2) or the direct Fick technique. Although the techniques used in this study can be used for absolute quantification of perfusion, we were interested in using the measurement technique to measure changes in the heterogeneity of blood flow. Therefore, we did not perform absolute quantification of perfusion in this study and cannot comment on changes in overall pulmonary blood flow.

Effects of interstitial pulmonary edema on pulmonary physiology

The redistribution of body fluids from caudal to cephalic regions of the body during head-down tilt increases pulmonary capillary pressures (11) and thoracic fluid volume, which would likely cause a concomitant increase in total lung water (intravascular and extravascular) and perhaps lead to interstitial pulmonary edema. Such fluid efflux has previously been suggested to alter ventilatory inhomogeneity, presumably via changes in the geometry of distal airways (23). As lung water accumulates, peribronchial and/or perivascular cuffing may result and thus compromise airway and blood vessel caliber. In particular, accumulation of fluid in an airway or blood vessel wall may isolate it from the

surrounding lung parenchyma, reducing parenchymal tethering and thus reducing the caliber of the airway or blood vessel, and resulting in a more heterogeneous distribution (increased relative dispersion) of pulmonary blood flow (32).

It is widely believed that interstitial pulmonary edema can occur during exercise, as a consequence of the effect of exposure of the pulmonary capillary bed to high pressures and flows (6, 13, 22, 25). An increase in pulmonary extravascular water has been described in trained cyclists at 90 min postexercise with use of a different MRI technique (19). Exercise-induced interstitial edema has also been suggested to alter the distribution of blood flow and ventilation distribution in the lung (3, 6, 9, 14, 30). In keeping with this idea, ventilation-perfusion inequality with exercise is accentuated by hypoxia (10, 31) and reduced by breathing 100% oxygen (6), both of which alter pulmonary vascular pressures. The abnormality persists during recovery from heavy exercise, even after ventilation and cardiac output have returned to normal (30).

Limitations

There are some limitations regarding our methodology that must be considered when evaluating our results. We imaged a single, two-dimensional coronal slice of the lung, and therefore changes in pulmonary blood flow heterogeneity could have been more or less pronounced in regions of the lung that were not imaged. Still, we have no reason to believe that blood flow heterogeneity would be different in nonimaged regions of the lung in a healthy subject. Although it is possible to acquire data from the entire lung from multiple adjacent slices, these data take more than 10 min to acquire. Because we were interested in the time course of any changes that occurred as a result of tilt, a limited scan was performed to avoid the possibility of conditions changing over the course of a more complete evaluation. Also because we did not image the entire lung, we could not obtain a direct measure of total lung volume. Instead we used the number of voxels within the lung field to determine whether there was a loss of lung volume due to atelectasis posttilt. Although this measure did not change, it is possible that small atelectatic changes in other regions of the lung that were not evaluated could have contributed to our findings of increased spatial heterogeneity. The lack of any changes in arterial oxygen saturation over the course of the study suggests that any such regions of atelectasis are likely very small.

ASL-FAIRER provides an image of any tagged blood that has moved into the imaging slice during the delay between tagging and image acquisition, whether it is arterial or venous blood. Therefore, the blood flow image contains components of both pulmonary arterial and venous blood flow, the effect of which is at present unclear. Nevertheless, this limitation applies to all images (whether before or after head-down tilt) and therefore does not change the interpretation that blood flow heterogeneity was altered (i.e., increased) after 1 h of 30 degree head-down tilt.

It was not possible to obtain relative dispersion measurements during head-down tilt because of physical constraints of the scanner's horizontal bore. Thus we could only obtain images before and after tilt with the subject in the supine position. We have obtained reliability data for relative dispersion of pulmonary blood flow obtained in 10 healthy nonsmoking volunteers by using the same methodology as this study. Relative dispersion was determined from two coronal images of blood flow obtained within a 1- to 2-wk interval. There was a highly significant relationship between the two measures with a correlation coefficient (r) of 0.97 and a variability of ~4.3%. Thus this measure of pulmonary blood flow is highly reliable, and the changes we observed are not likely because of positional changes from the subject being moved for the head-down tilt intervention.

Although we speculate that the mechanism most likely responsible for our findings is that steep head-down tilt increases pulmonary capillary pressures and fluid efflux in the lung, we did not measure pulmonary capillary pressures or lung water content directly.

In summary, 1 h of steep (30°) head-down tilt resulted in an increase in pulmonary blood flow heterogeneity as measured by relative dispersion (standard deviation/mean signal intensity) for up to 1 h after tilt. Lying supine in the scanner also caused an increase in relative dispersion, but this was much smaller than that which occurred after tilt. Lung volume (based on the total number of voxels in the right lung field) at the time of imaging before and after tilt remained unchanged, suggesting that elevations in relative dispersion posttilt were not due to atelectasis. Thus we conclude that 1 h of 30° head-down tilt increases the heterogeneity of pulmonary blood flow. We speculate that the mechanism most likely responsible for our findings is that head-down tilt increases pulmonary capillary pressures and fluid efflux in the lung, resulting in a higher relative dispersion and greater heterogeneity of pulmonary blood flow.

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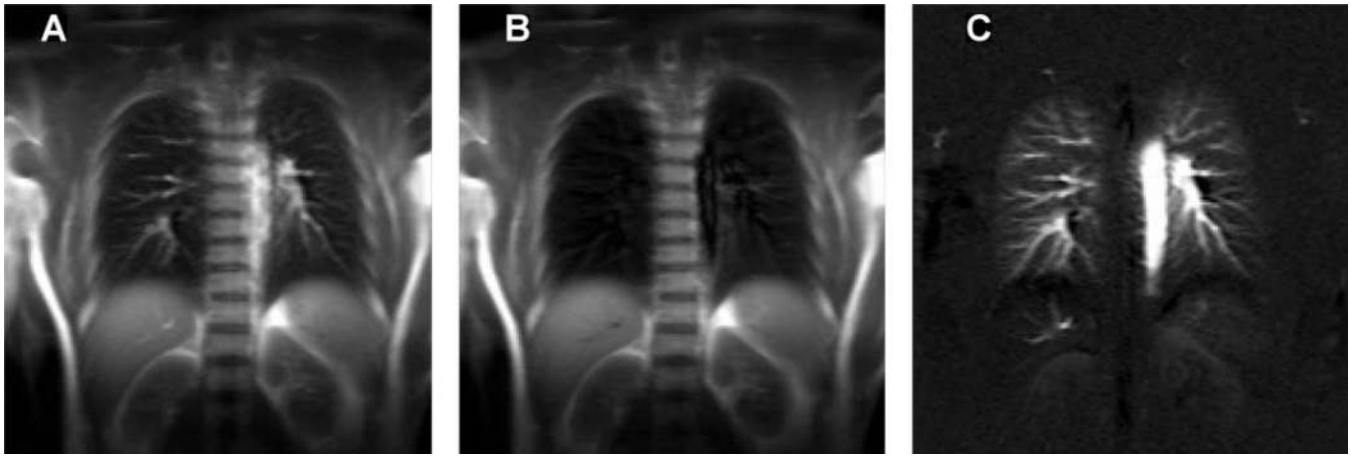

Fig. 1.

Creation of pulmonary blood flow image from ASL-FAIRER. Subtraction of selective and nonselective images (*A* and *B*, respectively) generates a blood flow-weighted map (*C*). The pulmonary vessels are identified as bright, tubular structures, many of which are seen to bifurcate to successively smaller vessels. A diffuse blush throughout the lungs is seen from flow within the microvasculature. The image is presented with the standard radiographic convention: the patient's left is to the right side of the image. The large, vertically oriented vessel just to the (patient's) left of midline is the aorta.

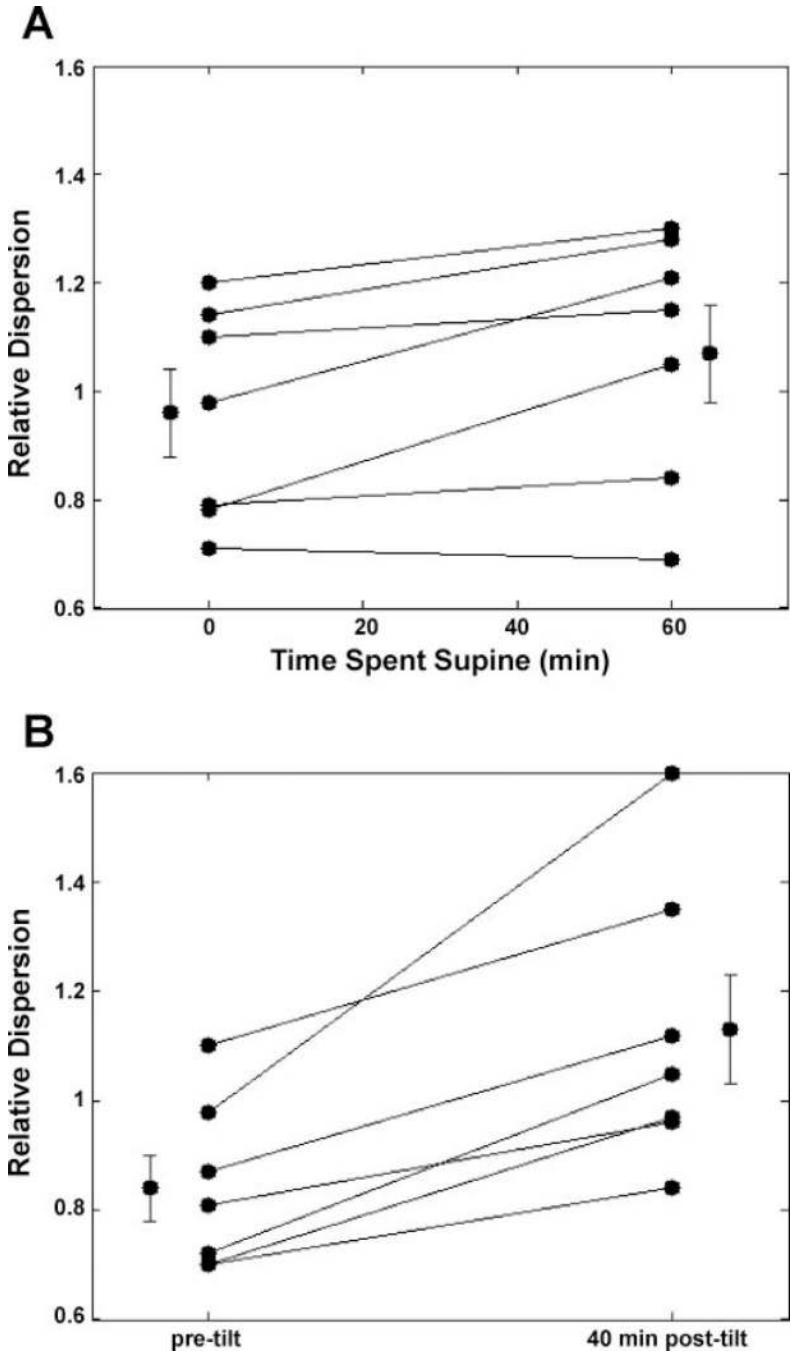

Fig. 2.

A: relative dispersion at baseline and after lying supine in the scanner for 1 h for the 7 healthy subjects shown in Table 1. Relative dispersion was either unaffected or slightly increased as a result of lying supine in the scanner for 1 h. *B*: relative dispersion at baseline (pretilt) and at 40 min posttilt for the 7 healthy subjects shown in Table 2. Note that every subject's relative dispersion was increased at 40 min posttilt compared with baseline.

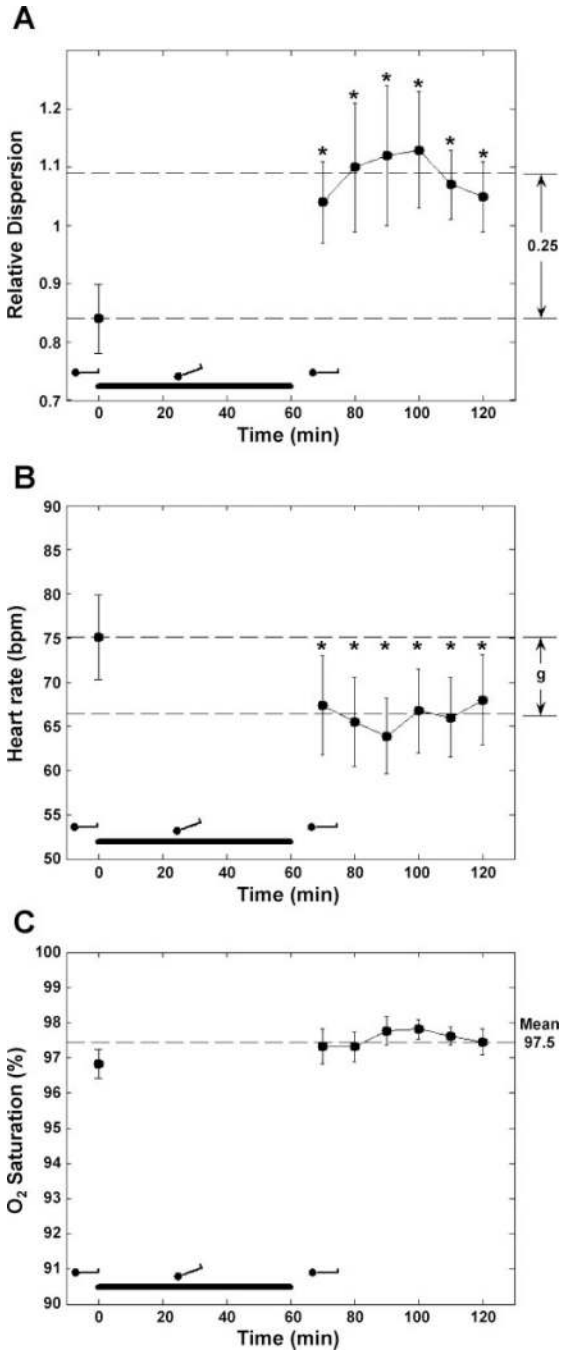

Fig. 3.

Relative dispersion (standard deviation/mean signal intensity; *A*), heart rate (*B*), and oxygen saturation (*C*) at baseline and at 10-min intervals up to 1 h after a 1-h period of 30° head-down tilt for the 7 healthy subjects shown in Table 2. Relative dispersion was elevated after a 1-h period of 30° head-down tilt and remained elevated for 1 h after tilt. Heart rate was decreased during the 1 h of measurements made posttilt, whereas arterial oxygen saturation showed no significant change. *Significantly different from pretilt value, $P < 0.05$

Table 1

Subject descriptive characteristics for experiment 1 in which subjects lay supine for 1 h

Subject	Age, yr	Sex	Height, cm	Weight, kg	FVC, %	FEV_1, %	FEV_1/FVC, %
1	37	M	175	71	112	105	101
2	39	M	183	78	127	105	83
3	42	F	165	52	106	112	107
4	34	M	178	79	116	94	81
5	25	M	170	60	113	107	95
6	48	M	185	109	100	108	108
7	35	M	188	107	101	96	95
Means ± SE	37 ± 3		178 ± 3	79 ± 8	111 ± 4	104 ± 2	96 ± 4

M, male; F, female; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; %, percentage of predicted values.

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Table 2

Subject descriptive characteristics for experiment 2 in which subjects underwent 1 h of 30° head-down tilt

Subject	Age, yr	Sex	Height, cm	Weight, kg	FVC, % pred	FEV_1, % pred	FEV_1/FVC, % pred
1	21	F	175	75	125	120	95
2	26	M	173	67	96	98	102
3	26	M	175	77	108	109	101
4	21	F	165	64	95	96	100
5	35	F	163	68	107	107	100
6	48	M	185	109	100	108	108
7	35	M	188	107	101	96	95
Mean ± SE	30 ± 4		175 ± 4	81 ± 7	105 ± 4	105 ± 3	100 ± 2

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