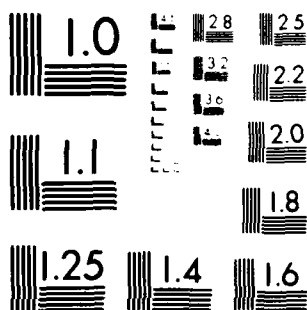


AD-A122 297 INTERACTION OF ANTI-G MEASURES AND CHEST WALL MECHANICS 1/1
IN DETERMINING GAS EXCHANGE(U) VIRGINIA MASON RESEARCH
CENTER SEATTLE WA H I MODELL MAY 82 AFOSR-TR-82-1046
UNCLASSIFIED F49620-79-C-0058 F/G 6/19 NL



END
DATE
FILMED
FBI
DTIC



MICROCOPY RESOLUTION TEST CHART
 NATIONAL BUREAU OF STANDARDS-1963-A

AFOSR-TR- 82 - 1046

AFOSR Contract Number F49620-81C-0055
Annual Progress Report
May, 1982

(4)

AD A 122297

INTERACTION OF ANTI-G MEASURES AND CHEST WALL
MECHANICS IN DETERMINING GAS EXCHANGE

VIRGINIA MASON RESEARCH CENTER
SEATTLE, WASHINGTON 98101

Dr. Harold I. Modell

Controlling Office: USAF Office of Scientific Research/NL
Bolling Air Force Base, DC 20332

DEC 13 1982
A

ANIMAL USE STATEMENT:

Care has been taken in this study to ensure that all animal experimentation complies with all federal animal welfare regulations and the "Guide for the Care and Use of Laboratory Animals".

Approved for public release;
distribution unlimited.

DATE FILE COPY

82 12 10 043

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER AFOSR-TR- 82 - 1046	2. GOVT ACCESSION NO. AD-A122 297	3. REPORT NUMBER CATALOG NUMBER
4. TITLE (and Subtitle) Interaction of Anti-G Measures and Chest Wall Mechanics in Determining Gas Exchange		5. TYPE OF REPORT & PERIOD COVERED Progress Report 1 Apr 81 - 31 Mar 82
7. AUTHOR(s) Harold I. Modell, Ph.D.		6. PERFORMING ORG. REPORT NUMBER
9. PERFORMING ORGANIZATION NAME AND ADDRESS Virginia Mason Research Center 1000 Seneca St. Seattle, Washington 98101		8. CONTRACT OR GRANT NUMBER(s) F49620-79-C-0058
11. CONTROLLING OFFICE NAME AND ADDRESS USAF Office of Scientific Research/NL Bolling Air Force Base, DC 20332		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 61102F 2312/A1
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)		12. REPORT DATE May 1982
		13. NUMBER OF PAGES 23
		15. SECURITY CLASS. (of this report) UNCLASSIFIED
16. DISTRIBUTION STATEMENT (of this Report) Approved for public release; distribution unlimited.		15a. DECLASSIFICATION DOWNGRADING SCHEDULE
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number) Acceleration Chest Wall Gas Exchange G-Suit		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) During the last twelve months, progress has been made in three experimental areas: 1) determination that right ventricular blood provides an accurate mixed venous blood sample in the canine; 2) investigation of gas exchange during repeated canine +Gz exposures; and 3) characterization of pressure-volume relationships of the pig lung and chest wall. It is generally assumed that pulmonary arterial blood best represents mixed venous blood. Under some conditions (e.g. during +Gz stress) sampling from this site is difficult. To determine if the right ventricle is as good a		

DD FORM 1 JAN 73 1473

EDITION OF 1 NOV 65 IS OBSOLETE

UNCLASSIFIED
SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)
JUN 82

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

sampling site as the pulmonary artery in the dog, samples were drawn from both sites and compared for blood gas composition. No physiologically significant differences were detected between the two sites in this species.

Results of our earlier studies indicated that exposure to +Gz stress in the presence of G-suit abdominal bladder inflation and breathing air leads to a gas exchange detriment lasting as long as three minutes post-exposure. To determine if this detriment is cumulative on repeated exposure, dogs were exposed to two episodes of +Gz stress separated by a three minute recovery period. Blood gas status during the last 20 seconds of each 60 second exposure was assessed. Results indicated that the same degree of detriment occurred during both exposures. The proposed mechanism of the detriment is airway closure created by the pressure generated by the abdominal bladder and +Gz stress rather than frank atelectasis.

Pig chest wall shape and compliance is closer to man's than is the dog chest wall. Adequate data characterizing the pig chest wall and lung mechanics, however, are not available. Studies have been initiated to provide this necessary base line data.

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

ABSTRACT

During the last twelve months, progress has been made in three experimental areas: 1) determination that right ventricular blood provides an accurate mixed venous blood sample in the canine; 2) investigation of gas exchange during repeated canine +Gz exposures; and 3) characterization of pressure-volume relationships of the pig lung and chest wall.

It is generally assumed that pulmonary arterial blood best represents mixed venous blood. Under some conditions (e.g. during +Gz stress) sampling from this site is difficult. To determine if the right ventricle is as good a sampling site as the pulmonary artery in the dog, samples were drawn from both sites and compared for blood gas composition. No physiologically significant differences were detected between the two sites in this species.

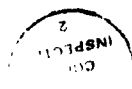
Results of our earlier studies indicated that exposure to +Gz stress in the presence of G-suit abdominal bladder inflation and breathing air leads to a gas exchange detriment lasting as long as three minutes post-exposure. To determine if this detriment is cumulative on repeated exposure, dogs were exposed to two episodes of +Gz stress separated by a three minute recovery period. Blood gas status during the last 20 seconds of each 60 second exposure was assessed. Results indicated that the same degree of detriment occurred during both exposures. The proposed mechanism of the detriment is airway closure created by the pressures generated by the abdominal bladder and +Gz stress rather than frank atelectasis.

Pig chest wall shape and compliance is closer to man's than is the dog chest wall. Adequate data characterizing the pig chest wall and lung mechanics, however, are not available. Studies have been initiated to provide this necessary base line data.

AIR FORCE OFFICE OF SCIENTIFIC RESEARCH (AFSC)
NOTICE: THIS IS A FINAL COPY
THIS DOCUMENT IS UNCLASSIFIED
DATE 11-11-81 BY 1011A, AFR 100-12.
Distribution is unlimited.
MATTHEW J. KILMER
Chief, Technical Information Division

High sustained gravitational stress (HSG), such as that experienced by pilots of high performance aircraft, affects cardiovascular and respiratory function adversely (Burton et al., 1974). Cardiovascular function is compromised because of changes in hydrostatic relationships caused by the increased G. Similar mechanisms influence distribution of ventilation and pulmonary perfusion (Bryan et al., 1966; Jones et al., 1969; Glaister, 1970a; von Niding and Krekeler, 1973). In addition, the HSG may alter chest wall mechanics (Hershgold, 1960) and impair gas exchange. A number of protective measures are presently employed in an attempt to restore normal arterial blood pressure and, thus, increase pilot tolerance to high sustained gravitational forces. Some of these measures (e.g. anti-G suits) have been associated with additional detriment to pulmonary gas exchange (Barr, 1962; Nolan et al., 1963; Hyde et al., 1963), whereas others (e.g. positive pressure breathing) may enhance pulmonary gas exchange under HSG conditions. Review of the literature reveals little information concerning the effects of standard anti-G measures on gas exchange. In view of this, several questions relevant to HSG tolerance must be answered if more effective protective measures are to be developed:

1. To what extent do commonly used protective measures enhance or impair pulmonary gas exchange?
2. What is the time course of any gas exchange detriment resulting from use of protective devices (e.g. anti-G suits) during HSG?
3. Is there a cumulative effect associated with gas exchange detriment resulting from use of protective devices?



A

4. By what means can these measures be modified to optimize gas exchange during HSG?

This project, initiated under AFOSR Contract F 49620-78-C-0058, has focused on these questions. Work conducted prior to 1 April 1981 (summarized in our final report entitled, "Effects of Anti-G Measures on Gas Exchange" dated May, 1981) dealt primarily with the influence of +Gz stress on chest wall-pulmonary mechanics and gas exchange in dogs. This year's effort has been directed toward extending those studies and examining similar relationships in the pig, an animal whose chest wall compliance and shape more closely approximates that of man than does the canine chest wall. By re-examining the relationships in the pig, an indication of direct influence of chest wall shape and compliance on gas exchange under these conditions may be obtained. During the last twelve months, progress has been made in three experimental areas: 1) determination that right ventricular blood provides an accurate mixed venous blood sample in the canine; 2) investigation of gas exchange during repeated canine +Gz exposures; 3) characterization of in vivo pressure-volume relationships of the lung and chest wall in the pig.

1. COMPARISON OF "MIXED VENOUS" BLOOD SAMPLING SITES IN THE CANINE

The blood gas status of mixed venous blood must be assessed if a complete picture of pulmonary gas exchange under various conditions is desired. Early studies dealing with the Fick technique for cardiac output determination addressed the importance of the mixed venous blood sampling site. Because of technique limitations, most of these studies compared blood sampled in the vena cavae or right atrium to right ventricular blood (Holt and Knöfel, 1944; Cournand, 1945; Shore et al., 1945; Cournand et al., 1945; Warren et al., 1946; Dexter et al., 1947; Barratt-Boyes et al., 1957). The general conclusion was that blood returning to the heart was not well mixed prior to its entrance into the right ventricle. Little attention was focused on the right ventricle versus the pulmonary artery as sampling sites. Development of the Swan-Ganz, balloon-tipped, catheter has provided relatively easy access to the pulmonary artery. Hence, most investigators now avoid the question of adequate mixing by sampling directly from the pulmonary artery rather than from a heart chamber. Under certain experimental conditions (e.g. high Gz stress) it is not always possible to obtain blood from this site. Furthermore, Shapiro and colleagues (1974) have shown that contamination of mixed venous blood by pulmonary capillary blood can occur at the pulmonary artery level. Because little data are available assessing the blood gas status of the right ventricular blood relative to pulmonary arterial blood, this study was designed to determine if, in the dog, the right ventricle is as good a sampling site for mixed venous blood as is the pulmonary artery.

Methods

Six adult mongrel dogs weighing 21.5 ± 3.97 (SD) kg were anesthetized with 30 mg/kg pentobarbital sodium and intubated with a cuffed endotracheal tube. A 7 Fr Swan-Ganz catheter was introduced through the right external jugular vein and positioned with its tip in the pulmonary artery. A second 7 Fr catheter with multiple side holes was also introduced through the right external jugular vein and positioned with its tip in the right ventricle. Placement was determined by observing the pressure profiles measured at the catheter tips, and care was taken to position the Swan-Ganz tip just beyond the pulmonary valve. The femoral artery and vein were cannulated for systemic arterial blood pressure monitoring and supplemental anesthesia administration.

Ten pairs of blood samples were drawn at five minute intervals for blood gas comparison. Each pair consisted of a 2 cc sample drawn from the pulmonary artery and 2 cc sample drawn from the right ventricle. These were drawn sequentially, and the order in which they were obtained was alternated with each pair. All samples were placed immediately in an ice bath, and analyzed subsequently for P_{O_2} , P_{CO_2} and pH using an Instrumentation Laboratories model 113 blood gas analyzer. All blood gas determinations were performed in duplicate, and calibration of the instrument was checked after each sample. Hemoglobin concentration for each sample was determined using the cyanmethemoglobin method. Data analysis was performed using a paired t-test.

Results

Results are shown in Figures 1 and 2. Figure 1 shows oxygen and

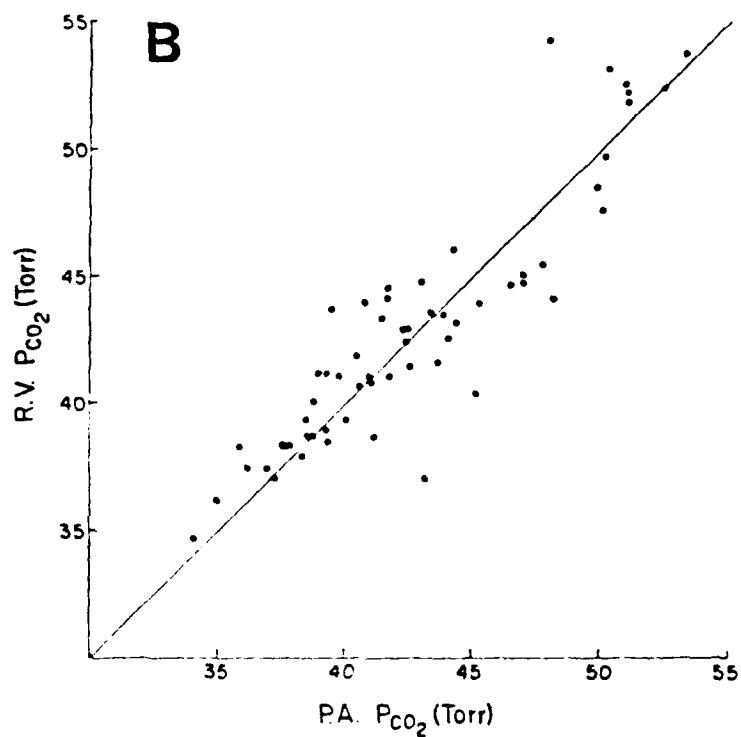
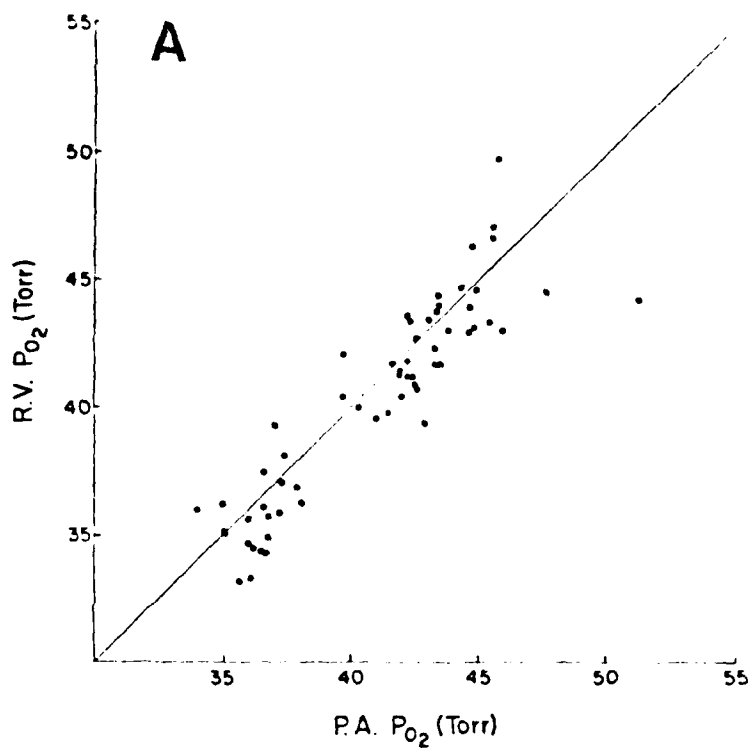


FIGURE 1. Comparison of oxygen (A) and carbon dioxide (B) tensions from blood samples drawn from the right ventricle (R.V.) and pulmonary artery (P.A.) of 6 dogs. Line of identity is indicated. No physiological differences were detected between sampling sites.

carbon dioxide tensions measured from right ventricular samples compared to those measured from pulmonary artery samples. Oxygen tensions measured ranged from approximately 35 to 50 Torr. The mean Po_2 measured from samples drawn from the pulmonary artery was 40.9 ± 3.85 (SD) Torr. The mean Po_2 measured from the corresponding right ventricular samples was 40.1 ± 3.94 Torr. Statistical analysis using a paired t-test indicated that pulmonary arterial oxygen tension was significantly higher than that in the right ventricular samples ($P < .005$). Although the values differed statistically, the difference was less than 1 Torr and most likely not significantly different physiologically.

A comparison of Pco_2 measured at the two sites is shown in Figure 1B. No statistical difference was found in this measurement between pulmonary arterial and right ventricular blood. Mean carbon dioxide tension in the pulmonary arterial samples was 42.74 ± 4.76 (SD) Torr, while in the right ventricular samples the mean tension was 42.7 ± 4.8 Torr.

Comparisons of pH and hemoglobin measurements from the two sites are shown in Figure 2. These determinations were made in four of the six animals. Hence, the data represent 40 rather than 60 sample pairs. No significant difference was found between pulmonary artery and right ventricle in either pH or hemoglobin determinations. Mean values for pH and hemoglobin from pulmonary artery samples were 7.347 ± 0.032 (SD) and 17.71 ± 1.08 gms/100 ml, respectively. Mean values for pH and hemoglobin from right ventricular samples were 7.346 ± 0.032 and 17.72 ± 1.09 gms/100 ml, respectively.

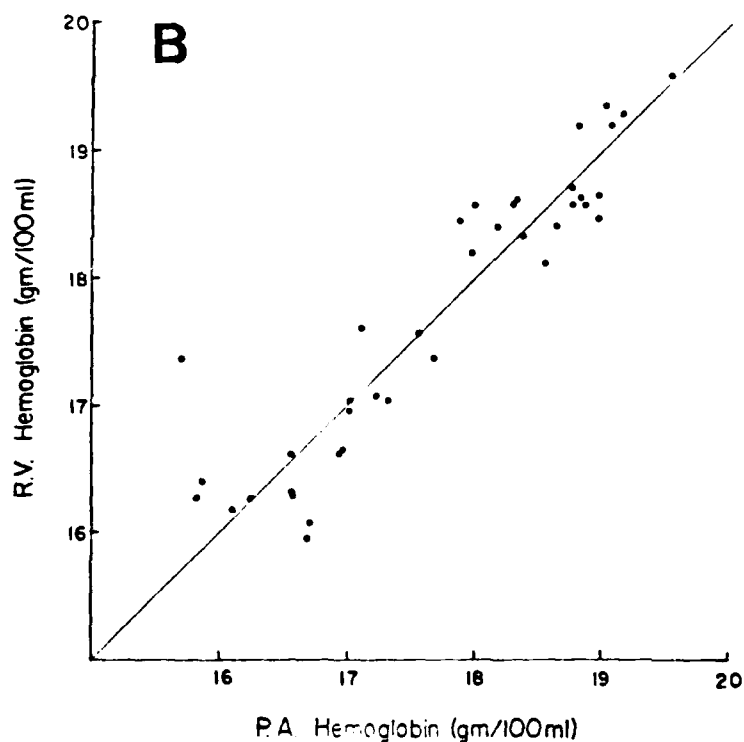
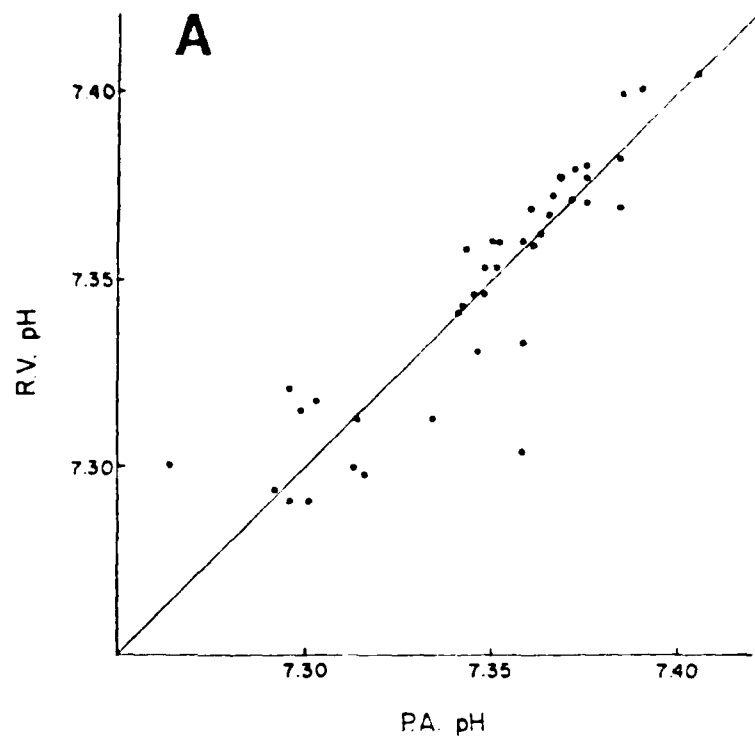


FIGURE 2. Comparison of pH (A) and hemoglobin concentration (B) from blood samples drawn from the right ventricle (R.V.) and pulmonary artery (P.A.) of dogs. Line of identity is indicated. No statistical differences (paired t-test) were detected between sampling sites.

Discussion

The importance of obtaining samples representative of "mixed venous" blood has been emphasized since the Fick determination for cardiac output gained wide acceptance in experimental and clinical situations (Holt and Knoefel, 1944; Cournand, 1945; Shore et al., 1945). Early investigators considered sampling sites ranging from the vena cavae to the right ventricle (Holt and Knoefel, 1944; Cournand et al., 1945; Shore et al., 1945; Warren et al., 1946) and concluded, on the basis of oxygen content measurements, that mixed venous blood was most reliable obtained from the right ventricle. Dexter et al. (1947) and Barratt-Boyes and Wood (1957) extended their studies in humans to include the pulmonary artery as a sampling site. Although the right ventricular and pulmonary arterial blood oxygen content determinations reported by Dexter et al. revealed no statistically significant difference between these two sites, these authors concluded that the pulmonary artery provided less variation among samples and was, therefore, the sampling site of choice. Barratt-Boyes and Wood, measuring blood oxygen saturation from various sites suggested that mixing of venous blood was relatively complete when it reached the right ventricular outflow tract. Nevertheless, these authors assumed that only pulmonary artery samples represented mixed venous blood. More recently, Weber and associates (1980) re-examined the various sites in healthy infants and children and concluded that, of the sites considered, the mean oxygen saturation from the right ventricle most nearly approached the mean oxygen saturation of the pulmonary artery. Results from these studies suggest that the best estimation of mixed

venous blood composition is best obtained by sampling from the pulmonary artery. The majority of these studies were conducted in humans. Little data are available in other species.

Shapiro and colleagues (1974) examined possible sources of errors when blood is sampled from the pulmonary artery. These investigators noted that, in humans, contamination from pulmonary capillary blood occurred only when the Swan-Ganz catheter was in the wedge position with the balloon inflated. In the dog, however, contamination of the pulmonary arterial sample by pulmonary capillary blood was seen even though a good pulmonary arterial blood pressure trace was recorded from the catheter prior to sampling. This finding suggests that sampling pulmonary arterial blood as "mixed venous" in the dog may introduce errors. Our data, in contrast to the earlier human studies, suggest that blood drawn from the right ventricle has essentially the same composition as that sampled just beyond the entrance to the pulmonary artery. This implies that, in the dog, either mixing is relatively complete within the right ventricle, or the catheter with multiple side holes enhances right ventricular blood mixing during sampling relative to a catheter having a single sampling port. In either case, our data suggest that right ventricular sampling in the dog provides a good estimate of mixed venous blood composition and minimizes the potential error of contamination by pulmonary capillary blood. Whether this is the case in other species remains to be determined.

II. GAS EXCHANGE DURING REPEATED +Gz STRESS IN DOGS

It has been generally agreed that acceleration atelectasis occurs only when +Gz stress is accompanied by 100% oxygen breathing and use of a G-suit (Glaister, 1970). Results of our earlier study focusing on the effects of G-suit abdominal bladder inflation per se on gas exchange indicated that abdominal bladder inflation with +Gz stress greater than +4Gz is associated with increased venous admixture (Modell, 1982). Furthermore, this detriment persists for at least 3 minutes post-G. Since repeated +Gz stress within short periods of time may be common in high performance aircraft, it is important to know if the G-suit induced detriment is cumulative on repeated exposures. The purpose of this study was to gain information related to this question.

Methods

Five adult male mongrel dogs weighing 22.06 ± 1.57 (SD) Kg were anesthetized with 30 mg/kg pentobarbital sodium and intubated with a cuffed endotracheal tube. A Millar, catheter-tip pressure transducer was introduced through the right femoral artery and positioned in the thoracic aorta for arterial blood pressure monitoring. A 7 Fr catheter was introduced through the left femoral artery and positioned in the thoracic aorta for arterial blood sampling. A similar sized catheter with multiple side holes was introduced through the right external jugular vein and positioned in the right ventricle for sampling mixed venous blood. Another catheter, placed in the right femoral vein, served as an injection site for supplemental anesthesia.

A standard G-suit abdominal bladder (CSU-12P) was placed around the

animal, and the animal was secured to a V-board restraint in the supine position. All +Gz exposures were made using the human centrifuge at USAFSAM (3.97 M radius). Remote sampling techniques for blood samples have been described in our previous report.

Animals were exposed to +4Gz (onset rate = 0.1 G/sec) or +5Gz with G-suit inflation using the standard inflation scheme (1.5 psi/G beginning at +2.2 Gz). Acceleration levels and G-suit status were randomized within the experimental design.

Prior to the first +Gz exposure, the animal was heparinized with 3000 units of heparin sodium administered intravenously, and control samples of arterial and mixed venous blood were drawn for blood-gas analysis. The experimental procedure paralleled the earlier studies. Animals were exposed to a given Gz level for approximately 60 seconds. After 40 seconds of the exposure, arterial and mixed venous blood samples were drawn (sampling time was approximately 18 seconds), and the animal was returned to 0Gz. When 0Gz was reached, a stopwatch was activated, and the blood samples were iced for later analysis. After 3 minutes, the animal was again exposed to the same +Gz stress, and blood samples were again drawn beginning at 40 second of the exposure. The animal was returned to 0Gz, the stopwatch activated, and the second set of blood samples were iced for later analysis. Three minutes after reaching 0Gz, a third set of blood samples were drawn and iced. The animal's lungs were then inflated several times with a large volume using an Ambu bag. At 10-15 minutes post-G stress, another set of arterial and mixed venous blood samples were drawn as 0Gz controls. The animal was then exposed to the next test condition.

After all +Gz exposures were completed, the blood samples were analyzed for PO_2 , PCO_2 and pH using an Instrumentation Laboratories Model 113 blood gas analyzer. Instrument calibration was checked after each sample.

Results

Data obtained from 6 +4Gz trials in 5 animals and 5 +5Gz trials in 3 animals are presented in Figure 3 where the arterial PO_2 measured three minutes after the second +Gz exposure are compared to control values. At each acceleration level, arterial oxygen tension remained 10-15 Torr below control values ($P < .05$, Student's t-test) three minutes post-G stress. These data are consistent with results of the earlier study where the animal was exposed to a single period of +Gz stress. These data are also summarized in Figure 3.

Arterial oxygen tension measured from samples drawn during the first +Gz exposure are compared to those measured from samples drawn during the repeated exposure in Figure 4. As indicated in this figure, the oxygen tension of arterial blood during the second +4 or +5Gz exposure was essentially the same as that seen during the initial exposure. When arterial PCO_2 and pH were examined, no significant differences between the initial and repeated exposure were detected. These data suggest that, with repeated +Gz stress during air breathing, the same degree of gas exchange detriment accompanies G-suit abdominal bladder inflation.

Discussion

Acceleration atelectasis as a clinical entity is characterized by the presence of cough, chest pain, and the inability to take a deep breath (Glaister, 1970). In studies aimed at examining factors

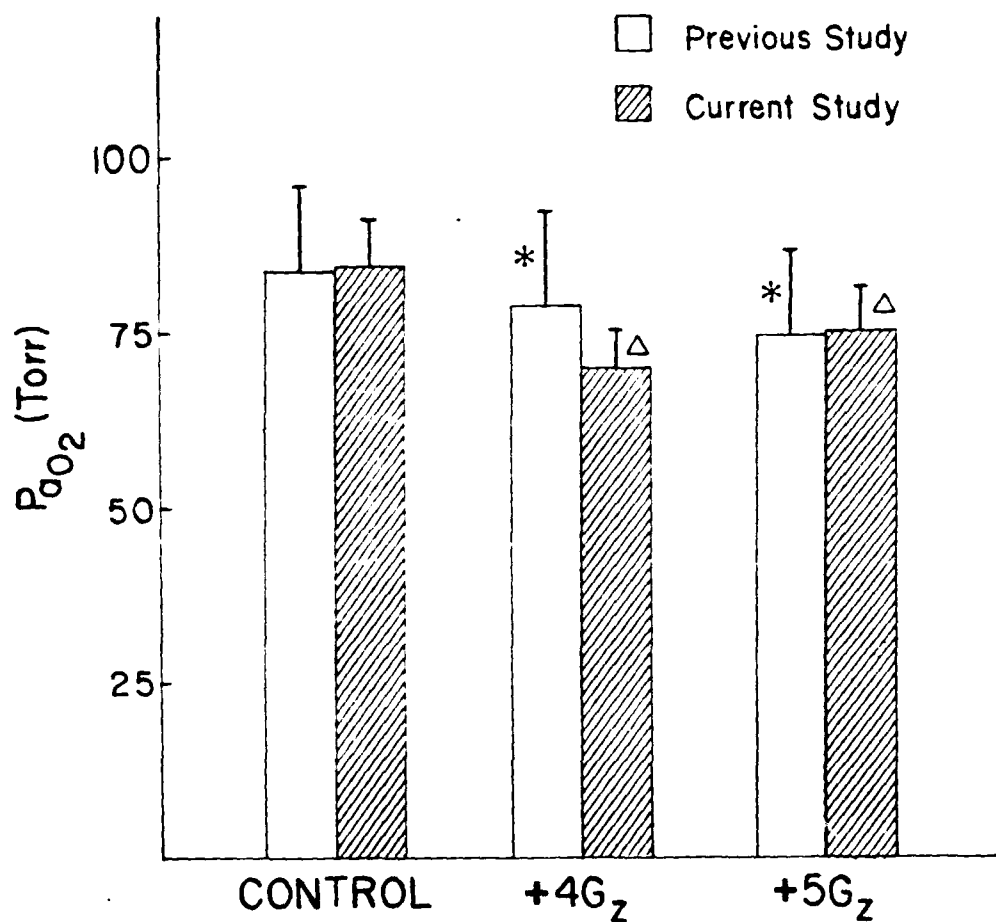


FIGURE 3. Arterial oxygen tension measured before and 3 minutes after exposure to +4 and +5G_z with G-suit abdominal bladder inflation. Open bars indicate data from previous study (Modell, 1982). Hatched bars indicate data from current studies. In both sets of experiments, arterial oxygen tension was significantly below control values after 3 minutes of recovery (P<.05, paired t-test).

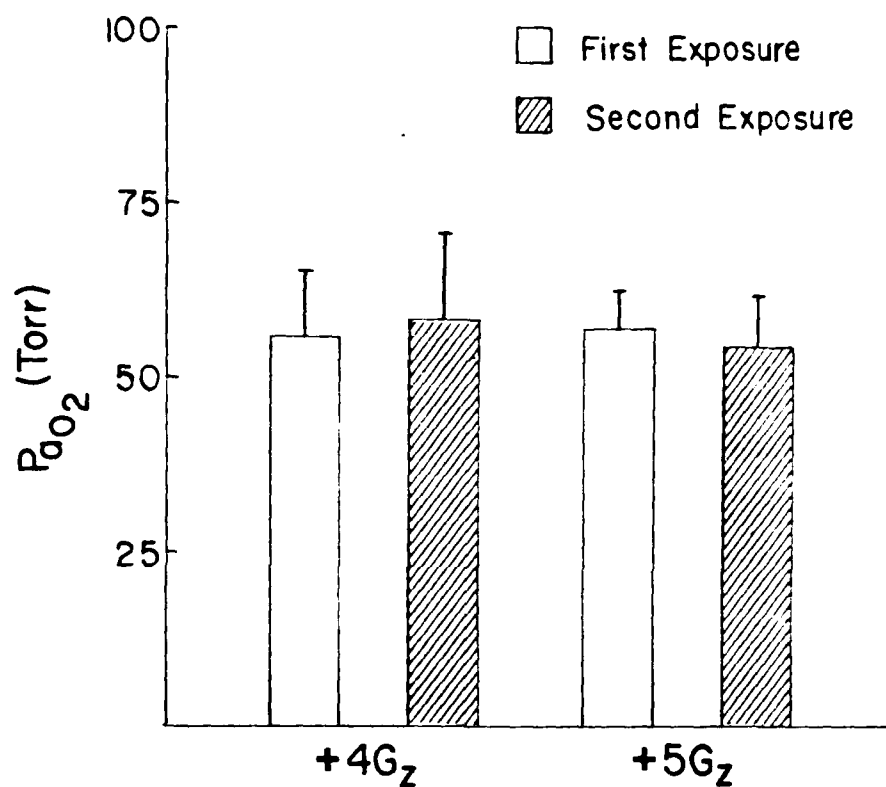


FIGURE 4. Arterial oxygen tension during the last 20 seconds of first exposure to +4 or +5G_z (open bars) compared to arterial oxygen tension during the repeated exposure after a 3 minute recovery period (hatched bars). No significant differences were detected (paired t-test).

contributing to acceleration atelectasis in man, Glaister (1965) assessed the presence and degree of atelectasis on the basis of vital capacity measurements. On this basis, he demonstrated that G-suit abdominal bladder inflation was necessary for vital capacity reductions to occur. He further demonstrated that significant vital capacity reductions occurred only when oxygen was breathed during application of +Gz stress. Green and Burgess (1962) provided radiographic evidence of lung collapse along with their measurements of lung volumes in man. The proposed mechanism of acceleration atelectasis development is airway closure created by mechanical forces associated with G-suit abdominal bladder inflation followed by alveolar collapse due to oxygen absorption (Glaister, 1970). Our measurements of regional intrapleural pressure during +Gz stress with abdominal bladder inflation in the dog (Modell, 1981) indicate that regional transpulmonary pressure favors airway collapse as +Gz stress and inflation pressure increase.

Studies in man assessing acceleration atelectasis development have not included arterial blood gas measurements. If either airway closure alone or in conjunction with atelectasis occurred during the exposure, increased venous admixture would result, and arterial PO_2 would fall, since, in both cases, areas with low ventilation-perfusion ratios would result. Lung inflation subsequent to the exposure would reopen areas of airway collapse or atelectasis. However, less pain would be encountered during voluntary re-expansion of the region with airway collapse than that encountered during re-expansion of the atelectatic area. Hence, regions of airway collapse would be more likely opened during a vital capacity maneuver than would atelectatic areas. Under the conditions of

our experiments, the anesthetized dogs most likely did not make large inspiratory efforts during the 3 minute recovery period, and, therefore, some degree of venous admixture remained (Fig. 3). The detriment was easily removed by rapid reinflation with an Ambu bag, suggesting airway closure rather than atelectasis.

Repeated exposure to +Gz stress after atelectasis development could result in a greater gas exchange detriment than that seen during the initial exposure, since the mechanical forces would act on an altered lung-chest wall configuration. Repeated exposure after airway closure, however, would be expected to result in the same degree of detriment since lung-chest wall configuration and the mechanical forces generated would replicate the initial conditions. Although atelectasis development can not be ruled out at this point, the more likely explanation for the data shown in Figure 4 that would also be consistent with Glaister's (1965) findings is that the increased intrapleural pressure generated by abdominal bladder inflation creates a significant amount of airway closure resulting in increased venous admixture. The detriment would increase at higher +Gz levels because of the continued increase in intrapleural pressure, but repeated exposures at a given acceleration level would not exhibit a cumulative effect.

III. CHARACTERIZATION OF IN VIVO PRESSURE-VOLUME RELATIONSHIPS OF THE LUNG AND CHEST WALL

Results of our previous studies using dogs suggest that the degree of gas exchange detriment seen with G-suit use may depend on chest wall compliance and configuration. The shape of the pig's chest wall is considerably different from that of the dog, and it is closer to that of man. Pig chest wall compliance is also closer to man's than is dog (Attinger and Cahill, 1960), although adequate data characterizing pig chest wall and lung mechanics are not available. This project has been initiated to obtain base line data for our upcoming experiments examining lung-chest wall interactions and gas exchange during +Gz stress in pigs.

Experiments are currently in progress to define in vivo lung and chest wall pressure-volume relationships. Anesthesia is induced with ketamine and xylazine and maintained with pentobarbital sodium. A tracheostomy is performed, the external jugular vein is cannulated, and the carotid artery is cannulated for arterial pressure monitoring. Two cannulae modified from those described in our earlier reports for monitoring intrapleural pressure are placed in the 3-4 and 6-7 intercostal space. The animal is then ventilated mechanically at a rate sufficient to lower the monitored mixed expired P_{CO_2} to approximately 10 Torr. This level is below the animal's apneic threshold, and when removed from the ventilator, its apneic time is greater than 1 minute.

To ensure a constant lung volume history prior to a test run, the animal is removed from the ventilator, its lungs are inflated to 30 cm

H₂O airway pressure. The animal's lungs are allowed to deflate passively, and lungs are inflated with 0.8 to 1 liter of air. This volume is held for 5 to 10 seconds after which the lungs are deflated in 200 ml steps. Five to ten seconds are allowed to elapse at each deflation step to ensure static conditions. The process is repeated until airway pressure becomes negative. Upon completion of the maneuver (approximately 45 seconds), the lungs are again inflated to 30 cm H₂O airway pressure.

By monitoring airway and intrapleural pressures relative to atmospheric pressure, and assuming that the chest wall muscles are relaxed during apnea, pressure-volume curves of the lung and chest wall may be derived. An example of such curves from one experiment is shown in Figure 5.

To provide a more accurate estimation of the volume axis and correct for volume changed due to gas exchange during an experimental trial, Functional Residual Capacity, oxygen consumption and carbon dioxide production will be measured in future experiments.

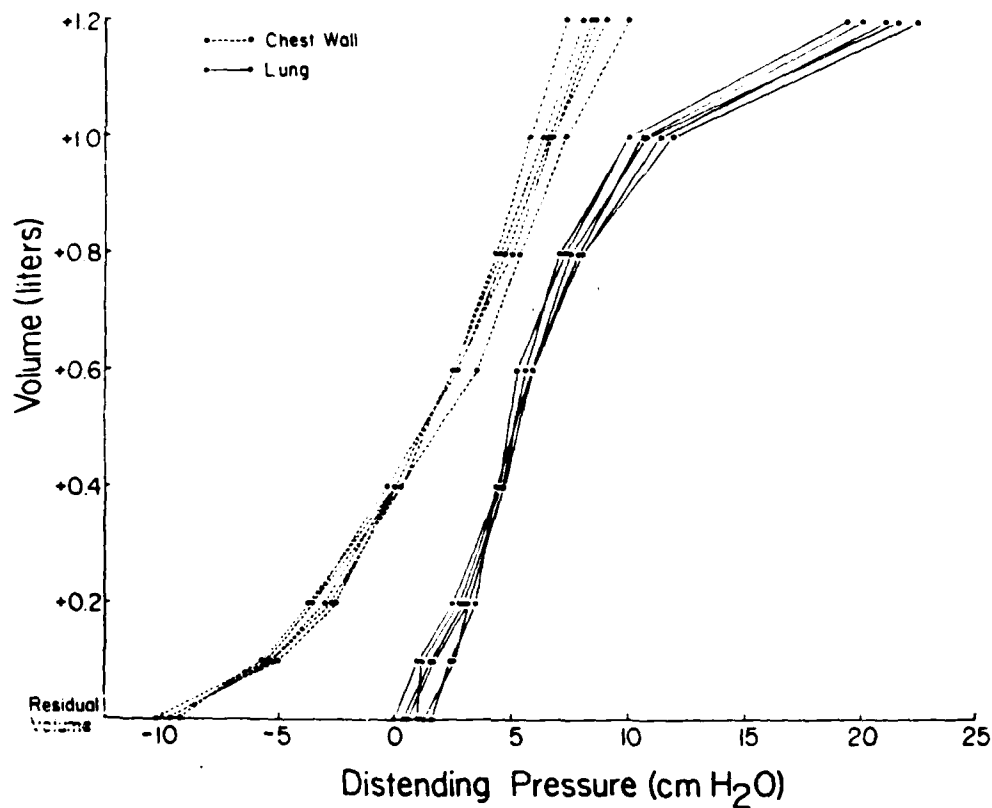


FIGURE 5. Deflation pressure-volume curves of the pig chest wall and lung obtained from one experiment of the current series. Future experiments will also include Functional Residual Capacity measurements so that the volume axis may be expressed in absolute rather than relative terms.

References

1. Attinger, E.O. and J.M. Cahill. Cardiopulmonary mechanics in anesthetized pigs and dogs. Am. J. Physiol. 198: 346-348, 1960.
2. Barr, P.-O. Hypoxemia induced by prolonged acceleration. Acta Physiol. Scand. 54: 128-137, 1962.
3. Barratt-Boyes, B.G. and E.H. Wood. The oxygen saturation of blood in the venae cavae, right-heart chambers, and pulmonary vessels of healthy subjects. J. Lab. and Clin. Med. 50: 93-106, 1957.
4. Bryan, A.C., J. Milic-Emili and D. Pengelly. Effect of gravity on the distribution of pulmonary ventilation. J. Appl. Physiol. 21: 778-784, 1966.
5. Burton, R.R., S.D. Leverett, Jr. and E.D. Michaelson. Man at high sustained +Gz acceleration: a review. Aerospace Med. 45: 1115-1136, 1974.
6. Cournand, A. Measurement of the cardiac output in man using the right heart catheterization. Fed. Proc. 4: 207-212, 1945.
7. Cournand, A., R.L. Riley, E.S. Breed, E.deF. Baldwin and D.W. Richards, Jr. Measurement of cardiac output in man using the technique of catheterization of the right auricle or ventricle. J. Clin. Invest. 24: 106-116, 1945.
8. Dexter, L., F.W. Hayes, C.S. Burwell, E.C. Eppinger, R.P. Sagerson and J.M. Evans. Studies of congenital heart disease. II. The pressure and oxygen content of blood in the right auricle, right ventricle and pulmonary artery in control patients, with observations on the oxygen saturation and source of pulmonary "capillary" blood. J. Clin. Invest. 26: 554-560, 1947.
9. Glaister, D.H. Acceleration atelectasis - some factors modifying its occurrence and magnitude. FPRC/Memo 220, January, 1965.
10. Glaister, D.H. The effects of gravity and acceleration on the lung. AGARDograph 133. England: Technical Services, 1970.
11. Green, I.D. and B.F. Burgess. An investigation into the major factors contributing to post flight chest pains in fighter pilots. FPRC Report 1182, 1962.
12. Hershgold, E.J. Roentgenographic study of human subjects during transverse accelerations. Aerospace Med. 31: 213-219, 1960.
13. Holt, J.P. and P.K. Knoefel. The oxygen content of blood in the right auricle and right ventricle. Fed. Am. Soc. Exptl. Biol. 3: 19-20, 1944.

14. Hyde, A.S., J. Pines and I. Saito. Atelectasis following acceleration: a study of causality. Aerospace Med. 34: 150-157, 1963.
15. Jones, J.G., S.W. Clarke and D.H. Glaister. Effect of acceleration on regional lung emptying. J. Appl. Physiol. 26: 827-832, 1969.
16. Modell, H.I. Effects of acceleration (+Gz) on the intrapleural pressure gradient. The Physiologist 24: 96, 1981.
17. Modell, H.I. Influence of abdominal restriction on gas exchange during +Gz stress in dogs. The Physiologist (In press).
18. Nolan, A.C., H.W. Marshall, L. Cornin, W.F. Sutterer and E.H. Wood. Decreases in arterial oxygen saturation and associated changes in pressures and roentgenographic appearance of the thorax during forward (+Gx) acceleration. Aerospace Med. 34: 797-813, 1963.
19. Shapiro, H.M., G. Smith, A.H. Pribble, J.A. Murray and F.W. Cheney. Errors in sampling pulmonary arterial blood with a Swan-Ganz catheter. Anesthesiology 40: 291-295, 1974.
20. Shore, R., J.P. Holt and P.K. Knoefel. Determination of cardiac output in the dog by the Fick procedure. Am. J. Physiol. 143: 709-714, 1945.
21. von Nieding, G. and H. Krekeler. Effect of acceleration on distribution of lung perfusion and on respiratory gas exchange. Pflugers Arch. 342: 159-176, 1973.
22. Warren, J.V., E.A. Stead, Jr. and E.S. Brannon. The cardiac output in man: a study of some of the errors in the method of right heart catheterization. Am. J. Physiol. 145: 458-464, 1946.
23. Weber, H., T. Grimm and J. Albert. The oxygen saturation of blood in the venae cavae, right-heart chambers, and pulmonary artery, comparison of formulae to estimate mixed venous blood in healthy infants and children. Z. Kardiol. 69: 504-507, 1980.

Publications 1 April 1981 - 31 March 1982

Modell, H.I. and M.M. Graham. Limitations of Kr-81m for quantitation of ventilation scans. J. Nucl. Med. 23: 301-305, 1982.

Graham, M.M. and H.I. Modell. Limitations of Krypton-81m use for ventilation scans. J. Nucl. Med. 22: P92, 1981 (Abstract).

Modell, H.I. Effects of acceleration (+Gz) on the intrapleural pressure gradient. The Physiologist 24: 96, 1981 (Abstract).

Professional personnel associated with research effort

Harold I. Modell. Assistant Member, Virginia Mason Research Center

Michael M. Graham. Assistant Professor, Division of Nuclear Medicine, University of Washington.

Interactions

In the period 1 Apr 1981 to 31 March 1982, papers relating to this research effort have been presented at the following meetings:

28th Annual meeting of the Society of Nuclear Medicine 16-19 June 1981, Las Vegas, Nevada.

1981 Fall meeting of the American Physiological Society, 11-16 October 1981, Cincinnati, Ohio.

Review of Air Force sponsored basic research in environmental and acceleration physiology, 15-17 March 1982, San Antonio, Texas.

LME
-83