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BLOOD OXYGEN CHANGES INDUCED BY FORWARD (+G_x) ACCELERATION

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BLOOD OXYGEN CHANGES INDUCED BY FORWARD ($+G_x$) ACCELERATION

NATALIO BANCHERO
LUCILLE CRONIN
A. CLARK NOLAN
EARL H. WOOD

FOREWORD

The studies on which this report is based were accomplished in the cardiovascular and human centrifuge laboratories of the Mayo Graduate School of Medicine (University of Minnesota), Rochester, Minnesota under the direction of Dr. Earl H. Wood under Air Force Contract No. AF 33(657)-8899, NASA Interagency Transfer Fund R-43, Project No.7222, "Biophysics of Flight".

Dr. Alvin S. Hyde, Multienvironment Division, Biophysics Laboratory, Aerospace Medical Research Laboratories, was the contract monitor.

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Dr. Natalio Banchero, First Assistant, Mayo Graduate School of Medicine, was supported during the period of this study by an International Postdoctoral Research Fellowship from the National Institutes of Health. Mr. Ralph E. Sturm also assisted in these studies. Work on this project started on April 1, 1962 and continued until November 1, 1964.

These studies were made possible by the unstinting co-operation of many of our technical and professional colleagues in the Section of Physiology and Section of Engineering, among which Donald Hegland, Julius Zarins, Willis Van Norman, Robert Hansen, William Hoffman, Ronnie Wilcox and Mrs. Jean Frank are deserving of particular mention.

This technical report has been reviewed and is approved.

J. W. HEIM, PhD
Technical Director
Biophysics Laboratory

ABSTRACT

Six dogs under morphine-pentobarbital anesthesia were exposed to forward accelerations of 2, 4 and 6G for one minute and 6G for three minutes while in the horizontal, 15° head-up and 15° head-down positions breathing room air. Exposures to 6G were repeated breathing 99.6% oxygen. Oxygen saturation and opacity at 800 millimicrons of blood were recorded continuously by cuvette oximeters. Pulmonary arterial-venous shunting was estimated from blood oxygen saturations.

No systematic changes in femoral artery oxygen saturation occurred at 2G while a small average decrease was observed at 4G (4%). Decreases occurred at 6G averaging 11 (5-17) per cent at the end of the 60-second exposure. Return to control (1G) values was nearly complete 50 seconds after the exposure. Oxygen inhalation delayed but did not prevent the desaturation. These decreases are believed due to pulmonary arterial-venous shunting. The average increase in pulmonary arterial-venous shunt over 1G values estimated at the end of 60-second exposures to 6G when breathing air, was 17 (11-31) per cent. Values for shunts at 6G, when breathing oxygen, were similar.

The oxygen saturation of mixed venous blood decreased during the exposures to 2, 4 and 6G, minimum values occurring about 20 seconds after return to 1G. The average decrease with exposures to 6G was 17 (8-22) per cent, recovery from which was not complete at 50 seconds after the exposure.

Changes in blood oxygen saturation were not systematically affected by the presumed differences in intrathoracic blood volume and position of the diaphragm associated with the 15° head-up or 15° head-down positions.

Respiratory amplitude and rate were variable but usually increased during the exposure and recovery periods. The degree of the resultant increase in ventilation tended to be directly related to the magnitude of acceleration.

Progressive decreases in transmission of infrared light of arterial and mixed venous blood were observed indicating hemoconcentration presumably due to loss of fluid from dependent capillaries during the exposures.

The average dorsal-ventral diameter of the lungs of the dogs selected for this study was similar to that of adult human beings. It is of interest that the decreases in oxygen saturation of arterial blood observed in these dogs during exposure to acceleration were also similar to those observed previously in healthy unanesthetized human beings. This similarity supports the belief that the arterial desaturation is caused by the occurrence of dependent pulmonary atelectasis, the degree of which would, on a physical basis, be predicted to be directly related to the level of acceleration and the dorsal-ventral diameter of the lungs.

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BLOOD OXYGEN CHANGES INDUCED BY FORWARD ($+G_x$) ACCELERATION *

Decreases in arterial blood oxygen saturation have been reported in man and in dogs exposed to forward ($+G_x$) acceleration (9,10,11,14,15,16, 19,20,22,25 and 28). Evidence suggests that these changes are related to atelectasis in the dependent regions of the lungs and magnification of the inequalities of the ventilation-perfusion ratios normally seen in different parts of the lungs in the 1G environment.

Since the maximum dorsal-ventral dimension of the thorax of adult mongrel dogs is similar to that of human beings, it was suspected that the hydrostatic effects of forward acceleration on the pulmonary circulation of the two species would be similar.

The present study was undertaken to further analyze the changes in arterial blood oxygen saturation during forward acceleration with the dog in the supine position, perpendicular to the vector of acceleration and with the body tilted 15 degrees head-up and 15 degrees head-down.

MATERIAL AND METHODS

Six mongrel dogs (20 to 22 kg. weight) anesthetized with morphine sulfate (5 mg./kg.) and sodium pentobarbital (25 mg./kg.) were studied in the 15-feet radius centrifuge of the Mayo Clinic during exposures to forward acceleration ($+G_x$) with the animal restrained in the supine position on a special support. Anesthesia was maintained during the experiment by additional administration of sodium pentobarbital as needed. Exposures to 2, 4 and 6G for periods of 60 seconds were carried out during spontaneous breathing of room air with the dog in the horizontal position and with the body tilted 15 degrees head-up and 15 degrees head-down. Repeated exposures in the horizontal position were performed in only five dogs. Figure 1 depicts the average sequence of exposures carried out in the first 8 hours of the experiment.

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* Presented at the Aerospace Medical Association Meeting in Bal Harbor, Miami, Florida, May 13, 1964.

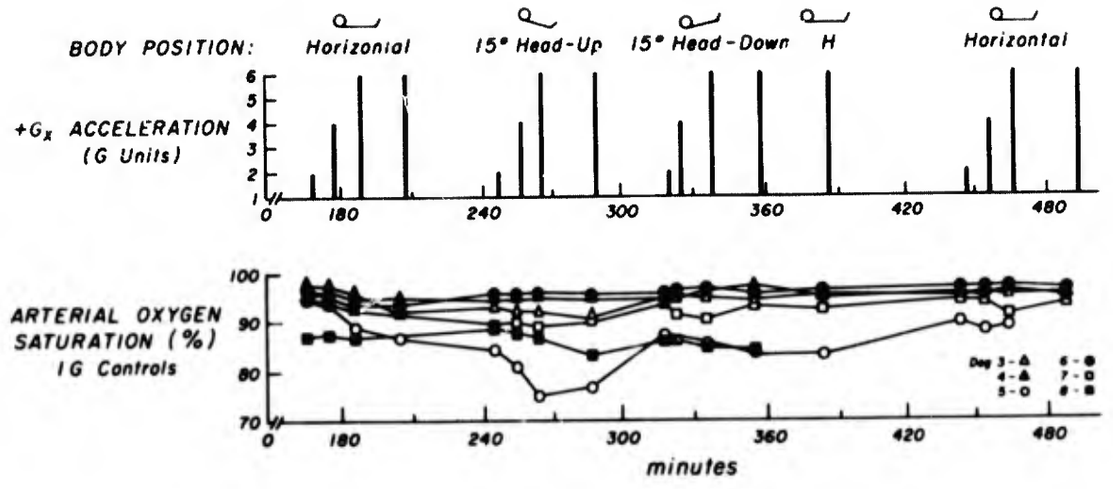

Figure 1 Average temporal sequence of exposures of 6 dogs to forward ($+G_x$) acceleration for studies of cardiorespiratory effects. Zero time was taken as the time when the dog was placed in the centrifuge cockpit in the supine position. The first series of horizontal exposures to 2, 4 and 6G were performed after about 170 minutes and were repeated 5 hours later. Exposures with the body tilted 15 degrees head-up and 15 degrees head-down were performed in between the two series in the horizontal position. A single horizontal exposure to 6G (indicated as H) was done in 5 dogs at an average interval of 28 minutes after the last exposure with the body tilted head-down. Additional exposures to 6G with the dog breathing oxygen were performed in 3 animals later in the experiment and are not shown in this figure.

In the lower panel, individual mean control values of arterial blood oxygen saturation, measured at 1G just prior to each exposure, are plotted. Note that animals maintained a fairly constant arterial oxygen saturation throughout the prolonged experiment. In this and in the next figure each dog is identified by a symbol.

In three dogs, additional horizontal exposures to 6G were repeated for three-minute periods while breathing room air and 99.6 per cent oxygen. The average total cumulative exposure to the 3 levels of acceleration studied is shown in Figure 2 with values for cardiac output determined by the dye dilution technic at intervals during the experiment when the dogs were at 1G.

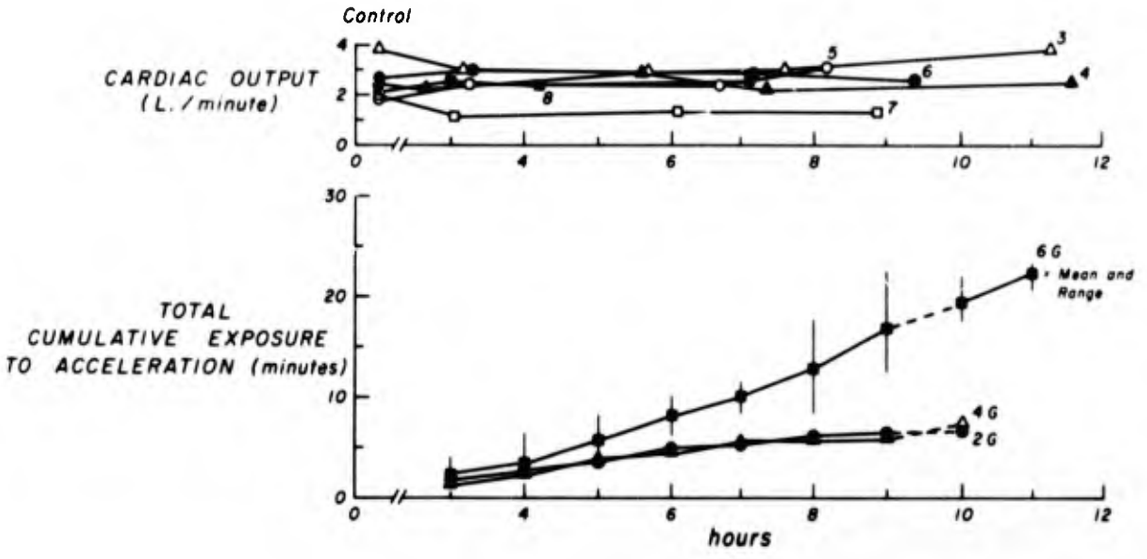

Figure 2 Cardiac output values measured at 1G and average total cumulative time of exposure to forward acceleration of 2, 4 and 6G in 6 dogs. As in Figure 1, zero time was taken when the dog was set up in the centrifuge cockpit. The values after 9 hours, in the lower panel, are the mean values from the three dogs studied for these extended periods.

A #6 Lehman catheter (length: 60 cm., I.D.: 0.8 mm.) was inserted percutaneously in the left jugular vein and advanced until its tip was in the main pulmonary artery. A nylon catheter (length: 21 cm., I.D.: 0.7 mm.) was introduced in the femoral artery through a #17 needle. Cuvette oximeters were placed in series between these two catheters and their respective strain gauges (Statham P23D) and connected by three-way stopcocks and polyethylene tubing to a double barrel Harvard withdrawal-infusion syringe. A flexible vinyl tube was introduced in the trachea, its pressure cuff inflated and the variations in airway pressure at mouth level recorded by means of a Statham strain gauge (PM5d).

Oxygen saturation, by single and double scale methods, and opacity at 800 millimicrons (hemoglobin concentration) of arterial and mixed venous blood were continuously measured by cuvette oximeters and recorded on a photokymographic camera (23,24). The rate of blood sampling from femoral and pulmonary arteries for these continuous spectrophotometric analyses was 9.9 ml./minute, before, during and after each exposure. At this rate of flow, the time delay in the dead space in the cuvette-catheter assemblies were approximately 6 seconds for the pulmonary artery assembly and 2 seconds

for the femoral system. The cuvettes were calibrated against manometric analyses by the method of Van Slyke and Neill of samples withdrawn from the femoral and pulmonary arteries while the animal was breathing air and 99.6 per cent oxygen.

The degree of pulmonary arterial-venous shunting was estimated from simultaneous oxygen saturation values of arterial and mixed venous blood as per cent of systemic blood flow by the formula (3):

$$Q_s = \frac{C_c O_2 - C_a O_2}{C_c O_2 - C_{\bar{v}} O_2} \; 100$$

Q_s = per cent of systemic blood flow through shunt

$C_c O_2$ = concentration of oxygen in pulmonary end capillary blood

$C_a O_2$ = concentration of oxygen in arterial blood

$C_{\bar{v}} O_2$ = concentration of oxygen in mixed venous blood

The rate of rotation of the centrifuge (rpm) was measured by a tachometer previously calibrated with a synchronous motor. The angle of tilting of the centrifuge cockpit (outward swing during the period of acceleration) was measured by a potentiometer attached to the cockpit axle. A Statham strain gauge A3-10-350 accelerometer was used to estimate the acceleration. The electrical output of these instruments was continuously recorded on the photokymographic camera.

Additional catheters to measure intravascular pressures were introduced percutaneously via the external jugular veins and advanced to the right and left atria respectively and a third catheter advanced from the femoral artery to the aorta. Two #4 catheters were inserted into the potential right pleural space to measure intrapleural pressure (26,27). Data from these measurements are not reported here. A necropsy was performed after each experiment.

RESULTS

Animals maintained a fairly constant physiological status throughout the prolonged experiment as judged from the measurements of arterial blood oxygen saturation, intracardiac pressures and cardiac output measured at 1G at periodic intervals during the course of the experiment (Figures 1 and 2).

Horizontal Exposures In the first series of exposures (Figure 3), mean control value of femoral arterial oxygen saturation was 92.8 per cent, individual values ranging from 86.2 to 97 per cent.

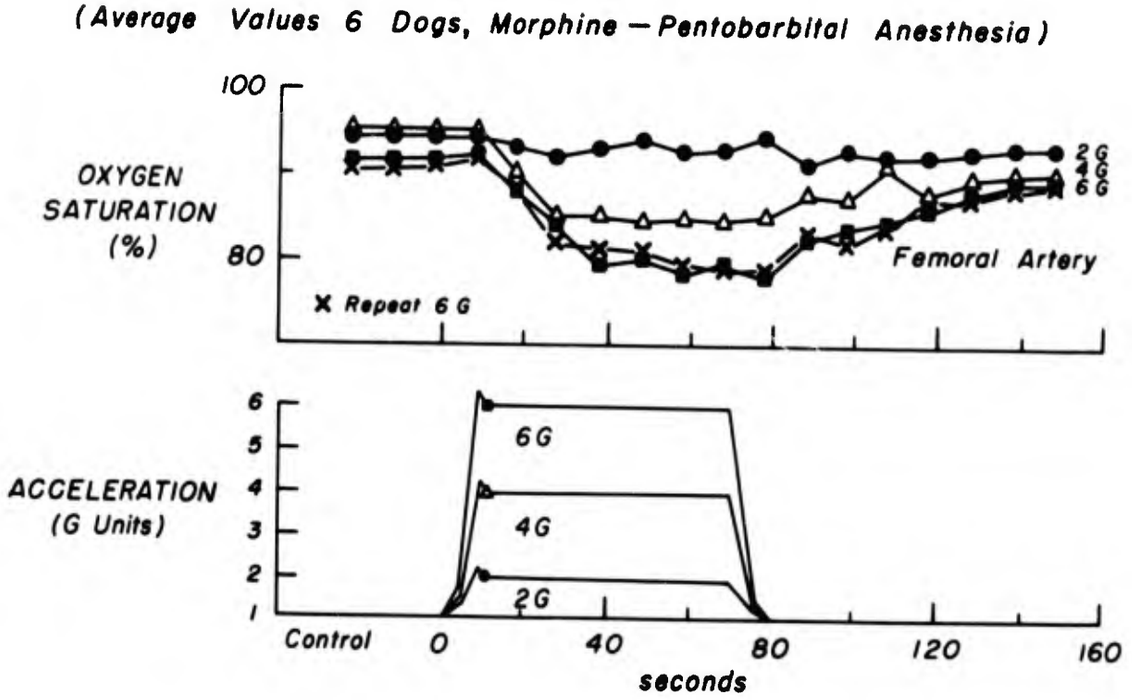

Figure 3 Average changes in arterial blood oxygen saturation in 6 dogs during 60-second exposures to forward acceleration at 2, 4 and 6G with the dog in the horizontal position. Animals were spontaneously breathing room air. Control values were determined for 20 seconds. Zero seconds indicates the onset of centrifuge rotation in this and the following figures. The time lag of 2 seconds due to the dead space of the catheter-cuvette sampling system was corrected for in this and subsequent plots.

No systematic changes in arterial oxygen saturation occurred at 2G. After the attainment of the plateau level at 4G, decreases in arterial saturation uniformly occurred which progressed down to an average value of 85 per cent. Upon termination of the exposure, there was a slow return to control values which was not complete 70 seconds after the centrifuge was stopped. At 6G, decreases in arterial oxygen saturation began about 10 seconds after the onset of the plateau level and rapidly reached mean saturation values of nearly 78 per cent. Repeated exposures to 6G, carried out 19 minutes later, showed similar control values and average changes. In both instances recovery began immediately when exposure stopped but it was not complete in 70 seconds.

Approximately 5 hours later, the horizontal series of exposures to forward acceleration were repeated in 5 dogs. The average changes in oxygen saturation of arterial and mixed venous blood are shown in Figure 4.

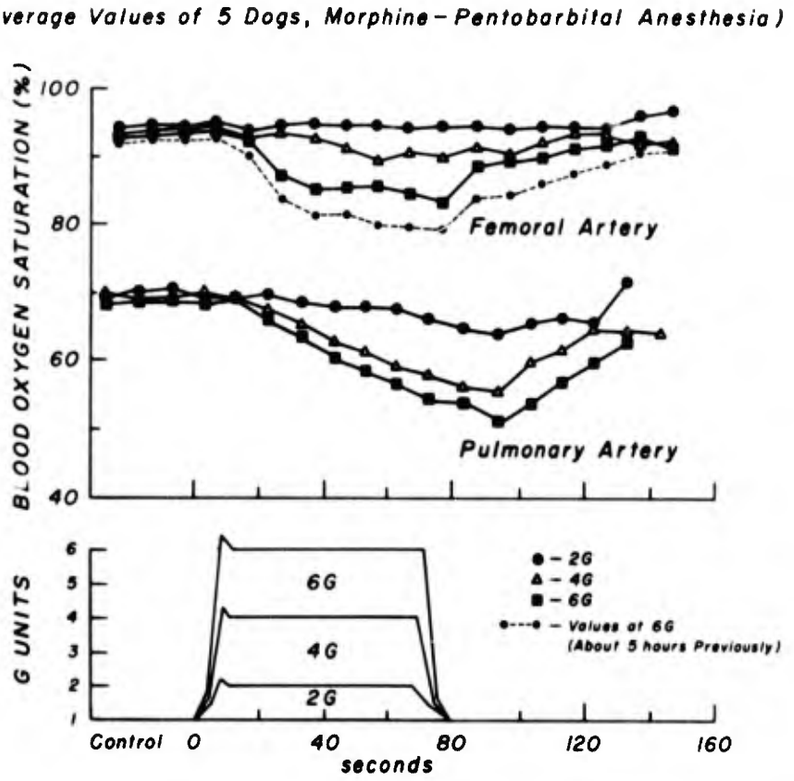

Figure 4 Average changes in oxygen saturation of arterial and mixed venous blood of 5 dogs during forward ($+G_x$) exposures to 2, 4 and 6G when spontaneously breathing room air. For comparative purposes, the average decreases in femoral arterial oxygen saturation (dashed line) observed during the initial exposures to 6G approximately 5 hours earlier in the experiment are shown. The sampling lag time of 2 seconds in the femoral artery and 6 seconds in the pulmonary artery catheter-cuvette systems were corrected for in these plots.

Mean control value of femoral artery saturation was 93.9 per cent with individual range from 88 to 96.9 per cent. As observed before, no systematic changes in femoral artery oxygen saturation occurred at 2G.

Only small decreases occurred at 4G and values of about 90 per cent occurred toward the end of the exposure. At 6G, the average decrease in arterial oxygen saturation was less than the average decrease observed in the same five dogs during the first series of exposures five hours previously. Recovery of arterial saturation at 4 and 6G began immediately after the exposure stopped and was nearly complete in 50 seconds.

The oxygen saturation of mixed venous blood showed a mean control value at 1G of 69.5 per cent, individual values ranging from 51.3 to 80.1 per cent. Decreases in pulmonary artery oxygen saturation occurred during exposures to 2, 4 and 6G, reaching minimal mean values of 56 per cent at 4G and 51 per cent at 6G, about 15 seconds after return to 1G. These decreases were significantly greater than the corresponding decreases in femoral artery oxygen saturation. Recovery to control values after 4 and 6G exposures was not complete 40 seconds after the centrifuge was stopped.

Respiratory amplitude, recorded as airway inspiratory pressure changes, increased during acceleration but no consistent pattern was evident. A transient increase in respiratory rate was usually observed after the onset of the exposure, however, this increase rate was not always maintained during the acceleration period. Some dogs showed a decreased respiratory rate which was usually associated with an increase in the amplitude of respiration (Figure 5).

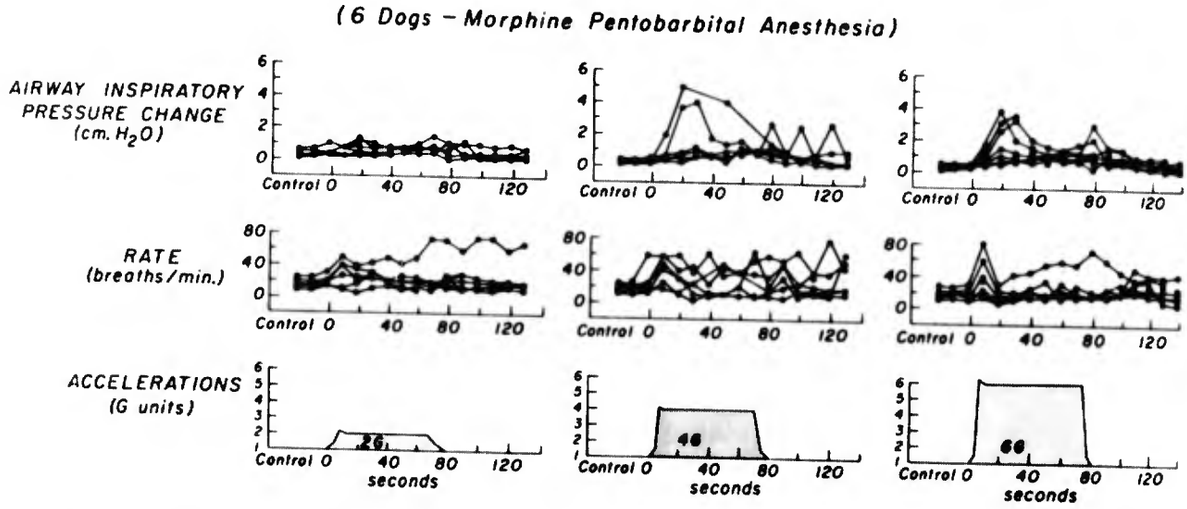

Figure 5 Amplitude and rate of respiration during exposure to forward ($+G_x$) acceleration of 2, 4 and 6G in the horizontal position.

The effects of forward ($+G_x$) acceleration of 5.9G maintained for three minutes were studied in three dogs while breathing room air and 99.6 per cent oxygen. When breathing room air, values of arterial blood oxygen saturation tended to be slightly lower toward the end of the 3-minute period of acceleration than at 60 seconds after the onset of the exposure. Recording of arterial blood oxygen saturation showed return to control values 80 seconds after the exposure. Decreases in oxygen saturation of mixed venous blood followed the changes of arterial blood but were more marked (Figure 6, upper panel). The administration of oxygen delayed but did not prevent the arterial oxygen desaturation and its magnitude was reduced. Changes in mixed venous blood followed these occurring in the femoral artery, the absolute values being higher than the corresponding values when breathing room air (Figure 6, lower panel).

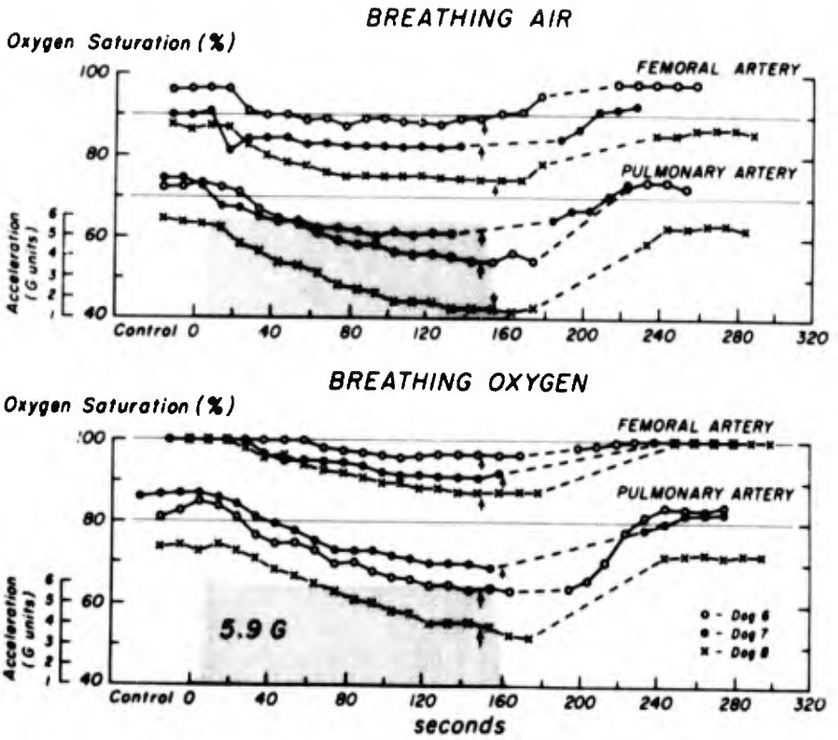

Figure 6 Effects of 3-minute exposures to forward ($+G_x$) acceleration on the oxygen saturation of arterial and mixed venous blood of 3 dogs when breathing room air (upper panel) and 99.6 per cent oxygen (lower panel). Arrows indicate when the acceleration stopped. Since it was necessary to reinfuse the blood after 30 ml. were sampled, measurements were interrupted temporarily during the recovery period, as indicated by dashed lines.

In an attempt to quantitate the changes occurring at different levels of acceleration, pulmonary arterial-venous shunting was estimated from blood oxygen saturation values. No systematic changes in pulmonary arterial-venous shunt were observed at 2G. During 4 and 6G exposures, the estimated pulmonary arterial-venous shunt increased immediately after the onset of the exposure and the magnitude of shunt increased as the level of acceleration increased, being higher at 6G. Shunts were about 15 per cent higher during the plateau level at 6G when compared with the control values at 1G (Figure 7).

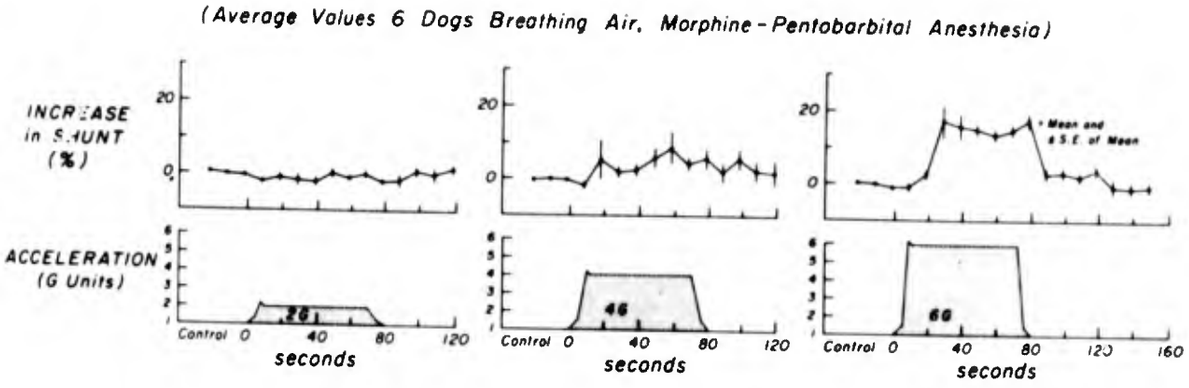

Figure 7 Average per cent changes and standard error of the mean of estimated pulmonary arterial-venous shunt during forward ($+G_x$) exposures to 2, 4 and 6G in six dogs in the horizontal position spontaneously breathing room air.

The increase in per cent of shunt at different levels of acceleration was independent of the estimated pulmonary arterial-venous shunt at 1G. Figure 8 shows the lack of correlation between the control pulmonary arterial-venous shunt at 1G plotted against the increase in shunt after 60 seconds of exposure.

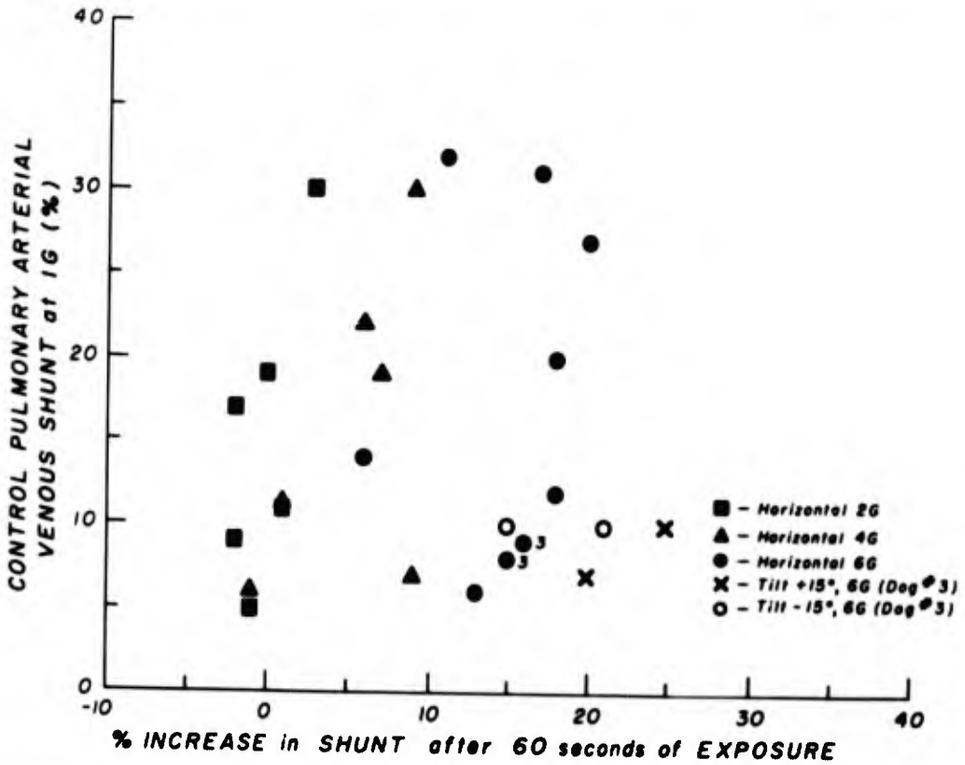

Figure 8 Relation of increase in pulmonary arterial-venous shunt during forward ($+G_x$) acceleration to the control shunt at 1G. Comparative values of estimated shunt when the body was horizontal and when tilted 15 degrees head-up and head-down are depicted for dog 3. Note the lack of correlation between these two variables.

When pulmonary arterial-venous shunt was estimated during the three-minute exposure to 5.9G breathing room air, there was a 15 per cent increase in magnitude of the estimated shunt in the three dogs studied, which was independent of the estimated shunt at 1G (control). When shunts were estimated during oxygen inhalation, absolute values of pulmonary arterial-venous shunts, similar to those on room air, were obtained during the exposures (Figure 9).

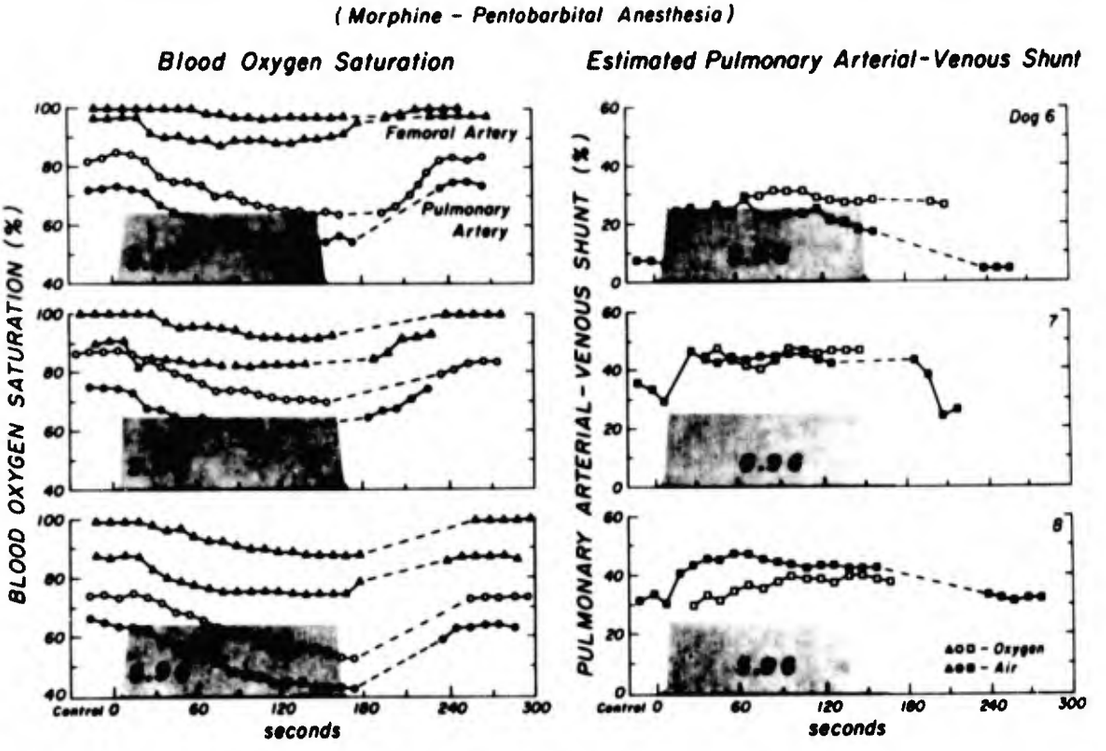

Figure 9 Estimated pulmonary arterial-venous shunt in 3 dogs during exposures to forward ($+G_x$) acceleration when breathing room air or 99.6 per cent oxygen. Note that the increase in magnitude of the estimated shunt of about 15 per cent during the exposure was similar from dog to dog, and the absolute magnitude of the shunt during acceleration was similar when breathing air and 99.6 per cent oxygen.

Effects of Body Tilting No systematic differences were observed in the changes in femoral arterial oxygen saturation produced by exposures to forward ($+G_x$) acceleration of 2, 4 and 6G with the animal horizontal and 15 degrees head-up and 15 degrees head-down positions while breathing room air. During exposures to 6G, similar control and average changes were observed (Figure 10).

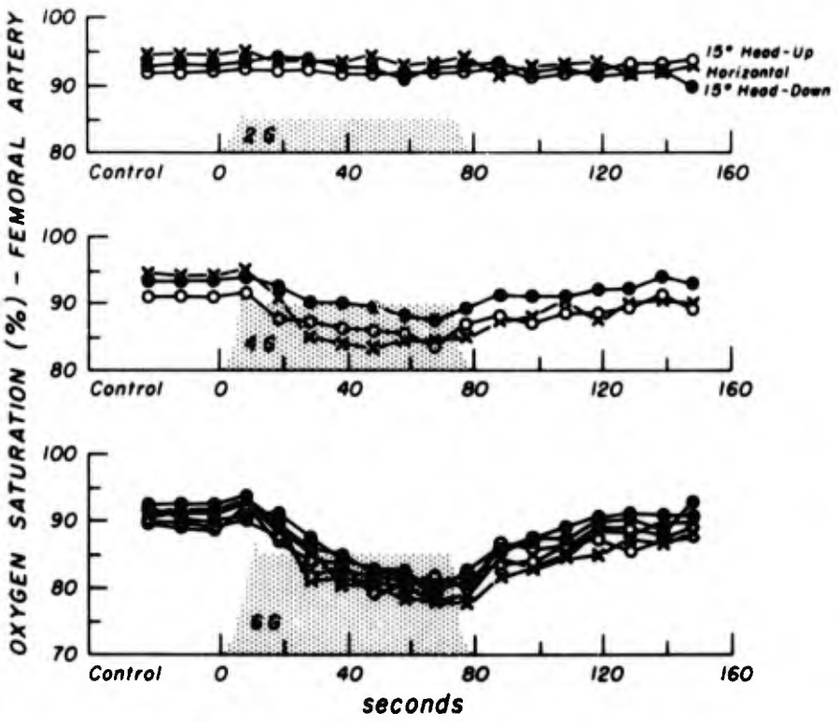

Figure 10 Average changes in oxygen saturation of arterial blood of 6 dogs during forward ($+G_x$) acceleration when the body was horizontal and in the 15 degrees head-up and 15 degrees head-down positions. Values from the horizontal exposures are indicated by crosses, 15 degrees head-up position by open circles and 15 degrees head-down position by closed circles. Exposures to 6G were repeated in each position.

Changes in amplitude and rate of respiration and estimated pulmonary arterial-venous shunt were not systematically different in the three different positions as shown in dog 3, Figure 11.

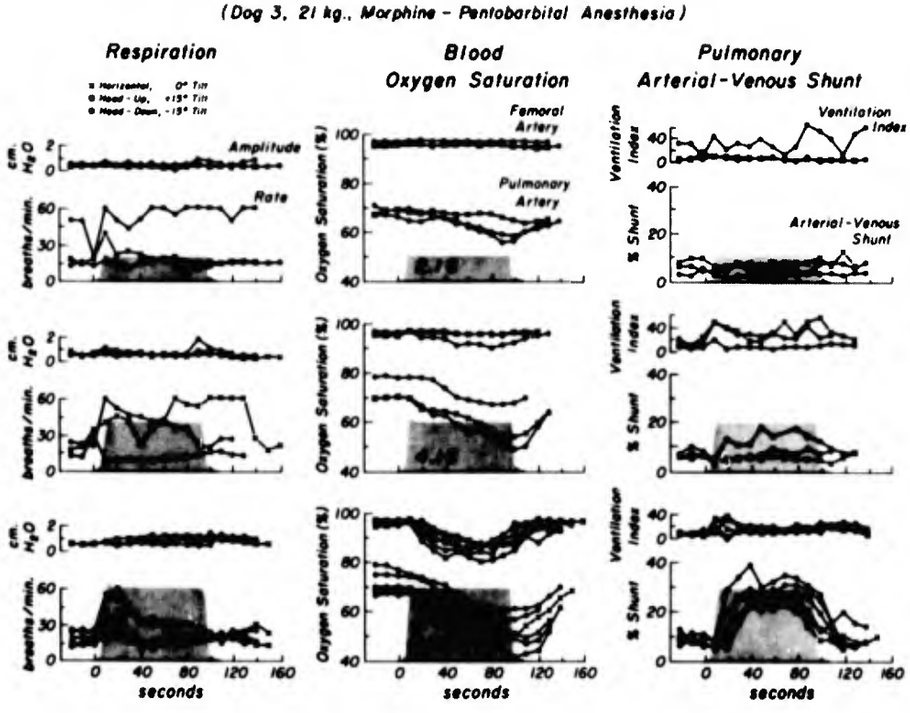

Figure 11 Comparison of the effects of forward acceleration when in the horizontal and 15 degrees head-up and 15 degrees head-down positions on the rate and depth of respiration, oxygen saturation of arterial and mixed venous blood and estimated pulmonary arterial-venous shunt during forward ($+G_x$) acceleration in dog 3. Note that during the exposures to 6G, there was no clearcut difference between the estimated degree of pulmonary arterial-venous shunting associated with different degrees of body tilting. The ventilation index is the product of the rate and amplitude (i.e., respiratory variation in endotracheal pressure) of each breath.

A progressive decrease in transmission of the infrared light of arterial and mixed venous blood indicating hemoconcentration was observed during acceleration presumably due to the loss of fluid from dependent capillary beds during exposures. Changes were related to the magnitude of acceleration and were higher at 6G. Infrared transmission values returned to 1G control values during the five to ten minute intervals between exposures (Figure 12).

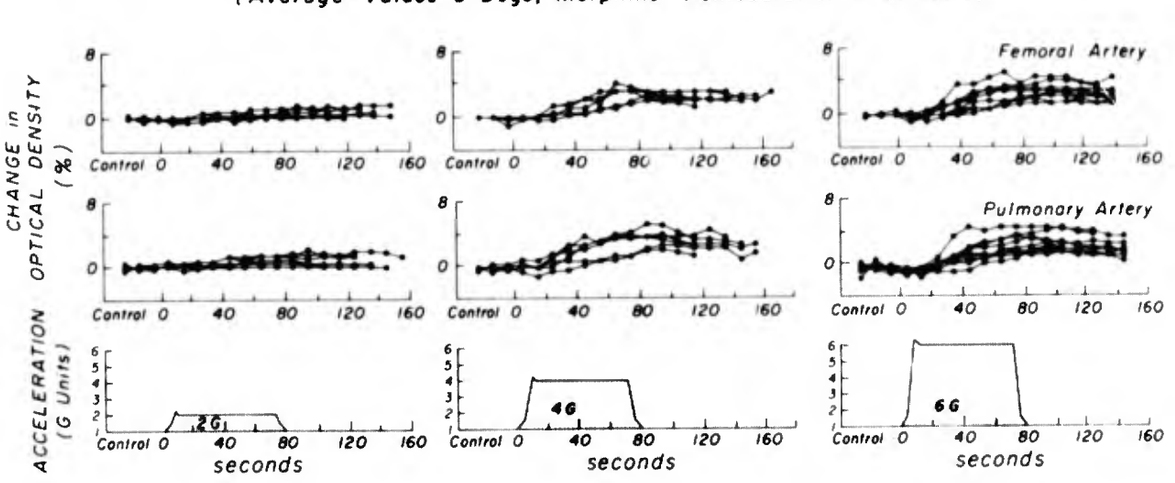

Figure 12 Changes in optical density of arterial and mixed venous blood at 800 millimicrons (hemoglobin concentration) during forward ($+G_x$) acceleration.

DISCUSSION

In the normal 1G environment, the inequalities of the ventilation-blood flow ratios within the lungs are mainly related to the effect of gravity acting upon the blood contained in the pulmonary vascular tree (specific gravity about 1) and the air contained in the tracheo-broncheo-alveolar tree (specific gravity virtually zero) (12,17,29). During acceleration, the increased gravitational force produces a proportional increment in the effective weight of the fluid (blood) and gas (air) contents of the lungs, proportional to their specific gravity thus magnifying the inequalities of ventilation-perfusion ratios normally present (15,22,28).

Edmundowicz et al have found that in dogs studied at 1G in the supine horizontal position the pressure in the intrapleural space is higher in the dependent, dorsal portions of the thorax as compared to that found ventrally (4,5). Recent studies in this laboratory have confirmed that pressures are higher in the most dependent sites of the pleural space both in supine and prone body positions (18). During forward acceleration, the increase in the weight of the thoracic contents, proportional to their specific gravity, produces a much higher pressure in the dependent dorsal sites of the thorax, and at 6G positive pressure values of about 20 centimeters of water were recorded while highly negative pressure occurred ventrally (about -30 centimeters of water) (18,26).

These pressure imbalances occurring in superior and dependent regions of the thorax between blood and air contained in the lungs and within the pleural space are probably responsible for the overdistention of alveoli in the superior, ventral parts and collapse of alveoli in the most dependent dorsal parts of the lungs (28). Anatomical and histological evidence of this type of changes has been found in dogs after exposures to forward acceleration. Steiner and Mueller (22) found "marked overexpansion of the alveoli and striking absence of red cells" in the anterior, ventral portions and "large areas totally devoid of patent alveoli and massive atelectasis" in the dorsal parts in the lungs of one dog killed 30 seconds after 10-minute exposure to 14 $+G_x$. Wood et al (28) found similar changes in dogs exposed to 2, 4 and 6 $+G_x$.

Since values of arterial oxygen saturation recovered within a few minutes after exposures to forward acceleration, it is probable that the anatomical changes seen in the pulmonary parenchyma of dogs killed at 1G subsequent to acceleration do not reflect adequately the severe degree of atelectasis that occurs during the actual exposures. Also, it is necessary to bear in mind that pulmonary atelectasis may be observed in post mortem examinations of anesthetized dogs maintained in one body position and not submitted to any kind of gravitational force other than the normal 1G (13).

In radiological studies, Hershgold (6,7) and Nolan et al (15) have pointed out increase in radio-opacity, suggesting atelectasis, in the most dorsal dependent parts of the lung silhouette of healthy subjects exposed to forward acceleration. Increase in radiolucency in the ventral parts was interpreted as a decrease in vascularity and blood content. It is thought that decreases in arterial oxygen saturation observed during forward acceleration are probably due to magnification of the inequalities in the ventilation-blood flow ratios, with pulmonary arterial-venous shunting occurring in the most dependent parts of the lungs which are perfused but possibly unventilated while the superior portions of the lungs are well ventilated but poorly perfused.

There is some evidence suggesting the production of edema during forward acceleration. Progressive and rapid increase of the optical density of the blood measured at 800 millimicrons indicates an increase in blood hemoglobin concentration which disappears 5 to 10 minutes after the exposures. During the recovery period, right and left atrial pressures have been found lower than in the control period (28). Both observations are probably related to loss of fluid from the vascular system due to pressure imbalances in the capillary system between the coloidal-osmotic and hydrostatic pressures during acceleration. Edema at pulmonary level could be a contributing factor for the collapse of alveoli and deficient oxygenation of blood; however, the magnitude of its contribution remains unknown.

Apparently no impairment in pulmonary ventilation was produced in the different conditions studied. Animals responded by increasing the amplitude and/or the rate of respiration to achieve a better ventilation during exposures to forward acceleration. This observation is in agreement with data reported by Zechman et al (30) and Cherniack et al (2) showing that pulmonary minute volume and alveolar ventilation increased progressively with acceleration.

The fact that absolute values of estimated pulmonary arterial-venous shunt observed during forward accelerations to 6G were similar when breathing room air and 99.6 per cent oxygen indicates that the right-to-left shunt cannot be explained by hypoventilation, impaired diffusion or unevenness of gas distribution but must be due to capillary blood passing through non-ventilated atelectatic alveoli presumably in the most dependent parts of the lungs. It has been reported that cardiac output is maintained practically unchanged or slightly increased during forward acceleration (8,9,25). Therefore, pulmonary capillary blood flow is assumed to be unaltered.

In spite of the prolonged time of exposure, no evidence of deterioration in the physiologic status of the animals was observed during the experiments. Small differences were observed comparing the horizontal exposures carried out five hours apart with slightly higher saturations in the second series. The progressive decrease in arterial oxygen saturation described by Barr et al (1) during repeated exposures to positive (headward) acceleration has not been observed in this nor in previous series studied at the Mayo Clinic.

Acceleration produces a redistribution of blood volume with pooling of blood in the most dependent parts of the body. Only at the level of hydrostatic indifference, blood pressure and blood flow will remain the same. Likewise, the intrathoracic blood volume is also changed by the hydrostatic effects of increased gravitational force. In addition, the pulmonary circulation and the pulmonary ventilation may be affected by the increased weight of the abdominal contents during acceleration. When comparing average values of femoral arterial oxygen saturation in animals in the horizontal position with values in the 15 degrees head-up and 15 degrees head-down positions, no systematic differences were obtained. This fact tends to indicate that, in dogs, the deleterious effects of forward acceleration on the pulmonary circulation are not essentially modified by tilting the body within the range $\pm$15 degrees and the presumed differences in intrathoracic blood volume associated with these positions.

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13 ABSTRACT Six dogs under morphine-pentobarbital anesthesia were exposed to forward accelerations of 2, 4, and 6 G for one minute and 6 G for three minutes while in the horizontal, 15° head-up and 15° head-down positions breathing room air. Exposures to 6 G were repeated breathing 99.6% oxygen. Oxygen saturation and opacity at 800 mmμ of blood were recorded continuously by cuvette oximeters. Pulmonary arterial-venous shunting was estimated from blood oxygen saturations. No systematic changes in femoral artery oxygen saturation occurred at 2 G while a small average decrease was observed at 4 G (4%). Decreases occurred at 6 G averaging 11 (5-17)% at the end of the 60-second exposure. Return to control (1 G) values was nearly complete 50 seconds after the exposure. Oxygen inhalation delayed but did not prevent the desaturation. These decreases are believed due to pulmonary arterial-venous shunting. The average increase in pulmonary arterial-venous shunt over 1 G values estimated at the end of 60-second exposures to 6 G when breathing air, was 17 (11-31)%. Values for shunts at 6 G, when breathing oxygen, were similar. The oxygen saturation of mixed venous blood decreased during the exposures to 2, 4, and 6 G, minimum values occurring about 20 seconds after return to 1 G. The average decrease with exposures to 6 G was 17 (8-22)%, recovery from which was not complete at 50 seconds after the exposure. Changes in blood oxygen saturation were not systematically affected by the presumed differences in intrathoracic blood volume and position of the diaphragm associated with the 15° head-up or 15° head-down positions.

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