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Mayo Association

STUDIES OF THE EFFECTS OF ACCELERATION ON CARDIOVASCULAR AND RESPIRATORY DYNAMICS

This is a progress report on the work which has been accomplished to date in approaching the problem of determining the effect which gravitational and inertial forces play in the regional distribution of pulmonary blood flow and the associated severe disturbances in ventilation/perfusion ratios observed during exposures to high levels of acceleration. This study is a direct extension of the investigations concerning the effect of gravitational and inertial forces on intrathoracic pressure relationships and the resulting severe degrees of arterial hypoxemia and instances of physical disruption of pulmonary parenchyma encountered at high levels of acceleration. These results have been published or are in press:

1. Rutishauser, W. J., Banchero, N., Tsakiris, A. G., Edmundowicz, A. C. and Wood, E. H.:
   Pleural pressures at dorsal and ventral sites in supine and prone body positions.
   Journal of Applied Physiology 21:1500-1510 (September) 1966.

2. Banchero, N., Rutishauser, W. J., Tsakiris, A. G. and Wood, E. H.:
   Pericardial pressure during transverse acceleration in dogs without thoracotomy.
   Circulation Research 20:65-77 (January) 1967.

3. Rutishauser, W. J., Banchero, N., Tsakiris, A. G., and Wood, E. H.:
   The effect of gravitational and inertial forces on pleural and esophageal pressures.
   Journal of Applied Physiology (in press).

4. Banchero, N., Rutishauser, W., Tsakiris, A. G. and Wood, E. H.:
   Respiratory changes in pleural pressures at different sites and body positions.
   Journal of Applied Physiology (in press).

5. Banchero, N., Schwartz, P. E., and Wood, E. H.:
   Intraesophageal pressure gradient in man.
   Journal of Applied Physiology (in press).

6. Banchero, N., Cronin, L., Rutishauser, W., Tsakiris, A. G., and Wood, E. H.:
   Effects of transverse acceleration on blood oxygen saturation.
   Journal of Applied Physiology (in press).

7. Banchero, N., Schwartz, P., Tsakiris, A. G. and Wood, E. H.:
   Pleural and esophageal pressures in the upright body position.
   Journal of Applied Physiology (submitted).

Introduction and Review of Literature

It has long been argued, and in recent years verified, that there is an inhomogeneity of blood flow per unit volume of lung parenchyma. It is further argued that this inhomogeneity is in some way systematically related to gravitational forces.

The study of the distribution of pulmonary blood flow has, by and large, been intimately associated with studies of the distribution of ventilation. Rahn in 1956 (1) reported, on the basis of the derived ventilation perfusion ratios determined simultaneously from the various lobes of the dog lung and the ventilation distribution as determined from the distribution of radioactive dust particles inhaled, that the perfusion to the upper lobes of a dog's lung is reduced and the perfusion to the lower lobes increased when the animal is moved from the supine to the upright position. Further, he found that the flow under this latter condition is 2.5 times greater in the lower than upper lobes.

Since the advent of radioactive gases, the distribution of perfusion has been more directly approachable. Studies have been generally accomplished on either the isolated lung preparation or on humans. Dollery (2), and later West and Dollery (3) reported the use of radioactive CO_2 and CO to determine regional blood flow. The gas is inhaled, the breath held, and the disappearance of the radioactive gas into the circulation was used as an index of the perfusion at the region of measurement.

The results of these experiments gave evidence in support of perfusion inequalities between apex and base of human lungs in subjects in the erect seated position.

In 1962 Ball (4) reported on the use of $Xenon^{133}$ as a methodology for studying regional ventilation and perfusion. More recently Millic-Emili (5) has extended this methodology and has shown the correlation of the human data to the isolated preparation of West.

In 1962 Permutt (6) presented a mechanical model based on the Starling resistor

as analogous to certain aspects of the pulmonary circulation. This model, he has termed the "vascular waterfall". About the same time Bannister and Torrance (7) put forth a similar model which they termed the "sluice" mechanism.

Figure 1 shows the Permutt "vascular waterfall" model. The hydrostatic blood column is representative of the pulmonary arterial pressure (PAP), the water column around the flexible, collapsible tubing (pulmonary capillaries) represents the alveolar pressure (AP) and the hydrostatic blood column on the opposite side represents the pulmonary venous pressure (PVP).

Flow through the pulmonary capillary (flexible collapsible tube) under the conditions in which the PAP > PVP > AP, as represented in the left-hand drawing, is proportional to the difference between PAP and PVP, and the changes in the AP have little effect on flow. When PAP > AP > PVP, then in order for flow to occur, the collapsible tube must be open at least part of the time. Under these conditions, flow is similar to a water flowing over a precipice, hence the name, "vascular waterfall", and, in this case as shown in the right-hand figure, flow is proportional to the difference between PAP - AP with PVP having little effect on the flow.

Figure 2 shows a flow ($\dot{Q}$) vs. (PVP) curve for this model. Here PVP was varied while AP and PAP were kept constant. Note that flow increased linearly from zero when PVP equaled zero to a maximum when PVP equaled AP, and that beyond this point, further decreases in PVP did not change the flow.

A test for this model was afforded by the isolated lobe of a dog lung, as shown in Figure 3. The co-ordinates are the same as in Figure 2. Here, with PAP and AP constant, PVP was varied and flow measured. Note the general agreement with the model except for maximum value, the hysteresis loop, and the decrease in flow as PVP is made more negative.

On the basis of this agreement, Permutt suggested that flow is a function of the absolute values of the three pressures, PAP, PVP, and AP.

John West, in a series of articles (8,9), supported this theory with experiments on the isolated dog lung. On the basis of his results, the following model of the

lung is presented as shown in Figure 4. West defines three flow zones on the basis of height from the base to apex of the lung. Zone 1 has no flow as PAP is less than AP and, therefore, no flow can occur since the driving force is zero. Zone 2 shows a linear increase in flow with distance, as shown in the right-hand side of the figure. In this zone, flow is proportional to the difference between PAP and AP. Note also this zone has the largest change in flow per unit distance in the gravitational field. Zone 3 also shows a linear increase in flow from the point where PVP = AP to the base of the lung. Flow here is supposed to be proportional to the difference between PAP and PVP, but this is constant in Zone 3. Why then, is there this increase in flow? West explains this by stating that the flow is proportional to transmural pressure and, since both PAP and PVP are increasing with distance toward base by the hydrostatic column above them, so is the transmural pressure; hence in Zone 3, flow is proportional to PAP - PVP plus the transmural pressure gradient. Note that the postulated change in flow per unit distance in Zone 3 is small.

Most of the data with $Xenon^{133}$ obtained on erect humans showed a linear decrease in flow with height up the lung from base to apex. This does not agree with what West and Permutt had shown since there should be an inflection point at PVP = AP, and the slope should go through zero flow at PAP = AP. Millic-Emili, using Xenon, showed that the linear relationship was due to the fact that alveolar size was not taken into consideration (10). If the scans were adjusted to total lung capacity, where a more uniform alveolar size is to be expected, then the inflection points in the perfusion curves occurred and these were approximately at the levels where pulmonary venous pressure and alveolar pressure were estimated to be equal.

Results

The following presents, in chronological sequence, our efforts in this problem area using intact dogs as our experimental animal. The experimental approach was designed so that the Mayo centrifuge could be used as a tool to amplify the effects

of gravitational forces, thus providing an opportunity to obtain additional information concerning the question "to what extent does regional pulmonary flow depend upon inertial forces".

We have used several methodologies to approach this problem. The first is the use of the Mayo videodensitometer system, as shown in Figure 5 (11).

The system is composed of an x-ray source (A), which is beamed through the chest of the animal. Those x-rays not absorbed by the tissue are detected on the face of an image-intensifier tube (B), which is coupled by a lens system to the image-orthicon television camera (C).

The electrical output of the camera is fed via the slip ring assembly of the centrifuge to a recording console (shown in Figure 6) where the x-ray image of the thorax is recorded at 60 frames/second. Analysis of this recorded tape is done by the use of the videodensitometer, which is an electronic device and allows the operator to select a rectangular area of the image and record the changes in density which occur at that site as a function of time. This system allows: adjustment of the position of the sampling window to any site in the roentgen silhouette of the thorax, replaying of the same tape while recording the densogram from this site, and comparison of the roentgen density-time curves between two or as many sites in the thorax as desired.

Generally, a radio-opaque solution, such as Renovist (a highly iodinated compound), is injected in the right side of the heart and the resulting angiogram of the thoracic circulation is recorded on videotape.

This system has been tested with various depths and concentrations of Renovist, and it has been found that the absorption of x-ray, as recorded on tape and as measured by the densitometer, bears an exponential relationship. In order to use this relationship to measure flow related events in tissue, it is necessary to know the depth of vascular tissue under the window.

When Renovist is injected into the right ventricular outflow tract, it is assumed that this circulatory indicator is completely mixed as it passes into the pulmonary

artery, and therefore, the roentgen density-time curve over various areas of the lung, when corrected logarithmically and adjusted linearily to the anatomical thickness of chest cavity at those sites selected, gives an estimation of the distribution of blood flow per unit volume. Another way of saying this is that, after normalization to make all measured sites of the lung field uniform with respect to depth, the area under the curve is proportional to the flow. Figure 7 shows a series of density-time curves from a prone dog at 1G and during centrifugation at 2, 4, and 6G.

The window was positioned at midchest level and the tape replay of recordings made at 1, 2, 4, and 6G shows curves which have approximately the same area in all cases. There is more than one run at some levels. For the curves on the right, the sampling window was positioned above midchest in the superior portion of the lung field. In this region, a progressive decrease in area of the curves occurred as the level of acceleration increased, indicating less flow to this area as the inertial forces increase.

In Figure 8, the photograph of the television tube face taken from a video-recording shows the x-ray image of a dog's thorax in the right lateral position with the superior lung (left) on the right and the dependent right lung on the left. The top is headward and the bottom is tailward. The square boxes, which are numbered, represent the sampling window positions at 1G at which dilution curves were measured.

Figure 9 shows a plot of the results of the analysis of the Renovist dilution curves recorded from the sampling sites, as shown on the previous figure. The animal is in the right lateral position. The area of each curve is plotted on the ordinate and the distance from the arbitrary zero pressure reference level at mid-chest is plotted on the abscissa. Values from superior lung regions (i.e., the sampling sites above the zero midchest reference level) are indicated by negative values and those from the dependent lung by positive values. The large numerals indicate the level of acceleration at which the measurement was made and the subscripts indicate the respective sampling windows, the position of which are

shown in the previous figure.

The values for cardiac output, mean pulmonary artery pressure, and estimated height above midchest level at which the mean pulmonary artery pressure would be zero at various levels of acceleration, are tabulated.

On hydrostatic principles, the greatest height that a given pressure would be able to support is given by the formula:

$$d = P/g$$

where d = distance in centimeters above the zero reference level
P = mean pulmonary artery pressure
g = gravitational force units (normal g = 1)

The vertical dashed lines indicate the height above the midchest level at which the mean pulmonary artery pressure would be zero at levels of acceleration of 3, 5, and 7G. No contrast media was detected at sites superior to the estimated zero hydrostatic pressure levels at the three levels of increased inertial force environment studied.

It is of interest that, at 1G a decrease in flow occurred in the most dependent portion of the lung field, and above this level, the flow decreased linearly with vertical height.

The second methodology used was embolization of the lungs with radioactive microspheres.

The technic consists of injecting into the right side of the vascular system, usually the right ventricular outflow, a small volume containing spheres which have a diameter of 20-40 microns. These particles are assumed to mix uniformly with the blood as it passes into the pulmonary artery and, therefore, embolize the pulmonary capillary beds in proportion to the volume flow to these beds. The first particles used were aggregated serum albumin tagged with radioactive I^{131}. Reports were appearing in the literature in early 1965 on the use of this method to assess the distribution of pulmonary perfusion. Tauxe and Burchell used this methodology on humans studied in various positions here at the Mayo Clinic. Bryan reported (12)

in that year, the use of this technic in humans who were subjected to centrifugation in the seated position. His series of five subjects was small, one each of whom was studied at 1, 2, 3, and 4G, respectively. The scans of the thoraces of these subjects show a shift in pattern with increasing G, which he concludes fit the Permutt-West model. His assumptions that the direct scan uncorrected for tissue volume under the probe is directly proportional to flow, that intravascular pressures are similar under the various gravitational levels, and that all his subjects had a cardiac output of 6.1 L/m, make his conclusions tenuous.

Attempts to use this technic in dogs in this laboratory were not fully satisfactory. Because of the inhomogeneity in I^{131} albumin aggregates sizes which allow as much as 30% of the isotope to pass through the lung and lodge in other areas of the body, especially the liver where it interferes with lung scan, the albumin tracer was abandoned for one which was more uniform in size and thus would embolize only the lungs and not pass into the systemic circulation. The 3M Corporation produces plastic microspheres of uniform size which can be tagged with radioisotopes suitable for counting. These microspheres were used with a diameter of 35 ± 5 microns. Figure 10 shows the scan data from one dog, which was injected with microspheres while in the prone position at 1G, scanned, then moved to the centrifuge and subjected to 6G during a second injection of microspheres containing the same isotope. The first scan was subtracted from the second to provide the 6G scan.

This figure is an attempt to present a 3-dimensional projection of the scan data. The sagittal plane on which the plots are superimposed represents the lateral x-ray image of the animal's thorax. The vertebral column is to the right, sternum to the left, tail into the screen, and head out of the screen. The uncorrected counts/second are shown as elevated lines in the direction of the inertial forces which point from right to left. The dependent, sternal region is on the left, the superior, vertebral region on the right.

A general shift in the shape of these curves is evident from peaks above midchest line at 1G to peaks which are more central in position and of greater amplitude at 6G. Note that the shapes of the curves are different from one scan line to the next.

This is due to the fact that variable volumes of lung pass under the probe as it scans across the animal at different levels. The correction for the probe efficiency due to collumation and the unknown geometry of the tissue being counted, make this procedure difficult to quantitate. These curves demonstrated that redistribution was occurring and the method should provide information that could be quantitated. In an attempt to quantitate the external scan technic, sections of these lungs were counted and comparisons made with the scan data. The most promising quantitative methodology followed as a consequence of this.

The following data were obtained at 1G since we have not used this method with centrifugation as yet. The current procedure is as follows: Dogs are prepared by percutaneous insertion of catheters into the pulmonary artery, left atrium, right ventricle, and femoral artery for the measurement of vascular pressures and obtaining cardiac output by dye dilution. The animal is placed in the desired position, which is maintained by a molded half-body cast, and vascular airway pressures are recorded during the injection of the microspheres. The lungs are then removed and inflated in a fiberglass cast of the thoracic cavity as shown in Figure 11.

When the lungs have air dried, usually in 48-72 hours, they are removed from the cast and imbedded in urethane foam in the form of a rectangular block. This block is then sliced into uniform 1 centimenter slides by means of a band saw. The slices are counted in unit areas, a representative result from 5 dogs is shown in Figure 12.

Counts per unit volume are plotted on the ordinate and distance in centimeters above (-) and below (+) the hydrostatic zero reference at midchest on the abscissa. RLB and LLB indicate the positions of the lateral borders of the right and left lungs, respectively. Vascular (MPA: mean pulmonary artery; MLA: mean left atrial) and airway (AP) pressures are tabulated on the figure. Note that the values from the dependent lung area are about 2.5 times greater than for the superior lung areas. Note also the change in slope in the lung region where mean left atrial pressure (MLA) was equal to the estimated alveolar pressure (AP). There are several differences between these data and that reported for the isolated lung. In Zone 2, which by

West's criteria is the region superior (-2 to -7 cm) to the region marked MLA = AP, the data show a shallow slope (i.e., small change in flow with vertical height) rather than a steep slope and a steeper slope in Zone 3, in which region mean left atrial pressure exceeds the alveolar pressure. Furthermore, there is a decrease in embolization in the most dependent portion of the lung.

What are possible reasons for these differences? Perhaps the microspheres are not distributed in proportion to regional blood flow but rather in proportion to their difference in density from that of blood. This was discounted because one would expect with the relative high specific gravity of 1.4 for the beads used, that the slope in Zone 2 would be steep rather than the very shallow slope which was observed. To give this a more rigid test, low density beads (specific gravity less than 1/5 that of blood) were used. Figure 13 illustrates the distribution of these 0.2 specific gravity beads. Note the similarity to Figure 12.

The Permutt-West model of the lung is based upon the very non physiological conditions of the isolated lung. In their isolated lung preparation, a constant surrounding pressure is utilized, thereby negating any pleural pressure gradient effect. Recently Milic-Emili (5) has supported the thesis proposed by Wood and coworkers (13-15) that the alveolar size gradient (i.e.,superior alveoli are more expanded than dependent alveoli) is due to the pleural pressure gradient and that ventilation distribution is a consequence of this. West has just reported immersing the isolated lung in a low density medium, the results of which support the Milic-Emili thesis. Further support for this comes from Glazier, who reported in an abstract that intact dog lungs frozen with dry ice and sectioned show alveolar size diminishes with depth from superior lobes. Now, with this evidence, one might postulate a means by which decrease in flow with height, which we have seen in the intact dog, which is less than expected on a simple hydrostatic basis can be explained: (1) Assume that there are two forces acting on the alveoli, one with the tendency to collapse the alveoli which is the inherent elastic and surface tension force, and the other a tendency to hold the alveoli open, which is the negative pleural pressure.

(2) Assume that the collapsing tendency is the same, irrespective of position in the lung (support for this is that the ventilation pattern in the lungs is reversed when body position, in relation to the gravitational vector, is reversed). Therefore, in order to hold the alveoli at a larger volume, the force holding the alveoli open must be greater, or in other words, the tissue pressure must be more negative at superior sites in the thorax where the alveolar size is greater.

But, on hydrostatic principles, the intravascular pressures must be decreasing as the rate of 1 cm H_2O pressure/cm increase in height. If one assumes that the pulmonary tissue gradient is proportional to the pleural pressure gradient which is keeping the lungs expanded, it is evident that the transmural vascular pressure (i.e., intravascular minus tissue pressure) will decrease in proportion to the difference in the pressure gradients (i.e., between vascular and pleural pressure) at different heights in the lung. The decrease in transmural vascular pressure will, therefore, be significantly less than would be predicted by consideration of the hydrostatic vascular effect alone.

To summarize, a model of the pulmonary circulation has been presented in the literature which is based on data from isolated lung preparations and is supported in general by the use of radioactive gas studies in man. The former have the disadvantage that they are non physiological, the latter have the disadvantage that there are large errors in counting and correcting scan data by a geometrical factor to provide a measure of the flow per unit volume of lung. Data are presented herein from intact dogs studied in the right lateral position by a more direct methodology.

The results of these studies indicate that the distribution of regional blood flow to the lungs is not a simple function of the differences in various vascular and airway pressures. It appears highly probable that regional differences in pleural pressures present in the intact thorax, which are weight dependent and and hence determined by the direction and magnitude of resultant inertial and gravitational force vectors (13-15), play an important role as a determinant of blood flow to different regions of the lung.

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Journal of Applied Physiology (in press).

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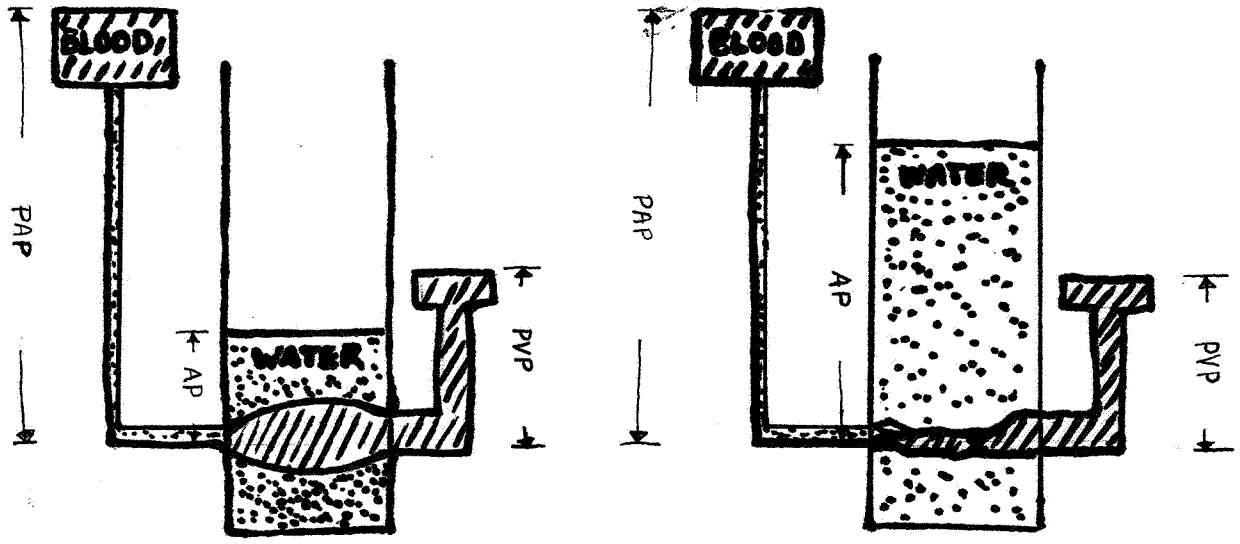
March 31, 1967

EHW:JHR:JF

$PAP > PVP > AP$
$\therefore \dot{Q} \propto PAP - PVP$

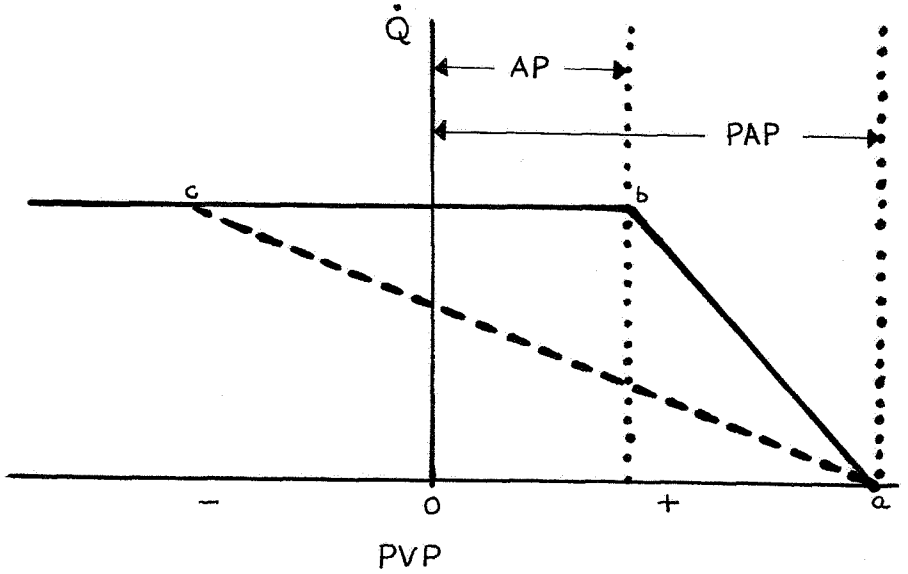
$PAP > AP > PVP$
$\therefore \dot{Q} \propto PAP - AP$



Model of the Lung (after Permutt) 1962
(STARLING RESISTOR)

FIGURE 1

Relationship Between Flow and Pulmonary Venous Pressure (after Permutt) 1962



$\frac{1}{\text{slope } ab}$ = true resistance = $\frac{PAP - PVP}{\dot{Q}}$ PVP > AP

$\frac{1}{\text{slope } ac}$ = measured resistance > true resistance PVP < AP

FIGURE 2

Relationship between Flow and Pulmonary Venous Pressure

(after Permutt) 1962

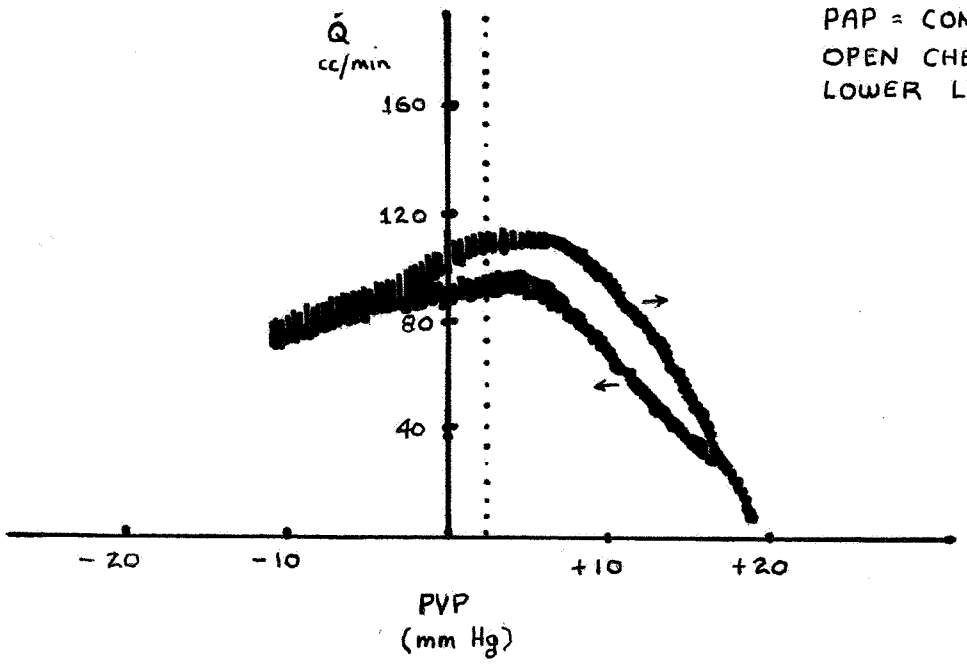
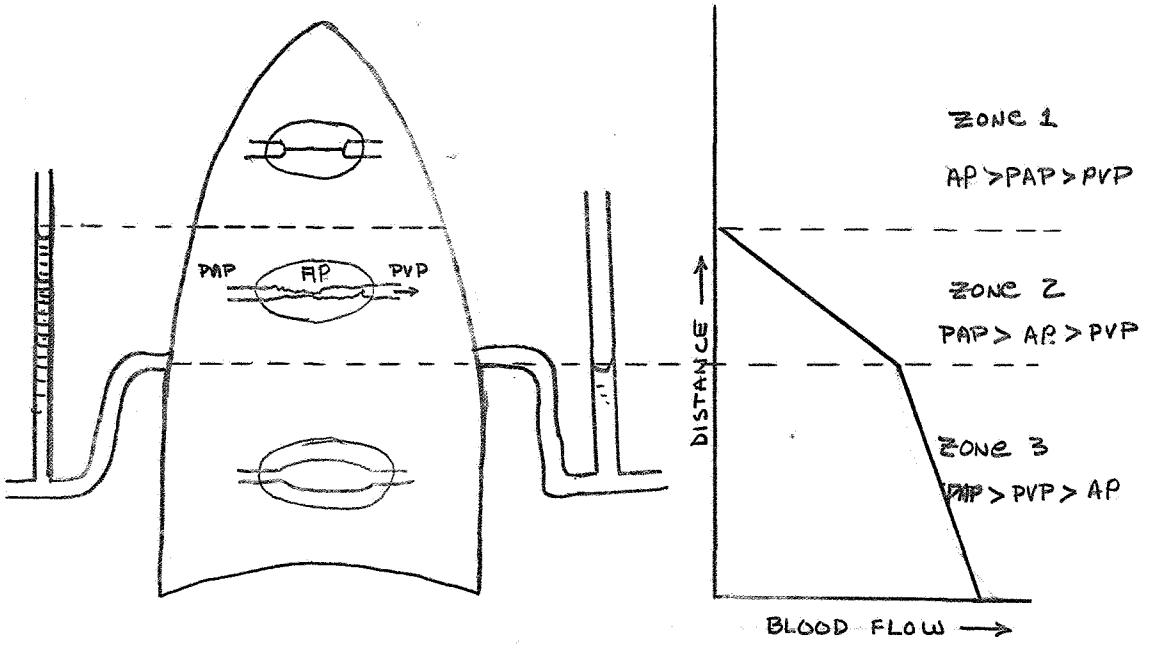


FIGURE 3



MODEL OF THE PULMONARY CIRCULATION INDICATING REGIONAL DIFFERENCES IN BLOOD FLOW.

(modified after West et al 1964)

FIGURE 4

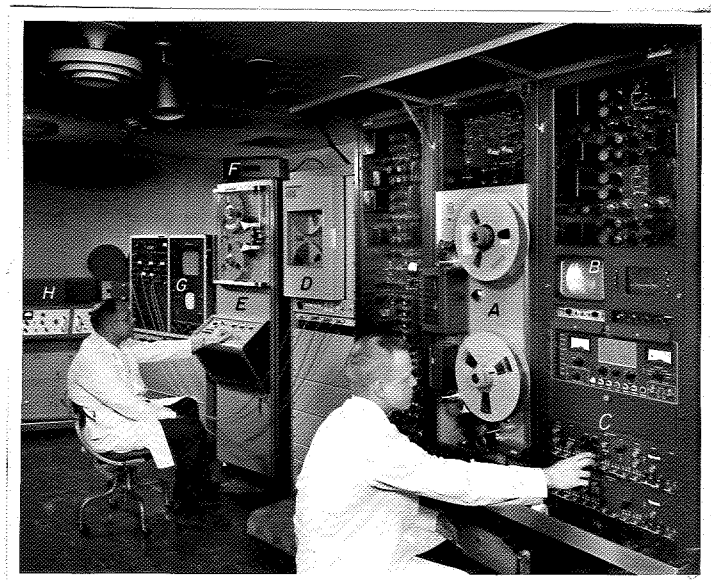


FIGURE 5

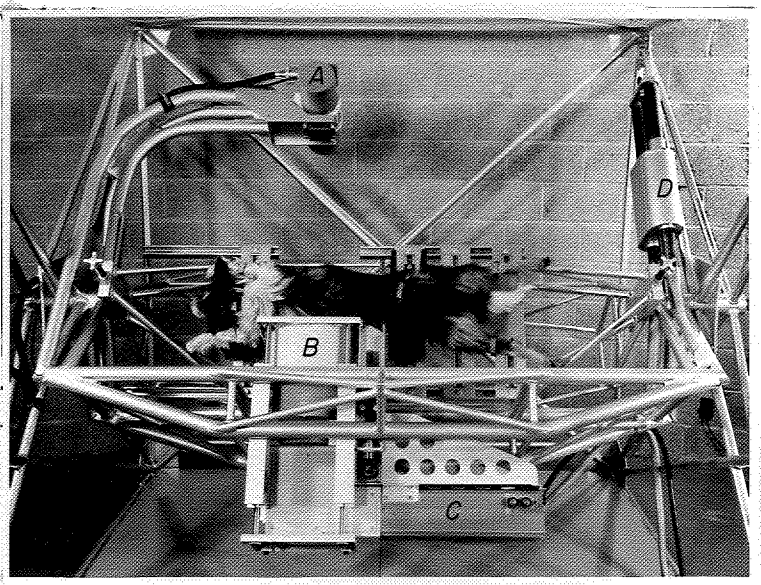


FIGURE 6

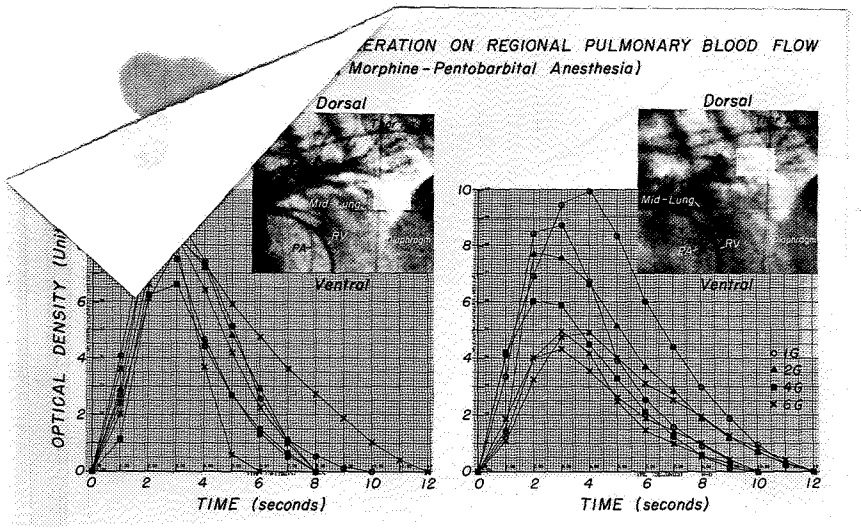

ERATION ON REGIONAL PULMONARY BLOOD FLOW
Morphine - Pentobarbital Anesthesia)
Dorsal
Ventral
Mid-Lung
RV
PA
OPTICAL DENSITY (Uni
TIME (seconds)
Dorsal
Ventral
Mid-Lung
RV
PA
1G
2G
4G
6G
TIME (seconds)

FIGURE 7

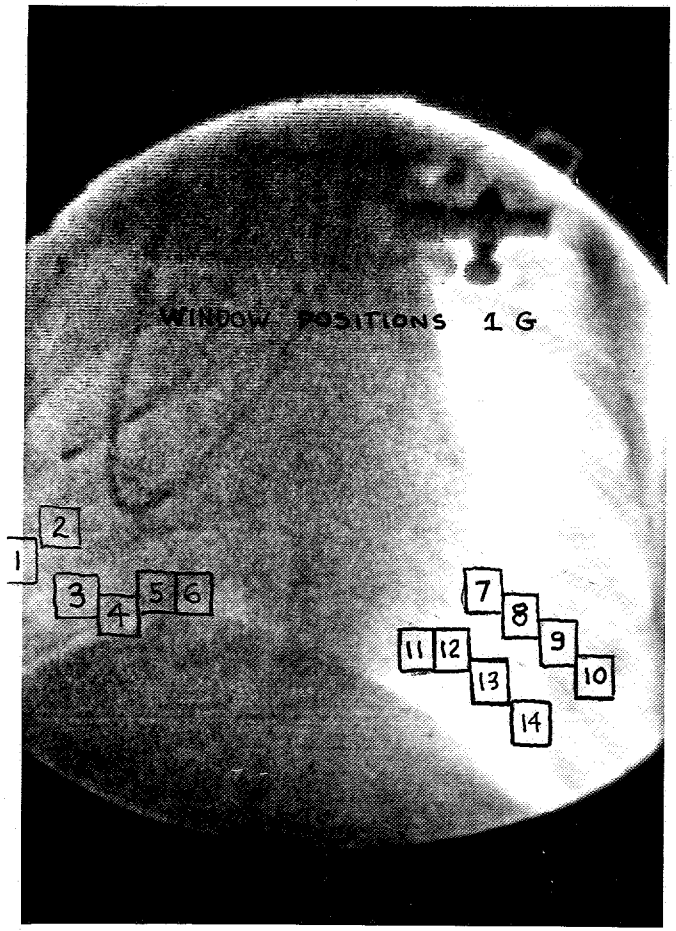
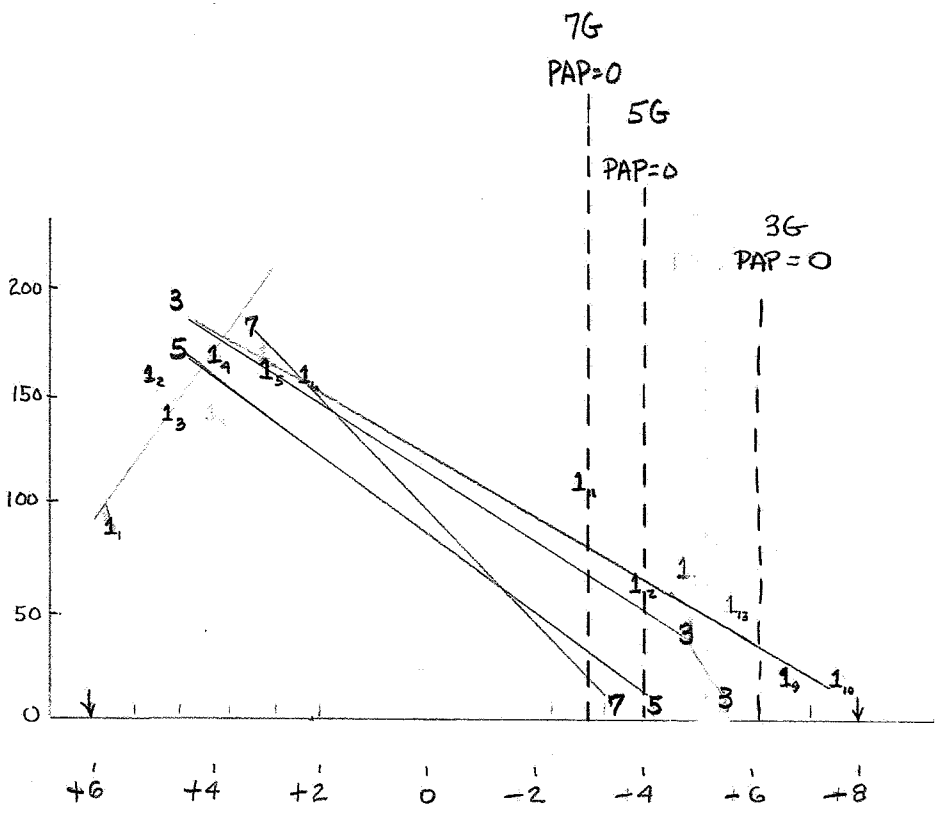


FIGURE 8.

EFFECT OF LATERAL ACCELERATION ON REGIONAL PULMONARY BLOOD FLOW (RIGHT LUNG DEPENDANT)

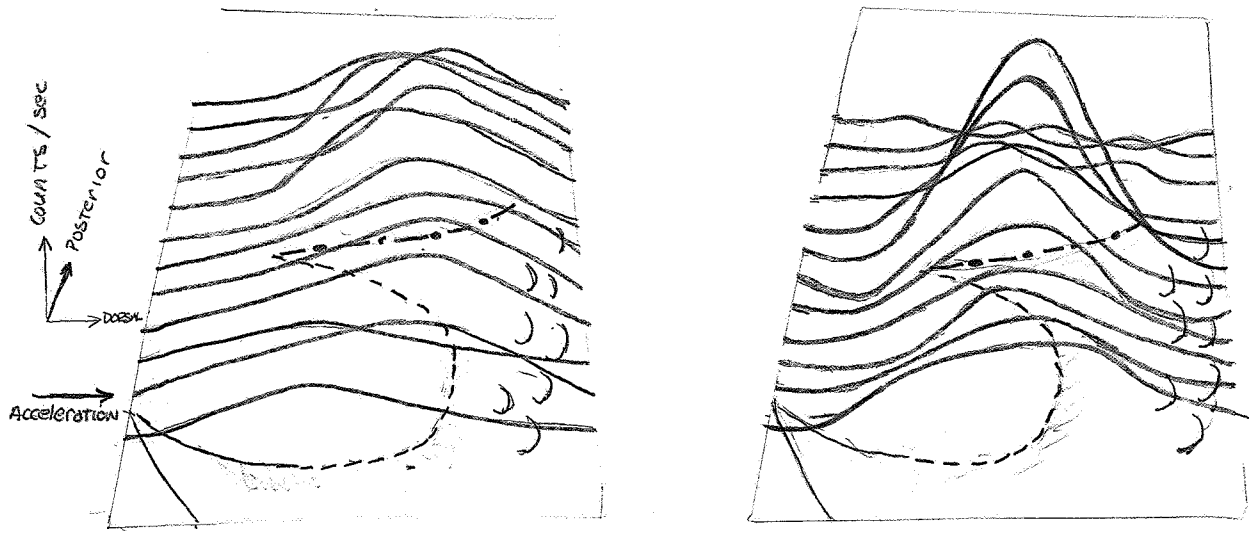


G	PARAMETERS CO L/Min	MPAP Cm H_2O	ZERO-PAP Cm above mid Lung
1	2.7	13	13
3	3.1	18	6
5	3.0	20	4
7		22	3
1	3.1	12	12

FIGURE 9

Distribution of counts over lung from radioactive microsphere emboli injected in right ventricular outflow tract during 1G and 6G (Dog in prone position)

DOG E-15



1G

6G

Key for Sagital plane of Thorax:

BROKEN LINE	Represents	heart shadow on X ray plate (lateral)
DASH-DOT	Represents	Diaphragm shadow on Xray plate (lateral)
))	Represents	vertebra interspaces " " "

FIGURE 10

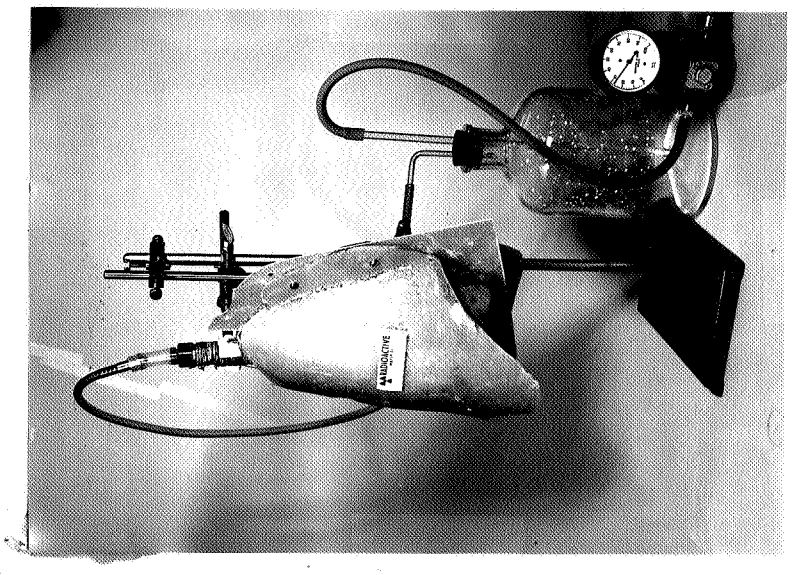


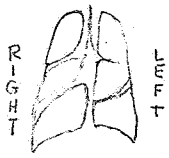
FIGURE 11

DISTRIBUTION OF MICROSPHERES EMBOLIZED IN LUNG
(injection: RIGHT LATERAL position in [illegible] outflow tract)

MPA = 18 cm H_2O
MLA = 5 cm H_2O
AP = 1 cm H_2O

before injection
C.O. = 3.1 l/min
P.A. = 28/9 cm H_2O
L.A. = 10/3 cm H_2O
H.R. = 150 beats/min

after injection
C.O. = 2.95 l/min
PA = 26/16 cm H_2O
LA = 10/2 cm H_2O
HR = 174 beats/min



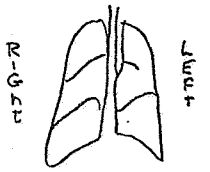
⇦ INERTIA

Dog G 812
Yb^{169}
$\rho = 1.4 - 1.$

FIGURE 12

DISTRIBUTION OF MICROSPHERES EMBOLIZED IN LUNG
(injection: RIGHT LATERAL position in Right Ventricular outflow tract)

MPA = 19 cm H_2O
MLA = 7 cm H_2O
AP = 5 cm H_2O



⇦ INERTIA

before injection
C.O. = 3.0 l/min
PA = 28/15 cm H_2O
LA = 10/6 cm H_2O
HR = 162 beats/min

after injection
C.O. = 2.65 l/min
PA = 28/15 cm H_2O
LA = 10/6 cm H_2O
HR = 176 beats/min

DOG = H 495
Kr^{85}
P = 0.2

FIGURE 13