AD-A189 671

CHARACTERIZATION AND MODELING OF THORACO-ABDOMINAL RESPONSE TO BLAST WAVES

Volume 5. Experimental Investigation of Lung Injury Mechanism

Annual/Final Report

May 1985

Y. C. Fung and M. R. Yen
University of California, San Diego

Contract No. DAMD17-82-C-2062

88 2 12 05 2

The findings in this report are not to be construed as an official Department of the Army position unless so designated by other authorized documents.

Accesion For		
NTIS CRA&I	☑	
DTIC TAB	☐	
Unannounced	☐	
Justification		
By		
Distribution /		
Availability Codes		
Dist	Avail and / or Special	
A-1		

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1a. REPORT SECURITY CLASSIFICATION UNCLASSIFIED	1b. RESTRICTIVE MARKINGS

2a. SECURITY CLASSIFICATION AUTHORITY	3. DISTRIBUTION/AVAILABILITY OF REPORT
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE	Approved for public release; distribution unlimited

4. PERFORMING ORGANIZATION REPORT NUMBER(S)	5. MONITORING ORGANIZATION REPORT NUMBER(S)

6a. NAME OF PERFORMING ORGANIZATION JAYCOR	6b. OFFICE SYMBOL (If applicable)	7a. NAME OF MONITORING ORGANIZATION

6c. ADDRESS (City, State, and ZIP Code) 11011 Torreyana Road San Diego, California 92121	7b. ADDRESS (City, State, and ZIP Code)

8a. NAME OF FUNDING/SPONSORING ORGANIZATION U.S. Army Medical Research & Development Command	8b. OFFICE SYMBOL (If applicable)	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER DAMD17-82-C-2062

8c. ADDRESS (City, State, and ZIP Code) Fort Detrick Frederick, Maryland 21701-5012	10. SOURCE OF FUNDING NUMBERS			
	PROGRAM ELEMENT NO. 61102A	PROJECT NO. 3M1 61102BS10	TASK NO. CG	WORK UNIT ACCESSION NO. 087

11. TITLE (Include Security Classification)
(U) Characterization and Modeling of Thoraco-Abdominal Response to Blast Waves
Volume 5. Experimental Investigation of Lung Injury Mechanism

12. PERSONAL AUTHOR(S)
Y. C. Fung and M. R. Yen

13a. TYPE OF REPORT Annual/Final	13b. TIME COVERED FROM 2/15/82 TO 5/31/85	14. DATE OF REPORT (Year, Month, Day) 1985 May	15. PAGE COUNT 35

16. SUPPLEMENTARY NOTATION
Annual covers time period of 15 February 1984 - 31 May 1985.
Annual/Final published in 8 volumes

17.	COSATI CODES		18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
FIELD	GROUP	SUB-GROUP	
06	21		
06	17		

19. ABSTRACT (Continue on reverse if necessary and identify by block number)

20. DISTRIBUTION/AVAILABILITY OF ABSTRACT ☐ UNCLASSIFIED/UNLIMITED ☒ SAME AS RPT. ☐ DTIC USERS	21. ABSTRACT SECURITY CLASSIFICATION Unclassified
22a. NAME OF RESPONSIBLE INDIVIDUAL Mary Frances Bostian	22b. TELEPHONE (Include Area Code) 301-663-7325	22c. OFFICE SYMBOL SGRD-RMI-S

DD Form 1473, JUN 86   *Previous editions are obsolete.*   SECURITY CLASSIFICATION OF THIS PAGE

FOREWORD

This Annual/Final Report has eight volumes. The titles are as follows:

1. Project Summary

2. Blast Load Definition on a Torso Model

3. Lung Dynamics and Mechanical Properties Determination

4. Biomechanical Model of Thorax Response to Blast Loading

5. Experimental Investigation of Lung Injury Mechanism

6. Biomechanical Model of Lung Injury Mechanisms

7. Gastrointestinal Response to Blast

8. Effect of Clothing on Thoracic Response

CONTENTS

INTRODUCTION

This work focuses on the _quantitative_ evaluation of lung damage due to blast loading. This is part of the objectives of the work at UCSD Biomechanics Laboratory. The overall objectives are: (1) determination of mechanical properties of lung parenchyma, (2) investigation of wave propagation in the lung, (3) determination of pulmonary stress distribution under blast loading, and (4) examination of damage mechanism in the lung. The first two objectives have been accomplished (see previous reports). In order to achieve the third and fourth objectives, experiments involving damage of an excised lung as a result of blast loading were performed.

Edema and hemorrhage are the most evident visible injuries of the lung when it is subjected to pressure shock waves. A quantitative evaluation of these injuries is of interest. Since they develop gradually, it is necessary to make measurements sequentially in time. In this report we describe a gravimetric method which is based on first establishing an isogravimetric condition of the lung before the impact load is applied. The total lung weight is measured continuously so that the rate of progress of edema is obtained. The edema rate provides a quantitative measure of lung injury. To complement this data, stereometry of the hemorrhagic area on the pleura and in the lung can be done to provide data on the injury to the vasculature. Some preliminary data are presented in this report. It is shown that edema depends very much on the rigidity of the surface with which the lung is in contact. If the pleura is supported on a lightweight soft cloth (such as a nylon net), the edema produced by a pressure wave is much less than that produced in the same lung supported on a rigid boundary.

The excised lung was perfused. A blast wave was then imposed on the lung. The wave speed of the pressure wave was faster than the speed of propagation of the stress wave in the lung so that a shock condition was established. If the lung were injured by the blast wave to the degree that the permeability of the blood-tissue barrier was changed, then interstitial edema may occur if the lymph system drainage could not keep up with the fluid transfer. If the injury

1

were more severe so that the epithelial barrier between the interstitium of the lung tissue and the alveolar gas were damaged, then alveolar edema due to the movement of fluid into the alveoli will occur. The interstitial edema will be reflected by a moderate rate of increase of lung weight with time. The alveolar edema can be recognized by a substantial increase in the rate of increase of lung weight with time. Thus the evaluation of the lung weight increase from an initially isogravimetric condition would provide a quantitative measure of the lung injury due to blast wave.

Furthermore, when alveolar edema occurs in the alveoli immediately underneath the pleural surface, the color and texture of the lung change in such a way that edema becomes visible to a trained eye. Hemorrhage in the lung tissue close to the pleural surface is also visible. Hence a description of the sites of edema and hemorrhage can be done.

Results of the lung weight gain experiments are contained in the first paper attached, while observations on color and textural changes to the pleural surface are contained in the second paper. Since these findings are quite recent, general conclusions must be considered as preliminary. In particular it will require further investigation to confirm the exact origin of the weight gain seen.

EDEMA OF LUNG DUE TO IMPACT INJURY

by

.R.T. Yen, H.H. Ho, Z.L. Tao, and Y.C. Fung

Department of AMES/Bioengineering
University of California,San Diego
La Jolla, CA 92093

ABSTRACT

Edema formation is the most evident injury of the lung when it is subjected to an impact load. A quantitative evaluation of the lung injury is of interest.) Since edema develops gradually, it is necessary to make measurements sequentially in time. In this report we describe a gravimetric method which is based on first establishing an isogravimetric condition of the lung before the impact load is applied. Excised rabbit lung was tested. The total lung weight was measured continuously so that the rate of progress of edema was obtained. The edema rate provides a quantitative measure of lung injury. It is shown that the rate of edema formation depends on the rigidity of the surface on which the lung rested. If the lung was "freely" supported on a lightweight soft cloth (a net made of nylon stockings), the edema produced by an impact was much less than that produced in the same lung supported on a rigid plate. This undoubtedly indicates that the reflection of a stress wave on a hard surface imposes a more severe condition for lung injury. It is also shown that the initial velocity of impact at the surface of the lung is a principal parameter to correlate with the rate of edema formation after impact.) For a freely supported rabbit lung the rate of edema formation was 20% of the initial total lung weight one hour after impact when the impact velocity was 11.5 m/s, but it increased to 100% of initial lung weight in one hour when the velocity of impacting pellet exceeded 13.5 m/s. These velocities are much lower than the speed of automobiles on our highways (55 mph $\doteq$ 25 m/s), suggesting that lung injury may be significant in considering the highway traffic safety problem.

> Keywords: wounds and injures

4

INTRODUCTION

It has been known for a long time that when a man or animal is subjected to an explosive pressure wave of sufficient magnitude, the gas-containing soft organs are the ones that get injured first: the ears, the lung, the intestines, the larynx, (see Refs. 1-9 and the bibliographies therein). Compared with other parts of the body, these organs can change volume to a large extent. In the process of nonuniform rapid motion tissues become overstretched locally and transiently, and injury results. The ear drum may be broken; the intestine may bleed; the lung may have hemorrhage and edema. Severe edema may be lethal. Qualitatively this description is convincing. But quantitatively, the mechanism of injury is still unknown.

The objective of the present paper is to evaluate the impact injury of the lung by correlating the rate of edema formation with the initial velocity of the lung surface subjected to impact load. The initial impact velocity is selected as the principal parameter because the impact injury must be related to the magnitude of the stress in the tissue, and the stress induced in a material due to the passage of a plane progressive wave is equal to $\rho c v$, where ρ is the density of the material, c is the velocity of the wave in the material, and v is the velocity of the material particles in the wave motion. ρ and c are material properties of the lung, and are unrelated to the shock. But v characterizes the shock. Since the stress depends on v, v is the parameter of choice to evaluate the lung injury.

In Yen et al.(1985), we presented data for c in the rabbit and goat lung. c lies in the range of 20 to 70 m/s depending on the transpulmonary pressure $P_A - P_{PL}$, P_A being the airway pressure, P_{PL} being the pleural pressure. In the following, we shall show that severe edema occurs when v exceeds 13 m/s.

Hence trauma occurs when v/c is less than 1, i.e., when the impact is of subsonic speed. It follows that we need to consider only sonic waves in the lung, not supersonic strong shock waves. Incidentally this again justifies the selection of the initial velocity at the lung surface as the principal parameter to correlate with lung injury. The velocity is, however, only one of the many parameters that affects injury, because injury is obviously affected also by the history and spatial distribution of the impact load and the state the lung was in initially.

Clemedson and Jönsson (1964a) were, perhaps, the first to have clearly stated that the initial surface velocity of the lung is the key parameter with respect to lung injury. Jönsson (1979) has constructed a drop tower to test lung injury based on this concept. Clemedson and Jönsson (1964 a, b) have indicated that the lethal injury level lies at a velocity of around 15 m/s.

We cannot find in the literature any data on the edema formation due to impact injury other than gross statements. Hence we thought it would add to the understanding of pulmonary trauma by measuring the rate of edema formation in excised lung of the rabbit after impact. The key to such a measurement is that the lung must be so perfused that there be <u>no</u> edema before impact. This is the <u>isogravimetric condition</u> that must first be established. Once established, then the lung can be impacted and the course of the weight changed measured.

Our results show that significant pulmonary edema may result at speed of impact considerably lower than the speed of automobiles on our highways. Hence the results are relevant to the highway traffic safety problem, as well as to the blast overpressure problem

METHOD

Excised rabbit lungs were used. Rabbits weighing between 2 and 2.5 kg were anesthetized with intraveneous pentobarbital (40 mg/kg) and heparanized. A high dosage of a smooth muscle relaxing drug, papaverine, was injected intraveneously to ensure that the pulmonary vasculature was in a relaxed state. After a midline chest incision, cannulas were tied into the pulmonary artery, trachea, and left atrium. The lung was then excised and placed on a screen made of nylon stockings stretched on a double-ring frame used in embroidery. As a normal lung is a soft and compliant structure, the nylon screen provides an excellent support that eliminates stress concentrations. The specimen and the support system were then put on a weighing platform together, and the weight of the lung was recorded continuously during the experiment to assess the edema development. All connections to the lung were made of soft penrose tubing.

Fig. 1

Figure 1 shows a schematic diagram of our experiment. The lung was perfused at a constant pulmonary arterial pressure from an open reservoir. The left atrial pressure was controlled by a Starling resistor whereas the airway pressure was controlled via an air source and monitored by a water manometer. In each experiment, the lung was perfused with Macrodex (6% dextran in normal saline) with the pulmonary venous pressure fixed at 3 cm H_2O and the airway pressure held at 10 cm H_2O. A constant pulmonary arterial pressure was chosen so that an isogravimetric condition of the lung could be established. When the lung preparation was isogravimetric, a shock tube or an impact pellet gun was activated with a chosen reservoir air pressure and a certain distance between the shock tube or pellet gun and the lung surface. With a touch of the control

key, the shock tube or the pellet was fired. An impact load was then imposed on the lung surface.

Two different kinds of boundary conditions were examined. One is referred to as "freely supported" when the nylon net was hung free. The other is referred to as "rigidly supported" when a metal plate was placed underneath the nylon screen. The rigid support was intended to simulate the condition in which a lung comes in contact with the heart or the ribs. Drastic differences were observed in the lung injury under different boundary conditions.

Figs.
2,3,4   Figures 2, 3, and 4 show the design of the shock tube, the pellet gun, and the weighing platform.

The shock tube used compressed air supplied by bottles. The pressure in the accumulator was in the range of 30 to 40 psi. A paper diaphragm was ruptured by a knife activated by an elastic signal to produce a weak supersonic shock. The nozzle diameter was 2.5 cm. The shock wave, however, was followed by a bulk flow resulting in a trianglular pulse with an approximate duration of 340 μsec. Because of this complex waveform the shock tube was used only for the injury experiment, and not for the wave speed measurement (for which we used a water jet, see Yen et al.(1985). The dynamic pressure of the shock wave was measured by a pressure transducer located at a position where the wave would hit the surface of the lung. The peak pressure was used to

Figs.
& 6   correlate with edema formation in Figs. 5 and 6.

The pellet gun was designed to propel a lightweight pellet of 1.5 cm diameter at controlled velocities. The pellets were made of neoprene plastic and each weighed 1.48 gm.   They had skirts which stabilized them in the internal and external trajectories. They were commercially available at $ 2.6 per 100 pellets. Velocity of the pellet was measured by two photosensors located in the gun barrel.

The weighing platform used force transducers to measure the weight change of the lung. The steady weight was counter balanced by adjustable weights and springs. It had a sensitivity of 60 gm,   and was calibrated by dead weight.

RESULTS

Figure 5 shows the results of edema in freely supported lungs after an air shock from a shock tube. Figure 6 shows the results of edema caused by the reflection of the compressive wave in the lung supported on a rigid metal plate which was placed beneath the pleura surface of the lung. The ordinate shows the gain in lung weight after 30 minutes or 1 hour after the arrival of the shock wave, expressed in percent of the initial lung weight measured at the isogravimetric condition. The abscissa is the intensity of the blast wave pressure when the wave arrived at the surface of the lung. The solid circles show the data taken at 30 minutes after the shock. The open circles show the data of weight gain one hour after the shock. These data show that the lung can survive a shock with little edema if the peak dynamic pressure of the shock wave was less than 2 psi. Above 2 psi, the rate of formation of

edema increases with increasing level of blast pressure. The injury to a freely-supported lung caused edema of less than 20% of the initial lung weight in half an hour after impact for blasts of intensity up to 2.4 psi. Reflection of the compression wave in the lung on a rigid plate, however, induced a much higher degree of tissue damage even at such a low blast intensity level. Figure 6 shows that the edema levels are increased if the lung surface was in contact with a rigid wall, as compared with that with a free wall at any given impact load intensity. At a shock level of 2.4 psi at half an hour the edema will be about 40% of initial lung weight if reflection from solid surface took place. Edema became more pronounced one hour after the shock: the edema weight gains of the lungs were of the order of 35% and 75% for "free" and "rigid" boundaries, respectively, when the peak shock wave pressure was 2.4 psi.

Fig. 7

Test results using the light-weight impacting pellets are shown in Figure 7. The lungs were freely supported. Here it is seen that there was no edema if the impacting pellet velocity was less than 11 m/s. But edema reached 20% of initial lung weight one hour after impact when the impacting pellet velocity was 11.5 m/s. At an impact velocity of 13.5 m/s and above the edema became massive, reaching 5 or more times the initial lung weight in one hour.

DISCUSSION

When the isogravimetric condition is established, that is, when
the arterial and venous pressures are so adjusted that the total lung weight
remained constant with respect to time, the static and osomotic pressures of
the blood in the capillary blood vessels of the lung are such that the net
fluid movement from the capillary blood vessels into the interstitial space
of the lung tissue is drained away by the lymphatic system at exactly the
same rate. When the lung is subjected to an impact load, the stress induced
in the tissue may cause changes in the permeability of the blood-tissue
barrier, and interstitial edema may occur if more fluid moves into the
interstitial space and the lymph drainage cannot keep up with the fluid transfer.
If the injury were more severe so that the epithelial barrier between the
intersititum of the lung tissue and the alveolar gas were damaged, then alveolar
edema due to the movement of fluid into the alveoli will occur. The interstitial
edema will cause only a small rate of increase of lung weight with time because
the total interstitial space is small. The alveolar edema may cause a
substantial increase in the lung weight with time if the injury is severe,
because the alveolar space account for more than 70% of the volume of the lung.
Thus the evaluation of the lung weight increase from an initially isogravimetric
condition would provide a quantitative measure of the lung injury due to blast
wave.

In a desire to better understand the relationship between edema formation
and impact load, we tested isolated excised lung of the rabbit rather than
subject the whole animal to impact and then evaluate the injury by dissection.

The advantage of the excised-lung approach is the better defined measurements and simpler interpretation. The disadvantage is that the effects of many other factors are omitted from consideration, factors such as the chest wall, muscles and bones, the diaphragm, the heart, spine, and abdomen, and the posture of the subject, clothing, restraints (e.g. seat belt), as well as the nature of the shock — whether it is single pulse or multiple pulses, supersonic or subsonic, and the values of the overpressure and the pulse history (whether it is an explosion or a decompression, or an impact due to collision with some hard or soft object). The complexity of the general subject of safety of man in his environment is evident, and our contribution provides only a clue to the pulmonary trauma. Unlike penetration wounds or rupture of the heart or blood vessels, or concussion of the brain, which are evident immediately after impact, edema of the lung can develop only if the blood continues to circulate and if sufficient time is available for the fluid to be transferred from the blood to the alveolar space. Hence we suspect that lung injury is under reported in those accidents in which blood circulation was interrupted too soon.

Our experiments were motivated by the assumption that lung injury is caused by stress waves. Because of the refraction and reflection of the stress waves in the lung, injury does not have to occur at the place of the external impact. This concept is supported by our experimental results. Different kinds of stress waves exist in the lung in response to a disturbance of the lung surface. It will be interesting to find out which of these wave types is responsible for lung injury. Our results show that the reflected

waves are important because edema was seen to be more severe when the support of the lung surface was changed from a fine nylon screen to a rigid plate. Since an incident compression wave can be reflected as a combination of a shear wave and another compressive wave, and can also generate surface waves, the strong influence of the rigid support yields an important clue to the mechanism of injury which must be investigated in the future.

Aside from edema, hemorrhage may occur in severe impact injury of the lung. Previous photographs of the lungs of the sheep and rabbit exposed to blast loading have revealed "rib markings" which was suspected to be microvascular hemorrhage (Clemedson and Jönsson, 1964 b, Firscoli and Cassen, 1950). We have found that this interpretation is incorrect. The markings are indications of collapsed alveoli, of atelectasis, and are not marks of hemorrhage. Hence the evaluation of hemorrhagic injury should be based on more positive evidences of hemorrhage than the rib markings.

REFERENCES

1. Bowen, I.G., Holladay, A., Fletcher, E.R., Richmond, D.R., and White, C.S. (1965). A fluid-mechanical model of the thoraco-abdominal system with applications to blast biology. Report No. DASA-1675, Lovelace Foundation for Medical Education and Research, Albuquerque, N.M.

2. Clemedson, C.-J. (1956) Blast injury. Physiol. Rev. 36: 336-354.

3. Clemedson, C.-J. and Jönsson, A. (1964a) Dynamic response of chest wall and lung injuries in rabbits exposed to air shock waves of short duration. Acta Physiol. Scand. Vol. 62, Supp. 233, 31pp.

4. Clemedson, C.J. and Jönsson, A. (1964b). Differences in displacement or ribs and costal interspaces in rabbits exposed to air-shock waves. Amer. J. Physiol. 207: 931-934.

5. Correy, E.L. (1946) Medical aspects of blast. Nav. Med. Bull. (Wash.) 46: 623-652.

6. Frisoli, A., and Cassen, B. (1950) A study of hemorrhagic rib markings produced in rats by air blast. J. Aviation Med. 21: 510-513.

7. Jönsson, A. (1979) Experimental investigations on the mechanisms of lung injury in blast and impact exposure. Linkoping Univ. Medical Dissertation No. 80, Dept. of Surgery, Linkoping Univ. Sweden.

8. White, C.S., Jones, R.K., Damon, E.G., Fletcher, E.R., and Richmond, D.R. (1971) The biodynamics of airblast. Report No. DNA 2738T, Lovelace Foundation for Medical Education and Research, Albuquerque, N.M.

9. Yen, M.R.T., Fung, Y.C., and Ho, H.H. (1985a) Speed of stress wave propagation in the lung. AMES/Bioengineering, Report No. 85-1, University of California, San Diego.

Figure 1   A schematic drawing of the experimental set-up for
measuring the edema of the lung subjected to a blast
loading produced by a shock tube. The shock tube is
replaced by an impacting pellet gun when the lung
is tested for injury by a pellet of given velocity.

Figure 2   The construction of the shock tube and a typical
dynamic pressure trace.

Figure 3   The impact pellet gun and the pellet.

Figure 4   The construction of the weighing platform.

Figure 5   Edema produced by air blast from the shock tube when
a rabbit lung was freely supported.

Figure 6   Edema produced by air blast when the rabbit lung was
rigidly supported on a metal plate underneath the nylon net.

Figure 7   Edema produced by the impacting pellet in a freely
supported rabbit lung.

Figure 1

Figure 2

Figure 3

18

Figure 4

FREELY SUPPORTED RABBIT LUNG

○ 1 hr.
● 0.5 hr.

WEIGHT GAIN %

SHOCK WAVE PEAK DYNAMIC PRESSURE psi

Figure 5

RIGIDLY SUPPORTED RABBIT LUNG

○ 1 hr.
● 0.5 hr.

WEIGHT GAIN %

SHOCK WAVE PEAK DYNAMIC PRESSURE psi
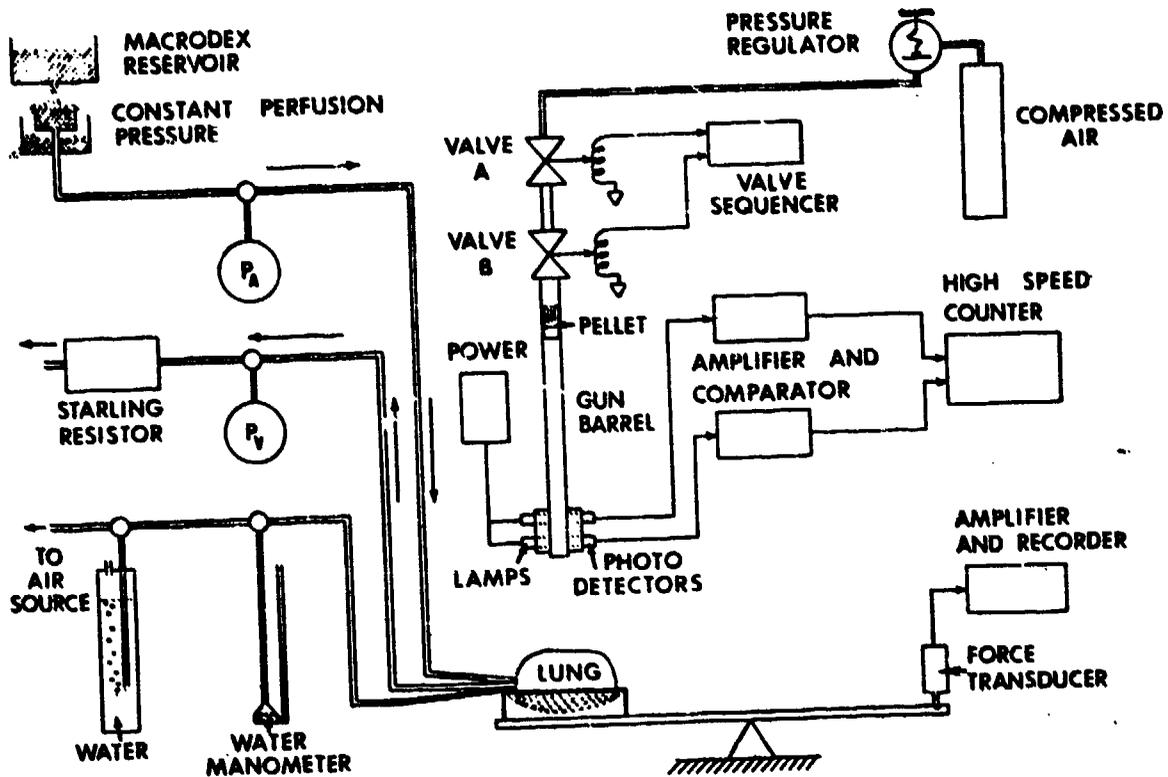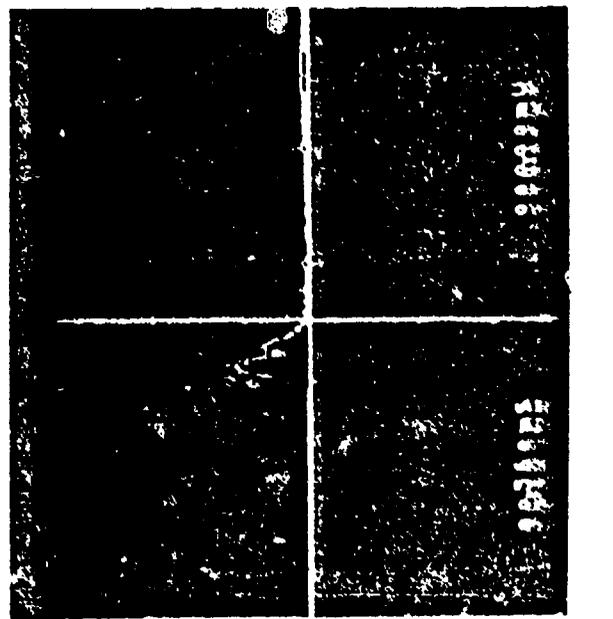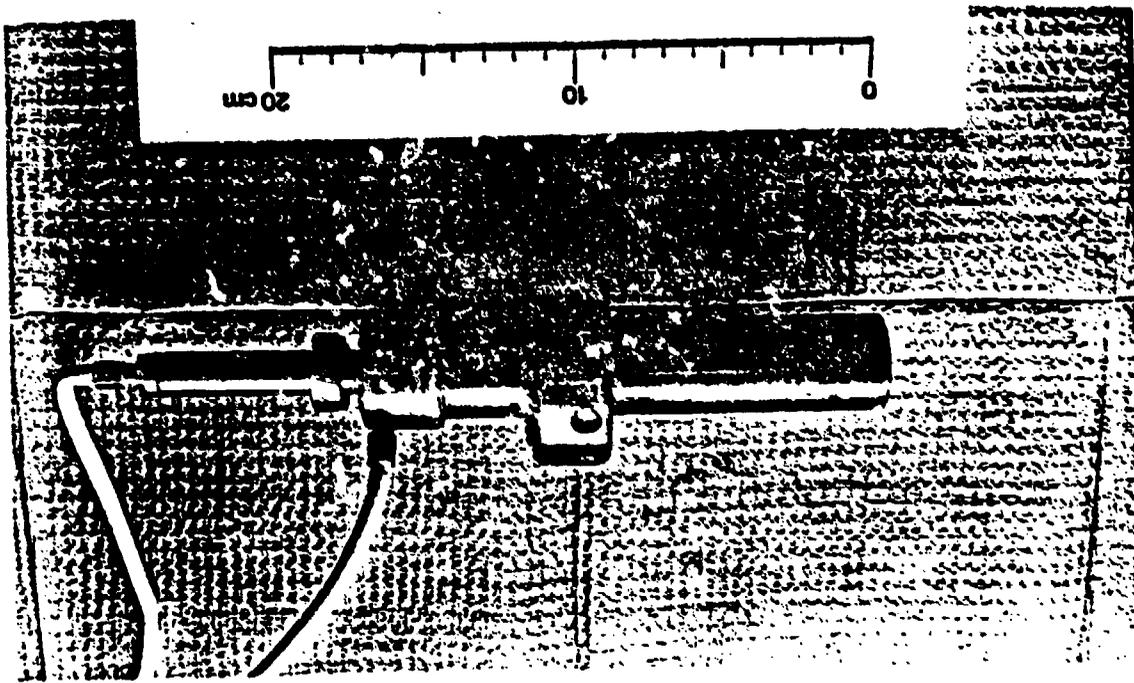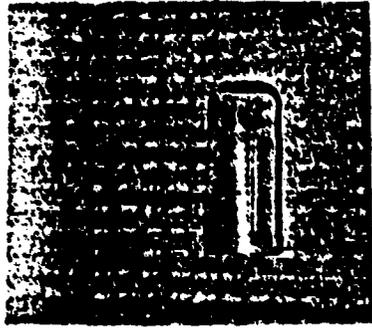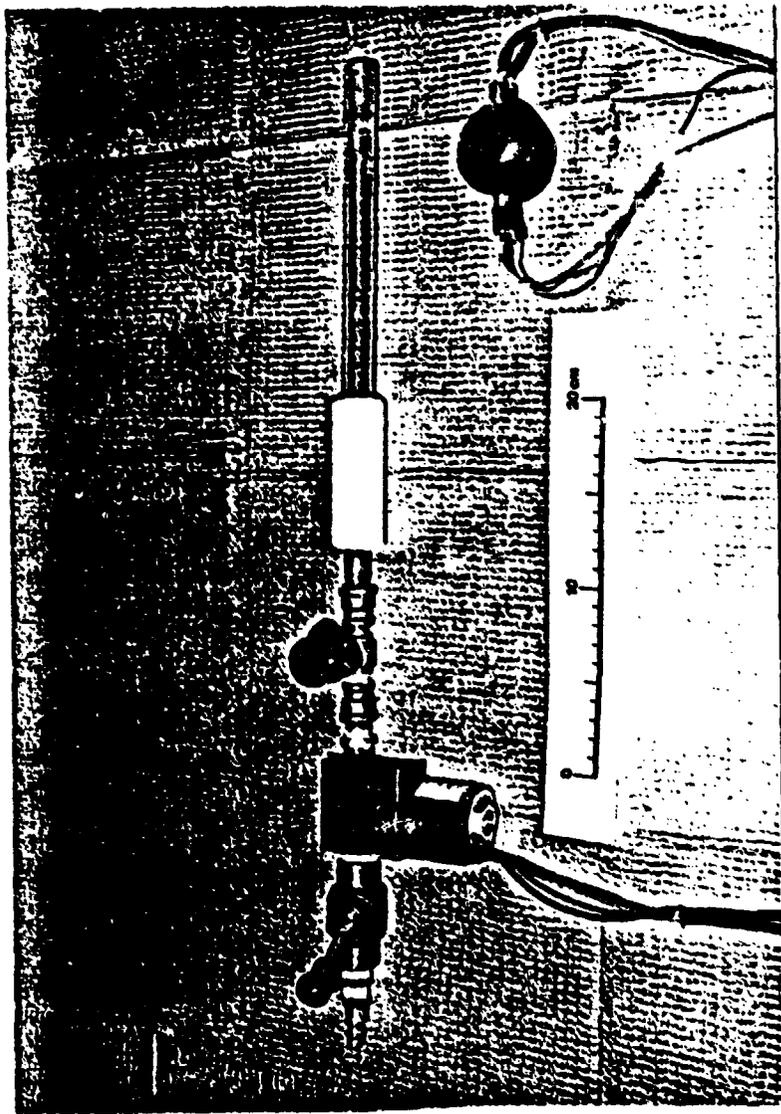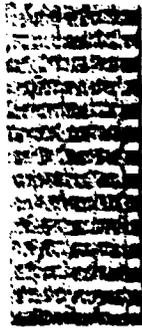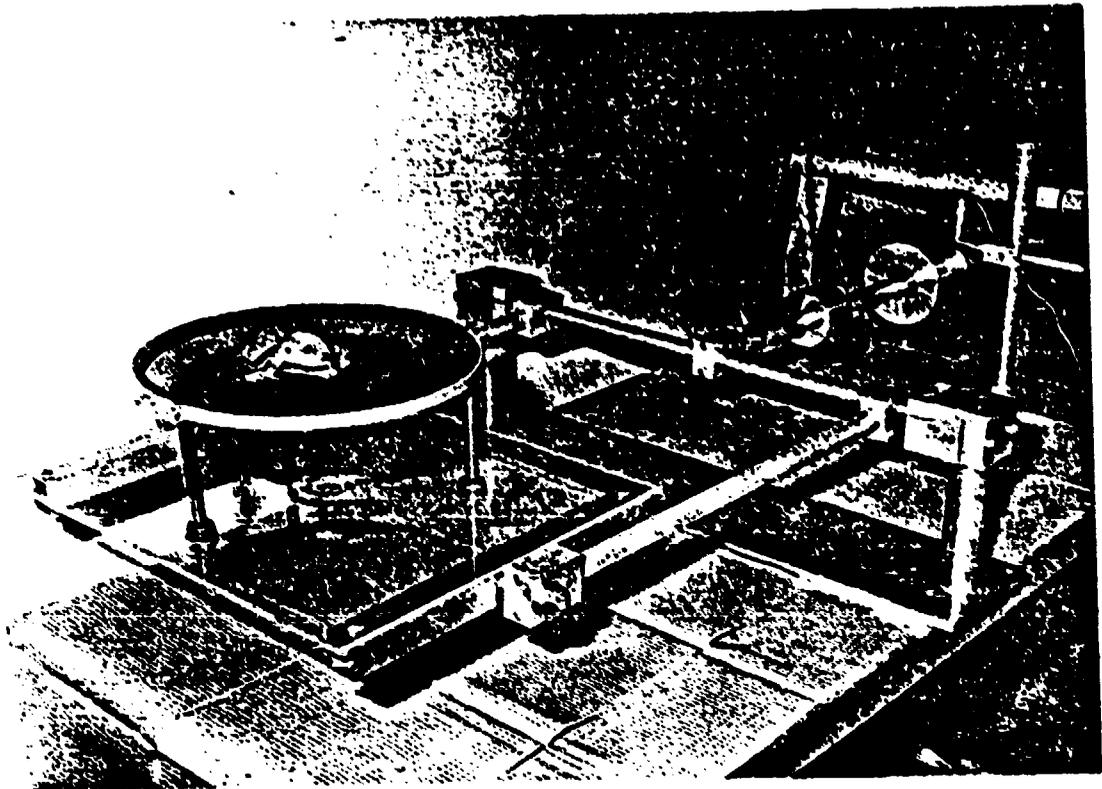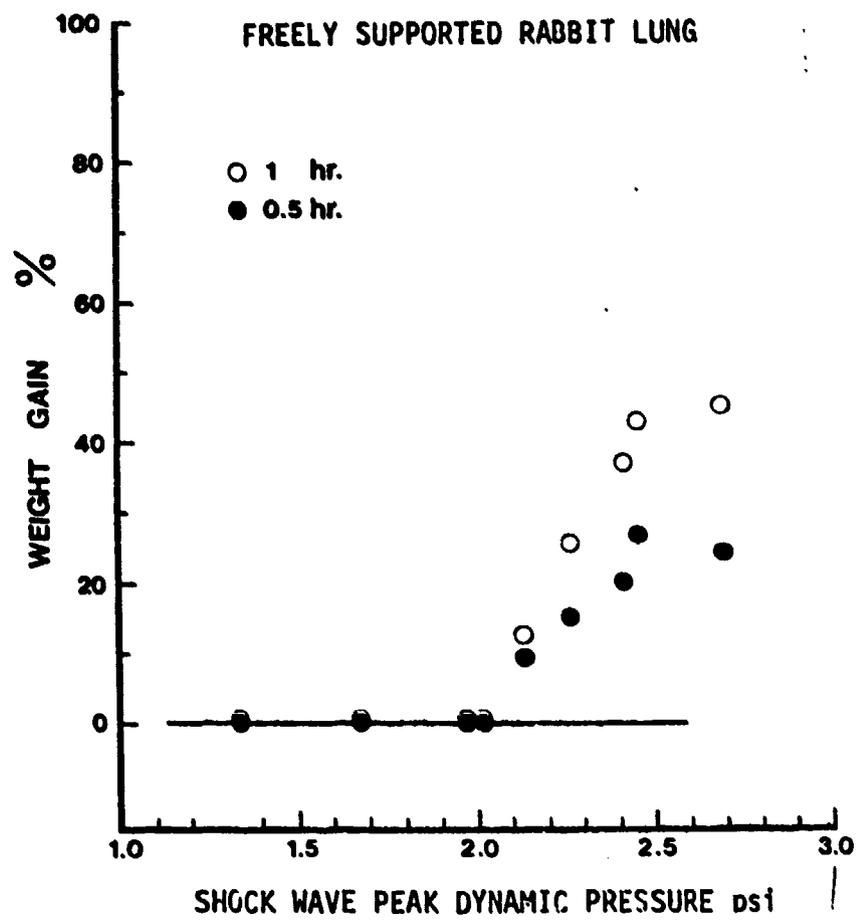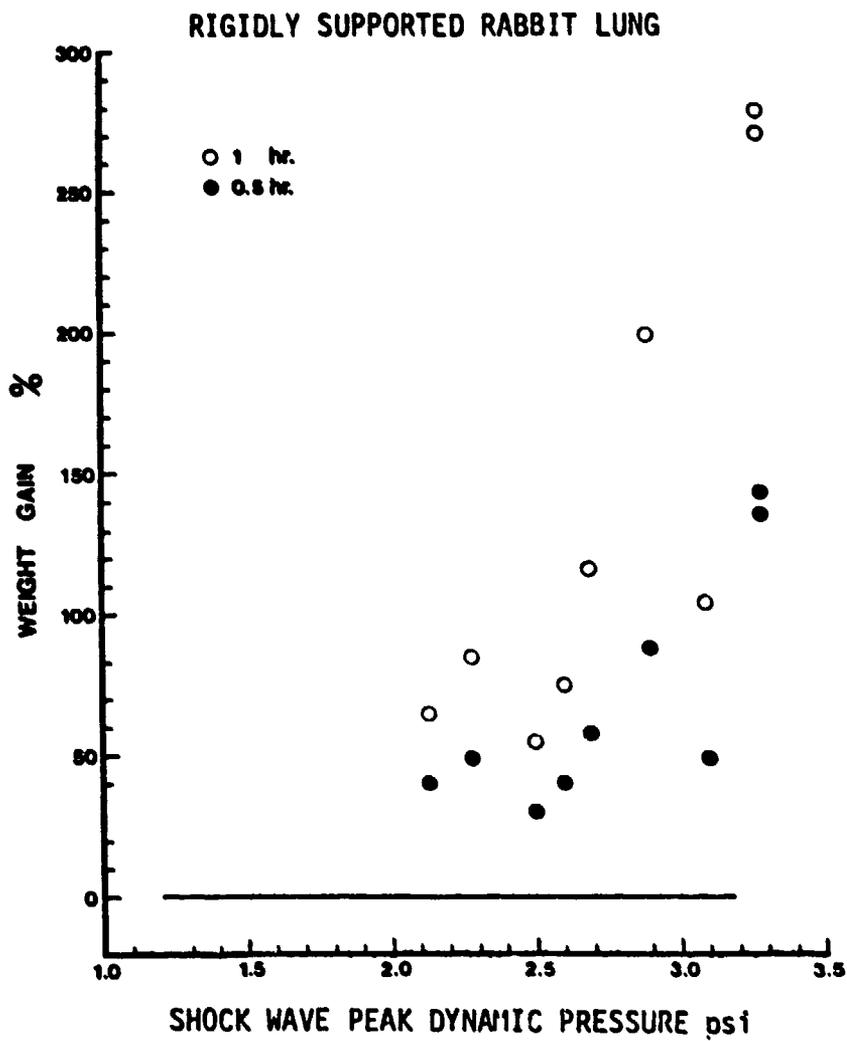
Figure 6

FREELY SUPPORTED RABBIT LUNG
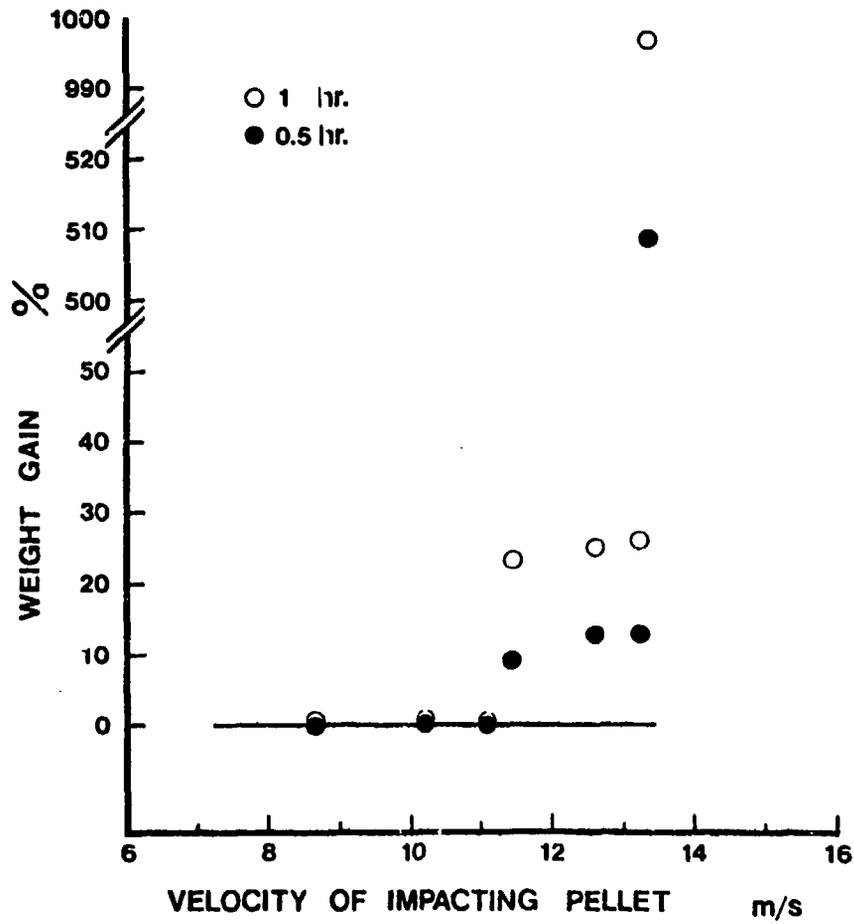
Figure 7

Brief Note

THORACIC TRAUMA STUDY:
RIB MARKINGS ON THE LUNG DUE TO IMPACT ARE
MARKS OF COLLAPSED ALVEOLI, NOT HEMORRHAGE

by

R.T. Yen and Y.C. Fung

Department of AMES/Bioengineering
University of California, San Diego
La Jolla, CA 92093, USA

When an animal is subjected to a strong pressure wave, as it occurs in a gun or bomb blast or an explosion accident, it is known that the lung injury is the most frequent cause of death, (see bibliographies listed in the references of Yen et al. (1985a). In post-mortem examination of victims of such accidents it was often noted that there were red marks on the surface of the lung which can be identified as the point of the ribs. Such markings can be seen also in simulated impact tests of isolated lung to be described below. Rib markings are seen on a rabbit lung subjected to the impact of a lightweight pellet at a velocity of 13 m/s. These simulated experiments are relevant to car accidents, since the initial velocity of impact of 13 m/s is only about half of the speed limit on our highways (55 mph).

Since the rib markings are of red color, they are often considered as a sign of hemorrhage of the delicate pulmonary capillary blood vessels (Clemedson and Jönsson, 1964. Frisoli and Cassen, 1950). Hemorrhage and edema are two most visible evidences of impact injury of the lung. We have reported our study of edema in Ref. (4). In the following we would like to show that the rib markings are in general not signs of hemorrhage. They are signs of collapsed alveoli - atelectasis.

Method

Rabbit lung was used in this study. The method of animal preparation and the instrumentation are the same as those described in Yen et al. (1985b) and will not be repeated here. The anesthetized rabbit was subjected to either a low supersonic wave from a shock tube, or an impact pellet propelled to a

specified velocity by a compressed air cylinder. After shock a midline thoractomy was made to open the chest and observe the lung. Some lungs with rib markings were artificially respirated for further observation. Other animals were artificially respirated for a period of time after being shocked, and then opened up for observation.

Result

We noticed that for those lungs that acquired rib markings after impact, the color of the marking appeared darker when the lung volume was small. If the lung was reinflated to a larger volume, the color of the marking becomes lighter. Following up this lead, we found that most of the markings can be removed by a few repeated inflations to larger volumes.

Figure 1 shows the rib markings of a lung when first excised from an animal which was subjected to an shock wave of peak dynamic pressure around 40 psi was put on a respirator and breathed at 60 cycles per minute for 30 minutes. The appearance of the lung changed to the one shown in Fig. 2. The rib markings were removed by respiration.

Two rabbits of approximately the same weight were subjected to impacts of the same intensity. One was opened immediately to observe the lung and ribs markings were found. The other was put on the respirator and breathed for sixty minutes and then the chest was opened: The lung appeared normal, there was no rib markings.

A shocked lung with clear rib markings was frozen, then sectioned and examined under a microscope. It was found that the alveoli were collapsed but there was no hemorrhage.

Conclusion

The rib markings are not marks of hemorrhage. These marks are removable by rebreathing or reinflation. They are, therefore, marks of collapsed alveoli - of atelectasis.

Under severe impacts it is possible to see hemorrhage of the lung. Usually the region where the lung is in contact with the heart show the first evidence of hemorrhage. The lesson we learned about the rib marking is that in evaluating hemorrhage injury of the lung a more positive evidence than the rib markings should be used.

REFERENCES

Clemedson, C.J. and Jönsson, A. (1964) Differences in displacement of ribs and costal interspaces in rabbits exposed to air shock waves. Amer. J. Physiol. 207: 931-934.

Frisoli, A., and Cassen, B. (1950) A study of hemorrhagic rib markings produced in rats by air blast. J. Aviation Med. 21: 510-513.

Yen, M.R.T., Fung, Y.C., and Ho, H.H. (1985a) Speed of stress wave propagation in the lung. AMES/Bioengineering Report No. 85, Univ. of California, San Diego.

Yen, M.R.T., Ho, H.H., Tao, Z.L., and Fung, Y.C. (1985b) Edema of lung due to impact injury. AMES/Bioengineering Report No. 852. University of California, San Diego.

LEGENDS

Figure 1   This shows the rib markings of a lung when first

excised from a rabbit which was subjected to a

shock wave of peak dynamic pressure around 40 psi

on the chest.

Figure 2   The same lung as shown in Figure 1 was put on a

respirator and breathed at 60 cycles per minute

between inspired and expired pressure of 4 to 16 cm H_2O.

After half an hour, the appearance of the lung is shown

here. The rib markings were removed by respirator.

Figure 1
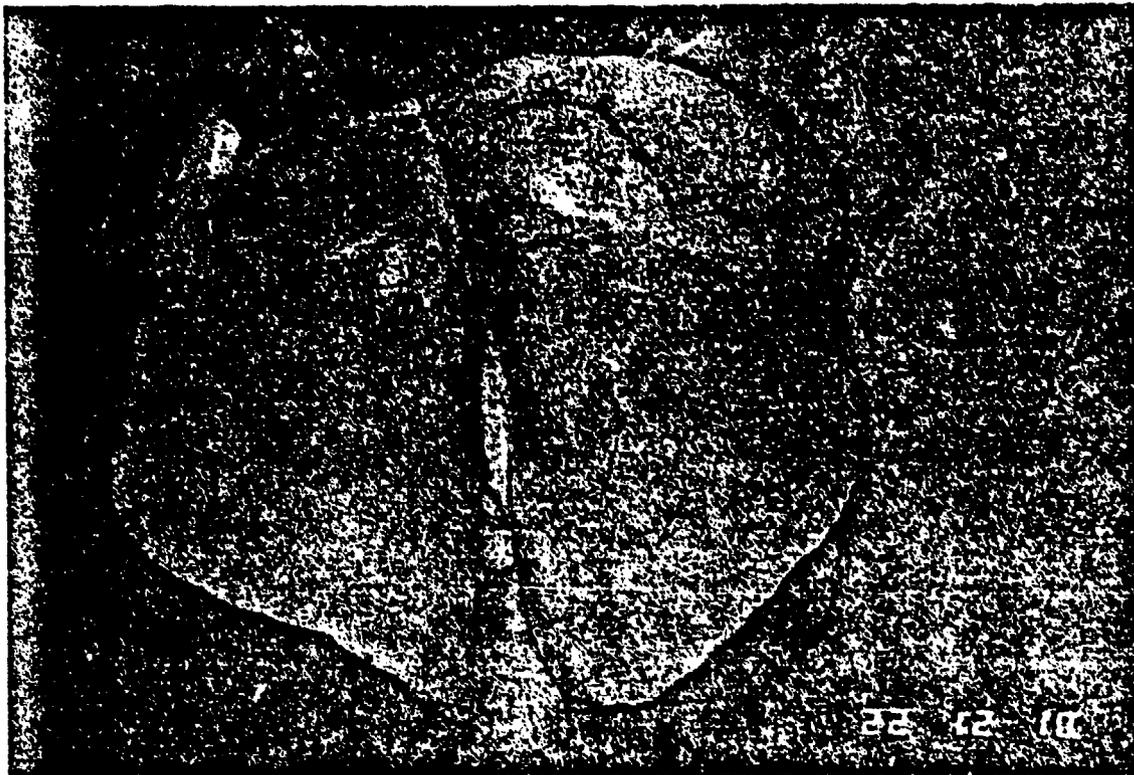
Figure 2

DISTRIBUTION LIST

12 copies
    Director
Walter Reed Army Institute of Research
Walter Reed Army Medical Center
ATTN: SGRD-UWZ-C
Washington, DC 20307-5100

1 copy
    Commander
US Army Medical Research and Development Command
ATTN: SGRD-RMI-S
Fort Detrick, Frederick, Maryland 21701-5012

12 copies
    Defense Technical Information Center (DTIC)
ATTN: DTIC-DDAC
Cameron Station
Alexandria, VA 22304-6145

1 copy
    Dean
School of Medicine
Uniformed Services University of the
  Health Sciences
4301 Jones Bridge Road
Bethesda, MD 20814-4799

1 copy
    Commandant
Academy of Health Sciences, US Army
ATTN: AHS-CDM
Fort Sam Houston, TX 78234-6100